ASPECTS OF THE RESUSCITATION OF THE NEWBORN WITH REFERENCE TO CAESAREAN SECTION

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The number of infants who die during the period 1-12 months of age has been greatly reduced during the last half century, but there has not been as marked a reduction in the number of infants who die during the 1st week of life.[1, 2] Although advances in obstetrical knowledge can be expected to produce a reduction in the number of stillbirths and neonatal deaths, it is possible that an improvement in the treatment of neonatal asphyxia will also make some contribution to the reduction of the neonatal mortality. In presenting this paper our aim is to deal with the problem of the treatment of neonatal asphyxia.

THE ONSET OF RESPIRATION AT BIRTH

At the time of birth the infant heart is a well-developed organ which has been called upon for some time to provide a circulation to the foetus and to a large mass of placental tissue. In contrast, however, the respiratory system is relatively primitive and it is upon the successful initiation of respiratory function at this time that the survival of the foetus is mainly dependent.

The onset of coordinated respiration follows closely on birth in a large majority of normal infants. The mechanism whereby regular respiration is initiated is not fully established but is probably a result of sensory and proprioceptive impulses received by the respiratory centre at birth.[3] If the foetal respiratory centre is depressed in function at birth the onset of respiration is delayed until the chemical effects of occlusion of the cord and mounting anoxia cause the foetus to gasp. If these gasps do not secure enough oxygen to bring the

higher parts of the respiratory centre into action, so that a coordinated respiratory rhythm establishes itself, the gasps fade away and the foetus dies.[4] Thus it has been postulated that there are 2 mechanisms which initiate respiration: a primary mechanism which acts more or less immediately after birth and a secondary reserve mechanism which acts if the foetus is not born in a physiologically alert condition. The immediate onset of respiration may be regarded as normal while the delayed and gasping response may be regarded as abnormal and less desirable, though life-saving, and is associated most frequently with deliveries involving unnatural or difficult circumstances.[3]

THE CASE FOR RESUSCITATION

Whatever the contributing causes of post-partum apnoea in each individual case, it is our experience that the act of supplying oxygen to the lungs by intermittent positive pressure results in the prompt termination of the apnoea and the adoption of rhythmical coordinated respiration. This suggests the possibility that in many deliveries which are followed by apnoea neonatorum there is superimposed on the effects of drugs or toxic products on the foetus the additional effects of an intrapartum anoxia. Drugs or toxaemia may exert their effect in part by their direct action on the foetal central nervous system and in part by reducing the placental circulation below a critical level for adequate oxygen exchange. The induction of anaesthesia is accompanied by a greatly increased flow of blood through skin and skeletal muscle due, probably, to a temporary suppression of vasomotor activity,[5] and the placental circulation may well be reduced at this time. This might explain the fact that some babies with a normal heart rate before the induction of anaesthesia are delivered by Caesarean section with a slow heart rate and cyanosis when there has been no lack of oxygen to the mother during the procedure.

The vast majority of infants, who have a period of postpartum apnoea do, of course, survive and regular breathing is established even after long periods of evident anoxia in some cases. Sufficient has been written about the possibility of anoxia consequent upon prolonged postpartum apnoea, as a possible cause of subsequent mental impairment, to suggest that it is undesirable to allow a baby to continue in a state of anoxia in the postpartum period for more than a few minutes.[6-8]

It is often stressed that the newly-born baby stands anoxia well and this contention is supported by experimental evidence that many newly-born animals can survive for much longer periods than the corresponding adults when subjected to low oxygen tensions. However, this prolonged time is measured in minutes and, although the explanations are not clear, the ability of the newborn to stand anoxia well might just as well be the result of an efficient heart or a lowered body temperature as the result of some anaerobic metabolic process peculiar to the newborn.

