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Risk of uterine rupture in Australian women attempting vaginal birth after one prior caesarean section: a retrospective population-based cohort study

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Objective Higher risks of uterine rupture have been reported among women attempting vaginal birth after caesarean (VBAC) particularly following induction with prostaglandins, compared with women who do not labour. This study aimed to estimate these risks as well as that associated with oxytocin use.

Design Population-based retrospective cohort study involving all women who had their first births by caesarean. In their second birth, risks of uterine rupture among women without labour and women who had labour augmented or induced were compared with women who gave birth after spontaneous labour.

Setting Four Australian states in 1998–2000.

Population Women on pregnancy outcome databases with a second birth after a prior caesarean for their first birth.

Methods From 29 008 women identified from the databases, those with uterine rupture were identified and validated using hospital case records.

Main outcome measure Uterine rupture.

Results The risk of complete uterine rupture among women without labour was 0.01%. The risk in spontaneous labour without augmentation was 0.15%, considerably higher when there was augmentation with oxytocin (1.91%). The risk with induction of labour was 0.54% for oxytocin alone, 0.68% for prostaglandin alone, 0.63% without either and 0.88% when they were combined. Compared with spontaneous labour, risks were increased three- to five-fold for any induction, six-fold for prostaglandin combined with oxytocin and 14-fold for augmentation with oxytocin.

Conclusions Careful consideration should be given to the use of oxytocin for augmentation of labour or induction by any method for women with a previous caesarean in view of increased risks of uterine rupture.

Keywords Uterine rupture, vaginal birth after caesarean.

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Introduction

Primary caesarean section rates have increased worldwide in the last 30 years.[1] Consequently, increasing numbers of women have to consider the risks and benefits of vaginal birth after caesarean (VBAC). Edward Cragin's 1916 dictum 'once a caesarean, always a caesarean', which was intended to reduce unnecessary caesareans and had many exceptions,[2] was largely followed in the USA until the late 1970s.[1] Following a series of studies in the 1980s reporting the relative safety of VBAC,[3] the rates of VBAC increased in most developed countries, in particular in the USA because of its previous very low uptake of VBAC, from 1% in 1978 to 28% in 1995.[1] The US VBAC rate then fell to

11% in 1999[1] because of concerns about safety.[4] However, high rates of VBAC were reported in the UK and Europe even in the 1950s and remained high (around 60%) in the late 1980s.[1]

In 2001 Lydon-Rochelle *et al.*[5] reported that, compared with the risk in women who had an elective caesarean section without labour, the relative risk of uterine rupture was 3.3 (95% CI 1.8–6.0) among women with spontaneous onset of labour, 4.9 (95% CI 2.4–9.7) among women with induction of labour without prostaglandins and 15.6 (95% CI 8.1–30.0) among women induced with prostaglandins. They found a risk of 2.45% in the group induced with prostaglandins.

This publication had a major international impact. The American College of Obstetricians and Gynecologists discouraged the use of prostaglandins for labour induction[6] whereas the British Royal College advocated caution but still considered prostaglandins 'safe' and preferable to oxytocin for induction.[7] The proportion of women attempting VBAC declined, with the US VBAC rate falling further to 9% in 2004.[8] Surveys of obstetricians found only a third in Australia[9] and 25% in Canada[10] indicating willingness to use vaginal prostaglandins in induction of labour for women with a previous caesarean.

The aim of this Australian study was to quantify the risk of uterine rupture in the second (singleton) birth among the following subgroups of women with a previous caesarean section, and to determine their relative risks, compared with women who have a spontaneous labour without augmentation with oxytocin:

1 Repeat caesarean section without labour.
2 Spontaneous onset of labour with augmentation with oxytocin.
3 Induction of labour with oxytocin (without prostaglandins).
4 Induction with prostaglandins (without oxytocin).
5 Induction with both oxytocin and prostaglandins.
6 Induction with neither oxytocin nor prostaglandins.

