CONVULSIVE MOVEMENTS OCCURRING IN THE FIRST 10 DAYS OF LIFE*

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It has been estimated that approximately 6% of children under the age of 5 years have convulsions due to one cause or another. Moreover, it is generally recognized that this liability to convulsive incidents is greatest in infancy. There is little mention in the literature of the extent to which these generalizations apply to the early days of life, although it is well known that convulsions in newly-born infants occur in a wide variety of circumstances. I present here the results of a study of convulsions occurring for the first time within 10 days of birth.

The Clinical Material

Three hundred and seventy-five babies are included in the study, of whom 158 died and came to autopsy, and 216 survived the neonatal period. With a few exceptions the infants were born in the Simpson Memorial Hospital, Edinburgh, or the Maternity Hospital, Leeds. The exceptions had been delivered elsewhere and subsequently transferred to one or other of the above-mentioned hospitals. All 374 babies were seen personally and were under continuous clinical supervision for a period of not less than 10 days unless death intervened.

Fatal Cases (158): Pathological Findings

The pathological findings in the 158 fatal cases are analysed in Tables 1 to 5. Significant non-infectious intracranial pathological changes were present in almost two thirds of the babies. Infection, developmental anomalies and haemorrhage (other than intracranial) were present in that order of frequency in the remaining fatal cases. Findings were completely negative at nine autopsies (Table 1).

There was evidence of intracranial infection (Table 2) in 18 and of septicaemia without intracranial involvement in seven babies. Infection was

TABLE 1
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
FATAL CASES: MAJOR AUTOPSY FINDINGS

Infection	29
Developmental anomalies	14
Acquired (non-infectious) intracranial pathology	97
Haemorrhage other than intracranial	9
Nil	9
	158

coliform in nine, streptococcal (haemolytic) in two, pneumococcal in one and staphylococcal in two of the 14 babies with septicaemia and meningitis. Blood obtained during life from the seven infants with septicaemia without meningitis gave cultures of *Esch. coli* in four, *Staphylococcus aureus* in two, and streptococcus in one. The strain of streptococcus was not established. Of the four cases of otitis media three were the result of pneumococcal and one of staphylococcal infection. Pyelonephritis was associated with coliform infection in two babies and with *Staphylococcus aureus* infection in one. The primary focus in babies with staphylococcal infection was in the eye in one, the ears in one and the skin in four cases, the majority of which occurred before the advent of antibiotic therapy. Neither brain nor meninges was the site of pathological changes in the babies with pyelonephritis and congenital syphilis.

With regard to deaths contributed to by developmental anomalies (Table 3) the three examples of

TABLE 2
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
FATAL CASES: DEATHS DUE TO INFECTION

Septicaemia with meningitis	14
Septicaemia	7
Otitis media with secondary meningitis	3
Pyelonephritis	3
Otitis media with secondary brain abscess	1
Congenital syphilis	1
	29

* Based on lectures given at the University of Bergen and to the Medisinsk Selskap, Oslo.

internal hydrocephalus all occurred in small premature infants. The renal and cardiovascular abnormalities were of a major character and incompatible with survival. Infants with evidence of cerebral agenesis are referred to later.

TABLE 3
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
FATAL CASES: DEATHS CONTRIBUTED TO BY
DEVELOPMENTAL ANOMALIES

Cerebral agenesis	6
Internal hydrocephalus	3
Cardiac with associated transposition of great vessels	2
Hydronephrosis (with secondary infection)	2
Cystic kidneys	1
	<hr/> 14

Haemorrhage contributed to death in a variety of ways (Table 4). Massive intrapulmonary haemorrhage was a feature in two cases, in one of which there was microscopical evidence of early secondary staphylococcal infection. Of the two examples of fatal exsanguination one was the result of incision of the placenta in the course of delivery by caesarean section and the other of massive intestinal haemorrhage in a baby with untreated haemorrhagic disease who had been born outside hospital. All the examples of suprarenal haemorrhage were massive and associated with widely distributed tardieu spots. In none of the cases was there a history of trauma or evidence of infection and I cannot say whether the suprarenal haemorrhages were the cause or the result of associated asphyxia. Suprarenal haemorrhage certainly was the immediate cause of death in each case.

