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## Abstract and Introduction

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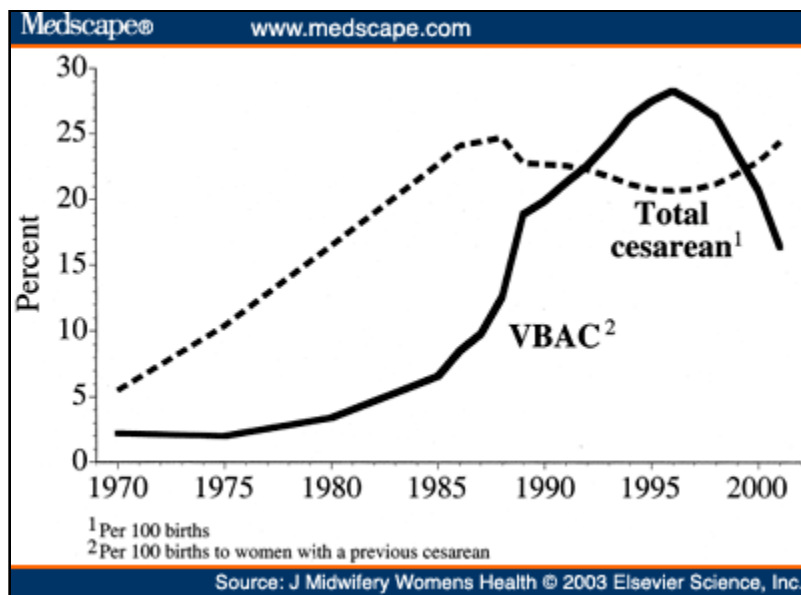
### Abstract

For the woman with a prior uterine scar, neither repeat elective cesarean birth nor vaginal birth after cesarean birth (VBAC) trial of labor (TOL) is risk-free. When VBAC-TOL is successful, it is associated with less morbidity than repeat cesarean birth. However, when VBAC-TOL fails due to uterine rupture, severe consequences often ensue. The challenge for clinicians today is to provide women who desire TOL after cesarean birth, a more individualized risk assessment of uterine rupture, thereby enhancing success and optimizing outcome. This article examines major risk factors for uterine rupture during VBAC-TOL. In addition, fetal response to uterine rupture and neonatal outcomes are reviewed.

### Introduction

In 1980, the U.S. National Institute of Child Health and Human Development (NICHD) cosponsored a conference on cesarean childbirth with the National Center for Health Care Technology and concluded that vaginal birth after cesarean section (VBAC) was an appropriate option by which to decrease the increasing cesarean section rates.<sup>[1]</sup> Widespread interest in VBAC trial of labor (TOL) ensued, with clinical research demonstrating its relative safety.<sup>[2-5]</sup> Both obstetric care providers and women desiring an alternative to cesarean birth, as well as government and private insurance company payers, enthusiastically embraced VBAC-TOL. As a result, the number of women who had a successful VBAC in the United States increased dramatically from 3.4 per 100 women in 1980 to a peak rate of 28.3 per 100 women in 1996.<sup>[6, 7]</sup>

In recent years, however, renewed controversy about the relative safety of VBAC-TOL has resulted in a rapid decline in the number of women who experience VBAC, falling from 28.3 per 100 women in 1996 to 16.4 per 100 in 2001, a 42% decrease (Figure 1).<sup>[8]</sup> Attention has focused primarily on symptomatic uterine rupture, a potentially catastrophic event, which can have serious consequences to mother and fetus. Although the overall estimated rate of uterine rupture is less than 1%, the incidence varies significantly depending on the presence of specific risk factors.<sup>[9-12]</sup> Concerns related to uterine rupture have prompted the American College of Obstetricians and Gynecologists (ACOG) to recommend that a physician be "immediately available throughout active labor, capable of monitoring labor and performing an emergency cesarean delivery"<sup>[13]</sup> when women undergo VBAC-TOL.



**Figure 1.** Total cesarean and vaginal births after previous cesarean (VBAC) rate: United States, 1970 to 2001.<sup>[6-8]</sup>

Neither repeat cesarean birth nor TOL after cesarean is risk-free for women with a prior uterine scar. When VBAC-TOL is successful, it is associated with less morbidity than repeat cesarean birth.<sup>[2-5, 9, 13]</sup> However, when VBAC-TOL results in uterine rupture, neonatal death or permanent neonatal injury can occur even in facilities with immediate access to cesarean birth.<sup>[14-17]</sup>

The challenge for clinicians today is to provide women, who desire VBAC-TOL, a more individualized risk assessment of uterine rupture, thereby enhancing success and optimizing outcome. A woman and her health care provider must evaluate the following: 1) risk of complications associated with VBAC-TOL versus repeat elective cesarean birth, 2) capabilities of the birth facility, 3) personal choice, and 4) the probable success rate of VBAC-TOL. Recent research has better defined factors that influence probable success of VBAC.<sup>[18-22]</sup> This article addresses uterine rupture, the major complication that can occur during VBAC-TOL, and the subsequent fetal response and neonatal outcome when uterine rupture occurs.

A literature search was conducted in MEDLINE and COCHRANE databases using the following search terms: vaginal birth after cesarean, TOL, uterine rupture, risk factors, fetal heart rate, neonatal outcome. A Boolean search operator was used throughout (AND), and the search was performed in "all fields" mode. Only articles published in English since 1990 were included. Additional studies were identified under related articles. The studies presented are not exhaustive, but they represent those that have the greatest significance for clinical practice. Earlier published works can be found in extensive reviews by both Lavin et al.<sup>[23]</sup> and Lieberman.<sup>[24]</sup>

## Risk Factors for Uterine Rupture

Several factors complicate the evaluation of risk factors for uterine rupture during VBAC-TOL. First, uterine rupture is a relatively rare event, requiring large sample sizes to identify significant statistical associations between various risk factors and uterine rupture. Most studies reported in the literature are small, making interpretation of results difficult. Second, randomized controlled trials are scarce in the VBAC literature. The majority of studies are observational series that used a case-control or cohort study design. Third, most studies define uterine rupture as a defect that involves the entire uterine wall that is symptomatic and requires surgical intervention, whereas uterine dehiscence is defined as asymptomatic scar separation or thinning that does not require intervention. However, the

definition of uterine rupture varies slightly from study to study.

