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ETIOLOGY OF INTRACRANIAL HEMORRHAGE IN THE NEWBORN

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INTRODUCTION

The French physicians, Denis (1826), Billard (1828) and
Cruveilhier (1829-1835), were the first to describe intra-
cranial hemorrhage in newborn infants[1].

Since (1826) there has been a vast amount of material
presented on the subject of intracranial hemorrhage in the
newborn. Upon looking over this material I find that most
of the literature deals with the treatment rather than the
etiology of the bleedings. I am of the opinion that before
adequate treatment can be given to any pathogenetic condi-
tion the etiology must first be well in mind and each factor
correctly evaluated as to importance. Few of the authors
who have dealt with the etiology of these bleedings have
tried accurately to correlate the factors concerned. Many
men present only one phase of the etiology and do it beau-
tifully, however, without much regard as to what has been
learned before them or the possibility of a great deal more
being learned in the future. It must be admitted that the
future will undoubtedly enlighten us on several phases of
intracranial bleedings with special reference to vitamins,
and problems of nutrition which are being investigated at
the present time.

As might be expected there has been considerable con-
troversy as to the etiology of intracranial hemorrhage in

the newborn. This controversy as I have already inferred has probably arisen because of the fact that each worker has attempted to explain fully the etiology with one specific mechanism or theory. I will attempt to show in this paper that the etiology is determined by a number of inter-related factors and that the same factors do not hold true in each individual case.

The principles underlying the most important pathogenetic explanations of the accurance of intracranial bleeding may be divided into two large groups; (A) the mechanical theories that cause the actual intracranial stress and (B) the predisposing factors which make possible the accentuation of intracranial bleeding after intracranial stress has taken place. These two large divisions will be used in this paper.

ETIOLOGY OF INTRACRANIAL HEMORRHAGE IN THE NEWBORN

A - Mechanical Theories

In order to understand the mechanism and development of intracranial hemorrhages, it is necessary to recall the anatomy of the dural septa. They are the falx cerebri, the tentorium cerebelli and the falx cerebelli. The falx cerebri arises from the crista galli and passes upward and backward, as a sickle-shaped structure, to insert into the upper leaf of the tentorium cerebelli. Enclosed between the superior edges is the longitudinal sinus; its free margin extending down into the sagital sulcus of the brain between the two hemispheres.

The tentorium cerebelli is attached laterally along the lateral sinus from the petrous portion of the temporal bone and the anterior surfaces of the occipital bone. Its two leaves, fusing at its inner margin, surrounding the midbrain. The falx cerebelli, which divides the two halves of the cerebellum, extends downward from the lower leaf of the tentorium to the foramen magnum.

These structures are simple thickenings of the dura which invest the entire surface of the brain and are continuous with the dura of the spinal cord. Its outer layer forms the periosteum of the cranial vault on the inner surface, and its inner layer is in contact with the brain surface. The falx cerebri and the ten-

torium are so placed that they are supporting structures tending to hold the cranial bones in more or less normal relationship to one another during the process of moulding at birth[2].

Rydberg[1] states that there are the following mechanical theories for production of intracranial hemorrhage. These will be listed and then discussed in full.

1) The bleedings arise through the tearing of certain veins, caused directly or indirectly by the sliding of bones of the vault under the compressive effect on the head during the passage through the birth-channel (Virchow, Kundrant, Sietz and others).

2) The deformation of the head during parturition causes a stretching and sometimes ruptures of the supportive apparatus, especially of the tentorium cerebelli; in such cases bleedings may arise from nystured vessels in the tentorium (Beneke).

3) As par. 2, except that the bleedings do not arise from ruptured vessels of the torn dura duplicatures but are caused by an obstruction in the venous efflux notably through kinking or tearing of the vein of Galen (Holland).

4) During the parturition the pains bring about by a suction effect a general stasis in the head, causing venous engorgement and vascular ruptures (Schwartz).

It can be said that none of the above give the clue to the pathogenesis of more than at most a limited number of cases of intracranial hemorrhages.

Tentorial tearing does not occur in all cases, and the theory of Beneke that the bleedings generally arise from ruptured tentorial vessels in cases of a torn tentorium with neningeal hemorrhage, has not been proven.

The pathogenetical opinion of Virchow and Kundrant perhaps accord with the actual course of events in some cases, but observations of ruptured veins are, to say the least, exceptional, and the assumed compression of the sinus longitudinalis as a causal factor of bleedings has likewise very little foundation in fact.

The theory of Holland, though in principle unassailable, may be applicable to cases of strong alteration of the cranial shape and excessive displacement of the cranial content, but there remains unexplained the numerous cases that show no signs thereof at the necroscopy.

There is in fact one reason why all these above mentioned mechanical theories are insufficient as general explanations of the intracranial bleedings: In many cases no such specific signs of mechanical strains as the theories imply can be demonstrated from the post-mortem examination.

