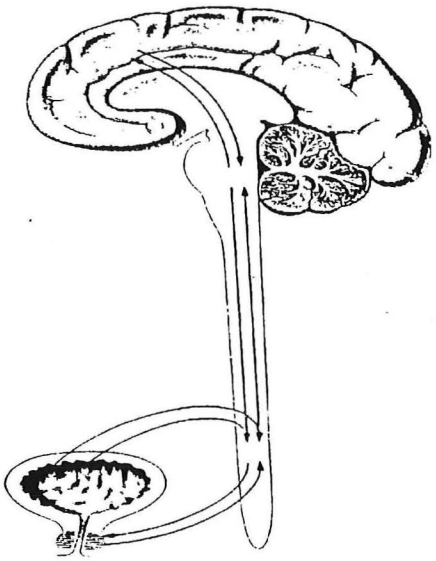
Urinary Incontinence in the Elderly



Medical Grand Rounds

University of Texas Southwestern Medical Center

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INTRODUCTION

Urinary Incontinence (UI) is defined as "a condition in which involuntary loss of urine is a social or hygienic problem and is objectively demonstrable"[1]. It is prevalent, costly, morbid, and neglected[2]. Patients may dismiss symptoms as a normal part of aging[3] and not seek help from health professionals. The presence of adult diapers lining grocery store shelves support this notion. Even when patients do confront health care providers with their problem, often little is done to evaluate the problem[4] because health professionals may share the myth that UI is a normal consequence of aging. In addition, many physicians may not be certain how to evaluate the problem and often respond by treating the symptom with diapers or catheters instead of trying to elucidate and treat the underlying cause as[5] one would do with other clinical problems. Until the last 10 years very little research has been done on UI in this country. In 1980 the first N.I.H. conference on this topic was convened. This topic has been considered "to be less than a fit subject for physicians to investigate seriously", and was often done by nurses[6]. Many of the studies that have been done on UI have commonly involved few or no elderly patients and/or omit important information such as associated illnesses, medication usage, functional descriptions, psychiatric profiles, or lack of adequate controls[7].

It has become apparent that UI when rationally approached results in cure or significant amelioration in the majority of patients, and invariably without an indwelling catheter[8],[9],[10].

Last month an NIH conference was convened in an effort to reach a consensus on the most appropriate ways to address UI.

During this Grand Rounds I have taken a broad approach to this very complex problem. I will discuss epidemiological issues, the anatomy and physiology of the lower urinary tract, the pathophysiological and functional factors leading to UI, the diagnostic approach, the treatment and management strategies available and what the future research needs related to UI are. Although UI affects all age groups my comments and approach are specifically directed to the elderly patient.

PREVALENCE

The prevalence of UI in the elderly depends on a variety of factors. Rates for community dwelling[11],[12] hospitalized[13],[14] and nursing home residents[15],[16] differ (see Table 1)[17].

Table 1.

CATEGORIES OF SURVEY SETTING AND THE RANGE OF PREVALENCE OF URINARY INCONTINENCE

STUDY SETTING	NO. OF STUDIES	RANGE IN PREVALENCE (%)
COMMUNITY	3	14-51
ACUTE-CARE INSTITUTION	3	15-34
LONG-TERM CARE INSTITUTION	6	38-55

The number of people who have experienced any incontinent episode in a single year may be quite different from the number who have experienced UI causing impairment in their lifestyle. Since UI is an embarrassing symptom it may be under reported in surveys. The frequency of episodes is not particularly useful since one patient may be incontinent daily and not be affected socially as compared to the patient who has a single episode of UI and is no longer able to leave their apartment for fear of having an episode in public with humiliating consequences.

When one considers studies that define severe UI in community dwellers, the range is between 3 and 10 percent[18]. This is useful since it may give us a better idea of the number of patients in this population who truly need evaluation and treatment.

MORBIDITY

The clinical, social, psychological and economic impact of UI can be significant. Clinically, UI has been associated with skin rash, pressure sores, cellulitis, urinary tract infection and urosepsis, and falls with their resultant trauma (e.g. hip fractures).

Socially, the consequence of UI are substantial. These include reduced daily activities including shopping, physical recreation, entertainment, travel, and volunteer work. Social interactions are impaired such as participating in social activities outside the home and sexual relations[19].

Psychologically, patients can develop a poor self image, become withdrawn and depressed. The strain on caregivers is also significant and it is incontinence that is often cited as the reason for placing a family member in a nursing home[20].

The economic burden is also considerable. Hu[21,22] has calculated that $10.3 billion (using 1987 prices) is spent annually in direct care costs for urinary incontinence (see Table 2.). The prevalence figures used include nine percent of elderly community dwellers, 50 percent

50 percent of nursing home residents and 2 percent of females between the ages of 25 and 64 have incontinence.

Table 2.

TOTAL DIRECT CARE COSTS FOR UI	
ELDERLY NURSING HOME PERSONS	$3.3
ELDERLY IN THE COMMUNITY	$4.8
WOMEN BETWEEN 25 AND 64 YEARS	$2.2
TOTAL	$10.3 BILLION

FROM HU

Of the $10.3 billion cost figure he further divides this into $3.3 billion for elderly persons in nursing homes, $4.8 billion for elderly persons in the community and $2.2 billion for adult females between the ages of 25 and 64 years. The routine care costs are the largest, estimated at $6.6 billion. Of note the routine care cost under private insurance, and medicare or medicaid or not reimbursable. This reimbursement scheme provides more economic incentives for nursing homes to catheterize their incontinent residents than to give them disposable or reusable absorbent briefs.

Table 3.

Costs of Urinary Incontinence

- *Direct Costs*
  - Diagnostic and evaluation costs
    - Physician consultation and examination
    - Laboratory
    - Diagnostic procedures
  - Treatment costs
    - Surgery
    - Drugs
  - Routine care costs
    - Nursing labor
    - Supplies
    - Laundry
  - Rehabilitation costs
    - Nursing labor
    - Supplies
  - Incontinence consequence costs
    - Skin breakdowns
    - Urinary tract infection
    - Falls
    - Additional nursing home admissions
    - Longer hospital stays
- *Indirect Costs*
  - Time costs of unpaid caregivers for treating and caring for incontinent elderly persons
  - Loss of productivity because of morbidity
  - Loss of productivity because of mortality

ANATOMY AND PHYSIOLOGY

The basic role of the bladder and the urethra is the storage and emptying of urine at suitable times. We need to understand the normal anatomic and physiologic means by which this function is carried out in order to understand what can go wrong and then how to evaluate and treat the underlying cause of urinary incontinence.

The way in which the bladder fills, stores, and empties urine is complex and many of the details of the process are still poorly understood, with significant disagreement among authorities on the topic[23]. The following represents an up to date overview of the physiology of lower urinary function but the reader is referred to the references for a more detailed discussion[24],[25],[26]. The functional units of the bladder can be divided into the detrusor (or bladder body) and bladder base. The detrusor is made up of smooth muscle arranged in an interlacing meshwork of muscle fibers such that when the detrusor contracts it does so in all dimensions. The bladder base can be subdivided into trigone and bladder neck. The trigone is a triangular area located on the posterior wall of the bladder. The ureterovesical junctions are the superiolateral borders and inferiorly is the interior urethral meatus. The trigone is divided into the superficial and deep trigone. This is a somewhat confusing description in that the muscle cells of the "deep" trigone are indistinguishable from the detrusor. The superficial trigone is a thin layer of smooth muscle that is a morphologically distinct component of the trigone and is continuous proximally with those of the ureters. Inferiorly it becomes continuous with the proximal urethra in both sexes.

Anatomically the bladder neck in the male and female are different. In the male there is a circularly arranged layer of smooth muscle that forms a collar which appears to have a genital function by contracting during ejaculation and thereby preventing retrograde ejaculation. It is not clear if the proximal sphincter has an active role in maintaining continence. Distally the bladder neck muscle becomes indistinguishable from the stroma and capsule of the prostate. In the female, the smooth muscle bundles in this area are arranged longitudinally and are contiguous with the urethra. There is no anatomic proximal sphincter in the female and it is unlikely that it plays any significant part in maintaining continence in the female. The urethra functions as a conduit for the passage of urine during micturition. During continence the urethral walls are in apposition. There are a number of differences in the male and female urethra. The urethra in the male and female differ in length. The female urethra is about 4 cm in length and the male urethra 20 cm.

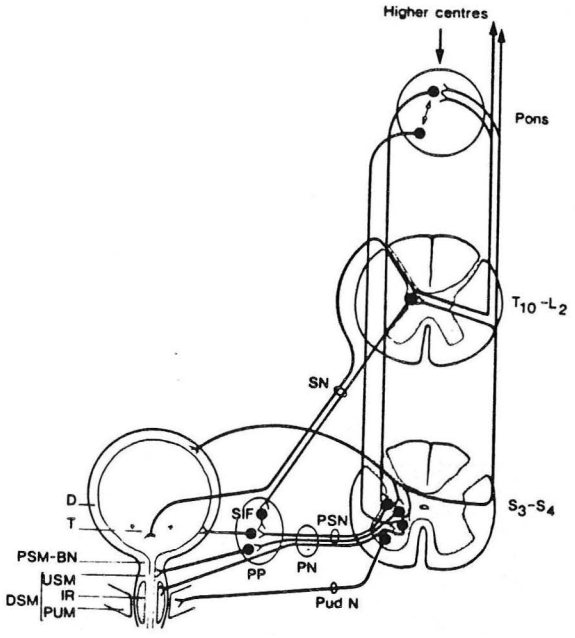
There is a superfical layer of smooth muscle fibers in both the female and male urethra that probably does not play a role in maintaining continence. But most importantly, there is a well defined area in the midportion of the urethra in women and in the area of the membranous urethra in men that is composed of circularly arranged striated muscle composed of slow twitch fibers that make up the rhabdosphincter or external sphincter. It is this structure that is believed to be the "true" external sphincter and responsible for maintaining continence between normal micturition. This sphincter has often been omitted in anatomy textbooks where the only sphincter described is the skeletal muscle of the urogenital diaphragm. This muscle is separated from the urethral wall by a connective

tissue plane. Unlike the rhabdosphincter, this periurethral muscle possesses morphologic features (slow and fast twitch fibers) similar to other "typical" voluntary muscles. This extramural sphincter does play a role in maintaining continence during brief sudden rises in intra-abdominal pressure such as coughing and sneezing.

