

*Fig. 1, 2, missing.*

A CONTRIBUTION

to the study

of the Subchorial Haematoma

of the Decidua

by

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During a year's work (1903-4) at the Hospice, 219 High Street, my interest in the Subject of blood moles was roused by the fact of six such specimens coming into my hands - Other two have since been lent for examination, making a total of eight.

I was still further interested and stimulated to make a careful study of these Specimens, with a view to if possible, throwing some fresh light on the Subject, by the fact that four out of the six cases had to be curetted on account of continuance of symptoms. These curettings will be described later, along with the macro- and microscopical descriptions and histories of each case. It is first intended however to review up to date the various theories put forward as to the causation of blood-moles, these having been gleaned from the literature on the subject.

#### THEORIES.

The earliest writers on the subject date from the time of Hippocrates and Galen, to Mitschik in 1845, Müller in 1847, and Scanzoni in 1849.

#### "MÜLLER

Writes about "flesh-mole", which is characterised  
by/

by subchorial hemispherical haematomata, and, as a rule a small foetus, that "from this medium form transitions to normal ova are met, or to striking deformities, where the foetus is unrecognisable or lost, or the whole cavity is destroyed, and the mass presents outwardly an irregular lumpy shape" -

PERNICE, writing in 1852, separates moles into Hydatid and Flesh moles - He says the latter result from blood extravasations, and the name is therefore false.

He believes the foetus dies when the ovum is compressed by the blood exudations, and the chorion villi are torn from their insertions - If the latter does not happen, the foetus may live a little while longer, but never reaches complete development.

Among later writers must be mentioned, Jakobson, Schwalbe, Spiegelberg, Leditsch, and in 1891 Galabin.

BREUS, however, in his paper published in 1894, was the first to give any detailed description of these sacs, and a definite theory formulated as a result of his investigations. He described a form of mole which he believed to be a variety by itself, and not to be confused with blood and flesh moles - he called it "Hamatoma Subchoriale der Decidua" and described, as its peculiar characteristic, the presence of many broad-based polypoid blood-effusions. If the placenta is/  
is/

is not differentiated, these are scattered over the whole surface of the ovum, and are mostly long stalked and isolated; if the placenta is developed, they are generally limited to the serotina, thickly pressed and broad based. In this form, the haemorrhage is only secondary to diverticular formation. In blood and flesh moles, he says, the tissue of the ovum is almost entirely destroyed by haemorrhages, and of flesh-moles he says: "The ovum consists in such cases only of extravasated blood, and more or less altered fibrin, in which the characteristic part of the ovum is destroyed almost beyond recognition, so that the whole structure presents only a compact flesh like mass."

Breus holds that the Tuberoso Subchorial Haematoma originates in the following way:

First, death of the embryo occurs from unknown causes, about the second month. The foetal membranes however continue to grow in spite of the arrested circulation in the chorionic villi, as, through their connection with the decidua, they are in a position to be nourished by osmosis from the maternal blood. *and are only nourished till Second month.*  
 (Example - the chorion in Hydatid mole, increasing in size in its myxomatous degeneration, to the end of the pregnancy.)

If now a large quantity of liquor amnii is not produced/

-ed (its non-production being easily explainable by the death of the embryo, imperfect placental formation and circulation, and increased osmotic processes in the amnio-chorion), the membranes become too large, and as any expanding pressure is lacking owing to the contents not having kept pace with the growth of the membranes, these fold inwards and form diverticula of the most complex shapes, projecting into the amniotic cavity - A haemorrhage now occurs from the decidual vessels and finds a place for itself in these preformed sacs - The decidua is thus not affected till possibly months later, and accordingly the mass, seldom exceeding the size of a fist, may not be expelled till almost the end of the pregnancy or beyond it, the embryos in such sacs remaining in good condition.

Breus lays special stress on three points, as proving the primary formation of the diverticula:

1. Their extremely complicated shapes and stalks.
2. The presence of empty sacs, which show no sign of having had blood in them.
3. The fact, that the membranes retain their various shapes (when emptied of blood clot), and show strands of connective tissue.

Neumann in 1897, attacked Breus's paper, and a lively discussion followed, Breus's mole being held by many to be in no way different from an ordinary flesh-mole.  
Neumann/

Neumann refuted the idea of any continued growth of the membranes after the death of the foetus, by raising such questions as:

"How can one prove that the membranes live after the death of the foetus, and therefore continue growing?"

"If the membranes grow, why not the Placenta?"

"Since degenerative changes are found in placentae after the death of the foetus, is one not much more likely to get an arrested than a further growth of the membranes?"

Neumann laid no importance on the varied shapes of the blood masses, nor on the fact of the membranes retaining these shapes when emptied of coagula, pointing out that no membranes once lifted up by a haemorrhage ever again recover their smooth form - The strands of connective tissue present in them, he showed to be a feature of normal placentae, acting there as a scaffolding, leading to stem villi, and preventing the chorion from being so easily lifted up, a ball-shaped swelling being the natural result on the occurrence of a haemorrhage. Hydatid mole and Deciduoma malignum he considered to be quite wrongly cited as instances of further growth, there being in both these cases degeneration.

Neumann's own view as to the formation of blood-mole is as follows:

He/

He believes that the blood exudations and diverticular formation depend on one another, and that the altered nourishment conditions of the ovum lead to death of the foetus. The empty folds he believes to be formed by shrinking after the death of the foetus. He leaves open the question whether the anomalies which lead to the disturbances of development are maternal or foetal, and the nature of these anomalies he considers doubtful.

To this criticism Breus retorted that some of the ten cases described by Neumann were simple flesh moles and the remainder retained abortion ova, the foetus being far too long. He also pointed out that none of Neumann's sacs were preformed and that such placental haemorrhages are quite common in flesh moles, retained and macerated ova, or in placentae of normal fruit.

GOTTSCHALK now stepped in as a mediator and expressed his views as follows:-

"There is a special form of mole, described according to its character as a haematoma mole, which is generated by the following circumstances:- most likely owing to an insufficiency of the primitive heart germ, the chorionic villi are not so vascularised that a placental circulation can be introduced. In such ova the embryo dies as soon as the yolk sac circulation/

ulation fails to satisfy its nourishment needs. But since the foetal coverings of the foetal sac can of their own accord, and without foetal circulation, continue to grow for a few weeks, a striking misproportion results between the size of the prematurely destroyed embryo, and that of the ovum sac. This misproportion can be greater than it seems, as the foetal membranes fold inwards, on account of the resistance of the uterine wall. By these folding processes there naturally results - with the added help of the intervillous blood stream - a partial lifting up of the chorionic membrane from the yielding portions of the placental structure, which at the same time -, also naturally -, keeps hold where there is an attaching villus. In this way subchorial gaps are formed in which the blood circulating between the villi exerts pressure more freely and so the folds of the chorionic membrane are bulged into the ovum cavity as protruding diverticula of different sizes and forms. Coagulation processes now occur in them, completing the haematoma formation."

There was for some time after this a cessation of any literature of importance on the subject, till in 1901 Dr. Davidsohn's paper, "Zur Lehre von der Mola Hämatomatosa", appeared. After a short introductory sketch of Breus's, Neumann's and Gottschalk's views, Dr./

Dr. Davidsohn proceeds to criticise these, and state his explanation of the formation of bloodmoles, flesh moles being included as simply an older and more organised form of the same, and Breus's mole as a variety by itself, he refuses to recognise. In his criticism Davidsohn lays emphasis specially on the fact that none of these three theories explain how it is possible for an ovum of only a few centimetres to reach a circumference of 10 centimetres through the formation of subchorial haemorrhages, without the uterus reacting in the least with contractions; and to regard the misproportion between the foetus and its sac as simply a result of passive pressure through haemorrhages, he considers entirely disproved by the fact that the chorionic membrane is quite thickly set with villi, and if by chance these are thinned out in one part through pressure from the extravasated blood, they are formed proportionately thicker in neighbouring parts. In addition he would expect to find a much greater degree of shrinking. In short, Davidsohn believes that the striking misproportion between foetus and sac wall is not apparent but real, and as this increased size cannot be followed after the death of the foetus (Breus's theory of further growth of the membranes being quite untenable) the conclusion is that this condition already existed when death of the foetus occurred and that therefore hydramnios of the ovum is the only feasible/

feasible solution.

The course of events Dr. Davidsohn believes to be as follows:

A primary hydramnios of the ovum is present. If abortion does not occur, the fluid is reabsorbed, or trickles away. There is thus lessened hydrostatic pressure in the amniotic cavity, and subchorial haemorrhages occur, which loosen the amnio-chorion from the uterine wall and form haematomata which speedily fill the emptied spaces. The ovum is now of the same size, but contains subchorial haemorrhages in place of a superfluity of liquor amnii. Shrinking now sets in leading to almost complete absorption of the fluid and folds are formed between the haematomata. No maceration results on account of the diminished amount of fluid. As the ovum does not increase in size, there is no inclination to abortion. Separation is caused in time by slow necrotic changes.

TAUSSIG and ENGEL must here be shortly mentioned as the exponents of a secondary hydramnios.

TAUSSIG'S theory of origin is as follows:  
After the death of the foetus, the membranes, amniotic fluid and uterus increase in size. A secondary hydramniotic ovum results by osmosis. From unknown causes the postmortem overgrowth stops. The ovum is retained, and the amniotic fluid gradually absorbed. The membranes lay/

lay themselves in folds which are partly filled with blood and finally form broad based and stalked haematomata."

He also remarks: "In many cases there existed previously some genital disease. But such moles also develop in cases where one seeks in vain according to accepted methods for any evidence of genital disease."

Taussig also gives a more detailed microscopical description of Micholitsch's hydatid and blood mole, than is found in Micholitsch's own paper (to be mentioned later.)

ENGEL in his paper, "Die Rückbildungsvorgänge an Abortiven Embryonen," states that he found in his three cases a striking misproportion between foetus and amnion sac. As endometritis was present in 2 of them, he throws out a suggestion (which cannot be considered more than that) that the disease may have passed over from the mother to the foetus and membranes. He believes "that after the death of the foetus through unknown causes (this death being partial or complete), hydramnios sets in."

In 1904 BAUEREISEN contributed a paper offering a fresh theory for the causation of blood moles, based upon his investigation of a case of his own.

His theory rests upon the fact that on microscopical examination/

examination he found villi either in the lumen of decidual vessels, or appearing to compress these by their position - Also, as the foetal membranes were healthy, he accepts Breus's view of their further growth as practically proved: and the occurrence of a hydramnios he believes to be shown by the appearance of the interior of his sac.

Linking these together his theory is as follows:

The deported villi in decidual veins or pressing upon them act as a barricade to the outflow of blood from the sinuses and a stasis results - a condition of hydramnios being also present at this time.

The stasis by disturbing the nourishment of the foetus causes its death, but the membranes continue to grow for a time. Haematoidal swellings are finally caused by the accumulated blood in the sinuses, aided by infolding of the overgrown membranes as a result of reabsorption of the fluid and cessation of growth of the uterus.

The first British writer on the subject was DR. BERRY HART in his paper in the Journal of Obstetrical Transactions on "The Nature of the Tuberosc fleshy Mole."

DR. HART, before entering upon his views, emphasises in a short resumé of the structure of the normal/

normal placenta, the presence of a certain number of chorio-basal septa which unite the basal serotina and the chorion.

He also points out that there occurs a physiological thrombosis of the sinuses in the uterine wall and basal serotina, observed during and after the 8th month by Friedländer in the uterine wall, and in the serotina at full time by Leopold. Dr. Gulland and he also observed at the 7th week venous sinuses in the compact layer of the serotina with proliferating endothelium or young connective tissue.

In the microscopical examination of his specimen, Dr. Hart found a curved chorio-basal septum persisting at the sulcus bounding the base of the tuberosities. He also found a blocking of the thin-walled sinuses in the large celled layer of the serotina.

His view is as follows:

1. Undue blocking of the serotinal sinuses in the large celled layer, which leads to
2. Slow engorgement of the intervillous circulation. This bulges out the chorio-basal septa, and as they tack down the chorion at definite points, amnion and chorion will bulge up between.
3. The embryo dies as a result of interference with its circulation, its death being secondary.

4./

4. The placenta becomes a thrombosed mass, and is retained a certain time before expulsion.

Before entering upon a criticism of these theories, mention must be made of MICHOLITSCH'S case of combined hydatid and blood mole, reported in the "Archiv. für Gynaekologie", since one of the eight cases to be described later is a similar combination of the two forms of mole.

It is however much to be regretted that Micholitsch has given no microscopical description of the sac, simply remarking that "histologically important is the inflammatory infiltration of the Decidua."

The macroscopical description is that of a sac covered for the most part with vesicles, varying from the size of shot to grape-like groups - in its interior showing a number of typical haematomata and a well preserved embryo 11 m.m. long.

TAUSSIG, as has been mentioned, has supplemented this in microscopical detail, but at the same time has thrown doubt upon the possibility of its being a true hydatid for two reasons -

- a. The absence of Langhan's layer.
- b. The insufficient amount of syncytial masses and buds.

CRITICISM OF THEORIES

Before venturing on a short criticism of each of these theories, commencing with Breus's, I feel bound to express for him the respect due to all those who act as pioneers, and by whose very mistakes their successors in investigation profit.

BREUS.SUMMARY OF HIS VIEW.

Death of the embryo occurs from unknown causes, the membranes however continuing to grow. From lack of expanding pressure an infolding of the same results, forming diverticular projections into the amniotic cavity. Haemorrhages occur from decidual vessels, which find a lodgement in these preformed sacs. The decidua is thus not affected till much later, accounting for the long retention of these ova.

CRITICISM.

Some such theory as this seemed to Breus absolutely necessary to account for that form of sac, filled with haematomata of all shapes and sizes, which he insisted upon regarding as a variety quite by itself, and not to be confounded with ordinary blood and flesh moles. As a matter of fact however a microscopical study of these three forms serves to convince one that they are identically the same, though resulting from haemorrhages of different degree and age. It follows therefore/

therefore (even allowing the possibility of further growth of the membranes) that Breus's theory of origin can only apply to a small number of blood moles. But there is no proof of this further growth, the likelihood being rather that degeneration sets in after a time, since the placentae of more mature dead fetuses show degenerative changes. Nor does there seem any difficulty in picturing the formation of the various shapes and sizes of the haematomata and their stalks, when one remembers the distensibility of the foetal membranes, the presence of chorio-basal septa in the normal placenta, and the fact of shrinking occurring in all haemorrhages.

