premedication failed to prevent a slowing of the heart rate on the subsequent injection of suxamethonium. Similarly, the intravenous injection of 0.6 mg. of atropine also failed to afford protection. In this investigation, however, the intravenous administration of 1 mg. of atropine sulphate prevented the subsequent development of bradycardia or arrhythmia in every case, even when large doses of suxamethonium were used.

The clinical significance of these findings is that the intermittent use of large doses of suxamethonium—designed to produce prolonged muscular relaxation—may give rise to a dangerous arrhythmia. Although a proved fatality from this method has not yet been reported, these results suggest that were such a technique used for a patient with myocardial ischaemia a fatal outcome might arise. Furthermore, if it is necessary to give large doses of suxamethonium intermittently, it is recommended that a dose of atropine (1 mg. intravenously) should precede the second injection of suxamethonium.

Summary

A slowing of the heart rate was found in 39 of a series of 41 patients after repeated suxamethonium injections.

Electrocardiographic recordings showed this slowing was a sinus bradycardia in over two-thirds of the patients, with irregularities of rhythm occurring in the remainder.

The nature of this arrhythmia was a depression of excitation and conduction of the cardiac impulse which in its severest form produced a cardiac arrest of seven seconds.

The bradycardia or arrhythmia appears to be related to the size of the repeat dose used, and not to the number of doses or to the total amount of suxamethonium given.

Until further work has revealed the cause of these changes, atropine is suggested as a safeguard against these potentially dangerous effects.

We are indebted to Dr. Evan Jones for his reading and interpretation of the electrocardiographic tracings. We thank Professor J. B. Kinmonth and Mr. F. B. Cockett for kindly allowing us to investigate their patients, and Mr. T. W. Brandon for photographing the electrocardiographic tracings.

REFERENCES

Bullough, J. (1959). *Brit. med. J.*, **1**, 786.
Leigh, M. D., McCoy, D. D., Belton, M. K., and Lewis, G. B. (1957). *Anaesthesiology*, **18**, 698.
Martin, K. H. (1958). International Symposium on Curare and Curarelike Drugs. Atti XI Congresso Societa Italiana de Anestesiologia, Venezia, September, 1958, p. 362.
Telford, J., and Keats, A. S. (1957). *Anesthesiology*, **18**, 841.
Verner, I., and Comty, C. (1959). *Brit. med. J.*, **1**, 1239.

An experiment in helping mentally handicapped men, women, and young people to lead normal working lives is to begin shortly in Northern Ireland. Twelve women and girls, aged 18 and over, will be taken out of special institutions, jobs will be found for them, and they will live in the ordinary community surroundings in a small hostel. The hostel will be in charge of a warden, who will be available to discuss personal problems that arise. The women and girls going into the first hostel are being transferred from special-care centres, where they have received expert training, and they will go out to work each day in the normal way. Some of them will work in local hospitals in the kitchens and laundries, and others may be placed in jobs with outside firms. If this experiment succeeds there will later be separate hostels for men.

EFFECTS OF EARLY AND LATE CLAMPING OF UMBILICAL CORD ON INFANT'S HAEMOGLOBIN LEVEL

BY

PHILIP LANZKOWSKY, M.D., D.C.H.*

Department of Child Health, University of Capetown, and Red Cross War Memorial Children's Hospital, Rondebosch, Capetown, South Africa

This paper describes a controlled trial designed to compare the haemoglobin levels of infants whose umbilical cords were clamped immediately after birth with those whose cords were stripped and clamped later.

The objective was to determine whether there was any appreciable difference in haemoglobin values or in general well-being of the infants which could be ascribed to the timing of the clamping of the umbilical cord. This trial was one of the investigations conducted in order to study the reasons for the local prevalence of iron-deficiency anaemia in infancy and childhood (Lanzkowsky and McKenzie, 1959; Lanzkowsky, 1959, 1960a, 1960b).

Many workers have suggested that early clamping of the umbilical cord deprived the infant of blood from the foetal side of the placenta which would otherwise be pumped into the circulation of the infant by post-partum uterine contractions and that the deficit might be considerable, even as much as 215 ml. There is controversy about the quantity. If it were large it would mean a considerable reduction in the potential iron content of many newborn infants.

This subject can only be considered against a background of physiological and other factors.

