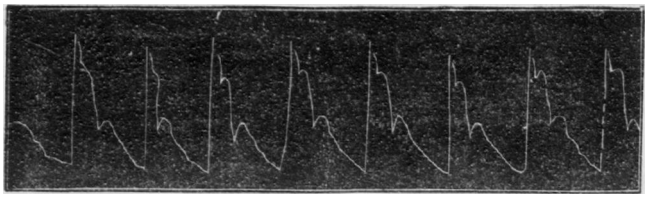
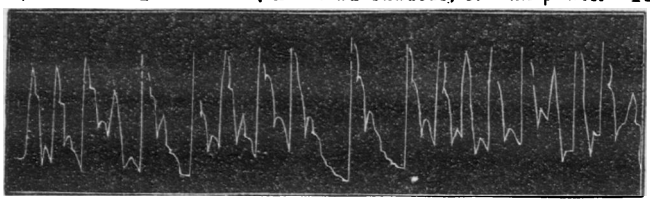
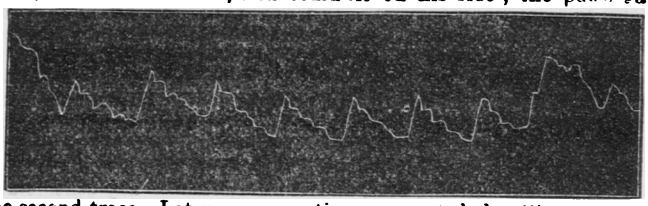
case in which acute dilatation with hæmoptysis had occurred from over-exertion; a mitral systolic murmur remained; the artery was not large.



In the case from which the second was taken there were breathlessness, difficulty of lying down, and enlargement of the liver, all of which were relieved by treatment. The patient whose pulse is represented by the third, was liable to stagger and even fall, on rising from a semi-recumbent position, with momentary loss of consciousness, and, on slight exertion, he would suddenly lose all power. The



artery was always full between the beats, notwithstanding the appearance of the trace. He improved under treatment, could walk eight miles, was more cheerful, and could lie on his side; the pulse gave



the second trace. Later, over-exertion aggravated the dilatation, and thrombosis of the left middle cerebral artery occurred, the case ending fatally.

THE LUMLEIAN LECTURES

ON

THE PATHOLOGY OF INTRA-UTERINE DEATH.

Delivered at the Royal College of Physicians of London, March, 1887.

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Lecture II.—Diseases of Fœtal Appendages.

I proceed now to speak of the causes of intra-uterine death which are associated with faulty conditions of the fœtal envelopes and fœtal appendages, and I shall take those first which properly belong to the earlier part of pregnancy.

In the earlier stages of its development, the ovum is in shape like an egg, and consists of the central embryo, with its surrounding envelopes. The outer one, or decidua, which lies next to the uterine wall, is the earliest in its formation, for it appears in the uterine cavity before the descent of the fecundated ovule from the Fallopian tube. It is now well known to be the product of the uterus itself, and to consist of the mucous membrane lining the interior of the womb, thickened and modified in such a way as to fulfil the necessary requirements of a fœtal envelope. A membrane in all respects like this, both in external appearance and minute structure, is sometimes expelled from the unimpregnated uterus at the catamenial periods in cases of dysmenorrhœa. It is then produced by an over-activity of formative elements, which occurs as the result of some reflex irritation, probably in the ovary, and which simulates the commencement of pregnancy. The expulsion of such membranes, therefore, does not always mean that an abortion has taken place, for they may be formed and extruded without impregnation; but, in a considerable number of cases in which these membranes are thrown off by married women, they are the result of conception. This is inferred from their frequent recurrence so long as sexual relations are continued, and their cessation when coitus is intermitted. Besides, the catamenial period is often missed once, at least, before expulsion, in women who at other times are perfectly regular. When entire, these membranes generally appear as membranous sacs, of triangular form, corresponding to the shape of the uterine cavity, and the walls of the sac are thick or thin according to the amount of organised material of which they are composed. The outer surface is commonly rough, and, if the preparation is placed in water, shreds of flocculi float out in the fluid. When laid open, it is a common thing to find the cavity quite empty. The inner surface is smooth, and marked everywhere with the apertures of hypertrophied glandular follicles, but there is no embryo. The ovule has either missed getting into the decidual cavity, or it has been endowed with so small an amount of vitality that it has undergone solution before it could plant itself in the decidual tissue. The reflex stimulus which has come from the ovary or tube, in whichever locality fecundation took place, has been enough to evoke the formation of the decidua in the womb; but the ovule being wanting, development is arrested, no decidua reflexa is formed, and the useless decidua vera is eventually thrown off as a foreign body. Here the fault in procreating power, as distinguished from generative power, may be either with the male or female; and, if the woman is otherwise healthy, it is quite as likely to be some defect in the fertilising fluid as faulty ovulation on the part of the female. These deciduæ of early pregnancy are in many cases found covered with coagulated blood, and the cavity may be filled with clot, soft or semi-fluid, if recently deposited there—firm and partially decolorised, if some time has elapsed since its extravasation. But these membranes are not always expelled in their entirety. More frequently they are thrown off in detached portions or shreds at variable intervals, and mixed with the fluid or coagulated blood which escapes at the same time. It is no uncommon occurrence for a patient, or even the medical man, to infer that the supposition of pregnancy has been erroneous because no substantial mass has been expelled—the discharges being taken for an over-profuse and perhaps deferred period. Eigenbrodt and Hegar,[1] in a memoir "On Apoplectic Destruction of the Decidua," found that in some cases the presence of the elements of the decidua could only be ascertained by the aid of the microscope. This accords with my own observation.