TABLE 4
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
FATAL CASES: DEATHS CONTRIBUTED TO BY
HAEMORRHAGE OR CONGESTION

Intrapulmonary haemorrhage	2
Exsanguination	2
Suprarenal haemorrhage	5
Intracranial haemorrhage, venous congestion or oedema	97
	<hr/> 106

Haemorrhage within the cranium was found in a number of different situations (Table 5). In many instances post mortem findings pointed to asphyxia having been secondary to traumatic haemorrhage. In an equal number it was not possible to differentiate the relative aetiological significance of coexistent haemorrhage and asphyxia. This was especially true of a high proportion of infants with subarachnoid and intraventricular haemorrhage. In two infants intracranial haemorrhage was associated with bleeding into the peritoneal, pleural and pericardial sacs which was

regarded as evidence of haemorrhagic disease; and in one baby bleeding arose from septic thrombosis of the choroid plexus in the presence of staphylococcal septicaemia without meningitis. Aseptic thrombosis of the choroid plexus with secondary intraventricular haemorrhage was found in six babies all of whom were markedly premature and died after the tenth day of life. In two of these babies there was, in addition, thrombosis of the renal vessels and in one, dry gangrene of the toes secondary to thrombosis.

Severe generalized venous congestion or extensive oedema was the only finding in about 15% of the fatal cases with acquired non-infectious intracranial pathology.

Pneumonia was present in 13 of the infants coming to autopsy, but was considered to be a terminal complication in each instance.

TABLE 5
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
FATAL CASES: ACQUIRED (NON INFECTIONOUS)
INTRACRANIAL CONDITIONS

Haemorrhage: subdural	30
subarachnoid	14
intraventricular	23
multiple perivascular in brain substance	6
Venous congestion involving meninges and brain substance	9
Generalized oedema	7
Sinus thrombosis	4
Kernikterus	4
	<hr/> 97

Surviving Cases (216): Diagnoses

Surviving cases are best considered according as the clinical diagnosis was proven, being confirmed by appropriate laboratory studies, or was conjectural, being based upon clinical observations.

Proven Diagnoses. Table 6 lists the 38 cases in which a firm diagnosis was made. Infection was present in 15. Pyelographic investigations were not considered desirable or practicable in the cases of urinary infection. The possibility of renal abnormalities in these cases was not therefore excluded, but none of the infants concerned had a recurrence of signs or symptoms by the age of 2 years. Exsanguination resulted in one infant from prolonged, severe intestinal bleeding due to haemorrhagic disease, and in the other infant was thought after examination of the mother's blood to be due to foetal intraplacental haemorrhage. Both infants were born at home and there was delay in diagnosis and treatment. The patients with cerebral agenesis are described later. Convulsions started on the tenth day of life in each of the babies with subdural effusions. The fontanelle

TABLE 6
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
SURVIVING BABIES: PROVEN DIAGNOSES (38)

Diagnosis	Confirmation	No. of Cases
Meningitis: <i>Esch. coli</i>	Bacteriological	3
Pneumococcus	Bacteriological	2
Meningococcus	Bacteriological	1
<i>B. friedlander</i>	Bacteriological	1
<i>Proteus</i>	Bacteriological	1
Pyelonephritis	Cytological:	
	Bacteriological	7
Exsanguination	Clinical, haematological and therapeutic	2
Cerebral agenesis	Air encephalography	11
Subdural effusion	Aspiration:	
	Air encephalography	2
Kernikterus	Serological and biochemical	6
Tetany	Biochemical	2

was a little tense and the resistance on percussion of the two sides of the cranium differed. A tentative diagnosis leading to confirmatory aspiration was not made until some weeks later by which time convulsions were recurring at intervals of several days. In the two infants with tetany, convulsions occurred as the culmination of particularly exhausting periods of severe generalized neuromuscular hyperexcitability and coincided with depression of the serum calcium level below 6 mg./100 ml. Both babies were the offspring of mothers with diabetes mellitus (Craig, 1958).

