STILLBIRTH: ITS CAUSES, PATHOLOGY,
AND PREVENTION.*

BY

FRANCIS J. BROWNE, M.D., CH.B. ABERD.,

F.R.C.S. EDIN.,

RESEARCH PATHOLOGIST, ROYAL MATERNITY HOSPITAL, EDINBURGH.
(Working under the Medical Research Council.)

THIS communication is based upon the *post-mortem* examination of 200 consecutive cases of stillbirth and neo-natal death occurring in the Edinburgh Royal Maternity Hospital from August 1st, 1919, to November 30th, 1920. Of these, 120 were stillbirths and 80 neo-natal deaths. The term "stillbirth" is used in the popular sense to include infants born dead at and after twenty-eight weeks' pregnancy, as well as those which, though born with the heart beating, failed to breathe after complete birth of the head and body. The term "neo-natal" death, on the other hand, is used to include all infants in whom respiration took place after complete birth, even though the child only gasped a few times. In every case an effort has been made to co-ordinate the *post-mortem* findings with the clinical history of the parents and of the labour, and from the results of this co-ordinated inquiry certain practical conclusions have been drawn with regard to the supervision of pregnancy, conduct of labour, and care of the newly-born infant. The problems presented by the termination of pregnancy at a period earlier than the seventh month are in many respects so different that it seems preferable to make this the subject of a separate paper at some future date. It is impossible to give in any detail the numerous facts that have been observed during the course of the investigation, but certain broad principles have emerged, and it is these that I wish to state with as much conciseness as is consistent with clearness and continuity.

I. CRANIOTOMIES.

Of the 19 craniotomies, the operation was performed upon the after-coming head in 3. In all cases the cause of difficulty was disproportion between the head and the pelvis, due in 12 cases to pelvic contraction, in 5 to excessive size of the child, the pelvis being normal, and in 2 cases to hydrocephalus. It is an interesting fact that 9 out of the 17 non-monstrous children were born to multiparae, and that 7 of these multiparae (or 77 per cent.) had previously given birth to full-time living children, in many cases even without instrumental interference. In only 3 of the 9 multiparae in whom it was necessary to perform craniotomy was there any history of dystocia in previous labours, and one, a 5-para, had had four previous full-time natural labours.

II. ASPHYXIA NEONATORUM.

Of 120 cases of stillbirth, asphyxia was the cause of death in 49 (or 40 per cent.). In addition to these there were 15 cases of craniotomy in which the child was dead before craniotomy was performed, and in which the cause of death was probably asphyxia during prolonged and unsuccessful attempts at delivery. The real number of deaths therefore due to asphyxia would probably be 64, or 53 per cent. For the purpose of this analysis, however, the lower figure has been taken as not being open to question.

The 49 cases may be divided into those occurring before labour—"ante-partum asphyxia"—11 cases (22 per cent.), and those occurring during labour—"intra-partum asphyxia"—38 cases (77 per cent.). The chief causes of ante-partum asphyxia were placenta praevia, accidental haemorrhage, and eclamptic convulsions. The foetus in these cases had been dead some hours, or even a day or two before birth, and showed slight maceration; in the internal organs the signs of asphyxia were extremely well marked, much more so than in those cases in which asphyxia occurred during labour, for certain reasons that will become apparent later.

With regard to intra-partum asphyxia, the chief causes were disproportion between the head and the pelvis, primiparity, prolapse of the cord, and difficulty with the after-

coming head in breech presentations. The two first act chiefly by causing prolongation of the second stage of labour. In at least one case the cause of this prolonged second stage seemed to be the cord round the child's neck. There were a few cases in which there was no undue prolongation of labour and no apparent reason why asphyxia should have occurred, and these emphasize the importance of keeping a careful watch on the foetal heart during the second stage. When the child is lying in a posterior position this is not always possible, and the correction of posterior positions by external manipulation before the onset of labour should therefore be attempted as a matter of routine. It must be admitted, however, that to follow the foetal heart when the head is low in the pelvis, the uterus firmly contracting, pains coming frequently, and the mother crying loudly, is not always possible. Neither is it always possible to feel pulsations in the anterior fontanelle even when this is palpable, nor to pass the examining finger over the advancing head, to palpate directly the cardiac area. Often a dangerous condition of the foetus is only indicated by passage of meconium, and when this occurs it is generally too late to render effective aid. We have, in fact, at present no constantly reliable method of discovering the condition of the foetus during even a normal second stage of labour.

Of the 38 cases of intra-partum asphyxia 11 were delivered by the breech. If we reject 2 cases of vertex presentation in which podalic version was performed after forceps had failed, and in which asphyxia might reasonably have been expected to occur had the presentation remained as vertex, and if we assume 3 per cent. to be the normal frequency of breech presentations, we arrive at the conclusion that asphyxia is eight times as likely to occur in breech as in vertex delivery.

The Post-mortem Appearances in Asphyxia Neonatorum.

These *post-mortem* appearances are, as a rule, definite, and seldom leave any doubt as to the cause of death.

1. *The External Appearances.*—The body is limp on account of the late onset of *post-mortem* rigidity. It is livid on account of deficient oxygenation of the blood. The conjunctiva is injected and the lips and finger-nails are almost black.

