travel upwards for this distance from the foramina in the lumbar and sacral regions. When, then, the circulation becomes languid, the mechanical difficulties of this arrangement make themselves felt. The symptoms attending the early stage of paraplegia due to failing circulation in the lumbar enlargement of the cord are very interesting. As the nutrition of the lower end of the cord begins to suffer, there is at first muscular weakness and loss of control over the legs only after a night's rest. The patient has some difficulty in standing and walking steadily when he first gets out of bed; but, after he has moved about a little, the legs regain power, and he can walk perfectly. A similar state of things is observed with regard to the bladder. He cannot pass water on rising, but, when he has had a little walking, he empties the bladder easily. Whenever he sits down for any length of time during the day, there is more or less impairment of mobility and strength in the lower extremities, which quickly passes off with movement. Sensation is not affected at first, but there may be feelings of numbness. The coming on of the weakness during the night is due to the slackening down of the circulation which takes place during sleep, and is paralleled by the morning depression in melancholia and debility.

I have met with this train of symptoms at the two extremities of high and low pressure. When there is high pressure it is that the general resistance in the periphery has overtaken the powers of the heart, so that the whole circulation is sluggish, and the languid flow is most easily brought to a standstill where the difficulties are greatest. Usually the symptoms come on very gradually, but I have known their onset to be determined by the occurrence of acute dilatation of the heart. When the tension is low and the heart weak, no explanation of the impeded circulation is needed.

I end these lectures with a feeling that I have done but scanty justice to the subject, but with feelings also of gratitude for the patience and indulgence with which my poor attempt has been received.

THE LUMLEIAN LECTURES

OR

THE PATHOLOGY OF INTRA-UTERINE DEATH.

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

By W. O. PRIESTLEY, M.D., F.R.C.P., LL.D.,
Consulting Physician to King's College Hospital; and late Professor of
Obstetric Medicine, King's College.

LECTURE II.—DISEASES OF FETAL APPENDAGES.

[Concluded from page 718.]

Morbid Changes in the Chorion.—The pathological change affecting the outermost true foetal envelope or chorion which has received most attention is what is commonly called "cystic or vesicular or hydatid degeneration." In this affection small or large quantities of cyst-like bodies are expelled at intervals from the uterus of the pregnant woman, their expulsion being attended with hæmorrhage, and commonly with the discharge of a large quantity of serous fluid of pinkish colour, which has been compared to red-currant juice. Madame Boivin²⁵ stated that she found this disease occurring only twice in 20,375 pregnancies, but I suspect this account does not represent the true frequency of the affection, for most accoucheurs in large practice have met with it, and specimens of cystic chorion in its various stages are to be found in almost all our museums. In its earlier phases it is not uncommon, but then may be readily overlooked. When fully developed the appearance of the growth is very remarkable, and must at once arrest the attention of the medical attendant. Although commonly thrown off at first in detached portions, a time comes when uterine action fully sets in, and then a large quantity of cysts may be expelled in one mass, which, in some instances, is sufficient to fill a large basin. The vesicles or cysts of which the mass is composed vary from the size of a millet seed to that of a grape, and these are intimately united together at various points by thin stems or pedicles. They have been compared to bunches of grapes, but the cysts do not necessarily terminate the stem on which they are suspended like grapes, but are united in a plexiform arrangement one with another in a sort of network.

In Paré's *Surgery* it is recounted "that the Countess Margaret,

daughter to Florent IV, Earl of Holland, and spouse to Count Herman of Heneberg, on Good Friday, in the year of our Lord 1276, and of her age 42, brought forth at one birth 365 infants, whereof 182 are said to have been males, as many females, and the odd one an hermaphrodite, who were all baptized, those by the name of John, these by the name of Elizabeth, in two brazen dishes, by Don William, Suffragan Bishop of Treves." It is added: "the basins are still to be seen in the village of Loedun, where all strangers go (on purpose) from the Hague, being reckoned among the great curiosities of Holland." This account is not regarded by modern authorities as a pure invention. Ambrose Paré was eminently honest, and these 365 children are now regarded as having been merely vesicles of a cystic chorion, magnified into infants by someone whose interest it was to promulgate the illusion.

Fig. 3.—A portion of cystic chorion as seen with the naked eye. The arrangement of the vesicles is seen and also their attachments to the chorion membrane (Ercolani).