Presumptive Diagnoses. The presumptive diagnoses in 178 surviving infants are given in Table 7. In over 80% of these babies the diagnosis was intracranial disturbance. This term is used deliberately as it is only rarely possible to differentiate clinical signs of intracranial disturbance during life due to cerebral haemorrhage, congestion and oedema. It might be argued that lumbar puncture would have assisted in differential diagnosis. In my opinion this is open to question and lumbar puncture should be reserved for those cases in which the need arises to exclude meningitis. Potter (1952) draws attention to the limited value of examination of the cerebrospinal fluid as a diagnostic measure in cerebral haemorrhage.

With regard to other cases included in Table 7 the

TABLE 7
CONVULSIONS IN THE FIRST 10 DAYS OF LIFE
SURVIVING BABIES: PRESUMPTIVE DIAGNOSIS (178)

Poliomyelitis	1
?-Aseptic meningoencephalitis	1
Alimentary disturbance (non-infectious)	7
Aspiration of feeds	4
Hypothermia	9
Cold injury	4
Intracranial disturbance	152
	178

diagnosis of poliomyelitis in a baby in whom two isolated convulsions occurred on the tenth day of life was based on a history of contact, a temporary pronounced increase of lymphocytes in the cerebrospinal fluid and a residual flaccid paresis and muscle wasting still present in the left lower limb at the age of 4 years. No convincing explanation of the case labelled '?-aseptic meningoencephalitis' can be offered. There was no clue in the birth history. The child was born at term. Progress was uneventful apart from a succession of violent general convulsions on the eighth day of life and the presence of nuchal rigidity for several days. Cells in the cerebrospinal fluid were greatly increased for a week, lymphocytes predominating, but successive bacteriological cultures were consistently negative. At 3 years of age the child's condition was in every way normal.

Five of the seven cases listed as alimentary disturbance were similar. A violent convulsion in the second week of life in a previously healthy baby occurred simultaneously with the passage of an abnormally bulky stool containing numerous large undigested curds. The association between convulsion and manifestly abnormal stools was inescapable. Convulsion and stool were completely isolated incidents. There was neither forewarning nor aftermath. The other two infants in this group were very small premature babies in whom the initial expulsion of meconium was delayed until the sixth day of life due, it was considered, to lack of muscle tone. There was no question of meconium ileus or mucoviscidosis. Generalized twitching movements appeared on the sixth day and did not recur after the removal of the retained meconium by gentle low colonic lavage. Asphyxial convulsions due to aspiration of feeds were due to a lapse in nursing technique in the case of one infant; to difficulty in feeding another baby with a suspected but at the time undemonstrated high, short tracheo-oesophageal fistula; and to injudiciously repeated feeds given to two infants with undiagnosed oesophageal atresia.

Use of the term hypothermia in Table 7 implies that a rectal temperature below 95° F. persisted for a period of not less than 24 hours. Hypothermia was a feature also of the four babies with cold injury, being associated with the hard infiltration of skin, subcutaneous tissues and muscles found in this condition which is variously, confusingly and frequently incorrectly referred to in the literature as sclerema, scleredema, scleroderma and cold injury (Macgregor, 1960). For the purposes of this paper the term cold injury is employed throughout.

