

Birth Trauma in the Head and Neck

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Objectives: To review the medical records of neonates found to have birth-associated trauma of the head and neck region. To describe the anomalies, physical findings, and possible sequelae of these injuries and to bring attention to the cause of mechanical birth injury as a potential cause of anomalies in the infant.

Design: Case-controlled retrospective chart review of a cohort of patients identified with birth-associated trauma to the head and neck from January 1, 1991, to March 1, 1997.

Setting: Academic tertiary care medical center.

Patients: Medical records from infants born or transferred with the diagnosis of birth trauma were reviewed. Medical records from a control group of 148 uninjured full-term infants born during the same period were reviewed for comparison. Neonatal charts, including labor and delivery records, were analyzed.

Main Outcome Measures: Each patient record was reviewed for diagnosis, associated injuries, maternal statistics, gestational age, birth weight and size, Apgar scores, type of delivery, length of labor, complications of labor, and length of hospital stay.

Results: One hundred sixty-four infants (incidence, 0.82%; prevalence, 9.5 per 1000 live-births) were identified

with 175 birth-associated injuries to the head and neck. The most common finding was cephalhematoma (56.6%). Other findings included scalp and/or facial lacerations (12%) and hematomas (2.3%), facial nerve palsy (8.6%), brachial plexus injuries (5.1%), clavicular (9.1%) and skull fracture (2.9%), nasal septal dislocation (0.6%), and phrenic (1.7%) and laryngeal nerve injuries (0.6%). Risk factors included birth weight ($P = .001$), vaginal delivery ($P = .001$), primiparity ($P = .02$), forceps delivery ($P = .005$), vacuum delivery ($P = .001$), infants categorized as large for gestational age ($P = .02$), and male infant sex ($P = .03$). Apgar scores were also noted to be lower in our study population ($P = .001$). Risk factors for specific types of injuries varied. However, facial nerve paralysis was associated with multiple birth injuries ($P = .001$), and 2 of 3 phrenic nerve injuries occurred with brachial plexus injuries. Correlation coefficients for factors such as maternal age, gravidity, and race were low.

Conclusion: Birth-associated head and neck trauma is rare. However, mechanical birth-associated trauma must be considered when assessing anomalies, injuries, respiratory difficulty, or feeding difficulties in the neonate or infant. A comprehensive approach is required to diagnose and manage these patients.

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THE DEFINITION of birth injury as defined by Potter¹ is any condition that affects the fetus adversely during delivery. These traumatic injuries can be subdivided into those caused by hypoxia and those caused by mechanical forces.² After being involved in the care of individuals meeting this definition and reviewing existing literature, we thought that an article illustrating the otolaryngical aspects of birth trauma would be a valuable addition to the otolaryngology–head and neck surgery literature. In the assessment of neonates with stridor, respiratory distress, feeding difficulty, and/or

cosmetic deformity the cause of birth-associated trauma should be entertained. Our objective is to review the medical records of neonates found to have birth-associated trauma of the head and neck region, to describe the anomalies, physical findings, and possible sequelae of these injuries, and to bring attention to the cause of mechanical birth injury as a potential cause of anomalies in the infant.

RESULTS

In our search 669 patients were identified, and 505 did not meet criteria and were excluded from the study. Exclusion rep-

PATIENTS, MATERIALS, AND METHODS

We used medical records of patients born or transferred as newborns to Georgetown University Hospital, Washington, DC, an academic tertiary care medical center, between January 1, 1991, and March 1, 1997. During this period, there were 10 591 newborns admitted and 9310 deliveries at this institution. Medical records were retrieved by specific *International Classification of Diseases, Ninth Revision (ICD-9)*³ codes describing birth trauma. Only those patient records indicating birth-associated trauma to the head and neck during the prescribed period were included in this study. All available data were reviewed, including diagnosis, associated injuries, demographic and maternal statistics, gestational age and size, birth weight, Apgar scores, type of delivery, length of labor, and complications. Medical records from 148 full-term infants born at Georgetown University Hospital during this same period but without the diagnosis of birth trauma were reviewed and used for comparison as a control group. Data were collated into a computerized database and were statistically reviewed.

Variables of sex, race, type of delivery, length of labor, Apgar scores, size for gestational age, and maternal gravidity and parity were compared by χ^2 , Fisher exact test, and Mann-Whitney *U* analysis. Birth weight was compared by 2-sample *t* test. *P* values < .05 with odds ratios with 95% confidence intervals were considered statistically significant. Specific injuries were then analyzed in the same manner.

resented birth injuries outside the head and neck region, and minor injuries such as superficial abrasions and scratches.

