1. The third paragraph of the introduction states, “Some women undergoing prolapse repair may have a relatively asymptomatic rectocele and may experience defecatory dysfunction.” Please clarify this statement since many providers feel that if a patient has defecatory dysfunction and a rectocele it cannot be asymptomatic by definition.

2. The study design is a retrospective cohort comparison between all patients who underwent surgery for apical/anterior pelvic organ prolapse using different approaches, and compared those patients who received a traditional native tissue rectocele repair, perineorrhaphy or perineoplasty to those who did not. Clearly, there is potential of surgeon selection bias in who is offered a posterior repair. This bias was demonstrated in Table 1 where the reason more women that received a posterior repair had worse prolapse of the posterior vaginal wall (Bp -1.0) but better apical support (C -4.0) compared to women who did not receive a posterior repair who had better posterior vaginal wall support (Bp -2.0) but worse apical prolapse (C -2.0). If the authors wanted to control for baseline risk factors, this would lead some to believe that POPQ point Bp (and even potentially POPQ point C despite lack of significance) should be included in the regression model rather than or in addition to overall POPQ stage. Why did the authors not include this more specific point in the model? Should they have?

3. The author’s second statement in the discussion is “those who underwent a PR had a significantly greater margin of improvement in bowel symptoms compared to those who did not undergo a PR, and these differences persisted when controlling for potentially confounding factors in a regression analysis.” In fact, this regression score is most reflective of the comparison after controlling for risk. However, do you the authors believe that a predicted regression model score of 4.9 CRADI-8 points better in the women who underwent PR is a clinically
meaningful difference compared to those who did not receive a PR? If this is indeed unknown as mentioned in the discussion do the authors feel they can conclude that those who receive a PR have greater improvement with reasonable certainty?

4. Can the authors speculate as to why symptoms may be different but quality of life does not appear to be different between those who receive a PR compared to those who do not? Could it be that posterior repair actually results in more patients whose quality of life gets worse or is less likely to improve as much despite symptoms improving?

5. The authors end the discussion with the statement that one option is to correct a rectocele if a patient has symptoms but they don’t describe what qualifies as a surgically correctable rectocele by anatomy. They also don’t completely describe what qualifies as defecatory dysfunction. Can you elaborate on these important details to guide surgeons in the potential selection? Are there other options that should be described based on the findings?