The published literature on this single disease of the chorion is more extensive than of any affection of the foetal membranes, and both detached observations and separate monographs exist in several different languages. Cloquet,²⁶ Percy²⁷ and Boivin regarded the vesicles of the chorion as true acephalocysts; but it has been abundantly proved that they are not so, being furnished neither with hooklets nor other minute structures characteristic of true hydatids. Bidlos and Soemmering believed cystic chorion to be a disease of the lymphatic vessels; and Bartolin, Müller, and Cruveilhier²⁸ a disease of the blood-vessels. Ruysch²⁹ attributed the formation of the cysts to accumulation of fluid in the cellular tissue which unites the vascular tunics of the foetal membranes.

Nearly all modern authorities agree in regarding them as the results of pathological changes of one kind or other in the villi of the chorion. The single exception, I think, is Ancelet, who in 1868 published a paper in the *Gazette des Hôpitaux*, and he there reverts to an old idea that the hydatid mole is a disease of the decidua.

²⁵ No. 1 de la Faune des Méd.

²⁶ Ancien Journ. de Méd., 1793.

²⁷ Vide Charpentier, *Maladies du Placent*.

²⁸ *Obsér. Anat. Clin.*, 1691.

²⁹ *Mém. de l'Art des Accouch.*

Velpeau³⁰ seems to have been the first to indicate that the cysts were not true or independent vesicles. He regarded them as swollen chorionic villi, the terminal extremities being enlarged by the accumulation of fluid, as a sponge is distended by water. H. Meckel³¹ and Gierse³² said there was hypertrophy of the natural structures, with cedema of the villi, which they considered secondary. An excellent description of the different stages of this transformation and its mode of growth after Mettenheimer is to be found in Sir James Paget's *Surgical Pathology*; and Dr. Barnes,³³ Dr. Graily Hewitt,³⁴ and Dr. Braxton Hicks³⁵ in this country have further illustrated the various stages of its development in separate papers. In France, C. Robin's observations have attracted much attention, and his views are adopted by M. Cayla, who wrote a well-known thesis on the subject. Probably the most lucid and notable contribution to the pathology of this subject is from the pen of the celebrated Virchow.³⁶ He points out that the many discrepancies in the ideas promulgated concerning the true nature of cystic chorion are due to incomplete knowledge of the villous structure in normal conditions. According to him, the villi consist of a prolongation of the same mucous tissue which forms the gelatin of Wharton in the cord. The villi have two essential portions—an epithelial covering (exochorion) and a substratum or body of mucous tissue (endochorion), which, non-vascular when the villi first appear as buds on the outer surface of the chorion, later contain blood-vessels. The proliferation of the epithelium which Heinrich Müller³⁷ regarded as the *point de départ* for the production of cysts, Virchow regards as the expression of regular and normal development. The buds or cysts are formed in the same way as in normal growth, and it is in the interior of the villus—not in its epithelium—that the particular and morbid transformation takes place. In effect, Virchow regards the cystic chorion as a typical form of myxoma, and he avers that he has found the same productions in other parts of the envelopes. Both Ruysch and Virchow have found these cyst-like bodies in the umbilical cord. Virchow noticed certain buds of the external epithelial covering of the villi which contained transparent cavities; these he called "physalides." Single large dilated cells containing a hyaline liquid he called "physalifores." Wedl had previously described them in his *Histological Pathology*; and Virchow, while confirming Wedl's observations, regarded them as accidental products. Ercolani interpreted their presence after another fashion, and as affording evidence of a different theory of cyst development.

Virchow further states that the mucous metamorphosis of cells seen in the villi of the chorion may disappear in fatty transformation, or the cells may be changed into fibrous elements, thus constituting a "myxoma fibrosum." This change I shall describe later.

Fig. 4.—A a, Extremity of a villus in early stage of cystic degeneration, a, shows the first stage of enlargement in the cells of the villus trunk; b, a somewhat more advanced stage, showing by a line cells escaping from the ruptured capsules of a young cyst. (Priestley.) B Terminal villus of cystic chorion, a, stellate connective tissue; b, c, inner and outer layers of wall; d, early stage of b (Braxton Hicks).