The considerable collagen and elastic tissue present around the bladder neck also contributes to the passive closure of the proximal urethra[27]. Lastly, an important consideration is the position of the bladder neck and proximal urethra (and rhabdosphincter) above the pelvic floor which is effectively part of the abdominal cavity. This ensures pressure transmission to the proximal urethra during increases in intraabdominal pressures and ultimately the most important physiologic consideration in maintaining continence. That is, urethral pressure must exceed vesicle pressure.

The neurologic control of micturition can be divided into local innervation of the lower urinary tract and the modulating effect of the central nervous system. The lower urinary tract is innervated predominantly from the autonomic nervous system. Most innervation actually emanates from peripheral ganglia, which are a short distance from, adjacent to, or within the organs they innervate[26]. Parasympathetic efferent supply originates from the sacral spinal cord segments S2-S4.

Figure 1.



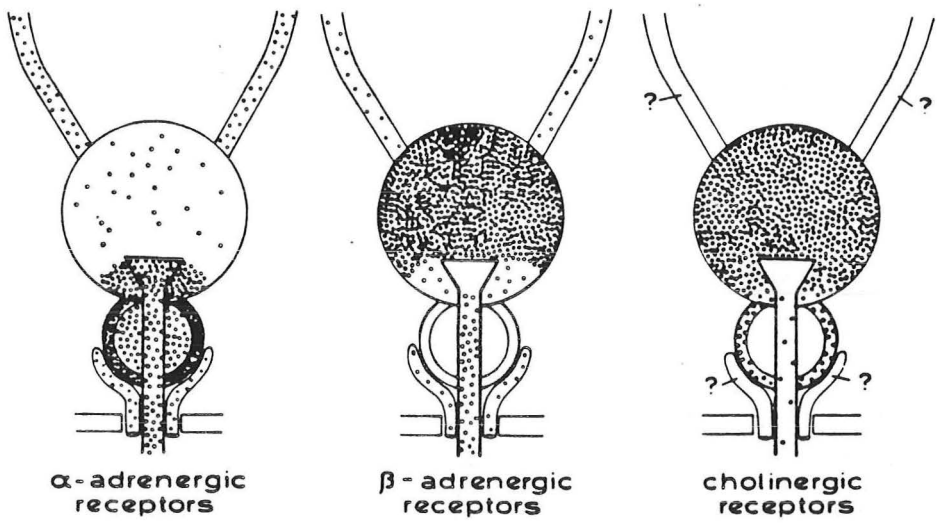
A concept of the innervation of the bladder and sphincter-active urethra. Key: D, detrusor; T, trigone; PSM, proximal sphincter mechanism; BN, bladder neck; DSM, distal sphincter mechanism; USM, urethral smooth muscle; IR, intrinsic rhabdosphincter; PUM, periurethral musculature; SN, sympathetic nerves; PSN, parasympathetic nerves; PN, pelvic nerves; PP, pelvic plexus; SIF, small intensely fluorescent cell; PudN, pudendal nerve.

This preganglionic supply is conveyed via the pelvic nerve. The sympathetic nervous system input originates from the thoracolumbar spinal cord segments T10-L2 and travel via the hypogastric nerves. Bilaterally, at a variable distance from the bladder and urethra the hypogastric and pelvic nerves branch to form the pelvic plexus. Divergent branches of the plexus innervate the pelvic organs. Somatic input is via the pudendal nerve comprised of nerve roots from the sacral cord S2-S4 and innervates the skeletal muscle of the urogenital diaphragm and is also the origin of sensory input from the anal and saddle region of the body.

Sensory input during bladder filling is poorly understood but is felt to be carried via afferents in the pelvic, hypogastric, and pudendal nerves[29].

The detrusor has abundant cholinergic (muscarinic) innervation and the parasympathetic fibers from the pelvic nerve are the principle motor supply to the detrusor. Stimulation of this nerve results in detrusor contraction. The role of the parasympathetic nervous system in the bladder base and urethra is uncertain. Although cholinergic receptors are present here, their density is less than in the body of the detrusor. An area of major controversy is the innervation of the rhabdosphincter. This was once thought to have somatomotor innervation (S2-S4 pudendal nerve) but many feel it has a unique[29] somatomotor plus diautonomic innervation, particularly with pelvic nerve input. The detrusor and bladder base also have adrenoceptors present. There is a density of alpha adrenergic receptors is the bladder neck and proximal urethra with sparse distribution in the bladder body itself. B-adrenergic receptors are present in the bladder body with sparse distribution in the bladder base and urethra.

Figure 2.



Distribution of autonomic receptors in the human urinary tract. The relative densities are roughly indicated. α receptors are α 1 in type; β receptors are thought to be β 2. (Caine 1984)

In spite of this there is disagreement as to the presence of postganglionic sympathetic innervation in the bladder and proximal urethra and if they have any active role in maintaining continence.

In addition to the classic transmitters (i.e. acetylcholine, norepinephrine) a variety of other substances appear to play a role in the innervation of the lower urinary tract. Some have speculated that noncholinergic\nonadrenergic innervation such as purinergic or peptidergic nerves may exist. Although immunohistochemical techniques have revealed the presence of neuropeptide-immuno reactive nerves in the lower urinary tract there is no convincing evidence to suggest either a "purinergic" or "peptidergic" nerve exist as a separate structural entity in the innervation of the lower urinary tract. What seems most likely is that multiple compounds may be released from the same nerve ending once stimulated.

Table 4.

Proven coexistence of neurotransmitters and neuromodulators (adapted from Hökfelt et al. 1986)

"Classical" transmitter	Coexisting petide
Acetylcholine	Enkephalin
	Substance P
	Vasoactive intestinal polypeptide (VIP)
	Calcitonin gene-related peptide (CGRP)
	Galanin
Noradrenaline	Enkephalin
	Neuropeptide Y
	Vasopressin
Serotonin	Substance P
	Thyrotrophin-releasing hormone (TRH)
	Cholecystokinin (CCK)
	Enkephalin

It is the brain stem, more specifically the rostral pons that is the center at which the coordination and integration of bladder and sphincter activity occur in the adult. Lesions above this level lead to coordinated incontinence,lesions below this lead to uncoordinated detrusor sphincter dyssynergia.

This pontine micturition center receives information from a number of higher cortical areas including the frontal lobes, (which have predominantly inhibitory input) basal ganglia, cerebellum, thalamus, paracentral lobule, hypothalamus and genu of the corpus callosum.

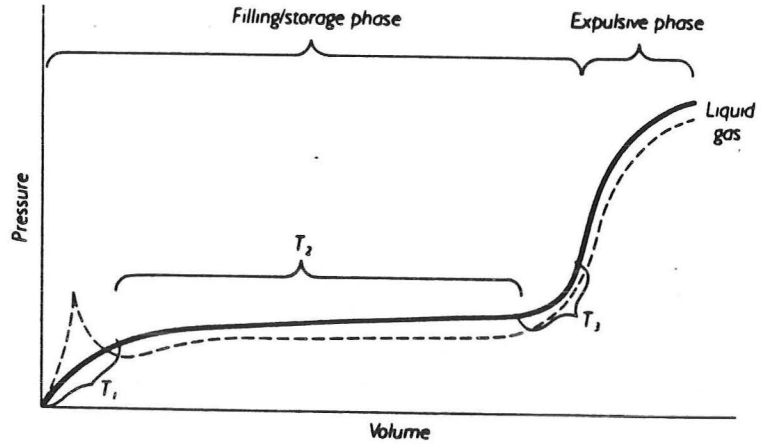
The brainstem micturition center receives input from the cord and lower urinary tract as well as the above mentioned cortical areas and delays voiding until an appropriate time (i.e. a commercial). For instance, lesions in the frontal area result in uninhibited

bladder contraction and urge incontinence or socially inappropriate or unconscious incontinence. In the spinal cord segments S2-S4 (with the major portion at S3) a sacral micturition center exists, although this "center" does not transmit a spinal detrusor reflex. It cannot fully coordinate sympathetic, parasympathetic and somatic activity related to filling and voiding.

Micturition Cycle

The bladder functions as a low pressure reservoir. It fills at around 2cc per minute and with no appreciable increase in pressure until a volume of about 400cc is attained. Initially, this compliance is accomplished by the fibro-elastic (viscoelasticity) properties of the bladder. Conditions that increase collagen content in the bladder such as chronic infection, radiotherapy, and hypertrophy result in decreased compliance[30]. As volume further increases the pressure remains low due to the active response of mechanoreceptors. This accommodation may be mediated through B adrenergic activity. As the bladder fills, the bladder neck and urethra must remain closed. Continence is maintained as long as maximal urethral pressure exceeds the intravesical pressure. As volume continues to increase further there is finally a steady increase in intravesical pressure.

Figure 3.



Idealized cystometrogram. During bladder filling there is an initial rise in pressure (T_1) that is due to the resting intravesical pressure and the viscoelastic response of the bladder to stretch. As the bladder is filled, it accommodates to increasing volume by increasing its wall tension and the intravesical pressure remains fairly constant (T_2). The third segment (T_3) is a sudden rise in intravesical pressure due to stretching of the bladder to its physiologic limit. (From Blaivas, J. G.: Urodynamic testing. In Raz, S. [ed.], *Female Urology*. Saunders, Philadelphia, 1983.)

