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Causes of Birth Asphyxia and Trauma

JOHN R. O'BRIEN, M.D., C.M., F.R.C.S.[C],
ROBERT H. USHER, M.D., C.M. and
GEORGE B. MAUGHAN, M.D., C.M., F.R.C.S.[C], *Montreal*

ABSTRACT

Causes of birth asphyxia and trauma were determined in the 208 most severely affected infants of 10,995 consecutive live births; 159 infants had cerebral disturbances, 39 had fractures and palsies, and 10 had fractures or palsies in addition to cerebral disturbances. Most frequent causes of birth asphyxia and trauma were: prolonged labour, midforceps or breech delivery in full-term infants; abruptio placentae, difficult breech delivery, and maternal sedation in premature infants; and unattended precipitate deliveries in immature infants. Asphyxia following normal labour and delivery usually occurred in infants with fetal malnutrition.

Improved obstetrical management with more frequent use of Cesarean section delivery might have been of value in preventing much of this fetal injury.

Asphyxia and trauma due to complications of delivery were twice as frequent on the ward as on the private service. This may have been due in part to a lower Cesarean section rate on the ward service. A monthly review of birth asphyxia and trauma is recommended to help maintain a high standard of obstetrical practice.

SOMMAIRE

On a établi les causes d'asphyxie et de traumatisme ayant affecté 208 des nourrissons les plus gravement atteints sur 10,995 naissances d'enfants vivants. Parmi les nourrissons on a trouvé 159 qui souffraient de troubles cérébrals, 39 de paralysie et de fractures, et 10 nourrissons avaient des fractions et des paralysies aussi que des troubles cérébrals. Les causes les plus fréquentes étaient: travail prolongé, emploi de forceps au détroit moyen ou présentation de siège chez des enfants à terme; abruptio placentae, accouchements difficiles dans des présentations de siège et sédation maternelle chez des prématurés et, enfin, accouchements précipités inattendus chez des plus prématurés. L'asphyxie après un travail et un accouchement normaux a été habituellement observée chez des nourrissons dont la nutrition fétale était insuffisante.

De meilleures méthodes d'obstétrique et un recours plus fréquent à la section césarienne comptent parmi les éléments qui auraient pu prévenir bien des accidents fâcheux. L'asphyxie et le traumatisme, comme complications de l'accouchement, ont été deux fois plus nombreuses dans les salles qu'en clientèle privée, cette disproportion pouvant être attribuée partiellement à un recours moins fréquent à la césarienne dans les salles. On propose de publier chaque mois un relevé de l'asphyxie et du traumatisme à la naissance pour maintenir la réputation de la pratique obstétricale.

IN 1861, Little[17] described a cause-and-effect relationship between asphyxia and trauma at birth and permanent cerebral injury. This association has been confirmed repeatedly since that time in retrospective studies of children with cerebral palsy.[8, 9, 12, 20] According to these studies it can be estimated that even with modern obstetrical methods, cerebral palsy develops in one out of every 300 births. Because the majority of such cases are directly attributable to obstetrical causes,[8] greater

From the Departments of Obstetrics and Gynecology, and of Pediatrics, Royal Victoria Hospital and McGill University, Montreal, Quebec.
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Address reprint requests to: Dr. G. B. Maughan, Obstetrician and Gynecologist-in-Chief, Royal Victoria Hospital, Montreal 2, Quebec.

understanding of the causes of birth asphyxia and trauma is needed if these injuries are ultimately to be prevented.

The purpose of the present study was to determine the relative role of various obstetrical and fetal factors in the production of birth asphyxia and trauma today. Neonatal sequelae of these processes were investigated in a consecutively delivered live-born population at one maternity hospital; all cases of severe asphyxia neonatorum, abnormal cerebral signs, convulsions, fractures and paralyses were included. The incidence, probable primary cause, and factors associated with these conditions were analyzed. In seven out of every eight affected infants a definite obstetrical complication or fetal pathological finding was detected which was apparently responsible for the neonatal injury. Certain obstetrical complications and procedures were associated with a risk of asphyxia or trauma to the fetus of greater than 10%.

These were the findings in a university teaching centre which has complete facilities and a specialized attending staff, and whose obstetrical results compare favourably with those generally prevailing (Table I).

TABLE I.

	Royal Victoria Hospital (1960-1962)	*Obstetrical Statistical Co-operative[14] (1959 and 1960) (white patients only)*
1. No. of births	10,995	69,175
2. Cesarean section rate	5.1%	5.9%
3. Perinatal mortality rate (per 1000 births)		
—over 500 g.	22.0	28.2
—over 1000 g.	16.5	20.0
—over 2500 g.	6.1	9.0

In this hospital 40% of all deliveries are carried out on the ward service. It is probable that the incidence and causes of birth asphyxia and trauma here are representative of those obtaining in most modern maternity centres.

