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Some Aspects of Neonatal Convulsions.

by

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Introduction

Not many articles can be found in the literature which consider neonatal convulsions as the main subject. Most of the authors regard the convulsions as an irritative cranial disorder, being a consequence of accompanying symptoms (Bound, Butler and Spector, 1956; Craig, 1960).

Burke (1954) in his study of 46 newborn infants, has already pointed out the significant prognostic value of convulsions.

Craig (1965) in his paper on convulsions during the first 10 days of life, reported autopsy findings and put forward suggestions in the probable etiology of the convulsions, he also did a follow-up study of the remaining children for a period of more than 3 years.

Schwartz (1965) in his literature study on neonatal convulsions gave a full description of the clinical picture of various types of convulsions in the neonatal period and the most probable etiologic factors.

It is obvious from the literature that the occurence of convulsions in the neonatal period is a significant disorder, especially in relation to the prognosis and the high number of neurologic sequelae found in follow up studies (Peterman, 1954; Mc. Inerny and Schubert, 1969).

The purpose of this paper is to review and evaluate the clinical data on incidence, sex, body-weight, morbidity and mortality and the possible etiology of convulsions occurring in the neonatal period (i.e. the first 30 days of life) and to study the outcome in follow up examination at the age of more than 6 months. The age of 6 months is taken as a limit because previous experiences and those of other authors (Billing, 1964)

Received 9th Dec. 1973.

*) Presented at the Second National Indonesian Pediatric Congress, Bandung 21-25 April 1971.

have shown that only after the age of 6 months neurological and mental disorders remain evident and permanent.

Material and Methods

All babies under 30 days of age, who had convulsions and were admitted to the Department of Child Health, Dr. Tjipto Mangunkusumo General Hospital, Jakarta, between January 1, 1969 and October 31, 1970, were used as material. Neonatal tetanus was excluded.

Collection of data was done by retrospective review of the medical records of all infants under 30 days of age, who were discharged with the diagnosis of neonatal convulsions or who developed convulsions in the course of a certain disease. Of babies born in the above mentioned hospital perinatal data were available.

Examinations consisted of routine physical and neurological examinations, including head circumference, skull transillumination, X-Ray picture of the skull and in a number of cases also electro-encephalography. Laboratory examinations in the majority of those babies consisted of blood glucose, calcium, phosphor, sodium and potassium level determinations, the cerebrospinal fluid determination and urine analysis including phenylketon detection.

Follow up was carried out by persuading the parents to come for further examinations in the Neurologic Subdivision of the Child Health Department, when the babies reached the age of 6 months and over. A thorough evaluation of the perinatal history, examination of the mental and motoric development, and of the behaviour of the child at home, were carried out next to the conventional physical and neurological examinations. If required a psychologist was consulted. Examinations were done regularly once a month.

Results

Ninety two babies were found to have convulsions before the age of 30 days in an observation period of 22 months. Out of the 92 babies, 35 were born in the above mentioned hospital, whereas 57 came from outside.

There were 4987 deliveries in the hospital during the observation period, so that the hospital incidence of neonatal convulsions was 0.70% (35 : 4987).

The cases were divided according to the age when the first convulsion occurred, respectively before the age of 24 hours, between 24 hours and 7 days, and over 7 days. (table 1) The majority the cases developed their first convulsions before 7 days of age. There was no significant difference in sex.

Etiologic factors which possibly caused the convulsions were shown on table 2.

More than half of the babies (49) experienced difficulties at the time of birth. Out of the 19 cases classi-

TABLE 1 : *Number of cases according to the age of occurrence of the first convulsion.*

AGE	SEX		NUMBER OF CASES
	MALES	FEMALES	
I. Less than 24 hours	17	26	43
II. 24 hours - 7 days	15	18	33
III. More than 7 days	9	7	16
TOTAL NUMBER	41	51	92

fied as unknown, some had no sufficient data to establish a diagnosis, and some had no underlying cause to determine the etiology. Of the remaining babies, 3 had their convulsions while suffering from gastroenteritis with dehydration and acidosis, 10 were prematures, 5 had C.N.S. infections, of whom 4 suffered from purulent meningitis and one was discharged with the diagnosis of encephalitis.