2. *Internal Appearances.*—The membranes of the brain are injected, the veins being filled almost to bursting point. The lateral ventricles of the brain contain, as a rule, excess of serous fluid, sometimes blood-stained. The chief signs, however, are found within the thorax. There are subpleural haemorrhages in the lungs—the so-called Tardieu spots, found chiefly around the roots on the posterior surface, on the basal surface, and on the surfaces between the lobes. There are subpericardial haemorrhages in the heart, with a peculiar grouping, mainly on the anterior surface, along the interventricular groove, and on the posterior aspect of the ventricles, along the auriculo-ventricular sulcus. In a well marked case there will also be subcapsular haemorrhages in the thymus, and occasionally one sees large interstitial haemorrhages in the thymus extending downwards from underneath the capsule. Punctate haemorrhages on the front of the aorta and pulmonary artery are frequent, and even in the fibrous pericardium. Occasionally one sees them also on the upper surface of the diaphragm and on the anterior aspect of the part of the vertebral column within the thorax. Intrathoracic haemorrhages therefore constitute one of the most important signs by which asphyxia may be recognized, but sometimes they are entirely absent. I had one case especially, where the child was born dead soon after prolapse of the cord. I was present at birth, and carried out the *post-mortem* examination soon after. In this case not a single intrathoracic haemorrhage was present, and the only internal signs of asphyxia were two other important signs that have not yet been mentioned—fluidity of the blood due to its increased carbon dioxide content, and great congestion of the internal organs.

Causes of Intrathoracic Haemorrhages.

When the child's blood supply is suddenly cut off by pressure on the cord, placental separation, or otherwise, the accumulation of carbon dioxide in the blood, by stimulating the respiratory centre, causes the infant to make a premature inspiration and expands the chest wall.

* Read at a meeting of the Edinburgh Obstetrical Society, May 11th, 1921. Abstract of report to the Medical Research Council, May, 1921.

If there is a little air in the maternal passages, as generally happens if the hand of the accoucheur has been introduced for any purpose, it is drawn into the lungs, which therefore imperfectly follow the chest wall, a negative pressure is created within the thorax and blood is sucked into it, with consequent giving way of the tiny venules and capillaries in places where they are least supported—that is, on the surfaces of the organs. We thus see why it is that in ante-partum asphyxia these intrathoracic haemorrhages are most marked, and why it is that in premature infants they are often entirely absent. In the premature infant the respiratory centre is poorly developed, attempts at respiration during asphyxia therefore ineffective, and the consequent negative pressure inside the chest comparatively slight. In ante-partum asphyxia no air whatever is available; therefore the negative pressure created inside the chest is greater, with consequently greater haemorrhage. The chief points, then, that require emphasis with regard to asphyxia are the great danger of breech presentation and the necessity for keeping a careful watch on the foetal heart during labour.

Also, the greatly increased liability to the occurrence of asphyxia in primiparae on account of rigidity of the tissues, even when the pelvis is normal and the child not excessively large, points to the desirability of inducing labour in some of these cases a little before full time, unless the pelvis is more than usually roomy or the child below the average size. Finally, most of the cases of asphyxia might have been prevented by adequate ante-natal supervision, which would have led to the discovery of such conditions as contracted pelvis, excessive size of the child, breech presentations, and albuminuria, and to the institution of preventive treatment.

III. MACERATION.

In the series are included 22 macerated foetuses, in 14 of which the cause of foetal death was syphilis, in 3 albuminuria, in 1 diabetes, and in 4 the cause was doubtful, further observation of the mother being required, especially during a subsequent pregnancy. The number of cases labelled doubtful show that it is occasionally a matter of some difficulty to decide definitely as to the cause of the foetal death. The chief points to be relied upon in arriving at a diagnosis of syphilis in any particular case are:

1. The presence of spirochaetes in the foetal organs.
2. Enlargement of the foetal spleen.
3. Presence of chondro-epiphysitis.
4. Presence of active syphilis in the mother, or in both parents.
5. Strongly positive Wassermann reaction in the mother.
6. A maternal history of repeated stillbirths, abortions, or neo-natal deaths, without other discoverable cause.
7. Presence of syphilitic changes in the placenta.

At this point I shall only say one word about the value of the placental appearances in distinguishing between the causes of foetal death. In the syphilitic macerated foetus the placenta is sometimes, but not always, larger and heavier than normal. It is always more or less pale on section, and is usually thickened from the maternal to the foetal surface. It is rare to find infarcts or haemorrhages in the syphilitic placenta, whereas in the albuminuric placenta large white or red infarcts and numerous circumscribed haemorrhages are common. The albuminuric placenta in maceration is, like the syphilitic placenta, pale and avascular, but it is not usually enlarged, and is not thickened from the foetal to the maternal surface. Microscopically it is very difficult to tell the one from the other, both being almost entirely avascular, but in the syphilitic placenta the villi are larger and thicker than normal, and consequently the intervillous spaces are diminished in extent.

In deciding, therefore, as to the cause of foetal death in the case of any particular macerated foetus it is necessary to weigh carefully all the available evidence—that obtained from the parents, the placenta, and that from the examination of the foetus itself—and only rarely, and more seldom with increasing experience, should the observer be left in doubt as to the cause of death.