SUBJECTS AND METHODS

The subjects consisted of 208 affected infants from a series of 10,995 live births at the Royal Victoria Montreal Maternity Hospital during 1960, 1961 and 1962. These infants had one or more of the following conditions:

(A) CEREBRAL DISTURBANCES

(i) *Severe asphyxia neonatorum.*—Infants who had an Apgar score[2] of less than 4 at one minute of age, and required at least three minutes of positive pressure resuscitation before the onset of sustained respirations.

(ii) *Cerebral depression and irritation.*—Infants who had severe cerebral depression and/or irritation for more than 12 hours (usually 24 to 48 hours) after birth, unrelated to co-existing disease.

(iii) *Convulsions.*—Infants who had one or more generalized seizures during the first week of life.

(B) PERIPHERAL INJURIES

(i) *Paralyses.*—Brachial plexus or facial, phrenic or radial nerve palsies.

(ii) *Fractures.*—Fractures of the clavicle, skull or humerus.

Almost all of the infants were seen during the neonatal period by one of the authors who also supervised weekly chart rounds at which diagnoses were assigned.

The obstetrical and neonatal charts of each affected infant were reviewed and, wherever possible, a specific obstetrical complication or type of fetal pathology was assigned as the primary cause of asphyxia or trauma. Causative factors were classified as follows: complications of labour, complications of delivery, problems in management, fetal pathology and unknown. Cord loops or knots were considered significant when associated with fetal distress. Labour was considered prolonged when it exceeded 24 hours in a primipara or 16 hours in a multipara. Trial of labour was considered prolonged when it exceeded six hours before a Cesarean section performed for cephalopelvic disproportion. Fetal distress was diagnosed when meconium appeared in a vertex presentation, or when the fetal heart was recorded at less than 100/min., or more than 160/min., or when it was irregular. All full-term breech deliveries were considered to be potentially asphyxiating or traumatic, but only those premature breech deliveries were included in which difficulty or delay was encountered. Congenital anomalies were considered to have caused asphyxia only if they could have interfered with onset of respirations, and erythroblastosis only if the infant's hemoglobin concentration was less than 7 g. % at birth.

The second part of the study consisted of a statistical analysis of the association between vari-

TABLE II.—INCIDENCE OF CEREBRAL DISTURBANCES

Number of live births	*Full-term (over 2500 g.) (10,222)*		*Premature (1001-2500 g.) (717)*		*Immature (501-1000 g.) (56)*		*Total (10,995)*	
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Cerebral disturbances:								
Total number of affected infants	99	0.98	42	5.87	28	50.00	169	1.54
Severe asphyxia neonatorum	53	0.52	40	5.58	28	50.00	121	1.09
Cerebral depression and irritation	55	0.54	6	0.84	1	1.80	62	0.56
Convulsions	18	0.18	2	0.28	0	0.00	20	0.18

ous obstetrical complications and neonatal injury. Control figures for this analysis were obtained from 3431 full-term (over 2500 g.) and 264 premature (1001-2500 g.) live births delivered during 1961, and 28 unaffected immature (501-1000 g.) infants delivered during the three-year period of the study.

RESULTS

Incidence of Cerebral Disturbances (Table II)

A total of 169 infants (16 per 1000 live births) had one or more cerebral disturbances. The incidence of severe asphyxia neonatorum was 121 (11 per 1000), of cerebral depression or irritation 62 (5.6 per 1000), and of convulsions 20 (2 per 1000). Different forms of cerebral disturbance tended to occur together in the same infants. Thirty-one infants had combinations of two or all three cerebral disturbances; such combinations would have been expected by chance alone in only one of the 10,995 live births.

The incidence of severe asphyxia neonatorum increased with degree of prematurity from 0.5% in full-term infants (over 2500 g.), to 5% in premature infants (1001-2500 g.), to 50% in immature infants (501-1000 g.).

TABLE III.—CAUSES OF CEREBRAL DISTURBANCE

Number of affected infants	*Full-term* (99)	*Premature* (42)	*Immature* (28)
Complications of labour:			
Abruptio placentae	0%	12%	14%
Prolapsed cord	3	7	0
Cord loops or knots	4	0	0
Prolonged labour, no fetal distress	5	2	4
Prolonged labour, fetal distress	15	0	0
Prolonged trial of labour	8	0	0
Unexplained fetal distress	9	0	0
Total	44	21	18
Complications of delivery:			
Midforceps delivery	18	0	0
Shoulder dystocia	5	0	0
Breech delivery	10	19	4
Birth weight over 4500 g.	2	0	0
Malpresentation	1	0	7
Precipitate delivery	3	0	0
Total	39	19	11
Problems in management:			
Sedation	4	7	18
Unattended delivery	0	0	14
Inadequate resuscitation	0	3	3
Total	4	10	35
Fetal pathology:			
Congenital anomalies	0	7	0
Rh erythroblastosis	1	12	0
Intrauterine infection	2	2	7
Total	3	21	7
Unknown			
Fetal malnutrition	6	24	4
Not malnourished	4	5	25
Total	10	29	29
Total	100%	100%	100%

Causes of Cerebral Disturbances (Table III and Fig. 1)

Because the causes of all three types of cerebral disturbance were similar, they are considered together.