Akin to this is another defect of development, which has been described by Matthews Duncan[2] and copied into the various text-books. This consists, according to the author named, in a defective development of the decidua round the ovum. The egg then comes in contact only with a limited surface of the mucous membranes which corresponds to the serotina, and as it is not supported, as usual, by growth of the decidua round it, it becomes, as it were, pediculated, and is easily detached from its limited adhesion. This is probably one of many faults of development occurring in early gestation. In one specimen I examined, which had been expelled at an abortion, the ovule had evidently failed to get inside the decidual cavity, and began its development on its outer rough surface, attaching itself, as it does in extra-uterine pregnancy, to the surrounding structures. The surrounding structures were partly the outer surface of the decidua, and partly the wall of the uterus, and for three-fourths of the circumference of the ovum the clear transparent chorion was quite uncovered, its villi there being either atrophied or never developed. The decidua seems in some instances to be detached from the uterine wall, and to lie so loosely in the uterine cavity, more especially if atrophied or imperfectly developed, that one can imagine the ovule as it enters the uterus from the Fallopian tube dropping between the decidua and the uterine wall.

We get some sort of idea, now that our knowledge is more precise concerning the formation of the decidua, and of the behaviour of the ovule when it enters the uterine cavity, of the way in which placenta prævia and other variations in the site of the placenta are produced. Ordinarily the thickened decidua fills pretty fully the cavity of the uterus, and the ovule is received into the cushion so formed, and is sustained in the upper part of the uterus. When, however, the decidua is less luxuriant in growth, its cavity will be larger, and the fertilised ovule may then drop down to the lowest part of its cavity, and become implanted there. Dr. Robert Lee[3] has recorded the appearances of a very early placenta prævia, which bears out this idea, and such an anatomical fault favours the occurrence of abortion, although we know by experience that those cases may go on to the later periods of pregnancy.

[1] *Monatsschr. für Geburts*, Bd. xx, 1863.
[2] *Researches in Obstet.*
[3] *Theory and Practice of Midwifery.*

The varieties of insertion of the ovum into the decidua, and the several forms of hypertrophy and atrophy, with other anomalies of the same membrane, have been elaborately studied by Kussmaul,[4] Hegar,[5] and Dohrn,[6] in special and excellent memoirs, and are well worth perusal. It seems obvious that a too scanty formation of the decidua may lead to the growth of a placenta too small for the needs of the embryo. Charpentier believes that an arrest of development in the decidua reflexa, or its premature destruction, are frequent causes of death in the embryo. The egg then is covered in a large part of its extent only by the chorion, and may be suspended by a sort of pedicle from the decidua serotina. It has thus a very limited attachment to the decidua, and is easily disturbed. Probably, indeed, pregnancy cannot go on with so slight an attachment, and it is a variety of a similar kind which Rokitansky[7] has called "Cervical Pregnancy."

When the ovule has been successfully implanted in the decidua, and the normal development of the decidua reflexa and other structures is going on in progressive series, there is great tendency in some women to go wrong, apparently from mere weakness of the outer structures which form the ovum. The decidua is commonly composed of so lax a tissue, and is so abundantly supplied with blood-vessels undergoing various modifications of size and distribution, that during early pregnancy it is prone to suffer from extravasations of blood into its parenchymatous substance, more especially in delicate women. It is true that the uterus is so suspended in the maternal pelvis as to be affected in the least possible degree by ordinary locomotion and by accidental concussions; yet, in some women, the union between the pregnant uterus and the decidua is so unstable, that a fall or stumble, or a shaking of any kind, may be quite sufficient to detach a portion of the latter, rupture the intervening vessels, and cause extravasation of blood. Small and circumscribed clots produced in this way are frequently found between the uterus and decidua; at other times they are in the meshes of the decidua itself, or both may be conjoined. If limited, these extravasations need not interrupt the continuance of pregnancy. If more extensive, and separating a larger portion of the decidua, they necessarily interfere with the nutrition of the ovum, produce death of the embryo, and precipitate abortion. When the escape of blood from the vessels is confined to a limited space at the upper part of the uterus, it may cause uneasiness, but no external hæmorrhage will be noticed. If it takes place near the cervix, the blood more readily finds exit from the uterus, and is discharged by the vagina, thus becoming a manifest symptom of threatened abortion. Blood-clots in every stage of transition, and every variety of firmness and colour, may be observed in some aborted ova. The latest are deep red or purple, and those older pass through the several tints of chocolate brown to yellow, like apoplectic clots elsewhere.