IV. CEREBRAL HAEMORRHAGE.

Out of the 200 there were 59 cases of cerebral haemorrhage, or 29.5 per cent., and of these 20 were delivered by the breech and 39 as vertex. Of the 39 cases delivered as vertex, 17 were forceps and 22 non-forceps. Of the 17

forceps cases, 9 were classified as difficult, and in 8 no special difficulty was encountered. The causes of difficulty in the difficult cases were either contracted pelvis or excessive size of the child, and all except one occurred in primiparae. Of the 8 cases in which forceps delivery was effected without any special difficulty, 3 were premature, and in the remainder forceps had been applied early in the second stage to hasten delivery, the patient in two cases being eclamptic; in one the mother had mitral stenosis, and in another ventral hernia.

Non-forceps Cases with Cerebral Haemorrhage.

Of these there were 22 in all, and all occurred in premature infants, 16 occurring at from seven to seven and a half months, 6 at eight months, while at eight and a half months there were none. The liability to cerebral haemorrhage in premature infants is thus greatest at from seven to seven and a half months, still considerable at eight months, while at eight and a half months the liability would appear to be no greater than at full term. Further, no case of cerebral haemorrhage occurred in a full-time child in which delivery was a natural vertex.

Again, of the 59 cases, 23 were at full time and 36 premature. If, therefore, we take the normal proportion of full-time to premature infants over a large number of cases as 10 to 1, we arrive at the conclusion that the liability to cerebral haemorrhage in premature infants is sixteen times that of those at full time. Further, of the 20 cases delivered as breech, only 9 were originally breech, the remaining 11 being converted into breech by internal version for various reasons. If we reject 3 cases in which the version was done for dystocia and in which cerebral haemorrhage might reasonably be supposed to occur had they remained as vertex, there remain 17 cases of breech presentation in which cerebral haemorrhage resulted out of a total of 59. Taking, again, 3 per cent. as the normal frequency of breech delivery, it is evident that the liability to cerebral haemorrhage in breech presentation is ten times that in vertex.

Operability.—It was a remarkable fact that probably none of the haemorrhages could have been removed by operation with any hope of permanent cure. This was owing to the site being in some cases inaccessible or to the brain being too much ploughed up and damaged by the haemorrhage, or to the presence of fatal lesions in other organs, such as kidney or liver.

Relationship between Cerebral Haemorrhage and Tears of the Dural Septa.

The condition of the septa of the dura mater within the cranium was carefully observed in the last 94 cases, with the result that tears, complete or incomplete, of the tentorium cerebelli were found in 35 (37 per cent.). Of these 35 cases, delivery was by breech in 22 (63 per cent.), by vertex in 12 (34 per cent.), and by face in one (3 per cent.). Reasoning in the same way as in the case of cerebral haemorrhage, we arrive at the conclusion that breech delivery is sixteen times as likely to give rise to tentorial tears as is vertex.

Further, in every case of breech delivery at full term some degree of tentorial tearing was apparent. It was also evident from my figures relative to methods of delivery that, while complete tears of the tentorium cerebelli are exceedingly likely to occur in difficult breech cases, yet they may also occur in breech cases in which there has been no difficulty whatever—in easy and premature labours; and while in vertex cases complete tears may frequently occur in forceps delivery, they are not confined to these, but may also occur in cases where delivery has been spontaneous but prolonged and somewhat difficult. It is evident, therefore, that the problem of the prevention of injuries to the dural septa is inextricably bound up with that of the prevention of breech and of difficult vertex deliveries.

Relationship between Tears of the Dural Septa and Cerebral Haemorrhage.

An examination of the 50 cases in which either tears of the dural septa, or cerebral haemorrhage, or both together, occurred, reveals the fact that there were 21 cases in which tears of the dural septa occurred, and in which there was no cerebral haemorrhage. This means that out of a total of 35 cases of tentorial tearing, 60 per cent. were

unassociated with cerebral haemorrhage in any situation or of any degree. If, on the other hand, we examine the cases in which cerebral haemorrhage occurred with no tears of the dural septa, we find there are 15 cases of such out of a total of 29 cases, or 57 per cent. Again, if we take the cases in which tears of the dural septa and cerebral haemorrhage coexisted, we find there are only 14 cases out of the total 50 in which septal tears and cerebral haemorrhage existed either together or separately—that is to say, tears of the septa and cerebral haemorrhage existed together only in 28 per cent. These 14 cases are tabulated in my report to the Medical Research Council and the causes of the haemorrhage analysed.

The matter is too detailed to enter into in this paper. Suffice it to say that there were only three in which it was necessary to invoke the theory advocated by Holland, of tearing of the dural septa, allowing excessive moulding of the head with consequent kinking of the vein of Galen and rupture of it or its tributaries from the base of the brain and cerebellum. In some cases the haemorrhage was in regions remote from that drained by the vein of Galen; in others it was obviously due to the tears in the tentorium cerebelli, extending deeply into the straight or lateral sinus.

Intraventricular Haemorrhage.

Of these there were 22 cases, but I shall only indicate here the points with regard to it that have a practical bearing. (1) It never occurred except in premature births, being most frequent from seven to seven and a half months, less at eight months, and at eight and a half months it seemed to be no more likely to occur than at term. As a matter of practical experience one does not expect to find intraventricular haemorrhage in infants born at full term. (2) Breech delivery was ten times as likely to cause it as vertex, a result exactly corresponding to cerebral haemorrhage in general. (3) In none of the subjects were any signs noted that might have led to diagnosis. The babies were all premature; one or two were blue from birth; they sucked and cried badly, or not at all. Some of them lived as long as forty-eight hours, even with both lateral ventricles distended with blood clot.