Of the group with metabolic disorders 3 babies had hypoglycemia, hypocalcemia and kernicterus. Developmental anomalies were found on 3 babies. The number of cases with different birth-weights were shown on table 3.

The highest frequency was found between 2500 - 3500 grams.

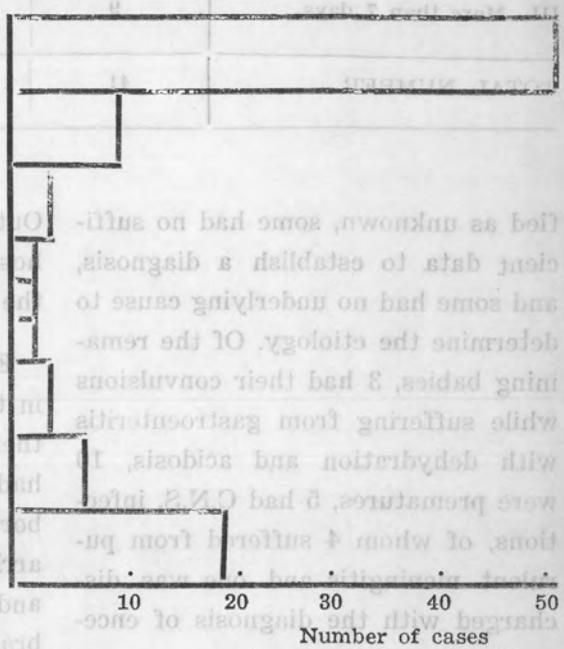
Out of the 92 infants, 40 died during hospitalization (Table 4), 33 before the seventh day of life.

E.E.G. was performed on 21 cases in the neonatal period or one month there after; 12 were found normal, 5 had an irritative focal lesion, 2 were borderline abnormal, 1 showed hypsarrhythmia and 1 had dysfunction and inactivation on both sides of the brains (Table 6).

Out of the 52 cases that were discharged from the hospital, 1 died at the age of 2 months, 17 were lost for follow up, 3 were not included in the follow up as they were less than 6 months of age on the last examination, and 31 were examined almost regularly every month. The youngest baby on follow up was 6 months and the oldest 23 months old.

TABLE 2 : *Etiology of neonatal convulsions*

PERINATAL COMPLICATIONS	49
PREMATURITY	10
DEPELOPMENTAL ANOMALIES	3
HYPOGLYCEMIA	1
HYPOCALCEMIA	1
KERNICTERUS	1
GASTROENTERITIS + DEHYDRATION + ACIDOSIS	3
INFECTION	5
UNKNOWN	19



PERINATAL COMPLICATIONS :

difficult resuscitation	11
forceps/vacuum extraction	11
breech	8
Cesarian Section	15
miscellaneous	4