Blood Volume of Newborn

There are several reports on total blood-volume determinations in neonates (Schücking, 1879; Lucas and Dearing, 1921; Bakwin and Rivkin, 1924; Robinow and Hamilton, 1940; Brines *et al.*, 1941; DeMarsh *et al.*, 1942; Mollison *et al.*, 1950). Brines *et al.* and DeMarsh *et al.* used the Evans blue method, and Mollison *et al.* employed isotopes in blood-volume estimations. The other authors used less reliable techniques—the perfusion method (Schücking) or the brilliant vital red dye method (Lucas and Dearing; Bakwin and Rivkin; Robinow and Hamilton). In the reports prior to that of Brines *et al.* (1941) the estimated blood volume of infants varied from 6.5% to 14.9% of the body weight. This wide discrepancy may be due in part to the unreliability of the method employed for volumetric estimation and in part to failure to consider the mode of handling and the time of clamping of the cord and the time of the estimation in relation to birth.

DeMarsh *et al.* (1942) estimated the blood volumes of 35 newborn infants. In 18 of these infants the umbilical cord was clamped immediately after birth. Blood-volume determinations were made between 15 minutes and 3 hours after birth, and on the third day of life. They obtained an average total blood volume of approximately 361 ml. between birth and the third day of life in infants receiving placental blood, and an average of 301 ml. in those infants deprived of it by early clamping of the cord. This indicates that infants

*Present address: Paediatric Unit, St. Mary's Hospital Medical School, London.

whose cords were clamped late had an average of 60 ml. greater blood volume. These average blood-volume values compare reasonably closely with that of approximately 300 ml. obtained by Brines *et al.* (1941). The mean values of 460 ml. obtained by Lucas and Dearing (1921) and 570 ml. obtained by Bakwin and Rivkin (1924) were determined by methods which are now considered to be inaccurate. Mollison *et al.* (1950) estimated blood volumes at birth by an isotope dilution method and found a mean value of 84.7 ml./kg. and a range of from 70 to 100 ml./kg. in 34 normal infants. These values closely approximate those determined by Brines *et al.* and DeMarsh *et al.*

Gairdner *et al.* (1958) have drawn attention to the occurrence of the post-natal fluid shift from the vascular compartment with resultant haemoconcentration occurring within the first hour or two after birth. They have stated that this must be taken into account when interpreting studies involving blood-volume measurement in the immediate post-natal period. Gairdner (1958) states that at birth most infants have a blood volume appreciably larger, by 10% or more, than when it is measured a few hours after birth.

Placental Blood Volume

Many workers have determined the amount of blood present in the placental circulation and available to the baby after birth if delayed clamping of the cord is practised. Budin (1875, 1876) and Schücking (1877) observed that about 100 ml. of blood flowed from the placenta to the infant after birth. Haselhorst and Allmeling (1930) found that the average amount of blood in the vessels of 120 placentas was 104 ml. Goodall *et al.* (1938) obtained an average of 125 ml. of blood for use in a blood bank, whereas Grodberg and Carey (1938) salvaged a maximum of 215 ml. from one patient. DeMarsh *et al.* (1942) stated that the placental blood averages 107 ml., and Windle (1950) reported an average of 108 ml. Colozzi (1954) quoted an experiment conducted at Cambridge City Hospital in which approximately 500 samples of cord blood were drawn and the average volume of placental blood was found to be 75 to 80 ml. Gairdner (1958) has stated that placenta and cord contain rather more than 100 ml. of blood.

The aforegoing reports suggest that approximately one-quarter to one-third of the foetal-placental blood volume remains in the placenta if the cord is clamped early. DeMarsh *et al.* (1942) have estimated that between 26 and 34% of the average total foetal blood is in the placental circuit at the end of intrauterine life. This is of special importance in regard to premature infants, since the placenta, which reaches maximum size at about the fifth month, contains a greater proportion of foetal-placental blood volume in the case of premature infants than the case of full-term infants. This is in conformity with the report by Engel (1885) that the mortality in premature infants whose cords were tied early was twice as high as in premature infants whose cords were tied late.

Neonatal Haemoglobin Level

There are several reports dealing with the effect of early and late clamping of the cord on the haemoglobin level of the neonate. Schiff (1901) and Scipiades (1903) noted that if the cord was clamped late the red blood cell count and haemoglobin levels at some hours or days after birth were higher than values found if the cord was clamped early. Börner (1928), using the Bürker method, showed that the mean haemoglobin level in five infants whose cords were clamped immediately was 16.94 g./100 ml. as compared with the mean haemoglobin level of 19.49 g./100 ml. in 30 cases in which the cord was clamped late. Phillips (1941) found in 37 cases a mean haemoglobin level of 15.6 g./100 ml. when the cord was clamped immediately and 19.3 g./100 ml. when the cord was clamped late.

