General Œdema of the Fœtus.*

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(Received February 22, 1922.)

Introduction.

It is probable that the condition described as General Œdema of the Fœtus occurs more commonly than has formerly been believed.

In this communication an attempt is made to review our present knowledge of this morbid state. Moreover, notes are given regarding eight cases of stillbirth, seven of which showed general œdema of the fœtus; while the eighth, though not exhibiting general œdema, was characterized by several features usually associated with that condition.

In almost every case hydramnios is also present.

General œdema of the fœtus is incompatible with post-natal life, the longest reported period of extra-uterine existence being six days (Bourret and Lathoud, quoted by Crozier [6]). In a large majority of cases death occurs during or immediately after birth.

Definition.

Ballantyne's definition [3] of general fœtal œdema is undoubtedly the best. It is as follows:—

> "A rare condition of the fœtus, characterized by general anasarca, by the presence of fluid effusions in the peritoneal, pleural and pericardial sacs, and usually by œdema of the placenta; and resulting in the death of the fœtus or infant before, during or very soon after birth."

It must be clearly noted that the dropsical state called "hydrops sanguinolentus fœtus," which sometimes supervenes upon intra-uterine death of the fœtus, and which is a secondary event, is not included in this definition.

Certain cases, such as that reported by Link,[22] do not strictly conform with Ballantyne's definition; for the fœtus may be born before it has fully assumed the œdematous state.

* The expenses of this investigation have been defrayed by a grant from the Medical Research Council.

Etiology and Pathology.

The primary causes may be sought in paternal or maternal disorders, or in a morbid state of the fœtus or placenta. There may even be a conjunction of one or more of these causal factors, each of which will now be considered in turn.

Maternal Causes.

Ballantyne laid emphasis on what he believed to be the frequency with which œdematous fœtus are born to women well advanced in the child-bearing period, and especially to those who present an unsatisfactory obstetrical history in so far as the fœtus has been concerned. If this were a fact it might support the possibility of some maternal toxæmia being a causal factor. But it must be noted that, since 1901, there have been many cases reported among younger and apparently more healthy mothers. In a review of 30 cases occurring between 1901 and 1915—cases in which the age of the mothers was stated—Schumann [33] draws attention to the fact that 17 were between 25 and 30 years of age; nine were between 30 and 35; while only four were over 35 years of age.

The health of the mother during the pregnancy which terminates in the birth of an œdematous fœtus is, however, generally bad.

In Schumann's list, mentioned above, there was, in a majority of instances, evidence of a maternal toxæmia, namely, dropsy, albuminuria or excessive vomiting. Further, finding vacuolation and degenerative swelling in the chorionic cells, Schumann assumed that maternal toxins had passed into the fœtal circulation, producing results somewhat similar to those seen in the mother. Lamouroux, quoted by Crozier,[6] in 1825, and Billiard, quoted by Crozier,[6] in 1828, were impressed by the association of maternal and fœtal œdema. Brœckhuizen, quoted by Schmidt and Mönch,[30] in 1908, estimated that in 30 per cent. of these cases maternal albuminuria and œdema were present, but that in a few the mother appeared to be quite healthy. Rautmann,[26] Liegner [20] and Schmidt and Mönch,[30] arrived at the conclusion that maternal toxæmias are causative; and the last mentioned quoted, in support of their view, the statement of Doi,[8] namely, that in the blood of pregnant women, especially of those with the so-called "pregnancy kidney," and even more definitely in that of eclamptic patients, erythroblasts may be found, and that these disappear during the puerperium. It is inferred that a toxin which can stimulate the maternal blood-forming organs can stimulate those of the child also; in this way the fœtal erythroblastosis observed by these workers may be explained.

In opposition to this hypothesis it must be remembered that

maternal toxæmia is relatively common, whilst fœtal œdema is comparatively rare.

Special attention has been drawn to the possible importance of the following maternal diseases.

Syphilis. In a majority of cases of fœtal œdema, syphilis of the mother can be excluded. In a few cases the maternal blood may give a positive Wassermann reaction, and in still fewer there may be more positive evidence of syphilis. In Liegner's case [20] the mother had leucoderma and a relapsing exanthem, in addition to a strongly positive Wassermann reaction. In the case reported by Fleischmann and Wolff [12] the maternal Wassermann was positive and that of the child negative; the infant showed no signs of syphilis, but the mother, who died during the puerperium of the next pregnancy, was found to have syphilitic aortitis. With the single exception of Lahm's case,[19] spirochætes have not been found in the fœtal tissues, nor in the œdematous placenta. Hence, if syphilis enters at all into the etiology of general fœtal œdema it must be through the medium of the toxins arising within the mother as the result of a syphilitic infection, rather than by the direct action of the specific organisms themselves.

Albuminuria. It has already been stated that during the pregnancy which results in the birth of an œdematous fœtus the mother frequently suffers with albuminuria and œdema. Doubtless the unusual abdominal distension caused by a large and œdematous fœtus and placenta, together with the excess of liquor amnii which so frequently accompanies general fœtal œdema, is partly responsible for these maternal conditions.

That the fœtus may often suffer as a result of maternal albuminuria is well recognized. In eclampsia the life of the child is in actual danger.

Grulee [15] has recorded the results of a post-mortem examination on a child born alive to an eclamptic mother five days previously. The kidneys and liver exhibited an acute parenchymatous degeneration, and there were hæmorrhages into the meninges, lungs and right perirenal tissue: the urine contained a few leucocytes and red blood-cells and an occasional granular cast. This author quotes the case of Vecchi, in which an infant showing general fœtal œdema and "peritonitis" was born to an eclamptic primigravida, and also the cases described by Gillmore.[14]

On the other hand, Paul Bar [4] has shown that in 17 children born to eclamptic mothers, definite kidney lesions other than congestion were found only in two.

General fœtal œdema is a much more rare state than eclampsia, and more frequently occurs in multiparæ, whilst eclampsia is most common in primigravidæ.

It does not seem, therefore, that there is any direct association between eclampsia or albuminuria and fœtal dropsy. We must assume that in those cases in which the two states are associated there must be some third, and at present unknown, factor involved.

Anæmia. Ballantyne [2] has indicated the frequency with which the mothers of œdematous fœtus showed no special symptoms of disease, but were anæmic. This observation has been supported by several more recent reports. Wienskowitz [42] has recorded a case in which the mother was very anæmic, and he regarded this anæmia as the probable cause of a maternal toxæmia. Nevertheless the mother of an œdematous child is not always anæmic. So, in those cases in which the two conditions—maternal anasarca and fœtal œdema—are associated another cause must be sought.

It is reasonable, therefore, to assume that in all cases in which maternal disease or toxæmia is present there is some other, and at present unknown, factor concerned, either alone or as a link in the chain of events. It has been suggested (Schmidt and Mönch [30]) that such a link may be constituted by an inherited capillary narrowness, which is said to be a feature of infantilism in the mother and to be associated with orthostatic albuminuria and the occurrence of eclampsia. Such an hypothesis needs convincing proof.

Paternal Causes.

Various writers have suggested that the following paternal disorders may produce a diseased condition of the fertilized ovum :

(*a*) Syphilis.
(*b*) Œdema and icterus.
(*c*) Alcoholism.
(*d*) Anæmia.
(*e*) Lead poisoning.

While it is well to bear in mind the possibility of paternal influences, no reliable evidence is at present forthcoming as to the importance that should be attached to them.

Fœtal Causes.

Causal factors in the fœtus itself may be considered in two groups—mechanical and non-mechanical.

The chief objection to this subdivision lies in the vagueness of the word "mechanical." Circulatory disorders may be caused by influences which are not visible to the naked eye : they may be physiological rather than anatomical. Hence it is possible that with further knowledge certain of the cases now regarded as non-mechanical may come to be included with those of mechanical origin.

It is common knowledge that the post-natal causes of œdema are many and various. Renal and cardiac lesions, and states of anæmia and of cachexia may be the precursors of more or less general œdema. Local œdemas may result from discrete venous or lymphatic obstructions, or from trauma. Moreover, there are œdematous conditions of obscure origin, such as angio-neurotic œdema and hereditary œdema.

It is possible that these factors which give rise to œdema in the child after birth may also produce œdema in the fœtus during intra-uterine life, when, owing to the special characteristics of fœtal structure and physiology, œdema may be more easily produced.

Cases of Mechanical Origin. In this group may be placed those cases in which some structural defect has been found—whether this were a developmental error or the result of intra-uterine disease or injury—which could be regarded as the cause of an obstruction in the vascular or lymphatic circulation of the fœtus. The following are examples of such lesions:—

(*a*) Cardiac anomalies: for example, closure of foramen ovale (Lawson Tait [41]) and acardiacus acephalus.
(*b*) Umbilical cirrhosis (Andrews [1]; de Sinéty, quoted by Crozier [6]).
(*c*) Umbilical hernia (O. Fischer [9]).
(*d*) Absence of lymphatic system (Smith and Birmingham [38]).

In estimating the influence of mechanical factors it must be remembered that in many cases of fœtal œdema, believed by those who have recorded them to be due to mechanical obstructions, the post-mortem reports have been incomplete, and the actual way in which the lesion found produced the œdematous state has not always been manifest.

Certain authors, notably Flamma [11] and Seyffert,[34] have put forward the view that in all cases of general œdema a mechanical cause can be discovered if a full post-mortem examination be made. This statement appears to be exaggerated and to lack support on general pathological principles. Furthermore, there are many well-recorded cases in which a thorough search to find a mechanical cause of the œdema was fruitless.

Examples of fœtal œdema undoubtedly secondary to vascular obstruction of mechanical origin have recently been reported by Flamma,[11] Seyffert [34] and Link.[22]

Flamma [11] demonstrated cardiac deformities in his case which he regarded as the cause of the general œdema. The heart was double the normal in size, chiefly on account of hypertrophy of the right auricle and ventricle, and to some extent of the left ventricle; there was marked congenital hypoplasia of the left

ventricle; the pulmonary artery was enormously dilated, especially beyond the bifurcation of the main trunk; the aorta was normal; the ductus arteriosus was widely patent, and equal in size to the aorta; the foramen ovale was pervious, but a little less so than in the normal heart; the ventricles communicated at the upper limit of the inter-ventricular septum, and the left auriculo-ventricular opening was much smaller than the right.

Seyffert [34] found in his case an enormous tumour of the left lung which had displaced the heart to the right.

In Link's case,[22] which, as stated, did not conform with Ballantyne's definition, in that there was no accumulation of fluid in the pleural, pericardial and peritoneal cavities, there was an old calcified thrombus in the inferior vena cava extending into both renal veins and into the left suprarenal vein, in association with œdema of the skin, especially over the abdomen, and of the retro-peritoneal tissues. There were several other deformities, including hydrocephalus, hydrencephalocele and cervical spina bifida. The placenta and umbilical cord were normal. Hydramnios was present. This case may be regarded as an example of a local œdema due to a local cause.

It is probable that in many of the earlier cases recorded in which lesions such as narrowness of foramen ovale (Lawson Tait [41]), umbilical hernia and short cord (O. Fischer [9]) and congenital absence of ureter (Stevens [39]) existed, the anomalies were only chance associations, or related to a common cause. It has been stated that a congenital defect of one type or another is found in about 10 per cent. of all stillbirths, so it is only to be expected that a certain proportion of œdematous fœtus will show malformations probably having no connexion with the œdematous state. This was obvious in the cases of Behm (spina bifida), quoted by Ballantyne,[3] Vecchi (polydactyly) and Commandeur (imperforate anus), both quoted by Schumann.[33]

Crozier, indeed, has aptly remarked: "It is difficult to see an immediate relationship between the anasarca and the reported fœtal diseases . . . . . for one or all may be absent, and there is no one pathognomonic feature."