Details Concerning Convulsions

The clinical characteristics of convulsions seen in the course of the present study varied considerably. Examples were seen of a tonic phase followed in succession by clonic movements and unconsciousness; of isolated, prolonged tonic incidents; and of both brief and persistent clonic phases not preceded by tonicity or followed by unconsciousness. In some instances the onset of a convulsion was immediately preceded by or coincided with a short and sometimes piercing cry; in others grunting or crying was noted during or at the termination of a phase of tonicity. A cry was absent in most cases. Violent generalized convulsions were seen with noteworthy rarity in premature babies. Rhythmic clonic movements were common; involved one or several limbs at any one time; frequently affected different limbs at different times; and were often associated with transient unilateral twitching of muscles at the angles of the mouth or eyes. Associated cyanosis, neck rigidity and opisthotonus were occasional findings.

Convulsions Considered in Relation to Diagnosis.

INFECTIONS. Convulsions in babies with septicaemia or meningitis consisted of fitful phases of subdued clonic movements with irregularly interspersed brief events of apnoea. In some infants these incidents occurred spontaneously and in others only when disturbed. Convulsive movements did not appear until after the third day of life even in infants in whom the obstetrical history suggested that infection had been antenatal. The fact that all the infants concerned were premature and frail probably accounted for the convulsions not being severe. In fatal cases there was no relationship between the clinical picture and the location, the extent or the bacteriological nature of the infective process as seen at autopsy. A feature of white blood cell counts during life was that a severe leucopenia was as common as a pronounced leucocytosis. Convulsions occurring in infants with pyuria were generalized but not severe in character, and commenced before the onset of the swinging temperature characteristic of the condition. They ceased as diuresis increased.

DEVELOPMENTAL ANOMALIES. Convulsions seen in babies with major anomalies of the cardiovascular system and of the kidneys were violent terminal events. The infants with internal hydrocephalus were premature and small, and the convulsions consisted of sporadic twitching movements. In marked contrast, babies with cerebral agenesis presented clinical pictures remarkable for their severity and similarity of pattern. The convulsions

first appeared about the second day of life, thereafter occurring spontaneously at frequent but wholly irregular intervals, and persisted despite large and frequent doses of chloral hydrate and phenobarbitone. Structural asymmetry or unusual configuration of the face or cranium was a feature in a significant number of these infants.

HAEMORRHAGE. The convulsions which took place in the presence of external bleeding were isolated but severe and general in distribution. Those that occurred in fatal cases were terminal events. In the two babies who survived exsanguination the convulsions were similar in character and occurred when the infants arrived at hospital *in extremis*, having been transported without previous treatment over distances exceeding 40 miles. Copious haemoptysis preceded the terminal convulsions in babies with massive intrapulmonary haemorrhage. Convulsions in infants with massive suprenal haemorrhage at autopsy began suddenly a few hours before death and assumed a violent form even when the baby was small and premature. They were accompanied by collapse and tachycardia and in two cases by sweating of abnormal degree.

INTRACRANIAL DISTURBANCE. Convulsions in babies developing kernikterus were subdued in character, recurred at intervals of widely varying length over a period of days and showed great variability in the parts of the body involved. Cycling movements of the lower limbs and boxing movements of the arms were among other features in these infants. The convulsions in the infants with subdural effusions began in the middle of the second week, were vigorous and general and associated with occasional vomiting. Personal experience suggests that the cases were exceptional in that vomiting attributable to subdural effusion usually precedes convulsions which more commonly first appear after the second week of life.

Consideration of intracranial disturbances presents special difficulties. Among babies with subdural haemorrhage major generalized convulsions were a feature in about 50%. Regardless of their severity and whether they were generalized or localized, convulsions tended to recur over a period of 24-36 hours. They contrasted with convulsive movements associated with other forms of intracranial haemorrhage in the readiness with which they were induced by external stimuli. Progressively deepening cyanosis during life was usually accounted for by pressure exerted on the mid brain by large amounts of blood which had gravitated downwards. There was no constant association between generalized convulsions and the distribution of blood over the cortex. Localized convulsive

movements were seldom explained by the anatomical distribution of bleeding.

