Brachial plexus injury in vaginal delivery

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ABSTRACT

Objective: To study the incidence and persistence of brachial plexus injury (BPI) and the ability to predict its occurrence pre-natally depending on patients' criteria and antepartum-partum course.

Methods: During a 3-year period from June 2001-June 2004 at Queen Alia Military Hospital, Royal Medical Services, Amman, Jordan, all newborns with BPI were identified (patients group) and followed-up for a one-year period. Obstetric details were compared with a control group who were delivered during the same period. Obstetrical neonatal features of both groups were compared.

Results: Over the mentioned period, 30 cases of BPI were identified from a total 11560 deliveries (0.25%). Persistency for more than one year was present in 5 cases (17%). Both groups were compared regarding certain known maternal and intra-partum risk factors. Significant risk was history of having shoulder dystocia in previous and current pregnancy ($p<0.05$) and duration of labor ($p<0.05$).

Conclusion: Brachial plexus impairment was encountered in the presence of normal course of labor and delivery. No predictable risk factors were found that could be avoided antenatally to prevent BPI.

Neurosciences 2005; Vol. 10 (2): 168-170

Brachial plexus injury (BPI) is one of the neonatal injuries that are diagnosed on clinical examination after birth. Historic obstetric teachings have stated that BPI results from traction and flexion exerted on the infant's neck during delivery in an attempt to dislodge an impacted anterior shoulder.[1-3] Shoulder dystocia complicates less than 0.6% of all vaginal deliveries,[4,5] with a range of 0.15-0.63%.[6] A BPI occurs in 8-23% of shoulder dystocia cases,[7-9] so other factors should play a role in BPI. Recent reports supported the idea that BPI occurs without evidence of birth trauma,[10-12] so intrauterine mal-adaptation may play a role.[13] Although most cases of BPI resolve within the first year of life, 5-8% persist for one year and in some records, this reaches 20%,[14,15] with persistent damage of approximately 1.6%, due to impairment of motor neuron activity.[16] The purpose of the current study was to detect the incidence and persistence of BPI at our hospital, and the effect of clinical antecedents that have been documented in association with BPI.

Methods. Each newborn delivered is routinely examined in the day post-delivery, before discharge by a pediatrician. The files of the mothers whose newborns were found to have BPI during a 3-year period from June 2001-June 2004 at Queen Alia Military Hospital, Royal Medical Services, Amman, Jordan were studied in an attempt to detect risk factors for such neurological deficit. Data recorded included maternal age, gestational age, maternal diabetes mellitus (DM), maternal obesity (body weight >90 kg), weight gain during pregnancy (>15 kg), history of having huge babies (>4kg), and history of shoulder dystocia. Partographs of these patients were also studied to detect oxytocin

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Received 7th August 2004. Accepted for publication in final form 10th November 2004.

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augmentation of labor, duration of first and second stages of labor, mode of delivery, shoulder dystocia, and newborn birth weight. A control group was selected as follows: the partograph of the next 2 mothers who delivered after the mother who delivered a newborn with BPI was studied after being matched for age (±3 years), parity (±1 child), gestational age (±1 week), weight (±3kg) and birth weight (±250gram). The newborns with BPI were followed in the pediatric clinic, referred for physiotherapy and followed for one year. Persistent cases for more than one year were referred to a neurologist.

Results. During the 3-year study period, a total number of 11560 newborns were delivered. Thirty (0.25%) newborns had BPI. Persistence after one year was present in 5 cases (17%). All the cases were unilateral. A comparison of the supposed maternal risk factor between the affected group and the control is shown in **Table 1**. **Table 2** shows a comparison between the affected group and control regarding intrapartum risk factors. From these results, the most significant risk factor for BPI is a history of shoulder dystocia, 50% (n=15) compared with the control group of 3% (n=2) and 80% (n=24) of shoulder dystocia in the current delivery compared with the 3% (n=2) of the control (p<0.05). The only other significant factor was prolonged first and second stage of labor (p<0.05). Of the 5 cases of persistency for more than one year, 4 were with shoulder dystocia, 4 were with birth weight >4 kg, and 2 were delivered after 41 weeks gestation.