The same result may ensue from contractions of the uterus, either provoked by local irritation directly applied, or from reflex causes, as, for example, suckling a child after a fresh pregnancy has commenced. It is well known that abortion is frequent if a new pregnancy begins during the time that a mother is suckling her child.

Occasionally, during the very early stages of gestation, blood flows directly into the decidual cavity, and fills it with coagula. The result is either that all traces of the embryo and its immediate surrounding structures are obliterated, or the embryo is compressed and destroyed by the invading flood, rudimentary portions only of it being discovered. More than once, under these conditions, I have found the embryo, wrapped in its small and budding chorion, detached from the decidua and floating in the semi-fluid blood contained in the decidual cavity. The decidua reflexa had not yet been sufficiently developed around it to give it a stable attachment. The small aborted ova of this early period are frequently thrown off entire in triangular or ovoid form, the outer layer being the decidua, which encloses, first, firm layers of fibrin, and, as one proceeds to the centre, soft clot.

In the second to the third months, the decidua reflexa or ovuli is fully formed, and the villi of the chorion are everywhere imbedded in its substance. Then another form of hæmorrhage may occur. In this, the blood is not alone extravasated between the decidua vera and the uterine wall, or into the meshes of the decidua itself, but between the decidua reflexa and serotina, and the chorion or outermost of the true fœtal membranes. In the process of development the villi of the chorion are pushed into the decidual tissue, and are soon surrounded by the blood-vessels which are to form the maternal placenta. The decidua is then a highly vascular membrane, especially at the site of the future placenta, and the maternal vessels which everywhere ramify through it undergo a development which in the human body is unique. Appearing first as capillaries, they rapidly enlarge, and eventually become sinuses which are filled with maternal blood, and in which the fœtal villi are eventually suspended.

It can scarcely excite surprise that these delicate and rapidly dilating maternal vessels should be very liable to rupture. Hæmorrhage taking place in this locality, floods the loose tissue of which the decidua reflexa is composed, and destroys or compresses the chorion villi implanted there. It may be so extensive as entirely to surround the embryo. At the earlier periods, when there is a considerable space between the decidua uteri and reflexa, it may be limited to the circumference of the latter membrane, which, when cut into, is found to contain clot with or without traces of the chorion and embryo. Later, when the decidua reflexa is in apposition with the decidua uteri throughout its whole extent, and the amnion is formed, the cavity, if exposed from within, exhibits eminences or projections from the walls, which may seem like projecting cysts if the blood is imperfectly coagulated; but they represent hard and firm bosses or nodules, when the blood has been long enough deposited to become solid. The force of the extravasation has in fact pushed forward both the chorion and the amnion, and diminished the size of the cavity. There are all possible gradations in the amount of blood so extravasated, and the morbid appearances vary from slight thickening or consolidation to the extreme forms indicated in the drawings. The nutrition of the embryo is necessarily arrested if the extravasation be extensive enough to interrupt the normal circulation through the imperfectly formed placenta, and the embryo may be found stunted and shrivelled, suspended by its umbilical cord from some part of the amniotic cavity. If the ovum has been long retained after these morbid changes have taken place, the embryo will appear small in proportion to the size of its containing cavity, for there can be no doubt that when the central embryo has perished, the membranous envelopes may go on growing until they are expelled by uterine action. In some specimens again, the embryo deprived of its nourishment breaks down and dissolves, leaving only the remains of a slender or rudimentary umbilical cord, and not uncommonly also a distinct umbilical vesicle, imbedded between the chorion and amnion.

These extravasations of blood between the chorion and the decidua constitute the typical "apoplexy of the ovum" described by many authors. It is found in a multiplicity of forms and modifications, and is, no doubt, produced by a variety of causes. Thus it may be produced by faults in early development, or by any cause which produces detachment of the well-formed decidua from the uterine walls, or from rupture of its blood-vessels. Gendrin said he saw one case where blood tore its way through both chorion and amnion, and overwhelmed the embryo, and Hegar gives another. The drawing by Westmacott shows the cavity of the decidua reflexa entirely occupied by a blood-clot, no trace of embryo or its special envelopes being found.

Wagner, in his *Traité de Physiologie*, states that in the majority of cases an extravasation of blood at the time of the formation of the decidua reflexa is the first cause of the death of the fœtus. In some cases, notwithstanding this, the egg may grow, but the embryo is then not properly nourished, and so a disproportion of different parts of the egg occurs. The extravasation of blood may therefore be the cause of disease in the decidua and of malformation in the fœtus.

