THE LUMLEIAN LECTURES ON THE PATHOLOGY OF INTRA-UTERINE DEATH.

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LECTURE III—DISEASES AND ANOMALIES OF THE PLACENTA.

ONE of the chief difficulties in studying the literature of these diseases as they have been described by various observers arises from the tendency on the part of authors to regard the particular morbid change which they have had the opportunity of investigating as the chief or only disease with which the organ is affected. All other morbid appearances are for them but consequences or complications of a specific and cardinal lesion upon which the rest depend. To give an example, Charpentier avers that the placenta is attacked by only one morbid change. He accepts the investigations of Robin as illustrating the whole range of placental pathology, and believes that when disease has invaded the placenta, it commences always in fibro-fatty change; and consequent on this, blood extravasations, and successive transformations of effused blood, account for all the various pathological appearances associated with the death or enfeebling of the foetus.

Verdier and Bustamente take entirely another view of these changes. It seems to me that one cannot long investigate the diseases of the placenta without discovering that they are most complex in their nature, and that they proceed from a variety of causes inextricably intermixed—sometimes one pathological condition having the precedence, sometimes another. The placenta is, in truth, as liable to be affected by a variety of diseases as the liver or the lung, and some of its diseases bear not only a striking resemblance to diseases occurring in those organs, but have affinities with them, and may depend on the same causes. Again, confusion has arisen from investigators describing the same morbid condition under a different name, probably because it was observed only in one stage of progress or with some variations, and some have fallen into the error of ranging affections which are intrinsically different under the same appellations.

After the third month of utero-gestation, the various membranes surrounding the embryo have come into apposition, there is no distinct interspace between them, and the foetus lies suspended in its liquor amnii in the centre of the ovum. By this time the placenta has assumed a definite form, and the embryo is now chiefly dependent upon it for the supply of those materials which are necessary for its growth. It is as yet, however, in a rudimentary form, and instead of having those sinuses, or cavities, everywhere permeating its substance which are peculiar to its structure at a later period, it consists of villi of the chorion, round which loops of maternal vessels have been thrown, in such fashion as to form a complex, but very distinct, network throughout the placental mass. I have repeatedly seen these maternal loops naturally injected in young placenta, diseased, as well as normal, for in some forms of indurated young placenta they are almost as distinctly seen as when the organ is healthy, and microscopic sections are more easily made. Their prototypes are seen in the placenta of some mammalia, which have been described more especially by Professor Turner, and I have thrown out the suggestion that by dilatation and further development these maternal vessels, appearing at first in the young placenta as capillary loops, become eventually the sinuses of the maternal portion of the placenta, into which the foetal villi are suspended as the gills of a fish are suspended in water. There they are bathed by the maternal blood-current, and both absorb materials for the growth of the embryo, and also effect the aeration of the foetal blood, which is so necessary for its well-being. It is obvious, therefore, that a healthy state of the placenta is necessary for the safety of the foetus, both in the earlier and later months. Until the placenta is fully organised, the nutrition of the embryo is carried on by the chorion villi more or less over the whole sphere of the ovum. But when the placenta is formed the whole nutritive and respiratory function is concentrated upon it, and the chorion villi degenerate and atrophy over the rest of the chorion membrane.

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Extravasations of blood into the young placenta are very common, and they occur as the result of rupture of some of those vascular maternal loops which I have just described. If the extravasations be limited in extent, the life of the embryo may not be compromised; but a more extended apoplexy at once stops its nutrition, and the root-like processes of the young villi become so compressed, that the circulation can no longer be carried on through the minute foetal blood-vessels which they contain. It is common enough to see clots, in varying stages of change in accordance with the date of their formation, in young placenta which have been expelled as the result of abortion, and if the ovum has been long retained after extravasation has been of such extent as to kill the embryo, the whole placenta is generally found to have become indurated and in a measure contracted. The clots have been consolidated, decolorised, and contracted, while the villi in other parts of the organ, not in the immediate vicinity of the altered blood-clots, have become atrophied, and so firmly fused together, that it is almost impossible to separate them for microscopic purposes.

Apoplexy takes place into the substance and on the uterine surface of the full-grown placenta also very frequently. There is, however, this marked difference from the earlier period, that now the placenta is larger and thicker, and isolated clots may be deeply imbedded in its substance, only to be exposed by incision. Others may have been formed between the uterine walls and the maternal surface of the placenta, making a deep depression into the centre of a placental cotyledon, and compressing the placental structures. It is further to be remarked, as a difference in the results of blood extravasations in the young and more mature placenta, that in the latter several successive effusions of blood may take place, forming apoplexies of varying sizes and dates, without so seriously jeopardising the life of the foetus as it would do at an earlier period of gestation. The larger size of the placenta and its more extended surface afford a better chance of so much of the potential part of it retaining its healthy relations, and so carrying on the placento-foetal circulation that embryonic life is sustained. This may be, notwithstanding that the whole amount of intended pabulum is not furnished, for the simple reason that a considerable portion of what may be called absorbing radicles have been destroyed, or their function impaired by the local lesion.

Apoplexy, and the various morbid changes associated with it, is, perhaps, of all others, the most frequent pathological condition easily recognisable with the naked eye in the placenta. This being so, it can scarcely be a matter of surprise that some authors have regarded it as the chief of placental diseases, being, indeed, the prime factor in all placental disease, and the forerunner of all other pathological appearances. To it has been attributed the occurrence of the so-called tubercle, and also the formation of scirrhus in the placenta.

Sir James Simpson,⁵⁹ in his Memoir on Congestion and Inflammation of the Placenta, which is remarkable for its literary research, describes placental congestion as the forerunner of apoplexy. The congestion, according to him, consists of an extraordinary accumulation of blood in the spongy tissue of the placenta. This only requires to be pushed a degree further to lead to rupture of the vascular coats, and escape of blood into the parenchyma or on the maternal surface of the organ. He then goes on to describe the changes which take place in the extravasated blood, and the effect on the placental structure.

In 1839, M. Jacquemier wrote a communication on Extravasations of Blood in the Placenta which was regarded as classical in France, and he gives an excellent description of the pathological appearances observed. He points out that the effects of extravasation vary with the stage at which gestation has arrived. According to him, at an early period the villi of the chorion are so loosely imbedded in the decidua serotina, that there may be said to be space between the two membranes. If, therefore, a rupture takes place in a utero-placental blood-vessel, the extravasation is the more general, and will spread, because of the laxity of tissue, and thus pregnancy is the more likely to be interrupted. In the later period the placenta is more developed and compact—the chorion and decidua have become fused together, and if extravasation occurs into the substance of the placenta it is more limited in extent—generally, indeed, confined to the lobe in which the apoplexy has occurred, although more than one lobe may be affected. Jacquemier describes three varieties of placental apoplexy in the later months, which I need not give in detail here.

Verdier, who wrote a thesis in 1868, entitled *Researches on Apoplexies and Hamatomas of the Placenta*, goes so far as to say that fibrous changes—scirrhus hardness, fibro-fatty, and those which

⁵⁹ Vide Obstet. Works, 397.

appear due to inflammation—are due only to extravasated blood and the changes taking place in it.