Methods

The data used were from the population-based perinatal data collections of the four largest states that collect the relevant data—Victoria, Queensland, Western Australia and South Australia—and that together include 60% of the approximately 257 000 births/year in Australia.[11] All women having their second birth, a singleton one, during 1998–2000 were included, where the first birth was a live-born singleton delivered by caesarean section. The period 1998–2000 was used because data on previous caesareans were not available before 1998 and this period preceded the introduction of misoprostol for induction of labour in Australia and the publication of the Lydon-Rochelle *et al.* paper,[5] i.e. captured a cohort with a reasonable number of women with a previous caesarean section subjected to prostaglandin induction of labour. The caesarean section rate in the four states during this period increased from 22.3% to 24.6%.[11] The VBAC rate, in one state, decreased from 30.4% to 28.0%.

The study was approved by the Human Research Ethics Committees of the South Australian Department of Health, the Aboriginal Health Council of South Australia and the Victorian Department of Human Services.

Cases of uterine rupture were identified from the International Classification of Diseases (ICD) codes of the perinatal data collections and the hospital morbidity collections provided by hospitals to the state Departments of Health. Case notes were reviewed by one of the doctors or midwives at the hospital of birth. Information was requested on the onset of labour, whether labour was augmented or induced and the method used, method of birth and details of the rupture, so that the perinatal data could be validated for these women. Copies of the relevant sections of the data retrieved for all women were examined by one of the researchers (GAD), an obstetrician, who was blinded to the labour status including the use of prostaglandins and intravenous oxytocin, and the state of origin. Only women with confirmed uterine rupture were included in the final analysis. Both women who had complete uterine rupture, representing the main substance of this paper (a rupture through the full thickness of the uterus with fetal parts and/or amniotic fluid in abdominal cavity) and those with partial rupture (a rupture through the full thickness of the uterus with ballooning of thin membrane but no fetal parts or amniotic fluid in abdominal cavity) were included. Analyses were then undertaken, comparing the risk of uterine rupture for each of the six groups (subgroups a–f) of women with women who went into spontaneous labour and received no oxytocin augmentation. Analyses were undertaken for all ruptures as well as for complete ruptures only because these are more likely to have significant clinical effects.

The following variables were examined for risk of uterine rupture, yielding univariate odds ratios (ORs):

Maternal age (in 5-year age groups), indigenous status, area of residence (metropolitan or country), patient type (public or private), medical conditions (diabetes and pre-existing hypertension), obstetric complications (prelabour rupture of membranes, gestational diabetes, genital herpes, pregnancy hypertension, breech presentation, antepartum haemorrhage from placenta praevia, placental abruption and other causes), labour complications (failure to progress, cord prolapse), gestational age and birthweight (both as continuous variables), sex and congenital abnormality.

Gestational age and birthweight and all other variables with a P value <0.10 in univariate analysis were entered into a multivariable logistic regression to examine the association

between the occurrence of uterine rupture and predictor variables and to indicate levels of statistical significance. Backwards elimination using STATA software (Statacorp., College Station, TX, USA) yielded adjusted odds ratios (AOR) for risk of uterine rupture where this did not reduce model fit ($P \geq 0.05$). Assumptions underlying these tests, such as an absence of co-linearity, were tested and found to be satisfactory.

Results

In this 3-year period 29 008 women fulfilled the inclusion criteria. Their mean age was 31.2 years with 22% 35 years or older. Two percent were indigenous, 74% lived in the metropolitan area and 43% were private patients. Mean gestation was 38.6 weeks with 5.8% preterm births (<37 weeks of gestation) and mean birthweight was 3396.1 g with 4.4% low birthweight (<2500 g). Fifty-three incidents of uterine rupture were identified from ICD codes: 20 were in both data collections (perinatal and hospital morbidity), and 15 and 18 each in only one data collection. Case notes for six women were not accessed. As these were identified in both collections and all the remaining 14 identified in both collections were found to have complete uterine rupture, these were also included as such. Of the 53 women, 48 had a confirmed uterine rupture, an incidence of 0.17%. Thirty-seven women (0.13%) had complete uterine ruptures.