Sensory stimuli steadily increase and at about 300 cc the first desire to void occurs. The intensity of this desire progressively increases as bladder distention increases along with more frequent and stronger sensory input via the spinal cord to the brainstem and cortical areas of the brain. At this "first desire to void volume" motor impulses to induce the detrusor to contract are propagated but are inhibited at the brainstem micturition center. When the individual finds a place to void, the CNS role becomes facilatory by enabling the detrusor to contract by input via the brainstem micturition center to the sacral cord which stimulates the detrusor to contract via the pelvic nerve. This may be assisted by the release of an inhibiting role the sympathetic nervous system plays on the parasympathetic ganglia. At the same time the detrusor contracts the urethral pressure falls due to relaxation of the sphincter mechanism and flow of urine ensues. The coordination of detrusor contraction and sphincter relaxation is critical and is accomplished by central nervous system activity. The inhibition of detrusor contractions is essential for normal bladder filling and storage to occur. A variety of disease processes (and perhaps normal aging) are believed to result in the loss of CNS ability to inhibit detrusor contraction resulting in a sudden (urge incontinence) loss of urine that cannot be voluntarily prevented (see below).

Figure 4.

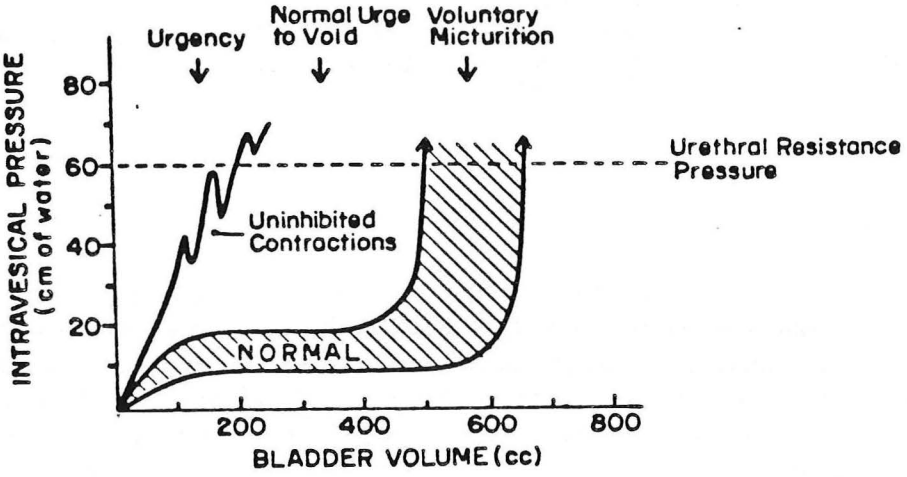
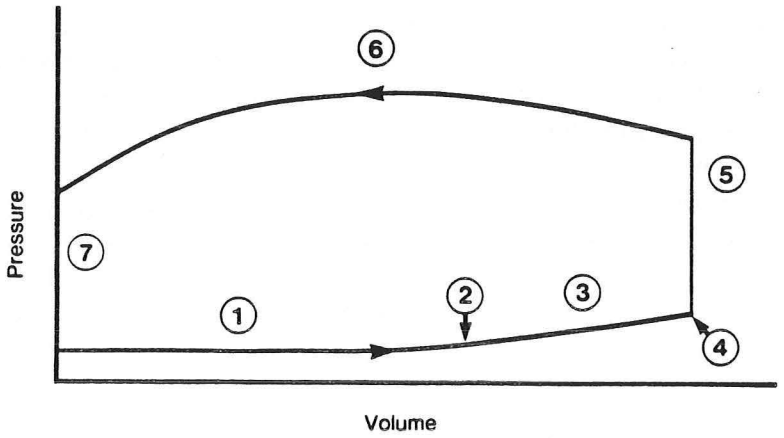


Figure 5.



Micturition cycle represented as a pressure-volume loop. Various phases, indicated by numbers are as follows:

1. Accommodation—partly because of intrinsic properties of bladder wall and partly because of unconscious nerve-mediated inhibition.
2. First conscious sensation—mediated by tension receptors, since no rise in detrusor pressure is necessary.
3. Postponement—cortical function lost in certain cerebral lesions.
4. Initiation of voiding—coordinated by pontine micturition centre.
5. Isometric detrusor contraction—phase before flow starts.
6. Sustained detrusor contraction—phase in which detrusor pressure remains relatively constant throughout voiding.
7. Relaxation phase, majority of which occurs after voiding has ended—in some cases detrusor pressure may rise as the bladder neck closes (isometric after contraction). (Torrens 1984)

The above is a very brief account of the normal micturition cycle. However by considering the above one can easily classify the ways in which incontinence can occur. There are basically four mechanisms which have there clinical correlates. (1) The bladder contracts when it should not. (2) The bladder doesn't contract when it should. (3) The outlet resistance is too low. (4) The outlet resistance is too high, and of course, any combination of the above.

Since urinary incontinence is not a normal aspect of aging
and yet the prevalence in this group of patients is so high, we must reconcile this inconsistency. First, there seems to be a number of physiologic changes in the lower urinary tract in the elderly that may predispose them to urinary incontinence. Bladder capacity, urine flow rate, and detrusor contractility may all decline with aging[10,31,32]. Some of this data is contradictory and many of the studies have major flaws in their design. Post void residual likely increases somewhat but is probably less than 50 cc. Uninhibited

bladder contractions which are virtually non-existent in healthy young individuals seem to increase in the elderly[31],[32]. Again, these studies are often flawed in design. In Andersen's series, 9 of 17 reportedly healthy subjects had uninhibited contractions but obstruction was not clearly ruled out. The latter is a well known cause of uninhibited contractions. Vaginal and urethral atrophy may occur in up to 50% of post-menopausal female[10]. The female urethra has estrogen receptors and is estrogen sensitive[33]. Because of estrogen deficiency urogenital atrophy can result in a decrease in urethral closing pressure.

Prostatic enlargement occurs in all men if they live long enough and except in the case of obstruction does not by itself result in UI. Aging appears to be associated with an increase in nocturnal excretion of urine. Kirkland[34] found that healthy elderly individuals excreted more than 50% of their daily fluid intake at night compared to 25% in a younger group. Both groups were healthy and had no medical complaints. This may explain why nocturia is such a common complaint in the elderly even in the absence of heart failure or venous insufficiency.

None of the above by themselves cause UI in the elderly. However, once faced with challenges associated with disease, drugs, functional impairment or environmental factors the marginally continent system may be overwhelmed and UI results.

CAUSES OF URINARY INCONTINENCE IN THE ELDERLY

One of the challenges in caring for the elderly patient is the multiplicity of problems that often exist in each patient. The elderly are a very heterogenous population physiologically when compared to younger age groups, and chronologic age often does not correlate with physiologic age. Community dwelling individuals have on average 3.5 important disabilities and in institutionalized individuals the average is 6[35].

The patient with UI may have multiple underlying problems, any one or combination of them potentially causing UI. For example, a man with a history of a cerebrovascular accident and osteoarthritis may be incontinent because of immobility from arthritis or uninhibited detrusor contractions from his prior CNS injury, or he may be obstructed from benign prostatic hypertrophy. We may find that he has a urinary tract infection and with treatment the UI resolves. However, uninhibited detrusor contractions documented by cystometrogram may persist. In fact uninhibited contractions may occur in as many as 11% of elderly people who have no urinary tract symptoms[36]. The important point here is that abnormalities detected during urodynamic evaluation may be neither the primary cause of the patient's incontinence nor require treatment in order to restore continence[37],[10]. Clinically it is useful to arbitrarily divide UI into transient (or acute) and established (or persistent) causes. There are a number of useful mnemonics to use as a checklist for those precipitating factors that can result in acute UI and by reversing them we can restore continence[38],[39]. I will use the one put forth by Resnick - DIAPPERS which represents delirium, symptomatic infection, atrophic vaginitis, pharmacologic, psychologic, endocrine, restricted mobility, and stool impaction.

TRANSIENT INCONTINENCE

Delirium

Acute confusional states whether from infection, drugs, or metabolic disturbances can result in incontinence. The patient may not be aware of bladder filling and probably doesn't care. The important point is not to miss the diagnosis of delirium. The underlying etiology must be established and treated. The UI will resolve with resolution of the confusional state.

Symptomatic Infection

Sensory input from the infected bladder may overwhelm the ability to inhibit bladder contractions and the individual with minor mobility problems may no longer be able to reach a toilet fast enough because of urinary urgency and frequency. Asymptomatic bacteriuria does not appear to cause incontinence[40]. However, since urinary tract infections can present with atypical symptoms and if initial evaluation in an elderly patient reveals infection it should generally be treated and the response observed.

Atrophic Vaginitis and Urethritis

As discussed previously the urethral tissue is under estrogen influence. This condition can mimic a urinary tract infection and symptoms of urge and stress incontinence may be present. The physical findings of atrophic vaginitis include a reddened, thin mucosa, punctate hemorrhages and erosions, easy friability and a scant watery discharge. A common clinical picture is the elderly woman who presents with symptoms of urinary urgency and frequency who has a totally benign appearing urine to the surprise of the examining physician who presumed the patient had a urinary tract infection. The treatment of course is estrogen therapy which will be discussed below.

Pharmacologic

Pharmacologic causes of UI are common in the elderly. Those agents that are commonly implicated include diuretics, sedative\hypnotic agents, anticholinergic agents, adrenergically active compounds and muscle relaxants. The elderly are the largest consumers of over-the-counter preparations and many of these medications may contain anticholinergic or adrenergic agents. An example would be the 80 year old asymptomatic man who goes out and takes a combination cold medicine with both anticholinergic properties which would impair detrusor contraction and an adrenergic component (pseudoephredrine or other similar decongestant) which has alpha adrenergic agonist properties which can stimulate alpha receptors in the bladder neck and prostate gland. The situation created is one of acute urinary retention. If such a patient is evaluated he would likely be found to have a large prostate (virtually all men by the time they reach 80 do) and may mistakenly undergo a TURP for presumed acute urinary retention secondary to BPH. In actuality it was caused by his medication and the treatment should be stopping

the offending agent and inserting an indwelling foley catheter until the bladder is decompressed and normal detrusor function resumes. Patients often do not mention non-prescription medications when asked what medications they take and physicians may fail to specifically ask about OTC medicines.