Although the foetus *in utero* exists in an environment in which the oxygen tension in its blood is half that of the adult, the amount of oxygen extracted from foetal blood is roughly similar to that occurring in the circulation of an adult at rest (i.e. 5 c.c. of oxygen per 100 c.c. of blood). The relative inactivity of the foetus *in utero* makes a reserve supply of oxygen in its blood unnecessary and indeed this supply is small, but when the newly-born infant is subjected to anoxia resulting from postpartum apnoea, the effects are obvious on the baby almost immediately in the form of a depression of reflexes, activity and alertness. The fact that a newly-born baby can achieve a limited respiratory exchange from the mucous membranes of the upper respiratory and alimentary tracts accounts for many of the long survivals that have been reported which have been accompanied by apnoea.

From a survey of the causes of perinatal mortality, it is apparent that even when unavoidable causes of death are omitted, such as congenital malformations and stillbirths due to haemolytic disease, we are left with a considerable problem of mortality and morbidity.[9] Thus Bound *et al.*[10] reviewing the necropsies of 221 neonatal deaths following 10,044 live births, found that the pulmonary syndrome, birth trauma, pneumonia and intraventricular haemorrhage comprised $\frac{2}{3}$rds of all the causes of death. They found that the clinical picture of these 4 conditions was similar and difficult to differentiate. Delay in the onset of respiration occurred in half the cases and poor condition at birth was common. Of the cases which showed cerebral irritation only 24% had evidence of intracranial trauma, while 63% had evidence in their lungs of periods of abnormal pulmonary ventilation. Of the cases which died from the pulmonary syndrome, which comprised 27% of the total number of deaths, 50% of the babies were in poor condition at birth and from that time showed persistent difficulty with respiration. The aetiology of the pulmonary syndrome of the newborn remains uncertain but it is believed by some that the hyaline membrane may be partly the result of failure to establish efficient respiration.[10, 11] Of the babies who died from this syndrome 43% weighed between 1,000 and 2,000 g. (4 lb. 6 oz.). Premature babies often have difficulty with the onset of respiration and it has been shown that they are not able to oxygenate their blood as well as mature infants under the same environmental conditions.[10] Premature babies weighing below 1,500 g. comprised as much as 60% of deaths occurring during the first day of life.[2]

All degrees of severity of the abovementioned pathological conditions occur and where they exist with less severity, and where the infant is more vigorous at birth, he will have a greater chance of survival. As difficulty with the onset of respiration, and consequently anoxia, often occurs in these conditions, this difficulty provides an excellent empirical reason for treating apnoea neonatorum promptly so as to ensure effectively the maximum vigour and chances of survival for these babies.

CAESAREAN SECTION

The following observations are of particular interest with reference to the resuscitation of the newborn at Caesarean section:

1. Postpartum foetal apnoea following delivery by Caesarean section is common, particularly when a general anaesthetic has been administered to the mother.[12, 13] Comparison of a group of uncomplicated Caesarean sections with a group of normal deliveries (with the use of spinal anaesthesia in both groups), showed that the babies delivered by Caesarean section were in poorer condition and that the onset of respiration was less prompt than in the group delivered through the pelvis.[14]

2. The type of anaesthesia used to deliver babies weighing over 2,000 g. had no significant effect on foetal mortality in a series of 671 Caesarean sections reported by Lund,[15] but babies weighing under 2,000 g. fared better when delivered by conduction anaesthesia than when delivered by general

anaesthesia. This does not mean that general anaesthesia cannot be improved to give equally good results.

3. The foetal mortality was low where no foetal or maternal complications were present (1·8%, excluding stillbirths and malformations), but high when these complications were present (13·4%).[15]

4. 'Hyaline membrane' is significantly commoner in babies delivered by Caesarean section.[10]

Anaesthetic Technique for Caesarean Section

We have used the following technique for a series of Caesarean sections: The mothers were premedicated with atropine and a stomach tube was passed before operation. Before anaesthesia was induced the mothers were allowed to inhale 100% oxygen for a few minutes and this was followed by the intravenous injection of up to 0·25 g. of thiopentone and 60 mg. of succinylcholine. A cuffed endotracheal tube was inserted and the patients were vigorously inflated with nitrous oxide and 40% oxygen. The operation was then started and ether introduced to supplement anaesthesia. The babies were delivered on an average 9 minutes after the induction of anaesthesia.

