In *Case 13* cortisone was discontinued because of a perforated gastric ulcer. For the next four weeks the patient progressed satisfactorily, and he was then discharged from hospital at his own request. A week later he deteriorated rapidly. He was readmitted with cachexia, peripheral nerve lesions, and gangrene of the fingers and toes, and died shortly afterwards.

Clearly this was a difficult case to manage. It is pure conjecture whether the risk of restarting cortisone would or would not have been greater than the risk of leaving active polyarteritis untreated.

In *Case 14* the patient died with malignant hypertension 11 months after the start of treatment. Cortisone, which was originally started at the standard dosage of 200 mg. daily, had been decreased after four days because of the dramatic improvement in the patient's general condition. Thereafter the patient received only from 37.5 to 75 mg. daily, except for a terminal increase to 100 mg. Throughout this period there was persistent evidence of active polyarteritis in the skin and joints, and the E.S.R. (Westergren) ranged from 35 to 100 mm. in one hour. This level of cortisone dosage cannot be held directly responsible for the development of malignant hypertension, which was probably due to renal involvement by polyarteritis.

Discussion

This paper illustrates very clearly the difficulties of using retrospective controls. We tried to minimize the factors responsible for differences between treated and control series by choosing consecutive periods and drawing cases mainly from the same hospitals. The two series, as shown in Table II, are closely comparable except in one feature—namely, the incidence of hypertension, which is much higher in the control than in the treated group. Unfortunately, this is an important difference, since analysis of other cases shows hypertension to be one of the most important factors influencing prognosis. When the cases with hypertension are included, the results seem better in the treated than in the control series; when these cases are omitted, the differences are small, though still in favour of the treated series. But since the numbers are so small, and the series are dissimilar in an important respect, comparing them does not justify conclusions about the effect of cortisone on expectation of life in polyarteritis nodosa; though it is to be said that our results are consistent with the idea that cortisone may prolong life.

In view of the importance of the question asked, we felt a moral obligation to try to answer it; but, for the reasons explained earlier, the method of retrospective control seemed the only one possible. The uncertain meaning of the results obtained makes us wonder whether the labour was worth while. Its chief importance is perhaps to emphasize that the assessment of therapeutic activity by the use of retrospective controls is an inherently fallacious method and no substitute for the strictly controlled clinical trial (Hill, 1951).

Those who have used cortisone therapy in polyarteritis nodosa agree that nearly always it improves symptoms conspicuously and suppresses the major manifestations of active disease. To maintain suppression of symptoms may require doses of cortisone which provoke troublesome and occasionally dangerous side-effects. Whether the treatment is better or worse than the disease is a matter on which we have no unequivocal evidence to present.

Summary

Survival rates at the end of one year are recorded in a series of 17 cases of polyarteritis nodosa proved by biopsy and in a retrospective control series of 19 cases proved in the same way.

The results illustrate the fallacies of retrospective controls; at the time of diagnosis the incidence of hypertension, which materially affects prognosis, was greatly

different in the two groups. Differences in survival rate may thus be due to variations over these periods in such factors as the indications for biopsy or the course of the disease.

Cortisone treatment usually produces quick relief of symptoms and suppression of signs. To secure sustained suppression of outward manifestations may require large doses producing troublesome side-effects.

The panel is greatly indebted to Dr. G. A. Rose for compiling the data contained in this report.

The following hospitals co-operated by permitting access to their case material for the collection of information on the retrospective untreated group: Canadian Red Cross Memorial Hospital, Taplow (Special Unit for Juvenile Rheumatism); Guy's Hospital; London Hospital; Middlesex Hospital; Radcliffe Infirmary, Oxford; Royal Infirmary, Edinburgh, and hospitals served by the Edinburgh University Department of Pathology; Royal Infirmary, Manchester; Royal Victoria Infirmary, Newcastle upon Tyne; St. Bartholomew's Hospital; St. Mary's Hospital; St. Thomas's Hospital; University College Hospital; Westminster Hospital.

