Placental Infarcts

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323, SHANKILL ROAD,
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28th Jan 1904.

Dear Sir,

My father obtained from You about 3 months ago the loan of my thesis upon Placental Infarcts. I sent it to the Editor of the Journal of Pathology & Bacteriology with the view to publication, and I regret to say it has not yet been returned.

I write to ask whether you will allow me to leave the essay in the hands of the publishers, promising to return it to you immediately I receive it:

Obliged,

Yours respectfully

Paul Kirkhope

With the Compliments of the

Medical Officer of Health.

David C. Kirkhope

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See note opp. p. 13

Summarise pp 1–13

The pathology of the Placenta has received such inadequate consid-eration, and, Considering the importance of the subject, so little has been written upon it, that further study may very profitably be devoted to it.

~~Thomas Watts~~ Eden (1896-7) describing the changes that occur in the placenta as it approaches maturity writes; "The importance of a right interpretation of the changes in the ripe placenta cannot be overestimated. It is impossible to make any progress with the study of its diseases until the normal structure of the organ in all its stages has been clearly defined. When this is accomplished it may be possible to enter the entirely unexplored field of placental pathology and throw light upon some of its dark corners, such as the causes of abortion and of

intra-uterine death, and the transmission and inheritance of disease".

White Infarction of the Placenta Thus Late Professor Coats (2)(1889) writing upon the subject of "The White Infarction" and its relation to "Periarteritis Nodosa" says; "The whole subject is in need of further elucidation."

The very name by which these pathological products are called indicates how little the matter has been considered, since, except in their naked-eye appearance as they bear no resemblance to infarcts.

Ackermann, (1) 1884 however, considering that these placental nodes are caused by an obliteration of the blood-vessels to the villi involved which, becoming anaemic, undergo Coagulation-necrosis; compares the process of placental infarct-formation with that Series of changes in the kidney which leads to wedge-shaped

infarctions.

~~The following method was, as far as possible, followed in this investigation.~~ The history of the parents was ascertained, and the occurrence of previous pregnancies or abortions noted; and any circumstances likely to have an adverse influence on the product of conception were enquired into, such as alcoholism, syphilis, renal or heart affection, anaemia, tuberculosis etc.. Portions were removed from the placenta to include normal and pathological tissue. Altogether about forty placentas in different stages of development were examined. After hardening in formalin, decalcifying and embedding in paraffin, sections for microscopic observation were cut and stained with haematoxylin and eosin. Where it seemed advisable to amplify the description of the

changes observed, photomicrographs were prepared and are submitted herewith.

The purpose of this essay is to ascertain, if possible, what circumstances govern the formation of infarcts (so called), which are of two distinct varieties, fibrinous and non-fibrinous.

History In the latter half of the seventeenth century Mauriceau (1712) described these nodes; calling them "schirrus of the placenta". During the eighteenth century there appears to have been great difference of opinion regarding the nature of "infarcts", which were designated; Atrophy, Inflammation; Hepatization; Apoplexy; Fatty degeneration and Hyaline degeneration. Most observers regarded them as of inflammatory origin, probably because of the regular occurrence in them of fibrin. This seems to have been the opinion of

1836

Simpson (5) ~~in 1836~~. Ten years later Gierse (6) referred their formation to haemorrhages which had to some extent undergone metamorphosis, and in their origin quite analogous to organized thrombi, or to the connective tissue metamorphosis of the Corpus luteum. To a certain extent Meckel (7) agreed with this view, but regarded "chronic inflammation" as influencing the change. Barnes (8) considered that fatty degeneration accounted for the appearances of the structures he described. Scanzoni's contributions (9) seem to indicate that he is in agreement with Gierse and Meckel, ~~and~~ explains that some of the inforcis are the result of haemorrhages for "he had frequently succeeded in demonstrating within the nodes dead blood corpuscles and accumulations of blood-pigment." Others,

however, he regarded as the result of inflammatory exudate, remarking however at the same time that "it is in many cases difficult or even impossible to distinguish between them."

Spaeth and Weal (1855) regard them as being due, not to inflammation, properly so called; nor do they consider that haemorrhage in every case determines their formation; but they describe them as resulting from "a simple deposit of superfluous fibrin which can no longer circulate in the capillary system."

Robin (1854) first separated them from the inflammations and classified them according to the changes observed in them into fibrous, fibrinous and scirrhus; all of which he considered to be due to a fibrous degeneration of the villi making them impermeable

to the blood, and as really identical with the normal villi which do not take part in the formation of the placenta.

