Observations of Intracranial Hæmorrhage in 400 Consecutive Newborn Babies.*

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The condition of intracranial hæmorrhage in the newborn has only infrequently been diagnosed in the acute stage within ten days after birth, unless the hæmorrhage and the associated cerebral œdema were of such a large amount that either death occurred or the baby exhibited signs of a severe cerebral lesion. Until recent years the literature of intracranial hæmorrhage in the newborn has been concerned with these extreme cases, either as post-mortem studies of extensive intracranial hæmorrhage due to laceration of the large sinuses, or falx, or as clinical observations of the marked signs of a probable intracranial lesion of hæmorrhage—stupor to the degree of unconsciousness, refusal to suck or even repeated general convulsive seizures—and the description of methods to increase the coagulability of the blood and in this manner lessen the cerebral damage and its resulting chronic condition of cerebral spastic paralysis with mental retardation.

In the period of 1826 to 1835, Denis,[1] Billard,[2] and Cruveilhier[3] published several papers intimating and suggesting the causal relationship of intracranial hæmorrhage at birth and the later development of cerebral spastic paralysis. In 1843, Little, of London, stated in his first monograph upon the subject of "Deformities of the Human Frame,"[4] that difficult labour might be an etiological factor, but that lack of development of cerebral tissues and meningitic inflammatory processes were the usual cause; only nineteen years later (1862), in his second monograph upon this subject,[5] Little stated that as a result of further study and especially of a large series of post-mortem examinations, he was of the opinion that almost 75 per cent. of the cases of cerebral spastic paralysis were due to an intracranial hæmorrhage at the time of birth. In 1885, Sarah McNutt[6] confirmed this opinion by careful pathological studies. More recently, Beneke[7] in 1910, Pott[8] in 1911, Mayer[9] in 1915, Holland[10] in 1920, and within the past four years Schafer,[11] Schwartz,[12] Saenger,[13] and Capon[14] and others have contributed valuable post-mortem observations and yet the literature of the

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clinical observations has been most meagre. In this country Green,[15] Brady,[16] Sidbury,[17] Warwick,[18] Rodda[19] and Huenekens[20] have all added to the knowledge of the subject during the past decade, and the present paper is a similar effort to facilitate the diagnosis and thus make possible the *early* rational treatment of the condition.

In 1913, one of the writers became interested in the chronic condition of cerebral spastic paralysis in its various forms with and without marked mental impairment. It was very impressive at that time[21] and during the past eleven years to note repeatedly in the histories of a large percentage of these patients—those having an increased intracranial pressure as disclosed by the ophthalmoscope and more accurately by the spinal mercurial manometer—that they were chiefly first-born fulltime males. The majority of these children had been delivered after a prolonged labour with or without the use of instruments. During the first week, and even two weeks after birth, they had been considered rather drowsy and stuporous with a lack of the normal demand for food, and even refusal to suckle, and in many of them there was a history of twitchings of the orbital muscles and of the hands and legs or of even generalized stiffenings to the degree of convulsive seizures. After a period varying from several days to two or three weeks, the baby rapidly improved and was considered a normal child at the end of one month after birth and continued to be so apparently, until several months later. Usually about the seventh or eighth month, it was observed that the child was not holding up its head nor attempting to sit up, and frequently at this time a spasticity appeared in the arm and leg of one or of both sides; later, when the child did not walk or talk, if at all, until months after the usual time, this chronic condition developing in an apparently normal child, was most puzzling. Frequently the diagnosis of Little's disease implied a tainted heredity and lues as the etiological factors in the lack of development. (In our series of these chronic cases, the blood serum and spinal fluid Wassermann tests have been positive in only half of one per cent. (1 in 200 cases). That the condition might be the result of an intracranial hæmorrhage at the time of birth was very improbable, as it was believed and taught that intracranial hæmorrhage was of such an extensive character and of such great danger to the life of the newborn that the baby usually did not survive. The earlier study, moreover, of the acute condition of intracranial hæmorrhage in the newborn was limited chiefly to post-mortem examinations and therefore to the study of the extreme condition sufficient to cause death at the time of birth or within several days after birth.