This type of anaesthesia is widely used for Caesarean section. It has the following advantages:

1. No pre-operative narcotic drugs which may depress the foetus are given to the mother.

2. The use of a stomach tube before induction and the apnoea produced by succinylcholine during induction makes the passing of an endotracheal tube almost free from the danger of the inhalation of vomit.

3. Complete oxygenation of the mother can be assured before and during the anaesthesia.

4. Light ether anaesthesia for a period of 4-5 minutes is unlikely to depress the foetus but does give added analgesia to the thiopentone-relaxant technique. This technique has the disadvantage that thiopentone, ether, and possibly succinylcholine,[16] cross the placental barrier and affect the foetus if used in excessive dosage.

RESULTS

The following results were obtained in 242 consecutive Caesarean sections for which we administered the anaesthetic and in which the foetus was considered alive 1 hour before the operation: Of the 242 cases, 103 (42·6%) sections were performed for foetal or maternal complications exclusive of disproportion. Foetal distress was diagnosed in 60 cases (24·8%), prolonged labour in 27 cases (11·2%) and toxaemia of pregnancy in 26 cases (10·7%). Antepartum haemorrhage occurred in 10 cases, prolapsed cord in 5 cases and a ruptured uterus in 3 cases. The remaining 139 cases were operated on for indications such as previous Caesarean section, disproportion or abnormal lie or presentation. Over 90% of the cases were in labour at the time of operation. There were 31 premature babies of which 5 weighed under 2,000 g. (4 lb. 6 oz.), while the remainder weighed between 2,000 and 2,500 g. (5 lb. 8 oz.). There were no maternal deaths at or after the operation, and no babies were born dead.

Neonatal Deaths

There were 8 neonatal deaths.

Case 1

This baby had a tracheo-oesophageal fistula and hydrocephalus, and died on the 4th day after an operation for correction of the fistula.

Case 2

The mother of this baby was admitted having been in the second stage of labour for 12 hours, and foetal distress was present. The baby died 7 minutes after delivery by Caesarean section without gasping or responding to resuscitation. At necropsy the trachea and bronchi contained meconium and the postmortem appearances were consistent with death due to asphyxia.

Case 3

This baby died 16 minutes after operative delivery for a ruptured uterus without gasping or responding to resuscitation. Necropsy showed large amounts of amniotic debris in the lung alveoli.

Case 4

This baby died on the 2nd day after delivery from a ruptured uterus. He was in poor condition at birth but responded to resuscitation and was breathing regularly 9 minutes after delivery. Necropsy showed large amounts of amniotic debris in the lung alveoli and areas of unexpanded lung.

Case 5

This baby was delivered prematurely in good condition after an antepartum haemorrhage. He weighed 2 lb. 12 oz. and died on the 6th day. Bronchopneumonia was found to be present at postmortem examination.

Cases 6 *and* 7

These babies survived for 45 minutes and 15 hours respectively and at necropsy subdural haemorrhages were evident. The 6th baby never breathed and the 7th one never achieved more than an occasional uncoordinated gasp and life was maintained by artificial respiration.

Case 8

The mother of this baby had eclampsia and had had 1 fit before operation. A baby weighing 5 lb. 2 oz. was delivered in poor condition but after resuscitation was breathing regularly 8 minutes later. The baby died 1 week after birth. The lungs showed patchy atelectasis but no sign of pneumonia. The kidneys contained numerous immature glomeruli.

While such classifications as described by Flagg[17] are useful in assessing the condition of the baby after delivery, the heart rate appeared to us to be the most useful single sign in this respect. The heart rates of 150 babies were recorded immediately after Caesarean section. Of these babies 48 (32%) had a heart rate below 100 per minute, 20 (13·3%) had heart rates ranging from 100–119 per minute and 82 (54·7%) had heart rates above 119 per minute. Two minutes after delivery 44 (91·7%) of the 1st group, 15 (75%) of the 2nd group and only 3 (3·7%) of the 3rd group were not breathing in a co-ordinated and rhythmical manner, and were in fact apnoeic.