Ehrenfest[3] states that in necropsies properly performed on all stillborn infants dying within the first few days of life, some lesion, clearly traumatic in origin, is discovered within the

skull in approximately one half of the cases. Most common among them are tears of the tentorium. In a considerable number of cases, estimated at from 20 to 25% the intracephalic injury is not directly responsible for the death but represents merely an incidental finding. In these instances, as a rule, the lesion is accompanied by only a negligible hemorrhage or none at all.

Observations and experimental studies, extensively recorded in the literature of obstetrics, have now determined the exact mechanical factors which effect the traumatization of the meninges, especially of the dura mater.

Compression of the fetal head, resulting in a relatively small reduction of the volume of the skull, but leading to a rather marked change in its configuration, represents an integral element of every labor. Experience proves that, in general, this compression is free of any noteworthy harmful effect on the child. Under normal conditions a process is at work which precludes a pathologic increase of intracranial pressure. As now understood, this protection of the skull contents and, most important, of the brain tissue, is procured by the escape of a small amount of cerebrospinal fluid toward the spinal canal, and furthermore by the reduction of the volume of blood within the brain.

The process of molding effects a change in the relative position of adjoining skull bones which is most pronounced in the sagittal suture. As the result of the overriding of the parietal

bones. The subjacent dura is both folded and stretched. If this
overlapping is excessive (mechanical dystacia) or is very suddenly
accomplished (forceps extraction, large doses of pituitary extract),
or if the dura is abnormally fragile (prematurity), the strained
dura may break. The longitudinal sinus itself may be torn open,
or, as is more commonly the case, the tear may involve only the
veins on one side, exactly at the site of their entrance into the
sinus. If these vessels are engorged (asphyxiation, or passive
congestion in the aftercoming head), they naturally will break
under a relatively smaller strain. Under, these conditions, ob-
viously, the resulting hemorrhage will be more profuse. If coin-
cidentally, there is an abnormal delay in the blood clotting
(hemorrhagic diathesis), the hemorrhages will continue, and may
attain dangerous proportions, even if the tear is small and in-
volves but a few, and those small vessels.

In a breech extraction not properly managed, exaggerated
lapping of the parietals over the squama of the occipital bone
in an identical manner may cause the rupture of veins emptying
into the transverse sinus, or laceration of the sinus itself, or
might push the cerebellum with sufficient force against the over-
lying tentorium to cause its laceration.

Far more significant, however, than this overlapping of ad-
joining skull bones is the changed configuration of the molded
head in the direct mechanical causation of meningeal lacerations.

Compression of the head in only one direction results
in the shortening of the diameter lying in the direction of
the pressure, and a simultaneous compensatory lengthening
of the diameter perpendicular to the one reduced. There-
fore, compression of the head, during the second stage of
labor, along either the anteroposterior or the lateral
diameters, always leads to an increase in the length of
the vertical diameters of the infant's head. Baker[4]
seems to be of the opinion that forces in the occipito-
frontal diameter is of the greater concern. The cra-
nium[3] becomes more convex and the falx is pulled upward.
It is generally believed that certain histolgic structural
details of such tissues as bones or ligaments permit re-
liable deductions in regard to the functional purpose of
the textures. The longitudinal arrangement of the fibers
of the falx and their lateral extension on either side in-
to the tentorium then would indicate that it is the chief
mechanical task of the falx to prevent an abnormal exten-
sion of the long diameters of the cranium; i.e., to counter-
act during moulding the effect of lateral compression. But
sudden or excessive compression in either a lateral or a
longitudinal direction, by raising the falx, would exert a
definite pull at the place where the fibers of the falx
diverge to either side to form the upper blade of the

tentorium. This also represents the relatively weakest
part of the falx and, as a matter of fact, it is the site
of predilection for tentorial tears.

Holland[5] states that alteration in shape of head
is brought about partly by a displacement of the bony vault
as a whole and partly by bending of the individual bones
which compose it. The displacement of the bones as a
whole takes place chiefly at the junction of the plate
with the base of the occipital bone; this hinge-joint
allows a considerable range of movement backwards and
forwards. The greatest bending of the individual bones
occurs in the parietals and frontal; the occipital bone
is more rigid and, although it becomes bent to a certain
extent, confines itself chiefly to the role of moving
backwards or forwards on the occipital hinge, carrying
with it the rest of the vault. The vault of the skull
is plastic as a whole and can, within limits, change shape
in most directions, but alterations in the shape of the
head are chiefly dependent on the backward or forward
movement of the occipital plate at the occipital hinge;
bending of the bones is secondary to this and is, of
course, a very necessary accompaniment.

Sachs [6] sees in the springing apart of the parietal
bones after the child's head leaves the birth channel, a

reason for the occurrence of subdural hematoma on the convex side of the brain. The difference in pressure between the intrauterine cavity and the vagina is noted, as a cause of intracranial hemorrhage by Seitz, Hannah and others.