During the past eleven years we have had the opportunity to examine, both at the operation and at autopsy, the brains of a large series of the chronic cases of cerebral spastic paralysis having a definite increase of the intracranial pressure. The pathology has been practically the same—"wet" œdematous brains under varying degrees of increased pressure, and along the supra-cortical veins in the sulci has been demonstrated the cloudy whitish new-tissue formation with a fibrous thickening of the walls of these veins—the organization residue of a former layer of supra-cortical hæmorrhage which had occurred most probably at the time of birth and of larger amount than could be entirely absorbed by the natural means of excretion. Since it has now been demonstrated that over 80 per cent. of the cerebrospinal fluid is normally excreted through the walls of the supra-cortical veins, the partial blockage of this main channel of excretion and cerebro-spinal fluid, by the organization-residue of a former hæmorrhage in these patients, explains the "wet" œdematous condition of the brain under varying degrees of increased pressure and the resulting spastic paralysis in its various forms associated with a mental retardation of varying degree. The treatment of these chronic conditions in the older patients can, at best, be directed only toward an improvement—lowering the increased intracranial pressure, mental training, the use of orthopædic measures (braces, tendon lengthenings, muscle transplantations) and the more recent peripheral nerve operations of Stoeffel, including the sectioning of the rami (ramisectomy) of the sympathetic ganglionic chain as advocated by Royle and Hunter.

During this same period of eleven years, we have had the opportunity of examining in consultation and treating 59 newborn babies within the first ten days after birth. The signs of an acute severe intracranial lesion were very evident—stupor and coma, refusal to suckle and muscular twitchings to the degree of convulsive seizures. Lumbar puncture in these early cases revealed bloody cerebrospinal fluid under varying degrees of increased pressure. Repeated lumbar punctures or spinal drainage in the milder cases or the modified subtemporal decompression or cranial drainage in the more severe cases made possible a recovery of life in a small percentage of them. One of the patients in this series (in 1920), on the third day after a difficult instrumental birth, appeared to be a normal baby with the exception of spasmodic twitchings of the left orbital muscles two or three times each hour. Careful neurologic examination was negative, but a lumbar puncture was advised merely to confirm these negative findings, so that the parents could be assured that no intracranial complication had occurred. Two hours later and *before* the lumbar puncture was performed, the baby

suddenly died. At the autopsy there was disclosed a supra-cortical hæmorrhage of over one inch in thickness upon both cerebral hemispheres—the longitudinal sinus having been torn at the posterior margin of the anterior fontanelle. This case, and the mildness and even apparent lack of the definite signs of severe intracranial hæmorrhage in the other acute cases, as disclosed both at operation and at autopsy, led to the belief that possibly an intracranial hæmorrhage of mild degree at the time of birth did not produce marked clinical signs of its presence owing to the less highly developed cortical cells at this early age. Possibly, therefore, the complication of an intracranial hæmorrhage at the time of birth was of more common occurrence than was ordinarily suspected.

First Series.

To determine the frequency of this complication in the newborn, permission was obtained in January, 1923, from Drs. F. A. Dorman and Wilbur Ward to perform a routine lumbar puncture upon a series of consecutive newborn babies, at the City Hospital, Welfare Island. In the first series[22] of 100 consecutive newborn babies, the cerebrospinal fluid was bloody in four and blood-tinged in five, that is, a total of nine (9 per cent.) and under varying degrees of increased pressure (the normal pressure in the newborn after one week being approximately 3 to 5 mm. as registered by the spinal mercurial manometer). As many as four lumbar punctures of spinal drainage were necessary in only one case to obtain clear cerebrospinal fluid and under normal pressure. Two of these babies died and the autopsy revealed a large ventricular hæmorrhage in one and a mild subarachnoid hæmorrhage with extensive cerebral œdema in the other. The post-mortem findings of free blood along the supracortical veins in the sulci confirm the observations made at autopsy and at operation upon the other acute cases in the newborn. These support the belief that if this free blood cannot be entirely absorbed, then an organization-residue occurs *in situ* with the resulting partial blockage of the excretion of the cerebrospinal fluid—the usual pathology in the cases of cerebral spastic paralysis having an increased intracranial pressure and due to an intracranial hæmorrhage at the time of birth. Repeated lumbar punctures of spinal drainage at intervals of 12 hours were sufficient to make the cerebrospinal fluid clear and under normal pressure in the remaining seven cases. No instruments had been used in these nine cases; one was a version with breech extraction; in two, the cord was round the neck. The low forceps had been applied in ten cases of this series, but no intracranial hæmorrhage had occurred in these particular instrumental deliveries. The cord-Wasserman was negative in every case. Two were reported as "jaundiced."