The points to be kept in mind in the prevention of cerebral haemorrhage would therefore seem to be:

(a) Avoidance of breech deliveries, by cephalic version of breech presentations at a period when this is always possible, not later than the seventh or eighth month.

(b) Avoidance of difficult forceps deliveries; this is only possible by antenatal supervision of every case.

(c) Avoidance of the induction of premature labour, certainly before eight months, better still before eight and a half months.

(d) Manual dilatation of the parturient canal for the passage of the premature infant and preservation of the membranes unruptured as long as possible. We shall see that the latter has also an important bearing on the question of infection of the newly-born infant.

In other words, every effort should be made to prevent pressure upon the premature infant during its birth. Before eight and a half months the infant is unfitted to pass with impunity through even a normal and easy labour.

Finally, after a study of this series of cases, it is impossible to avoid the conclusion that it is only by adequate antenatal supervision of every pregnant woman that the present high morbidity rate from cerebral haemorrhage can ever be seriously diminished.

V. SYPHILIS.

There were altogether 35 cases of syphilis, including 14 macerated foetuses. I should like to state very briefly the chief points that emerged from a study of these 35 cases.

(1) *With Regard to the Mother.*—Only very occasionally does one find that the mother has any signs or even any history of syphilis. In only 6 cases of the 35 did the mother show any signs or give any history of syphilitic infection. Again, while a positive Wassermann reaction is of great value, a negative Wassermann in a woman with a history of repeated stillbirths, abortions, or neonatal deaths, without any obvious reason to account for them, such as birth injuries, albuminuria, or retroverted uterus, has little or no value in excluding syphilis. So much has

this been impressed upon us that in the presence of such a history, even though the Wassermann is repeatedly negative, we do not hesitate to carry out antisyphilitic treatment. In the case of a primiparous patient the bad obstetric history is, of course, wanting, but in these cases it is, in our experience, less common to find latent syphilis and a negative Wassermann.

(2) *With Regard to the Child.*—What are the points that guide us to a diagnosis of syphilis at *post-mortem* examination? It should be borne in mind that the evidence obtained from this *post-mortem* examination of the child is often the only evidence obtainable of the presence of syphilis in the mother.

In the first place, the syphilitic child is usually premature, as were twenty-six out of my thirty-five cases. Syphilis is the commonest cause of premature labour. In the second place, there may be marasmus, but only if the child has lived at least a few days, so that if it has died in the first day or two it may be perfectly well nourished. Third, it may be jaundiced, and sometimes the jaundice is present from birth—it may be born jaundiced or the jaundice may come on immediately afterwards. Fourth, there is sometimes an oedematous condition—a hard oedema of the body and the limbs. And fifth, there may rarely be a few pustules or vesicles or areas of desquamated skin about the body, especially on the buttocks or lower limbs. These five signs are all that may be seen externally and they may be all absent, and it is rare to find more than one or two present at the same time.

The Internal Signs of Syphilis.

There are four organs, or parts, of the foetus in which we may find naked-eye changes due to syphilis.

1. *Lungs.*—As a rule, there are no naked-eye changes present. The lungs may look perfectly healthy, well aerated, float buoyantly in water, and show the normal pink colour. The typical naked-eye pneumonia alba of the textbook is rarely seen in the newly-born foetus—it was only present in one case of my series. When seen it is easily recognized; the lung is heavy and voluminous, the cut section is more or less solid, in colour it resembles grey marble, and it sinks in water. On squeezing the cut surface, more or less purulent-looking fluid exudes. But in most cases of syphilis of the newborn the lungs look perfectly healthy to naked-eye examination, except for some terminal oedema in the lower lobes.

2. *Liver.*—It is commonly said that the liver is enlarged in syphilis. I have rarely seen an enlarged liver. In only two of my cases was there enlargement, and then it was not definite; in only one case was the liver tough on section, and its surface was smooth and bile-stained. As a rule the only change present is that, in cases of marasmus, the liver is pale and somewhat fatty, which is due to the marasmus and not to the syphilis.

3. *Spleen.*—An enlarged spleen is one of the most reliable signs of syphilis in the newly-born infant. When a definitely enlarged spleen is found in an infant that is stillborn, or that dies a few weeks after birth, it may be taken as pathognomonic of syphilis. But an enlarged spleen is not always found. It was only found in five of my series of 35 cases, or 14 per cent. One sometimes has a case where the spleen appears to be enlarged, but the enlargement is not definite; it is on the border line, and one cannot be sure, for the spleen varies a good deal in size in normal cases. Speaking generally, however, I think I am nearly correct in saying that a spleen weighing more than 16 grams may be taken as definitely enlarged.

4. *Chondro-epiphysitis.*—We come now to the fourth point in the *post-mortem* diagnosis, chondro-epiphysitis. Textbooks of midwifery lay great stress upon the presence of chondro-epiphysitis as proof of the presence of syphilis. No doubt it is when present. The trouble is that one very seldom sees it. Out of my series of 21 fresh syphilitic infants I have only found it definitely present in one, and so indefinitely in two others as to be useless in diagnosis.