In 1848, Bustamente published a memoir on Placental Disease, which attracted much attention in France and elsewhere. Admitting the accuracy of previous observers as to the frequent presence of so-called apoplexies in the placental tissue, and their influence in producing death of the fœtus, he contends that the masses of blood so found are not due to vascular rupture and extravasation, but are produced by thrombosis in the maternal sinuses of the placenta. He points out that deposits are very frequent in the deeper structures of the placenta, and even near its fœtal surface. The mechanism of their production he believes to be somewhat as follows: The whole of the villi constituting the fetal portion of the placenta, and found equally distributed throughout its entire mass, are bathed with maternal blood in the lacunæ in which they are immersed. The maternal circulation through these lacunæ is necessarily slow, because it is poured into the placenta by maternal arteries, and, before passing back into the maternal veins, it must pass through the dilations which constitute the placental sinuses or lacunæ. Anything, therefore, which disturbs the utero-placental circulation, favours stasis in the placental lacunæ, and more especially in those portions of the placenta which are most distant from the maternal supply. The increase of fibrin observed in the blood of pregnant women, various general pathological conditions which render the blood more readily coagulable, syncope by slowing or arresting the circulation, even for a limited period—all favour stasis of the utero-placental circulation and resulting thrombosis. The villi, once imbedded in the blood of a thrombosis, although at the moment healthy, speedily degenerate. The loops of fetal capillaries become granular, and eventually disappear. The epithelial covering of the villus and the proper fibroid structure of the stem become fatty and disorganized, and the whole tissue eventually shrivels. But this is not the only change which takes place, according to Bustamente, when placental thrombosis has occurred. The blood-clot itself becomes organized. He quotes O. Weber, in Billroth's *Surgery*, as an authority on the organisation of extravasated blood, and avers that he has himself seen leucocytes in the centre of placental clots, not near living tissue. These leucocytes develop, and form a connective tissue, with nuclei readily seen by the addition of acetic acid, and firmly unite the villi to each other, thus producing a dense and resisting tissue, the component elements of which it is difficult to separate.

Other and later authors have expressed the opinion that no apoplexy takes place into the substance of the placenta, either in the early or later periods of gestation, without some previous degeneration of structure. Charpentier quotes from a contribution of M. Bailly, who endeavours to prove that some morbid change invariably takes place prior to blood extravasation, and in this way the morbid condition bears analogy to apoplexies in the brain and elsewhere. Charpentier himself, in his elaborate essay on *Diseases of the Placenta*, says it is a law that clots are only found in cotyledons of the placenta previously altered by disease. There is a very general consensus of opinion that when effusion of blood takes place into the placenta, it is always, or almost always, from the maternal system; and there are some curious coincidences occasionally noticeable between blood extravasations in the placenta and apoplexies in other parts of the body of the same patient, indicating a feeble resistance in the blood-vessels in more than one organ at the same time. Latour (*Rev. Méd.*, 1843) has put on record the case of a woman who died of cerebral apoplexy after spontaneous abortion at twenty-eight years. A painful case occurred in my own practice some years ago illustrating the point. A lady, most anxious to have children, lost three successively in the latter half of gestation. In each pregnancy uterine hæmorrhage came on, apparently without cause, and ended in the expulsion of the child before it was viable. No disease of the child or placenta could be detected, beyond the fact that the twisting or spiral arteries in the centre of each maternal lobule were found to be in a state of partial fatty degeneration. In the fourth pregnancy, as the result of great care and absolute rest, gestation was carried on to the eighth month, when uterine hæmorrhage set in as before. The labour being speedy the child was born alive, but within an hour of what seemed a comparatively happy delivery a sort of convulsive seizure supervened, followed by deep coma, and in two hours later the patient died with all the signs of apoplexy of the base of the brain.

Charles Robin is one of the chief and explicit exponents of the doctrine that morbid change goes on in the placental tissue preliminary to blood extravasation. He differs *in toto* from Jacquemier, Verdier, Bustamente, and others, as to the order of pathological events in the placenta. He insists that no blood extravasations take place in the placenta without previous disease in the tissue, and the morbid change

which is the most common forerunner of apoplexy is not mere vascular change, but an alteration of the whole structure of the villus.

As Robin's researches on this subject are regarded in France as the most important contribution of late years, I venture to speak of them in some detail. M. Robin points out that in the early formation of the chorion a certain proportion of the villi do not contain vascular loops, but the centre becomes fibro-cellular, and the nuclei are seen, by the addition of acetic acid, to be of an elongated form. This centre is easily distinguished from the elastic sheath, which retracts and corrugates when lacerated. In some of these non-vascular villi are fine granules and distinct drops of oil or fat, highly refracting the light, and attacked by potash, but not by acetic acid. The fibro-fatty degeneration of villi is, therefore, according to Robin, a normal process, by which useless and non-vascular villi undergo atrophy; but this fibro-fatty change may become a morbid process, and, by extending to other and active villi, may first compress and then obliterate the contained fetal blood-vessels, thus producing a solid villus which is composed of dense fibro-cellular structures admixed with fat, and whose function is entirely abrogated by the change. This morbid transformation may occur in the mature placenta as well as in the earlier stages of its formation, and the ultimate effect is to produce a firmness and density of tissue, in which it is most difficult to separate the component elements. The amount of fatty deposit and its proportion to fibroid change varies in different cases, and the larger the amount of fatty granules and molecules, the less distinct are the nuclei of the fibroid structures.

According to Robin, extravasations of blood are always consecutive to placental disease of this or a kindred form, and he contends that this prior change is fibrous obliteration of the villi with fatty degeneration of tissue.

I have mentioned the views of M. Robin here because they have a direct bearing on the production of placental apoplexy, and are associated by him, and those who think with him, as cause and effect—the fibro-fatty degeneration being the invariable precursor of hæmorrhage into the placental substance.

Supposing it to be admitted that some pathological change not infrequently precedes blood-extravasation into the deeper structures of the placenta, there can be no doubt that effusion of blood takes place with comparative frequency on the maternal or decidual surface of the placenta from other causes. The most common of these causes is an accident of some kind, or uterine contraction, which tears the utero-placental vessels across, and causes the formation of apoplectic clots between the placenta and uterine walls. Such hæmorrhage may go on to separate the membranes from the uterus down to the os uteri, and then it appears as flooding from the genital passages. If, on the other hand, the results remain at the point of extravasation, the amount of clot may be so considerable that it bulges forward the uterine wall, and can be felt as a projection by abdominal examination, constituting the so-called "concealed hæmorrhage." When the clot is small, it remains in the locality of its formation, and, if pregnancy is not further disturbed, it undergoes all the changes peculiar to retained clot elsewhere. Large or small, the clots before disappearing go through various phases. According to Ercolani, the fibrin assumes a lardaceous, yellowish aspect, the appearance of fibrillæ gives place to two kinds of granulations, the one of proteic nature, soluble in alkalies and acetic acid; the other of a fatty nature, resisting these reactive agents. If a clot is to become organised through the process originally described by O. Weber, and afterwards confirmed by Virchow, it may eventually form a distinct neoplasm in the placenta. These neoplasms are rare, but cases have been put on record by Virchow and also by Danyau.

The interference with the life of the child or with the course of gestation depends upon the amount of extravasation and the excitability or quiescence of the uterine walls under the provocation to contraction.

Inflammation of the Placenta, or Placentitis, has been described by Brachet, Dance,⁶⁰ Simpson, Cruveilhier,⁶¹ and other authors. Simpson described it as consisting of three stages, the first being one of congestion, in which the tissue is engorged by an unusual accumulation of blood in the vessels; the second characterised by the exudations of coagulable lymph, producing a greater density of tissue and eventual induration; the third stage being that in which purulent matter is formed.

Brachet, in the *Revue Médicale* for 1828, gave a very full and precise description of four cases in which the placenta bore a resemblance to the red hepatisation of the lungs, was friable, engorged with blood, and dense in tissue. In some sections the structure was yel-

⁶⁰ *Report. Gén. d'Anat.*, Tom. iii.

⁶¹ *Anat. Pathol.*

lowish, and strewn with fatty degeneration analogous to fatty liver. He further states that he has seen circumscribed abscesses in the placenta, such as occur in inflammation of other parenchymatous organs. Dance and Brachet both aver that they have seen a deposit of pus covering the entire surface of the placenta, while Jacquemier and Cruveilhier each report the presence of purulent matter as the result of inflammation in the placenta and adjacent uterine veins. Jacquemier teaches that, especially when a portion of the placenta has been separated, inflammation may be set up and pus formed.

In later days a good deal of scepticism has been expressed about the existence of placentitis; at least, so far as its parenchyma or foetal portion is concerned. Bustamente disbelieves in its existence altogether, and says the evidence rests on the supposed presence of pus, which has been shown to be fallacious; the supposed purulent matter, according to Robin and other observers, being merely pseudo-pus, produced by broken-down fibrin.

