CEREBRAL   BIRTH   TRAUMA

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Thesis

submitted by

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P r e f a c e.

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INTRODUCTION.

The object of this work is to investigate the causation of cerebral birth trauma, and assess to what extent traumatic damage to the child's brain at birth can be held responsible for the subsequent development of Mental Deficiency, Cerebral Paralysis and Epilepsy.

The thesis is made up of three parts.   In the first part there is a pathological and clinical investigation into the causation of cerebral birth trauma in a series of cases where stillbirth or neonatal death had occurred.

The second part consists of an investigation into another group of cases in which the clinical histories were similar to those in the first series investigated, but where the cerebral birth trauma was not sufficient to result in stillbirth.   Upon these cases an attempt was made to find the extent to which birth trauma might influence the child's later mental and physical development.   This second part of the work was amplified by a subsidiary investigation into the birth records and history of a number of cases where mental deficiency was known to exist, in an attempt to find whether the mental deficiency could be directly attributed to cerebral birth trauma.

The third part of the work consists of a study of the relevant literature on the subject, in which the views of

the various authors are discussed and commented on.

The work was commenced in April 1939. The pathological examination of 100 stillborn babies was completed. With the outbreak of war, however, in September, the author was recalled for service and had, of necessity, to curtail the scope of the second part, namely, that dealing with the clinical follow-up and the examination of the mental defectives. While on leave a further attempt was made to complete the follow-up but, on account of the evacuation of school children, this had to be abandoned. It is thought, however, that, although the numbers in the second part of the investigation are not as large as was originally intended, they are sufficiently large to record and to throw some further light upon a subject of considerable interest.

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CHAPTER I.

PLAN OF PATHOLOGICAL INVESTIGATION.

This investigation was carried out in the Glasgow Royal Maternity and Women's Hospital, and commenced in April 1939.

The first part of the research was planned in order that the etiology of cerebral birth trauma could be determined. It was thought desirable to study, not only the immediate pathological damage, but also the circumstances surrounding the birth of the child.

In order to become familiar with the general necropsy findings in the crania of the newborn, some thirty practice cases were examined. Thus it was possible to evolve a routine technique for every case, before the examination of the series of 100 consecutive cases was undertaken.

Each post mortem examination was completed before any reference was made to the history of the confinement.

Permission for the post mortem examination was not always immediately obtainable on account of the mother's condition after delivery. This necessarily caused some delay in fixing the brain tissue. The delay, however, was seldom greater than twelve hours, and post mortem changes were controlled as far as possible by wrapping the child immed-

iately after death, and putting it in the cool post mortem room.

CHAPTER   II.

PATHOLOGY.

SECTION I: SURVEY OF CASES EXAMINED.

The clinical features of the 100 cases examined from a cerebral pathological aspect were as follows:

(a) Time of Death:

        Antepartum Mortality .... 17 cases
        Intrapartum Mortality .... 54 cases
        Neonatal Mortality .... 29 cases.

Antepartum Mortality: This group included cases where death had occurred in utero before the onset of labour. In none of these cases had the foetus undergone sufficient maceration to interfere with dissection. The great majority were associated with accidental haemorrhage. One case was a deformed child with exomphalos and only one leg, but the cranium was normally developed.

Intrapartum Mortality: This group included all cases where a foetal heart-beat could be heard clearly on abdominal auscultation before the onset of labour, but the child was born dead.

Neonatal Mortality: This group consisted of children who died within the first three weeks of independent life.

(b) <u>Parity of Mother</u>:

      The mother was a primigravida in ... 44 cases.

      The mother was a multipara in ...... 56 cases.

(c) <u>Mode of Delivery</u>:

      Normal spontaneous deliveries ... 50 cases.

      Abnormal deliveries .... 50 cases.

Twenty of the abnormal cases were delivered by forceps: low forceps, 6; mid-forceps, 8; high forceps, 6.

Twenty-three cases were delivered as breeches: spontaneous type, 10; manual type, 13.

Seven cases were delivered by Caesarean section.

(d) <u>Maturity of Children</u>.

    Mature children (weighing 2.5 kgs. and over), 44 cases.

    Premature children (" less than 2.5 kgs.), 56 cases.

(e) <u>Duration of Labour</u>.

The duration of labour in 70.7 per cent of the primigravidae was up to 24 hours; almost half of these cases were in labour less than 12 hours. The duration of labour in 61.5 per cent of the multiparae was below 12 hours.

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SECTION II: PATHOLOGICAL TECHNIQUE.

The routine method of post mortem examination was as follows:

    (a) Fixation.

    (b) Exposure of brain tissue.

    (c) Removal of brain and cord.

    (d) Preparation for microscopic sections.

(a) **Fixation**:

It was found by trial to be valueless to attempt any fixation of the tissues of premature and macerated foetus, and thus only mature subjects underwent the process. In the case of the macerated ones in general the tissues had undergone slight autolytic changes and fixation was impracticable. The brain tissue in the premature babies could be easily dissected, and the presence of a relatively larger amount of cerebro-spinal fluid enabled the hemispheres to be delivered from the skull with comparative ease and little handling.

In the case of the mature subjects fixation was performed by the use of half-strength Liquor Formaldehyde injected (by means of a Higginson's syringe, attached by rubber tubing to a canula) into the common carotid vessels on either side. An incision was first made in the midline running from immediately below the chin to the manubrium

sterni. The internal carotid arteries were next exposed
and ligated proximal to the canula. The internal jugular
vein of the same side was next cut across, and water was
then pumped through the canula until the return flow from
the jugular veins was practically clear of blood. The
syringe was then filled with the formaldehyde solution, and
this in turn was permeated through the arteries of the
brain tissue until its return along the jugular vein was
detected by smell. The vessels were then ligated, and the
same process carried out on the opposite side. It was
found by experiment essential to inject both carotids as
the anastomosis of the cerebral vessels was inadequate to
permit a sufficient flow to the brain tissue on the side
opposite to the carotid injected. Having firmly ligated
the carotid arteries and jugular veins, the initial incision
was sutured, and the child left flat in a cool atmosphere
for twenty-four hours.

(b) Exposure of brain tissue:

In all cases the same approach was made to expose the
tissue without leaving any obvious signs of the necropsy.

The sagittal incision used by many previous workers
was regarded as being in itself a likely cause of damage
to the dural structures, and was thus abandoned.

A coronal incision was first made, extending between the mastoid processes and penetrating the complete scalp. The scalp was then stripped anteriorly and posteriorly over the skull, when the presence and consistence of any caput formation was noted. The presence of moulding of the bones of the vault, or any undue sub-pericranial haemorrhage was next observed.

By means of a small incision in either side of the centre point of the lambdoidal suture, the blades of a pair of Mayo's dissection scissors were introduced extradurally, and the parietal bones cut out separately. The frontal bones were dealt with in like manner.

The dura mater was then stripped off the surface of the cerebral hemispheres, and the presence of any subdural congestion or haemorrhage detected.

(c) Removal of brain tissue:

The peduncles of the cerebrum were next cut through, and the hemispheres permitted to drop out of the skull. The tissue, which was of firm consistency, was then sectioned and examined for the presence of congestion, haemorrhages, or other abnormality.

The dural folds were next examined in situ for the presence of free-blood clot, and any traumatic damage of the

falx cerebri and tentorum cerebelli carefully observed.

The falx cerebri was then freed by an incision at the anterior attachment, and both tentoria cut by an incision commencing at the mid-point of the free edge and extending to the margin of the lateral sinus. The cerebellum, mid-brain and cord were then withdrawn and examined sectionally.

(d) Preparation of tissue for microscopic sections:

Where it was proposed to cut microscopic sections the brain was not subjected to partial fixation by the formalin solution. In this way the vessels could be examined microscopically in their congested state, and the histology could present no changes which might be attributable to the fixation solution.

FIXATION:

Complete immersion in corrosive sublimate solution for 16 hours.

Washed in running water for 24 hours.

Immersed in methylated spirit (64 O.P.) for 24 hours.

Immersed in pure alcohol (74 O.P.) on two occasions for 4 hours each.

Immersed in chloroform on two occasions for 4 hours each.

Finally imbedded in paraffin.

STAINING:

Haemalum immersion for 5 minutes.

Washed in running water for 15 minutes.

Eosin immersion for 15 minutes.

Then dehydrated, cleared, and mounted in Canada Balsam.

SECTION III: PATHOLOGICAL FINDINGS.

The following pathological findings were noted particularly:

1. Caput succedaneum.

2. Cephalhaematomata.

3. Cranial moulding.

4. Fractures of the skull bones.

5. Oedema of the foetal brain.

6. Congestion of the brain tissue.

7. Intracranial haemorrhage.

8. Injuries to the dural septa.

9. Injuries to the spinal cord.

1. Caput succedaneum:

Occurrence: This was present in 42 of the cases, and was quite independent of the time of the child's death.

Thus in 6 cases a caput was observed at necropsy in spite of the fact that the child had died before the onset of labour. This finding is not in agreement with the older view, which ascribed caput formation to an obstruction of the venous blood from the presenting disc of the scalp.

In 29 of the 42 cases the child had died during labour. The caput was generally formed over the vertex of the head. From its situation one could frequently assess the degree

of flexion of the child's head.   The more anterior the
caput the greater the deflexion, and the greater the liability
of intracranial damage.

In 7 cases the child had died during the antenatal
period, and the caput had persisted.   Of these 7 cases the
maximum time of persistence of the caput was two days.

Pathology: The caput succedaneum was found to consist
of an infiltration of sero-sanguinous fluid into the subcutan-
eous tissue of the scalp and tissue beneath the galea aponeur-
otica.   In most cases (88 per cent) there was slight haemorr-
hagical subpericranial staining beneath the site of the caput.
Occasionally in this position the meningeal vessels were di-
lated.

In 9 of the cases the caput was of grossly haemorrhagic
consistence: these cases showed no common factor to account
for this production.

The formation of the caput was found to bear a direct
relationship to the length of the mother's labour.   Out of
the 16 cases in the series where labour was of over 24 hours'
duration, caput formation occurred in 14 cases;   the other
two cases were breech deliveries, and the caput was not
therefore encountered during cranial examination.

The development of the caput was seen more frequently
in mature than in immature children.   The incidence of caput

formation in premature children was only 30 per cent, where-
as in mature children the incidence was 57 per cent.   In
this series caput formation was therefore almost twice as
frequent in mature children.   This finding is explained by
the fact that the rupture of the membranes is generally
earlier in the birth of mature babies. This explanation is
substantiated by the fact that in none of the seven babies
delivered by Caesarean section before the rupture of the
membranes was caput formation observed.

Caput formation was found to be a more common occurrence
in the case of first children than in the children of multi-
parous mothers.

The incidence in children of primigravidae was 50 per cent
The incidence in children of multiparae was 36 per cent.
This finding can be accounted for by reason of the firmer
nature of the soft parts of the primigravidae.

In conclusion, it may be said that several factors play
their part in the formation of a caput succedaneum. The
size of the caput is proportional to the length of time which
elapses between the rupture of the membranes, and the expul-
sion of the child. Caput formation is favoured by the de-
livery of a large child by a primigravida, where the mem-
branes have ruptured early and the labour has been prolonged.

2. <u>Cephal-haematomata</u>:

    Occurrence: There were 3 cases in which cephalhaemato-
mata had developed over the parietal bones. In two of the
cases the effusion of blood was present beneath the peri-
cranium of both parietal bones; in the third case it was
unilateral.

    Etiology: No common etiological factor could be dis-
covered to account for its production. Two cases were instru-
mental deliveries, and one spontaneous.

    Two cases had short labours and one prolonged.

    In two cases the mother was multiparous.

    All three cases showed other evidence of cerebral birth
trauma.

    Pathology: Cephalhaematomata probably develop by rupture
of the sub-periostal capillaries, which are alternately
stretched and relaxed by the recurrent displacement of the
soft tissues against the underlying bone during the uterine
contractions. They are usually formed before birth, as is
shown by the fact that in all three cases the child was born
dead. An increased coagulation time of the infant's blood
would favour their occurrence.

3. <u>Cranial moulding</u>:

    Occurrence: Overlapping of the foetal skull bones was

present in 34 per cent of the cases.   Moulding was observed in all types of cases irrespective of the actual time of death, although it was more commonly found in cases where the child had died during labour.

Etiology: The presence of moulding is much more common in mature babies, and especially those over the average weight. Thus, in the case of the mature babies 48 per cent showed evidence of moulding, whereas only 23 per cent of the premature children had this feature. The incidence in post mature cases was 64 per cent. The explanation of this finding is obvious, as is the fact that moulding was found to be a more common finding in the children of primigravidae than in those of multiparous mothers.

Similarly, prolongation of labour was a usual accompanying clinical feature. Thus 69 per cent of the cases where labour was more than 24 hours showed marked moulding of the skull bones. Of the 84 cases where labour was less than one day only 22 cases showed moulding (26 per cent).

Pathology: The majority of cases where moulding was present showed the occipital and frontal bones overlapped by the parietals, and generally the right parietal bone overlapped the left one.

In three of the cases there was complete separation of the dura mater along the medial aspect of one parietal bone;

in two of these cases the free edge of the medial border
of the right parietal bone overlapped the sagittal sinus
and also the left parietal bone; and in one case the left
parietal overlapped the right one. The mothers in these
cases were all primigravidae, and in two cases at least,
the child had died before the onset of labour; the third
case was one of mixed accidental haemorrhage where a foetal
heart-beat was reputed to have been heard before the onset
of labour.

Excessive moulding leads to an increase in intracranial
pressure and hence congestion of the brain tissue. The
latter feature was marked in 58 per cent of the cases show-
ing moulding.

4. Fractures of foetal skull bones.

Occurrence: There was only one case of fractured skull
in this series.

In this case, the child was delivered in the interest
of the mother, who suffered from mitral disease. The
delivery was performed under spinal anaesthesia, and a pre-
mature child was delivered by the use of forceps. The child
survived six hours.

Pathology: The fracture was of the linear type, and
extended from the centre point of the medial border of the

right parietal bone laterally for one inch. As pointed out by Ehrenfest,[23] this type of fracture is frequently due to trauma, and generally occurs in cases where the ossification is deficient.

Hemsath (36) has recently drawn attention to the separation of the posterior intra-occipital synchondrosis found on pathological examination of stillborn children, and has reported a series of 32 cases. This writer explains how excessive pressure in the sub-occipito-bregmatic diameter may cause internal displacement of the squama of the occipital bone and consequent overriding. In this way the posterior atlanto-occipital ligament becomes ruptured, and permits the squamous segment to press on the vital medullary centres of the child's brain.

This type of injury, according to Hemsath, is not infrequently discovered after forceps or breech deliveries, and is produced by the sub-occiput of the child being forced against the maternal symphysis pubis.

In a series of 166 consecutive cases Hemsath (36) demonstrated this feature in 32 cases. There were no cases of occipital osteodiastasis in this series.

5. Oedema of the foetal brain:

Occurrence: This was present in 26 cases of the series. In 22 of these the child was premature, an in-

cidence of 39 per cent of all prematures. Only 4 mature children showed this feature.

Oedema was found in cases where death had occurred during labour, and in cases dying during the antenatal period.

While oedema of the brain was not infrequently associated with intracranial congestion, it was indeed exceptional to encounter intracranial haemorrhage and cerebral oedema in the same infant.

Etiology: The exact causation of cerebral oedema in infants is difficult to understand. In 5 of the cases the children were delivered by Caesarean section, and on this account it is impossible to explain the occurrence on mechanical grounds. Shannon (56) seeks to explain it as a deficiency of foetal blood calcium. This cannot be the complete explanation, however, as 2 of the cases in this series were twin pregnancies, and the child that survived showed no abnormal clinical features.

It would seem that a degree of cerebral oedema is quite physiological in premature children, but the exact factor or factors which produce an excess are as yet unknown.

6. Congestion of brain tissue:

Occurrence: There were 62 cases in the series.

Intracerebral congestion was present irrespective of

the time of the child's death.   It was present in 12 ante-partum deaths, 39 intrapartum deaths, and 11 postpartum deaths.

The presence of congestion did not seem to bear any direct relation to the mode of delivery, or the parity of the mother.   Of the 62 cases, 36 were normal deliveries and 26 were abnormal.   There was an equal number of mature and premature babies showing this feature.

Prolongation of labour was a frequent finding in assoc-iation with brain tissue congestion.   In all 16 cases where labour was over 24 hours congestion was noted.

Pathology: In these cases the meningeal vessels were seen to be markedly congested, and on incision the brain tissue was found to be purple in colour, the degree of in-tensity varying with the congestion.   The choroid plexuses were congested, and the vessels coursing through the adjacent tissue engorged, causing a radiation of dark red streaks. This dark red, strand-like arrangement proved, on microscopic examination, to consist chiefly of grossly engorged veins, which in some instances had ruptured causing extravasation of blood into the surrounding tissues in the form of small haemorrhages.

7. Intracranial haemorrhage:

Occurrence: A vast amount of medical literature has

been published on the subject of intracranial haemorrhage
of the newborn, which represents the most serious accompani-
ment of cerebral birth trauma.

The incidence of intracranial haemorrhage varies
greatly in pathological reports.

In 1926, Ford (25) pointed out the frequent occurrence
of blood in the cerebro-spinal fluid of newborn children.
In 400 cases he found the incidence of this feature to be
10 per cent.

Warwick (66), writing in 1919, found 50 per cent of
young infants died of cerebral haemorrhage; in 1921, (67),
reporting on 200 post mortems, she found the incidence of
intracranial haemorrhage to be 44 per cent.

Browne (7), in 1921, found the incidence of intracran-
ial haemorrhage in 200 infants who had been born dead, or
who had died within the neonatal period, to be 29.5 per
cent.   Later, in 1922, he reported 53 cases of intracranial
haemorrhage in neonatal post mortems of a total of 153 cases.

Serbin (55) reported in 1928 that 19.7 per cent of a
total of 320 foetal deaths were due to intracranial haemorr-
hage.

Cruikshank (17), in 1930, found gross intracranial
haemorrhage to be the cause of death in 20.1 per cent of his
series.

In 1934 Emmerich von Haam (31), from a study of 317 foetal necropsies, recorded the incidence as 16 per cent.

Schwartz (54) found 105 of 110 cases studied, in which there were indications of cerebral haemorrhage.

Hemsath and Canavan (35) report that 64 per cent of 53 cases showed microscopic haemorrhages in the medulla oblongata.