Of 164 newborns, 175 birth-associated injuries were identified for an incidence rate of 0.82%. Prevalence did not vary significantly from year to year, ranging from 22 to 34 cases per year, and averaged 9.56 cases per 1000 live-births per year. Cases included 92 males and 72 females (ratio, 1.3:1; *P* = .03).

Race included 108 whites, 27 blacks, 28 patients categorized as other, and 1 patient with an unspecified category. These percentages were consistent with our normative data.

Types of deliveries included spontaneous vaginal delivery (SVD), 42 (25.6%); vaginal delivery of unspecified type (VD), 18 (11%); vacuum-assisted delivery, 46 (28%); cesarean section, 28 (17%); low forceps-assisted delivery, 14 (8.5%); forceps delivery of unspecified type, 15 (9.1%); and midforceps delivery, 1 (0.6%). The distribution of types of delivery between groups is significant (*P* = .001). The rate of vaginal deliveries (SVD+VD; *P* = .001), forceps deliveries (*P* = .005), and vacuum-assisted deliveries (*P* = .001) also reached significance individually (**Table 1**). Rates for cesarean section and indications for cesarean section were not significantly different between groups (**Table 2**).

Table 1. Types of Delivery*

Types of Delivery	Population		<i>P</i>
	Study (n = 164)	Control (n = 148)	
CS	28	29	.48
SVD	42	93	.001†
VD	18	2	...
VAD	46	12	.001
LFD	14	10	.005‡
MFD	1	0	...
FD	15	0	...
Unspecified	0	2	...

*CS indicates cesarean section; SVD, spontaneous vaginal delivery; VD, vaginal delivery of unspecified type; VAD, vacuum-assisted delivery; LFD, low forceps-assisted delivery; MFD, midforceps delivery; FD, forceps delivery of unspecified type; and ellipses, not applicable.

†SVD + VD.

‡LFD + MFD + FD.

Table 2. Indications for Cesarean Section*

Reason	Population		<i>P</i>
	Study (n = 28)	Control (n = 29)	
FTP	8	10	.57
Repeat	3	6	.29
FD	7	3	.29
CPD	4	1	.36
Breech	0	6	.009
Unspecified	6	3	

*FTP indicates full-term prolonged; FD, forceps delivery of unspecified type; and CPD, cephalopelvic disproportion.

Table 3. Length of Labor*

Length of Labor, h	Population	
	Study (n = 164)	Control (n = 148)
<12	45	48
12-24	20	28
24-36	7	5
>36	1	2
Unspecified	91	65

**P* = .07.

Length of labor results included less than 12 hours, 45 (27.4%); 12 to 24 hours, 20 (12.2%); 24 to 36 hours, 7 (4.2%); and more than 36 hours, 1 (0.6%). Length of labor was unspecified in 91 cases (55%). Our normative results are shown and compared in **Table 3**. There appears to be a trend toward an association with increased length of labor, which fails to reach significance. This may be due to the high number of unspecified labor times in the cohort group.

Seventy-five patients (45.7%) were characterized as average size for gestational age; 31 (18.9%) as large for gestational age; 5 (3.0%) as small for gestational age; and 53 (32.3%) were of an unspecified size. The rate of large

Table 4. Size for Gestational Age*

Size for Gestational Age	Population	
	Study (n = 164)	Control (n = 148)
AGA	75	111
LGA	31	19
SGA	5	5
Unspecified	53	13

*P = .02. AGA indicates average size for gestational age; LGA, large for gestational age; and SGA, small for gestational age.

Table 5. Maternal Parity*

Parity	Population		P
	Study (n = 164)	Control (n = 148)	
0	85	57	.02
1	54	58	>.05 (NS)
2	17	19	>.05 (NS)
>2	7	12	>.05 (NS)
Unspecified	1	2	>.05 (NS)

*NS indicates not significant.

for gestational age was significantly increased in the study population ($P = .02$). Maternal parity was 0 in 85 cases (51.8%), 1 in 54 cases (32.9%), 2 in 17 cases (10.4%), greater than 2 in 7 cases (4.3%), and unspecified in 1 case (0.6%). An increased rate of primiparity was present in the study group ($P = .02$). Size for gestational age and maternal parity are shown and compared with normative data in **Table 4** and **Table 5**, respectively.