One baby in whom convulsive twitchings developed ten hours after birth had clear cerebrospinal fluid and the muscular twitchings ceased within six hours after the lumbar puncture—being the result possibly of the cortical irritability of a mild cerebral œdema.

Second Series.

In the second series[23] of 100 consecutive newborn upon whom a lumbar puncture was performed within 12-24 hours after birth, bloody cerebrospinal fluid of varying degree was found in 13—that is in 13 per cent.; nine were bloody, three blood-tinged, and one was yellow, containing many red blood cells. There was no mortality in this series and the intracranial pressure in the bloody cases ranged from 4-26 mm.; five punctures of spinal drainage were the largest number necessary to obtain clear cerebrospinal fluid under normal pressure. The anterior fontanelle did not appear to be an accurate index to the intracranial pressure. Two of the babies exhibited muscular twitchings of the face, hands and feet and also cyanosis of the face, which disappeared within six hours after the lumbar puncture. Marked signs, such as stupor, refusal to suckle or convulsions were not present in any of the cases. The labour in these 13 bloody cases was normal cephalic in six, prolonged in three, two of which terminated as cephalic presentations and one as a breech; medium-forceps was used in two; one was a face presentation and one the child of an eclamptic mother. Jaundice was present in one case only. The coagulation time of the blood was normal in all 13 bloody cases; one case clinically diagnosed as hæmorrhagic disease of the newborn did not have an intracranial hæmorrhage and the 13 cases having bloody cerebrospinal fluid did not have the disease. Ten babies were of luetic mothers and in only two of them was the cerebrospinal fluid bloody.

Third Series.

In the third series[24] of 100 consecutive newborn babies upon whom a lumbar puncture was performed within 12—24 hours after birth, bloody cerebrospinal fluid of varying degree was found in 10, that is 10 per cent.; the fluid was red with blood in four and straw-coloured and yellow with many red blood cells in six. Six was the highest number of lumbar punctures necessary to obtain clear cerebrospinal fluid under normal pressure. There were no deaths in this series. Seven babies born of luetic mothers all had clear cerebrospinal fluid. The forceps was applied in nine cases in this series; of the two medium-applications, in one the cerebrospinal fluid was blood-tinged and in the other bloody, and in the seven low applications only one baby had free blood in the cerebrospinal fluid. The one case of version with breech extraction had clear cerebrospinal fluid. The intracranial pressure ranged from

3-15 mm. and again the anterior fontanelle was not a reliable indicator of the intracranial pressure. The blood coagulation time was not lengthened in any of the four bloody cerebrospinal fluids, and in only one of the yellow fluids was it slightly prolonged to 10 minutes (normal clotting time being considered as five to eight minutes). However, in four cases having clear cerebrospinal fluid, the blood-clotting time was 8½—11 minutes, so that it seems that the hæmorrhagic tendency in the newborn is only of minor importance as an etiological factor in the production of the intracranial hæmorrhage. Four babies, in whom the cerebrospinal fluid was clear within 27 hours after birth became jaundiced on the second, third, sixth and seventh day respectively; in two of these babies the blood coagulation time was four minutes, and in the other two, 10 and 11 minutes, respectively.

Fourth Series.

In the fourth series of 100 newborn babies upon whom a lumbar puncture was performed within 24—48 hours after birth, the cerebrospinal fluid was bloody in four and straw-coloured in three—a total of seven (7 per cent.). One baby having bloody cerebrospinal fluid died and the autopsy revealed extensive intraventricular, basal and supracortical hæmorrhages. Jaundice was present in four babies, in three of whom the spinal fluid was clear and in one a golden yellow, slightly turbid with numerous red blood cells. In the latter case the jaundice appeared on the seventh day, and it is interesting that the first lumbar puncture, performed fourteen and a half hours after birth, was clear, under 6 mm. of pressure and the blood-clotting time was seven minutes; whereas when jaundiced, the cerebrospinal fluid was yellow with red blood cells, under 8 mm. of pressure and the blood-clotting time was four minutes.