#### NEUMANN.

#### SUMMARY OF HIS VIEW.

That some anomaly, maternal or foetal, exists, leading to disturbance of development. Haemorrhages occur causing the death of the foetus by altered nutritive conditions, and the blood effusions and diverticular formation depend on one another, the empty folds and stalk formation following as a result of shrinking.

#### CRITICISM.

This is certainly an advance on Breus's theory, in that it suggests some anomaly, maternal or foetal, as a cause of the haemorrhages, and through them of the death/

death of the foetus - There is however no indication as to what these anomalies might be, or in what way they hinder foetal nourishment.

But quite apart from his theory, Neumann has aided us greatly by his free criticism of Breus's view, more especially as regards its central point, - further growth of the membranes after the death of the foetus.

#### GOTTSCHALK.

##### SUMMARY OF HIS VIEW:

That an insufficiency of the primitive heart germ causes early death of the foetus. The membranes however continue to grow, forming folds into which blood flows for weeks and months, finally causing haematomata by its sudden coagulation.

##### CRITICISM.

This being in the main Breus's view, there is no necessity for any separate criticism of it.

As to "insufficiency of the primitive heart germ" it need only be pointed out that the presence of a normal heart has been demonstrated by other authors, and that such abnormalities as acardiac monsters do occur.

#### DAVIDSOHN.

##### SUMMARY OF HIS VIEW:

That blood moles arise from hydramnios ova (i.e. ova with/

with real enlargement of the membranes)- The super-abundant fluid reabsorbs or trickles away, causing a lessened hydrostatic pressure in the amniotic cavity - Subchorial haemorrhages now occur, which loosen the amnio-chorion from the uterine wall, so forming haematomata - Shrinking takes place, with the formation of folds, and a confluence of neighbouring masses to form a variety of shapes.

#### CRITICISM.

Davidsohn's theory can only apply to the development of the very marked forms of blood moles, and for this reason primarily, it is unsatisfactory.

In order for it to work out satisfactorily, the excess of liquor amnii should only be present for a time, and then conveniently disappear, with apparently a cessation of any fresh formation. And yet the pathological conditions which originally caused it, remain, these being according to Davidsohn himself, abnormalities of the decidua, chorion, placenta or foetal organs. It must also be mentioned that there was an excess rather than a diminution of liquor amnii in three of the cases to be described later.

Since this theory can only explain the formation of those moles with haematomata rising into the cavity, what of the sacs with simple finger tip projections? These present the same picture microscopically and are by Davidsohn considered to be less marked forms of the other/

other, and yet by virtue of their striking haematoma formation they are put on a higher plane and supposed to be developed from a hydramniotic ovum.

TAUSSIG AND ENGEL.

It seems unnecessary to offer a separate criticism of the theory of a secondary hydramnios as expounded by Taussig and Engel, since the points brought out against Breus's and Davidsohn's theories apply here also, namely the absence of any proof or likelihood of further growth of the membranes after the death of the foetus, the difficulty of explaining why the overgrowth and excessive production of liquor amnii should suddenly cease, and the inapplicability of the theory to those forms of blood-mole with only finger tip projections.

BAUEREISEN.

SUMMARY OF HIS VIEW:

That deformed villi are found in or pressing on decidual vessels (veins), the circulation is thus blocked and stasis results. The disturbance of its nourishment causes death of the foetus, but the membranes continue to grow. Hydramnios is also supposed to be present at this time.

Haematoidal/

Haematoidal swellings result from the accumulation of blood in the sinuses, and following on the reabsorption of the hydramniotic fluid and cessation of growth of the uterus, folding occurs.

#### CRITICISM.

This strikes one at first as rather novel, but after reading Poten's most interesting paper, there seems no likelihood whatever of its offering any solution. Bauereisen found what he took for deported (verschleppte) villi in decidual veins, but there is of course no real proof that these are deported villi, till as in Poten's cases the whole thickness of the wall has been reduced to serial sections and an absence of any connection with the chorion demonstrated.

Moreover Poten and others have demonstrated that fragments of villi and syncytium are in every pregnancy being carried into the systemic circulation, and only find a lodgement in the minute lung capillaries, or, by back pressure in the vaginal veins. Since then deportation of villi is a feature of every pregnancy, why should it in these cases be supposed to cause barricading of the efferent vessels so leading to stagnation in the intervillous spaces, and haematoma formation.

BERRY HART.SUMMARY OF HIS VIEW:

1. That there is an undue blocking of the serotinal sinuses in the large celled layer, which leads to
2. Slow enlargement of the intervillous circulation- This bulges out the chorio-basal septa, and as these tack down the chorion at definite points, the amnion and chorion will project between them (tuberose swellings.)
3. The embryo dies as a result of interference with its circulation - its death being secondary.
4. The placenta becomes a thrombosed mass, and is retained a certain time before expulsion.

CRITICISM.

Berry Hart's theory seems to me unsatisfactory because it stops short at suggesting any cause for this undue blocking of the sinuses. Granted that there is a physiological thrombosis in the early months of pregnancy, it has certainly crossed the border line and become pathological when it leads to such a series of events as result in the form of abortion sac known as blood mole. Does it not therefore seem reasonable to look deeper into the subject in order to arrive at some conclusion as to what the altered conditions may be which have caused this physiological process to become pathological; and if it is not/

not pathological then why does every pregnancy not end in a blood mole?

Having now put before you the main theories offered up to the present time, as to the causation of bloodmole, and shortly indicated the defects in each, it will be most convenient to give here the details of the eight cases mentioned, before entering upon any fresh theory.

#### CASES.

CASE I. Aet. 24 -- 1-para.

HISTORY. Patient was married 5 years ago, and had a normal confinement a year afterwards, the child not being nursed. Amenorrhoea followed for 9 months, succeeded by an interval of combined amenorrhoea and irregular periods. In May 1903, profuse bleeding came on after cessation of the periods for 3 months, and a lump was passed which no doctor saw. A week later patient sought medical advice for persistent bleeding, and was prescribed for. Amenorrhoea again occurred till the end of December of the same year, when there was bleeding for a few days, followed by "floodings" at irregular intervals during the next month. Patient was/

was admitted to the Hospice on January 30th 1904 for a chloroform examination. The uterus was found enlarged to about the size of a 2 months' pregnancy, and the fornices clear. She was therefore sent home, and an expectant line of treatment was followed.

On February 18th the blood mole to be described later was passed with a fair amount of bleeding, which then ceased.

In May 1904, patient again came under observation, as she was admitted to the Hospice suffering from acute bronchitis. On account of her delicate, tubercular appearance, the lungs were specially carefully examined, and the sputum tested for tubercle bacilli, but with a negative result in both cases. The bronchitis entirely disappeared and on May 24th patient was curetted under chloroform, as she gave a history of irregular **haemorrhages** since the last miscarriage. The periods then returned and remained regular for 8 months, when she again became pregnant. During the latter part of this time, she had been in attendance at the Victoria dispensary, where she had been told that the condition of her lungs was such, that a pregnancy would be a very serious matter. On March 31st there was slight bleeding and pain and some dilation of the os. On April 3rd the foetus was extracted, as its feet were presenting in the os, and the placenta and membranes were removed under chloroform/

form. The temperature was 103.8 the same afternoon, and during the next three days patient complained of severe pain in a hip joint and one finger joint. On the fourth day she developed pneumonia of the right lung and succumbed to it very quickly. The lochia were normal throughout.

DESCRIPTION OF SAC. (Fig. 1. Stereoscopic.)

At the time of expulsion it was a closed sac of greatly thickened decidua, of about the size of a 3½ month's pregnancy, and with no differentiation of the placental area.

It was carefully opened down one side, when a small quantity of fluid escaped. There was no evidence of a foetus.

In its present shrunken condition the sac measures 8½ C.M. in length, by 5¼ c.m. in breadth. A considerable amount of thickened decidua (3) has been removed, and the interior shows haematomata (2) bluish coloured, of varying consistence, sessile, and firmly adherent. Some show facetting through pressure, and the largest one has shrunk from its covering of amnio-chorion, which lies loosely folded on it. Soft brown pigmented material is sprinkled over the amnio-chorion (1), and portions of the wall show a brown tinge.

MICROSCOPICAL DESCRIPTION.

Low Power.

Amnio-Chorion/

Amnio-chorion. The epithelium shows as a broken granular looking line - *masses of fibrin lying on it in places.* which is stripped off in parts.

The connective tissue has a hyaline thickened appearance in places - pigment cells and granules are present in quantity - (Fig.6.) many projections and infoldings of it are seen - with one large haematoma (Fig.5) and in places it is stripped up. The amnio-chorion rests on a fibrin layer, which consists of irregular clumps or wavy strips - pigment granules are present here also, and a few hyaline degenerated villi are embedded in it.

Below this are fairly recent looking haemorrhages, with wavy strips of fibrin running through them, then more fibrinous haemorrhages of a trellis-work appearance, shading for the most part gradually into deeply stained fibrin of irregular conformation (clumps and strands) with hyaline degenerated villi lying in it. Possibly there is some degenerated decidua beyond this. No appearance of anything that can be described as a vessel, but some spaces and clefts are seen here and there.

#### High power.

Amnio-chorion. The epithelium of the amnion, and of the chorion where it can be distinguished, shows extreme granular degeneration. The connective tissue varies from wavy fibrous tissue to an appearance of hyaline/

hyaline degeneration -there are many cells in it, pigmented or non-pigmented, of various shapes and sizes, and also granular pigment.

In the subjacent fibrin layer are seen:

- a). clumps of fibrin.
- b). cells of various shapes and sizes.
- c). red blood corpuscles.
- d). granular pigment.

The haemorrhages show gradations from commencing coagulation to fibrin formation - deep in the fibrin again are spindle and other cells - granular pigment everywhere.

VILLI. All show hyaline degeneration, with granular degeneration of the epithelium - no vessels in them, but cells of various shapes.

#### MICROSCOPICAL DESCRIPTION OF SCRAPINGS.

Low power. (Fig.27.)

The superficial epithelium shows a broken outline. The glands are dilated, and some contain blood.

The stroma has very extensive haemorrhages into it, indeed they have the appearance of replacing it over large areas, with stroma cells, glands, and blood vessels scattered through them. The number of thin-walled vessels is greatly increased, many/

many of them showing considerable dilatation - many thick-walled vessels are also seen.

High power - confirms these points.

CASE II. Aet. 31. IV - para.

HISTORY.

Patient had 2 miscarriages between the first and second child. The fourth child was nursed for 11 months, and died in the end of June 1904. Amenorrhoea continued till the end of September of the same year, when the sac to be described came away. The bleeding appeared to stop at the time, but on visiting patient recently to get the subsequent history, she stated that for 3 weeks after she bled at intervals on exertion or with any excitement. Two normal periods followed, and then 3 months of amenorrhoea, which ended in an abortion on April 2nd of this year.

DESCRIPTION OF SAC. (Fig. 3)

The sac came into my hands entire. It was about the size of a 3 months' pregnancy, and covered with thickened decidua, excepting an area the size of a penny, where the amnio-chorion showed bare. There was no differentiation of the placental area. It was opened with great care, and an unusually large amount of sparkling liquor amnii escaped - there was no sign of/

of a foetus anywhere.

The sac now measures 6 c.m. by 7 c.m.

The interior has a blue-red appearance and shows as it were solid projections of the wall into the cavity, covered by amnio-chorion, and varying from the size of a sixpence to that of a shilling: these on cross section have the appearance of clotted blood.

MICROSCOPICAL DESCRIPTION. Slide 1.

Low power.

Amnio-chorion is of varying thickness - fairly normal

*in all, abnormally few villi -*

A very few villi are lying almost in connection with it, then haemorrhages right back to the decidua which forms quite a thick layer, running into fibrin - a leucocyte infiltration is seen in it. A few villi are gaining attachment to the decidua, others lying free - ~~some fairly normal, others~~ showing myxomatous degeneration. Fresher haemorrhages again beyond the decidua.

High power.

Amnio-chorion shows more or less altered red blood corpuscles and crystals. (These may possibly be an artifact.)

*Many spindle cells, some pigmented cells, some leucocyte infiltration.*

The haemorrhages in the intervillous space are fairly recent - also those in the decidua, which is torn up by them. The fibrin layer is broken up into clumps and strips, and has red blood corpuscles scattered/

(28) *Showing that it is not a normal fibrin layer.*

-tered through it in almost its whole extent. The fi-  
-brin layer passes into an area of altered decidual  
cells lying in an almost colourless homogeneous exuda-  
-tion (Fig.7). The small celled infiltration appears  
under the high power as necrosing fibrin, decidual  
tissue and leucocytes. ~~Crystals are seen in all the  
spaces.~~

VILLI are degenerated (myxomatous and hyaline)  
~~with the exception of a few fairly normal ones~~ - many  
are lying in a thick layer of fibrin - one or two ap-  
-pear to have vessels.

Vessels. These are seen as large and medium  
sized sinuses and small vessels. With the exception  
of a few of the largest and smallest, they have a  
broken and altered outline, and many appear to be  
pouring their contents into the surrounding tissue  
(Fig.8) - a few contain thrombi (Fig.7).

## SLIDE 2.

Low power.

Amnio-chorion is for the most part oedematous -  
in 2 places much condensed - Amnion epithelium strip-  
-ped off in parts.

The amnio-chorion for the most part of its course  
is resting on the fibrinous edge of haemorrhages, into  
which fibrin processes extend forming spaces filled  
with/

*corpuses - (29)*

with blood. There are a very great many more villi than in Slide 1, giving a ~~much~~ nearer approach to the appearance of a permanent intervillous space and for the most part showing degrees of degeneration (myxomatous and hyaline ~~or fibrinous~~) and lying in thick fibrin. (Fig.9.) This has at one end of the slide the appearance of a normal Nitabuch's layer (deeply stained and minus red blood corpuscles). Towards the other end, it is not easy to determine where the fibrinous haemorrhages end and the normal fibrin layer begins. There are large sinuses and smaller vessels in the decidua, and an area of leucocyte infiltration.

*Area of decidua seen. Numerous of pigmented round cells are seen breaking down throughout area of decidua luteo haemorrhages. High Power.*

Amnio-chorion The epithelium of the amnion is stripped up and degenerated looking in parts.