From these pathological reports quoted above it would seem that much controversy exists regarding the significance and frequency of intracranial haemorrhage in infant post mortems.

Schwartz (54) finds intracranial haemorrhage to be present in over 95 per cent of his cases, while von Haam (31) finds intracranial haemorrhage in only 16 per cent of his series of 317 cases. These misleading statistics are readily explained by the fact that von Haam deals with macroscopic haemorrhages, as do most workers, while Hemsath (35) and Schwartz (54) refer solely to microscopic haemorrhages.

On reading the publications referred to, there was difficulty in deciding whether intra-cranial haemorrhage was in fact the cause of death in some cases. The present writer failed to find in most instances a statement which revealed the large number of cases where intracranial haemorrhage was present, but was not considered of sufficient severity to

account for the child's death.   It was also noted that
only occasional reference was made to the occurrence of
more than one type of haemorrhage in each individual case.

In the present series there were 60 cases in which
intracranial haemorrhage was actually noted;   yet it was
considered that in only 28 of these was intracranial haemorr-
hage of sufficient severity to prove fatal. Difficulty was
experienced in assessing the importance of small areas of
haemorrhage, and due regard was paid to the position and
size.   The 28 cases so earmarked were all cases in which
intracranial haemorrhage was a marked feature and quite
incompatible with life.

Mortality: Of the 28 cases of fatal intracranial
haemorrhage in this series, 25 children were born dead, and
3 died within 5 days of birth.   From these figures it will
be seen that in this present series 11 per cent of the in-
fants who suffered intracranial haemorrhage at birth, died
shortly after birth.   Capon (9) records 6 cases who died
after delivery in a series of 30 cases of intracranial
haemorrhage; the other 24 cases were stillborn.

Browne (7), however, shows a higher rate of survival
in his first series of 200 foetal necropsies; in his collec-
tion 59 cases died of intracranial haemorrhage, 24 of which
were born dead, and the large number of 35 cases died during

the neonatal period.

Classification: Various attempts have been made to classify cases of intracranial haemorrhage in accordance with the supposed predominant factors, on the assumption that such methods of grouping would prove of practical value for diagnosis and treatment.

Warwick (66) suggests the following classification:

(a) Traumatic cases: resulting from excessive cranial stress.

(b) Cases of congestion or stasis, with rupture of veins.

(c) Cases where the haemorrhage was due to a diseased condition of the child.

It will be seen that this classification mixes etiological and pathological factors, and in view of more recent knowledge is unsuitable.

Munro and Eustis (47) choose to modify Warwick's classification with the substitution of an Asphyxial group in place of the Congestive group.

Ehrenfest (23) classified the pathological conditions found in intracranial haemorrhage thus:

(a) Cephalhaematoma internum.

(b) Subarachnoid haemorrhage.

(c) Dural haemorrhage: (A) Supratentorial
                          (B) Infratentorial
                          (C) Mixed type.

(d) Brain haemorrhage: (A) Ventricular
                        (B) Diffuse or circumscribed.

Von Haam (31) with justification discards this method of grouping from the pathologic-morphologic point of view in that it does not include all pathological possibilities.

Von Haam's (31) classification which is formed on an anatomical basis adequately covers all pathological features. This classification has been utilised for the present work, with slight alterations in nomenclature, and one addition.

(A) Epidural

(B) Intradural

(C) Subdural

(D) Subarachnoidal

(E) Intracerebral: (a) Multiple and minute haemorrhages.
                    (b) Single and diffuse haemorrhages.

(F) Intraventricular.

The present writer has preferred to classify the intra-cerebral type into two separate categories according to the size of the haemorrhage. The intraventricular type has been added to the classification of von Haam (31) to ensure path-ological completeness.

In this series the 28 cases of intracranial haemorrhage are so called because a large quantity of free blood was plainly evident on dissection. This fact is re-emphasised

as it is frequently difficult on reading a pathological report to calculate in which cases more than one type of haemorrhage was present.   In this series the incidence of each type of haemorrhage will be entered separately under its own particular group; and thus it will be seen on addition of the numbers in each group the total may exceed the total number of cases examined. This is obviously explained by the fact that two or more types of haemorrhage have occurred in one case. For the complete findings in each case the reader is referred to the Appendix.

(A) Epidural Haemorrhages.

This type of haemorrhage is comparatively rare, and practically only occurs in association with some severe injury to the skull. The haemorrhage may be of arterial or venous origin. This type is referred to by Ehrenfest as cephalhaematoma internum, and he states that from a theoretical standpoint it may occur in the absence of a bony lesion. The same writer describes its formation as similar to cephalhaematoma externum in that the blood originates from the vessels of the cranial periosteum, which pass through the skull directly into the dura mater. The firm attachments of the dura mater to the skull, however, limit the extension of the haemorrhage.

In this series there were 4 cases, all of which were

associated with cranial damage, and in none of which was
the haemorrhage extensive.   One case, in which epidural
bleeding was present, was associated with a linear fracture
of the right parietal bone. The other 3 cases were assoc-
iated with complete separation of the medial aspect of the
·parietal bone from the dura mater.   In none of these cases
was the amount of haemorrhage large.

Few authorities attach much importance to this type
of haemorrhage in the newborn; so much so that most leading
authorities fail to report on its occurrence.

It is quite conceivable that this form of haemorrhage
not infrequently occurs in association with external cephal-
haematoma, as pointed out by Ehrenfest (23). If its occurr-
ence were non-fatal, as must be frequent in view of the firm
dural fixation limiting its spread, it might cause cortical
irritation in later life.

(B) Intradural Haemorrhage.

This type of haemorrhage may be found in cases of
asphyxia and, according to von Haam, in infants with a
haemorrhagic diathesis. These haemorrhages, which are
commonly multiple and of small size, are usually of little
clinical significance. There are two situations, however,
where these haemorrhages may be of larger size and endanger
the life of the child, viz: (a) when they occur between the

folds of the falx cerebri, and (b) when they occur between the blades of the tentorium cerebelli.

(a) Haemorrhage of the falx cerebri: Small haemorrhages are frequently noted between the two dural folds of the falx cerebri. They occur chiefly at the broader basal portion and are usually to be found adjacent to the superior sagittal and straight sinuses. It is not unusual to find these haemorrhages associated with lacerations of the falx.

In this series there were 42 cases of this type of intradural bleeding, but, with the exception of 3 cases, the haemorrhage was of small size.

In one case the child was born after Caesarean section.

In 12 of these cases there was tearing of the falx cerebri.

It was noted that while the inferior portion of the falx beside the inferior sagittal sinus was more liable to lacerations, the superior portion was more liable to intradural haemorrhage. This is explained on purely anatomical grounds as the inferior portion is thinner and more firmly anchored, and thus tears on being strained; whereas the blades in the superior portion are of stronger consistence but yet more easily separated, which permits blood to seep

between the two folds.

(b) Haemorrhages between the tentorial blades: Intra-tentorial haemorrhage occurred in 19 cases of this series, but was severe in only one case. It was associated with tearing of the tentorium cerebelli in 4 cases. In 2 of these 4 cases the tearing was confined to the upper blade, and in 2 cases the tearing was completely through both blades.

In the one severe case of intratentorial haemorrhage the child was mature and born spontaneously. In the 18 slight cases 13 were premature infants and 12 were born spontaneously. Less than half the cases were first chil-dren. These findings suggest that birth trauma does not play such a vital role in the production of the intra-tentorial type of haemorrhage.

(C) Subdural Haemorrhages.

Occurrence: This type of intracranial haemorrhage was present in 40 cases.

According to their location this type is subdivided into 3 distinct varieties:

     (1) Supratentorial haemorrhages.
     (2) Subtentorial Haemorrhages.
     (3) Mixed type including haemorrhages in
         the subdural space over the cerebral
         convexities.

(1) Supratentorial haemorrhages: There were 26 cases

in which clotted blood was present on the superior surface
of the upper blade of the tentorium. In 15 of these cases,
the haemorrhage was considered to be of sufficient severity
to hasten the death of the infant; in the remaining 13
cases it was probably of slight significance.

Severe supratentorial haemorrhage: These cases are
usually associated with dural tearing. Of the 15 cases,
12 had accompanying tentorial tears. Only 2 of the cases
were prematurely born infants.

In most cases it was extremely difficult to ascertain
from which vein the haemorrhage had issued. Where the
supratentorial haemorrhage was associated with tearing of
the tentorium it was frequently found that the straight
sinus had been opened, and that bleeding originated at
this point. In some cases where no dural damage was evident
the vena magna was found to have been torn across, and to
be the source of the bleeding. In 5 of these cases there
was gross subtentorial haemorrhage accompanying the supra-
tentorial haemorrhage. These cases showed adjacent lacer-
ations of the tentorium cerebelli.

Slight supratentorial haemorrhage: These cases were
usually accompanied by laceration of the tentorium. Of
the 11 cases, 10 had adjacent tears of the tentorium cere-
belli. Similarly the incidence of mature infants showing

this feature was very high, 9 of the 11 cases were over 2.5 kg. at birth.

In the total of 26 cases where supratentorial haemorrhage was present, it is of importance to stress the fact that only 5 cases were spontaneous vertex deliveries.

(2) Subtentorial Haemorrhages: All writers are agreed on the vital importance of haemorrhage occurring in this critical position. Most authorities ascribe this importance to the fact that such haemorrhage depresses the respiratory centre by a congestive stasis. Other workers uphold the original theory of Beneke (4) "that the medulla becomes wedged into the foramen magnum" by the pressure of the haemorrhage under the tentorium. The modern trend of thought is to associate in the mind the congestion of the medulla under such circumstances with the presence of minute petechial haemorrhages in its substance. Hemsath and Canavan (35) report 34 cases in their series of 53 in which death was due to microscopic haemorrhages in the medulla oblongata.

In this present series there were 32 cases in which some free blood was found beneath the tentorium cerebelli. The amount of haemorrhage was severe in 8 cases and of little importance in 24 cases.

Severe subtentorial haemorrhage: All 8 cases were

associated with tearing of the tentorium. Six of the
children were mature and only 2 premature. Two of the
cases were spontaneous vertex deliveries.

Slight subtentorial haemorrhage: There were 24 cases
where slight haemorrhage was present beneath the tentorium
cerebelli. In 17 of the cases the tentorium was torn,
and in 16 delivery was abnormal. In 13 cases the infant
was mature and in 11 cases premature. In one case the
medulla was surrounded by clotted blood and wedged into
the foramen magnum.

Congestion of the medulla and cerebellum was fre-
quently present in cases of subtentorial haemorrhage. In
25 cases of the total 32 the medulla and cerebellum on
section were purple in colour. Thus 72 per cent of cases
of subtentorial haemorrhage show congestion of the medulla
and cerebellum.

(3) Mixed type of subdural Haemorrhage: This group
includes all types of haemorrhage into the subdural space
not previously described in relation to the tentorium cere-
belli. For the most part this refers to haemorrhages over
the surface of the hemispheres outside the leptomeninges.

In this series there were 25 cases of subdural haemorr-
hage over the convexity of the brain; in 21 cases of this
total, haemorrhage was also present around the tentorium

T A B L E I – SUBDURAL AND INTRADURAL HAEMORRHAGE.

TYPE OF HAEM.	No. of cases showing type of haem.	No. of cases with associated tentorial tearing.	MODE OF DELIVERY		MATURITY		PARITY OF MOTHER		
			Normal	Abnormal	Premature	Mature	Primip.	Multip.	
Supra-tentorial	26※	22	5	21	4	22	11	15	} Subdural Haemorrhage 40 cases.
Sub-tentorial	32※	25	10	22	13	19	13	19	
Mixed type	25※	19	7	18	8	17	9	16	
Into the tentorum	19※	4	13	6	13	6	9	10	} Intradural Haemorrhage 45 cases.
Into the falx	42※	12	21	21	21	21	20	22	

※ The number of cases in the first column is in excess of the total number of subdural and intradural cases in the series by the number of individual cases showing more than one type of haemorrhage.

cerebelli. In 19 cases tearing of the tentorium was also present. Of the total 25 cases only 7 cases were delivered normally. Seventeen of the infants were mature while 8 were prematurely born.

This type of haemorrhage may occur through injury to the superior sagittal sinus, although this was an infrequent finding. Most cases of this type were secondary to haemorrhage in the region of the tentorium, which had spread over the surface of the cerebri. These haemorrhages may also be caused by excessive compression of the temporal bones which so increases the distance between the dura mater and the leptomeninges over the vault of the skull that the subdural veins are stretched and finally torn across. Hence the frequent association of this type of haemorrhage in cases of large children and instrumental deliveries. It will also be noted that most cases were accompanied by tearing of the tentorium cerebelli, an indication of the traumatic origin of subdural haemorrhage.

Examination of Table I reveals the following important facts:

(i) In most cases (80 per cent) of subdural haemorrhage there is associated subtentorial bleeding.

(ii) Subdural haemorrhage is frequently associated with tearing of the tentorium cerebelli.

(iii) Mature infants and those who are not born in the normal manner are especially prone to suffer subdural haemorrhage.

(iv) Intradural haemorrhage occurs in 45 per cent of the cases and is not commonly associated with tentorial tearing.

(v) The frequent presence of bleeding into the falx would not appear to be influenced by the maturity of the child or the delivery and parity of the mother.

(vi) Intratentorial haemorrhage would seem to be more common in premature infants.

(D) Subarachnoid Haemorrhage.

This type of haemorrhage was present in 12 cases of the series. Of all the cases which showed evidence of intracranial bleeding on post-mortem examination, the incidence was 19 per cent.

Ehrenfest (21) considers this type of bleeding to be common, and reports its presence in 24 per cent of his cases; he calls attention to the association of instrumental and breech deliveries and this finding.

Von Haam (31) stresses the fact that a history of syphilis or haemorrhagic diathesis is a common feature in premature children.

Craig (13), who found 29 of his 36 cases of subarach-

noid haemorrhage were premature, helped to establish the importance of prematurity as an etiological factor.

In this series 9 of the 12 cases occurred in premature infants.

Abnormal delivery was present in 9 of the cases; 3 cases were extracted with forceps, and 5 were breech deliveries; the remaining child was delivered before the commencement of labour by Caesarean section.

Half of the cases were associated with tearing of the tentorium cerebelli, and in one-third of the cases the child was the first-born.

The relation of asphyxia to intracranial meningeal oozing of blood was described in detail by Cruickshank (17) in 1930. He states that meningeal capillary oozing is common, and generally associated with asphyxia. He also maintains that it is not an uncommon finding in premature infants, where it is characteristically associated with oedema.

In this group of 12 cases of subarachnoid haemorrhage 9 cases showed congestion of the brain tissue. It would seem that small meningeal oozing of blood may be produced in a similar manner to the serosal haemorrhages found in other parts of the body in cases dying slowly in a state of passive congestion.

These leptomeningeal haemorrhages were of small size and generally situated on the convexity of the brain adjacent to the midline. They were occasionally bilateral (2 cases), but in these two cases were more pronounced on one side.

<div align="center">(E) Intracerebral Haemorrhage.</div>

Two types will be described, (a),minute haemorrhages; and (b) single and diffuse haemorrhages.

(a) Minute multiple haemorrhages. This type of haemorrhage was present in 56 per cent of cases in the series. This high incidence is in agreement with the finding of Schwartz (54), Hemsath and Canavan (35).

Schwartz (54) in his original paper on the subject states: "Small haemorrhages or distinct areas of degeneration in the substance of the brain apart from the fairly common pial and tentorial haemorrhages can even macroscopically be recognised in 65 per cent of infants up to the age of five months. The arrangement and position leave no doubt concerning their origin. Their distribution clearly expresses cerebral trauma by the mechanism of labour."

Hemsath and Canavan (35) found microscopic haemorrhages to be present in 64 per cent of their cases, and are in agreement with Schwartz on their frequency and etiology. They did not find minute multiple haemorrhages in any of

the cases which were breech extractions.

Of the 56 cases observed in this work 33 cases were normal vertex deliveries. However, in controversy with the opinion of Hemsath and Canavan (35) this type of haemorrhage was present in 12 cases of breech delivery. Ten of the cases were instrumental deliveries. In 28 of the cases there was other evidence of cerebral trauma, and 24 of the infants were first children. The children were prematurely born in 30 cases, and mature in 26; these results agree with the reports of other workers.

Cruickshank (17) expresses doubt as to the supposed frequency of minute intracerebral haemorrhages and considers most of the petechial haemorrhages that occur during birth are due to increased intravascular pressure.

Schwartz (54) disagrees with this view and explains their origin as follows: "During labour, so long as the membranes are intact, any increase of intrauterine pressure caused by uterine contraction will, according to the law of hydraulics, be equally distributed. The rupture of the membranes however, brings about a significant change. The presenting part is now subjected to a much reduced pressure, i.e. atmospheric pressure. Hence under the influence of this negative pressure or suction the body fluids of the foetus are drawn towards the presenting part and the caput

succedaneum is produced.   In the adjacent region within
the skull the veins in the meninges, brain and venous
sinuses become overfilled and eventually might rupture
when the mechanical factor of the moulding process is super-
added."   In this way, Schwartz (54) considers small multi-
ple haemorrhages are found in the brain tissue.   He is also
of the opinion that the blood stasis so induced by this
suction force may interfere with the proper nutrition of
the brain cells to such an extent as to cause degeneration
processes.

An examination of the results in this series forces
the conclusion that trauma and asphyxia are both responsible
for these haemorrhages, for the following reasons:

If their occurrence was produced purely on the mechan-
ical grounds of suction, then the finding would be absent
in breech deliveries. However, in fact, 12 breech deliver-
ies of a total of 23 such deliveries in the series showed
this feature.

If such haemorrhages were produced purely by asphyxia
it would be reasonable to expect that in all cases showing
petechial haemorrhages the brain tissue would be congested.
However, in 12 of the 56 cases showing multiple small
haemorrhages the surrounding brain tissue was not congested.

