Intracranial hemorrhage in the new born

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INTRACRANIAL HEMORRHAGE
IN THE NEWBORN

SENIOR THESIS

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JOHN O. HEALD
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INTRODUCTION.

Cranial and intracranial damage in the newborn, commonly referred to as intracranial hemorrhage in the newborn, has been subjected to sporadic investigation for many years. Literature cited here covers approximately one hundred years, and shows what advance has been made in the various phases of intracranial hemorrhage of the newborn.

To make the subject-matter of this paper more comprehensible and so as not to be lead into a maze of more or less closely related topics, it has seemed logical to formulate a definition. The close adherence then to the development of this definition into a rational conception is the object of the following dissertation. Let it here be said, however, that some migration from the strict meaning of the title "Intracranial Hemorrhage in the Newborn" will be necessary, but bearing in mind that the migration will be restricted only to that which is necessary.

This term has been used to designate the history, the incidence, the etiology and pathology, the symptoms and diagnosis, the prognosis, and the treatment which have to do with the occurrence during or after birth, of
hemorrhage on the surface of the brain into the brain tissue, or into the ventricles.

The subdivision of certain of these main divisions is made to meet a chronological need. The reasons for these subdivisions are given preceding their discussion occurring in the following pages.

An attempt has been made to cover the literature on this subject comprehensively if not extensively, with an added attempt to show that traumatic intracranial hemorrhage in the newborn, hemorrhagic diathesis, and asphyxia neonatorum are very closely related, and that intracranial hemorrhage is the ultimate finding in many cases.

J.O.H.
HISTORY.

The subject of hemorrhage in the newborn is one to which a good deal of attention has been drawn recently, but is by no means a new one. Scattered through medical literature of the past century, and more, one finds references to it.

In considering this topic historically it seems striking that it was not the obstetrician but the neurologist who first manifested interest in the clinical aspect of the problem, Béchet (1852) and Little (1862).

Cruveilhier (1835-1842) offered excellent illustrations of intracephalic hemorrhage of the newborn along with some indications that this type of hemorrhage was being found and discussed.

Kennedy (1836) discussed some of the causes of the predominance of apoplexy in the newborn infant. It was his opinion that suddenly altered circulation after birth was of etiologic importance. The structure of the brain also occurred to him as being soft and vascular and with the dependent position to which it is liable at birth, pressure might cause an intracranial hemorrhage.
An excellent case and autopsy report are offered by Bechet (1852). In this report Bechet gives some of the clinical findings now recognized as very diagnostic of intracranial hemorrhage, as, for instance, bulging of the fontanels, dilation of the pupils, vomiting and convulsions, the child dying in extreme marasmus. At autopsy, after making an incision over the anterior fontanel, blood was found outside the longitudinal sinus in the subarachnoid space. An incision was made into the left cerebral hemisphere where more blood was found. Incisions made in other parts of the brain substance also showed hemorrhage.

In a second paper published by Little (1862), he asserts that the cerebral spastic palsy in children is the result of an intracranial birth hemorrhage in about three-fourths of all instances.

Dr. Abbot (1872) described a case of difficult labor. After extreme traction with forceps applied on a head high in the pelvis, he delivered a child with a large indentation "in the superior portion of the left frontal bone, extending to within a quarter of an inch of the coronal and sagittal sutures, two inches in length
and half an inch deep'. The child was dead.

It is interesting to note that up to this time very little attempt was made to determine the cause of death by a pathological investigation such as would be obtained with an autopsy.

At a meeting of the Clinical Society of the New York Post Graduate Medical School and Hospital, McNutt (1885) opened a discussion on the subject of Intracranial Hemorrhage in Children. In this discussion several case histories were given and the subject taken up more from the neurologist's point of view, with emphasis laid on the later sequellae of birth hemorrhage. Autopsies were quite common at this time. Dr. McNutt had collected details of thirty-four autopsies himself and had come to the conclusion that intracranial hemorrhages of children were usually meningeal.

It is most interesting to observe what advances had been made, not only in the technique of performing autopsies, but in the actual knowledge regarding intracranial birth injuries.
This is well typified in the case reports by Partridge (1886). He reports two cases, giving very good brief histories and quite remarkable autopsy findings. He concludes by stating that "cerebral hemorrhage might occur in the new-born independently of any operative interference".

It is a fact, historically interesting, that only the new method of opening the infant’s skull at autopsy, described by Beneke ('10), has permitted the formation of a definite and clear conception of the exact pathology and actual frequency of intracranial injuries.

The neurologist was the first to manifest any interest in the problem of intracranial birth injury and he has maintained it ever since.

If we were to set down in chronological order the outstanding men who have been interested in intracranial hemorrhage in the newborn, we would find that following the neurologist the pediatrician has to be mentioned next as concerning himself with the question.
The last to become interested in this problem was the obstetrician. Ehrenfest ('22) believed that the obstetrician's researches have led to a much better and clearer understanding of the etiology and pathology of intracranial birth lesions.
INCIDENCE.

It is the opinion of many authors that Beneke's ('10) method of examining the skull at autopsy has been revolutionary regarding the incidence of intracranial lesions, especially of tentorial lacerations. Due to this fact it is believed that older statistics are valueless.

When Pott ('11) became interested in this problem he discovered 14 tentorial lacerations in 101 autopsies. With a more careful technique and with a more minute study of the findings he found evidences of tentorial injuries in 6 out of the last 15 autopsies.

Benthin ('12), in a series of 1239 consecutive labors, including premature, found that 107 infants were stillborn, or died soon after birth. Of these 73 came to autopsy showing 8 tentorial tears evidently responsible for death, (11 percent of all autopsies).

In an exhaustive study, dealing with the etiology of hemorrhagic pachymeningitis, based on 6,000 autopsies of young children, Kowitz ('14) states that fatal intracranial hemorrhages, both after spontaneous and artificial deliveries, arranged in order
of their relative frequency, involve most often the dura, and least frequently the cerebral tissue itself, with hemorrhages of the arachnoid and into the ventricles ranging between them.

Of 36 autopsies done on stillborn infants and those dying in early infancy Warwick (1919) found that 50 percent showed indisputable evidence of hemorrhage, either on the surface of the brain or in the ventricles.

Bailey (1920), performing 100 autopsies in the Manhattan Maternity Hospital in New York, found intracranial hemorrhages in 40 cases, 30 of these being stillborn infants, the others having died within four days after birth. In 17 cases of these latter, the deliveries were spontaneous and normal.

It is Browne's (1922) contention that the liability for premature infants to be stricken with cerebral hemorrhage is greatest at from 7 to 7 1/2 months. At 8 months it is less and when they reach 8 1/2 months there does not seem to be any greater liability than when they are at full term.
Grulee and Bonar ('26) were of the opinion that in view of the fact that a conception of the frequency of this condition was almost wholly obtained from autopsy statistics, there were two chances of error. They thought this for two reasons; (1) because a majority of these cases live, and (2) because intracranial hemorrhage certainly accounts for a very large percentage of deaths as compared with the survivals.

Holland and Claypon ('26), in their review of 1673 newborn deaths, draw certain conclusions. To quote Holland and Claypon ('26), the following statements are made:

1. "Of all presentations with spontaneous delivery, that of breech shows the highest percentage of tentorial tears.

2. "In all the cases in which forceps were applied about half of the cases had tentorial tears. Delivery by podalic version appears to be the next largest cause of tentorial tears.

3. "The highest incidence of tentorial tears occurs among those cases in which the primary cause of death was some complication of labor."
A sharp concise statement regarding the incidence of intracranial hemorrhage in the newborn is made by Greene (’28) when he says: "Intracranial hemorrhage is the most frequent cause of death in the newly born and was found in 55 of 177 consecutive autopsies". This is roughly 35 percent.

Fleming and Morton (’30), in a review of 103 cases, state that the immediate mortality was 48 percent; that in premature infants only, the mortality was 75 percent.

In these 103 cases frequency was being based upon mortality. We know that these startling figures would probably be made more startling if the autopsy findings had been recorded. Fleming and Morton do, however, go on to say that tentorial tears were the most common post-mortem finding.

Irving (’30) has listed a number of conditions with the percentage of relative frequency of intracranial hemorrhage in each case. These are as follows in order of greatest percentage:

1. Mid forceps 3.0%
2. High forceps and breech extraction 2.6%
3. Version 1.7%
4. Low forceps 0.5%  
5. Normal delivery 0.4%  
6. Caesarian Section 0.3%

Grulee ('31) seems to believe that Peterman's ('30) statement is entirely unjustified and does not think that most convulsions in the newborn period of life can be attributed to hemorrhage. It is Grulee's opinion that the nervous disorders attributed to intracranial hemorrhage may be rather on the basis of a congenital brain defect.

Kutscher ('31) says that intracranial hemorrhage accounts for practically 50 percent of the natal and neonatal deaths. Farther along in his article it is brought out that premature infants suffer more from intracranial hemorrhage than any other group.

In a study and a review of 2256 deliveries made by Tyson and Crawford ('31) the general incidence of hemorrhage was 1.9 percent. Of the stillborn babies that came to autopsy 32.1 percent had intracranial hemorrhage.

Ehrenfest ('32) believes it is only a relatively small percentage of cases in which cerebral traumatization proves promptly fatal on account of its severity.
We may add Taylor ('32) to the long list of those who have shown that intracranial injury is the most frequent cause of fetal death during labor and the first week of life after delivery. He shows that the frequency of intracranial hemorrhage in European and American clinics is very nearly alike. Taylor's compilation shows that out of 3011 European autopsies 1002 cases showed brain hemorrhage; out of 617 American autopsies 226 cases showed brain hemorrhage. From this it is found that about 30 percent of all deaths at birth or during the first week of life are caused by intracranial injury.
ETIOLOGY AND PATHOLOGY.

In the consideration of this phase of the subject "Intracranial Hemorrhage in the Newborn" the striking condition which confronts one is the diversity and confusion afforded by the literature.