The chorion epithelium shows no Langhan's layer, is blurred and indefinite, and runs into the fibrin layer in many places - the latter appearing to be the thrombosing edge of a haemorrhage. (Fig.10) Tacking of the chorion to the fibrin layer is well seen. (Fig 11.) The spaces before mentioned have no distinct endothelial lining, and contain red blood corpuscles and ~~round cells.~~ *leucocytes.*

Some of the deeply staining fibrin may be due to normal pregnancy changes, but the remainder is evidently a more advanced stage of coagulation of haemorrhages.

Decidual Tissue/

Decidual tissue is present in fair amount, but for the most part much altered - necrosed, torn up by haemorrhages, in one place decidual cells are lying in an almost colourless homogeneous exudation. In one part is seen a large area of necrosing decidual tissue and leucocyte infiltration - there is much brown pigment scattered through the decidua, in the form of round cells breaking down.

Villi. The majority show degrees of myxomatous degeneration or are oedematous. Some show ~~fibrinous~~ or hyaline change. One or two appear to have vessels. There is only a small amount of syncytial budding and but few off-shoots. On most of the large myxomatous villi it is only seen as a thin strip. Here and there are clumps of fibrin with degenerated decidual cells - The villi are embedded in thick fibrin.

CASE III. Aet. 42. 9- para.

HISTORY.

Patient had 6 normal confinements in succession, then a miscarriage at about the 5th month. Then again three normal confinements followed by a second miscarriage (at about the 2nd month.) After this the periods came three-weekly and remained a week (instead/

-stead of four-weekly or longer and remaining 2 or 3 days), with thin discharge between the periods, which became coloured with any unusual exertion or excitement.

Patient says she always had a tendency to bleed at her confinements, and with the last one had a "flooding" 8 days afterwards.

After the second miscarriage, she sought medical advice, and was prescribed for. In October 1904 after 2 months' amenorrhoea she had a third miscarriage (the blood-mole to be described) and in spite of the fact that the sac came away entire, was on the third day losing so much blood, that she was curetted - the curettings were inadvertently thrown away. The periods have since been normal.

#### DESCRIPTION OF SAC.

The sac which was about the size of a 2 months' pregnancy was opened carefully at the time, and there was no appearance of a foetus. In its present condition it measures 6 c.m. in length and  $3\frac{1}{4}$  c.m. in breadth. There are no projections into the cavity, though there is an irregularity of the surface which may be due simply to hardening. But on cross section of any part of the wall the appearance is that of solid blood clot (either bright red or darkly coloured), in fact an exactly similar appearance to that of the cross/

cross section of a projection in Sac 2.

MICROSCOPICAL DESCRIPTION. SLIDE 1.

Low power.

Amnio-chorion is lifted up in a great part of its course and haemorrhages are lying on it. Amnion epithelium shows a very irregular outline. Chorion epithelium has heaped nuclei in parts, thinned in other parts. The connective tissue is undergoing a hyaline degeneration. *There is an infiltration of small cells.* There are haemorrhages from the amnio-chorion to the decidua, intersected by syncytial strands, and a few villi lying in them towards the decidua, which is torn up by haemorrhages, and bounded outwardly by further extravasations.

*Few amount of small called capillations.*

High power.

Amnion epithelium has almost disappeared - a suggestion of a cubical cell here and there - red blood corpuscles are lying on it in most of its course.

The connective tissue shows a swollen and degenerating appearance of many of its cells - some leucocytes and red blood corpuscles are also seen.

Chorion epithelium is only present here and there in the unattached portions, and a leucocyte infiltration has practically taken its place in the attached portions. Two bent septa are seen enclosing a haemorrhage. (Fig 12.) A few cells of Langhan's layer are present/

present here and there - the syncytium has nuclei one or more rows deep.

The haemorrhages of thickly massed red blood corpuscles show no coagulation - they are more darkly stained in the deeper parts.

Decidua is present in much smaller amount - a large area of it shows decidual cells swollen and fusing to form a faintly staining mass - there is a leucocyte infiltration into this, with necrosis in one part. The decidua is in places split up into strips by haemorrhage, or completely inundated. The fibrin layer is present, but very faintly staining, and with red blood corpuscles in parts of it; some of it appears to be coagulated haemorrhage.

*In infiltration area  
are seen: large  
faintly stained cells,  
smaller deeply  
stained & some  
smaller bodies, or  
of nuclei set free*

Villi Some show commencing myxomatous degeneration, others fibrinous or hyaline. An occasional cell of Langhan's layer is seen in places - almost no proliferation of the syncytium.

Vessels.- are seen as sinuses and smaller vessels - they have the same indefinite outline described before.

SLIDE 2.

Low power.

Amnion and chorion with the intervening connective tissue/

tissue are very much better preserved - some tendency to hyaline degeneration - buds of chorion epithelium are being given off. At one end of the specimen there is almost the appearance of a permanent intervillous space, with red blood corpuscles and villi, many syncytial buds and giant cells - (Fig.13.) most of the villi show definite myxomatous degeneration, others advanced or slight <sup>hyaline</sup> fibrinous change - one or two have most distinctly got capillaries. They are gaining attachment to the thin strip of decidua, which at one end shows a fibrinous projection well supplied with vessels. At the other end of the specimen there is extensive haemorrhage with 2 groups of villi and some isolated ones, lying in it, for the most part swollen and degenerated (Fig.14) Haemorrhages again beyond the fibrinous decidual strip, with strands of fibrin.

#### High power.

Amnion epithelium is for the most part very indefinite - red blood corpuscles lying on it in places. Langhan's layer is seen in parts - buds of syncytium are being given off in two or three places. The connective tissue shows hyaline degeneration.

The haemorrhage area shows commencing coagulation in places - there is much dark pigment in the form of round cells breaking down. In the intervillous space

of/

of the more normal portion, fibrin strands and leucocytes are seen.

There is no decidua that can be described as such, but simply a meshwork of fibrin, containing red blood corpuscles and leucocytes scattered over it in indefinite spaces, which stand out more distinctly with the low power.

Villi. As described under the low power these show swelling or myxomatous or <sup>hyaline</sup> fibrinous degeneration - in 2 or 3 vessels are seen (thrombosed in 2 cases.) A few cells of Langhan's layer are present, and a fair amount of syncytial budding. The villi of the haemorrhagic part show much more marked degeneration both in their outlines and in the connective tissue - they are lying in fibrin. Many villi are firmly attached to the decidua (?) by proliferation of Langhan's layer.

Vessels. The only appearance describable as vessels are the indefinite spaces in the fibrin layer.

CASE IV. Aet. 39. viii- para.

#### HISTORY.

Patient had eight children by her first marriage, and no miscarriages. The second marriage was in March 1904./

1904. Amenorrhoea in April, May, June, and a miscarriage in July which no doctor saw. Patient went on bleeding for a fortnight. The periods returned in August and September in fairly large quantity, with a thin white discharge in the intervals. Amenorrhoea in October and November, then bleeding began a week before the new year, and the sac to be described was expelled in a day or two, with practically no bleeding afterwards. Regular periods since.

DESCRIPTION OF SAC.

The sac was about the size of a  $2\frac{1}{2}$  months' pregnancy in length, but narrow in proportion to the length. There was no differentiation of the placental area. On cutting it open so deep had the knife to be carried before the amnio-chorion was laid bare that it was at first thought to be possibly only bloodclot.

The sac now measures 8 c.m. by  $2\frac{1}{2}$  c.m. The wall has the appearance of firmly clotted blood, and its thickness is so great that the actual cavity is reduced to the size of a narrow tube, which in the fresh state did not measure more than a quarter millimetre across. The amnio-chorion is seen lying deep in the incision, and a probe can be pushed between its folds (i.e. into the amniotic cavity.)

MICROSCOPICAL DESCRIPTION./

MICROSCOPICAL DESCRIPTION.Low power.

Amnio-chorion is undergoing hyaline degeneration - it is well supplied with cells, and there is an appearance of vessels. There are haemorrhages on and into the amnion, which appears to have lost its epithelium. No appearance of Langhan's layer. The syncytium runs along (with one, two, or multiple rows of nuclei), either attached, in process of being separated, or lying at a distance, touching the thrombosed edge of haemorrhages (Fig.15) which extend right back to the decidua. These are coagulating in parts, and at one end of the specimen form successive fibrin layers from the chorion to Nitabuch's layer, fresher haemorrhages and villi lying between them. (Fig. 17.) At the other end villi are scattered throughout them, and are apparently becoming obliterated in one part.

Nitabuch's layer shows areas of red blood corpuscles, beyond this is a big strip of decidua with again smaller strips and haemorrhages.

High power.

Amnio-chorion is rather more cellular than normal, a few cells being pigmented. Several vessels are seen, and in parts an appearance of hyaline degeneration.

The/

The amnion shows an endothelial-like covering here and there, but for the most part a broken outline, and haemorrhages on and into it. No Langhan's layer. Syncytium as described under low power.

The haemorrhages are coagulating deeper, and villi are being obliterated. In the fibrin layers at the other side of the specimen one can see that the fresher haemorrhages are advancing to fibrin formation - these layers show degenerated cells and fragments of nuclei or leucocytes. Nitabuch's layer and the decidua have also extensive haemorrhages into them. (Fig.18) The decidual cells are much altered, smaller than normal, and in parts there are large necrosed tracts. Extensive leucocyte infiltration is seen. (Fig.16.)

Villi - show myxomatous or hyaline degeneration, some have vessels. The syncytium on most of them is undergoing fibrinous degeneration, though in parts better preserved, and in the oedematous villi is frequently reduced to a layer of flattened cells.

Vessels - are seen in fair number, but none with the exception of a few very small ones, have any distinct lining, and many appear to be discharging their contents into the surrounding tissue. A few vessels in the outermost portions of decidua show some evidence of commencing thrombosis.

CASE V.    Aet. 32.    VI - para.

HISTORY.

Patient had first two healthy children, then a miscarriage at the  $2\frac{1}{2}$  month, after which she bled for one month. She was curetted under chloroform, when some adherent bits were removed, and had to be treated for resulting anaemia. She had again 2 normal confinements, no trouble with the placenta, but inclined to bleed a little afterwards, and rose anaemic, needing to be re-stored with iron and arsenic.

In June 1903 she was 5 months pregnant and feeling ill - a swollen labium, varicose veins etc. On July 20th there was a premature birth of a 6 months' foetus - the placenta was adherent and had to be manually detached. Patient had again 2 periods, and then amenorrhoea till March 14th 1904, when the sac to be described was passed. In February of this year, she had a small full time child which only lived a week.

DESCRIPTION OF SAC.

At the time it was passed, the sac was about the size of a  $2\frac{1}{2}$  months' pregnancy.

It now measures 6 c.m. long and 3 c.m. broad. It is formed of thickened decidua, of the shape of a cotyledon. It has been cut open through the middle, avoiding/

avoiding the cavity, which consists of a slit between the 2 halves of the divided cotyledon. The cavity has a markedly convoluted appearance and is lined with amnio-chorion which has soft pigmented material scattered over it. That this convoluted appearance is not due simply to shrinking is evidenced by a glance at the cross section which shows a dark haemorrhagic area opposite such a convolution.

Attached to the inner wall of the cavity by a very short cord ending in a folding of the amnio-chorion is a shapeless soft mass about  $\frac{1}{2}$  centimetre in length and also covered with pigmented material. (the foetus?)

#### MICROSCOPICAL DESCRIPTION.

Amnio-chorion shows thickening, hyaline degeneration and much folding - pigmented cells are present. Masses of fibrin are seen lying on the amnion and in the amniotic cavity.

Fibrin layers are seen along the chorion outwardly, filling up the blood spaces between the villi, and one large blood space in the chorion and a villus conjointly, at the point where the latter is being given off. Outwardly from this at one end of the specimen are fresher but thrombosing haemorrhages going on finally to fibrin formation, with hyaline degenerated/

-generated villi lying in them - in the remainder of the specimen, there is almost entirely fibrin encircling hyaline degenerated villi and blood spaces, and then decidua.

High power.

The amnion epithelium is for the most part stripped off, attached here and there, but showing extreme granular degeneration. Balls of fibrin can be seen working their way into the amnion, as described by Taussig. There are numbers of pigmented cells and leucocytes. No Langhan's layer or syncytium can be described - the chorion in its whole length either adheres to the fibrin layer or is tacked at short intervals over a large area (Fig. 19). The fibrin layer is broken into masses and strands, with much dark pigment and leucocytes.

The haemorrhages are in various stages of coagulation.

Decidua. The decidual cells show very faintly staining. Where haemorrhages have occurred into the decidua, strands of fibrinous material are seen running through them.

Villi. Most of them are embedded in fibrin and show extreme hyaline degeneration - on one or two, fragments/

fragments of syncytium are present and also syncytium undergoing fibrinous degeneration.

Vessels. Most of the large sinuses, both superficial and deep show a tendency to thrombosis of their contents, and in others the lumen is outlined by what appears to be proliferated endothelium and leucocytes combined - (Fig. 20.)

CASE VI. Aet. 28. V- para.

HISTORY.

By her first marriage, patient had one child and no miscarriages. By the second marriage there were 4 children, the youngest being nursed for twenty months, and weaned in May 1904. In June patient had a "flooding," which lasted a week, then cessation of the periods till August 20th of the same year, when patient was seen for the first time on account of bleeding. The uterus was found to be about the size of a 2½ months' pregnancy, and the cervix, not dilated, was noted as lacking the typical velvety feeling. Sedatives were given, but 2 days later bleeding came on in fair quantity, and the sac was passed entire. It unfortunately disappeared from the collection, and so is described from memory.

DESCRIPTION OF SAC.

It/

It was entire, consisted of greatly thickened decidua, and was about the size of a 2-2½ months' pregnancy. On carefully opening it, a large quantity of sparkling liquor amnii escaped, with a few small fragments. The interior showed several finger-tip projections into the amniotic cavity, covered with glistening blue amnio-chorion.

MICROSCOPICAL DESCRIPTION LACKING.

SUBSEQUENT HISTORY.

Patient reported herself on September 27th as having had some irregular bleeding since going about again, and what might be described as an extremely profuse period during the last 5 days, and still not diminishing. Patient was admitted to the Hospice the following day and curetted under chloroform. One normal period followed, and patient is now 6 months pregnant.

The Laboratory report of the scrapings was:

"Microscopical examination doubtful, possibly sarcomatous."

