Trends and recurrence of placenta praevia: A population-based study

Christine L. ROBERTS,1 Charles S. ALGERT,1 Janna WARRENDORF,2 Emily C. OLIVE,3 Jonathan M. MORRIS1,4 and Jane B. FORD1
1The Kolling Institute of Medical Research, University of Sydney, Sydney, 2University of Amsterdam, Amsterdam, The Netherlands, 3Royal Women’s Hospital, Melbourne, Victoria, and 4Royal North Shore Hospital, Sydney, New South Wales, Australia

Abstract
We determined recent trends and recurrence rates of placenta praevia in 790,366 deliveries in NSW. From 2001 to 2009, the rate of placenta praevia increased by 26%, from 0.69% to 0.87% (trend P < 0.001). The placenta praevia recurrence rate in a second birth was 4.8%. Two-thirds of the increase in placenta praevia was accounted for by trends in known risk factors, and the unexplained portion may reflect changes in unidentified risk factors or in the threshold for placenta praevia diagnosis.

Introduction
Placenta praevia occurs when the placenta is implanted in the lower segment of the uterus, presenting ahead of the leading pole of the fetus.1,2 Risk factors for placenta praevia have been well documented. Factors associated with increased risk include advancing maternal age, multiparity, multiple gestation, previous placenta praevia, caesarean section and abortion, infertility treatments, smoking and cocaine use during pregnancy, gestational diabetes and male fetuses.3–7 Decreased risk of placenta praevia has been associated with gestational hypertension.3,6 Trends in placenta praevia are the result of the effects of changes in all these factors and, on balance, are likely to be driving an increase in placenta praevia rates. Contemporary estimates of recurrence risk are lacking. The aims of this study were to determine recent trends and recurrence rates of placenta praevia in an Australian population.

Materials and Methods
The study population included 790,366 deliveries (to 525,822 women) during the period 1 January 2001 to 31 December 2009. Data were obtained from linked NSW population-based birth and hospital discharge records. Birth data are from the Perinatal Data Collection, a legislated population-based surveillance system covering births _20 weeks’ gestation or _400-g birthweight. Hospital discharge data are from the Admitted Patient Data Collection (from July 2000), a census of all NSW inpatient hospital discharges (public and private) with diagnoses and procedures coded [according to the 10th revision of the International Classification of Diseases (ICD10)] for each admission based on information from the medical records. The data sets were probabilistically linked and de-identified for analysis using methods that have been described previously.8 The study was approved by the NSW Population and Health Services Research Ethics Committee.

Placenta praevia was identified by the ICD10 code O44 in the hospital data for women who were delivered by caesarean section at or after 26 weeks’ gestation.6 Women who had a vaginal birth (n = 881) were not considered to be cases in this study. Placenta praevia identification in hospital data has a sensitivity of 88–100%, specificity of 100% and positive predictive value of 100% when compared with the medical record.9,10
accurately ascertained.11–13

Analysis
The trend in placenta praevia rates from 2001 to 2009 among all deliveries was assessed using the Cochran-Armitage trend test. We used predictive modelling as described elsewhere14 to ascertain whether the observed trend could be explained by changes in the maternal risk factors over time. If the observed and predicted trends are not significantly different, this implies that the available explanatory variables account for all of the increase in placenta praevia.14 Conversely, any difference between the observed and predicted trends would be because of factors not included in the model. Crude and adjusted odds ratios and 95% confidence intervals (95% CI) were determined for placenta praevia risk factors only for 2006–2009 (so that prior uterine surgery could be included in the logistic regression model.)

The recurrence analyses were limited to women who had a consecutive first and second delivery. Women with a first delivery prior to January 2001 or nonconsecutive births (ie second birth outside NSW) were excluded. We determined the rate of the first occurrence of placenta praevia in first, second or third births and the recurrence rates for women with a history of placenta praevia in their first and/or second births using contingency table analysis. Singleton and multiple births were examined separately.

Results
From 1 January 2001 to 31 December 2009, there were 6152 (0.78%) deliveries complicated by placenta praevia, with a higher rate among multiple (1.11%) than singleton births (0.78%) [relative risk (RR) = 1.42; 95% CI 1.20–1.69] and among private (1.12%) compared with public (0.62%) patients (RR = 1.80; 95% CI 1.72–1.90). The rate of placenta praevia increased with parity: first births 0.74%; second births 0.81%; third births 0.80%; fourth births 0.88%; fifth and higher parity 1.09%. Maternal risk factors for placenta praevia for the period 2006–2009 are presented in Table 1.