Cases of Non-Mechanical Origin. This subdivision includes those cases in which there is no macroscopical evidence of vascular or lymphatic obstruction.

In this class the features of excessive hæmatopoiesis predominate; that is to say, there is enlargement of liver and spleen with histological appearances of increased blood-formation of an idiopathic or reparative nature.

Recent reports upon examples of general fœtal œdema, and especially those of German investigators, have given full accounts

of the pathological conditions found within the fœtus and its placenta. In the cases reported under this heading since 1910, when Schridde [31, 32] first drew attention to certain hæmatological features to be described below, there has been a fairly close agreement in regard to pathological findings. As a result of these observations the use of the adjective "toxic" in connexion with the causative agents has become more justifiable. Whether the toxin arises primarily in the mother or in the fœtus is still unsettled.

The actual appearances noted in the fœtal structures lend support to the belief that they represent the fœtal response to a toxæmia; and this view is supported by the findings in certain post-natal conditions believed to be toxæmic in origin, such as anæmia pseudoleukæmia infantum. Schridde [31] has demonstrated the presence of deposits of greenish-yellow, pointed pigment-particles in the epithelial cells of the kidney tubules. Fischer [10] compares this phenomenon with the deposit of pigment which occurs in the same locality in cases of experimental poisoning, and employs the comparison to support the view of the "toxic" origin of general œdema of the fœtus.

In order properly to understand the question of hæmatopoiesis in the œdematous fœtus, an enquiry into the processes seen in the normal fœtus and syphilitic fœtus is necessary.

It is a well-recognized fact that during intra-uterine existence blood-formation occurs in several localities:

(1) The various so-called "vascular areas" of the organism produce the solid elements of the blood during early embryonic development. This process is of brief duration.
(2) At a later period the liver, the spleen and the bone-marrow take over the production of these cells, which may also be formed in certain other glands.

With regard to embryonic blood-formation in the liver, the process may be seen in progress in bays projecting from the capillaries and in the periportal tissue, especially that which surrounds the small veins. Later in ante-natal life, red-cell formation in the liver is said to occur in two ways; most commonly these cells are produced by division of preformed basophil erythroblasts; but sometimes they arise from endothelial capillary cells (Lobenhoffer [23]; M. B. Schmidt [29]).

Of these formative structures the bone-marrow is the only one which retains during adult life its power of forming blood-cells; whilst the spleen, which loses before the liver its capability for forming red cells, probably possesses in post-natal life the power of producing lymphocytes.

The examination of the livers of premature infants, and, to a

lesser degree, of the newly-born, reveals the presence of hæmatopoietic areas (M. B. Schmidt [29]; Lobenhoffer [23]); that is, clusters of erythroblasts scattered fairly evenly through the liver, but placed for the most part in the spaces between capillary walls and the hepatic cells.

It is assumed that those erythroblasts which are formed outside the vessel walls find their way into the blood-stream by amœboid movements.

The livers of syphilitic infants contain many of these cell clusters (Swart [40]); and it might be assumed that a hæmatopoietic response has been called forth owing to the anæmia which is secondary to the effects of specific toxins. In opposition to such an assumption it must be remembered that the hepatic cells of such syphilitic livers do not show the presence of iron-containing pigment. Blood-pigment in the liver might be expected if destruction of erythroblasts were taking place.

There is to be seen in the livers of dropsical fœtus, however, hæmatopoietic development which in degree quite surpasses that observed in premature and in syphilitic children: erythroblasts and myeloid cells appear in numerous clusters—"Herde" in the German literature—and to such an extent that the liver cells proper are isolated and appear as islands of varying size. Within the hepatic cells a greyish-golden pigment containing iron is found. This would suggest that some blood-cell destruction is occurring at the same time as active formation.

The periportal connective-tissue is frequently increased in quantity, and the greatest "Herde" formation is in the region of the vessels within this periportal tissue. The erythroblasts show an extensive mitosis, and their protoplasm is often basophilic. König [18] has shown that this feature may be a sign of immaturity; that is to say, regeneration of red-cells is taking place very rapidly.

The same "Herde" arrangement is frequently found, though to a lesser extent, in the kidneys of œdematous fœtus. Here they almost invariably occur in close relationship to the vessels at the junction of medulla and cortex. Until recently it was agreed that clusters of red-cell precursors do not occur in the kidneys of normal or syphilitic children. In 1920, however, Richard Bloch [5] reported the finding of typical "Herde" formations in the kidneys of a baby which had died of syphilis at the age of six weeks. These formations may also occur in the adrenals and lymphatic glands of dropsical fœtus. The spleens of fœtus showing general œdema are almost invariably enlarged; this hypertrophy is found, on microscopical examination, to be due to a myeloid proliferation. The normal lymphatic structure has almost disappeared, but myeloblasts and erythroblasts, showing extensive karyokinesis,

occur in numbers so great that the histological picture may be not unlike that of a bone-marrow preparation. The splenic pulp cells remain as a supporting framework and contain pigment, generally iron-free, though in Schridde's case[31] it gave the iron-reaction.

This author found an overgrowth of myeloblasts in the bone-marrow; and observations by recent investigators upon the blood in such cases indicate the great preponderance of erythroblasts—53 per cent. to 75 per cent. of all nucleated cells. These cells, like those occurring in the "Herde," show active karyokinesis and are frequently basophilic. Next to erythroblasts, megalocytes are most frequently found in the fœtal blood; after these, myeloblasts and myelocytes are most prominent. Leucocytes and lymphocytes are much less commonly seen. The myocardium is frequently hypertrophied.

Basing their views upon these findings, Schridde,[31, 32] and later Loth,[24] have regarded general fœtal œdema as the result of an anæmia caused by some unknown toxin, probably arising within the fœtus. W. Fischer[10] appears to incline towards the same opinion. It is unfortunate that an accurate red-cell count and hæmoglobin estimation cannot be performed in these cases, for other workers, including Rautmann,[26] who have demonstrated the occurrence of almost similar changes, are not prepared to admit that the œdema is associated with anæmia, but favour the idea that hæmatopoiesis results directly upon toxic irritation of blood-forming tissues, and is not a reparative process in relation to a toxic anæmia. Swart,[40] also, in recording the results of post-mortem examinations upon four infants has drawn attention to a state of hæmatopoietic activity which he believes followed some toxic stimulation—possibly syphilitic, though the subjects examined showed no evidence of syphilis.

The characteristic and outstanding feature, then, in general œdema of the fœtus is the extreme activity of the hæmatopoietic structures. All potential sources of blood formation are excited.

Before it was proved that the large number of round-cells seen, especially in the liver, were erythroblasts, and not lymphocytes and leucocytes, numerous writers (Klebs, quoted by Ballantyne[3]; Sänger[27]; Nachtigäller, quoted by Croizier[6]; Siefart[36]; Lahs, quoted by W. Fischer[10]) believed that they were dealing with fœtal leukæmia. Improvements in staining methods have enabled observers to show the inaccuracy of these earlier conclusions.

Of other non-mechanical fœtal conditions which may possibly be causative, it is necessary only to call attention to hyperchloridæmia and nephritis.

Hyperchloridæmia. Sauvage[28] demonstrated an increase in the

chloride content of the tissue fluids in the œdematous fœtus. It is questionable whether this condition can be regarded as causative of the œdema, or whether it is only an associated condition.

Nephritis. Sitzenfrey [37] and Lieven [21] have considered the possibility of fœtal nephritis being a causal factor, and they have demonstrated lesions which resembled those found in inflammation of the kidney. In this connexion it is well to remember that autolytic changes occur with remarkable rapidity after fœtal death, and that the appearances thus produced in the kidney are not unlike those of a nephritis—cloudy swelling of the more highly specialized epithelial cells, with nuclear karyorrhexis and pyknosis (Cruikshank [7]).

Placental Causes.

Whether the placenta plays a part in the causation of fœtal œdema is exceedingly difficult to determine; we do not know, in any particular case, whether the placental œdema is primary or secondary in so far as the general fœtal œdema is concerned.

Cases of fœtal œdema are on record in which the placenta has been reported to be normal (Nyhoff [25]; Link [22]); but it is usual to find both fœtus and placenta affected.

The dropsical placenta generally shows an œdematous swelling of the villi; in some cases there is also a connective-tissue hyperplasia (O. Fischer [9]). There is little evident change in the syncytium or cells of Langhans (Schridde [31]; Himmelheber [16]); though Schumann,[33] as previously mentioned, demonstrated in his case vacuolation and degenerative swelling in these cells. Spirochætes are not found.

If the placenta becomes œdematous, either primarily or secondarily to some maternal disorder, we may assume that the fœtal circulation will be considerably impaired thereby—a mechanical obstruction will be produced in the fœtal circulation, the fœtal arterial pressure will rise, and, if cardiac hypertrophy is not sufficient to re-establish equilibrium, venous back-pressure with general œdema will occur. The failure of fœtal excretion in such circumstances will augment the factors producing an œdematous state of the fœtus; for the fœtus which is attempting to excrete waste products through an œdematous placenta is in a state closely comparable with the adult who is endeavouring to excrete from inefficient kidneys.

Führ [13] thinks that endometritis causing chronic hypertrophy and vascular obstruction may be the cause of fœtal œdema. If this were true, though the autopsy revealed no mechanical cause within the fœtus, there would be a mechanical obstruction within that part of the fœtal circulation which is extra-corporeal, namely, within the placenta.

On the other hand, if the cause of the œdematous state originates within the fœtus it is reasonable to suppose that the placenta will be affected, just as in the cardiac œdemas of post-natal life it is the most distal portion of the circulation which first shows the effects of back-pressure.

It is unlikely that toxins arise *de novo* within the substance of the placenta; but rather that under the influence of poisons reaching it from either one side or the other, the placental tissues add to the deleterious effects of the toxin mechanically, by reason of the œdema which develops, and, chemically, because of the cellular degeneration which is almost certain to follow in a greater or lesser degree.

Diagnosis and Treatment.

In a large majority of cases diagnosis of general œdema of the fœtus is not made until the birth has taken place. The condition may be suspected, however, in the elderly mother who develops albuminuria and dropsy, whose abdomen is too large, either as a result of hydramnios, or because of the large size of the contained fœtus and placenta, and whose obstetrical history is bad—especially if it include records of one or more œdematous fœtus.

It may be mentioned that Victor Kafka [17] believes that the fœtal heart sounds can be heard clearly during the intra-uterine life of an œdematous fœtus, even though the accompanying hydramnios might be expected to cause their disappearance.

Until we become certain of the causes of the condition, and the means by which they can be recognized before they give rise to the fœtal disease, treatment cannot do more than attempt to prolong life in the newly-born child.

It has been suggested that a salt-free diet might reduce the possibilities of fœtal œdema, but there is no means of testing such treatment until ante-natal diagnosis is more exact.

After the birth of the child aspiration of fluid from the serous sacs has been recommended by Ballantyne.[2]

Fleischmann and Wolff [12] withdrew 200 ccs. of yellowish-red fluid from the peritoneal cavity of an œdematous child, which breathed very much more easily after the operation, but death followed. Such a procedure is not likely to be of permanent benefit, but it might be useful if it were combined with treatment directed against the primary, as yet unknown, cause of the condition.

DESCRIPTION OF CASES.

Case 1.

Mother. Age 27. Stated to have had valvular disease of the heart, with periodic swellings of the feet, for the past twelve years.

