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Cystoscopy at the time of benign hysterectomy: a decision analysis

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TO THE EDITORS: We were excited to read your timely and important publication "Cystoscopy at the time of benign hysterectomy: a decision analysis"[1] because safety in and quality of gynecologic surgery is a major focus. We wish to express several concerns regarding the methods of this study and possible implications for its interpretation and conclusions.

First, we suggest the outcomes used within the authors' models are incomplete. In the paper, Cadish et al[1] account for only 90-day outcomes of diagnostic testing, treatment, and readmission for treatment without fully considering other documented infectious and systemic sequelae. Recently, Blackwell et al[2] demonstrated the profound and far-reaching consequences of a delayed ureteral injury, with includes increased odds of rehospitalization, kidney injury, sepsis, and death. A robust model of lower urinary tract injury at the time of hysterectomy should include these substantive complications to ensure appropriate cost estimates of delayed injury recognition.

Second, although we agree that the use of Medicare fee schedules represents an appropriate cost basis, our concern is with the use of these data. The reported costs appear to include only the professional component of each procedure.[1] In our experience, it is customary also to include facility, anesthesia, diagnostic interpretation, and hospitalization fees where applicable. Exclusion of these fees, which are published by the Centers for Medicare and Medicaid Services,[3,4] systematically underestimates all costs that are used within the proposed model. For example, the authors used a cost of $858.41 for laparoscopic hysterectomy (CPT 58571). When accounting for the facility fee of an ambulatory surgical center and the anesthesia fee for a presumed 2-hour case in a low-risk patient, we calculate a cost estimate of $8198.66.[3] Appropriate tabulation of these costs may change conclusions significantly regarding the marginal cost of cystoscopy and the reported threshold analysis findings.

Third, we question the assumptions used in the model of "selective" cystoscopy. The authors describe performing cystoscopy in "the group with above-average risk" but provide no further enumeration of the probability of cystoscopy or risk calculation/distribution.

Given our concerns regarding the assumptions and cost calculations in this analysis, we caution against the use of this study's conclusions to drive decision-making regarding cystoscopy at the time of hysterectomy and will continue to support its universal implementation at our institution while awaiting further research that will address this important topic. ■

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REPLY

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We agree that delayed ureteral injury causes significant morbidity after hysterectomy. We intentionally modeled hysterectomy modalities separately, accounting for increased thermal injury with laparoscopic or robotic approaches. Blackwell et al[1] published their study of delayed ureteral injury sequelae after our analysis was complete, but even so, delayed injury rarely is diagnosed beyond the 90-day postoperative period, even when

time to definitive management extends beyond >90 days. Our model included any injury diagnosed within 90 days, even if treatment extended further. Selli et al[2] reported delayed ureteral injuries with laparoscopic gynecologic procedures, all of which were diagnosed 15−20 days postoperatively; Morrow et al[3] reported a median time to diagnosis of 16 days after intraoperatively unrecognized injury. Those diagnosed at >90 days comprise such a minute proportion that they would likely not impact outcomes.

Although it is customary to include facility, anesthesia, and hospitalization fees in total costs, it is acceptable to exclude costs that are common between strategies. We believe that differential costs by strategy, which includes the cost of injury diagnosis/treatment and additional hospital days when prolonged stay was required, were appropriately modelled.

The letter accurately notes that we did not thoroughly describe our methods for the risk required for selective cystoscopy. Indeed, we struggled with the level of detail for the article. We used a "suspicion multiplier" that was arbitrarily set initially to 2. In a hypothetical population with 10% injury rate, the high-risk group has injury rate 10%×2=20%, and the low-risk group has 10%/2=5%. However, with equal groups, this averages to 12.5%, not 10%, which necessitates adjustment in group proportions. The high-risk group proportion is 1/(1+suspicion multiplier), and the rest are low-risk. Different values for the suspicion multiplier that was assessed in the sensitivity analyses did not impact conclusions. As the suspicion multiplier increases, there are fewer people in the high-risk group, which we feel mirrors clinical practice.

We omitted this lengthy description from the article, fearing it might obscure our message that any suspicion of injury is sufficient to prompt intraoperative cystoscopy. The threshold for selective cystoscopy should be low, and selective cystoscopy even at a low threshold may be cost-saving.

The letter's authors, like us, are subspecialists in female pelvic medicine and reconstructive surgery and favor routine cystoscopy. We carry a similar bias, because it is the standard of care for pelvic floor disorder procedures. However, our results show that selective cystoscopy with a low threshold was the better alternative for benign hysterectomy without concomitant procedures. ■

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B.M.R. has been a consultant for Coloplast, Inc. and has provided legal expertise for Ethicon, Inc. The remaining authors report no conflict of interest.

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Uterine and fetal placental Doppler indices are associated with maternal cardiovascular function

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TO THE EDITORS: In their recent paper, Tay et al[1] assert that uterine and fetal placental Doppler indices are significantly associated with measures of maternal cardiovascular function, and go on to state that classic descriptions of uterine and fetal Doppler changes being initiated by placental maldevelopment are a "less plausible explanation" for the pathogenesis of preeclampsia and fetal growth restriction (FGR) than are maternal cardiovascular changes.[1] This conclusion is not supported by the findings described in the paper. Furthermore, their hypothesis fails when applied to pregnancies characterized by high rates of preeclampsia, such as molar pregnancy or trisomy 13, or the case of twin pregnancies with discordance for fetal growth restriction and preeclampsia. We[2] and others[3] have described cases where selective feticide of the FGR-affected twin has led to resolution of preeclampsia in a short timeframe. The uterine environment and cardiovascular performance for both twins were clearly identical, yet elimination of the putative morbid placenta resolved the clinical syndrome.[2,3] Another example along the same lines, hinting at the importance of the fetus in the development of preeclampsia, comes from a randomized controlled trial published just recently in *The Lancet*,[4] which described higher rates of preeclampsia in pregnancies achieved with