Birth weight ranged from 770 to 5443 g, with an average birth weight of 3474 g and a median (SD) of 3535 (741.56). This is compared with a range of 2438 to 4990, an average of 3485.9, and a median of 3480 for our normative data. Birth weight distribution between groups was significant ($P = .001$).

Apgar scores (APG) were greater than 8 at 1 minute (APG1) in 102 cases (62.2%), greater than 8 at 5 minutes (APG5) in 143 cases (87.2%), and unspecified in 1 case (0.6%). Apgar scores for our control group were greater than 8 for APG1 in 125 (84.5%) and greater than 8 for APG5 in 145 (97.9%). Distribution between groups was significantly different ($P = .001$). Apgar scores are shown and compared in **Table 6**.

One hundred seventy-five injuries were identified. These included 99 (56.6%) cephalhematomas, 21 (12%) lacerations, 16 (9.1%) clavicular fractures, 15 (8.6%) facial palsies, 9 (5.1%) brachial plexus injuries, 5 (2.9%) skull fractures, 4 (2.3%) hematomas, 3 (1.7%) phrenic nerve injuries, 1 (0.6%) vocal fold paresis, 1 (0.6%) nasoseptal dislocation, and 1 (0.6%) Horner syndrome (**Figure 1**). Nine patients were diagnosed as having multiple birth trauma injuries, which were the following: cephalhematoma, laceration, and facial paresis; cephalhematoma and laceration; cephalhematoma and facial paresis; clavicular fracture and brachial plexus injury; cla-

Table 6. Apgar (APG) Scores*

Apgar Score	Population		P
	Study (n = 164)	Control (n = 148)	
APG at 1 min >8	102	125	.001
APG at 5 min >8	143	145	.001
Unspecified	1	0	...

*Ellipses indicate not applicable.

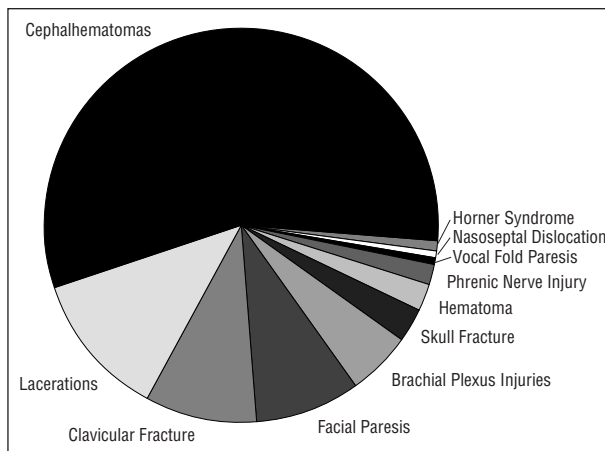


Figure 1. Distribution of specific birth injuries.

Table 7. Injury vs Type of Delivery*

Type of Delivery	Cephal-hematoma	Facial Paresis	Phrenic Palsy	Brachial Plexus Injury			Skull Fracture	Clavicular Fracture
				Injury	Fracture	Fracture		
CS	15	2	1	1	0	2		
FD	12	10	2	3	4	2		
VAD	31	2	0	2	1	3		
SVD	37	1	0	3	0	9		
Total	97	15	3	9	5	15		

*CS indicates cesarean section; FD, forceps delivery of unspecified type; VAD, vacuum-assisted delivery; and SVD, spontaneous vaginal delivery.

vicular fracture and facial palsy; brachial plexus injury, facial palsy, phrenic nerve injury, and skull fracture; brachial plexus injury and phrenic nerve injury; facial palsy and skull fracture; and facial palsy and vocal fold paresis.

Specific injury vs type of delivery are compared in **Table 7**. Clavicular fractures (n = 15) were more common in vacuum-assisted delivery and SVD+VD and in infants considered large for gestational age (n = 13) compared with average size for gestational age (n = 2), small for gestational age (n = 0), and unspecified (n = 1). Clavicular fractures and brachial plexus injuries were more common with shoulder dystocia. Additionally 2 (66.6%) of 3 phrenic nerve injuries occurred in conjunction with brachial plexus injuries. Facial paresis was more common in the multiply injured patient ($P = .001$), and is shown in **Table 8**.

Table 8. Associations With Facial Paresis*

	Single Injury	Multiple Injury	Total
Facial palsy	9	6	15
No palsy	146	3	149
Total	155	9	164

*P = .001.