Where neither asphyxiation nor traumatization were
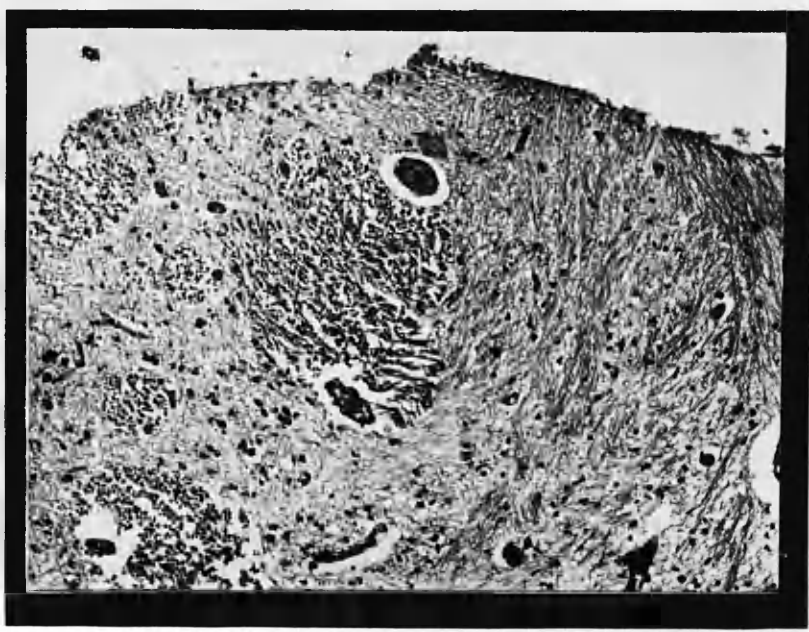
The microphotograph shows congestion of the cerebral cortex with several intensely engorged vessels. Adjacent to these vessels are seen areas of degeneration which probably developed immediately before death as there is little evidence of phagocytic reaction.

present in the 7 cases delivered by Caesarean section, no cases of multiple minute haemorrhage were present.

That these haemorrhages may be caused by both trauma and asphyxia is strongly suggested by their localization.

These haemorrhages are found to be more numerous in two main sites; adjacent to the ventricles, and in the cortex. Those found in the region of the ventricles are probably caused by the local asphyxial congestion; whereas the haemorrhages in the white substance in the region of the cortex are more likely to have a traumatic origin.

Small areas of fatty degeneration as demonstrated by Schwartz were also occasionally observed, but the incidence of this occurrence was not recorded (see microphotograph on page 36a).

(b) <u>Single and diffuse haemorrhages</u>. There were no cases of large intracerebral haemorrhage in this series. While most authors agree on the rarity of this type of haemorrhage on anatomical grounds it seems reasonable that the soft infant brain tissue would permit of such haemorrhages. It is probable that the delicacy of the vessels and the predominance of venous haemorrhages do not allow such a phenomenon to occur frequently.

Von Haam (30) reports 4 cases in a series of 317 post mortems.

Craig (12) observed 6 cases in a series of 126 cases of intracranial haemorrhage.

(F) Intraventricular Haemorrhage.

In this series there were no cases of intraventricular haemorrhage. The absence of this type of haemorrhage is not in agreement with the majority of pathological reports.

Warwick (67) reports 2 cases in a series of 53 cases of intracranial haemorrhage.

Browne (7) found the incidence of intraventricular haemorrhage to be 8.5 per cent in a series of 200 post mortems.

Craig (13) found this type to be present in 22 of his 126 cases of intracranial haemorrhage.

The latter, in agreement with Cruickshank (17) and Browne (7), is of the opinion that this type is commoner in premature children and frequently occurs in cases where the mother has been ill during the pregnancy.

8. Injuries to the Dural Septa:

Occurrence: Injuries to the dural septa represent the most concrete evidence of cerebral trauma at the time of birth. According to Ehrenfest [23] these lesions are clearly traumatic in origin and are found in approximately half of all cases. Injury to the dural septa results in tearing

and this damage is seen in the tentorium cerebelli and falx cerebri.

In this series there were 40 cases in which the tentorium cerebelli was torn, and 20 cases in which damage to the falx cerebri was present. The total number of cases in the 100 necropsies which showed injury to the folds in either or both sites was 47. From these figures it will be realised that almost half the cases of this series showed pathological evidence of trauma. This high incidence of dural laceration is in keeping with the findings of other writers.

Holland (38), in 1922, from a study of 167 cases found tearing of the tentorium cerebelli to be present in 81 cases (i.e. 48 per cent).

Capon (9) records tearing of the tentorium in 25 per cent of cases dying within the neonatal period and in 59 per cent of stillborn children.

Browne (7) observed this feature in 37 per cent of his cases.

Hemsath and Canavan (35) in a series of cases dealing with another subject mention that in 53 foetal post mortems 14 cases showed tentorial tears, an incidence of 26 per cent.

Few writers refer to the incidence of tearing of the

falx cerebri; Browne (7) noted the fact in 3 of 94 cases,
and Holland (38) reports tearing of the falx cerebri in
5 of 167 cases, i.e. 3 per cent.

Laceration of falx cerebri: In the 20 cases of tear-
ing of the falx cerebri only 7 cases occurred without the
associated presence of tentorial tearing. While it is not
suggested that damage to the falx in the absence of hae-
morrhage produces a fatal issue, it must be noted that
such tearing is evidence of excessive cranial stress, and
from this aspect is of equal importance to other dural
lacerations. In spite of numerous articles on cerebral
trauma few authors seem to have attached any significance
to this rather frequent finding. Since the majority of
these cases will be included in the cases of tearing of the
tentorium it is unnecessary to enter into their statistics
separately.

The damage in the majority of these cases consisted
of small oval perforations of the falx along its free
border, but not involving the inferior sagittal sinus.
These injuries were generally complete perforations through
both layers of the falx and were larger towards the base.
In 4 cases the inferior sagittal sinus had been damaged
with a severe laceration along the free border of the falx
extending from the middle to the base, where it coincided

with tentorial tearing.   In 1 case a large perforation was present at the base of the falx which did not damage the inferior sagittal sinus, but involved the straight sinus;   a similar type of case was described by Ehrenfest (23).

Tearing of the Tentorium Cerebelli: Tentorial tears were present in 40 per cent of the cases.   The appearance of the tears was similar to the original description by Beneke (4) in 1910.   In the majority of cases the tear commenced at a point joining the medial and middle thirds of the free edge of the tentorium and extended in a posterior direction, occasionally penetrating the straight sinus. Only in one of these cases was the free border of the tentorium intact, and in this case the tear was in the form of a complete perforation just posterior to the usual site of damage.

In approximately half the cases only the upper layer of the tentorium was damaged;   and in these cases the cerebellum could be seen shining through the intact thinner lower leaf when viewed from above.   In spite of the finer texture of the lower half of the tentorium this portion is not subjected to so much strain as the upper half.

These tears may be found on either side of the falx cerebri or quite commonly on both sides.   Thus the tears

are referred to as complete or incomplete, according to
whether both layers of the tentorium are involved or not,
and right and left-sided according to the side damaged.

TABLE II.

ANALYSIS OF THE POSITIONS OF TENTORIAL TEARING.

Right Side	Left Side.		
Complete	Complete	= 11)	Cases with com-
Complete	No tear	= 1)	plete tear - 14.
No tear	Complete	= 2)	
Incomplete	Incomplete	= 6)	Cases with In-
No tear	Incomplete	= 5)	complete tears - 15.
Incomplete	No tear	= 4)	
Incomplete	Complete	= 4)	Mixed cases with
Complete	Incomplete	= 7)	Complete and In-
			complete tears - 11.

Total 40 Cases

Bilateral tearing in 28 cases: unilateral in
12 cases.

Browne (22) records 21 cases of Complete and 18 cases of
Incomplete in his series of 35 cases, but does not reveal
into which groups the mixed groups have been included.

Holland (38) found 64 of his cases showed bilateral
and 17 unilateral tearing of the tentorium.

Several American writers have referred to the work of
Benthin who demonstrated an association between the inci-
dence of tearing of the tentorium on the right side and
the first vertex position. This worker was of the opinion

that the stronger pressure against the left parietal bone
and the resulting flattening in left occipito-anterior
presentations would cause a dent in the right parietal
bone with corresponding increased tension on the falx
fibres running over to the right.

The results of the present investigation do not
support this finding.   In fact, it was found that in the
majority of first vertex presentations the tearing was
present on the left side, and in cases showing bilateral
damage the more severe laceration was also frequently
present on the left side.   The positions of these cases
were ascertained by the overlap of the parietal bone found
at necropsy, and not on clinical findings.

The frequent occurrence of dural lacerations in first
born children is universally accepted.   In this series
from a total of 44 first born children 21 suffered tentorial
damage, i.e. an incidence of 48 per cent.

Instrumental and breech deliveries are commonly a
feature in cases showing tentorial tears.   Twelve of the
40 cases in this collection were spontaneous deliveries,
and 28 were abnormal.

The 40 cases were delivered as follows:

Spontaneous Vertex .... 12 cases

Forceps Delivery: high ..... 5)
                   mid .... 5) .... 15 cases
                   low ...... 5)

Breech Delivery: spontaneous ... 2)
                  manual .... 11) ..... 13 cases

(a) <u>Normal Delivery</u>.

In these infants complete tears were rarely found;
the majority of these cases had unilateral incomplete tears.
It is concluded therefore that in this small number of cases
from a total of 50 normal cases in the series, when tearing
does occur following a spontaneous vertex delivery it is
rarely of severe type. In the 7 infants delivered by
Caesarean section no tentorial tearing was present.

(b) <u>Abnormal Cases</u>.

(i) Instrumental deliveries: In the complete series
of 100 cases 20 infants were delivered by the aid of for-
ceps; and of these, 20 cases tentorial tears were present
in 15 infants. These figures indicate that 75 per cent
of the instrumentally delivered children who succumb have
tentorial tears. Only 2 of these cases survived birth,
and the maximum survival period was $1\frac{1}{2}$ days. Contrary to
expectation it was found that of the instrumental delivery
the most serious damage was caused by the low application
of forceps. In this group 4 of the 5 low forceps cases

had complete tears, but it must be noted that 3 of these cases were first children and in the other 2 cases there was prolongation of labour.

(ii) Breech deliveries: Tentorial tears were present in 13 cases of breech delivery from a total of 23 such deliveries in the series. Only 2 of these cases were spontaneous; the other 11 were manual breech extractions. The tearing was of severe degree in 8 of these 13 cases, all of which 8 were delivered manually.

Most writers agree that damage to the tentorium is more probable in breech than in spontaneous deliveries. In this series the incidence of tentorial tears in breech deliveries was 57 per cent, and the incidence in spontaneous vertex cases was 24 per cent. Thus tentorial tears are over twice as common in breech cases. Holland (39) found the incidence of tentorial tears in foetus following breech extraction to be as high as 88 per cent.

The length of labour in cases showing tentorial tears was found to be rather longer than the normal duration.

TABLE III.

ASSOCIATION OF LENGTH OF LABOUR WITH TEARING OF TENTORIUM.

HOURS IN LABOUR	0-6	6-12	12-18	18-24	24-48	48 & over	Total No.
Number of Primigravidae	3	1	4	3	6	4	21
Number of Multiparae.	2	5	3	5	2	2	19

In the 21 primigravida cases the total length of labour was over 24 hours in 48 per cent of cases, and over 48 hours in 19 per cent of cases; in 62 per cent of cases the duration of labour was between 12 and 48 hours.

In the multiparous cases the duration of labour exceeded 12 hours in 63 per cent of cases. In 79 per cent of the cases the duration was within 24 hours.

In the majority (70 per cent) of these cases the child died during parturition. Seven of the cases showing tearing died before the onset of labour, the laceration having presumably been produced after the death of the child. Only 5 cases with tearing of the tentorium cerebelli survived birth and of these the maximum duration of life was 36 hours.

Only 2 cases were not associated with some degree of

intracranial haemorrhage. Fatal cerebral haemorrhage was present in 70 per cent of the cases of torn tentorium.

There were tentorial tears in 27 mature and 12 premature children. The incidence in mature infants is therefore 61 per cent as opposed to an incidence of 21 per cent in premature children.

It must be pointed out however that the majority of the so-called mature infants were really post-mature. Of the 25 post-mature infants in the series of 100 cases, 18 had tentorial tears.

Incidence in prematures .... 21 per cent

Incidence in matures .... 47 per cent

Incidence in postmatures .... 72 per cent.

9. Injury to the Spinal Cord:

There are two main types of damage which the infant's spine may sustain at birth. Firstly, the primary type which is usually caused by forcible traction on the spine resulting in dural tearing, haemorrhage and subsequent compression of the cord. The second main type includes injuries which are secondary to vertebral damage causing compression of the spinal cord.

The flexible nature of the foetal spine helps to protect it from injury. The spinal cord, however, does not have this protection, as it is anchored above by the

brachial plexus and below by the cauda equina, and is
therefore a fixed structure enclosed in a tube which is
capable of slight expansion. Thus excessive elongation
of the spinal canal by over-traction will result in in-
tolerable strain and possibly tearing of the delicate
spinal vessels.

Bronson Crothers (14) considers "there is every
reason to suppose that pathologic statistics underestimate
the frequency of cord injuries." He maintains that in
breech extractions injury to the cord is an important
cause of foetal death and of paraplegia in childhood.

Pathological reports on spinal cord damage in the
infant are rare. This can probably be accounted for by
the intricacy of the dissection necessary to expose the
membranes and cord in position and intact.

In this series the cord was not dissected from the
spinal canal, but withdrawn through the foramen magnum.
For this reason no results are available regarding the
condition of the surrounding membranes.

There were 18 cases in which the cord was able to be
withdrawn intact and a proper examination undertaken.
Fifteen of these cases had previously been fixed in
formalin solution. No abnormality was detected macroscop-
ically except in one case in which the following were the

The photographs show multiple scattered areas of haemorrhage in the Spinal Cord.

Fourth Cervical Segment

Medulla Oblongata.
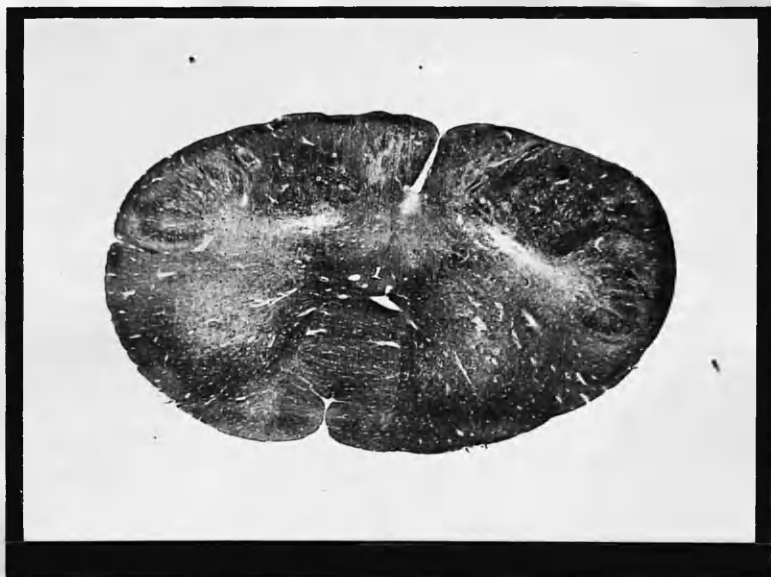
findings:

Case 51: On withdrawing the spinal cord from the canal an area extending downward for a distance of 1.4 cm. from the medulla was found to be stained bright red in colour.

Microscopic examination revealed small areas of haemorrhage scattered throughout the grey matter of the medulla oblongata and the cervical spine. No ruptured vessel or line of blood flow could be detected and the small haemorrhages were therefore presumed to have originated from the capillaries of the grey matter.

The case was one of face presentation which had been converted into a vertex by Thorne's manoeuvre and extracted with forceps applied in the mid-pelvis. The damage might have been caused by over-enthusiastic flexion of the foetal head with consequent spinal capillary damage. The child was stillborn.

Whereas the medulla is fully developed at birth, the spine and the rest of the brain are not. It is reasonable to suppose therefore that injury to the cord at birth could be quite considerable without causing any immediate clinical manifestations.

No conclusion can be expressed regarding the frequency of spinal cord injury from the examination of one case. It is of interest to note, however, that of the 18 cases examined, 6 cases in which no abnormality was detected were manual breech deliveries. Of the other cases 9 were instrumental and 3 spontaneous births.

CHAPTER   III.

CONCLUSIONS FROM PATHOLOGY.

The results of this investigation suggest that the presence of intracranial haemorrhage is probable, but not positive, evidence of cerebral birth trauma. Two cases in this series, which were born by Caesarean section before the onset of labour, suffered slight intracranial haemorrhage. In one case the haemorrhage was subarachnoidal, and in the other between the blades of the falx cerebri.

Tearing of the dural folds, however, is positive evidence of excessive intracranial stress, and was present in 47 per cent of the cases.

Cerebral birth trauma may occur in spontaneous normal deliveries, but is more frequently produced by instrumental and breech deliveries. In this series the incidence was 24 per cent in normal cases; 57 per cent in breech cases; and 75 per cent in instrumentally delivered cases.

The children of primigravidae are more liable to receive cerebral trauma during birth than the children of multiparae. In this series 48 per cent of firstborn children suffered cerebral trauma, whereas the incidence in children of multiparae was 34 per cent.

The results of this pathological investigation stress the liability of post-mature children to suffer cerebral trauma at birth. The incidence here was 72 per cent.

The length of labour bears a direct relationship to the production of cerebral birth trauma, which occurs more frequently in prolonged labours. In 62 per cent of the primigravidae investigated here labour lasted longer than 18 hours; and in 63 per cent of the multiparae labour lasted longer than 12 hours.

PART II.

CHAPTER IV.

PLAN FOR EXAMINATION OF CHILDREN.

CHAPTER V.

EXAMINATION OF CHILDREN.

SECTION I: EXAMINATION OF CASES KNOWN TO HAVE
SUFFERED CEREBRAL BIRTH TRAUMA.

Group I. (a) Convulsion Cases.
(b) Intra-cranial Haemorrhage Cases.

Group II. Cases subjected to Birth Trauma.

SECTION II: REVERSE CHECK ON KNOWN FEEBLE-MINDED
CHILDREN.

CHAPTER VI.

CONCLUSIONS DRAWN FROM THE EXAMINATION OF CHILDREN
AND BIRTH RECORDS.

P A R T   II.

CHAPTER   IV.

PLAN FOR EXAMINATION OF CHILDREN.

This investigation consisted of two sections:

I. The mental and physical examination of a group of children who were born in this hospital, and whose births were associated with dystocia, complete records of which were present in the hospital journals.