In *full-term infants* the cause of the cerebral disturbance was considered to be a complication of labour in 44%, a complication of delivery in 39%, sedation in 4%, and fetal pathology in 3%, leaving 10% unexplained. The most frequent specific causes were prolonged labour, midforceps delivery and breech delivery, these together accounting for one-half of the cases.

Among *premature infants* the cause of the cerebral disturbance was a complication of labour in 21%, a complication of delivery in 19%, sedation or inadequate resuscitation in 10%, and fetal pathology in 21%. In 29% no cause was determined. The most common specific causes of cerebral disturbance in this weight group were difficult breech delivery, abruptio placentae, and Rh erythroblastosis.

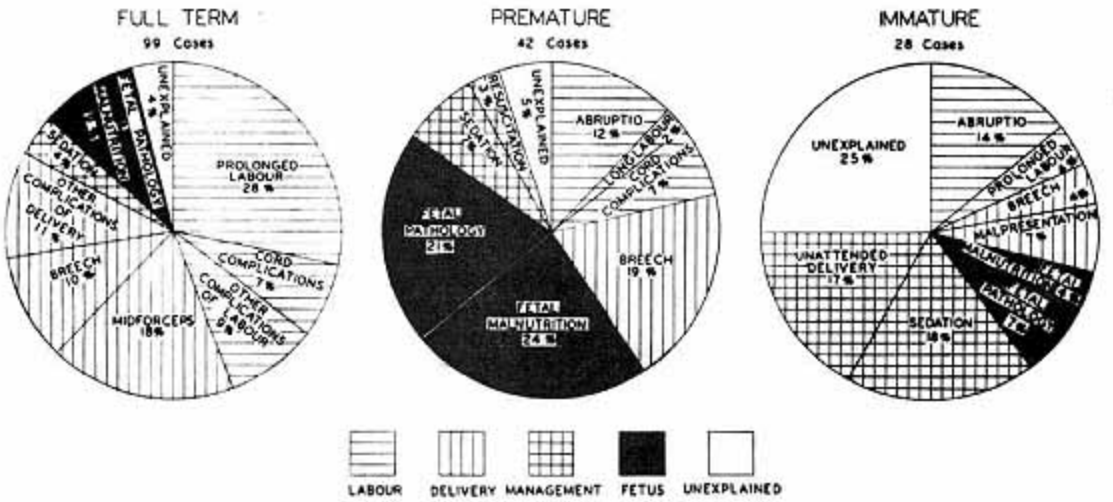

Fig. 1.—Causes of cerebral disturbances (severe asphyxia neonatorum, abnormal cerebral signs, and convulsions) in 99 full-term, 42 premature and 28 immature infants drawn from 10,995 consecutive live births.

TABLE IV.—INCIDENCE OF OBSTETRICAL COMPLICATIONS IN INFANTS WITH CEREBRAL DISTURBANCES AND IN CONTROLS

	Full-term		*Premature*		*Immature*	
Number of infants	*Cerebral disturbance (99)*	*Control (3431)*	*Cerebral disturbance (42)*	*Control (264)*	*Cerebral disturbance (28)*	*Control (28)*
Obstetrical complication:						
Labour						
Abruptio placentae	1%	0.6%	12%*	2.7%	18%	20%
Prolapsed cord	5 *	0.4	10 *	1.5	0	0
Prolonged labour, no distress	11 *	3.5	12 *	3.4	4	0
Prolonged labour with distress	19 *	1.3	2	0	0	0
Prolonged trial of labour	8 *	0.5	0	0	0	0
Unexplained fetal distress	12	14.7	0	12.9	0	12
Delivery						
Midforceps	29 *	5.6	0	0	0	0
Shoulder dystocia	8 *	0.5	0	0	0	0
Breech	15 *	3.0	43 *	9.5	32	29
Birth weight over 4500 g.	8 *	1.1	0	0	0	0
Malpresentation	5 *	0.8	7	3.4	14	0
Sedation within four hours	31	30.3	24	16.7	25	5

*Significantly increased incidence among infants with cerebral disturbance ($P<0.05$).

In *immature infants*, only 18% of cases were due to complications of labour, 11% to complications of delivery, and 7% to fetal pathology. In these small infants maternal sedation, unattended delivery and inadequate resuscitation accounted for 35%, and a further 29% remained unexplained.