To illustrate this point and to give the reader a clearer conception of the various ideas, beliefs and probably theories, statements made by various authors relative to the etiology and pathology will be referred to here.

Of necessity the subjects of etiology and pathology are to be considered together because of their close relationship. The pathology incident to the fetus and to the first two stages of labor has, in a large measure, a direct etiological bearing upon the occurrence of processes which may result in the death or permanent disability of the infant.

As was referred to before, there are many different ideas as regards the etiology of intracranial hemorrhage in the newborn. Some authors believe there is only one etiological factor, while others mention as many as eight, as does Ehrenfest (122). It has also been
found that any combination of causes may be dealt with by these various authors.

After several attempts were made to work all these etiological factors into chronological order it was decided that an outline including all the different causative factors would have to be made. It was for this reason then that the following outline was developed. By adhering to this arrangement it is felt there will be nothing omitted and a much better chronology will be effected. Each division will be discussed separately. The outline is as follows:

1. Prematurity or immaturity.
2. Traumatic or mechanical.
3. Hemorrhagic diathesis.

Prematurity or Immaturity.

The first topic to be discussed is prematurity or, as some authors consider, immaturity in its relation to the causation of intracranial hemorrhage in the newborn.

As early as the work of Kennedy (1836) the fact is mentioned that the fetus has a very soft and vascular brain, which in combination with another cause, to
be dealt with later, has a tendency to produce intracranial hemorrhage.

Whether or not Kennedy (1836) had reference to a premature condition of the brain we do not know. It is extremely interesting, however, that the structure of the brain should be even thought about at this early a date as regarding a possible etiological factor in intracephalic hemorrhage.

Spencer (1891) states that intracranial hemorrhages occur in almost any condition relating to the fetus, the mother or the delivery, for several reasons. The chief reason with which we will be concerned in this part of our discussion will be the one which Spencer lists first.

To quote Spencer, "If we look at the list of organs in which hemorrhage occurs we shall find this most frequent and most severe in those viscera which contain a large quantity of delicate vessels. It is also observed that with equal difficulty of delivery the hemorrhage is apt to be more severe the younger the fetus is, that is to say, the more delicate is the
structure of the vessels and surrounding tissue".

It is important to notice that here is, beyond a doubt, the first record we have that prematurity or immaturity was recognized even vaguely as an important etiological factor in the production of intracranial hemorrhage. It should also be noticed that there is a period of approximately thirty years following Spencer's (1891) work during which time there were no important contributions to this phase of the etiology of intracranial hemorrhage in the newborn as far as we were able to discern.

Strachauer ('20) states that cerebral hemorrhage occurs as frequently in premature births as after stormy labors. No direct mention is made that prematurity is a causative factor, but it is felt that it would not be amiss to place his statement in this part of the discussion.

It is Sidbury's ('20) contention that premature babies have very fragile blood vessels. Due to this fragility he feels that these vessels are not strong enough to undergo the amount of pressure necessary even in a normal delivery, hence their predisposition to hemorrhage.
The structure of the cerebral vessels in the newborn and in the premature has been worked out to a much greater degree by several other authors, namely, Ehrenfest ('22), Magner ('23 and '24), Kutscher ('30), Kugelmass ('32) and Taylor ('32). The work of these men will be dealt with in more detail later, when the proper chronology permits.

Ehrenfest ('22) says that prematurity will represent a definite etiological factor which will predispose the intracranial tissues to more extensive damage. He mentions further that the percentage of premature infants is high in all the various types of intracranial parturitional lesions. Instead of ascribing this fact to the fragility of the blood vessel walls in the brain, Ehrenfest believes that this fact that he has mentioned is due also to fragility of the dura and of the brain itself, resulting from incomplete development.

Holland ('23) says "The soft plastic head of a premature fetus is much more liable to excessive head-molding and injuries of the dural septa than the firm head of the mature fetus."
After a review of a series of cases by Magner ('23 and '24) he decides that intraventricular hemorrhage does not occur in the full time child. He concurs with Sidbury ('20) and others that the bleeding is due to an excessive fragility in the majority of cases. Magner also believes that hemorrhagic diathesis and defective formation of the capillary walls are somehow interlocked in the formation of intracranial hemorrhage in the newborn.

Grulee and Bonar ('26) after a careful investigation, decide that intracranial hemorrhage is much more frequent in premature infants.

Schwartz and Fink ('27) find that the great majority of microscopic bleedings occur in premature babies.

The work referred to previously on the structure of the blood vessel walls is quite adequately dealt with by Capper ('28). He believes that the lack of elastic tissue in the blood vessel walls is responsible for the hemorrhage by rhexis. Capper considered vaso-liability as one of the most important predisposing causes of intracranial hemorrhage.
Unlike the firm skull bones of the normal full-term child, Ehrenfest ('29) feels that those of the premature infant offer but little protection. In the premature infant both the dura and the vessel walls are notoriously fragile. As Ehrenfest states, it is well known that they often are severely lacerated merely by a forced or quick passage of the soft head through a not fully dilated cervix. He feels then that these two conditions, the soft skull bones of the premature and the fragility of the dura and vessel walls, act as significant contributory elements in the development of serious lesions.

Kutscher ('30) refers to the capillaries of the brain as "mere endothelial tubes". He states that they contain no elastic or muscle fibers in the premature brain, and by these findings places prematurity or immaturity as an etiological factor in the production of intracranial hemorrhage in the newborn.

As can readily be seen from the foregoing references prematurity is a definite etiological factor, and, as is further shown by these same foregoing references,
it derives its importance from the nature of the capillaries in the brain. These same findings are mentioned by Kugelmass ('32) and Taylor ('32).

Kugelmass ('32) believes that due to the structure of these capillaries they yield more readily to pressure and produce intracranial hemorrhage more easily than the capillaries found in the full term child.

Taylor ('32) states, "The more immature or premature the fetus the more common the injury. This is due to the lack of development of the elastic fibers in the walls of the vessels, especially of the veins".

**Traumatic or Mechanical.**

Traumatic and mechanical etiological factors do not appear to be so widely separated as some authors would have us believe. If they are separated and discussed separately considerable confusion results and there is a great deal of overlapping from one to the other. Consequently these two phases of the etiology of intracranial hemorrhage in the newborn
will be considered together, and further, they will be considered almost as synonymous terms. In addition, all of the isolated factors which have been encountered in our readings that pertain to trauma or the various mechanisms related to labor will be included under this heading. There will be in addition to trauma proper, one fairly large subdivision dealing with asphyxiation neonatorum.

In considering trauma proper we automatically become interested in a variety of conditions. These conditions, such as trauma and its various causes, lacerations of the sinuses and blood vessels and location of hemorrhage, or in other words the general pathology of intracranial hemorrhage in the newborn, will be discussed in chronological order. When there are a number of authors writing on one subject, each will be discussed as his work falls into the chronological structure of this paper so as to show the progress of science relative to this subject.
The subject of this section of this paper can be much more logically dealt with if it is subdivided into three separate but related parts, as follows:

1. Trauma and its causes.

Under this heading the work of Spencer (1891) mentioned the fact that mechanical squeezing of blood into some parts of the body during the act of birth, plus external violence led to an increased blood pressure producing intracranial hemorrhage.

It was not until the work of Ehrenfest (1'22) that we have any available references made to the causes of trauma. It is his belief that severe pressure produced by the use of forceps or a forcible traction of the head along protruding portions of the rigid pelvic canal is adequately sufficient to cause trauma resulting in intracranial hemorrhage.

Grulee and Boner (1'26) agree with Ehrenfest that instrumental deliveries are responsible, in a large portion of cases, for trauma and the resultant intracranial hemorrhage. They further believe that the
overriding of the parietal bones or molding of these bones over the occipital and frontal, and also of one bone over the other is beyond a doubt responsible for severe trauma, if not actual hemorrhage.

Injury to the child's head during delivery, especially if force is applied suddenly, Greene ('28) believes, is the most important cause of trauma, if not intracranial hemorrhage.

The importance of trauma as an etiological factor, according to Burpee ('28), is established by the fact that the death rate from intracranial hemorrhage is higher in the first born, and in the male infant over the average size. He also believes that trauma is due to too rapid compression of the head, whether due to passage through a too narrow birth canal, to the improper use of forceps, to the use of too powerful doses of solutions of pituitary or to breech deliveries.

Caesarian section is mentioned by Burpee, as well as Ehrenfest ('22), as a cause of trauma. He believes that the long unsuccessful test of labor is no doubt responsible.

Ehrenfest ('29) states, "Since there is such a
large number of clinical proofs of natal traumatization encountered in a very large number of newborn infants, and especially first born babies, and since, with relatively few exceptions, they disappear promptly and permanently, it is my belief that we are justified in speaking, in analogy with a similar traumatic effect of labor on cervix or perineum, of a physiologic intracranial birth trauma".

This is, as far as we are able to ascertain, the first reference to a physiologic intracranial birth trauma, and is the only reference.

Partridge ('30) believes that pressure upon the head, due to the lack of full flexion, results in trauma frequently, but intracranial hemorrhage more frequently.

Barbour ('30) states that the greatest factor in the production of intracranial hemorrhage is trauma.

Kutscher ('30) believes that puerperal eclampsia produces trauma in two ways. First, he believes, that eclamptic mothers are prone to deliver themselves
rapidly and forcibly. Second, during the convulsions of the mother the tetanic contractions of the abdominal muscle and the uterine contractions subject the child to terrific pressure.

In a large series of complete autopsies performed by Ehrehfest (1932) which were subsequent to breech labors, and particularly after versions and extractions, the immediate cause of death was found to be of traumatic origin in 80 to well over 90 percent.

2. Lacerations of the sinuses and blood vessels.

It is to Beneke (1910) that we owe much of our present knowledge regarding intracranial hemorrhage in the newborn. Up until the time that he developed his particular technique of opening infant's skulls at autopsies, no absolutely accurate statistics could be assembled as regards the location and thus the frequency of tears of the sinuses and blood vessels.