It has been further argued that, according to modern theories, inflammation of the placenta is impossible, since there are no capillaries in the maternal portion, and there are no nerves to regulate the contractility of vascular walls in the entire structure. This seems to me not conclusive reasoning. There are foetal capillary loops in all the active villi, and there are also minute capillaries of maternal origin in processes or dissepiments of the decidua which circumscribe the foetal villi.

It is true, nevertheless, that the presence of purulent matter in the placenta in most of the reported cases is supported by imperfect evidence. There are only some ten cases of abscess in the placenta recorded, and as no account is given of the microscopic examination of the supposed pus, the evidence is plainly unsatisfactory. Purulent matter has, however, been found and duly authenticated on the surface of the placenta, and in the uterine sinuses at or near the placental site.

That some morbid change analogous to inflammation does take place in the placenta or its neighbourhood is apparently indicated by the firm adhesions which are occasionally formed between the placenta and the uterine walls,⁶² and from the traces of an exudation which agglutinates and compresses the villi of the placenta. The progress of such inflammatory action is, moreover, often marked by the presence of tenderness over the gravid uterus, with general indications of pyrexia. Braun, Schroeder, and Spiegelberg regard this placental inflammation as not a form of true placentitis, but of chronic endometritis affecting the mucous membranes of the uterus primarily, and only extending to the foetal structures incidentally. Hegar and Mäier⁶³ describe it as a form of interstitial endometritis, in which the villi are agglutinated and compressed by the hypertrophied elements of the serotina, and the development of a new connective tissue.

Fig. 7.—Placental tissue showing granular or slightly striated matrix, with elongated decidua cells, constituting the first stage of interstitial placentitis (Hegar and Mäier).

According to Spiegelberg, it is only through the work of Hegar and Mäier that we have got precise knowledge concerning the so-called inflammation of the placenta. Their memoir is certainly an example of careful work, and is well worth perusal. I regret that I must pass it with so brief a notice. This inflammation begins, according to them, in the formation of soft granular exudation, and ends in the production of induration and disorganisation. The decidua tissue

⁶² Desormeaux and Dubois say that inflammation not only makes the placenta adhere to the uterus, but also to the fetus, and so produces monstruities. On the other hand, Robin holds that adhesions between the placenta and uterus are not due to inflammation, but result from the persistence of the firm condition of the uterine mucous membrane found in the unimpregnated uterus, instead of softening as usual in pregnancy.

⁶³ Virchow's *Archiv*. 1871.

normally consists of elongated cells imbedded in a small quantity of amorphous intercellular substance. When inflammation begins there is an enormous development of fusiform cells, with an increase of intercellular granular material. Then the cells become deformed, distended with fine fat granules, and eventually the whole structure is converted into fibroid tissue. It is apt to repeat itself in the same individual, and the chronic morbid condition of the mucous membrane, upon which it depends, may lead to earlier abortion in other pregnancies. The recognition of the true cause of this placentitis, as Spiegelberg points out, affords a clue for treatment, and also a hope that its recurrence may be prevented on future occasions.

Fig. 8.—a. Strong development of connective tissue taking the place of the earlier cellular formation seen in the normal placental structure. b. Indication of vessels obliterated by the growth of connective tissue. c. Extremities of villi atrophied and in fatty degeneration (Hegar and Mäier).

Under the influence of this morbid change, the utero-placental vessels and the villi undergo varied modifications, of which the constant result is atrophy and degeneration by compression, in such sort that, passing through various intermediate stages, they become hard, whitish nodules or cords, like old cicatrices, imbedded in the spongy substance of the placenta. This chronic and progressive production of connective tissue, on the surface and in the substance of the placenta, constitutes a work of induration, the net result of which is the obliteration of the double system of foetal and maternal vessels, and secondary disturbances in the circulation which are evidenced in thrombosis or in apoplexy more or less extended. The whole morbid process, say these authors, bears the closest resemblance to cirrhosis of the liver.

Neumann⁶⁴ has described a condition which he also regards as produced by inflammation, and which he terms "sclerosis" of the placenta. In this form of disease the villi develop in such luxuriance as to encroach upon the maternal sinuses, and eventually to obliterate them. It may therefore be regarded essentially as an affection of the foetal part of the placenta. Neumann recognises the fact that this sclerosis of the placenta, in some cases at least, may be the result of an inflammatory process in the decidua, and result in a hyperplasia of its connective tissue.

Meyer, of Freyburg,⁶⁵ while he says that he has never found the condition which Neumann calls "sclerosis of the placenta," describes a morbid process which he has carefully investigated, and which consists of hypertrophic proliferation of the connective tissue in the placenta, with consecutive induration. The more this connective tissue grows, the more the proper parenchyma of the placenta dwindles by pressure and wasting of the vessels; the whole organ shrivels, and the diminution in the size of the organ results as much from the loss of normal, as the contraction of newly formed, tissue. In the normal placenta there is, he says, only a small amount of connective tissue, but there is a large amount in these abnormal cases. This author does not agree with Simpson, Rokitsansky, Scanzoni, and others, who suppose inflammation followed by exudation to be the cause of the formation of connective tissue.

There is a sort of general resemblance between the descriptions

⁶⁴ *Königsb. Med. Jahrb.*

⁶⁵ *Monats. f. Geburt.*

given by Hegar and Mäler of placentitis originating in the decidua part of the placenta and Meyer's "hypertrophic proliferation of the connective tissue in the placenta." The differences are as to the nature of the process and in the details of microscopic change. Henning has farther made an important contribution on the subject of placentitis, and explained how it produces adhesions between the uterus and placenta and membranes.

Placental Phthisis.—At different times extending over years, I have seen specimens of a disease of the placenta, which does not correspond accurately with any of these descriptions. In some of its stages, it bears a close resemblance to morbid changes which have already been described by the authorities I have quoted, and I have endeavoured to trace its connection with these other morbid processes. It may, indeed, combine more than one of them in the progress of its development. It differs, nevertheless, in some important particulars of its pathological history, which I shall endeavour to indicate. It has so many analogies to phthisis in the lung, that it may with propriety be named "phthisis of the placenta." Sir James Simpson has previously used the term "placental phthisis;" but he comprehended under this appellation various forms of disease in the placenta which interfere with intra-uterine respiration and nutrition.

In its earlier stages, repeated investigations have convinced me that it is not, primarily at least, a mere increase of the cell structure, nor of the fibroid element in the placenta, as described by Hegar and Mäler, or by Meyer; but that, in its first stage, it consists of an exudation or deposit, thrown out among the villi, and that it is probably due to some modification of a low inflammatory process.

The disease is not really tubercular in its character, nor have bacteria been found in its substance, neither is it primarily or essentially a fatty degeneration, for the deposit is not affected by the reagents which show the presence of fat. Its corpuscles, unlike fat granules, are thoroughly coloured by the material used for staining the microscopic sections. It is not due to changed blood-clot, for there are neither altered blood-corpuscles nor crystals in its substance. Its general appearance, both before and after section in the earlier phases, is rather that which has been described as fibrinous or other analogous deposit, and it has the chemical characters of protein compounds.

I can scarcely go into the minute pathological anatomy of this so-called placental phthisis in these lectures. This I propose to do in a paper to be offered to the Obstetrical Society; but I may state broadly that, in the specimens I have examined, masses of exudation were found in the substance of the placenta which rendered its tissue firmer and denser, and produced, so to speak, "hepatisation" of the part affected. The hepatisation was partial or more general in accordance with the amount of disease present, and the localities affected were commonly the centre of a lobule, but the deposit might be more irregularly placed, and even diffused. Some of the masses were dense throughout, gradually shading off into the healthy surrounding tissue; others were undergoing a process of crumbling or disintegration in their centre, after the fashion of tubercular masses in the lungs; and in a third form the disintegration of the centre had attacked the blood-vessels, blood was extravasated, and apoplectic clots, limited by layers of fibrin, were undergoing all the usual changes in accordance with the date of extravasation. The primary exudation, when recent, was amorphous or finely granular, and somewhat striated, with round interspersed corpuscles, but no fat particles, and it glued the villi together so as to fuse them into a firm mass. Where the tissue was breaking up and becoming disorganised, the villi were wrinkled and broken, and undergoing fatty degeneration. In places, again, where exudation was older and was not undergoing the destructive process seen in the centre of some of the exudation masses, it was being rapidly converted into connective tissue, firmly binding the now exsanguine villi into one solid mass, and completely abrogating their functions.