Maier (12?) finds the cause of the whole process in the decidua serotina and especially in the processes which arise from this layer and are found between the cotyledons and the individual villi of the placenta. He describes three stages in the formation of the infarct, firstly, a proliferation of decidual cells which, secondly, give rise to the formation of granulation tissue. This granulation tissue contracts and hardens, and, thirdly, in the process of contraction ensnares the villi. These villi undergo fatty degeneration on account of the surrounding pressure on their substance but especially on their blood-vessels.

Apparently he is describing the non-fibrinous infarct and mistakes the fibrin on the surface of the decidual processes for granulation tissue.

Langhans (1877), however, returns to the view that infarcts are composed of fibrin. He regards their origin as being in close relation to a layer of fibrin which is situated on the placental side of the chorion, previously described by Winkler and Kölliker, and supposes this layer to be a continuous layer in the ripe placenta varying in thickness from a few 1/100 ths m.m. to 1 m.m; but at certain places rising to a height of about 1 c.ms. While infarctions, he believes to be due to this heaping up of fibrin.

Ackermann, on the other hand, in his earlier paper (3) (1884) declares that the nodes or infarcts are not by any means constantly

united to this chorionic layer, but indeed, in most cases are separated from it by free villi. He describes the infarct as consisting mainly of three constituents, fibrin, villi and connective tissue. The fibrin occurs within the nodules either in connected masses not broken up by placental villi, or it surrounds the villi in layers of various thickness so that they appear embedded in it. The infarcts consist, therefor, in many places almost entirely of fibrin, at others of villi held firmly together by the fibrin. The third constituent, connective tissue, was described as occurring round the villous blood-vessels leading to the obliteration of their lumen, so that they become for the most part impermeable to injection fluid. Nevertheless it is possible in some fresh infarcts

to inject the vessels of the engaged villi. The stratum underlying the syncytium is said to suffer first in consequence of the periarteritis, undergoing coagulation-necrosis and conversion into fibrin. The syncytium then suffers in a similar way and fibrin from the intervillous space supplements that developed from the syncytial covering of the villus and the subjacent layer, representing the remains of Langhans' layer. Ackermann subsequently (1891) found occasion to change his opinion regarding the causation of infarcts, or, at any rate, to modify it, and described the changes in the blood vessels as partly due to "endarteritis obliterans" and not entirely to "periarteritis fibrosa multiplex". Further he considered that he had overestimated the

importance of the changes in the blood in the intervillous space and believes that most of the fibrin is derived from necrosis of cells. Later observers uphold Ackermann's opinion including Orth (1893), Eden, 1896-7, Kermauner and Williams (1900 & 1903), and Norris & Dickinson (1902) are also in agreement.

Steffeck (1890) does not agree with Ackermann, but found the immediate cause of infarction in the proliferation of decidual cells growing up around the villi, and believed that the vascular changes were of secondary importance since they could not regularly be demonstrated, and Jacobsohn (1890) arrived independently at a precisely similar opinion, and these observers had many followers.

Others again thought the primary cause was the change which the maternal blood-vessels underwent, and with this opinion I am disposed to a certain extent to agree. Amongst those are included Rossier (1888), Rohr (1889) and Ribemont-Dessaignes (1894).

I have not attempted to give a complete list of observers and their opinions; but I think I have succeeded in ~~expressing~~ summarizing the views which met with greatest acceptance.

The exact nature of the material which lies between the villi in an infarct has been much discussed, and opinion is divided as to whether it is fibrin or hyaline. Since it corresponds very closely in staining properties with fibrin, and as it has, for the most part, a finely

Re literature - few references
+ summaries

reticular structure, I shall designate it "canalized fibrin" in agreement with most recent observers.

Normal Placenta.

About mid-term, changes in the blood-vessels which empty themselves into the intervillous space are found to occur, whereby the vessels, principally the arteries become more and more occluded.

~~Fortunately~~, I have not had the opportunity of examining the condition of the blood vessels of the uterus after delivery, but, judging from the scanty amount of haemorrhage which occurs normally from so large a surface, and from the thickness of the vessel-walls in the part of the decidua shed; and also from the pathological reports

of those who have had opportunity of examining the maternal vessels, one may fairly assume that as term approaches the calibre of the maternal arteries is very much reduced; and the blood current through the intervillous space becomes less rapid, favouring the conditions which cause fibrin to be deposited.

The same changes in the blood vessels are found in the foetal portion of the placenta. The villous stems have their main blood-vessels much reduced in calibre by a process of endarteritis obliterans. From the time that changes begin to occur in the blood-vessels, changes also occur in the intervillous space. The maternal surface of the inter-villous space becomes lined with a deposit of fibrin. This layer gradually

spreads covering the dissepiments which lie between the cotyledons. The foetal surface of the intervillous space also partakes in the same change, and the villous stems springing from the foetal surface come to have a coating of fibrin which may reach to a varying length along the stems and their branches.