As in the preceding three series of the newborn, neither sex predominated and approximately 50 per cent. were first-born babies. The signs observed in this series of newborn were the following: in five muscular twitches of the extremities and face and convulsive seizures, in two of whom the cerebrospinal fluid was bloody, but in the other three clear. Poor suckling in one, and in this particular baby the fluid was yellow with many red blood cells. Cyanosis was present in one baby having clear fluid. Normal intraspinal pressure (5-8 mm. of mercury within 48 hours after birth) was present in 45 cases, one of them having yellow fluid; above normal in 32, six of them having bloody or yellow fluid, and below normal in eight, and in these the fluid was clear. Seven premature babies were not punctured. Venous contamination of puncture occurred in two cases, and in five babies the spinal canal could not be tapped, due presumably either to faulty technique or to anatomical variation. In one case the pressure was not recorded.

The blood-clotting time was normal (3-8 minutes) in 81, seven of the babies having hæmorrhagic cerebrospinal fluid; prolonged in two (8½ and 10 minutes respectively); below normal in one and not recorded in 16 (these included the babies not punctured or successfully tapped and the premature babies). The anterior fontanelle was flush in 38, and of these four had hæmorrhagic fluid; depressed in 39, of whom three had hæmorrhagic fluid; bulging in 14, and in them all the fluid was clear. The status of the fontanelle was not recorded in nine.

In five mothers the Wassermann test was 4 plus, and in four babies the Wassermann was 4 plus in three and 2 plus in one; only one luetic child had bloody fluid. Seven of the nine premature babies were not punctured on account of their low vitality; three of these babies died and the autopsy did not reveal an intracranial hæmorrhage.

Type of labour. The position was left occipito-anterior in 49, of whom four had hæmorrhagic fluid; in 23 it was right occipito-anterior, of whom one had yellow fluid, and right occipito-posterior in one with clear fluid. The low forceps was used in 10, of whom one had bloody fluid. Prolonged labour in two, one having blood-tinged cerebrospinal fluid. One dry labour, one Cæsarean section, one frank breech and one induced labour had clear fluids.

Impressions. Acute intracranial hæmorrhage and cerebral œdema of varying degree occur more frequently in the newborn than formally suspected. Unless the hæmorrhage is of large amount, the usual signs of intracranial hæmorrhage in the newborn are meagre (drowsiness, difficulty in nursing and slight muscular twitchings) or even apparently absent. At present, early lumbar puncture is the only accurate means of diagnosis and especially in the mild and so-called signless cases. The therapeutic lumbar punctures of spinal drainage should be repeated as frequently as the severity of the intracranial hæmorrhage warrants—every 12-24 hours, and even more frequently. Babies of low vitality, and especially if premature, or babies in a state of shock, should not be punctured, even in the presence of signs of an intracranial hæmorrhage, until the general condition improves. This is true of brain injuries in adults as in the newborn. The treatment should be directed to the general condition of shock rather than to the local intracranial condition. If the patient is unable to survive the shock, no examination or tests will aid, but rather increase the shock. Muscular twitches of fingers, extremities, face and orbits are very suggestive as signs of increased cerebral irritability and due to supra-cortical hæmorrhage or cerebral œdema. The use of early and correctly applied forceps, especially the application of the low forceps, does not increase the liability to intracranial hæmorr-

hage. In prolonged labour causing extreme intracranial venous stasis, especially when instruments are used as a last resort, and particularly delayed medium forceps, the danger of the occurrence of intracranial hæmorrhage is greatly increased. Hæmorrhagic disease of the newborn does not appear to be an important etiological factor. Yellow spinal fluid in the newborn is most probably the result of transudation of the blood-plasma when red blood corpuscles are absent (the hæmoglobin not being demonstrated by the guaiacum test) and due to minute hæmorrhage when red blood corpuscles are present. Early lumbar puncture as a diagnostic and therapeutic aid is not advocated as a routine procedure, but only in doubtful and suspected cases of intracranial hæmorrhage. Early recognition of intracranial hæmorrhage at the ideal time for treatment should permit the greatest possible recovery, both of life and of future normality. By this procedure that large group of physically and mentally impaired children having the chronic condition of cerebral spastic paralysis with varying degrees of mental retardation, may be lessened.

