

Study of the Impact of Urinary Incontinence on Quality of Life in Egyptian Elderly Females

Thesis

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LIST OF ABBREVIATIONS

BMI	Body mass index
CS	Caesarean section
CT	Computerized tomography
FUSS	Female Urinary Symptom Score
ICI	International Consultation on Incontinence
IIQ-7	Incontinence Impact Questionnaire
IPAQ	International Physical Activity Questionnaire
I-QOL	Incontinence associated quality of life
IVP	Intravenous pyelography
KHQ	King's health questionnaire
LUTS	Lower urinary tract symptoms
MRI	Magnetic resonance imaging
OAB	Overactive bladder
QOL	Quality of life
SUI	Stress urinary incontinence
UDI-6	Urinary Distress Inventory
UI	Urinary incontinence
UK	United Kingdom
UPP	Urethral pressure profilometry
USA	United States of America
vs	Versus
Yrs	Years

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INTRODUCTION

Urinary incontinence (UI) is defined according to the International Continence Society as a condition of involuntary urine loss that is objectively demonstrable and is a social or hygienic problem (*Nadir et al., 2009*).

Up to 35% of the total population over the age of 60 years is estimated to be incontinent, with women twice as likely as men to experience incontinence. One in three women over the age of 60 years is estimated to have bladder control problems (*Thom et al., 1997*).

In the United States of America (USA), UI affects approximately 13 million Americans, with the highest prevalence being in the elderly. For non-institutionalized people older than 60, the prevalence of UI ranges from 15- 25 % (*Zimmern et al., 2002*). While in the nursing homes, this increases to about 50% of nursing home residents (*Diokno, 2001*).

Urinary Incontinence imposes a significant psychosocial impact on individuals, their families, and their caregivers regardless of its type. It is also a major cause of institutionalization in the elderly. Elderly rank UI among the 4

most distressing disorders after angina, difficulty with ambulation and psychiatric disorders (*LEE, 2004*).

Although UI is not generally a great threat to life, yet it is more often an everyday annoyance and because of this it can lead to emotional distress. Moreover if incontinence is left untreated, complications can occur. Complications include physical complications such as urinary tract infection, pressure sores, candida infection, sleep deprivation due to nocturnal urination, falls and fractures (*Grimby et al., 1997*).

Complications can also be psychological as about 80% of incontinent elderly have negative psychological complications. This includes emotional disturbance such as anxiety, sense of insecurity, shame and embarrassment, reduced social activities, weakened interpersonal relationships as a result of social withdrawal, low self-esteem and reduced sexual activity. Unlike the past; it is now clear that regardless the type of UI, there is great impact on the quality of life (QOL) of those affected (*Grimby et al., 1997*).



AIM OF THE WORK

The main objective of the study is to identify the impact of UI on the quality of life of Egyptian elderly females suffering from persistent UI.

THE STRUCTURE OF THE LOWER URINARY TRACT AND AGE-RELATED CHANGES IN THE URINARY SYSTEM

The Structure of the Lower Urinary Tract

The main organs involved in urination are the bladder and the urethra. The smooth muscle of the bladder, known as the detrusor, is innervated by sympathetic nervous system fibers from the lumbar spinal cord and parasympathetic fibers from the sacral spinal cord. Fibers in the pelvic nerves constitute the main afferent limb of the voiding reflex. The parasympathetic fibers to the bladder that constitute the excitatory efferent limb also travel in these nerves (*Rajaofetra et al., 1992*).

Smooth muscle bundles pass on either side of the urethra, and these fibers are sometimes called the internal urethral sphincter, although they do not encircle the urethra. Farther along the urethra is a sphincter of skeletal muscle, the sphincter of the membranous urethra (external urethral sphincter). Part of the urethra is surrounded by the external urethral sphincter, which is innervated by the somatic pudendal nerve originating in the cord, in an area termed Onuf's nucleus (*Rajaofetra et al., 1992*).

The bladder's epithelium is termed transitional epithelium which contains a superficial layer of dome-like cells and multiple layers of stratified cuboidal cells underneath when

evacuated. When the bladder is fully distended the superficial cells become squamous and the stratification of the cuboidal is reduced in order to provide lateral stretching (*Rajaofetra et al., 1992*).

Mechanism of Micturition:

Micturition is fundamentally a spinobulbospinal reflex facilitated and inhibited by higher brain centers such as the pontine micturition center and is subjected to voluntary facilitation and inhibition (*Yoshimura et al., 2003*).

