injections of typhoid-paratyphoid vaccine (dosage 25 to 75 millions at intervals of 3 to 6 or more days) being definitely benefited thereby. Vaccine therapy is, however, definitely contra-indicated in old age or infancy and in debilitated patients or those with cardiac or renal complications.

Edwenil (1 to 4 c.cm. subcutaneously every 4 hours) is a harmless remedy which produced definite improvement in 3 cases of my series, and it seems probable that other patients might also have benefited if the dosage had been pushed. S.U.P. 36 seems also to have helped in a few cases (4 out of 12).

Many other remedies such as Mercurochrome, Argochrome, Trypaflavine, Bayer 205, Solganal, neoarsphenamine, Stabilarsan, nuclei acid, colloidal solutions of silver, manganese, and sulphur, and injections of whole blood and of milk have been used, but the results appear to have been disappointing, and the intravenous antiseptics are by no means free from risk.

Finally, reference should be made to brucellin or abortin, the substance employed for the intradermal diagnostic test. This usually consists of the filtrate of a three weeks' broth culture of the causative organism. Considerable success has been claimed abroad for treatment with this substance, usually by intramuscular injection after a preliminary skin dose, but I have no details of the results of such treatment in this country though I have done my best, with the coöperation of Prof. G. S. Wilson, to encourage its trial.

INTRANATAL AND NEONATAL DEATH

A REVIEW OF NINETY-NINE CONSECUTIVE CASES

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THIS paper is based on the clinical and post-mortem records of a series of infants whose deaths had been reported to H.M. coroner for the city of Birmingham. All of them had died suddenly or unexpectedly within the intranatal or neonatal periods. The causes of death as ascertained by autopsy were as follows:—

	Cases.
Stillborn	21
Asphyxia neonatorum	37
Intracranial hæmorrhage	17
Broncho-pneumonia	11
Congenital defects—	
Congenital heart disease	3
Cleft palate	1
Suprarenal hæmorrhage	2
Intestinal volvulus	1
Septicæmia	1
Violent death—	
Intraperitoneal hæmorrhage	1
Shock and asphyxia	1
Suffocation	3
	99

STILLBIRTHS

Stillbirths are reported to the coroner when no doctor or midwife is present at the birth, or when there is any suspicion of foul play. In several of my cases birth was premature, but all the infants were viable as judged by their length, weight, and general development. Many had tears of the dural septa but these were all incomplete and the hæmorrhages that had resulted were insignificant and insufficient to have caused fatal issue.

Failure of survival, therefore, must be considered as due to the non-establishment of normal respiration. One cannot go so far as to say that all would have lived if modern methods of treatment had been available immediately, but there is no doubt that in a very large proportion death could have been avoided. In none was any condition found that was incompatible with life. In other words, the problem is similar in all respects to that encountered in dealing with neonatal asphyxia to which I refer next.

ASPHYXIA NEONATORUM

This group contains 37 of my cases. Of these, 23 died within twenty-four hours of birth and 14 lived for more than a day. Of the latter 10 died within forty-eight hours of birth but 3 lived for more than two days, and 1 for four days. In a considerable number a midwife was in attendance at the time of the birth, no difficulty was experienced in getting respiration established, and the infants were stated to have cried well. On the other hand, a certain number of the mothers had concealed their pregnancy and were alone when the baby was born; and when help arrived the infant was dead.

There is nothing of importance in the obstetric histories of any of those cases in which reliable information could be obtained; all except one were vertex deliveries and in none was instrumental aid necessary.

In all the infants the lungs were very imperfectly expanded; in fact in none did they fill more than half their respective pleural sacs. The other pathological changes encountered were those commonly found in death from asphyxia from any cause and do not require reiteration here, with the exception of the increased intracranial pressure to which reference will be made presently.

