

On fatty degeneration of the placenta : and the influence of this disease in producing abortion, death of the foetus, hæmorrhage, and premature labour / by Robert Barnes.

Contributors

Barnes, Robert, 1817-1907.

Publication/Creation

London : [C. and J. Adlard], [1851]

Persistent URL

<https://wellcomecollection.org/works/x2zzfbkr>

License and attribution

This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.



Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
<https://wellcomecollection.org>

From his friend the author
ON

FATTY DEGENERATION

OF

THE PLACENTA,

AND

THE INFLUENCE OF THIS DISEASE

IN PRODUCING

ABORTION, DEATH OF THE FÆTUS, HÆMORRHAGE,
AND PREMATURE LABOUR.

BY

ROBERT BARNES, M.D. (LOND.)

OBSTETRIC SURGEON TO THE WESTERN GENERAL DISPENSARY,
AND LECTURER ON MIDWIFERY.

*[From Volume XXXIV of the 'Medico-Chirurgical Transactions,'
published by the Royal Medical and Chirurgical Society of
London.]*

LONDON:

PRINTED BY

C. AND J. ADLARD, BARTHOLOMEW CLOSE.

1851.

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

THE JOURNAL OF THE

ON
FATTY DEGENERATION OF THE PLACENTA,
AND
THE INFLUENCE OF THIS DISEASE
IN PRODUCING ABORTION, DEATH OF THE FÆTUS, HÆMORRHAGE,
AND PREMATURE LABOUR.

BY
ROBERT BARNES, M.D. (LOND.)
OBSTETRIC SURGEON TO THE WESTERN GENERAL DISPENSARY,
AND LECTURER ON MIDWIFERY.

COMMUNICATED BY
PROFESSOR MURPHY.

Received February 19th.—Read May 13th, 1851.

CASE I.—In October, 1850, Mr. Humby sent me a placenta taken from a lady who had been prematurely delivered. In the sixth month of pregnancy, this lady was seized suddenly, and without pain, with flooding, while walking in the garden, but not having used any extraordinary exertion. The flooding stopped on this occasion, and returned without pain or any obvious cause in about three weeks. This time premature labour followed. The child presented every sign of having been dead some time before delivery.

Appearance of the Placenta.—The uterine surface was studded with fatty masses, varying in size from that of a bean, to one mass which was as large as a pigeon's egg. These masses extended inwards into the substance of the placenta, and the largest occupied the whole thickness, and was visible on the foetal surface.

X Although the difference of structure and consistence between these masses and the normal placental tissue was striking, it was difficult to isolate them by dissection. The placental tissue appeared to have undergone a fatty transformation in the seat of these masses, and not that fatty tumours had become developed independently. The fatty masses were traversed like the rest of the placenta, by branches of the umbilical vessels. Indeed it was obvious from the dissection, as was subsequently demonstrated by microscopical analysis, that the fatty masses were constituted of placental structure variously altered, either by the simple deposition of fat, or by actual degeneration. They were of dense structure and firm consistence, of a pale yellowish colour; the knife used in incising them was rendered greasy; and portions laid upon paper caused a greasy stain. No vestige of blood could be seen in them. They appeared as hard foreign bodies, interspersed in the softer spongy structure of the healthy placenta.

X It was manifest that this diseased condition of the placenta was the cause of the hæmorrhage, of the death of the foetus, and of the premature labour; and I shall presently endeavour to explain in what manner I conceive these events were brought about. I deferred publishing any account of the case until I should receive a report from my friend Dr. Hassall, upon the minute anatomical structure of these fatty masses, having forwarded to him specimens for examination. Dr. Hassall did examine them; but being too much engaged at the time to make drawings and commit to writing an account of his observations, preserved specimens until his leisure should permit him to complete what I required.

CASE II.—In December, 1850, Mr. Humby sent me another placenta for inspection, which had been taken from a patient who had also been delivered prematurely. In this instance there was no hæmorrhage, but labour had come on suddenly at the seventh month. As in the former case, the child appeared to have been dead some time before delivery.

Appearance of the Placenta.—Nearly one-half of the uterine surface was thickly studded with fatty masses, varying in size from that of a pea to that of a walnut. Adipose matter appeared to be aggregated into firm defined masses, isolated from the surrounding placental tissue. No large umbilical vessels could be traced in their substance. In this respect they differed from the tumours in the first placenta: possibly the vessels had been obliterated or replaced by cellular tissue, in consequence of pressure and cessation of function. The minute structure of the masses in this case differed, however, in no particular from that of the first: like them, they were composed of umbilical capillaries and villi, with the investing chorion in various stages of fatty deposition and degeneration. In the grosser physical characters also, the masses found in both placentas entirely corresponded. In this case, as in the former, the masses were chiefly seen on the maternal surface of the placenta, the larger ones penetrating more or less deeply into its substance; some even occupying its entire thickness. There were only two or three small spots in which it could be perceived that fatty masses were being developed on the foetal surface. The masses were of solid consistence, and appeared as hard bodies imbedded in the softer spongy structure of the healthy placenta.

This second case confirmed me in my opinion as to the importance of this alteration in the structure of the placenta. It showed that not only hæmorrhage might be occasioned by it, and hence premature labour; but also that premature labour might be excited in consequence of the death of the foetus, which had, in all probability, been brought about by the necessarily imperfect performance of the functions of the placenta. I forwarded portions of this placenta also to Dr. Hassall, and procured from time to time healthy recent placentas, that we might be enabled to compare the healthy and diseased structures.

