

with a scarred uterus” is our acknowledgement that association and causation are separate and that all statistics must be interpreted within clinical practice. Furthermore, this statement reflects provider differences in the management of women with and without a uterine scar, because many obstetric providers minimize the use of labor induction and oxytocin augmentation in the setting of a previous uterine scar. The use of oxytocin may increase the risk for primary uterine rupture, because it is a known risk factor for rupture of the scarred uterus.²

Ms Cohain references Tversky and Kahneman’s “Belief in the Law of Small Numbers” to question our results and interpretation.³ We acknowledge the (thankfully) few primary uterine rupture cases as a study limitation. Tversky and Kahneman caution the observer from making assumptions from small numbers (although they never define what constitutes a “small number”—in their paper, 20 subjects is seen as a reasonable sample size). As physician scientists, however, we must make do with clinical realities.

In conclusion, we urge all clinicians providing obstetric care to women to recognize that risk factors and associations are powerful tools but they are not absolutes. Although extremely rare, devastating obstetric outcomes such as primary uterine rupture (even in the labor absence of induction or augmentation) may occur in any gravida, with potentially catastrophic morbidity and mortality.

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REPLY

We appreciate Ms Cohain’s interest in our manuscript and concerns regarding the association between oxytocin use and uterine rupture.¹ Unfortunately, oxytocin use was only one obstetric covariate and not our main focus. In addition, the facts presented in Ms Cohain’s letter are incorrect; 4 women with primary uterine rupture were neither induced nor received oxytocin augmentation. The claim that “the unscarred uterus that is not artificially forced to contract, will not contract so hard as to explode itself” should not, as the majority of situations in medicine and in obstetrics, be considered absolute. We speculate that underlying undiagnosed connective tissue aberrations or genetic factors may have influenced the development of uterine rupture in these 4 women. Unfortunately, our study design precludes an investigation of causation.

The incidence of oxytocin use was greater in unscarred cases (80% vs 37%, odds ratio 6.7, $P < .001$). This statistic shows an association between oxytocin use in unscarred uterine ruptures compared with scarred uterine ruptures but does not prove causation. Our comment that the increased rate of oxytocin use in primary uterine rupture cases “likely reflect differences in provider management among women