## THE CAUSES OF DEATH OF THE VIABLE FETUS BEFORE LABOR.\*

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A CONSIDERATION of this subject, in order to bring the discussion within reasonable bounds, makes it advisable to consider that class of cases in which the fetus, having attained to the eighth month of pregnancy, for reasons often apparent, sometimes doubtful, and occasionally absolutely inexplicable, dies without any of the contributing trauma of parturition. This eliminates the cases in which the partly formed ovum, or nonviable fetus perishes in utero from the results of unhealthy endometrium, or faulty development, and brings us to the consideration of the fetus which has become viable.

The continued health of the child in utero depends on the mother through the amount of nourishment furnished, and poison eliminated, through the placenta as the intermediary organ of a imentation, respiration, and elimination, and upon the healthy functionating of the fetal organs. The factors of fetal death, therefore, might be classified as, first, maternal; second, intermediate conditions, due to invo vement of the placenta; third, conditions intrinsically fetal.

But of most of these classifications it will be at once recognized that they overlap, that is, that any maternal condition may affect placental health; that placental damage invariably results, from fetal disturbances, and vice versa. So delicate is the adjustment between the two living organisms, that disturbance of one must result to some extent in disturbance to the other. For the safety of the child nature has interposed certain well-recognized means of protection between it and the maternal organism—in a physiological sense, by the layer of syncytial cells which is a barrier against invading parasites. In a mechanical sense, by the water-bag inside the membranes, and by the further protection of a staunch wall of uterine muscle.

The toxemia of pregnancy is a factor of very grave importance. Be its origin due to the wandering syncytial cell embolism of Veit or to the new fibrin theory of Dietz, or whatever it may be,

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its danger to the child in utero is unquestionably great. If the onset of the poisoning has been gradual, the explanation is obvious through the tremendous destruction of the placental tissue by infarcts, evident by gross inspection. The maternal toxemia also reacts on the child's vitality. In proof of this, postmortem examination of fetuses dying of eclampsia shows visceral lesions similar to those of eclampsia. There is also added the danger from carbonic-dioxid poisoning occurring during the time of convulsions. The cases of pure toxemia, whether eclampsia, hyperemesis, or coma, are difficult to differentiate in many cases, and may be uncomplicated by nephritis, associated with acute nephritis, or with chronic nephritis.

As a general rule it may be observed that with acute nephritis the death of the fetus is common, and with chronic nephritis, stil more common. So frequent is this condition that it often seems as if the death of the ovum was one of nature's conservat ve processes. Davis says that the tendency of nephritis is to rescue the mother at the expense of the fetus through interstitial changes in the blood-vessels of the placenta.

In another class of cases true uremia seems to exist, distinct from the toxemia of pregnancy. To determine how frequently the death of the fetus before labor occurs, a series of 100 eclampsia cases at the Sloane were analyzed, and in 15 per cent. of these the fetus was found to have evidently died some time before the beginning of labor. Of these, 6 per cent. had not reached the time of viability, leaving 9 per cent. of deaths of viable children, before labor, in cases of eclampsia. Unfortunately it was impossible to determine in these cases the degree of relationship of the eclampsia to kidney disease, nor do these statistics show the effect of the toxemia of pregnancy on intrauterine life, because we are only considering cases that have progressed to real eclampsia, leaving out of account the many cases of toxemia which do not culminate in convulsive seizures

To get at this more accurately, there were collected all the cases of fetal deaths antepartum in a series of 10,000 consecutive deliveries at the Sloane Maternity. The evidence of the time of these deaths was clearly set forth by the history of the patient and the degree of maceration of the infant. A series of 229 cases was secured. Of these forty-seven or 18 per cent. showed evidence of toxemia or renal defect, that is, nine were eclamptic, two had had hyperemesis, and the rest showed the presence of albuminuria.