The fibrin also insinuates itself between the cells of the decidua serotina, separating them into clumps or layers surrounded by fibrin. These separated cells undergo retrograde changes, their protoplasm becoming granular and staining darker than the, as yet, unaffected cells; the nuclei also change in their staining properties; and the fibrin, once deposited on them, tends to accumulate when exposed to the blood-stream, especially when the

current is sluggish. Williams believes that these altered cells are not really decidual cells, but are of foetal origin and belong to the ectoderm. I think, however, Eden has quite clearly demonstrated that there is a transition in appearance from the most markedly affected cells to the normal decidual cells, and that it is only those which have been for the longest time separated by the fibrin from the rest of the layer that have suffered in greatest degree.

Examining sections from the margin of the mature placenta, clumps, similar in appearance to these altered cells, may be found distributed quite across the intervillous space from the maternal to the foetal surface. (Photos I & II) They occupy the intervals between the

villi, to which they are attached by their fibrinous covering. These cells are large and have round or oval nuclei of considerable size, and are easily identified as being of maternal origin, by their resemblance to decidual cells. Photo I is from the maternal, and Photo II from the foetal surface of the placenta at the margin of the intervillous space. The decidual cells can be traced for a certain distance from the maternal towards the foetal surface, and in the interval between the point where the continuous layer of decidual cells ceases and the chorionic surface, groups of these large cells can be distinguished plentifully distributed. In other words, the characteristic cells of the decidua serotina are not limited to the

maternal portion of the placenta, but are distributed throughout its entire thickness, and are surrounded by a thick layer of fibrin.

It may be that, at the time when the ovum becomes implanted in the uterine wall, attachments are formed between the decidua serotina and the chorionic surface by means of columns of decidual cells, and the clumps seen on section may represent these columns.

Examining sections from a centrally situated portion of a mature placenta, large processes of decidual cells are found arising as a protrusion from the maternal surface (photo III), and penetrating deeply into the intervillous space. These processes consist of centrally situated large cells surrounded

by a layer of fibrin. In the inter-villous space they divide up into branches (photo IV), and with each division becoming smaller and smaller, they are, for the most part, inserted into the chorionic surface by fine filamentous radicles. (photos V and VI.)

The fibrin covering these processes is continued over the chorionic membrane, passing along the villous stems for some distance, following the course of their branches, and in some instances reaching to the decidual surface where the "anchoring" villi have their attachment.

The intervillous space, then, is occupied by a system of branching processes, some villous and some decidual.

It has been stated above

that fibrin extends along the villous stems and branches by continuity of surface. In making this statement I am not supported by the accepted opinion of Ackermann. Yet I have been led to form this opinion from examination of longitudinal sections of villi. Photo VII shows the extension of fibrin along a villous stem. It shows that the fibrin extends, not along the sub-epithelial layer, but along the plasmodium of the syncytium converting it into fibrin. It also shows that the blood-vessels of the villus do not suffer to any considerable degree from obliterating endarteritis since the central vessel and the smaller one lying underneath the syncytium are widely open and contain blood, apparently in normal

condition. When, however, the syncytium is destroyed the further changes observed by Ackermann and others do occur. Indeed, the fibrin, having reached the underlying tissue, may spread so rapidly through its substance as to overtake the extension of fibrin along the syncytium, since the process is there retarded on account of the syncytium being nourished from the maternal blood; and on cross section of the villus beyond the part where the syncytium is involved the appearance figured by Williams is seen. Photo VIII is copied from Williams' paper (16) p. 794. The intervillous space between two villi is obliterated and fibrin extends from the obliterated space along the peribsyncytial stratum. Here the vessels appear to be almost

completely closed, and yet the only peripheral part of the villus affected is where two villi have become cemented together by fibrin. The syncytium still exposed to the blood stream in the intervillous space remains intact. It seems reasonable to infer that the syncytium suffers when there is some hindrance to its deriving nourishment from the maternal blood, since it is the maternal and not the foetal blood that nourishes the foetal appendages. As proving further that the changes in the villous blood-vessels are not responsible for the formation of fibrin in their peripheral parts; it will be shown, when discussing the formation of non-fibrinous infarcts, that although changes do occur in the blood-vessels, and the

current through them is arrested, neither the subepithelial layer nor the syncytium undergo fibrinous change.

Fibrinous Infarcts.

