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A hypothesis explaining weight loss cure of obesity-linked female urinary incontinence

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ABSTRACT

Objectives: The vagina has an important role in transmission of 3 opposite striated pelvic muscle forces acting against pelvic ligaments to close urethra distally and at bladder neck. The same muscle forces stretch vagina to support the urothelial stretch receptors to prevent them activating the micturition reflex at low bladder volumes, and to open the urethra prior to micturition. Striated muscles have a limited contractile force. The hypothesis the weight of the intraabdominal contents on the vaginal membrane adds additional weight for the striated pelvic muscles to move when they tension the vagina to close the urethra for continence, open it for evacuation and prevent urothelial stretch receptors from activating micturition prematurely (interpreted as urgency).

Materials and Methods: To test if a binary musculo-elastic model of bladder control can explain significant cure/improvement of lower urinary tract symptoms following weight loss after bariatric surgery.

Results: The results for improvement of stress urinary incontinence (SUI), overactive bladder syndrome (OAB) and mixed symptoms were consistent with the concept of an extra intraabdominal intestinal load on the vagina membrane which prevented the 3 opposite striated muscle forces stretching the vagina sufficiently to close the urethra (SUI) and/or prevent excessive afferent impulses from the urothelial stretch receptors activating micturition prematurely (OAB).

Conclusion: Removing the burden of intestinal contents on the vagina restored several different areas of incontinence, SUI, mixed incontinence, urgency (OAB) seemingly validates the hypothesis.

Keywords: Urinary incontinence; integral theory; weight loss; obesity; bariatric surgery, damaged ligaments

INTRODUCTION

In 2006, O'Boyle et al.[1] tracked urinary symptoms in 240 female patients who lost weight following bariatric surgery. The results were quite remarkable, and are quoted directly:[1] "The prevalence of urinary incontinence preoperatively was 45% (108). Eighty-two (76%) completed urinary function questionnaires pre-operatively and post-operatively. Fifty-seven (70%) underwent laparoscopic gastric bypass, twenty-four (29%) underwent sleeve

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gastrectomy and one underwent a banding procedure. Thirty-one (38%) reported leaking on sneezing or coughing-stress urinary incontinence (SUI). Thirteen (16%) complained of leaking before reaching the toilet-overactive bladder syndrome (OAB). The remaining thirty-eight (46%) reported mixed symptoms. The mean pre-operative weight and body mass index (BMI) were 133 (18) kg and 50 [standard deviation (SD) =6.2] kg/m respectively. The mean post-operative BMI drop was 16 (SD =5.2) kg/m(^{2}). Preoperatively, 61 (75%) reported moderate to very severe urinary incontinence compared to 30 (37%) post-operatively. Twenty-seven (33%) patients reported complete resolution of their urinary incontinence. Fifty-one (62%) patients required incontinence pads on a daily basis pre-operatively, compared to 35 (43%) post-operatively. The mean international consultation on incontinence questionnaire-urinary incontinence short form (ICIQ-UI SF) score was 9.3 (SD =4.4) pre-operatively compared to 4.9 (SD =5.3) post-operatively (t=7.2, p=0.000). The improvement score post-operatively was 8 (SD =3). A significant difference in the ICIQ-UI SF was identified between OAB and SUI groups when adjusting for age, number of children, type of delivery and pre-op BMI."

Hypothesis for Obesity Causation of Incontinence and Cure by Weight Loss

With reference to Figure 1, the increased weight of the organs pressing on the bladder and vagina is an additional load for the opposite muscle forces which stretch the vagina to perform their natural reflex functions of urethral closure during effort; to support the stretch receptors "N" to control urgency; to externally open the urethra prior to micturition by lowering resistance, thus facilitating urine flow. The hypothesis is based on the Integral Theory of Female Urinary Incontinence (IT), which states that control of bladder function is binary, and not from the bladder itself, but from structures outside it, the vagina, its supporting ligaments, and pelvic muscles.[2]

MATERIALS AND METHODS

To test if a binary musculo-elastic model of bladder control, Figure 1, can explain cure/improvement of lower urinary tract symptoms following bariatric surgery.

Normal Binary Control of the Bladder

The vagina has an essential role in all bladder functions.[2] With reference to Figure 1, under reflex binary cortical control, the 3 directional muscle forces (large arrows) contract against the pubourethral ligaments anteriorly and the uterosacral ligaments (USL) posteriorly to stretch the vagina to enact 3 functions: To close the urethra during effort; to stretch the vagina in opposite directions to prevent activation of the micturition reflex at low volume (OAB);[2,3] to open it prior to micturition.

