Perinatal risk factors for developmental dysplasia of the hip

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Abstract

***Aims*—To identify perinatal risk factors for developmental dysplasia of the hip (DDH) and define the risk for each factor.**

***Methods*—In this case control study, using logistic regression analysis, all 1127 cases of isolated DDH live born in South Australia in 1986-93 and notified to the South Australian Birth Defects Register were included; controls comprised 150 130 live births in South Australia during the same period without any notified congenital abnormalities.**

***Results*—Breech presentation, oligohydramnios, female sex and primiparity were confirmed as risk factors for DDH. Significant findings were an increased risk for vaginal delivery over caesarean section for breech presentation (as well as an increased risk for emergency section over elective section), high birthweight (≥4000 g), postmaturity and older maternal age; multiple births and preterm births had a reduced risk. There was no increased risk for caesarean section in the absence of breech presentation. For breech presentation, the risk of DDH was estimated to be at least 2.7% for girls and 0.8% for boys; a combination of factors increased the risk.**

***Conclusions*—It is suggested that the risk factors identified be used as indications for repeat screening at 6 weeks of age and whenever possible in infancy. Other indications are family history and associated abnormalities.**

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Keywords: congenital hip dislocation; perinatal risk factors; screening; breech presentation.

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Developmental dysplasia of the hip (DDH) embraces conditions of varying severity, from dislocated, dislocatable, or subluxatable hips to stable or clicky hips with radiological or ultrasound evidence of acetabular dysplasia. Early identification of affected infants is important for optimal outcome, as results of treatment become worse with delayed diagnosis after the neonatal period.[1] Neonatal clinical screening programmes for the condition have been operative since the 1950s,[2] but have varying levels of sensitivity. Attempts to improve sensitivity have been based on the identification of infants at increased risk[3] and ancillary procedures such as ultrasound scanning, which may identify dysplastic hips that are clinically normal.[1 3] Prevalence of the clinical condition has been reported to vary from 0.8 to 1.6 per 1000 births in populations not screened neonatally, but with high rates of 10 to 100 per 1000 births among ethnic communities, where infants are traditionally cradled or clothed with their hips extended and adducted; in screened populations, rates of 2.5 to 20 per 1000 births have been reported, but reach 40–90 per 1000 births in some communities.[4] Differences in reported prevalence may be due to genetic differences and differences in clinical skills and methods used in detection as well as definition of the condition.

DDH has been associated with other "congenital postural deformities"[5] such as sternomastoid torticollis, scoliosis, talipes, genu recurvatum, Potter's or compression facies (associated with oligohydramnios) and plagiocephaly. It has been suggested since Hippocratic times that these result from mechanical factors during later fetal life, altering the form of previously normally formed parts. This is supported by the rarity of the conditions in fetuses before 20 weeks of gestation.[5] DDH has also been associated with severe abnormalities such as meningomyelocoele, arthrogryposis, and muscular dystrophy,[5] when it is classified as pathological, teratological, or paralytic dislocation.

Family studies[6–11] have provided evidence that there is a genetic predisposition to DDH based on polygenic–multifactorial inheritance. These have shown a much higher concordance of the condition in monozygotic compared with dizygotic twins, and a significantly higher prevalence of DDH among siblings (4.3–14.0%) and parents (1.6–2.3%) of probands than would be expected from the population prevalence. Of environmental factors, breech presentation, with a prevalence of 11% to 50% in DDH[6 7 9–16] has been considered the most important since the 19th century; female sex[2 9 10 14 15 17] and primiparity[2 9 11 13 15 17] have been consistently associated with it. Postmaturity,[13 14 16] and, in single studies, upper social class,[7] and miscarriage in the previous pregnancy[18] have also been associated with it. There has been no consistent finding regarding month or season of birth,[2] maternal age,[9 11 14 17] or birthweight.[2 10 11 13 14 16 17]

The aim of the present study was to identify perinatal risk factors for DDH by linking cases with the perinatal data routinely collected on each birth in South Australia, to identify an "at risk" group of children and contribute to understanding of the aetiology of the condition.