Hegar says, and Verdier[8] agrees with him, that anomalies in the insertion of the placenta, abnormal insertions of the allantoid and umbilical vessels, predispose to apoplexy, also the hypertrophy and atrophy of the various parts of the decidua.

In examining ova which have been extruded as the result of abortion during the earlier period, one is often struck with the amount of compound granular cells, as they are called, and of distinct fat or oil-particles, not only mixed up with the parenchyma of the decidua, but contained even in the interior of the decidual cells and fibro-cells, thus constituting a true fatty degeneration. Sometimes the decidual blood-vessels, namely, those which permeate everywhere the decidual tissue, and ramify among the villi at the site of the future placenta, have undergone fatty change, and this is more marked near the seat of the hæmorrhages. At one time I thought this was due merely to the after-changes in the extravasations of blood produced by traumatic and other causes, but I subsequently found this fatty change in portions of the membranes distant from the clots, and occasionally I have found the whole decidual structures affected by this form of granular degeneration.

I have elsewhere pointed out[9] that in the parietal decidua which

[4] *Von dem Mangel, etc., der Gerbärmutter*, 1859.
[5] *Monatsschr. für Geburts*, Bd. xxi, Supplement. Heft.
[6] *Monatsschr. für Geburts*, 1863.
[7] *Lehrbuch der Path. Anat.*

[8] *Thèse*, 1868.
[9] *Devel. of Gravid Uterus.*

does not take part in the formation of the placenta, a granular or fatty degeneration sets in as soon as its function is superseded by the organisation of the placenta. The drawing there shows the glandular follicles in the third month, filled with fat-granules prior to their final disappearance, and similar fatty molecules are scattered over the parenchyma generally. In the normal condition this fatty degeneration does not extend beyond the border of the placenta, but it can readily be understood that in such close proximity it may invade the placental decidua also, when impaired nutrition or other cause predisposes it to decay.

On becoming acquainted with the remarkable researches of Ercolani,[10] I found he believed the true cause of extravasation and of the formation of clots between the decidua and chorien in early pregnancy, to be this fatty degeneration of the cells of the decidua serotina, which ought to be forming the young placenta. These cells so transformed out of the decidua, when in a state of fatty degeneration, imperfectly support the pressure of blood in the sinuses, and hence, as he says, there is a breakdown and veritable interstitial hæmorrhage. The rupture of the layers of serotina permits the blood to pass from one cotyledon to another, and necessarily produces a slackening of the maternal blood in the imperfectly formed sinuses. Thus a true thrombosis is produced, but preceded by morbid change of structure, not arising out of a normal state as supposed by Bustamente[11], whose views I shall notice presently. Sirelius,[12] of Helsingfors, also attributed hæmorrhage in the early formed placenta to the breaking down or rupture of the decidual structures forming the boundaries of the lacunæ, thus producing coalescence and arrest of circulation, with extravasation.

That the presence of a pathological fatty degeneration in the structures of the decidua will, in many cases, account for the occurrence of hæmorrhage both in the parietal decidua and in the serotina is no doubt true, but there are many cases where no such explanation can apply, otherwise there would be very little chance of checking a threatened abortion when once indications of hæmorrhage have set in. The frequent recovery of patients from the symptoms of abortion, with the successful continuance of pregnancy to the full time, is sufficient proof either that hæmorrhage is due to other causes than fatty degeneration, or that, in particular cases, the degeneration was at least so partial that only a very limited area of the structures was affected. Thickening and fatty degeneration to a marked extent in the decidual structures are noticeable in many instances of recurring early abortion; and, as we shall see presently, this may be associated with an inflammatory process. In other cases, separation of the ovum is clearly traceable to some violence or traumatic cause; then the structures show no indication of morbid transformation, and the extruded ovum frequently tears away with it from the interior of the uterus shreds of uterine muscular fibre which are found attached to its outer surface.

Apoplexy of the ovum deserves careful study, because it is a very common cause of embryonic death, and, if one may judge from the large number of specimens preserved in the various museums, compared with others, it is by far the most frequent of all the pathological changes affecting the early ovum. In almost every museum in London are examples of the apoplectic ovum in its various phases, even where there is an absolute poverty of other specimens illustrating diseases of the ovum, and it may be inferred therefore that it is probably the most frequent of the immediate causes producing intra-uterine death in the early months of gestation. The specimens are often wrongly described in the museum catalogues as "tubercular ova," or "cystic ova," and other misnomers are applied to them.

The largest number of the preparations of this kind in our museums are of ova so advanced that the two deciduæ are united in close apposition. This corresponds to about the third month of gestation. The chorion has imbedded its tufts everywhere in the decidua, and the amniotic cavity is distinctly formed. On account of the size of the aborted ova, the morbid changes are so marked that they are obvious to the most casual inspection, and hence they come to be preserved.

We have no accurate means of knowing the comparative frequency of apoplexy and resulting abortion in ova at an earlier stage than this, because in the first weeks aborted ova have less marked characteristics—they are often passed unobserved, or, if noticed, they are, from their fragility, likely to be torn and injured during expulsion. For both these reasons preserved specimens of this kind are much rarer in the several museums than otherwise they might be.