As long ago as 1858,³⁸ before I knew anything of the researches of Virchow, I had pointed out that the cysts of the chorion were not mere dropsical dilatations of the terminal villi, but that the fluid distending them, in the earlier stages at least, was formed or secreted in the interior of beautiful thin-walled cells, which had their origin in

the centre of the villus. The microscopic examination I described in my lectures on the Gravid Uterus as follows:—"Placing a terminal branch under the microscope which seemed to the naked eye nearest to the normal condition, it was seen to be enveloped by a granular covering, probably derived from the altered decidua, the club-shaped extremities were observed to be distended with large nucleated cells, and bearing little resemblance to the small and more uniform cellules composing a terminal villus in a healthy state. By compression, the envelope bounding the villus could be ruptured, and gave egress to the cells. The fully developed cysts had two coats, like a normal villus, the external epithelial, the internal delicately fibrous, but firm and dense. An incision being made through these (if the preparation was fresh) what appeared to be a viscous or gelatinous fluid escaped from the aperture, and this was found to be contained in large transparent cells, with walls of extreme tenuity, assuming the polyhedral form from the pressure to which they had been subject. The entire contents had much the appearance of the vitreous humour of the eye, which has transparent partitions running across, separating the fluid into compartments; the presence of a nucleus, however, in each compartment clearly proved it to consist of largely developed thin-walled cells. In the largest cysts, the pellucid cells were not readily discernible, having probably undergone solution. Scattered over the vesicles and their connecting peduncles were little nodular projections, consisting of cellular buds for new branches, such as exist in healthy villi. The narrow stems uniting the cysts were fibrous with a cellular covering, and sometimes enclosed a small vessel full of blood. Fat granules were copiously deposited in the texture of both stems and vesicles."

There is no discrepancy between this account from actual observation and the view taken by Virchow, that the morbid change is a true myxoma. The only difference is that Virchow seems to look upon the distension of the villi as due to the accumulation of mucus in the inter-cellular tissue, while I found the distension produced during early development at least in the rapid production of thin-walled, nucleated cells. Possibly both may be right; it seems highly probable that, in the smaller cysts, large cells with fluid contents constitute the first stage of the disease, and, later, these cells form themselves into spaces traversed by trabeculae of connective tissue, and containing the fluid. This is identical with the process by which the gelatin of Wharton is produced in the cord. The exuded fluid in both cases has all the chemical reactions of mucine. It is by no means uncommon to find in the extremity of a comparatively healthy villus one or more large transparent hyaline cells. These represent the beginnings of the cystic change, or the first transformation from the more uniform cell-structures which compose the parenchyma in a normal state.

Ercolani³⁹ (who wrote in 1876) differs *in toto* from Virchow and other authors in his view of the production of cystic chorion, and, disbelieving in its myxomatous origin, he regards Virchow's appellation for it as misleading. He, like Virchow and Robin, describes a villus as composed of two parts: one internal or parenchymatous (*tissu chorion* of Robin, mucous tissue of Virchow) which is in direct communication, and in fact continuous, with the chorion. Besides this is an external portion, which constitutes an outer envelope or casing to the villus (the exochorion of Robin). Before the villus penetrates the decidua, this outer envelope is constituted of an epithelium which belongs to the foetal structures, but directly the villus is embedded in the decidua to form the placenta this epithelial envelope disappears, and its place is taken by a layer of cells derived from the decidua, which he calls the "glandular organ," because in this glandular organ he conceives is carried on the process of elaboration by which materials are provided from the mother's blood for the nutrition of the foetus. Now it is from this outer envelope—the epithelial covering in the primitive chorion villus, or, if the disease begins later, from the glandular organ in the villi of the placenta—that, according to Ercolani, the proliferations take place which form the cystic disease of the chorion, and not from the mucous tissue or parenchyma, as stated by Virchow. Heinrich Müller had in 1843 declared that these morbid changes commence with an enlargement of the external envelope of the villi, and that later these buds or enlargements give place to internal cavities which become vesicles. Virchow so far agreed with Müller that the epithelium is the point of departure for the formation of cysts, but he contends that, in the production of vesicles, the parenchymatous or basic substance of the villi must penetrate the new epithelial product, thus carrying with it the mucous cells continuous with the chorionic structure, which are necessary to the production of the true myxoma. From this view Ercolani emphatically dissents, and he gives various details of in-

³⁰ *De l'Art des Accouchements*.

³¹ *Handbuch der Mensch. Anat.*, 1820.

³² *Verhandl. der Gesells. für Geburt.*, 1847.

³³ *British and Foreign Medical Review*, 1855.

³⁴ *Obstet. Trans.*, vol. 1.

³⁵ *Guy's Hospital Reports*, 1864.

³⁶ *Die Krank. Geschw.*, t. 1.

³⁷ *Der Bau der Molen.*, 1867.

³⁸ *Lecture on Development of Gravid Uterus*.