Sixteen or near y one-third had a notable degree of accidental hemorrhage. So frequently is a mild degree of albuminuria found to exist with death of the fetus, and with infarcted placenta, that the question arises whether the so-called physiological kidney of pregnancy is not often evidence of a much more serious condition, as far as the child's life is concerned, than is usually supposed. The presence of albumin in the urine, should, in my opinion, always be regarded with suspicion. Another renal condition, in which fetal death has been reported, is with high temperature associated with pyelitis. Diabetes in pregnancy, possibly developing at that time, while a rare complication, has a most deleterious effect on fetal life. Most of the fetuses die in utero and the majority of these before the period of viability.

Endocarditis is a maternal condition, if advanced, with serious consequences to fetal life. The placenta in these cases shows occasional hemorrhage due to circulatory status. The fetal deaths in these cases are from malnutrition, or from asphyxia, from placental interference with the circulation, or from carbonic-dioxid poisoning from maternal asphyxia. There is also ncreased likelihood of renal insufficiency.

Exophthalmic goitre has been noted as a cause of fetal death, usually from the asphyxia associated with mechanical pressure of the growing gland against the trachea, although the interference with normal metabolism due to faulty thyroid secretion may be a factor. It seems possible that the parathyroid secretion may be one of the essential factors in the neutralization of the toxines of eclampsia.

Of disturbances of the blood structure, pernicious anemia, although rare, has been found to be peculiarly fatal to the child. Findlay, in a recent review of the literature of these cases, reports that in a majority of instances the child dies in utero shortly before birth.

Chorea is a maternal condition, probably partly due to nervous susceptibility, partly to toxemia. While comparatively rare, in its severer forms, it exacts an intrauterine mortality in one third of the cases.

Of acute maternal infections, pneumonia is a frequent cause of fetal death. Asphyxia may be the immediate factor but there is also the possibility of hyperpyrexia. It is well known that high temperature, even for a short time, is poorly borne by the fetus in utero. Rarely the pneumococcus may invade the fetal circulation through the syncytial membrane.

Typhoid, and recently paratyphoid, have been proven to be transmissible to the fetus, though probably rarely, and so may destroy fetal life. Colive, of Johns Hopkins, has recently reported a still-birth, in which the bacillus typhosus was obtained in pure culture from the amniotic fluid and heart-blood, spleen and umbilical vessels.

Malaria is dangerous to the fetus because of the fatal effect of sudden hyperpyrexia, and of the cachexia sometimes occurring. The transmission of the plasmodium to the fetus is a matter of extreme improbability, as lesions of the placenta are not caused by the infecting organism.

Severe attacks of influenza have been noted to have a tendency to cause premature labor, and of these labors the majority terminate in still-births. Cholera acts virulently on the fetus, probably from direct infection.

The acute exanthemata are of especial interest because of the common transference of the specific organism through lesions of the placenta. In addition to this factor are the effects of high temperature and asphyxia. In measles a maternal pulmonary lesion may be the cause of fatal ending for the fetus. In scarlatina a renal lesion may be the actual cause of fetal One of the cases in my series gave birth to a still-born macerated fetus, at the sixth month, showing the partly healed skin lesions of variola. The mortality of the fetus in variola is placed by Oueriel as 50 per cent. Lesions of the placenta are unusual, but in hemorrhagic cases the placental attachments are injured.

Syphilis is the infection most commonly existing in cases of the death of the child in utero, and yet these cases of the death of the viable fetus are only a small part of the total number of cases of syphilitic fetal mortality, that is, when compared with the number of syphilitic abortions and fetal deaths postpartum. Of the total of 229 deaths in this series, forty-eight were syphi-There were sixty-two cases in which the cause of the death could not be determined from the facts on hand. doubtedly some of these were of sepecific origin. It will be interesting to learn positive y in view of our knowledge of the spirocheta, and of the reliability of Wasserman's serum test, how many more cases of fetal death are due to this infection. Probably the percentage will be much larger than here shown.