MICROSCOPICAL DESCRIPTION OF SCRAPINGS.

Low power.

In parts there is a very marked increase of the interstitial tissue, which appears in some places to be pressing upon and destroying the glands. In other parts/

parts this increase is less marked or the stroma appears to be even rarefied. There is a multiplication of the thin-walled capillaries, many of which are greatly dilated. Extensive haemorrhages are scattered through the stroma in every part of the slide.

High power.

Confirms these details, and shows very plainly the destruction of the glands by the increase of interstitial tissue.

CASE VII. Aet. 32. 2-para.

HISTORY.

Patient had her first child 8 years ago - there was no special delay during labour, to account for a sharp haemorrhage which occurred before the expulsion of the placenta.

The second child was born 5 years ago, confinement normal. After this normal periods till April 15th 1904, then amenorrhoea up to January 29th 1905, when the specimen to be described was expelled. Patient states that when about  $4\frac{1}{2}$  or 5 months pregnant, she got a fright from a dog, and a dark-coloured discharge began, which lasted for 2 days, then stopped for a time and returned again for one day. Patient was very nervous after this, but had no suspicion of anything/

anything abnormal in her pregnancy.

Normal periods since the expulsion of the sac.

DESCRIPTION OF SAC. (Fig. 2. Stereoscopic.)

At the time it was opened a small amount of liquor amnii escaped, and there was a large quantity of clay-coloured material in the sac.

The specimen consists of a well differentiated thickened placenta (2), of about 5 or  $5\frac{1}{2}$  months according to development, fairly triangular in shape, the sides of the triangle varying from  $6\frac{1}{2}$ -7 c.m. Its external surface is shaggy. On its internal surface the amnio-chorion (1) is drawn over solid swellings of varying sizes, one large round one (3) - the appearance, (as in specimen VI,) being best described as one of marked convolutions. These on cross section are seen to be formed by bloodclot, though paler and more organised looking than in VI, and less sharply defined from the remainder of the wall. The surface of the amnio-chorion is covered with soft reddish material, giving the appearance of a sprinkling of cayenne pepper.

From the edge of the triangular placenta, rise the foetal membranes, having some prolongations of the united decidua vera and reflexa onto them. The same also persisting as broad bands at the corners of the placenta (5). Rising from between two small haematomata, towards the edge of the placenta, is a slender cord/

cord  $11\frac{1}{2}$  c.m. in length(4) suspending the  $6\frac{5}{4}$  c.m. long foetus, which is well preserved. Judging from its length and stage of development it has the appearance of a 3 months foetus, the special points being, a) loss of webbing of toes and fingers, and appearance of nails as fine membranes, b) eyes protected by lids, c) progressing differentiation of sex.

#### MICROSCOPICAL DESCRIPTION.

##### Low power.

Amnio-chorion in a large part of its course is stripped off and much broken up (possibly during preparation). Where detached it shows some amount of hyaline degeneration at either side, with many pigmented cells, especially in one portion.

The amnion-epithelium has for the most part the appearance of a granular interrupted line - no chorion epithelium can be described. A fibrin meshwork occupies the intervillous space, with some degenerated villi lying in it towards the amnio-chorion and again deeper. It consists of fibrin waves, staining much deeper in some parts than in others, and also showing tracts of fresher but thrombosing haemorrhages. Towards the decidual end, hyaline degenerated villi and calcium crystals are lying in it. Beyond this is an indescribable area of much degenerated and fragmentary villi, calcium crystals and shreds and strands. There is no definite appearance of vessels, but some spaces are seen.

##### High power.

The amnion epithelium where present, is so degenerated, that/

that a description is not easy: it is an amorphous granular mass, with an almost crystalline appearance in parts. There is no chorion epithelium that can be described as such. Many pigmented cells and scattered pigment granules are seen, and throughout is the same refractile appearance already described. Fibrin clumps are seen in the amniotic cavity.

The intervillous space is described under low power - pigmented cells are scattered through the haemorrhages in parts.

Villi-- for the most part show a combination of hyaline degeneration and fibrous tissue change, with shrunken and pigmented nuclei scattered through them. In some, calcium crystals are seen. Many of them in the area towards the decidua are greatly degenerated and broken up or mixed with fibrin masses. The only appearance of any epiblastic covering is that of a homogeneous glancing line round some. There are also fibrinous shreds and strands, with much débris and crystalline material, in addition to the calcium crystals.

Some altered decidual cells are seen in a stretch of what is apparently degenerated decidual tissue.

The following case is described here as being a combination of hydatid and bloodmole::

CASE VIII.    Aet. 21.    0-para.

HISTORY.

Patient was married in July 1903. The three periods succeeding her marriage were missed, and an abortion followed in the fourth month, no doctor being in attendance. There was regular menstruation for the next 6 months, then four periods were missed, and early in September 1904, bleeding began. The uterus was then enlarged to about the size of a 3 months' pregnancy. A day or two later, sharp pains came on, and the sac was passed. As the bleeding still continued and because of the diagnosis of hydatid mole patient was curetted a few days later.

Following this there was one normal period, and patient is now 6 months pregnant, active foetal movements have been felt by palpation, and the foetal heart heard.

DESCRIPTION OF SAC.    (Fig. 4.)

It was about the size of a  $3\frac{1}{2}$  months' pregnancy in length, but rather narrower in breadth - an ordinary amount of liquor amnii escaped on opening it, and there was no appearance of a foetus.

The sac now measures 11 c.m. in length and 5 c.m. in breadth.

It shows as its outermost covering, soft, thickened decidua vera (this has been almost entirely destroyed/

destroyed at one side during preparation of slides.)

At the uppermost end there is an appearance of placental differentiation in the form of a raised area of vesicular villi crowded together (much less marked now). The lower end of the sac is simply a rounded thickened end of decidua, almost forming a cast of the cervix. Within the covering of decidua vera is a second sac, united to the outer one at the placental area, and in the lower part, but separate in the inter-vening portion. This shows outwardly reflexa, and inwardly amnio-chorion, and between these two but not growing through the reflexa, thickly massed vesicular-ly degenerated villi, but of much smaller size than those at the placental area.

#### MICROSCOPICAL DESCRIPTION.

##### SLIDE I. Placental area.

##### Low power.

Amnio-chorion is doubled on itself in the specimen - and is normal looking - buds and villi are being given off from the chorion.

The majority of the villi show either advanced myxomatous degeneration, or are oedematous or fibrin-ous.

The fibrin layer is quite well marked - following on this a layer of decidual cells, an area of necrosis, a stretch of haemorrhages and leucocyte infiltration - then/

then again decidual tissue in strips.

High Power.

The amnion epithelium is well preserved in parts, but with broken intervals - it is tending to become cubical. Areas of fibrinous haemorrhage are seen on it. The connective tissue is oedematous in parts. There is an interval between the amnion and chorion with connecting fibres. The cells of Langhan's layer can be followed in long stretches. The syncytium is reduced to a strip or degenerated looking. In two places (the one extending over a long stretch) fibrinous thrombi are attached to the chorion.

The fibrin layer of Nitabuch shows degenerated decidual cells and nuclei in its meshes. Then there is a layer of decidual cells, staining in varying degrees, and showing a blurred outline in parts. In it are many large sinuses, two communicating with the intervillous space, and all containing strands of fibrinous thrombi. These sinuses have in parts a distinct endothelial lining, or are degenerated. Below this layer and sharply defined from it is an area of leucocyte infiltration and necrosing decidual tissue, and further over in the specimen another very much larger area of intense necrosis containing shadows of decidual cells, and sinuses with much degenerated walls, filled/

filled with fibrinous thrombi. In one place a large sinus has overflowed into the surrounding tissue, and the whole area is thrombosing. Beyond this again is a strip of more normal decidual tissue, and empty but fairly healthy sinuses. Across a stretch of villi is a much larger piece of decidual tissue with the same conditions more varied. Three round sinuses act as a landmark. Again are seen well marked decidual cells towards the fibrin layer, necrosed tissue below - then a narrower neck of decidual cells with 3 or 4 empty vessels containing fragments of fibrin strands, and a few larger empty ones, followed by one blocked with fibrinous thrombus. This leads on to a long stretch of leucocytes infiltration and necrosis. Wide sinuses are seen in great numbers, empty or containing fibrin threads, and also stretches of haemorrhages into the tissues, commencing to thrombose.

In the intervillous space one is struck by:

- 1). The absence of red blood corpuscles, and the presence of strands of fibrin.
- 2). The unusual size of many villi.
- 3). The fragmentary and shrunken condition of other villi.
- 4). The number of plasmodial masses.

Villi show extreme myxomatous degeneration, or are/

are oedematous or fibrinous (Fig.23.) There is a proliferation of Langan's layer in the plasmodial masses, on the surface of the decidua and in some villi. The syncytium is stripped off in many places, and degenerated masses of it are also seen.

SLIDE 2. Reflexa, Amnion and Chorion.

Low power.

Amnio-chorion is doubled on itself in the specimen. The amnion epithelium is interrupted here and there. Villi and buds are being given off from the chorion. Many villi show advanced myxomatous degeneration, and the remainder are oedematous.

Nitabuch's fibrin layer is seen, with villi gaining attachment to it, then a long stretch of leucocyte infiltration and necrosis (Fig.24) and in another portion of the decidua, extensive haemorrhages are seen. (Fig.24)

High power.

The amnion epithelium is for the most part cubical, in places more endothelial in character - proliferated masses of amnion epithelium are seen.

The connective tissue is undergoing myxomatous degeneration. Langan's layer is much proliferated, especially in one part on the chorion where it also shows/

shows extreme vacuolation. (Fig. 25.)

Decidua shows a normal fibrin layer with remains of decidual cells - then a layer of decidual cells, faintly staining, swollen, and coalescing - and beyond this a layer of leucocyte infiltration and necrosis - then two stretches of thrombosing haemorrhages, and a detached portion of decidua with haemorrhages even in-  
-to the fibrin layer. One or two sinuses can be seen as in slide I., with fibrinous thrombi. A few hyaline degenerated villi are lying in thick fibrin.

Villi show advanced myxomatous degeneration or are immensely swollen - in many is seen the remains of Langhan's layer. The syncytium is proliferated in parts irregularly, and elsewhere stripped off in stretches - no vessels are seen with any certainty. There are different stages of red blood corpuscles ad-  
-vancing to fibrin formation in the intervillous space.

SLIDE 3. Vera only.

Low power - shows decidual tissue partly broken up into strips, and full of large sinuses and smaller vessels. The sinuses appear blocked or are quite empty. The smaller vessels are of two kinds, both seen in groups:

- a). thin-walled.
- b). thick-walled, almost to obliteration in 1 or 2 cases. (Fig. 26.)

High power./

High power. The decidual tissue is very broken up looking. Here and there sinuses are emptying into the surrounding tissue, and fibrin formation following. In others the contents are more or less altered or thrombosing, or are empty. The endothelium shows a broken outline in places, or is being cast off. The thickened vessels appear to be an exaggeration of the thick-walled capillaries described under the normal endometrium.

#### MICROSCOPICAL DESCRIPTION OF SCRAPINGS.

##### Low power.

Much decidual tissue is seen, faintly staining and oedematous, and in parts tending to revert to the appearance of normal endometrial stroma. There are degenerated villi, losing their characteristic appearance, scattered through it. The vessels are all greatly dilated, some showing a normal endothelial lining, others a degenerated outline. In one part of the field is a group of large sinuses, bounded by broad fibrinous bands (Fig.28.) The contents of some vessels show thrombosis, others are empty or nearly so. There are extensive haemorrhages into the decidual tissue, showing some amount of fibrin formation and an area of necrosis.

While/

While quite recognising that in each of the theories reviewed there is more or less of truth, it yet seems from a study of these eight cases, that there is need for a fresh view of the subject, which would go nearer to the root of the matter by especially directing attention to the endometrium as the most important factor in determining the production, either of a normal foetus or an abortion sac. As has been already indicated, Taussig and Engel have vaguely suggested this, without incorporating it in their theories, or in any way attempting to work it out. Dr. Berry Hart has touched on it by his particular study of the condition of the sinuses in this form of abortion sac, and three authors, Gottschalk, Taussig and Bauereisen, having described endometrial scrapings after bloodmole, without however offering any special comment.

On comparing the histories of the present cases, the prominent feature was undoubtedly the existence of an unhealthy endometrium in conjunction with the production of these moles. The recognition of this fact led naturally to the study of the normal endometrium, in relation to the implantation of the human ovum. It is thus advisable to first shortly discuss its structure, the normal pregnancy changes occurring in it, and such abnormal endometria as might be supposed to have a bearing on the production of blood-moles./

-moles.

THE NORMAL ENDOMETRIUM consists of:

- 1). Lining epithelium.
- 2). Glands.
- 3). Interglandular stroma.

The lining epithelium is composed of ciliated columnar cells, with their nuclei (round-ended rods) placed, as a rule, deeply. A basement membrane of flattened connective tissue cells may sometimes be seen.

Invaginations of this lining epithelium form the glands. These are tubular, simple or branched, the branchings occurring for the most part in the deeper layers of the endometrium. The majority of them run obliquely to the surface, and they have in parts a basement membrane similar to that on the surface of the mucosa.

The interglandular stroma is composed of connective tissue of an embryonic type. The cells have no distinct outline, but a well-marked round or oval nucleus, and are connected by anastomosing processes, thus forming a network with well-marked spaces, which contain lymph and leucocytes. Groups of round cells are also seen here and there, without anastomosing filaments.

Vessels./

Vessels.

Arteries and veins extend in from the muscular part of the wall, the former running a tortuous or spiral course, the latter a straighter one. These pass into capillaries about the middle of the mucosa, two varieties being found: a). thin-walled, with an endothelial lining, b) thick-walled with encircling layers of flattened connective tissue cells. Arteries with an adventitia are found in the muscular layer.

Keeping to the same order, a resumé of the Physiological Pregnancy Changes in the Endometrium (as decidua vera, reflexa and serotina) will now be given. (b and c of course only applying to the serotina and reflexa, and all the changes described running a shorter course in the reflexa.)

- 1). Lining epithelium. This undergoes degeneration, and has entirely disappeared by the fourth month.
- 2). Glands. Narrowing and obliteration of the portions in the compacta occurs from pressure of the increased decidual tissue, and enlargement of the portions in the spongy layer up to the fourth month, then an increasing elongation parallel to the surface, till by full time hardly any spaces exist. The glandular epithelium after a preliminary hypertrophy in the spongy layer, shows progressive/

progressive degeneration.