In nearly every mature placenta, on careful examination, white masses are to be seen, varying in size from a millet seed to an almond or even larger. They are variously situated, sometimes on the maternal surface, more frequently on the foetal, and in the case of the larger masses, reaching from the one surface to the other. They may be few or many; so few as to require some search to find, or so many as to involve the greater part of the placental tissue. In the latter case the child is usually dead-born,

having perished in all likelihood from a deficiency of nutriment on account of the impervious condition of the greater portion of the placenta. Where the placenta is invaded in lesser degree, the child, if born alive is small shrivelled and puny.

The fibrinous infarcts are composed of villi, which have become quite functionless. The blood-channels of the villi, for the most part, are occluded, or contain a few corpuscles which have become necrotic. The stroma is represented by a few ill-shapen nuclei supported by strands of what was the cell protoplasm. The syncytium has disappeared, and the intervillous space is occupied with fibrin which cements the villi together that compose the groundwork

or basis of the infarct. The peripheral part of the infarct has villi adherent to it which are in process of destruc-tion, the syncytium on the attached border disappearing or being altogether unrecognisable; the syncytium exposed to the open space being intact.

The central part of the infarct has mostly become so altered by necrosis as to present an almost homogeneous appearance; and, therefore, this part is unfavourable for study as to causation. The peripheral part will receive further consideration.

It was noted, in examining the margin of the placenta, that strands of decidual cells surrounded by fibrin reached across the inter-villous space from the maternal to the foetal surface, and that they

were united by their fibrinous covering to the adjacent villi. The presence of these cells in the vicinity of the chorionic surface of the placenta, so constantly embedded in fibrin, and so constantly occupying the marginal area of a mass, morphologically indistinguishable from a fibrinous infarct, suggested the study, of which this paper is the outcome, to discover, if possible, whether these processes of decidual cells have any causal relationship to the formation of infarcts.

Changes occurring in the foetus and its appendages, depend upon altering maternal conditions. Since all the nourishment to the foetus and placenta is derived from the maternal blood passing through channels in the decidua

perotina; obstruction to the free blood supply will, in the first place, exercise its immediate effect upon the decidua perotina. It is generally agreed that changes leading to narrowing of the lumen do occur in the maternal blood-vessels of the uterus, commencing shortly after mid-term and steadily progressing to term. Immediately following the narrowing of the calibre of the vessels fibrin is deposited on the lining of the intervillous space, and it penetrates between the superficial decidual cells cutting them off from the more deeply situated, and causing thereby the altered appearance of the cells already noted.

When it is considered that the whole placenta is divided up into cotyl-

-edons of comparatively small size; that between the cotyledons the decidual layer dips down, reaching almost to the chorionic surface, and that this layer is thickly coated in the ripe placenta throughout its whole extent with canalized fibrin; that the chorionic membrane has also an extension of this fibrin over its surface; and, further, that these limited areas contain decidual processes and villous stems, each with their branches covered to a greater or less extent with fibrin, forming a network of processes giving rise to eddies in the blood-stream already slowed by narrowing of blood-channels, thereby tending to further deposit of fibrin; it is not surprising that infarcts should

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be found; but rather it is astonishing that so extensive an area of tissue forming a suitable foundation for fibrinous deposit, should not lead to almost complete obliteration of the intervillous space. The fibrin once deposited continues to be added to. As the intervillous space between the separate villi becomes invaded, villi are added to the mass; not only as the result of the space being encroached upon by the invading fibrin attached to a particular stem, but by a progressive extension along the branches of the stem and their many offshoots. All stages of the process of infarct formation are to be found in every mature placenta. Villous processes are to be seen entirely surrounded by fibrin side by side

with villous processes entirely free from fibrin; or a large mass of fibrin enclosing villi that have become functionless, having at its margin newly affected villi, the syncytium of which, at the line of contact, has become destroyed, whereas the syncytium exposed to the still open intervillous space shows no appearance of destruction. Invariably also, in such a mass, the remains of large cells are to be distinguished. Where they occupy the periphery they are found lying together in clumps, having their characteristic appearance; where they lie more deeply all that may remain of them are the large nuclei, mis-shapen and staining badly, with strands of necrotic tissue uniting them to the remains of their cell

walls. More deeply still, a cluster of large nuclei is all that is left to indicate the position of a decidual process.

The fibrin as it encroaches upon villous stems and branches, exercises its destructive effect, firstly, on the syncytial covering. The epithelium is cut off from its source of nourishment and undergoes a process of coagulation necrosis. This is followed by a fibrinous change affecting, as demonstrated above, in the first instance, the plasmodium of the syncytium; and later the sub-syncytial layer is engaged. The stroma appears to resist destruction for a much longer time than the epithelium; probably because it derives sufficient nourishment from the foetal blood. The blood-vessels occupying the

central positions of the recently affected villi are found to contain blood having a normal appearance, the channels themselves being patent until the mass forming the infarct has become very large. Then the central villi are deprived of their blood supply and the vessels are found to contain merely debris. The component parts of the villi thus affected fail to stain with the usual stains, and present an almost homogeneous appearance, only the outlines of the villi being recognisable.