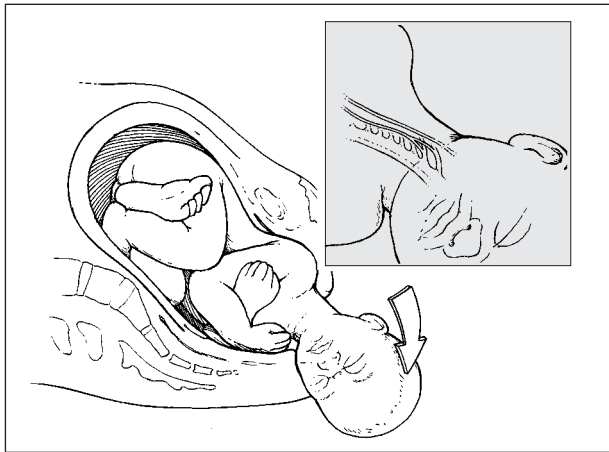


Figure 2. Lateral neck traction injury.

COMMENT

The incidence of birth trauma varies with the articles²⁻⁵ cited. Although decreased in comparison with historical references, recent articles^{2,4,5} report that major birth trauma occurs in 3% of all live-born infants, accounts for 2% of all neonatal mortality, and accounts for 10% of all neonatal deaths in full-term infants. Salonen and Uusitalo⁴ assessed 14 265 live-born infants in Finland. The reported incidence of major birth trauma was 3.16%, which included clavicular fracture, brachial plexus injury, fracture of the long bones, and facial nerve injury. Levine et al⁵ established an incidence of 1.54% when looking at brachial plexus, facial nerve injuries, and clavicular fractures. When other injuries were added, the most common of which included caput succedaneum and cephalhematoma, that incidence increased to more than 23%. In our study, regarding birth trauma specifically in the head and neck, we find an incidence of 0.82% with a prevalence of 9.56 per 1000 births per year.

Predisposing factors to birth injuries that have been previously reported include fetal macrosomia, malpresentation, prolonged labor, forceps delivery, and primiparity.^{5,6} Macrosomia usually occurs in neonates with a birth weight of more than 3500 g and is associated with an increased incidence of birth trauma. The most common malpresentation previously reported to be associated with birth trauma is breech delivery. In most cases, the mechanism of injury is extensive traction of the neck during delivery (**Figure 2**). This most frequently occurs with shoulder dystocia (difficult childbirth because of shoulder engagement).

Prolonged labor also increases the likelihood of complications, as do primiparity and forceps delivery. In our study, risk factors for birth trauma in the head and neck included birth weight ($P = .001$), vaginal delivery ($P = .001$), primiparity ($P = .02$), forceps delivery ($P = .005$), vacuum-assisted delivery ($P = .001$), infants categorized as large for gestational age ($P = .02$), and male infant sex ($P = .03$). Apgar scores were also noted to be lower in our study population ($P = .001$). The specific birth injuries are described below.

CEPHALHEMATOMA

Cephalhematoma is caused by subperiosteal bleeding and is thought to occur as a result of friction separating the pericranium from the skull during the birth process. The incidence range reported is 0.4% to 2.49% of all live births.² Incidence in our study was 0.5%. Cephalhematoma, which is blood effused beneath the pericranium, can be differentiated from caput succedaneum and subgaleal hemorrhage by not extending past suture lines due to periosteal attachments.⁷ Caput succedaneum is an effusion of serum that overlies the periosteum. These processes are usually self-limiting and resolve within 4 weeks.

Subarachnoid hemorrhage, the most common intracranial lesion detected after birth, can occur with cephalhematoma, caput succedaneum, skull fracture, or even in uncomplicated birth. Subarachnoid hemorrhage alone is rarely associated with subsequent complications but symptoms of somnolence, lethargy, absent Moro reflex, or general failure of normal motion should alert the physician to a more serious injury.

SKULL FRACTURE

Skull fracture must be suspected in any cephalhematoma or subarachnoid hemorrhage. Reports state that 10% to 25% of cephalhematomas are associated with skull fracture,² although none occurred together in our study. These fractures are usually linear, involving the parietal bones, or depressed, forming the so-called ping-pong fracture. These fractures can occur with forceful attempts by forceps, spontaneous, or cesarean delivery.⁸ Neurosurgical consultation is highly recommended with any neonatal skull fracture.