Before the problem of asphyxia neonatorum can be solved the mechanism whereby normal respiration is established in the newly born baby has to be elucidated. A vast literature is growing up round this subject which it is impossible for me to summarise fairly here, for much is controversial and much speculative. The observations that have recently been published by Barcroft [1] are, however, illuminating. He discusses three possible causes for the first breath, viz.: (1) oxygen-want, (2) excess of carbon dioxide, and (3) increased blood pressure in the brain. His original investigations have enabled him to produce positive evidence that lack of oxygen may lead to respiration, whereas no such evidence was forthcoming concerning the two other suggested causes. At the same time, Barcroft qualifies this opinion by stating that whilst oxygen-want is the essential cause of the first breath the amount of carbonic acid in the blood cannot be ignored. Confirmation of this work will be awaited with interest, as it may go a long way towards the solution of asphyxia in the new-born infant.

Asphyxia neonatorum is commonly divided into two groups—asphyxia livida and asphyxia pallida. The former is regarded as a condition akin to suffocation and the latter to surgical shock. Moncrieff [2] does not subscribe to this classification but maintains that the colour of the baby is no clear guide to the degree of respiratory failure. He does agree that the pallid infants present a more serious problem, and as regards causation states that almost any of the ætiological factors may result in either cyanosed or pale babies. In the cases here considered all the infants died from blue asphyxia, and many were reported to the coroner because of suspicion that death was due to suffocation.

Cruickshank [3] in his monograph on neonatal death draws attention to the fact that œdema of

the brain and meninges is present in almost all cases of asphyxia neonatorum, and my experience fully confirms this. What part the increased intracranial pressure thus produced plays in the production or aggravation of the asphyxia is difficult to decide. It is obvious that the respiratory centres must be embarrassed by such pressure, but it is equally important to bear in mind that asphyxia will produce cerebral congestion and œdema. Which is cause and which effect is the problem. I have separated my cases of frank intracranial hæmorrhage from those of asphyxia neonatorum, and my opinion is that the cerebral œdema found in asphyxia is in the main the result of the asphyxia and not the cause. At the same time it cannot be controverted that the more severe the asphyxia the greater the cerebral œdema, and the greater the cerebral œdema the greater will be the embarrassment of the respiratory centres.

The correct methods of treatment of the asphyxiated newly born infant are now founded on sound knowledge, but the old-fashioned and useless methods die hard. These infants are suffering profoundly from shock; therefore great care in handling and the provision of warmth are of paramount importance. Next comes "the toilet of the air-way,"[4] and thirdly inhalation therapy.[2 4 5] It is not my intention to enter into the details of these methods of resuscitation but I will content myself with drawing attention to three important points in connexion with inhalation therapy. Firstly, carbon dioxide is the normal stimulant to the respiratory centres, but in excessive amount it is a respiratory depressant. Secondly, carbon dioxide can only be produced in the tissues from oxygen. And, thirdly, a cylinder of oxygen is of much greater value than a cylinder of carbon dioxide; the former can do nothing but good, whereas the inspiration of pure carbon dioxide, except for a very brief period, will prove fatal.

Lastly, the association between asphyxia and increased intracranial pressure, to which attention has already been directed, has to be considered. In this connexion treatment by means of hypertonic rectal saline as advocated by Moncrieff[6] undoubtedly constitutes a real therapeutic advance. He advises the introduction of 2–3 oz. of 10 per cent. saline into the rectum of the new-born babe, as soon as any symptoms appear. This should be done slowly, with the baby in the cot and with the minimum of disturbance, and the buttocks should be held together to get as long a retention as possible.

NEONATAL INTRACRANIAL HÆMORRHAGE

I have included in this division only those of my cases with gross intracranial hæmorrhage. In all free blood or blood-clot was present over the surface of the cerebral hemispheres in part or in whole. In some, in addition, hæmorrhage had occurred beneath the tentorium cerebelli but none showed subtentorial hæmorrhage only. Demonstrable tearing of the tentorium was present in 12 instances—in 6 of which the tears were complete—and in a few the falx was also damaged. In one case the source of the hæmorrhage was a tear of the superior longitudinal sinus. Of the 12 in which the hæmorrhage was associated with partial or complete tentorial tears 10 had been born precipitately, 1 was an instrumental delivery due to protracted labour, and the twelfth was a normal labour.