In the order of successive changes, therefore, the disease runs in somewhat parallel lines with phthisis in the lungs. First comes solidification; then softening, with breaking up of tissue and resulting hæmorrhage. In parts of the placenta where the exudation has been more scanty, or endowed with higher vitality, it does not break up, but forms fibroid connective tissue, bearing some analogy again to fibroid phthisis, or perhaps to cirrhosis of the liver, as remarked by Hegar and Mäler.

A notable feature in many of the microscopic sections was the great hypertrophy of the contractile coat of the foetal blood-vessels. This was observable in the larger branches, but was most marked in those of intermediate size before they divide into capillaries. A similar hypertrophy of the vascular tissues was observed by Fränkel in a placenta, which he described as syphilitic, and also by Lawson

Tait in his description of a form of placental disease where no syphilis was traceable. Lawson Tait regarded it as the result of a compensating effort on the part of the arteries to overcome the obstruction found in the extremities of the villi, which eventually ends in their contraction and closure, and he points out its analogy to the hypertrophy of the vessels observed by Dr. George Johnson in renal disease. Hegar and Mäler had previously spoken of this compensating action as likely to lead to extravasation of blood and apoplexy in sounder portions of the placenta.

There is a marked difference between this affection and primary fatty degeneration of the placenta, which is comparable, for example, to fatty degeneration of muscle. Here there is an adhesive exudation or infiltration distinctly seen to be thrown in among the villi, as the first step of the morbid process. It pushes some villi aside, while it crowds others into small space, stopping the circulation, both foetal and maternal. When the deposit degenerates because of its adventitious origin—which it may do as one of the results of the pathological process—it produces also degeneration of the villi, which gradually break up and soften. Thus cavities are formed which become full of blood. Women who lose their children from such placental disease as I have described are not necessarily phthisical, but have sometimes a strumous family history, and I have seen like cases associated with anæmia and syphilis. In anæmia it is well-known there is a deficiency of red globules in the blood with a larger proportion of fibrin. All these conditions may predispose to a low type of inflammation, as in other organs of the body, and to such deposits as those described.

It is not an uncommon form of disease, and there is some reason to believe that, although commonly it runs a chronic course, yet occasionally its progress is very rapid. Dead children are sometimes born plump and in good condition, with the placenta so largely affected with the first stage of infiltration that intra-uterine respiration could no longer be maintained. The well-nourished condition of the child seems to indicate that in these instances the disease is of recent origin, and that it killed the child more speedily than in the chronic form usually observed.

Fatty Degeneration of the placenta, as first described by Dr. Barnes⁶⁶ in this country, is, according to the author named, analogous to fatty change attacking other organs, as the heart and arteries, for example. In its simplest form it is seen in cases where the death of the child has arisen from some other cause than disease in the placenta. In the case of a child which died near the full time from intra-uterine small-pox, and was retained some time after, all the villi, as well as the maternal portion of the placenta which I examined, were crowded with oil-globules and compound granular fat particles. The villi were swollen and cedematous, and their external covering was readily separated. The entire placenta was paler and softer than usual, and contained an abundance of serous fluid in all its meshes. Dr. Barnes is careful to draw a distinction between this sort of passive fatty change following the death of the fœtus, which he calls "fatty transformation," as distinguished from "fatty degeneration," which, according to him, commences previously in the placenta, and, if extensive enough, kills the child. This he calls "true fatty degeneration," and compares it to the fatty degeneration of the brain, liver, and muscles occurring in the adult. It is characterized by the morbid changes being partial, invading one or more cotyledons, and the diseased masses are often imbedded in the midst of healthy tissue. The child may be living if only limited portions of the placenta are diseased. If the disease is extensive, it kills the child, and there is an intermediate stage in which the child lives; but its nutrition is interfered with, and it becomes puny and starved. Dr. Barnes figures both the decidua cells forming the maternal part of the placenta, and also the foetal villi studded everywhere with fat globules. The villi are brittle and firmer than natural. Under the microscope they are seen to be thickly covered with oil-globules; the proper structures are disorganized; and the blood-vessels which are empty have their courses marked out by the clusters of oil-molecules which follow their tortuous course.

Kilian⁶⁷ had, prior to Barnes, written on "A New Disease of the Placenta," which partly anticipated Dr. Barnes's observations on "fatty placenta;" and, soon after Barnes, C. Robin published his monograph to which I have previously alluded. Robin, as we have seen, does not believe in a purely fatty degeneration, and regards the fibrous transformation of the villi in so-called fatty placenta as the chief lesion, the fatty deposit being associated with it only as a complication in the obliteration of the villi.

The description of Barnes is certainly accurate so far as some phases

⁶⁶ *Medico-Chir. Trans.*, 1851, etc.

⁶⁷ *Neue Zeitschr. für Geburt.*, 1850.

of placental change are concerned. The question is, whether fatty degeneration of the placenta during the life of the child really occurs as a primary pathological change, or is invariably preceded by some exudation or other morbid change which initiates the fatty metamorphosis. Adventitious deposits are more liable to fatty degeneration than normal tissues; and it is to be noted that, in the majority of examples where fatty degeneration has attacked the placenta, the portions affected are not softer, but firmer and denser, than usual, the villi being almost inseparably united together. It is conceivable, nevertheless, that some depraved and impoverished conditions of the maternal blood may lead to such deterioration of vitality in the placenta that it begins to degenerate and die, and thus kills the foetus.

Fig. 9.—Fatty Degeneration of Placental Villi. (Barnes.)

The deposit of minute fatty molecules and of larger oil-globules is associated with most forms of morbid change in the placenta—in some stages, at least. In consonance with the general law that effete and useless parts of the organism undergo degeneration into fat, and so are more amenable to absorption, the placental structures, when altered by disease so far as to be unfit for the exercise of their normal function, begin to undergo fatty change, and this results in atrophy of the part affected.

Fatty degeneration, whether it occurs as a primary change or is the secondary effect of some other pathological condition which precedes it, may produce softening and disorganisation of the placental tissue, and so favour extravasation of blood or apoplexy. In other placenta it leads to atrophy. All the structures shrivel and condense, so that the organ seems like a mass of dense fibroid tissue; it undergoes, in fact, the fibrous transformation which I have previously described. The tendency of modern investigators is evidently to regard fatty degeneration not as a primitive change, but a phase or stage of some other morbid condition which precedes it or is associated with it.

Dr. Druitt⁶⁸ regarded fatty degeneration of the placenta as a normal condition towards the end of pregnancy, and as preparatory to detachment at the time of delivery. For him, therefore, fatty degeneration was only the extension to a morbid degree of a condition which exists normally to a certain extent towards the end of pregnancy in all placenta. There is, it is true, some amount of fatty change towards the end of pregnancy in certain portions of the after-birth which have now no active purpose to serve. This is notable round the margins and in the membranes. The nuclei in the epithelial cells of the amnion are surrounded with a ring of fat particles in nearly all placenta at the full term; but normally there is no fatty degeneration in the potential villi as pregnancy advances, and it is obvious that the nutritive and respiratory functions have to be carried on for the well-being of the child just as actively up to the termination of pregnancy as at any previous period.

Hiffelsein and Laboulbène,⁶⁹ Charpentier, Simpson,⁷⁰ Goodell,⁷¹ Whitaker,⁷² Cowan,⁷³ and others, have all made contributions to the subject, and all regard it, however it may arise, as intimately associated with the processes which produce destruction of fetal life.

Ercolani has described as synonymous with fatty degeneration of the placenta what he calls "cellular hyperplasia and hypertrophy of the parenchyma of the placental villi." He agrees with the general accuracy of Robin's observations in regarding the presence of fat-granules with fibroid degeneration of the villi as an accidental and not essential

complication. The firm and dense placenta, which have been written upon by Bustamente and Neumann as "sclerosis of the placenta," he considers as essentially due to a hyperplasia not of the connective tissue, but to a cellular hypertrophy of the parenchyma of the villi, either simple or complicated with obliteration of the contained vessels, and also in some cases with lesion of the glandular organ or decidual covering.