³⁹ *Loc. cit.*

vestigation to sustain his position. Thus, besides his asseveration that the buds take their origin in the epithelial or glandular layer surrounding the villi, he points out what had before been indicated by Ancelet, that the vesicles in their development do not necessarily spring from the parent villus trunk, but take their origin in other vesicles, and that some vesicles not only proliferate from large vesicles, but also that the largest often spring from those which are much smaller. This he holds is quite incompatible with the idea of the mucous tissue from the chorion penetrating or sending prolongations into all the vesicles.

Fig. 5.—Extremity of a chorion villus, hypertrophied, and beginning cystic degeneration. The cellular structure seen at *h*, is the epithelial covering of the villus. Neoplasms are springing from the surface of the villus (*ee*), and large dilated cells (physaliformes) are seen at (*g*). (From Ercolani.)

In *Ulmine* Ercolani holds that there may be a formation of cyst-like bodies beginning in the epithelial envelope of a villus, when disease attacks the chorionic villi at a very early period. But when the chorion has united with the serotina to form the young placenta, the typical form of cystic degeneration begins in morbid proliferations of the decidua or glandular organ, and so the disease apparently belongs more to the mother than her offspring. It is worth while to appreciate this point, if it be true, as it may afford some clue to subsequent treatment, which must necessarily be on the maternal side. Ercolani's researches and speculations on this subject are marked by great painstaking and ingenuity, but I confess I am not convinced of the truth of his view as opposed to Virchow's. Ercolani has certainly contributed to produce some confusion in the pathology of the condition by describing myxoma of the chorion and myxoma of the placenta as different affections, and giving to each a separate chapter, while there can be no doubt that both are essentially the same disease with but slight modifications.

The general relation of the cystic chorion to the decidua is sometimes very distinctly seen in the incipient stages of the morbid change. Many years ago the late Dr. McClintock showed me in Dublin a preparation which is mentioned by Dr. Montgomery in his *Signs of Pregnancy*, in which a mass of cysts is enclosed between two layers of decidua—the decidua vera and the decidua reflexa. Dr. Grailly Hewitt⁴⁰ has also figured a specimen in which both deciduæ are well shown, and the relation of the cyst-like structures to the serotina is delineated. When cystic disease is further advanced the relations of the various membranes become so confused that the vesicles are inextricably mixed up with the hypertrophied decidual structures, and separation of the two is impossible.

There are all possible varieties in the degree and stage of the disease to be met with, and the different appearance of the mass which is expelled from the uterus has given rise to a classification of these "moles," as they are called. Thus we have the "vesicular mole," in which the chief part of the substance passed consists of the pellucid cysts just described, varying in size from a pin's head to the size of a grape. With the cysts, however, is always mixed up a certain amount of a denser envelope—the altered decidua—which either ad-

heres to the cysts or is loosened from them and expelled separately. If the thickness of the outer envelope of the mass greatly preponderates over the cystic structures within, we have another modification of the so-called "carneous mole," which may with propriety be named "carneo-cystic," to distinguish it from the other carneous mole I have described as a mere thickened decidua. In this the mass is made up of a more or less fleshy substance, granular in appearance; and blood-stained, with a small proportion of vesicles. Some time ago I carefully examined a specimen of this kind, which had been expelled from the uterus with all the symptoms of miscarriage. The mass was half the size of a cocoa-nut, and, when cut into, was seen to be composed of a granular dark red substance, interspersed here and there, at distant intervals, with vesicular bodies. In this case the chorionic villi had degenerated as in the more marked form of vesicular mole; but the decidua had become proportionately much more morbidly developed than the chorionic villi, and hence the different appearance of the mass. In this and in other instances I have repeatedly examined the microscopic structure of the granular matrix in which the cysts were sparingly or more amply imbedded, and I have rarely failed to find distinct evidences of a definite arrangement of glandular structure, such as exists in the normal decidua. All the elements were, however, enormously hypertrophied; the glandular particles were infiltrated with fat-granules and the debris of blood-corpuscles and crystals. The blood-vessels themselves were surrounded with fat-granules and molecules, and crumbling in degeneration. This fatty degeneration must cause a tendency to separation from the walls of the uterus. The mass then becomes a foreign body, which at length excites uterine contraction, and ends in its expulsion.

When cystic disease of the chorion is far advanced, the embryo either entirely disappears, or, if found, it is commonly shrivelled and wrapped in its amnion in the centre of the mass. In some rare cases, even when the embryo is found, no trace of the amnion is present. It has either not been developed, or, once formed, it has disappeared, and the shrivelled and distorted embryo remains attached directly to the inner surface of the chorion.