While waiting for the drawings of the intimate structure of these fatty placentas, which Dr. Hassall had undertaken

X to prepare for me, I observed in the 'Brit. and For. Med.-Chir. Rev.,' for January, 1851, an account of a case of fatty degeneration of the placenta by Professor Kilian. The case, in its pathological history, nearly resembles the second case related in this paper; death of the foetus and premature labour are correctly ascribed to the placental disease. But I cannot think it necessary, with reference to the cases I have observed, to discuss and to refute, as Professor Kilian does, the possibility of these fatty masses being of post-mortem production, or the supposition that they are the result of the conversion of blood-globules into fat. In the perspicuous statement of the microscopical examination which follows, Dr. Hassall has, I think, clearly established the true character and origin of this important affection in the cases which form the subject of this communication.

I have here to mention that I am not acquainted with any description of fatty degeneration of the placenta, or even with any account of a case of the disease, excepting Professor Kilian's and those recorded in this paper, sufficiently precise and minute to establish its exact nature and importance. In the numerous obstetric works I have consulted, I find only the most casual and general allusion to the disease.

The minute pathological changes observed in the placentas, are accurately described in the following report of Dr. Hassall, and faithfully delineated in the accompanying drawings.

I transcribe the report:

"I am sorry to perceive that the delay which has occurred in the preparation of the drawings, illustrative of the general anatomy of the normal and abnormal placenta, has been the cause of your being anticipated, although to a slight extent only, in the publication of your important observations on that organ; and I regret the circumstance the more when I consider that I am mainly responsible for that delay.

"The pathological changes which occur in the placenta from fatty deposition and degeneration, will be more clearly comprehended if we take, in the first place, a brief survey of the normal structure of that organ.

"The disease in question, so far as my observations have

yet extended, originates in, and is mainly limited to, the foetal portion of the placenta; it is this, therefore, that we shall have to describe more particularly, which, it is to be remembered, however, constitutes almost the entire substance and bulk of the organ.

“The placenta is divisible into numerous lobes and lobules of variable size, and consists of two portions, a maternal and a foetal.

“The *maternal portion* is made up of blood-vessels, arteries, and veins, dilating into sinuses, derived from the mother, and which are covered externally with a layer of cellular decidua (see fig. 1). The cellular decidua is usually described as enveloping each villus, of which it is enumerated as forming one of the coats. This description I have not been able to verify; it is extremely rare to observe this membrane in connection with the villi; and I believe that the *tufts* of villi are *not in union*, but merely *in apposition* with this layer, which covers and dips down between and into the lobes and lobules of the placenta.

“The *foetal portion* consists of the ramifications of the umbilical vein and arteries enclosed in a layer of chorion. Each umbilical artery, after numerous divisions, and when reduced to a certain diameter, gives off consecutively a series of loops, the last of which terminates in a radicle of the umbilical vein. The placental villi are either *simple* or *compound*, according as they consist of a single loop, or are made up of an arterial and venous trunk, with numerous intervening loops. The plan of development of the terminal divisions of the blood-vessels constituting the foetal placenta, is very peculiar, and is not, I believe, generally recognised. Each vessel is *double*, that is to say, the same vascular sheath is divided by a septum into two channels. This formation is best seen in fragments of recent placentas which contain blood, and which have been immersed in a weak solution of common salt, or in injected preparations.

“A second very great peculiarity is the unusually large diameter of the capillaries forming the loops of the villi, and

which is such as to admit two or three rows of blood-corpuscles at a time.

"The walls of the placental blood-vessels, like those of other vessels of the same diameter occurring elsewhere, are thickly studded with elongated nuclei, which, in fresh healthy placentas, are obscured, though not concealed, by the chorion (fig. 3 *a*). It is best seen in fragments of placenta which have been immersed, for a few minutes, in water, or extremely dilute acetic acid. Such fragments, viewed with an object-glass magnifying 350 diam. lin., present to observation innumerable nuclei; some of these belong to the chorion, but others to the vessels beneath. Skirting the margins of the vessels will be noticed, in particular, a series of cells of a slightly oval form; these belong exclusively to the chorion (fig. 3 *a*). In perfectly fresh placentas the chorion adheres so firmly to the vessels that it is difficult to detach it; when, however, the placenta has been kept a day or two, it separates readily enough, and in fragments torn up with needles, the membrane will be found, in some cases, to be attached to the vessels, and in others to have become removed (fig. 3 *b*).

"Such is a very brief outline of the structure of the normal placenta. In the *placenta affected with fatty degeneration*, certain of the lobes, in place of presenting the red spongy texture of healthy tissue, exhibit a fatty appearance, and are of a yellow colour, glistening, firm, and exsanguine, while the remaining lobes present their ordinary characters, at least to the unaided eye.

"I will now endeavour to determine the nature of the changes which have ensued in these altered lobes, and examine both the maternal and foetal portions of the placenta as contained in them.

"The maternal portion, as already described, consists of the fibrous walls of the internal blood-vessels, covered on the outside by cellular decidua. Both these structures presented, to a great extent, their normal characters; the walls of the vessels were nucleated, and the cells of the decidua were of the usual size. The important difference, however, was

noticed that both vessels and cells were studded over with numerous minute spherules, some of which appeared to be upon the surface, but others evidently were contained within the coats of the vessels, and in the cavities of the cells (fig. 2). The maternal portion of the placenta was therefore not free from disease.