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FIG. 1. HÆMORRHAGIC CASES.

No	Name	Para	Labour or Position	Anterior Fontanelle	Character of Spinal Fluids	Press. In Hg.	Blood Clot. Time	No. L.Ps. to Clear Fluid	Miscellaneous
1	M. S. (White)	i	L.O.A.	Flush	Yellow (clear)	6 mm.	3½ m.	3	—
2	E. P. (Coloured)	ii	L.O.A.	,,	Bloody	10 mm.	6 m.	—	Died at 14hrs. of age. P.M. Hæmorrhage of Base, Cortices & Ventricles.
3	M. Z. (Coloured)	i	Prolonged	,,	Blood-tinged	10 mm.	5 m.	4	Convulsion 4th day. Recovery.
4	W. F. (White)	iii	Low Forceps	,,	Bloody	10 mm.	7 m.	3	4 + Wass.
5	E. P. (Coloured)	i	L.O.A.	Depressed	Bloody	9 mm.	5½ m.	3	—
6	R. F. (White)	iii	L.O.A.	,,	Yellow (clear)	16 mm.	7½ m.	2	Cord around neck.
7	E. H. (Coloured)	ii	R.O.A.	,,	Yellow c R.B.C.	10 mm.	3½ m.	2	Suckled poorly.

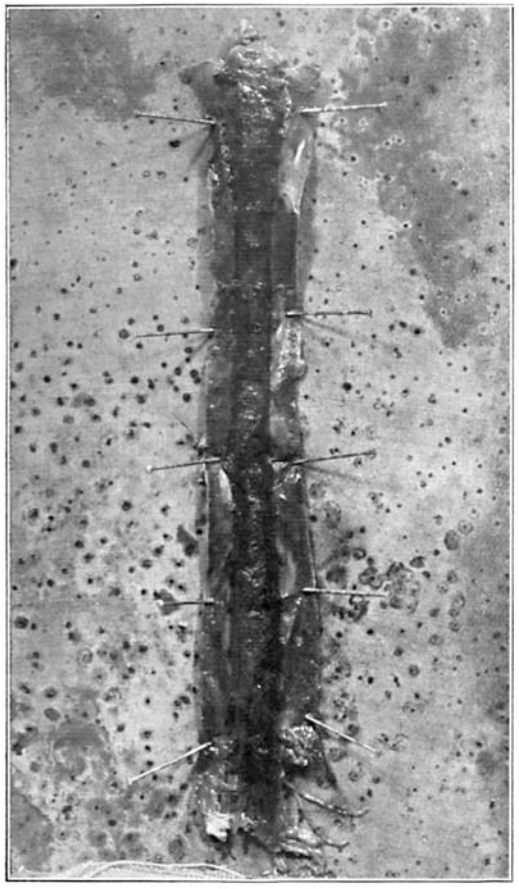
FIG. 2. CLEAR FLUID CASES & SIGNS.

No.	Name	Para	Labour or Position	Anterior Fontanelle	Character of Spinal Fluids	Press.	Clot. Time	Signs.
1	S. K. ♀	i	R.O.A.	Flush	Clear	8 mm. Hg.	7 m.	Cyanosis.
2	G. S. (Coloured)	ii	R.O.A.	,,	,,	6 mm.	4 m.	Twitches of arms & legs.
3	S I. (Coloured)	i	Prolonged	Bulging	,,	12 mm.	3 m.	,,
4	R.A. (White)	i	Dry	,,	,,	12 mm.	3½ m.	Convulsions.