Although the overall rate of uterine rupture in women attempting VBAC-TOL is quoted to be less than 1%, women who elect a repeat cesarean birth without labor still have a uterine rupture risk of 0.03% to 0.2%.<sup>[9-12]</sup> Among those women attempting VBAC-TOL, rates of uterine rupture vary significantly, depending on associated risk factors ( Table 1 ). Characteristics in a woman's obstetric history (type of uterine scar, single-layer versus double-layer uterine closure, number of prior cesarean births, number of prior vaginal births, interdelivery interval, maternal age, maternal fever following cesarean), in addition to factors related to current labor management (induction or augmentation with prostaglandins and/or oxytocin), have been found to significantly influence uterine rupture rates during VBAC-TOL. Characteristics of questionable significance, such as macrosomia and postdate pregnancy, will not be discussed in this article; however, they are presented in an excellent review by Lieberman.<sup>[24]</sup>

**Table 1. Major Risk Factors for Uterine Rupture During VBAC-TOL**

Classical uterine incision
Single-layer uterine closure
Prior cesarean births $\geq 2$
Interdelivery interval $\leq 24$ months
Maternal age $\geq 30$ years
Maternal fever $>38^{\circ}\text{C}$ following cesarean
Induction of labor with oxytocin
Prostaglandin $\text{E}_2$ or misoprostol use

## **Risk Factors for Uterine Rupture in Obstetric or Maternal History**

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### **Type of Uterine Scar**

The risk of uterine rupture during VBAC-TOL varies on the basis of the type and location of the prior uterine scar. A prior classical uterine incision extending into the uterine fundus is associated with a 12% risk of uterine rupture<sup>[3]</sup> and is a contraindication for TOL.<sup>[13]</sup> Thus, Craigin's dictum of "once a cesarean always a cesarean" still applies to women with a history of a classical uterine incision.<sup>[25]</sup> In contrast, women with the more common, prior low transverse uterine incision, have an estimated risk for uterine rupture during TOL of less than 1%.<sup>[2, 4, 9, 26]</sup>

Clinical management options for women with a previous low vertical uterine incision have not been as clear due to the imprecise definition of a low vertical incision, the relatively small study series, and the rare occurrence of uterine rupture. The two largest observational series carefully excluded women in which the surgical record indicated that the vertical incision extended into the uterine fundus. Naef et al.<sup>[27]</sup> found a 1.1% incidence of uterine rupture in 174 women who had a previous low vertical uterine incision and a subsequent VBAC-TOL. Shipp et al.<sup>[28]</sup> compared the outcomes of 3289 women with either low transverse or low vertical scars who underwent VBAC-TOL. Uterine scar disruption occurred in 1.3% ( $n = 38/2,912$ ) of the women in the low transverse group and in 1.6% ( $n = 6/377$ ) in the low vertical group ( $P = .6$ ). Symptomatic uterine rupture occurred in 1.0% ( $n = 28/2,912$ ) of the women in the low transverse group and in 0.8% ( $n = 3/377$ ) in the low vertical group ( $P > .999$ ). On the basis of these findings, the authors concluded that women with a prior low vertical uterine incision are not at increased risk for uterine rupture during TOL compared with women who had a prior low transverse uterine incision.

## Single-Layer Versus Double-Layer Uterine Closure

Single-layer uterine closure gained clinical acceptance after the publication of several series demonstrating comparable short-term maternal morbidity and shorter operating times compared with double-layer closure.<sup>[29-32]</sup> However, potential morbidity associated with single-layer closure on subsequent VBAC-TOL was not addressed.

Tucker et al.<sup>[33]</sup> reviewed the medical records of 292 women who had VBAC-TOL and found the incidence of scar separation, uterine rupture, and adverse perinatal outcomes were similar for those women with one- or two-layer uterine closure. Because the choice of single- versus double-layer closure was originally at the discretion of the surgeon performing the cesarean birth, selection bias may have confounded real differences between the two closure types after a subsequent labor. Chapman et al.<sup>[34]</sup> compared the incidence of uterine rupture in 145 women who were originally randomized to either one- or two-layer uterine closure at the time of the original cesarean birth, who then subsequently underwent VBAC-TOL. One case of uterine dehiscence occurred in the cohort of women with single-layer uterine closure, whereas no cases of uterine rupture occurred in either the single- or double-layer closure groups. However, the sample size was too small to determine a statistically significant effect.

In a recent, larger, observational cohort analysis, Bujold et al.<sup>[35]</sup> identified a nearly four-fold increased incidence of uterine rupture during VBAC-TOL in women who had a single-layer closure of the previous lower uterine segment incision compared to women who had a previous double-layer uterine closure (OR: 3.95; 95% CI: 1.35-11.49). Medical records, including the operative report of the initial cesarean birth, were reviewed for 1980 women who had VBAC-TOL. At the time of the initial cesarean, single-layer closure was used in 489 women and double-layer closure in 1491 women. Uterine rupture occurred in 15 (3.1%) of the women with previous single-layer closure and in 8 (0.5%) of the women with a previous double-layer closure ( $P < .001$ ). On the basis of these findings, the authors recommended that surgeons consider using a double-layer closure technique for women who may subsequently experience a TOL.