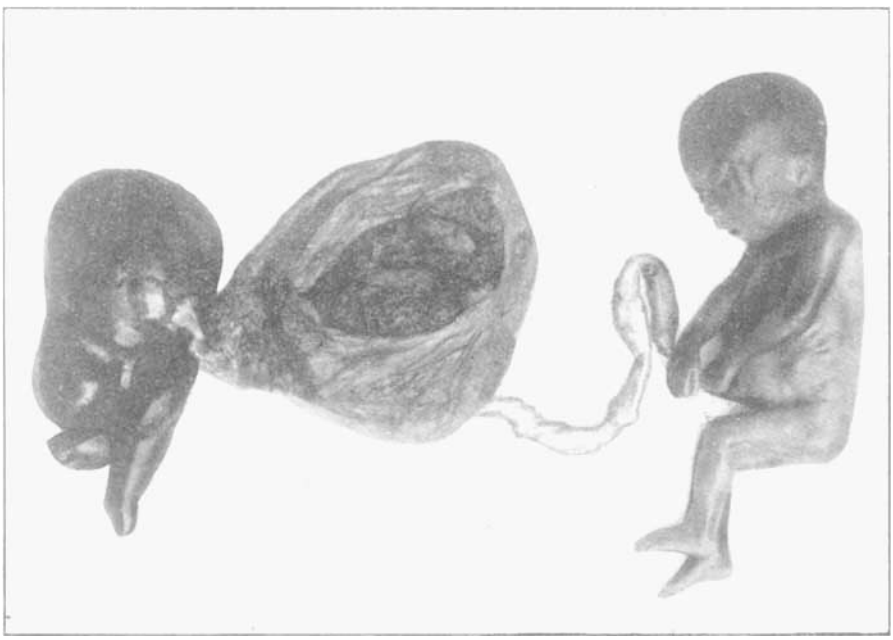
Previous Pregnancies. None.

Present Pregnancy. Duration: $23\frac{4}{7}$ weeks. General health: Good until two weeks before labour; breathing then became difficult and the feet were swollen. Liquor Amnii: In excess.

Present Labour. Twin labour. First child vertex; 2nd child breech. Fifteen minutes elapsed between the births of the two fœtus. The placenta was born immediately after the second fœtus.

FATHER. Age 35. Fitter's labourer. Said to be "regular and steady; a good athlete, though rather stunted."

FŒTUS AND PLACENTA. The normal fœtus, a female, weighed 1 lb. $2\frac{1}{2}$ ozs., and was 12 in. in length (Fig. I). The apparent age was 25 weeks.



Œdematous fœtus Normal fœtus

Fig. I.

The œdematous acephalic acardiac twin weighed 1 lb. $1\frac{1}{2}$ oz., and was 6 in. in length. The head and upper extremities were represented by three small fleshy anterior protrusions situated one inch below the upper end of the fœtus. The lower extremities were comparatively well developed. The œdematous phallic papilla was $\frac{5}{8}$ in. in length, and at first sight suggested that the fœtus was of male sex; but the healthy fœtus was female and the twins were definitely uniovular; hence the œdematous fœtus must have been of female sex. At the abdominal attachment of the thin umbilical cord, which contained only one artery and one vein, there was a hernial sac containing an intestinal protrusion which ended blindly. The anus was absent. All the tissues were so under-developed that differentiation was difficult.

On dissection it was found that the rudimentary vertebral column made a sudden anterior right-angled bend at a distance of $1\frac{1}{2}$ in. from its upper extremity. The heart could not be found. As previously stated the placenta was uniovular. It weighed 15 oz. and its dimensions were $5\frac{1}{2}$ in.× $4\frac{3}{4}$ in.×1 in. The cord of the healthy fœtus was inserted almost in battledore fashion; whilst the cord-insertion of the œdematous twin was velamentous.

The septum between the amniotic sacs consisted only of two layers of amnion and was so attached along the fœtal placental surface as to suggest that about five-sixths of the total placental substance belonged to the healthy fœtus, and the remaining one-sixth to the abnormal twin. When the two vessels in the cord of the œdematous fœtus were dissected, it was impossible to find any branches passing into the placental substance. The single umbilical artery passed directly into one of the umbilical arteries of the healthy twin; and the umbilical vein also ran across the placental substance into the similar vein of the other fœtus. The œdematous fœtus, therefore, was a parasite, not upon the mother through the substance of the placenta, but directly upon the other twin. The vitiated blood passing through one of the umbilical arteries from the healthy fœtus had evidently proceeded directly into the unhealthy fœtus through its single umbilical artery. This reversed circulation of impure blood was the obvious cause of the developmental abnormalities and general œdema described.

CASE 2.

MOTHER. Age 28. Married 6 years. Does not take alcohol.

Previous History. At 12 years of age she suffered with "dropsy and creeping paralysis." She states that "dropsy" continued until her marriage. Good health prior to present pregnancy.

Previous Pregnancies. (1) February 1916. Enteric fever and pneumonia during the third and fourth months. Normal labour. Full-time male child which died of "meningitis" when one year old. (2) December 1918. Normal pregnancy and labour. Full-time female child, alive and well.

Present Pregnancy. Duration: $34\frac{6}{7}$ weeks. General health: Sickness after every meal. Early morning headaches. Swellings: None. Urine: Normal volume.

Present Labour. Normal vertex labour.

FATHER. Age 26. Dock labourer. Army service from 1916 to 1919. Total abstainer. "Appears to be a healthy, industrious man."

FŒTUS. Male. Weight=6 lbs. 11 oz. Length=17 in.

General Appearance (Fig. II). General œdema. In certain regions, such as the lateral cervical, the fluid had accumulated in definite collections. Testes both within scrotum. Chest circumference=$12\frac{3}{4}$ in. Abdominal circumference=$14\frac{1}{4}$ in.

Skin and Subcutaneous Tissue. Skin of limbs and trunk was white and glistening. The face was slightly blue. A clear amber-coloured fluid flowed from an incision in the subcutaneous tissue. Within the scalp, the effusion was partially gelatinous.

Muscles: Pale and œdematous.

Cranium : Punctiform hæmorrhages of pericranium. Endocranial sinuses contained red translucent blood-clots.

Brain : Weight=$9\frac{1}{4}$ oz. Appeared to fit the cranial cavity more closely than usual. Cerebral tissue œdematous and glistening. Ventricles not distended : no venous congestion nor hæmorrhages.

Thymus : Weight=21 grs. Small gland with venous effusions between tissue lobules.

Thyroid : Weight=16 grs. Normal.

Thoracic Duct : Normal.

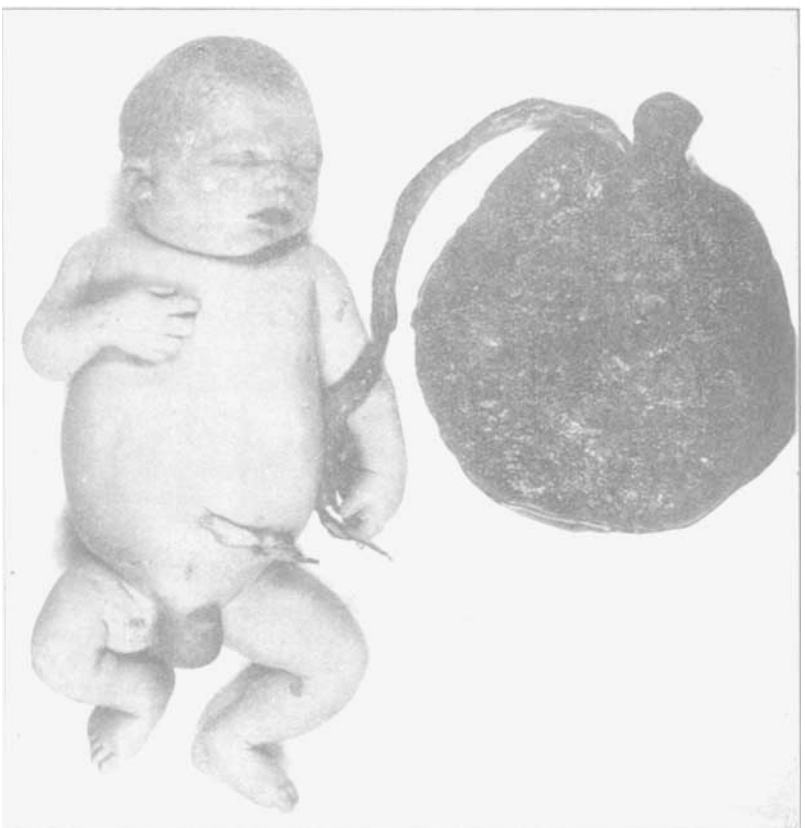


Fig. II

Pleural Cavities : Contained clear amber-coloured fluid. Pleura normal.

Lungs : Weight, Rt.=293 grs.; Lt.=226 grs. Pink in colour. Sank in water. Milky fluid exuded on the cut surface being squeezed.

Heart : Cardiac muscle pale. Ductus arteriosus, foramen ovale and all valves normal. Pericardial sac contained clear amber-coloured fluid. Pericardium normal.

Peritoneal Cavity : Contained 200 ccs. of fluid similar in appearance to that found in pleural and pericardial sacs. Albumen content=24 gms. per litre. Peritoneum normal.

Liver: Weight=4⅔ oz. Reddish-brown in colour. Cotton wool wiped across the cut surface was stained green. Umbilical vein ⅜ in. in diameter and patent. Ductus venosus also patent. Gall-bladder distended with fluid similar to that found in the body-cavities.

Spleen: Weight=304 grs. Colour normal; consistence soft.

Pancreas: Weight=25 grs. Normal.

Suprarenals: Weight, Rt.=54 grs.; Lt.=60 grs. Normal.

Kidneys: Weight, Rt.=138 grs.; Lt.=144 grs. Both paler than normal. Prominent fœtal lobulation. Zone of venous congestion surrounding bases of pyramids. Cortex slightly more narrow than usual.

Bladder: No urine contained. Urethra patent.

Intestines: Normal.

Testes: Collection of amber-coloured fluid in each tunica vaginalis.

Blood: Partial coagulation of blood: blood-count impossible.

Epiphyseal Line: Broader than normal; distal edge blurred by vascular zone.

Ossification: Centre for lower end of femur very small. Manubrial and upper three meso-sternal centres present.

Placenta and Umbilical Cord. Weight=2 lbs. 7 oz. Dimensions=8 in.×8 in.×1¾ in. Length of umbilical cord=21½ in.

Thick, pale, œdematous placenta which exuded fluid on pressure. Cotyledons large, separated by deep sulci. Placental tissue contained numerous small apoplexies, ranging in size up to that of a hazel-nut. They were at different stages of organization. Numerous small white infarcts visible on the fœtal surface; also two venous aneurysms, in which blood-stasis and disintegration had occurred.

Umbilical cord normal.

Case 3.

Mother. Age 24. Married 3 years.

Previous History: Suffered with anæmia, palpitations, and fainting attacks, but had no serious illnesses until March 1919, when she developed a primary labial chancre with syphilitic rash and sore throat. Her Wassermann reaction was then strongly positive (++). She was treated with mercury, iodides, and five injections (total dose=3 gms.) of neokharsivan.

Previous Pregnancies: (1) March 1918. Normal pregnancy: twin labour, both infants of female sex: one alive and well: the other died age 1½ years, cause unknown. (2) End of 1918. Miscarriage at 3½ months.

Present Pregnancy: Duration: 34⅘ weeks. General health: Very poor; frequent vomiting after food; fainting attacks and headaches. Fœtal movements: Normal. Swellings of feet developed at 18th week. Liquor amnii: Excessive volume.