DeMarsh *et al.* (1941), using the Hellige–Sahli apparatus calibrated by the oxygen capacity technique for haemoglobin estimation, found that the mean haemoglobin level in capillary blood at 20 to 75 minutes after birth was 18.94 g./100 ml., in 25 normal healthy infants when the cord was clamped within 30 seconds after birth, and 21.6 g./100 ml. in 25 similar infants when the cord was clamped after the placenta had separated from the uterus. Throughout the first post-natal week the infants whose cords were clamped early had significantly lower haemoglobin values (19.5 g./100 ml.) than those in whom the cords were clamped late (22.1 g./100 ml.). DeMarsh *et al.* (1948), who used blood from the superior sagittal sinus, showed that when the umbilical cords were clamped immediately the mean haemoglobin level was 16.4 g./100 ml. at birth and 17.4 g./100 ml. on the third day of life as compared with values of 20.6 g./100 ml. and 20.8 g./100 ml. respectively when the cords were clamped after separation of the placenta. Siddall *et al.* (1952) showed that in 50 infants delivered by caesarean section the mean haemoglobin level was 15.9 g./100 ml. when the cord was clamped immediately, whereas the value was 17.2 g./100 ml. in 50 infants in whom the cord was stripped. They excluded all cases in which haemorrhage occurred from the foetal-placental circulation due to extension of the uterine incision into the placenta, as well as infants showing evidence of rhesus incompatibility. They did not observe any ill-effects from stripping the cord. Colozzi (1954) used blood samples from the heel and made estimations by the Haden–Hausser method. He determined the haemoglobin level of 100 normal healthy infants in whom the cord was dealt with

TABLE I.—*Summary of Literature on Effect of Mode of Clamping of Umbilical Cord on the Haemoglobin Level (g./100 ml.) in the Newborn*

Author	Early Clamping	Late Clamping	Stripping	Postnatal Time of Hb Estimation	Method
Schiff (1901)		Higher		Hours–days	
Scipiades (1903)					
Börner (1928)	16·94 (5)	19·49 (30)			Bürker
DeMarsh *et al.* (1941)	18·94 (25)	21·60 (25)		20–75 minutes	Hellige–Sahli
	19·50 (25)	22·10 (25)		During 1st week	
Phillips (1941)	15·6 (37)	19·3 (33)		20–30 hours	Osgood–Haskins
DeMarsh *et al.* (1948)	16·4	20·6		"Immediately"	Hellige–Sahli
	17·4	20·8		3rd day	
Siddall *et al.* (1952)	15·9 (50)		17·2 (50)		
Colozzi (1954)	14·85	17·50	18·62	24 hours	
	14·71	17·30	18·96	72 ,,	Haden–Hausser

Figures in parentheses denote number of cases.

in four different ways. He showed that the mean haemoglobin level at approximately 24 and 72 hours after birth was 18.62 and 18.96 g./100 ml. respectively when the infant was placed below the level of the placenta and the cord stripped four to eight times, as compared with 14.85 and 14.71 g./100 ml. respectively when the cord was clamped immediately after birth. McCausland *et al.* (1950) maintained that stripping accomplishes more than delayed clamping. Duckman *et al.* (1953) showed that the haemoglobin level was, on average, 3.4 g./100 ml. higher in infants who acquired an extra 50 g. or more of blood by delayed ligation of the cord as compared with controls.

Table I summarizes the literature on the effect of the mode of clamping of the umbilical cord on the haemoglobin level of the newborn.

According to DeMarsh *et al.* (1941), cessation of pulsation of the cord is not an indication that the placental vessels are empty. Expulsion of blood from capillaries and veins of the placenta into the infant appears to be accomplished by uterine compression of the placenta as well as vasoconstriction of its vessels. Colozzi (1954) also demonstrated that uterine contraction played a major part in forcing blood through the umbilical vein. Windle (1950) stated that the arteries stopped pulsating shortly after delivery, so that only the veins through which the blood is forced into the foetus with each uterine contraction remained distended. Duckman *et al.* (1953) and Whipple *et al.* (1957) stated that when the cord stops pulsating transference of blood from the placental vessels and cord to the infant is almost entirely by gravity.

It is difficult to make a precise assessment of the published data on the effect of early and late clamping of the umbilical cord on the infant's haemoglobin level. Many workers have based their conclusions on haemoglobin and blood-volume estimations which were made by unreliable methods. Some of the reports do not record essential information such as the post-natal time and site of blood-sampling and the weights of the infants. These factors are important considerations in assessment of infant haemoglobin levels.

Although all authors have agreed that there is a significantly higher haemoglobin level in the immediate neonatal period in infants whose cords have been clamped late, no series of cases except that of Wilson *et al.* (1941) has been followed to show whether or not there is any lasting benefit. This follow-up must be done not later than three months of age, after which time dietary factors affect the haemoglobin level.

An investigation was therefore conducted on a large series of infants from birth to three months of age. This work was adequately controlled in respect of the aforementioned factors which may affect results in an investigation of this nature.