One of the morbid conditions of the decidua, the presence of which is inferred rather than demonstrated, is congestion.[13] The inference is drawn from the fact that in some ova expelled during abortion the decidual vessels are found gorged with blood, not only on the outer or uterine surface, but in the deeper layers, and no evidence of other pathological change is observable. The women chiefly predisposed to it are the plethoric, those suffering repeatedly or habitually from disorders of the portal circulation, from heart-disease, and also those who, from any other cause, are the subjects of pelvic congestion generally. Hegar noticed the uterus proportionately larger in those prone to congestion and apoplexy of the ovum.

It is believed that congestion of the decidua is a sufficient and adequate cause, irrespective of any other, to produce rupture of its blood-vessels and extravasation into its substance. It depends, of course, upon the amount and extent of the congestion and its consequent results as to whether abortion is provoked and pregnancy comes to an end. If blood is extravasated as the result of congestion, it goes through the same changes and phases as when extravasated from other causes. But there is reason to suppose, also, that hyperæmia, arising from whatever cause it may, apart from extravasation, can have the effect, by the distension and irritation it produces, of bringing on uterine action and thus precipitating abortion. This is the more probable as the whole uterus must partake of the congestion, and be proportionately irritable.

Inflammation of the decidua, taking various forms, has been described by Virchow, Hegar, Schroeder, and Spiegelberg. Slavjansky has described an acute form associated with cholera in pregnant women, and leading to hæmorrhage with the death of the fœtus.[14] These inflammations are, however, generally chronic, and are continuations or extensions of previously existing inflammation in the unimpregnated mucous membrane of the uterus, and three forms have been described as producing different alterations in the decidua.

I. The first, "chronic diffuse endometritis," of authors, produces a thickening or hyperplasia of the uterine mucous membrane, which, when conception occurs, renders it unfit for the reception and growth of the fecundated germ. According to Spiegelberg, the hypertrophy consists in a development of connective tissue, which not only thickens, but indurates the membrane, and extends down to the muscular fibres themselves. The arrangement of the hypertrophied tissues is such as often to give rise to the appearance of cysts, which, however, are probably only the "cups" of Montgomery enlarged. Schroeder regards it as a chronic and diffuse proliferation of the mucous cells, both parietal and reflected. The thickened membrane presents, he says, the large cells of the decidua, united *en masse*. By their proliferation, particularly in the deeper layers, they may produce a cavernous structure, and even form cysts. This alteration, he says, produces death of the embryo and abortion, by the irritation which the inflammation produces on the uterine nerves.

Other cases have been described in which inflammation was still more chronic, and where the nutrition of the germ was not interfered with. Madame Kaschewarowa,[15] a Russian woman doctor, whose researches are sufficiently accredited to find a place in Virchow's *Archives*, alleges that she has found the membranes of a fœtus at term thickened, not only by proliferation of connective tissue and decidual cells, but even by smooth muscular fibres of new formation. Hofe [16] has described other alterations of form, and Schroeder [17] and Spiegelberg [18] both state that there may be proliferation of decidual cells, which is secondary to the death of the fœtus, although, if it begins before, it is apt to deprive it of vitality.

In examining early deciduæ thrown off as the result of early abortion I have repeatedly observed a morbid condition, which differs in its histology from the description just given by Spiegelberg, Schroeder, and others, and which does not seem to have been sufficiently noticed. In this the membrane is distinctly thickened and hypertrophied, and its structure is firmer than normal, while its outer surface exhibits none of these floating filaments seen in comparatively healthy deciduæ, but is more or less nodular, and shows indications of being slowly separated from the uterine walls. Microscopic examination shows that there may be an attempt to increase the cell element peculiar to the decidual structure, and that the cells themselves are more frequently split up than usual and otherwise deformed; but in addition to the cell element there is besides a copious exudation, which is at first simply amorphous or granular, but soon degenerates into fat. The granular deposit is in much larger proportion than the increase of the cell element or of connective tissue, and it may be free

[10] *Delle Malattie della Pl.*, Memoria, 1871, and Translation by Marcy.
[11] *Thèse Inaug.*, 1868.
[12] *Vide* Ercolani.
[13] See Devilliers, *Rev. Méd.*, 1842.
[14] *Archiv. für Gynäkol.*, 1882.
[15] Virchow's *Archiv.*, 1868.
[16] *Archiv. für Gynäkol.*, 1869.
[17] *Lehrb. der Geb.*
[18] *Lehrb. der Geb.*

from fat in some portions of the membrane, but is elsewhere pervaded with compound granular cells and oil globules. A condition somewhat similar to this has been briefly noticed by Hegar. It is, I believe, the result of a low form of inflammation, and as it degenerates, which it readily does if at all abundant, it involves the decidual cells, fibro-cells, and blood-vessels in the same morbid change. The inflammatory action is probably due to an unhealthy condition of the uterine mucous membranes prior to conception, or to impaired nutrition afterwards from faults in the maternal blood. Had the exudation been less abundant, or endowed with greater vitality, it would probably have been converted into connective tissue as described by the authors named.