Kundrant[7] has shown that certain conditions must be present for hemorrhage to occur. The skull bones must have firm, hard edges and a fairly wide membrane so that the bones can over-ride one another. If the bones are soft or the interstitial membrane very wide, over-riding is not so apt to occur, because the pressure of delivery compresses the brain together with the skull. Also when the bones are rigid and the interstitial membranes are very narrow, little or no displacement can take place, and hemorrhage is less likely to occur.

As has already been shown there has been some contraversy as to the exact location of the hemorrhage, and in my estimation there is no definite proof of any common sight. Some of these sights have been already mentioned. McGuinness[8] states that the most common site of injury is the triangular area at the junction of the falx and tentorium. This site was damaged in 92.5% of deaths from cerebral hemorrhage. Baker[4] states that cranial stress causes rupture of the vein of Galen or of its

tributaries. A further discussion on this point need not
be attempted in this paper.

Holland[5] states that it is obviously impossible,
during the course of labor in any given case to analyze
the exact nature of cranial stress, to measure accurately
its intensity, or predict its effects. For practical
purposes no more can be said than that the stresses and
their effects vary chiefly with (1) the absolute amount
of the forces acting on the head at any given time, (2)
the direction in which these forces act on different
parts of the head, (3) the degree of plasticity, or
ductility, of the head, and (4) whether the forces attain
their maximum effect suddenly or gradually. The only
means of estimating the intensity of cranial stress is
by observing its effects after birth. Baker[4] states
that cranial stress depends on two main factors - the
outside pressure to which the intracranial contents are
subjected, and the degree of congestion of the cerebral
veins and dural sinuses.

(B) PREDISPOSING CAUSES OF INTRACRANIAL HEMORRHAGE
AND CRANIAL STRESS

In the preceding section I have attempted to show
what the actual results of cranial stress are and some
of the basic fundamental factors concerning cranial stress.
I shall now discuss the factors which are directly respon-
sible for the cranial stress. I will call these factors,
predisposing factors, since they predispose to the cranial
stress, and make concentration of bleeding possible as a
result of the cranial stress.

There are many causes for excessive head molding at
delivery. Capon is of the impression that the most im-
portant cause in disproportion in size between the mater-
nal bony pelvis and the fetal head; for example, pelvic
contraction and oversize of the fetus. Deficient cephalic
flexion, the rigid cervix and outlet of the primegravida,
and pelvic growths are less frequent causes. Some writers
(Holland[5] Ehrenfest[3] Martin[12] have called attention
to the fact that measures undertaken to support the peri-
neum may be so vigorously carried out that intracranial
trauma is caused by compression of the squamous occiptal
bone against the maternal pubic arch. Baker[4] lists the
primary factors responsible for tentorial tears are (a)
Breech delivery, (b) Forceps application, (c) rapidity
of the second stage of labor, (d) prolongation of the

second stage of labor, (e) contracted pelvis, (f) prema-
turity of fetus. Brandt[10] reminds us that a quick com-
pression of the fetal head also occurs in multiparae in
whom the fetal head is not firmly engaged during the first
stage of labor. These infants are frequently born in one
or two pains as soon as cervical dilatation is complete.
This rapid expulsion causes momentary compression and may
cause severe intracranial injury.

I shall also discuss in this section such factors as
asphyxia, certain drugs which are frequently misused,
Caesarean section, and resuscitation. All these factors
have been discussed in detail by many authors and it seems
that they all seem to reach certain definite conclusions
as to their importance.

The type of delivery which the infant was subjected
to seems to be of great importance with respect to intra-
cranial hemorrhage.

Craig[11] constructed a chart showing the nature of
the delivery in relation to intracranial hemorrhage with
respect to his findings.

Spontaneous		Instrumental			Caes. Sect.	Total
Vertex	Breech	Vertex	Vertex Preceded by Rotation of Head (Manual or Instrument)	Breech		
60	15	28	16	2	5	126

Some writers point out certain facts that they have found to be true with respect to delivery of the infant and intracranial hemorrhage. According to Kubn[13] the incidence of intracranial hemorrhage is ten times as great among infants delivered surgically as among those delivered spontaneously. Chase[14] states that in half of his cases of subdural hematoma the infants were delivered surgically. Crathers[15] found large intracranial hemorrhages with tears of the tentorium in 88 percent of his cases of premature infants delivered surgically.

I think that one may safely conclude from the above stated information that surgical proceedure does cause increased intercranial stress which may or may not be avoided as will be shown later.

Becker[16] found a history of difficult and prolonged labor in 100 percent of his cases of intracranial hemorrhage. Seventy-five percent of these infants were first born. Sharpe[17] [18] enlarges somewhat on Becker's statement by neting that first born, full-term males having difficult prolonged labors are more liable to this complication, forceps being used as a last resort. It may be noted here that the above authors in giving their statistics have not proven or made mention that the intracranial hemorrhage may have occurred before the application of forceps. It

is very possible that the contractions of the uterus and resistance of the soft tissues may have caused the injury. It is easy to see that first born children are subjected to more traumatizing forces since the duration of labor is usually about six hours longer in primiparae than in multiparae. Generally speaking the average for the former is about eighteen hours; for the latter about twelve hours.[19]

Craig[11] noted a predominance of intracranial hemorrhage in first born in his series of 126 cases. He shows this relationship to the number of pregnancies with the following figures:

Number of Pregnancies	1	2	3	4	5	6	7	8	9	10	11	Total No. of Infants
Number of Infants with Hemorrhages	79	14	16	7	4	1	0	0	1	2	2	126

With respect to Craig's figures it seems that the prolonged labor of the primiparae is responsible for more cases of intracranial hemorrhage in the newborn than is the quick and momentary compression of the fetal head in multiparae when the infant is born in one or two pains as soon as cervical compression of the head is complete - as suggested by Brandt[10].