5. *Placenta.*—If you are fortunate you will also have the placenta to guide you. It is generally taught that the syphilitic placenta is enlarged, greasy and pale, but nothing is further from the truth. The placenta of a syphilitic child born alive looks perfectly normal and healthy. It is not increased in size; its vascularity is normal; and even on microscopic examination it is

impossible to tell it from the placenta of the healthy child. Had it been otherwise the child would have died *in utero*. If, however, the child has been born macerated, the placenta is frequently enlarged; on section it is pale and avascular, thickened from the maternal to the foetal surface, and the larger vessels stand out on the cut surface as thick white cords. The reason for this is that the enlargement of the placenta as a whole is due to the enlargement of the individual villi, and these owe their enlargement to a cellular proliferation in their stroma which blots out the capillaries; and so the child dies from the cutting off of its blood supply, and the same cause that leads to the death of the child causes the enlargement of the placenta. It is not, therefore, strictly correct to say that the syphilitic placenta is never enlarged, for it may be in the case of the syphilitic macerated foetus.

It will be seen, therefore, that the naked eye *post-mortem* examination may be completely negative. The child may appear normal and well nourished, and the lungs, liver, spleen, epiphyses and placenta may seem perfectly healthy. How, then, are we to arrive at a diagnosis of syphilis? This is achieved by histological examination in the fresh foetus of the organs, especially the thyroid, thymus, lungs and liver.

Histological Changes in these Organs.

Thyroid.—Five of my 21 fresh foetuses showed well marked fibrosis of the thyroid, which, as a result, contained few acini. In addition to this there is a peculiar proliferation of cells into the acini that remain, filling them so that little or no colloid may be visible. An interesting question arises here as to whether normal colloid-containing acini would become regenerated if specific treatment were carried out, or whether such regeneration would not take place, and the child become a cretin. Between the acini of the normal thyroid there are groups of cells which afterwards form new acini. Many of these are still present in the syphilitic thyroid, and it is probable that from these the thyroid would become sufficiently regenerated to carry on its normal function.

Thymus.—Several of my cases showed well marked fibrosis, increase of reticulum cells and plasma cells, with a peculiar change in the corpuscles of Hassal. These were enlarged, and the concentric body inside the capsule was replaced by granular debris and leucocytes, chiefly polymorphs and a few lymphocytes. In some the replacement is so complete that none of the concentric body is left, while in others some part of the latter is still left in the centre of the otherwise granular mass.

Lung.—The histological changes present in the lungs were: (1) Increased thickness and comparative avascularity of the alveolar walls, due to a fibro-cellular proliferation; (2) thickening of the vessel walls and of the walls of the bronchioles; (3) thickening of the pleura, liable to be irregular, and not always present; (4) in one case a few small microscopic gummata were found in which the chief cells distinguished were plasma cells, lymphocytes, and a few polymorphonuclear leucocytes; (5) the alveoli are usually loosely filled with catarrhal phagocytic cells derived from the endothelial cells lining them.

Liver.—Three chief types of histological changes were found: (1) Coarse, or peri-portal, cirrhosis was present in 10 cases, in which lymphocytes, plasma cells, and fibroblasts were numerous in the portal tract. (2) Fine, or intercellular, cirrhosis was present in 3 cases. Both in this and in the coarse type there was also present more or less thickening of the hepatic vein in the centre of the lobule. (3) The haemopoietic liver. In this the sinusoids are filled with large cells, chiefly lymphocytes, a few nucleated reds, also a few large cells lying in groups of two or three, and containing in their protoplasm large coarse eosinophile granules. This indicates a continuation of the blood-forming function of the liver into post-natal life. The large number of the lymphocytes present gives the liver the appearance of a case of lymphatic leukaemia, but the excess of lymphocytes is confined to the sinusoids, and is not found in the vessels of the portal canal or even in the central hepatic vein.

Spleen.—The splenic enlargement, in the cases in which it was present, was associated with peri-portal cirrhosis in the liver. Microscopically it was of the nature of a hyperplasia, while in three cases there was fibrosis. There was no definite or constant increase in reticulum cells as compared with several normal cases examined, adult and

foetal. As regards plasma cells, their number in normal cases varied very much, and there was no characteristic or constant increase in their number in syphilis.

Kidney.—Only one case showed any change. In this there was marked interstitial haemorrhage between the tubules, strictly confined to the pyramids, and not all of these were affected. Microscopically there was thrombosis in the veins between the cortex and the pyramids. There was no thrombosis in the arteries, but their intima was much thickened by a cellular proliferation, and in some cases the lumen was obliterated. The condition was probably one of infarction.

Pancreas.—Fibrosis of the pancreas was well marked in several cases. The vascular coats were thickened and a young fibrous tissue spread between the acini, widely separating and destroying them. Islands of Langerhans appeared to be more numerous than in sections of the normal pancreas. In the latter they are rather scarce and somewhat difficult to find, whereas in some sections of a fibrotic syphilitic pancreas there were frequently found as many as eight in a microscopic field with 1/3 objective.

Finally, there only remains the examination for spirochaetes. It is another common idea that the spirochaete is always found in the tissues of the syphilitic foetus. That this is by no means true will be evident when I say that out of fourteen macerated syphilitic foetuses the spirochaete was only found in eight, while of twenty-one fresh syphilitic infants it was only found in one. The absence of the spirochaete was particularly unexpected in one case where twins were born at seven and a half months. The first was born macerated, and spirochaetes were very numerous in the organs, including the uterus, ovaries, tubes, and even in the wall of the heart. The other twin died, at the age of 16 days, of pneumonia. There were well-marked syphilitic changes in the liver and lungs, but no spirochaetes were found in any organ, in spite of searching examinations by dark-ground and Levaditi methods. Dr. Amand Routh has suggested to me that the infants may belong to different fathers, but there was nothing in the clinical history to suggest this, and the twins were both females and at the same stage of development. The placentas were separate, and that of the living child was much less avascular than that of the other. It would seem that it is only on the assumption that the spirochaete goes through a definite sexual cycle that its repeated absence in proved syphilitic foetuses can be explained.