TABLE 3 : *Birthweights of infants with neonatal convulsions.*

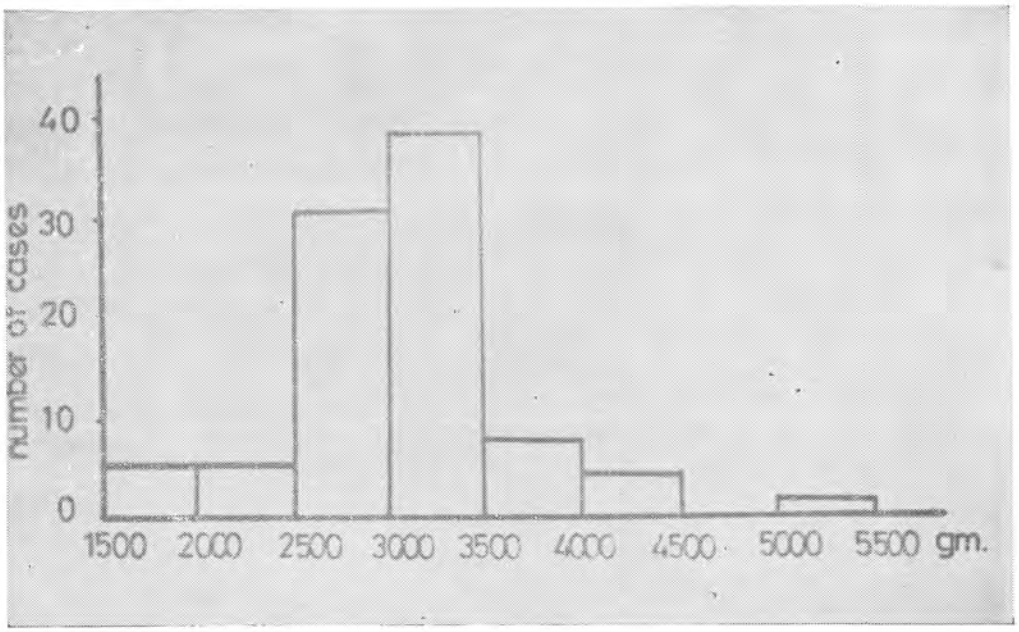


TABLE 4 : *Number of deaths in the three groups*

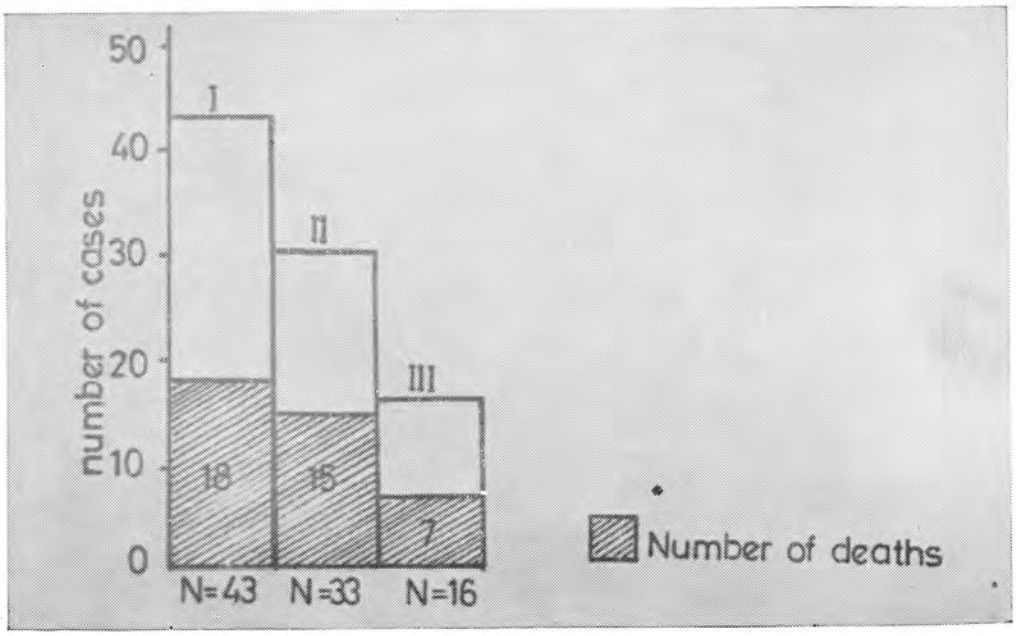
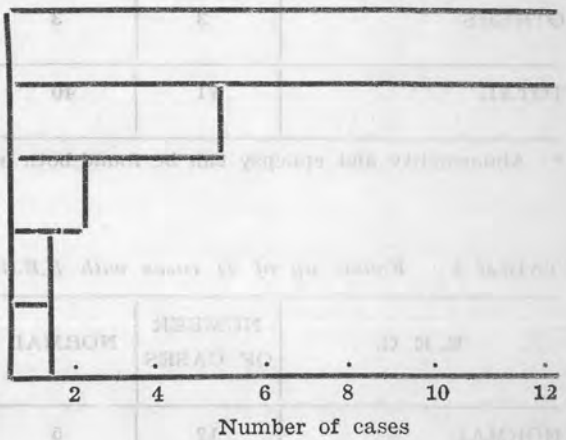


TABLE 5 : *Type of convulsions found in neonatal convulsions.*

TYPE OF CONVULSIONS	NUMBER OF CASES
I. GENERALIZED	52
II. FOCAL	35
III. UNKNOWN	5
TOTAL	92

TABLE 6 : *E.E.G. Pattern of 21 cases.*

NORMAL	12
IRRITATIVE FOCI	5
BORDERLINE ABNORMAL	2
HYPSARRHYTHMIA	1
DISFUNCTION AND INACTIVATION	1



Discussion

Of the 92 babies who had convulsions in the first 30 days of life, 76 (80%) experienced their first convulsions during the first 7 days.