Forceps have been responsible for a great number of intracranial hemorrhages. It may be said, however, that this incidence is decreasing due to better training as to

their use. Beck[20] states that under no circumstances
should forceps be used as compressors, because of the
great danger of injury to the child's brain. The more
skillful the operater, the greater is his endeavor to
lessen the compression of the child's head.

Spencer[4] suggests two causes for the frequency of
hemorrhage in forceps delivery: (a) Clamping of the
great anastomotic vein by the lower anterior angle of the
parietal bone, leading to hemorrhage around the Sylvian
fissure, (b) clamping of the internal jugular vein by the
forceps blade.

Sharpe and Maclaire[21] state that forceps as a
factor causing intracranial hemorrhage becomes more im-
portant where the application is a late difficult one, or
where it is a medium or especially a high, rather than a
low forceps. Sharpe[22] also states that the use of
early and correctly applied forceps, especially low for-
ceps, does not increase the danger of intracranial hem-
orrhage; but in prolonged labor, causing extreme intra-
cranial venous stasis, and especially when instruments
are used as a last resort, the danger of intracranial
hemorrhage is greatly increased.

There are cases of intracranial hemorrhage occurring
in Caesarean section, which substantiates the fact that

hemorrhage may occur in cases where there is little stress applied to the fetal head.

Ehrenfest[3] states that serious intracranial hemorrhages have been recorded in infants delivered by Caesarean section. In some of these cases, the traumatization undeniably occurred before operation as the result of the effort of the uterus to force the relatively too large fetal head into the narrowed pelvic inlet. In other instances, in which operation was actually performed before the onset of labor, the infant's head evidently was severely traumatized solely by its forced extraction through a uterine incision of insufficient length.

Meyer and Hauch[23][24] reported that during a Caesarean section, the wound shut down on the child's head for a few seconds. The child died in ten hours and at autopsy a complete double rupture of the tentorium was found, with abundant hemorrhage.

Lapage[25] states he knew of a case of extensive cerebral hemorrhage at Caesarean section. He believes it was due to the passive congestion and tendency to thrombosis. At autopsy there was also association of thrombosis and hemorrhage in the adrenals, and was probably recent. Such an extensive cerebral hemorrhage with no observable relation to mechanical force of any

kind indicates that factors other than mechanical, such as
deficiency of vitamin K, may be potent in causing cerebral
hemorrhage in the newborn. These factors will be discussed
in full later.

Asphyxiation was formerly regarded as an important
cause of intracranial hemorrhage, and some authors still
consider it as an important etiological factor. In my
opinion it is a predisposing cause but not the direct cause.

Brandt[10] states that the importance of asphyxiation
has greatly changed since autopsies performed after Beneke's
method have established the great frequency of tentorial
lacerations. It is now believed that in many of the seem-
ingly asphyxiated newborn infants, the asphyxia is the
result rather than the cause of the intracranial lesion,
and that efforts to overcome the asphyxia often exaggerate
the effects of the intracranial lesion. Along this same
manner of thinking Ehrenfest[3] states that the effect of
a meningeal tear on the infant in the main is determined
by the hemorrhage resulting from it. The occurrence of
a hemorrhage and the size of the hematoma, therefore,
actually depend on whether any vessels are torn and whether
they are small or large, empty or full. Thus asphyxiation
enters into the problem of intracranial birth hemorrhages,
but only as a predisposing and contributory cause, and not

as the direct cause of the injury. Asphyxiation positively
cannot have any bearing on the fact that so often in labor
the tentorium is torn.

Jahr[26] says that aside from the direct pressure on
the child's head there is the disturbance of the circulation
incident to the decreased pressure associated with the
"cupping" process by which the caput succedaneum is formed.
The passive congestion in the longitudinal sinus always con-
stitutes a danger point. Then there is the problem of
asphyxial stasis, and the friability of blood vessels and
birth. The danger of anoxemia cannot be overstated. Even
under normal conditions, placental blood going to the fetus
has only one-fifth the oxygen content of adult artereal
blood. The returned venous blood shows a four fifths dimi-
nution of oxygen as compared with adult diminution of one-
third. Until he becomes physilogically independent, there-
fore, the infant is in constant hunger for oxygen. In the
experimental work of Landis[27], he showed that fluid passes
through the walls of the capillaries at four times the
normal rate after complete oxygen want lasting three min-
utes. The capillary walls may be permeable not only to
blood plasma but also to the red cells so that cuffs of
hemorrhage may be seen.

