ASPHYXIA AND BIRTH INJURIES AS THE CAUSE OF SPASTICITY AMONG PREMATURE INFANTS*

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Little in 1861 was the first person to think that, with rare exceptions, cerebral palsy was due to complications of labor with the common factor in all cases being asphyxia.[1] This problem has been much debated among many investigators and there have been some who supported his theory and others who opposed it. In 1940, Schreiber called attention to the frequency of neurological defects present in infants who were in serious oxygen need at the time of birth.[2] Lilienfeld and Parkhurst also showed that the incidence of cerebral palsy was above average among families which had a high incidence of neonatal deaths in siblings.[3]

Evans, on the other hand, called attention to the high incidence of neonatal anoxia particularly in the athetoid group.[4] Courville in 1961 also defended the theory of anoxia and pointed out that anoxia at birth might be due to a severe hemorrhage caused by premature separation of the placenta (as in placenta previa).[5] This situation usually followed depression of the fetal cardiac and respiratory centers secondary to increase in the intracranial pressure. He noted that if the respirations alone failed (asphyxia livida) the outlook was less serious, but if both respiratory and cardiac activity were depressed (asphyxia pallida) the possibility of residual brain damage in the infants who survived was greatly increased.

In reviewing different statistics, several controversial ideas and figures were encountered. For instance, Keith and Gage, in their investigation of 180 children who were anoxic at birth, found that the only neurological manifestation was febrile convulsions. On the other hand it was thought that anoxia was the most important factor in brain damage even though the fetus and the newborn were more resistant to oxygen lack than in later childhood.[6] Also premature detachment of the placenta, interruption of the circulation in the cord

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(prolapsed cord), tonic contracture of the uterus with pressure on the fetal head, prolonged labor, oxytocic drugs used in the first stage of labor, intrauterine manipulations, early rupture of the membranes and analgesics given under certain conditions could endanger the supply of oxygen to the fetus and cause neonatal asphyxia.

Atelactasis, aspiration of amniotic fluid, mucus, blood or meconium in addition to the delay in the onset of respiration are the most common causes of asphyxia in the neonatal period. In Nabors' statistics, in 1960, anoxia showed an incidence of 10 per cent in all spastics, 26 per cent in all athetoid and 31 per cent in all "rigidities".[7] McPhail and Hall in their work on the possible late effects of anoxia in the newborn infant published in 1941, noted that the incidence of severe asphyxia was more common among cases born after a very short labor, after early ruptured membranes, after administration of analgesics and among babies with a birth weight between 1,000-2,500 grams.[8] This last figure also confirmed the relation of birth weight to other associating factors such as anoxia, and supports the theory of the fragility of vessels of the premature baby.

Sjösted in 1957 recommended that the role of anoxia not be overestimated and gave as an example the survival of infants delivered by cesarean section of mothers under inhalation anesthesia even when the mother had been dead in some cases from 15 to 20 minutes.[9] After extensive studies measuring the oxygen saturation in the umbilical arteries and veins, Sjösted accepted the view that asphyxia could be due not only to hypoxia but also to other factors such as brain injury or deformity. In two of their clinically asphyxiated children, who died from brain injury within a few days of birth, the oxygen saturation was normal, and therefore traumatization during delivery was suggested as the main factor in producing these brain injuries.

Obstetrical factors as possible causes of spastic diplegia: Asher and Schonell in their studies in 1950 of 221 full-term babies, found 87 cases with a history of abnormal labor and showed that the incidence of abnormal labor among children who were born with a cerebral birth injury was remarkably increased (65.5 %).[10] Those who were born without birth injury and abnormal parturition made up only 34.5 per cent. On the other hand, the incidence of birth injury among children whose mothers had normal labor was only 22.6 per cent. Many statistics give evidence that abnormal labor and birth injuries are more common among athetoids than among spastics. It is interesting to note that the athetoid group showed the largest percentage

of mothers who had abnormal labor (65 per cent) and the lowest percentage of premature babies (28 per cent). The opposite was true for spastic diplegia: Similarly, in their series of twins, there was a higher incidence of spastic twins than athetoid.

Potter in 1949 showed that prematurity itself in infants was not the significant reason for increased mortality. He also observed that breech and cesarean sections were the most common methods of delivery in the high mortality group.[11]

Smith in his analysis of the problem ten years later concluded that with the exception of breech delivery there was very little difference in the mortality rate among the common obstetrical methods of delivery.[12]

It should be remembered that, even at the present time, cesarean section babies are not completely free from possible intracranial hemorrhage. Earlier, Ehrenfest had compiled 19 such cases.[13] In Ford's study, in 1960, of 33 cases in which the children were born of mothers who had had difficult labor and had symptoms of injury at birth, there was only one case which developed cerebral diplegia.[14] In a very large series of 539 newborn babies with signs of intracranial irritation, Craig, in 1950, observed 56 cases with residual mental and physical defects, 41 of these cases were also associated with various types of cerebral palsy.[15]

Material and Methods

The spastic probands were ascertained by examining the patients and reviewing the medical records of the probands of those who are registered with the Cerebral Palsy Rehabilitation Center of the Montreal Children's Hospital. All cases who showed definite evidence of spasticity, without other forms of cerebral palsy, were included in this study. These children were divided into two groups: (1) those born prematurely, and (2) those born at full term. The histories of 431 spastic children were investigated of which 146 had a birth weight of five and a half pounds or less.