The incidence of 0.70% is comparable to that quoted in the literature, i.e. from 0,2% to as high as 0,8% (Harris and Steinberg, 1954; Craig, 1960).

It is obvious that perinatal complications involve more than half of the total number of neonatal convulsions (table 2).

Peterman (1949) reported cerebral birth injury as the cause of 68% of 176 infants with convulsions during the first month of life.

Craig (1960) found 78% with cerebral etiology on postmortem examinations of babies who died in the neonatal period with convulsions before the tenth day of life. Mc. Inerny and Schubert (1969) on the other hand, found only one third of 95 cases of

TABLE 7 : *Etiology.*

	NUMBER OF CASES	DEATH	NORMAL	ABNORMAL	EPILEPSY
PERINATAL COMPLICATION	40	20	12	7*	7*
PREMATURITY	10	10	—	—	—
GASTRO-ENTERITIS DEHYDRATION AND ACIDOSIS	3	1	2	—	—
INFECTION	5	2	2	1	—
UNKNOWN	10	4	1	5*	2*
OTHERS	3	3	—	—	—
TOTAL	71	40	17	13	9

* Abnormality and epilepsy can be found both in the same patient.

TABLE 8 : *Follow up of 21 cases with E.E.G. examinations.*

E. E. G.	NUMBER OF CASES	NORMAL	ABNORMAL	EPILEPSY	UNKNOWN
NORMAL	12	5	2*	2*	4
BORDERLINE ABNORMAL	2	—	2*	1*	—
IRRITATIVE FOCI	5	—	5*	3*	—
HYPSARRHYTMIA	1	—	1*	1*	—
DISFUNCTION AND INACTIVATION	1	—	1*	—	—
TOTAL	21	5	10 11	7	4

* Abnormality and epilepsy can be found both on the same patient.

convulsions in the first 30 days, that had perinatal complications as the etiologic cause.

From the 49 cases with difficulties at birth we could not exclude the possibility of developmental anomalies, such as porencephaly, anomaly of the cerebral vessels etc., which was often found on postmortem examinations of infants who died due to perinatal complications (Schwarzt, 1965). This supported the finding of Keith (1963), that many cases of epilepsy were caused by an anomaly of the cerebral vascular arteries.

Forty five of the 49 cases with perinatal complications had their first convulsions in the first week, whereas in 3 cases who developed convulsions after the 7th day, subdural effusion was detected by skull transillumination and subdural tapping. The fluid showed a high protein content without any signs of infection. It was put forward that there had been subdural hematomas caused by birth injury, which after one week or more became effusion with a high protein contents (Rabe, 1967).

There were only 10 premature infants with convulsions, as cerebral lesions in prematures are more often presented with cyanosis, respiratory distress or hypotania, whereas convulsions were usually found in the mature infant (Thorn, 1969).

Developmental anomalies were found in 3 babies, i.e. hydrocephalus, primary microcephaly and spina bifida with probable Arnold Chiary's syndrome in 1 infant. This baby died on the 12th day of life and unfortunately autopsy could not be performed due to refusal by the parents.

Five babies were found to suffer from C.N.S. infections, of which 4 were diagnosed as neonatal purulent meningitis respectively on the 2nd, 4th, 5th and 15th day of life; one baby was diagnosed as suffering from encephalitis and was admitted in the third week of life.

The temperatures of the first 4 babies during hospitalization varied between 37 and 37.5 degrees Celsius, whereas the highest temperature noted in the baby with encepalitis was 38.2° C. It is known that convulsions are not a common manifestation of neonatal meningitis (Schwarzt, 1965), and elevation of the temperature is seldom seen. Zai and Haggerty (1958) in their investigation of 83 babies with neonatal meningitis found that only 35 had convulsions, whereas the majority of the remaining babies had only nonspecific symptoms such as abnormal respiration, difficult feeding and inactivity.