All the babies that were not breathing rhythmically 2 minutes after delivery were inflated with oxygen. This procedure was carried out in 76 (31·4%) of the cases. In 242 cases of Caesarean section only 4 babies (cases 2, 3, 6 and 7) did not establish a satisfactory respiratory pattern within 10 minutes of birth. All the other babies had pink mucous membranes and a heart rate above 120 per minute within 4 minutes of delivery and only a few of the 76 cases which were inflated with oxygen took longer than 4 minutes after delivery to establish a satisfactory respiratory pattern.

DISCUSSION

There seems to be some doubt whether unexpanded foetal lungs can be inflated without damage occurring to lung tissue. In 1933 Wilson and Farber[18] showed that a minimum pressure of between 25 and 30 cm. of H_2O was necessary to inflate unexpanded foetal lungs. In 1937 Wilson, Torrey and Johnson[19] submitted the lungs of 3 stillborn foetuses to a pressure of 25 cm. of H_2O for 4 seconds and the lungs of

1 stillborn foetus to this pressure for 20 seconds, and were able to demonstrate damage to the alveoli on histological sections. They concluded that damage occurred to the lungs when they were inflated at the minimum pressure required to expand the alveoli. In 1952 Day *et al.*[20] showed that the degree of expansion of lungs submitted to inflation depended on both the pressure and the time interval during which the expanding force was applied. Thus satisfactory expansion of alveoli could be obtained with comparatively high pressures delivered over periods of 0·1-0·2 seconds without causing alveolar damage or overdistension, except in very immature foetal lungs (e.g. from a foetus weighing under 2 lb.) where overdistension of alveolar sacs occurred.[20]

Working on these principles Goddard has established that patchy aeration occurs in full-term foetal lungs at pressures between 30-40 cm. H_2O delivered for 0·2 second, and that uniform expansion can be achieved at pressures between 50-60 cm. H_2O delivered for 0·2 second without causing damage to lung tissue. A Goddard-Bennett-Lovelace infant resuscitator has been designed and used to resuscitate apnoeic infants using these high pressures without any clinical or X-ray evidence of harm occurring to the lungs.[21] It should be noted that the time interval for the pressure wave, which has been found satisfactory, is $\frac{1}{20}$th of the time interval used by Wilson *et al.*[19] when they were able to show lung damage.

Once unexpanded alveoli have been expanded, a much reduced pressure is subsequently required to inflate them and it is most probable that the cohesion of moist alveolar walls is mainly responsible for the initial high resistance offered to inflation.[3]

We have been using a resuscitator shown in Fig. 1 to inflate apnoeic babies with air or oxygen. It consists of a rubber bulb connected to a face mask of firm construction. The pressure at which the lungs are inflated is measured through an outlet interposed between the bulb and the face mask connected to a clinical mercury manometer when in use. At the opposite end of the bulb is a non-return valve. When oxygen is used it is led in by a T-piece attachment which is plugged in distal to this valve. The open arm of the T-piece ensures that an increased oxygen pressure will not build up in the face mask when the bulb is not being squeezed.

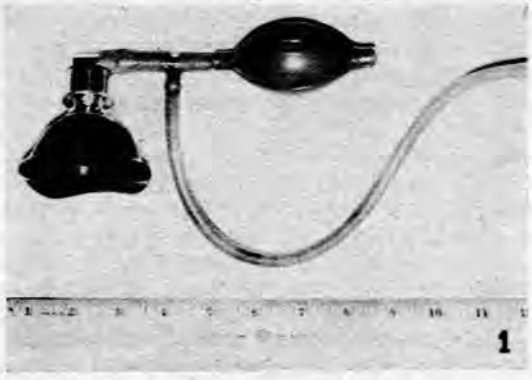

Fig. 1. An infant resuscitator, incorporating a mask, a bulb and a non-return valve. The pressure in the mask is measured with a standard sphygmomanometer connected *via* the tubing which is shown.