Present Investigation

The survey was conducted at one of the maternity institutions attached to the University of Capetown. This hospital has two labour wards, and the patients are admitted at random alternately to each ward. Before the beginning of this trial it was decided that the umbilical cord would be clamped immediately—that is, within 15 seconds of delivery—in every infant delivered in the one ward, and that in every infant delivered in the other ward the cord would be clamped only after the signs of placental separation had occurred and after the cord had been stripped four or five times from the vulva to the infant's umbilicus. In the period between birth and the clamping of the cord, the infant lay on the bed between the mother's legs—that is, slightly below the level of her uterus. The use of two labour wards simplified administration and excluded bias in selection.

In order to keep the series as homogeneous as possible infants of rhesus-negative mothers were not included in this survey. Twins, premature infants, and infants born to mothers who had haemorrhagic episodes before or during birth, as well as infants delivered by caesarean section, were also excluded. Cases of mid-forceps, rotation, and breech delivery, and cases in which any traumatic factors were involved, were omitted. Only infants delivered without untoward incident were included. There were no deaths among the infants in this investigation.

A total of 133 white women and their infants were studied. In 63 cases the umbilical cord was clamped immediately the infant was born (group 1), and in 70 only after placental separation had occurred and after the cord had been stripped (group 2).

Serological tests for syphilis were negative in all the mothers. None of them had received any iron medication during pregnancy. The mean age and mean parity

TABLE II.—*Comparison of Maternal Factors and Haematological Findings in Group 1 and Group 2 Women*

	Age (Years)			Parity			Haemoglobin g./100 ml.			P.C.V. %			M.C.H.C. %		
	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.
Group 1 ..	63	26·70	5·92	63	2·6	1·71	63	11·54	1·36	62	40·05	4·05	62	28·78	1·46
„ 2 ..	70	25·57	5·63	70	2·2	1·76	68	11·54	1·44	68	39·75	4·33	68	28·94	2·12
Significance t:	p>0·25 (N.S.)			p>0·10 (N.S.)			t=0 (N.S.)			p>0·05 (N.S.)			p>0·25 (N.S.)		
Significance F:	p>0·05 (N.S.)			p>0·05 (N.S.)			p>0·05 (N.S.)			p>0·05 (N.S.)			p<0·01		

N.S.=Not significant.

TABLE III.—*Comparison of Mean Birth Weights and Haemoglobin Levels in the First 96 Hours of Life in Infants Born to Group 1 and Group 2 Women*

	Birth Weight (Pounds)			Haemoglobin Levels (g./100 ml.)								
				Birth to 12 Hours			13 to 24 Hours			72 to 96 Hours		
	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.
Group 1 ..	63	7·77	0·95	17	19·80	2·15	46	18·20	2·01	62	18·13	1·88
„ 2 ..	68	7·49	1·05	31	20·14	1·96	39	19·86	1·92	69	19·74	1·82
Significance t:	p>0·10 (N.S.)			p>0·25 (N.S.)			p<0·001			p<0·001		
Significance F:	p>0·05 (N.S.)			p>0·05 (N.S.)			p>0·05 (N.S.)			p>0·05 (N.S.)		

N.S.=N significant.

of the women in the two groups were similar. In the group 1 mothers the mean haemoglobin, packed cell volume, and mean corpuscular haemoglobin concentration values were 11.54 g./100 ml., 40.05%, and 28.78% respectively; compared with 11.54 g./100 ml., 39.75%, and 28.94% in the group 2 mothers (Table II). In none of these respects was the difference between the two groups statistically significant, and the groups were strictly comparable.

The mean birth weight of the infants was 7.77 and 7.49 lb. (3,525 and 3,395 g.) in groups 1 and 2 respectively. The difference is not statistically significant (Table III).

Methods

Blood investigations were done on venous blood in the mothers. In the infants capillary blood was used, taken in every instance from the heel. The haemoglobin estimations were done by the oxyhaemoglobin method, using a Klett–Summerson colorimeter previously calibrated for the purpose against standard haemin and cyanmethaemoglobin solutions. Packed cell volume estimations were made by the standard Wintrobe (1929) procedure.

Results

The mean haemoglobin levels between birth and 12 hours, 13 and 24 hours, and 72 and 96 hours were 19.80, 18.20, and 18.13 g./100 ml. respectively in the infants in group 1, compared with 20.14, 19.86, and 19.74 g./100 ml. respectively in the infants in group 2. The difference between the two groups in respect of haemoglobin values between birth and 12 hours is not statistically significant, whereas the differences between the values at the other two ages are statistically significant (Table III).