Complications of labour and delivery accounted for 83% of cerebral disturbances occurring in full-term, 40% in premature, and 29% in immature infants so affected. Fetal pathology accounted for 8% of cases. Congenital anomalies were noted in three infants, Rh hemolytic disease in six, and intrauterine infection in five.

Thirty infants had cerebral disturbances which could not be attributed to obstetrical complications or to fetal pathology. Of these, 17 were underweight as judged from the calculated period of gestation or showed soft tissue wasting at birth. This incidence of fetal malnutrition (57%) among infants with otherwise unexplained cerebral disturbances can be compared to the incidence (10%) of such malnutrition in infants in the total hospital population.[22] If fetal malnutrition is accepted as a cause of intrauterine asphyxia, only 13 of 169 infants (7.5%) had cerebral disturbances without obvious obstetrical or fetal cause; seven of these 13 were immature.

ASSOCIATION BETWEEN OBSTETRICAL COMPLICATIONS AND CEREBRAL DISTURBANCES (Table IV)

The most probable primary cause of cerebral disturbance in each infant in this study was presented earlier. In order to demonstrate whether an association did in fact exist between obstetrical asphyxia or trauma and neonatal cerebral disturbance, a statistical analysis was performed. The incidence of various obstetrical complications in infants with cerebral disturbances was compared with that in controls. The following statistically significant differences were noted.

Full-term infants with cerebral disturbances had a higher incidence of prolapsed cord, prolonged labour with or without fetal distress, prolonged trial of labour, midforceps or breech delivery, shoulder dystocia, malpresentation, and a birth weight exceeding 4500 g. than did controls. Prolonged labour or prolonged trial of labour was noted in 38% of full-term infants with cerebral disturbances and in only 5% of the unaffected full-term population ($P < 0.001$). Midforceps delivery, breech delivery or other malpresentation occurred in 49% of full-term infants with cerebral disturbances but in only 9.4% of the unaffected full-term population ($P < .001$). A statistically significant association was not observed between cerebral disturbance and unexplained fetal distress, or maternal sedation within four hours of delivery.

Premature infants with cerebral disturbances had a higher incidence of abruptio placentae, prolapsed cord, prolonged labour and breech delivery than did controls. Abruptio placentae, prolapsed cord or prolonged labour occurred in 36% of affected premature infants and in only 8% of unaffected premature infants ($P < .001$). Forty-three per cent of premature infants with cerebral disturbances were the product of breech deliveries, compared with 9.5% of the unaffected premature infants ($P < .001$). An association could not be demonstrated between cerebral disturbance and unexplained fetal distress, midforceps delivery, malpresentation, or maternal sedation within four hours of delivery.

Among *immature infants*, maternal sedation was administered within four hours of delivery in seven of the 28 severely asphyxiated infants and in only one of the 28 unaffected ($P = 0.051$).

RISK OF CEREBRAL DISTURBANCE WITH OBSTETRICAL COMPLICATIONS (Table V)

On the basis of the relative incidence of obstetrical complications in affected infants and in the unaffected population, it was possible to de-

TABLE V.—RISK OF CEREBRAL DISTURBANCE OR TRAUMA

	Risk of cerebral disturbance (%)	*Risk of birth trauma* (%)
In all live births:	1.7	0.45
With complications of labour:		
Abruptio placentae	12.4	
Prolapsed cord	17.6	
Prolonged labour without fetal distress	4.4	
Prolonged labour with fetal distress	15.3	
Prolonged trial of labour	15.6	
Unexplained fetal distress	0.7	
With complications of delivery:		
Midforceps	5.0	3.6
Shoulder dystocia	13.3	22.0
Breech delivery (full-term only)	6.2	1.0
Large baby (over 4500 g.)	6.2	4.6
Malpresentation	11.1	0.0

termine the incidence or risk of cerebral disturbances in the infant whose birth was associated with specific obstetrical complications. The incidence of cerebral disturbance was highest (greater than 10%) with prolapsed cord, prolonged trial of labour, prolonged labour with fetal distress, shoulder dystocia, abruptio placentae, and malpresentation. There was a somewhat lower incidence following full-term breech delivery, midforceps delivery or prolonged labour without fetal distress, or when the birth weight exceeded 4500 g.

Fractures and Paralyses

(a) *Incidence*

Fracture of the clavicle occurred in 10 infants (0.9 per 1000 live births), and of the humerus and skull in two infants each. Facial nerve paralysis occurred in 25 infants (2.3 per 1000 live births), brachial nerve paralysis in 15 infants (1.4 per 1000 live births), and radial and phrenic nerve paralysis in one infant each. These 56 lesions occurred in 49 infants. Seven of these infants had combined lesions (five with combined brachial and facial nerve paralyses, one with brachial and phrenic nerve paralyses, and one with fractured humerus and radial nerve paralysis). All but two infants who had fractures or paralyses were full-term by weight.