It is obvious from the description that the disease described by Ercolani is different from fatty degeneration as described by Barnes, and I have not seen any specimen having the exact characters of those described by Ercolani.

Myxoma Fibrosum.—Virchow has described a very curious morbid transformation of the villi of the placenta, which he terms "myxoma fibrosum." It is quite different from the cellular or fibrous degeneration of the villi described by Ercolani and Robin. It consists of such enlargement of the stems and villi by fibroid hypertrophy that they form in some cases distinct tumours in the placental structure. The cases are somewhat rare, but I saw two excellent examples in Copenhagen which have been described in Virchow's *Archives* in 1878, by Storch.

Fig. 10.—Myxoma Fibrosum of the Placenta.—Virchow (after Storch).

Fig. 11.—Section of a Tumour showing Fibroid Structure.

In one, the tumours were as large as hazel-nuts, attached by pedicles to the deeper seated trunks of the placental villi, and, when an incision was made into the substance of the placenta, they projected through the opening like so many adherent hard nodules, or perhaps I may say digitated radishes. Microscopic examination showed that they were composed entirely of fibroid elements formed by a cellular hyperplasia of the central part of the villi, and that they were enlargements or excrescences on the villous trunks, with an external arrangement somewhat analogous in appearance to cystic chorion. Virchow regards the disease as a transformation of the mucous element pertaining to the villus structure into fibroid tissue, instead of the soft myxoma which is seen in cystic chorion. The fibroid change is, however, unlike the cystic chorion, more frequently connected with the later period of pregnancy, although indications of it are sometimes seen in the earlier months, and, therefore, it is described as a disease of the fully developed placenta. Hildebrandt has also described a good example in *Mon. für Geburt.*, Bd. xxxi; and Dr. Sinclair, of Boston, United States,⁷⁴ another. It is probable that the case

⁶⁸ *Lancet*, 1853.

⁶⁹ *Soc. de Biol.*, 1865.

⁷⁰ *Obstet. Works*.

⁷¹ *American Journal of Obstet.*, vol. ii.

⁷² *American Journal of Obstet.*, 1870.

⁷³ *Edinb. Med. Journ.*, 1864.

⁷⁴ *Journal of the Obstetrical Society of Boston*, 1871.

of sarcoma in the placenta given by Hyrtl is one of the same kind. In Storch's first case the child was born alive, but feeble, and died an hour after birth. If the placenta is only partially affected, the child, though emaciated, may live; if the disease is more universal, the child dies.

I pass over oedema of the placenta, melanosis, calcareous or osseous concretions, cysts and tumours, so that I may be enabled to say a few words on the subject of syphilitic placenta.

Syphilitic Placenta.—It is important, if possible, to answer the question, Is there a syphilitic placenta?—that is, a form of disease in the placenta associated with syphilis which may be recognised by examination. Much diversity of opinion has been expressed concerning this matter, and various descriptions have been given of the syphilitic placenta which do not agree.

That syphilis is a potent cause of placental disease and abortion has been recognised by some of the oldest authors. Astruc mentioned it as far back as 1796, but Murat (according to Deschamps⁷⁵) was the first to describe certain appearances as belonging to the syphilitic placenta. To illustrate the diversity of opinions in recent times, I may mention that Verdier regards a thickening of the arterial walls produced by a form of arteritis in the fetal part of the placenta as characteristic of syphilis. After thickening, the vascular walls degenerate, and the vessels become obstructed. Rokitsky⁷⁶ associated some of the fibrinous deposits found in the placenta with the syphilitic taint. These had a syphilitic aspect on section, whatever that may mean. Lebert⁷⁷ before him had observed yellow granulations having the same structure as tubercles, but he did not regard them as of special importance.

Gusserow and Klebs⁷⁸ observed a morbid hyperplasia of the decidual cells in a case where the father was syphilitic, but Straussman⁷⁹ and Madame Kaschewarowa have since shown the same changes in the placenta where there was no suspicion of syphilis. Virchow,⁸⁰ who always brings a certain originality into his work, made some observations on syphilitic placenta. He points out that in studying this subject we must remember the envelopes consist of two portions, the fetal and the maternal. He then proceeds to point out that syphilis may produce two forms of endometritis during pregnancy, the one attacking that part of the decidua which enters into the formation of the placenta, the other the parietal decidua, or vera, which is away from the placental spot. The first form produces thickening and fibrous indurations which may cause atrophy of the villi. In the second form the most marked effects were shown upon the decidua vera. It was thickened and covered on its uterine side with growths almost polypoid, composed of very vascular proliferating mucous tissue, with no trace of fatty degeneration. Virchow considers this change is analogous to mucous papulae, or flat condylomata, observed elsewhere on mucous membranes in connection with syphilis.

Straussman has thrown some doubt on the supposed specific character of this lesion by finding it in the case of a mother who had not the least symptom of venereal disease. Henning regards these proliferations as simple angiomas. Virchow's observations on this subject seem to me to refer rather to the effects of syphilis on the ovular membranes in early pregnancy than to the full-grown placenta. Fränkel⁸¹ is the author whose researches in later days have received most attention in Germany and France, and they bear the impress of great care. Fränkel's conclusions, which he says are founded on the examination of over one hundred placenta, point distinctly in favour of there being a syphilitic placenta, which may be recognised by certain definite characteristics. The placenta mostly belonged to syphilitic mature, or immature children, and he took the indications pointed out by Wagner, of Berlin,⁸² as peculiar to syphilitic foetuses, to be unfailling evidence that syphilis was present. Wagner has pointed out that in all syphilitic children there is a peculiar band of tissue between the shaft of the bone and the cartilage of the epiphysis, which is in a state of inflammatory irritation. The microscope, as well as the naked eye, shows that the changes thus produced are unlike any other changes, and consequently are specific. Taking this test as a basis, Fränkel states that the evidences of placental syphilis vary according to whether the syphilitic virus is derived from the mother or the father. If from the male parent, the villi constituting the fetal part of the placenta are chiefly or primarily affected, but the disease may extend thence to the maternal part. The villi undergo a

peculiar cellular hypertrophy, with thickening and proliferation of their epithelial covering, which alters and distorts their form in a remarkable manner. There is a particularly marked hypertrophy in the coats of the vessels, with consecutive obliteration of their canals.

Fig. 12.—Villi of placenta, affected with "disfiguring granulation cell disease." (Fränkel.) a, Portion of villus covered with granulation cells; b, normal villus deprived of its epithelial covering by maceration; c, junction between sound and diseased structure.

This morbid change Fränkel calls by the awkward name of "disfiguring granulation cell disease." The placenta in their entirety were, in the early stage of the disease, unusually large, notwithstanding that in most cases the foetuses were atrophied and shrivelled. After the hypertrophy, came one of atrophy, in which the vessels were completely obstructed, the epithelial covering disintegrated, and fatty degeneration in full progress. Fränkel insists strongly on one point, namely, that although fatty degeneration of the villi, blood extravasations, etc., may be present in placental syphilis, they are secondary, the primary disturbance being increase in the normal cells of the villi, a revolutionary growth brought about by diseased blood.

Fig. 13. Section of villi affected with "disfiguring granulation cell disease." aa, Luxuriant cell-development in the interior; vv, lumen of blood-vessels with hypertrophied walls; b, villus in which only a trace of blood-vessels can be seen at ss; c, villus without trace of vascular canal; b, epithelial covering. (Fränkel.)

When the mother was the subject of syphilis the pathological lesion was more complex, and the changes were especially marked in the decidual or maternal part of the placenta, but also might extend by contiguity to the villi or foetal portion. The changes in the decidua took the form of disease described by Virchow as placental endometritis, or by Slavjansky⁸³ and Kleinwächter⁸⁴ as "placentaris gummosa." These consisted of increased growth of the connective tissue, and enormous hypertrophy of the large decidual cells, these so crowding and compressing the villi that their circulation was interrupted, and the result was interference with the nutrition and placental respiration of the foetus. I believe Henning was the first to call attention to the intimate relation of the cell-growth to the vessels in syphilitic placenta, and his observations are confirmed by Fränkel. If both father and mother were syphilitic, the lesion was of course a mixed one, and in all there were variations, consisting of changes of

⁷⁵ Thèse, 1880.