The engraving of a hypertrophied decidua given by Duncan at p. 292 of his *Researches on Obstetrics* is probably an example of one of the forms of hyperplasia produced by endometritis, syphilitic or otherwise. The membrane is described as much increased in thickness, its walls thrown into prominent folds or rugæ, and the cavity was very large in comparison with the small size of the embryo attached to the fundal portion; the embryo had evidently ceased to grow soon after conception, while the decidua had gone on vegetating.

Dr. Duncan remarks that fatty degeneration was present in this as in other specimens of hypertrophied decidua which he examined, and I may observe that fatty change is noticeable in all preparations of this kind which have been retained after the death of the embryo. It is often difficult to say whether the fatty degeneration was a primary change arising from faulty conditions of the maternal blood, and which caused the death of the embryo; or whether it was secondary to fœtal death from some other cause. In either case the fatty change, by the friability it produces, loosens the attachments of the decidua to the uterus, and so precipitates abortion.

A variety of this chronic diffuse endometritis constitutes the "adhesive endometritis" of Braun.[19] It takes place in the later months and it then attacks the utero-placental mucous membrane, setting up such irritation that it imperils the life of the fœtus and precipitates premature labour. Strictly speaking the affection may be regarded as a disease affecting the placenta. One of its results is to produce adhesions between the placenta and uterus, and so complicate delivery. Its presence is characterised by the fœtal movements giving pain, and often, besides this, acute pain and tenderness are experienced in the walls of the womb, which some have described as "uterine rheumatism." It may be produced by chills, overwork, etc.; and according to Kaschewarowa, it is one of the results of syphilis, or a pre-existing endometritis.

II. A second form of inflammation affecting the decidua has been described by Virchow[20] under the name of "polypoid endometritis."

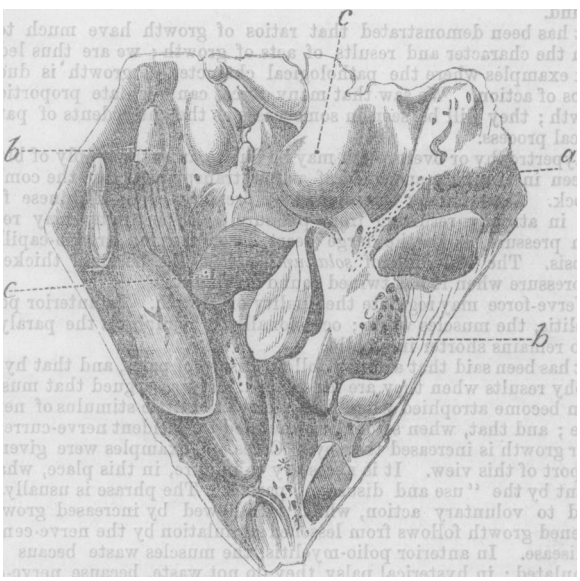

Fig. 1.—Polypoid Endometritis. (Virchow.) *a*, fine apertures of the glands; *b*, larger apertures of glands; *c*, protuberences, or polypi.

It would seem to be only a more advanced degree of the inflammatory condition just described, but the mucous membrane is thickened to twice or thrice its ordinary depth, and prominences and projections like polypi protrude from the free internal surface. Where the eminences exist, the uterine gland apertures are obliterated, while they are apparent on other parts of the surface. According to Virchow, the microscopic element which grows here in excess is the interstitial mucous tissue, which increases and proliferates in such wise as to cause the hypertrophy. Spiegelberg, Schroeder, Dohrn, and Gusserow[21], have all verified Virchow's observations with some modifications as to detail in structure. Virchow's case was attributed to syphilis, but there was no proof of this in others. This change is only seen in very young ova, and it always produces an alteration in the chorial villi which may be in contact with it, disturbing and altering their form; and in some cases, as pointed out by Dohrn and Müller[22], causing them to show commencing myxoma. The embryo in nearly all the recorded cases had disappeared.

III. A form of inflammation affecting the decidua has been named "catarrhal endometritis." It is characterised by a persistent discharge from the gravid uterus, which constitutes the "hydrorrhœa" of pregnant women. A woman, generally about the sixth month of pregnancy, loses suddenly and at intervals a quantity of transparent colourless fluid, analogous to ascitic fluid. This is succeeded by a dribbling more or less prolonged and without pain. The pregnancy may go on to term; and the membranes, as a rule, are found intact. The affection has given rise to much discussion, and there is an extended literature on the subject. Naegele has seen it persist for sixteen weeks without interrupting pregnancy.