So far the following facts have been demonstrated clearly. The presence of the spirochetæ in the placenta, the maternal part as well as the fetal portions, and more commonly in the blood of the fetus, and in lesions of the liver and of the lungs. Further, the practically constant positive reaction of the mother's serum to the Wasserman test in cases of fetal syphilis, seems to point the way to the discrediting of Colle's law. Pauli, in the November number of the Johns Hopkins Bulletin, believes that the spirocheta is rarely found in the syphilitic placenta, and that the anatomical changes in the placenta are the result of toxins produced by the spirochetæ in the fetal organs.

The immediate cause of fetal death is usually the placental condition. The constant lesion has been described as "profuse productive chorionitis." This shows on gross inspection a thickened and heavy placenta, often twice the normal weight, lighter in color, ranging to a light fatty appearance. The microscopic examination shows an hypertrophy, malformation, and friability of the villi, with thickening of the intima of the chorionic vessels, and with much infiltration and increase of the interstitial tissues. The vessels of the cord also show great thickening. There is more or less mottling of the placenta from infarcts. The fetus is subjected by these conditions to a gradual process of starvation and asphyxia. A thickened amnion is a frequent cause of hydramnios.

There is also the fairly constant factor of fetal infection, acting upon the child's vitality, and shown by the interstitial and occas onally gummatous changes in the liver and lungs. The blood-vessels show an obliterating endarteritis and periarteritis. There may be such obstruction of the ductus arteriosus as to cause death.

As to the effect of the time of the infection, the following points are well established: First, that recent infections are the more virulent; and conversely, that time shows in many cases a tendency toward lessened fetal involvment; second, that infection acquired in the last third of pregnancy may not be conveyed to the fetus; third, the marked effect of antisyphi itic treatment in preserving fetal life.

The other common chronic maternal infection, tuberculosis, infrequently acts as a cause of still-birth. True fetal infection by tuberculosis is quite rare. Warthin and Cowie in 1900, reported the careful examination of a still-birth from a fatal case of acute miliary tuberculosis. They reported agglutination thrombi in the maternal sinuses, enlarged caseous areas throughout the decidua. Tubercle bacilli were found in numbers in the thrombi

and in the blood of the sinuses. In the fetal portion, agglutination thrombi were found in the intervillous blood spaces. the fetal circulation numerous virulent tuberc e bacilli were It is considered demonstrated, but no tuberculous lesions. undecided but unlikely that the bacilli pass through undamaged syncytial tissue. In fetal death associated with tuberculosis, it is more apt to be the maternal condition, with its fever and asphyxia that causes death.

Maternal conditions against which the fetus has no protection are poisonings from dialyzable substances. Two, particularly, are fatal to the fetus, even though the mother recovers, namely, poisoning from coal gas and from phosphorus. Chronic plumbism produces an effect on the fetus analogous to syphilis. Other common chronic poisons, such as alcohol, morphin and nicotin, while injurious to the fetus have not been proven lethal.

The cachexia associated with advanced cancerous lesions has been noted as being fatal to the fetus. One of the cases in this series was of this sort. Grimoud reports 50 per cent. of stillbirths in cancer of the cervix.

When we come to local conditions affecting the placenta as a cause of fetal death, an unhealthy endometrium must be considered as a factor. While endometritis usually prevents conception, it may permit of gestation. If this occurs, the decidua develops as diseased tissue, and abortion is the usual result. is probable that endometritis as the original cause of placenta previa, is most significant in the causation of fetal death. Faulty nutrition due to the abnormally developed placenta and the atrophic effect on the villi of pressure of the fetal head, affect the child's vitality. Further, there is the early separation of sections of the placenta. Nine cases of macerated fetuses were found complicated by placenta previa. In two of these the membranes were ruptured and the cord prolapsed.

Fibroids, the cause of the associated endometritis, seem occasionally to be a cause of fetal uterine death. There were four such cases in this series.

Infarction of the placenta is due either to a condition of thrombosis of the maternal sinuses extending into the intervillous spaces and thus causing necrosis and degeneration of the neighboring villi, or, according to other authorities, is the result of changes in the chorionic vessels, and is therefore essentially The point is undecided, but infarction is certainly to some extent physiological, when occurring moderately in the later

months of pregnancy. In its marked degree, the condition seems closely associated with albuminuria and toxemia, and is a cause for fetal death.