⁷⁶ Lehrbuch der Path. Anat.

⁷⁷ Comptes Rendus, 1852.

⁷⁸ Virchow's Archiv., Bd. xxvii.

⁷⁹ Verhandl. d. Berlin. Geburtsh., 1863.

⁸⁰ Path. der Geschw., Bd. ii.

⁸¹ Arch. für Gynäkol., 1873.

⁸² Virchow's Archiv., Bd. i, p. 305.

⁸³ Prager Vierteljahrs., 1871.

⁸⁴ Ibid., Bd. 114.

colour, dark hyperæmic patches, alternating with paler structure, and frequently smaller or larger blood extravasations.

The late Dr. Angus Macdonald,⁸⁵ of Edinburgh, wrote a paper confirming these observations of Fränkel, and asserted that although the syphilitic placenta may be mistaken for a fatty placenta, and, in fact, had been so mistaken by Kilian and Robin, this is easily rectified by microscopic examination. In these cases the death of the foetus is the result of progressively increasing defective blood-supply, owing to the changes described.

I have on different occasions had the opportunity of examining placenta in the early months of gestation with certain peculiarities of morbid change which I considered were undoubtedly due to syphilis, and which bore some resemblance to Fränkel's descriptions. For example, I examined the placenta in two separate pregnancies of the same individual. She had contracted syphilis immediately after her marriage, and at the same time became pregnant. During the early part of her pregnancy the vulva became the seat of specific sores, mucous tubercles, and warts. Later her body was covered with patches of syphilitic psoriasis. She miscarried in both the first and second pregnancies in the fifth month. Both the placenta exhibited changes very closely resembling each other. On the uterine side of the placenta the decidua was much thickened, and there was great increase of all the cellular and fibroid structures which normally constitute that membrane. Prolongations of this dense tissue were sent down into the substance of the placenta, and seemed to terminate about half way through its thickness. The effect of this dense layer was apparently to compress and contract the sinuses or lacunæ, to do away with the spongy character of the organ, and so to prevent the growth of the villi into the sinuses. The villi next to this layer were stunted and atrophied. Those near the foetal surface were hypertrophied, and were beginning to undergo fibroid or fatty degeneration or both. In all the microscopic sections, hypertrophy of the arterial coats in the foetal blood-vessels was noted, and those nearest the maternal surface were absolutely obliterated. Here was a case in which syphilis in the mother was clear and distinct, and where the changes in the maternal portion of the placenta were most marked, supporting the doctrine that it is this portion which is most affected when the mother is syphilitic in early pregnancy.

So far as my observation goes, I do not think we are yet able to say with precision that any one specific lesion of the placenta belongs alone to syphilis, although some morbid appearances are more constant than others in connection with syphilis, as, for example, the changes described by Fränkel. In addition to the hypertrophy of the villi described by Fränkel, and the morbid changes in the decidua just described, I have seen fibroid deposits, such as those described by Rokitsky, some unchanged, others undergoing fatty transformation. Again, I have seen the yellowish granulations of varying sizes looking like tubercles, as observed by Lebert, but I have also seen most, if not all, of these pathological appearances where no syphilitic history could be traced. The nearest approach to precision in this respect is to say, as a general rule, when the decidua or maternal portion of the placenta has become so far changed by hyperplasia as to arrest the utero-placental circulation and the full development of the placental villi, that this is probably due to maternal syphilis. It finds its analogy in the changes which take place in the mucous membrane in the uterus and elsewhere when the blood is undoubtedly poisoned by syphilis, and also in the thickening of the decidua during the early pregnancy of syphilitic women, which has been described by Virchow and Dohrn as "endometritis papulosa et tuberosa." When fibrinous and pseudo-tubercular deposits are found in the placenta in connection with syphilis, they are probably only the expression of a depraved or impoverished condition of the blood, which may be equally associated with anæmia or with some form of dyscrasia. When there is marked hypertrophy and degeneration of the villi, the maternal portion of the placenta being less affected, the syphilitic taint more probably comes from the male parent, and the mother may show no signs of the disease. Both Depaul and Tarnier, among modern observers, dispute the existence of absolutely specific lesions in the placenta as the result of syphilis.

De Sinéty, one of the latest writers quoted by Charpentier, did not find placental lesions in all women affected with syphilis, but where lesions were present he was able to demonstrate three important points: (1) Hypertrophy of the placental villi; (2) fibrous degeneration of them; (3) islands of granulation belonging to the caseous form of degeneration. This coincidence of the fibrous and caseous forms of degeneration is found in syphilitic gumma, notably in gummata of the liver. De Sinéty had not found this combined degeneration except in syphilis. He does not know if any other disease may produce the

combined changes, and does not decide the question as to a specific placental lesion in connection with syphilis.

In reference to all diseases which affect the placenta, it is to be noted that the effect on the life of the child bears a direct relation to the amount of damage done to its tissues, and impairing its double function as an organ for respiration and absorption. In cases of separation of the placenta, a portion still adherent to the uterine walls may be enough to sustain the life of the child, for a time at least; and, in like manner, when the placenta has become diseased, if some portions of it only remain sound, vitality may still be maintained in the body of the foetus. If the morbid process be slow and chronic there will probably be progressive emaciation, and if the child be born alive it will have all the appearances of being starved during its development. If the placental disease is more acute and rapid, and affecting a large area of tissue, the child's movements become at first more restless than usual, and then become less marked and distinct as they subside into absolute quiescence. With the aid of the stethoscope the beats of the foetal heart have repeatedly been noted in cases of suspected placental disease to become slower and slower, and thus to furnish important indications for the induction of premature labour.

Among the further causes of foetal death *in utero* which I cannot here overtake are the pathological conditions of the umbilical cord, *ante-partum* hæmorrhage, extra-uterine gestation, and allied conditions, the malformations and diseases of the unborn child—the most fertile cause of the latter probably being syphilis.

Did time permit I might say something besides, concerning the changes taking place in the body of the foetus when it is long retained in the uterus after its death; of the inferences to be drawn as to the cause of its death from the appearances it presents after longer or shorter periods of retention, and other kindred topics.

I must, however, hasten to say a few words on the subject of preventive treatment.

Treatment.—It may be asked, what are the remedies suggested for these many and varying causes of intra-uterine death? The answer must, I fear, in regard to a large number of them, be considered as eminently unsatisfactory. Notwithstanding all that has been done, we are yet only on the threshold of those investigations which must reveal to us eventually the best methods of obviating death in unborn children. In the meantime, so far as the prevention of some of the forms of intra-uterine death is concerned, we are absolutely in the dark, and the therapeutics of the subject are still as a closed book.

Fortunately this need not be said of all. A careful study of the several pathological conditions in the parents, combined with the local expression of the results of those conditions, enables us in some cases to formulate methods of treatment and lay down rules for guidance which in practice have been attended with happy results.

Whenever, therefore, a woman has once or more frequently lost the product of conception at an early or later period, careful inquiry should be made into the health of both parents, and any previous history of illness should be accurately scrutinised. If the constitution of either parent be found at fault, measures must be taken to amend this. The question of syphilis is so important, and the venereal poison is so persistent and all-pervading, that no pains must be spared to ascertain whether this is the root of the misadventures. If a patient has once had syphilis it is impossible to say when the effects on the constitution have entirely passed away, and many men who have believed themselves to be cured and free from every taint of the disease find the evidence still remaining in their wives' frequent abortions, or in the indications of syphilis in their living children. When, therefore, either patient has suffered from syphilis in the near or distant past, and the wife has recurring abortion, or has her children die *in utero* at a later period of gestation, the presumption is that syphilis is the cause. Both parents ought at once to be put under anti-syphilitic treatment before a fresh conception is permitted, and this ought to be sufficiently prolonged to give it a fair chance of producing satisfactory results. Diday recommends that the physician should not be contented with submitting both parents to anti-syphilitic treatment prior to the occurrence of pregnancy, but that with each successive pregnancy, as it occurs, the mother should at once recommence a mercurial course, even if she has no visible indications of the disease. I can testify that I have repeatedly seen good effects from small doses of bichloride of mercury, with bark, given during the first three months of gestation, when there has been no opportunity of commencing the treatment before conception began; and under this method, with a careful diet and regimen, women who had repeatedly miscarried before went to the full time.

