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Part 2

Origin of OBPL and natural history

In 1746, British obstetrician William Smellie was called to a difficult labour due to a face presentation. The child was delivered by forceps, but "the long compression had rendered the arms paralytic for several days". This case is generally accepted to be the first description of obstetric brachial plexus palsy.

William Smellie, Treatise on the Theory and Practice of Midwifery. 1754

Chapter 4

Correlating birthweight with neurological severity of obstetric brachial plexus lesions

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Objective To investigated the nature and extent of neurosurgically treated obstetric plexus lesions with obstetric and neonatal precedents.

Design Retrospective analysis of prospectively collected data.

Setting Leiden, the Netherlands.

Population A 9-year cohort of infants (n=206) neurosurgically treated for obstetric brachial plexus lesion at a tertiary referral centre for nerve lesions.

Method Obstetric and neonatal data (parity, diabetic status, pregnancy gestation, mode of cephalic delivery and birthweight) were collected using a standardised protocol and correlated to neurological severity of the brachial plexus lesion.

Main outcome measure Neurological severity of the brachial plexus lesion.

Results Nulliparous women delivered significantly lower birthweight newborns ($p=0.016$), injuries in those infants were associated with the least severe injury classification. The most prominent association in ordinal logistic regression was between neurological injury severity and larger birthweight ($p<0.001$).

Conclusions Birthweight is correlated with neurological severity of the injury in a group of infants experiencing brachial plexus injury resulting from cephalic vaginal delivery.

Many obstetric studies on brachial plexus injury focus on shoulder dystocia and its risk factors and management and, more recently, on injury prevention.[1-4] From a paediatric perspective, microsurgical papers on obstetric brachial plexus lesion (OBPL) focus solely on permanent lesions, on indication and timing for surgery and on treatment outcome.[5-11] Only seldom are obstetric data combined with the intraoperative pathological findings of the nerve lesion or the neurological severity of the injury.

In previous studies examining the obstetric precedents of neurosurgically treated children, a subgroup of infants with OBPL had a specific pattern of nerve lesion: C5 and C6 nerves of infants born in breech delivery were more often torn from the spinal cord (root-avulsion) than ruptured at the level of the brachial plexus (neurotmesis) in comparison with infants born with a cephalic presentation.[12,13] Because of substantial injury risk, obstetricians in many countries have virtually stopped performing breech deliveries for term infants.[14]

The largest group of infants with OBPL, however, consists of macrosomic infants born in cephalic presentation.[15-18] High birthweight, shoulder dystocia and forceful downward traction during delivery have been previously identified as the strongest risk factors for the occurrence of an OBPL.[18-22] Additionally, infants with a permanent injury were shown to have had a higher birthweight compared with those with a transient injury (4519 versus 4143 g).[20,23]

Detailed research to relate obstetric antecedents with the neurological *severity*, i.e. the number of spinal nerves that was damaged, has been performed twice. With 104 infants, Poggi et al[24] concluded that operative delivery does not worsen the degree of injury. Mollberg et al[20] found a correlation between the amount of downward traction and injury severity. No study has systematically examined the relationship of obstetric factors and birthweight with the neurological severity of the OBPL. In the present study, we classified OBPL severity as a lesion of the upper part of the brachial plexus (C5, C6), a lesion comprising both the upper and middle part of the brachial plexus (C5-C7) or a global lesion (C5-T1). The objective of the present paper is to determine whether there is a relationship between obstetric characteristics and birthweight and the neurological severity of the lesion in a cohort of surgically treated infants with OBPL.

Methods

All neurosurgically treated infants that were evaluated for an OBPL in our tertiary referral centre for nerve lesions during a 9-year period (1 January 1994 – 31 December 2002) were analysed (n=228). Although there is debate in the neurosurgical community about criteria for surgery[11] our decision to perform surgery was based on the following two criteria: either a complete paralysis of the arm and hand at 3 months of age or because recovery of shoulder function and of the biceps muscle did not occur between 4 and 6 months, as outlined in previous reports.[5,6]

The severity of neurological impairment was determined by clinical examination at a mean age of 3.4 months and verified during surgery (mean age 5.9 months). The