Table 5.

MEDICATIONS THAT CAN AFFECT CONTINENCE

TYPE OF MEDICATION	POTENTIAL EFFECTS ON CONTINENCE
DIURETICS	POLYURIA, FREQUENCY, AND URGENCY
ANTICHOLINERGICS	URINARY RETENTION, OVERFLOW INCONTINENCE, AND FECAL IMPACTION
PSYCHOTROPIC	
ANTIDEPRESSANTS	ANTICHOLINERGIC ACTIONS AND SEDATION
ANTIPSYCHOTICS	ANTICHOLINERGIC ACTIONS, SEDATION, RIGIDITY, AND IMMOBILITY
SEDATIVES/HYPONOTICS	SEDATION, DELIRIUM, AND MUSCLE RELAXATION
NARCOTIC ANALGESICS	URINARY RETENTION, FECAL IMPACTION, SEDATION, AND DELIRIUM
ALPHA ADRENERGIC BLOCKERS	URETHRAL RELAXATION
ALPHA ADRENERGIC AGONISTS	URINARY RETENTION
BETA ADRENERGIC AGONISTS	URINARY RETENTION
ALCOHOL	POLYURIA, FREQUENCY, URGENCY, SEDATION, DELIRIUM, AND IMMOBILITY

Psychological causes such as depression may result in urinary incontinence. The severely depressed patient may not care if they are incontinent and simply do not bother to use the toilet.

Endocrine causes include hyperglycemia and hypercalcemia which result in polyuria that can overwhelm the older patient's ability to get to the restroom in time.

Restricted mobility is a common cause of acute UI especially in the hospitalized patient. The patient with enforced bedrest because of a hip fracture or myocardial infarction without sufficient assistance or a bedside urinal out of reach can easily result in a humiliating episode. A person who develops a flare up of arthritis which impairs their mobility may not reach a toilet fast enough.

Stool impaction is a fairly common cause of incontinence[41]. This should especially

be considered in the bedridden or immobile patient that develops fecal oozing in addition to urinary incontinence. The mechanism is unclear and has variably been attributed to obstruction of the bladder outlet or reflex bladder contraction by rectal distention.

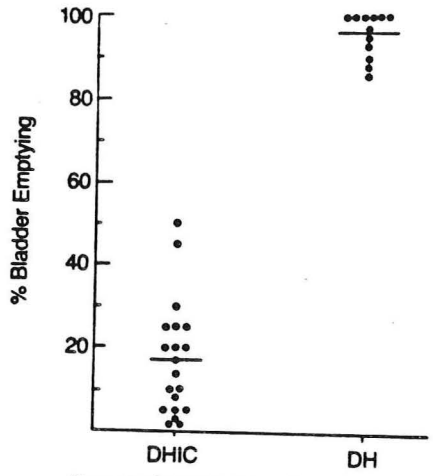
ESTABLISHED CAUSES

The established causes of incontinence can be divided into four basic subtypes, urge, overflow, stress, and functional. Urge incontinence occurs when patients sense the urge to void but are unable to postpone micturition. The most common underlying cause is uninhibited detrusor contractions (UDC). Other terms for this include detrusor instability, spastic bladder, or detrusor hyperreflexia. If the contractions are due to a neurologic abnormality the latter is the most appropriate term[1]. However, in the patient who may have had a lacunar infarction and have uninhibited contractions it may be impossible to clearly state that the contractions originate from the area of infarction or from another source.

In patients with urge incontinence secondary to uninhibited detrusor contractions urge symptoms are clearly documented during filling cystometrogram just before a detrusor contraction that cannot be voluntarily inhibited and that exceeds 15cm of H2O. UDCs in the elderly are associated with a variety of neurologic disorders such as stroke, parkinsons, spinal inhibitory pathways interfered with by spondylosis or metastasis. The clinical picture is of a patient complaining of the sudden onset of urgency with inadequate time to get to a toilet. Since the bladder usually contracts fully once the UDC occurs the contents of the bladder are emptied and a moderate amount of urine is loss. Generally the patient remains dry between incontinent episodes, that is there is no history of continuous leaking. Postural changes may precipitate incontinence and the patient typically also has nocturia. The post void residual is predictably small.

Recently a new cause of incontinence in the elderly has been described[42]. Detrusor hyperactivity with impaired contractile function (DHIC) seems to be a distinct physiological subset of detrusor instability. In a study of urinary incontinence in institutionalized frail elderly patients (mean age 89) Resnick found this condition present in 33% of the patients evaluated. Those with DHIC did not differ clinically from those with detrusor hyperreflexia (UDC). In these patients there is impaired detrusor contractility associated with bladder trabeculation, slow velocity of bladder contraction, and a significant residual urine because of ineffective emptying. One possible explanation of these findings is that DHIC represents the advanced stage of the natural history of detrusor instability (UDC). If these findings can be confirmed it would have important therapeutic implications.

Figure 6.



—Percent of total bladder volume evacuated by uninhibitable voiding contraction. Bimodal distribution can be seen, with mean value of 17% for detrusor hyperactivity with impaired contractile function (DHIC) and 96% for detrusor hyperreflexia (DH) ($P<.001$).

Figure 7.

—Voiding Parameters During Uroflowmetry*

Parameter	DHIC†	DH†	P
Fraction of patients with normal flow rates, %‡	33	80	<.05
Fraction of bladder volume voided, %	37 ± 10	86 ± 4	<.001
Total bladder volume, mL	278 ± 44	156 ± 45	.07
Postvoiding residual, mL	95 ± 15	14 ± 4	<.05

*Presented as mean ± SE.
†DHIC indicates detrusor hyperactivity with impaired contractile function; DH, detrusor hyperreflexia.
‡Standardized to reference nomogram.[15]

—Cystometric Parameters of the Uninhibited Contraction*

Parameter	DHIC†	DH†	P
Fraction of bladder volume evacuated, %	17 ± 3	96 ± 2	<.001
Rate of rise in detrusor pressure, cm H_2O/s†	2 ± 0.4	6.1 ± 0.8	<.0001
Maximum contraction pressure, cm H_2O	42 ± 3	42 ± 3	>.5
Isometric pressure change, cm H_2O	3 ± 1	35 ± 8	<.0005
Contraction duration, s	64 ± 9	53 ± 8	.45
Bladder volume before contraction, mL	215 ± 24	166 ± 26	.16

*Presented as mean ± SE.
†DHIC indicates detrusor hyperactivity with impaired contractile function; DH, detrusor hyperreflexia; and cm H_2O, centimeters of water.

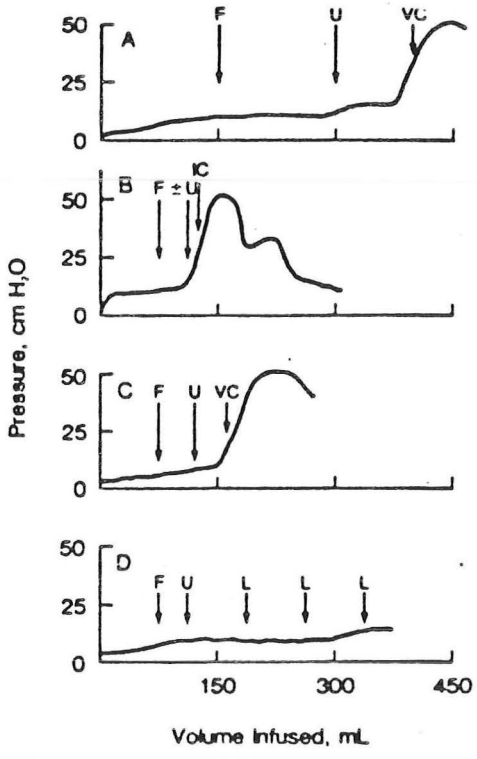
Unhibited detrusor contractions can also occur secondary to outlet obstruction such as from prostatic hypertrophy. It may be very difficult to tell whether these UDCs are primarily or secondary. Prostate size is not helpful in the elderly especially since the posterior lobe of the prostate which is palpated during rectal exam is the only lobe which the urethra does not travel through. Post void residual may also be misleading because a patient can valsalva and have a post void residual which does not suggest obstruction. If the UDCs are secondary to obstruction they often will resolve when the obstruction is resolved. UDC can also occur secondary to sudden increase in intrabdominal pressure such

as from coughing or sneezing. The history is often that there is a delay of a few seconds from the precipitating activity to the sensation of urge and then involuntary loss of urine.

In many cases no specific etiology of UDC can be identified even after a careful clinical and laboratory evaluation has been completed.

Another group of patients with UI have urgency but when studied have no evidence of lower urinary tract disease. On filling cystometrogram no UDCs are seen but bladder capacity is usually reduced and a strong urge to void (at low volumes) is accompanied by a voluntary void. Patients may also be seen who have very early first sensation during filling followed by frequent leakage. In the first group of patients one possibility is that there is a dysfunction of bladder sensors. The second situation has been related to transient drops in urethral pressure called urethral instability[43],[44],[45].

Figure 8.



Cystometrograms illustrating three types of incontinence associated with detrusor instability and/or urgency. A, Normal subject. B, Detrusor instability. C and D, Urgency without detrusor instability. F indicates first sensation of filling; U, urge to void; VC, voluntary detrusor contraction; IC, involuntary detrusor contraction; L, urine leakage not associated with detrusor contraction; plus-or-minus symbol, involuntary detrusor contraction may or may not be preceded by urge sensations. Urgency incontinence not associated with detrusor instability may either be associated with small bladder capacity (panel C) or with leakage unaccompanied by any detrusor contraction (panel D) related to transient drops in urethral resistance.