It is evident therefore that the *post-mortem* diagnosis of syphilis is not always a simple matter, but that it frequently entails a careful weighing of all the available evidence—the obstetrical and clinical history of the mother, the Wassermann test, the placenta, a complete naked-eye and microscopic examination of the foetal organs. Even when all these data are available there will occasionally be a case where there is difficulty in deciding whether syphilis is present or not.

VI. PNEUMONIA.

Pneumonia is a common cause of death amongst infants during the first week after birth, and accounted for 21 deaths out of 80, or 26 per cent. I shall only draw attention very briefly to one or two points regarding it: (1) In the infant there is little defensive reaction as compared with the adult against the infecting agent. In the lung alveoli there are remarkably few polymorphonuclear leucocytes present. (2) Pneumonia in the first few days of life is an extremely insidious disease, generally presenting no characteristic symptoms which might lead to its presence being suspected, and to the carrying out of physical examination. (3) Even at *post-mortem* examination its presence may be missed if microscopic examination of the lungs is not carried out. The reason of this, of course, is that a small pneumonic patch, insufficient to alter the normal naked-eye appearance of the lung, may frequently be sufficient to kill the infant. (4) It is sometimes due to antenatal infection from premature rupture of the membranes, and the infant may be not only born infected, but may be even suffering from an advanced degree of pneumonia before its birth. This is illustrated by the following case:

A primipara at full time was admitted to the Royal Maternity Hospital after long delay on the district, due to primary inertia. The membranes had been ruptured for some time before admission. Forceps were applied in hospital, and a

"blue baby" was born, breathing badly, which died eight hours later. The child was at full time, very well nourished, and considerably above the average size—3,800 grams. Each pleural cavity contained about 2 oz. of blood-stained serous fluid, the lower lobes of both lungs were of solid consistence, and on squeezing the cut surfaces they exuded a yellowish frothy fluid. Microscopically the lungs showed an advanced degree of catarrhal pneumonia, in the stage of grey hepatization. At some parts no cells could be made out, and the whole appeared to be a necrotic mass in which even the alveolar walls could not be distinguished. Cultures from the lung on blood agar yielded an organism of the pneumo-bacillus group. It is impossible that such an advanced degree of grey hepatization could have taken place in the eight hours in which the child lived, and its blueness and difficulty in breathing at birth, and continuing till death, are strong evidence in support of antenatal pneumonia. Another child, in whom the membranes had been ruptured three days before birth, died on the third day, and at *post-mortem* examination there was found pneumonia, double empyema, and septic endocarditis, the causal organism being the *Bacillus coli*. Breathing from birth had been wheezy and rapid.

I should like to draw attention to a type of pneumonia that would seem to be peculiar to the newly-born infant, and which occurred in six of my cases. This might appropriately be called "acute haemorrhagic pneumonia of infants," and its course is somewhat as follows:

A child is born, perhaps at the full time or a few days prematurely. It seems perfectly healthy at birth and afterwards, when, without any warning symptoms whatever, it is found dead in bed. The face may be blanched, there may or may not be a little blood staining about the mouth and nose, and nothing more is to be seen. Suspicion of overlying or of foul play may arise. What has happened is this: the child's lungs have become infected in some way and an acute and rapid congestion occurs. In the adult this would be the first stage of a pneumonia, the next stage would be consolidation, followed, if the patient recovers, by resolution. But in the young infant the fragile vessels cannot stand the strain thrown upon them by the engorgement, and they give way and blood is poured out into the alveoli and bronchi, and the child dies suddenly, drowned in its own blood, the whole process occurring with dramatic suddenness and probably not occupying longer than a few minutes.

This acute haemorrhagic pneumonia of infants, then, seems to be a distinct clinical and pathological type, and while the etiological factor in most cases is organismal, there is some evidence to support the theory that it may occasionally be of the nature of an anaphylaxis. The important points to be kept in mind are the necessity for guarding the newly born infant against infection, and of avoiding, when possible, the premature rupture of the membranes during labour.

VII. SUPRARENAL HAEMORRHAGE.

There were 18 cases of haemorrhage into the suprarenal body. All were born dead except two, the cause of death being in most cases asphyxia. In one case the child lived four hours, the haemorrhage being severe and into the medulla of the left suprarenal body. Of the 16 cases that were born dead, the suprarenal haemorrhage was the probable cause of death in one, no other cause being found.

Site of Haemorrhage.