This destruction is progressive from the centre towards the ever-widening periphery. In very large infarcts cretaceous deposits are found scattered throughout, an instance of the well-known pathological law that lime

salts tend to be deposited in dead or dying material.

That the decidual processes take a very active part in causing the formation of infarcts is clearly to be seen by careful examination of the region immediately surrounding an infarct. Here it is not unusual to find the intervillous space between two or more villi occupied by a decidual process (Photo IX). The adjacent surfaces of the villi have lost their syncytial covering, and by extension of the spreading fibrin ultimately these villi will be included in the infarct.
By examining the periphery of the infarct in nearest relationship to the process between the villi it is sometimes possible to distinguish the presence of large cells there,

rendering it probable that the process is merely an offshoot from one of those which have already taken part in the production of the infarct already formed.

The theory advanced; besides being capable of abundant demonstration, has the additional merit of explaining the difficulties which other observers had in accounting for the appearances they observed. Langhans was right so far as he went in explaining the origin of infarct formation but he failed to follow the process beyond its commencement. It explains how the process, having commenced at the chorionic surface, may not, and as Ackermann pointed out, does not as a rule reach its full

development there. Ackermann was unable to explain how the villi which showed very little change in their blood-vessels were at times affected by coagulation-necrotic changes, when villi whose vessels were almost obliterated escaped. I believe the villous vessels have nothing to do with the fibrinous degeneration of the syncytium and underlying stratum. Steffeck only stated a part of the truth when he ascribed the cause of infarct formation to proliferation of decidual cells growing up around the villi.

Infarct formation, then, is a steadily progressive process which, once begun, may advance to such a degree as seriously to compromise the prospects of the product of

conception.

So far as my investigations have gone, it is this variety of infarct-formation that is found most frequently in Syphilis; often studding the placenta throughout its entire extent so abundantly as to render it perfectly hopeless that sufficient tissue would be left in the placenta in condition to serve as the channel of nourishment to the foetus, to carry it to full term. In connection with Syphilis as a cause of infarct-formation and abortion; it is worthy of notice that the blood vessels in this disease are liable to become seriously affected, and, with their normal tendency to become narrowed as to their lumen during the course of pregnancy, the superadded syphilitic

disposition to obstruction determines the serious results to the foetus.

Again, in examining the placentas from early abortions it is invariably found that an haemorrhage, separating the foetal from the maternal portions, has caused the throwing off of the early ovum. This might also be explained by the changes occurring in the blood vessels of a syphilitic mother. Alcoholism is another fertile cause of abortion, and in it also the vascular system is the one which suffers in greatest degree.

In Kidney disease, infarcts are found in great number in the placenta, and in this disease also the blood vessels are liable to suffer.

In all these affections the tendency is for the lumen of the blood vessels

to become narrowed. It would therefor appear that any effort made to reduce the liability to haemorrhage and infarct formation, the two most frequent immediate causes of abortion and premature birth, should be in the direction of keeping the blood Vessels, in the first half of pregnancy in the best possible condition for fulfilling their function probably by the use of drugs such as reduce tension, thus preventing undue overstrain upon them; and in the latter half, using such remedies, if there be such, as tend to preserve the fluid condition of the blood and restrict fibrin formation.

Non-fibrinous Infarcts.

The non-fibrinous infarct

has its central part composed of villi which, also, are functionless. They remain, however, quite distinct even in the most central part. Indeed the villi in the centre may be separated by a space from each other, while those nearer the periphery are in contact. Their structure has undergone considerable change, but of a different kind from that of the fibrinous variety. The blood-vessels are patent, but contain only debris. The cells of the stroma do not take up the haematoxylin stain. The outline of the syncytium is preserved, staining faintly with eosin, although the individual nuclei are not differentiated. The intervillous space is almost obliterated, so that the villi come to lie very close to one another or quite in

contact. Where they are separated by a little interval, the space is filled with the debris of blood; fibrin does not take any considerable part in the obliteration of the space. Where they are in contact, the nuclei of the syncytium become fused, and the clumps of what were the nuclei occupy the angles between two or more contiguous villi. The periphery of the non-fibrinous infarct has a different structure which will be treated of more in detail. Suffice it for the present to say, that the peripheral part has, more or less, the character of the fibrinous variety. The part which is distinctly non-fibrinous is well-defined as to its limits, whereas the peripheral part, which forms a kind of capsule to the central part is