Clinical features in babies with subarachnoid haemorrhage lacked the drama of those in cases of subdural haemorrhage. Localized slight convulsive or twitching movements were present in about half the infants with subarachnoid haemorrhage. Generalized convulsions were rare. There was no significant correlation between the extent of the subarachnoid haemorrhage and the severity or localization of the convulsive movements. An unexplained finding was the frequency with which twitching was preceded by abnormal movements of the eyes. Intraventricular haemorrhage is sometimes found unexpectedly at autopsy without any signs of cerebral irritation during life. When they did occur convulsions were dramatically sudden in onset, of extreme violence and accompanied by uncoordinated movements of the eyes and piercing cries of intense cerebral pain. These signs were present in 10 babies, of whom seven died in the first, and three in the second week. In five babies isolated slight twitchings were noted for a few hours before the development of major convulsions. The clinical pictures in the six babies with widely distributed intracranial perivascular haemorrhages were strikingly similar. In all the appearance of convulsions was delayed until the second week. From the time of their onset the convulsions were general, of extreme violence and completely exhausting. Persistent restlessness was characteristic. Death occurred suddenly after a period of wild uncontrollability similar to that sometimes seen in small babies with fulminating encephalitis. I have known only two babies survive after presenting this clinical picture. Both were eventually severely

handicapped, physically and mentally, and both were ultimately ataxic.

During life the clinical pictures presented by infants with severe intracranial oedema at autopsy were indistinguishable from those due to other forms of intracranial disturbance.

HYPOTHERMIA AND COLD INJURY. Convulsions in babies with hypothermia were general but not violent, and although not frequent, occurred at lengthy irregular intervals until the body temperature was raised. Similar findings were encountered in the two babies with cold injury, but it was evident that as the stiffness or rigidity characteristic of the condition increased, fine frequent convulsive movements became more coarse and deliberate.

The Incidence of Convulsive Movements

In one hospital to which admissions are subject to obstetrical selection, and at which detailed clinical notes have been kept over many years, the records suggest that one infant in approximately every 120-125 live births has convulsive movements at some time during the first 10 days of life. The incidence is certainly considerably less, although exact figures are not available, in another hospital, with which I am associated, which is of about the same size and which admits only multiparous and uncomplicated maternity cases.

The incidence of convulsions in relation to certain clinical conditions seen in newly-born babies was also studied. Table 8 summarizes the findings.

The Severity of Convulsive Movements and Long Term Prognosis

A follow-up study was made of 141 of the 152 infants who survived the neonatal period and who

TABLE 8
INCIDENCE OF CONVULSIONS OCCURRING IN THE FIRST 10 DAYS OF LIFE
IN ASSOCIATION WITH CERTAIN CLINICAL CONDITIONS

Clinico-Pathological Group	Number of Infants						% of Infants with Convulsions
	Dying†		Surviving†		Total		
	Convulsions		Convulsions		Convulsions		
	With	None	With	None	With	None	
Cerebral agenesis	6	2	11*	2	17	4	81
Septicaemia and/or meningitis	24	10	8	2	32	12	73
Intracranial disturbance	97	†	152	488	249	†	Less than 33
Renal anomalies	3	11	—	—	3	11	
Kernikterus	4	6	6	4	10	10	50
Severe urinary infection	3	3	7	58	10	61	14
Cold injury	—	3	4	15	4	18	18
Haemorrhagic disease	1	1	2	149	3	150	2
Hypocalcaemic tetany	—	—	2	63	2	63	3
Suprarenal haemorrhage	5	10	—	—	5	10	—
Internal hydrocephalus	3	6	—	—	3	6	—
Intrapulmonary haemorrhage	2	8	—	—	2	8	—

* Six of these infants died in the second year of life.

† Relates to first four weeks of life.