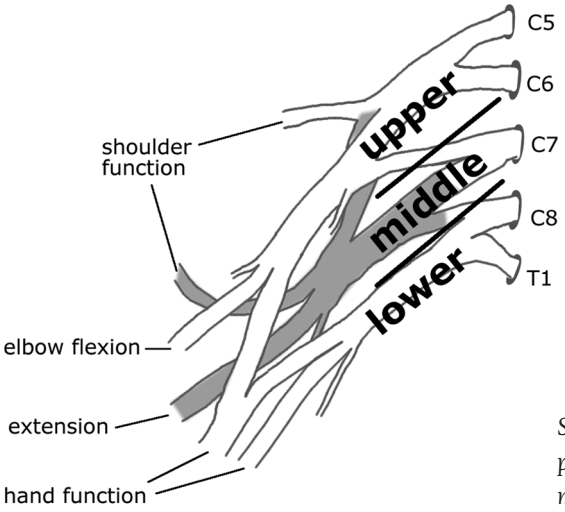
neurological examination was performed by a neurosurgeon experienced at the examination of infants with OBPL (either WP or MJAM). When a paralysis was found of abduction, elevation and external rotation of the upper arm, together with paralysis of elbow flexion and supination, only the spinal nerves C5 and C6 ('upper plexus') were involved in the lesion. When an additional weakness of the extensors of the wrist and fingers was present, the spinal nerve C7 was also clinically involved ('upper and middle plexus'). When flexion of the fingers was weak or absent, C8 or both C8 and T1 were involved as well ('upper, middle and lower plexus') (Figure 1). In this way, the infants were divided into three groups depending on the neurological examination: C5-C6 lesions; C5-C7 lesions and C5-T1 lesions.

Routinely, characteristics of gestation and labour were collected from the parents according to a standardised questionnaire developed by the treating surgeons. This protocol included parity, pregnancy history, gestation length, diabetic status, mode of delivery, and birthweight.

Infants that were born in breech presentation were excluded. Using recent Dutch epidemiological data, birthweights in this study were compared with national birthweights (corrected for gender, parity, and length of pregnancy).[25] In this surgical group, two infants had a sibling that had been neurosurgically operated because of an OBPL.

The SPSS package (version 17; SPSS Inc, Chicago, IL, USA) was used for statistical analysis. Tabular categorical data were analysed using Pearson's chi-square test; comparison of means was performed using the independent t-test. Ordinal logistic regression[26] was applied to model the dependence of the severity of the nerve lesion (defined by neurological examination) on a set of dichotomous and continuous parameters (birthweight, parity, gender, length of pregnancy, assisted delivery, side of lesion, age

Figure 1: **The brachial plexus**



Semi-schematic drawing of the brachial plexus divided in upper (C5-C6), middle (C7) and lower (C8-T1) parts

at examination). This regression results in calculation of odds ratios to assess the impact of the parameters. A level of $p<0.05$ was considered significant.

Results

In all, 206 infants that were born in cephalic presentation were included in the study. A further 22 infants were neurosurgically treated in this period for breech-delivery-associated OBPL, no infants were seen where the OBPL had resulted from caesarean section. The study group consisted of 94 males and 112 females, with 85 left-sided lesions (41%) and 121 right-sided lesions (59%). Ninety-two assisted deliveries had been performed. Mean pregnancy length was 39.8 weeks; 60 women were nulliparous, the others were multiparous. Patient details and missing data are provided in Table 1.

Birthweights were available in 98% of the patient population. The range was from 2895 to 5900 g. Of the infants, 73% had birthweights > 4000 g, 38% were > 4500 g. The weight distributions by 500 g increments are shown in Table 2. For 14 infants (6.7%)

Table 1: **Patient data**

	group			missing (n)
	C5-C6	C5-C7	C5-T1	
Group size (n)	63	98	45	-
Male/Female (n) *	29/34	39/59	26/19	-
Diabetes (%) *	14%	17%	29%	10
Parity (nulliparous / multiparous) **	27/36	24/72	9/36	2
Length of pregnancy (mean ± SD; weeks)*	39.8 ± 1.6	39.8 ± 1.5	39.9 ± 1.5	9
Instrumental delivery (%)*	49%	43%	42%	9
Birthweight (mean ± SD; g)***	4153 ± 479	4327 ± 588	4638 ± 474	4
Affected side (left / right; n)	22/41	49/49	14/31	-
Age (mean ± SD; months) *	3.4 ± 1.7	3.5 ± 2.6	3.1 ± 1.7	-

group: neurological impairment divided in three groups
* not statistically significant
** p=0.016 (chi-square test)
*** C5-C6 vs. C5-C7 p=0.052; C5-C7 vs. C5-T1 p=0.003; C5-C6 vs. C5-T1 p<0.001.(t-test)