Methods

South Australia has a population of 1.46 million people and about 20 000 births every

year. Since 1981, details of mother and baby for births occurring in the State have been routinely provided on a perinatal data collection form by midwives for the South Australian Health Commission's Pregnancy Outcome Unit, for the purpose of monitoring perinatal trends. This has been provided under legislation drawn up in 1986. The data comprise more than 99.5% of all births and include: sociodemographic details and information on previous pregnancies; medical and obstetric complications; presentation and method of delivery; birthweight; gestation; congenital abnormalities; and outcome up to 28 days of age. The notifications of congenital abnormalities identified at birth are complemented by notifications up to the child's fifth birthday to the South Australian Birth Defects Register from hospitals, special investigation and treatment centres, and practitioners dealing with children. The Register collates the statistics and reported a prevalence of DDH of 7.4 per 1000 births in South Australia for 1986–93.[19]

The present study was undertaken using the 1127 live born cases of DDH in 1986–93, including two multiple births, from which cases with major associated abnormalities had been excluded (as they may have other aetiologies, such as chromosomal abnormalities). All 164 live born cases of isolated DDH in one year (1991) which had been notified to the Register had been validated (as having dislocated/dislocatable/subluxatable hips/acetabular dysplasia) by an orthopaedic surgeon for an earlier study.[20] This earlier study identified 42 other cases of isolated DDH born in that year in South Australia by contacting all clinicians who might have been involved in identification and treatment. Thus 80% of DDH cases in 1991 had been notified to the Register and this included a larger proportion of the more severe cases. This study also showed that the perinatal risk factors identified were the same for the more severe category (dislocated and dislocatable hips) and the less severe category (subluxatable and acetabular dysplasia). This finding supported the use of all types of cases as one clinical entity in the current study. It is also assumed that diagnoses of DDH in the Register for the remaining years are also accurate. All 150 130 live births without any notified congenital abnormalities in 1986–93 were used as controls, and odds ratios were calculated using EpiInfo[21] for mother's age, race, country of birth, region of residence, parity, specific medical conditions, obstetric complications listed in the data set, and oligohydramnios, presentation and method of delivery, month of birth, birthweight, gestation, sex of baby and plurality. This study had 80% power to detect a twofold difference at the 5% level for variables with 1.0% prevalence among controls.

Variables with crude odds ratios (OR) showing significance ($P<0.05$) or borderline significance ($0.10>P\geq0.05$) in association with DDH were entered into an unconditional logistic regression analysis using SPSS for Windows[22] to determine which were independently associated after adjusting for confounding from the other variables included. All models were examined for goodness of fit, multicollinearity (using SAS/STAT[23]) and interaction among variables. Adjusted odds ratios (95% confidence limits) were obtained for the variables in the final models by taking the exponentials of the respective regression coefficients ($\pm1.96 \times$ standard error).

The coefficients obtained from logistic regression permitted the calculation of the probability of being a case, in this study the probability of having DDH, for each of the South Australian live births. The formula used for calculating the probability was:

$$p = \frac{\exp(\sum_i \alpha + \beta_i x_i)}{1 + \exp(\sum_i \alpha + \beta_i x_i)}$$

where α is the regression constant and the βi are the regression coefficients.[24] Furthermore, population attributable fractions (PAF) were calculated for possibly modifiable risk factors using the formula:

$$PAF = \frac{P(OR-1)}{P(OR-1)+1} \times 100$$

where P = prevalence of the factor in the population (from the perinatal data collection for 1986–93).

Results

The variables found to be associated with DDH ($P<0.10$) in univariate analysis (crude OR) were maternal age, region of residence, parity, oligohydramnios, presentation and method of delivery, baby's sex, birthweight, gestation and plurality. The adjusted OR for multiple births was 0.06 (0.01, 0.22) showing a 94% reduction in risk for multiple births compared with singletons. However, collinearity was found between plurality and birthweight, and all 4091 multiple births were then excluded from the analyses which are presented in table 1 for singleton births alone.

Logistic regression analysis using singleton live births showed that the following were independent risk factors for DDH, in order of strength of association (adjusted OR) (table 1).