Table 1 shows the absolute risks (and 95% CI) of uterine rupture and complete uterine rupture for each group. It also shows the ORs and AORs of uterine rupture as well as complete uterine rupture for each group compared with women who had spontaneous labour without augmentation with oxytocin. The other co-variables in the final multivariable models are provided in the footnotes. For all ruptures, there were no co-variables in the final models and ORs and AORs were identical. For complete ruptures, placental abruption was the only co-variable showing increased risk of uterine rupture in all models. Table 1 also provides the percentage of women achieving vaginal birth in the various subgroups, the overall rate of vaginal birth in the total VBAC cohort was 54.3%.

Table 1 shows that the lowest risk for uterine rupture (0.02%) and complete uterine rupture (0.01%) occurred with elective caesarean section with AORs of 0.11 and 0.04, respectively, compared with spontaneous labour without oxytocin augmentation (which had absolute risks of 0.19% and 0.15%, respectively). Compared with the latter group, the risk was increased three- to five-fold when labour was induced, whether with oxytocin alone or prostaglandins alone or other methods (artificial rupture of membranes). The risk was slightly higher for induction with prostaglandins combined with oxytocin, a nine-fold increase for all ruptures and six-fold for complete ruptures. However, the highest risk (10-fold increase for all ruptures and 14-fold for complete ruptures) was obtained for augmentation with oxytocin after spontaneous onset of labour (1.91%).

Table 2 compares the risks of uterine rupture for no labour and trial of labour with other major studies in the recent literature and Table 3 compares them for no labour, spontaneous labour and induced labour.

Discussion

The optimal mode of delivery after one previous caesarean section remains a topic of heated debate both within countries and also at an international level. The recent Cochrane reviews by Dodd *et al.*[12,13] concluded that no randomised controlled trials had been completed up to 2006.

This large retrospective Australian study confirmed the findings of many studies that the lowest risk for uterine rupture occurs among women with no labour (0–0.2% generally but 1.9% in one meta-analysis of studies in the 1980s), and risks are higher for a trial of labour (0.3–2.1%, with higher risks in earlier years[4,12–17] (Table 2).

Our study found lower absolute risks for uterine rupture than the study by Lydon-Rochelle *et al.*[5] both among women who have an elective caesarean section (0.02% compared with 0.16%) as well as among women who had prostaglandins for induction of labour (0.99% compared with 2.45%). Some studies[17–19] found high rates of uterine rupture for prostaglandin induction but others did not.[7,16] We also found that induction of labour with oxytocin was associated with an equivalent absolute risk for uterine rupture to that for prostaglandin (0.82% compared with 0.68%). However, the highest absolute risk of uterine rupture occurred among women who had either augmentation with oxytocin after spontaneous onset of labour (1.91%); or induction with both oxytocin and prostaglandins (1.77% but 0.88% for complete ruptures).

Although the significantly increased risk associated with oxytocin used for augmentation is a clinically relevant finding it needs to be acknowledged that this finding is based on the 12 cases of complete uterine rupture in this particular subgroup.

Lieberman[20] concluded that most large series suggest that oxytocin use is associated with some increase in risk of uterine rupture (three to five times that when oxytocin is not used). Whether it was used in augmentation or induction of labour was often not defined but two studies reported similar risks of rupture for augmentation and induction. Zelop *et al.*[18] reported a higher risk with induction (2.3% versus 1.0%) and even higher when used with prostaglandin (4.5%), but only the increased risk with induction with oxytocin was statistically significant. Landon *et al.*[4] found increased risks among women who had spontaneous labour augmented with oxytocin compared

Table 1. Incidence of all uterine ruptures and complete uterine ruptures in the second birth, by onset of labour, women with one previous caesarean section for four Australian states, 1998–2000