"The foetal portion of the placenta, as already noticed, consists of the umbilical vessels and chorion. On placing a small portion of one of the diseased lobes in water, the first thing which strikes the observer, is that the tufts of villi do not expand or float out in the same way as in the healthy placenta, and on endeavouring to separate the fragments into its component villi with needles, the extreme brittleness of the whole structure becomes apparent. Examined with the half-inch object-glass, the villi are observed to be much broken up, and darker than usual, especially near their terminations, which reflect a yellowish colour. Viewed with a glass of 420 diam. lin., a variety of structural changes are detected.

"1st. We observe that the villi are thickly studded with innumerable minute spherules of oil (figs. 4, 5).

"2d. The chorion is much altered; it is thickened, and destitute of nuclei.

"3d. The walls of the vessels no longer contain nuclei; these having, in all probability, become degenerated into spherules of oil (fig. 5).

"4th. The spherules of oil are contained, some in the chorion, some in the walls of the blood-vessels, and many in the intervals or spaces between these (fig. 5).

"5th. The cavities of the vessels are almost invariably free from fatty deposition.

"6th. The vessels are destitute of blood (figs. 4, 5).

"Such was the usual condition of the several component structures; sometimes, however, in places, the disease appeared to have progressed still further, and to have produced almost complete disorganisation and disintegration of tissue.

"Turning our attention to those lobes of the placenta which, to the eye, present a normal appearance, we detect

in them manifest evidences of the same destructive changes in progress ; considerable fatty deposition is visible, and the nuclear structure of the parietes of the blood-vessels and of the chorion is, to some extent, implicated (fig. 4). In these portions, the distribution of the oil-molecules coincides exactly with the course of the blood in the vessels. This observation shows that the condition of the blood itself is intimately connected with the origin of the deposit.

“ It must not be forgotten, however, that a very small quantity of fat, in the form of minute spherules, is almost constantly present in the placenta.

“ In order to judge how far the pathological changes in the placenta resulting from fatty degeneration above given, accord with those observed by Professor Kilian, I will quote his description.

“ ‘ Examined with the microscope, the *extreme ends of the vessels* of this portion were found to form little knobbed swellings composed of *fat-globules*, strongly reflecting the light. From these ends of the vessels filled with fat droplets closely packed together, the blood-corpuscles of the placental vessels were quite absent ; but in proportion as the vessels were traced back from their terminations, the fat-globules were progressively replaced by blood-globules, the walls of their vessels which, at their terminations, were also loaded with fat, recovering their natural appearance. The remotest ramifications of the healthy portions of the placenta continued entirely normal ; and where the yellow portion joined the coloured, many of the terminating vessels were destitute of fat ; others contained droplets, but none showed the dense masses observed at the uterine surface.’

“ I will quote another passage conveying Professor Kilian’s remarks on the origin of this disease. ‘ But it may be inquired whether this fatty formation occurs during the healthy conditions of life, and becomes the *causa mortis* ; whether it results from decomposition after death, or whether, originating during life, it may be but the expression of other causes, inducing the degeneration of the ovum, and a symptom of retrogressive metamorphosis of the placenta.

On examining the foetus, all parts were found apparently normal, except a contused state of its head and face, and a large effusion of blood between the scalp and the cranium; so that it might be inferred that the child died from some cause of pressure on the brain, and the described changes in the placenta were only the product of decomposition consequent on its death. Against this view, however, many objections may be urged. There is no fact known of the conversion of stagnant blood into fat. Virchow and Reinhardt have only seen the formation of fatty corpuscles within the colourless blood-corpuscles. The changes which occur in the terminations of the blood-vessels, or other metamorphosis after death, authorise no such view. The author, too, (Professor Kilian) after macerating healthy placentas in water until approaching putrefaction, never found this obturation produced, a few droplets here and there being formed only on the walls of the vessels.'''

Connected with the anatomical facts thus related by Dr. Hassall, are various physiological, medical, and obstetric considerations of great interest and importance. Some of these I will endeavour to point out as comprehensively as possible.

The researches of Rokitansky, Bowman, Handfield Jones, George Johnson, R. Quain, Paget and others, taken collectively, fully assert the prominent position which fatty degeneration is destined to occupy in general pathology. To demonstrate the existence of a disease in the placenta, an organ intermediate between the mother and her offspring, similar in its nature, development, progress, and termination to that which the observers I have referred to, have shown to be of such importance, when affecting the liver, kidneys, heart, muscles, vessels, and other structures of the body, cannot be uninteresting. The demonstration of fatty degeneration in the placenta, not only supplies a new chapter in the history of the disease, aiding in the acquisition of a more comprehensive knowledge of its nature, but it also throws a new light upon a question of the deepest interest in the study of this and many other of the most formidable diseases. Long-con-

tinued observation and accurate analysis have established the hereditary nature of insanity and phthisis; our acquaintance with fatty degeneration is too recent and too little advanced to have admitted of any extended investigations into the transmission of this disease to successive generations. There exist, therefore, at present few facts from which any trustworthy conclusion can be drawn. The occurrence, therefore, of fatty degeneration in the placenta, the organ by means of which the embryo derives the materials for its growth from the mother, and in which the elements of mother and child are being constantly interchanged, affords the strongest presumptive evidence, that the germs of that disease which pervade the system and circulate in the blood of the mother, may be directly transmitted to her offspring.