Only 3 of these 17 infants survived for more than twenty-four hours, and most of them died very shortly after birth. One lived for forty-two hours, this being an infant of 6¼ lb. who was born with a normal labour, breathed well, and at autopsy had much free blood over the surface of both cerebral hemispheres.

It is now generally agreed that trauma plays the significant part in the production of neonatal intracranial hæmorrhage. This subject has been studied exhaustively by Holland,[7] who has shown that when the fœtal head undergoes changes in shape alterations in the tension of the dural septa are inevitable, owing to the nature of their attachments to the cranial bones. Most of Holland's cases were the result of breech presentation and forceps delivery through a contracted pelvis, but a few resulted from apparently normal labour and only one was precipitate labour. Capon[8] gives as the determining causes of hæmorrhage excessive head-moulding, too rapid head-moulding, and fœtal asphyxia. Of my 17 cases, the labour was precipitate in 9 and protracted with instrumental delivery in 2; one was a breech presentation, and in the remaining one the labour was stated to be normal or at all events more or less uneventful. These figures thus indicate that too rapid head-moulding is a potent factor in the production of neonatal and intracranial hæmorrhage. Hence the danger of precipitate birth.

Ford[9] maintains that prematurity is an important contributing cause, and my figures support this contention in that over half were under 6 lb. in weight.

Little of importance emerges from a study of the clinical histories of these infants. As most died very shortly after birth, and as almost none were born or treated in any institution, or indeed had been examined by a medical man, no facts of significance can be elicited. In no case was the diagnosis suspected during life, and even if it had been it is difficult to believe that any form of therapy could have saved life, such was the extent of the hæmorrhage.

In my experience with coroners' post-mortems fatal neonatal intracranial hæmorrhage is more likely to follow precipitate labour and too rapid head-moulding than prolonged, difficult, and instrumental deliveries. The obstetricians have, in my opinion, been unjustly blamed in the past for many of these catastrophes.

BRONCHO-PNEUMONIA

If the lungs of all the infants in this series had been examined microscopically there is no doubt that consolidation would have been found in many, for atelectasis and neonatal broncho-pneumonia are closely allied pathological conditions. However, I am here concerned only with those in which the consolidation was so extensive as to be evident to the naked eye, and therefore the primary cause of death. There are 11 of these, of whom the youngest was nine hours, the eldest twenty-eight days, and the average eighteen days old. In 6 the history was to the effect that the infants had appeared to be thriving satisfactorily and had been free from cough or other symptoms of respiratory disorder. Their deaths were unexpected, and in several the information received pointed to death from suffocation.

CONGENITAL DEFECTS

A congenital defect was the cause of death in only 4 of my cases. In several others congenital abnormalities of one kind or another were an accidental post-mortem finding—e.g., solitary kidney—but in none had they contributed to the fatal issue.

All three dying from *congenital heart disease* had some degree of aortic stenosis. The baby with the *cleft palate* was 8½ lb. at birth: various methods of feeding were tried without success, and he really died from marasmus, for at the time of death the weight had dropped to 5½ lb.

SUPRARENAL HÆMORRHAGE

Suprarenal apoplexy is a fascinating and baffling condition, the ætiology of which appears to be wrapped in obscurity. Arnold[10] states that the causal factors mentioned in the literature include asphyxia, trauma, eclampsia in the mother, purpura and fever in the mother, thrombosis of the suprarenal veins, syphilis, hæmophilia, and toxic factors such as burns and diphtheria. In both my cases the infant died shortly after birth, and in both massive hæmorrhage had occurred into one suprarenal gland. They were fully mature, and there is nothing in the histories of either mothers or babies to suggest possible causes.