A Hypothesized Anatomical Pathway for Obesity-induced Incontinence

The pathway is graphically indicated in Figure 1. Essential to explaining the hypothesis, is the well-known fact that striated muscles (large arrows, Figure 1) have a finite contractile force.4 The additional intraabdominal weight of fat-laden organs is an extra weight added to the contractile force required for the opposite muscle forces pubococcygeus muscle, levator plate/conjoint longitudinal muscle of the anus to stretch the vagina to perform the 3 functions of the musculo-elastic control complex. In consequence, all 3 functions of the musculo-elastic control complex, Figure 1, weaken, and can be expressed clinically as described, stress urinary incontinence (SUI), "mixed incontinence", OAB (1). According to hypothesis predictions, these same patients1 would have symptoms of inability to empty adequately "UAB" ("underactive bladder"), Figure 1.

DISCUSSION

The pre-operative and post-operative results[1] are consistent with the predictions of the hypothesis, that weight loss would relieve some of the burden on the contractile force of the striated pelvic muscles, thus allowing them to perform their natural control functions unhindered. Pre-operatively, the symptom incidence was: SUI (38%), OAB (16%), mixed symptoms (46%). Post-operatively 33% of these patients reported complete resolution of their urinary incontinence, and daily pad use reduced from 62% to 43%, With reference to Figure 1, the anatomical pathway to the described cure,[1] is the reduction of the added load on the striated muscles, allows the muscles to close and open the urethra normally.

Urinary incontinence is one of many co-morbidities associated with the intraabdominal pressure (IAP) from obesity. Sugerman[5] reported increased IAP increases pleural pressure, cardiac filling pressures, femoral venous pressure, renal venous pressure, systemic blood pressure, and vascular resistance, renin and aldosterone levels, and intracranial pressure. Varela et al.[6] reported systemic hypertension, type 2 diabetes mellitus, gastroesophageal reflux disease, urinary stress incontinence, lower extremity edema, obstructive sleep apnea, and abdominal wall hernia. Jordan and Tincello[7] stated that weight loss has a profound effect upon urinary, anal, and prolapse symptoms: achieving a target weight loss of 5-10% of baseline body weight is associated with 30-70% cure of urinary leakage, around 65% improvement of anal symptoms, and 75% resolution of prolapse symptoms. They recommended that weight loss should

be recommended as first-line intervention for obese incontinent women. Surgery for incontinence carries a slightly lower cure rate in obese women but no higher risk of complications.

The Hypothesis and the Integral Theory

This is the first time the IT has been able to explain how restoration of the muscle forces themselves can restore normal bladder function, and therefore continence.

The hypothesis itself, and the anatomical analysis, both flow from the IT which is summarized by Figure 1. The anatomical analysis is an example of Karl Popper' dictum, that a universal theory (for example, the IT), must be simply stated, falsifiable and predictive.[8] In all surgical studies to date, surgical cure has been based on native ligament repair[9] or harnessing the wound reaction of a precisely implanted tape to create new collagen to repair weak collagen-deficient ligaments[10] against which the