Into medulla of both	...	...	...	5
Into medulla of right	...	...	...	4
Into medulla of left	...	...	...	5
Under or into capsule of both	...	...	...	1
Under or into capsule of right	...	...	...	1
Under or into capsule of left	...	...	...	2

Six cases were delivered by vertex and 12 by breech. Of the 6 cases delivered by vertex, in 3 the haemorrhage was into or underneath the gland capsule, and in only one of these was it severe. Rejecting for statistical purposes the two slight cases, there remain 4 cases in which delivery was by vertex. In 3 of these the haemorrhage was into the medulla, in all into the right only, and in one beneath the capsule. Of the 12 breech deliveries, in 3 podalic version had been done after forceps had failed on the forecoming head. If we reject these 3 cases, as it might reasonably be supposed that suprarenal haemorrhage would have occurred had they remained vertex, and assuming again 3 per cent. as the normal frequency of breech presentation, we arrive at the conclusion that breech delivery is twenty-two times as likely to give rise to suprarenal haemorrhage as delivery by the vertex. In

vertex presentations suprarenal haemorrhage only occurred in difficult forcps deliveries, while in breech cases it occurred also in labours in which no special difficulty was encountered. Again, of the 15 severe cases, 9 were at full time and 6 premature. Assuming 1 in 10 to be the normal proportion of premature to full-time labours, we find that suprarenal haemorrhage is four times as likely to occur in a premature labour as in a labour at full time.

Causes of Suprarenal Haemorrhage.

Various causes have been invoked from time to time to explain these suprarenal haemorrhages. It has been suggested that pressure upon the liver compresses the inferior vena cava between the liver and the vertebral column, thus damming back the blood into the suprarenal veins (Mather). Too early ligation of the cord has been suggested, but this seems to be ruled out by the fact that the increased pressure would not fall directly upon the inferior vena cava, neither would this cause operate more frequently in breech than in vertex presentation. Syphilis, being a frequent predisposing cause of haemorrhage in other organs, would no doubt also predispose to suprarenal haemorrhage by furnishing a capillary toxin, but it was not present in any of my cases.

It was found from a study of my tables that of the 16 cases in which the child was stillborn, 15 were associated with asphyxia, and it seems probable that this is an important factor in its causation. It is certain that haemorrhages over the cerebral hemispheres, around the small veins of the meninges, are frequently found as a result of asphyxia in premature infants, while from the same cause large subcapsular and even interstitial haemorrhages may be met with in the thymus, even in full-time infants. In the premature infant the vessels are extremely fragile and their muscular coats undeveloped, and it is possible that they give way easily under the strain of the sudden congestion of the internal organs in asphyxia. In no organ would this be more likely to occur than in the suprarenal, which is excessively vascular and in which there is a minimum of supporting fibrous stroma. The organ also is so situated, close to the termination of the superior vena cava, that it is likely to receive the full force of the backward pressure of blood from the distended right auricle. In discussing asphyxia, however, we found that breech delivery was eight times as likely to give rise to asphyxia as vertex delivery, while it is twenty-two times as likely as vertex to give rise to suprarenal haemorrhage. It would appear from this discrepancy that there must be some other factor at work in producing suprarenal haemorrhage, and coming much more prominently into play in breech than in vertex deliveries. This factor is probably the direct trauma incidental to the passage of the breech and abdomen through the comparatively undilated parturient canal and the pressure of the child's lower limbs against the abdominal organs.

VIII. PREMATURE LABOUR.

Out of the 200 cases 95 were born prematurely. Bearing in mind that every child should be carried to full time unless there is some reason for the contrary, an effort was made to ascertain this reason by a co-ordination of the clinical history of the mother with the *post-mortem* findings in the foetus, with the result that in every case except two some cause for the premature onset of labour was discovered, the chief causes being, in order of frequency, syphilis 28, twins 16, induction of labour 12, albuminuria and eclampsia 11, placenta praevia 8.

Causes of Death in the Premature Infant.

In the 56 cases of neo-natal death among premature infants the causes were as follows:

Cerebral haemorrhage	...	...	...	22
Syphilis	...	...	...	12
Syphilis with catarrhal pneumonia	...	...	...	6
Catarrhal pneumonia	...	...	...	5
Septic peritonitis	...	...	...	2
Septic meningitis	...	...	...	1
Suprarenal haemorrhage	...	...	...	1
Omphalorrhagia	...	...	...	1
Moist gangrene of foot	...	...	...	1
Asphyxia (gasped a few times)	...	...	...	1
No cause found	...	...	...	4

56

With regard to the 4 cases in which no cause for death was discovered, it can only be supposed that the infant had

insufficient vitality to maintain life, or that fatal cooling of the body had occurred through insufficient development of the heat-regulating mechanism. Examination of the causes of premature birth reveals the fact that the latter is, in a large percentage of cases, unpreventable as far as present knowledge goes. Thus the only preventable cases are those of syphilis and albuminuria, including eclampsia, comprising 39 cases, or about three-fifths of the entire number, and these might have been prevented by antenatal care.

An examination of the causes of death among premature infants, on the other hand, reveals the fact that most might have been prevented, (1) by proper supervision and treatment of the mother during pregnancy, (2) by the adoption of certain precautions to prevent injury to the foetus during birth, and (3) by improved care of the infant during the first month of post-natal life. This improved care can only be obtained by the provision of specially fitted rooms or *couveuses*, on the Continental principle, in which the child's body heat can be maintained until such time as its own heat-regulating mechanism is properly developed, and in which the necessary measures can be taken to guard against infections that are such a serious and constant menace to the life of the premature infant.

IX. SCOPOLAMINE-MORPHINE NARCOSIS.

There were three cases in which the cause of death would seem to have been twilight sleep—at least, asphyxia and other known causes of death were excluded. All three occurred in primiparae, and in all repeated doses of morphine had been given four-hourly, and hyoscine hourly. In all cases the thymus was much enlarged, but was specially so in two, in which it weighed 30 grams and 24 grams respectively, the normal being 10 grams. It seems more than a coincidence that these two thymuses should be the two largest I have met with in 350 necropsies on infants. Instances of increased susceptibility to the action of morphine are not infrequent in the case of infants and young children, and it does not seem unreasonable to suppose that a like idiosyncrasy may exist in the unborn foetus, or that the maternal metabolism in certain cases is slower than in others, thus allowing more of the toxin to pass over into the foetal circulation.

