

so in a satisfactory fashion. I feel most strongly that the question of the availability of oxygen at home confinements merits the attention of every one of us.

### FAILURE TO MAINTAIN RESPIRATION

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I would like to start by making three points. The first is that when an infant fails to maintain respiration after he has started breathing the most common causes have their origin before birth, and we must therefore look for failure of respiration in babies who have initially shown difficulty in establishing respiration.

My second point is that respiratory difficulty in a newborn infant is never benign and is always an indication for admission to hospital. One reason for this is that the diagnosis of the cause of respiratory distress in a newborn infant is very difficult and always requires x-ray facilities. I am surprised at how often x-ray facilities are not easily or quickly available in neonatal units. It is thought to be essential to have chest x-rays available to diagnose respiratory trouble in an adult, and yet for the newborn, where the diagnosis is much more difficult, the x-ray machine is often either not there or not easily available. Another reason for the infant's admission to hospital is that the treatment of respiratory difficulty in the newborn today has become too sophisticated to be adequately done outside hospital.

The third point I would like to make is that if a newborn infant is to be transferred to hospital he should always be taken in a portable incubator. If he is taken in an ordinary ambulance, or in a taxi or something of the sort, in the winter, he is likely to reach the hospital with a dangerous level of hypothermia which only adds to the difficulties he already has to suffer.

Before I go on to discuss some of the common causes of respiratory distress I would like to make one further clinical point. This is that the normal newborn baby, particularly if he is premature, will often have tachypnoea for the first hour after birth—60, 70 or even 80 respirations per minute—and this is not necessarily very serious or very significant. But if his respiratory rate is still above 50 in the

second hour of life, he is likely to be developing respiratory distress from some cause or other, and therefore this is a fairly early indication of probable trouble to come.

I now come to the causes of respiratory distress, and the commonest of these is the *respiratory distress syndrome of the newborn*, sometimes called "Hyaline Membrane Disease". This is a condition very largely seen in premature infants but not confined to prematures. There are some well-known factors which predispose to the syndrome, including antepartum haemorrhage, maternal diabetes, delivery by Caesarean section, and of course prematurity itself. The infant who develops the respiratory distress syndrome has almost always had some difficulty in establishing respiration at the beginning, some resuscitation has usually been required, and then after two, three or four hours he develops respiratory distress, shown by marked sternal recession. There is inspiratory recession of the lower end of the sternum and very often also intercostal recession, subcostal recession and recession of the suprasternal notch. This is associated with expiratory grunting loud enough to be heard outside the incubator and very considerable tachypnoea—the respiratory rate may go up to 80 or 90 per minute or even higher. He will be cyanosed without oxygen. To begin with, and in milder cases, the cyanosis can be relieved with 35 per cent oxygen, but in the severe case, or the case that is becoming more severe, you have to keep raising the oxygen concentration and ultimately you may find that you cannot abolish the cyanosis even in 90 per cent oxygen at normal atmospheric pressure. Oedema is often quite marked in these babies but it is not usually present at birth, or at least is very slight at birth. It develops in the next few hours after birth.

The cause of the respiratory distress syndrome is unknown, and therefore, of course, primary treatment directed at the cause is not possible, but by biochemical correction in Glasgow in the last four years or so, we have reduced the mortality in severe cases from about 45 per cent to 12–15 per cent.

The radiological features of the syndrome are characteristic. There is a fine reticular or granular mottling throughout the lung fields, and the bronchi are well outlined by air against the atelectatic lungs. This appearance is not seen in respiratory distress from any other cause.

The biochemical features of the respiratory distress syndrome can be summarized very briefly. One is a lowered arterial oxygen tension ( $pO_2$ ), and in severe cases even the use of 90 per cent oxygen will not raise the arterial  $pO_2$  to normal because there are large right to left shunts going on in these very engorged progressively collapsing lungs.

The second biochemical feature is carbon dioxide retention with respiratory acidosis, and the third is a very low plasma bicarbonate with metabolic acidosis. It is by recognizing these biochemical abnormalities and doing our best to correct them that we have been able to reduce the mortality to some extent.