According to Spiegelberg, Schroeder, Braun, and Hegar,[23] the fluid comes from the cavity of the decidua, which is a secreting membrane even during pregnancy, and hydrorrhœa is only a hyper-secretion of this membrane depending on chronic inflammation of the decidual glands. A great diversity of opinion has, however, been expressed as to the source of this fluid. Tarnier and Budin, besides the decidual form, speak of an "amniotic hydrorrhœa," which occurs as the result of perforations of the amniotic sac far up in the uterine cavity, and, on account of the position of the aperture, do not necessarily precipitate labour.

A drawing of a portion of the amnion, with such perforation, is given in their conjoint work on *Midwifery*. It is a somewhat rare

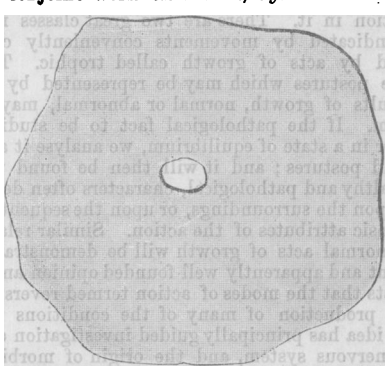

Fig. 2.—Aperture in the bag of membranes found in the upper part of the sac, distant from the usual seat of rupture. (Tarnier and Budin.)

condition, only seventy cases having been collected in the most recent essay on the subject. It is only under exceptional circumstances that simple hydrorrhœa compromises the course of pregnancy; most women affected by it go to the full period. I need not, therefore, dwell further on the subject. Those interested may be referred to the *Thesis* of M. Stapper,[24] and to the *Traité des Accouchements* by Tarnier and Budin, which contains the last and most able exposition of this topic.

A form of catarrhal endometritis I have sometimes met with in early pregnancy, and attended with sero-sanguineous or dark grumous discharges, is more formidable so far as pregnancy is concerned. Its persistence generally indicates chronic disease going on in the fœtal envelopes—of the decidua more especially—and it commonly ends in abortion. There is usually, in such cases, some antecedent history either of injury or of endometritis existing before the advent of pregnancy.

Some of the so-called "moles" consist of the decidua altered and distorted by one of the morbid processes I have just de-

[19] *Gesammt. Gyn.*, 1881.
[20] Virch. *Arch.*, 1861 and 1865.
[21] *Mon. für Geburt*, 1866.
[22] *Bau der Molen*, 1867.
[23] Hegar, *Monatsschr. für Geburt.*, 1863.
[24] *Vide* Charpentier, *Tr. des Acc.*

scribed. A "carneous mole" is so termed because it looks like a fleshy mass expelled from the uterus, and it usually consists of the morbidly thickened decidua which forms the outer covering; and the contents are either laminæ of blood-clot arranged like the layers of an aneurysm, or such remains of the disorganised embryo and its special membranes as have escaped entire destruction. Most frequently, in the case of carneous moles, no trace of the embryo can be discovered.

[*To be continued.*]

ABSTRACT OF LECTURES
ON
THE ACTION OF NERVE-CENTRES AND MODES OF GROWTH.

Delivered at the Royal College of Surgeons of England.

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Lecture III.—The Study of Pathology.

In studying any fact in physiology or pathology, as in studying any scientific problem, we must first describe it in exact physical terms, such as connote what may be observed. In giving descriptions for scientific purposes, it is advisable to avoid using metaphysical terms, as also to avoid speaking of pathological states as special entities. Let us, then, seek to describe pathological states in terms connoting facts capable of observation. The work in hand is divided into two parts:—to describe facts; and to seek their explanation in the antecedents and sequents of these facts. The importance of pabulum and its supply in bringing about pathological processes has often been insisted on; I would insist on the equal importance of the forces or stimuli incident to the subject observed as necessary factors in determining the action in it. There are two great classes in pathological facts—those indicated by movements conveniently called kinetic; those indicated by acts of growth called trophic. The results of movements are postures which may be represented by casts or drawings; the results of growth, normal or abnormal, may be preserved in the museum. If the pathological fact to be studied is kinetic, either active or in a state of equilibrium, we analyse it as we analysed movements and postures; and it will then be found that in many cases, both healthy and pathological, characters often depend upon the antecedents, upon the surroundings, or upon the sequents, rather than upon the intrinsic attributes of the action. Similar relations between normal and abnormal acts of growth will be demonstrated. There is a very prevalent and apparently well-founded opinion among biologists and pathologists that the modes of action termed reversion are potent factors in the production of many of the conditions termed pathological. This idea has principally guided investigation concerning the action of the nervous system, and the origin of morbid growths. I shall not attempt to prove any view, but put forward an hypothesis for the purpose of illustrating what I believe to be the advantages derived from the definite observation and study of movements. The theory may be advanced that "chorea is a condition of the brain analogous to that found in healthy infancy, such brain-state occurring at a period of life when the face, generated by nutrition, is greater than in infancy." A reversion has been defined as a repetition of a series of movements or trophic acts, similar to a series previously existent; the criteria indicating similarity have also been defined. The child can never again become an infant, but it may became "infant-like;" its body is heavier and larger than that of an infant, its quantity of nutrition and movement are greater. In chorea the reversion is indicated by a repetition of the ratio of the attributes of the infantile state; the ratio of kinesis to weight is repeated, it is infant-like in the ratio of kinesis to trophic action.