(1) *Breech presentation*: this considerably increased the risk compared with non-breech presentation; on the other hand, caesarean section in the absence of breech presentation was not associated with an increased risk compared with vaginal delivery, OR 1.10 (0.92,1.31);

(a) *Vaginal or caesarean delivery*: the risk was significantly higher for vaginally delivered breech births, OR 17.15 (12.79, 22.99), than for breech births delivered by caesarean section, OR 10.03 (8.58,11.72).

(b) *Emergency or elective caesarean delivery*: the risk was higher for emergency section usually performed after the onset of labour, OR 13.19 (10.67,16.29), than for elective section performed as a planned procedure before the onset of labour, OR 7.56 (6.27, 9.12). (There were 116 cases, 1905 controls for emergency and 141 cases, and 2853 controls for elective caesarean, giving

crude ORs of 10.53 and 8.54, respectively.)

(2) *Oligohydramnios*: this increased the risk nearly four times, OR 3.97 (1.69, 9.35).

(3) *Female sex*: the risk was also increased nearly four times for females compared with males, OR 3.94 (3.41,4.56).

(4) *High birthweight*: an increasing risk was seen with increasing birthweight. Babies of 4000–4499 g had an OR of 1.55 (1.26, 1.91) and those of 4500 g and above had an OR of 2.67 (1.81, 3.94). Low birthweight (<2500 g) was protective.

(5) *Primiparity*: there was an increasing risk with decreasing parity, with primiparous women having more than twice the risk, OR 2.19 (1.83, 2.62), of women of parity 2 or more.

(6) *Older maternal age*: there was increasing risk with increasing age, with mothers of 30 to 34 years having an OR of 1.71 (1.26, 2.32) compared with mothers of under 20 years.

(7) *Postmaturity*: there was an increasing risk with increasing gestation above term, with postmature babies (≥42 weeks of gestation) having an OR of 1.48 (1.02, 2.13) while gestation up to 38 weeks was protective.

(8) *Metropolitan region of residence*: metropolitan, OR 1.45 (1.23, 1.69) and outer metropolitan residents, OR 1.86 (1.41, 2.46), had an increased risk compared with country residents.

Interaction occurred between sex and presentation and method of delivery. Therefore, separate models were used for each sex. The model for females (table 2) which included most DDH babies (n = 882), was remarkably similar to the model which included all babies. The same risk factors emerged, with similar levels of risk. In the model for males (table 3), on the other hand, the much reduced number of DDH cases (n = 243) and controls resulted in no significant increased risk for ages 30 years and above, parity 1, breech delivery over caesarean delivery for breech presentation, any region of residence, oligohydramnios, birthweight 4000–4499 g, or late gestation, and less consistency in the trends noted for maternal age and birthweight.

The probability of being a case for any variable or combination of variables is presented in tables 4 (females) and 5 (males), where the probability for the reference infant (maternal age <20 and parity 2 or more, country resident, non-breech presentation, absence of oligohydramnios, birthweight 3000–3499 g and gestation 40 weeks) is 0.3% for females and 0.05% for males. The population attributable fractions (PAF) for possibly modifiable risk factors—breech presentation delivered vaginally, breech presentation delivered by caesar-

Table 1 Adjusted and crude odds ratios of factors associated with DDH in South Australian singleton live births (1986-93)