Table 2: **Birthweight distribution and diabetic status**

birthweight	n	diabetic status		
		yes	no	?
unknown	4		3	1
2500 – 2999	1		1	
3000 – 3499	11	2	8	1
3500 – 3999	39	4	34	1
4000 – 4499	73	17	51	5
4500 – 4999	52	10	41	1
5000 – 5499	20	5	14	1
5500 – 5999	6	2	4	
total	206	40	156	10

Values in the shaded area represent the cutoff values according to North-American and British guidelines for macrosomia in relation to caesarean section (n=36)

the birthweight percentile could not be determined because of one or more missing variables. Of the total group, 53% of the birthweights were above the 97.7th percentile (2 standard deviations above mean). Birthweight was statistically different in the three neurological severity groups (t-test, equal variance: C5-C6 versus C5-C7 p=0.052 NS; C5-C7 versus C5-T1 p=0.003; C5-C6 versus C5-T1 p<0.001). Nulli-parity was unequally distributed between the groups (chi-square test, p=0.016). No statistical difference was noted in distribution of diabetes (chi-square test, p=0.186), instrumented delivery (chi-square test, p=0.715), length of pregnancy (t-tests), age at neurological diagnosis (t-tests). A possible confounder was parity: nulliparous women had children with a lower mean birthweight (4133 g), compared with multiparous women (4424 g) (t-test; p<0.001).

In the multivariable ordinal regression model using the three groups only birthweight (p<0.001) and parity (p=0.042) remained as statistic significant variables. An odds ratio of 2.7 was calculated for the continuous risk factor birthweight: the effect of an increase in birthweight of 1 kg, the odds ratio was 2.7 that this infant would sustain a more severe nerve injury. Because of missing data of one or more variables, the multivariable model included 181 / 206 infants (88%). Gender, length of pregnancy, diabetic status, assisted delivery and affected side proved not to be statistically significant, both in multivariable and univariable logistic regressions. Table 3 presents the results from multivariable and univariable analysis of the ordinal regression model.

In the group under study, two infants had a sibling that had also undergone neurosurgery because of an OBPL. To avoid the impression that the data from the siblings could result in data-clustering, the statistical analysis was repeated excluding these infants. The results were equivalent.

Table 3: **Ordinal logistic regression model**

Factor	Test value	multivariable model				univariable models			
		OR	95% CI		p	OR	95% CI		p
			min	max			min	max	
Male/Female	Male	0.92	0.51	1.65	0.776	1.28	0.77	2.15	0.341
Diabetes	Yes	1.54	0.70	3.39	0.281	1.82	0.94	3.52	0.076
Parity	Nulliparous	0.46	0.23	0.93	0.029*	0.44	0.25	0.79	0.006*
Length of pregnancy	(weeks)	0.90	0.73	1.11	0.330	1.02	0.86	1.21	0.831
Instrumented delivery	Yes	1.50	0.80	2.84	0.209	0.92	0.54	1.56	0.766
Birthweight	(kg)	2.71	1.52	4.83	0.001*	2.79	1.70	4.56	0.000*
Affected side	Left	1.26	0.71	2.26	0.430	1.00	0.59	1.68	0.990
Age	(months)	0.95	0.83	1.09	0.469	0.96	0.86	1.08	0.545

gender, diabetic status, parity, instrumental delivery, and affected side were entered as dichotomous variables; length of pregnancy, birthweight and age as continuous variables to predict the outcome of neurological severity in ordinal scale (C5-C6/C5-C7/C5-T1); OR – Odds Ratio; 95% CI – 95% confidence interval of OR; * statistically significant values