Chorionic villi which have undergone cystic degeneration have a remarkable way of forcing themselves not only through the decidua, but into the very substance of the uterine walls. This is strikingly illustrated in a preparation in St. Thomas's Hospital, which is recorded in the *Obstetrical Transactions* by Dr. Cory. The woman from whom it was taken was forty years of age and had had eight children. When more than four months pregnant, she passed a large hydatid mole. Bleeding, with serous discharge, continuing, the uterus was eventually injected with perchloride of iron. The patient died very suddenly after, and it was found that the iron had penetrated to the peritoneal cavity. The uterus shows a portion of cystic chorion still attached to the interior of its cavity, and the cystic villi have forced themselves along the uterine sinuses so far down into the deep structures of the womb that they are close to the peritoneal surface. There was consequently very little tissue left between the cavity of the uterus and the peritoneal cavity. Somewhat similar cases have been recorded by Waldeyer and Jarotzky,⁴¹ Spiegelberg,⁴² and Volkmann.⁴³ (Fig. 6.)

Cystic degeneration of the chorion is essentially a disease of the membranes in early pregnancy. It has been supposed to begin before the allantois reaches the chorion, and, therefore, before the villi are supplied with vascular loops. That this cannot be always the case is proved by the fact that foetal blood-vessels are sometimes seen in the stems of the diseased villi and in the vesicles, and also by the cases of partial myxoma which have been recorded.

In early gestation, the whole outer surface of the chorion is furnished pretty equally with villi. Those which are to form the placenta, and which are imbedded in the decidua serotina, soon become more developed, and the rest, under ordinary circum-

Fig. 6.—Cystic chorion forcing its way through the muscular walls of the uterus towards the peritoneum. (After Spiegelberg.)

⁴¹ Virchow's *Arch.*, Bd. xlv.

⁴² *Lehrb. der Geburt.*

⁴³ Virchow's *Arch.*, xli.

stances, remain for a while stationary or retrograde. If cystic disease begins, it is generally at the crisis of development, when the placenta is about to take a definite shape, and the villi are concentrating themselves on the placental spot. Then the villi over the whole sphere of the ovum have about an equal growth, although they may be somewhat more luxuriant at the placental spot; and all may be alike affected by the myxomatous change. Not only do the villi destined to form the placenta dilate, but the chorionic villi which should become atrophied increase in volume and hypertrophy. This accounts for the general appearance of the cystic mass when it is expelled from the uterus, the vesicles being pretty equally distributed everywhere through it. There are some few exceptions, in which the placenta is attacked by cystic change after the placenta is already localised; but the disease then rarely shows itself in an advanced form, the dilations of the villi being comparatively small. Instances, however, have occasionally been recorded in which living fetuses have been born with the placenta in a state of partial cystic degeneration, and some of these seem sufficiently authenticated by modern observers to be admitted.

Partial myxomas have been described in which one or more branches of villi alone have been affected, the rest remaining healthy and the child well developed. These cases are, however, rare. When the disease does occur, as before stated, it generally attacks all the villi alike. Still rarer are instances which have been recorded where a twin embryo has perished in early pregnancy, and has eventually been expelled with cystic degeneration of its membranes at the same time as a living child with its placenta healthy. Siebold gives an example of this, and Dr. Hall Davis has recorded another in the *Obstetrical Transactions* for 1862.

The crucial question, and one which has been much discussed, is, whether the disease of the chorion precedes the death of the embryo, and so causes its death, or whether the embryo dies first, and the cystic degeneration takes place subsequently, and is in some sense the consequence. Most modern authors regard the disease of the chorion as first in the order of events; but Mikschik,⁴⁴ Gierse, and Dr. Graily Hewitt have endeavoured to show that the disease is consequent on the death of the embryo. The reasons advanced for this view are, that, in many cases, the embryo has either been arrested in growth or has disappeared altogether; and, further, that in some twin conceptions one chorion has degenerated, the other remaining healthy until term. This view is vitiated, in the first place, by the recorded instances of living fetuses where the placenta was cystic, notably by Martin, of Berlin,⁴⁵ Villers,⁴⁶ and Krieger;⁴⁷ and, further, by the fact that we may have partial myxoma of the chorion with continued growth of the embryo. Graily Hewitt gives, as one of his reasons for regarding the death of the embryo as first in the order of events, its small size in all cases where the chorion has become cystic; the largest he had been able to find was Dr. Granville's example, in which the embryo was only one inch and three quarters in length.