INTESTINAL VOLVULUS

This was a mature infant that suddenly collapsed on the third day and died without warning. Post-mortem examination revealed a general peritonitis with fæcal material in the peritoneal cavity. Intestinal obstruction had been caused by a volvulus of the cæcal variety and the cæcum itself had perforated.

SEPTICÆMIA

This infant had panophthalmitis. It had lived for thirteen days and had received only some sporadic treatment for ophthalmia during the first few days of life. Death was due to septicæmia.

DEATH DUE TO VIOLENCE

One infant died from *intraperitoneal hæmorrhage* due to a rupture of the liver.

This was the second child of a single woman who had concealed her pregnancy. The baby was born after she had been in labour for some hours and while she was standing upright over a blanket. At autopsy the body was that of a fully mature infant weighing 7 lb. 10 oz. with cord and placenta still attached. On opening the abdomen the peritoneal cavity was seen to be filled with blood due to a rupture of the liver just where the umbilical vein entered. The pull on the umbilical cord just as the baby hit the floor must have produced sufficient tension to cause this rupture of the liver but insufficient to damage the cord externally.

The death from *shock and asphyxia* took place in circumstances which are instructive and probably unique.

A healthy male infant, eighteen days old, was being washed by his mother on her knee and over a bowl of water on the floor when the woman fainted. When she came round she was lying on the floor and the baby's head was in the bowl of water and its feet sticking out. At the post-mortem examination the body was that of a well-nourished infant weighing 7 lb. 12 oz. There was no external bruising of the head or elsewhere. The child was not drowned, and my explanation of this catastrophe is that it was concussed by a blow on the head from hitting the bowl in its fall; and this, combined with the sudden immersion in the water, produced death from shock and asphyxia.

Only three of my cases died from *suffocation*. In view of the fact that in a considerable number of the total cases under review both the circumstances and the external examination of the bodies suggested that death might have been due to suffocation, the very small number in which this was actually proved to have been the cause is arresting. In particular, the external appearances of a baby that has died from pneumonia and one that has been suffocated are usually identical, and in fact differential diagnosis, in spite of the fullest history of all the circumstances, can only be established by post-mortem examination. In Birmingham all these cases are subjected to a detailed autopsy, and my experience leads me to fear that where this practice is not carried out there is a very real danger that mothers may be admonished for causing the deaths of their babies when they are innocent. In consequence I feel that I cannot urge too strongly the universal adoption of the Birmingham policy. It is only by post-mortem examination that the cause of death can be irrefutably ascertained, and by such procedure the risk of stigmatising the parents when such would be unwarranted and unjust will be entirely avoided.

SUMMARY

The clinical and post-mortem findings in 99 cases of death in the intranatal and neonatal period that were reported to the Birmingham coroner have been here reviewed. Of these, 75 failed to survive because of factors directly attributable to birth (accidents associated with labour or inability to establish normal respiration); 15 died from disease not directly associated with birth factors; 4 died from congenital abnormalities; and 5 met their death from violence.

It is appreciated that these constitute a highly selected group and form but a very insignificant proportion of the total stillbirths and neonatal deaths in the city of Birmingham. Furthermore, this series is not complete in itself in that it does not include all the cases in this age-period that were reported to the coroner. Nevertheless, they do constitute a valuable collection for at least two reasons: the causes of death are based on personally performed post-mortem examination, and they occurred amongst a section of the population outside institutional guidance or control and with a minimum of medical supervision; in fact, in the very large majority no medical man had been in attendance. No useful purpose, therefore, could be served by attempting to compare my series with those recorded by other observers, most of which have been obtained from institutional practice. Whilst hospitals must of necessity deal very largely with the abnormal and difficult case, on the other hand modern methods of diagnosis and treatment of the newly born child are always available immediately. In view of these circumstances my cases can best be regarded as a complement to those recorded by others. It is hoped that they will help to shed light from this unaccustomed angle on the extremely important and complex subject of stillbirth and neonatal death.

I have to thank Dr. W. H. Davison, H.M. coroner for the city of Birmingham, for permission to make use of the clinical and post-mortem records of these cases.

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