not well-defined, having villi attached to its outer border; and an outlying area of newly invaded territory as in the fibrinous infarct.
Eden advances the ingenious theory, that non-fibrinous infarcts are caused by the obliteration of the maternal arteries, as follows.
When an artery becomes almost or quite closed, there is a deficient supply of blood to the cotyledon or part of the cotyledon supplied from that artery. The blood-stream carries the floating villi towards the still open vein. But in the affected part there is an insufficient amount of pressure, owing to the closure of the artery, to keep the villi apart. There may even be negative pressure in the area involved, and with the positive pressure in the remaining part of

the cotyledon or surrounding cotyledons, the Villi are compressed. He is careful to state that this is merely a suggestion, but supported by the fact that in the vicinity of non-fibrinous infarcts, arteries are usually found to be very much narrowed in their lumen or altogether closed.

Against this theory may be advanced the arguments: that the occlusion of blood vessels is a usual occurrence towards term; that the communic-ations with neighbouring cotyledons would allow of the pressure being equalized; that the Villi involved are definitely circumscribed in area; and that immediately outside the peripheral wall or "capsule" of the non-fibrinous infarct the intervillous space is open and the Villi contained

in it normal in appearance. One would expect that if the blood current is the cause of the infarct, there would be a gradual shading off into normal tissue, and not the abrupt change from pathological to normal usually found. Should, also, for any other reason than the one given by Eden, the circulation in a part of a Cotyledon be arrested, then the vessel supplying that part would become thrombosed, so that, what Eden suggests as the cause of the infarct may be the result of the infarct formation. Referring to Eden's paper Williams writes "We have not infrequently observed such structures, but as yet are unable to express a definite opinion as to the correctness of Eden's explanation concerning their production." (16).

The large processes stretching from the maternal to the foetal surface, and having decidual cells as the groundwork upon which fibrin is deposited, have branching offshoots uniting with branches of the same or other processes, or with the stems or branches of villi (Photo X). Both decidual processes and chorionic villi stretch across the intervillous space, and by their branches enclose areas of villi by forming a network around them. The arrangement might be thus described. The processes of decidual tissue, and villous stems with their "anchoring" villous branches, form columns of support in the intervillous space, keeping the two surfaces, maternal and foetal in their respective

relationship to one another and to the contained villi.

The fate of these areas of villi will depend greatly upon the structure of the network surrounding them. If the meshes are wide allowing of free interchange of blood in the area, the villi will continue to functionate, but if the meshes are narrow, not only will the blood stream be retarded, but the very condition is produced which favours the deposit of fibrin. Thus the meshes are further closed, and a "capsule" is formed round a certain number of villi. In this condition we have the "Red Infarct", and there is every gradation between it and the typical non-fibrinous infarct. As the process of the formation of the "capsule" proceeds, the blood is driven

out of the space by the encroachment of fibrin and by shrinking of the fibrin already deposited.
So long as the villi are capable of absorbing nutrition, they take up a certain amount of the fluid in the enclosed area, thereby further diminishing the space in which they are contained.

But as the stems, the branches of which compose the non-fibrinous infarct, must pass through the enveloping structure, they are likewise compressed, and the circulation through them arrested. The vessels of the villi, though patent, contain blood which, after the circulation ceases, undergoes necrosis. The syncytial covering, no longer bathed in nutrient fluid, undergoes coagulation-necrosis, and the nuclei

lose their individuality, and are found in clumps in the angles at the junction of two or more villi.

Support is given to this theory of the formation of non-fibrinous infarcts by the following facts. The decidual processes can be demonstrated in every placenta, and in the vicinity of a non-fibrinous infarct- I have not failed to identify large groups of decidual cells and even large decidual processes (photo X1), both in the envelope itself and in the surrounding area, those in the envelope penetrating between the engaged villi to some considerable depth.

With regard to the envelope of a non-fibrinous infarct, there are changes in it of a different-

character. The fibrin deposited on the network not only affects the area surrounding the non-fibrinous portion, but spreads along the branches in an outward direction involving the villi in the neighbourhood and obliterating the intervillous space; precisely as in the case of a fibrinous infarct.

Why the same fibrinous change does not spread inwards towards the centre of the infarct to any considerable depth is, I believe, because the formation of the non-fibrinous portion is of rapid development. The blood is, within a comparatively short time, driven out of the region, and too little remains from which fibrin could be formed to obliterate

the spaces between the villi.