Graham[28] claimed that hemorrhages in new-born in-

fants are probably the result of a deficiency in oxida-
tion, and that similar hemorrhages can be produced by
direct asphyxiation or by the administration of chloro-
form. Asphyxia can occur as a result of: (1) Diseases
of the placenta or premature separation of the same (2)
prolonged interval between the birth of the head and the
remainder of the body and (3) pressure on the cord.
Boyd[29] states that the cord about the neck and decreased
placental blood supply which may result from continued
and vigorous contractions of the uterus after giving
pituitrin will also contribute to asphyxia. According
to MacHoffie[28] the above factors can lead to cerebral
venous congestion; which in turn will cause hemorrhage
of the cortical and subcortical vessels and cerebral
veins; the resulting hemorrhage will cause increased
intracranial pressure and medullary edema, which may
lead to death or asphyxia at birth.

Irving[30] states that in any case of asphyxia
associated with brain hemorrhage, the asphyxia is the
primary cause of the hemorrhage. Rydberg[1] makes the
statement that asphyxia is in itself sufficient to cause
bleeding into the cranial cavity.

Munro and Eustie[31] seek to explain the majority
of such hemorrhage as a result of intrauterine "asphyxia."

This asphyxia from whatever cause (pressure on the umbilical cord, rigidity of the mothers' soft parts, dry labor, etc.) raises the cerebral venous pressure and produces a venous congestion. They interpret the hemorrhage as an effect of the increased intracranial pressure that follows. These authors are of the opinion that intracranial hemorrhages are but rarely traumatic in origin.

Brandt[10] pointed out that in the Schultze method of resuscitation of the asphyxiated infant, the shoulders are grasped on either side and the head is fixed between the wrists. In the swinging there is frequently considerable lateral compression of the head. Such compression may cause tentorial laceration and since such an infant is asphyxiated and its vessels engorged, even a small tentorial injury may cause considerable extravasation of blood. All vigorous manipulations during resuscitation where the baby is swung or held head downward, will make a severe intracranial lesion of one that might have been mild or given no signs of existence.

Hansen when referring to Keegan's discussion of Johr's article[29] on "The Mechanics of Intracranial Injury in the New-Borne", states that, "I cannot refrain from adding emphasis to the point which Dr. Keegan has made, relative to the analogy between an injury to the

brain of an infant and an injury to the brain of the adult.
Many times the obstetrician is responsible for increased
intracranial damage by the use of unwise procedures in
attempting to resuscitate the patient. I am sure if you
gentlemen (addressing the members of Nebr. Med. Association)
were called to see an individual who had been in an auto-
mobile accident and had a severe trauma to his head, that
you would not put him into a hot bath and then into a
cold bath, and swing him through the air, and do a lot
of other things some men do to babies such as spanking,
slapping him on the body, which tend to increase the
amount of bleeding from the intracranial vessels."

Hall[32] reminds us that syphilis has long been re-
garded as one of the foremost causes of intracranial hem-
orrhage. Except in the case of the premature, this is
not now thought to be very often true.

Von Haam[13], Ehrenfest[3], Signarelli[33], all
agree with the statement made by Bloom[34]. Syphilis,
toxemic conditions of the mother, early low forceps, and
hemorrhagic diseases of the newly born are relatively un-
important as factors in the production of intracranial
hemorrhages of the newly born. Burpee[35] adds cardiac
lesions to the above list. Roberts[36] made an inten-
sive study of the effect of syphilis in 1,853 patients

with positive Wassermann. He came to a definite conclusion
that the disease had little to do with the etiology of
intracranial hemorrhage in the newborn. Ehrenfest states
that among the many writers on the subject only the French
assert that syphilis does play an important role in the
causation of intracranial hemorrhage. Haam[13] states that
the modern concept that syphilis is a cause of intracranial
hemorrhage is probably true only so far as the condition
is responsible for the prematurity of the infant.

Prematurity is one of the most important predisposing
causes of intracranial hemorrhage. It is stated, however,
by Holt and McIntosh[37] that the susceptibility of pre-
mature infants to cerebral hemorrhage, formerly ascribed
to their fragile, delicate blood vessels, may well be due
in considerable part to their exceedingly low prothrombin
concentration. Recent studies suggest that the frequency
of cerebral hemorrhages can be greatly reduced by the ad-
ministration of vitamin K to the mother toward the end of
pregnancy or during labor. These factors will be discussed
in detail later in this paper.

Crathers[15] found large intracranial hemorrhages
with tears of the tentorium in 88 percent of his cases
of premature infants delivered surgically. Craig[11]
shows the following figures with respect to 126 of his
cases.

MATURITY IN RELATION TO INTRACRANIAL HEMORRHAGE

Premature	Full-term	Postmature	Total
67	48	11	126

When we speak of prematurity according to Griffith and Mitchell[38] we mean a birth weight of less than 2500 gm. (5.5 lbs.) and a length under 45 cm. (17.7 in). These factors are usually indications that the infant is premature or immature. The statistics given for the frequency of premature births vary greatly, most clinics reporting from 5 to 15 percent.