LACERATIONS AND HEMATOMAS

No rates for lacerations or hematomas in birth trauma were found. Incidence in this study was 0.1%. Twenty-one lacerations occurred, 15 involving the scalp, 3 the face, and 1 each for the cheek and ear; and 1 case was not further specified.

CLAVICULAR FRACTURE

Clavicular fracture is associated with brachial plexus, phrenic, and right recurrent nerve injuries. These injuries must be ruled out when a clavicular fracture is diagnosed in the newborn. The incidence is reported at 2.7 to 5.7 per 1000 live-births^{2,9} and is most often associ-

ated with large neonates and shoulder dystocia. In our study clavicular fracture was associated with large for gestational age (100%), increased birth weight (range, 3785-5273 g), and shoulder dystocia (5 [29.4] of 17). Clavicular fracture was also more common in vacuum-assisted delivery and SVD+VD. Isolated clavicular fractures usually heal without sequelae.

FACIAL NERVE INJURY

Injury to the facial nerve is usually associated with seizing the head obliquely. In forceps delivery, the posterior blade exerts pressure on the stylomastoid foramen or compresses the bone overlying the vertical segment of the facial canal.¹⁰ However, 33% of cases occur in spontaneous delivery. This is believed to be due to compression against the maternal sacral promontory.⁵ Incidence ranges from 1.8 to 7.5 per 1000 live-births. Acquired congenital facial nerve palsy by birth trauma must be differentiated from developmental facial paralysis. Developmental paralysis is associated with Mobius syndrome, hemifacial microsomia, or hypoplasia of the depressor anguli oris muscle.¹¹ Falco and Eriksson¹¹ reviewed 92 congenital facial nerve palsies among 44 299 live-birth infants. They found 81 acquired injuries, and 74 (91%) of these were associated with forceps delivery. We find an incidence of facial paresis secondary to birth trauma of 0.8%. Nine (60%) of the 15 facial palsies occurred in assisted deliveries and it was significantly more common ($P = .001$) in the multiply injured patient (Table 8). Facial nerve palsy was also identified in males at twice the rate of females (10 males and 5 females). The prognosis of acquired facial paralysis by birth trauma is good with spontaneous recovery in a matter of hours to weeks. Electromyography is recommended, as well as auditory brainstem response to rule out auditory nerve involvement. Some authors recommend^{15,12,13} surgical intervention as early as 1 to 3 months; however, most reports favor observation for 1 year, or up to 2 years if there have been improved electromyographic findings.

BRACHIAL PLEXUS INJURY

Brachial plexus injury is usually caused by a stretch injury caused by lateral traction of the neck (Figure 2). The incidence is reported as 1 per 1000 live-births (current study, 0.5 per 1000) and has been associated with macrosomia and shoulder dystocia. Erb palsy is paralysis involving the fifth and sixth cervical nerves and represents 90% of brachial plexus palsies (Figure 3). Paralysis of the deltoid, infrascapular, and flexor muscles of the forearm are involved. Diagnosis is made with the presence of an asymmetric Moro reflex. The arm falls limply to the side but grasp remains intact. The infant shows the so-called waiters'-tip grasp. Klumpke paralysis involves the fifth to seventh cervical nerves and is diagnosed when the grasp reflex is also absent (Figure 4). Horner syndrome (ptosis, miosis, and anhidrosis) can also be present with involvement of the sympathetic fibers from the first thoracic root.¹⁴ Spontaneous recovery is the rule. Electromyography is recommended. Treatment is conservative with physiotherapy and the application of dy-

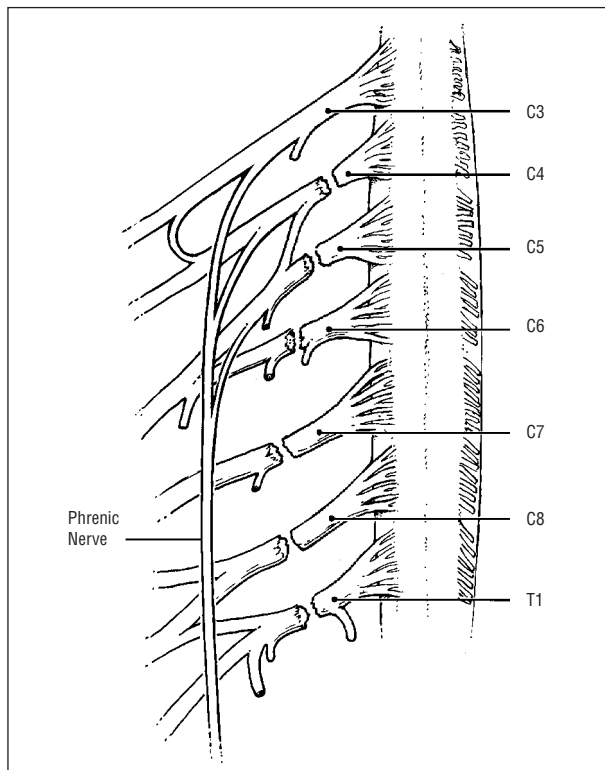


Figure 3. Brachial plexus injury.