Rodda ('20,a) finds that the pacchionian bodies, which afford support to the veins extending from the subarachnoid space to the superior longitudinal sinus, are absent in the newborn so that destruction of the
continuity of these vessels is very easy.

In those infants where the dura is abnormally fragile and the overlapping excessive or very suddenly accomplished, as Ehrenfest (122) believes, the dura breaks and the longitudinal sinus itself tears open. He further believes that the overlapping of the parietals over the squama of the occipital bone is prone to cause rupture of the veins emptying into the transverse sinus, or laceration of the sinus itself. It is thought by this same author that the destruction of vessels by a syphilitic process will produce rupture of these same veins, or the sinus.

The histology of the falx and the tentorium have first been described in their relationship to intracranial hemorrhage by Ehrenfest (122). He finds that the longitudinal direction of the fibers in the falx, and their lateral extension from either side into the tentorium, indicate that it is the chief mechanical task of the falx to prevent an abnormal extension of the long diameters of the cranium. By a lateral compression
the compensating elongation of the cranium thus might exert on the falx a strain severe enough to cause it to tear.

The tentorium, according to Ehrenfest, is occasionally torn simply as a result of the forced passage of the fetal head through an incompletely dilated cervix, or a rigid vulvar ring. Ehrenfest, and others, feel that all violent methods of resuscitation should be done away with. Some of them tend to produce lateral compression of the head with its dangerous results, while others favor the escape of blood "even from very small ruptured vessels".

Holland ('23) speaks of "cranial stress". By this term he has reference to the state of the head during labor. He gives a very comprehensive discussion of the mechanics taking place within the cranial vault during labor. This discussion is too long to be more than mentioned here. From his description of the anatomy of the falx and tentorium it is readily seen how, in the common form of head molding, the vein of Galen becomes altered in direction, kinked and stretched. This results in the rupture of this vein.
and its tributaries. Holland also shows how the fibers of the falx and tentorium are arranged so as to meet the strain to which the head is subject during labor. He states that tears of the tentorium usually occupy this free border near its junction with the falx. He further considers that a stress reaching its maximum suddenly is less liable to be counteracted by the septa than if this stress had reached its maximum gradually.

Sacks ('26) states that "If the head is suddenly born the springing apart of these overlapping bones leads to rupture of some of the veins entering the longitudinal sinus".

Holland and Claypon ('26) mention breech deliveries as causing the highest percentage of tentorial tears. Forceps then podalic version rank next in causing tentorial tears. They believe that the highest incidence of tentorial tears is present after some complication of labor.

With a rupture of the falx or tentorium, Crothers ('26) believes that the tributaries of the vein of Galen are involved. He also mentions tearing of all layers of the meninges.
Due to the fact that the vessels on the surface of the brain, in these infants, are comparatively free, Grulee and Bonar ('27) believe, as did Ehrenfest ('22) and others, that pressure either laterally or anteroposteriorly on the head produces tension on the tentorium, and if severe enough, this structure is torn. They further believe that overriding of the cranial bones is likely to result in rupture of the blood vessels leading from the sinuses. Whether or not malpresentations play a primary or secondary part in the production of lacerations has not been fully decided by Grulee and Bonar.

It was found after work on a series of 117 cases by Munro ('28) that some degree of laceration of the falx and tentorium was present in nearly all cases. According to Munro the source of hemorrhage was, in the large majority, either from the great vein of Galen or from one or both lateral sinuses.

A method discussed by Roberts ('29) and modified by Allen and McClure ('31), dealing with the detection of tears in the various blood carrying structures of
the brain without performing an autopsy should be very beneficial in adding to our knowledge in the event that autopsy is refused. The exact technique of this method will not be discussed here. Suffice it to say, however, that when a substance known as röntgum is injected into the jugular veins, and the skull exposed to Röentgen rays the presence and location of tears will be disclosed.

Chase (‘30) carefully discusses tentorial splitting in a recent article on subdural hemorrhage.

Fleming and Morton (‘30), after a review of 103 cases, find that tentorial tears were the most common post-mortem finding.

McClintic (‘31) lists the various vessels that he finds lacerated as follows:

1. Vessels of the cerebellar tentorium.

2. The great vein of Galen rupturing at its junction with the straight sinus.

3. The cerebral veins may give way at their entrance into the superior longitudinal sinus.
4. The superior longitudinal, transverse and straight sinus may give way.
5. Rupture of the choroidal veins may take place in the ventricles.
6. Laceration of intracortical vessels may occur.

Sharp ('31) agrees with McClintic and others that contusion and laceration of brain tissue will not only sever sinuses but will lacerate small vessels in the substance of the brain giving rise to microscopic hemorrhages.

The sudden release of the molded head, Clein ('32) believes, will allow a spreading of the bony plates of the skull with a tearing or stretching of the smaller meningeal vessels and those leading to the sinuses. He further remarks that this may also explain the large number of tentorial tears.

Young ('32) states that cerebral hemorrhage in the newborn is venous. He states that "It results from the laceration of the dural folds or sinuses, or from the laceration or rupture of the cerebral veins."
3. Location of Hemorrhages.

It is Pott's (11) contention that hemorrhage coming from tears through the free edge of the tentorium escapes downward and upward. He believes that most of it spreads above the tentorium forward into the temporal fossa. It may, however, rise laterally over the surface of the occipital bone. Pott also mentions another type of laceration resulting in the formation of a small hematoma between the blades of the tentorium or at the end of the falx.

Ehrenfest (12) divides intracranial lesions into two classes, those characterized by a large hemorrhage and those in which but little blood is extravasated.

1. Cephalhematoma internum. This type of hemorrhage is located on the inner side of one of the cranial bones and is limited by the attachment of the periosteum and dura to the skull bones.

2. Subarachnoid hemorrhage. These hemorrhages are situated on the convex surface of the brain near the midline.

3. Dural hematomas.
4. Brain hemorrhages. With hemorrhage into the lateral ventricles Ehrenfest believes that the blood flows into all the ventricles and finally into the spinal canal.

Holland ('23) found that blood was nearly always confined to the area in which is located the vein of Galen and its tributaries.

Three sites of hemorrhage are listed by Schwartz and Fink ('25) as places drained by the great vein of Galen. These locations are (1) the frontoparietal region; (2) the parietotemporal region, and (3) the posterior horn region.

Munro ('25) speaks of an intraventricular hemorrhage which damaged the neighboring cortex. This amounted, in one case he states, to actual perforation.

According to Crothers ('26) there are three important locations for intracranial hemorrhage:

1. Gross meningeal hemorrhage. He states that no direct damage will result from pure subdural or subarachnoid hemorrhage unless pressure and consequent
anoxemia of cells occur. Also that only those meningeal hemorrhages which damage the pia arachnoid will result in adhesions.

2. Intraventricular hemorrhages.

3. Hemorrhages into the substance of the brain. Crothers says that the "ultimate effect of actual intracerebral hemorrhage cannot be predicted at the onset".

Hemorrhage below the tentorium is spoken of by Crothers. He states that if this hemorrhage is at all large the medulla is at once embarrassed.

Grulee and Bonar ('26) state that the striking characteristics of intracranial hemorrhage are due to the fact that bleeding takes place where it affects tissue, which, when destroyed, does not recuperate.

In the order of their frequency the locations of hemorrhages are listed by Grulee and Bonar as follows:

1. Supratentorial. These are most frequently found over the parietal region.

2. Infratentorial. Hemorrhages here spread over the surface of the cerebellum and the basal ganglia.
It is possible for hemorrhage to occur between the upper and lower surfaces of the tentorium, Grulee and Bonar believe, without producing any clinical signs.

3. Intraventricular.

4. Intracortical. When hemorrhage is not sufficient to cause death, destruction of brain tissue takes place. If the hemorrhage is on the surface of the brain the nervous tissue below the site of hemorrhage shows definite retraction. Grulee and Bonar find that the gyri become small and worm-like and the blood vessels in the region become large and dilated. If the condition of hemorrhage occurs in the ventricles or in the substance of the brain itself cavities are formed.

Munro ('28), studying 117 cases, representing 7 years work, finds that almost half of these hemorrhages were subarachnoid in location. He also mentions massive meningeal hemorrhages and multiple intracranial hemorrhages. The microscopic hemorrhage is most commonly subarachnoid or intracortical.
Hemorrhage within the layers of the tentorium is spoken of by Kutscher ('30).

McClintic ('31) gives a very good description of the locations hemorrhages may select:

1. Collections of blood upon the surface of the cerebellum, or drainage may take place around the occipital lobes.

2. Hemorrhage may be located about the dorsal surface of the mid-brain and flow downwards around and beneath the cerebellum, pons and medulla.

3. Hemorrhage into the subdural space on one or both sides.

4. Intraventricular hemorrhage.

5. Intracortical hemorrhage rarely.

The basal areas, according to Tyson and Crawford ('31), show a large proportion of hemorrhage.

Clein ('32) divides cerebral hemorrhage into three main groups:

1. "Cortical or supra tentorial. This may be localized or massive and is usually subdural.

2. "Subcortical or infratentorial. This occurs
usually about the cerebellum and brain stem and is subarachnoid.

3. "Meningeal irritation."

Young ('32) lists the locations of hemorrhage in order of frequency. We find this order to be subdural, subarachnoid, intraventricular and intracerebral.

The location of microscopic hemorrhage within the brain tissue had not been definitely worked out until we find Hemsath and Canavan ('32) publishing their study of 53 infants. They state that microscopic hemorrhage in the medulla occurred in 34 cases of the 53, or 64 percent of this series. They realize that a study of this number of cases is not sufficient to base any definite conclusions on but they do feel that the finding of these hemorrhages reduces the number of cases classified as "cause of death undetermined".

No attempt is being made to cover all phases of asphyxia neonatorum, nor of hemorrhagic diathesis, the part immediately following this. The only thing that interests us is its relation to the etiology of intracranial hemorrhage in the newborn. This and nothing
more shall be discussed here.

