Pathophysiological Rationale for Surgical Treatments of Stress Urinary Incontinence

Urology Grand Rounds
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Herman Christopher Kwan R4

A familiar case?
- 62 year old female – initial presentation urinary incontinence 1999
- Cystoscopy, UDS
- Urologist diagnosed “SUI”
- After few months conservative therapy, a surgical procedure was performed successfully
- She returns w/ sx’s urinary incontinence 2005

Surgical treatments of SUI
- 29.2% are re-operations
  (Olson, O&G 1997)

Why did her incontinence recur?
- Patient factors
  - change in pt's status
  - age, additional injury
- Procedural factors
  - Wrong material
  - Procedure prone to failure
- Surgeon factors
  - incorrect diagnosis
  - treatment did not address the pathophysiology of her SUI

objectives
- We have a poor understanding of the pathophysiology of SUI…it is still evolving
- Introduce single factor theories for SUI
- Gain a more holistic view of SUI
  - Pathophysiology of SUI is multifactorial
  - Introduce concepts of “shear force” & “continence threshold”
- Introduce the “Trampoline Theory” of female continence

“I think we’re tooting our own horn”
Dr. Christopher Payne  NWUS meeting 2004
- High cure rates reported for SUI
- Considering only symptom of SUI
- Voiding dysfunction, post-op enteroceles or prolapse, detrusor instability
  - Reported in 10-25% of pts operated on for SUI
“I think we’re tooting our own horn”

Dr. Christopher Payne  NWUS meeting 2004

- therefore many pts classified as surgical success continue to have significant problems despite alleviation of their stress incontinence

Current therapy for SUI is empiric!

One’s urologist’s ideal pt. for pubo-vaginal sling is another’s ideal TVT or another’s Burch

UI prevalence/incidence

- Flow between continent and incontinent groups over time (n=382)
  - Samuelsson, AM J O & G ’2000

SUI is most common type in women

Pathophysiology of SUI & Treatment Options

- existing treatment modalities based on existing understanding of pathophysiology of SUI
  - Not well understood!!
Pathophysiology of SUI & Treatment Options

- Deficiencies in understanding of pathophysiology lead to failure of therapies
- “Can’t fix it if we don’t know where it is broken”
  
  Omar Nazif P. Eng.

Physiology of Continence

- Central control mechanisms
- Peripheral control mechanisms

Peripheral control mechanisms

- Lower Urinary Tract Structures
  - Subject of most investigation
  - Basis of most treatment modalities of SUI in women
  - (Delancy O&G ’86)

Ligaments and fasica of pelvis is actually one continuous sheet of levator fascia and not surgically distinct

Endopelvic fascia

Levators

Pubocervical fascia
  - periurethral fascia or urethropelvic ligament
  - perivesical fascia or vesicopelvic ligament
  - sacrouterine/cardinal ligaments
Factors for SUI

- Bladder descensus
- Hypermobility
- ISD
- Fixed, open bladder neck
- Rotational descent
- “pipestem” urethra
- Hammock hypothesis
- Mid-urethral continence zone

Single factor theories on pathophysiology of SUI

- Position of proximal urethra¹
- Intrinsic sphincter deficiency²
- Hammock hypothesis³
- Integral theory⁴

1. Enhorning Urol Int. 1976
2. McGuire 1993

Urethral Position Theory

*Goran Enhorning 1961*

- Bladder, urethral pressure measurements in 250 women: normal, postpartum, and SUI
  - During stress, increase in urethral pressure was not as great in SUI compared to normal subjects

**Hypothesis:** absence of additional pressure during stress is due to extra-abdominal position of urethra
Urethral position theory

- Urethra needs to be above pelvic floor
- With stress, there would be pressure transmission to bladder and proximal urethra

Normally continent women.
- Well supported proximal urethra
- Retropubic, intraabdominal position
- Does not change position with stress (i.e., NOT HYPERMOBILE)

Normally continent women. Well supported proximal urethra. Retropubic, intraabdominal position. Does not change position with stress (i.e., NOT HYPERMOBILE).

Forms basis for retropubic suspensions.

MMK 1st reported successful RP approach for SUI 1949.

Modified by Burch in 1961.
- Pereyra
- Stamey
- Gittes
- Raz

Why is the urethral position theory incorrect?

“No correlation b/t intraabdominal position of bladder neck and SUI.” Constantinou et al. 1981

- Hypermobility is equally prevalent in continent and incontinent females
- Women w/ large cystourethroceles often continent

Recorded urethral pressure profiles and transmission of abdominal pressures to urethra in:
1. continent women,
2. stress incontinent
3. Stress incontinent after stamey needle suspension
In UPP demonstrating maximal pressure transmission at level of mid-urethra in normally continent women.