In support of this view, that the placenta may be the medium through which the various morbid diatheses may be propagated from the mother to the embryo, and also as affording further proof that the placenta is in nowise exempt from those diseases which affect the permanent organs of the female, it deserves to be remembered, that the placenta may be attacked by inflammation and congestion; that it may be the seat of the deposition of fibrin, cartilage, bone, and even cancerous matter; and the case of a phthisical patient is related¹ in which small crude tubercles were formed on the external surface of the uterus, under the peritoneum, and eight or ten on the uterine surface of the placenta.

With regard to the frequency of fatty disease in the placenta, it is at present difficult to form an accurate opinion. I am in the habit of minutely examining the placenta on every opportunity; and I have not unfrequently seen fatty masses in various stages of growth in placentas expelled at the full period of gestation. In these cases it is to be presumed, that the balance of healthy placenta preserved was sufficient for the development of the fœtus. There are certain physiological considerations which render it probable,

¹ M. Hardy, 'Arch. Gén. de Méd.,' Juin 1834.

that the placenta is especially prone to become the seat of fatty deposition. The decarbonising function of that organ is one; but there are several others which deserve attention. Pregnancy itself appears to predispose to the formation of fat. The increased proportion of fibrin in the blood; the tendency to albuminous urine; the constant presence of fat in that secretion; the secretion of fat in the breasts; and the observation that a certain amount of free fat is always present in the healthy placenta, constitute a series of facts, which, viewed in their relation to each other, may elucidate those morbid processes which lead to the production of an excess of fat in the circulating system. Certainly, the elimination of this material from the economy, is obstructed by the encroachment on the cavity of the chest, the pressure upon the liver, kidneys, and other abdominal organs, and the growing disinclination to exercise, which attend the progress of gestation.

The observation of fatty degeneration in the placenta presents another point of interest with reference to the study of the general pathology of that disease. It furnishes an example of the *rapidity* with which it may proceed, which is capable of close limitation. The placenta being a caducous organ lasting but a few months, all the pathological changes observed in it, we know, must have been wrought within a certain brief period of time. When we find, therefore, that the disease has proceeded from simple deposition to degeneration, and even disintegration of the original tissues in a placenta expelled at the seventh month of pregnancy, we are in possession of accurate data by which to form an estimate of the rapidity with which these changes may occur. No similar data can be obtained with reference to the like changes in other organs.

I will not, however, dwell longer upon the general relations of fatty degeneration of the placenta, as I am anxious to advert to the practical bearings of the disease on obstetric practice.

The effects that must result, sooner or later, according to the rate of increase of the morbid alteration of an organ so essential to the foetus as the placenta, are not difficult to

understand. In exact proportion as the growth of the embryo requires a larger and more unimpeded extent of placental structure, in which its blood may be brought into multiplied points of contact with the blood of the mother, large portions of placenta are being gradually rendered useless for this purpose; and the coats of the villi throughout the remainder are probably undergoing a disorganisation which must materially impede the performance of their function, of permitting the interchange of elements between the maternal and foetal blood. If this conversion of healthy placental structure proceed at such a ratio as to exceed the normal increase of healthy placental tissue, a period must arrive when the healthy portion of placenta will be insufficient for the production of those changes in the blood of the foetus which are essential to its growth and life. When this period arrives, then, the foetus will perish, and premature labour will follow,—unless, indeed, premature labour be induced during the life of the foetus by a process to which I will next advert.

One of the conditions essential to the perfect cohesion of the placenta to the uterus, is the preservation of the uniform spongy structure of the placenta. The whole uterine surface of the placenta must present an equal consistence, so that every part may permit of equable distension, whether from the maternal or the foetal circulating system, and be equally adapted to the opposing surface of the uterus. This condition is necessary to enable it to preserve this adaptation throughout the peristaltic movements to which the walls of the uterus become more and more liable as gestation advances. But when, as is the case in these examples of fatty tumours in the placenta, certain parts of the uterine surface are hard and unyielding, while intervening portions are of a soft and yielding character, the entire placenta is no longer in a condition to follow the movements of the uterus and preserve its connections. The hard portions, moreover, in which fat has occupied the walls of the foetal vessels, and by pressure obliterated the cavernous structure, no longer maintain any vascular communication with the uterus. The maternal

or cavernous portion of the placenta being either closed up, or otherwise rendered useless, the channels of communication with the uterus necessarily disappear. Accordingly, in these portions no oblique valvular openings, no remains of arteries, could be discerned, such as usually exist, and such as were observed on the maternal surface of the more healthy portions of the diseased placentas. The consequence of this loss of homogeneity of tissue, and cessation of vascular connection with the uterus, is, that when the peristaltic movements of the uterus assume an active character, as they often do towards the seventh month of pregnancy, or even earlier, the cohesion between the placenta and the uterus is soon destroyed at those points where the fatty masses are largest and hardest. This partial disruption can hardly occur without involving some separation of the placenta not diseased, in the immediate proximity of these masses. Hæmorrhage results; the effusion of blood and the formation of coagula excite increased action of the uterus; further detachment follows; premature labour is imminent; and if it ensue immediately, the child may be born alive. But this event may not occur on the first appearance of flooding, as the first case I have related will show. Should it not so happen, the favorable opportunity for the child will be lost. That amount of disease which is sufficient to cause partial detachment of the placenta is scarcely compatible with prolonged life of the foetus; and in all probability, the foetus will have been cut off from defective nutrition, before a further detachment and hæmorrhage could bring about the expulsion of the contents of the uterus.