In St. Thomas's Hospital, however, is a fetus of from three to four months' growth, with cystic chorion fully developed. The fetus is not shrivelled, as commonly happens in such cases, but seems as though, up to a certain time, it must have been fairly nourished, notwithstanding a very marked twisting of the umbilical cord. The largest portion of the placental mass had in this specimen undergone advanced cystic degeneration, but enough of the villi had, up to a certain time, remained capable of supplying nourishment to the fetus. A time came at length when the distension of the uterus, from the rapidly-growing chorion, brought on uterine contraction and eventual expulsion of the contents. The placenta in this preparation is seen to be as large as that organ commonly is at the full time.

I take it, therefore, that both the direct and indirect evidence is clearly in favour of cystic disease of the chorion preceding the death, and in most cases being the cause of death in the fetus. There may be exceptional cases where cystic degeneration takes place after the death of the embryo. This may be the case where one placenta of twins only is affected by disease; but this does not invalidate the main position; it only indicates that cystic change in the villi may take place both before and after the death of the embryo.

As to the cause of the disease, Virchow believes that the original fault is in the decidua, which, becoming inflamed and thickened before impregnation, is incapable of receiving and supporting the chorionic villi which try to take root in it. It is converted, so to speak, into a soil incapable of affording nourishment for normal

growth, and the result is the production of a monstrosity, which acquires all the characters of a parasite, and kills the embryo by appropriating all the nutrient materials to its own vegetation. It may not be regarded as fanciful to remark that the various forms of mole seem not only to bear analogy, as previously remarked, to monstrous flowers and fruits, but also that the structural parts of these monstrosities find their counterparts in these abortive masses from the human subject. Thus, the carneous mole, with its overgrown decidua, may represent the deformed calyx, and the vesicular mole the distorted and over-developed corolla, being developed at the expense of the central embryonic portion.

Virchow's theory—that the cause of cystic disease in the chorion is to be found in a faulty condition of the uterine mucous membrane—is supported by the fact that the same woman will often produce several vesicular moles in succession, and Virchow asserts that in these women the decidua bears traces of inflammatory thickening, sometimes with polypoid excrescences on its free surface. Depaul⁴⁸ mentions a woman who had the affection three times. There is, nevertheless, abundant evidence to show that a woman may have living children at some later period, after being the subject of cystic disease of the chorion. The only objection to Virchow's view which strikes me is the fact, previously mentioned, that twin ova have been expelled in abortion, one of which had cystic chorion and the other not. It is difficult to understand, if disease of the decidua was the cause of cystic degeneration, how one embryo should be affected and the other escape.⁴⁹

The last point in connection with this subject is the question: Can cystic or vesicular chorion ever occur in women without conception? This question came before a Court of Inquiry in India some years ago, and involved both the character of an unmarried woman and the reputation of a medical man whose opinion had impugned her chastity. The medical man collected the opinions of all the leading obstetricians in this country, and, although the balance of opinion greatly preponderated in favour of vesicular chorion being always the result of impregnation, there was at least one notable dissident, who believed that the vesicles might be formed in the virgin uterus. We now know so accurately the way in which these vegetations are produced that doubt should no longer exist on the matter. With our present knowledge it would be just as reasonable to suppose that a child might be expelled from an unimpregnated uterus as a true vesicular chorion.

A portion of cystic chorion may be long retained *in utero* without giving rise to active symptoms, and thus unfounded suspicion may arise concerning the chastity of women when this is expelled. The late Dr. McClintock⁵⁰ specially called attention to this, and said "Hydatids may be retained *in utero* for many months or years, or a portion only may be expelled, and the residue may throw out a fresh crop of vesicles to be discharged on a future occasion."

There can be no doubt also of the fact, which is well illustrated in Dr. Cory's preparation previously described, that after a cystic mole has been expelled, a portion of the cyst growth may remain behind and proliferate in the uterine walls, thus keeping up exhausting discharges, and imperilling the safety of the patient. In these cases therefore the prognosis should be a guarded one. I need not say that cystic chorion must not be mistaken for true hydatids or acrophalocysts, which on very rare occasions have been expelled from the unimpregnated uterus.