All these infants were followed in the first three months after birth. Any who were given iron medication or who developed infections during these three months were excluded from further study. Of the original 133 infants, 112 (54 of group 1 and 58 of group 2) were examined at the end of three months. The feeding of the two groups was comparable; 44% of group 1 and 40.7% of group 2 infants were breast-fed. None of the artificially fed babies received milk fortified with iron. Table IV shows that the mean body weight in group 1 infants was 12.07 lb. (5,475 g.) compared with 11.87 lb. (5,385 g.) in group 2 infants. This difference was not statistically significant.

TABLE IV.—*Comparison of Mean Haemoglobin Levels and Weights at 3 Months of Age in Infants Born to Group 1 and Group 2 Women*

	Haemoglobin g./100 ml.			Weight (Pounds)		
	No. of Cases	Mean	S.D.	No. of Cases	Mean	S.D.
Group 1 ..	54	11·08	1·03	54	12·07	2·21
,, 2 ..	58	11·09	0·93	58	11·87	1·64
Significance { t:	p>0·25 (N.S.)			p>0·25 (N.S.)		
Significance { F:	p>0·05 (N.S.)			p<0·025		

N.S.=Not significant.

The mean haemoglobin level at 3 months of age in the two groups was almost identical. In group 1 it was 11.08 and in group 2 it was 11.09 g./100 ml.

Discussion

The primary purpose of the present investigation was to ascertain and compare the effect on the infant's haemoglobin level of early and late clamping of the umbilical cord, with special reference to the possible bearing of the mode of clamping on the incidence of anaemia in later infancy.

Hitherto information on this subject has been almost entirely confined to observations made in the immediate neonatal period. Search of the literature has revealed only one report on a small series of cases followed for 9 to 10 months post-natally (Wilson *et al.*, 1941). Wilson and his co-workers found higher mean haemoglobin levels in the infants whose cords had been clamped late than in the early-clamped group of infants. Information about their feeding was inadequate. The mean weight of the infants whose cords were clamped early was 525 g. more than those in whom the cord was clamped late. The groups were, therefore, not comparable and, furthermore, illnesses which were present in a number of the infants would probably have had an influence on their haemoglobin levels. For these reason the significance of the findings of Wilson *et al.* cannot be accurately interpreted.

Divers factors such as method of feeding, infection, and rate of growth play an important part in influencing the haemoglobin level during infancy. The demonstration that transplacental ^{55}Fe is diluted by dietary iron when the infant is 3 to 4 months old (Smith *et al.*, 1955) is clear evidence that after this time dietary iron is a very important source of supply to the infant. Theoretically it might appear preferable to assess the difference in haemoglobin levels at 1 year of age. If such an investigation were to be continued up to this age, the groups of infants would have to receive identical diets, be kept completely free from infection, and demonstrate identical weight gain under strictly controlled conditions. This was beyond the competence of the present investigation, and indeed it is doubtful if such a long-term study could ever be done. For these reasons the present investigation was not continued in these infants beyond three months of age.

Most other investigations on the effects of clamping the cord at different times have not been conducted on strictly comparable groups of mothers and infants. Details of the mother's blood picture, age, and parity, and essential information such as weights of infants, are often not stated in the records. In the present series the two groups of mothers were strictly comparable. No statistical difference was found between the two groups of infants in respect of weight either at birth or at 3 months of age.

Many workers have called attention to the fact that the principal iron reserves of the newborn infant are in the circulating haemoglobin and that the tissue stores are small (Fullerton, 1937; Stearns and McKinley, 1937; Lintzel *et al.*, 1944; Hemmeler, 1946; Neander and Vahlquist, 1949; Langley, 1951; McCance and Widdowson, 1951; Josephs, 1953). DeMarsh *et al.* (1941), Wilson *et al.* (1941), and Sturgeon (1956) have calculated theoretically the extent to which the haemoglobin level is increased by the amount of iron present in the placental transfusion. If it is accepted that only a small amount of blood is transferred after delivery of the infant, prevention of transfer of extraplacental blood to the infant by early clamping means only relatively negligible deprivation of potential iron.

If estimates of 100 to 250 ml. are correct, the extra blood volume gained by infants whose cords are clamped late should be reflected by an extra 3 to 8 oz. (85 to 225 g.) birth weight of these infants compared with infants whose cords are clamped early. In the

present series the mean birth weights of group 2 infants was less than that of group 1.

The mean haemoglobin level in group 2 infants was higher than in group 1 in the immediate neonatal period. At 3 months of age there was no difference statistically between the mean haemoglobin levels of the two groups of infants. It is possible that if this investigation was duplicated in non-white infants, who have generally lower mean haemoglobin levels than are found in white infants, the placental transfusion might be sufficient to affect the haemoglobin levels at 3 months of age. For administrative reasons it was not possible to do this investigation in non-white infants in Capetown.