Discussion

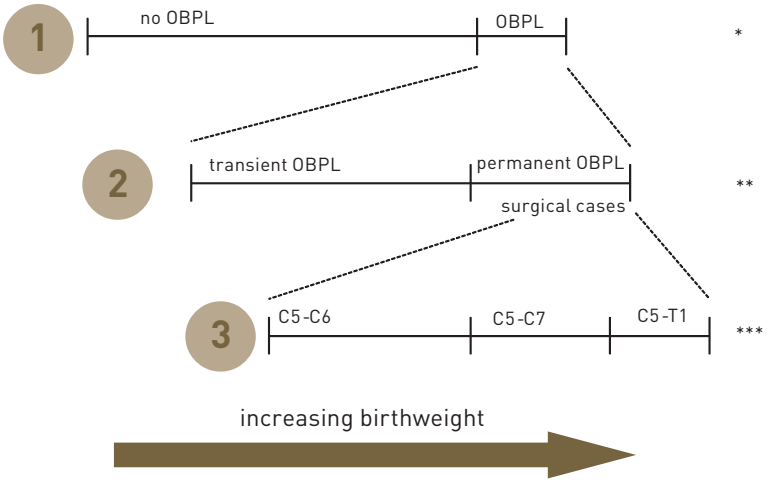
In this neurosurgical series of 206 OBPL infants, it is shown that the neurological severity of the OBPL correlated with the birthweight of the child. Although disproportion between the fetal passenger and the pelvic passageway is a well-known principal risk factor for the *occurrence* of shoulder dystocia leading to permanent OBPL, it is now established that it is also a risk factor for the *neurological severity* of the permanent OBPL.

This disproportion probably reflects the severity of the shoulder dystocia and the probably stronger clinician-applied traction that occurred during birth.[20] That the traction was usually downward comports with the 60% right sided injury in our population as left occipitoanterior or left occipitotransverse presentation occurs more frequently (less than twice as often) than right occipitoanterior or right occipitotransverse.

The current standard method to assess the size of a newborn is its birthweight. It is well established that a higher birthweight results in a higher risk of shoulder dystocia-related OBPL.[1,15-18,27,28] In a case-control study, with birthweight as control variable. no additional risk factors could be identified for the occurrence of an OBPL.[29] So on a first level to analyse the relationship between birthweight and OBPL, it is well established that increasing birthweight leads to a higher risk of an OBPL. (Figure 2)

On a second level, those infants in which an OBPL has occurred are investigated. Nehme et al[30] previously established that lighter birthweight is associated with

Figure 2: **Three levels of relationships of birthweight and OBPL**



Increasing birth weight leads to (1) increased risk of OBPL, *(2) increased risk of permanent* OBPL *and (3) increased neurological severity of surgically treated* OBPL. * [18,27] ** [23,30] *** *present study (Figure was not published in original paper)*

whether spontaneous recovery would occur. Gherman et al[23] have confirmed that birthweight is higher in infants with a permanent brachial plexus injury than in those with a transient paresis. So, on a second level, a higher birthweight is correlated with a permanent OBPL (hence more severe stretch injury) compared with a transient OBPL. On the third level – the present study – we focused on children with a surgically-warranted permanent brachial plexus injury who were born in cephalic presentation by vaginal birth. In this subgroup we found a clear *dose-effect gradient*[31] between birthweight and neurological severity of the OBPL.

This dose-effect gradient for trauma mechanism is in accordance with two historical papers describing mechanical experiments with stillborn fetuses.[32,33] Increasing lateral traction to the head with fixed shoulders resulted in a stretch injury of the brachial plexus. First, the upper part of the brachial plexus was injured, and with subsequently increasing traction, the middle and lower parts were also injured. This pattern of injury is well recognised by surgeons who treat brachial plexus stretch injuries in paediatric and adult patients.

Fetuses heavier than 4000 g are more likely to encounter a shoulder dystocia delivery, which often results in more forceful traction applied to the fetal head, and greater risk for temporary and permanent injury.[19-23] When forceful traction or twist is applied to the fetal head, the angle between neck and shoulder is widened, thereby hyperextending the ipsilateral brachial plexus. Recently, forceful downward traction expressed on a visual analogue scale was identified as the only independent risk factor for OBPL, and its neurological severity.[19,20]

Similarly, it has been shown that an operative delivery, and also duration of the instrumental delivery, increases the risk of shoulder dystocia and the associated increased risk of permanent OBPL.[28,34] That operative delivery does not factor into the severity of the injury confirms previous results.[24]