I think the two cases I have related are interesting and valuable as illustrations of the two processes just described. In one case, premature labour was induced by partial detachment of the placenta and hæmorrhage; in the other, the premature labour was the result of the death of the foetus. Certainly in one, probably in both, the death of the foetus was occasioned by the gradually increasing obstruction of the placental circulation.

I have thus referred to the different states of fatty

degeneration in the placenta, entailing different consequences to mother and embryo, according to the ratio of increase the disease may have followed. In the first kind, the disease has made so little advance, that the child may be carried in safety to the end of the natural term of gestation. In the second kind, of which I have given two examples, the disease has proceeded at such a rate as not necessarily to involve the destruction of the child. At the seventh month the child is viable, and if, as I have shown, premature labour should, by any means, be induced before the child has perished, a living child may be born. I have now to indicate a third kind or degree, which not only places the mother in danger, but necessarily destroys the embryo. If the disease proceed so rapidly as to have invaded a large portion of the placenta in the *early* months of pregnancy, it may be the immediate cause of abortion.

The various causes of abortion usually enumerated by systematic authors, are mostly insufficient to account for the event. I believe there is a growing disposition to attribute to diseases of the ovum the first place; and those who are in the habit of examining the ova of women said to have aborted in consequence of injuries, mental emotions, habit or other causes assigned to the mother, will, perhaps, be disposed to admit, that such ova exhibit, for the most part, appearances of disease which deserve a large share of attention in determining the cause of the abortion. Observation inclines me to conclude that disease of the ovum mostly precedes its detachment from the uterus; and perhaps future experience will show that fatty deposition is a disease to which the ovum is particularly obnoxious.

As a *cause of hæmorrhage* before delivery, this disease of the placenta is especially deserving of consideration. When this event occurs in the course of pregnancy, it is usual to infer that the placenta has been in part, or wholly, detached. When the placenta has been attached to the cervix uteri, the hæmorrhage is easily explained; but when this is not the case, then I would hazard the opinion that the detachment was preceded by disease of the placenta.

With regard to the treatment or obstetric management of these cases, I wish to offer a few observations. There are few conclusions which have acquired a more settled possession of the minds of obstetric practitioners than this; namely, that patients are liable to a recurrence of affections in future labours similar in character to those which had occurred in former labours. Consequently when a particular anomaly or disease has occurred before, and especially more than once, to place the mother or her offspring in peril, it is an established rule to endeavour, in subsequent pregnancies, to anticipate the expected unfavorable result by timely interference. As a striking illustration of this recurrent tendency of some disease of the placenta, I am tempted to quote the following case:—

In 1847, I saw a placenta exhibited to the Pathological Society, by Dr. Ramsbotham.¹ It was enormously hypertrophied, but did not appear to consist of more than ordinary placental tissue. The patient from whom it was taken had lost six children successively, which had all been expelled putrid. In each instance the placenta had been hypertrophied, adherent, and generally contained indurated masses of lymph. On her being a seventh time pregnant, it was deemed expedient to induce premature labour with the view of saving the child. The attempt was not successful: the child gasped heavily two or three times, and died. I entertain little doubt that the fatty degeneration of the placenta is a disease peculiarly apt to recur in successive pregnancies, and to blight the fruit of successive conceptions. I believe, therefore, that the rule I have referred to finds an appropriate application in this case. When it has been observed that the embryo has been lost in one or more pregnancies, in consequence of this disease, it must be a matter for serious consideration whether the induction of premature labour should not be recommended in a future pregnancy.

Many other points having more or less bearing upon this

¹ Transactions of the Pathological Society, 1847-8.

subject present themselves ; but I am anxious that this paper should not be extended to an improper length ; and that its more practical features should not be overlaid by speculations. In concluding, I cannot but express my sense of the many imperfections of this paper. But however imperfect, I am not without hope, that this account will be accepted as a useful contribution in extending the knowledge of an important subject in general pathology ; and in directing attention to a source of danger to mother and child, comparatively unnoticed in obstetric practice.

APPENDIX.

Since the foregoing paper was submitted to the Society, I have, through the kindness of Mr. Bartlett, of Notting-hill, been put in possession of the following case, illustrative of a new feature in the history of granular deposit and degeneration of the placenta.

A lady, about three months and a half pregnant, was riding over a very rough road in a dog-cart ; she was much shaken ; and making a false step in getting out, she suffered further succussion. Symptoms indicating the death of the fœtus followed, but miscarriage did not ensue. She went on to what she calculated to be the full term of gestation before labour took place. The ovum was expelled entire ; it was found that the embryo had attained the development only of the fourth month ; there was no sign of putrefaction. The placenta was an inch in thickness, nodulated in parts, universally firm in texture, and of a yellowish-white colour. It had undergone general granular metamorphosis. The entire ovum has been preserved in spirit for some months ; but the adipose character of the placenta was remarked by Mr. Bartlett in the recent state. Portions of it have been submitted to microscopical examination by Dr. Hassall and myself, and appearances identical in character with those described in the placentas of the two cases related in my former communication have been ob-

served; the main difference being, that in this instance every part of the organ is affected.