Constantinou’s conclusions

- 1. pt of maximal urethral pressure transmission is in mid urethra (almost always below pubis)
- 2. after needle suspension, mid urethral hump is re-established
- 3. a reflex muscular contraction augments the pressure transmission as % transmission is >100%
  - Rise in urethral pressure PRECEDED rise intravesical pressure during a cough
  - active contraction of midurethra through a neuromuscular reflex

Invalidity of intra-abdominal pressure theory

“little advance in surgery of SUI over past 20yrs from uncritical acceptance of intra-abdominal pressure transmission theory”

ISD/Open bladder neck

- 1980s, McGuire introduced concept of ISD
  - theory based on urodynamic observations of women who failed anti-incontinence surgery
  - intrinsic problem (anatomical or physiological) from multiple surgeries in urethral area
  - classically described as SUI w/ non-mobile retropubically fixed urethra Type III

The Effects of Sacral Denervation on Bladder and Urethral Function

- No activity of pudendal nerve and still retain normal urethral pressure profile
- “urethral SM function alone provides satisfactory urinary continence”

Clinical Assessment of Urethral Sphincter Function

- Table 3: Abdominal pressures required to induce leakage
Conclusions

- Maximal Urethral Closing Pressure (MUCP) does NOT correlate with severity of incontinence and thus sphincter function.

- Abdominal pressure required to induce leakage predicts severity of incontinence (Low ALPP...larger amount of incontinence).

ISD/Open bladder neck

- Urethra unable to generate enough outlet resistance to retain urine in bladder. Lack of ‘washer’

- He believed this problem of urethral coaptation could be improved with:
  - Slings “McGuire Sling”
  - Collagen injection

ISD/Open bladder neck

- Important because it divided females with SUI into:
  - SUI from hypermobility: ↑ VLPP
  - SUI from ISD: ↓ VLPP

ISD/Open bladder neck

- VLPP today: technique not standardized
  - “Still is a useful test and clinical utility is proven” Bump ’97
  - Largely replaced UPP, which are falling out of favor
Anatomical studies of fresh and embalmed cadavers

- Urethra lies on supportive layer: -endopelvic fascia -anterior vaginal wall

Am J Obstet Gynec 1994

Tissues below bladder neck and proximal urethra provide strong backboard to allow occlusion of urethra during increased abdominal pressure, thus preventing SUI

Hammock Hypothesis

John DeLancy 1994

Urethral support is not only factor involved in stress incontinence

- Acknowledged Constantinous observation of a possible NM reflex contributing to ↑ urethral pressure
- Greatest ↑ pressure in mid-urethra
  - Abdo pressure transmitted mostly to prox urethra

Hammock Hypothesis

John DeLancy 1994

This layer gains structural stability through its lateral attachment to

- arcus tendineus fascia
- levator ani muscle

Hammock Hypothesis

John DeLancy 1994

Explains why hypermobile urethras can still be continent

Integral Theory

Ulmsten 1992
Integral Theory
Ulmsten 1992

- Integral theory
  - suggests pelvic muscles play a more active role in maintaining continence (vs suburethral hammock)
  - Tries to explain etiology of urge incontinence in SUI

- “stress and urge symptoms arise from same anatomical defect…”
  - Lax anterior vaginal wall
    - Activation of stretch receptors in bladder neck and proximal urethra...triggers inappropriate micturition reflex...urgency, frequency
    - Does not allow efficient transmission closure pressure by 3 separate closure mechanism

Integral Theory
Ulmsten 1992

- TVT re-establishes integrity of pubo-urethral ligaments

Putting it all together...

- Hypermobility/Rotational descent
- ISD
- Suburethral support
- Integrity of configuration
  - introduce concept of
    - “incontinence threshold” and “shear force”

Incontinence threshold

- Pubourethral ligaments tether anterior portion of urethra
- Kinetic energy of anterior vaginal wall pulls the posterior urethral wall away

- Shear forces pull's bladder neck open depending on...
  - Configuration of urethra
  - Intrinsic sphincter function
  - Transmission of pressure
  - Integrity of configuration
Pathophysiology of SUI
Conclusions

- SUI is NOT a single factor disease but a spectrum of complex anatomical and physiological disorders based on central and peripheral control mechanisms

- Physiology of continence in women is MULTIFACTORIAL, so is the pathophysiology of SUI

Pathophysiology of SUI
Conclusions

- a shearing effect occurs at continence threshold when:
  - Urethral walls are separated (position/hammock)
  - Urethral washer is damaged (ISD)
  - Support structure is damaged (hammock, PUL)

Pathophysiology of SUI
Conclusions

- “Continence Threshold”
  - Incontinence occurs when this “threshold” is overwhelmed

Pathophysiology of SUI
Conclusions

- We need to adapt a more holistic view of SUI
  - The “Trampoline Theory”

The Trampoline Theory of Female Continence

- **Predispose** (gender, race, anatomic, neurologic, collagen)
- **Incite** (childbirth, radiation, nerve damage, surgery)
- **Promote** (constipation, occupation, smoking, COPD, obesity, infection, meds)
- **Decompensate** (aging, dementia, decreased mobility)