It is Little's (1861) belief that asphyxiation is due more to an interruption of the proper placental relation of the fetus to the mother than from direct mechanical injury to the brain and spinal cord.

Spencer (1891) mentions that asphyxia is one of the causes of increased blood pressure which in turn produces intracranial hemorrhages.

Nothing definite seems to have been written up to this time as regards the relation of asphyxia to intracranial hemorrhage; its relation is merely mentioned.

Strachauer (1920) states that spontaneous rupture of the delicate vessels of the cranium may occur in the cerebral congestion of asphyxia neonatorum.

Asphyxiation is classed by Ehrenfest (1922) as both a predisposing and a contributory factor due to the fact that an engorged sinus or vein is not only "more prone to rupture, but also to extravasate more blood". He goes on to state that in many infants dying of cerebral hemorrhage the various serosa show
small petechial hemorrhage characteristic of death from suffocation.

Ehrenfest does not want to leave the impression that asphyxiation can explain all intracranial lesions, but he does believe that it has its place as an etiological factor.

Holland (’23), after a study made upon 92 cases of intracranial hemorrhage, makes the statement that 17 cases were due to asphyxiation. His idea as regards asphyxiation is as follows:

"Stimulation of the respiratory center by the accumulation of carbon dioxide in the blood leads to respiratory movements, and the resultant alteration in the intrathoracic pressure causes venous obstruction and a great rise of venous pressure throughout the body."

Cruickshank (’23) believes there are three types of asphyxia, closely related. These are prenatal, intranatal and neonatal. He further believes that asphyxia and trauma are the only two etiologic factors in intracranial hemorrhage in the newborn.
The statement is made by Grulee and Bonar (1926) that congestion with stasis and rupture of the veins may occur as a result of a breech presentation or a prolonged labor with transverse presentation. They believe that trauma to a vessel engorged with blood would be more apt to produce rupture than if it were not engorged, thus placing asphyxia either as a primary or secondary etiological factor.

An entirely new idea is brought forward by Schwartz and Fink (1925) in the production of asphyxia. They mention the marked difference in pressure between the contents of the uterus and the outside world during expulsion of the baby. This, according to these co-authors, produces a point of application of a damaging force, mostly influencing the venous system, and results in hemorrhage. They believe, however, that asphyxia is of secondary importance in the production of intracranial hemorrhage.

A statement made by Ehrenfest (1929) tends to corroborate the idea of Schwartz and Fink (1925). Ehrenfest states that pressure and suction represent the primary mechanical forces producing hemorrhage.
A severe congestion of the intracranial vessels with increased pressure result from the cervix constricting about the neck or head in breech deliveries is thought by Kutscher ('30) to cause intracranial hemorrhage.

There is no attempt made, Irving ('30) states, to have us believe that all asphyxiated babies are victims of intracranial hemorrhage. He does think, however, that the subject of asphyxiation in its relation to intracranial hemorrhage in the newborn has never received the attention that it deserves. It is his contention, based upon delivery and autopsy reports, that the two conditions are very closely related.

Taylor ('32) deals with the idea of Schwartz and Fink ('25) and Ehrenfest ('23) referred to previously regarding the mechanical cause of intracranial hemorrhage in the newborn. He calls this the "negative pressure", or the "difference between the atmospheric pressure on the presenting part, and the internal pressure of the uterus on the fetal head." This causes a "slowing of the circulation and an increased
congestion in the vessels of the presenting part", resulting in hemorrhage.

Eastman ('32) makes several noteworthy statements in his excellent article upon the chemical nature of asphyxia neonatorum. His first outstanding, but not original, statement is the assumption that the sensitivity of the fetal respiratory center, in respect to carbon dioxide at least, is definitely depressed while the fetus is in utero. His next pertinent statement is that "the primary blood chemical change in asphyxia neonatorum is a reduction in the oxygen content of the fetal blood to extremely low levels."

An editorial in the Lancet ('32) gives about the same opinion as Eastman.

It is readily seen, then, how congestion of the cranial vessels will bring about the condition of asphyxia neonatorum.

**Hemorrhagic Diathesis.**

Only so far as this subject concerns the etiology of intracranial hemorrhage in the newborn will it be discussed.
It is quite an old subject and in its early reference no particular correlation was developed between hemorrhagic diathesis and intracranial hemorrhage in the newborn.

Spencer (1891) lists alterations in the blood among those factors he believes cause intracranial hemorrhage.

Morse ('12) is the first writer, we have record of, who did work on the blood of a newborn showing bleeding. He states that "In two cases of hemorrhagic disease of the newborn, the platelet count was within normal limits when bleeding began but a rapid rise in the count followed the cessation of bleeding under treatment."

"The so-called 'hemorrhagic disease of the newborn'," Warwick ('19) states, "is a much neglected but very important cause of cerebral hemorrhage in infants." It occurred in 44 percent of the deaths in her series of cases.

A combination of the normal trauma incident to childbirth and a phase of the hemorrhagic disease of the newborn is believed by Strachauer ('20) to cause
majority of cases in intracranial hemorrhage.

Rodda ('20,a) did some very remarkable work on the coagulation time of the blood in newborn infants.

Rodda could conceive of massive hemorrhage resulting from rupture of the large veins, sinuses or tears in the tentorium, but he could not understand what caused the large group of cases of cerebral hemorrhage which develop insidiously and not until several days after birth. It was this type of intracranial hemorrhage that at necropsy reveals a "large clot with a thick center, thin edges, usually unilateral and covering the parietal area". According to Rodda there is no demonstrable source of hemorrhage in these cases, no torn veins or rents in the sinuses.

It was his belief then that other factors besides instrumentation at delivery with trauma were concerned in intracranial hemorrhage. It was this then that lead Rodda to studies on the question of disturbances of the coagulation properties of the blood in the newborn.
Rodda ('20,b), performing determinations on 126 newborn infants within the first 24 hours of life, reached an average coagulation time of seven minutes. An approximate range was worked out which was from five and one-half to eight and one-half minutes. He states that the return to the level of the first day is reached during the first twenty-four hours next before the tenth day.

Rodda ('20,a) has found that there is a prolongation of both the coagulation and bleeding times, or, in other words, a variation in these times. Considering the average determinations on the first day as normal, there is a steady increase in time on the second, third and fourth days with a maximum on the fifth day, and a steady fall from there on with the arbitrary normal reached by the tenth day. He goes on to remark that these facts are significant in that they show a relation between the coagulation times of the newborn infant and the age incidence of hemorrhagic disease and certain cases of cerebral hemorrhage.

In an article by Jarcho ('21) he states "These calcium estimations go to show very clearly that in
the blood of the newborn calcium is rarely at fault in any problem related to hemorrhage of the newborn, except in those cases of obstructive jaundice in which case the calcium is bound with the bile salts and is, therefore, invaluable for entering into the process of coagulation."

Lucas and Fleischner ('21) find a definite tendency to prolongation of coagulation time in newborn infants. The average coagulation time was 15 minutes, and no coagulation time below 5 minutes was found on the first day of life. They find that the average bleeding time was from one and a half to three minutes. During the first days of life the studies of the authors showed a definite diminution in the prothrombin element of the blood, the platelets, however, are not diminished. They conclude "that the delayed coagulation time is due to a qualitative defect in the platelets which are deficient in prothrombin."

Magner ('23 and '24) believes that the so-called "hemorrhagic diathesis" embraces both a defective formation of the capillary walls and a deficient clotting power of the blood, and are probably the underlying lesions in intracranial hemorrhage.
The conclusion was reached by McLean and Caffey ('25) that, while the platelet count in infants and children on the whole approximates the normal count in adults, "in the newborn and in premature infants it is fixed within narrower limits and has lower averages".

Grulee and Bonar ('26) contend that there is a great difference of opinion as to whether or not the majority of the cases of this condition are on the basis of hemorrhagic disease or are of traumatic origin. These authors also mention a distinct increase in the coagulation time of the blood and feel that it is responsible for the delayed type of intracranial bleeding.

In contradistinction to the author's already quoted, and others to be quoted later, we find Capper ('28) believing that the term hemorrhagic diathesis is erroneous. He believes that the trouble lies not in the blood itself, but in the unripeness or vasolability of the cerebral vessels referred to previously.

He does find a somewhat longer coagulation
time in most newborn infants, but feels that this is due to the bile pigments that circulate in the blood stream.

Greene ('28), Burpee ('28) and others mention hemorrhagic disease as a distinct etiological factor in the production of intracranial hemorrhage in the newborn.

A delay in blood coagulation, according to Ehrenfest ('29), will permit slight oozing from a small vessel to continue until it produces the picture we recognize as intracranial hemorrhage in the newborn.

In agreement with other authors, referred to previously, Jarcho (130) states that as a rule, the platelet count is low in newborn infants and during the first few days of life. He goes on to state that not until the platelet count has reached 60,000 are there any associated hemorrhagic symptoms. Qualitative changes in the platelets may be of importance as well as the total count, Jarcho says.

Tyson and Crawford (131) believe as did Capper ('28) that hemorrhagic disease of the newborn does not seem to be important as a causative factor.
"Abnormal continuous bleeding in the absence of infection is indicative of hemorrhagic disease of the newborn", is Kugelmass' (131;2) statement.

In completing such a discussion as the preceding one, it is felt that some space should be given to the normal physiology of blood coagulation. With this thought in mind we concern ourselves with the work of Kugelmass (131,b). He says "Physico-chemical studies reveal that blood plasma, so long as its constituents are not dissociated by extraneous forces, is a single complex in equilibrium rather than a mixture of substances."

In addition, he remarks that when blood is liberated it exhibits a physiological function on which is a dissociation into elements which are necessary for the clotting reaction. Whether or not this dissociation is adequate enough to yield a clot is a very important factor. He gives a graphic presentation of this phenomena which is as follows:
By a study of the individual components of this phenomena Kugelmass gives some very illuminating and interesting facts.