The volume of gas delivered by the bulb on each manual compression is 20 c.c. which is roughly equal to the average tidal volume of a full-term infant. It is a safe volume even for premature infants of 2,000 g. because lung compliance allows a much larger volume than this to be introduced without pressure in the bronchial tree building up to dangerous levels.[21] When the bulb is briskly squeezed, the contained gases are delivered in approximately 0·2 second and the maximum pressure registered is about 30 mm. Hg. (40 cm. H_2O) when applied to unexpanded lungs. The pressure achieved depends upon the compliance of the lungs and when some lung expansion has occurred, the delivery of this small fixed volume of gas will result in a smaller rise in pressure, which—as already stated—is what is required.

In addition to the experimental and clinical evidence that inflation pressures of 40 cm. H_2O are safe when applied for a limited time to the lungs of a newborn infant, Smith and Chrisholm[22] have shown by an ingenious device that the average alert newly-born baby is capable of exerting a negative pressure of between 40-50 cm. H_2O during its early inspiratory efforts. It can be deduced from this that there is little danger in applying a similar positive pressure. This deduction should be qualified by stating the fact that occasionally a premature infant produces alveolar rupture and interstitial emphysema by his own respiratory efforts and presumably the same effect could result from an applied positive pressure.[23]

Resuscitative devices which are designed to deliver oxygen at pressures below 25 cm. H_2O are unable to expand unexpanded lungs. Their efficacy in such cases is due to the fact that they bring a flow of oxygen into contact with the bronchi and allow a limited respiratory exchange. If the baby is still able to respond to tactile stimuli, or has in fact already gasped, the application of such devices will stimulate the foetus to breathe or will aerate the already expanded alveoli. Such desirable effects can be achieved by insufflation with an oxygen catheter.

The use of the rocking bed has similar limitations when used in circumstances of total apnoea, but is of value in aiding respiratory and circulatory activity in babies who have started breathing.

When using positive pressure to inflate babies it is dangerous to introduce oxygen into the trachea at unknown pressures for periods much longer than 0·2 second. A rubber balloon will burst if exposed to the initial critical expanding pressure for a long enough period and so will lung alveoli.

When an infant is inflated with a face mask one can observe the bilateral descent of the diaphragm as the lungs expand. *Fig.* 2 is a chest X-ray showing partial expansion of the lungs

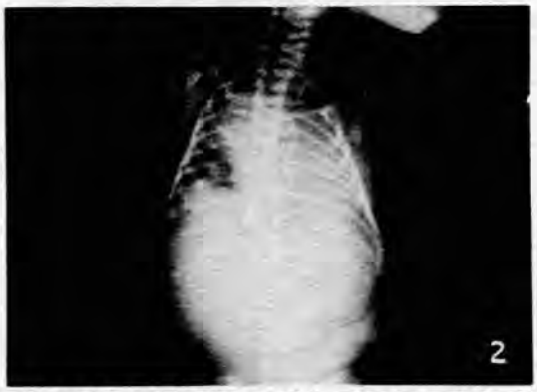

Fig. 2. An X-ray showing partial expansion of the lungs achieved by the application of positive pressure *via* a face mask in a stillborn baby.

in a full-term stillborn infant submitted to positive pressure.

In some cases gas can be seen to enter the stomach by the appearance of a small swelling in the left upper quadrant of the abdomen. This never causes a problem since such resuscitation is confined to a very brief period and the stomach quickly deflates as the gas passes into the upper alimentary tract. The stomach can also be deflated *via* the oesophagus by pressing gently on the stomach, bearing in mind that any regurgitated gastric contents must be removed from the pharynx.

RESUSCITATION BY INTERMITTENT POSITIVE PRESSURE

The resuscitator described is used to terminate apnoea following delivery if apnoea is prolonged for a period considered undesirable by the medical attendant. Only partial expansion of the lungs is achieved, but a rhythmical respiration is initiated.