Discussion. This study showed that BPI is an unpredictable event ante or intrapartum. Although it is a transient event in most cases (persistent only in 17%), the obstetrical events that surround persistency did not differ from those in the transient group. The cause of congenital BPI is controversial. Empiric experiences have suggested that this injury can occur at the time of the birth as a direct result of excessive traction on the fetal head in an attempt to dislodge the impacted anterior shoulder.[17] Recent reports have shown that some cases of BPI have an intrauterine origin. In a study carried out by Ouzounian et al,[18] it was found that there is no risk factor in most cases; 89% were not diabetic, 76% were not obese, and normal labor was present in 91% of cases. He stated that "in cases with permanent Erb's palsy in the posterior shoulder of the delivering infant we hypothesize that the injury was not a product of traction applied at delivery, but rather preceded expulsion of the fetal head. The nerve damage possibly occurred during decent of the fetus through the maternal pelvis via impaction of shoulder on sacral promontory; the brachial plexus could have been injured at that time."[19] This could explain why a prolonged second stage of labor is important as a risk factor in BPI in our study.

Use of antenatal, intrapartum, and neonatal variables predicted only 19% of BPI in a study carried out by Perlow et al,[20] other authors noted that these risk factors have only 10% predictive value for BPI.[21,22] Nearly half of all BPI is not associated with shoulder dystocia,[23,24] and it is remarkable that there has never been a series of cases from any institution that has found more than 80% of BPI to be associated with shoulder dystocia,[16,21] comparable to our study, in which 50% of cases had a previous history of shoulder dystocia, and 80% had shoulder dystocia in the current delivery.

The finding of BPI in the posterior arm of infants with antecedent anterior shoulder dystocia or

Table 1 - Maternal risk factors.

Features	BPI (n=30) n (%)	Control (n=60) n (%)	*p*-value
Parity			
Nullipara:	12 (40)	25 (42)	>0.05
Multipara:	18 (60)	35 (58)	>0.05
Post date			
> 41 weeks	15 (50)	32 (53)	>0.05
Maternal DM	2 (6)	3 (5)	>0.05
Maternal obesity	2 (6.6)	1 (1.6)	>0.05
Weight gain	5 (16.6)	7 (12)	>0.05
History of having huge babies	8 (26)	15 (25)	>0.05
History of shoulder dystocia	15 (50)	2 (3)	<0.05

BPI - brachial plexus injury, DM - diabetes mellitus

Table 2 - Intrapartum risk factors.

Features	BPI (n=30) n (%)	Control (n=60) n (%)	*p*-value
Oxytocin use	18 (60)	39 (65)	>0.05
Mean duration of first stage	4.5 hrs	3.5 hrs	<0.05
Mean duration of second stage	50 min	40 min	<0.05
Normal vaginal delivery	26 (85)	52 (86)	>0.05
Shoulder dystocia	24 (80)	2 (3)	<0.05
Huge baby	18 (60)	30 (50)	>0.05

BPI - brachial plexus injury
hrs - hours, min - minutes

associated with cesarean section delivery, strongly suggest an in utero mechanism.[24,25] Other evidence for the hypothesis of in utero BPI is cases of complete brachial plexus palsy after infection with infectious mononucleosis, toxoplasmosis, mumps and pertussis.[26] On the basis of adult derived data, it was found that 10-14 days is necessary before electromyography studies will demonstrate fibrillation potential in denervated brachial muscle group in the newborn. Its finding immediately after delivery differentiates an in utero cause from intrapartum etiology.[27,28]

In our study, shoulder dystocia was a cause in 80% of cases, alternately, 20% did not have traumatic delivery and this supports the concept that not all BPI are traction mediated, and many cases of BPI may be attributable to unavoidable intrapartum or antepartum events, not to actual mismanagement. Even when shoulder dystocia is present it should not be considered as a cause of BPI in infants with prolonged second stage of labor; a significant degree of pressure may have already been applied to the fetal brachial plexus before actual recognition of shoulder dystocia.

In conclusion, it is clear that BPI cannot be anticipated before delivery and every effort should be directed to inhibit traumatic treatment of shoulder dystocia rather than anticipation of this problem before delivery. There is no longer any question that intrauterine maladaptation is responsible for some cases of BPI.

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