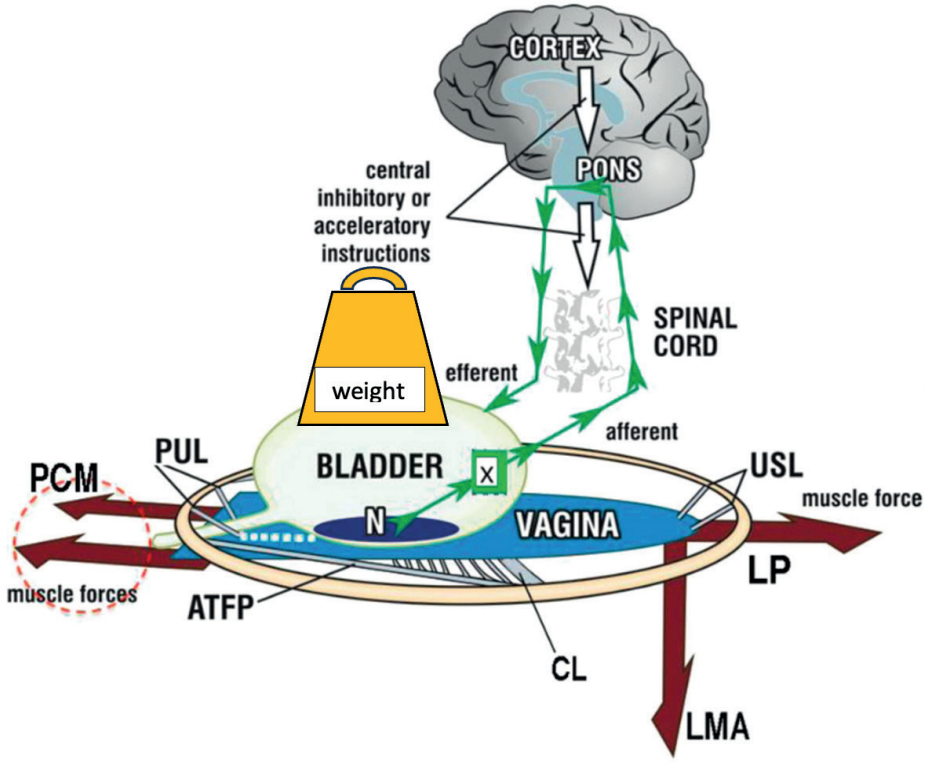

Figure 1. Binary model for bladder control by 2 opposing reflexes, EITHER closure OR micturition. Schematic 3D sagittal view, System in normal closed mode. PCM=pubococcygeus muscle; LP=levator plate; LMA=conjoint longitudinal muscle of the anus; PUL=pubourethral ligaments; USL=uterosacral ligaments; N=urothelial stretch receptors; CX=cervix; CL=cardinal ligament; ATFP=arcus tendineus fascia pelvis.

Control of SUI: PCM stretches distal vagina forwards to close distal urethra from behind; LP/LMA stretch proximal vagina and bladder base back and around PUL to close bladder neck.

Cortical control of OAB: Afferent impulses "X" from stretch receptors "N" are reflexly suppressed cortically (white arrows). Efferent impulses prevent coalescence of micromotions which are necessary for smooth muscle contraction during micturition.

Peripheral control of OAB: Is by a musculo-elastic mechanism which responds to cortical efferents (small arrows) to stretch vagina in opposite directions to support "N" and decrease afferent impulses "X". The three directional muscles (large arrows), forward, (PCM), backward (LP), and downward, (LMA) contract against the supporting ligaments, PUL and USL, to stretch vagina tightly, much like the membrane of a drum. The stretched vagina supports the urine column, preventing activation of the stretch receptors "N", decreasing afferent impulses to the cortex.

Micturition: The closure reflex is shut down and the micturition reflex is activated. Central control (white arrows) relaxes, as does PCM (broken circle); PCM relaxation allows the posterior muscles LP and LMA to unrestrictedly open out the posterior wall of urethra (white broken lines below urethra) just before bladder evacuation by detrusor contraction.

Dysfunction: PCM, LP, LMA contract against PUL and USL. As they are striated muscles, they require a firm anchoring point to contract against. If the ligament is weak, the contractile force of the muscles weakens, and inhibits their function (4): the muscles cannot mechanically close urethra (causing stress incontinence), cannot open it (causing obstructed micturition) or they cannot stretch the vagina sufficiently to support the urothelial stretch receptors "N"; these may fire off excess afferent impulses to activate the micturition reflex, experienced as "urge to go" ("OAB").

Obesity: The extra weight of the abdominal organs weighs down on the bladder and vagina. The opposite forces are not sufficiently strong to stretch the vagina to close the urethra or to support "N", so control of all the above functions, SUI, urge, emptying is weakened. Improvement in all the foregoing parameters following bariatric surgery (1) provides support for this anatomical explanation

pelvic muscles (large arrows, Figure 1) contracted. However, as is evident from,[11] no surgery was performed on the ligaments, so only restoration of contractile strength to the muscles could prevent the symptom cure reported.[1] Removing the burden of intestinal contents on the vagina restored several different areas of incontinence, SUI, mixed incontinence, urgency (OAB) seemingly validates the hypothesis, and the underlying Integral Theory itself.

CONCLUSION

Loss of weight, either by diet or by bariatric surgery to lower caloric intake is a well documented treatment for urinary and even bowel incontinence. The vagina has an important role in closure and opening of the urethra by the pelvic muscles. The hypothesis of additional weight of the intrabdominal contents imposing an additional contractile force to stretch the vagina seems to explain the relief of SUI and urge reported by several investigators. It also supports the statement that control of the bladder is from outside the bladder from the vagina, muscles and ligaments.[2] The hypothesis predicts significant cure of emptying symptoms also, which would be a further test of the hypothesis.

DISCLOSURES

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