‡ Reliable figures not available.

in the first few days of life had shown convulsive tendencies attributed at the time to intracranial disturbance. Of the 141 babies nine died before the age of 2 years and the remaining 132 were last examined between the ages of 3 and 3½ years. Findings were analysed according as the postnatal convulsions had been violent and general, or transient and slight; and were compared with those in a control series consisting of 488 babies who had had no convulsions but had shown signs of postnatal anoxia and distress, and who were re-examined at or shortly after the age of 3 years (Table 9). The figures do not purport to be statistically accurate but reflect a higher incidence of sequelae in babies with a history of neonatal convulsions than in babies with no such history.

TABLE 9
NEONATAL ASPHYXIA/INTRACRANIAL DISTURBANCE
WITH AND WITHOUT CONVULSIONS: SURVIVING
BABIES: FINDINGS AT OR OVER
THREE YEARS OF AGE

Findings at 3 Years of Age or Over	Neonatal Convulsions Number of Babies		
	None	Prolonged/ Severe	Transient/ Slight
Normal	473	85	45
Physical handicap ..	6	1	1
*Mental handicap ..	3	1	1
*Physical and mental handicap	6	5	3
Total	488	92†	49‡

* Does not include children considered to be primary aments.

† Includes 9 babies of birth weight of 5½ lb. or less.

‡ Includes 31 babies of birth weight of 5½ lb. or less.

Discussion

The evidence presented indicates that whereas it has been variously stated that 2-4% of children under 3 years (Green and Richmond, 1954) and approximately 6% under 5 years (Davidson, 1953) have convulsions, considerably less than 1% of babies in maternity hospitals have convulsions within 10 days of birth. This is of interest in view of the greater liability to convulsions of infants as compared with older children (Davidson, 1953) and of the findings of Crump (1945) which show that the incidence of convulsions is lowest in the first six months of life and highest between the ages of 6 months and 3 years.

Asphyxia, cerebral birth injury, meningitis, systemic infections, kernikterus, congenital malformations and metabolic disturbances such as tetany are among causes of neonatal convulsions given in the majority of reviews. In a study of 176 babies aged less than 4 weeks, Peterman (1946) considered that convulsions were accounted for by birth injury in 69%, acute infection in 10%, develop-

mental anomalies in 4% and tetany in 3%. The present series includes examples of all these conditions together with instances of convulsions associated with exsanguination, intrapulmonary haemorrhage and cold injury. Clifford (1939), Ford (1952), Stone (1945) and McQuarrie and Baird (1959) consider that haemorrhagic disease contributes to a proportion of cases of intracranial haemorrhage. Findings in this study confirm the views of other workers that this is not a frequent occurrence (Craig, 1938; Potter, 1945; Sanford, Shmigelsky and Chapin, 1942). Kagan (1959) makes specific mention of gastro-enteritis. This is contrary to my experience. In a detailed review of 53 cases of acute enteritis in the newly-born in both epidemic and sporadic form no instance of convulsions was encountered.

Recent literature does not refer to alimentary disturbances other than gastro-enteritis as a cause of convulsions in infancy. Thomson (1921), Still (1927) and Hutchison (1931), however, all mention the occurrence of convulsions as a result of the retention of undigested food in the bowel or stomach. They ascribe the convulsions to reflex irritation. Today this explanation is considered inadequate. Nevertheless, in the absence of a convincing alternative and in the absence of evidence of metabolic disturbance the explanation merits consideration in connexion with the few babies in the present series in whom isolated convulsions coincided with the evacuation of undigested stools.