3). Interglandular tissue.

- a). The most important change in it is the development of the decidual cells by hypertrophy of the preexisting embryonic cellular elements, this process commencing in the outer part of the compact layer. These cells are of various shapes (round, oval, polygonal or spindle-shaped), and have a large round or oval nucleus; connecting processes may be present or absent. The decidual cells show degeneration by the second month (rather earlier in the serotina.). This is apparent as flattening parallel to the surface, with swelling and fusion of cells into a faintly staining vacuolated mass, containing irregular nuclei. During the later months of pregnancy they tend to revert to embryonic interglandular tissue.
- b). As early as the sixth week (earlier in the reflex-a), the superficial portion of the compacta is occupied by an irregular layer of fibrinous material; called Nitabuch's layer. It contains degenerating cells, but no blood corpuscles, and stains deeply with eosin. This continues to increase till the end of pregnancy, becoming denser and more structureless, and sending prolongations downwards, even into the spongy layer.

c)./

c). Absorption of the decidua, with a compensatory new formation, is going on throughout pregnancy, in part due to the phagocytic action of the foetal epiblast. The latter is seen in the earliest ovum described (Peters' 5 days old one), in the form of strands of nucleated cells extending into the decidua. These show vacuolation and intervening spaces, many of which contain maternal blood: moreover a transformation of their outer layers (i.e. those in contact with blood) into the nucleated protoplasmic masses of syncytium, can be followed.

It is not necessary to devote more attention to this reticulated structure, which fixes and nourishes the ovum during the first two weeks. It is then replaced by the syncytium, which, increasing till the fourth month, continued on the down-grade till the end of pregnancy. This leads to a consideration of the very important part played by the syncytium in the maintenance of foetal nourishment, as has been worked out by several authorities, British and German.

SCHOLTEN and VEIT have shown that there is a mutual action going on constantly between syncytium and red blood corpuscles. The syncytium has a haemolytic action on the latter, dissolving out the haemoglobin, anchoring it to itself, (Ehrlich's "Seitenketten" theory)/

theory) and passing it on to the foetal blood corpuscles in the intervillous capillaries. The maternal red blood corpuscles in turn have a syncytiolytic action on the syncytium, dissolving it in the maternal blood, and thus eliminating the material of foetal retrogressive metamorphosis.

KWOROSTANSKY says:

"Why does one find foetal elements for example, in the capillaries and veins of the muscle? Because they are not only the agents of haemoglobin and gas exchange between foetal and maternal blood, but also contain the excretion products of the foetal blood, and the fragments of maternal red blood corpuscles. The destruction of corpuscles takes place with the normal function and structure of the placenta, and on account of lack of oxygen, the foetal epithelial cells must reach into the maternal veins."

VESSELS.

d). Dilatation of the capillaries to form sinuses, has already begun in Peter's specimen. These having reached their highest point of development in the serotina in the 5<sup>rd</sup> month, then show degenerative changes which will be discussed later. These sinuses communicate on the one hand with the intervillous space, and on the other with the tortuous arterial vessels, which have been noted in the description/

description of the normal endometrium, the blood-stream being thus reduced to a slowly moving mass. The communication with the intervillous space is effected gradually by the extension of processes of trophoblast and syncytium into the sinuses. Nature has thus done everything in her power to reduce to a minimum the shock of any sudden changes in the maternal circulatory system, in order that the villi may be permitted to retain their hold upon the decidua, and time be given for the interchange of food-stuffs and gases between foetal and maternal blood.

- e). By the 6th month fibrinous changes are seen in the walls of many sinuses, diminishing their size. (This applies of course only to the serotina, for those of the vera are already to a great extent obliterated, and the reflexa is by this time out of count.) Thickening of the intima is also seen in many of the smaller vessels of the decidua and muscular layer, and endothelial proliferation in others.
- f). Blood extravasations have been noted in all three deciduae in the first three weeks.

Having thus shortly indicated the changes which the normal endometrium is called upon to undergo, in order to/

to play its part in the complicated process of the development of the embryo, it seems not unreasonable to lay some stress on pathological conditions of the same, as a factor in variations in nourishment of the ovum.

#### ABNORMAL ENDOMETRIA.

Pathological variations in the endometrium are very great and very numerous, and on this account it is here intended only to touch upon those which would be likely to have a bearing on the formation of blood-moles. That is to say those showing a change in the vascular conditions, since haemorrhage is the characteristic feature of all blood-moles.

#### 1). Chronic congestive oedema of the endometrium (Glandular Endometritis.)

In this form there is oedema of the stroma cells and glandular epithelium, enlargement of the glands, and increase in the number of the thin-walled veins and capillaries, especially near the surface of the endometrium. These dilate easily, occasionally forming what appears more like venous sinuses than capillaries, and show a ready tendency to haemorrhage.

#### 2). Sarcoma-like changes in the endometrium.

The interstitial tissue is here increased in such amount/

amount that the glands become irregular or are destroyed. There is also in this form an increased production of thin-walled capillaries, showing a ready tendency to haemorrhage. From the nature of the stroma and vessels of the endometrium it is difficult to differentiate between very early sarcoma and simple hyperplasia. For this reason the term sarcoma-like has been employed here.

3). Arterio-Sclerosis of the vessels of the endometrium.

There is in this variety a proliferation, thickening and hyaline degeneration of the arterioles of the endometrium, associated with similar changes in the general arterial system of the uterus, but usually not in the general vascular system. Its characteristic feature is severe and intractable haemorrhage.

Having now clearly before us a picture of the changes occurring in the normal endometrium during the development of the embryo, let us consider how and at what stage in the development, any of the three pathological endometria mentioned, might fail to similarly respond to the stimulus of the growing ovum.

Kworostansky has worked out in detail, through an interesting series of cases, the great fact that the method/

method of ovum implantation varies according to the varying structure of the **mucosa**, and that the latter most particularly affects the intimacy of attachment of the villi. He has also in this connection drawn special attention to glandular endometritis (chronic congestive oedema of the endometrium), as being from its structure peculiarly ill adapted to effect any firm union with the foetal elements.

This form of endometritis may be accepted as the one most commonly met with, its chief characteristics being:

- a). oedema of the stroma.
- b). hypertrophy of the glands.
- c). multiplication and dilatation of the thin-walled capillaries at the surface of the endometrium - and,
- d). tendency to haemorrhage.

In the variety of endometrium described as showing sarcoma-like changes, multiplication of the capillaries is specially to be noted, and in the arterio-sclerotic form, degeneration of the arterioles, and since, in both these, there is a ready tendency to haemorrhagic extravasations into the stroma, the latter cannot be considered in either case a structure calculated to produce a normal implantation. The ovum thus starts life handicapped by an inability to effect

a/

a firm hold on the decidua, by reason of the over-oedematous and over-glandular condition of the latter. It must not however be forgotten that the cause of this inability may in part be foetal, and due to a maldevelopment of the trophoblast. The influence of an imperfect union between ovum and decidua is so much the greater, since, according to the most recent teaching, the ovum may be regarded as from its earliest days dependent upon the decidua for its nourishment, this being the most feasible explanation of the early established trophoblastic connection between foetal epiblast and maternal blood. The yolk supply in the human ovum is thus of very insignificant importance, and the allantois may be regarded simply as serving to guide the foetal vessels through the belly stalk to the placenta.

The failure of such an intimate connection as this, will naturally lead to impairment of nutrition of the villi, and a resulting imperfect development of the ovum.

Having now considered the bearing of the abnormal stroma on the development of the young ovum, the vascular consideration next deserves attention, particularly in the first and second forms of abnormal endometria, since these are much more commonly met with, and are exemplified in two of the eight cases.

To/

To repeat: in the first there is a multiplication and dilation of the thin-walled capillaries near the surface of the endometrium, and in the second, a multiplication alone, with in both cases an increased tendency to diapedesis or to rhexis.

These pathological capillaries are now called upon to form large sinuses, communicating on the one hand with the intervillous space, and on the other with the tortuous arterial vessels of the deep mucosa and uterine wall. It must further be remembered that during this process of dilatation they have to undergo the additional strain of the monthly recurring crises. These "physiological crises of pregnancy" as described by Bossi, and more minutely by Verf, correspond to the menstrual period in abeyance, and "as far as the genital apparatus is concerned, evidence themselves by a disposition to bleedings, a diminution of the strength of the uterine tone, and most particularly in the increased tendency to abortion on those days"

If this strain be greater than these pathological capillaries can bear, they rupture here and there, thus acquiring additional weak points, in spite of which they continue their process of conversion into sinuses, which are on the up-grade till about the third month. A succession of haemorrhages can then be pictured, primarily into the intervillous space/

space (Fig.14), and later involving the decidua also. That this is what actually occurs is proved by a microscopical study of the specimens, which shows haemorrhages of various periods, in the form of fibrin waves and fresher extravasations, (Fig.21) pigmented cells and scattered pigment (taking on the iron stain or not) (Figs. 6 and 19) crystalline material, and even (in one case) calcium crystals (Fig.22). There is also the additional evidence of the pigmented material seen in the interior of some sacs.

It cannot be supposed that villi, whose hold on the decidua normally depends upon the extremely delicate arrangement of the foetal circulatory system, can withstand these successive shocks. On the contrary, they are torn from their decidual attachment in greater or lesser number, and left suspended in the intervillous space, there to undergo degeneration.(Figs.9 and 14.) The foetal system of food and gas interchange is thus partially thrown out of working order, since the ovum depends for its nourishment not only upon a superficial firm attachment of the villi, but upon the prolongations of the foetal epiblast into the deep decidua, to draw upon fresher maternal blood; for so only can the syncytium efficiently perform its all-important work of food and gas interchange between foetal and maternal elements.

In all the sections examined, the amount of syncytium/

cytium on most of the villi was below normal, and degenerative changes were present. This condition as far as the endometrium is concerned, is secondary to the imperfect attachment of the villi, or to the excessive haemorrhages present, since recent writers have shown that the blood and syncytium have a mutually antagonistic action. The possibility of a primary mal-development of the syncytium must be kept in mind, but the present facts are not a sufficient warrant for the expression of an opinion on this point.

Thus an inadequate amount of nourishment is carried to the rapidly developing foetus, and conversely its excretory products are imperfectly got rid of.

It would follow naturally that a young ovum would die, and undergo partial or complete absorption, while a more mature foetus might die, or continue its development, but imperfectly, so that, at the time of expulsion, its size would be entirely out of proportion to the stage of pregnancy. The exact age of the foetus at death cannot be determined, as the ordinary criteria (length, state of preservation) are not applicable. The quantity of liquor amnii present will also, as has been suggested by some writers, have a considerable influence on the early absorption of the foetus, or its good preservation.

From a study of these eight cases it appears that the haemorrhages/

haemorrhages with their resulting changes, may occur before or after the formation of a definite placenta.

In order to investigate the point as to whether, in these uniformly thickened sacs, placental differentiation was in progress or not, before the onset of the haemorrhages, slides were prepared in Cases I, II, and III, from two different portions of the sac, these naturally being chosen at random. In II and III it was interesting to find that in spite of the absence of any appearance of a placental area externally, the second set of slides prepared showed a strikingly larger number of villi, giving at the first glance a much nearer approach to the appearance of a normal intervillous space (Figs. 9 and 13), and also more evidence of successive haemorrhages. As regards case I, no difference was found in the two sections, but since II and III had clearly demonstrated the possibility of placental differentiation being in progress, the investigation was not further pursued.

It follows therefore that in those cases where the haemorrhages occur during the progress of placental differentiation, they are most marked at the placental site, but also occur over the remainder of the decidua, as would seem natural since sinuses are still present there. If the placenta however is definitely formed, the haemorrhages are limited to it, as is well demonstrated/

demonstrated in case VII, where the placenta is seen as a thickened mass, with well-marked solid haematomata, while the united decidua vera and reflexa are only present as bands and shreds.

In the first case, the final result may be either a bloodmole (of the type described by Breus, or a less marked form) or a fleshy mole, the latter being simply a more organised variety of the former. Each of these again is either empty or contains a foetus of imperfect development.

In the second case, there is a fleshy mole as a placenta, with a foetus of a proportionately much lower grade of development, as in case VII, where the placenta is of about 5 or 5½ months development, and the foetus not more than the third month.

Taking up the first, i.e., a blood or flesh mole, let us study the details of its formation, returning to the point where the overstrained sinuses are yielding and discharging their contents into the intervillous space and superficial decidua. As already stated, microscopical study has demonstrated the occurrence of a succession of such haemorrhages, and with the suddenly increased volume of blood in the intervillous space, the amnio-chorion is pushed forward, either en masse or in the form of commencing haematomata.

Dr. Berry Hart has demonstrated the formation of these by the bulging forward of amnio-chorion between two/

two chorio-basal septa. True chorio-basal septa (i.e. those uniting decidua and chorion) have not been seen in any of the slides described, but a haemorrhage close under the chorion enclosed between two septa, which have not a direct communication with the decidua is figured (Fig. 12).

In the slides of cases II, V, and VII, an appearance of tacking of the chorion to the subjacent fibrin layers is well seen (Figs. 11 and 19) and suggests the possibility of these attaching points acting in the same way that chorio-basal septa have been supposed to.

From a further study of the slides, however, there seems every likelihood that a secondary thrombosis occurs in the sinuses of the compact layer, causing actual stagnation in the intervillous space, and completing the process of haematoma formation (Fig. 5)

Vessels showing early hyaline thrombi or fibrinous thrombi have been described and are figured (Figs. 7 and 20) and their occurrence is not to be wondered at when one remembers that two causes of thrombosis are present in these cases:

- a) altered vessel-walls.
- b) stagnation in some vessels through the pressure of an extensive haemorrhage.

An extension of the haemorrhages throughout the decidua/

decidua (Fig.18), and from the deeper vessels takes place later, along with, in some cases, a leucocyte infiltration (Fig. 16.), and in others necrosis (Fig. 24) according to the time and extent of its occurrence depends the separation and expulsion of the sac.

To sum up.