II. The examination of the birth records of another group of children who were attending special schools on account of known mental deficiency.

SECTION I: THE EXAMINATION OF CHILDREN KNOWN TO
HAVE SUFFERED CEREBRAL BIRTH TRAUMA.

Two separate groups of children were mustered for examination.

Group I. This group consisted of children of four years of age who had been treated in the Sick Nurseries after birth on account of known intracranial haemorrhage, frequent cyanotic attacks, or repeated convulsions.

Group II. This group consisted of children of nine years of age, most of whom had suffered trauma at birth.

Group I: Sick Nursery Children.

This category consisted of 8 children who were born

in 1935. Four of these children had suffered repeated convulsions before dismissal from hospital, and four were known to have had intracranial haemorrhage after birth.

The examination of the children was performed in the following manner:-

History of Child's Health from Mother.

The mother was questioned regarding the child's general health with special reference to the following points:

Previous accidents or illnesses.

Age of teething, walking and talking.

General behaviour and sociability.

Undue restlessness, convulsions or other signs of instability.

Physical Examination.

Firstly the general appearance of the child was noted. Particular attention was paid to the facial expression, the size and shape of the head, the general development in relation to the age, and the presence of any tics or tremors. The strength of the child's limbs were roughly estimated and the superficial and deep reflexes tested.

Mental Examination.

In children of this age it was considered of more importance to study the child's general attitude and interest in surroundings, than to rely on set mental tests. Each

child, however, was examined in the method laid down in the Stanford Revision of the Binet-Simon Measurement of Intelligence, in order that a uniform standard of mental ability could be recorded.

Group II.

This category comprised children of nine years of age, of whom the birth records were available. In all, 200 cases were written for, but only 40 presented themselves for examination. The physical examination and history recording were similar to the cases in Group I, but a more detailed examination was undertaken to assess the children's mental development.

Mental Examination.

In most cases the children had been absent from school for six months prior to the examination on account of the National Evacuation Scheme. In a few cases partial attendance at school had recommenced and the children were attending for two mornings per week or for one hour daily.

It was therefore considered unfair to subject these handicapped children to mental tests which only gauged their scholastic attainments, and thus special tests of common sense were devised. The standard of mental development was assessed by the results from:

(i) Serial intelligence tests standardised to age, (Binet-Simon).

(ii) Common sense tests of general knowledge.

Further details of the above mental tests are to be seen on pages 65, 66 and 67.

CHAP. IV.

SECTION II: THE REVERSE CHECK ON KNOWN FEEBLE-MINDED CHILDREN.

By the courtesy of the Department of Health for Scotland, access was obtained to the roll of children in the City of Glasgow who were attending special schools by reason of their feeble-mindedness.

In all 250 case records belonging to the Educational Department were examined to ascertain the birth records of these children. But, since it was considered inadequate to rely on the maternal statement noted on these case sheets, only the children out of these 250 who had been born in the Glasgow Royal Maternity and Women's Hospital were used.

The Hospital journals of 1924, 1925 and 1926 were consulted and where the name, sex, birthday and religion of the maternity records coincided exactly with that of the feeble-minded child it was considered justifiable to presume that the birth record was that of the feeble-minded child under consideration. From the 250 records investigated, in 43 cases the exact birth records were available.

By the study of these records a reverse check was obtained on the results of the first section of this chapter.

- CHAPTER V.

EXAMINATION OF CHILDREN.

SECTION I: EXAMINATION OF CASES KNOWN TO HAVE SUFFERED CEREBRAL BIRTH TRAUMA.

Group I.

This first group contains eight children of four years of age, who had been delivered in this hospital and had suffered from convulsions or intracranial haemorrhage after birth.

These eight cases are divided into two sections, (A) The four cases who had suffered convulsions during the neonatal period, and (B) the four who had shown evidence of intracranial bleeding. The report of the examination of these cases will be modelled as follows:

Birth Record.

Sick Nursery Record.

History of Child's Health (from Mother).

Physical Examination.

Mental Examination.

(A) Convulsion Cases.

Birth record of these cases. The following table shows the birth record of each case.

TABLE LV.

CASE	SEX	WEIGHT	MATURITY	PARITY	LABOUR	DELIVERY
D.	M	7 lb	Mature	9th	26½hrs.	Spon.Vert.
C.	M	8 lb	Mature	1st	3 days	L.U.Caesar. section.
R.	M	9⅛lb	Mature	1st	2 days	Spon.Vert.
B.	M	8⅛lb	Mature	2nd	13 hrs	Spon.Vert.

From the above table the following facts are revealed:

Labour. The average duration of labour in these four cases was forty hours.

Weight. The average weight of the children at birth was 8½ lbs.

Delivery. One child was delivered by Caesarean section, and the other three were spontaneous vertex deliveries.

Parity. The mothers of two of the cases were primigravidae.

Sick Nursery Records. All four cases had been admitted to the Sick Nursery on account of convulsions or repeated twitchings, which had developed immediately after birth. Lumbar puncture was performed in each case, and the cerebro-spinal fluid was found to be faintly yellow in colour and under normal pressure. On dismissal all

four cases were apparently quite normal, and no case was older than ten days.

History of Child's Health from Mother. In none of these cases was there any history suggesting retardation of development or illness during infancy. There was no history suggestive of epilepsy. In each case the mother was satisfied with her child's progress in every respect.

Physical Examination. No abnormality noted in any case.

Mental Examination. The following intelligence tests were used in combination with the general attitude of the child in assessing the mental development. The child was asked:-

(i) To point to four different parts of its body, e.g. nose, mouth, hand and ear.

(ii) To name the following familiar objects: a key, penny, knife, watch and pencil.

(iii) To name any three objects seen in a simple picture.

(iv) What sex it was - boy or girl?

(v) What it's name was.

(vi) To repeat three digits.

(vii) To compare the lengths of two horizontal lines, and say which was the longer.

(viii) To discriminate colours.

The children all performed these tests in a satisfac-

tory manner with the exception of the last test. According to normal standards a child should be capable of differentiating blue from green at the age of three years. These colours were frequently confused. It was also noted that quite frequently the child, instead of mentioning the colour itself, stated the name of an object having this colour.

The standard of mental development in all four children was within normal limits -

Case D. Actual age 4.7/12yrs. Mental age 4.8/12yrs.

Case C. Actual age 4.7/12yrs. Mental age 4.6/12yrs.

Case R. Actual age 4.1/12yrs. Mental age 4.1/12yrs.

Case B. Actual age 3.8/12yrs. Mental age 3.9/12yrs.

(B) Intracranial Haemorrhage Cases.

In this group each of the four cases will be described independently.

(1) CASE O.

Birth Record. During the pregnancy the mother suffered from pre-eclamptic toxaemia and hyperpiesis; the Wasserman reaction was negative. This was the mother's seventh pregnancy and the child was the first of twins. Medical induction was performed and the mother was given Morphine gr.$\frac{1}{4}$, $3\frac{3}{4}$ hours before the child was delivered. The dura-

ation of labour was 13½ hours; and the child was a normal
spontaneous vertex delivery, weighing 6 lbs. 14 ounces.
The child was deeply cyanosed at birth.

Sick Nursery Record. The child was admitted to the
Sick Nursery immediately after birth and kept in charge
for ten days. Generalised rigidity of the child's limbs
was a notable feature. The anterior fontanelle was tense
and the child suffered repeated convulsions. Lumbar
puncture revealed bloodstained cerebro-spinal fluid.

History of the child's health from mother. The
mother, who was an intelligent woman, volunteered the in-
formation that the child had always been very backward.
She was 18 months old before she sat up, and was over 2
years of age before she attempted to speak. The child had
not suffered from convulsions or fits. The twin sister
of the child is apparently normal and at school. No
familial history of idiocy was admitted.

Physical Examination. The child was found to be
poorly developed and quite incapable of standing unsupport-
ed. Both lower limbs and the right upper limb were
spastic and paralysed; the tendon reflexes were exagger-
ated.

Mental Examination. The child was most inattentive
and uncooperative. She showed little interest in her

surroundings. She was quite incapable of answering the test questions, and on being questioned either grinned idiotically or babbled indistinctly a few incoherent words of the question she had been asked.

Actual age, 4⅛ yrs.   Mental age, Nil.

CASE G.

Birth record. The child weighed 6¾ lbs. at birth, labour lasted 2⅛ hours, and the child was extracted with forceps applied in the mid-pelvis. He was a third child and easily resuscitated.

Sick Nursery record. The child remained in the Sick Nursery for eight days after birth. The anterior fontanelle was full and "boggy" and the child had a cerebral type of cry. Ophthalmic examination revealed numerous retinal haemorrhages. The cerebro-spinal fluid was uniformly bloodstained and under increased pressure. The child seemed normal on dismissal.

History of the child's health from mother. Development and progress had been satisfactory. There was no history of fits.

Physical Examination. General development normal. The only abnormality noted was a partial paralysis of the right eyelid, but the eyesight was good.

Mental Examination. The intelligence was above normality. Mental age, 4.10/12 yrs. Actual age, 4.1/12 yrs.

CASE F.

Birth record. The mother was in labour for two days and received medical induction. The child was extracted with forceps as a persistent occipito-posterior. The child was the mother's third and was mature at birth.

Sick Nursery record. The child was feeble at birth and remained in the Sick Nursery for eight days. The anterior fontanelle was tense and bulging, and the C.S.F. was bloodstained. The child had occasional twitchings, but appeared to be normal on dismissal.

History of child's health from mother. The child walked at one year and spoke at two years. Apart from requiring to wear glasses, the mother considered she had made good progress. No illnesses or convulsions during infancy.

Physical Examination. The child was considered to be of good development.

Mental Examination. The child was of normal intelligence. Mental age, 4.10/12 yrs. Actual age, 4.9/12yrs.

CASE S.

Birth record. Not available.

Sick Nursery record. The infant remained in the Sick Nursery for eight days. During this time she suffered from occasional fits and the anterior fontanelle was tense. Lumbar punctures revealed the cerebro-spinal fluid to be bloodstained.

History of child's health from mother. The child's progress was satisfactory; she had not suffered from convulsions.

Physical Examination. The child was of normal development, but suffered an internal squint of the left eye, and a spastic paralysis of the right upper limb.

Mental Examination. The child was extremely "spoiled" but mentally normal. Mental age, 3.8/12 yrs. Actual age, 3.10/12 yrs.

From examination of the results of the cases in Group I it will be seen, that while a history of convulsions after birth would not appear to foretell later developments, this is not always the case in children who have suffered from intracranial haemorrhage in addition.

CHAP.V., SECT.I cont.

Group II. Cases subjected to Birth Trauma.

This group is composed of 40 children of nine years
of age who had been subjected to birth trauma, and whose
birth records were available. The choice of cases in
this group was based on the conclusion drawn from the path-
ological findings.

Birth records.

TABLE V.

BIRTH RECORDS OF THE 40 CASES WHO HAD SEVERE BIRTHS.

PARITY OF MOTHER		WT.OF CHILD IN LBS				MODE OF DELIVERY		
Primigr.	Multip.	Under 7 lbs	7-8 lbs	8-9 lbs	over 9 lbs	Normal	Forceps	Breech.
24	16	10	14	14	2	2	25	13

Examination of Cases. These children of 9 years of age
were examined in the following manner:

(1) History of child's health from mother.

(2) Physical examination.

(3) Mental examination (a) Serial intelligence tests.
                        (b) Tests of general knowledge.

The examination of (1) and (2) was similar to that des-
cribed in Group I.

Mental Examination. (a) Serial Intelligence Tests,
   (Binet-Simon).
The children were asked -

(i) What is the date - day, month, and year.

(ii) To repeat four digits backwards.

(iii) To name six coins and give total value,
(2/6, 2/-, 1/-, 6d, 1d and ½d).

(iv) To form a sentence using the words boy,
ball and river.

(v) To say a word rhyming with a given word.

(vi) To explain how he or she would look for
a lost ball in a circular field.

(b) Tests of General Knowledge.

Attention and Form Perception. The power of directing
all one's mental effort in one direction was combined with
the perception of form.

A simple shape of a single dimension such as a square,
triangle, or diamond was drawn on a sheet of paper, and
the child was asked to point out this particular shape from
a selection of various different shapes printed on a card.

In this manner the children were forced to concentrate
their attention on the one sheet of drawings, and also to
recognise the particular shape, as in the Sequin test. At
the same time any change in size was perceived by the chil-
dren.

MEMORY. The children were encouraged to converse
with the examiner about their brothers and sisters; en-
quiries were made regarding the names and ages of their
playmates; and they were asked what they had seen on their

journey to hospital.   In these ways the recent memory of the children was assessed.

The children were then asked about their last holidays or birthdays to gauge the power of their remote memory.

ASSOCIATION.   This power was investigated in the manner suggested by the Joint Mental Deficiency Committee. The children were asked to describe the similarity between two things, such as an apple and an orange, or wood and coal.

CAPACITY OF MENTAL IMAGERY.   This quality of brain power was estimated by asking the children to describe one of the pictures contained in the Terman testing material for the measurement of intelligence.   The Healy Pictorial Completion Test (34) which is more valuable in assessing the progress of mental development, was not used.

RESULTS OF EXAMINATION OF CHILDREN IN GROUP II.

In 39 of the 40 children examined there was no evidence of any physical or mental defect.   No useful purpose therefore would be served by recording each individual result, but some general conclusions on the tests used are of interest.

The "date" test was well done by all children.   The repetition of digits produced a few failures, which is

understandable in view of the poor schooling of these children at that time. The "coin" test and formation of sentences was performed by most children in a satisfactory manner. The rhyming test was done very poorly. The Ball and Field test was performed quite well, especially by the boys.

Only one case of the forty examined failed to reach the normal standard. The details of this one case are as follows:-

EXAMINATION OF THE ONLY DEFECTIVE CASE.

<u>Birth Record</u>. The child was delivered by forceps after a prolonged 21½ hours' labour. The child was the mother's 7th, and weighed 8½ lbs. at birth.

<u>History of child's health from mother</u>. The mother explained that since birth the child had been backward and very troublesome. She stated the child was generally dull and sullen, but occasionally took mad turns. Education was only possible in a special school for feeble-minded children. The other six living children are of normal development and mind.

<u>Physical Examination</u>. The child appeared younger than his age. No physical abnormalities were detected.

<u>Mental Examination</u>. The child was dour and negatistic and most unresponsive. On examination he was reserved and

shy and his intelligence below normal.   Intelligence
Quotient = 63.

SECTION II: REVERSE CHECK ON KNOWN FEEBLE-
MINDED CHILDREN.

In the previous Section, 48 cases have been reported in which late developments of cerebral birth trauma might have been expected.   This second section deals with the birth records of 43 children who were attending special schools on account of known mental deficiency, and whose birth records were at hand.

1. Analysis of Birth Records of the 43 feeble-
minded Children.

(a) MODE OF DELIVERY.

All 43 cases were delivered spontaneously.

Spontaneous vertex delivery .... 41 cases.

Spontaneous breech delivery .... 2 cases.

From the figures it is calculated that of the 43 feeble-minded children, 95 per cent were born normally.

(b) PARITY OF MOTHER.

The mother was a primigravida in 7 cases.

The mother was a multipara in 36 cases.

(c) WEIGHT OF THE CHILD.

The weight of the child at birth was recorded in only 13 of the 43 cases, and consequently is of no statistical value.

Premature children 4

Mature children 9

(d) DURATION OF LABOUR. (No record in one case).

Duration.	Number of Cases.
0 - 12 hours	35
12 - 24 hours	6
24 - 36 hours	1
	42 cases.

It will be noted from these figures labour lasted less than 12 hours in 83 per cent of the cases.

There were only two cases of the 42 recorded in which labour was less than two hours.

2. General Observations on the Mentality of these 43 Cases.

These children were attending special school in which their intelligence quotient was checked at least once every three years by the school medical officer.

In none of these cases were the parents of the child mentally defective.

The average Intelligence Quotient was 70.

The physical development in 40 of the cases was normal. Only three cases were underdeveloped. Two cases had defective articulation. One child was an epileptic; two suffered from rickets, and one had pseudo hypertrophic dystrophy.

CHAPTER VI.

CONCLUSIONS DRAWN FROM THE EXAMINATIONS OF THE

CHILDREN AND BIRTH RECORDS.

For consideration in this chapter there is a total collection of 91 cases. Forty-eight of these had suffered trauma at birth, and were examined for the presence of any subsequent developments, such as mental deficiency, cerebral paralysis or epilepsy. The remaining 43 cases were known to be feeble-minded, and their birth records were investigated to find out if birth trauma had been present.

Cases of Convulsions and Intracranial Haemorrhage:

Convulsion Cases:

It is reasonable to conclude from the examination of these 4 cases that the occurrence of convulsions during infancy in the absence of intracranial haemorrhage does not indicate subsequent mal development.

Intracranial Haemorrhage Cases:

Examination of the 4 intracranial haemorrhage cases did not show a similar absence of late developments. In 2 of the cases spastic paralysis was present in one or more limbs, and in a third case there was partial paralysis of the right eyelid. All these physical defects had

been present since birth.   Mental retardation was present in one case, and in the others, the standard of intelligence was normal.

It may be concluded therefore that in a proportion of the cases which survive intracranial bleeding at birth various forms of paralysis may develop, some cases of which may also have mental retardation.

Cases which suffered Trauma at Birth:

From the results of the pathological investigation and the birth records it seems justifiable to conclude that these cases suffered cerebral birth trauma.

Examination of these 40 children showed mental impairment in one case, and no physical deformity in any case.   In this one case there was no family history of mental deficiency.   The labour was prolonged, the delivery instrumental, and the child was post-mature.

Thus it may be concluded that in one of the 40 cases mental deficiency might reasonably be ascribed to cerebral birth trauma.

Birth Records of Known Feeble-minded Children:

There is no evidence in the birth records of these 43 cases to permit a conclusion that their mental deficiency was caused by cerebral birth trauma.

GENERAL CONCLUSION.

The conclusions drawn from the examination of these 91 cases may be summarised thus:

Amentia: The incidence of amentia resulting from cerebral birth trauma in the selected cases of this investigation is 2.2.per cent.

Cerebral Paralysis: From the results of this investigation the occurrence of cerebral paralysis as a late result of intracranial haemorrhage at birth would not seem to be uncommon.

Epilepsy: There were no cases which underwent cerebral birth trauma who had developed epilepsy before the age of nine years.