Prevention of shoulder dystocia and the associated brachial plexus lesion is difficult. Although recent results demonstrate that reducing birthweight and the incidence of shoulder dystocia in a population of women is possible with tight glycaemic control[35], it is not possible to predict shoulder dystocia in a specific infant. Many shoulder dystocia deliveries occur in women with no risk factors and many high-risk women (such as those who carry a macrocosmic fetus, who are diabetic and who have experienced a prior shoulder dystocia) will not encounter shoulder dystocia. Prophylactic caesarean section is the only way to prevent shoulder dystocia and related permanent OBPL, and yet caesarean section has its own risks to mother and newborn, and is not cost-effective on a widespread scale.[36-39] Calculations have been presented that to prevent one permanent OBPL, between 1000 and 10 000 caesarean sections would be necessary.[37,40]

Treatment guidelines state that caesarean delivery may be discussed with the woman for suspected foetal macrosomia with estimated fetal weights greater than 5000 g in women without diabetes (according to guidelines in North America) and

greater than 4500 g in women with diabetes (according to both British and North-American guidelines).[41,42] Recognizing the impreciseness of antepartum estimation of birth-weight, 36 of our patients (17%) could have been included in one or both categories. If this had led to caesarean section, the brachial plexus injury would have been avoided.

A special situation occurs when a previous delivery was complicated by shoulder dystocia and permanent OBPL: should a caesarean section be performed for the index pregnancy in such a case ?[43] In the present patient series, two siblings (<1%) suffered a permanent OBPL. In another series reported from Saudi Arabia, the incidence of repeat injury was much higher, 14%.[44] Diabetes and macrosomia were present in both pregnancies for our siblings and most of the eight siblings in the Saudi study. It seems reasonable therefore, in women for whom a previous delivery resulted in a permanent OBPL to not offer a trial of labour in a subsequent pregnancy, especially in pregnant women with diabetes or macrosomia, or both.

A main weakness of our study is that the obstetric data were derived from parents recalling their child's birth. However, the obstetric data collected – gestation, diabetic status, parity, birthweight – comprised data that parents were likely to recall correctly, especially only 3 months after birth.[45] A possible bias might concern exaggeration of birthweight as the delivery might be regarded as traumatic by the parents. This would, however, probably occur in the three severity groups in a similar fashion. Shoulder dystocia, perhaps the most important obstetric variable, was not included in the data collection and statistical analysis. The reason for this is that in our outpatient clinic almost all parents state that their child was 'stuck' during birth. In some cases birthing records were available, but typically objective measures like head-to-body-interval or number of manoeuvres performed were absent. Such measurements are probably indispensable for the classification of shoulder dystocia.[46] Pre-existing diabetes and gestational diabetes were not scored separately in the out-patient clinic protocol and because of this imprecision the effect of diabetes in the statistical analysis may not be accurate. Ideally, gestational and obstetric data should be recorded prospectively for correlation with neurological severity of potential OBPL. This would include a multicentre collection of data because of the low incidence of neurosurgically treated OBPL. For data on a group of 200 infants with neuro-surgically treated OBPL, gestational and birthing records of one million births should be prospectively collected (assuming an incidence of 2 per 1000 births, of which 10% need surgery). Such a study is unlikely to be performed.

Another weakness is the composition of the group under study. A double inclusion bias exists, as firstly, these children are referred to our tertiary centre for peripheral nerve lesions which selects the more serious lesions. Additionally, from this group of referred patients, only the surgical patients were selected for analysis, which represents those infants that show insufficient neurological recovery. However, the likelihood that a severely injured child would not be referred for surgery is unlikely in our medical system.

One strength of our study is that all records of infants with OBPL in our department are collected prospectively according to a standardised protocol that included standard questionnaires to collect data; 88% of infants could be entered in the ordinal regression model, and for 93% the data were sufficient to define the birthweight-percentile.

The correlation in the general population between birthweight and risk for an OBPL and for a permanent OBPL is already sufficiently documented in the available literature. The group under study is sufficient to strengthen the hypothesis that there is a dose-effect relationship between birthweight and neurological severity of OBPL.

Conclusion

In the present series of 206 infants with neurosurgically treated OBPL a higher birthweight was significantly correlated with a greater neurological severity of their OBPL. Although our study was hampered by selection bias and parental recall of obstetric data, the described dose-effect relationship between birthweight and neurological severity is convincing. This finding may help in developing updated obstetric guidelines to aid in the prevention of shoulder-dystocia-associated permanent brachial plexus injury.

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