The following hospitals are co-operating in the clinical trial of cortisone treatment: Canadian Red Cross Memorial Hospital, Taplow (Special Unit for Juvenile Rheumatism); Hammersmith Hospital; Kingston Hospital; Norfolk and Norwich Hospital; Northern General Hospital, Edinburgh; Paddington Hospital; Royal Infirmary, Manchester; Royal Sussex Hospital; St. Mary's Hospital; St. Thomas's Hospital; University College Hospital.

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PERINATAL DEATH FROM FOETAL EXSANGUINATION*

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Foetal exsanguination may not be a frequent cause of perinatal death, but it is a risk to the child's life that can be treated successfully if recognized early, and may on occasion be prevented. For these reasons we record the experience we have had of foetal exsanguination during the years 1951 to 1956. We have previously drawn attention to blood loss from the foetal circulation incurred at caesarean section (Neligan and Russell, 1954, 1955). This paper is concerned mainly with foetal blood loss as it may occur during vaginal delivery and with a method of detecting the foetal source of such bleeding with rapidity and certainty.

Foetal Blood Loss Associated with Vaginal Delivery

In the past five years we have noted at least nine cases where foetal blood has been lost before delivery of the

*This article is based on a symposium presented to the Edinburgh Obstetric Society on December 12, 1956.

baby. Seven of the nine babies lost their lives. In three cases blood was lost from ruptured vasa praevia, and in three others the ruptured vessels lay at a distance from the cervical os and were associated with a velamentous insertion of the cord or a succenturiate lobe of the placenta. In one case there was a circumvallate placenta, but no damage to foetal vessels could be demonstrated. In a further case the mechanism of foetal blood loss remains uncertain, for placenta and cord appeared normal in all respects. The most unusual case was one in which a large foetal vessel, in a normally situated placenta, was damaged at the time of induction of labour with a Drew Smythe catheter.

Brief clinical details of nine cases follow.

Case 1.—A gravida-3 aged 25 was admitted to hospital at term. During the 12 hours before admission there had been slight intermittent vaginal bleeding. The foetal heart was not heard on admission and a few hours later spontaneous delivery occurred of a fresh stillborn child; weight 8 lb. 6 oz. (3.8 kg.). Examination of the placenta revealed ruptured vasa praevia.

Case 2.—A gravida-2 aged 35 was admitted to a private nursing-home at term, following a sudden small vaginal haemorrhage. A slight ooze was noted once or twice during the next 24 hours and the patient was then transferred to our hospital; the foetal heart at this time was quite regular. Over the next few hours a trickle of blood was noted on one or two occasions, but the foetal heart remained regular. Suddenly, eight hours after admission, there was a short brisk vaginal loss and the foetal heart sounds disappeared. A stillborn child weighing 6 lb. 7 oz. (2.9 kg.) was delivered spontaneously two hours later and examination of the placenta showed ruptured vasa praevia.

Case 3.—A primigravida aged 24 was admitted to hospital at 38 weeks, having had a small haemorrhage a few hours previously. The foetal heart was noted to be regular. Four hours after admission a further loss was reported, but this soon ceased. Eight hours later there was a trickle of blood from the vagina, but again this quickly settled. Eleven hours later labour began with rupture of the membranes and the passage of blood-stained liquor. Without warning the foetal heart sounds disappeared. A vaginal examination at this stage revealed a cord-like structure running in the membranes. A fresh stillborn child weighing 5 lb. 14 oz. (2.7 kg.) was delivered spontaneously a few hours later. The placenta showed ruptured vasa praevia.

Case 4.—A primigravida aged 35 was admitted to hospital in labour at term. The foetal heart was regular. Labour progressed normally until the second stage was reached, when the foetal heart sounds suddenly disappeared. Ten minutes later the foetal head appeared at the vulva and was accompanied by a small gush of blood. A fresh stillborn child weighing 7 lb. 14 oz. (3.6 kg.) was delivered; the child was noted to be very pale. Examination of the placenta showed a velamentous insertion of the cord, and one large foetal vessel had ruptured.

Case 5.—A primigravida aged 20 had a surgical induction of labour at the 36th week of pregnancy because of pre-eclampsia associated with twin pregnancy. Twin babies were subsequently delivered spontaneously; neither before nor during labour was any haemorrhage observed. The second twin showed marked pallor at birth and the haemoglobin was 73%. There was a velamentous insertion of the cord and two foetal vessels were seen to be torn across. A blood transfusion was given and the baby recovered.