P A R T III.

CHAPTER VII.

GENERAL DISCUSSION OF THE SUBJECT.

Brief History of the Literature.

Cranial Anatomy of the Newborn.

Cranial Stress and Moulding.

Asphyxia.

Anoxaemia.

Anaesthesia and Analgesia.

Causation of Cerebral Birth Trauma.

Mental Deficiency.

Cerebral Paralysis.

Epilepsy.

FINAL CONCLUSION.

P A R T   III.

CHAPTER   VII.

GENERAL DISCUSSION OF THE SUBJECT.

Brief History of the Literature.

During the past century considerable work has been published on the subject of cerebral birth trauma;   but references to the correlation between it and subsequent developments are indeed rare.

The earliest writings on the causal relationship of intracranial haemorrhage at birth and later developments were published by Dennis (19), Billard (19), and Cruveilhier (19) between the years 1826 and 1835.   These writers over one hundred years ago were the first persons to suggest that birth trauma might cause mental retardation or deformity later in life.

In 1862 Little (42) wrote his classical monograph "On the Influence of abnormal parturition, difficult labours, premature birth, asphyxia neonatorum on the mental and physical condition of the child, especially in relation to deformity."

Even at this early date Little foresaw the possibility of repeated cyanotic attacks after birth causing physical and mental changes in the child.   He states "that even

the want of a few breaths, if not fatal to the economy
may imprint a lasting injury upon them." In the same
paper speaking of intracranial haemorrhages he says, "when
they do not destroy life they give rise to permanent de-
formity of the cranium, to atrophy of injured portions
of the brain, and are the cause of many cases erroneously
described as congenital idiocy."

The work of Dr. McNutt (45) in 1885 is inaccurately
stated by most writers to have confirmed Little's views
on the etiology of infantile spastic paralysis. In actual
fact, without any pathological evidence, she generalised
that meningeal haemorrhage was the universal cause of in-
fantile spastic states from the time of birth, and assoc-
iated with difficult labour.

The next valuable contribution to the literature was
by Beneke (4) of Marburg in 1910, when he demonstrated for
the first time a method of cranial dissection which left
the tentorium cerebelli intact, and free for inspection
after removal of the cerebral hemispheres. This worker
showed how occipito-frontal pressure could readily produce
tearing of the dural septa and consequent intracranial
haemorrhage. At the conclusion of his article Beneke,
having shown how gross haemorrhage by compression of the
medulla will cause asphyxial death, points out how lesser

degrees of haemorrhage would form a plausible explanation for the every-day vague nervous symptoms, and might even cause epilepsy and idiocy.

In 1919 Margaret Warwick (66) drew attention to haemorrhagic diathesis of the newborn as an important etiological factor of intracranial haemorrhage in the newborn.

Eardly Holland (38) in 1922 investigated the pathological and mechanical aspect of cerebral birth trauma and advanced many important theories on its causation; he referred especially to the incidence of tearing of the tentorium cerebelli and submitted a detailed description of 81 cases.

During the last decade many statistical studies have been published on Intracranial Haemorrhage of the Newborn and Neonatal Deaths. Of the well known writers on these branches of the subject, Cruickshank (17), Craig (13), Ehrenfest (23), Hemsath (36), Sharpe (59) and von Haam (31) have made valuable contributions.

Within the last few years American writers, including Courville (12) and Schreiber (51), have stressed the importance of cerebral anoxaemia at birth as a cause of mental retardation in later life.

In 1932 Doll (20), Phelps (20) and Melcher (20) wrote

an interesting book which comprehensively deals with birth trauma and subsequent mental and physical changes.

Cerebral Anatomy of the Newborn.

Although many of the undermentioned facts were learnt from the pathological investigation, it has been considered apt to present them at this stage in order that the various differences between the skull and brain of the newborn child and those of the adult may be appreciated.

(a) CRANIUM. The skull of the child represents the all-important protective factor against brain traumatization at birth. The fact that the foetal skull must be capable of moulding in order that it may pass along the birth canal has been shown by experiment to depreciate the value of the protection.

Ehrenfest (83) quotes an experiment which was carried out on newborn animals with thick skull bones and no fontanelles. These animals were used as control studies and subjected to trauma similar to severe human birth trauma. In not a single instance could evidence of trauma be discovered in the brains of these animals.

The infant's skull is covered by skin and superficial fascia in layers similar to the adult. In the region of

the fontanelles, however, underneath the superficial fascia, a membraneous interosseous layer consisting of fused periosteum and dura mater separates the superficial fascia from the blood of the cerebral sinuses.

The adult skull bones consist of one layer of cancellous bone sandwiched between two layers of compact bone. In the newborn child, however, the bones are surprisingly thin, translucent and brittle and have only one layer of thickness. The diploe of the skull, according to Munro and Eustis (47), does not develop until the second year of life.

(b) BRAIN TISSUE. The brain tissue of the infant while it enjoys an immunity from many of the degenerative changes of the adult, has the distinct disadvantage of immaturity. The consistence of the tissue is softer; and this lack of firmness renders it more easily damaged. Moreover, as stated by Ashby[1] "damage to a brain as yet partially developed is likely to affect greater mischief in the long run by interfering with the growth, than the same damage to an already developed brain."

In discussing the development of the child's brain, Smith (61) points out that at birth the association pathways of the cerebral cortex are not fully myelinated, and the normal infant's behaviour is dependent mainly on the

tracts of the inner cortex. It will be readily under-
stood therefore that damage to the outer cortex, which
would receive the principal birth trauma, will not present
any clinical changes in the child until some time after
infancy.

After birth two main types of growth take place.
Firstly, the development of association pathways connect-
ing one cell with another, and secondly, the growth of
the dendritic processes which produce connection between
the cortex and spinal cord. Where development of these
pathways has been interfered with by accident, according
to Smith (61), "we find transitions, both in the neurolo-
gical and mental picture, in which the deficiency may be
any gradation from that of the lowest idiot to the border-
line individual, in whom defects of the higher powers of
association, insight, and judgment, as well as true
personality deviations, are the only outward manifestations
of previous injury."

(c) BLOOD VESSELS. In the cerebral blood vessels
of the adult degenerative changes and haemorrhages of the
brain are chiefly of arterial origin; and in the case of
haemorrhages are generally intracranial in type. In the
infant's brain the vessels are immature and delicate, and
more frequently damage is of venous origin, and only

rarely is macroscopic intracerebral haemorrhage found.
In the infant, the cerebral veins which feed the sinuses
are extremely numerous, especially in the fronto-parietal
region. These veins are particularly prone to rupture,
partly on account of their thin walls, but chiefly owing
to the fact that their terminal segments after emerging
from the pia-arachnoid are free and unsupported.

(d) CEREBRO-SPINAL FLUID. As in the adult, the
cerebro-spinal fluid of the healthy infant is secreted
from the fourth ventricle and choroid plexuses, and cir-
culates at a pressure equal to that of the blood in the
venous sinuses.

The normal pressure of cerebro-spinal fluid in the
newborn child, according to Munro and Eustis, is 5-8 mmHg,
whereas in the normal adult the cerebro-spinal fluid is
under the slightly greater pressure of 8-10 mmHg.

The moulding of the foetal head by occluding the
cerebral sinuses produces a rise in the pressure of the
cerebro-spinal fluid. This is due partly to the diminu-
tion of available capacity, and partly to a decrease in
the amount of cerebro-spinal fluid absorbed in the venous
circulation on account of the general congestion.

Since the infant brain has no Pacchionian bodies it
must be assumed that the avenues of escape of cerebro-

spinal fluid are diminished. Thus there are several factors which can increase the pressure of the cerebro-spinal fluid during parturition. If a blockage occurred in the flow of cerebro-spinal fluid, these factors would play an important part in accelerating the production of hydrocephalus.

Ehrenfest (31) maintains that under normal conditions the physiological increase of cerebro-spinal fluid pressure during parturition is prevented from effecting pathological changes by an escape of fluid down the spinal canal and furthermore by a reduction of the volume of blood within the brain.

Cranial Stress and Moulding.

According to Holland (38), cranial stress consists of a general compression of the child's skull combined with a simple longitudinal compression which is antero-posterior in direction in occipito-anterior and breech deliveries; and in face and brow deliveries the longitudinal pressure is exerted from the vault to the base of the child's skull.

The cranial stress which is naturally increased with the greater intra-uterine pressure during the second stage of labour results in the overlapping of the cranial bones of the child's head. Excessive stress will result in an

excessive amount of moulding; _and, if the strain were further increased, over-stretching of the dural stays might lead to their rupture and this in turn probably to subsequent intracranial haemorrhage.

The dural stays therefore act as strengthening bands to the skull, and by taking the strain, protect the brain tissue from direct injury. Where the compression of the skull is suddenly applied, as in the case of instrumental and breech deliveries, these strengthening bands are more liable to tear.

The main dural bands are the falx cerebri and tentorium cerebelli, which are formed from the periostal layer of the dura mater. These have their fibres especially arranged so as to bear the cranial strain.

The exact mechanism of rupture of the tentorium and falx, which was first described by Beneke (4) and later fully confirmed by Holland (38) and Capon (9), depends mainly on the sudden or excessive alteration of the normal cranial diameters. Thus, to quote Ehrenfest (23), "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 lengthening of the diameter perpendicular to the one reduced." In the falx cerebri the fibres are arranged longitudinally, and

sweep down posteriorly on either side into their lateral
extensions, where they are joined by the superficial
fibres of the tentorium in the white line.   This direc-
tion of fibres would therefore indicate that the chief
duty of the falx is to prevent an abnormal extension of
the longitudinal diameter of the cranium, or in other
words, to counteract the effects of lateral compression.
In a similar manner the deep horizontal fibres of the
tentorium likewise counteract severe antero-posterior
compression.

Either lateral or longitudinal compression by raising
the falx will exert a strain on the fibres of the upper
blade of the tentorium, and it will be at the weakest
point in this structure that tearing of the dura will first
reveal itself.

Two years after the publication of Eardly Holland's
(39) concise and lucid paper, Osborne Greenwood (30) wrote
an article on "The moulding of the foetal head and its
consequences". This interesting research caused most work-
ers to modify their views on the changes in configuration
when the head is moulded.   Greenwood (30) in his intro-
duction agrees that "moulding and its consequences often
furnish the newborn infant with the first of its many
mundane troubles". Firstly he used a lead cirtometer to

keep a permanent record of the diameters of the skull at
birth, and then again at the age of one week. This
method was soon replaced by the making of plaster casts
of the child's head at similar times. These casts covered
the vault of the skull and extended below the frontal
eminences anteriorly and the occiput posteriorly. In this
way the entire surface subjected to moulding could be
measured. By these experiments Greenwood clearly proved
that in the majority of vertex presentations the lengthen-
ing of the foetal head is antero-posterior and not ver-
tical. In occipito-posterior and breech cases the ver-
tical diameter is increased as previously demonstrated by
Holland.

The moulding of the skull increases very considerably
the cerebral congestion during birth, and hence renders
the delicate vessels and sinuses more prone to rupture.

Thus in agreement with Ehrenfest (21) and others we
may conclude that the overlapping of the adjoining skull
bones in sutures and fontanelles during moulding yields
two important effects. Firstly, through distortion of
the underlying venous sinuses the intracranial blood cir-
culation is markedly embarrassed. Secondly, that com-
pression of the skull in one direction generally causes
compensatory elongation in another direction; in this

manner certain of the dural folds are subjected to a definite strain which seemingly often proves too great for their integrity.

Asphyxia.

Ever since the early publications on the consideration of cerebral birth injury, asphyxia has been proclaimed as a factor of paramount importance. Until 1910, when mechanical trauma was introduced as an etiological factor by Beneke (4), writers on the subject insisted that asphyxia in itself was a common cause of children dying at birth and during the first few days of life.

The prime importance of asphyxia as a cause of neonatal death was strongly supported, as, it was frequently noticed that stillborn children were delivered in a cyanotic condition with evidence of cerebral venous congestion. Similar evidence was produced following necropsy examinations of neonatal deaths on children born in the condition known clinically as asphyxia livida.

Warwick (67), in 1921, and later Munro and Eustis (47) have given wholehearted support to this view, and consider asphyxia is frequently a direct cause of intracranial haemorrhage.

These writers point out how asphyxia may be either intra-uterine or intrapartum, and state that venous con-

gestion can be sufficiently great to cause rupture of
one or more cerebral vessels. Ehrenfest (23) takes issue
at the views of Munro and Eustis (47) on this subject and
maintains that "the etiologic relation of asphyxiation to
intracephalic parturitional lesions is practically limited
to the plausible assumption that a congested sinus or vein
is more likely than an empty one to rupture under pres-
sure strain."

Most modern writers share the opinion of Ehrenfest
and consider asphyxia as an important contributory factor
but one of secondary importance to mechanical trauma in
the production of intracranial damage. Thus von Haam (31),
1934, writing on the Pathology of Intracranial Haemorrhage,
regards "asphyxiation only as a predisposing cause or a
contributory factor; it may result from injury to the
respiratory center."

Cruickshank (17) in his detailed report on the Causes
of Neonatal Death does not differentiate asphyxial and
traumatic cases in his classification. This writer agrees
with Serbin (55) and Capon (9) that "the effects of the
injury, and, to a certain extent, the liability to the
injury, are increased by the asphyxial congestion which is
present in the newborn child."

Asphyxia neonatorum results when the respiratory
centre of the foetal brain is depressed. This depression

can be brought about by trauma to the brain, or by actual
failure of circulation in this vital portion of the brain.
From a consensus of opinion it would seem that per se
asphyxia is a symptom of medullary damage and plays a re-
latively insignificant role as a "direct" cause of neo-
natal death.

It has frequently been asserted by writers on the
subject of asphyxia neonatorum that, while the temporary
state of cerebral anoxaemia experienced during post-partum
apnoea may not in itself be fatal it may produce permanent
degenerative changes in the nerve cells of the child's
brain. Speaking of apnoea, Little (42) in 1862 declared
"that even the want of a few breathings, if not fatal to
the economy, may imprint a lasting injury upon them."

An experimental investigation into the effects of
asphyxia on the brain was undertaken by Ford (28) in 1928.
The purpose of this investigation was to determine whether
asphyxia alone could produce permanent brain damage, or
more specifically, whether asphyxia neonatorum played any
part in the etiology of infantile cerebral paralysis.
Ford (27) used cats in his experiments which were designed
to duplicate the conditions during human parturition, and
discovered that anoxaemia brought the heart to a standstill
before any lasting damage was inflicted on the nervous

system.   By subsequent experiments Ford showed "If the animal were asphyxiated only once its nutrition did not suffer, but repeated asphyxiation for prolonged periods caused interference in development, and the kitten would be definitely smaller than its siblings."   Yet even in these cases no gross or microscopic changes could be detected.

During the last ten years workers in the Continent and more recently in North America have done much research on the pathology and consequences of cerebral anoxaemia and their results to a large extent contradict the earlier experimental work of Ford.

Anoxaemia.

The clear perception of the state of anoxaemia has been presented to us by the publications of Haldane (32) and Barcroft (2).   Haldane writing on the clinical effects of the onset and prognosis of anoxaemia in one sentence of concentrated thought concludes that "anoxaemia not only stops the machine but wrecks the machinery".

To Barcroft (2) we owe the lucid differentiation of the types of anoxaemia -

      (i) Anoxic type
     (ii) Anaemic type
    (iii) Stagnant type.

In anoxic anoxaemia the arterial blood is insuffic-

iently saturated with oxygen.   Any type of suffocation
in the newborn such as aspiration of mucus, an excessive
amount of amniotic fluid in the pulmonary alveoli, or
partial strangulation by the cord, may produce this type
of anoxaemia.

In anaemic anoxaemia the quantity of functioning
haemoglobin is insufficient, and hence the oxygen capacity
of blood is too low.   Schreiber (51) describes the case
of a mother suffering from splenic anaemia who produced
a mentally defective child;   the subsequent children, born
after the mother was cured of her anaemia, were normal in
every respect.

In stagnant anoxaemia the blood is normal in quality,
but is supplied to the tissues in insufficient quantity.
This may occur in cases of heart disease resulting in a
low maternal blood pressure, and consequent failure to
maintain adequate foetal circulation.   Similarly, in
cases of dystocia where cranial pressure due to forceps
will cause a stagnation of foetal cerebral circulation.
In these cases cerebral anoxaemia may develop if the oxygen
deprivation is acute, or of sufficient duration.

It is reasonable to assume, and is generally believed
by modern workers, that during the course of even normal
labour the cerebral hemispheres will be subjected to some

degree of anoxaemia as the head is moulded. Several writers are of the opinion that cerebral anoxaemia is the causative factor in the production of small areas of cerebral necrosis found on post-mortem examination. That such patches do exist is verified by the microphotograph on page 36a which shows a typical area of degeneration. The congestion of the adjacent vein supports the theory that this lesion was caused by stagnant anoxaemia.

Courville (12), writing on the mechanism of cerebral necrosis following anoxaemia, describes how the interchange of oxygen and carbon dioxide, as far as the nerve cells are concerned, takes place in the pericellular fluid. He claims therefore that it is here that the earliest evidence of damage from cerebral anoxaemia should be found. This he finds to be the case and by means of microphotographs has demonstrated the development of the actual morphological changes. The resulting pathological changes in the brain tissue, according to Courville (12), which depend upon the degree of anoxaemia and its duration are: (a) a sclerosis of scattered pyramidal cells, (b) an occurrence of discreet pale areas on the cortex, (c) a patchy necrosis of superficial or deep, or of all the cortical layers, (d) a subtotal destruction of the cortex or, if the patient survives for a sufficient interval,

(e) a vascular scar may result due to the formation of new blood vessels.

Whether these pathological findings can be ascribed to local anoxaemia is doubted by many writers. Cruickshank (17), for example, maintains that the rapidity with which the central nervous system of the newborn infant undergoes autolysis, makes it unwise to draw conclusions from histological and cytological findings without very careful control of observations.

Other writers maintain that not only does anoxaemia produce cerebral necrosis, but also that such necrosis may cause permanent changes in the brain with clinical neurological manifestations. Thus Schreiber (52) considers that "spasticity, convulsions and mental retardation are merely reflections of the area of brain damage due to the anoxic lesions, and should not be considered as clinical entities, but rather as expressions of anoxic brain damage."