There can be little doubt that the foetus really perished in consequence of the succussion the lady experienced in the early period of pregnancy. Whether the ovum had been previously affected with a disposition to granular degeneration, or any other disease, must be a matter of conjecture only. In all probability the granular metamorphosis of the placenta, if it did not take its rise at that date, was, at all events, completed subsequently to the death of the foetus. During this process of conversion, the placenta probably preserved its connection with the uterus. One singular purpose it evidently fulfilled, was the preservation of the ovum from putrefaction, and its retention in the womb for some months after the death of the foetus.

Authors have recorded cases in which the placenta has been retained for a lengthened period after delivery, without giving rise to those symptoms which usually attend putrefaction or the presence of a foreign body in the uterus. Saxtorph even says, "I have reason to believe that a placenta, which is entire and uninjured, enclosed in the uterus, adherent to it, and shut out from access of air, *never becomes putrid.*"

In many of these cases it is highly probable that granular metamorphosis is the conservative process.

In connection with the present case, I am desirous of defining the meaning I wish to attach to the terms "fatty deposition" and "fatty degeneration," more especially as they are apt to convey different ideas to different pathologists.

Fat, properly so called, is a normal organised structure, consisting of blood-vessels and cells, the cavities of which contain oily matter. This, like other organised structures, is subject to various lesions, such as hypertrophy and atrophy, or degeneration. It is to this latter condition, as affecting the organised adipose tissues, that the term "fatty degeneration" ought in strictness to be confined. This, the true fatty degeneration, differs entirely from that affection of the



placenta which I have, in my first communication, brought under the notice of the Society. In this affection of the placenta there is no formation of fat, but deposit of oil in the form of minute granules or spherules, and consecutive degeneration of the structures in which the oil is deposited. "Granular oily deposit in, and consecutive degeneration of, the proper tissues of the placenta," would be a more correct and precise expression to indicate the nature of the changes which that organ undergoes in consequence of the affection which, for the sake of brevity, and uniformity with others, I have denominated "fatty degeneration of the placenta." In its anatomical characters it corresponds with that affection of the liver and kidney to which the term "fatty degeneration" has been generally applied.

This distinction between true fatty degeneration and granular deposit, with accompanying degeneration, is important to bear in mind; it has been pointed out by several recent pathologists. By many the term "fatty deposit" is applied to the abnormal development of true fat; and "fatty degeneration" to the deposit of oil and degeneration of structure which occur in the liver, kidney, &c. Employed in the above sense, it is obvious that these terms are inaccurate.

The granular oily and disorganising affection of the placenta may manifest itself under very different conditions:—

1st. It may, no doubt, occur during life as the result of the transformation of fibrin or albumen effused through inflammation. We have analogous examples in the change which clots of blood, fibrin, &c., undergo in other organs of the economy. I have examined a placenta in which osseous matter, fibrin, and granular degeneration were all observed; but I was unable, in this instance, to determine whether or not the granular degeneration was the result of changes effected in the bony matter, and fibrin previously effused. The converse sequence of events may hereafter be observed. It is possible that the coats of the umbilical vessels may be so weakened by granular degeneration, at a period when blood is still circulating in them, that rupture and hæmor-

rhage may ensue. This may be one cause of placental apoplexy, resembling that form of cerebral apoplexy described by Mr. Paget as occurring as a consequence of granular degeneration of the capillaries of the brain. 1x

2dly. It may originate in functional derangement either in the placenta or in remote organs, and apart from organic lesion. I believe the two cases which form the subject of the first communication must be referred to this head. It would appear that organs having a decarbonising function to perform,—as the liver, kidney, and placenta,—are especially prone to suffer granular oily degeneration.¹

3dly. It may occur as a post-mortem change, and resemble the metamorphosis of muscular tissue into oil, and the formation of adipocire. Of this form, the case recorded in this Appendix offers a striking example. Although of less importance in relation to obstetric practice, it is still not without pathological interest to establish the fact, that granular metamorphosis may take place in the placenta while retained in the living womb.—May 24, 1851.

¹ Dr. Mackenzie brought me (May 29th) a placenta quite fresh, healthy-looking, of average size. It had been expelled at full period; the child was living; the mother had enjoyed good health throughout gestation. Extending over about half the foetal surface, under the membranes, there is a layer of a glistening yellowish-white substance, exactly resembling true fat. On making a section, this layer was found to be in parts nearly half an inch thick. In colour and firmness it offers a remarkable contrast with the healthy placental tissue. It may be separated into two or three laminae but is intimately adherent to the proper placental structure. This fatty-looking substance was examined while quite recent with Dr. Hassall. The basis of it was found to be *fibrin*; it showed obscure fibrillation, but no organic structure. From whatever part a section is taken, innumerable spherules of oil are observed in it. This specimen offers a clear example of an effusion of fibrin in the placenta undergoing granular degeneration.

PLATE I.

FIG. 1.—*Normal Placenta.*

Portion of the maternal placenta.

A. Wall of cell.

B. Cells of decidua.

Magnified 420 times.

FIG. 2.—*Abnormal Placenta.*

Portion of maternal placenta affected with fatty degeneration.