Local irritating factors such as bladder tumors, stones, diverticuli, and cystitis can also cause urge symptoms. In these cases it is thought the sensory input overwhelms the central inhibitory influence on the brainstem micturition center and detrusor contraction occurs.

Overflow incontinence occurs basically in one of two circumstances. During obstruction such as from the prostate gland, urethral stricture, or very large cystocele causing kinking of the urethra, or because of a detrusor that cannot contract (such as from diabetic autonomic neuropathy, or from lower motor neuron disease such as lesions of the sacral nerve roots, conus medullaris, or cauda equina[46]). Impaired sensory input from the bladder can also result in overflow and is most commonly secondary to diabetes. Medications such as anticholinergic agents are not infrequently responsible. The clinical features of overflow incontinence include a history of frequent almost continuous leakage of small volumes of urine. There may be lower abdominal tenderness and bladder distention. Urine flow rates are low and the post void residual is usually large.

Stress Incontinence occurs during sudden increases in intraabdominal pressure that results in simultaneous lost of urine. Urine is lost because the intravesicular pressure exceeds the urethral pressure. In the elderly patient this most commonly occurs because of hypermobility of the proximal urethra, which in essence herniates out of the pelvis so that the sudden rise in intraabdominal pressure is no longer transmitted to proximal urethra. Vesicular pressure now exceeds urethral pressure and urine is lost. This form of SI is often associated with multiparity and pelvic wall laxity. Stress Incontinence can also occur as a result of destruction of the urethral sphincter (sphincteric incontinence) such as from surgery or radiation fibrosis.

Clinically, patients lose small to moderate amount of urine (depending on the volume in the bladder before the stressor event) simultaneously with coughing, sneezing, laughing or when bending over or lifting a heavy object. If sphincteric incontinence is present urine loss may be continuous. Nocturia is often absent because intravesicular pressure is reduced in the supine position. Typically the post void residual (PVR) is small. In patients with a cystocele, puddling of urine may occur and the PVR may erroneously be interpreted as being large. It is important to remember that the presence or absence of a cystocele (except for the rare exception) or rectocele has nothing to do with stress incontinence. Many continent women have cystoceles just as women without them can have stress incontinence. It should be remembered that detrusor instability can occur with stress incontinence.

Functional incontinence applies to those cases in which the lower urinary tract is intact but other factors such as immobility, severe cognitive impairment, psychologic unwillingness or environmental barriers result in urinary incontinence. Finally, many elderly incontinent patients can have two or more of the above mention types of incontinence simultaneously.

Table 5. *Basic Types and Causes of Persistent Geriatric Urinary Incontinence*

TYPE	DEFINITION	COMMON CAUSES
Stress	Involuntary loss of urine (usually small amounts) with increases in intra-abdominal pressure (eg, cough, laugh, or exercise)	Weakness and laxity of pelvic floor musculature Bladder outlet or urethral sphincter weakness
Urge	Leakage of urine (usually larger volumes) because of inability to delay voiding after sensation of bladder fullness is perceived	Detrusor motor and/or sensory instability, isolated or associated with one or more of the following: Local genitourinary condition such as cystitis, urethritis, tumors, stones, diverticuli, and outflow obstruction Central nervous system disorders such as stroke, dementia, parkinsonism, suprasacral spinal cord injury or disease*
Overflow	Leakage of urine (usually small amounts) resulting from mechanical forces on an overdistended bladder, or from other effects of urinary retention on bladder and sphincter function	Anatomic obstruction by prostate, stricture, cystocele Acontractile bladder associated with diabetes mellitus or spinal cord injury Neurogenic (detrusor–sphincter dyssynergy), associated with multiple sclerosis and other suprasacral spinal cord lesions
Functional	Urinary leakage associated with inability to toilet because of impairment of cognitive and/or physical functioning, psychological unwillingness, or environmental barriers	Severe dementia and other neurological disorders Psychological factors such as depression, regression, anger, and hostility

*When detrusor motor instability is associated with a neurologic disorder, it is termed "detrusor hyperreflexia" by the International Continence Society.

EVALUATION

The comments that follow are from the perspective of the primary care physician and that which pertain to the initial evaluation of UI. That is, the evaluation short of urodynamic testing that can be done in an office or nursing home setting. There has been considerable debate regarding the diagnostic approach to evaluate UI in the elderly, especially with regard to the need for urodynamic testing. A number of researchers have devised algorithms to outline a clinical approach with consideration of the cost, time, availability and desire to avoid unnecessary invasive testing[47,48,49]. Hilton and Straton retrospectively reviewed UI in 100 elderly women and developed an algorithmic method for assessing patients. They showed that 69% of invasive investigations could be avoided by utilizing their method with minimal loss of diagnostic accuracy. They reported a diagnostic accuracy of 83% and a therapeutic accuracy of 95%. In a prospective study, Eastwood used a clinical algorithm modified from the one published by Hilton and compared it to the urodynamic diagnosis (see following figure).

Figure 9.

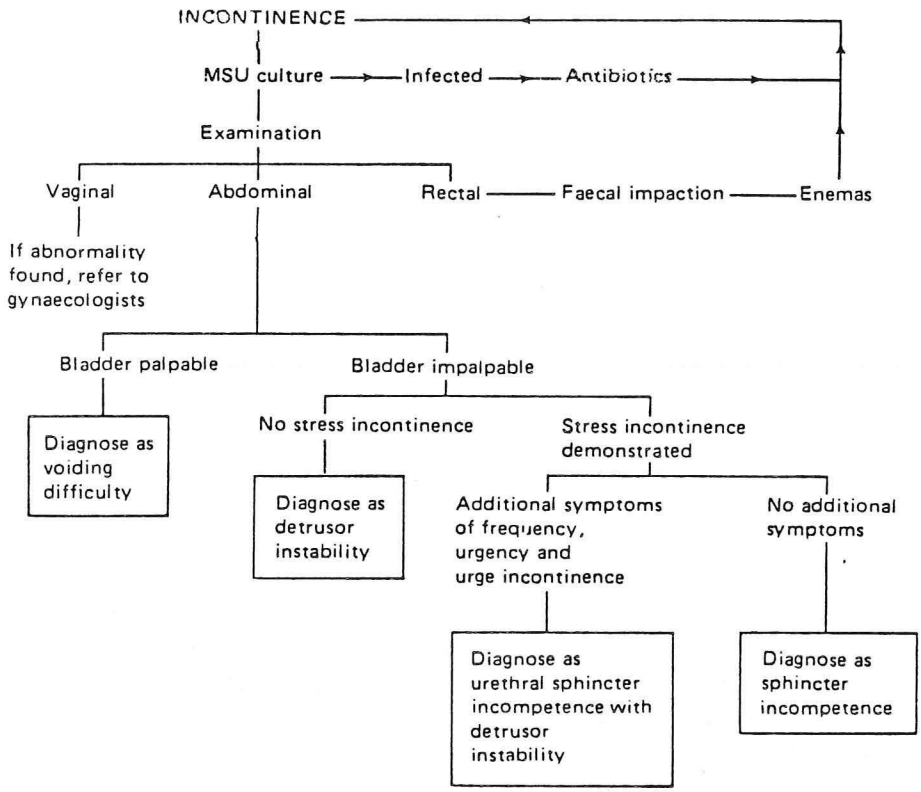


Figure. Clinical algorithm for the diagnosis of urinary incontinence in elderly women modified from published algorithm (Hilton & Stanton 1981).

They found 70% of the women they assessed were correctly diagnosed, but up to 20% might have been harmed by the treatment inaccurately suggested by the algorithm. However, both algorithms assessed for overflow incontinence by the examiners ability to palpate the patients bladder which is notoriously unreliable and particularly difficult in obese patients. In fact, except for one case of voiding difficulty diagnosed by a palpable bladder which was confirmed by urodynamic testing as well, the algorithmic approach missed 12 other urodynamically proven cases of urethral stenosis or acontractile bladder.

Table 6.

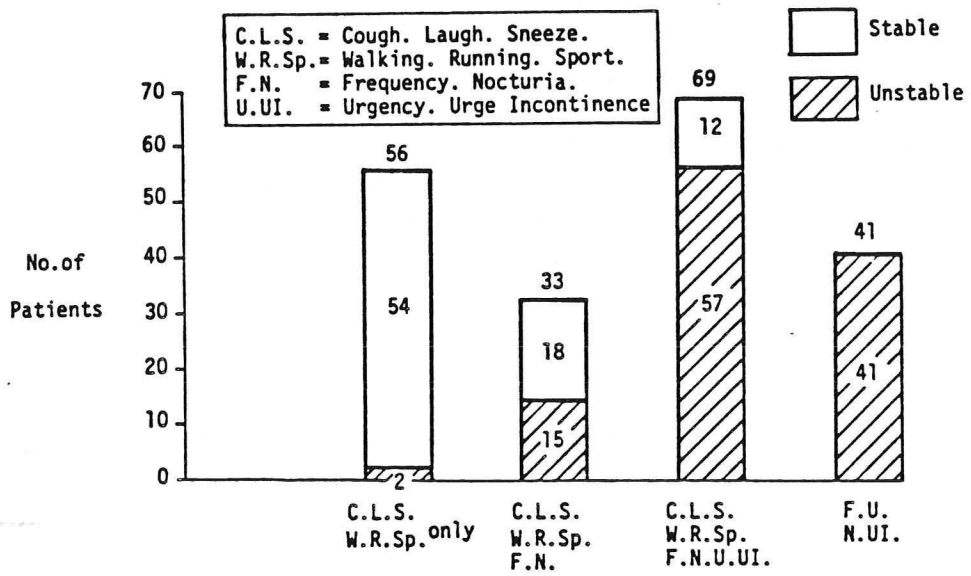
Comparison of algorithmic and urodynamic diagnoses in prospective series at Southampton geriatric unit.