Present Labour: April 14th 1920. Vertex L.O.A. Child made a few ineffective respiratory efforts. Severe hæmorrhage occurred before the birth of the placenta.

Puerperium: Normal.

After History: The fourth pregnancy, lasting 16 weeks, terminated in abortion on February 19th 1921.

Clinical Examination: March 29th 1921. General condition poor: no definite clinical signs of disease. Blood count: red corpuscles, 3,450,000 per

cmm.; white corpuscles, 6,562 per cmm. Hæmoglobin percentage=75. Differential count: polymorphonuclear leucocytes 15 per cent.; large lymphocytes 68 per cent.; small lymphocytes 1 per cent.; mononuclear cells 16 per cent. Film: slight anisocytosis. Wassermann reaction: positive (+).

FATHER. Age 26. Scaler. History of "fever and ague" several years ago. Said to be "strong and healthy."

FŒTUS. Male. Weight=7 lbs. 5 oz. Length=18¾ ins.

General Appearance: Dropsy, especially of face, abdomen, scrotum and penis. Eyelids swollen and in apposition; mouth open and distorted by œdema of lips. Tongue œdematous. Attached umbilical cord yellow and almost translucent. Testes situated in upper half of scrotum. Nails extended beyond finger-tips. Chest circumference=14 in. Abdominal circumference=14¾ in. Scrotal dimensions=2 in.×2 in.×2 in.

Skin and Subcutaneous Tissue: Forehead, neck, abdomen, and scrotum of a purple tinge and stippled with petechial hæmorrhages. Skin of arms and legs was glistening. Clear yellow fluid flowed from incised sub-cutaneous tissue. Scalp tissues occupied by a gelatinous sero-sanguinolent effusion.

Muscles: Œdematous. Hæmorrhage had occurred into the right temporal muscle.

Cranium: Petechial hæmorrhages in the deep scalp tissues. General venous congestion of cranial contents, especially in the posterior fossa.

Brain: Sulci contained greyish, translucent fluid. Cerebral tissue soft. Lateral ventricles slightly distended with greyish faintly-turbid fluid.

Thymus: Weight=22 grs. Small congested gland.

Thyroid: Weight=15 grs. Smaller than normal and greatly congested.

Thoracic Duct: Not found.

Pleural Cavities: 3 to 4 ccs. of clear yellow fluid in each sac.

Lungs: Weight, Rt.=275 grs.; Lt.=237 grs. Both pink in colour, with a few small subpleural petechiæ. Both lungs sank in water.

Heart: Weight=309 grs. Transverse diameter=2¼ in.; vertical diameter =1½ in. Right auricle and ventricle greatly distended with partially-clotted blood. Heart muscle deep purple in colour. All valves normal. Ductus arteriosus patent, but narrow at its mid-point; the upper half contained a blood clot of recent origin. Foramen ovale normal.

Pericardial cavity contained not more than 1 cc. of clear yellow fluid.

Peritoneal Cavity: Contained 170 ccs. of turbid blood-stained fluid, the albumen content of which was 17 gms. per litre. Two blood-clots, of dimensions 1 in.×½ in.×¼ in., and ½ in.×½ in.×⅛ in., lay in the peritoneal cavity and were tethered to the spleen by fibrinous strings. No peritoneal inflammation.

Liver: Weight=5 oz. 320 grs. Firm consistence. Early "nutmeg" mottling. Cut surface presented a slightly green tinge. Direct examination of liver serum by dark-ground illumination: no spirochætes found. Ductus venosus wider than normal. Gall-bladder filled with clear viscid colourless fluid resembling glycerine.

Spleen: Weight=2 oz. Dimensions 3½ in.×1¾ in.×1¼ in. Normal consistence; rounded borders; cut surface of a very deep purple colour, upon which the fibrous tissue was prominent as lighter strands.

Pancreas: Weight=66 grs. Larger than normal. Congested.

Suprarenals: Weight, Rt.=30 grs.; Lt.=36 grs. Both medullæ congested.

Kidneys: Weight, Rt.=192 grs.; Lt.=200 grs. Both larger than normal and showed general congestion. Uric acid infarcts were present in the collecting tubules. Ureters normal.

Bladder: No urine contained. Urethra normal.

Testes: Both tunicæ vaginales very congested; the right contained blood-stained fluid.

Intestines: Mucous membrane of small intestine congested; both small and large intestines contained meconium.

Lymph Glands: Mesenteric glands larger than normal.

Blood: Wassermann reaction: Positive (++).

Epiphyseal Line: Blurred and uneven. A zone of hyperæmia was observed in the cartilage 1 mm. from the epiphyseal line.

Bone Marrow: Paler than normal.

Ossification: Centre for lower end of femur not present. Manubrial and upper three mesosternal centres present.

PLACENTA. The placenta was not obtained for examination, but the midwife reported that it was the largest she had ever seen.

CASE 4.

MOTHER. Age 38. Married 18 years.

Previous History. States that she has always been healthy. During the 10th pregnancy she received 8 injections of grey oil. Hydrarg. c. Cret. gr. ii t.d.s. was administered during part of the 6th month of the 11th pregnancy. This treatment was repeated during March and April 1918; she was not then pregnant.

Previous Pregnancies. (1) December 1902. Normal. Male, now alive and well. (2) March 1905. Normal. Male, now alive and well. (3) October 1906. Normal. Female, died aged 12 hours; cause of death unknown. (4) to (9) inclusive. Still-born fœtus; no pregancy longer than 7 months. (10) January 1916. Female. General œdema. Weight=5 lbs. 2 oz. Length =15 in. No chondro-epiphysitis. Spleen and liver both large. No spirochætes found in liver. Weight of placenta=2 lbs. 3 oz. (11) December 1917. Stillbirth at 7th month. Hydramnios.

Present Pregnancy. Duration, $28\frac{2}{7}$ weeks. During the last 15 weeks she received 7 injections (total dose=2.1 gms. of novarsenobillon, and three injections of grey oil, each containing 1 gr. of mercury. Fœtal heart sounds inaudible on April 10th 1920.

Present Labour. April 26th 1920. Face presentation; left mento-anterior position.

FATHER. Age 42. Ship's foreman. Had "fever and ague" at sea. Said to enjoy good health.

FŒTUS. Female. Weight=2 lbs. Length=12 in.

General Appearance. Maceration and skin-peeling. General anasarca, especially of scalp, face, fingers and toes. Mouth open; tongue protruding. Chest circumference = $9\frac{1}{2}$ in. Abdominal circumference = 9 in.

Skin and Subcutaneous Tissue: Œdematous and blood-stained.

Muscles: Œdematous.

Brain: Cerebral tissue soft, especially internally where disintegration had begun: colour greyish-pink. The following fissures were present: Sylvian, Rolandic (not complete), callosal, parieto-occipital and calcarine (incomplete).

Thymus: Weight=2 grs. Surrounded by œdematous connective tissue.

Thyroid: Weight=3 grs. Very small pale gland.

Pleural Cavities: Each contained opaque blood-stained fluid.

Lungs: Weight, Rt.=61 grs.; Lt.=42 grs. Both pale pink in colour. Sank in water.

Heart: Weight=73 grs. Cardiac muscle soft: pale pink in colour. Ductus arteriosus, foramen ovale and all valves normal.

Peritoneal Cavity: Contained 30 ccs. of turbid, blood-stained fluid, the albumen content of which was 9.2 grms. per litre.

Liver: Weight=440 grs. Mottled yellowish-grey in colour Softer than normal in consistence. Direct examination of liver-serum by dark-ground illumination: no spirochætes found. Umbilical vein and ductus venosus patent. Gall bladder contained clear, bile-stained, viscid fluid.

Spleen: Weight=52grs. Reddish-grey in colour; consistence slightly softer than normal.

Pancreas: Weight=2 grs. Small, œdematous gland.

Suprarenals: Weight, Rt.=21 grs.; Lt.=23 grs. Of soft consistence; otherwise normal.

Kidneys: Weight, Rt.=24 grs.; Lt.=23 grs. Macerated, uniform brownish-pink colour without differentiation on the cut surface into cortex and medulla.

Bladder: Contained no urine.

Uterus, Fallopian Tubes and Ovaries: Normal.

Intestines: Lower half of small intestine, pelvic colon and rectum contained meconium.

Blood: Cell-count and Wassermann reaction impossible on account of blood disintegration within the vessels.

Epiphyseal Line: Wider than normal; edge blurred, but not definitely jagged.

Bone Marrow: Very pale.

Ossification. Centre for lower end of femur not present. Sternal manubrial centre present.

Placenta and Umbilical Cord. Weight=2 lbs. 3 oz. Dimensions=9 in.×8 in.×1 in. Length of umbilical cord=15 in.

Large, pale, œdematous placenta. Membranes normal and completely attached around the placental periphery. Fluid flowed from the cut surface of the cotyledons, which were large and became pitted on pressure. Numerous small white infarcts upon the fœtal surface. The umbilical cord was $\frac{1}{2}$ in. in diameter; the Whartonian jelly appeared more translucent than usual.

Case 5.

Mother. Age 30. Married 5 years. Healthy family history.

Previous History: No serious illnesses.

Previous Pregnancies: (1) March 1916. Miscarriage at third month after running for a train. (2) February 1918. Supposed ten-months' pregnancy. Stillbirth in hospital; said to be caused by birth-trauma.

Present Pregnancy: Duration, $29\frac{3}{7}$ weeks. General Health: Not good. Fœtal movements: "Quickened" in the 20th week. Swellings: Feet, ankles and right leg swollen from the second month; thighs and abdominal wall became œdematous later. Abdominal girth increased considerably during last month of pregnancy. Urine: Volume reduced. Doctor reported albuminuria. Liquor Amnii: Excessive volume. Pelvic measurements: 9 in.: $10\frac{1}{2}$ in.: $7\frac{1}{2}$ in.

Present Labour: June 15th 1920. Twin labour. First child breech, L.S.A. with extended arms and legs. Second child, breech, L.S.P., with extended arms and legs. Fœtal heart sounds of second child heard immediately before its delivery. Hydramnios of first sac, but not of second sac.

Puerperium: Normal. Urine (catheter specimen) June 16th 1920. Sp. gr. 1026. Reaction acid. Slight trace of albumen. Deposit: Leucocytes, epithelial cells, some granular casts.

Clinical Examination: August 27th 1920. No pathological condition found. Wassermann reaction: Negative.

FATHER. Age 30. Cableman. Healthy man.

FŒTUS. (*a*) *First Fœtus*: Female. Weight=4 lbs. $3\frac{1}{2}$ oz. Length= $14\frac{3}{4}$ in.

General Appearance (Fig. III): Diffuse anasarca, especially of head, chest, abdomen and external genitalia. Attached umbilical cord swollen and partially translucent. Eyelids in apposition: mouth open: tip of tongue protruded. Chest circumference=$11\frac{1}{2}$ in. Abdominal circumference =$11\frac{3}{4}$ in.

Skin and Subcutaneous Tissue: Pale, glossy skin, without petechiæ. On the chest, abdomen and right labium majus were scattered blebs, $\frac{3}{4}$ in. to 1 in. in diameter, some of which contained clear yellow fluid, whilst of others the raised superficial layer had been torn away, leaving pink raw areas. The pectoral superficial tissues were $\frac{1}{2}$ in. to $\frac{3}{4}$ in. in thickness, and from their cut surface there flowed a clear yellow fluid. The scalp tissues contained an opaque red effusion, with some gelatinous clots.

Muscles: Pale and œdematous.

Cranium: Dural septa uninjured.