Antithrombin, according to Kugelmass, is a stabilizing component of the plasma complex. When bleeding occurs the plasma is dissociated. As this takes place clotting components are produced which by mass action on the antithrombin decreases its stabilizing effect.

Kugelmass says, "Prothrombin is a protein substance identified with albumin and globulin and is electronegative in blood. It acts as a nucleus of electrical condensation of the clotting components in the formation of a gel. Its concentration
is directly proportional to the clotting activity of blood."

He goes on to say that "Fibrinogen of body fluids is a globulin formed in the liver and coagulable by thrombin. It constitutes the potential clot structure dispersed in the plasma in the most readily precipitable form. Its transformation into soluble fibrin completes the clotting reaction."

"Platelets are lipoidal protoplasmic separations from megakaryocytes in the bone marrow. Clotting of blood takes place, other factors being equal, when there is an adequate platelet content and a rapid platelet disintegration liberating phosphatides".

The ability of a specified blood to clot may, then, be calculated from these findings.

This procedure has likewise been graphically illustrated as follows:
Prothrombin Platelets
-----------

Fibrinogen

or

\[
\frac{(\text{Prothrombin}) \times (\text{Fibrinogen}) \times (\text{Platelets})}{\text{(Antithrombin)}} = 1
\]

The clotting function index is thus produced which is:

\[
I = \frac{(\text{Prothrombin}) \times (\text{Fibrinogen})}{\text{(Antithrombin)}}
\]

And according to Kugelmass is a very valuable guide to the condition of the patient, and the value of each of the clotting substances of a patient gives an insight into the nature of the clotting deficiency and hence the hemorrhagic status of the child.

In closing the subject of etiology of intracranial hemorrhage in the newborn it should be said that there are several etiological factors such as syphilis, puerperal eclampsia, and others, which were not discussed separately. It was felt that they were sufficiently related to the main etiologic factors to
warrant their being placed with them. The other less common causes of intracranial hemorrhage have been dealt with similarly.
SYMPTOMS AND DIAGNOSIS.

Here, as in the section just preceding, two related subjects are to be considered together. This has been done because it was found that as a general rule the references used dealt with these two subjects as if they were more or less closely interlocking.

It is the attempt here to set before the reader abstracts from the available literature which will show the beliefs and disbeliefs of various men in regard to this subject.

A space of roughly twenty years has been covered during which time there have been many symptoms and diagnostic points set down as valuable, according to their sponsors. After a varying length of time other authors produce, what are to them, just as valuable findings but which may entirely refute those made by previous writers. The steady increase of knowledge which would be expected may not be as smooth as one would wish throughout these years, but the material to be found on the succeeding pages, it is hoped, shows a certain definite chronology.
Greene ('14) advocates lumbar puncture or cranial puncture as a means of establishing a positive diagnosis. He also states that this will afford some therapeutic relief. His statement in this last regard was merely placed here to show the close relationship of some diagnostic means and some therapeutic measures.

"One of the earliest indications of the existence of increased intracranial tension is determined" Kearney ('17) asserts, "by a direct method of ophthalmoscopic examination of the fundus of the eye."

When there is the usual amount of intracranial hemorrhage there is a slight blurring of the upper and lower margins of the disk in the first few days after birth. At a later date there is found to be anedematous blurring over the entire surface of the nerve head. When massive hemorrhage occurs there are gross edematous changes which take place quite early.

Some authors, whose work will be discussed later, do not believe that study of the fundus of the newborn's eye is of any value, while an equal number of others agree with Kearney ('17) and go further to
advocate routine fundi examinations (Rodda, '20,a).

The classical signs of cerebral hemorrhage in the newborn are, according to Rodda ('20,a) bulging fontanel, respiratory and cardiac disturbance, spasticities and convulsions. It is his belief that these may present themselves early, late, or not at all.

The diagnosis of prolonged and progressive hemorrhage is made by Rodda when the blood shows prolonged coagulation and bleeding times. The hemorrhage continues under these circumstances until the intracranial pressure is increased to the extent of producing symptoms.

According to Strachauer ('20) when respiration, in infants suspected of having intracranial hemorrhage, is established it is frequently slow, shallow and irregular. Strachauer identifies two types of pulse; first, the slow type pointing to a moderate degree of compression, and second, the rapid type diagnostic of imminent medullary paralysis.
Sidbury ('20) believes, as did Green ('14), that in all cases of suspected birth hemorrhage, a lumbar puncture should be done, not only for diagnostic purposes but for the therapeutic value as well.

A classification of symptoms has been adopted by Ehrenfest ('22) which "is based rather on the requirements for a prompt clinical diagnosis, than on the anatomic-pathologic conditions which, indeed, determine the specific symptoms."

Intracranial Hypertension. Ehrenfest, as did Rodda ('20,a) lists this as one of the classic symptoms. This symptom, Ehrenfest believes, is often the result of cerebral edema which in many cases develops as a direct sequelae of a hemorrhage. He states further, however, that the large fontanel may remain normal if the hemorrhage is small or if it is primarily or entirely subtentorial. Restlessness and incessant crying is thought by Ehrenfest to be due to the pain caused by the stretching of the dura mater. Refusal to nurse is found, he declares, in cases where the hemorrhage is not excessive.
"Convulsions represent the most obvious of all symptoms of an abnormal increase in intracranial tension". These convulsions vary in frequency, duration and interval. They are easily brought on by any manipulation of the infant. If a careful examination is made it is found that the infant is in a state of distinctly increased reflex irritability or spasticity just preceding the first convulsion. Following the convulsive state, and as the blood continues to be extravasated, there comes a paralytic state, found especially in the muscles of the extremities.

Ehrenfest gives a localizing classification of intracranial hemorrhage as well as the general classification already mentioned. He also gives some of the most noteworthy differences in the typical symptomatology of hemispheric and of infratentorial hemorrhage. From his work ('22) we find that he bases his diagnosis upon the following things:

1. Symptomatology, as mentioned before.
2. History of a difficult labor, such as a difficult forceps extraction.
3. Prematurity.
4. Asphyxiation.
5. Fetal tachycardia.
6. Delayed coagulation time and prolonged bleeding time.
7. External traumatism. "with the exception of evidently perforating injuries, fracture of skull bones and very deep indentations, other external traumatic birth injuries of the head do not permit reliable conclusions concerning intracephalic conditions."
8. Ophthalmic examination.

In a series of 18 cases of the delayed type of hemorrhage which Foote ('24) has seen, "12 gave a history of disinclination or refusal to nurse on the second day". In cases where the hemorrhage is infratentorial the cyanosis is early and marked.

Grulee and Bonar ('26) have decided that there may be practically no symptoms and that there are many light forms of intracranial hemorrhage which are never diagnosed because the symptoms are only very slight and
are attributed to other conditions.

In the delayed type of hemorrhage these authors have found that lethargy assails the child after 24 to 48 hours. The first thing noticed is that the child refuses to take the breast. As the condition progresses these symptoms become more and more exaggerated; the skin becomes pale. Following this slight twitchings begin, either spontaneously or after slight irritation. These become generalized convulsions. Grulee and Bonar content that the site of the convulsions has no bearing upon the location of the hemorrhage. Other symptoms described by Grulee and Bonar which follow in the wake of those already mentioned, are bulging of the fontanel, cyanosis, labored and irregular respiration, bradycardia. These authors do not believe, however, that these symptoms are always diagnostic of intracranial hemorrhage. They find that the lethargic state is not uncommon and that in premature infants cyanotic attacks are quite common.

After studying the work of Grulee and Bonar we may list some of the conditions upon which they base
their diagnosis:

1. Decrease in hemoglobin and red cells greater than normally found. They consider a hemoglobin of 100 and a red cell count of from 4,500,000 to 5,000,000 as distinctly low.

2. Increased spinal fluid pressure with the finding of a xanthochromic spinal fluid containing blood cells. Certain definite findings are necessary in this particular case. It must be positively determined whether the yellowish tinged spinal fluid is produced by decomposed blood or by bile pigment. There must be broken down blood cells or at least very crenated cells in the fresh fluid so as to differentiate from hemorrhage of the spinal cord due to the spinal needle. The position of the child while the puncture is being done is very important so as not to increase the cerebral congestion.

Sacks (1926) feels that due to the fact that the child's nervous system is undeveloped and that "most of the cortex does not functionate nor do the
pyramidal tracts which are still nonmedullated-- and besides does not have its brain encased in a closed box", that nearly all of the symptoms occurring in older children and adults are totally lacking in the newborn infant.

After a review of 450 cases of intracranial hemorrhage, Roberts ('27) found that 17 percent showed microscopic blood in the spinal fluid. They are very serious in their belief that routine punctures be done so as not to let so many of the cases of intracranial hemorrhage go undiagnosed. In his series only 46 percent showed symptoms of hemorrhage.

An article by Glaser ('28) rather refutes the statements made by Grulee and Bonar ('26). Glaser, after studying 100 infants and performing 129 lumbar punctures says that "spinal fluids free from microscopic red blood cells occur but rarely during the first two weeks of life". He believes that the bloody spinal fluid from infants of this age is suggestive but not diagnostic of intracranial hemorrhage. He has found that fatal cerebral
hemorrhage without any gross blood or even an exceptional number of microscopic red cells in the spinal fluid may occur in premature infants. Another striking statement that Glaser makes is in regard to xanthochromia. He finds that this is a "physiologic phenomena in premature infants during at least the first four weeks of life." He believes that a positive direct van den Bergh reaction of spinal fluid suggests either an intracranial hemorrhage or "hern icterus".

Burpee ('28) mentions the fact that epidural and subarachnoid hemorrhages are usually without symptoms.

Kutscher ('30) believes that manifestations of asphyxia in a newborn infant should always place a tentative diagnosis of intracranial hemorrhage upon him. He states that the rapid and severe symptoms are due to pressure about the medulla. The symptoms, slow in onset, are due to an oozing rather than a frank massive hemorrhage.
Tests carried out by Preissecker (1950) on the changes in the serum of infants suspected of intracranial hemorrhage show that a diagnosis of intracranial hemorrhage may be made when the serum shows a yellowish-brown discoloration. Because this test is based upon the fact that every extravasation in the body causes changes in the serum the diagnosis cannot be positive until all other extravasates are ruled out. It is possible to differentiate the pure yellow color as found in icterus neonatorum from the yellowish-brown pigment of intracranial hemorrhage.