Figure 4. A patient with Erb palsy.

namic splints. However, some authors¹⁵⁻¹⁷ advocate early surgical intervention with mixed results. Gordon et al¹⁵ investigated 59 children with brachial plexus paralysis at birth. Of these, paralysis was transitory in 56 (95%). Wickstrom¹⁶ studied 87 children with brachial plexus injuries at birth. Seventy-seven (88.5%) of the 87 patients were found to have some degree of residual deformity, and 28 (32%) of 87 required procedures to correct the defects. Procedures include release of contracture, teres major transplantation to the external rotator, open reduction of posterior dislocations, and osteotomy of the humerus. Boome and Kaye¹⁷ reported a series of 70 patients and concluded that if recovery of the upper roots had not started by 3 months of age, a significant residual functional deficit would occur. They propose early exploration and sural nerve grafting in patients meeting these criteria.

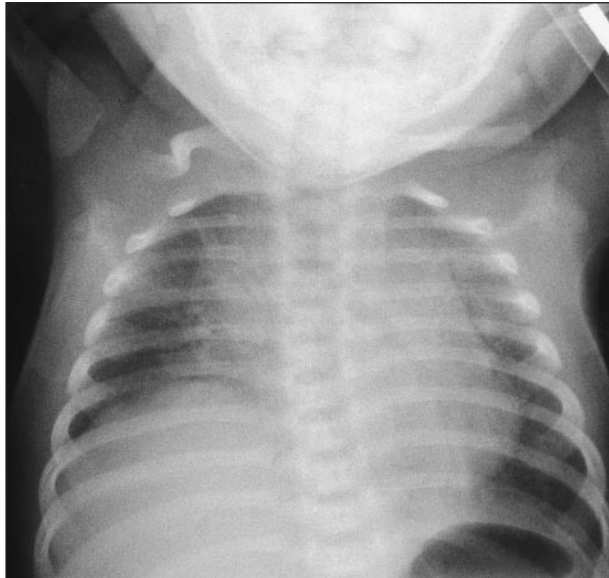


Figure 5. Chest radiograph demonstrating phrenic nerve paresis.

PHRENIC NERVE INJURY

In birth trauma, phrenic nerve injury is usually associated with a concurrent brachial plexus injury (Figure 3). Our study shows this association in 2 of 3 patients. Phrenic nerve injury should be suspected in infants with respiratory distress who have a history of complicated labor, especially in the presence of brachial plexus injury. Symptoms may begin as early as the first day of life, but may be delayed and not present until more than 1 month of age.¹⁸ Classically, breathing is labored and thoracic, with decreased breath sounds on the affected side. Diagnosis can be confirmed by chest radiography with elevation of the hemidiaphragm (**Figure 5**). Fluoroscopy shows paradoxical “seesaw” movement of the affected side. However, radiography can be negative especially if the patient is receiving mechanical ventilatory support. The diaphragm is of vital importance to respiration, especially in the infant. The first thoracic vertebra and manubrium sternum are at the same level in the infant. With growth, the manubrium descends to its final position at approximately 10 years of age. In addition, the costal arc is flat and does not increase in curvature until approximately 6 months, reaching its normal shape after 2 years of age.

These factors contribute to make the diaphragm the vital motive force for spontaneous respiration in the infant.¹⁹ Recovery is usually spontaneous but pulmonary infection is a serious complication. Prophylactic antibiotic therapy may be beneficial. Treatment includes placing the infant on the involved side with respiratory support as necessary. Feeding is by gavage to save the infant the energy required for breathing. Diaphragmatic pacing may be a possible option, and surgical plication may be necessary.