X. MISCELLANEOUS.

There were a few cases in which the cause of death fell into none of the above-mentioned classes. They may be tabulated as follows:

Septic peritonitis from cord infection ...	2
Septic meningitis ...	1
Omphalorrhagia ...	1
Neo-natal asphyxia ...	3
Encephalocele with malformation of the heart	1
Anencephaly ...	2
Double congenital hydronephrosis ...	1
Ruptured liver ...	1
Large thymus (status lymphaticus) ...	1
Deformed chest and rudimentary lungs ...	1

(The case of double congenital hydronephrosis has been reported by Dr. R. W. Johnstone and the writer in the *Transactions of the Edinburgh Obstetrical Society* 1920-21.)

NECESSITY OF EXPERT SUPERVISION OF PREGNANCY.

In conclusion, the great fact that emerges from this investigation is that a very large percentage of the stillbirths and neo-natal deaths could have been avoided by adequate antenatal supervision. Especially is this the case in deaths from craniotomy, asphyxia, and in cerebral haemorrhage occurring in breech and in difficult forceps deliveries. Only about 3 per cent. of the 200 cases had undergone adequate antenatal supervision.

Arguments directed against the preservation of the lives of the unfit certainly do not apply in these cases, for the child that is lost in birth from these causes is generally very large and well nourished, and it is usually just because of this large size that stillbirth occurs. Thus, in the cases of craniotomy the average weight of the child without a large part of the brain was 3,180 grams; of the cases of asphyxia in full-time foetuses the average weight was 3,480 grams—the largest weighed 6,160 grams—

and of the fatal cases of cerebral haemorrhage in full-time infants the average weight was 3,460 grams.

Nor do such arguments apply even in the case of syphilis. If we were certain that every syphilitic infant would be born dead it might reasonably be contended that such was the best possible solution of the problem; and if we were sure that every infant born with congenital syphilis would, if untreated, die, to allow it to do so might be the best course to adopt, both in the interest of itself and of the race as a whole. Fortunately, or unfortunately, however, we cannot be certain that such a termination will occur, either of the pregnancy of the syphilitic woman or of the life of the congenitally syphilitic infant. The latter may, even if untreated, live to bear in its body the major portion of the punishment of the sins of its parents, and therefore the only justifiable course to adopt is the early and adequate treatment of the expectant syphilitic mother, especially as such apparently excellent results in the case of the child are now being obtained with modern methods.

The ideal is antenatal supervision and care of every unborn infant by the compulsory, or preferably by the voluntary, notification of the pregnancy of every expectant mother. Parents of children of school age may be compelled to have remediable physical defects in those children treated. It would seem that such compulsory care of the child may begin too late if only undertaken after birth. Such conditions as mental backwardness due to small non-lethal cerebral haemorrhages or tentorial tears occurring during birth cannot be remedied in the child of school age. They could and should have been prevented. In short, it seems illogical that expectant parents should be allowed to risk the life of their unborn child with impunity, whilst the failure to provide even for its physical comforts after birth renders them liable, and rightly so, to the condemnation of the law and of public opinion.

I wish to express thanks to the head of the antenatal department and to the staff of the Royal Maternity Hospital for their cordial co-operation in this investigation; also to Professor Lorrain Smith for allowing the pathological part of the investigation to be carried out in his department in the Royal Infirmary, as well as for much invaluable advice upon difficult matters connected therewith.

THE PSYCHOLOGIST IN PUBLIC LIFE.

BEING THE PRESIDENTIAL ADDRESS DELIVERED TO THE
METROPOLITAN COUNTIES BRANCH OF THE
BRITISH MEDICAL ASSOCIATION.

BY

H. B. BRACKENBURY, M.R.C.S., L.R.C.P.LOND.,
CHAIRMAN INSURANCE ACTS COMMITTEE, BRITISH
MEDICAL ASSOCIATION.

EXPERIENCE in the great war has created, I think, a greater revolution in psychology than in any other science. That experience has, indeed, proved, as nothing else probably could, the enormous advances in surgery and in preventive medicine, but for this demonstration we were prepared. In psychology the effect of the war has been to establish a new view-point, to compel a new outlook. For some years before 1914 pioneers had been indicating new regions for exploration and marking out new roads; but either, as in the case of McDougall, those who followed the pioneer, in spite of the invaluable nature of his work, were disappointingly few, or, as in the case of Freud, the explorer himself, after brilliantly showing the new way, wandered slightly astray into a not very savoury swamp lying by the path. The great war has afforded abundant opportunities for the investigation of cerebral physiology and pathology and of the psychoneuroses, and the work of such investigators as Head and Rivers has directed us more and more to the new regions and has confirmed the fundamental truth of the teachings of the pioneers. Psychology has become not merely the science of mind, mainly concerned with the analysis of consciousness by the method of introspection, but the science of behaviour, whose subject matter is conduct in general and whose conclusions are based on wider observations and experiment than was formerly considered unquestionably legitimate. More and more the human mind is being studied in its biological and its social connexions. New conceptions have arisen of its evolutionary history and its driving forces as