Case 6.—A gravida-5 aged 37 was admitted to hospital at term because four days previously there had been a small haemorrhage followed by intermittent staining. Vaginal examination in the theatre revealed a type I placenta praevia. The membranes were ruptured with a catheter and labour began shortly afterwards. The foetal heart was regular. At the end of the first stage of labour there was further small loss of blood and the foetal heart was not heard. The baby was stillborn and weighed 7 lb. 8 oz. (3.4 kg.). The praevia portion of the placenta was noted to be a succenturiate lobe

and one of the vessels joining it to the main placenta had ruptured.

Case 7.—A gravida-10 aged 30 was admitted to hospital at term in labour. Two hours before admission she had had a small vaginal haemorrhage. One hour after admission there was a further slight loss and the foetal heart became irregular. The patient was examined under anaesthesia; no placental tissue was felt and the membranes were ruptured. A baby weighing 6 lb. 4 oz. (2.8 kg.) was subsequently delivered by forceps and was noticed to be pale at birth. The haemoglobin was 95% and fell in one hour to 75%. A blood transfusion was given and the baby made good progress. The placenta was circumvallate, but no damage to foetal vessels was found.

Case 8.—A primigravida aged 32 was admitted to hospital at term in labour. When the cervix was 4 fingerbreadths dilated there was a loss of 2 to 3 oz. (60–90 ml.) of fresh blood and the foetal heart became irregular. A precipitate delivery occurred 15 minutes later. The baby was pale and died 45 minutes later. Post-mortem examination revealed marked exsanguination of all foetal organs. Examination of placenta and cord failed to show any injury to the foetal circulation.

Case 9.—A gravida-5 aged 29 had a surgical induction of labour at the 38th week of pregnancy because of pre-eclampsia. No placenta was felt, but a "blood-stained tap" was obtained with a Drew Smythe catheter. For the next 28 hours there was an intermittent slight ooze of blood from the vagina and then the foetal heart suddenly became irregular. The baby was delivered by caesarean section and was noted to be pale at birth. The baby's haemoglobin was 70% and a transfusion was given. The baby improved but died 52 hours later. Examination of the placenta revealed a puncture hole in a large foetal vessel and this area was surrounded by blood clot.

Comment on the Cases

It will be noted that in eight of these cases small antepartum or intra-partum haemorrhages occurred. The origin of the blood loss, however, was not appreciated until after delivery, and this drew our attention to the need for a simple and reliable test to differentiate foetal from maternal blood. In order to ascertain whether the application of such a test would be of practical value it was first of all necessary to determine the time interval between the onset of vaginal bleeding and the delivery or intrauterine death of the foetus. We believe that the test described below would have been of value in Cases 2, 3, 7, and 9 (Table I). The test might also

TABLE I.—Time Intervals in Hours Between Onset of Vaginal Bleeding and Delivery or Intrauterine Death of Foetus

Case No.	Intra-partum Death	Born Alive but Died	Recovery Following Blood Transfusion
2	32 hours		
3	25 "		
7			
9		29 hours	3½ hours

have been of value in Case 1, but we cannot be sure, for we do not know when the foetal heart sounds disappeared in relation to the repeated small vaginal haemorrhages. In Case 4 the foetal heart sounds disappeared before there was any bleeding, and in Case 5 no vaginal bleeding was noted. We are uncertain about Case 6; for the first bleeding, four days before delivery, may well have been due to partial separation of the succenturiate placental lobe.

Detection of Foetal Bleeding before Delivery

Adult and foetal blood differ in several respects (Lancet, 1954), and, though methods for distinguishing the two types of blood on the basis of different properties are available, most of them, at present, are not suitable as a diagnostic test. Williamson (1912) suggested that foetal bleeding might

be detected in a vaginal blood loss by recognition of nucleated red blood cells, but this test is impracticable.