Onset of labour	Total number of women who gave birth	Uterine rupture Number (%) (95% CI)	OR and AOR for uterine rupture (95% CI)	Complete uterine rupture Number (%) (95%CI)	OR for complete uterine rupture (95% CI)	AOR for complete uterine rupture (95% CI)
Spontaneous onset of labour, no augmentation with oxytocin	8221	16 (0.19) (0.11–0.32)	1.00 (Reference group)	12 (0.15) (0.08–0.26)	1.00 (Reference group)	1.00 (Reference group)
VB achieved %	52.6					
1. No labour (elective caesarean)	18 050	4 (0.02) (0.01–0.06)	0.11 (0.04–0.34)	1 (0.01) (0.00–0.03)	0.04 (0.00–0.29)	0.04 (0.01–0.30)*
2. Spontaneous onset of labour, augmentation with oxytocin	628	12 (1.91) (0.99–3.31)	9.99 (4.71–21.21)	12 (1.91) (0.99–3.31)	13.33 (5.96–29.79)	14.04 (6.22–31.65)*
VB achieved %	61.6					
3. Labour induced, oxytocin only	735	6 (0.82) (0.30–1.77)	4.22 (1.65–10.82)	4 (0.54) (0.15–1.39)	3.74 (1.20–11.64)	3.76 (1.21–11.69)*
VB achieved %	64.5					
4. Labour induced, prostaglandins only	586	4 (0.68) (0.19–1.74)	3.52 (1.17–10.58)	4 (0.68) (0.19–1.74)	4.70 (1.51–14.62)	4.72 (1.52–14.70)*
VB achieved %	51.4					
5. Labour induced, oxytocin and prostaglandins	226	4 (1.77) (0.48–4.47)	0.24 (3.06–27.86)	2 (0.88) (0.11–3.16)	6.11 (1.36–27.45)	6.29 (1.39–28.37)*
VB achieved %	60.2					
6. Labour induced, no oxytocin or prostaglandins	320	2 (0.63) (0.08–2.24)	3.23 (0.74–14.09)	2 (0.63) (0.08–2.24)	4.30 (0.96–19.30)	4.66 (1.03–21.12)*
VB achieved %	61.2					
Labour induced, unspecified method**	242	0	–	0	–	–
VB achieved %	55.4					
Total	29 008	48 (0.17) (0.12–0.22)	–	37 (0.13) (0.09–0.18)	–	–

VB, vaginal birth.
Vaginal birth achieved % = percentage of women achieving vaginal birth in a particular subgroup.
*1 adjusted for placental abruption AOR 12.29 (1.57–95.98); two adjusted for placental abruption AOR 10.60 (1.35–83.53); three adjusted for placental abruption AOR 9.65 (1.25–74.63); four adjusted for placental abruption AOR 9.67 (1.25–74.97); five adjusted for placental abruption AOR 11.70 (1.49–91.62); six adjusted for placental abruption AOR 13.00 (1.65–102.38).
**Data from one state in 1998: distribution of these women among groups 3–6 according to their frequencies in that state in 1999–2000 would result in slightly reduced risks of rupture for these groups.

with no augmentation (0.9% versus 0.4%) and also for induction with prostaglandin and oxytocin (1.4%) and for oxytocin alone (1.1%). Macones *et al.*[14] found no significantly increased risk of rupture with induction with prostaglandin or oxytocin alone, but with their sequential use (4.54 times that of spontaneous labour). Zwart *et al.*[21] reported relative risks of around two for augmentation after spontaneous onset of labour and induction with oxytocin alone or prostaglandins alone compared with spontaneous labour.

The Lydon-Rochelle *et al.* study[5] included 1 year with misoprostol. We believe that misoprostol was not used in Australia for term induction till after 2000. The Australian Misoprostol trial only started in April 2001. The study

Table 2. Comparison of risks of uterine rupture for trial of labour and no labour among women with a previous caesarean section, major studies