Out of the 48 cases with complete laboratory examinations, only one fulfilled the criteria of Cornblath for hypoglycemia, i.e. less than 30 mg/100 ml in the first 72 hours; this minimal finding is probably due to the fact that the Hagendorn-Jensen method used locally usually gives

TABLE 9 : *Prognosis.*

AUTHOR	TOTAL	FOLLOW-ED UP CASES	MORTA-LITY	NORMAL	ABNOR-MAL	EPILEPSY
BURKE (1954)	46	45	40%	49%	11%	not mentioned
GRAIG (1960)	375	299	56%	40%	4%	not mentioned
HARRIS & TIZZARD (1960)	41	12	30%	40%	23%	7%
TIBBLES & PRICHARD (1964)	135	126	16%	23%	48%	13%
MC. INER-NY & SCHUBERT (1969)	90	70	26%	40%	23%	11%
AUTHORS (1971)	92	71	59%	23%	18%	13%

10 to 30% higher glucose levels than other methods (Monintja et al, 1969). 1969).

One baby with hypocalcemia had a serum calcium level of 6.4 mg/100 ml and a phosphor level of 8.4 mg/ 100 ml, it developed the first convulsion on the 11th hour of life and died several hours after-wards before any specific treatment could be given.

Routine urine examination for phenylketon did not reveal a positive result.

There were more babies with generalized convulsions (52) as compared with the 35 with focal convulsions (table 5).

The follow up attempt was successful in 71 cases. The overall prognosis is shown on Table 7. The mortali-

ty rate is 41 out of 92 neonates i.e. 59%, which is comparable to Craig's finding, but higher if compared with others (table 9). There were only 17 babies considered normal in the follow up study (23%).

Prematures have the worst prognosis with a mortality of 100%, whereas both of the 2 cases of gastroenteritis with dehydration and acidosis are normal in the follow up. Out of the 31 babies who could be further examined at the Out-Patient Clinic of the subdivision of Pediatric Neurology, 12 showed motoric and mental retardation, 4 of whom with a marked degree of neurologic sequelae with cerebral palsy and tetraspasticity, whereas 9 babies developed epileptic seizures; some of these latter combined with motoric and mental retardation or cerebral palsy.

Behaviour disorders were found in 2 babies; both showed uncontrollable hyperactivity. Visual as well as hearing defect was detected on one baby who was also mentally retarted and tetraparetic.

One baby had strabismus of the eyes but was otherwise normal.

Out of the 21 cases on whom E.E.G. could be performed only 17 could be followed up (Table 8). Out of 12 babies with normal E.E.G. only 5 were considered normal on follow up, 2 had motoric and mental retardation and spastic tetraparesis, 2 developed epilepsy; the remaining cases could not be evaluated due to absence on further follow up.

It was noted that of the neonates with borderline abnormal or abnormal E.E.G. pattern such as an irritative focal lesion, hypsarrythmia and dysfunction with inactivity, none was found normal on follow up.

Three of the 5 cases with focal irritative lesion developed epilepsy.

The low number of cases that were followed up does not allow a statistically reliable evaluation. However, it can be assumed that E.E.G. examination in neonates with convulsions has a prognostic value. This was suggested earlier by Tibbles and Prichard (1965).

They found that about 70% of the babies who had a normal E.E.G. pattern, remained also normal on follow up examinations.

On the contrary, one third of the babies with abnormal E.E.G. died, another one third was retarded and only one third was normal on follow up. Harris and Tizzard (1960) found that more abnormal babies could be detected after one year follow up of neonatal convulsions in babies with a formerly abnormal E.E.G. pattern as compared to babies whose E.E.G. had been normal; more abnormal cases were also found when the E.E.G. abnormality was bilateral.

Table 9 shows the overall prognosis as compared with the findings of other authors. The present mortality rate is comparable to Craig's finding (59%), it is however very high as compared with others; consequently

the number of abnormal cases in the follow up became very low, because the majority of them died earlier.

Conclusion

The incidence of neonatal convulsions in infants born at the Dr. Tjipto Mangunkusumo General Hospital, Jakarta is 0.70%.

The prognosis of neonatal convulsions is poor in terms of a high mortality rate and a high number of abnormal cases or epileptics in future life.

Perinatal complications form the largest group of etiologic factors of neonatal convulsions. Approximately 80% of the cases develop their convulsions in the first 7 days of life.

E.E.G. is of value in predicting the outcome of neonatal convulsions.

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