Abt and Feingold (1930) contend that the rise in blood pressure in the newborn infant is diagnostic of intracranial hemorrhage. They do not give any reading for infants' blood pressure that may be quoted as that of normal.

It is felt by Peterman (1930) that the diagnosis of intracranial hemorrhage is not made at birth unless the symptoms are very acute. He even feels that when the infant is older and cannot sit alone or shows incoordination, athetosis or even convulsions, that
cerebral birth injury is seldom considered. He attributes this neglect to lack of careful histories. The value of careful histories and accurate details of the labor was mentioned at an earlier date by Capon ('22).

The cardinal sign of intracranial hemorrhage, Irving ('30) states, is a disturbance in respiration which continues past a reasonable length of time.

The most common symptoms, as given by Munro ('30) are, first, hypertonicity. This child shows "Spastic extremities, increased deep reflexes and a characteristic excessive muscular reaction to sudden sensory stimuli". Second, an abnormal or poor cry. Munro considers any variation from the normal in volume, tone or frequency as being an abnormal or poor cry. Third, cyanosis, which may or may not be associated with abnormal respiration. The fourth and last most common symptom, according to Munro, is the failure of the baby to nurse, which he believes, and his belief is substantiated by other men, is due to the loss of the sucking reflex. Three minor symptoms whose presence is suggestive of a grave prognosis are
flaccidity, pallor and birth asphyxia. Munro believes that the most reliable of all symptoms is the increase in intracranial pressure.

Loeber ('30) considers the history of the newborn in regard to the labor, the recovery of bloody cerebrospinal fluid and an amelioration or relief of the symptoms following the puncture as highly diagnostic or intracranial hemorrhage. The cisternal puncture rather than the spinal is recommended by Loeber for two reasons. In the first place there is immediate relief without danger of causing added congestion because of the position assumed by the infant during the procedure, and second, because of the comparative facility of the procedure as compared to the spinal puncture.

Lethargy, refusal to nurse, and the whining cry are not considered by Grulee ('31) as outstanding symptoms because he believes that most any newborn infant, normal or otherwise will exhibit these symptoms. He considered bulging of the anterior fontanel, pallor and muscular flaccidity as very indicative of increased intracranial pressure. Flaccidity is much more important than rigidity, for Grulee believes that infants of this
age normally exhibit some muscular rigidity. Twitchings, in his estimation, are valueless, as are lumbar punctures as diagnostic measures because of the position in which it is necessary to have the child as well as the possibility of getting blood in the spinal fluid by puncturing one of the veins on the inner wall of the canal. "Cisterna puncture, as advocated by Loeber ('31) would seem to be a much more rational procedure if we are to obtain spinal fluid from which we may draw conclusions".

Tyson and Crawford ('31) reviewing 45 cases of intracranial hemorrhage, add excessive loss of weight and fever which occurred in 85 percent of these cases to the list of symptoms already mentioned.

Some results from spinal puncture done on 66 normal babies show an average of 8 mm. of mercury as the spinal fluid pressure. Bloody fluid was found in 14 cases, yellowish fluid in 32 cases and clear fluid in 20 cases. These findings bear out some of Glaser's ('28) contentions.
Tyson and Crawford (131) state that history of delivery, clinical symptoms, and lumbar punctures or cisternal are essential points of diagnosis.

Braid (131) believes that the diagnosis of intracranial hemorrhage should be made upon the clinical picture and that no conclusions should be based upon the presence of absence of blood in the cerebrospinal fluid.

As was referred to previously some authors advocated ophthalmoscopic examinations of the newborn infant, while others did not. The main objections being the difficulty of the procedure plus the fact that after it had been done nothing was learned. Sykes (131) strongly advocates this type of examination because of its accuracy and because it can be done in such a short time. In his series of cases Sykes states that 60 per cent of those who died of intracranial hemorrhage presented retinal hemorrhage.

Clein (132) bases his diagnosis upon the presence of one or more of the following symptoms:
1. Cyanosis, he believes, is the most constant and important symptom. It is usually intermittent and occurs at irregular intervals shortly after birth, the intermittency differentiating it from the continuous progressive cyanosis due to other causes.

2. Asphyxia which is present at birth.

3. Apathy. Klein states that with this apathy the infant seems lifeless. There is a flaccidity of all muscle groups.

4. Failure to cry, a low moan, or an occasional grunting sound.

5. Respirations which are short, shallow and labored.

6. Difficulty in swallowing liquids and vomiting.

7. Convulsions and a bulging fontanel which are later manifestations.

8. Change in color of spinal fluid which ranges from a xanthochromic golden yellow to a deep, dark, pure bloody fluid.

It is Moore's (1932) contention that there is no definite group of symptoms early in intracranial hemorrhage in the newborn. He lists the most common
symptoms which are as follows:

1. Signs of cortical irritability, crying, twitching of muscles, spasticity and convulsions.
2. Refusal to nurse.
3. Disturbance of respiration.
4. Cyanosis or pallor.
5. Signs of increased intracranial pressure.
6. Paralysis.
7. Ocular signs.
8. Changes in the coagulation and bleeding time of the blood.

Kugelmass ('32) agrees with Grulee ('31), Moore ('32), and others when he states that "intracranial injury reveals no well defined symptomatology in the newborn".
PROGNOSIS.

It is evident that Greene ('14) considered the prognosis relative to intracranial hemorrhage as generally bad when he remarks that "the gravity of the prognosis demands an enlightened prophylaxis".

Rodda ('20,a) in concluding his noteworthy work upon the coagulation and bleeding times in newborn finds that if the coagulation and bleeding times are normal, recovery may take place. He goes further to state that with prolonged coagulation and bleeding time and with no relief of symptoms, death will ensue.

It has been definitely established by Ehrenfest ('22) that those children who have external cranial lesions at birth, but show no noticeable symptoms of intracranial hemorrhage, not only live, but are physically and mentally normal in later life. He has divided prognosis into prognosis of life and prognosis of health. In the former he has found that small hemorrhages fail to cause any noticeable symptoms. With the formation of a clot which hinders the normal function of certain vital centers in the medulla death
ensues, either immediately or during or within a few days after birth. Even small hemorrhages complicated by their infection, or a pneumonic process cause the babies' death. When convulsions occur, especially if these follow each other in short intervals, Ehrenfest gives these infants a decidedly unfavorable prognosis. In the latter cases or those dealing with the prognosis of health, Ehrenfest shows that up to fifteen years ago the obstetricians had not shown any interest or belief in the claims of the neurologists that birth injuries lead to "certain physical and mental anomalies which manifest themselves only in the later life of the child". He has reference here to imbecility, idiocy, epilepsy, hydrocephalus, strabismus, congenital spastic paralysis, etc.

In cases of intraventricular hemorrhage Crothers ('26) finds that if the child survives he may recover completely. Where there are massive hemorrhages, he concludes, the dangerous late results are not so prevalent. "Diffuse intracerebral hemorrhages are likely to result in serious trouble". Crothers goes
on to state that "if ever 'guarded prognosis' is justified it is in babies with suspected birth injury of the nervous system".

Grulee and Bonar ('26) believe that a certain percentage of children who have had intracranial hemorrhage go on into later life without handicaps. Many of these children have had definite and even severe signs of intracranial hemorrhage, yet "to all intents and purposes become perfectly normal individuals".

The statement of Fleming and Morton ('30) corroborates the statements made by previous authors when these co-authors say that in a series of 103 cases of intracranial hemorrhage 28 patients had no mental or physical defects when over a year old.

Grulee ('31) says that the prognosis is good for life and for normal mental and physical condition if adequate drainage is performed early.
TREATMENT.

Treatment has been arbitrarily divided into three sections. It is felt that this division will give a much clearer conception of the work which has been done, therapeutically, on intracranial hemorrhage in the newborn.

The divisions of treatment to be dealt with here are prophylactic, medical and surgical. Such treatment as spinal and cisternal puncture and injection of blood have been placed in the latter section, for it is believed that they are more of a surgical than a medical procedure.

There is no doubt that an immediate and rational treatment must be instituted, but such treatment can only be accomplished by a thorough understanding of the underlying etiology and pathology.

Prophylactic.

Greene ('14) feels that the prophylactic treatment of intracranial hemorrhage is very important and may be accomplished in one way, namely, by avoiding any fetal traumatism.
Ehrenfest ('22) mentions and discusses the following in this connection:

1. Forceps. He states "The forceps should never be employed in an effort to overcome difficulties arising from a marked disproportion between head and pelvis."

2. Management of the after coming head. A slow and deliberate extraction of the head through the vagina and vulvar ring, properly dilated or widened by means of an episiotomy will greatly lessen the danger of intracephalic traumatization.

3. Premature labor. There should be no induction used, according to Ehrenfest.

4. Protect the perineum by doing an episiotomy.

5. Violent means of resuscitation should not be used.

6. Twilight sleep should not be considered for it lengthens labor, necessitates the application of forceps in many cases, and causes some degree of asphyxiation.

Grulee and Bonar ('26) believe that routine coagulation and bleeding times should be taken on all
infants so as to detect any hemorrhagic tendencies.

Henderson ('28), as did Ehrenfest (22), sets forth a prophylactic measure when he states that the old fashioned methods of cutaneous "stimulation" should not be used to make the newborn breathe.

Episiotomy in the case of a rigid perineum is again advocated by Ehrenfest ('29) as being of great prophylactic value; this and the application of truly perineal forceps to a head compressed to the maximum.

As a prophylactic measure Kugelmass ('31,a), and others, believes daily clotting and bleeding time determinations should be made.

Ehrenfest ('32) feels that because traumatization and artificial delivery are so closely related, the trend toward operative obstetric practice should be curbed.