The incidence of associated cerebral disturbances among full-term infants with fractures and paralyses was 10/47 or 21%, considerably higher than the 0.87% incidence in the remainder of the full-term infant population ($P < .0001$).

(b) *Causes of Fractures and Paralyses* (Fig. 2)

Sixteen infants with facial nerve paralyses were delivered by midforceps, while in the remaining nine infants there were no evident abnormalities of labour or delivery to account for the lesion.

Of 15 infants with brachial paralysis, seven were delivered by midforceps, six had shoulder dystocia,

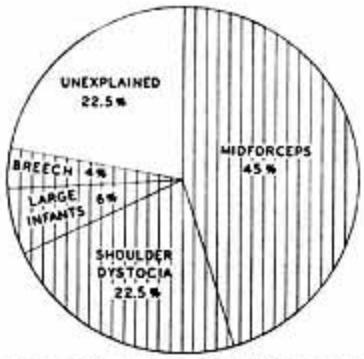

Fig. 2.—Causes of fractures and peripheral nerve paralyses in 47 full-term and two premature infants in a series of 10,995 live births.

one was a second twin delivered by breech extraction, and the last was a 4840-g. infant delivered to a primiparous mother.

Of 10 infants who had fracture of the clavicle, six had shoulder dystocia, another was a large infant (4760 g.) without recorded shoulder dystocia, and in the remaining three the cause was unexplained. The infant who sustained a fractured humerus and radial nerve paralysis weighed 5440 g. and was delivered by version and extraction because of a prolapsed cord.

Of the two skull fractures, one was associated with a difficult midforceps delivery carried out after a 36-hour labour, the infant dying at age 12 hours with subdural and scalp hemorrhages. The other skull fracture was caused by difficult forceps delivery of the aftercoming head in a breech presentation after a 32-hour labour, the infant dying immediately after birth.

(c) *Risk of Fracture or Paralysis with Obstetrical Complications* (Table V)

Fracture or paralysis occurred in 22% of deliveries complicated by shoulder dystocia, in 4.6% where the infants weighed more than 4500 g., and in 3.6% where midforceps were used.

TABLE VI.—ASSOCIATION BETWEEN OTHER OBSTETRICAL FACTORS AND BIRTH ASPHYXIA AND TRAUMA

	Population	*Incidence of asphyxia and trauma* No.	%	*Statistical significance of difference*
Parity: primiparity	3440	92	2.67	
multiparity	7555	116	1.54	$P < .001$
Sex: male	5560	127	2.28	
female	5435	81	1.49	$P < .01$
Gestational age: (over 2500 g. only)				
42 weeks and less	9573	136	1.42	
43 weeks and more	649	8	1.23	Not significant
Maternal age:				
(a) Primipara: 35 and over	110	8	7.27	
up to 34 yr.	3317	84	2.53	$P < .01$
(b) Multipara: 35 and over	1118	30	2.68	
up to 34 yr.	6450	86	1.33	$P < .05$
*Hospital status:** public	4453	65	1.46	
private	6542	46	0.70	$P < .001$

*Including only those cases of asphyxia and trauma associated with complications of delivery

ASSOCIATION BETWEEN OTHER OBSTETRICAL FACTORS AND BIRTH ASPHYXIA AND TRAUMA (Table VI)

A statistically significant increase in the incidence of asphyxia and trauma was noted in primiparous mothers, in elderly mothers, and in male infants. A higher incidence was not encountered in post-mature infants. The incidence of asphyxia and trauma associated with complications of delivery was twice as high on the ward service as on the private service.

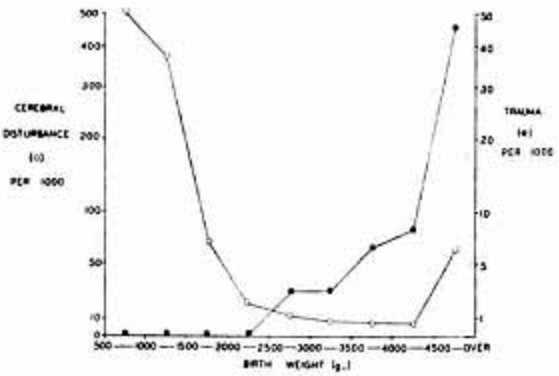

Fig. 3.—Incidence by 500 g. weight groups of cerebral disturbances (O) and of fractures and paralyses (●) per 1000 live births.

The incidence of asphyxia and trauma by 500 g. weight groups is shown in Fig. 3. An increased incidence of cerebral disturbance was seen among low-weight infants, and of fractures and paralyses among high-weight infants.