- 1). An abnormal condition of the endometrium exists, (especially of its vascular system), and possibly of the foetal epiblast.
- 2). As a result, implantation of the ovum is less securely effected than normally.
- 3). Successive haemorrhages occurring from the abnormal capillaries during the process of sinus formation detach villi in greater or lesser numbers, and sever the connection between the foetus and the extensions of foetal epiblast into the deep decidua.
- 4). The ovum if young, dies, and is partially or completely absorbed: if more mature, it continues its development, but imperfectly.
- 5). Haemorrhages into the intervillous space and a secondary thrombosis of the sinuses in the compact layer lead to haematoma formation or lesser projections.
- 6). Should the haemorrhages occur before the formation/

tion of a definite placenta a blood or flesh mole results: if after, there is a fleshy mole as a placenta.

Before concluding it will be well to attempt an answer to the most obvious objections to such a theory.

These might be stated as follows:

- 1) Granted that there is an abnormal endometrium in these cases, why should it cause a bloodmole in one woman, and an ordinary abortion sac in another, or the two in succession in the same woman?
- 2) Would one not expect every case of bloodmole abortion to be associated with definite symptoms of endometritis?

The answer to 1. may be indicated as follows:--

Each writer in turn on the subject of blood moles appears to have set off with the assumption that a bloodmole is entirely different from an ordinary abortion sac, and therefore of necessity demands a different theory of origin.

But the study of these eight cases has served rather to/

to bring out the points of resemblance between the two, and to impress most forcibly the necessity for microscopical examination of all abortion sacs obtained, as the only way in which one can hope to arrive at any understanding of bloodmoles.

Is it not the case, that out of the numbers of abortion sacs passed, (and excluding those which are seen by no doctor), but few, in proportion to the total numbers, undergo more than a very superficial examination, and of those few again, only such as come into the hands of those who are specially interested, are subjected to a microscopical examination. In this way not only are the points of resemblance between the two overlooked, but also, no doubt, many of the less marked forms of blood mole.

This thought was suggested by the quite unexpected revelations made by the study of a slide of what was to all external appearances an ordinary well developed placenta and foetus of about  $4\frac{1}{2}$  months, which had indeed been selected on this account, in the hopes of obtaining more normal material to contrast with the strikingly abnormal bloodmole slides. It may be very roughly described as consisting, at either end of the intervillous space, of an area of greatly degenerated villi, embedded in fibrinous haemorrhages, and gradually losing their outlines. The intervening portion, though/

though free from haemorrhage, contains villi, which are far from being normal, and the decidua is torn up by haemorrhages. (The slide has been submitted for inspection.)

The suggestion that bloodmoles are simply a form of abortion sac, seems to find considerable support from the fact that there is no peculiar appearance in them such as cannot be described in any abortion sac. On the contrary, haemorrhage is the chief characteristic of both, the difference apparently being rather one of intensity, combined with more gradual development, and a later involvement of the deep decidua.

Further points in reply to the first objection, are also applicable to the second, and are as follows:

1) There are many facts regarding menstruation and abortion for which no satisfactory explanation can be given.

For instance: why does one woman abort on the smallest provocation, while another woman undergoes the most violent treatment, and yet the pregnancy suffers no interruption?

Or again: why should change of climate, work, or circumstances, cause either a temporary cessation of the menses, or one or two very profuse periods?

2) It is a fact, not only as regards the eight cases mentioned, but also as regards the numbers reported by previous/

previous writers, that the overwhelmingly large majority are of the working class and multiparae, a combination which does not lead one to expect a very normal endometriun.

- 3). The question of curetting is always considered in the case of a repeated abortion, where no adequate cause can be found for it, and even though there are no definite symptoms of endometritis: and further, when carried out, it often leads to a healthy pregnancy.

The bearing of the corpus luteum on the development of the ovum (as it has been worked out by Fraenkel) has not been overlooked, but of necessity can in these cases only be considered a theoretical factor, since no positive information is obtainable.

Many points in the formation of bloodmoles still lack a satisfactory explanation, and invite further and more searching investigation. The theory which has been offered, has been suggested on account of the fact/

fact that so far, insufficient attention has been paid to the endometrium, which is the most important factor in maintaining the nourishment of the embryo.

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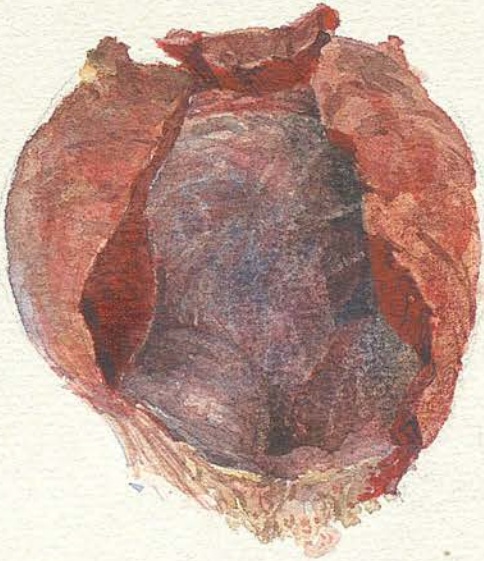
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CASE II.

DESCRIPTION OF SAC.

The sac came into my hands entire. It was about the size of a 3-months' pregnancy, and covered with thickened decidua, excepting an area the size of a penny, where the amnio-chorion showed bare. There was no differentiation of the placental area. It was opened with great care, and an unusually large amount of sparkling liquor amnii escaped there was no sign of a foetus anywhere.

The sac now measures 6 c.m. by 7 c.m. The interior has a blue red appearance and shows as it were solid projections of the wall into the cavity, covered by amnio-chorion, and varying from the size of a sixpence to that of a shilling: these on cross section have the appearance of clotted blood. *And a slight villous projection at the lower end.*



CASE VIII.

DESCRIPTION OF SAC.

It was about the size of a 3½ months' pregnancy in length, but rather narrower in breadth - an ordinary amount of liquor amnii escaped on opening it, and there was no appearance of a foetus.

The sac now measures 11 c.m. in length and 5 c.m. in breadth.

It shows as its outermost covering, soft, thickened decidua vera (this has been almost entirely destroyed at one side during preparation of slides.)

At the uppermost end there is an appearance of placental differentiation in the form of a raised area of vesicular villi crowded together (much less marked now). The lower end of the sac is simply a rounded thickened end of decidua, almost forming a cast of the cervix. Within the covering of decidua vera is a second sac, united to the outer one at the placental area, and in the lower part, but separate in the intervening portion. This shows outwardly reflexa, and inwardly amnio-chorion, and between these two but not growing through the reflexa, thickly massed vesicularly degenerated villi, but of much smaller size than those at the placental area.



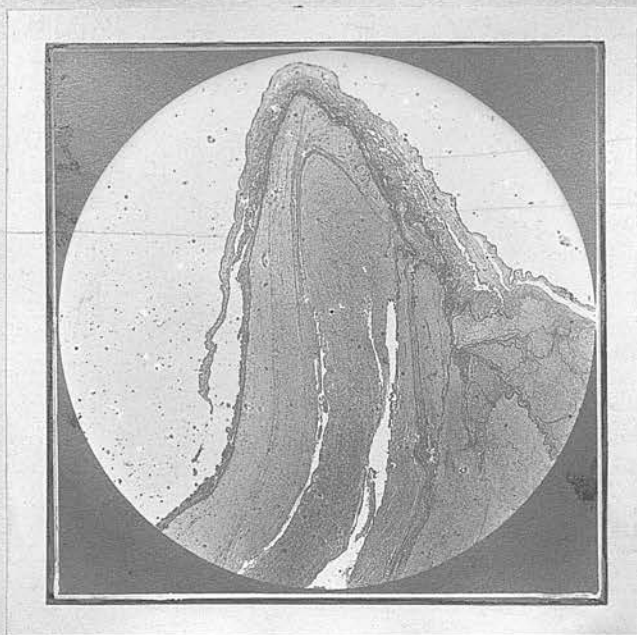


Fig. 5 (x 15.)

a) Haematoma. b) stripping up of amnio-chorion. c) fibrin layer.

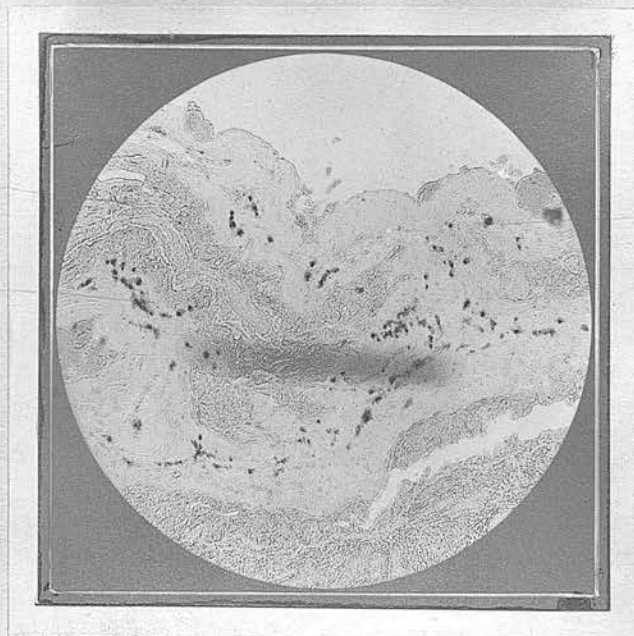


Fig. 6 (x 50)

a) pigment cells and granules in the hyaline degenerated amnio-chorion and villi. b) haemorrhage.

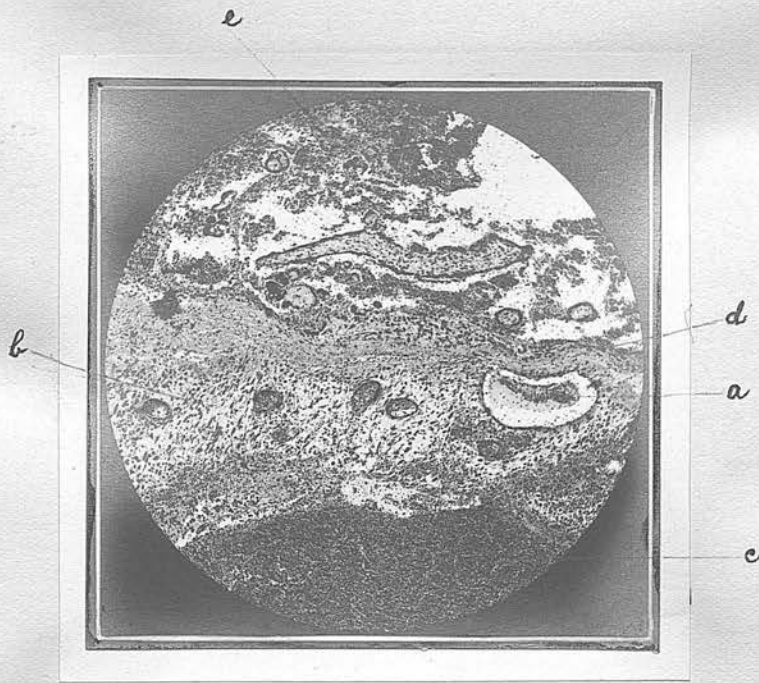


Fig. 7 (x 40)

a) Thrombus in vessel. b) decidual cells lying in homogeneous exudation. c) haemorrhage. d) fibrin layer. e) ~~haemorrhage~~ in intervillous space. f) villus.



Fig. 8 (x 40)

a) Vessels discharging their contents into the decidua. b) thrombi in 2. c) haemorrhage.



Fig. 9 (x 20)

a) Villi showing myxomatous degeneration, and b) fibrinous degeneration  
 c) Clumps of fibrin with degenerated decidua cells. d) ~~haemorrhage~~ *thrombus*  
 in intervillous space. e) amnio-chorion.

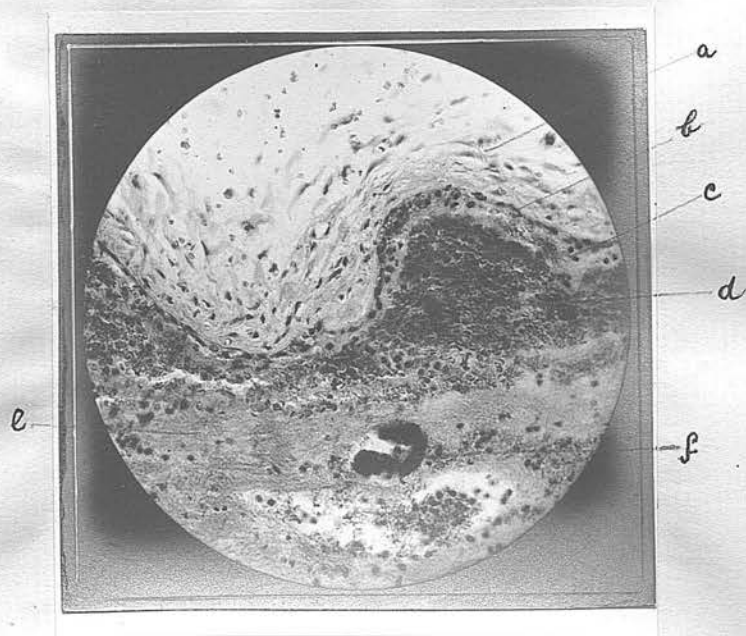


Fig. 10 (x 160)

a) Chorion. b) indefinite appearance of Langhans' layer and Syncytium.  
 c) thrombosing edge of a haemorrhage. d) haemorrhage. e) fibrin.  
 f) giant cell.

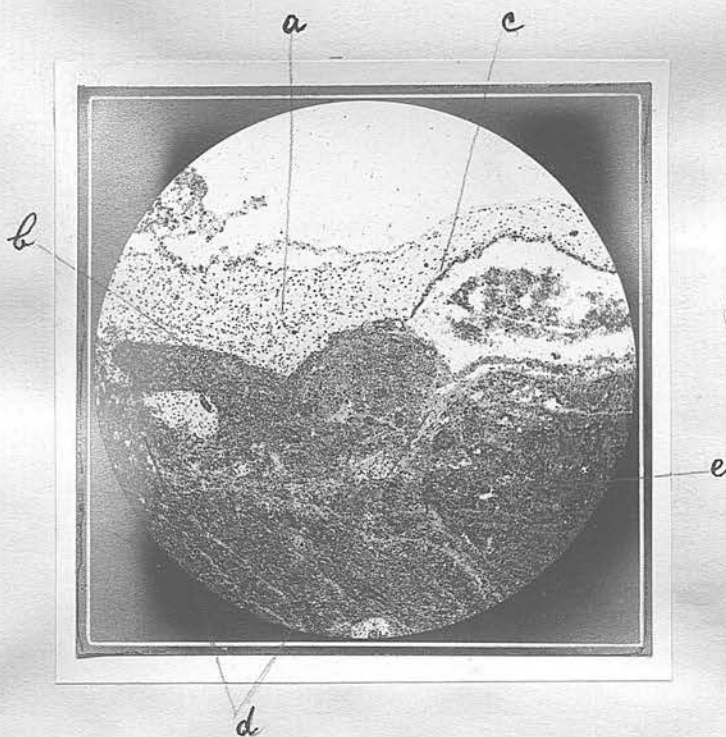


Fig. 12 (x 30)

a) Amnio-chorion showing leucocyte infiltration. b) line of leucocytes replacing chorion epithelium. c) amnio-chorion lifted up by haemorrhage  
 d) septa enclosing localised haemorrhage. e) haemorrhage in inter-villous space.