From 2001 to 2009, the rate of placenta praevia increased by 26%, from 0.69% to 0.87% (trend P < 0.001). The observed and predicted trends are shown in Figure 1 and demonstrate that 65% of the increase can be accounted for by the trends in maternal age, smoking, private care, previous caesarean, multiple gestation, diabetes, hypertension and assisted reproductive technology.

The number of singleton first births over the entire period was 324,227. Of these, 147,468 (46.6%) also recorded a consecutive singleton birth before the end of the study period, and 29,693 also had a third consecutive delivery. The women who had a second birth differed from those who did not; at their first birth, they were younger (mean age 28.1 years vs 29.1 years) and less likely to have had a caesarean section (26.5% vs 31.5%) or placenta praevia (0.58% vs 0.85%).

For the 856 (0.58%) women with placenta praevia in their first singleton birth, the recurrence risk for a second birth with placenta praevia was 4.8% (Fig. 2). Only five of the 41 women with a recurrent placenta praevia in their second birth had a third birth in the study period, and two had a third placenta praevia.

Among singleton births, the rate of first occurrence of placenta praevia was significantly higher following first birth caesarean section compared with vaginal first birth (1.08% vs
0.58%, RR = 1.87, 1.65–2.12) (Fig. 2). Among the 2,049 women with a multiple birth in their second birth, the rate of first occurrence of placenta praevia following vaginal birth was 0.76% and following caesarean section was 1.49%.

Discussion
Placenta praevia rates increased by 26% between 2001 and 2009, and two-thirds of the increase was explained by increases in established risk factors. For women with a first birth placenta praevia, one in 20 will have a recurrence in their second birth. For those having a third birth, a second birth without placenta praevia does not appear to mitigate the recurrence risk. In second births, the rate of placenta praevia occurrence differed for women with multiple gestations and for women with a prior caesarean, and together, the risk was higher than either risk factor in isolation. Our restriction to delivery by caesarean section provides a conservative estimate of placenta praevia rates, but is consistent with previous research and avoids the misclassification of low-lying placenta as placenta praevia.

The increasing rates of placenta praevia were only partially explained by changes in the known risk factors. Although we did not have information on all the established risk factors for placenta praevia, the lack of information on prior uterine surgery or other unidentified risk factors is unlikely to entirely explain the difference between the observed and predicted trends in placenta praevia. We could not include uterine surgery in the trend model because there was no ‘lookback period’ for women at the start of the study period. Given the risk associated with prior uterine surgery (aOR 1.71 for the period 2006–2009), this could account for some of the placenta praevia increase if the proportion of women with a history of prior uterine surgery is increasing.

Increased ascertainment is a possible explanation for the unexplained increase in placenta praevia. However, this seems unlikely, given the high (88-100%) reported ascertainment rates. Changes in the method or threshold for diagnosis and/or intervention could also account for some of the observed increase in placenta praevia. Transvaginal scans (TVS) are more accurate than transabdominal scans (TAS) for placental localisation in second and third trimester. Thus, widespread use of TVS or Trans Perineal Scanning for confirming placental location in the 3rd trimester might be expected to decrease the rate of placenta praevia diagnoses, but information on the safety, choice of technique, and timing of such assessment in the presence of suspected placenta praevia is lacking.

There have been few population-based studies reporting the recurrence risk of placenta praevia. Two older (pre-1993) Scandinavian studies reported recurrence risks for second births of 2.3% and 2.4%, respectively. A recent study from England 2000 to 2009 had a recurrence risk of 4.3%, similar to our estimate of 4.8% for the same period. The doubling of recurrence risk since the 1970s and 1980s likely reflects the changes in diagnostic ability, from a period of largely clinical diagnosis in women with painless vaginal bleeding and a high presenting part or abnormal lie to current widespread availability of ultrasound and the choice of threshold of placental edge-os distance for undertaking a caesarean section. In conclusion, the incidence of placenta praevia has risen faster than can be explained purely by known risk factors. Population-based rates of placenta praevia occurrence and recurrence provide information for counselling. Women with placenta praevia are at high risk of haemorrhage and serious morbidities, including hypovolaemic shock, disseminated intravascular coagulopathy, renal failure, liver failure and adult respiratory distress. Delivery in a hospital with an onsite blood bank and an experienced multidisciplinary team is recommended.
Acknowledgements
We thank the NSW Ministry of Health for access to the population health data and the NSW Centre for Health Record Linkage for linking the data sets. This work was supported by a National Health and Medical Research Council Project Grant (#512162). CLR is supported by a NHMRC Senior Research Fellowship (#457078).

References