Febrile convulsions are described by Green and Richmond (1954) and McQuarrie and Baird (1959) as especially common in the first two years of life. Davidson (1953) stressing their occurrence in infants considers that of convulsions in all children 2.5% are regarded as 'so-called febrile convulsions'. According to Friderichsen and Melchior (1954) febrile convulsions are rarely seen under the age of 6 months. The youngest patient in 256 children with simple febrile convulsions reported by Livingston (1958) was aged 2 months. Discussing febrile convulsions in general, McQuarrie and Baird (1959) refer to the onset of extracranial infections and to a high environmental temperature as possible contributory factors. An outstanding feature of the series under discussion is the absence of any baby with convulsions which could reasonably be termed febrile in the strict sense of the term. In the few babies with severe urinary infection in whom convulsions occurred the temperatures were subject to fluctuation and were elevated on occasions to 102°-103° F. Pyrexia of the same degree was a feature of the considerably more numerous cases of pyuria lacking any convulsive tendency. Among

infants with meningitis and septicaemia a normal or slightly subnormal temperature was as frequent a finding as an elevated temperature. Pyrexia when present was usually slight. There was no obvious connexion between body temperature and the occurrence of convulsions in these cases. Experience gained in the course of the present study warrants the view that febrile convulsions if they do take place in the first 10 days of life do so with the utmost rarity. There is support for this opinion in the fact that dehydration fever is not accompanied by convulsions. Moreover, over a period of many years I have seen and made records of five newly-born babies with extreme hyperpyrexia attributable to faulty nursing technique or defective incubator control. The rectal temperature ranged from 106° to 108° F. and in each case hyperpyrexia was present for not less than six hours. All the babies recovered without sequelae. None had convulsions. Unfortunately records were not made concerning the rapidity with which the temperatures rose, an aspect of febrile convulsions considered to be important by Wegman (1939).

Severe, extensive cerebral oedema was the sole finding at autopsy in five cases. It was also found in less severe degree in association with other forms of intracranial pathology in three babies born prematurely and in two born at term. The clinical simulation of intracranial haemorrhage by babies with cerebral oedema is noted by Hughes (1952), and McQuarrie and Baird (1959) include acute cerebral oedema among the causes of convulsions in infancy. Judging by the incidence of cerebral oedema among fatal cases in the present series it is possible, indeed probable, that cerebral oedema may account for a considerable number of convulsions in surviving newly-born babies. Certainly cerebral oedema provides one possible explanation for the convulsions occurring in two of the surviving examples of cold injury.

Hypothermia is characteristic of cold injury and was a feature of the two cases in this series. It was a feature also of several of the premature babies dying of infection, and of several infants dying as a result of intracranial haemorrhage. Furthermore, among large babies surviving neonatal cerebral disturbance, convulsions occurred in a number within 36 hours of birth while the body temperature was still below 96° F. There were several examples of twitching movements occurring simultaneously with a sudden drop in body temperature on the third and fourth days of life in particularly small premature babies who eventually survived. These observations do not warrant any conclusions but suggest that a closer relationship may exist between

convulsions in the first few days of life and hypothermia than between convulsions and hyperpyrexia.

Bound, Butler and Spector (1956) in their study of perinatal mortality refer to the occurrence of convulsions in 20, and of cerebral irritation without convulsions in six premature babies dying as a result of the cardio-respiratory syndrome. Their experience contrasts with that at the Leeds Maternity Hospital where over a number of years continuous detailed clinical observations have been made on all infants in whom there was a possibility that signs of the cardio-respiratory syndrome might develop.

During the period in question 38 babies dying with clinical and pathological evidence of the syndrome were observed from the time of delivery in the labour ward until death, with as one particular object the recognition of any convulsive tendencies. All the babies were premature and delivered by caesarean section. Entirely contrary to expectations no convulsions were seen in any of the infants. Insofar as a limited number of cases permits of tentative deductions it seems that the anoxia occurring in fatal cases of the cardio-respiratory syndrome does not of itself give rise to convulsions. A further point is that these cases are not associated with the generalized tonicity and apnoea sometimes seen in very small premature babies. It is of interest in this connexion that McKay and Smith (1959) do not include convulsions among the clinical manifestations of hyaline disease.