The same writer (50) also considers that similar damage is produced by apnoea of the newborn infant, and substantiates his theory with the results of an investigation into the birth histories of 685 children with the following defects:

Suffering from convulsions .... 131 cases.

Showing evidence of spasticity .... 69 cases.

Combination of symptoms .... 248 cases.

Mental retardation .... 130 cases.

Miscellaneous group .... 107 cases.

Excluding these cases which developed infectious diseases
after birth, and also those cases in which there was an
appropriate familial tendency, Schreiber discovered that
of the 500 remaining cases 70 per cent had suffered apnoea
at birth.

While there is microscopic evidence of cerebral
necrosis in cases which have been delivered abnormally or
have been difficult to resuscitate after birth, it is
hardly justifiable to assume that such cerebral damage
produces symptoms in later life.

In the series of cases quoted above no reference is
made to the incidence of intracranial haemorrhage at birth.
In Case O of the present investigation the child was appar-
ently morphinised and suffered prolonged apnoea at birth
and therefore cerebral anoxaemia, but it would seem more
likely that the subsequent development of mental retarda-
tion and spastic paralysis was caused by the co-existing
intracranial haemorrhage.

Anaesthesia and Analgesia.

Anaesthesia. Many discussions have taken place, and much has been written during the last decade on the various methods employed to produce relief of pain during child-birth. While it is outside the sphere of this work to deal in detail with the drugs administered for this purpose recent publications have suggested that anaesthesia and analgesia may affect, under special circumstances, the ultimate fate of the child.

As a rule general anaesthesia is not administered until the second stage of labour when it may be given either intermittently throughout this stage, or merely to render the patient unconscious during the actual delivery.

Chloroform has until recently held first place in the field of obstetrical anaesthesia mainly on account of its cheapness, ease of portability and administration. This anaesthesia is capable of producing complete perineal relaxation and although the maternal and probably the foetal blood pressures tend to become lowered the period of post-partum apnoea is not unduly prolonged. There is no evidence that this anaesthesia is in any way harmful to the newly born child.

Ether anaesthesia in obstetrical practice has several disadvantages. Under light anaesthesia the patient is

frequently excitable and difficult to control. Respiratory embarrassment is commonly encountered and consequently before birth the child's brain is deprived of an adequate supply of sufficiently oxygenated blood. According to Lloyd-Williams (42) ether seems to have a more prolonged effect on the child than chloroform and its first respiration is often delayed.

Gas and oxygen, which is the most popular anaesthesia in obstetrical practice today, does not inhibit uterine contractions, and is comparatively safe, pleasant, and non-toxic to the patient. The induction using this anaesthesia generally produces slight cyanosis of the mother, and consequent deficient supply of oxygenated blood to the child. The child is occasionally apnoeic at birth.

Spinal anaesthesia is the most suitable anaesthetic in cases requiring Caesarean section. This form of anaesthesia removes the spinal nerve control of the uterus which results in the spasmodic contraction of the uterine muscles. Some obstetricians have condemned spinal anaesthesia in instrumental deliveries on the grounds that the severe contractions of the uterine muscle so obtained may endanger the life of the child. It would seem that unless it is used improperly before the commencement of the second stage of labour any contractions would be of an

expulsatory nature and beneficial to the progress of the labour. There was only one case in this series where spinal anaesthesia was used in an instrumental delivery, and the child sustained a fractured skull.

It will therefore be realised that anaesthesia, while it relieves the maternal suffering, is occasionally responsible for causing a slight prolongation of the period of post-partum apnoea, and may therefore be harmful to the child.

Analgesia. For the average patient who approaches her labour in a calm and satisfactory psychological state there is as a rule no indication for prescribing drugs during the early part of the first stage. When the second stage is reached, the patient retires to bed and an analgesic is prescribed.

There are numerous analgesics at the disposal of the obstetrician, but in this article they will be considered only in respect of their effects upon the foetus.

The chloral and bromide group act as sedatives and are mildly analgesic. There is no evidence to suggest that these drugs have any effect on the foetus.

The barbiturate group includes many proprietary preparations which have an excellent hypnotic and analgesic effect. When used in sufficient strengths to pro-

duce amnesia and analgesia these drugs frequently result
in the child being born in a limp and apnoeic condition.
As a rule, however, the child is easily resuscitated.

The opium and hyoscine groups are frequently combined
to produce a narcosis named 'Twilight Sleep'. This method
of analgesia is used in most countries and when adminis-
tered with skill is probably the method par excellence.

The condition of the child at birth following the
abuse of such therapy has led to numerous publications
by research workers interested in cerebral anoxaemia. The
clinical manifestation of toxaemia of the newborn child
by excessive maternal analgesia may be summarised as
follows:

The baby is born in a limp and drowsy condition, and
is generally pale in colour. The respirations are shallow,
infrequent and irregular, and cyanosis is usually present.
The muscles are in a flaccid state, and the child lies
still. The babies generally recover quite quickly from
their period of apnoea on the administration of oxygen.

Attention has already been drawn to the serious conse-
quences which some authorities attribute to this period
of apnoea. While it is true that certain analgesic drugs
do lengthen the period of apnoea it is unlikely that their
popular administration would continue if such unfortunate

sequelae resulted.

Causation of Cerebral Birth Trauma.

In the pathological investigation of this work the most important findings were intracranial haemorrhage and dural lacerations.   While the former is probable evidence, the latter is positive evidence of cerebral birth trauma. The causation of these two features will now be considered.

(A) INTRACRANIAL HAEMORRHAGE:

The etiology of intracranial haemorrhage has produced great diversity of opinion among the numerous authorities, who have made contributions to the extensive literature. Following the pioneer work of Beneke,[4] birth trauma was held responsible until the explanation proved unsatisfactory as intracranial haemorrhage was noted in cases unattended by dystocia and even in infants whose mothers were delivered by Caesarean section.   Consequently many other causative factors have been added.

The causes of intracranial haemorrhage fall into three distinct groups.

   (i)   Immediate causes.

   (ii)   Predisposing causes.

   (iii)   Contributory factors.

(i) The immediate cause of haemorrhage in the large

majority of cases is traumatization during birth. In
this present investigation, of the 28 cases of fatal
intracranial haemorrhage there was dural laceration in
23 cases. The combination of a severe delivery and the
subsequent development of intracranial bleeding is of too
frequent occurrence to be passed off lightly as a coinci-
dence. While it is more usual to find intracranial hae-
morrhage in cases where the child is brutally dragged
between the promontory of the sacrum and the symphysis
pubis with forceps, it may also develop in some cases
born spontaneously in which little or no moulding may
have been necessary. There were 5 such cases in this
series. Likewise intracranial haemorrhage may occur
following delivery by abdominal section; Craig (13) had
5 such cases in a series of 126 cases of haemorrhage;
Ehrenfest (23) has notes of 19 cases of this type. In this
series there were 2 such cases; in neither case had
labour commenced. These facts force the conclusion that
in a small proportion of cases intracranial haemorrhage
may be produced by one other, or several other factors.

(ii) Predisposing causes. (a) Asphyxiation: formerly
this factor was considered to be the direct cause of
intracranial haemorrhage. The frequent association of
haemorrhage with the clinical and characteristic signs of

asphyxia is stressed by Cruickshank (17), who classified deaths due to gross injury in the same group with cases which he considered died of asphyxiation. The same writer is of the opinion that acute cerebral oedema, which he considers results from trauma, embarrasses the respiratory centre and so produces asphyxiation. In this present series 22 of the 28 cases of fatal intracranial haemorrhage showed marked congestion of the brain tissue. Large haemorrhages inside the skull are most likely the direct results of trauma, but there is good reason to believe that the minute multiple type described elsewhere may frequently result from asphyxiation alone. Asphyxia is the most important predisposing cause of intracranial haemorrhage. Until such time as it is possible to differentiate between asphyxia due to oxygen deprivation and asphyxia due to trauma of the respiratory centre it cannot be considered as the most important direct cause.

(b) Syphilis. As a predisposing cause this disease has in the past been greatly overrated. This disease is probably only of significance by virtue of the fact that, as pointed out by Cruickshank (16), it may cause a premature birth with consequent intracranial haemorrhage and death of the foetus. As a predisposing cause of intracranial haemorrhage it is of little or no importance. There

were no mothers infected with syphilis in this series of cases.

(c) Prematurity. In the above paragraph prematurity has been assumed to predispose to intracranial haemorrhage. While this is the general opinion, Craig (13) has recently shown that prematurity is frequently present in cases of subarachnoidal and intraventricular haemorrhage, but that mature infants are more liable to develop the more common subdural type of haemorrhage. These findings concur with the present series in which the number of mature children with subdural haemorrhage was almost double the number of premature children. Similarly in agreement with Craig (13), 75 per cent of the cases of subarachnoid haemorrhage were found in premature infants. From a study of fatal cases of intracranial haemorrhage it would seem that the development of intracranial haemorrhage in post-mature infants merits more attention. In this series there were 13 cases (i.e. 46 per cent) which died of intracranial haemorrhage which were over 3.2 kilos in weight.

(d) Diseases of the mother. Sporadic observations suggest an increased susceptibility to intracranial hae-morrhage in cases where the mother has suffered a serious illness during her pregnancy. In this series the number of cases of this kind was negligible.

(e) Parity of mother. The number of the pregnancy plays an important role as a contributory factor in the causation of intracranial haemorrhage. First children are more liable to suffer intracranial haemorrhage. Of all the 60 cases which showed some intracranial bleeding the large majority were first born children.

TABLE VI.

PREGNANCY:	1	2	3	4	5	6	7	8	9	10	11	12
Cases of Intracranial Haemorrhage.	29	8	9	2	3	3	1	2	1	0	0	2

The above table shows the incidence of intracranial haemorrhage and demonstrates the liability of first born children; these findings are in agreement with other works.

(iii) Contributory Factors. (a) Haemorrhagic Diathesis: Formerly this diathesis was thought to be of paramount importance in the causation of intracranial haemorrhage. The prominence which this disease received was largely due to a publication by Warwick (66) in 1919. In the concluding summary of this article Warwick states that "Haemorrhagic disease of the newborn is a much neglected but very important cause of cerebral haemorrhage in infants, occurring in 44 per cent of the cases of our

series."

The same writer (67) in 1921, in a series of 53 cases of intracranial haemorrhage found haemorrhages in other organs in 20 cases. In 13 of these cases the coagulation time was increased beyond the normal 5-9 minutes.

Munro and Eustis in 1922 (47) drew attention to the frequency of this disease in cases of intracranial haemorrhage.

Serbin (55) in 1928 found 2.5 per cent of his 320 foetal post mortems had suffered from haemorrhagic disease of the newborn.

Modern workers, including Cruickshank (17), Sharpe and Maclaire (57), do not find this disease to be a common cause of haemorrhage, but consider it is an important contributory factor. The recent research of Macfie, Bachrach and Change (44) should be of considerable therapeutic value in reducing the incidence of haemorrhagic disease, which has been demonstrated by these workers to be due to a deficiency of Vitamin K with a consequent fall in the prothrombin index.

The coagulation time was not determined in any of the neonatal infants of this series. The post mortem examinations were confined to the head of the infants.

(b) Miscellaneous contributory factors. The abuse of

the Schultze method of resuscitation and the too frequent
desire to suspend an asphyxiated child by the feet may
contribute to the production of intracranial haemorrhage.
Similarly the untimely administration of Morphine to the
mother may so depress the respiratory centre of the child
that asphyxial congestion will result; in this way pre-
disposing to intracranial haemorrhage.   Likewise the
administration of other analgesics and narcotics.

The administration of Pituitrin before parturition
produces in some instances a spasmodic contraction of the
uterine muscles, and so causes a sudden moulding of the
foetal head;   a contributory factor in the production of
intracranial haemorrhage.

Prolongation of labour likewise increases the likeli-
hood of an asphyxiated child and so contributes to the
production of intracranial haemorrhage.   In 15 of the 28
cases of fatal intracranial haemorrhage in this series
labour was of abnormal length.

(B) INJURY TO THE DURAL SEPTA:

Tearing of the tentorium cerebelli is the most re-
liable evidence of intracranial traumatization.

(i) _Immediate cause._   The immediate cause in all
cases is birth trauma, which may, of course, occur in a
spontaneous normal delivery.   It has been shown how 12

cases of the 40 cases showing tentorial tearing were born spontaneously. There were no cases of dural laceration in children whose mothers were delivered by Caesarean section, which suggests that it is during the passage of the child through the birth canal that such tearing occurs.

In this series 88 per cent of the cases showing tearing of the tentorium died before birth. In 5 of the cases the child lived for some hours. The frequency with which dural tears are encountered during pathological examinations suggests that many cases of torn tentorii survive without producing any marked symptoms. No doubt cases showing evidence of intracranial birth trauma are much more frequent than is indicated by statistics which include only the fatal cases. It is in fact reasonable to suppose that careful examination of the tentoria in persons dying later in life would show some evidence of damage sustained at birth. The writer has only seen one such case described.

The mechanism by which an increase in vertical diameter, due to compression of the child's head, causes straining and rupture of the dural stays has already been described elsewhere. When the tentoria are torn the support which prevents the full compression of the head being transmitted to the medulla is removed and consequently

the vital centres are embarrassed.   In this way, and in producing intracranial haemorrhage, tearing of the tentoria may prove fatal.

(ii) <u>Predisposing causes</u>. (a) Instrumental and Breech deliveries: The results of the pathological investigation demonstrate the higher incidence of tentorial tears in these types of confinement.   Seventy per cent of the cases showing tentorial tears were instrumental or breech deliveries.   In the instrumental deliveries, dural laceration was present in 75 per cent of cases.

In the breech deliveries dural lacerations were present in 57 per cent of such cases.   There is general agreement on the high incidence of dural lacerations in these two types of delivery.

(b) First-born Children: The results of this investigation have shown the higher incidence of tearing in infants of primigravidae. The incidence in these cases was 48 per cent whereas the incidence in infants of multiparae was only 34 per cent.

The chief reason for this occurrence is the increased resistance of the firm maternal parts of the primigravida, which causes extra moulding of the child's skull. Also the longer duration of labour and the greater frequency of instrumental deliveries in primigravidae are important

factors.

(c) Maturity of the Foetus: The results of this
investigation show that prematurity as a contributory in
the cause of intracranial injury is not of such signifi-
cance as post maturity.   The incidence of tentorial tear-
ing in post mature infants was 72 per cent while it was
only 21 per cent in premature infants.   This is in agree-
ment with the findings of Ehrenfest (23).

(d) Length of Labour: Examination of Table III re-
veals the unquestionable fact that prolonged labour in-
creases the liability to intracranial haemorrhage. This
is partly in view of the fact that most of these cases are
eventually instrumental deliveries and partly due to the
increased congestion of the brain resulting from prolonged
compression.

This concludes the consideration of the factors which
may predispose the infant to cerebral birth trauma.

In the next section the possible late effects of
cerebral birth trauma will be discussed.   These sections
will consist therefore of the current views on the causa-
tion of mental deficiency, cerebral paralysis, and epilepsy
with especial reference to their association with cerebral
birth trauma.

Mental Deficiency:

Mental defectiveness means a "condition of arrested or incomplete development of mind existing before the age of eighteen years, whether arising from inherent causes or induced by disease or injury."   Amentia has thus been classified into two distinct types.

(a) Primary Amentia includes all cases where the mental deficiency is caused by an intrinsic or endogenous factor. According to Tredgold (65), this "germinal type" accounts for approximately 80 per cent of all cases of Amentia.

(b) Secondary Amentia includes the cases of mental deficiency which have no familial origin, but are caused by extrinsic or environmental factors such as disease. This type is numerically of less significance.

Most writers subdivide these two basic types of Amentia into the five clinical varieties adopted by Tredgold (65):

(1) Germinal, (2) Traumatic, (3) Infective, (4) Degenerative, and (5) Deprivative. For the purposes of this work, the following classification, modelled on that of Wyllie (71), will be found more instructive, as it includes those cases of secondary Amentia which are associated with paralysis.

(A) PRIMARY AMENTIA:

    (i) Simple primary Amentia.

    (ii) Microcephalus.

    (iii) Mongolism.

    (iv) Tuberose Sclerosis.

(B) SECONDARY AMENTIA:

    (i) Ante and Intrapartum Group.

        (a) Congenital Hydrocephalus.
        (b) Congenital Porencephaly.
        (c) Traumatic Amentia.
        (d) Amentia with Cerebral Paralysis.

    (ii) Post Partum Group.

        (a) Encephalitis.
        (b) Meningitis.
        (c) Acquired Hydrocephalus.
        (d) Congenital Syphilis.
        (e) Epilepsy.
        (f) Amaurotic Family Idiocy.
        (g) Endocrine deficiencies.

While it is outside the scope of this work to deal in detail with the numerous types of Amentia classified above, it was found essential to have a clear conception of the entire subject, in order that an accurate account of the clinical varieties, which might have been encountered in the examination of the children, could be recorded. Moreover, as it has recently been hinted that many cases of simple primary amentia should be classified under the secondary type of amentia which results from birth trauma,

an inquiry was therefore undertaken into the etiological factors of these two types in particular.

(A) PRIMARY AMENTIA:

(i) <u>Simple Primary Amentia</u>. This type accounts approximately for 73 per cent of all cases of Amentia. The etiology therefore becomes a matter of paramount importance.

HEREDITY. Most authorities agree that primary amentia can be ascribed to defective germ potentiality. While this is probably the most important etiological factor, it does not follow that the majority of mentally defective children are the offsprings of certifiable mentally deficient parents.

This fact was pointed out by Williams (69) who concluded that only about 5-10 per cent of mentally deficient children had certifiable parents. He concludes that while direct transmission of amentia is found in comparatively few children, the great majority of defective children are the offsprings of persons not themselves certifiable, but of inferior and unstable mentality.

Herd (37) discovered in his series of cases that in 15 per cent of the children direct transmission was present and either one or both parents were mentally defective.

Goddard (29) is of the opinion that normal-mindedness is, or at least behaves like, a unit character; is dominant

and is transmitted in accordance with the Mendelian law
of inheritance.   In the totals of all the matings he
recorded the expected number of feeble-minded persons
according to calculations was 704;   the actual number
found on examination was 708.   The expected number of
normal-minded persons according to calculations was 352,
and the actual number found on examination, 348.   Such
results are difficult to account for on any other basis
that feeble-mindedness is frequently transmitted in accord-
ance with the Mendelian formula.