*Thrombus*

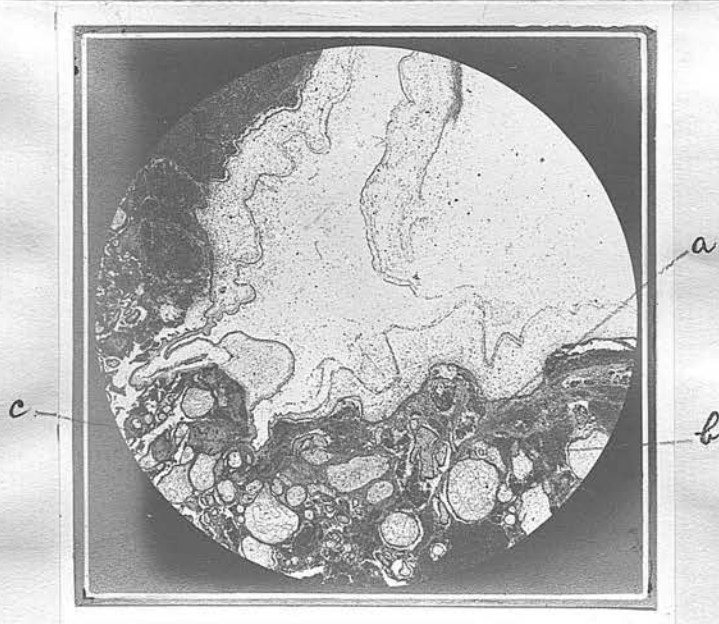


Fig. 11 (x 20)

a) tacking of chorion to fibrinous band. b) haemorrhage. c) condensation and tacking of the whole amnio-chorion.

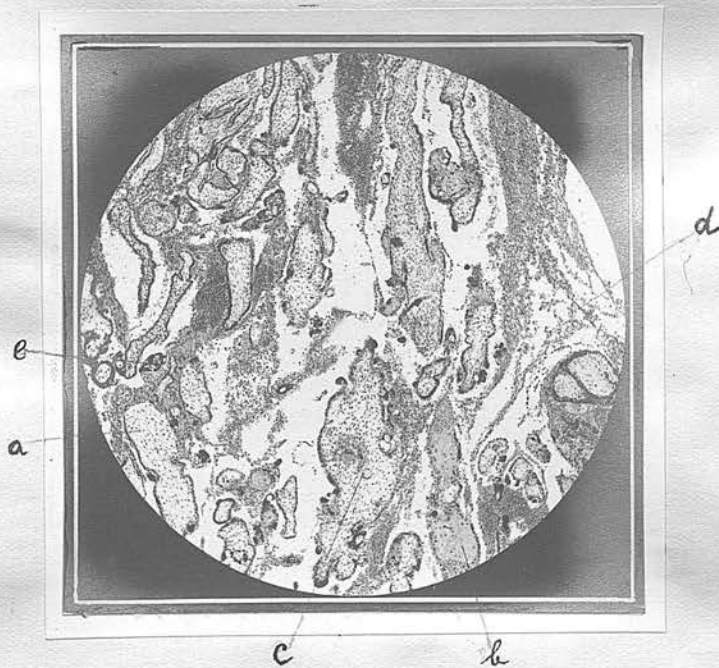


Fig. 13 (x 30)

a) Oedematous villus. b) fibrinous villus. c) vessel. d) fibrin strands  
e) giant cell.

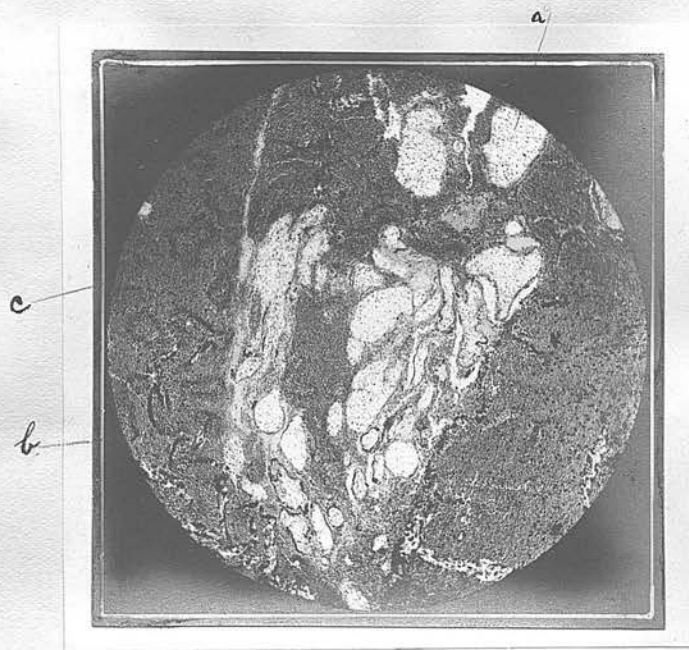


Fig. 14 (x 30)

a) degenerated villus. b) fibrin. c) haemorrhage.



Fig. 15 (x 40)

a) Syncytium attached to chorion. b) stripping off. c) lying in the thrombosed edge of a haemorrhage. d) haemorrhage. e) fibrin. f) villus.

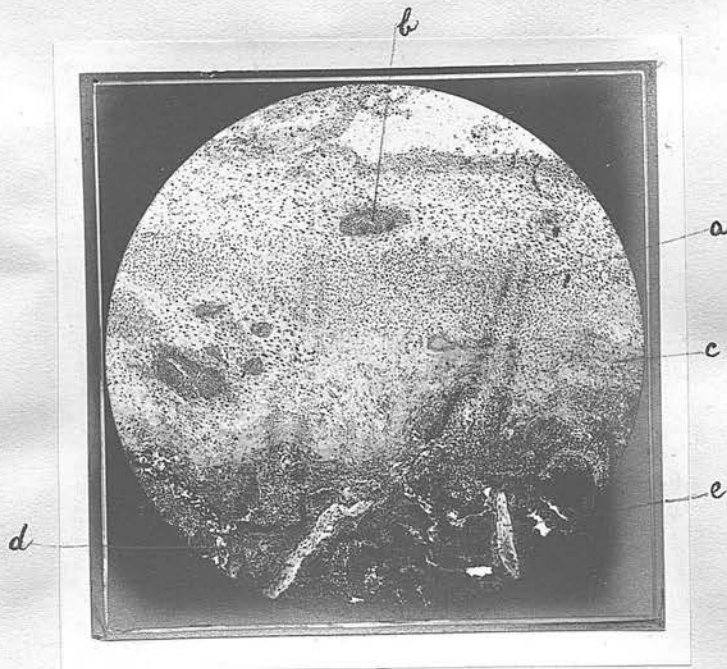


Fig. 16 (x 40)

a) leucocyte infiltration into decidua. b) vessel. c) fibrin layer of Nitabuch. d) ~~haemorrhages~~ *thrombus* in intervillous space. e) villus.

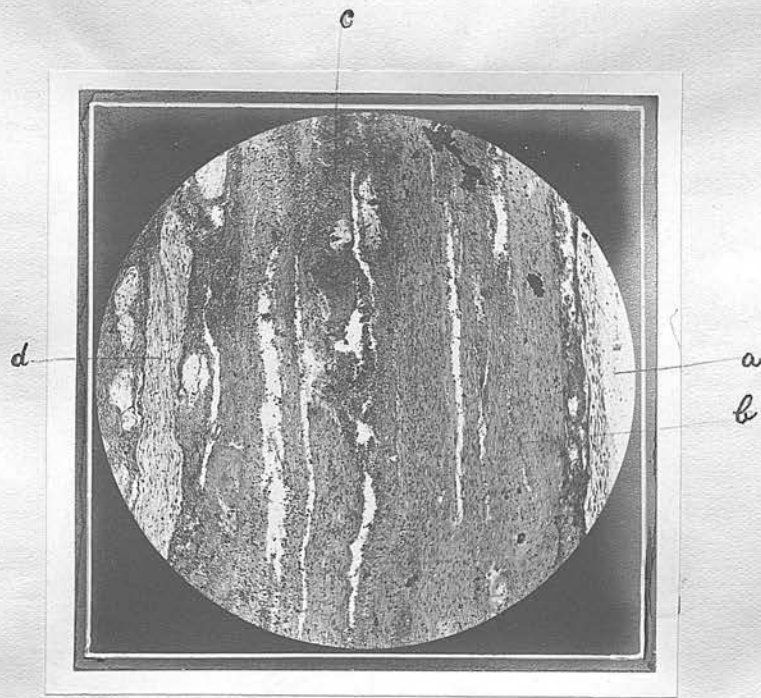


Fig. 17 (x 40)

a) amnio-chorion. b) fibrin layer. c) fresher haemorrhage. d) oedematous villus.

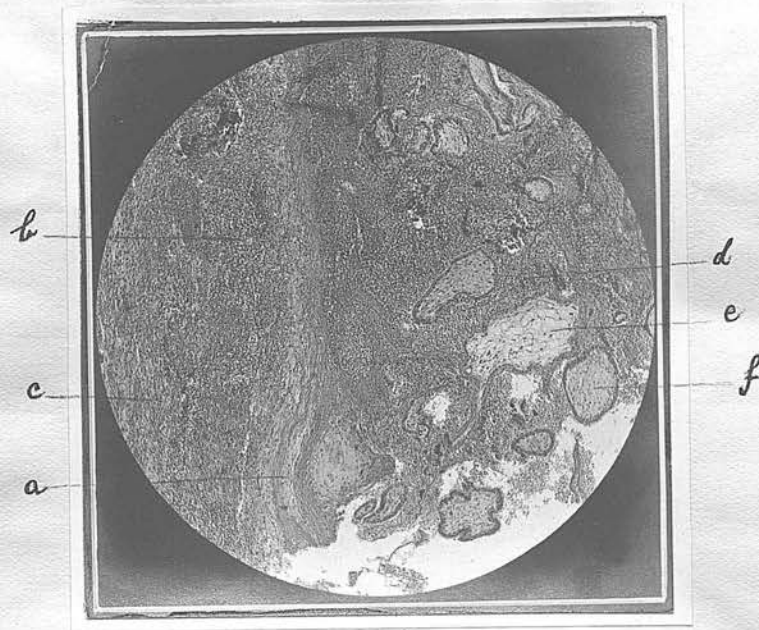


Fig. 18 (x 60)

a) Nitabuch's fibrin layer. b) haemorrhage. c) decidual tissue. d) haemorrhage in intervillous space. e) myxomatous villus. f) fibrous villus.

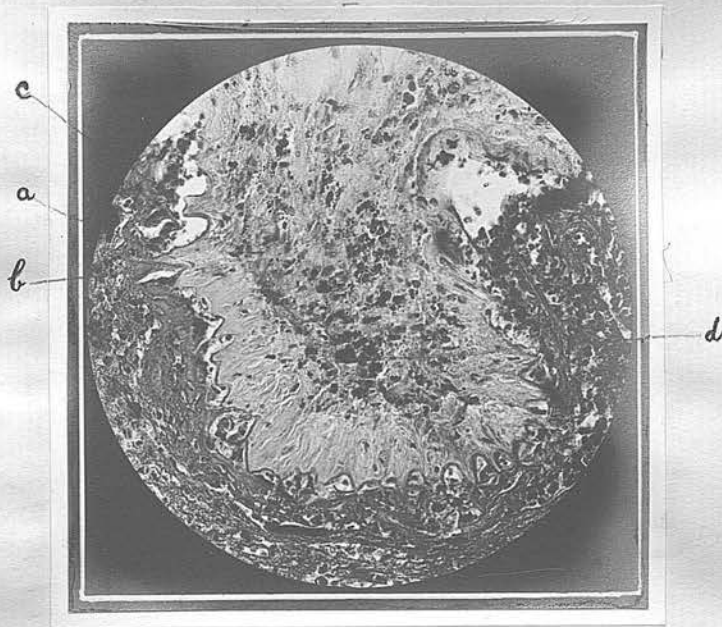


Fig. 19 (x 200)

a) Tacking of chorion. b) fibrin. c) leucocytes in its meshes. d) pigment cells.

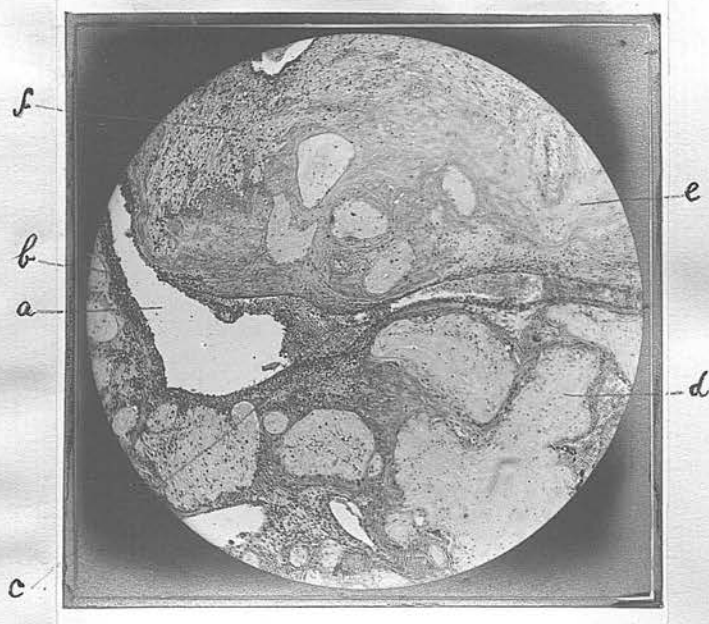


Fig. 20. (x 45)

a) large sinus. b) proliferated endothelium (?) and leucocytes lining it. c) red blood corpuscles. d) hyaline villus. e) fibrin. f) decidua tissue.