Similar favourable results have been observed to follow the administration of iodide of potassium. M. Goshkevich, quoted in the JOURNAL (July 1st, 1885) advises its use in habitual abortion, even

⁸⁵ *Obstetric Journal*, 1875-6.

when no syphilitic symptoms have been recognised, and testifies to its utility in saving fetal life. It must be left for the medical man in charge of a case to determine whether in any given case some of the approved forms of mercurial treatment are most appropriate, or a course of iodide of potassium. Possibly both may be judiciously combined, or one course succeed the other. Depaul has such confidence in mercurial treatment that he urges it should be adopted in all cases of recurring abortion, even when no syphilitic history is traceable in either parent. Charpentier, by way of enforcing this precept, records a case in which a woman had four consecutive abortions without known cause, and in which both parents not only denied any knowledge of syphilis, but no traces of the disease could be found upon them. The two parents were then subjected to an anti-syphilitic course, and two children were born subsequently quite healthy, and with no trace of disease upon them.

I may mention, for what it is worth, the case of a patient who had aborted in five successive pregnancies. In the sixth she was salivated by accident in the third month. On this occasion she went her full time, and was delivered of a living child.

In attempting to form a just estimate of the effects of mercurial treatment in syphilitic cases, it is but fair to say that the venereal poison has a natural tendency to attenuate itself and wear out in such way, that, apart from treatment, pregnancy may at length be continued to the normal term in women who have repeatedly miscarried from this cause before. In these cases each successive pregnancy may have longer duration, until eventually a living child is born. The value of mercurial treatment is nevertheless almost universally acknowledged. So far as my reading goes, there is only one notable dissentient to its utility. Desprès, a French author, looks upon mercury as the cause of all the evils which it is expected to remedy, and to be itself a cause of abortion. It is probable that some of the forms of mercury (the bichloride, for example) in very small doses over a continued period, and so regulated as not to produce pyalism, have a beneficial effect in other than syphilitic cases, for more than one reason. The treatment certainly has a good effect on some constitutions. It restores the secretions, regulates the bowels, and, if combined with bark, improves the appetite and aids nutrition. I have frequently seen patients fatten while taking it. Further, it is an acknowledged remedy for certain forms of uterine hypertrophy, and more especially acts on the mucous membranes. In those instances, therefore, where the uterine walls are thickened, and their elasticity impaired—in those cases where chronic endometritis so hinders the formative power of the uterine mucous membrane that a faulty or diseased decidua is produced—an alternative mercurial course may best fulfil the indication of treatment demanded, and indirectly hinder the repetition of abortion. If inquiry shows that the health of either parent is disordered or deranged from some other cause than syphilis, care must be taken to trace out the nature of the deviation from health, and so to define it that treatment fulfils its purpose. It cannot be too much insisted upon that not the mother only, but the father also must be put under supervision. If there is any constitutional peculiarity or diathesis, it must be met by appropriate means—the strumous diathesis by tonics and cod-liver oil, with such improved climatic conditions as may be feasible; the gouty rheumatic by limitations of diet, careful regimen, and alkaline medicines. The gouty rheumatic constitution is often associated with congestion of the portal circulation, and this may become so important a factor in bringing on abortion that it deserves special mention. The late Dr. Edward Rigby constantly dwelt on the importance of keeping the bowels unloaded by aperients in women liable to abortion, and he preferred saline aperients to aloes or other drastic purgatives, as easier in action and less likely to be injurious during pregnancy. The effect of carefully regulating the bowels is to lessen the tendency to congestion in the pelvic organs, and so aid in averting engorgement of the fetal membranes and placenta. Care must be taken to choose such laxatives as are not likely to stimulate undue action of the bowels or straining, else the medicines may stir up the very mischief they are given to avoid. Compounds of sulphur, like the German compound liquorice powder, the confection of senna, and salient aperients, seem best to fulfil the needful indications. Venesection has been recommended in cases of general plethora, and local depletion where local congestion seemed to threaten abortion from the pain and sense of engorgement which it entailed. General bloodletting is now rarely employed in these instances. Its advantages are doubtful, and its employment may lead to harm instead of good. Where fainting is produced in the mother, the embryo may be destroyed and abortion follow. Local depletion by leeches is less objectionable, and leeches applied round the anus, as the best point for abstracting blood, will readily relieve tension in the hæmorrhoidal vessels. They may require

repetition two or three times during the early months of gestation, if indications of congestion recur, and as a rule they are of most use when applied just before the time which corresponds to the catamenial period.

The constitutional condition which, next to syphilis, seems to hold the most prominent place as predisposing to abortion, is anæmia, and for this it is plain a plan of treatment is called for the very opposite to a depleting one. Preparations of iron must be administered in some form least likely to disturb the digestive organs of the patient; and these should be given not only antecedent to the occurrence of conception, but they should be continued with such modifications and in such combinations as may be suitable during the progress of pregnancy. It has been thought by some that iron is counter-indicated when gestation has once commenced, as it might favour the occurrence of abortion by inducing hæmorrhage. As a general rule there is no ground for such supposition, and iron, if cautiously given, may be taken by anæmic patients throughout pregnancy without harm. There is often the greatest tolerance of it where it is most urgently needed. Cazeau remarks that in ordinary pregnancy a state of chlorosis is produced—there is a relative anæmia, for there is a diminution of red globules, with increase of fibrin in the blood. A slight increase, therefore, of this anæmia may have the effect of deranging the progress of gestation by lessening the nutrition of the fetus and predisposing to fibrinous deposits in the placenta or membranes.

It is probable that some of the forms of degeneration found in the fetal membranes and placenta are due to defects in the constitution of the blood in one or both parents. Whether the fault lies with the male or the female parent may not be easily determined. If there is obvious anæmia or deterioration of health in one or other, it may be enough to place the one apparently deranged under treatment; but this indication not being forthcoming, it will be desirable to put both parents on iron, combined, perhaps, with quinine, arsenic, or other tonic remedies, as may seem suitable in each particular case.

Where local conditions have been ascertained or suspected to be the cause of repeated abortion, or of later fetal death, the treatment must be directed in accordance with the special requirements of the case. Especial care should be taken to remove as far as possible all indications of endometritis prior to the commencement of pregnancy, as an unhealthy condition of the lining membrane of the uterus is regarded by most authorities as a potent cause of disease in the fetal membranes and placenta.

In all instances where past experience has proved the proneness to miscarriage, it is an essential part of any plan of treatment that the patient should be habitually at rest in the recumbent position, so that the uterus may be affected as little as possible by the effects of gravitation. It is doubtful in most cases whether the patient should be entirely precluded from taking exercise and fresh air, lest the general health should so suffer as to undo some of the advantages of rest. This point deserves especial consideration, so far as anæmic and naturally bilious patients are concerned, both suffering if deprived of fresh air and exercise. A moderate amount of walking out of doors may, therefore, be permitted, with repose in the recumbent posture at other times. All carriage exercise and other forms of locomotion likely to jar the body should be sedulously avoided, and a carrying chair may be advantageously used to take the patient up and down stairs.