## Number of Prior Cesarean Births

Numerous retrospective reviews prior to 1990 have attempted to examine the association between the number of previous cesareans and subsequent risk of uterine rupture during VBAC-TOL.<sup>[23, 24]</sup> However, most of these series are small, each consisting of less than 100 women with a history of two or more prior cesareans.

In the largest published series to date, Miller et al.<sup>[5]</sup> identified 12,707 women with prior uterine scar who underwent TOL. Uterine rupture occurred in 1.7% of the women with two or more previous cesareans compared with a uterine rupture incidence of 0.6% in those with only one prior cesarean birth (OR: 3.06; 95% CI: 1.95-4.79;  $P < .001$ ). However, this retrospective analysis did not control for other aspects of the women's obstetric history or labor management. Similarly, Asakura and Myers<sup>[36]</sup> reviewed the medical records of 302 women undergoing TOL who had two or more prior cesarean births. Uterine rupture occurred in 1% (3/302) of the women with two or more prior cesareans compared to 0.5% (5/1,110) in the women who had one prior scar on the uterus.

More recently, Caughey et al.<sup>[37]</sup> conducted a retrospective analysis of 3871 women who underwent a VBAC-TOL. The rate of uterine rupture was 3.7% among 134 women in the two-scars group compared to 0.8% in the 3,757 women with one previous uterine scar (RR: 4.5; 95% CI: 1.8-11.5;  $P = .001$ ). After controlling for maternal age, epidural analgesia, oxytocin induction, oxytocin augmentation, use of prostaglandin E2 gel, birth weight, gestational age, type of prior hysterotomy, year of TOL, and prior vaginal delivery, women with two prior cesarean scars were still 4.8 times more likely to experience uterine rupture during VBAC-TOL than women with one prior uterine scar (OR: 4.8; 95% CI: 1.8-13.2). In summary, women with two or more prior uterine scars have a significantly increased risk of uterine rupture during VBAC-TOL compared to women with only one prior uterine incision.

## Prior Vaginal Birth

Although the number of previous cesarean births appears to increase a woman's risk for uterine rupture during a VBAC-TOL, prior vaginal birth appears to be somewhat protective. Several scoring systems, developed to predict the likelihood of successful TOL, have included prior vaginal births as a predictor of success.<sup>[18-20, 22]</sup>

Zelop et al.<sup>[38]</sup> recently published the results of a study that evaluated the effect of prior vaginal birth on the risk of uterine rupture during TOL in 3783 women. Vaginal birth, which occurred either before or after the incident cesarean birth, lowered the risk of uterine rupture in subsequent VBAC-TOL. The rate of uterine rupture among women with prior vaginal birth was 0.2% (n = 2/1,021), compared to a rate of 1.1% (n = 30/2,762) among the women with no prior vaginal births ( $P = .01$ ). After controlling for birth weight, use of epidural analgesia, duration of labor, maternal age, year of TOL, induction with oxytocin, induction with prostaglandin E2 gel, and oxytocin augmentation, women having one or more prior vaginal births had a rate of uterine rupture that was one fifth the rate noted in women without prior vaginal birth (OR: 0.2; 95% CI: 0.04-0.8).

### **Interdelivery Interval**

Three studies have examined the association of uterine rupture and interdelivery interval, or time interval between prior cesarean and subsequent VBAC-TOL. In each retrospective review, analysis was limited to women with singleton pregnancies, at term, who had one prior cesarean birth and no prior vaginal births.

Shipp et al.<sup>[39]</sup> found that in women who underwent VBAC-TOL with an interdelivery interval of 18 months or less, the incidence of uterine rupture was 2.3% (n = 7/311) compared to an incidence of uterine rupture of 1.1% (n = 22/2,098) in women with a longer interdelivery interval ( $P = .07$ ). After controlling for maternal age, public assistance, length of labor, gestational age, and oxytocin use, women with a shorter interdelivery interval of 18 months or less were 3 times as likely to experience uterine rupture (OR: 3.0; 95% CI: 1.2-7.2). In contrast, Huang et al.<sup>[40]</sup> found no increase in the incidence of uterine rupture in 81 women with an interdelivery interval of 18 months or less compared to 1104 women with an interdelivery interval of 19 months or more. Three cases of uterine rupture occurred, all in the group with interdelivery interval 19 months or more. However, this difference was not statistically significant ( $P = 1.00$ ).

Recently, Bujold et al.<sup>[41]</sup> examined the medical records of 1527 women who underwent VBAC-TOL. Uterine rupture occurred in 2.8% of the women with an interdelivery interval of 24 months or less compared to 0.9% in women with an interdelivery interval greater than 24 months ( $P < .01$ ). After controlling for potential confounding variables, women with an interdelivery interval 24 months or less were almost 3 times more likely to experience uterine rupture (OR: 2.65; 95% CI: 1.08-5.46). Furthermore, the combination of an interdelivery interval 24 months or less and single-layer uterine closure of the previous uterine incision increased the incidence of uterine rupture to 5.6%.

### **Maternal Age**

Two large population-based studies of VBAC-TOL reported an increased rate of uterine rupture in women who were older than 35 years of age.<sup>[9, 26]</sup> However, neither study was able to control for potential confounding variables.

Shipp et al.<sup>[42]</sup> compared the incidence of uterine rupture in women who were younger than 30 years old with the incidence of uterine rupture in women who were at least 30 years or older. Among women with only one prior cesarean and no prior vaginal births, the incidence of uterine rupture was 0.5% (n = 5/1,065) for women younger than 30 years and 1.4% (n = 27/1,950) for women 30 or more years old ( $P = .02$ ). After controlling for birth weight, labor induction, labor augmentation and interdelivery interval, women 30 or more years of age were 3.2 times more likely to experience uterine rupture during VBAC-TOL than women younger than 30 years old (OR: 3.2; 95% CI: 1.2-8.4).