Brain: Weight=$4\frac{1}{2}$ oz. Congestion and œdema of meninges. Cerebral tissue normal.

Thymus: Weight=30 grs. Normal.

Thyroid: Weight=26 grs. Dark red colour; friable; œdematous.

Thoracic Duct: Normal.

Pleural Cavities: Together contained 40 ccs. of clear yellow fluid of sp. gr. 1011 and containing 6 gms. of albumen per litre. Pleura normal.

Lungs: Weight, Rt.=93 grs.; Lt.=76 grs. Small pink lungs, which sank in water.

Heart: Cardiac muscle soft, but of normal colour. No cardiac hypertrophy. Foramen ovale, ductus arteriosus and all valves normal. Pericardial sac contained about 1 cc. of clear yellowish fluid. Pericardium normal.

Peritoneal Cavity: Contained 130 ccs. of clear yellow fluid of sp. gr. 1010, and containing 10 gms. of albumen per litre. Two transparent clots were in this fluid.

Liver: Weight=2 oz. Of normal size, colour and consistence. Umbilical vein and ductus venosus normal. Gall bladder contained viscid green bile.

Spleen: Weight=42 grs. Normal.

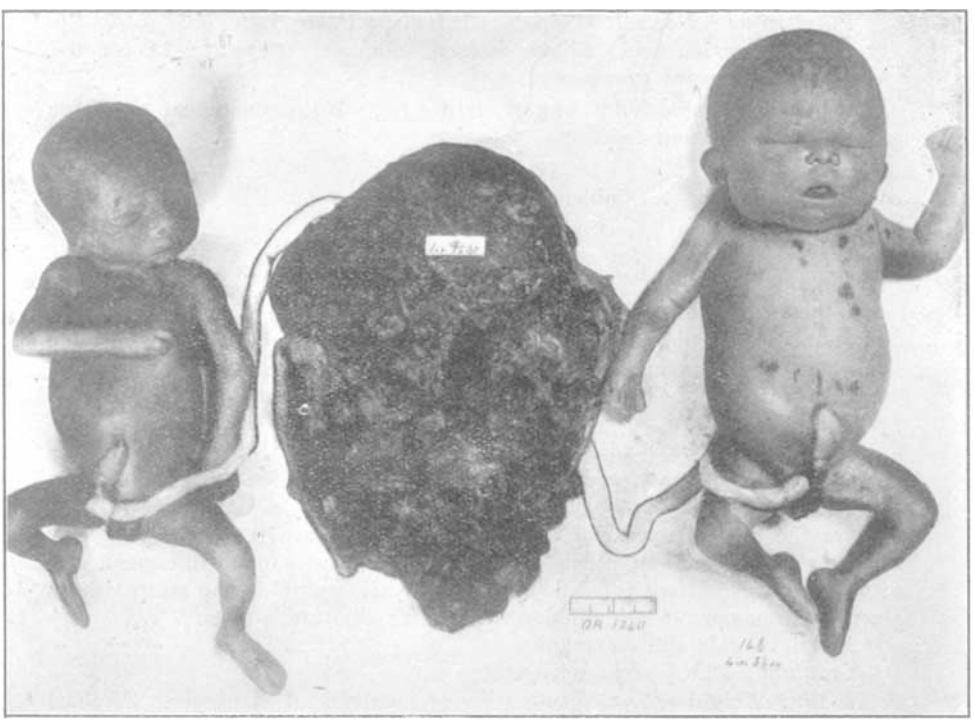
Pancreas: Weight=10 grs. Normal.

Suprarenals: Weight, Rt.=28 grs.; Lt.=27 grs. Darker and softer than normal. Medulla congested.

Kidneys: Weight, Rt.=80 grs.; Lt.=87 grs. Cortex paler than normal. Ureters normal.

Bladder: No urine contained. Urethra patent.

Uterus, Fallopian Tubes and Ovaries: Normal.



2nd Fœtus. 1st Fœtus.

Fig. III.

Intestines: Small collections of meconium in the transverse and pelvic portions of the colon.

Blood: Wassermann reaction negative.

Epiphyseal Line: Normal.

Bone Marrow: Normal.

Ossification: Centre for lower end of femur not present. Manubrial and first mesosternal centres present.

(*b*) *Second Fœtus*. Female. Weight=2 lbs. 7½ oz. Length=14½ in.

General Appearance: No external abnormalities, except slight abdominal protrusion. Eyes easily opened: mouth closed. Nails did not extend to finger-tips.

Skin and Subcutaneous Tissue : Not œdematous.

Cranium : Numerous petechiæ in pericranium. Sutures wider than normal.

Brain : Weight = 5½ oz. Meningeal congestion and œdema. 50 ccs. of red-coloured fluid flowed from the subarachnoid space. Cerebral tissue normal.

Thymus : Weight = 58 grs. Normal.

Thyroid : Weight = 33 grs. Slightly congested and enlarged.

Thoracic Duct : Normal.

Pleural Cavities : Together contained 20 ccs. of clear yellow fluid. Pleura normal.

Lungs : Weight, Rt. = 146 grs.; Lt. = 154 grs. Both lungs sank in water.

Heart : Cardiac muscle soft and slightly congested. Ductus arteriosus, foramen ovale and all valves normal. Pericardial sac contained a few ccs. of clear yellow fluid.

Peritoneal Cavity : Contained 10 ccs. of clear yellow fluid. Peritoneum normal.

Liver : Weight = 1 oz. 220 grs. General congestion : one subcapsular hæmatoma upon the superior surface. Umbilical vein and ductus venosus both patent. Gall bladder contained green viscid bile.

Spleen : Weight = 42 grs. Normal.

Pancreas : Weight = 16 grs. Normal.

Suprarenals : Weight, Rt. = 21 grs.; Lt. = 19 grs. Both congested.

Kidneys : Weight, Rt. = 92 grs.; Lt. = 102 grs. In the right there was prominent pallor of the pyramids; in the left the pyramids were a little paler than usual and were surrounded by a congested zone. Ureters normal.

Bladder : No urine contained. Urethra patent.

Uterus, Fallopian Tubes and Ovaries : Normal.

Intestines : Large intestine contained meconium.

Epiphyseal Line : Normal.

Ossification : Centre for lower end of femur not present. Manubrial and upper two mesosternal centres present.

Placenta and Umbilical Cord. Weight = 1 lb. 9½ oz. Dimensions = 9½ in. × 7 in. × ½ in. to 1 in.

Binovular placenta. Membranes appeared healthy and were complete around the placental periphery except at a segment 2½ in. in length. Cotyledons pale, œdematous, and friable; this condition was especially prominent in the placental tissue of the first fœtus. Both umbilical cords showed a left-handed spiral and both were inserted very near to the placental margin.

Case 6.

Mother. Age 43. Married 21 years. Healthy family history.

Previous History : Rheumatism in 1912 : since then has had pains in the back, and occasional swellings of hands and eyelids.

Previous Pregnancies : (1) April 1901. Normal full-time female; died age 10 weeks, "wasting." (2) October 1904. Normal full-time female; now alive and well. (3) August 1912. 3½ months' miscarriage : thinks abortion was caused by fright. (4) August 1913. Abortion—about 3 months. (5) October 1918. Normal full-time female; now alive and well.

Present Pregnancy : Duration 35⅚ weeks. General health : Poor; felt

drowsy and unfit for work; epigastric pains. Swellings: Great abdominal swelling during the month before labour; also swelling of legs. Urine: Dark coloured. Micturition four or five times in the night, but only a small volume passed each time. No albumen present in sample tested ten days before labour. Liquor Amnii: Excessive volume.

Present Labour: August 6th 1920. Duration 14 hrs. 25 mins. Vertex, R.O.A. No post-partum hæmorrhage.

Puerperium: Normal.

Clinical Examination: August 11th 1920. No pathological condition found. Wassermann reaction: slightly positive. Urine, August 17th 1920: Faint cloud of albumen upon boiling. No casts.

FATHER. Age 44. Carter. No history of illness. Appears to be healthy. Wassermann reaction, August 11th, 1920, slightly positive.

FŒTUS. Female. Weight=6 lbs. 9 oz. Length=18 in.

General Appearance: Abdomen protuberant; general anasarca, especially of scalp and face, chest, abdomen and external genitalia. Mouth slightly open; tongue not protruded. Only slight œdema of arms, hands, legs and feet. Finger nails extended to tips of fingers. Circumference of thorax=$13\frac{3}{4}$ in. Circumference of abdomen=15 in.

Skin and Subcutaneous Tissue: Scalp tissues contained sero-sanguineous clot $\frac{1}{2}$ in. in thickness. Clear yellow fluid containing 4 grms. of albumen per litre flowed from incised subcutaneous tissue. Blue mottling of subcutaneous tissue of face; tiny petechial hæmorrhages of abdominal skin.

Muscles: Pale; fibres separated by œdema.

Cranium: Petechial hæmorrhages of pericranium. Subarachnoid œdema.

Brain: Weight=7 oz. Cerebral tissue very soft.

Thymus: Weight=24 grs. Small congested gland not containing milky fluid.

Thyroid: Weight=32 grs. Normal.

Pleural Cavities: Each contained about 5 ccs. of clear yellow fluid.

Lungs: Weight, Rt.=$\frac{3}{4}$ oz.; Lt.=$\frac{1}{2}$ oz. Both lungs sank in water: apparently normal unexpanded lung tissue.

Heart: Weight=$1\frac{1}{4}$ oz. Dimensions 2 in.×$1\frac{3}{4}$ in.×$1\frac{1}{4}$ in. Right ventricular wall $\frac{3}{16}$ in. thick. Left ventricular wall $\frac{5}{16}$ in. thick. Foramen ovale, ductus arteriosus and all valves normal. Pericardial sac contained 3 ccs. of clear yellow fluid.

Peritoneal Cavity: Contained 90 ccs. of clear yellowish-red fluid, with a faint green tinge. Iodine reaction for bile pigments—negative. Albumen content of fluid=6.4 gms. per litre. Peritoneum normal.

Liver: Weight=$6\frac{1}{2}$ oz. Dimensions $4\frac{1}{2}$ in.×3 in.×2 in. Firm consistence. Cut surface presented a greenish tinge superimposed upon the normal liver colour. Umbilical vein and ductus venosus both patent. Gall bladder contained green fluid.

Spleen: Weight=$1\frac{1}{2}$ oz. Dimensions 3 in.×$1\frac{3}{4}$ in.×$\frac{3}{4}$ in. Firm consistence: borders rounded. Cut surface uniformly deep red in colour, resembling normal hepatic tissue. Malpighian bodies not visible. No "Herde" seen.

Pancreas: Weight=45 grs. Normal.

Suprarenals: Weight, Rt.=70 grs.; Lt.=84 grs. Small subcapsular and cortical puncta of a pale red colour. Medulla congested; no hæmorrhages.

Kidneys: Weight, Rt.=226 grs.; Lt.=220 grs. Slight congestion: no "Herde" seen. Œdema and congestion of peri-ureteral tissues; walls of ureters also congested.

Bladder: No urine contained: urethra patent.

Intestines: Small intestine showed congested areas. Meconium present in lower third of large intestine.

Uterus and Ovaries: Normal.

Lymph Glands: Mesenteric glands slightly larger than normal.

Blood: Clotting within veins prevented the collection of blood for Wassermann test, and for blood-count.

Epiphyseal Line: No epiphysitis, but the line was not sharp.

Bone Marrow: Paler than normal.

Ossification: Centre for lower end of femur not present. Sternum contained manubrial (double) and upper three mesosternal centres.