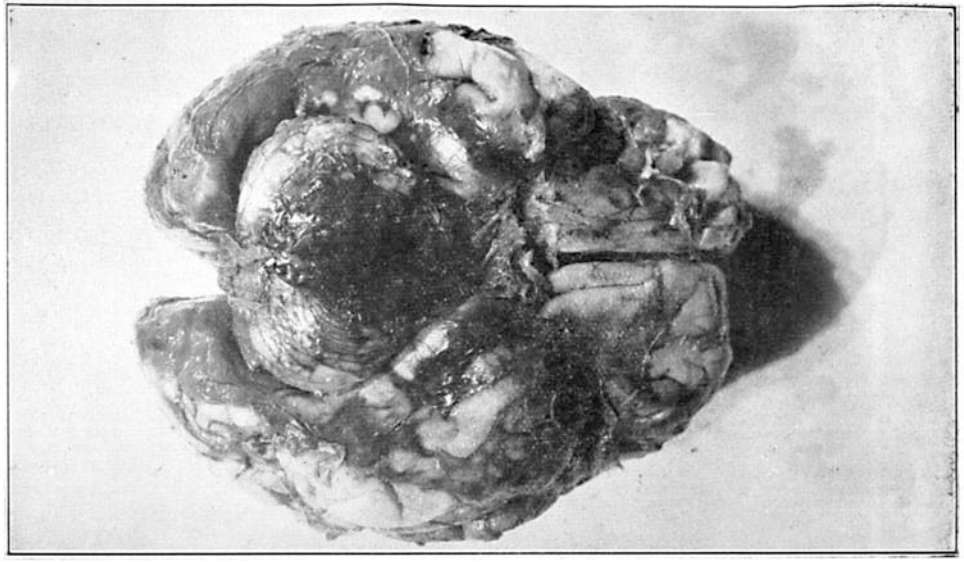
FIG. 3. TYPES OF LABOUR OR POSITIONS.

L.O.A. ...	... 49	... Of these 4 abnormal fluids	{ 2 yellow (clear) fluids 2 bloody fluids.				
R.O.A. ...	... 23	... „	„	1	„	fluid	(yellow c̄ R.B.C.)
R.O.P. ...	... 1	...					
Low Forceps	... 10	... „	„	1	„	„	(bloody).
Dry Labour	... 1	...					
Prolonged	... 2	... „	„	1	„	„	(blood-tinged).
Cæsarean	... 1	...					
Breech ...	... 1	...					
Premature labours.	9	...					
Not Mentioned ...	1	...					
Normal ...	... 1	...					
Induced ...	... 1	...					

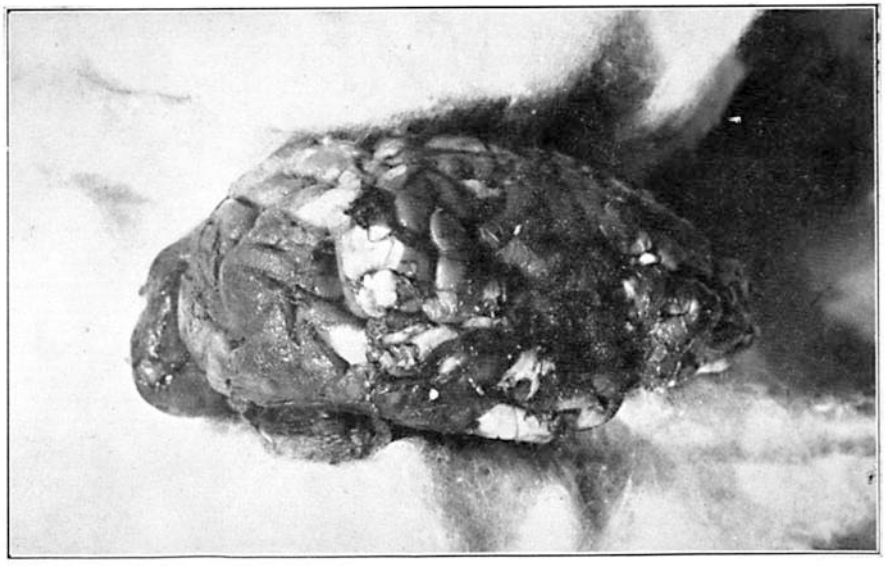
c̄ R.B.C. = Red blood corpuscles.



Case No. 2.—E. P.
Showing hæmorrhage within the spinal canal.



Case No. 2.—E. P.
Showing hæmorrhage at the base, both above and below the tentorium.



Case No. 2.—E. P. Showing sunra-cortical hæmorrhages