### **Maternal Fever Following Cesarean**

A recent case-control study evaluated the association between postpartum fever following cesarean birth and the incidence of uterine rupture in a subsequent VBAC-TOL.<sup>[43]</sup> Fever was defined as a temperature greater than 38°C. Twenty-one cases of uterine rupture were analyzed. The rate of maternal fever following the prior cesarean birth was 38% (n = 8/21) among the uterine rupture group and 15% (n = 13/84) in the control group of women who had a previous cesarean birth and no postpartum fever ( $P = .03$ ). After controlling for confounding variables, women with postpartum fever following a prior cesarean were 4 times more likely to experience a uterine rupture in a subsequent VBAC-TOL than case controls (OR: 4.0; 95% CI: 1.0-15.5). Although this is a striking association, the actual effect of infection and poor wound healing on future risk for uterine rupture requires further study in a prospective design.

## The Effect of Labor on Uterine Rupture

Three large population-based, cohort analyses have demonstrated that the risk for uterine rupture exists, whether a woman with a prior cesarean birth has a scheduled repeat cesarean or VBAC-TOL ( Table 2 ). Gregory et al.<sup>[26]</sup> reviewed California hospital discharge summary data for 1995 and reported a uterine rupture rate of 0.28% (n = 79/27,760) among women who elected repeat cesarean without labor and a rate of 0.53% (209/39,096) among women who underwent VBAC-TOL. Rageth et al.<sup>[10]</sup> examined a database of records from 17,613 women attempting VBAC-TOL in Switzerland and reported an overall uterine rupture rate of 0.4% (n = 70/17,613) in the VBAC-TOL group, which was slightly higher than the 0.19% uterine rupture rate in the women who underwent elective repeat cesarean birth (n = 22/11,433). Similarly, Lydon-Rochelle et al.<sup>[12]</sup> reviewed Washington State vital records and abstracted hospital discharge (ICD-9) diagnoses to report a 0.16% (n = 11/6,980) incidence of uterine rupture among women who underwent an elective repeat cesarean without labor and an incidence of uterine rupture of 0.6% (n = 80/13,115) among women attempting VBAC-TOL. In each of these population-based studies, the effect of labor appears to increase the incidence of uterine rupture in women who underwent VBAC-TOL. However, it is unclear whether this increased incidence of uterine rupture is secondary to other risk factors, specifically the additive effect of induction of labor with either oxytocin and/or prostaglandins.

**Table 2. Risk of Uterine Rupture in Elective Repeat Cesarean Birth Versus VBAC-TOL**

Author	Type of Study	N	Uterine Rupture Rate (%)	
			Repeat C/S	VBAC-TOL*
Gregory et al., 1999 <sup>[26]</sup>	Retrospective cohort	66,856	0.28	0.53
Rageth et al., 1999 <sup>[10]</sup>	Retrospective cohort	29,046	0.19	0.4
Lydon-Rochelle, 2001 <sup>[12]</sup>	Retrospective cohort	17,769	0.16	0.6

## Labor Induction and Augmentation With Oxytocin

Two large population-based studies have evaluated the effect of oxytocin induction and/or augmentation on uterine rupture during TOL. In a study by Rageth et al.,<sup>[10]</sup> 17,613 women underwent VBAC-TOL in which 70 women experienced uterine rupture. Women in the uterine rupture group had increased rates of induced labor (24%) compared to the women in the non-rupture group (13.9%) ( $P = .013$ ). Similarly, Lydon-Rochelle et al.<sup>[12]</sup> found women who experienced spontaneous labor had a 0.52% (n = 56/10,789) incidence of uterine rupture compared to an incidence of 0.77% (n = 15/1,960) in women whose labors were induced with oxytocin.

Few studies separately evaluate the effects of oxytocin induction versus oxytocin augmentation on uterine rupture during TOL. Zelop et al.<sup>[44]</sup> reported a uterine rupture rate of 0.7% (n = 16/2,214) among women attempting VBAC-TOL with spontaneous labor compared to 2.0% (n = 9/458) among women induced with oxytocin. After

controlling for birth weight, epidural anesthesia, duration of labor, maternal age, year of delivery, and years since last birth, induction of labor with oxytocin was associated with a 4.6-fold increased risk of uterine rupture, compared to the rate noted in women who had spontaneous labor (OR: 4.6; 95% CI: 1.5-14.1). Augmentation with oxytocin was found to be associated with a 2.3-fold increased risk of uterine rupture (OR: 2.3; 95% CI: 0.8-7.0); however, this difference was not statistically significant.

Leung et al.<sup>[45]</sup> conducted a case-control study of 70 women with prior cesarean birth attempting VBAC-TOL. After controlling for confounding variables, women receiving oxytocin were almost 3 times more likely to experience uterine rupture than the women in the control group (OR: 2.7; 95% CI: 1.2-6.0). Dysfunctional labor had an even greater effect on increasing the incidence of uterine rupture. Dysfunctional labor, primarily arrest disorders, increased the risk of uterine rupture 7-fold (OR: 7.2; 95% CI: 2.7-20.0). However, a majority of the women who received oxytocin also experienced dysfunctional labor. The presence of dysfunctional labor may be a factor for uterine rupture or a marker for its occurrence. Further study is needed to determine if the major risk for uterine rupture is dysfunctional labor, use of oxytocin, or both.