After delivery the baby is placed on an inclined plane with the head extended and the pharynx is cleared of amniotic debris with the aid of a laryngoscope and a sterile rubber catheter connected to a suction unit. The mask is firmly placed over the nose and mouth and the rubber bulb is squeezed briskly 6-8 times over a period of the same number of seconds and then removed from the face. A rise in the heart rate from the slow anoxic level of 50-70 per minute to 140-180 per minute can be expected within 10 seconds followed by the onset of rhythmical breathing within a similar period. Should the onset of breathing be delayed, as happens occasionally, the heart rate should be kept above 120 per minute by the periodic application of the resuscitator. (Before reapplication of the resuscitator the pharynx should be inspected to exclude any regurgitated gastric contents.) When oxygen is used for inflation, the response in the pulse rate and the onset of breathing is more rapid than when air is used.

If the heart rate does not respond to inflation as stated, a block in the trachea must be excluded, for even babies who have suffered from cerebral injury usually show an initial increase in the heart rate when oxygenated. There have been 5 such cases in this series; 2 (cases 2 and 3) were delivered early in the series. Neither responded to resuscitation and at postmortem examination much semi-solid matter was present in the bronchial tree. No tracheal aspiration was carried out in either case. Subsequently, 3 cases presented a similar clinical picture. At Caesarean section the amniotic fluid was heavily stained with meconium and there was some delay in delivery of the head. These babies showed no response to inflation with oxygen. One baby did gasp but appeared to have respiratory obstruction and all 3 showed signs of progressive anoxia with the disappearance of muscular tone and a progressively slowing heart rate. Tracheal toilet was performed in each case and a large amount of semi-solid material was removed. This was followed by an immediate and gratifying response to resuscitation (positive pressure) and the onset of a satisfactory respiratory pattern.

Thus, of 242 deliveries, we think that 5 babies needed tracheal toilet and that all 5 would have died without it. These deaths might then have been classified as stillbirths because they did not achieve pulmonary ventilation.[24] This definition of a stillbirth might allow preventable neonatal deaths to escape due attention as such.

A satisfactory tracheal suction catheter can be made from a 7-inch-length of polythene tubing with a 1·5 mm. bore. The distal end is rounded off in a flame and the proximal end is fitted over a large-bore needle and connected to a suction unit. The cathether is easier to introduce into the trachea before the suction apparatus is switched on and, although the lumen is small, the walls do not collapse as is often the case with a rubber catheter of this size when it is connected to a suction unit. When in use the lumen of the catheter is cleared of debris by periodically dipping the tip into a bowl of water.

It is generally accepted that the inhalation of amniotic fluid into the foetal lungs occurs *in utero*. It is possible that there is a limit to the volume of amniotic fluid that can be inhaled just before delivery and that a large quantity of inhaled fluid may prejudice the survival of a foetus already handicapped by intrapartum anoxia. After all, drowning is possible at any age. A baby about to be delivered through the pelvis is so compressed by surrounding tissue that its attempts to breathe are limited. The baby delivered by Caesarean section is not so limited and any anoxia, or the tactile stimulation caused by the surgical delivery, might result in increased respiratory activity and the inhalation of large amounts of amniotic fluid at a time when the oxygen supply is cut off with the severing of the cord. While an alert newly-born baby is able to dispose of a greater or lesser amount of inhaled amniotic fluid, it is difficult to believe that a baby will not benefit from the removal of solid matter from the trachea and pharynx and it requires some imagination to believe that such material will flow out of the trachea when the baby is on an inclined plane. We have observed this material lying between the true and false cords of a baby lying at 40° head down without any indication either that the baby or gravity would remove it.

We have had little experience with the use of intragastric oxygen as a means of treating neonatal asphyxia, but we have successfully introduced oxygen into the lungs after other methods had only been sufficient to maintain an asphyxiated baby alive long enough to make permanent damage to the central nervous system a distinct possibility.