Authors, year of publication and study	% Risk of uterine rupture (95% CI)		Relative risk or OR of uterine rupturefor Trial of labour versus No labour (95% CI)
	No labour	Trial of labour	
Rosen *et al.* 1991[12]: meta-analysis 32 cohort studies, USA 1982–1989, 11 417 women	1.9	1.8	OR 0.8 (0.6–1.2)
Boulvain *et al.* 1997[12]: meta-analysis 14 studies Sub-Saharan Africa 1978–1993, 4500 women	Not reported	2.1 (1.0–3.2)	
Mozurkewich and Hutton 2000[12]: meta-analysis 15 studies, 'high-income' countries 1989–1999, 47 682 women	0.16	0.39	OR 2.1 (1.45–3.05)
Guise *et al.* 2004[16]: systematic review, prospective cohort studies	–	0.38 (pooled risk) (symptomatic ruptures)	Increased risk by 0.27%
Landon *et al.* 2004[4]: prospective cohort studies 19 academic medical centres, USA 1999–2002, 33 699 women (15 801 no labour, 17 898 trial of labour)	0.0	0.7	$P < 0.001$
McMahon *et al.* 1996[15]: Nova Scotia Perinatal Database, Canada1986–1992, 6138 women (2889 no labour, 3249 trial of labour)	0.0	0.3	OR 5.2 (0.6–45.4)
Macones *et al.* 2005[14]: retrospective cohort 17 hospitals in Pennsylvania and Rhode Island, USA, 1996–2000, 25 005 women (11 299 no labour, 13 706 trial of labour)	0.004	0.98	RR 21.1 (8.6–51.5) $P < 0.001$
Lydon-Rochelle *et al.* 2001[5]: population retrospective cohort Washington State, US, 1987–1996, 20 095 women (6980 no labour, 13 115 trial of labour)	0.16	0.61	RR 3.87 (2.06–7.26)
Kwee *et al.* 2007[17]: prospective cohort 38 Netherlands hospitals (1295 no labour, 3274 trial of labour)	0.08 (symptomatic ruptures)	1.5 (symptomatic ruptures)	RR 18.99 (2.62–137.41)
Four Australian states 2009: populationretrospective cohort, 1998–2000, 29 008 women (18 050 no labour, 10 958 trial of labour)	0.02 0.01 (complete ruptures)	0.40 0.33 (complete ruptures)	RR 18.12 (6.51–50.41) RR 59.30 (8.13–432.46) (complete ruptures)

by Lydon-Rochelle *et al.* only used unvalidated ICD9 codes from hospital morbidity collections and these may overestimate uterine ruptures.[6] In our study we found that use of the perinatal data alone or the hospital morbidity collection alone to ascertain cases of uterine rupture resulted in under-ascertainment of cases. Moreover, case note review helped in the validation, e.g. three cases identified by ICD codes as uterine ruptures were merely extensions of uterine incisions during surgery.

The perinatal data in all four states are also subjected to several validation procedures during processing and validation studies have shown the data to have a high level of accuracy compared with hospital case records, particularly for socio-demographic and many variables used as co-variables.[22–24] Obstetric complications tend to be under-reported. In South Australia, comparison of perinatal data with hospital case records[22] demonstrated that nearly all the variables used in this study had values of kappa >0.75, indicating excellent agreement beyond chance. Audit of hospital morbidity data in South Australia has also reported on its overall consistency and reliability.[25]

Our study has the limitations of observational studies. There may be differences between the groups of women who have no labour and those who have a trial of labour.[4] Outcomes are also worse in women who have to have an emergency caesarean after a trial of labour.[1,3,4,15] Risk factors for uterine rupture are uterine anomalies, classical scar (rupture rate 12% versus 1% for low transverse incision, but not higher in low vertical scar), age 30 years or more, postpartum fever, more than one previous caesarean, inter-pregnancy interval of 9 months or less, oxytocin induction and prostaglandin induction.[4,5,14,18–20] A previous vaginal delivery reduces the risk.[14,17]

Our dataset did not allow a further analysis on perinatal outcome, particularly perinatal death, in the cohort. We could not study the effect of the length of inter-pregnancy interval and did not have data on the reason for the first caesarean. We also did not have data on the details of

Table 3. Comparison of risks of uterine rupture for no labour, spontaneous labour and induced labour among women with a previous caesarean section, major studies