Algorithmic result		*Urodynamic diagnosis*	
Treat as detrusor instability	52	Detrusor instability	42
		Normal	4
		Acontractile bladder	3
		Instability with urethral stenosis	3
Urethral sphincter incompetence	5	Urethral sphincter incompetence	2
		Posturally induced detrusor instability	2
		Acontractile bladder	1
Mixed detrusor instability with urethral sphincter incompetence	7	Detrusor instability	1
		Instability with urethral stenosis	1
		Mixed DI and USI	1
		Acontractile bladder	4
Voiding difficulty	1	Voiding difficulty	1
	65		65

Of note, obstruction to catherization for cystometry resulted in the diagnosis of 4 cases of urethral stenosis. These were not by themselves producing obvious symptoms of poor urine flow or clinically detectable retention. Three of these woman presented with urge symptoms, not an uncommon presentation of outflow obstruction in women. If the algorithm was modified to include a post void residual (pvr) one would suspect the diagnostic accuracy may have been better.

Some have stated that "the bladder is an unreliable witness" in reference to the lack of reliability the clinical symptoms provide in yielding the underlying etiology of UI. The sensitivity of urge and stress symptoms is above 80%[50]. No one symptom is 100% reliable but rather all the history and physical findings must be considered together to arrive at a correct diagnosis. Farrar[51] et al found that 54 of 56 women with symptoms of stress incontinence had genuine stress incontinence and all 41 women in their study with symptoms of urge incontinence had detrusor instability (see Figure 10). There series differed from most others because symptoms were carefully pursued and the gold standard they used for comparison included sophisticated urodynamics including videourodynamics.

Figure 10.



Analysis of symptoms for those in the incontinence group.

In a blind, prospective study Resnick found that more than 90 percent of patients referred to a tertiary care center could be correctly diagnosed by clinical evaluation, voiding record, pvr, and observed micturition[52]. They have also prospectively tested in blinded fashion, an algorithmic approach in 70 nursing home patients with an average age of 90. The overall diagnostic accuracy was 82% and lead to appropriate therapy in over 90% of cases[53].

Although urodynamics is often touted as the gold standard in assessing UI it must be remembered that a number of abnormalities in the elderly patient may be present without any clinical correlation. This is also true for findings on the physical examination. For example, Ouslander studied 167 women and 96 men all over the age of 65 years who were evaluated in an outpatient urodynamic laboratory. 19% of patients had uninhibited detrusor contractions present without incontinence. 23% of patients without incontinence presented with maximal urethral closing pressures less than 20cm of water. This finding is associated with incontinence in younger populations[54]. An abnormal prostate (enlarged and/or nodular) was present in 60% of the men presenting with incontinence as well as in 65% of the men without incontinence.

The following recommendations reflect my bias in the initial evaluation of the elderly patient after reviewing the present literature. There is a dearth of good data on this topic however and the next few years will hopefully supply more answers that are currently available. These recommendations are also consistent with those arrived at during a recent NIH consensus conference on urinary incontinence in the adult convened in October of 1988.

The evaluation of UI has certain routine components but a variety of considerations need to be taken into account. The setting in which the evaluation takes place, the patients functional and cognitive state and their overall medical status will help determine the diagnostic and therapeutic strategy which must be tailored to the individual. The physiologic changes that occur in the elderly as above noted must be taken into account when assessing symptoms and signs. The initial task is to assess for any factors that can cause transient incontinence (DIAPPERS). Transient incontinence may account for as much as a third of incontinence in the community-dwelling elderly persons and for half of incontinence in acutely hospitalized elderly patients[55]. [56]Once transient causes have been ruled out one then focuses on the persistent causes of UI.

The following outlines the key aspects of the history.

Active Medical Conditions:

Neurologic disorders, diabetes mellitus, congestive heart failure, venous insufficiency, and malignancy.

Medications (see Table 5.)

Fluid-intake pattern.

Type and amount of fluid (especially before bedtime, alcohol?)

Past genitourinary history

Childbirth, surgery, urinary retention, radiation, and recurrent urinary tract infections.

Symptoms of Incontinence

Onset and duration.

Type of incontinence should be described. Stress vs. urge vs. mixed. Frequency, timing, volume of incontinence. If stress related, timing of stress to incontinent episode-simultaneously?

Dysuria, hematuria, hesitancy, dribbling, slow or interrupted stream, incomplete emptying, straining to void, nocturia, urgency

Other symptoms

Neurologic (indicative of stroke, dementia*,[157]parkinsonism, normal pressure hydrocephalus, spinal cord compression).

Psychologic (depression)

Bowel (constipation and stool incontinence)

Perceptions of Incontinence

Patients' concerns or ideas about underlying cause(s)

Interference with daily life

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*Dementia appears to correlate poorly with detrusor hyperreflexia- 57.

Severity
Patient and caregivers perceptions of the problem and expectations from evaluation and treatment.

Environmental Factors
Location and structure of bathrooms
Availability of toilet substitutes.

After the history has been obtained (or even before the patient is formally evaluated) it is helpful to obtain an incontinence record completed by the patient or caregiver. This can assist in the history, serve as a guide to treatment and help monitor the response to therapy.

Table 6.

Bladder Record

Name_____ Week Starting _____ / _____ / _____
Month Day Year

Instructions:

Mark D for "Dry," each time urination occurs without leakage

Mark W for "Wet," each time leakage occurs
(If you cannot tell when the leakage occurred,
Mark W at the time closest to when you find the wetness.)

	7am	8am	9am	10am	11am	12n	1pm	2pm	3pm	4pm	5pm	6pm	7pm	8pm	9pm	10pm	11pm	12am	1am	2am	3am	4am	5am	6am
Mon																								
Tues																								
Wed																								
Thur																								
Fri																								
Sat																								
Sun																								
	Morning					Afternoon						Evening					Night							

Example of a bladder record for outpatient settings.

Physical Examination

The patient requires a complete physical examination with particular attention to the abdominal, neurologic (especially the lumbosacral innervation), rectal and pelvic exams and functional and mental status. There are a number of parts of the exam that deserve emphasis.

The rectal exam is important to check for fecal impaction as well as for a mass. Whether fecal impaction can be excluded by the absence of stool detected by the examining finger is unknown. An important point to emphasize again is that prostate size has little to do with whether obstruction is present or not. The posterior lobe of the prostate is palpated on rectal exam, but the urethra does not traverse through this lobe of the gland. Glands that are small by exam can be obstructing and the converse is also true.

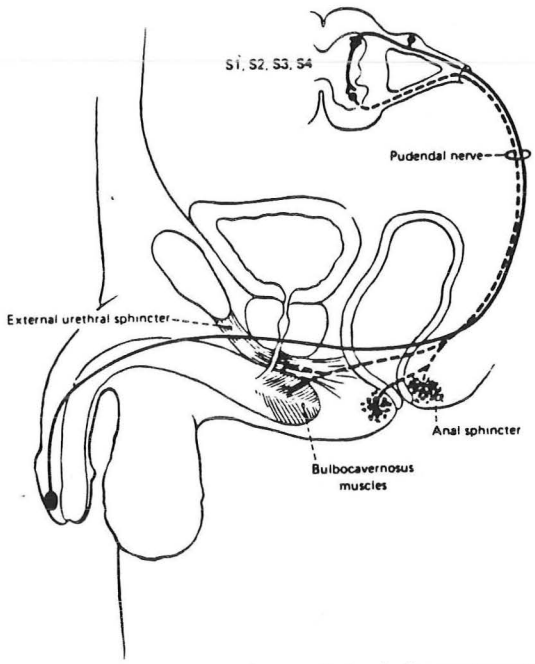
The remainder of the rectal exam serves as a neurourologic assessment. The same nerve roots (S2-S4) innervate both the anal and urethral sphincter.

The first aspect of the exam is to test for sensation in the saddle area to check the afferent loop of the reflex arc. Once this is done the patient is asked to contract the anal sphincter around the examining finger. This assesses the motor component. Some patients may have difficulty contracting the anal sphincter and this may not represent neurologic disease.

To further assess the reflex arc the anal wink or anocutaneous reflex is elicited: another reflex, the bulbocavernous reflex, is tested by inserting a finger into the anus to detect anal sphincter contraction while the glans penis or clitoris is squeezed.

Some uncertainty exists as to what proportion of neurologically intact subjects have reflex anal contraction following stimulation of the glans or clitoris[58,59]. While their absence does not necessarily suggest a pathologic process neither does their presence exclude detrusor underactivity due to diabetic neuropathy[39]. Therefore, although this maneuver is often mentioned as part of the neurourological evaluation it appears to be relatively useless. The tone of the anal sphincter should be assessed. Although a patulous anus may suggest paralysis of the anal sphincter, chronic over distention from severe constipation or prior rectal surgery can account for these findings[60].

Figure 11.



Reflex pathway of the bulbocavernosus reflex. Solid line-afferent; dotted line-efferent.

During the pelvic exam it is important to examine the patient for signs of atrophic vaginitis, and other anatomic abnormalities. The presence or absence of a cystocele or rectocele must be interpreted cautiously. In a study by Ouslander[54] 61% of incontinent patients had moderate or marked cystourethrocele but 41% with the same findings had no

UI. A study by Diokno[61] found that those with urethral incompetency were more likely to have severe cystocele and/or rectocele. The Q-tip test and bladder neck elevation (Bonney) tests are of limited usefulness.

A provoked full-bladder stress test is recommended to assess stress incontinence. The patient is asked to cough with a full bladder in the supine position. If urine is leaked this is highly suggestive of genuine stress incontinence. If there is a delay from the time of the cough to loss of urine this may represent stress induced uninhibited contraction. If the test is negative while supine the patient is asked to stand and the process is repeated. The sensitivity of the full-bladder stress test in identifying urethral incontinence exceeds 80-90%[62].

A post-void residual urine should be done in all patients. Although there may be some increase in the pvr in the elderly, values should still be under 50cc and over 100 cc should prompt further investigation. Falsely low pvr can be seen when patients are tested during low bladder volumes. Patients can can falsely reduce their pvr with a valsalva maneuver during voiding.