A. Wall of blood-vessel.

B. Cells of decidua.

Magnified 420 times.

Fig. 1.

MATERNAL PLACENTA.

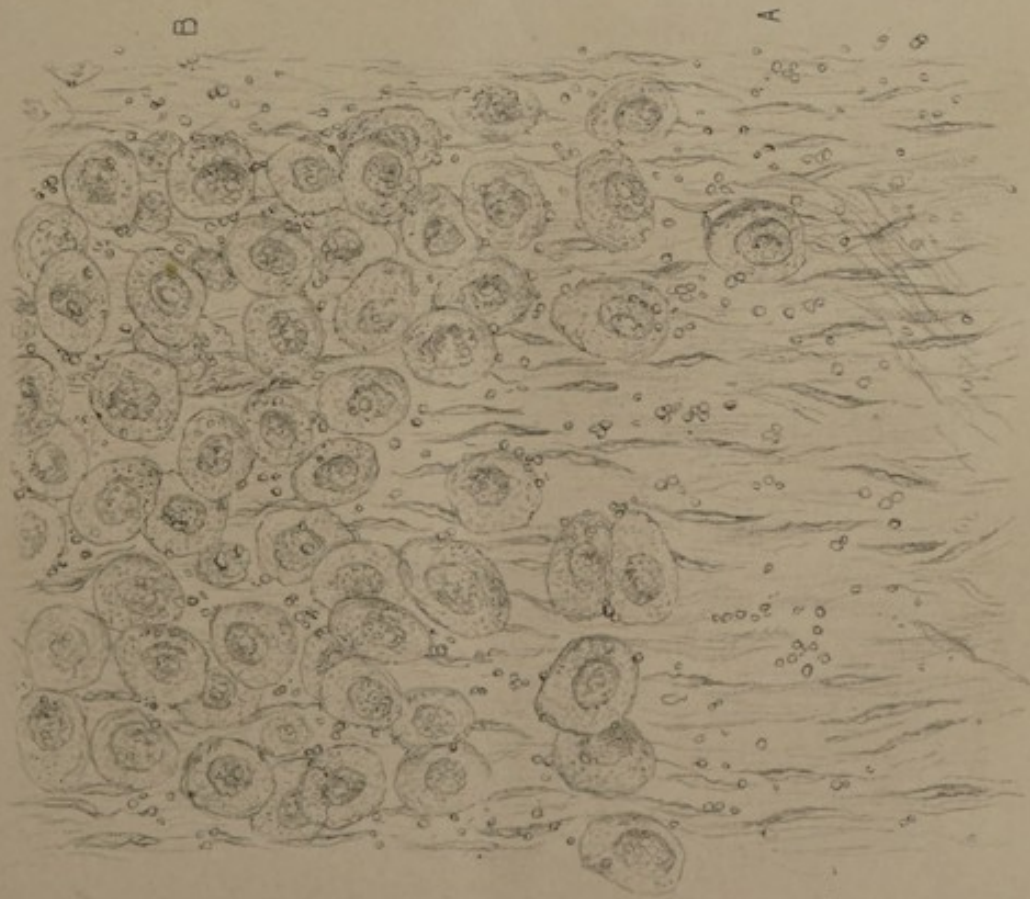


Fig. 2.