In summary, studies performed to date suggest oxytocin use may result in a higher risk of uterine rupture during VBAC-TOL. Therefore, it is prudent to exercise caution when inducing or augmenting labor in a woman with a scarred uterus. Labor progression must be carefully followed and the woman and her fetus must be monitored closely in an institution capable of immediate emergency response.<sup>[13]</sup>

### Labor Induction With Prostaglandins

Two types of prostaglandin agents are currently used for cervical ripening and labor induction: prostaglandin E2 (Prepidil, Cervidil) and the analogue of prostaglandin E1, misoprostol (Cytotec). The use of prostaglandin E2 (PGE2) gel in women with previously scarred uteri is associated with a clear increased incidence of uterine rupture. However, in several of the initial studies, it is unclear whether the cases of uterine rupture were related to PGE2 gel alone or to PGE2 gel in combination with oxytocin administration.<sup>[46, 47]</sup>

Zelop et al.<sup>[44]</sup> examined the effects of PGE2 gel on women who underwent VBAC-TOL. Among the 35 women in whom labor was induced by PGE2 alone, uterine rupture occurred in 2.9% (n = 1/35) compared to 4.5% (n = 3/67) among the 67 women who received both PGE2 and oxytocin for induction ( Table 3 ). Overall, the rate of uterine rupture among those who received any PGE2 gel was 3.9% compared to 0.9% among those who did not receive PGE2 gel (P = .02). However, 84 of the 102 women who received PGE2 gel also received oxytocin. After controlling for oxytocin induction and augmentation, birth weight, use of epidural anesthesia, duration of labor, maternal age, year of delivery, and years since last birth, induction of labor with PGE2 gel alone was not found to be a significant risk factor for uterine rupture. Although the odds ratio of 3.2 (95% CI: 0.9-10.9) suggests an association, it is not statistically significant.

**Table 3. Risk of Uterine Rupture in Women Undergoing VBAC-TOL: Spontaneous Labor Versus Induction or Augmentation**

Author	Type of Study	Number of Women	Methods	Uterine Rupture Rate (%)	
				Spontaneous Labor	Induction or Augmentation
Zelop et al., 1999 <sup>[44]</sup>	Retrospective cohort	2,774	Women at term with 1 prior cesarean and no other births:		

			Spontaneous labor vs. oxytocin augmentation	0.7	1.0
			Spontaneous labor vs. oxytocin induction	0.7	2.0
			Spontaneous labor vs. PGE2 gel	0.7	2.9
			Spontaneous labor vs. PGE2 gel and oxytocin induction	0.7	4.5
Lydon-Rochelle, 2001 <sup>[12]</sup>	Retrospective cohort	17,749	All women birthed in Washington State		
			Analysis of hospital discharge diagnostic codes		
			Spontaneous labor vs. induction of labor without prostaglandins	0.52	0.77
			Spontaneous labor vs. induction with prostaglandins	0.52	2.45

The recent population-based, retrospective analysis by Lydon-Rochelle et al.<sup>[12]</sup> noted a significant increase in the incidence of uterine rupture in women who underwent VBAC-TOL and were induced with prostaglandins. Uterine rupture occurred at a rate of 1.6 per 1,000 women who experienced a repeat cesarean birth without labor compared to an incidence of 24.5 per 1,000 women in the induction of labor with prostaglandins group (RR: 15.6; 95% CI: 8.1-30.0). Although the authors concluded that induction of labor with prostaglandins significantly increases the risk of uterine rupture among women with a previous cesarean birth, they did not have information on specific types and dosages of prostaglandins used. The authors acknowledged that their use of ICD-9 codes might have caused an overstatement of the actual incidence of uterine ruptures, secondary to the use of a single ICD code for both uterine incision extension and uterine rupture.

### Misoprostol

Misoprostol (prostaglandin E1) is gaining popularity as an option for cervical ripening and labor induction in women without previous uterine surgery. A number of studies have found the use of misoprostol to be a significant risk factor for uterine rupture in women with a prior cesarean birth.<sup>[48-54]</sup> However, some of the studies evaluating misoprostol in women who undergo VBAC-TOL are confounded by concomitant use of oxytocin. The definitive work in this area was a randomized controlled trial conducted by Wing et al.<sup>[50]</sup> Women were originally randomized to 25 mcg of misoprostol intravaginally every 6 hours to a maximum of four doses, or to intravenous oxytocin per a standard infusion protocol. Seventeen women received misoprostol and 21 women received oxytocin. The study was stopped prematurely secondary to two emergency cesarean sections with noted uterine disruption at the time of surgery in the women who received misoprostol. In a larger series, Plaut et al.<sup>[52]</sup> reviewed the uterine rupture cases from two hospitals over 20 months in 1996 to 1997. Of the 89 women induced with misoprostol in this combined series, uterine rupture occurred in 5.6% (5/89).



No studies or meta-analyses have been conducted that have large enough numbers to detect a statistically significant relationship between misoprostol use and uterine rupture during VBAC-TOL. A recent COCHRANE review<sup>[55]</sup> evaluated 62 trials in which vaginal misoprostol was used for cervical ripening or induction of labor in women with unscarred uteri. The review concluded that the studies were not large enough, either individually or cumulatively, to exclude the possibility of uterine rupture. In April 2002, the ACOG issued a committee opinion that discourages the use of prostaglandins for cervical ripening or induction of labor in women attempting VBAC-TOL.<sup>[56]</sup> ACOG supports the practice of VBAC-TOL under proper circumstances and with appropriate safeguards.<sup>[13, 56]</sup>

## **Fetal Heart Rate and Uterine Rupture**

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Studies examining the fetal heart rate (FHR) prior to uterine rupture consistently report that non-reassuring fetal heart rate patterns are the predominant sign suggesting impending or actual uterine rupture. Menihan<sup>[57]</sup> examined the FHR tracings of 11 women attempting VBAC-TOL and reported fetal bradycardia in 82% (n = 9/11) prior to uterine rupture. Prior to the bradycardia, 73% (n = 8/11) had variable decelerations, 36% (n = 4/11) had late decelerations, 27% (n = 3/11) had early decelerations, and none had tachycardia. In a review of eight fetal monitor tracings for 2 hours preceding uterine rupture, Ayres et al.<sup>[58]</sup> identified recurrent late decelerations in 88% (n = 7/8) and terminal bradycardia in 50% (n = 4/8) of the tracings.