The next cause of respiratory distress in the newborn in order of frequency is *intra-uterine pneumonia*. This is a good deal more common than we used to think. It is commoner in full-term infants than in prematures. If there has been a rupture of the membranes for longer than 72 hours before delivery one can be almost certain that it will be present. One would suspect it if the mother has pyrexia or other signs of infection during labour or just afterwards, and one would also suspect it if the delivery has been difficult or associated with an undue amount of manipulation. The clinical picture is much the same as in the respiratory distress syndrome. A few hours after the birth the infant develops marked tachypnoea, with grunting and sternal recession. The clinical signs over the lungs are usually very few in these ill babies—perhaps a few crepitations, perhaps none—but the x-ray film often shows quite a different appearance from the respiratory distress syndrome. In intra-uterine pneumonia you get larger, softer and more confluent opacities. I would like to stress that the diagnosis of intra-uterine pneumonia is really made by considering the whole circumstances of the birth and the mother's labour, and if there are reasons to suspect intra-uterine pneumonia, a broad-spectrum antibiotic should be started at birth and one should not wait until the infant is in obvious respiratory distress.

Another type of pneumonia that may be seen in a newborn infant is the pneumonia acquired immediately after birth as a complication of mouth-to-mouth breathing. Outside hospital and where intubation or hyperbaric oxygen is not available, mouth-to-mouth breathing may be life-saving. The only point I would make is that if an infant has been resuscitated in this way a broad-spectrum antibiotic should be started immediately afterwards.

Another cause of respiratory distress in the newborn infant is the *meconium aspiration syndrome*. This is seen in babies of low birth-weight, but unlike those with the respiratory distress syndrome, these babies are of low birth-weight because they are dysmature, and not because they are premature. These infants' birth-weights are lower than the length of gestation would lead you to expect; they have the typical appearance of the low-birth-weight baby—a long, scraggy infant with dry, wrinkled, cracked skin and often meconium staining of the nails, umbilical cord and so forth. There has often been meconium staining of the liquor during labour, and though normal liquor amnii does the bronchial tree no harm at all, meconium seems

to be a very severe irritant. The baby develops respiratory distress within a few hours of birth and the x-rays show a much coarser, streaky mottling, often with areas of emphysema, and in some cases the babies even rupture alveoli and get a spontaneous pneumothorax.

The next cause of respiratory distress is *spontaneous pneumothorax*. We have evidence in hospital that this is a good deal commoner than the number of cases diagnosed would suggest. If the infants are not x-rayed I am sure that cases are missed. Very often spontaneous pneumothorax follows resuscitation by intubation. In one such case there was an enormous tension pneumothorax herniating right across to the other side, pushing the mediastinum over and compressing the opposite lung. It is not surprising that this infant was in severe respiratory difficulty. Intubation is a most effective method of resuscitation but it does carry a risk of spontaneous pneumothorax, particularly if it is done by unskilled people, and to be skilled at intubation you have to be doing it regularly—it is not something that one can do once or twice a year and still be safe as an operator. The condition also occurs without intubation, and not so infrequently as we used to think. The treatment of spontaneous pneumothorax is easy provided it is recognized. A needle is put in, with a tube leading to an under-water seal, and the lung is allowed to expand as the air goes out. Nowadays we do not use a metal needle but an “intra-cath”. This is safer than leaving a needle in the chest which might lacerate the lung as it expanded.

I have been talking about what one might call medical causes of respiratory distress because their treatment is medical. I would now like to say a quick word about four, perhaps one might say, surgical causes of respiratory distress because their treatment is surgical, though this will be successful only if the surgeon gets the baby in good condition, and it is the physician who must make the diagnosis before the surgeon is called. The first of these is *oesophageal atresia*. When the baby with oesophageal atresia is fed he will develop acute respiratory distress with cyanosis. He will recover from this after a little while until the next feed, when he will do the same again, and after a few feeds he will then remain in permanent respiratory distress because he has aspiration pneumonia, and the surgeon's task is then well-nigh impossible. It should always be possible to diagnose oesophageal atresia before the infant has his first feed. The infant with oesophageal atresia always produces an excess of mucus in his mouth and pharynx. The corollary is that if an infant is excessively mucousy you should suspect oesophageal atresia. The diagnosis is made with the greatest ease: all one needs do is to attempt to pass a No. 8 soft-rubber catheter into the stomach. This is very easy to do in the newborn infant but if there is oesophageal atresia the

catheter will stick about 7–11 cm from the gums. If your suspicion is wrong and the catheter passes into the stomach, far from this doing any harm, you can take the opportunity to aspirate the contents of the baby's stomach, which often makes feeding start more smoothly in any case.