Etiological Factors were studied in three major groups which were also divided into subgroups:

1. *Prenatal factors:* a) Faulty placenta, b) Toxemia, c) Prenatal hemorrhage, d) Infection and e) Radiation. Among these factors,

thought to be the most important as a presumptive factor, only prenatal hemorrhage showed a significantly higher precentage among prematures than among non-prematures. This was also true for the total and separate groups of quadriplegics (see Table 1).

TABLE 1

PRENATAL FACTORS

	Prematures No. of Pt.	% affected	Non-prematures No. of Pt.	% affected	P
Faulty placenta	3	2.22 ± 1.27	2	0.89 ± 0.63	0.30
Toxemia	2	2.22 ± 1.27	5	2.27 ± 0.98	—
Prenatal hemorrhage	20	14.81 ± 3.06	6	2.67 ± 1.07	0.001
	—	— —	—	— —	—
Radiation	—	— —	—	— —	—

Faulty placenta is a term which covers generally the faulty placement of the placenta, such as placenta previa, or early separation of the placenta. According to our observations, faulty placenta was not a significant factor among prematures or non-prematures.

Toxemia did not prove to be one of the important factors in either group.

2. *Natal Factors:* a) Prolonged labor, b) Precipitated labor, c) Induced delivery, d) Forceps delivery, e) Presentation (cephalic, breech, cesarean section).

Among the factors which are the most important during the phase of actual birth, only prolonged labor and forceps delivery were found to be significantly higher among the prematures. Different presentations of the head did not seem to be a contributing factor to this disorder in either group. Incidence of breech delivery was high in both groups compared with the total population (see Table 2).

3. *Postnatal Factors:* a) Asphyxia, b) Blood incompatibility, c) Cerebrovascular accidents, d) Metabolic diseases, e) Infections, f) Head trauma, and g) Incubation. None of the several post-natal factors showed any significantly higher incidence among either group with the exception of incubation during the first two to three weeks of life (see Table 3). This finding among the prematures during the neonatal period is only natural, since most of the premature babies

are placed in incubators, regardless of the severity of their clinical condition. On the other hand, the incidence of incubation of the full-term spastics is very high and indicates that these children were obviously in difficulty at birth.

TABLE 2

FACTORS DURING THE PHASE OF ACTUAL BIRTH

	Prematures No. of Pt.	% affected	Non-prematures No. of Pt.	% affected	P
Prolonged labor	9	6.67 ± 2.15	39	17.33± 2.58	0.001..
Precipitated labor	14	10.37 ± 2.62	18	8.00 ± 1.81	0.40
Induced delivery	2	1.48 ± 1.04	3	1.33 ± ...76	0.90
Forceps delivery	17	12.59 ± 2.86	50	22.22 ± 2.77	0.02..
Cephalic presentation	121	89.63 ± 2.62	201	89.33 ± 2.06	0.90
Breech delivery	11	8.14 ± 2.35	23	10.22 ± 2.02	0.50
Cesarean section	3	2.22 ± 1.27	3	1.33 ± 0.76	0.50

TABLE 3

POSTNATAL FACTORS

	Prematures No. of Pt.	% affected	Non-prematures No. of Pt.	% affected	P
Asphyxia	59	43.70 ± 4.27	84	37.33 ± 3.22	0.20
Blood incompatibility	5	3.42 ±0.015	7	2.46 ±0.009	20.80
C.V.A.	0	— —	16	7.11 ± 1.71	—
Metabolic diseases	0	— —	4	1.78 ± 0.88	—
Infection	1	0.74 ± 0.74	9	4.00 ± 1.31	0.05
Head Trauma	1	0.74 ± .074	5	2.22 ± 0.98	0.20
Incubation	116	85.93 ± 2.99	74	32.89 ± 3.13	0.001

Conclusions

Among the 431 spastic children, the incidence of prematurity was found to be 33.8 per cent (146 cases). Comparisons were made between premature and full-term spastics according to the various associated characteristics, etiological factors, neurological involvement, severity of the disorder, intelligence quotient and seizure tendency. Only etiological factors are discussed in this paper. Our results were as follows:

1. *Prenatal:* No difference was found between premature and full-term children as far as various prenatal factors were concerned. An exception was antepartum hemorrhage which was more common in prematures. But this particular factor was also found to have a high incidence among normal prematures and could not be accepted as one of the main causes of spasticity among these children.

2. *Paranatal:* There was no significant difference in the duration of labor between the groups when they were compared with the total population. Prolonged labor was higher among full-term babies; precipitate labor was higher among premature babies. Forceps delivery was found more commonly among non-prematures. The various presentations of the child at birth were equally distributed between both groups although a significant observation was the higher incidence of breech deliveries among full-term spastics compared to the total population, whereas the premature spastics had a lower incidence of breech delivery compared with total premature births. Cesarean section showed an equal incidence between both groups even though this was relatively lower than the incidence among the total population.

Respiratory difficulties (neonatal anoxia): There was a considerably higher incidence of respiratory difficulties in both groups compared to normal newborns.

3. *Post-natal:* None of the considered factors, such as blood incompatibility, CNS disturbances, neonatal infections, and metabolic disturbances, were found significantly different or higher in either of these two groups. Incubation during the neonatal period showed a very high incidence among premature spastics and a considerably higher incidence among non-premature spastics which was thought to be very significant.

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