In a larger study, Leung et al.<sup>[14]</sup> analyzed the FHR and uterine contraction pattern immediately prior to 78 cases of uterine rupture. Prolonged deceleration was defined as a FHR less than 90 beats per minute that exceeded 1 minute without return to baseline. Prolonged deceleration (alone or preceded by either severe late or variable decelerations) occurred in 71% (n = 55/78) of the cases of uterine rupture. In addition, prolonged deceleration occurred in 100% (n = 36/36) of the FHR tracings in which total fetal extrusion occurred. If more than one type of periodic deceleration, in addition to a prolonged deceleration was present, only the most ominous pattern was recorded. Late decelerations were considered most ominous, followed by variable and early deceleration. Although late decelerations were more common preceding the prolonged deceleration that heralded uterine rupture, this finding may be an artifact of the study methodology.

## **Timing of Birth and Neonatal Outcome**

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Several studies have attempted to evaluate the timing of birth and neonatal outcome in women with uterine rupture during VBAC-TOL. Bujold et al.<sup>[16]</sup> compared neonatal morbidity associated with uterine rupture during VBAC-TOL to neonatal morbidity following umbilical cord prolapse when the median time from the first manifestation to birth was comparable (18 versus 17 minutes). Although similar in response times, neonatal neurological morbidity was more significant with uterine rupture than with umbilical cord prolapse. Neonatal arterial blood pH less than 7.1 occurred in 47% of the uterine rupture cases compared to only 3% in the umbilical cord prolapse group ( $P < .005$ ). Likewise, 5-minute Apgar scores less than 6 were found in 33% of the uterine rupture cases compared to 3% in the umbilical cord prolapsed group ( $P < .05$ ). At 2- to 6-month follow-up, three (20%) of the newborns in the uterine rupture group were diagnosed with ischemic encephalopathy and major neurologic impairment compared to none in the cord prolapse group ( $P < .05$ ).

Porter et al.<sup>[15]</sup> examined the neonatal outcome in 26 cases of uterine rupture occurring during VBAC-TOL in large hospitals in a single metropolitan region, all of which had 24-hour in-house anesthesia coverage. Six (23%) infants suffered either neonatal death or adverse neurologic sequelae as a result of the uterine rupture. Poor neonatal outcome was seen in 31% of the infants birthed within 30 minutes and in 33% of the infants birthed within 20 minutes of either severe variable decelerations or bradycardia.

In a larger study, Leung et al.<sup>[14]</sup> evaluated 78 cases of uterine rupture in a large tertiary care medical center and reported significant neonatal morbidity when 18 minutes or more elapsed between the onset of prolonged

deceleration and birth. When the prolonged deceleration was preceded by severe late or variable decelerations, fetal asphyxia occurred as early as 10 minutes from the onset of prolonged deceleration.

Similarly, Bujold and Gauthier<sup>[17]</sup> examined 23 cases of uterine rupture in a tertiary care medical center with in-house obstetricians and anesthesiologists. Nine neonates (39%) had severe metabolic acidosis. Among these, four neonates had seizures, two had multiple organ failure, and three were diagnosed with hypoxic-ischemic encephalopathy. Within this group, one intrapartum death occurred. All three neonates with hypoxic-ischemic encephalopathy were outside the uterus at the time of laparotomy. Contrary to the findings of Leung et al., less than 18 minutes elapsed between the time of prolonged deceleration and delivery in two of the three neonates diagnosed with hypoxic-ischemic encephalopathy (15 and 16 minutes).

Prompt intervention does not always prevent severe, fetal metabolic acidosis or neonatal death. Even in facilities with immediate access to cesarean birth uterine rupture can result in catastrophic outcome. A non-reassuring FHR pattern, occurring prior to the time of uterine rupture, further decreases the amount of time available before fetal insult occurs.

## Summary

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A pregnant woman with a previously scarred uterus is at increased risk for complications whether she has a successful VBAC-TOL, unsuccessful VBAC-TOL or elective repeat cesarean birth. Neither elective repeat cesarean nor VBAC-TOL is risk-free. Only by eliminating primary cesarean deliveries can we hope to obviate the need for repeat cesareans or VBAC-TOL.

Providing women desiring VBAC-TOL a more individualized risk assessment of uterine rupture is one component of the informed consent women need to participate in this decision process. A history of single-layer uterine closure, more than one prior uterine scar, an interdelivery interval less than 24 months, maternal age greater than 30 years, or maternal fever following a previous cesarean birth have been identified as significant factors increasing a woman's risk of uterine rupture during TOL. Factors in management of a woman's index pregnancy that increase her risk of uterine rupture are labor induction with either prostaglandins or oxytocin. The predominant sign suggesting impending or actual uterine rupture is a non-reassuring FHR pattern and/or prolonged deceleration.

Women attempting VBAC-TOL require close surveillance and continuous fetal monitoring. TOL should only be attempted in facilities equipped to respond to emergencies with physicians and personnel for emergency birth immediately available. After individualized counseling of the benefits and risks of VBAC-TOL, the ultimate decision to attempt TOL or elect repeat cesarean birth should be made by the informed woman and her health care provider.