Medical.

A symptomatic treatment, as outlined by Ehrenfest, ('22), consists of the following:

1. Infant manipulated as little as possible.

2. Keep in warm and quiet room.
3. Protect against all irritation, including glaring light.
4. Ice bag to head.
5. Bromides and chloral.
6. Tube feeding.

Grulee and Bonar ('26) find that after the hemorrhage has been present a certain length of time a definite coagulum is formed which prevents further hemorrhage. Any radical treatment before this coagulum has formed would then seem harmful. They come to the conclusion then that the best means of treating these cases is by absolute rest. This absolute rest is accomplished by the use of

Sodium or potassium bromide, 2 to 3 gr. by mouth

or

Chloral hydrate, 1 gr. diluted in ½ ounce of water, the first hour, by rectum, followed thereafter every four hours by ¼ to ½ gr. also diluted in ½ ounce of water and given by rectum,

or

Morphine sulphate, 1/200 to 1/100 gr. hypodermically.
By pursuing a policy of "masterly inactivity" Irving ('30) believes, in the average case, there will be more undamaged babies born from undamaged mothers.

Munro ('30) gives a retention enema of a drachm of saturated solution of magnesium sulphate; in order to force fluids to the greatest possible extent he gives the infant salt solution subpectorally.

Kugelmas and Samuel ('31) have found that the introduction of heterologous protein into the vascular system increases the blood clotting function transiently, with a rapid return to normal.

Riesenfeld ('31) sets out an entirely new method of treating intracranial hemorrhage in the newborn. He calls this a "postural treatment". He likens intracranial hemorrhage to a hemorrhage from a cut finger. When the finger is held downward bleeding persists, but when the finger is held upright the bleeding ceases. He places the infant in an almost vertical position with the head slightly extended.
and maintained in this position by small pillows placed along the sides of the infant. The child usually remains in the upright position for 8 to 10 days, or even longer should the symptoms of cerebral hemorrhage continue to manifest themselves."

Murphy, Bowman and Wilson ('31) advocate the use of the Drinker respirator in the treatment of newborn infants suffering from intracranial hemorrhage. They have had very good results in those infants showing the symptoms for the first time some hours or even days after birth.

The use of carbon dioxide in asphyxia, Eastman ('32) believes, is not only superfluous but it tends to aggravate an already existing acidosis. He feels that instead of carbon dioxide that oxygen should be used.

A Lancet editorial ('31) bears out Eastman in this regard.

Opposed to this idea, when asphyxia is present, Moore ('32) uses a mixture of 10 percent carbon dioxide in oxygen, either in a chamber, by a nasal tube or by an inverted funnel. It is used intermittently, relieving the cyanosis and establishing regular breathing.
Convulsions are controlled by keeping the infant quiet and at an even temperature. If spinal puncture does not relieve the convulsions sodium luminal, 1/8-1/4 gr. is given hypodermically and repeated as necessary. Fluid and nutrition is maintained, states Moore, by nasal gavage. He starts with a 5 percent glucose solution, 3 ounces every 3 hours with a slow change to breast milk or formula in 48 to 72 hours.

In reviewing the effect of ultraviolet radiation upon 20 newborn infants, Sanford, Gasteyer and Wyat ('32) have found that the formation of fibrogen and prothrombin is stimulated, thus producing a greater coagulability.

Clein ('32) has found, as have many others, that disturbance of the baby unnecessarily has a bad effect. He advocates feeding the infant in the crib. Small feedings should be given every 3 hours with water or weak tea between every other feeding. When there is difficulty in swallowing from a nipple he uses a medicine dropper or gavage.
Much has heretofore been said regarding how not to resuscitate an infant. Kugelmass (132) describes what he considers to be the correct way to manipulate the infant. This method is mouth-to-mouth insufflation. First the mucous is cleared from the air passages by a tracheal catheter and the operator closes the infant's nostrils by pressure. He breathes gently through gauze into the baby's lungs. When expansion is reached the lower ribs are gently compressed and the process continued. Once the infant has emerged from the gentle manipulations, Kugelmass states, the newborn should be placed in an oxygen tent or a Drinker box respirator, with a flow of three liters of oxygen in one hour. Feeding and care of the infant should be carried on under the tent with a minimum disturbance. In cases of cyanosis an inflow of 5 percent carbon dioxide into the tent along with the oxygen should be permitted.

In those infants where there has been proven a hemorrhagic tendency, Kugelmass finds that the coagulability of the blood can be accelerated within
several hours not only by the injection of blood but by the feeding of gelatin solution. He finds that a 3 percent gelatin solution reduces clotting time more rapidly than even blood injection.

This solution is administered hourly after birth for three days. Due to a lack of the sucking reflex in these infants a correct feeding must be given to eliminate regurgitation. Kugelmass has found that a thick milk mixture containing the gelatin solution is the most favorable.

He advocates calcium bromide in 3 gr. doses every four hours between feedings as the most successful means of controlling convulsions and spasticity. A rectal administration of 1 gr. of sodium amytal in an aqueous solution, or in one ounce of 1 percent magnesium sulphate solution alleviates persistent attacks of convulsions. Morphine is contraindicated, according to Kugelmass, for it further depresses an already injured respiratory center.
Surgical.

An argument is advanced by Cushing ('05) against the possibility of carrying out operative procedures on the newborn due to the fact that the blood of these infants has such a slow coagulation time that a fatal hemorrhage might result from the incision.

Mere mention is made by Green ('14) that some therapeutic relief is afforded by lumbar puncture or by cranial puncture. He continues by saying that if these measures fail operative decompression by craniotomy is indicated. No definite procedure is advocated by Green as to the technique of these various procedures.

When it is found that the coagulation time and bleeding time of the newborn's blood is prolonged Strachauer ('20) advocates injection or transfusion of blood. Repeated tests on the infant's blood determine the necessity for additional injection of blood. In the event that there is infratentorial hemorrhage Strachauer would remove as much as 60 cc to 80 cc of spinal fluid by lumbar puncture. It is his belief that craniotomy should be promptly per-
formed in those cases of supratentorial hemorrhage. This, he states, "may consist of a decompression operation or an osteoplastic craniotomy with the removal of the blood". Any surgical treatment for cerebral hemorrhage should be instituted early and, according to Strachauer, this should preferably be done during the first weeks of life. He considers the following points as important when surgery is performed:

1. The infant should be warmly wrapped during the operation and the temperature of the operating room be preferably around 80 degrees F.
2. Ether used as the anesthetic.
3. The operation performed with scrupulous attention to hemostasis.
4. The clot carefully removed with a spatula and cotton wound applicators aided by gentle irrigation with normal saline solution.

Rodda (1920) merely mentions that delayed coagulation and bleeding times were favorably affected, in his series of cases, by the subcutaneous administration of whole blood.
A spinal puncture or any puncture to drain the cerebrospinal fluid in cases of intracranial hemorrhage would avail the most if they were done as near as possible to the primary source of the hemorrhage. Ehrenfest ('22) describes a puncture between the second and third cervical vertebra to promptly give relief in the case of a peribulbar extravasation of blood even in its incipient stage. It is his contention, however, that when dealing with cases of a decreased coagulability of the blood a relief of the intracephalic pressure may foster a continuation of the hemorrhage. Therefore it seems to him that a routine spinal puncture in suspected cases preceded by blood or serum injections, whenever there seems to be a delay in clotting, is very desirable.

Ehrenfest ('22) considers the injection of blood or serum as the most satisfactory treatment. Subcutaneous injections of from 10 cc to 30 cc of father's blood, repeated, if necessary, at intervals of from 4 to 8 hours are advocated by him.
As far as the operative treatment is concerned all Ehrenfest says in regard to that is that results are far from satisfactory.

When there is middle meningeal arterial hemorrhage the only treatment suggested by Crothers ('26) is operation. He considers there are two explanations available when no further fluid can be withdrawn though intracranial pressure is obviously high. In the first place "the spinal canal may be filled by a thick mass of more or less gelatinous clot which does not flow through the needle". Second- "the blood within the cranial cavity may block the flow of fluid from the ventricles". When spinal puncture fails Crothers advocates drainage by puncture of the ventricle through the lower angle of the anterior fontanel.

Grulee and Bonar ('26) believe that lumbar puncture is only of value when the hemorrhage is infratentorial.

In operative procedures Sacks ('26) suggests giving infants subcutaneous salt solution before the operation.
Kutscher ('30) is a strong advocate of cisternal puncture in the treatment of intracranial hemorrhage. He states that as much as 20 cc of the fluid can be safely removed if under pressure. It is his contention that whole blood, in 20 cc to 30 cc amounts, from the father should be injected subcutaneously after each tap, not only for its coagulant value but also because it furnishes nutritious food.

The advantages of the cisternal puncture over the spinal route in cerebral hemorrhage have been discussed previously, as given by Loeber ('31).

With an external hydrocephalus a subtemporal decompression is advised by McClintic ('31), and with an internal hydrocephalus he recommends a bisection of the cerebellum and roof of aqueduct.

Kugelmass ('32) merely mentions intravenous injections of hypertonic glucose for decreasing the cerebral hydration and edema, because of the difficulty of intravenous therapy in infants. He does advocate either spinal, cisternal or ventricular
puncture although he states that the fontanel route is definitely contraindicated, and shows a preference for the cisternal puncture. Kugelmass believes that cisternal puncture should be resorted to immediately once asphyxia or other drastic manifestations of massive intracranial hemorrhage are found at birth.

As far as the injection of blood is concerned, Kugelmass states that "everybody suspected of intracranial hemorrhage should be given 15 cc of whole blood intramuscularly into each buttock immediately after resuscitation". These procedures should be carried on daily, the results being checked by a reliable method for determining the blood clotting function. Treatment may be discontinued when the clotting and bleeding times have been reduced to less than 5 minutes.