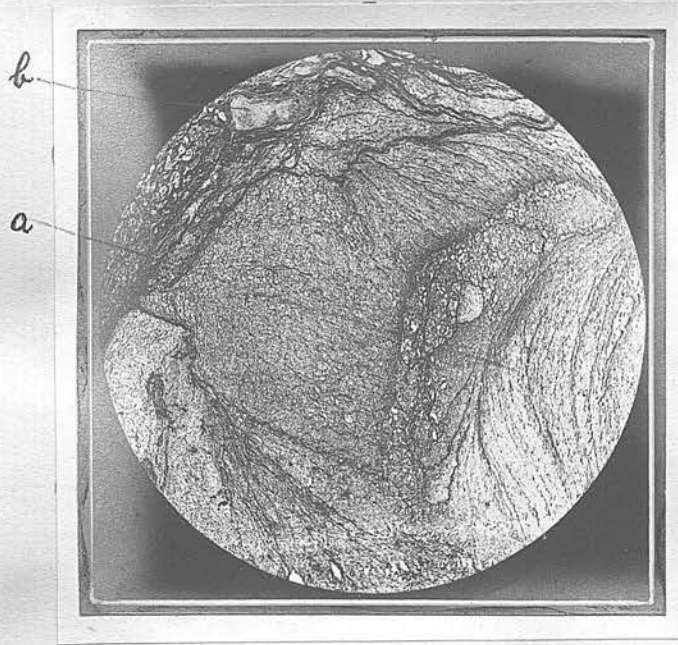


Fig. 21 (x 20)

a) grades of fibrin formation. b) degenerated villus.

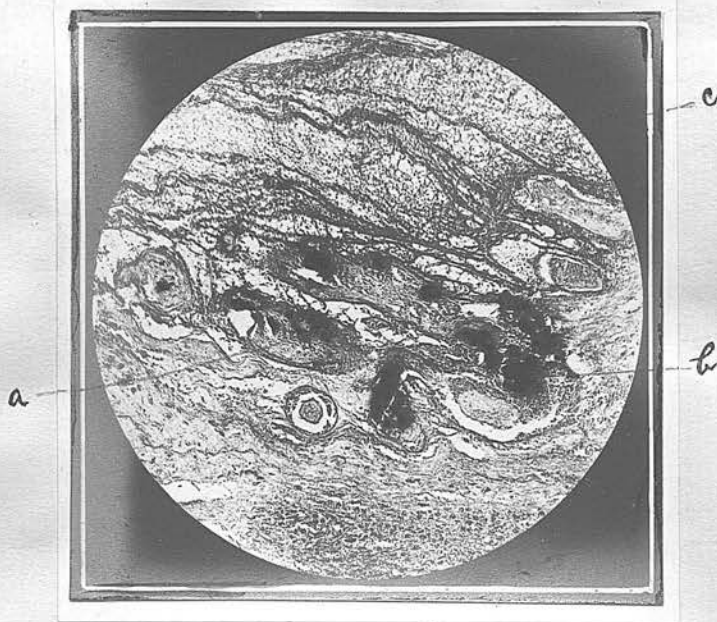


Fig. 22. (x 60)

a) calcium crystals in a villus. b) the same lying in the fibrin meshes. c) fibrinous haemorrhages.

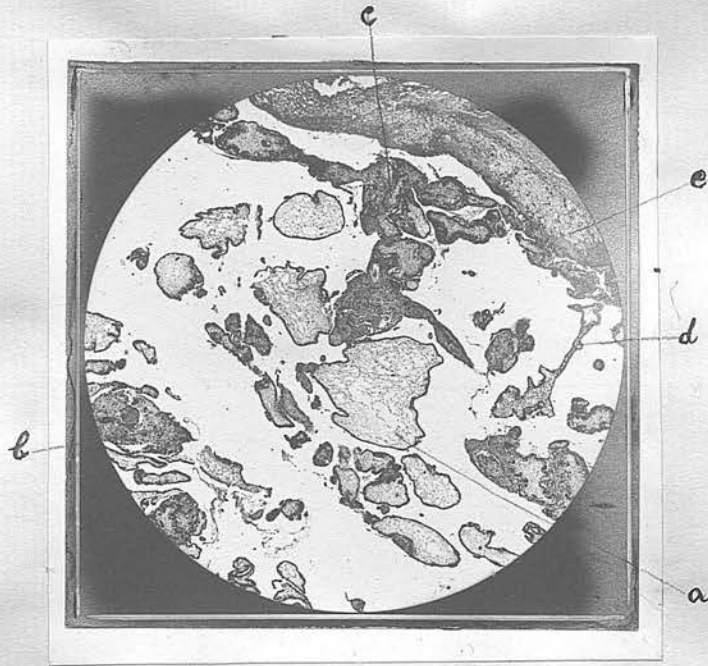


Fig. 23 (x 20)

a) myxomatous villus. b) fibrinous villus. c) plasmodial mass composed of villi with much proliferation of Langhan's layer and fibrin. d) shrunken villus. e) decidua.

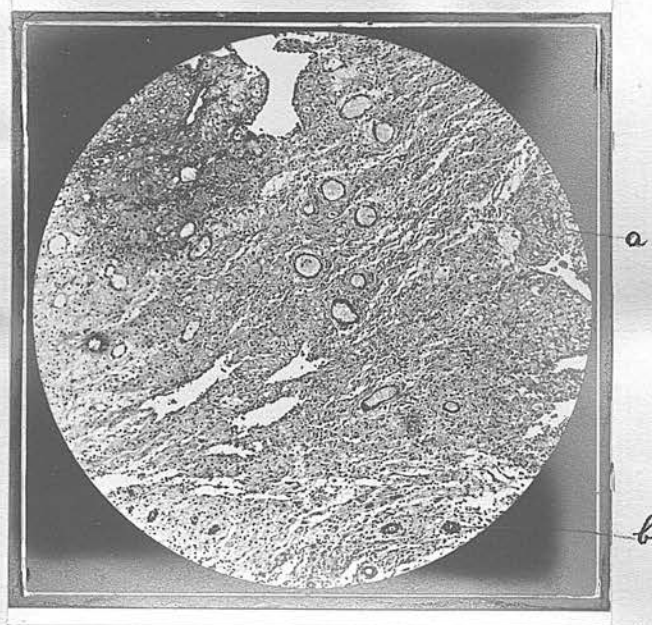


Fig. 26. (x 45)

a) Thinwalled vessel. b) thickwalled vessel.

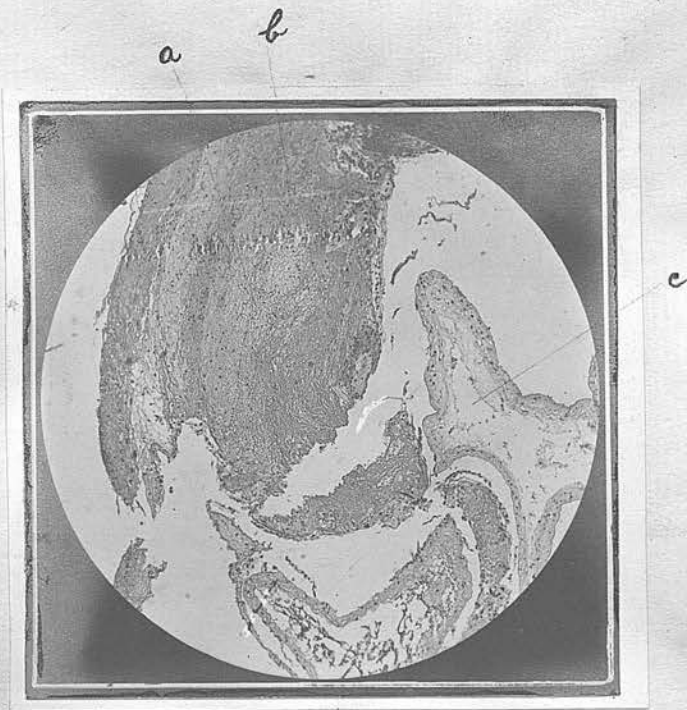


Fig. 24. (x 30)

a) haemorrhage into decidua. b) necrosis of decidua. c) myxomatous villus.



Fig. 25. (x 160)

a) amnio-chorion. b) proliferated Langhan's layer. c) vacuolation of the same.

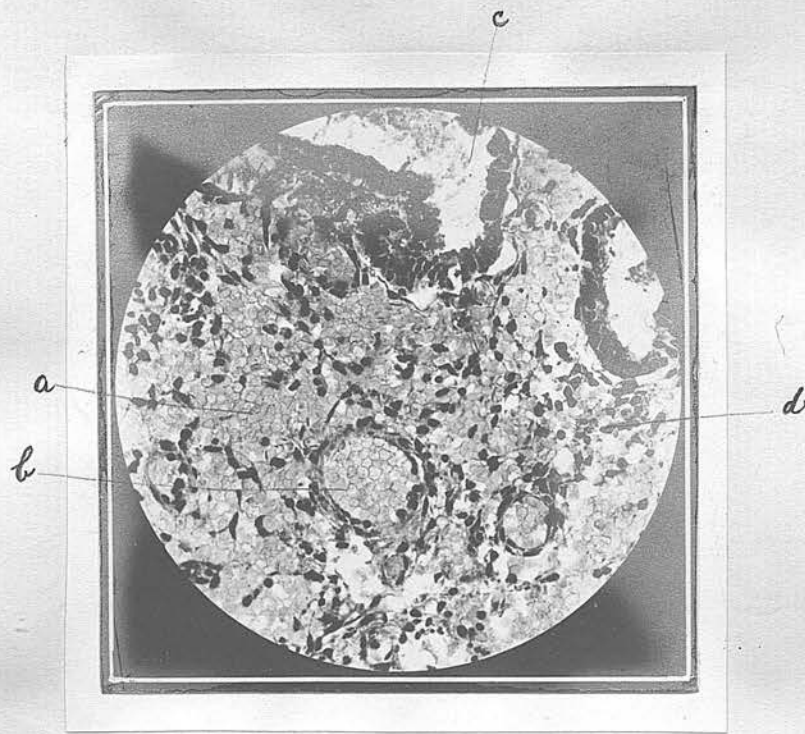


Fig. 27.(a) (x 200)

a) haemorrhage in interglandular tissue. b) dilated thinwalled vessel.  
 c) dilated gland. d) nuclei of stroma cells.

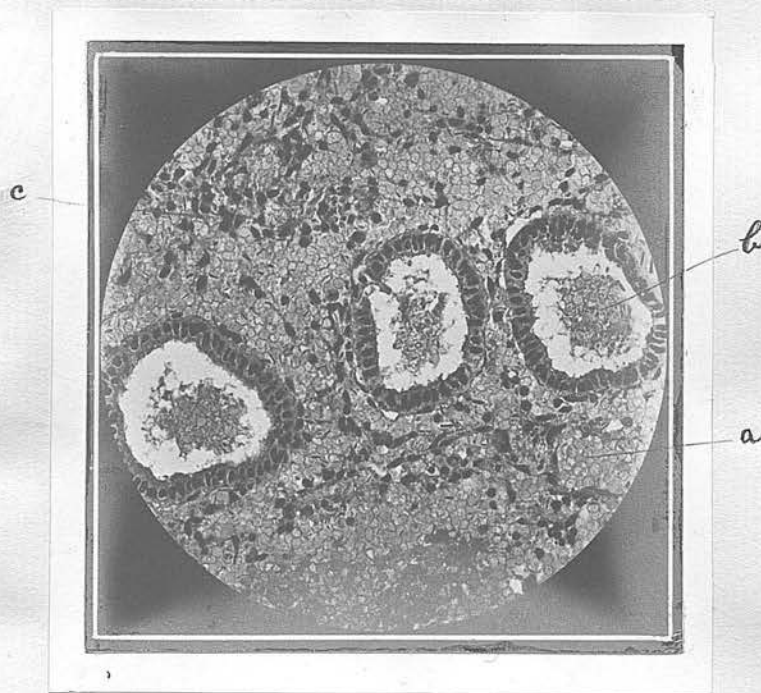


Fig. 27.(b) (x 200)

a) haemorrhage in interglandular tissue. b) dilated gland filled with  
 red blood corpuscles. c) nuclei of stroma cells.

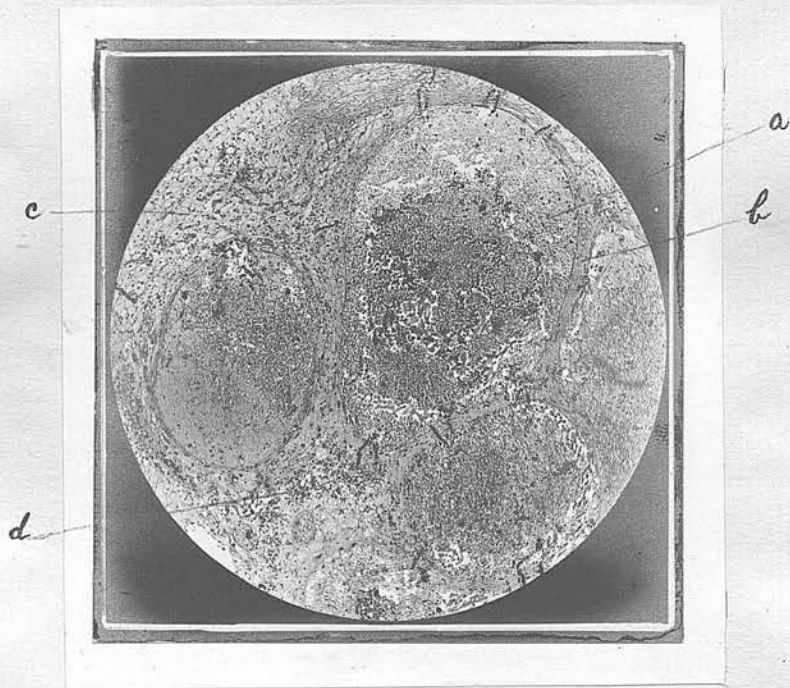


Fig. 28. (x 45)

a) large sinus. b) fibrinous band. c) decidual tissue. d) leucocytes