## References

1. National Institutes of Health. Cesarean childbirth. NIH publication no. 82-2067. Washington (DC): Government Printing Office, 1981.
2. Flamm BL, Newman LA, Thomas ST, Fallon D, Yoshida MM. Vaginal birth after cesarean delivery: Results of a 5-year multicenter collaborative study. *Obstet Gynecol* 1990;76:750-4.
3. Rosen MG, Dickinson JC, Westhoff CL. Vaginal birth after cesarean: A meta-analysis of morbidity and mortality. *Obstet Gynecol* 1991;77:465-70.
4. Flamm BL, Goings JR, Liu Y, Wolde-Tsadik G. Elective repeat cesarean delivery versus trial of labor: A prospective multicenter study. *Obstet Gynecol* 1994;83:927-32.
5. Miller DA, Diaz FG, Paul RH. Vaginal birth after cesarean: A 10-year experience. *Obstet Gynecol* 1994;84:255-8.
6. Morbidity and Mortality Weekly Report. Rates of cesarean delivery-United States 1993. *MMWR* 1995;44:303-7.

7. Menacker F, Curtin SC. Trends in cesarean birth and vaginal birth after previous cesarean, 1991-99. *National Vital Statistics Reports* 2001;49(13):1-15.
8. Martin JA, Hamilton BE, Ventura SJ, Menacker F, Park MM, Sutton PD. Births: Final data for 2001. *National Vital Statistics Reports* 2002;51(2):1-103.
9. McMahon MJ, Luther ER, Bowes WA, Olshan AF. Comparison of a trial of labor with an elective second cesarean section. *N Engl J Med* 1996;335:689 -95.
10. Rageth JC, Juzi C, Grossenbacher H. Delivery after previous cesarean: A risk evaluation. *Obstet Gynecol* 1999;93:332-7.
11. Mozurkewich EL, Hutton EK. Elective repeat cesarean delivery versus trial of labor: A meta-analysis of the literature from 1989- 1999. *Am J Obstet Gynecol* 2000;183:1187-97.
12. Lydon-Rochelle M, Holt VL, Easterling TR, Martin DP. Risk of uterine rupture during labor among women with prior cesarean delivery. *N Engl J Med* 2001;345:3-8.
13. American College of Obstetricians and Gynecologists. Vaginal birth after previous cesarean delivery. Practice Bulletin No. Washington (DC): ACOG, 1999.
14. Leung AS, Leung EK, Paul RH. Uterine rupture after previous cesarean delivery: Maternal and fetal consequences. *Am J Obstet Gynecol* 1993;169:945-50.
15. Porter TF, Clark SL, Esplin MS, Tooke-Miller C, Scott JR. Timing of delivery and neonatal outcome in patients with clinically overt uterine rupture during VBAC. *Am J Obstet Gynecol* 1998;178: S31.
16. Bujold E, Gauthier RJ. Comparative study of neonatal morbidity associated with uterine rupture during VBAC versus cord prolapse. *Am J Obstet Gynecol* 2000;182:S160.
17. Bujold E, Gauthier RJ. Neonatal morbidity associated with uterine rupture: What are the risk factors? *Am J Obstet Gynecol* 2002;186:311-4.
18. Troyer LS, Parisi VM. Obstetric parameters affecting success in trial of labor: Designation of a scoring system. *Am J Obstet Gynecol* 1992;167:1099 -1104.
19. Weinstein D, Benshushan A, Tanos V, Zilberstein R, Rojansky N. Predictive score for vaginal birth after cesarean section. *Am J Obstet Gynecol* 1996;174:192-8.
20. Flamm BL, Geiger AM. Vaginal birth after cesarean delivery: An admission scoring system. *Obstet Gynecol* 1997;90:907-10.
21. Holt VL, Mueller BA. Attempt and success rates for vaginal birth after caesarean section in relation to complications of the previous pregnancy. *Paediatr Perinat Epidemiol* 1997;11:63-72.
22. Shipp T, Zelop C, Cohen A, Repke J, Frigoletto F, Sachs B, Lieberman E. Prediction of the rate of uterine rupture using an obstetrical scoring system. *Am J Obstet Gynecol* 2001;185:S124.
23. Lavin JP, Stephens RJ, Miodovnik M, Barden TP. Vaginal delivery in patients with a previous cesarean section. *Obstet Gynecol* 1982;59:135-48.
24. Lieberman E. Risk factors for uterine rupture during a trial of labor after cesarean. *Clin Obstet Gynecol* 2001;44:609 -21.
25. Craigin EB. Conservatism in obstetrics. *NY Med J* 1916;104: 1-3.
26. Gregory KD, Korst LM, Cane P, Platt LD, Kahn K. Vaginal birth after cesarean and uterine rupture rates in California. *Obstet Gynecol* 1999;94:985-9.
27. Naef RW, Ray MA, Chauhan SP, Roach H, Blake PG, Martin JN. Trial of labor after cesarean delivery with a lower-segment, vertical uterine incision: Is it safe? *Am J Obstet Gynecol* 1995;172: 1666-74.
28. Shipp TD, Zelop CM, Repke JT, Cohen A, Caughey AB, Lieberman E. Intrapartum uterine rupture and dehiscence in patients with prior lower uterine segment vertical and transverse incisions. *Obstet Gynecol* 1999;94:735-40.
29. Hauth JC, Owen J, Davis RO. Transverse uterine closure: One versus two layers. *Am J Obstet Gynecol* 1992;167(4 Pt 1):1108 -11.
30. Jelsema RD, Wittingen JA, Vander Kolk KJ. Continuous, non-locking, single-layer repair of the low transverse uterine incision. *J Reprod Med* 1993;38:393-6.
31. Ohel G, Younis JS, Lang N, Levit A. Double-layer closure of uterine incision with visceral and parietal

peritoneal closure: Are they obligatory steps of routine cesarean sections? *J Matern Fetal Med* 1996;5:366-9.