	No of subjects		*Crude OR*	*Adjusted OR**	
Variables	*Cases (n=1125)*	*Controls (n=146041)*	*OR (95% CI)*	*OR (95% CI)*	*Coefficients (β) in logisitc regression analysis*
Mother's age (yrs):					
<20	52	8725	1.00	1.00	0.0000
20-24	228	32169	1.19 (0.87, 1.63)	1.30 (0.96, 1.76)	0.2618
25-29	431	5572	1.30 (0.97, 1.76)	1.46 (1.09, 1.96)	0.3798
30-34	313	36967	1.42 (1.05, 1.93)	1.71 (1.26, 2.32)	0.5360
35+	101	12608	1.34 (0.95, 1.91)	1.72 (1.22,2.44)	0.5440
Parity:					
0	624	58976	2.13 (1.79, 2.52)	2.19 (1.83, 2.62)	0.7825
1	322	51089	1.27 (1.05, 1.53)	1.29 (1.07, 1.56)	0.2553
2+	179	35976	1.00	1.00	0.0000
Presentation and delivery:					
Non-breech presentation	813	140533	1.00	1.00	0.0000
Breech, caesarean delivery	257	4758	9.34 (8.07, 10.80)	10.03 (8.58, 11.72)	2.3058
Breech, breech delivery	55	750	12.68 (9.46, 16.95)	17.15 (12.79, 22.99)	2.8420
Baby's sex:					
Female	882	70602	3.88 (3.36, 4.48)	3.94 (3.41, 4.56)	1.3717
Male	243	75439	1.00	1.00	0.0000
Region of residence:					
Metropolitan	858	102487	1.56 (1.33, 1.82)	1.45 (1.23, 1.69)	0.3690
Outer metropolitan	69	6751	1.90 (1.43, 2.52)	1.86 (1.41, 2.46)	0.6199
Country	198	36803	1.00	1.00	0.0000
Oligohydramnios:					
Present	6	209	3.74 (1.50, 8.73)	3.97 (1.69, 9.35)	1.3790
Absent	1119	145832	1.00	1.00	0.0000
Birthweight (g):					
<2000	6	2260	0.33 (0.13, 0.77)	0.30 (0.12, 0.77)	−1.1971
2000-2499	17	4468	0.48 (0.28, 0.79)	0.52 (0.31, 0.88)	−0.6469
2500-2999	174	21782	1.00 (0.84, 1.20)	0.90 (0.75, 1.08)	−0.1066
3000-3499	440	55214	1.00	1.00	0.0000
3500-3999	335	45627	0.92 (0.80, 1.07)	1.12 (0.97, 1.30)	0.1133
4000-4499	124	14318	1.09 (0.89, 1.33)	1.55 (1.26, 1.91)	0.4367
4500+	29	2372	1.53 (1.03, 2.27)	2.67 (1.81, 3.94)	0.9819
Gestation (weeks):					
<37	24	7428	0.44 (0.28, 0.67)	0.42 (0.25, 0.69)	−0.8703
37	31	5992	0.70 (0.48, 1.02)	0.59 (0.40, 0.86)	−0.5299
38	136	17993	1.02 (0.84, 1.24)	0.80 (0.66, 0.98)	−0.2214
39	224	25133	1.20 (1.03, 1.41)	0.90 (0.76, 1.06)	−0.1035
40	517	69833	1.00	1.00	0.0000
41	161	16862	1.29 (1.08, 1.55)	1.26 (1.05, 1.51)	0.2311
42+	32	2800	1.54 (1.06, 2.24)	1.48 (1.02, 2.13)	0.3891
Constant					−7.2099

*Logistic regression analysis.

ean section and for postmaturity—are presented in table 6.

Discussion

The perinatal data used in this study have already been shown to have a very high level of reliability for variables which were risk factors in the study—mother's age and parity, presentation and method of delivery, sex, plurality, birthweight and gestation, with κ values of 0.85 to 1.00 when compared with hospital case records.[25] Kappa values of greater than 0.75 indicate excellent agreement beyond chance.[26] The reliability of these perinatal data and the demonstration in the study of cases of DDH in 1991 of the uniformity of risk factors for both milder and more severe categories of DDH supported the use of data for 1986–93.

The trends in the odds ratios suggestive of a dose–response relation in age, parity, birthweight and gestation, provide strong support for the associations noted. The results confirm those of other studies in relation to breech presentation, female sex, oligohydramnios and primiparity as risk factors, but there are some new findings or risk factors that have had little previous support. These are: breech delivery increasing the risk for breech presentation; high birthweight; older maternal age; postmaturity; and metropolitan residence. Interestingly, in a Western Australian study which used Statewide perinatal data and a univariate analysis,[14] the increasing risks with increasing age and gestation were also noted.