NEONATAL DEATHS CAUSED BY ASPHYXIA AND TRAUMA

Twenty-seven of the 28 immature infants, 18 of the 44 premature infants, and seven of the 134 full-term infants who had cerebral disturbances, fractures or paralyses died in the neonatal period. It was difficult to determine the contribution of asphyxia and trauma to the fatal outcome in immature and premature infants. A definite cause-and-effect relationship between antenatal asphyxia or trauma and neonatal death was obvious in the full-term infants.

All seven full-term deaths were the direct result of complications of labour or delivery. As previously mentioned, two infants died from skull fractures. Three died from meconium aspiration which occurred in association with prolapsed cord in one case, with midforceps delivery in another, and with prolonged labour in the third. One infant died of a large subdural hemorrhage following midforceps delivery. The seventh died from cerebral edema resulting from prolonged trial of labour and three failed forceps applications, the infant appearing to be decerebrate from birth until death.

The neonatal mortality rate among full-term infants with cerebral disturbances or trauma was 5.2%, a rate 33 times greater than the neonatal mortality rate of 0.16% among unaffected full-term infants ($P<0.001$). These seven full-term deaths due to obstetrical asphyxia and trauma represented 30% of the total full-term neonatal mortality during this period.

DISCUSSION

This study showed that the neonatal conditions selected for investigation were valid indicators of birth asphyxia and trauma. The vast majority of infants with severe asphyxia neonatorum, abnormal cerebral signs, convulsions, fractures and paralyses either had a history of asphyxiating or traumatic obstetrical complications, or had associated fetal pathology which could have produced the result noted.

Studies of children with cerebral palsy have shown that at birth such children had a higher incidence of asphyxiating and traumatic obstetrical complications[1, 4, 8, 12, 19-21] and also a higher incidence of neonatal cerebral disturbances[9, 20] than normal control infants. The immediate effects of birth asphyxia and trauma seem to be manifested by asphyxia neonatorum, abnormal cerebral signs, and convulsions. In some instances permanent injury has resulted and cerebral palsy develops. In the majority, however, the injury is apparently temporary and reversible, cerebral function recovering completely.[5, 7]

In the present study no apparent differences were found between the neonatal cerebral manifestations of asphyxia and those of trauma. Similar cerebral disturbances were noted following potentially asphyctic complications of labour and potentially traumatic complications of delivery. Infants who sustained definite traumatic lesions (fractures and paralyses) during delivery often had associated cerebral disturbances, presumably asphyctic in origin. Burnard[6] has suggested that direct trauma to the brain is probably uncommon, brain damage in traumatic delivery usually being due to the associated asphyxia of a difficult delivery.

In two-thirds of the cases, birth asphyxia and trauma in full-term infants were the direct consequences of prolonged labour or difficult delivery. If it had been possible to predict these events in these cases and avoid them by abdominal delivery, the Cesarean section rate would have been increased by only 0.8%. The higher incidence of birth asphyxia and trauma on the ward service associated with complications of delivery (primarily midforceps or breech delivery, and shoulder dystocia) may have been due to less experienced obstetrical care, to a higher incidence of abnormal pelves, and to a lower Cesarean section rate (3.9% *vs.* 5.9% on the private service), common to many teaching institutions.[15]

Prematurity, although it predisposes to neonatal cerebral disturbances, was rarely the sole factor operating in any individual case. Even in premature infants abnormal delivery played a significant role, as one in five cases of cerebral disturbance were considered to be due to difficult breech delivery, and there was a statistically significant association between breech delivery and cerebral disturbance. Another one-quarter of the infants with cerebral disturbances in the premature-weight group were not truly premature at all. They were low-weight full-term infants who suffered from fetal malnutrition, a condition in which there is a predisposition to intrauterine asphyxia.[22]

Among immature infants, the difficulty in assessing progress of labour and in hearing the fetal heart during labour often resulted in sedation being given shortly before delivery, an unattended delivery, or inadequate resuscitation following delivery. Only too frequently the immature infant was delivered unexpectedly into the bed, or a living but sedated immature infant was born after being considered dead.

It was discouraging to note that in spite of obstetric teaching to the contrary, sedation was often given shortly before delivery to mothers delivering prematurely and to mothers with complications of labour and delivery which already represented severe handicaps to the fetus. Maternal sedation was employed in approximately one-sixth of all premature and immature infants delivered during the period under study. Mothers of one-third of the full-term infants with cerebral disturbances were sedated even though prolonged labour, fetal distress, and some degree of cephalopelvic disproportion were usually present.