Premature and immature infants may be classified as follows:

(I) Those who are normal for their fetal age and manifest no pathologic changes.

(II) Those with pathologic changes due to (a) acute infections in the mother during pregnancy; (b) constitutional diseases and chronic infections in the mother, such as cardiac disease, nephritis, diabetes, endocrine disorders, tuberculosis, and syphilis; (c) maternal factors influencing fetal nutrition, such as overwork and undernourishment; (d) local conditions in the mother, such as contracted pelvis, premature separation of the placenta, difficult presentation, uterine fibroids, and all other conditions producing asphyxia of the infant; (e) multiple pregnancies, which cause 10 to 15 percent of prematurity; (f) constitu-

tional defects and congenital malformations in the fetus; and (g) probably advanced age of the parents.

(III) Those who are born at full term but are immature and manifest pathologic changes due to the causes already enumerated.

From a careful histological study of the tentorium cerebelli, which is the usual site of the hemorrhage in the premature infant, Chase[14], thinks its structure is responsible for its greater friability in the premature infant. He found that the tentorium cerebelli of the premature child differed in three important respects from that of the mature child. In the tentorium cerebelli of the premature child there is (a) a predominance of fibroblasts with few collagen fibrils; (b) an absence of elastic tissue fibers; (c) a relative abundance of lymph spaces and thinned walled vessels. This immaturity of the fibrous connective tissue as shown by the absence of collagen and elastic fibrils accounts for its increased friability, and this predisposes to injury.

Capper[39] terms the immature and premature infants vasolabile individuals, ie., infants in whom a small trauma will produce hemorrhage. Their blood vessels are readily torn and injured. The elastic tissues are the last ones to develop in the body, and a study of the vascular systems

of immature and premature infants discloses a poverty of elastic tissue, which necessarily predisposes the vessels to trauma and the hemorrhages commonly found.

In the case of immature and premature infants, even if the obstetrician utilized the most ideal prophylaxis or watchful waiting policy, cerebral hemorrhage will nevertheless occur in many cases. This is so because that particular infant is vasolabile and the contractions alone are sometimes sufficiently traumatizing to cause hemorrhage.

Crowder[2] states that since the development of the dural septa does not begin until late in the fifth month of fetal life one can readily see that they could be and are more easily torn by a minimum amount of trauma during the last trimester. The more premature an infant, the more possibility there is of damage being done to the septa, even in a fairly normal expulsion.

Vaglio[40] quotes that the low vitality of the prematurely born may be due more to the trauma of the delivery than to the immaturity of the child. Brandt[10] says the premature infant is extremely vulnerable to the traumatisms of birth and in a large proportion of the fatal cases, labor is normal and spontaneous. However, again it must be emphasized that in spite of the fact that prematurity means a small baby, and therefore less

general compression, yet compression of the head may be rapid and of short duration, as might be caused by rigid soft parts in the birth canal..

Pituitrin should be considered as a predisposing factor since there is a great deal of misuse of this drug and other similar drugs.

In Sharp's article[41] pituitrin is mentioned only to condemn its use before delivery except when strictly indicated. It has been likened to a surgical instrument, to be used only by those of utmost judgment and knowledge of its effects. Comparative competency is required in the employment of drugs producing deep narcosis and interference with normal progress in the second stage. As in delayed labor from other causes, the child runs the risk of asphyxia with cerebral edema and hemorrhage, and the need for operative measures may be precipitated. In many of these cases, to leave a fetal head compressed for hours in a pelvis or on a perineum, in lieu of a properly executed instrumental delivery, is as bad for the child as too early interference. Boyd[29] adds to Sharp's statements by saying that pituitrin when given in a case of labor where for some reason labor is not progressing as rapidly as it apparently should, we have very much the same condition existing as the improper use of forceps. The rapid and

harder pains cause a quicker moulding of the head and the almost continuous pains do not give time for equalization in the circulation of the infant between pains.

In recent years considerable knowledge has been gained about vitamins. Certain vitamins such as "K" and "C" are very important with respect to hemorrhagic tendencies in the newborn. Sharpe and Maclaire[21] state that hemorrhagic diseases of the newborn is a very important cause of cerebral hemorrhage in infants, occurring in 44 percent of the deaths in their series.

Boyd[29] states that in autopsies of cases of intracranial hemorrhage, it is occasionally noted that there are smaller and larger hemorrhages in the mucosae and serosae of the body. These were formerly considered as evidence of the asphyxia, which was believed responsible for the intracranial lesion. Most likely all the hemorrhages in such cases are due to a common cause, the hemorrhagic disease of the newborn. Of course, intracranial trauma in the presence of hemorrhagic diseases of the newborn will result in a greater outpouring of blood from the injured vessel and result fatally sooner.

Ehrenfest[23] states that in fifty-three cases of intracranial hemorrhage found in 136 consecutive autopsies, hemorrhage in other organs was present in twenty.