While this latitude of movement is permitted ordinarily, absolute rest on the sofa or in bed should be enjoined at the times which correspond to the days of the catamenial period. A greater tendency to abort has been noted at those times than at others, and, if abortion does not immediately take place then, damage is done, which culminates in miscarriage later, and is traced back to the menstrual epoch for its first symptoms. At those times, even during pregnancy, there is a nœvus often distinctly observable in increased discomfort, or in some other way. It appears like a periodical tidal wave, which, in weakly women or those liable to go wrong, readily overflows its barriers, and ends in abortion. The influence of this menstrual wave is so fully acknowledged at all times in women that, by common consent, operations are avoided during its continuance, by way of avoiding the time of local hyperæmia with which it is accompanied. Charpentier remarks that, in certain women who abort frequently without known cause, there seems to be a peculiar irritability of the uterine fibre. The sphincter uteri seems weakened, and, when pregnancy occurs, the smallest effort promotes its dilatation. Irritability also determines premature contractions, and the sphincter being weak it easily yields, and so abortion occurs. It is obvious that in these cases, whatever else is prescribed, absolute rest in the recumbent posture is imperative so long as the irritability remains.

To Sir James Simpson we owe the suggestion that, in all cases where there is reason to believe the placenta is partially disabled by

disease, chlorate of potash should be given, with the object of keeping the child alive during its further detention *in utero*. Simpson's theory was that, because chlorate of potash contained six atoms of oxygen to one of chlorine and one of potassium, it would sensibly increase the amount of oxygen in the mother's blood, and afford a larger proportion of this element for the placental respiration of the fetus. The maternal blood-current being thus more abundantly charged with oxygen than under ordinary circumstances, a smaller portion of sound placenta could absorb more for the requirements of the fetus, and so supplement the function of those parts of the organ damaged or destroyed. Whether this theory be true or not, it is certain that many practitioners have testified to its utility as well as to its harmlessness. Dr. Playfair suggests that it may act, in virtue of its tonic properties, on the constitution of the mother; and I think it may act usefully as an alkaline salt in preventing the formation of coagula and fibrous deposits in the placenta. It is given in ten or fifteen grain doses three times a day, and may be administered at any period of pregnancy, but it is believed to be most useful in the latter half.

The induction of premature labour, also recommended by Simpson as a means of averting the death of the child *in utero*, is only available in those cases where it dies in successive pregnancies after the expiration of the seventh month. At any period antecedent to this the child would not be viable, or would be so feeble that nothing would be gained by the proceeding. But in a limited number of instances it has been observed that placental disease begins apparently just before the seventh month, and gradually and steadily advances until it kills the child and its movements cease to be felt by the mother. With such experience of past times to guide the practitioner, the induction of labour is perfectly justifiable if the movements of the child continue to be felt after the seventh month is completed; and the operation is the more imperative if the movements grow slower and feebler, and the stethoscope indicates a declension of strength in the beating of the fetal heart. In this way children's lives have been saved which would probably have been sacrificed by further delay.

In patients in whom intra-uterine death is threatened or feared as the result of some form of poison, inorganic or organic, to which I have alluded, the treatment must necessarily be prophylactic, and consists in the removal of the cause. In those special instances where the progress of zymotic disease or of inflammation in some organ of the patient's body is attended with high temperature, it is possible some attempt may be made to avert the usual consequences so far as the pregnancy is concerned. As the experiments of Runge and others have shown that peril comes to the child *in utero* directly the temperature of the mother rises persistently up to 105°, while the infant's temperature rises still higher, it may be possible, in the progress of science, not only to keep down the general maternal hyperpyrexia, but I would throw out the suggestion that, where it is feasible, an attempt should be made to lower also the temperature of the uterus and of the foetus, either by the application of ice-bags to the maternal abdomen, or of those tubular appliances so ingeniously invented for the application of cold, and which may be modified to fit any part of the body.

When all our most ingenious methods of diagnosis and treatment are exhausted in attempting to obviate intra-uterine death, there will still be a large number of cases in which no further clue can be found than an obvious constitutional feebleness, permanent or temporary, in one or both parents. In some of these the organic weakness or degeneration in health may be of such character that it is hopeless to expect a remedy, and perhaps it is well for the race in general that such constitutional faults or diseases should not be perpetuated in progeny. But where the constitutional weakness may be but temporary, attempts should be made to resuscitate the health. Everything that science and practice teach to be useful for the restoration of strength in single organs and in the entire system should be called into requisition.

Boerhaave is said to have recommended horse-riding as a remedy for abortion. In any case where it proved useful it would probably be by improving the general health. Apropos of this I may mention a case within my own experience, of a lady in India who, after aborting several times under the most careful system of rest—in some pregnancies amounting to absolute repose for many months in the recumbent posture—at length gave up the hope of bearing children, and took to riding on horseback, not only before a fresh gestation began, but after she had reason to believe that she was again pregnant. As the result she went for the first time to the full period, and was delivered of a living child.

Some of the baths and waters on the Continent have a high reputation for their tonic properties and their favourable influence on pregnancy. Aix-les-Bains is said to be useful in these cases. I have seen

courses at Schwalbach and Kissingen followed by happy results. Schwalbach, perhaps, is more appropriate for patients who are more or less anæmic; Kissingen for those in whom the digestion and portal system are at fault. One successful case is fresh in my memory where a mother who had previously borne healthy children was stricken with severe pleuro-pneumonia, and continued for some time after in comparatively feeble health, although she resumed her place in society again after an interval. Not long afterwards she conceived, and at the end of six months lost her child. A little later a fresh pregnancy occurred, and this terminated, apparently without cause, at the end of four months. After this she went to Kissingen, experienced a notable improvement in health, and subsequently bore two healthy children without misadventure.

My task is now ended, however imperfectly completed, and I thank those who have listened to me so patiently. In giving these lectures, I have had a sense as of rushing rapidly through a plentiful harvest-field; plucking, it may be, a few scattered ears of grain by the way, but leaving an abundant crop to be garnered by those reapers who may come after me, and who will patiently and slowly go over the same ground.

REMARKS ON "ANALGESICS."

BY JOHN KENT SPENDER, M.D. LOND.
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It is becoming, first, to acknowledge the honour of being asked to open a discussion on "Analgesics" in the Section of Therapeutics and Pharmacology. There are three ways, so it seems to me, in which this duty may be done: (a), I might offer you a compilation of what everyone in this room knew before, from dictionaries, encyclopædias, and text-books, some of which my learned friends now present may have written themselves; (b), or I might frame a didactic sketch, like a master to pupils, a teacher to disciples, or a preacher to a congregation; (c), but there is one other way, which I venture to call the best way, which consists in taking up a few points from a vast subject, and comparing notes with my brethren upon them; bringing the experience of one man to strengthen the experience of another, gathering illustrations from many sources, and garnering the knowledge harvested from all quarters so that it may illuminate the solemn and vital issue which we medical men are expected to grasp and control.

There is a charm of language in what Sir William Hamilton says when he tells us that "pleasure is the result of certain harmonious relations, of certain agreements; pain, on the contrary, is the effect of certain inharmonious relations, of certain disagreements." But Dr. Latham comes more truly home to us when he declares that "things which all men know infallibly by their own perceptive experience cannot be made plainer by words. Therefore, let pain be spoken of simply as pain." So far as the object of this paper is concerned, it is simply a waste of time to speculate upon the pathogeny of pain, or to try and define it with the precision of an axiom. And yet there lurks in every Englishman's mind a fervid desire to know the shape and the look of the enemy he has to battle with. Whenever pain is a sentinel and a beacon to warn us that something in the body is going wrong, it would be as absurd to abolish that pain as to put out the light of a lighthouse, or to obliterate the letters on a sign post; worse than absurd, it would be almost wicked. The faculty of the perception of pain, when needed as a safeguard against bodily peril, is to be cherished rather than destroyed. And it ought always to be distinguished from that disturbance of common sensibility which is purposeless, and therefore aggressive and disorganising.

A curious paradox, then, we have to face at the very opening of our debate—pain may save and pain may kill. The former comes within our scope now only as it may be out of proportion to the object which it serves; the latter claims our eager and quick regard, importunes us with shocks and cries, and will not be put out of court by the bland and trimmed phrases, "You will be better by-and-bye," "We will try and do you good," or by that most jejune formula, "The weather is against you." But, at the outset of our therapeutic business, we shall be met by the warning note which

¹ Read in the Section of Therapeutics and Pharmacology, at the Annual Meeting of the British Medical Association held at Brighton, August 10th-18th, 1886.