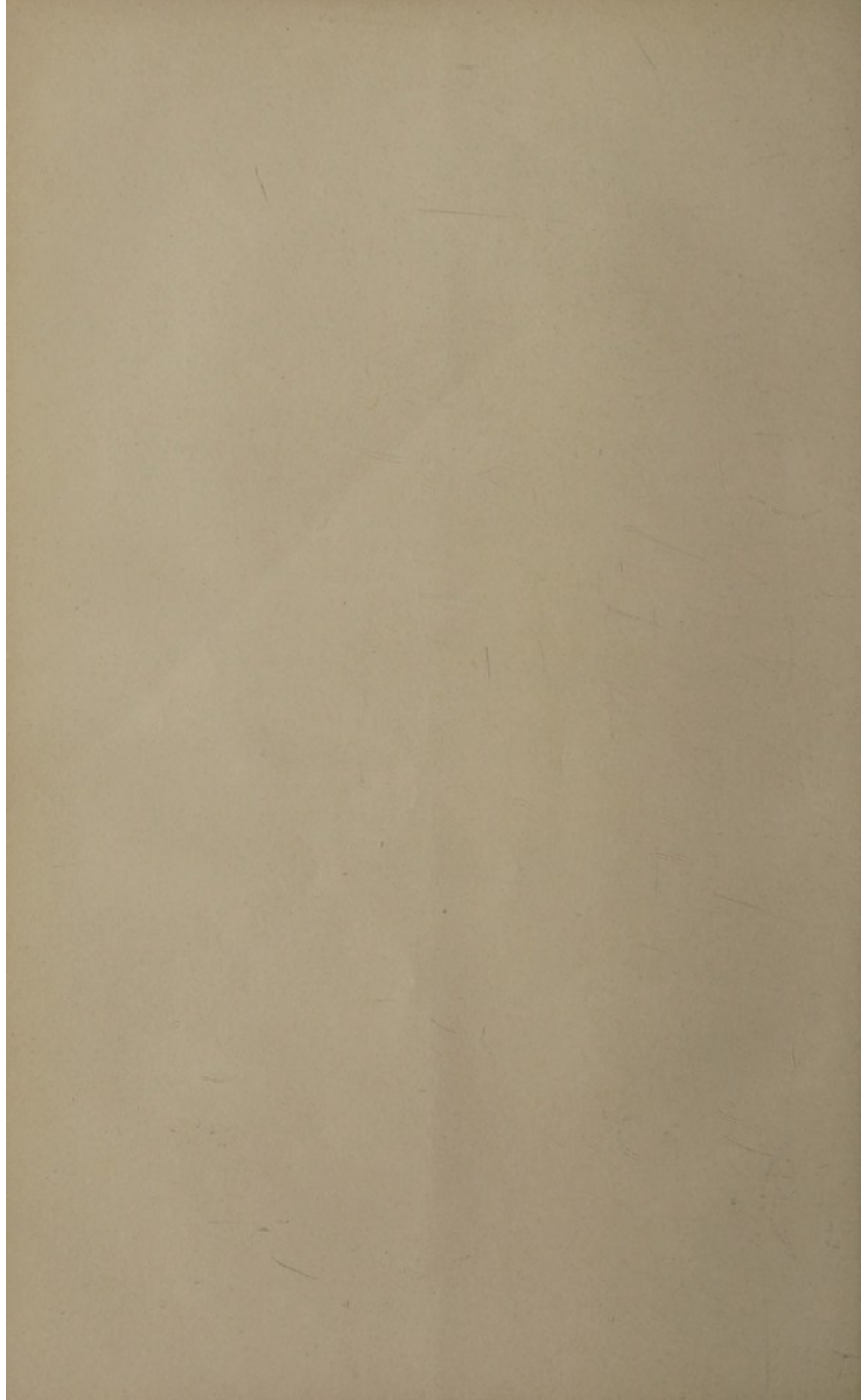


PLATE II.

FIG. 3.—*Normal Placenta.*

Portions of a villus.

- A. Vessel with its investing chorion.
- B. The same deprived of its chorion.
- C. Chorion detached, showing its cellular formation.

Magnified 420 times.

FIG. 4.—*Abnormal Placenta.*

Villi affected with fatty degeneration in an early stage.

- A. Vessels invested with chorion.
- B. Vessels denuded of chorion.

Magnified 220 diameters.

FIG. 5.—*Abnormal Placenta.*

Portions of a villus affected with fatty degeneration.

- A and B. Branches partially denuded of their investing chorion, and showing much fatty deposition in the form of minute spherules.

- C. A blood-vessel entirely deprived of its chorion.

Magnified 420 diameters.

Fig. 3.

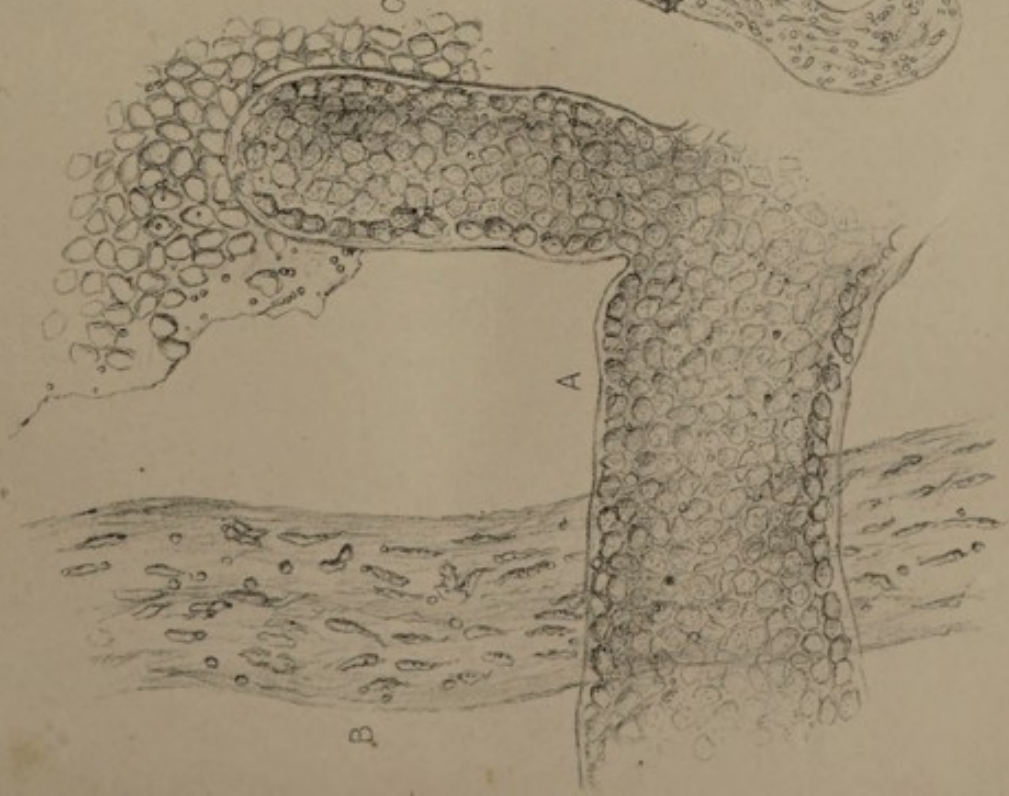


Fig. 4.



Fig. 5.