Another surgical cause of respiratory distress is *congenital lobar emphysema*, in which one lobe becomes enormously over-distended and ballooned with air. This is due to failure of development of the cartilage of an upper lobe bronchus. It always affects an upper lobe. The infants are perfectly well at birth, with no respiratory difficulty at all, but as they breathe in they fill the affected lobe with air which cannot get out during expiration, and after some hours or days the lobe becomes distended and they develop respiratory distress. At this age, an x-ray film is necessary for diagnosis. The treatment is lobectomy, which is a most successful and simple operation in the newborn, who withstand it very well. Here again, the physician must make the diagnosis before the surgeon can operate successfully.

The next cause of respiratory distress, *congestive cardiac failure*, is not as rare in the newborn period as we used to think. One of the many causes is paroxysmal tachycardia, and if the heart-rate is over 220 a minute that is the diagnosis. This would be a diagnosis not to miss because if you digitalize the baby he will quickly recover and he will have a normal life expectancy thereafter because these hearts are healthy. Another and commoner cause is congenital heart disease. The clue to congestive cardiac failure in the newborn baby is hepatomegaly. They develop a large liver early and usually have either a very fast heart from paroxysmal tachycardia or a cardiac murmur from a congenital cardiac lesion. If they are quickly digitalized they usually respond very well, and in many cases they are then amenable to surgery, whereas without surgery the mortality is nearly 100 per cent.

The last cause of respiratory distress in the newborn which I propose to discuss is *diaphragmatic hernia*. Most often these are on the left side. The gap is in the centre of the diaphragm—the pleuro-peritoneal foramen or foramen of Bochdalek. In these babies you often find half the abdominal contents in the hemithorax. Nevertheless, they can be resuscitated and made to breathe spontaneously without any trouble at all, and they will be all right for a few hours, because an infant can get along perfectly well on one lung. But when the bowel, which is often lying in masses and coils in the chest, becomes filled with air, as will happen in a few hours, then the contents of the chest will expand and the infant will develop acute respiratory difficulty. The important thing about a diaphragmatic hernia is that it is an easy operation, my surgical colleagues assure me, and

these cases do very well provided they are diagnosed. There is one other point I would make about diaphragmatic hernia. If it is diagnosed in a maternity unit, as it often is, and the baby has to be transferred to another hospital where paediatric surgery is available, it is important to transfer the infant with an intratracheal tube in position, accompanied by an anaesthetist giving intermittent positive-pressure respiration, because if you try to relieve the infant's cyanosis and respiratory distress by just giving him oxygen with a mask or funnel, you will merely fill his guts with oxygen and increase his respiratory distress, so that he is likely to die on the way.

## DISCUSSION

**Sir Hector MacLennan** thanked Professor Hutchison in particular for drawing attention to the importance of transferring these babies who show some signs of distress to a paediatric clinic at an early stage, if possible in an incubator, so that the paediatrician can diagnose the trouble and institute appropriate treatment.

**Dr B. Colville (Leeds):** I want to make some dogmatic comments, particularly addressed to Dr Kerr. I have been taught another way of resuscitating babies who fail to breathe. I agree that the first step is to remove what mucus you can by thorough suction, and very often this will start them breathing. If this does not work, the next thing is to use a proper mouthpiece to start mouth-to-mouth artificial respiration, and that will often start them breathing. I suppose it is the same as using oxygen, which you say works because it causes a cold blast. Otherwise I am sure that oxygen does not cause a baby to start breathing any better than air, unless there is something wrong with his lungs preventing them from taking the oxygen in sufficient quantities. If this mouth-to-mouth resuscitation does cause pneumonia, can we have the figures to show how much pneumonia you get without resuscitation, and whether a baby who requires resuscitation is much more likely to get pneumonia anyway? You say that intubation is the accepted procedure, but I know many hospitals where they will not allow intubation and I have myself seen some horrible results from intubation. Except for people who are specialists in intubation, with a straight baby's laryngoscope, I think there is probably no place for it at all. I see that you are getting good results with hyperbaric oxygen—you suggest similar to those with intubation. I suggest that the figures from Bradford, where they do not do either, are probably better still.

**Dr Kerr:** I do not honestly believe that the airway helps very much. I just feel that this is yet another bit of apparatus that you have to keep handy and that perhaps when you need it most you have not got it with