Commentary

The risks of reproduction are borne exclusively by females, who face the hazards of childbirth that arise from the "human obstetrical dilemma:" a narrow "hourglass" pelvis whose size and shape are constrained by the requirements of upright bipedal locomotion together with big babies who develop big brains as a result of our species' progressive encephalization[1]. These conflicting evolutionary forces mean that humans are predisposed to obstructed labor. When that obstruction is not relieved by timely intervention (assisted delivery, often by cesarean section), the consequences can be catastrophic. Large areas of soft tissue which line the boney pelvis may be injured or destroyed by pressure necrosis from the prolonged impaction of the fetal head, and the fistulas that result are often breathtaking in size and complexity. As a result, rarely are the tissues around an obstetric fistula completely healthy. Although the neighboring tissues are still living, often they have been severely injured, losing both vitality and elasticity. Sometimes the fistula is so deeply embedded in dense scar tissue that extensive dissection is needed even to locate the opening prior to any consideration of repair.

The "continence gap" that persists in patients whose fistulas have been closed successfully but who remain incontinent (transurethrally) after surgery has been a persistent puzzle[2]. For a long time this post-fistula incontinence was thought to be stress incontinence from severe urethral damage (so-called "Type III" stress incontinence), but urodynamic studies have demonstrated multiple intermingled pathophysiological processes in these patients[3]. Whatever it is, it is clearly not "simple" stress incontinence, but exactly how to describe it, how it originates, and (most importantly) how it should be treated, have all been elusive.

In this issue, Browning, Williams and Petros advance an idea – supported by intriguing preliminary clinical data – to suggest that one of the main culprits in the "continence gap" is tethering of the vagina occasioned by the scarring produced by the pressure effects of obstructed labor. Those familiar with the barely-mobile anterior vagina that may be found in many fistula patients will understand the underlying logic of their argument. The same phenomenon of vaginal tethering may explain the profound stress incontinence that develops in some women with post-hysterectomy vaginal vault prolapse, who, although continent before surgery, may develop debilitating stress incontinence after undergoing sacral-colpopexy. The pathphysiology that produces urine leakage in these cases presumably is also "tethering" of the anterior vaginal wall through excessive suspensory tension which alters the dynamics of the urethral closing mechanism in a similar way as that proposed here. In this case the incontinence is not "unmasked" by reduction of the prolapse; rather, it is created by altering the normal physiologic mechanisms of urethral closure. Further clinical research to verify this hypothesis is greatly to be desired.

REFERENCES

1. Wittman AB, Wall LL. The evolutionary origins of obstructed labor: Bipedalism, encephalization, and the human obstetric dilemma. Obstetrical and Gynecological Survey 2007; 62 (11): 739-748, November.
2. Wall LL, Arrowsmith SD. The "Continence Gap:" A critical concept in obstetric fistula repair. International Urogynecology Journal 2007; 18 (8): 843-844, August. Published online: DOI 10.1007/s00192-007-0367-z.
3. Goh JTW, Krause H, Tessema AB, and Abreha G; Urinary symptom and urodynamics following obstetric genitourinary fistula repair; International Urogynecology Journal 2013; 24: 947-951.

L. LEWIS WALL, MD, DPhil
Department of Anthropology, Department of Obstetrics & Gynecology
Washington University in St. Louis, USA
walll@wudosis.wustl.edu