The causes of fractures and peripheral nerve palsies were similar to those described previously;[10, 13, 16, 23, 25] it was again noted that many facial palsies and clavicular fractures are associated with normal, spontaneous deliveries. Excessive traction of the shoulder on the neck has been considered to be the cause of brachial plexus palsy.[23, 25] This accounts for the prevalence of this lesion in breech deliveries and in association with shoulder dystocia. However, seven of the 15 infants with brachial plexus palsy in the present series had neither of these complications, but were delivered by midforceps extraction. It seems likely that traction on the head or rotation of the head during midforceps delivery may exert sufficient tension on the brachial plexus to produce paralysis.

We would like to recommend that the 2% (approximately) of surviving infants who present the neonatal manifestations of birth asphyxia and trauma analyzed here, undergo monthly review at the maternity hospital perinatal mortality conferences. These cases provide a sensitive index of the standard of obstetrical practice, in many ways a more sensitive index than that obtained by review of perinatal deaths alone. Such a review would foster a critical attitude toward current obstetrical practices. It would focus attention upon those obstetrical complications and methods of management which most frequently lead to birth asphyxia and trauma, and thereby stimulate development of improved obstetrical techniques.

On the strength of the experience gained during this investigation, the following recommendations can be made. Anticipation of obstetrical difficulties begins with a careful review of the obstetrical history. Records of previous deliveries must be readily available. The progress of labour should be accurately monitored by hourly assessments. Labour should never be allowed to exceed 16 hours in a multipara or 24 hours in a primipara without consultation with a senior member of the obstetrical attending staff. Deliveries associated with prolonged labour, malpresentation, or high fetal risk such as prematurity, should always be attended by an experienced obstetrician.

Only by a more judicious use of Cesarean section in cases of borderline disproportion can the contribution of prolonged labour and difficult delivery in the production of birth asphyxia and trauma be eliminated. The risk of Cesarean section to the fetus in the form of respiratory distress syndrome appears to be nil in cases done for disproportion, probably because these are almost always done at term.[24]

Sedation should be avoided in premature labour and in full-term labour where the fetus is already jeopardized by obstetrical complications.[14] Conduction analgesia is a safer alternative in such situations.

The infant suffering from fetal malnutrition, whose existence can be suspected ante partum from the failure of maternal abdominal growth and from decreased maternal estrogen output, has a high risk of asphyxia during labour and at birth.[3, 11] Prolonged labour, difficult delivery, and sedation should be avoided when fetal malnutrition is suspected.

Case Reports

Four full-term infants in whom alternative obstetrical management would have avoided fetal injury are described. They represent the four most tragic cases among 208 instances of asphyxia and trauma occurring in 10,995 consecutive live births, in a hospital with a very low perinatal mortality record. These cases illustrate the need for constant vigilance if birth asphyxia and trauma are to be reduced to the absolute minimum compatible with present-day knowledge and techniques.

Case 1.—Prolonged trial of labour with fetal distress in an infant with fetal malnutrition syn-

drome, resulting in severe asphyxia neonatorum, abnormal cerebral signs, convulsions and death.

The mother, a 23-year-old primipara, was admitted to hospital at 40 weeks' gestation because of moderate pre-eclampsia and because the fetal head did not engage at term. She was discharged a few days later and readmitted in labour at 41⅛ weeks' gestation, having had contractions at home for 22 hours. On admission the cervix was 2 cm. dilated and the vertex was 2 cm. above the level of the ischial spines (spines minus two), with a left occiput transverse presentation. Radiographic pelvimetry at this time showed a small platypelloid pelvis with an anteroposterior diameter of 9.8 cm. After 12 hours of strong labour in hospital, the membranes ruptured, emitting a liquor heavily stained with meconium. The fetal heart was faintly audible at a rate of 118/min., later returning to normal. Vaginal examination showed 4-cm. cervical dilatation, the head at spines minus one, and the presentation still left occiput transverse. Six hours later a Cesarean section was performed under epidural anesthesia.

The infant weighed 2670 g., was severely asphyxiated with an Apgar score of 3, and required five minutes of resuscitation with bag and mask before respirations began. Definite soft-tissue wasting was noted. The neonatal course was marked first by cerebral depression, then hyperirritability leading to convulsions and finally status epilepticus at age 4 days. Although convulsions were eventually controlled, he remained in a decerebrate condition until death at 14 days of age. Postmortem examination showed meconium aspiration but no cerebral hemorrhage or tentorial tear. The cause of death was considered to be cerebral anoxia. This death was not reported as a neonatal death in this study, as it occurred after 7 days of age.

CASE 2.—Prolonged labour and difficult midforceps delivery resulting in moderate asphyxia neonatorum, facial paralysis, and multiple intra- and extra-cranial hemorrhages resulting in death.