It has been suggested that risk factors operate through increasing pressure on the fetus or through decreasing resistance to dislocation.[4] In primiparity, the high tone of the previously undistended uterine wall would restrict the fetus, as would high birthweight or the reduction of amniotic fluid in oligohydramnios and postmaturity; it is not clear how an increase in maternal age results in suboptimal fetal accommodation. An association between older maternal age and amniocentesis was explored by including amniocentesis in the logistic regression analysis, but it did not emerge as a risk factor (OR 0.62 (95% CI 0.30, 1.28)). This study found that preterm birth (<37 weeks of gestation) and multiple birth both reduced the risk of DDH (by 58% and 94%, respectively). The lower risk in early gestation may be related to the greater mobility of the smaller fetus in a relatively larger volume of amniotic fluid. Multiple births are of lower birthweight than singletons and showed collinearity with birthweight in the logistic regression analysis. The prevalence of DDH among multiple births in this study was 0.5 per 1000 live births compared with 7.7 for singletons.

It has been suggested that high concentrations of relaxin in DDH may contribute to connective tissue and hip joint laxity. This facilitates hip displacement, which interferes with the leg folding mechanism, resulting in breech malposition (with hips flexed and knees extended).[5 6] A recent study showed no signifi-

Table 2 Adjusted and crude odds ratio of factors associated with DDH in South Australian Singleton female live births (1986-93)

Variables	*No of subjects*		*Crude OR*	*Adjusted OR**	*Coefficients (β) in logisitc regression analysis*
	Cases (n=882)	*Controls (n=70602)*	*OR (95% CI)*	*OR (95% CI)*	
Mother's age (yrs):					
<20	44	4160	1.00	1.00	0.0000
20-24	181	15694	1.09 (0.77, 1.54)	1.21 (0.86, 1.69)	0.1888
25-29	314	26703	1.11 (0.80, 1.55)	1.25 (0.90, 1.72)	0.2196
30-34	262	18044	1.37 (0.99,1.92)	1.68 (1.21, 2.35)	0.5196
35+	81	6001	1.28 (0.87, 1.88)	1.63 (1.11, 2.40)	0.4907
Parity:					
0	482	28382	2.06 (1.70, 2.50)	2.17 (1.77, 2.65)	0.7744
1	256	24736	1.26 (1.02,1.55)	1.30 (1.06, 1.61)	0.2657
2+	144	17484	1.00	1.00	0.0000
Presentation and delivery:					
Non-breech presentation	655	67683	1.00	1.00	0.0000
Breech, caesarean delivery	185	2507	7.63 (6.42, 9.05)	8.65 (7.21,10.38)	2.1576
Breech, breech delivery	42	412	10.53 (7.49,14.76)	15.10 (10.79, 21.13)	2.7147
Region of residence:					
Metropolitan	672	49687	1.58 (1.32, 1.90)	1.48 (1.23, 1.77)	0.3901
Outer metropolitan	59	3222	2.15 (1.57,2.93)	2.12 (1.56, 2.89)	0.7521
Country	151	17693	1.00	1.00	0.0000
Oligohydramnios:					
Present	5	116	3.46 (1.25, 8.81)	4.07 (1.59, 10.43)	1.4030
Absent	877	70486	1.00	1.00	0.0000
Birthweight (g):					
<2000	6	1099	0.44 (0.18, 1.02)	0.36 (0.14, 0.95)	−1.0147
2000-2499	14	2480	0.46 (0.26, 0.80)	0.51 (0.28, 0.90)	−0.6819
2500-2999	142	12573	0.91 (0.75, 1.11)	0.88 (0.71, 1.08)	−0.1320
3000-3499	356	28751	1.00	1.00	0.0000
3500-3999	255	19901	1.03 (0.88, 1.22)	1.13 (0.95, 1.33)	0.1185
4000-4499	89	5115	1.41 (1.10, 1.79)	1.60 (1.26, 2.04)	0.4694
4500+	20	683	2.36 (1.46, 3.80)	2.95 (1.85, 4.70)	1.0801
Gestation (weeks):					
<37	21	3441	0.51 (0.32, 0.80)	0.47 (0.27, 1.23)	−0.7533
37	23	2829	0.68 (0.43, 1.05)	0.58 (0.38, 0.91)	−0.5392
38	102	8782	0.97 (0.77, 1.21)	0.78 (0.62, 0.98)	−0.2484
39	173	12320	1.17 (0.97, 1.40)	0.89 (0.74, 1.08)	−0.1115
40	410	34077	1.00	1.00	0.0000
41	128	7873	1.35 (1.10, 1.66)	1.28 (1.04, 1.56)	0.2433
42+	25	1280	1.62 (1.06, 2.48)	1.49 (0.98, 2.25)	0.3958
Constant					−5.7437