Morbid Changes in the Amnion.—The Amnion or innermost foetal envelope bears a very close resemblance to a serous membrane. In its earliest development it may be seen raised like a blister over the whole dorsal surface of the embryo, and as it gradually grows away from the fetus and becomes a more distinct cavity to surround it, it increases in firmness and density, and at length coalesces with the inner surface of the chorion to form the future bag of membranes. In the early stages of pregnancy the amnion does not always escape the effects of extravasation beginning in the decidual structures. The force of the blood extravasation may be such as to break down the resistance both of the chorion and amnion, and if the cavity of the amnion is invaded, the inevitable result is the destruction of the embryo, with abortion sooner or later. The earlier in pregnancy this extravasation takes place, the greater the likelihood of the inner envelope giving way, for it is then most fragile and most easily ruptured. There is great variation in the amount of resistance offered by the amnion in different cases. This is notable even to the end of pregnancy, for we sometimes find the

⁴⁴ *Zeit. der Wien. Aerzte*, 1845.

⁴⁵ *Monat. f. Geburt*, Bd. xxix.

⁴⁶ *Schmidt's Jahrb.*, Bd. xxxix.

⁴⁷ *Ibid.*, 1864.

⁴⁸ *Leçons de Clin. Obstét.*, 1872.

⁴⁹ Underhill asserts that cystic chorion, in the majority of cases, is associated with a cancerous or syphilitic dyscrasia.—*Obstet. Gaz.*, 1879.

⁵⁰ *Clin. Mem.*, 1863.

bag of membranes of which the amnion forms an integral part so thin that the first uterine contractions produce escape of the waters, while in other cases the membranes are so tough as to impede the progress of labour and require puncture for its acceleration. Dubois⁵¹ says the feeble resistance of membranes may be due to natural structure, I suppose as some women may have thinner skins than others, or it may be due to alterations consequent on inflammation. However produced, it may, by facilitating rupture and escape of the liquor amnii in early pregnancy and before the child is viable, lead to intra-uterine death, for it is well known that pregnancy cannot continue long after the entire evacuation of the amniotic fluid.

The question as to whether there is true inflammation of the amnion has been discussed, and various supposed pathological results have been pointed out. Jacquemier details three cases which he regards as clearly indicating the occurrence of inflammation on its free surface. One of the patients had peritonitis, and aborted five days later; another had been kicked in the abdomen at the third month, and although she went to the full time, there was a rapid development in size of the abdomen as the result of the injury, and a large quantity of liquor amnii was formed in the uterus. In all the cases the membranes were thickened and traversed by abundant vessels, and in one the containing fluid was cloudy and whitish in colour, with flakes like curd of milk floating in it, which were portions of false membrane detached from the free surface of the amnion.

The adhesions sometimes noticed between the amnion and the umbilical cord, between the amnion and the fetus, are almost invariably associated with some malformation or monstrosity of the child, and have been attributed to inflammatory action. The difficulty in accepting this theory is the general absence of other inflammatory signs in such cases, besides the mere adhesion and its associated monstrosity.⁵² The natural history of such malformations further points to some entanglements of embryonic structures in the process of development, these resulting in distortion and displacement of normal parts.

The condition which has received most attention in connection with the pathology of the amnion is what has been termed "Hydramnios," or superabundance of liquor amnii. The presence of a certain amount of fluid in the cavity of the amnion is necessary to the development of the fetus, but in some cases it is so abnormally large as to endanger the life of the child, and if it does not necessarily bring peril to the mother, it at any rate places her in very grave discomfort. Dr. Kidd, who wrote on the subject in the *Proceedings of the Dublin Obstetrical Society* for 1878, limits the term "hydramnios" to those cases in which more than two quarts of amniotic fluid are present. The quantity of fluid is sometimes very large. Baudelocque gives one case where thirty pints escaped from the uterus; other authors give thirty or forty pints. Coming on suddenly in some instances, more slowly in others, the fluid so distends the uterus that it acquires a volume entirely disproportioned to the stage of pregnancy. The result to the mother is that she may suffer greatly from the distension. Respiration is impeded, and the patient may not be able to lie down night or day. The digestion is deranged from physical pressure, and the heart's action is disordered, while other secondary symptoms, such as oedema of the lower limbs and blueness of the countenance, may be superadded.

The effect on the child is to impede its development, and, if born alive, it is commonly too feeble to survive its birth. Frequently the child is affected with dropsy, hydrocephalus, or other complication. Of thirty-three cases mentioned by McClintock, nineteen children only were born alive; ten of these died a few hours after birth, and nine survived. Often the fetus dies *in utero*, and may then be retained a considerable time after its death, thus prolonging the discomfort of the mother. The condition has a tendency to repeat itself in the same woman. I saw a patient some years ago in her tenth pregnancy, who was sent to me by Dr. Matthews Duncan, and who had suffered from hydramnios in nine out of the ten gestations, losing most of her children, the first pregnancy being the only healthy one.