The lower urinary tract has two discrete phases of activity: the storage phase, when urine is stored in the bladder; and the voiding phase, when urine is released through the urethra. The state of the reflex system is dependent on both a conscious signal from the brain and the firing rate of sensory fibers from the bladder and urethra (*Yoshimura et al., 2003*). At low bladder volumes, afferent firing is low, resulting in excitation of the outlet (the sphincter and urethra), and relaxation of the bladder. At high bladder volumes, afferent firing increases, causing a conscious sensation of urinary urge. When the individual is ready to urinate, he or she consciously initiates voiding, causing the bladder to contract and the outlet to relax. Voiding continues until the bladder empties completely, at which point the bladder relaxes and the outlet contracts to re-initiate storage. The muscles controlling micturition are controlled by the autonomic and somatic

nervous systems. During the storage phase the internal urethral sphincter remains tense and the detrusor muscle relaxed by sympathetic stimulation. During micturition, parasympathetic stimulation causes the detrusor muscle to contract and the internal urethral sphincter to relax. The external urethral sphincter is under somatic control and is consciously relaxed during micturition (*Lynch, et al., 2008*).

Storage phase

During storage, bladder pressure stays low, because of the bladder's highly compliant nature. The intravesical pressure will show a very slight rise as the bladder is filled. Therefore, the pressure increase is slight until the organ is relatively full. The bladder's smooth muscle has some inherent contractile activity; however, when its nerve supply is intact, stretch receptors in the bladder wall initiate a reflex contraction that has a lower threshold than the inherent contractile response of the muscle. Action potentials carried by sensory neurons from stretch receptors in the urinary bladder wall travel to the sacral segments of the spinal cord through the pelvic nerves. Since bladder wall stretch is low during the storage phase, these afferent neurons fire at low frequencies. Low-frequency afferent signals cause relaxation of the bladder by inhibiting sacral parasympathetic preganglionic neurons and exciting lumbar sympathetic preganglionic neurons (*Lynch, et al., 2008*). Conversely, afferent input causes contraction of the sphincter



through excitation of Onuf's nucleus, and contraction of the bladder neck and urethra through excitation of the sympathetic preganglionic neurons (*Yoshimura et al., 2003*).

Voiding phase

Voiding begins when a voluntary signal is sent from the brain to begin urination, and continues until the bladder is empty (*Blok et al., 1994*).

Bladder afferent signals ascend the spinal cord to the periaqueductal gray, where they project both to the pontine micturition center and to the cerebrum (*Blok et al., 1994*). At a certain level of afferent activity, the conscious urge to void becomes difficult to ignore. Once the voluntary signal to begin voiding has been issued, neurons in pontine micturition center fire maximally, causing excitation of sacral preganglionic neurons. The firing of these neurons causes the wall of the bladder to contract; as a result, a sudden, sharp rise in intravesical pressure occurs. The pontine micturition center also causes inhibition of Onuf's nucleus, resulting in relaxation of the external urinary sphincter. When the external urinary sphincter is relaxed urine flows from the urinary bladder when the pressure there is great enough to force urine to flow through the urethra. The micturition reflex normally produces a series of contractions of the urinary bladder (*Sie et al., 2001*).

Voluntary Control:

When urination must be delayed, the individual inhibits the micturition reflex by contracting the external sphincter, which is under voluntary control. When the opportunity to urinate becomes available, the individual voluntarily contracts the abdominal muscles, which raises the intra-abdominal pressure and assists in expelling urine from the bladder (*Guyton et al., 2000*).

The act of micturition involves a complex coordination of neural and muscular responses leading to:

1. Relaxation of the internal sphincters of the urethra under sympathetic control and of the external sphincter, under somatic control.
 2. Constriction of the sphincters of the two ureters under sympathetic control to prevent urine retrograde flow to the kidney.
 3. Contraction of the detrusor muscle under parasympathetic control.
- (Guyton et al., 2000)*

Physiologic Requirements for Continence: (*Timiras et al., 2007*).

Motivation to be continent

Adequate cognitive function



Adequate mobility

Normal lower urinary tract function

No involuntary bladder contractions

Appropriate bladder sensation

Closed bladder outlet

Low-pressure accommodation of urine

Normal bladder contraction

Lack of anatomic obstruction

Coordinated sphincter relaxation and bladder contraction

Absence of environmental or iatrogenic barriers

Age-Related Changes in the Urinary Tract

Several changes occur with aging that may affect a person's ability to control urination. With aging, the bladder capacity tends to decline from 400-600 in adults to 250 in elderly due to decrease in bladder elasticity results from atrophy and stiffness of the bladder muscle fibers. Also, the ability to postpone urination after feeling the need to urinate may decrease (*Mold, 1996*). Deterioration of sensory tracts between bladder, spinal cord and brain leading to abnormal bladder sensation of the urge to void at small bladder volumes (*Lekan-Rutledge, 2004*). In addition, the residual urine volume increases with aging due to decrease in the strength of the