*Logistic regression analysis.

cant difference in mean relaxin concentration between cord bloods of a group of 24 babies with DDH and a group of normal babies matched by gestation and sex; however, the authors suggest that relaxin receptor expression of the developing fetal hip joint needs to be explored.[27]

Earlier studies have not explored the association between caesarean delivery and DDH in the absence of breech presentation, yet it has been assumed that caesarean section is a risk factor.[1 3] They have also not explored the difference between elective and emergency caesarean section nor used a multivariate analysis on all available data: conflicting results concerning the prevalence of DDH among vaginally delivered compared with caesarean delivered breech babies have been reported,[12 16 28] some studies being limited by small numbers.

Table 3 Adjusted and crude odds ratios of factors associated with DDH in South Australian singleton male live births (1986-93)

Variables	No of Subjects		Crude OR	Adjusted OR*	Coefficients (β) in logisitc regression analysis
	Cases (n=243)	Controls (n=75439)	OR (95% CI)	OR (95% CI)	
Mother's age (yrs):					
<20	8	4565	1.00	1.00	0.0000
20-24	47	16475	1.63 (0.74, 3.73)	1.74 (0.82, 3.71)	0.5545
25-29	117	28869	2.31 (1.09, 5.10)	2.58 (1.25,5.34)	0.9492
30-34	51	18923	1.54 (0.70, 3.50)	1.75 (0.82, 3.75)	0.5614
35+	20	6607	1.73 (0.72, 4.27)	2.13 (0.92, 4.95)	0.7568
Parity:					
0	142	30594	2.45 (1.67, 3.62)	2.25 (1.52, 3.33)	0.8100
1	66	26353	1.32 (0.86, 2.04)	1.24 (0.82, 1.88)	0.2147
2+	35	18492	1.00	1.00	0.0000
Presentation and delivery:					
Non-breech presentation	158	72850	1.00	1.00	0.0000
Breech, caesarean delivery	72	2251	14.75 (11.02, 19.72)	16.35 (12.06, 22.16)	2.7942
Breech, breech delivery	13	338	17.73 (9.53, 32.36)	28.72 (15.91, 51.85)	3.3576
Region of residence:					
Metropolitan	186	52800	1.43 (1.03, 2.00)	1.35 (0.97, 1.87)	0.2984
Outer metropolitan	10	3529	1.15 (0.55,2.36)	1.07 (0.54, 2.12)	0.0641
Country	47	19110	1.00	1.00	0.0000
Oligohydramnios:					
Present	1	93	3.35 (0.46, 24.11)	3.47 (0.44, 27.49)	1.2435
Absent	242	75346	1.00	1.00	0.0000
Birthweight (g):					
<2000	0	1161	0.00 (0.00, 1.31)	0.02 (0, 82.96)	−4.0938
2000-2499	3	1988	0.48 (0.12, 1.56)	0.69 (0.43, 2.35)	−0.3775
2500-2999	32	9209	1.09 (0.71, 1.67)	1.09 (0.71, 1.66)	0.0865
3000-3499	84	26463	1.00	1.00	0.0000
3500-3999	80	25726	0.98 (0.71, 1.35)	1.10 (0.80, 1.51)	0.0953
4000-4499	35	9203	1.20 (0.79, 1.81)	1.45 (0.96, 2.18)	0.3688
4500+	9	1689	1.68 (0.79, 3.45)	2.44 (1.21, 4.96)	0.8940
Gestation (weeks):					
<37	3	3987	0.25 (0.06, 0.82)	0.24 (0.07, 0.83)	−1.4105
37	8	3163	0.85 (0.38, 1.79)	0.56 (0.26, 1.20)	−0.5754
38	34	9211	1.23 (0.82, 1.84)	0.86 (0.57, 1.30)	−0.1471
39	51	12813	1.33 (0.94, 1.88)	0.95 (0.67, 1.35)	−0.0497
40	107	35756	1.00	1.00	0.0000
41	33	8989	1.23 (0.81, 1.84)	1.21 (0.81, 1.80)	0.1873
42+	7	1520	1.54 (0.66, 3.43)	1.41 (0.65, 3.06)	0.3408
Constant					−7.6060

*Logistic regression analysis.