Additional bedside or "eye-ball" urodynamic techniques are being assessed in prospective studies and may prove useful in evaluating patients[63].

Other tests to be obtained in all patients are included in Table 7. At this point in the evaluation a decision for treatment or further evaluation by formal urodynamic testing is required. A number of considerations must be taken into account: the risk of empiric therapy, the overall health status of the individual, is surgery being planned or under consideration? It is useful to remember that non-invasive and reversible therapy does not demand as exact a diagnosis as invasive and irreversible therapy[64]. Also UI is not a fatal disease process that requires urgent treatment.

Table 8.

BASIC COMPONENTS OF THE DIAGNOSTIC EVALUATION OF PERSISTENT URINARY INCONTINENCE

ALL PATIENTS
- HISTORY
- PHYSICAL EXAMINATION
- INCONTINENCE CHART
- POST-VOID RESIDUAL DETERMINATION
- PROVOKED FULL BLADDER STRESS TEST IN WOMEN
- URINALYSIS
- URINE CULTURE
- BLOOD GLUCOSE, BUN, CREATININE, CALCIUM, ELECTROLYTES

Urodynamic evaluation is indicated when;

- Persistent diagnostic uncertainty exists after careful clinical evaluation.
- Morbidity associated with potentially misdirected empiric therapy is high.
- When empiric therapy has failed
- When surgical intervention is planned
- When the patient has overflow incontinence

Urodynamic testing is not a single test but consists of five different basic tests that should be selected depending upon the clinical situation.

These five examinations include;

-cystometry
-uroflow
-sphincter electromyography
-urethral pressure profilometry
-radiographic visualization of the lower urinary tract.

Cystoscopy may also provide important information in evaluating the patient presenting with hematuria regardless of the patients age. A further discussion of urodynamic testing is beyond the scope of this protocol but a number of excellent discussions on this topic are referenced[65],[66],[25].

TREATMENT

The first step in providing appropriate treatment for UI rests in the careful assessment of the patient. Consideration of the patients overall clinical status, environment, and desires are important factors that help guide treatment decisions. Evaluation and treatment options should never be determined on the basis of age alone. Most patients can expect significant improvement or cure of their incontinence. Any review of treatment options is difficult because many treatments have not been extensively studied by well designed clinical trials. Follow up is often lacking, studies are often done on a very selected population and the results may not be generalizable. The degree of evaluation before and after treatments may be limited and measures of efficacy may not be well defined or documented. Furthermore, study size are often small and frequently done in younger populations.

A number of different treatment options are available to treat UI. They include pharmacologic, behavioral, surgical, mechanical, catheters, supportive and palliative measures. The specific treatment depends on the underlying pathophysiologic process. I will review the treatments therefore under four major groups.

Table 9.

Treatment Options for Geriatric Incontinence

Drugs
Bladder relaxants
Alpha agonists
Estrogen
Surgery
Bladder neck suspension
Removal of obstruction or pathologic lesion
Other
Training Procedures
Pelvic floor exercises
Biofeedback
Bladder and habit retraining
Toileting procedures
Catheters (for Overflow Incontinence)
Intermittent
In-dwelling
Mechanical/Electrical Devices
Artificial sphincters
Intravaginal electrical stimulation
Anal electrical stimulation
Nonspecific, Supportive, and Palliative Measures
Toilet substitutes (eg, commodes and urinals)
Environmental manipulations
External collection devices
Incontinence undergarments and pads
Chronic in-dwelling catheters

I. Detrusor Instability (Uninhibited detrusor contractions)

There have been many medications which have been used to inhibit detrusor contractions producing increased bladder capacity and delay of and reduction in amplitude of involuntary contractions. These agents work by their anticholinergic effect, antispasmodic effect (direct smooth muscle effect) or a combination of the two. Propantheline bromide is a pure anticholinergic agent commonly used for its antimuscarinic effect in the lower urinary tract. Because of the myriad of side effects (dry mouth, impaired lens accommodation, increased intraocular pressure, constipation, tachycardia, esophageal reflux, and mental status changes, especially in those patients with cognitive impairment) these agents are best avoided in the elderly. Emepronium is an anticholinergic commonly used in Europe with varying success[67],[68],[69]. The agent probably most frequently used in the U.S. is oxybutinin. This drug appears to act directly on smooth muscle at a site metabolically distal to the cholinergic receptor mechanism. It also possesses some antimuscarinic and local anesthetic properties[70]. A randomized, double blind, placebo-controlled study has shown benefit with oxybutinin[71] both symptomatically and by urodynamics. It is generally well tolerated in the elderly and side effects are anticholinergic. Dicyclomine and flavoxate are two other agents in this class. There have been some favorable clinical reports but other reports have shown limited effect[72],[70]. The

tricyclic antidepressants have long been useful agents in facilitating bladder storage. The precise mechanism is not known but they have an inhibitory effect on the detrusor resulting in decreased contractility and an alpha agonist like effect which increases outlet resistance. Imipramine is most commonly used. Its utility in the elderly is probably limited because of its potential side effects of postural hypotension, sedation, and anticholinergic effects. Calcium channel blockers have been used in view of their smooth muscle relaxant properties. In particular terodiline is an agent having both calcium-antagonistic and anticholinergic properties that has shown benefit[73],[74],[75],[76]. How it compares to other anticholinergic agents and its side effect profile in the elderly remains to be seen. It is presently not available in this country. Other drugs such as prostaglandin inhibitors, bromocriptine, and beta adrenergic agonists have been used but seem to have limited or no benefit[77],[78],[79],[80].

Estrogens have been shown to alleviate the symptoms of urgency and frequency of micturition in post-menopausal women[81]. Its' effect on detrusor instability has not been studied and its benefit in urge symptoms is presumed secondary to the irritative symptoms from estrogen deprivation (atrophic urethritis). Behavioral therapy ,specifically bladder training therapy has benefited patients with urge incontinence. Since there is confusion regarding the terminology of these techniques Hadley et al[44] has attempted to divide these scheduling regimens into four conceptual categories. These are bladder training, habit training, timed voiding and prompted voiding.

In bladder training the patient voids at scheduled intervals which are progressively increased. Commonly a patient voids every two hours and if continence is maintained this is gradually increased to every three or four hours. Many studies have shown benefit in patients with urge and stress incontinence. Most studies have been done in middle aged patients. They are often highly motivated, educated, and without cognitive deficits. The physiologic basis of bladder training is not known. Improving central nervous system control of micturition reflexes is a likely mechanism. Habit training, timed voiding and prompted voiding are utilized in patients with cognitive deficits or functional incontinence and will be discussed below.

Biofeedback has been used in patients with urge incontinence. This is a learning technique to exert better voluntary control over the sphincter, detrusor, and abdominal muscles. Some studies have shown cure or significant improvement[82]. However, long term follow-up data is not available. It requires continued motivation and practice on the part of patient and because of the equipment and training required to provide this therapy the utility of this technique on a large scale is being debated.

Surgery has little role in the treatment of detrusor instability except in the case of detrusor instability secondary to outlet obstruction. Bladder transection has been performed in patients who have failed to respond to standard conservative treatment[83],[84]. This treatment has largely been abandoned for lack of efficacy.

Electrical stimulation of the sacral roots[85] or peroneal or posterior tibial nerve[86] have been used in patients with urge incontinence but there role in elderly patients is not known.

Ewing[87] has reported extra trigonal phenol injections as being successful in 23 of 30 patients refractory to pharmacologic treatment. Five developed chronic retention and were reported happy with this situation, resorting to self catheterization. Cox[88] reported a case of retention in a 71 year old female. Blackford treated 116 patients and reports no incidence of urinary retention[89]. The true incidence of this complication needs to be established.

II. Stress Incontinence

Pelvic muscle exercises (pelvic floor exercises, kegel) strengthen the voluntary periurethral muscles, which exert a closing force on the urethra. Studies have shown a 30-90% improvement with this technique with widely varying design and outcome measures among studies. Continued exercise is required for long term benefit. In view of the cost and lack of any side effects it is reasonable to employ this as part of any therapeutic regimen to treat stress incontinence. Exercise may be used in concert with biofeedback although additional benefit has not been consistently shown to date. Bladder training can also be useful as discussed above.

Medications

Because of the thinning of the urethral tissues and a decrease in closing pressure and coaptation in post menopausal women, estrogen therapy has been used in an attempt to reverse these changes and improve continence in patients suffering with stress incontinence. Early studies identified subjective improvement without urodynamic assessment. Three uncontrolled studies that evaluated urodynamic parameters such as maximal urethral pressure profiles revealed variable urodynamic results but subjective improvement was noted in all[90],[91],[92]. Wilson[93] in a double blind controlled prospective trial in 36 women showed no significant difference in treatment groups by symptoms or objective urodynamic measurement. After six weeks of treatment a greater reduction in the number of pad changes/24 hours in the estrogen treated patients were noted but did not reach statistical significance because of a marked response in the control group as well. In another purely subjective, double-blind, placebo-controlled, cross over study, oral estrogen showed improvement in urge incontinence but not SI[94]. Although the numbers of well controlled studies are small, most have shown no clear benefit in treating stress incontinence. More studies are needed. Because the bladder neck and proximal urethra contain a preponderance of alpha adrenergic receptor sites alpha agonists have been tried with and without estrogens in patients with SI. Pseudoephedrine, ephedrine, and phenylpropanolamine have all been used. The latter is most frequently used. Collste[95] (see Table 10.) in a double blind placebo controlled study of 24 women (35-65 years) treated SI of slight to moderate grade with phenylpropranolamine (PPA) 50 mg. bid. They showed a significant decrease in incontinent episodes but no decrease in frequency of urination. Also, a significant increase in the maximal urethral closure pressure was noted in the treated group compared to placebo. Subjectively patients preferred PPA as well. These agents may also augment the response to estrogen treatment in women with mild to moderate SI[96]. Side effects of alpha adrenergic agents especially in the elderly include anorexia, nausea, insomnia, confusion, restlessness, hallucinations and exacerbation of

hypertension and coronary artery disease.