Routine gastric aspiration has not been practised on babies following delivery although there is some evidence that such a practice prevents the inhalation of gastric contents and reduces neonatal mortality.[1, 25]

The use of respiratory stimulants has been confined to giving nalorphine to the mother before delivery or to the baby in cases where pethidine or morphia has been given to the mother within 3 hours of the Caesarean section.

To conclude, we would like to stress that the methods of resuscitation described need seldom be applied to babies delivered normally. Mainly as a result of the use of analgesic drugs during labour, neonatal apnoea, following normal delivery, is now sufficiently common to be thought of as normal. Neonatal apnoea is, however, not a normal phenomenon and, although a few minutes of apnoea following normal delivery probably has no significant effect on the baby, the period of apnoea following Caesarean section is often longer. We feel justified in bringing this abnormal period to a speedy end.

We cannot claim that our results are much better or worse than those of other hospitals where other methods of anaesthesia and resuscitation are used because we lack an adequate control series for comparison, and our series comprises a relatively small number of cases. We have, however, set out

our views for those interested in infant resuscitation and have outlined the limits and dangers in the use of positive-pressure inflation of newly-born babies.

SUMMARY

The causes and the consequences of the delayed onset of respiration in newly-born infants, particularly after Caesarean section, are discussed and a case is made out for the early oxygenation of such cases.

A method of anaesthesia for Caesarean section is described.

A simple resuscitator is described and the problems met in the use of positive-pressure inflation of newly-born babies are discussed.

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REFERENCES

1. Little, D. M., Hampton, L. J. and White, M. L. (1952): Anesthesiology, **13**, 518.
2. Bundesen, H. N. (1953): J. Amer. Med. Assoc., **153**, 466.
3. Smith, C. A. (1951): *The Physiology of the Newborn Infant*, 2nd ed., p. 38. Springfield: Charles C. Thomas.
4. Barcroft, J. (1946): J. Roy. Inst. Publ. Hlth., **9**, 86.
5. Churchill-Davidson, H. C. (1957): S. Afr. Med. J., **31**, 1017.
6. Preston, M. I. (1945): J. Pediat., **26**, 353.
7. Penfield, W. (1954): Curr. Res. Anesth., **33**, 145.
8. Schreiber, F. (1938): J. Amer. Med. Assoc., **111**, 1263.
9. Annotation (1957): Brit. Med. J., **1**, 216.
10. Bound, J. P., Butler, N. R. and Spector, W. G. (1956): *Ibid.*, **2**, 1191.
11. Taylor, E. S., Scott, W. C. and Govan, C. D. (1951): Amer. J. Obstet. Gynec., **62**, 764.
12. Ozinsky, J. and Harrison, G. G. (1956): Brit. Med. J., **1**, 725.
13. Bingham, W. (1957): Anaesthesia, **12**, 435.
14. Agpar, V. (1953): Curr. Res. Anesth., **34**, 1.
15. Lund, P. V. (1955): J. Amer. Med. Assoc., **159**, 1586.
16. Pittinger, C. B. and Morris, L. E. (1955): Curr. Res. Anesth., **34**, 107.
17. Flagg, P. J. (1931): Amer. J. Obstet. Gynec., **21**, 537.
18. Wilson, J. L. and Faber, S. (1933): Amer. J. Dis. Child., **46**, 590.
19. Wilson, R. A., Torrey, M. A. and Johnson, K. S. (1937): Surg. Gynec. Obstet., **65**, 60.
20. Day, R. L., Goodfellow, A. M., Agpar, V. and Beck, G. J. (1952): Pediatrics, **10**, 593.
21. Goddard, R. F. (1955): Curr. Res. Anesth., **34**, 1.
22. Smith, C. A. and Chrisholm, T. C. (1942): J. Pediat., **20**, 338.
23. Rees, J. G. (1958): Brit. Med. Bull., **14**, 38.
24. Resnick, I. (1957): S. Afr. Med. J., **31**, 559.
25. Gellis, S. S., White, P. and Pfeffer, W. (1949): New Engl. J. Med., **240**, 533.