In our attempts to find a practicable and reliable diagnostic test for the detection of foetal bleeding use has been made of the fact that foetal haemoglobin is much more resistant to denaturation by alkali than is the adult haemoglobin. In the human foetus traces of adult haemoglobin first appear by the tenth week and the amount slowly increases so that, at term, on average, 20% of the total haemoglobin is of adult type. After birth the percentage of foetal haemoglobin decreases, and by the age of 4 months 90% is of adult type: in the normal adult foetal-type haemoglobin is present, but in minute amounts.

Singer, Chernoff, and Singer (1951) devised a method using the alkali-denaturation technique for the detection of foetal haemoglobin, but in its original form it is not suited to the needs of routine obstetrical practice. Clearly the test would have to be done in all cases of ante-partum and intra-partum haemorrhage in order to detect the few cases where the blood was of foetal origin: speed and simplicity are essential, for the clinician himself must usually do the test and act upon the result.

Rapid Modification of Singer Test

The following test, which is a modification of Singer, Chernoff, and Singer's method, was accordingly devised by one of us (G. S. A.) to meet the necessary requirements: the test takes between five and ten minutes.

Principle.—A sample of blood is allowed to react with an alkali for a set period of time. The reaction is then stopped by an acid solution which also precipitates non-haemoglobin chromogens. The mixture is filtered immediately and the filtrate examined by the naked eye and in a colorimeter.

Reagents.—Alkali: N/12 NaOH or N/12 KOH.* Acid-precipitating solution: 800 ml. of 50% saturated $(\text{NH}_4)_2\text{SO}_4$ plus 2 ml. of 10 N HCl. Blood:—The sample is either pure blood or a mixture of blood and other contaminants issuing from the vagina. It should be collected in a clean heparinized bottle. If collected in an oxalated tube or by means of a siliconed syringe there is a danger of "foetal results" being obtained from adult blood.

Technique.—The haemoglobin level of the blood sample is first determined. To 2 ml. of alkali, 0.1 ml. of blood is added and the mixture is shaken. The reaction is allowed to proceed for one minute at room temperature, when 4 ml. of acid-precipitating solution is added and the mixture again thoroughly shaken. It is then filtered through a No. 1 Whatman filter paper and the filtrate collected.

Results of Test

The results are usually obvious by naked-eye examination. With adult type haemoglobin the filtrate is either clear and colourless or a faint brown-amber colour. With foetal type haemoglobin it varies from a faint pink to a distinct cherry red, depending on the amount of haemoglobin in the sample.

Readings on the colorimeter have been determined, first, so that foetal blood might be detected even when mixed with blood; and, second, to lessen the danger of a mistaken result that might be possible on naked-eye examination alone. The readings obtained by us on an E.E.L. colorimeter, using a green Chance glass filter OGRI with the filtrate in small-bore tubes ($3\frac{1}{8}$ by $\frac{1}{4}$ in.—8 by 0.6 cm.), are presented in Table II. It will be seen that the haemoglobin level of the sample makes an appreciable difference in foetal blood (12 or more units) and a slight difference in adult blood (4 units), but that a range of readings for both types of blood at different haemoglobin levels has been established. It will also be noted that the range for adult blood of 50% haemoglobin (7.4 g.) and above approaches the range for foetal blood of 50% (8.5 g.) and under—that is, samples of

TABLE II

Adult Blood		Foetal Blood	
Haemoglobin		Haemoglobin	
Above 50%	Below 50%	Above 50%	Below 50%
46 samples. Highest reading 10	40 samples. Highest reading 6	44 samples. Lowest reading 22	36 samples. Lowest reading 10

In all estimations of haemoglobin the M.R.C. grey-wedge photometer has been used.

foetal and adult blood may give a reading of 10 units, depending on the haemoglobin level. There seemed to be no advantage in making a more complicated division than the one adopted (50% haemoglobin level), for the reading of 10 units obtained with foetal blood was on one specimen only and this had been diluted to 16% (2.72 g.): all other samples gave readings well above 10 units. With adult specimens, only two gave readings of 10 units; four more gave readings above 7 units, but the remainder were below this figure.