In further vindication of this theory, it would appear that non-fibrinous infarction is probably due to some peripheral compression; for it can be observed in large non-fibrinous infarcts of recent origin, that the central part has its villi fairly well separated, whereas the more peripherally situated villi are closely wedged together. This, I would attribute to the compression of the "capsule" driving the still fluid blood to the centre of the affected part, where the villi being movable are kept separate, but the villi in the vicinity of the "capsule" are unable to leave their situation being anchored by their stems; and are compressed against

one another, and the space between them is quite obliterated (photos XII and XIII).

Foot-note. This theory is in general outline the one which Maier put forward; but I arrived at it quite independently, indeed, I had not the reference to his work until this essay was completed

Edin. of Lond. 1896 p. 449, 1897 p 265.

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note place of publication of book or Journal

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Description of Photographs.

Photo I x 100

From Placental Margin:

Maternal surface.

The upper is the decidual surface.

Decidual cells are seen dipping down towards chorionic surface.

Photo II x 100.

From Placental Margin:

Chorionic Surface.

In centre of field – clump of large decidual cells. Villi embedded in fibrin.

The amnion is seen at the lower border.

Photo I

Photo II

Photo. III × 80

Decidual process -
Maternal Surface to Left
of photo.

Photo IV × 80

Decidual process.
From centre of Intervillous
space - Showing branching.
Notice network formation
in the fork of the process.

Photo III

Photo IV

Photo. V.

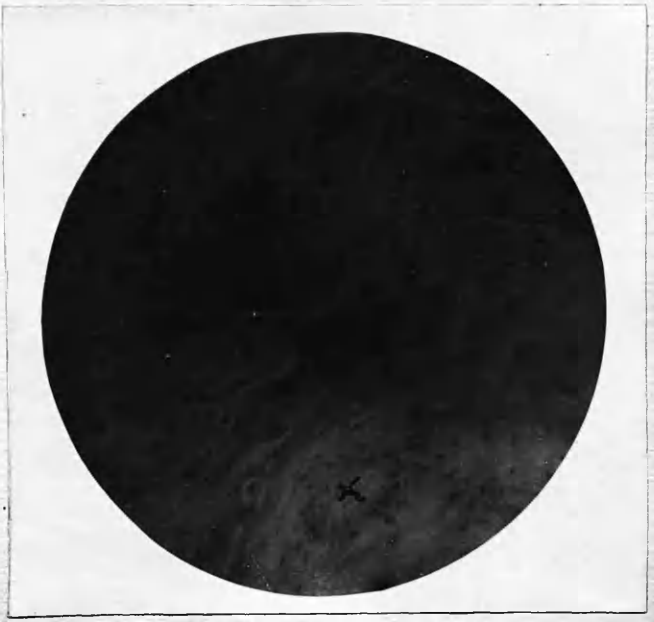

Photo. VI

Photo VII. × 450.

Longitudinal section of villus. Extension of fibrin within syncytium. The syncytium on proximal side of the villus has undergone fibrinous degeneration. The blood vessels are patent and are not thickened.

Photo VIII

Copy of fig. 4 – Williams's paper Shows engagement of an inter-villous space. The syncytium is lost only where the villi are in contact with the fibrin. Fibrin is seen extending from the area affected along the subsyncytial stratum. Vessels obliterated