Table 10.

*Micturition and leakage episodes per forty-eight hours (mean ± SD) in all patients and when patients grouped according to stress urinary incontinence grades I and II**

Variable	Initially M ± SD (n)	Placebo (P) M ± SD (n)	Rinexin (R) M ± SD (n)	Stat. Signif.† R-P (n)
Micturitions/48 hours				
All patients	16 ± 6 (15)	18 ± 13 (21)	19 ± 15 (21)	ns (21)
Grade I	14 ± 6 (11)	18 ± 15 (16)	19 ± 17 (16)	ns (16)
Grade II	19 ± 4 (4)	19 ± 6 (5)	18 ± 7 (5)	ns (5)
Leakage episodes/48 hours				
All patients	5 ± 4 (15)	6 ± 6 (21)	2 ± 3 (21)	p = 0.005 (21)
Grade I	3 ± 3 (11)	5 ± 5 (16)	2 ± 3 (16)	p = 0.014 (16)
Grade II	10 ± 5 (4)	9 ± 10 (5)	4 ± 4 (5)	p = 0.09 (5)

*Values initially and after placebo (P) and after phenylpropanolamine (R).
†Effects of Rinexin compared to placebo (R-P). Statistical significance test: Wilcoxon's signed rank sum test. ns = p > 0.10.

Biofeedback has been used in the treatment of stress incontinence with positive benefit. Burgio using bladder sphincter biofeedback studied 19 mentally alert, ambulatory outpatients (65 to 86 years). These patients achieved an 82% reduction in the frequency of SI[82]. Burton et al[97] studied a group of elderly patients with both stress and urge incontinence. They were trained in either bladder-sphincter biofeedback or behavioral training without biofeedback. Both groups had a reduction in accidents of about 80%. Again, these treatments are limited to highly motivated, cognitively intact patients. It is not useful in patients with post-surgical incontinence characterized by constant leakage.

Surgery is particularly effective in patients with stress incontinence secondary to urethral hypermobility (see Table 11.).

Table 11.

Surgical Procedures to Correct Female Stress Urinary Incontinence

Transvaginal
Anterior colporrhaphy (Kelly)
Retropubic
Suprapubic vesicourethral suspension (Marshall–Marchetti–Krantz)
Ileopectineal ligament urethrovesical suspension (Burch)
Combined transvaginal and retropubic
Needle bladder neck suspensions (Pereyra, Stamey, and Raz)
Sling operations
Artificial material
Periurethral Teflon injection
Artificial urinary sphincter

With the advent of needle suspensions (pereyra, stamey, raz) most elderly patients are potential surgical candidates[98,99,100,101]. A variety of other surgical procedures are available with success rates over 90%. In patients with sphincteric incontinence periurethral injections (teflon, microsperes, collagen)[102] and artificial sphincters are available. Because anterior colporrhaphy (Kelly) cannot elevate the bladder to the desirable high, fixed, retropubic position, it has a very high failure rate (50-65%). This procedure should be

abandoned in the treatment of stress incontinence.

Pessaries can be used in women with mild SI, it is believed they stabilize the bladder base and improve the transmission of pressure. They require considerable dexterity and have limited usefulness in older patients.

III. Overflow Incontinence

If obstruction is the cause of overflow incontinence surgical correction is usually indicated. Transurethral resections can be done under local anesthesia[103] or palliative decompression procedures such as prostatotamies can be performed allowing even some of the most frail patients to undergo surgical treatment. If a surgical procedure is still not feasible use of an alpha adrenergic blocker such as prazosin may be useful as a temporizing agent. [104]In patients with overflow incontinence secondary to an underactive detrusor medication probably has no role in geriatric incontinence[26]. Bethanechol, the drug most often used in this setting can cause bothersome cholinergic side effects including abdominal pain, diarrhea, and bronchoconstriction. Attempts to augment voiding using the Crede or valsalva maneuver may be useful. The primary treatment options in these patients are intermittent catheterization versus indwelling catheter. The former is preferred if the patient has the manual dexterity or if family or other caregivers are willing to learn and do the procedure.

IV. Functional Incontinence

A number of scheduled regimens can be used in patients with cognitive dysfunction. These include habit training, timed voiding, and prompted voiding. In habit training the patient is asked to void at scheduled times. If the patient needs to void before the designated time they are allowed to and the pattern is noted. The schedule is readjusted to the timing of the patients voids. Intervoiding intervals are either increased or decreased as dictated by the patient.

Table 12.

—Differences in Scheduling Adjustments of Bladder Training and Related Protocols

Regimen	Change in Intervoiding Interval Over Course of Regimen
Bladder training	Increased
Habit retraining	Increased or decreased
Timed voiding	Unchanged
Prompted voiding	Prompting schedule unchanged, voiding interval variable

In timed voiding patients are given fixed voiding schedules with intervoiding intervals remaining unchanged. This is a particularly common approach found in nursing homes[105].

In prompted voiding, the patient is asked at regular intervals if they need to void. They are assisted to the toilet only if the response is positive.

External collection devices. The condom catheter can be helpful in selected cases. It may reduce the patients willingness to comply with behavioral strategies or drug therapy. There are a number of well known complications associated with its use. These include balanitis, necrosis of tissue, and an increase risk of urinary tract infections[106],[107]. Newer products such as a self adhesive condom that does not require a potentially constricting piece of tape are available. Catheters should be changed daily and the skin inspected. Progress has been made in the development of a suitable female external catheter by NASA and others. These products are currently available although there is limited experience with their use in older patients.

There has been an explosion in the development of various absorbent products. A helpful product reference is the Help for Incontinent People (HIP) resource guide of continence aids and services (available for $3 from HIP Box 544 Union, South Carolina 29739). Patient needs vary depending upon each situation. Patients with small volume leaks may use inserts. If urine losses are large a thicker pad may be more appropriate. For bedfast patients launderable bed sheets may be best. Adult diapers should include fitted legs to help prevent leg leakage.

Lastly, environmental factors need to be considered. If manual dexterity is a problem buying clothes with velcro fasteners, wrap-around skirts, or elastic waistbands can be helpful. Location of the bathroom is important. Is it accessible to the patient? A bedside commode may be useful especially for night-time use. Handrails next to the commode and height of the seat (should be 15 to 20 inches) are important. Brink and Wells[108] offer a detailed discussion of these issues.

Table 13.

Important Features in the Design of Toilets, Toilet Supplements, and the Environment

Toilets
Location (ie, distance)
Access
Removal of barriers
Adequate lighting
Call system
Bathroom door design
Adequate space
Accommodation of wheelchairs, walkers, and caregivers' help
Seat height
Appropriately located grab bars
Commodes
Adequate number (in institutions)
Ease and safety of transport
Design to facilitate support and safety in patient transfer
Bedpans
Facilitate use in sitting position
Fracture pan design
Urinals
Adequate handles
Curvature to prevent leakage

SUMMARY

Urinary incontinence is common among older individuals. Although it is not a normal part of aging certain age related changes predispose the older patient to develop it. The consequences of urinary incontinence are significant. It is costly, morbid, and often leads to social isolation and institutionalization. The public and often professional perception is that nothing can be done for this problem. In fact, most cases can be cured or improved and every patient deserves evaluation and treatment.

Many controversies and unanswered questions still remain. Further research is needed from a wide range of disciplines to improve our evaluation and treatment of urinary incontinence.

Directions for Future Research[109]

1. Basic research on the biological mechanisms underlying the specific forms of urinary incontinence.

2. Epidemiologic studies with emphasis on elucidation of risk factors for development of urinary incontinence, its occurrence in specific populations, and the natural history of the various clinical and physiologic subtypes.

3. Studies of strategies to prevent urinary incontinence.

4. Randomized clinical trials, including longitudinal studies in well-specified populations, of algorithms for the systematic assessment of incontinent patients and of specific behavioral, pharmacologic, and surgical treatment, either alone or in combination.

5. Development of new therapies, including pharmacologic agents with greater specificity for the urinary tract and new behavioral and surgical strategies and other innovative techniques, including electrical stimulation.

DRUGS USED TO TREAT INCONTINENCE

Drugs	Dosages	Action	Type of UI
Anticholinergics			
Propantheline	7.5-15 mg tid	Increased Bladder Capacity	Urge or SI with UDC
Antispasmotics			
Oxybutinin	2.5-5.0 mg tid		
Flavoxate	100-200 mg tid		
		Diminish involuntary Bladder contractions	
Tricyclic Antidepressants			
Imipramine	25 mg q hs		
Calcium Antagonists (investigational)			
Terodiline	not available		
Alpha-Adrenergic Agonists			
Phenylpropanolamine	50 mg bid	Increase urethral smoooth muscle contraction	Stress
Pseudoephedrine	15-30 mg tid		
Imipramine	25 mg q hs		
Conjugatated Estrogens			
Oral	0.625 mg/d	Increased outlet resistance	Stress
Topical	0.5/1 gm per application	Diminish inflammation	Urge with atrophic vaginitis
Alpha-Adrenergic Blockers			
(Prazosin)	1 mg bid	Decrease urethral resistance	Temporizing measure overflow incontinence (BPH)

PRIMARY TREATMENT

Type	Medical	Behavioral	Surgical
Urge	Bladder relaxants Estrogens (if atrophic vaginitis/urethritis)	Bladder training Biofeedback	Electrical simulation? Removal of obstruction or other irritating pathologic process
Stress	Alpha agonists Estrogen? Pelvic muscle exercises	Bladder Training Biofeedback	Bladder neck suspension Artifical sphincter
Overflow	Catheters Indwelling Intermittent Augmented voids		Removal of obstruction
Functional	PADS Undergarment External collection devices	Prompted voiding Timed voiding Environmental manipulations	

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