PLACENTA. Weight=2 lbs. 9½ oz. Dimensions 9½ in.×9 in.×1 in. to 1½ in. Length of umbilical cord=20 in. Diameter=½ in. Large, thick, ragged placenta. Membranes normal but were torn away from one-fifth of the fœtal surface. Cotyledons large, pale and spongy; they exuded fluid on standing and contained numerous small laminated blood clots. Small white infarcts occurred on the fœtal surface. Umbilical cord showed excess of Whartonian jelly in places. The spiral was right-handed.

CASE 7.

MOTHER. Age 31. Married seven years. Healthy family history. Total abstainer.

Previous History: No general illnesses, but states that she has not been in perfect health since the age of 18, when she suffered with "anæmia."

Previous Pregnancies: (1) November 1913. 3½ months' male miscarriage. (2) October 1914. Full-time male; now alive and well. (3) Date uncertain. 3½ months' miscarriage. (4) December 1917. Normal. Full-time male, died age 3 days: "jaundice." (5) February 1918. Eight months stillborn female; "had been dead a few days." (6) January 1920. Seven months macerated female. The midwife states that all the placentæ have been "too large."

Present Pregnancy: Duration 31⅘ weeks. General health: Good until the fifth month: lassitude: difficulty in going upstairs. No headaches. Morning sickness: Normal. Fœtal movements: Vigorous. Swellings: Abdominal girth increased rapidly about one month before labour. Slight swelling of ankles; none of face. Urine: Normal in volume, but was pink in colour during two weeks before labour. Liquor amnii: Excessive volume.

Present Labour: March 20th 1921. Duration 22 hours. Vertex, L.O.A. Four quarts of liquor amnii collected.

Puerperium: Normal.

Clinical Examination: March 25th 1921. No œdema; no icterus; no stains of previous rash. Liver and spleen just palpable, but not tender. Blood count: Red corpuscles, 5,250,000 per cmm.; white corpuscles, 12,500 per cmm. Hæmoglobin percentage=70. Differential count: Polymorpho-

nuclear leucocytes 57 per cent.; eosinophil leucocytes 2 per cent.; large lymphocytes 29 per cent.; small lymphocytes 4 per cent.; mononuclear cells 8 per cent. Film showed anisocytosis; some erythrocytes were basophilic. Wassermann reaction: Negative. Urine: Normal.

FATHER. Age 32. Engineer. Healthy family history. No previous illnesses. War service; twice wounded. Does not take alcohol.

Clinical Examination: March 25th 1921. A healthy man. Wassermann reaction: Negative.

FŒTUS. Female. Weight = 4 lbs. $3\frac{1}{2}$ oz. Length = $15\frac{1}{2}$ in.

General Appearance: Abdomen protuberant; slight general anasarca, most evident of eyelids, scalp, hands, feet, back and external genitalia. Mouth open; tongue protruding. Lanugo on trunk; finger nails did not extend to the tips of the fingers.

Skin and Subcutaneous Tissue: Very small red puncta on abdominal skin. Scalp tissues contained yellow jelly-like clot.

Muscles: Paler than normal and œdematous.

Cranium: Numerous petechiæ scattered in pericranium. Extensive meningeal œdema, without vascular congestion. Dural septa normal.

Brain: Weight = 6 oz. Sylvian blood-vessels very tortuous. Cerebral tissue normal. Intracerebral ventricles, which had been distended, contained a few ccs. of almost clear, yellowish fluid.

Thymus: Weight = 29 grs. Gland œdematous; not congested.

Thyroid: Weight = 23 grs. Normal.

Thoracic Duct: Normal.

Pleural Cavities: Each contained a few ccs. of clear, reddish-yellow fluid. Pleura normal.

Lungs: Weight, Rt. = 196 grs.; Lt. = 154 grs. Both lungs solid and sank in water. In colour they were slightly paler than normal.

Heart: Weight = 270 grs. Dimensions of combined ventricles = $1\frac{1}{2}$ in. × $1\frac{1}{2}$ in. × 1 in. Left ventricular wall = $\frac{1}{4}$ in. thick. Right ventricular wall = $\frac{3}{16}$ in. thick. Ductus arteriosus, foramen ovale and all valves normal. Pericardial sac contained a few cc. of clear yellow fluid. Pericardium normal.

Peritoneal Cavity: Contained 135 cc. of bright yellow fluid in which floated strings of white fibrin. This fluid had a specific gravity of 1015 and contained 25 gms. of albumen per litre. Its total chloride content was 8 gms. per litre; the deposit after standing contained erythrocytes and mononuclear cells resembling large lymphocytes. The peritoneum appeared normal.

Liver: Weight = $5\frac{3}{4}$ oz. Dimensions $4\frac{1}{4}$ in. × 3 in. × $1\frac{1}{2}$ in. Rich red in colour. Tissue normal. Umbilical vein entered liver substance 1 in. above the inferior border; it was patent. Ductus venosus also patent. Gall bladder contained clear, greenish-yellow bile.

Spleen: Weight = 202 grs. Dimensions $2\frac{1}{4}$ in. × $1\frac{1}{2}$ in. × $\frac{1}{2}$ in. Deep purple colour. Consistence softer than normal. Malpighian bodies not visible. One spleniculus, also enlarged, lay at the splenic hilum.

Pancreas: Weight = 20 grs. Normal.

Suprarenals: Weight, Rt. = 47 grs.; Lt. = 53 grs. Both relatively large: cortex appeared thicker than normal for size of fœtus. Medullary congestion. No "Herde" visible.

Kidneys: Weight, Rt.=87 grs.; Lt.=73 grs. Both paler than normal. Congestion of boundary zone between cortex and medulla. No "Herde" visible. Slight dilatation of both ureters.

Bladder: Contained 5 cc. of clear, colourless fluid. No stenosis of ureteral openings. Urethra patent.

Intestines: Coils of small bowel were contracted and drawn back against the posterior abdominal wall, which showed retroperitoneal œdema. Large intestine contained much green meconium.

Lymph Glands: Mesenteric glands normal. Left supraclavicular gland deep-red in colour.

Blood: Dark in colour; viscosity diminished. Wassermann reaction: negative.

Epiphyseal Line: Normal.

Bone Marrow: A little paler than normal.

Ossification: Centre for lower end of femur not present. Manubrial and upper two mesosternal centres present.

Placenta and Umbilical Cord. Weight=2 lbs. $3\frac{1}{2}$ oz. Dimensions=10 in.×7 in.×1 in to $1\frac{3}{4}$ in. Length of umbilical cord=20 in.

A bulky, pallid, soft and friable placenta: the membranes appeared healthy and were completely attached around the placental periphery. The maternal surface appeared œdematous and pitted on pressure; to it were attached small blood clots which appeared very prominent on the pale background. No infarcts found.

The umbilical cord presented a left-handed spiral; its placental insertion was almost battledore. No abnormalities of the umbilical cord except the presence of a yellowish fluid in the portion $3\frac{1}{2}$ in. in length terminating at the abdominal attachment.

Case 8.

Mother. Age 38. Married 12 years. Healthy family history.

Previous History: No general illnesses, but she states that her health since the birth of the sixth child has not been satisfactory.

Previous Pregnancies: (1) to (6) born between 1908 and 1918. All normal. Five children now alive and well. Third child had intrauterine amputation of left forearm; she died of diphtheria at the age of 11 years. (7) March 1919. Seven months' macerated fœtus. Examined in Department of Obstetrics, University of Liverpool. Epiphyseal line not irregular; no spirochætes found. (8) December 1919. Seven months' stillborn fœtus. The patient thinks that the liquor amnii was in excessive volume in both the 7th and 8th pregnancies.

Present Pregnancy: Duration, $37\frac{2}{7}$ weeks. General health: Poor; sickness, anorexia, and pain in the right upper half of the abdomen after the fourth month. Morning sickness: None. Fœtal movements: Patient "quickened" during the fourth month. Fœtal movements never felt strongly. Swellings: abdomen large; feet and legs swollen during the last two months. Urine: ? diminished in volume. Liquor amnii: Excessive volume.

Present Labour: Onset of pains at 1 a.m., October 30th 1920; child born at 3 a.m. on the same day. Vertex presentation. Easy labour.

Puerperium: Normal.

Clinical Examination: November 13th 1920. Anæmia and chronic

bronchitis. Blood count: Red corpuscles 4,100,000 per cmm.; white corpuscles 11,875 per cmm. Differential count: Polymorphonuclear leucocytes, 64 per cent.; eosinophil leucocytes, 2 per cent.; large lymphocytes, 18 per cent.; small lymphocytes, 0 per cent.; mononuclear cells, 16 per cent. Film: Showed anisocytosis; the smaller erythrocytes were mostly of a deeper colour than the larger red corpuscles, some of which were basophilic. Two of the mononuclear cells closely resembled lymphoidocytes, both in staining quality and in nuclear vacuolation. Two Türck cells were seen. Wassermann reaction: Negative.

FATHER. Age 41. Casual dock labourer. Healthy family history. States that he enjoys good health except when he has been drinking.

Clinical Examination: November 13th 1920. Good physique: cardio vascular system normal. Wassermann reaction: negative.

FŒTUS. Female. Weight=8 lbs. 2¼ oz. Length=19 in.

General Appearance (Fig. IV): A large, jaundiced baby, with protuberant, evenly-distended abdomen. Tissues of face and limbs firm and not œdematous. Eyelids closed. Mouth slightly open: tip of tongue visible between separated lips.

Skin and Subcutaneous Tissue: Showed diffusely scattered blotchy hæmorrhages, of which the largest were ¼ in. in diameter. The subcutaneous fatty layer was well developed; on the forearm it was ¼ in. thick.

Muscles: Paler than normal and slightly œdematous.

Brain: Weight (without cerebellum)=9½ oz. Slight meningeal congestion and œdema. Cerebral tissues stained green; consistence firm. Macroscopically normal.

Thymus: Weight=57 grs. Normal.

Thyroid: Weight=20 grs. Normal.

Lungs: Weight, Rt.=1 oz.; Lt.=¾ oz. Both lungs stained greenish and appeared mottled; the paler areas were chiefly in the upper lobes. They sank in water, but small pieces of the paler portions tended to float. Cut surface appeared partially expanded; scanty air bubbles could be squeezed out.

Heart: Weight=1¼ oz. Ventricles together measured 2 in. in transverse, 1¾ in. in supero-inferior, and 1½ in. in antero-posterior diameter. Hypertrophy of the cardiac muscle; the walls of the ventricles were approximately equal in thickness (⅜ in. to ½ in.). Developmentally the heart was normal in all respects.

Peritoneal Cavity: Viscera all bile-stained. The abdominal distension was found to be caused by very great hepatic and splenic enlargements, and the presence of 7½ fl. ozs. of blood and clot. This blood had originated from a rupture of the spleen capsule 1½ in. in length upon its diaphragmatic surface.

Liver: Weight=7¾ oz. Dimensions=5 in.×3½ in.×2 in. A firm, green, enlarged organ with rounded borders. Lobular arrangement easily visible both on the external surface, and also on cross section. Umbilical vein and ductus venosus patent. Gall bladder contained a little greenish slimy bile.

Spleen: Weight=4 oz. 290 grs. Dimensions=4¼ in.×2½ in.×1¾ in. Very greatly enlarged and soft; borders rounded. Capsule very tense and had ruptured, as previously mentioned, on the diaphragmatic surface. The organ appeared to consist almost entirely of blood-clot.