It has been noted that although the mean haemoglobin level of the infants in group 2 was higher than those in group 1 from birth, the difference was statistically significant only from 13 hours of age. This is in keeping with the observations by Smith (1951) that differences assignable to the time of clamping of the cord are not demonstrable at the moment of delivery but appear in samples taken from the baby an hour or so later, and become even more striking in those obtained next day.

In normal infants, according to Schiff (1901), several earlier investigators claimed that those whose cords were cut late lost less weight and made a more rapid return to original birth weight than those whose cords were cut early. Duckman *et al.* (1953) stated that the infants in whom delayed ligation of the cord had been practised exhibited more activity and enjoyed better general health than those in a control series. DeMarsh *et al.* (1941) and Colozzi (1954) were of the opinion that the mode of handling of the umbilical cord did not influence the infant's gain in weight. Colozzi stated that no particular improvement in well-being was noted when the cord was clamped late. So many other factors, such as speed of the mothers' lactation and its quantity, influence the infants' general progress and the loss and recovery of birth weight that it is impossible to attribute achievement or failure in these respects solely to one isolated factor such as the time of clamping of the cord.

In infants born prematurely or those delivered after bleeding accidents from obstetrical abnormalities, or after traumatic delivery such as caesarean section, the beneficial effect of extra blood may be especially important. In 50 unselected cases delivered by caesarean section, Siddall *et al.* (1952) showed that the increase over birth weight at the time of discharge was 16.7% in babies whose cords were clamped immediately as compared with 21.6% when the cord was stripped.

Traumatic deliveries from various causes may be associated with severe shock, and it has been claimed that additional placental blood increases the possibility of survival. Landau *et al.* (1950) described infants delivered by caesarean section who at two to four hours post-partum developed symptoms consisting of cyanosis, respiratory distress, dyspnoea, costal retraction, and rapid weak pulse. They were not benefited by incubation, oxygen, caffeine, or similar stimulants, and died 18 to 20 hours after birth. Landau and his co-workers attributed this to haematogenic shock, since the clinical picture was similar to that observed in the older child or adult in shock from blood loss. They disagreed with the opinion of Potter (1943) that death was due to increased intracranial pressure in a clinically identical syndrome which she described. The same clinical picture has been ascribed to "meningeal oedema" (Potter and Rosenbaum, 1943) and to "aspiration pneumonia" (Russ and Strong, 1946; Gellis *et al.*, 1949). From the reports quoted there is a well-defined clinical syndrome in many infants who do not survive delivery by caesarean section. Landau *et al.*, in attributing this clinical picture to haematogenic shock, claimed that it was due to deprivation of a considerable amount of blood as a direct consequence of the early tying of the cord, and they devised a technique whereby about 90 ml. of placental blood flowed into the baby after delivery by caesarean section. In a series of 87 cases, in which the placenta was held above the infant so that the blood drained into it, none of the infants manifested this shock-like state. Those authors did not feel justified in running a control series. The picture is typical of what is to-day recognized as "hyaline membrane disease."

Colozzi (1954) stated that he had seen several infants with asphyxia pallida who were transformed within a few minutes to ruddy, lusty infants by slow methodical cord-stripping. Suction and oxygen were then resorted to secondarily. It is possible that in "traumatic" deliveries and in premature infants the additional placental transfusion occurring with late clamping might be of importance, but the evidence from the literature so far is not conclusive.

In the present series of cases in which the cord was clamped early no infants were reported to have manifested any of the above difficulties. Other explanations for them can be suggested, and on present evidence it seems that the extra amount of blood gained by late clamping is unlikely to be a major factor in their alleviation.

Certain objections have been raised to attempts at transference of the maximum possible amount of blood from placenta to baby. Franklin (1931) suggested that delayed cord-clamping resulted in a greater incidence of jaundice than did early clamping. On the other hand, DeMarsh *et al.* (1941) recorded jaundice in 3 out of 25 infants clamped immediately and in 5 out of 25 infants when the cord was clamped late, and concluded that jaundice was probably a normal phenomenon in most instances. No increased incidence of jaundice in association with late clamping was reported by DeMarsh *et al.* (1948), McCausland *et al.* (1949, 1950), Duckman *et al.* (1953), or Colozzi (1954). Whipple *et al.* (1957) showed that cord-stripping did not give rise to any bilirubinaemia beyond the range of normal for the newborn period. In the present study no difference in this respect was noted in the two groups.

Obstetricians may object that the presence of the baby on the operating-table until placental separation occurs interferes with prompt control of maternal bleeding and suturing of the uterine incision in caesarean section. This could be overcome by rapid stripping of the cord from the unseparated placenta towards the baby.