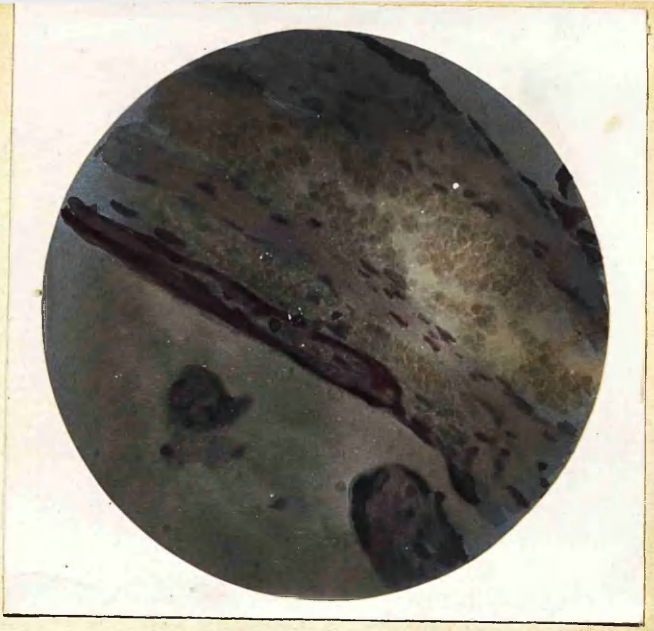

Photo VII

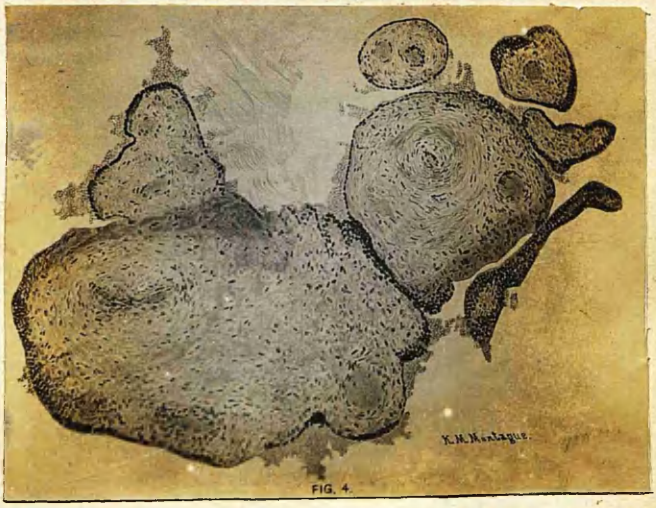

FIG. 4.

Photo VIII.

Photo IX. ×100.

Villi lying outside fibrinous infarct. The interval between the villi is occupied by large cells. The same cells can be traced within the infarct to some considerable depth.

Photo X. ×80.

Decidual process in centre with villi attached

will
reproduce

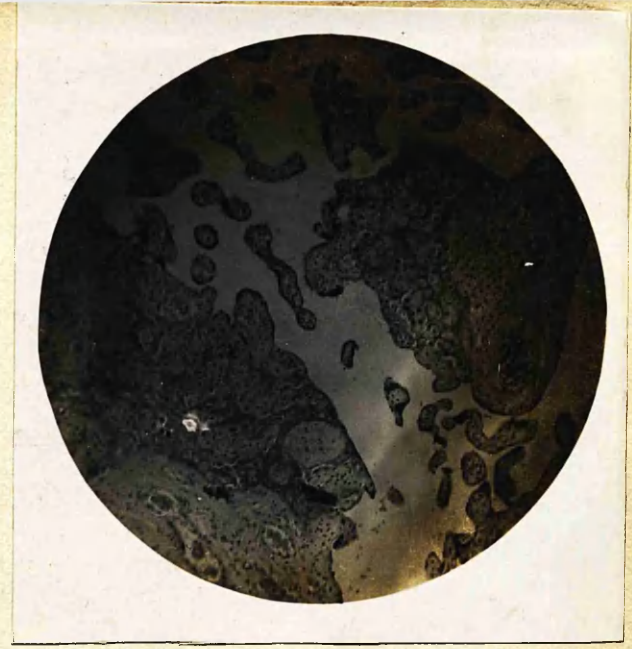

Photo IX

Photo X.

Photo XI ×80

Decidual process bordering a new-formed non-fibrinous infarct.

Photo XII ×80.

Centre of non-fibrinous infarct. Intervillous space open. Syncytium has undergone coagulation-necrosis. - No fibrin present. Owing to the failure to stain the villi of a non-fibrinous infarct, photographic reproduction was not successful.

Photo XI

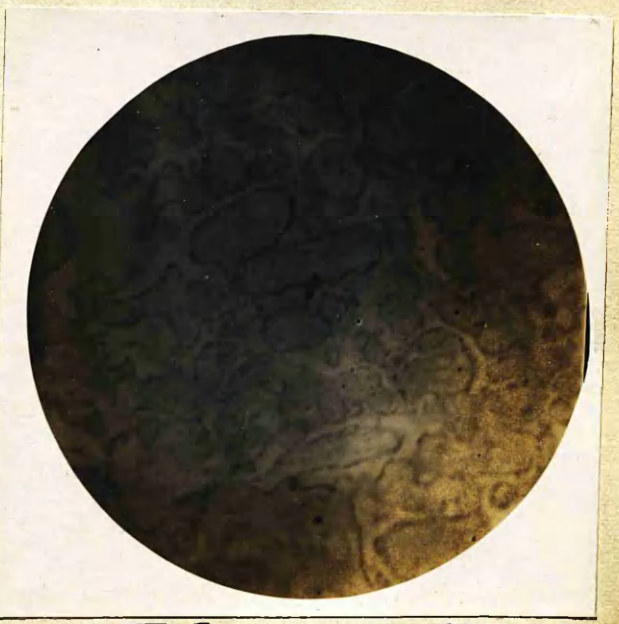

Photo XII

Photo. XIII ×80.

Periphery of non-fibrinous infarct.

Shows obliteration of intervillous space. The fibrinous "capsule" takes the eosin stain and appears dark on the R..

Photo XIII