There may be a failure to detect foetal blood when the sample contains very little haemoglobin—for example, below 15% (2.55 g.). The colour change under these circumstances may be so slight that the photometer reading falls into the adult range. This circumstance may arise when faintly blood-stained liquor is obtained at surgical induction of labour. With such a sample too little haemoglobin will be contained in 0.1 ml. to give either a clear visual change after alkali treatment or a reliable colorimeter reading after the rapid alkali denaturation test. The difficulty can be overcome by using a larger quantity of the blood-stained liquor. Into each of two test-tubes about 4 ml. of sanguineous liquor is placed and to one, the control, an equal amount of water is added. To the other an equal amount of N/12 alkali is added and the mixture shaken. With adult blood a colour change will be observed at five minutes, when the colour becomes greenish amber. Foetal blood does not show any changes at five minutes, but a colour change does begin at about 30 minutes, to be completed in several hours.

Meconium-stained liquor provides a complicating factor in that heavily contaminated liquor, if put through the rapid alkali denaturation test, will give a reading of 2 or 3 units. Pure meconium can give a reading of 10 units. Fortunately the occurrence of ante-partum haemorrhage with the escape of meconium-stained liquor is rare.

Mixtures of Adult and Foetal Blood

The circumstances under which loss of foetal blood might occur in a vaginal delivery or following a surgical induction of labour are such that only a small admixture by adult blood is likely. In this connexion it will be remembered that foetal blood itself is a mixture of adult (20%) and foetal (80%) haemoglobin at term, and before term the percentage of foetal haemoglobin is higher. Also the level of haemoglobin in the foetus is higher than the level in adults by 3 to 4 g. per 100 ml. It has been found by experiment that, provided the haemoglobin of the mixture of adult and foetal bloods is above 50%, as little as 1 part of foetal blood in 20 of adult can be detected. When the haemoglobin of the mixture is below 50%, this ratio becomes 1 in 10.

Foetal Haemoglobin in Adult Blood

Foetal haemoglobin in amounts that would be detected by the test described are not produced by normal adults. In sickle-cell anaemia and in thalassaemia (Cooley's anaemia; Mediterranean anaemia), however, appreciable amounts do occur. For all practical purposes these disorders are limited to the Negro and Mediterranean races, but an Italian woman with thalassaemia was recently seen in this hospital; foetal haemoglobin was present in the circulating blood and was detected by this test. The transplacental transfer of foetal red blood cells, first suggested by Wiener (1948), has been shown by Rucknagel and Chernoff (1955) to be an

*Singer *et al.* specify N/12 KOH at exactly pH 12.7. It has been found that the concentrated volumetric solutions of NaOH (B.D.H.) can be used to make a satisfactory N/12 solution when one ampoule is dissolved in 600 ml. of distilled water.

uncommon occurrence. Using a more sensitive technique than alkali denaturation, they detected foetal haemoglobin in significant amounts in 10 out of 91 pregnant women, although the highest amount detected was only 1.37%. The original alkali-denaturation test of Singer *et al.* is insensitive to amounts of foetal haemoglobin of approximately 2% or less. The modified test, as we describe, is even less sensitive, detecting 5 to 10% of foetal haemoglobin, depending on the haemoglobin level of the sample.

To obtain a 5% mixture in a normal adult, assuming the total blood volume in the adult to be six litres, would require 300 ml. of foetal blood, and this seems to us to be more than could be lost from a viable infant. Bleeding by the foetus into the maternal circulation would not therefore invalidate the test.

Application and Interpretation of the Rapid Alkali-Denaturation Test

The following procedure is carried out in this hospital. In cases of ante- and intra-partum haemorrhage, and when a blood-stained tap has been obtained at surgical induction of labour, the sample of blood is collected and the haemoglobin is estimated. Blood is obtained by venepuncture from the mother and the haemoglobin estimated. The test, as outlined above, is carried out on both samples of blood and the interpretation made against the reading obtained on the venepuncture specimen and against the general background of colorimeter readings (Table II).*

If the colorimeter reading of the vaginal specimen of blood is 11 or above, then foetal blood is clearly present, although the mother's venous-blood reading must always be obtained to check the technique and to avoid the possibility that she is the source of the foetal haemoglobin. Where the vaginal specimen of blood gives a reading of from 7 to 11, then the various colorimeter results given in Table II cease to be of value, and one must then critically compare the colorimeter reading obtained on a venepuncture specimen of blood in which the haemoglobin level has been equated to that of the vaginal specimen. Since the experimental error in performing the test is not greater than one unit, it is considered safe to allow that, if the vaginal specimen of blood gives a reading of 2 or more units higher than the venepuncture specimen at an identical haemoglobin level, foetal blood is present.