Theoretical objections have been raised that extra blood in the infant's circulation may have certain dangers. This is likely to occur in cases where there are anomalies of the cardiovascular system, or in cases of asphyxia with circulatory failure, or if there is intracranial haemorrhage and the "placental transfusion" might impose too great a load on the immature cardiovascular system. In cases of maternal-foetal blood incompatibility the transfer of the extra volume of blood to the infant might add, even though only slightly, to its difficulties. It is also possible, if the amount of extra blood were substantial, that stimulation of erythropoiesis in the marrow might be retarded by a "placental transfusion."

As premature babies and all abnormal deliveries were excluded from this investigation no information was obtained about the value of delayed clamping in such cases. In normal infants the conclusion must be that, although a delay in the clamping of the umbilical cord undoubtedly gives a higher level of haemoglobin in the first few days of life, it has no detectable value to the neonate in any other respect. This investigation was continued for the longest post-natal period of observation on record to date in any large controlled series. It did not reveal that delayed clamping results in any demonstrable benefit to the babies' haemoglobin level or weight at the age of 3 months, which is the latest time at which it is practicably possible to assess the effect of intranatal factors.

Summary

The literature on neonatal and placental blood volume and on the effect on the haemoglobin level of the neonate of early and late clamping of the umbilical cord is reviewed.

Haematological investigations on a series of 133 apparently healthy pregnant white women and their infants are described. In 63 cases the umbilical cord was clamped immediately the infant was born, and in 70 cases the cord was clamped only after the cord had been stripped.

The mean haemoglobin levels in the late-clamped infants between 13 and 24 hours and 72 and 96 hours of age were significantly higher statistically than the mean haemoglobin levels in the early-clamped infants.

At 3 months of age the mean haemogloblin level in the early-clamped infants was 11.08 g./100 ml., and in the late-clamped infants 11.09 g./100 ml. Three months is considered to be the latest time at which it is practicably possible to assess the effect of such a factor.

The advantages of late clamping of the umbilical cord may be of importance only in premature infants or in those infants born after traumatic deliveries.

The objections to late clamping of the umbilical cord are discussed. Most workers do not believe that an increased incidence of jaundice occurs. The theoretical implications of extra blood in the infant's circulation in the presence of anomalies of the cardiovascular system, intracranial haemorrhage, asphyxia with circulatory failure, and maternal-foetal blood incompatibility are mentioned.

I am indebted to Professor F. J. Ford, head of the department of child health, University of Capetown, for his constant guidance, advice, and constructive criticism; to Professor J. T. Louw, head of the department of obstetrics and gynaecology, University of Capetown, and Dr. A. W. Falconer, superintendent of the Mowbray Maternity Home, for facilities in the maternity home; to Dr. D. McKenzie for providing me with the facilities of his pathology laboratory; and to Dr. H. Myers for his advice and assistance in the presentation of this work. I acknowledge with gratitude the willing co-operation, unfailing courtesy, and assistance rendered by the matron and nursing staff of the Mowbray Maternity Home. This work was assisted by a Dr. C. L. Herman Research Grant, University of Capetown Staff Research Fund.