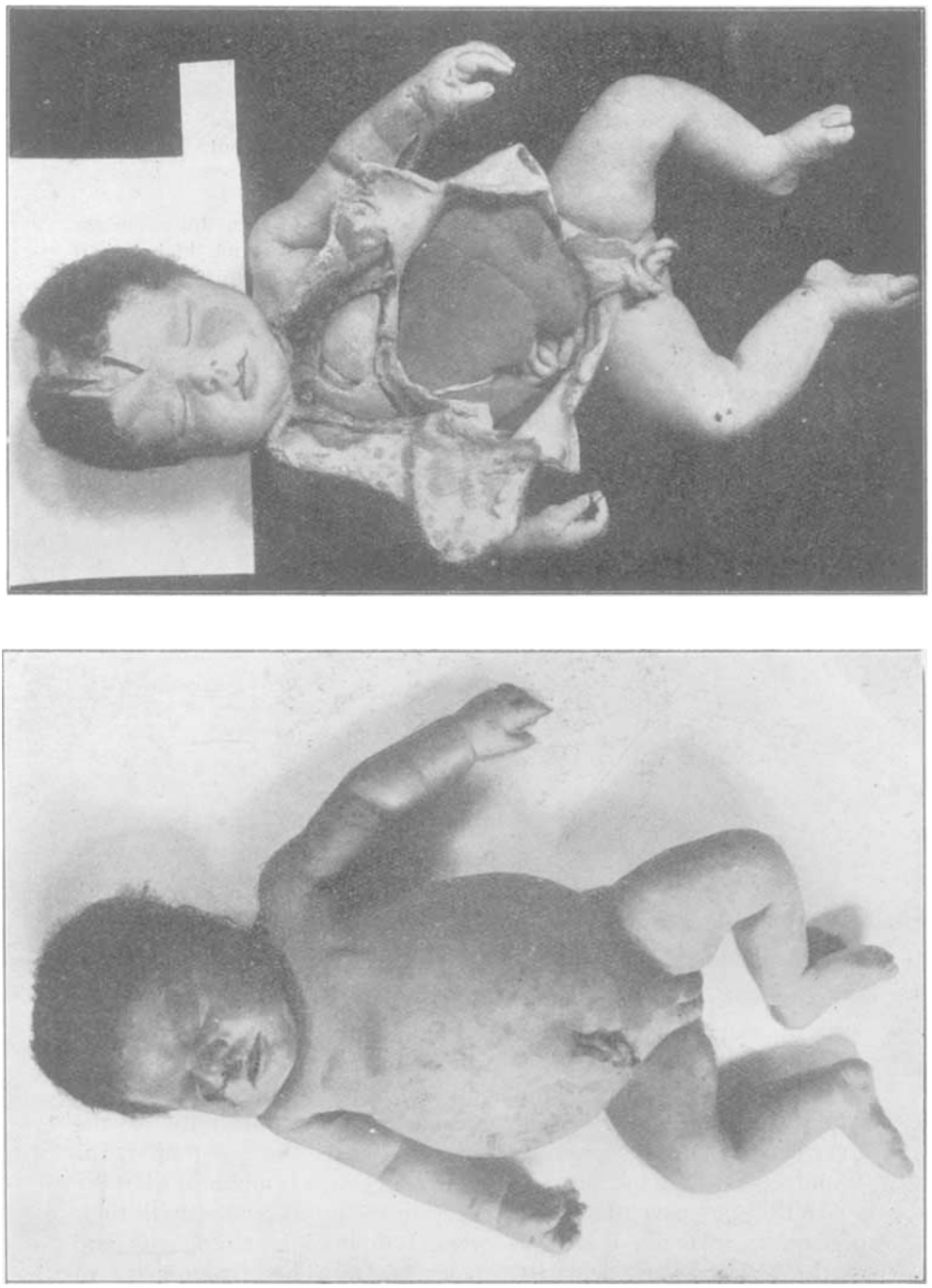


Fig. IV.

Pancreas : Weight=45 grs. Normal.

Suprarenals : Weight, Rt.=80 grs.; Lt.=97 grs. Both showed medullary congestion.

Kidneys : Weight, Rt.=180 grs.; Lt.=162 grs. Apparently normal.

Epiphyseal Line : Normal.

Bone Marrow : Redder and softer than normal.

Ossification : Centre for lower end of femur not present. Manubrial centre and upper three mesosternal centres present.

PLACENTA AND UMBILICAL CORD. The placenta was not sent for examination, but the midwife stated that it was very much larger and thicker than normal. The cord was about 1 in. in thickness, and very spiral.

HISTOLOGICAL FINDINGS.

In order to economize space the histological features have been summarized. The placentæ showed swelling of villi caused by a delicate connective-tissue cellular overgrowth more frequently than œdema. As a result the fœtal circulation within the villi was considerably impaired. Figure V shows the appearance seen in the

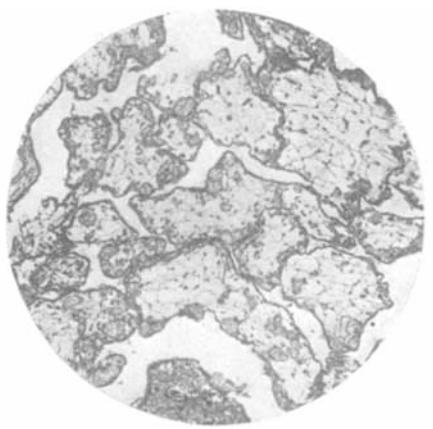


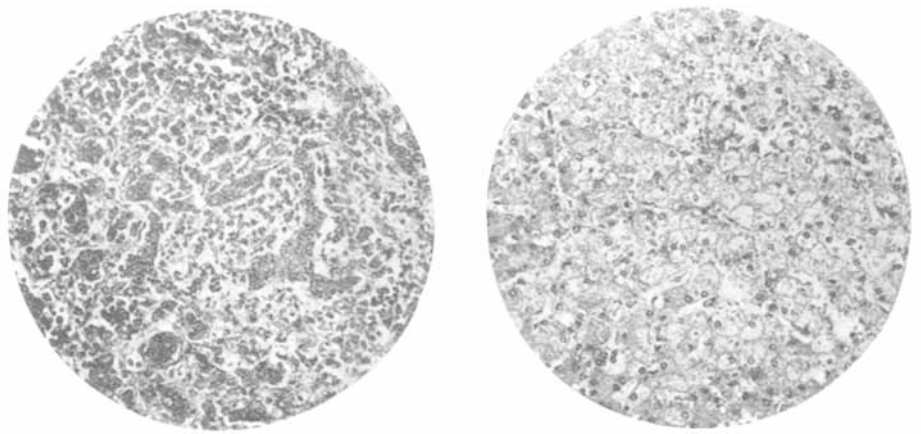
Fig. V. × 120.

placenta of Case 5. The chorionic epithelium showed no characteristic lesion, except where crowding of villi had occurred to such an extent that the maternal blood-supply had probably been cut off. At these points the epithelium had disappeared.

The characteristic appearance in the fœtal tissues was an intense activity of the hæmatopoietic structures, seen especially in the liver (Fig. VI), spleen and bone-marrow; to a lesser extent it was found in the thymus, suprarenals, kidneys and lymphatic glands.

In this excessive blood-cell formation the erythrocytes, with their precursors, namely, lymphoidocytes, lymphoid hæmatoblasts and erythroblasts, were specially concerned. The irregularity of erythrocytes, together with basophilia of erythroblasts, supported

this unusually active blood-formation. Myeloblasts occurred with a frequency which was undoubtedly greater than normal. The lymphoid tissue, on the other hand, was considerably less than normal, especially in the spleen. The pigment occurring in the hepatic cells, and to a lesser extent in other localities, such as the spleen and suprarenals, pointed to the fact that exaggerated blood-destruction was in process side by side with exaggerated blood-formation.



Liver (Case 3). Liver (non-dropsical fœtus).

Fig. VI. × 170.

Schridde's observation that in certain cases a fine yellowish pigment may be found in the cells of the renal convoluted tubules was confirmed. Connective-tissue overgrowth was not a constant feature.

Every attempt to find the spirochætes of syphilis was futile; nor did the tissues show histological features characteristic of syphilis. Degenerative changes were frequently noted in the parenchymatous cells of the liver and kidneys, and, to a lesser degree, of the lungs, pancreas and suprarenals. These changes consisted in granular degeneration and poor staining reaction, with nuclear karyorrhexis and pyknosis.

Whilst recording these observations, the possibilities of autolytic tissue-changes have been borne in mind; and it is believed that certain of the degenerative changes noted, especially in the more highly specialized cells, such as those of the convoluted renal tubules, had occurred post-mortem. This belief is confirmed by the experience gained in examining sections prepared from the tissues of fœtus not showing any of the signs of general dropsy. Appearances in the kidney tubules suggestive of fœtal nephritis cannot be taken as positive evidence of that condition.

Histologically, Case 8 resembled very closely the other

specimens examined. This fact, coupled with its macroscopical similarities in all points save the presence of fluid effusion and anasarca, constitutes the justification for its inclusion in this series. If intra-uterine life had been prolonged further it is probable that the fœtus would have become dropsical.

Summary of Clinical Details.

In the tabulation of the chief clinical features it will be noted that the mothers were generally about the middle of their child-bearing period; each had been previously pregnant at least twice. Their previous pregnancies totalled 36, of which 19 had ended in miscarriage or stillbirth; that is to say, 53 per cent. of their pregnancies had been failures.

The children which had been born alive were in nearly all cases the results of the earlier pregnancies; the capability of carrying offspring to the full time seemed to deteriorate progressively. The pregnancies under consideration invariably terminated before term; probably this was chiefly due to the presence of hydramnios.

The general health of the mothers during these pregnancies was unsatisfactory; the symptoms of which the patients complained were most commonly those associated with hydramnios.

In the histories of the mothers' previous health no uniform features were elicited. Case 3 was definitely, and Case 4 probably, syphilitic; the other mothers spoke vaguely only of "anæmia," "rheumatism" and "dropsy." Physical examination did not reveal the signs of gross disease.

The maternal blood was examined in Cases 3, 7 and 8. Anisocytosis occurred in each case, and basophilia in Cases 7 and 8. In each instance there was a relative increase in the proportions of large lymphocytes and mononuclear leucocytes, which may be accounted for in Case 3 by syphilis; in the other cases chronic toxæmia of unknown origin may have been causative.

The "slightly positive" Wassermann result in the mother and father of Case 6 is not of importance in the absence of confirmatory evidence in parents and child.

In regard to the presence of maternal kidney lesions, it is difficult to draw conclusions, especially since only two of the patients were delivered in hospital. There was certainly no case in which the so-called "pregnancy kidney" was a prominent feature, and no case of eclampsia. Œdema, when it occurred, was only in the legs; this clinical sign, together with the small urinary albumen content found in two cases, might be ascribed to the presence of hydramnios, especially in view of the rapid recovery which followed upon delivery.

The paternal histories did not reveal any important points.

Summary of Post-mortem Findings.

A tabulation of the most important findings is given. The condition of general fœtal œdema was a prominent feature in all these cases except in No. 8 and the second twin of No. 5. In the latter fluid had collected in the body cavities, but was not present in the subcutaneous tissues. In Case 8 no fluid collections nor œdema had occurred.

With the exception of Case 4, in which the fœtus showed advanced maceration, and Case 5, in which skin peeling had just commenced, all the fœtuses were quite fresh.

The thoracic duct was not found in Case 3; whilst in Case 4 the tissues were too macerated to permit the identification of such a delicate structure.

In four cases (Nos. 2, 6, 7, 8) advantage was taken of an X-ray examination of the leg.

So far as we know at present, the only important fœtal disease associated with X-ray signs is syphilis; in this infection the appearances produced on the X-ray plate are generally easy of diagnosis (Shipley and others [35]).