There is an extensive literature on the subject, but little is known as to the cause or causes. Mercier noted traces of inflammation in the amnion, but Dubois and Desormeaux cite cases where there was no appreciable lesion of the membranes, and there are no constant appearances in the placenta associated with the condition. Churchill attributed it to excessive secretory activity in the amnion, and

various other explanations have been given, but they do not add much to the precision of our knowledge of its causation.

In the case I have alluded to as occurring in my own experience, there was nothing to account for the phenomena beyond a short residence in India, which had impaired the general health, and I found no albumen in the urine.

It has been said to be rare before the fifth month, but in going through the museums, I find many examples of it in the earlier periods of pregnancy; and both from this and my own experience, I infer that it is far from infrequent in the first half of gestation. In St. Mary's Hospital there is a specimen in which the amniotic cavity is from three to four inches long, and the embryo is not larger than a horse-bean. In St. Bartholomew's, again, there is a large amniotic cavity with no embryo at all.

Merriman,⁵³ Robert Lee,⁵⁴ and others attribute hydramnios to a morbid state of the mother, and particularly to the effects of syphilis. That the liquor amnii may be affected by the state of the mother is evident from many facts. Dr. Duncan,⁵⁵ in a paper on Puerperal Diabetes, states that hydramnios is frequent in association with this condition, and sugar has been found in the liquor amnii. Two or three facts have been distinctly made out in relation to its pathology.

1. Scarpa⁵⁶ noticed a coincidence between this form of dropsy and the presence of twins. This has been fully confirmed by the observations of Winckel,⁵⁷ McClintock,⁵⁸ and others.

2. In pregnancies complicated with hydramnios, the child often presents some vice of conformation. Monsters are, in fact, common with this affection.

3. The children born with a superabundance of liquor amnii often present evidences of syphilis, and this whether they are born dead or alive.

4. Hydramnios is comparatively rare in first pregnancies as compared with others.

5. It is more frequent with female children than males. Of thirty-three cases collected by McClintock, only eight were males.

The impression derived from a careful study of all the circumstances is, that hydramnios is not a product of inflammation of the amnion, as some have supposed, but that it arises sometimes from constitutional conditions affecting the mother, sometimes from local causes. The idea of local origin, in some cases at least, is strengthened by the fact that it may occur with twins, one only being affected; and Charpentier mentions a case where the abdomen increased rapidly after local injury from a fall at the fifth month, and the woman was delivered of a dead child at seven months with seven to eight litres of liquor amnii. In this instance, the placenta was found to be diseased to a third of its extent. It would thus seem to be due to some fault in development, as cystic chorion is a fault of this kind.

ON THE ALLEGED DANGERS OF STARCH-CONTAINING FOODS DURING THE PERIOD OF INFANCY.¹

By ROBERT WILLIAM PARKER, M.R.C.S.ENG.,
Surgeon to the East London Hospital for Children, Shadwell, E.

SIR GEORGE CORNEWALL LEWIS, in his admirable essay on the Influence of Authority in Matters of Opinion, defines the "principle of authority" as the principle of adopting the belief of others, on a matter of opinion, without reference to the particular grounds on which that belief may rest. I do not think I am wrong in asserting that the belief in the dangers of starch-food during infancy is widespread, and generally accepted in our profession.

It appears to me that it may be advantageous to inquire into the grounds on which this belief rests; whether, in fact, it exists as an abuse of this principle of authority, and whether the grounds on which it rests are sufficient and trustworthy. In order to get a decided expression of opinion on this point from the Fellows present, I will somewhat dogmatically state the opinion I have come to adopt after many years' work in a children's hospital, namely, that the dangers of starch-foods are very greatly exaggerated. I may even go further, and say that I have never myself met with cases in which I could distinctly and directly trace disorder to starch-foods.

⁵³ *Synopsis*, etc., 1838.

⁵⁴ *Theory and Practice of Midwifery*.

⁵⁵ *Obstet. Trans.*, 1882.

⁵⁶ *Opuscoli di Chir.*, 1825-32.

⁵⁷ *Path. der Geburt*.

⁵⁸ *Diseases of Women*.

¹ A paper read before the Medical Society of London on April 4th, 1887.

⁵¹ *Diet. en 30 volumes*, Art. Hydrométrie.

⁵² Courty says when the phenomena are produced early, it is by the liquor amnii being absorbed, and the membranes then acquire adhesions to the integument of the fetus. In others, inflammation is the cause, and there are false membranes formed from the amnion.