Accidental hemorrhage, or partial separation of the normally situated placenta, shares a large part of the responsibility for antepartum fetal death. With it is usually an exaggerated degree of placental infarction. Its common remote cause is toxemia or nephritis. There were thirty cases with this complication in my series. Sixteen of them were shown to be complicated with albuminuria, two with syphilis. In some of them there was a traumatic element. Rupture of the uterus from its weakening by disease or postoperative scars may be mentioned as rarely causing fetal death.

The conditions producing still-birth, largely inherent in the fetus, are, first, entanglements or knotting of the cord. fetus in cases of excessive liquor amnia, or unusually long cord, may wind the cord about its neck or extremities to the point of stricture. The cord may be twisted to extreme attenuation. A case has been reported where the cord became strictured through an amniotic band. By diving through a loop of cord, the fetus may so knot the funis that circulation is cut off. By a premature rupture of the membranes, with delayed onset of labor, pressure on the cord may kill the fetus. Twin pregnancies impose a certain danger upon one of the fetuses, because of the possibility of a greater determination of blood to one of the twins. In these cases there is also a greater tendency to early separation of the placenta and to albuminuria.

Diseased conditions of fetal organs may exist to such an extent that death occurs. In such cases, where the expulsion of the fetus is apt to occur from one to two weeks following the fetal death, the maceration of the body makes careful autopsy work impossible. Some of the sixty-two unexplained still-births in this series are doubtless due to fetal diseased conditions that could not be determined.

Anasarca from disease of the fetal heart, fetal nephritis, leukemia, cystic degeneration of the kidneys, hemorrhage into the adrenals, and congenital new-growths are conditions which may cause fetal death.

The analysis of my series of still-births shows the following:

Total series of cases investigated . . . . . 10 000 Cases of fetal death occurring after the time of viability and before labor . . . .

Death from unexplained conditions	62
Syphilis, evident24	02
Syphilis, probable24	48
Albuminuria, eclampsia, toxemia	
- NG (CONTROL OF CONTROL OF CONT	47
Accidental hemorrhage: total 30, with-	
out albuminuria	14
Cord tight about neck	17
4 times	
3 times2	
2 times3	
I time	
Placenta previa, twice with prolapsed	
cord	9
Malaria	6
Fetal acites	4
Hydraminos	4
Fibroids	4
Dry labor	4
Congenital anomaly	5
Triplets 1, twins 3, anencephalous 1	0
Torsion of cord	2
Gonorrhea	2
Carcinoma, stenosis of cord	-
Ectopic and variola	100
Fever (causes unknown), each one	5
Under two classifications and so count-	
ed twice	

The prevention of antepartum fetal death brings us first to a consideration of the specific treatment of syphilis. In this condition heretofore difficulties have arisen in cases of uncertainty of diagnosis. It is probable that rigorous mercurial treatment has dangers for the fetus in cases not syphilitic. Therefore its use experimentally has been reluctantly employed. If the new serum test proves reliable, mercury can be more confidently used.

In the nephritic and toxemic cases much can be done by early dieting and regimen. As acute symptoms develop more active eliminative treatment and rest in bed is of great importance. Especially is rest in bed necessary in cases where some show of blood, fresh or black, indicates a tendency to the loosening of placental attachment. In these cases the condition of the fetal

heart must be carefully watched. An induction of premature labor may save the child's life. In fact, in some cases of this sort, where there seems to be a habit of intrauterine death, this method is the only way in which a living child may be secured.

The induction of premature labor is also a saving process to both mother and child in some cases of noncompensated heart lesion.

In malarial conditions, quinin may be freely used. In all cases where there is a condition of high temperature, the fever should be actively combated in the interest of the child.

General advice to the pregnant mother should include warning against exposure to contagious diseases, against catching cold, overeating, neglect of daily catharsis, and against violent physical exertion of any sort.

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