Table 4 Risk of DDH: female baby

Risk factors	Breech presentation		Oligohydramnios	High birthweight (≥ 4500g)	Primipara	Age 35+	Post-maturity
	Caesarean delivery	Vaginal delivery					
No other risk factor	2.7%	4.6%	1.3%	0.9%	0.7%	0.5%	0.5%
+Oligohydramnios	10.1%	16.4%		3.7%	2.7%	2.1%	1.9%
+High birthweight	7.5%	12.5%			2.0%	1.5%	1.4%
+Primipara	5.7%	9.5%				1.1%	1.0%
+Age 35+	4.3%	7.3%					0.8%
+Postmaturity	4.0%	6.7%					
+Oligohydramnios +high birthweight	24.9%	36.7%			7.7%	5.9%	5.4%
+Oligohydramnios +primipara	19.6%	29.9%				4.4%	4.0%
+High birthweight +primipara	15.0%	23.6%				3.2%	3.0%
+Age 35+ +postmaturity	6.3%	10.5%	3.1%	2.2%	1.7%		
+Oligohydramnios +age 35+	15.5%	24.3%					
+Oligohydramnios +postmaturity	14.3%	22.6%					
+High birthweight +age 35+	11.8%	18.9%					
+High birthweight +postmaturity	10.8%	17.5%					
+Primipara +age 35+	8.9%	14.6%					
+Primipara +postmaturity	8.2%	13.5%					

Table 5 Risk of DDH: male baby

Risk factors	*Breech presentation*		*Oligohydramnios*	*High birthweight (≥ 4500g)*	*Primipara*	*Age 35+*	*Post-maturity*
	Caesarean delivery	*Vaginal delivery*					
No other risk factor	0.8%	1.4%	0.2%	0.1%	0.1%	0.1%	0.1%
+Oliogohydramnios	2.7%	4.7%		0.4%	0.4%	0.4%	0.2%
+High birthweight	1.9%	3.4%			0.3%	0.3%	0.2%
+Primipara	1.8%	3.1%				0.2%	0.2%
+Age 35+	1.7%	3.0%					0.1%
+Postmaturity	1.1%	2.0%					
+Oligohydramnios +high birthweight	6.5%	10.8%			0.9%	0.9%	0.6%
+Oligohydramnios +primipara	6.0%	10.0%				0.8%	0.5%
+High birthweight +primipara	4.3%	7.3%				0.6%	0.4%
+Age 35+ +postmaturity	2.4%	4.1%	0.5%	0.4%	0.3%		
+Oligohydramnios +age 35+	5.7%	9.6%					
+Oligohydramnios +postmaturity	3.8%	6.5%					
+High birthweight +age 35+	4.1%	6.9%					
+High birthweight +postmaturity	2.7%	4.7%					
+Primipara +age 35+	3.8%	6.4%					
+Primipara, +postmaturity	2.5%	4.3%					