LARYNGEAL NERVE INJURY

The most common cause of vocal fold paralysis reported in infants is birth trauma. Cohen et al²⁰ described 100 infants and children diagnosed as having la-

ryngeal paralysis. In 19% of these children vocal fold paralysis was considered to be attributable to a birth injury. Symptoms included stridor, airway obstruction, dysphonia, dysphagia, and aspiration. These symptoms when caused by birth trauma had an early onset with a mean of 5.6 days of age, whereas other causes of childhood vocal fold paralysis had a somewhat later onset. Cohen et al²⁰ found 40% of vocal fold paresis were unilateral and 60% were bilateral. Vocal fold paralysis was found to occur in forceps, spontaneous, and cesarean deliveries.

Of 164 infants included in this study only 1 was diagnosed as having vocal fold paresis (0.6%) and this injury was associated with a concomitant facial nerve palsy. Delay in diagnosis could certainly be a factor in our study, in that the injury may not have been paired with *International Classification of Diseases, Ninth Revision (ICD-9)*³ codes for birth trauma and would not have met search criteria.

Vocal fold paralysis in infancy can be associated with other cranial nerve deficiencies and intrinsic central nervous system malformations such as Arnold-Chiari deformity, meningomyelocele, and hydrocephalus, or with diseases of the heart and great vessels. Laryngeal nerve paralysis has also been described in cases of syphilis.²¹

Investigation should include a modified barium swallow and consultation with a speech pathologist to optimize feeding. Unilateral paralysis can usually be followed clinically, whereas bilateral paralysis usually requires tracheotomy. In the study by Cohen et al,²⁰ the mean time before decannulation was possible in the tracheotomy group was 49 months. Narcy et al²² described 219 cases of newborn laryngeal paralysis, 22 of which required surgical management in cases where no spontaneous recovery or improvement occurred after 6 to 9 months. It is their belief that the tracheotomy tube should not remain after this point, and they recommend arytenoidopexy to lateralize laryngeal structures. Arytenoidectomy and reinnervation procedures were not believed to be advisable. Tucker²³ reported 30 patients younger than 5 years with abnormal vocal fold mobility; 22 of them had bilateral involvement and 8 had unilateral involvement. Eighteen patients in this group were considered good candidates for a muscle-pedicle reinnervation procedure. Nine (50%) were successfully decannulated with no further loss of voice. Better results were achieved in children in whom the diagnosis was described as traumatic paralysis than in children with a more central neurologic dysfunction. However, Tucker cautioned that these procedures may result in further loss of voice and should be delayed until the child is old enough to take part in the decision process.

NASAL INJURY

Dislocation of the cartilaginous portion of the septum from the vomerine groove and the columella may cause airway obstruction. Nasal respiration in the newborn is obligate for respiration and feeding. Dislocation must be distinguished from fetal modeling and other causes of nasal obstruction such as congenital stenosis of the pyriform aperture, choanal atresia, congenital cysts of the nasolacrimal duct, and dental alveolar cysts. Podoshin et al²⁴

examined 4090 consecutive newborns over a 2-year period and found an incidence of 0.93% of anterior or nasal septal cartilaginous dislocations (present study, 0.6%). Diagnosis is made by noting deviation of the tip of the nose to one side accompanied by leaning of the columella and flattening of the nasal aperture. Early detection and treatment is important and should occur within the first 3 days of life. Treatment by manipulation is recommended. The head is steadied, the dorsum of the nose is grasped, and a nasal elevator is lifted pushing the anterior end of the septum into the septal groove and columella.

CONCLUSIONS

We find, regarding birth trauma specifically in the head and neck, an incidence of 0.82% with a prevalence of 9.56 per 1000 births per year. Predisposing factors to birth injuries in our study included birth weight ($P = .001$), vaginal delivery ($P = .001$), primiparity ($P = .021$), forceps delivery ($P = .005$), vacuum-assisted delivery ($P = .001$), infants categorized as large for gestational age ($P = .02$), and male infant sex ($P = .03$). Apgar scores were also noted to be lower in our study population ($P = .001$). In addition, clavicular fractures were more common in vacuum-assisted delivery and SVD+VD and in infants considered large for gestational age; clavicular fractures and brachial plexus injuries were more common with shoulder dystocia; 2 (66.6%) of 3 phrenic nerve injuries occurred in conjunction with brachial plexus injuries; and facial paresis was more common in the multiply injured patient ($P = .001$).

The field of otolaryngology encompasses many aspects of birth trauma, and the otolaryngologist is consulted to assist in the diagnosis and management of these difficult cases. Paralysis of the phrenic and laryngeal nerves may cause respiratory and feeding difficulty and may require intervention to maintain an adequate airway.