The radiographs of Cases 2 and 7 showed quite definitely the absence of syphilitic signs.

In Case 6 a pale cap-like band was seen surmounting the ends of the bone-shafts. In Case 8 the ends of the diaphyses were slightly irregular and more dense than usual.

General Summary and Conclusions.

1. Maternal clinical histories, though similar in several respects, did not reveal any pathognomonic features.

2. A mechanical cause for the œdematous state was found only in Case 1.

3. Histologically, hæmatopoiesis was the most prominent finding in the cases of non-mechanical origin.

4. The spirochætes of syphilis were not found in any instance.

5. The ætiology of general fœtal œdema of non-mechanical origin remains obscure. It is probable that maternal toxins of unknown nature are causative. In these circumstances the placenta would respond to impurity of the maternal blood by increasing its total absorptive area. The chorionic villi would become elaborated in complexity and increased in number. This overgrowth of villi throughout the whole substance of the placenta would, however, defeat its own purpose, unless the placental margin were to make a very rapid advance across the surrounding decidual surface; for if this did not occur the hypertrophy of villi would take place at the expense of the intervillous spaces, and the supply of maternal blood would be correspondingly reduced.

The histological examination of the placentæ shows that crowding of villi does occur, and also that the overgrowth of the intra-villous connective-tissue has been out of proportion to the increase in the total epithelial surface. This placental change is dangerous to the fœtus, for the chorionic vessels become compressed, fœtal nutrition is impaired, and the increase in peripheral resistance in the placental vessels will cause a rise in fœtal blood-pressure, with cardiac hypertrophy. At the same time the fœtus responds to the impairment of nutritions by an increase in blood-cell formation. When the pathological changes have reached this stage and exaggerated red-cell formation within the fœtus does not suffice to maintain healthy tissue-metabolism, exudation of fluid into the hypertrophied placental villi and into the fœtal serous sacs and subcutaneous tissue will occur. It is certain that the rise in fœtal blood-pressure will play a part in this process.

6. There are several points of resemblance between polycythæmia of the fœtus, as observed in examples of general œdema, and the rare disease of adult life, erythræmia. Enlargement of the liver and spleen, the presence of erythroblasts and scanty myelocytes in the blood-stream, and sometimes superficial hæmorrhages, are common to both.

The ætiology of erythræmia is not well understood, but some cases are associated with lesions of the respiratory system, such as emphysema or syphilitic disease of the pulmonary arteries (Ayerza's syndrome); others appear to be caused by poisoning with one of the coal-tar derivatives.

It is probable that in fœtal polycythæmia a similar ætiological subdivision into respiratory and toxic causes will be eventually demonstrated.

This work was carried out under the general direction of Emeritus-Professor H. Briggs, to whom I am greatly indebted for encouragement and advice.

I am very grateful to Professor Blair Bell, who has spent much time in editing this paper. His criticisms and advice have been most helpful.

Professor Ernest Glynn has also assisted me in many ways.

My thanks are due to the surgeons and midwives who have given me the opportunity of examining the specimens; also to Mr. C. Thurstan Holland and his Staff at the Liverpool Royal Infirmary for taking the radiographs.

In the examination of the references I have received much assistance from the Librarians of the University Medical Library and of the Liverpool Medical Institution.

	Case 2.	Case 3.	Case 4.	Previous Œdematous Fœtus.	Case 5.		Case 6.	Case 7.	Case 8.
1. Sex.	Male.	Male.	Female.	Female.	Female.	Female.	Female.	Female.	Female.
2. Weight and Length.	$6\frac{11}{16}$ lbs. 17 ins.	$7\frac{5}{16}$ lbs. $18\frac{3}{4}$ ins.	2 lbs. 12 ins.	$5\frac{2}{16}$ lbs. 15 ins.	$4\frac{3}{16}$ lbs. $14\frac{3}{4}$ ins.	$2\frac{7}{16}$ lbs. $14\frac{1}{2}$ ins.	$6\frac{9}{16}$ lbs. 18 ins.	$4\frac{3}{16}$ lbs. $15\frac{1}{2}$ ins.	$8\frac{2}{16}$ lbs. 19 ins.
3. Thymus.	Diminished.	Diminished.	Diminished.	—	Normal.	Normal.	Diminished.	Diminished.	Normal.
4. Thyroid.	Slightly diminished.	Diminished.	Diminished.	—	Normal.	Slightly enlarged.	Normal.	Normal.	Normal.
5. Thoracic Duct.	Present.	Not found.	—	Search not made.	Present.	Present.	Search not made.	Present.	Present.
6. Albumen content of peritoneal fluid in gms. per litre.	24	17	9.2	—	10	—	6.4	25	—
7. Liver.	Slightly enlarged.	Enlarged.	Normal.	Enlarged.	Normal.	Normal.	Enlarged.	Enlarged.	Greatly enlarged.
8. Spleen.	Enlarged.	Greatly enlarged.	Normal.	Enlarged.	Normal.	Normal.	Enlarged.	Enlarged.	Greatly enlarged.
9. Epiphyseal line.	Broader than normal.	Blurred edge.	Blurred and a little broader.	Normal.	Normal.	Normal.	Not quite sharp.	Normal.	Normal.
10. Placenta.	2 lb. 7 oz.	Not sent with specimen but was reported to be very large.	2 lb. 3 oz.	2 lb. 3 oz.	Binovular 1 lb. $9\frac{1}{2}$ oz.		2 lb. $9\frac{1}{2}$ oz.	2 lb. $3\frac{1}{2}$ oz.	Not sent with specimen.
11. Fœtal Wassermann.	Not done.	Positive + +	Not possible.	Not done.	Negative.	Not done.	Not possible.	Negative.	Not done.

TABULATION OF CHIEF CLINICAL DETAILS IN CASES TWO TO EIGHT.

	Case 2.	Case 3.	Case 4.	Case 5.	Case 6.	Case 7.	Case 8.	Average.
Age of Father, in years.	26	26	42	30	44	32	41	$34\frac{3}{7}$
Age of Mother, in years.	28	24	38	30	43	31	38	$33\frac{1}{7}$
Previous Pregnancies.	2	2	11	2	5	6	8	$5\frac{1}{7}$
Number of stillbirths and miscarriages.	0	1	8—one showing general œdema.	2	2	4	2	$2\frac{5}{7}$
Pregnancies which terminated in above stillbirths or miscarriages.	0	2nd	4th to 11th inclusive.	1st and 2nd.	3rd and 4th.	1st, 3rd, 5th and 6th.	7th and 8th.	—
Duration of present pregnancy in weeks.	$34\frac{6}{7}$	$35\frac{4}{7}$	$28\frac{2}{7}$	$29\frac{3}{7}$	$35\frac{5}{7}$	$31\frac{4}{7}$	$37\frac{2}{7}$	$33\frac{2}{7}$
Presence of Hydramnios.	Not stated.	Present.	Not stated.	Present.	Present.	Present.	Present.	—
Paternal Wassermann.	—	—	—	—	Slightly positive	Negative.	Negative.	—
Maternal Wassermann.	—	Positive, together with clinical signs of syphilis.	Said to have been positive in 1916.	Negative.	Slightly positive.	Negative.	Negative.	—

REFERENCES.

1. Andrews, H. Russell. *Trans. Obstet. Soc.*, Lond., 1901, xliii, 166.
2. Ballantyne, J. W. "Manual of ante-natal pathology and hygiene. Edin., 1902.
3. ——. "The diseases and deformities of the fœtus." Edin., 1892.
4. Bar, P. *L'Obstétrique*, 1903, viii, 289.
5. Bloch, R. *Virchow's Arch. f. pathol. Anat.*, 1920, ccxxviii, 285
6. Crozier, L. Dissert. Lyon, 1913.
7. Cruikshank, J. *Journ. Pathol. and Bactol.*, 1911-12, xvi, 167.
8. Doi, M. *Arch. f. Gynäkol.*, 1912, xcviii, 136.
9. Fischer, O. *Zeitschr. f. Geburtsh. u. Gynäkol.*, 1911, lxix, 758
10. Fischer, W. *Deutsch. med. Wochenschr.*, 1912, xxxviii, 410.
11. Flamma, S. *Ann. di Ostet.*, 1920, xlii, 385.
12. Fleischmann, O., and S. Wolff. *Arch. f. Kinderh.*, 1914, lxii, 75.
13. Führ, O. Inaug. Dissert. Giessen, 1891.
14. Gillmore, R. *Surg., Gynæcol. and Obstet.*, 1906, iii, 621.
15. Grulee, C. G. *Arch. Pediat.*, 1907, xxiv., 510.
16. Himmelheber, K. *Monatsschr. f. Geburtsh. u. Gynäkol.*, 1910, xxxii, 370.
17. Kafka, V. *Deutsch. med. Wochenschr.*, 1921, xlvii, 86.
18. König, H. *Folia Hæmatol.*, 1910, ix, 278.
19. Lahm, W. *Arch. f. Gynäkol.*, 1914, cii, 284.
20. Liegner, B. *Monatsschr. f. Geburtsh. u. Gynäkol.*, 1919, l, 350.
21. Lieven, F. *Zentralbl. f. Gynäkol.*, 1911, xxxv, 804.
22. Link, G. *Ziegler's Beitr. z. pathol. Anat.*, 1914, lix, 371.
23. Lobenhoffer, W. *Ziegler's Beitr. z. pathol. Anat.*, 1908, xliii, 124.
24. Loth. No initial in original. *Deutsch. med. Wochenschr.*, 1912, xxxviii, 1642.
25. Nyhoff, G. C. *Zentralbl. f. Gynäkol.*, 1911, xxxv, 808.
26. Rautmann, H. *Ziegler's Beitr. z. pathol. Anat.*, 1912, liv, 332.
27. Sänger, M. *Arch. f. Gynäkol.*, 1888, xxxiii, 198.
28. Sauvage, C. *Ann. de Gynécol. et d'Obstét.*, 1913, x, 385.
29. Schmidt, M. B. *Ziegler's Beitr. z. pathol. Anat.*, 1892, xi, 199.
30. Schmidt, E., and G. Mönch. *Monatsschr. f. Geburtsh. u. Gynäkol.*, 1918, xlvii, 368.
31. Schridde, H. *Münch. med. Wochenschr.*, 1910, lvii, 397.
32. ——. *Deutsch. med. Wochenschr.*, 1911, xxxvii, 432.
33. Schumann, E. A. *Amer. Journ. Obstet.*, 1915, lxxii, 961.
34. Seyffert, M. *Arch. f. Gynäkol.*, 1920, cxii, 413.
35. Shipley, P. G., J. W. Pearson, A. A. Weech, and C. H. Greene. *Johns Hopkins Hosp. Bull.*, 1921, xxxii, 75.
36. Siefart, G. *Monatsschr. f. Geburtsh. u. Gynäkol.*, 1898, viii, 215.
37. Sitzenfrey, A. *Zentralbl. f. Gynäkol.*, 1910, xxxiv, 1381.
38. Smith, A. J., and A. Birmingham. *Journ. Anat. and Physiol.*, 1889 xxiii, 532.
39. Stevens, T. G. *Trans. Obstet. Soc. Lond.*, 1895, xxxvii, 5.
40. Swart, G. *Virchow's Arch. f. pathol. Anat.*, 1905, clxxxii, 419.
41. Tait, Lawson. *Trans. Obstet. Soc. Lond.*, 1875, xvii, 307.
42. Wienskowitz, H. *Deutsch. med. Wochenschr.*, 1914, xl., 1952, 1972.