This study has shown no increased risk for DDH for caesarean delivery in the absence of breech presentation. Interestingly, 85% of singleton breech presentations were delivered by caesarean section. Breech presentation imposes at least a sevenfold increased risk (that of breech delivered by elective caesarean section), but the risk for breech delivered vaginally is considerably greater (17 times that for non-breech presentation). The risk for breech delivery by emergency caesarean section is intermediate, suggesting that external forces during parturition affect hip joint stability. It could be argued that it is only the truly dislocated or dislocatable hip which is affected in this way by delivery method, and that the breech position for several months' gestation produces the acetabular dysplasia by a gentle moulding process. The association of type of breech presentation—breech with extended legs, with an increased risk for breech delivery—could not be evaluated, as examination of a sample of 47 case records of mothers of breech presentation DDH babies showed that in many cases the reason for delivery by caesarean section was not related to the type of breech presentation but to factors such as maternal age, inadequate pelvis, large baby, previous caesarean section, failure to progress, fetal distress and maternal request. Frequently, the type of breech presentation was also not noted in the case records. As the prevalence of breech delivered vaginally is only 0.9% among live births in South Australia, using population attributable fractions (table 6), the estimated reduction in prevalence of DDH as a result of delivering these breech presentations by caesarean section would be only 5.2% (12.7–7.5%). In any case, in the absence of other specific benefits of caesarean delivery[29] and the fact that DDH is readily treatable, such a recommendation would be untenable. Another factor to consider is whether external cephalic version of a breech presentation baby at 38 weeks of gestation would result in a reduction in risk of DDH. If this risk were eliminated, then the potential reduction in DDH prevalence from converting all breech to non-breech presentations (which cannot be achieved) would be 38.7% (12.7%+26.0%) (table 6), which is quite substantial. The identification of these risk factors supports the hypothesis that, although a genetic predisposition to DDH exists, DDH is probably a deformation occurring in later gestation in utero which may be aggravated by the passage through the birth canal in breech presentation. These findings need to be replicated in other studies. While boys have a much lower risk of DDH than girls (3.2 *vs* 12.3 per 1000 singleton live births), and also a lower risk of breech presentation (3.5% *vs* 4.4% being breech presentations in this study), the level of increased risk of DDH for boys with breech presentation was almost twice that for girls (OR 16.35 *vs* 8.65 for caesarean delivery) (tables 2 and 3).

Table 6 Population attributable fraction (PAF) for possibly modifiable risk factors

Risk factor	*Population prevalence*	*Adjusted OR*	*PAF*
Breech presentation delivered vaginally	0.009	17.15	12.7%
Breech presentation delivered by caesarean (those currently delivered vaginally only)	0.009	10.03	7.5%
Breech presentation currently delivered by caesarean	0.039	10.03	26.0%
Postmaturity	0.019	1.48	0.9%

The finding of an association between metropolitan or outer metropolitan residence and DDH is consistent with the higher prevalence among babies born in metropolitan hospitals in Western Australia.[14] This may be related to better reporting in metropolitan regions: the prevalence of total birth defects is higher in these regions, but there is no increased prevalence for sentinel defects (a group of severe defects readily identifiable at

birth and used internationally for monitoring purposes).[19]

We had no information on family history, which is believed to exercise its influence either through generalised joint laxity (more common in neonatally diagnosed cases), or the development of acetabular dysplasia (more common in late diagnosed cases, in which environmental factors have a smaller role).[7 10 14]

The neonatal screening programme in South Australia, using the Ortolani and Barlow tests, as in many developed countries, is a part of the routine physical examination of the neonate by a medical practitioner shortly after birth. We recommend further screening in infancy of those with normal tests who have identified risk factors. Examinations on certain occasions entail little additional costs, while considerable expense would be imposed by a special visit to a paediatrician or an orthopaedic surgeon or an ultrasound examination for all babies. These occasions include the following:

(1) before discharge from hospital after birth (as additional cases may be detected)[30]
(2) when babies are reviewed at about 6 weeks of age by paediatricians, obstetricians, and midwives; and
(3) the visits to the Child and Youth Health (or well baby) clinic.

It would therefore be reasonable to target an educational programme to all medical practitioners, particularly obstetricians and paediatricians, and child health nurses and midwives. We recommend that this programme include information on DDH, the importance of early detection, the clinical method of screening, the risk factors, and the need for repeated screening (to increase sensitivity[31]) for "at risk" groups until the child is walking, and for referral to an orthopaedic surgeon when DDH is suspected. Perinatal information is included in the personal health record of the baby which is provided by each obstetric unit on discharge after birth, and this record could be used to advise parents of "at risk" births to facilitate programme implementation.

The principal "at risk" groups would be those identified in the study. If we use a risk estimate of 1% for DDH derived from the logistic regression analysis as a basis for repeated screening, tables 4 and 5 suggest that the baby should be a breech presentation (risk at least 2.7% for girls and 0.8% for boys) to be included. This would apply to 4–5% of South Australian babies. Combinations of risk factors, a family history in first degree relative/s, and the associated abnormalities mentioned would warrant inclusion of other babies.

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