CHRONIC ADDICTION TO ALCOHOL or A TUBERCULOUS DIATHE-
SIS.   This is frequently noted in the parent of a mentally
deficient child.   These factors were formerly considered
of importance as a direct cause of simple primary amentia.
It is now realised that only in virtue of the parents be-
ing of weak stock can this be applied as an indirect etio-
logical factor.

CONSANGUINITY OF THE PARENTS.   This is not now con-
sidered to be a direct cause of amentia, although most
writers admit it bears an important relationship to the
occurrence of deaf-mutism.

(ii) Microcephalus.   This includes all cases where
the cranial circumference after complete development is
below 17 inches.   This type of amentia is probably the

result of a pathological variation of the germ cell. As a rule one or other parent is of a distinctly neurotic stock.

(iii) <u>Mongolism</u>. No definite opinion has been expressed regarding the exact cause of this type of amentia; there is no evidence whatever that the condition is due to a pathological process.

(iv) <u>Tuberose Sclerosis</u>. Hypertrophic nodular sclerosis of the brain is a rare condition in which the error in development seen in the cerebrum is also reflected in similar changes in the skin and other viscera. It is generally a heredo-familial affliction and is always associated with amentia and frequently with epilepsy.

(B) SECONDARY AMENTIA:

(1) <u>Ante and Intrapartum Group</u>.

(a) <u>Congenital Hydrocephalus</u>: In most cases of congenital hydrocephalus, who survive parturition, there is interference with the mental functions, and idiocy may be present; exceptionally, however, the child may be wonderfully intelligent. Cerebral pneumography has revealed the interesting fact that the majority of these cases have an obstructive origin. According to Dandy a large proportion of these cases are due to a congenital

atresia of the aqueduct of Sylvius.

Cases have been published from time to time with photographs demonstrating this type of hydrocephalus resulting from occlusion of the aqueduct by blood clot. In these cases it is probable that the condition was of traumatic origin. Ford (27) is of the opinion that this is one of the few types of amentia which can be reasonably ascribed to cerebral birth trauma.

(b) <u>Congenital Porencephaly</u>: Muir (46) and Hassin (33) distinguish two distinct varieties of porencephaly. The primary type is due to a malformation with resultant agenesia of the brain tissue. The secondary type may, according to these authorities, be a result of injury received at birth, or of encephalitis.

(c) <u>Traumatic Amentia</u>: Few writers deny the existence of such a condition, but its significance is frequently debated. Many exhaustive articles have been published by psychiatrists in the past emphasizing the importance of birth trauma as a cause of amentia. The statistics of these articles, however, must be nullified to a great extent for two main reasons. Firstly, the mere claim of a difficult delivery should not convince the psychiatrist unless obstetrical records can support the mother's evidence. Secondly, many cases of instrumental delivery which have resulted in the infant being

born dead, or succumbing in early childhood, are ex-
cluded from the calculations.

It is of interest to note that Tredgold (63), who
is one of the greatest authorities on amentia, writing
in 1908, considers "that the importance of labour as a
cause of amentia has been much overrated, and that the
total number of cases which are the immediate consequence
of these conditions is relatively very small, being pro-
bably not more than 1-2 per cent of all aments." Twenty-
one years later, the same writer (64) states "that these
cases of amentia which can definitely be ascribed to
injury during birth is about 4-5 per cent of all defects."

In a study of 103 cases of meningeal haemorrhage in
the newborn, Fleming and Morton (24) were able to follow
up 33 of these cases for over a year after birth. Five
of these were found to have either physical or mental
defects. Of these 5 cases 3 were spontaneous deliveries
and 2 were instrumental; 4 of the cases had spasticity
in one or more limbs; one child was hydrocephalic; and
in all cases the children were considered to be mentally
deficient. These writers conclude "that it does not seem
possible to find any common factor in the birth to account
for the intracranial damage." Moreover they state "that
some of the cases indeed who have made a complete recovery

seemed to have had far worse injury at birth than any of the 5 cases under discussion."

Werner (68), in a study of 96 cases delivered by low forceps and seen from 2-4 years after delivery, could find no instances of mental deficiency that could be attributed to instrumentation.

Schroeder (53), writing on the behaviour difficulties of 5000 children, in association with birth trauma, concludes (i) that difficult labours tend to produce mental retardation which in turn leads to behaviour problems; (ii) that distractibility and hyperactivity are characteristic personality traits associated with birth trauma, and (iii) that children with a history of cerebral birth injury without later palsy show the mental retardation and behaviour characteristics common to children with cerebral birth palsy.

Doll, Phelps and Melcher (20) attribute 6-10 per cent of their mentally deficient subjects to injury received at birth. These workers from a comprehensive review of the literature conclude that birth trauma may result in early mortality, in relatively minor damage which is subsequently overcome through the repair of natural growth; inmotor handicap, and in serious behaviour disorders or personality defects.

Ford (27) considers that only 3 types of mental defect have any possible relation to birth injury, namely the spastic, the hydrocephalic, and the epileptic. "It is evident then", writes Ford, "that birth injury is of some importance to the development of mental deficiency although it is far behind the heredity disposition in numbers. On the other hand it should not be overlooked that the cases due to birth injury are often of the most severe form which does not merely make the patient an idiot, but renders him completely helpless in every respect."

This supposition does not agree with the actual findings of Dayton (18), who performed a psychiatric examination of 20,473 retarded children in the public schools of Massachusetts. Dayton made a graphic representation between normal and abnormal labours and the Intelligence Quotient. From the analysis of this graph it appears that children of dull normal or low normal intelligence are more likely to be influenced by abnormal birth conditions than children who are definitely in the imbecile moron or borderline groups. The same worker also studied the relationship between labour and the average grade accomplishments of the children in the subjects, reading, spelling, writing, languages, and arithmetic. The results

did not suggest that there was any association between abnormal labour in the mother and low school accomplishments of the children in any of the above-named subjects.

This view is expressed by Little (42), who wrote the following extract 70 years before Dayton's (18) publication: "I have observed that in impaired intellect from abnormal birth the degree of impairment met with in private practice often does not exceed feebleness of intellect .... The individual may acquire a fair knowledge of music, the memory is good, the constructive tendencies may exist, a fair capacity for arithmetic and languages may be displayed, but there commonly exists a great want of application, and a slowness of intellect similar to the slowness of volition."

The cases of amentia following birth trauma described by these writers are of the dull, normal, emotional, and distractible type. The present series of known mentally defective children was of this mental grade, with an average Intelligence Quotient of 70, yet abnormal labour was almost unknown in these cases.

At the present time it will therefore be understood while most authorities on the subject of traumatic amentia are willing to admit its presence only a few workers are prepared to emphasize its importance.

Recently workers have attempted to establish the significance of traumatic amentia on pathological grounds, but these investigations have been of little avail. There seems little doubt that intracranial haemorrhage which is thought to be evidence of trauma occurs to some extent or other quite frequently during child birth.

A small amount of blood from a meningeal bleeding would most likely be quickly absorbed without producing any clinical signs or later results. A gross haemorrhage would most probably prove fatal. Between these two extremes is the intermediate type of haemorrhage which may result in permanent brain damage and subsequent amentia. This moderate haemorrhage may form a localised haematoma which in turn undergoes softening and sclerosis with arrest of development and disarrangement of the architecture of the cortical brain cells.

Likewise the small punctate haemorrhages described by Schwartz (54) may undergo cyst formation, softening and ultimate sclerosis. According to Courville (12) necrosis of the brain tissue is not demonstrable histologically unless the infant has survived at least 36 hours; he also states that these changes are probably responsible for the residual symptoms present in children who survive.

These degenerative changes, however, as pointed out

by Muir, Hassin and others are not specific to traumatic
lesions but can occur after any toxic action, or as a
result of vascular disease. Moreover until the third
month of extrauterine life the neopallium which is so
important to the psychic activity of the child is rela-
tively undeveloped. Thus it is questionable whether
small haemorrhages would be of much significance since
the cells of the neopallium would doubtless adapt them-
selves to these small areas of sclerosis, which would not
appreciably interfere with their function.

Pickworth (49), who has examined over 1000 brains of
persons suffering from amentia, points out that many cases
show areas of subcortical softening. Quite frequently
these areas are unsuspected during life and give rise to
no clinical features.

Biggart (5) maintains that in many cases of amentia
it is impossible to say on pathological examination "whether
the lesion is the result of maldevelopment or an injury at
birth."

The results of this present investigation strongly
suggest that, while cerebral damage does not infrequently
occur as a result of birth trauma, the percentage of those
cases so injured, which subsequently develop amentia, must
be extremely low: less than 2 per cent.   —

Cerebral Paralysis:

The physical defects which some workers attribute
to cerebral birth trauma, on account of their more con-
spicuous nature, have received great attention by the
medical profession.   Spastic monoplegias, hemiplegias
and quadraplegias are not infrequent amongst children,
but are by no means so common as the cases of spastic
diplegia. Thus Holt (40) records that in the Harriet Lane
Home diplegia occurs at least ten times as commonly as
hemiplegia.

There has been much discussion concerning the etio-
logy of cerebral diplegia since it was first described
by Little (42). According to his writings Little's dis-
ease, strictly speaking, only includes those cases of
cerebral diplegia associated with a difficult birth.
Little (42), as pointed out by Ehrenfest (23) and Collier
(11), did not consider, nor even suggest, that meningeal
haemorrhage was a cause of infantile spastic paralysis,
but asserted that asphyxia at birth was a causal factor
of the nervous lesion.

Little's valuable contribution to our knowledge of
cerebral diplegia established several important facts:

(i)   that the condition is often undeniably assoc-
      iated with abnormal birth.

(ii) that this disease is frequently associated
      with microcephaly.

(iii) that the causative pathology consists of a widespread fine lesion of the brain tissue which is unequally distributed throughout the cerebrum.

(iv) that the disease is not caused by gross injury to the brain during birth.

McNutt (45) in her thesis for membership of the American Neurology Association, written in 1885 described in detail one case of double infantile spastic paralysis, the necropsy findings of which, as agreed by Collier (11), strongly suggest a case of atrophic lobar sclerosis. In the same paper she referred to two cases of infantile spastic paralysis, and without sufficient evidence stated that meningeal haemorrhage was the universal cause of the infantile spastic state dating from birth and associated with difficult labour. This statement was readily accepted by the medical world, and meningeal haemorrhage for many years was considered to be the initial morbid process in cerebral diplegia.

Since a proportion of diplegic infants are premature, the condition was next ascribed to an arrest of development of the nervous tissue following premature birth. In this connection it is interesting to note that in an analysis of 235 cases of congenital diplegia investigated by Ford, abnormalities of labour such as prolonged or difficult labour, instrumental or breech delivery occurred in only

15 per cent of cases, and of these 235 cases one third were born prematurely.

Ford (27) is convinced that congenital diplegia, which constituted 84 per cent of the 280 cases of infantile spastic palsies examined, cannot be attributed to meningeal haemorrhage at birth, but is the result of various pathological processes of intrauterine origin.

In agreement with this idea Batten maintains that congenital chronic diplegia may be due to four pathological conditions:

        Atrophic lobar sclerosis.
        Arrested development of the brain.
        Intrauterine occlusion of the cerebral
           vessels.
        Various types of meningitis.

The exact cause of cerebral diplegia is still a matter for conjecture, but there are many sound reasons to support the view of Brain (6), Collier (11), Ford (27), Capon (9) and others that in the majority of cases at least its origin is not indicative of intracranial haemorrhage:

(i) In many cases the birth has been unattended by any abnormality which could be held responsible for the haemorrhage.

(ii) Meningeal haemorrhage of the newborn is frequently unilateral; and if bilateral it is generally of

unequal severity. On the other hand, cerebral diplegia
is bilaterally symmetrical.

(iii) The lesions in cerebral diplegia are more
symmetrical than one would expect to find in a case of
meningeal haemorrhage.

(iv) According to Sharpe, (59), in approximately
10 per cent of newborn children blood is found in the
cerebrospinal fluid. Yet one of every ten children does
not develop cerebral diplegia.

(v) While it is not uncommon to observe the cranial
circumference of children suffering from cerebral diplegia
to fall below the expected measurement, in children who
have suffered considerable intracranial haemorrhage the
cranium generally undergoes rapid enlargement.

(vi) The morbid anatomy of cerebral diplegia is such
that it does not conform with a traumatic origin. The
pathological changes found by Biggart (5) suggest that the
majority of the cases are probably the result of a degen-
erative process. In many cases the brain is small and
the convolutions are atrophic in patches. Histologically,
the findings signify degeneration. Cell counts reveal
the absence of many cortical cells, and imperfect develop-
ment of others. A proliferation of the glial cells is
usual.

Collier (11) at the conclusion of his paper submits "that the essential anatomical cause of diplegia is a primary degeneration of the cerebral neurons from causes which are at present elusive, with the exception of rare cases in which syphilitic infection of the brain is certainly a provoking agent."

This conception of the pathogenesis seems to be compatible with all the pathological evidence which has hitherto been recorded, and to explain best the symptomatology and clinical aspects of cerebral diplegia.

As the clinical features of the disease are not generally apparent until the child is a few months old, it is quite conceivable that the causative toxin or infection is not present in utero, but affects the child during its first few months.

Crothers and Putman (26), writing on the subject of birth injuries to the spinal cord, deprecate the use of unphysiological force in obstetrics, and consider this is the cause of many cases of flaccid paraplegia.

While the consensus of opinion, mainly based on pathological evidence, points to a primary neuronic degeneration to be the cause of cerebral diplegia, one is forced to conclude from the findings shown in this and other works, that intracranial haemorrhage at birth accounts

for a proportion of the remaining types of infantile
cerebral paralysis.

Amentia associated with Cerebral Paralysis. Most
medical men are familiar with the clinical syndrome in
children of spastic paralysis associated with some degree
of mental deficiency.

The frequency of this syndrome has recently been
investigated by Schroeder (53) who showed that of the 5000
children with behaviour problems there were only 146 cases
of infantile cerebral palsy. From these figures it must
be assumed that of all the aments only a small proportion
of them suffer from cerebral paralysis.

It is interesting to find that most authorities agree
that a similar small proportion of aments are due to birth
injury. In fact, it is difficult to imagine the location
of traumatic damage to the brain which prevents the
development of the brain tissue responsible for intelli-
gence and yet does not injure the motor cells of the
cortex.

In this series the associated syndrome of amentia
and cerebral paralysis was seen in Case O, which supports
the suggestion that the intracranial haemorrhage present
at birth was responsible for the subsequent amentia.

In agreement with this view, Herd (37) writes: "The

presence of a paralytic condition in association with
mental defect always involves the presumption of an
environmental causation after fertilization, probably
trauma at birth, or a meningeal or polio-cephalitic in-
flammation."

The degree of mental deficiency associated with
paralysis ranges from complete idiocy to a mild degree
of backwardness. Many of these cases, as shown by Doll,
Phelps and Melcher (20), show rapid mental improvement
when the orthopaedic handicap is removed. Schroeder has
shown that the differences in behaviour between the birth
injured group and birth palsied group are largely due to
the physical defects of the latter group.

Epilepsy.

Whether cerebral birth trauma bears any relation to
this disease is a question which for the present remains
debatable, since the exact cause of this relatively common
affliction still remains unknown.

The causes of true epileptiform convulsions have
been classified under different headings by all writers
since the time of Galen.

In the past, writers have discussed at length their
views on the various etiological factors appertaining to
epileptic patients. It is not proposed to deal with these

causes in detail, but a brief resume will presently be furnished. The following classification is adapted from that of Russell Brain (6):

ETIOLOGY:

1. General Causes. Including exogenous poisons, anox-
        aemia, disorders of metabolism or endocrines.

  (a) Age and Sex: Almost all true cases of epilepsy
            arise during the growth and development of the
            brain. Females are slightly more subject to
            the disease.

  (b) Heredity: Inherited predisposition is of impor-
            tance.

  (c) Metabolic: Impairment of general health fre-
            quently precedes an attack of epilepsy.

  (d) Psychological causes: In persons so disposed
            direct transmission is infrequent.

2. Local Causes.

  (a) Increased intracranial pressure.

  (b) Inflammatory conditions.

  (c) Traumatic damage at birth or in later life.

  (d) Congenital abnormalities, including diplegia,
            tuberose sclerosis and porencephaly.

  (e) Degenerations of the brain.

  (f) Cerebral circulatory disturbances.

3. Immediate cause of attack.

There is general agreement that the ordinary major

epileptic fit is the outcome of discharges of the cortical
nerve-cells throughout the middle physiological level of
both hemispheres, which is caused by a transitory loss
of power of the inhibitory cortical motor cells. It is
probable, according to Kinnear Wilson (70), that there is
another element in epileptic discharges than merely a
temporary cessation of inhibition from a higher level.

The precise mechanism which produces an epileptic
seizure is still obscure. It has long been held that the
underlying cerebral disturbance was of vascular origin.
Hughlings Jackson (41) put forward the following hypo-
thesis: "The pathology of most cases of epilepsy is that
it is the plugging of small cerebral arteries and its
consequences." This authority considered that the so-
called exciting causes of epilepsy were only the premature
development of a fit nearly due. Several observers have
actually witnessed cerebral vascular changes in the brain
of an experimental epileptic. Kinnear Wilson (70) des-
cribes these changes as follows: "Sudden blanching of
the cortex and pial vessels and the arrest of cerebral
pulsation are at once succeeded by pronounced hyperaemia
and venous engorgement during the convulsive movements."

The investigation of Berger in 1929 made it possible
to record changes in the electrical potential of the human

brain; this has done much to disprove Jackson's theory
of local ischaemia. Each cortical epileptic seizure
produces a characteristic fluctuation of the changes in
the electric potential of the brain or a dysrythmia.
While at present the exact interpretation of such dys-
rythmia in terms of functions of neurones is impossible,
according to Brain this recent work supports the view that
the physiological basis of a convulsion is a discharge
of cortical neurones rather than primarily impairment or
loss of cortical function.

Association of Epilepsy with Cerebral Birth Trauma. In
view of the fact that the immediate causation of an epi-
leptic attack is not properly understood the role played
by birth trauma must remain an open question. An injury
to the head at birth with subsequent intracranial haemorr-
hage, might eventually produce meningo-cerebral adhesions
with a gliosis surrounding the fibrous patch. The assoc-
iated adhesions in the brain substance thence may lead to
deformity of the adjoining brain substance, and hence the
possibility of epilepsy supervening. This hypothesis,
however, cannot hold as it has been shown by many workers
that the presence of a brain scar is in itself not suffic-
ient to produce epileptic attacks. Biggart maintains
that some associated constitutional error, which he calls

the epileptic diathesis, must also be present.