From this point of view, the first thirty-five consecutiv e
cases of intracranial hemorrhage in this series of fifty-
five were studied in detail. In twenty-two cases of intra-
cranial hemorrhage (from slight to severe in amount) there
was no hemorrhage elsewhere in the body although one child
was definitely a "bleeder". In thirteen cases (from slight
to severe in amount of bleeding), there was hemorrhage else-
where in the body, especially in the adrenals. These thir-
teen cases with hemorrhage, both intacranial and elsewhere,
were further studied to see if there was any relation be-
tween the severity of hemorrhage in the cranium and the
severity of the hemorrhage elsewhere in the body. Of nine
cases with slight vesical hemorrhage, five had extensive
and four had slight cerebral hemorrhage. Of four cases
with extensive visceral hemorrhage three had extensive and
one had slight cerebral hemorrhage. These figures show no
tendency for slight cerebral hemorrhage to accompany slight
visceral hemorrhage, but as far as the figures go, a tend-
ency is shown for extensive cerebral hemorrhage to accom-
pany extensive visceral hemorrhage. We may conclude from
above that hemorrhagic disease is probably not the cause
of intracranial hemorrhage, in general, but in certain
cases it must be an important factor as in the following
instances. A child died three days after a difficult

breech extraction. During life, the bleeding was four min-
utes, but the clotting time was over two hours and fifty
minutes. Autopsy revealed masses of blood clot over the
cortex and about the base of the brain, and also hemorrhages
into the scalp, one adrenal, and under the periosteum.

Sage[42] states that it has been shown by many workers
that all newborn infants have low prothrombin readings be-
tween the second and sixth days after birth.

He says that there is sound therapeutic basis for
administering vitamin K to prevent hemorrhagic diseases
of the newborn. Recent plasma prothrombin studies have
given us a new conception of hemorrhagic diathesis of the
newborn, which is really a hypoprothrombinemia which all
newborn infants exhibit to a certain extent and in various
gradations.

Work presented by D'Alessandro[43] substantiates Sage's
statements above, when he presents his article concerning
the role of vitamin K in blood coagulation.

D'Alessandro states that the exact mechanism of blood
coagulation is not fully understood, it is generally accepted
that prothrombin, thromboplastin and calcium interact to
form thrombin which combines with fibrinogen to form fibrin.
The only physiological anticoagulant known is heparin,
which is not found in circulating blood in any appreciable

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amount.

He continues by saying that vitamin K apparently is a
precursor or possibly an enzyme in the formation of pro-
thrombin, which is a product of liver metabolism. Absorp-
tion of the natural fat soluble vitamin K from the intestinal
tract requires the presence of adequate bile salts and a
normal mucosa. Its utilization after assimilation depends
on the presence of healthy liver tissue for the formation
of prothrombin.

D'Alessandro correlates all the above with hemorrhagic
diseases of the newborn by stating that Rodda, in 1920,
called attention to the defective coagulation of blood in
infants in the first few days of life, but no explanation
was offered until 1937 when Brenkhous, Smith and Warner re-
ported that the blood prothrombin level of infants is very
low when compared with adult values, the average being about
one-fourth the adult concentration. It has been shown that
the infant blood prothrombin falls from the second to fourth
or sixth day of life when it may again reach the birth level,
but does not reach the adult level until about the end of
the first year. This fall is even more pronounced in pre-
mature infants. It is a well known fact now that vitamin
K is produced by bacterial action. Logically it follows
that as soon as the baby ingests food or water, it infects

its intestinal canal, which at birth is sterile. With the establishment of an intestinal flora the synthesis of vitamin K begins, thus producing a rise in prothrombin after the first week of life. This explanation might account for Javert's observation that hemorrhagic disease was twice as common in babies in the New York Hospital, where feedings were prepared according to the most sanitary methods, as in babies born at home.

In 1939 Waddell, Guerry, Bray and Kelly reported on the administration of vitamin K concentrates to two infants with very low prothrombin levels and prolonged coagulation time with the dramatic and rapid restoration of normal values.

Sage[42] continues by saying that it is well to remember that vitamin K will never be a substitute for obstetric skill and gentleness in manipulation and handling of the newborn. In regard to cerebral bleeding, it is clear that vitamin K will have little or no effect if the tentorium is torn or a large cerebral or meningeal artery is severed.

Vitamin K is not a panacea for all hemorrages. If the trauma of labor has been severe enough to rupture one or more blood vessels in the infant's brain, vitamin K will not save that life. In a case of any difficult delivery in which the baby may have sustained cranial injury, vitamin K should be promptly administered either orally or

hypodermatically. If an emergency operation has to be done
on a newborn baby, vitamin K must be a part of the preopera-
tive treatment.

Beck[44] states that latent scurvy in the fetus has
been suggested as a factor in the etiology of cerebral and
other hemorrhages which occur during or soon after birth and
play no small part in neonatal mortality statistics.