The nerve-centres are observed at two periods of life, infancy and childhood. Comparison is made as to the quantities and time of their action. Observation shows the combination of movements in the infant and in the choreic child to be similar. There is said to be similarity as to the parts and their order of acting. The quantity of movement depends upon brain-nutrition. The total quantity of brain-nutrition may roughly be estimated by the weight of the body; say 15 lbs. in the infant, and 45 lbs. in the child. The nutrition in the child is three times as great as in the infant. If the ratio of kinesis and nutrition were maintained during development, there would be three times as much movement in the child as in the infant. I believe this is not usually the case; but, as development advances, kinetic action is lessened in its ratio to other functions. We say that in health the ratio of kinetic action to nutrition lessens as growth proceeds; if this ratio reverts to that of infancy, there will be an amount of movement altogether abnormal. It seems to me that this reversion to the infantile ratio between nutrition and movement is what we observe in chorea. Lessened nutrition tends to produce reversion. In chorea the weight falls, and the condition is not removed till it is restored to near the normal. Mitral regurgitation tends to produce irregular supply to the brain.

Coma may be described in terms of lessened movements and reflexes. All movements may be absent, except those of organic life. The head is not turned towards a source of light or sound; the word of command and pressure on the chin are not followed by protrusion of the tongue; there may be subsultus tendinum. The arm, held passively free, falls into the posture of the feeble hand. (See *Physical Expression: (International Scientific Series)* chap. ix.)

Further illustration was given by a comparative study of hemiplegia and hemispasm; the nerve-muscular area is the same in each. The decrease of quantity of motion in hemiplegia is proportional to its increase in hemispasm in the small and large parts respectively. Fatigue and exhaustion of the brain muscular power on both sides are common. If, however, one side of the body or one limb be powerless, there being no evidence of organic disease, then the state is often called hysteria. It appears, then, that a bilateral diminution of nerve-force of short duration is less pathological than a one-sided weakness.

In observing patients as to their movements and postures, note the action of small muscles; for example, interossei causing lateral movements of fingers, as distinguished from flexor movements produced by larger muscles. Asymmetry of action was then dwelt upon; it was suggested that lateral curvature of the spine often commences there. A nervous child was described. Let the child stand up, and observe it: the conditions of growth, make of skin, the form of the features; he is often too tall and too thin, the legs being less emaciated than the body. Let the hands be held out, the left upper extremity is usually at a lower level, the nervous hand posture is probably seen on either side, perhaps more marked on the left. There may be finger-twitching, with flexor or lateral movements; the spine is arched too forward in the lumbar region, often with slight lateral curvature. The face, as a whole, is usually too immobile, but there may be twitchings of the face, eyes, tongue, etc.; the head is often flexed, with inclination and rotation towards the same side. The teeth are often ground.

It has been demonstrated that ratios of growth have much to do with the character and results of acts of growth; we are thus led to seek examples where the pathological character of growth is due to ratios of action. We saw that many forces can regulate proportional growth; they will be seen in some cases as the antecedents of pathological process.

Hypertrophy or overgrowth may be due to an extra supply of blood, as seen in Hunter's specimen of a spur transplanted into the comb of a cock. Growth may be lessened by pressure, as in a Chinese foot, and in atrophy resulting from pressure. Hypertrophy may result from pressure, as in the large heart accompanying arterio-capillary fibrosis. The leaf-stalk of *solanum jasminoides* becomes thickened by pressure when it has twined round a support.

Nerve-force may regulate the quality of growth. In anterior poliomyelitis, the muscles waste; occasionally in hemiplegia the paralysed limb remains shorter in a child.

It has been said that atrophy follows disuse of parts, and that hypertrophy results when they are much used. It was argued that muscles often become atrophied when they receive too little stimulus of nerve-force; and that, when strongly stimulated by incident nerve-currents, their growth is increased thereby. Cases and examples were given in support of this view. It is necessary to inquire, in this place, what is meant by the "use and disuse of parts?" The phrase is usually applied to voluntary action, which is followed by increased growth; lessened growth follows from lessened stimulation by the nerve-centres in disease. In anterior polio-myelitis, the muscles waste becaus not stimulated; in hysterical palsy they do not waste, because nerve-currents do reach them, though not in quantity sufficient for motion. Certain crustaceans from the Mammoth Cave were shown, whose eyes had been lost from their long residence in darkness; other members of the species living in light had perfect eyes. What is called voluntary action appears to produce more growth than chorea, athetosis, etc.; it is probable that in the former cases stimulation is greater.