Flexible laryngoscopy has increased our ability to quickly assess vocal fold function and aids in our examination of the newborn. A comprehensive approach is required to diagnose and manage the effects of birth trauma. Acquired injury must be distinguished from developmental lesions on the basis of history, physical examination, and associated anomalies. Complete evaluation should also include radiographic, audiometric, and electrodiagnostic studies as indicated. It must be emphasized that most of these injuries are self-limiting and conservative management must be entertained.

It is our hope that this review facilitates discussion and emphasizes the importance of considering birth trauma when assessing anomalies in the neonate or infant. Recognition of these disorders, the differential di-

agnoses, and the treatment options will benefit both the children and parents we serve.

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REFERENCES

- Potter EL. *Pathology of the Fetus and Infant*. 2nd ed. Chicago, Ill: Year Book Medical Publishers Inc; 1961.
- Gresham EL. Birth trauma. *Pediatr Clin North Am*. 1975;22:317-328.
- World Health Organization. *International Classification of Diseases, Ninth Revision (ICD-9)*. Geneva, Switzerland: World Health Organization; 1977.
- Salonen IS, Uusitalo R. Birth injuries: incidence and predisposing factors. *Z Kinderchir*. 1990;45:133-135.
- Levine MG, Holroyde J, Woods JR, et al. Birth trauma: incidence and predisposing factors. *Obstet Gynecol*. 1984;63:792-795.
- Donn SM, Faix RG. Long-term prognosis for the infant with severe birth trauma. *Clin Perinatol*. 1983;10:507-520.
- Netter FH. Extracranial hemorrhage and skull fractures in the newborn. In: *The Ciba Collection of Medical Illustrations*. West Caldwell, NJ: Ciba; 1986:4.
- Saunders BS, Lazowitz S, McArthur RD, et al. Depressed skull fracture in the neonate. *J Neurosurg*. 1979;50:512.
- Perlow JH, Wigton T, Hart J, et al. Birth trauma. *J Reprod Med*. 1996;41:754-760.
- McHugh HE. Facial paralysis in birth injury and skull fractures. *Arch Otolaryngol*. 1963;78:443.
- Falco NA, Eriksson E. Facial nerve palsy in the newborn: incidence and outcome. *Plast Reconstr Surg*. 1990;85:1-4.
- Kornblut AD. Facial nerve injuries in children. *Ear Nose Throat J*. 1977;56:43-54.
- Manning JJ, Adour KK. Facial paralysis in children. *Pediatrics*. 1972;49:102-109.
- Netter FH. Brachial plexus and/or cervical nerve root injuries at birth. In: *The Ciba Collection of Medical Illustrations*. West Caldwell, NJ: Ciba; 1986:19.
- Gordon M, Rich H, Deutschberger J, et al. The immediate and long-term outcome of obstetric birth trauma. *Am J Obstet Gynecol*. 1973;117:51-56.
- Wickstrom J. Birth injuries of the brachial plexus: treatment of defects in the shoulder. *Clin Orthop*. 1962;23:187-196.
- Boome RS, Kaye JC. Obstetric traction injuries of the brachial plexus: natural history, indications for surgical repair and results. *J Bone Joint Surg Br*. 1988;70:571-576.
- Schullinger JN. Birth trauma. *Pediatr Clin North Am*. 1993;40:1351-1358.
- Mearns AJ. Iatrogenic injury to the phrenic nerve in infants and young children. *Br J Surg*. 1977;64:558-560.
- Cohen SR, Geller KA, Birns JW, et al. Laryngeal paralysis in children: a long-term retrospective study. *Ann Otol Rhinol Laryngol*. 1982;91:417-424.
- Gentile RD, Miller RH, Woodson GE. Vocal cord paralysis in children 1 year of age and younger. *Ann Otol Rhinol Laryngol*. 1986;95:622-625.
- Narcy P, Contencin P, Viala P. Surgical treatment for laryngeal paralysis in infants and children. *Ann Otol Rhinol Laryngol*. 1990;99:124-128.
- Tucker HM. Vocal cord paralysis in small children: principles in management. *Ann Otol Rhinol Laryngol*. 1986;95:618.
- Podoshin L, Gertner R, Fradis M, et al. Incidence and treatment of deviation of nasal septum in newborns. *Ear Nose Throat J*. 1991;70:485-487.