Starr (62) has drawn attention to the frequency with which a history of severe convulsions in infancy is found among epileptics. Ehrenfest (23) agrees, and maintains that if birth trauma can cause convulsions then it also causes epilepsy.

Epilepsy is relatively commoner amongst first-born children than among later members of the family, which suggests that the increased liability of the first child to cerebral birth trauma might be a causative factor.

There is no constant pathological change to be found in the brains of epileptics. Some show multiple patchy sclerosis, others diffuse gliosis of the cortex, others sclerosis of Ammon's horn. But absence of uniformity in their character and location has prevented their being accepted as the lesion of the disease. Thus, on pathological grounds there is no evidence for or against an origin in birth trauma.

In view of the present lack of exact knowledge of the disease the interpretation of the results of experimental investigations must act as guide in an attempt to correlate the relation of birth trauma and epilepsy.

Dayton (17) has shown a significant association between abnormal labour and the development of a child who

is emotionally unstable. Although it is true that exceptions are not uncommon it is generally agreed that most epileptics are emotionally unstable; on the other hand all emotionally unstable children are not epileptics.

Doll, Phelps and Melcher (20) in their study of cerebral birth trauma had no true cases of epilepsy in their series.

The absence of any history of epilepsy in the children of this series who were subjected to cerebral birth trauma and also in those who had suffered convulsions at birth does not exclude these circumstances as possible contributory factors, as epilepsy rarely develops before the years of puberty.

FINAL   CONCLUSIONS.

The results of this investigation suggest that all cases of intracranial haemorrhage of the newborn cannot be ascribed to cerebral birth trauma.

Evidence of cerebral birth trauma in the form of dural lacerations is found in a large proportion of infant post mortems. In this series the incidence was 47 per cent.

The following factors have been shown to predispose to the occurrence of cerebral birth trauma:

Instrumental and Breech deliveries.

The delivery of Primigravidae.

The delivery of Post Mature Infants.

The prolongation of Maternal Labour.

While the small number of cases examined do not permit any dogmatic statement, it would seem that convulsions at birth in the absence of intracranial haemorrhage, do not commonly predict any abnormal development in later life.

Intracranial haemorrhage at birth may result in the production of various types of assymetrical paralysis, a proportion of which cases have associated traumatic amentia.

In this selected series, the incidence of traumatic amentia following childbirth was 2.2 per cent.

There is no evidence to suggest that epilepsy is a manifestation of cerebral birth trauma.

A P P E N D I X.

Key to Pathological Table:

Delivery:
S.V.	-	Spontaneous Vertex.
C.S.	-	Caesarean Section.
L.F.	-	Low Forceps.
M.F.	-	Mid Forceps.
H.F.	-	High Forceps.
S.B.	-	Spontaneous Breech.
M.B.	-	Manual Breech.

Time of Death:
S.B.	-	Still Birth.
I.U.D.	-	Intrauterine Death.
D.	-	Days
H.	-	Hours.

Cord:
N	-	Examined and found normal.
H	-	Haemorrhage of Cord.

Tentorial Tearing:
C	-	Complete.
I	-	Incomplete.

CLINICAL AND PATHOLOGICAL FINDINGS IN 100 CASES.

The undernoted is a tabular summary of the Pathological findings:

| | CLINICAL | | | | | PATHOLOGICAL | | | | | | Intercranial Haemorrhage | | | | | | | | | Dural Lacerations | | |
| | | | | | | | | | | | | | | | | | | | | | Tentorial | | Falx |
Case Number.	Parity.	Labour (in hours)	Delivery.	Weight (kilos).	Time of Death.	Caput Succedaneum.	Cephalhaematomata.	Moulding.	Fractures.	Oedema	Brain Tissue Congestion.	Petechial.	Subarachnoidal.	Supratentorial.	Intratentorial.	Subtentorial	Into Falx.	Mixed Type.	Haem. into Cord.	Fatal Haemorrhage.	Right	Left	Falx
1	2	15	Sp.V	1.6	6 H	−	−	−	−	+	+	−	−	−	−	+	+	−		−	−	−	+
2	8	60	S.V.	4	S.B.	+	−	+	−	−	+	+	−	+	+	+	+	+		+	C	C	+
3	1	13½	S.V.	2.8	S.B.	+	−	−	−	−	+	+	−	−	−	+	−	−		+	I	−	+
4	2	22	S.V.	1.8	S.B.	+	−	−	−	+	−	−	−	−	−	+	−	−		−	I	−	+
5	2	1½	S.V.	3	S.B.	−	−	−	−	−	+	+	−	−	−	−	−	−		−	−	−	−
6	3	58	M.B.	3.6	S.B.	−	−	+	−	−	+	+	−	+	−	+	−	+	N	+	I	I	+
7	7	0	C.S.	3.4	IU.D	−	−	−	−	−	−	−	−	−	−	−	−	−		−	−	−	−
8	5	0	C.S.	.4	14 H	+	−	−	−	+	−	−	−	−	−	−	−	−		−	−	−	−
9	5	0	C.S.	.1	19 H	−	−	−	−	+	−	−	−	−	−	−	−	−		−	−	−	−
10	1	45	M.F.	4.6	S.B.	+	+	+	−	−	+	+	+	+	−	+	−	+		+	C	C	+
11	4	13	H.F.	4.1	S.B.	+	−	+	−	−	−	−	−	+	−	+	+	+		+	C	C	+
12	1	42	H.F.	4.7	S.B.	−	+	+	−	−	+	+	−	+	−	+	−	−		+	I	−	−
13	1	9	S.V.	1.1	IU.D	+	−	−	−	−	−	−	−	−	−	−	+	−		−	−	−	−
14	1	22½	S.V.	2.3	I.U.D	+	−	−	−	−	+	−	−	−	−	−	−	−		−	−	C	−
15	1	0	C.S.	2.2	I.U.D	−	−	−	−	−	−	−	−	−	−	−	−	−		−	−	−	−
16	3	6	S.V.	1.7	I.U.D	−	−	−	−	−	+	+	−	−	−	+	−	−		−	I	I	−
17	1	14	S.V.	2.8	72 H	−	−	−	−	−	−	+	−	−	+	+	−	−		−	−	−	−
18	1	10	L.F.	2.1	S.B.	+	−	−	−	+	+	+	+	−	+	+	−	−		+	C	I	−
19	1	15	S.V.	2	24 H	−	−	−	−	−	+	+	−	−	+	+	+	−		−	−	−	−
20	1	13½	S.V.	1.2	72 H	−	−	−	−	−	+	+	−	−	+	−	+	−		−	−	−	−
21	3	8	S.V.	1.6	12 H	−	−	−	−	−	+	+	−	−	+	−	+	−		−	−	I	−
22	1	2	S.V.	1.9	24 H	−	−	+	−	−	+	+	−	−	+	−	+	−		−	−	−	−
23	1	105	H.F.	3.9	S.B.	+	−	−	−	−	+	+	−	−	+	+	+	−		−	−	−	−
24	1	45	S.V.	2.1	S.B.	−	−	+	−	−	+	+	−	−	+	−	+	−		−	−	−	−
25	7	7½	L.F.	3.6	S.B.	−	−	+	−	−	−	−	−	−	−	+	−	−		−	−	−	−
26	2	13	M.B.	1.4	S.B.	−	−	+	−	+	−	+	+	−	+	−	−	−		−	−	−	−
27	1	1	S.V.	1.8	S.B.	−	−	−	−	−	−	−	−	−	+	−	+	−		−	−	−	−
28	1	33	H.F.	3.7	S.B.	+	−	+	−	−	−	−	−	−	+	−	+	+	N	−	−	I	−
29	12	11	L.F.	5.1	S.B.	+	−	+	−	−	+	+	−	−	+	−	+	+		−	C	C	+
30	7	12½	M.B.	3.9	S.B.	+	−	−	−	−	+	+	−	−	−	−	−	−	N	−	C	C	−

CLINICAL and PATHOLOGICAL FINDINGS in 100 CASES.

| | CLINICAL | | | | | PATHOLOGICAL | | | | | | | Intercranial Haemorr. | | | | | | | | Dural Lacerations | | |
| | | | | | | | | | | | | | | | | | | | | | Tentorial | | Falx |
Case Number	Parity	Labour (in hours)1	Delivery	Weight (kilos)	Time of Death	Caput Succedaneum	Cephalhaematomata	Moulding	Fractures	Oedema	Brain Tissue Congestion	Petechial	Subarachnoidal	Supratentorial	Intratentorial	Subtentorial	Into Falx	Mixed Type	Haem. into Cord	Fatal Haemorr.	Right	Left	Falx
31	2	0	C.S.	1.8	S.B.	-	-	-	-	+	-	-	+	-	-	-	-	-		-	-	-	-
32	1	4½	S.V.	1.5	S.B.	+	-	+	-	-	-	+	-	-	-	-	-	-		-	-	-	-
33	2	3	S.V.	1.3	S.B.	+	-	+	-	-	+	+	-	-	-	+	-	-		-	-	-	-
34	1	25	M.F.	3	I.U.D	+	-	-	-	-	+	+	-	-	-	-	-	-		-	-	C	-
35	9	13	S.V.	2	6 H	-	-	+	-	-	+	+	+	-	-	-	+	+		-	-	I	-
36	1	19½	S.V.	1.1	S.B.	-	-	+	-	-	-	+	-	-	-	-	-	-		-	-	-	-
37	5	4½	S.V.	1.6	12 H	-	-	-	-	+	-	+	-	-	-	-	-	-		-	-	-	-
38	3	8	L.F.	4	S.B.	+	-	-	-	-	-	-	-	+	-	+	-	-	N	+	C	C	-
39	6	8½	S.V.	2.2	S.B.	-	-	-	-	-	+	+	-	-	-	-	-	-	N	-	-	-	-
40	4	19½	S.V.	2.1	S.B.	+	-	+	-	+	-	+	-	-	-	+	-	-		-	C	I	-
41	9	47½	M.B.	5.4	S.B.	+	-	-	-	-	+	+	-	+	-	+	-	+	N	+	C	I	+
42	12	6	M.B.	3.8	I.U.D	+	-	-	-	-	-	-	-	-	-	+	-	-		-	I	I	-
43	3	9	S.V.	2	S.B.	-	-	-	-	+	+	+	+	-	-	-	+	-		+	-	-	-
44	3	8½	S.V.	1.7	S.B.	-	-	+	-	-	+	+	-	-	-	-	-	-		-	-	-	+
45	1	20	S.V.	.9	S.B.	+	-	-	-	+	-	-	-	-	+	-	+	-		-	-	-	-
46	2	5½	S.B.	1.5	S.B.	-	-	-	-	-	+	+	+	-	-	-	+	+		-	-	-	-
47	1	16	M.F.	3.6	S.B.	+	-	+	-	-	-	-	-	+	+	-	-	+		+	I	I	-
48	5	6½	M.B.	2.5	S.B.	-	-	+	-	+	-	+	-	+	-	+	-	+		+	I	C	-
49	3	5½	S.V.	2.5	6 H	+	-	-	-	+	-	-	-	-	+	-	+	-		+	-	-	+
50	3	5½	S.V.	2	84H	+	-	-	-	-	-	-	-	-	+	-	+	-		-	-	-	-
51	5	12	M.F.	4.1	S.B.	+	-	-	-	-	-	-	-	+	-	+	+	+	H	+	-	-	-
52	3	11	S.V.	1.9	108H	-	-	-	-	-	-	+	-	-	-	-	-	-		-	-	-	-
53	1	14	M.B.	3.1	S.B.	-	-	-	-	-	-	-	+	-	+	+	+	-		+	C	C	+
54	1	16½	M.F.	3.4	S.B.	+	-	+	-	-	+	+	-	+	-	+	-	-		-	-	-	-
55	7	31½	S.V.	3.4	I.U.D	+	-	+	-	-	+	-	-	-	-	-	-	-	N	-	-	-	-
56	1	66	M.F.	3	36H	+	-	+	-	-	-	-	+	-	+	+	+	+	N	+	C	I	+
57	6	6½	M.B.	3	S.B.	-	-	-	-	-	-	-	+	-	+	+	+	+	N	+	C	I	+
58	1	1¼	M.B.	3.1	S.B.	-	-	-	-	-	-	+	-	-	-	-	-	-	N	-	I	I	-
59	6	5¼	S.B.	2.6	S.B.	-	-	+	-	+	-	+	+	+	-	+	+	+		+	-	-	+
60	2	5	S.V.	2	6H	-	-	+	-	-	+	+	-	-	-	-	-	-		-	-	-	-
61	1	10	S.V.	1.2	15D.	-	-	+	-	-	-	-	-	-	-	-	-	-		-	-	-	-
62	5	4	S.V.	1.1	S.B.	-	-	+	-	+	+	+	-	-	+	-	+	-		-	-	-	-
63	1	6	S.V.	1.9	16D	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-	-	-
64	1	21¼	M.F.	3.7	48H.	+	-	-	-	-	-	-	-	-	-	-	-	-	N	-	-	-	-
65	8	21½	S.V.	2.1	1 H.	-	-	-	-	+	+	+	-	-	-	-	-	-		-	-	-	-

CLINICAL and PATHOLOGICAL FINDINGS in 100 CASES.

Case Number	Parity	Labour (in hours)	Delivery	Weight (kilos)	Time of Death	Caput Succedaneum	Cephalhaematomata	Moulding	Fractures	Oedema	Brain Tissue Congestion	Petechial	Subarachnoidal	Supratentorial	Intratentorial	Subtentorial	Into Falx	Mixed Type	Haem. into Cord.	Fatal Haemorrhage	Tentorial Right	Tentorial Left	Falx
66	2	7½	S.B.	1.7	18D	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-	-	-
67	3	6½	S.V.	2.6	I.U.D	-	-	-	--	-	+	-	-	-	-	-	+	-		-	-	-	+
68	1	4	S.B.	1.5	IU.D	+	-	-	-	-	+	-	-	-	-	-	+	-		-	-	I	-
69	1	17	S.V.	2.9	I.U.D	-	-	+	-	-	+	-	-	-	-	-	-	-		-	I	C	-
70	1	10½	S.V.	1.2	S.B.	-	-	-	-	+	-	+	-	-	-	-	-	-		-	-	-	-
71	2	1	S.B.	2.1	S.B.	-	-	-	-	-	+	-	-	-	-	-	-	-		-	-	-	-
72	2	25½	M.B.	4.1	S.B.	-	-	-	-	-	+	+	-	+	-	+	+	+		+	C	C	-
73	1	7	S.V.	1.1	S.B.	+	-	-	-	-	+	+	-	-	-	-	-	-		-	-	-	-
74	1	42	L.F.	2.2	6 H.	+	-	-	+	-	-	-	+	-	+	+	+	+	N	+	C	-	-
75	1	84	H.F.	3.8	S.B.	+	-	+	-	-	+	+	-	+	-	+	+	-	N	+	C	I	-
76	7	13	S.V.	2.7	I.U.D	-	+	-	-	-	+	+	-	-	-	-	-	-		-	-	-	+
77	1	3	L.F.	3.3	I.U.D	-	-	+	-	-	+	-	-	-	-	-	+	-		-	I	I	-
78	1	6	S.V.	2.8	7 D.	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-	-	-
79	8	17	S.V.	1.1	S.B.	-	-	-	-	-	+	+	+	-	+	-	+	+		+	-	-	-
80	1	12½	S.V.	1.6	I.U.D	-	-	+	-	-	+	-	-	-	-	-	-	-		-	-	-	-
81	2	19	H.F.	3.8	S.B.	+	-	+	-	-	+	+	-	+	-	+	-	+	N	+	C	C	+
82	6	5	S.V.	2.1	S.B.	+	-	-	-	+	+	+	-	-	+	-	-	-		-	-	-	-
83	1	0	C.S.	1.1	2 H	-	-	-	-	+	-	-	-	-	-	-	-	-		-	-	-	-
84	1	0	C.S.	1.2	2 H	-	-	-	-	+	-	-	-	-	-	-	-	-		-	-	-	-
85	1	22	S.B.	2.9	S.B.	+	-	+	-	-	+	+	-	-	-	-	-	-		+	-	I	-
86	2	7	S.B.	2.5	I.U.D	-	-	-	-	+	+	+	-	-	-	-	+	-		-	-	-	-
87	5	9	S.B.	1	S.B.	+	-	-	-	-	-	-	-	-	-	-	-	-		-	-	-	-
88	4	16	S.B.	3.2	I.U.D	-	-	-	-	-	+	+	-	-	-	-	+	-	N	-	-	-	+
89	1	5	S.V.	1.3	5 D	-	-	-	-	-	+	+	-	-	-	-	-	+		-	-	-	-
90	1	50	M.B.	3.2	S.B.	+	-	-	-	-	+	+	-	+	-	+	-	+	N	+	C	C	+
91	2	3	M.B.	2.4	I.U.D	-	-	-	-	-	-	-	-	-	-	-	-	-		-	-	-	-
92	9	3½	S.V.	1.4	7 D	-	-	-	-	+	-	-	-	-	-	-	-	-		-	-	-	-
93	2	5	S.B.	1.7	28 H	+	-	-	-	+	-	-	-	-	-	-	+	-		-	-	-	-
94	10	10	S.B.	1.8	3 D	-	-	-	-	+	-	-	-	-	-	-	-	-		-	-	-	-
95	1	85	M.F.	3.4	S.B.	+	-	+	-	-	+	+	-	+	-	+	-	+	N	+	C	C	+
96	1	30	S.V.	3.3	S.B.	+	-	-	-	-	+	+	-	+	-	+	-	+		+	C	I	-
97	3	21	S.B.	1.8	S.B.	-	-	-	-	-	+	+	+	+	-	+	-	+		+	I	C	-
98	3	21	S.V.	1.8	7 H	-	-	-	-	-	-	-	-	+	-	+	-	+		-	I	-	-
99	1	19	M.B.	2.4	S. B	-	-	-	-	-	+	+	+	+	-	+	-	-		-	I	C	-
100	3	19	S.V.	2.1	S.B.	+	-	-	-	-	+	+		-	-	-	-	-		-	-	-	-

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