'The causes of convulsions (in infants) are legion.' This statement of Still's (1927) can be applied with justification to the newly born. It is important, however, that the occurrence of convulsions in the early days of life should be seen in proper perspective. Although the causes are legion the analysis in Table 8 indicates that no one clinico-pathological condition is invariably associated with convulsions. The highest incidence was found in babies with cerebral agenesis. Of the 21 babies, no fewer than 17 had major convulsions. The clinical pattern in these was unlike that associated with other forms of intracranial pathology, and it is considered that it has a diagnostic significance. Particular attention should be paid in this connexion to major convulsions commencing within a day or two of life, persisting at irregular intervals despite heavy anticonvulsant therapy, and occurring in an infant with asymmetrical anomalies of the face or cranium.

There was a high incidence of convulsions among babies with septicaemia and meningitis. Convulsions cannot, however, be regarded as fundamental to diagnosis. They were absent in a number of babies with widely dispersed septicaemic foci and

thick inflammatory exudate covering the entire surface of the brain. Clinical features requiring the exclusion of infection as a cause of convulsions include the presence simultaneously of an unexplained low body temperature, grey cyanosis and reluctance to feed. A leucocytosis or leucopenia is in favour of infection whereas a normal white cell count is not.

This study makes it abundantly clear how rarely a precise diagnosis is possible during life. Convulsions in the presence of unconsciousness and of deepening cyanosis not responding to oxygen therapy suggest pressure on the mid-brain or brain stem by haemorrhage arising from a dural tear. An uncontrollable encephalopathic picture punctuated by convulsive seizures following difficult delivery of a large or post-mature infant is suggestive of intracerebral and/or intracerebellar multiple perivascular haemorrhages. The dramatically sudden appearance in a premature baby of violent, generalized convulsions, a bulging fontanelle and an agonizingly piercing cry is in keeping with intraventricular haemorrhage. Otherwise there is no clinical picture (apart from that already described in connexion with cerebral agenesis) characteristic of intracranial pathology of a particular kind or with a particular anatomical location.

Terminal convulsions irrespective of their pathological explanation and of the maturity of the infant tend to be violent and general. In other circumstances convulsions encountered in small premature babies are generally of a subdued character. Mention has been made of the occasional simultaneous occurrence in these babies of transient cyanotic apnoea and generalized tonicity. Incidents of this kind can occur as completely isolated events or as events interspersed between phases of subdued twitching or myoclonic activity. As Green and Richmond (1954) point out, young infants may demonstrate only the tonic phase of a major convulsion. There were occasions when even momentary apnoeic attacks might in reality have been minimal convulsive incidents, but they were not regarded as such for the purposes of this study. Nevertheless, the extent to which the frailty of prematurity tends to subdue the severe manifestations of convulsive tendencies was very evident. For this reason it is considered that assessment of the potential risks of convulsions in the early days of life must have regard to the maturity and physical robustness or otherwise of the baby.

Observations made in the present study do not agree with the conclusion of Keith, Norval and Hunt (1953) that 'difficulties during delivery have very little to do with persistent neurological abnor-

malities' in surviving infants, and do not support the view advanced by Hellström and Jonsson (1953) that asphyxial attacks appearing 'later on and after the child has breathed normally' have a less favourable prognosis. The findings in the follow-up series (Table 9) do suggest, however, that the general risk of morbid sequelae is as great in the case of premature babies with slight transient convulsions as in babies born at term having prolonged major convulsions.

Summary

The clinical findings in 374 newly-born babies who had convulsions commencing within 10 days of birth are presented.

The pathological findings in 158 fatal cases are considered in relation to clinical observations made during life.

Surviving cases are considered according as the diagnoses were proven or presumptive.

Attention is drawn to the characteristics of convulsions arising in cases of cerebral agenesis.

Emphasis is attached to the absence of 'febrile convulsions' and of convulsions in association with uncomplicated cardio-respiratory syndrome.

The occasional occurrence of convulsions in the presence of cerebral oedema and hypothermia is noted.

The influence of prematurity and the prognostic significance of transient minimal convulsions in premature babies are discussed.

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