Study name/authors	No labour	Number of women (% Risk of uterine rupture), RR or AOR (95% CI)			Induction of labour with prostaglandin
		Spontaneous onset of labour	Induction of labour	Induction of labour without prostaglandin	
Four Australian states 2009 (1998–2000 data) (complete ruptures only)	18 050 (0.01) RR 0.04 (0.00–0.29)**	8221 no oxytocin (0.15) 628 with oxytocin (1.9) RR 13.09 (5.91–29.02)**	2109 (0.57) RR 3.90 (1.75–8.66)**	1055 (0.57) RR 3.90 (1.47–10.36)** 320 without oxytocin (0.63) RR 4.28 (0.96–19.05)** 735 with oxytocin (0.54) RR 3.73 (1.21–11.53)**	812 (0.74%) RR 5.06 (1.90–13.45)** 586 prostaglandin alone (0.68) RR 4.68 (1.51–14.45)** 226 prostaglandin with oxytocin (0.88) RR 6.06 (1.36–26.93)**
Lydon-Rochelle *et al.* 2001[5]	6980 (0.16)	10 789 (0.52) RR 3.3* (1.8–6.0)	2326 (1.03) RR 6.6* (3.2–13.4)	1960 (0.77) RR 4.9* (2.4–9.7)	366 (2.45) RR 15.6* (8.1–30.0)
Zelop *et al.* 1999[18]	–	2214 (0.7) 1142 without oxytocin (0.4) 1072 with oxytocin (1.0) AOR 2.3 (0.8–7.0) compared with no oxytocin	560 (2.3)	458 with oxytocin (2.0) AOR 4.6 (1.5–14.1) compared with no oxytocin	102 (3.9) 35 prostaglandin alone (2.9) 67 prostaglandin with oxytocin (4.5)
Ravasia *et al.* 2000[19]	–	1544 (0.45)	575 (1.4) $P = 0.004$ compared with spontaneous labour	403 (0.74)	172 (2.9) RR 6.41 (2.06–19.98) compared with spontaneous labour
Landon *et al.* 2004[4]	15 801 (0)	12 694 (0.6) 6685 without oxytocin (0.4) 6009 with oxytocin (0.9) OR 2.42 (1.49–3.93)**	4708 (1.02) OR 2.86 (1.75–4.67)**	3555 (0.98) 1864 with oxytocin alone (1.1) 3.01 (1.66–5.46)**	926 prostaglandin with oxytocin (1.4) OR 3.95 (2.01–7.79)** 227 prostaglandin alone (0)
Kwee *et al.* 2006[17]	1295 (0.08)	2592 (1.04) 2056 with no uterotonic agents (0.8) 536 with oxytocin (1.9) OR 2.2 (1.04–5.0)**	682 (3.1) OR 3.8 (2.0–7.3)**	308 with oxytocin alone (1.3) OR 1.5 (0.5–4.4)**	203 with prostaglandin E_2 alone (5.9) RR 6.8 (3.2–14.3)** prostaglandin E_2 with oxytocin (4.3) OR 4.8 (1.6–4.6).

RR, relative risk.
*Reference group: women who had repeat caesarean section without labour.
**Compared with spontaneous labour without oxytocin.

induction or augmentation, e.g. the dosages and rates of administration of drugs. However, we used population databases that have been shown to have a high level of accuracy for the variables used and validated uterine rupture data from two sources.

One would hope that the final answer would come from randomised controlled trials. However, proper randomised trials are difficult because recruitment may be hampered by the high percentage of women with a fixed preference with regard to the mode of delivery of their second infant and the low frequency of clinically relevant adverse pregnancy outcome results in large sample size requirements in the order of 1000 truly randomised women per arm.

A recent large Californian study[3] with detailed data on more than 40 000 VBACs concluded that the ideal VBAC candidate should have no maternal, fetal, or placental conditions complicating pregnancy. VBAC is still a viable option but is safer with spontaneous onset of labour, in pregnant women without any significant associated perinatal or medical risk factors, and only for well-equipped hospitals with good 24-hour access to theatre for emergency caesarean section. Augmentation of spontaneous labour with oxytocin or induction of labour particularly with oxytocin and prostaglandins may increase the risk for uterine rupture of the scarred uterus.

The results derived from this large retrospective Australian study proving the risk for uterine rupture and the overall percentage achieving vaginal birth in the various subgroups may be considered for use in counselling pregnant women with a previous caesarean section who are contemplating their second birth options.

Disclosure of interest

The authors have no interests to declare.

Contribution to authorship

GAD and AC were responsible for the study design; all authors were involved with data collection and verification. GAD was responsible for validating all diagnosis of uterine rupture. GAD, AC, CGL, KP, JH and JFK were responsible for the statistical analyses and for writing the manuscript.

Details of ethics approval

The study was approved by the Human Research Ethics Committees of the South Australian Department of Health, the Aboriginal Health Council of South Australia and the Victorian Department of Human Services.

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