Discussion

It has long been known that foetal vessels in the placenta may be damaged during a caesarean section. Leopold (1888) and Kelly (1891) both warned against the practice of boring through an anterior placenta; they pointed out that if this were done the foetus might bleed to death. Siddall and West (1952) and Wickster (1952) have drawn attention again to this accident. Neligan and Russell (1954, 1955) reported upon the circumstances of the accident, and upon its frequency and management. An operative technique was described in which they stressed the importance of circumventing the placenta and clamping the umbilical cord as soon as the baby's face had been delivered. Russell and Warwick (1955) have shown how radiology can help in the management of these cases by warning the obstetrician of the presence of an anterior placenta praevia. The operator may even receive guidance on how best to circumvent the placenta without injuring it. Since 1952 the obstetric and paediatric staffs in our hospital have followed the routine described by Neligan and Russell, and no baby born by caesarean section has lost its life from foetal exsanguination.

Whereas we feel confident that the risk to the baby's life incurred by damage to foetal vessels at caesarean section has been overcome, the danger of foetal exsanguination from other causes still remains. We have given details of nine known cases in the last five years, but there may have been others of lesser degree. Reliance on clinical features

alone will result in a high foetal mortality. In six of the nine cases foetal distress was either not noted or did not precede the vaginal bleeding by a sufficient length of time to be of practical value. This observation fits in well with the findings of Neligan and Russell (1954), who noted that the exsanguinated baby may appear deceptively well until shortly before death. In one case, however, foetal distress was present for almost one hour before delivery was effected by forceps. This baby received a blood transfusion and survived (Case 7). In Case 9, foetal distress was noted for half an hour before delivery by caesarean section. The warning, however, was too late, for the baby died in spite of blood transfusion. The other case (No. 1), an emergency admission, resulted in the death of the foetus before arrival in hospital, and no records are available about the duration of foetal distress.

A characteristic feature of the bleeding in eight of the nine cases has been its small amount—enough, however, in each case to warrant a diagnosis of ante-partum haemorrhage. Cases 7 and 8 are of some interest in that we could demonstrate no damaged foetal vessel, yet there was clear evidence of foetal exsanguination. These two cases may be examples of the foetus bleeding into the maternal circulation as has been reported by Wiener (1948), Wickster (1952), and Chown (1954). In both cases, however, there was some vaginal bleeding, and this may have been foetal blood from an undiscovered source. The exact pathological mechanism of blood loss in these cases still remains uncertain, but we record them because they draw attention to the possibility of haemorrhagic shock in babies noticed to be pallid at birth, despite the failure to detect damage to foetal vessels.

In the series of nine cases that we report here, only two babies survived. We hope that routine use of the test described above in cases of ante-partum haemorrhage will enable us to diagnose foetal blood loss earlier and lead to a higher foetal survival.

Summary

Foetal exsanguination, although an infrequent cause of perinatal death, is on occasion preventable.

By careful attention to obstetric and paediatric techniques it has been found possible to overcome the risk to the baby's life incurred by damage to foetal vessels at caesarean section.

The risk of foetal exsanguination from other causes remains, and nine cases are described. In these cases reliance on clinical features did not give sufficient warning for prompt treatment to be undertaken.

Attention is drawn to the need for a rapid, reliable test to show when foetal blood is present in the vaginal loss in cases of ante-partum haemorrhage. A rapid alkali denaturation test for the detection of foetal blood is described.

We are indebted to Professor Harvey Evers and Professor Donald Court for their advice and encouragement and for permission to publish details of cases admitted to the Princess Mary Maternity Hospital. We also thank Dr. R. B. Thompson for his interest and help in the development of the test to differentiate foetal from maternal blood.

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*These results apply only to the E.E.L. colorimeter in this hospital.