Beck's statement may be better understood by Bodansky's[45]
statements that in vitamin C deficiency we see diminished
capillary resistance, marked tendency to hemorrhage, struc-
tural changes in cartilage, bone, and teeth, and enlargement
of the adrenals. According to Wolbach and Howe, the funda-
mental defect is the loss of ability of the supporting
tissue to produce and maintain intercellular substances.
As these cementing and supporting materials are resorbed
and nothing is provided to take their place, there being
no new formation of intercellular material, reparative pro-
cesses cease. As there is a lack of fibrous and collogenous
substance, structural weakness results. As a result of
these morphological changes, hemorrhage follows even slight
trauma. Harrow[46] shows that in the absence of ascorbic
acid, according to Harris, certain cells no longer function
normally in their oxidative capacity, with the result that
the blood vessels become weak and bleeding follows.

Munro, Lazarus, and Bell[47] state that estimation of capillary fragility has been widely used and regarded as an index of vitamin C intake. However, they find that there are many difficulties in accurately determining the capillary fragility - thus they are uncertain how much the effects of lack of vitamin C on the capillaries may be obscured by other factors. Hess (1920) noted in his monograph that even in cases of scurvy the capilla ry fragility was not uniformly increased. Munro, Lazarus, and Bell in their first investigations (1940) found that healthy people with increased capillary fragility usually showed a reduction in the petechial counts after a course of vitamin C.

It is conceivable that a latent scurvy with accompanying hemorrhagic tendency in the newborn may develop rather easily since Beck[44] states that there is an increased need for vitamin C in pregnancy is shown by the observation that the level of ascorbic acid in the blood decreases as gestation advances. Irrespective of the diet, the amount present at term (43 mg.) is little more than half that present in the earlier months (0.72 mg.) and it is difficult to maintain the normal level of 1 mg. per 100 cc. unless the diet is supplemented by additional ascorbic acid.

From the above information we may safely conclude that vitamin C deficiency is a predisposing factor in the etiology of intracranial hemorrhage in the newborn.

CONCLUSIONS

After carefully studying certain factors which I have presented. I have come to the conclusion that the etiology of intracranial hemorrhage of the newborn centers itself around the phrase "cranial stress" with or without the addition of vitamins C and K deficiencies and hemorrhagic diseases of the newborn.

I believe that I am correct in saying that compression of the fetal head resulting in a relatively small reduction of the volume of the skull, but leading to a rather marked change in its configuration, represents an integral element of every labor.

Compression of the head in any one direction results in the shortening of the diameter lying in the direction of the pressure, and a simultaneous compensatory length-ening of the diameter perpendicular to the one reduced. Therefore, compression of the head, during either the first or second stage of labor, along either the anteroposterior or lateral diameters, always leads to an increase in the length of the vertical diameter of the infant's head. The cranium becomes more convex and the falx is pulled upward. Thus this is the mechanism of the intracranial stress - no matter what forces act upon the head to cause it.

The more common causes of this intracranial stress according to my conclusions are; first, disproportion in size between the maternal bony pelvis and the fetal head, malpositions and difficult presentations which lead to long hard labors; secondly, the incorrect use of forceps; thirdly, the unwise use of pituitrin, followed by poor methods of resuscitation.

As far as Caecarean section is concerned we find that intracranial bleeding has taken place in certain instances. This bleeding may be due to stress (the methods which have been discussed) or to a vitamin K deficiency.

Prematurity frequently accompanies intracranial bleeding because the premature passes weaker structural development. From this one may safely conclude that they are much more susceptable to intracranial stress.

Syphilis with respect to the findings of this paper is important only as a factor in predisposing to prematurity of the infant.

Asphyxiation may be a result of intracranial hemorrhage, rather than an etiological factor. However, asphyxia will lead to a cerebral venous congestion, and oxygen lack undoubtedly does make the vessels more permeable. It is doubtful, however, if bleeding from these factors alone would be extensive enough to cause symtoms of bleeding.

It is granted that congested vessels may be more easily ruptured by cranial stress and the bleeding accentuated due to the engorged vessels. Asphyxiation may be considered only as a predisposing factor in intracranial bleeding in the newborn.

Vitamin C deficiency does cause a lack of cementing and supporting materials of the blood vessels, and other tissues. Vitamin C is also responsible for the formation of intercellular material, and reparative processes. It can be concluded that a vitamin C deficiency greatly increases the possibility of bleeding from slight amounts of trauma and does predispose to a bleeding tendency. However, it is doubtful if a vitamin C deficiency alone would cause a symtomatic intracranial hemorrhage which would demand treatment.

Hemorrhagic diseases of the newborn and vitamin K deficiency will undoubtedly in some cases allow enough bleeding to be of importance. However, in general the importance of these factors is that due to the low prothrombin level, bleeding is greatly increased after damage has already been done by intracranial stress.

We may now conclude that intracranial stress actually causes the damage to the blood vessels, and that the other factors which have been discussed make possible

predisposition to trauma and an accentuation of the bleeding
after injury has occurred.

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