32. Ferrari AG, Frigerio LG, Candotti G, Buscaglia M, Petrone M, Taglioretti A, et al. Can Joel-Cohen incision and single layer reconstruction reduce cesarean section morbidity? *Int J Gynaecol Obstet* 2001;72:135-43.
33. Tucker JM, Hauth JC, Hodgkins P, Owen J, Winkler CL. Trial of labor after a one- or two-layer closure of a low transverse uterine incision. *Am J Obstet Gynecol* 1993;168:545-6.
34. Chapman SJ, Owen J, Hauth JC. One- versus two-layer closure of a low transverse cesarean: The next pregnancy. *Obstet Gynecol* 1997;89:16-8.
35. Bujold E, Bujold C, Hamilton EF, Harel F, Gauthier RJ. The impact of a single-layer or double-layer closure on uterine rupture. *Am J Obstet Gynecol* 2002;186:1326-30.
36. Asakura H, Myers SA. More than one previous cesarean delivery: A 5-year experience with 435 patients. *Obstet Gynecol* 1995;85:924-9.
37. Caughey AB, Shipp TD, Repke JT, Zelop CM, Cohen A, Lieberman E. Rate of uterine rupture during a trial of labor in women with one or two prior cesarean deliveries. *Am J Obstet Gynecol* 1999;181:872-6.
38. Zelop CM, Shipp TD, Repke JT, Cohen A, Lieberman E. Effect of previous vaginal delivery on the risk of uterine rupture during subsequent trial of labor. *Am J Obstet Gynecol* 2000;183:1184-6.
39. Shipp TD, Zelop CM, Repke JT, Cohen A, Lieberman E. Interdelivery interval and risk of symptomatic uterine rupture. *Obstet Gynecol* 2001;97:175-7.
40. Huang WH, Nakashima DK, Rumney PJ, Keegan KA, Chan K. Interdelivery interval and the success of vaginal birth after cesarean delivery. *Obstet Gynecol* 2002;99:41-4.
41. Bujold E, Mehta SH, Bujold C, Gauthier RJ. Interdelivery interval and uterine rupture. *Am J Obstet Gynecol* 2002;187:1199-1202.
42. Shipp TD, Zelop C, Repke JT, Cohen A, Caughey AB, Lieberman E. The association of maternal age and symptomatic uterine rupture during a trial of labor after prior cesarean delivery. *Obstet Gynecol* 2002;99:585-8.
43. Shipp TD, Zelop C, Cohen A, Repke JT, Lieberman E. Post-cesarean delivery fever and uterine rupture in a subsequent trial of labor. *Obstet Gynecol* 2003;101:136-9.
44. Zelop CM, Shipp TD, Repke JT, Cohen A, Caughey AB, Lieberman E. Uterine rupture during induced or augmented labor in gravid women with one prior cesarean delivery. *Am J Obstet Gynecol* 1999;181:882-6.
45. Leung AS, Farmer RM, Leung EK, Medearis AL, Paul RH. Risk factors associated with uterine rupture during trial of labor after cesarean delivery: A case-control study. *Am J Obstet Gynecol* 1993; 168:1358-63.
46. Flamm BL, Anton D, Goings JR, Newman J. Prostaglandin E2 for cervical ripening: A multicenter study of patients with previous cesarean delivery. *Am J Perinatol* 1997;14:157-60.
47. Ravasia DJ, Wood SL, Pollard JK. Uterine rupture during induced trial of labor among women with previous cesarean delivery. *Am J Obstet Gynecol* 2000;183:1176-9.
48. Bennett BB. Uterine rupture during induction of labor at term with intravaginal misoprostol. *Obstet Gynecol* 1997;89:832-3.
49. Sciscione AC, Nguyen L, Manley JS, Shlossman PA, Colmorgen GHC. Uterine rupture during preinduction cervical ripening with misoprostol in a patient with a previous caesarean delivery. *Aust NZ J Obstet Gynaecol* 1998;38:96-7.
50. Wing DA, Lovett K, Paul RH. Disruption of prior uterine incision following misoprostol for labor induction in women with previous cesarean delivery. *Obstet Gynecol* 1998;91:828-30.
51. Cunha M, Bugalho A, Bique C, Bergstrom S. Induction of labor by vaginal misoprostol in patients with previous cesarean delivery. *Acta Obstet Gynecol Scand* 1999;78:653-4.
52. Plaut MM, Schwartz ML, Lubarsky SL. Uterine rupture associated with the use of misoprostol in the gravid patient with a previous cesarean section. *Am J Obstet Gynecol* 1999;180:1535-42.
53. Gherman RB, McBrayer S, Browning J. Uterine rupture associated with vaginal birth after cesarean section: A complication of intravaginal misoprostol? *Gynecol Obstet Invest* 2000;50:212-3.
54. Hill DA, Chez RA, Quinlan J, Fuentes A, LaCombe J. Uterine rupture and dehiscence associated with

intravaginal misoprostol cervical ripening. *J Reprod Med* 2000;45:823-6.

55. Hofmeyr GJ, Gulmezoglu AM. Vaginal misoprostol for cervical ripening and induction of labour (Cochrane Review). *Cochrane Database Syst Rev* 2003;1:CD000941.
56. American College of Obstetricians and Gynecologists. Induction of labor for vaginal birth after cesarean delivery. ACOG Committee Opinion 271. *Obstet Gynecol* 2002;99:679 -80.
57. Menihan CA. Uterine rupture in women attempting a vaginal birth following prior cesarean birth. *J Perinatol* 1998;18:440 -3.
58. Ayres AW, Johnson TRB, Hayashi R. Characteristics of fetal heart rate tracings prior to uterine rupture. *Int J Gynaecol Obstet* 2001;74:235-40.

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