The mother, a 28-year-old primipara, was admitted to hospital at 41 weeks' gestation in labour, the antepartum course having been normal. After eight hours of labour the cervix was 3 cm. dilated, and the vertex was at the ischial spines with a left occiput posterior presentation. After a further 28 hours of labour the cervix was only 4 cm. dilated. Radiographic pelvimetry showed an adequate gynecoid pelvis. Membranes were now ruptured and an oxytocin drip was started. Eighteen hours later (46 hours after the onset of labour), the cervix was fully dilated and the infant was delivered following a difficult midforceps procedure. At no point during the labour was fetal distress noted.

The infant weighed 3500 g. and was moderately asphyxiated (Apgar score of 6). He had a facial paralysis, bilateral large cephalohematomas, and a scalp laceration. Subsequently he developed a coagulation disorder, anemia with a hemoglobin concentration of 10 g. %, and congestive heart failure. There was evidence of severe cerebral damage with marked and continued depression, repeated convulsions, and a tense fontanelle with spread sutures. He died at 64 hours of age in spite of transfusions of fresh blood and plasma and subdural taps. Postmortem examination showed an extensive left subdural hemorrhage and softening and hemorrhage at various sites of the brain. The cause of death was considered to be a combination of cerebral trauma and anoxia.

CASE 3.—Difficult midforceps delivery resulting in severe asphyxia neonatorum, abnormal cerebral signs and permanent severe cerebral damage.

The mother, a 30-year-old foreign-born patient who spoke no English, had had one previous uneventful pregnancy terminating at 7½ months. After a normal antenatal course in the present pregnancy, she was admitted in labour at 37 weeks' gestation. Pelvic assessment early in pregnancy was considered adequate. After six hours of labour the cervix was fully dilated but the vertex was only at the ischial spines, in a left occiput anterior position, and a large caput was noted. After a further two hours of second-stage labour with little progress, she was anesthetized with ether and three attempts were made to apply forceps. On the third attempt the infant was delivered after the use of considerable traction.

The infant weighed 2920 g. and had severe asphyxia neonatorum with an Apgar score of 4, requiring 10 minutes of bag-and-mask resuscitation before the onset of spontaneous respirations. He showed marked cerebral depression and irritation for days after birth, and before 2 years of age was admitted to an institution because of severe mental retardation.

A postpartum pelvic examination showed a very short anteroposterior diameter, and Cesarean section delivery was recommended for subsequent pregnancies.

CASE 4.—Prolonged second stage of labour and failed forceps applications leading to severe asphyxia neonatorum, facial palsy, cerebral irritation, convulsions and finally neonatal death at age 5 days.

The same mother as described in Case 3 was admitted during her next pregnancy two years later at 37 weeks' gestation in strong labour of four hours' duration. She had been followed up in the antenatal clinic but, because of a language barrier and failure to review the previous chart, the history of the previous difficult delivery was not elicited. On examination at the present admission the cervix was fully dilated and the head was at the spines minus one. Forceps application was attempted three times, and finally abandoned because the head was too high. After a total of three hours in second-stage labour, a Cesarean section was performed under epidural anesthesia.

A 3300-g. infant was delivered who had severe asphyxia with an Apgar score of 1; three minutes of resuscitation was required before spontaneous respirations began. The infant had a left facial paralysis and developed cerebral irritation, convulsions, and congestive heart failure, dying at five days. Postmortem examination revealed aspiration of squames but no cerebral hemorrhage or tentorial tear. The cause of death was considered to be cerebral anoxia during a prolonged second stage of labour.

SUMMARY AND CONCLUSIONS

The causes of birth asphyxia and trauma at one maternity hospital were studied in 208 affected newborn infants selected from a total population of 10,995 consecutive live births. Common obstetrical complications accounted for 73% and fetal pathology for another 15% of the cases.

From this study it appears that efforts to reduce the incidence of birth asphyxia and trauma should be concentrated on the following areas:

1. Earlier recognition of compromised fetal status and more frequent consultation in prolonged labour.

2. Attendance of a senior obstetrician at all complicated deliveries.

3. More liberal use of Cesarean section in women with borderline disproportion.

4. Avoidance of sedation in association with premature labour or in the presence of potentially hazardous obstetrical complications.

5. Recognition that erythroblastosis and fetal malnutrition predispose to severe asphyxia neonatorum and require exceedingly cautious obstetrical management without sedation.

6. Adoption of measures to reduce the increased risk to the ward-service patient of birth asphyxia and trauma associated with abnormalities of delivery.

7. Provision of adequate forewarning of impending delivery of the high-risk infant—especially the immature—to allow time for a well-controlled delivery and to provide experienced resuscitation immediately thereafter.

It is suggested that a monthly review of deliveries resulting in birth asphyxia or trauma be adopted as standard maternity hospital procedure.

Figures were drawn by Miss Lois Coons, R.N., and statistical analysis was performed by Dr. J. D. Sharp.

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