Moore ('32) routinely injects from 10 cc to 20 cc of blood intramuscularly which is repeated in 12 hours if signs of persistent hemorrhage continue. In exceptional cases he advocates a definite localization of the hemorrhage, a decompression operation and the
removal of the clot and bleeding checked by ligature or packing. It is his custom to always do a puncture in cases which show bulging of the fontanels, neck rigidity or repeated convulsions.

Blood transfusion has been used by Moore in cases where the infant was in extremis. The direct method was used and 75 cc to 100 cc of blood was given.

Cisternal puncture, according to Clein ('32) "is the method of choice", as Loeber ('31) and others advise, "because it is (1) most direct route, (2) provides more adequate drainage, and (3) offers less chance of a dry tap". Clein lets the fluid drain until no more comes through the needle. No other puncture is done unless signs of increased tension develop. Whole blood is given in from 15 cc to 30 cc amounts intramuscularly.
CASE REPORTS.

Cushing ('05) gives some case reports illustrating his work upon the surgical intervention for intracranial hemorrhage in the newborn.

Case I. The patient, a male child three days old, was seen in consultation on the afternoon of August 22, 1903. The child was the first of twins, born at full term of a primipara thirty-five years of age. Though a normal head presentation, the labor was slow and prolonged, and low forceps were finally applied. The delivery was easily accomplished without the exertion of force. The child was deeply asphyxiated and it was some time before it could be made to take a breath. Its twin brother, born a few moments later, was normal in every respect. The first child did very badly for the first two days. It could not suckle, and would only occasionally swallow the drops of milk which were put in its mouth. It had a peculiar dusky, cyanosed appearance. There had been several convulsive seizures.

The symptoms were more obvious, as we had the normal child for a control. The fontanelle was tense and without pulsation. The left eye was kept closed and the pupil on this side was dilated, being twice the
diameter of that on the right side. Both reacted to light, the right more than the left. The winking reflex was present on both sides, but only when the lashes were touched. The supraorbital reflex was present, more active on the left. The child could move its arms and legs, and there seemed to be no spasticity nor difference in muscular tone on the two sides of the body. The convulsions had not been unilateral.

Contrasted with its twin, whose pulse was 110 and regular, this child's pulse was considerably slower, having an irregular action of about 92 beats per minute. The respiration, which had been rather irregular and shallow from the beginning, had on this last day assumed something of a Cheyne-Stokes type of rhythm, with periods of apnoea. The child was prepared for immediate operation, August 22d, 6 p.m. After the head was shaved and prepared, a few drops of chloroform were administered and a horseshoe-shaped incision was made through the skin just within and concentric with the border of the left parietal bone. An osteoplastic flap was then raised, including almost the entire parietal bone. The bone was cut through with a stout pair of blunt, curved scissors, very close to its thin and serrated edge. On
elevating the bone-flap a tense, dark, plum-colored dura was brought into view. When this membrane was opened and reflected, a clot about 1 cm. in thickness, apparently covering the entire hemisphere, was disclosed. This clot was lifted off in large pieces from the exposed area of the cortex with a blunt instrument, and a large amount of clotted blood which extended over the frontal and occipital regions was irrigated out from under the dura with warm saline solution. The wound was closed without drainage.

Despite its enfeebled condition, for it had taken practically no nourishment during its three days of life, the infant stood the operation remarkably well. A small subcutaneous infusion of salt solution was given, and two hours after the operation the child took some nourishment from a bottle by sucking the nipple for the first time since its birth. Though there seemed every prospect of a recovery, the infant suddenly died during the night, about eight hours after the operation.
Green ('14) gives a case which he believes indicates the type of clinical picture more often met in traumatic intracranial hemorrhage due to forceps extraction.

Case II. Female. Born August 29, 1909, by difficult forceps extraction through a contracted primiparous pelvic outlet. Weight 7 lbs. 10 oz. Baby easily resuscitated and cried vigorously. Face considerably traumatized. No cephalhematoma. Nursed well until the fourth day, when it began to refuse the breast, and to develop general pallor and progressive edema of the right side of the face. Cry feeble and moaning. Physical examination was entirely negative. Lumbar puncture yielded clear fluid. The baby was fed breast milk with a medicine dropper and stimulated with brandy and subpectoral salt solution, but continued to fail, and died, without convulsions, on September 5.

Post-mortem examination: On the right side of the head, the tissues were swollen and show dark reddish discoloration. On section, the subcutaneous tissues are swollen and markedly infiltrated with a brownish and bloody fluid. No fractures of the skull are made out. The sutures are intact. On opening the skull, there is a moderate amount of frank blood-clot resting here and
there over the right hemisphere, and a considerable amount of blood-like material in the right middle fossa. This blood is all subdural. No definite laceration of the brain tissue is made out. The sinuses are intact.

Rodda ('20, b), working on the coagulation time of blood in the newborn, reports the following case:

Case III. Girl, a twin, born March 11, 1919, with breech delivery, was in rather poor condition at birth, mouth to mouth breathing was employed. March 12, the child's condition was good. March 13, pallor, weak pulse and unequal pupils, which failed to react to light, were noted. The head was pulled to the left, the limbs spastic; the spinal fluid was blood tinged. There was hematoma of the scalp. The coagulation time was thirty-nine minutes. The infant was given 25 cc. of the mother's blood. March 14, the findings were as before, with paresis of the right side of the face; the spinal fluid was yellow. The coagulation time was ninety minutes. The infant was given 25 cc. of the mother's blood; this was repeated March 15. icterus and Cheyne-Stokes breathing were present. The spinal fluid findings were the same as on March 14. Twenty-five cc. of blood was given. The coagulation time
was eleven minutes. The child died at 10:45 p.m.

Roberts ('27), becoming interested in the frequent occurrence of blood in the spinal fluid, reports the following case:

Case IV. Age 7 years and 2 months, was premature (seven months). The labor was long and hard, a face presentation. Birth weight was 3 pounds 6.25 ounces. The condition at birth was poor; respiration was delayed, the cry was weak and the baby quite cyanotic. A puncture done thirty-eight hours after birth revealed a markedly bloody fluid. Treatment consisted in spinal drainage on three occasions at twelve-hour intervals and certain supportive measures. Fifteen cc. of whole blood were given in the buttocks although the coagulation time of the blood was normal. After a stormy period of ten days the infant began improving, though gain was slow.
Kugelmass ('33) reports several recent cases of intracranial hemorrhage in the newborn treated with 3 per cent gelatin solution in addition to other therapeutic measures.

Case V. A male infant; spontaneous birth; labor six hours; slight difficulty in delivery of shoulders. At birth the baby was extremely cyanotic to the extent of about thirty minutes. Preliminary mild resuscitation followed by the use of 10 per cent carbon dioxide-oxygen were necessary in establishing normal respiration. The cyanosis was persistent and continued to be more and more marked without any other manifestations during the second day after birth. The physical examination was negative in every respect including a roentgenogram for fractures of the skull and of the chest for thymus, insisted upon by the attending obstetrician. The birth asphyxia, the character of the labor, the massiveness of the infant and the persistent cyanosis and the subsequent lethargy I considered sufficient clinical criteria for the presumptive diagnosis of intracranial injury. A cisternal puncture revealed xanthochromic fluid under increased pressure. Once spinal fluid was removed the baby's respirations became normal in character and with a gradual,
but definite clearing of cyanosis. The temperature rose to 103 F. on the second day of birth before spinal puncture. The baby was given gelatin-glucose solution every hour from birth and later supplemented by thick feeding mixture. On the fourth day after an apparent two days of welfaring the baby began to reveal generalized convulsions which were cleared by repeated cisternal punctures and the oral administration of 1/8 grain of luminal t.i.d. as well as the rectal injection of 1 grain of sodium amytal. Five sequential cisternal punctures at twenty-four-hour intervals, together with the other therapeutic procedures previously indicated, have brought recovery to a desperately cyanotic infant from birth.

Case VI. An instrumental delivery after long labor. Ten hours postpartum the baby became restless and irritable, breathing irregular, intermittent cyanosis, right facial paralysis, inability to suck and difficulty in swallowing. Normal respirations were established by the administration of 5 per cent carbon dioxide-oxygen and continued without necessity of oxygen therapy. Gelatin-glucose solution was borne better than water and thick feeding was better tolerated than breast milk. The clotting and bleeding times were normal. A single cisternal puncture revealed blood-tinged fluid under
increased pressure. The distended fontanel receded rapidly and the baby recovered from the symptoms of cerebral pressure.
1. The work of Beneke ('10) regarding technique of opening the infant's skull at autopsy has had a marked influence upon the fundamental concepts pertaining to intracranial hemorrhage in the newborn.

2. Of all infants dying at birth, or within the first week of extra-uterine life, an average of 50 percent die of intracranial hemorrhage. The percentage of deaths in the premature infant is somewhat higher.

3. Prematurity, trauma, and hemorrhagic diathesis produce the necessary pathology to cause intracephalic hemorrhage. Due to the various birth traumata, asphyxia neonatorum plays a large part in the production of intracranial hemorrhage in the newborn.

4. Symptoms of bulging anterior fontanel, involvement of the cortex, refusal to nurse, respiratory difficulties, and cyanosis, plus the changes in the coagulation and bleeding time of the blood, and the relief of symptoms afforded by spinal or cisternal puncture, are adequate for a correct diagnosis in the majority of cases. Other symptoms and laboratory findings are helpful in the more obscure cases.
5. Prognosis is variable in cases of intracranial hemorrhage in the newborn. Many infants recover completely after having the typical signs and symptoms of intracranial hemorrhage, while others die immediately or shortly after birth. Then there are those who show no damage to the nervous tissue until years later. Prognosis depends to a considerable degree upon early and adequate institution of treatment.

6. Treatment may be divided into prophylactic, medical and surgical. The cisternal puncture seems to be replacing the spinal puncture, but any means of adequate drainage of spinal fluid is very beneficial. The prophylactic treatment of injecting parental blood intramuscularly is highly recommended. Surgical treatment is not employed at the present time as much as it was previously, possibly because of better diagnosis resulting in early adequate and less radical measures.
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