REFERENCES

Bakwin, H., and Rivkin, H. (1924). *Amer. J. Dis. Child.*, **27**, 340.
Börner, R. (1928). *Arch. ges. Physiol.*, **220**, 716.
Brines, J. K., Gibson, J. G., and Kunkel, P. (1941). *J. Pediat.*, **18**, 447.
Budin, P. (1875). *Progr. méd. (Paris)*, **3**, 750.
—— (1876). Ibid., **4**, 2
Chuinard, E. G., Osgood, E. E., and Ellis, D. M. (1941). *Amer. J. Dis. Child.*, **62**, 1188.
Colozzi, A. E. (1954). *New Engl. J. Med.*, **250**, 629.
DeMarsh, Q. B., Alt, H. L., and Windle, W. F. (1941). *J. Amer. med. Ass.*, **116**, 2568.
—— —— —— (1948). *Amer. J. Dis. Child.*, **75**, 860.
—— Windle, W. F., and Alt, H. L. (1942). Ibid., **63**, 1123.
Duckman, S., Merk, H., Lehmann, W. X., and Regan, E. (1953). *Amer. J. Obstet. Gynec.*, **66**, 1214.
Engel, G. (1885). *Zbl. Gynäk.*, **9**, 721.
Franklin, M. (1931). *Amer. J. Obstet. Gynec.*, **22**, 913.
Fullerton, H. W. (1937). *Arch. Dis. Childh.*, **12**, 91.
Gairdner, D. (1958). *Recent Advances in Paediatrics*, 2nd ed. Churchill, London.
—— Marks, J., Roscoe, J. D., and Brettell, R. O. (1958). *Arch. Dis. Childh.*, **33**, 489.
Gellis, S. S., White, P., and Pfeffer, W. (1949). *New Engl. J. Med.*, **240**, 533.
Goodall, J. R., Anderson, F. O., Altimas, G. T., and MacPhail, F. L. (1938). *Surg. Gynec. Obstet.*, **66**, 176.
Grodberg, B. C., and Carey, E. L. (1938). *New Engl. J. Med.*, **219**, 471.
Haselhorst, G., and Allmeling, A. (1930). *Z. Geburtsh. Gynäk.*, **98**, 103.
Hemmeler, G. (1946). *L'Anémie infectieuse*. Schwabe, Basle.
Josephs, H. W. (1953). *Medicine (Baltimore)*, **32**, 125.
Landau, D. B., Goodrich, H. B., Francka, W. F., and Burns, F. R. (1950). *J. Pediat.*, **36**, 421.
Langley, F. A. (1951). *Arch. Dis Childh.*, **26**, 64.
Lanzkowsky, P. (1959). "Iron-deficiency Anaemia in Infants and Pre-school Children in Three Racial Groups in Cape Town." M.D. Thesis, University of Capetown.
—— (1960a). *S. Afr. med. J.*, **34**, 469.
—— (1960b). In press.
—— and McKenzie, D. (1959). *S. Afr. med. J.*, **33**, 21.
Lintzel, W., Rechenberger, J., and Shairer, E. (1944). *Z. ges. exp. Med.*, **113**, 591.
Lucas, W. P., and Dearing, B. F. (1921). *Amer. J. Dis. Child.*, **21**, 96.
McCance, R. A., and Widdowson, E. M. (1951). *J. Physiol. (Lond.)*, **112**, 450.
McCausland, A. M., Holmes, F., and Schumann, W. R. (1949). *Calif. Med.*, **71**, 190.
—— —— —— (1950). *West. J. Surg.*, **58**, 591.
Mollison, P. L., Veall, N., and Cutbush, M. (1950). *Arch. Dis. Childh.*, **25**, 242.
Neander, G., and Vahlquist, B. (1949). *Acta physiol. scand.*, **17**, 97
Phillips, B. I. (1941). Quoted by Chuinard *et al.* (1941).
Potter, E. L. (1943). *Illinois med. J.*, **83**, 100.
—— and Rosenbaum, W. (1943). *Amer. J. Obstet. Gynec.*, **45**, 822.
Robinow, M., and Hamilton, W. F. (1940). *Amer. J. Dis. Child.*, **60**, 827.
Russ, J. D., and Strong, R. A. (1946). *Amer. J. Obstet. Gynec.*, **51**, 643.
Schiff, E. (1901). *Jb. Kinderheilk.*, **54**, 172.
Schücking, A. (1877). *Berl. klin. Wschr.*, **14**, 18.
—— (1879). Ibid., **16**, 581.
Scipiades, E. (1903). *Arch. Gynäk.*, **70**, 630.
Siddall, R. S., Crissey, R. R., and Knapp, W. L. (1952). *Amer. J. Obstet. Gynec.*, **63**, 1059.
Smith, C. A. (1951). *The Physiology of the Newborn Infant*, 2nd. ed. Thomas, Springfield, Illinois.
—— Cherry, R. B., Maletskos, C. J., Gibson, J. G., Roby, C. C., Caton, W. L., and Reid, D. E. (1955). *J. clin. Invest.*, **34**, 1391.
Stearns, G., and McKinley, J. B. (1937). *J. Nutr.*, **13**, 143.
Sturgeon, P. (1956). *Pediatrics*, **18**, 267.
Whipple, G. A., Sisson, T. R. C., and Lund, C. J. (1957). *Obstet. and Gynec.*, **10**, 603.
Wilson, E. E., Windle, W. F., and Alt, H. L. (1941). *Amer. J. Dis. Child.*, **62**, 320.
Windle, W. F. (1950). In *Modern Trends in Obstetrics and Gynaecology*, p. 152, edited by K. Bowes. Butterworth, London.
Wintrobe, M. M. (1929). *J. Lab. clin. Med.*, **15**, 287.

A British Standard for rectangular metal boxes for use in high-vacuum steam sterilizers (B.S.3281) has recently been published that specifies two sizes of rectangular metal sterilizing boxes, suitable for holding dressings and surgical rubber gloves and intended for use with high-vacuum steam sterilizers. Requirements are also laid down which will ensure that the container is of robust construction and that there is adequate means of ingress or egress of steam and air. The standard has been published to ensure that the best use is made in the available space in the sterilizing chamber of the sizes specified in B.S.3220, "Horizontal rectangular hospital sterilizers—pressure steam type." Copies of the standard may be obtained from the British Standards Institution, Sales Branch, 2 Park Street, London W.1, price 3s., postage extra, to non-subscribers.