Indirect Assessment of Oxygen Requirements in Newborn Babies by Monitoring Deep Body Temperature

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The general availability of pure O₂ in cylinders gave medicine a powerful therapeutic tool. Until 1952 oxygen was used freely in high concentrations in many premature baby units, in the knowledge that many babies were hypoxic and on the assumption that it would in any case do no harm. The demonstration that high concentrations of inspired O₂ could lead to retrolental fibroplasia in premature babies (Patz, Hoeck, and De La Cruz, 1952) led to more caution, and it became a generally accepted practice to restrict added O₂ so that inspired concentrations never exceeded 40% (Kinsey, 1956). This seemed particularly reasonable in view of the demonstration (Campbell, 1960) that O₂ concentrations of 40% would be able to overcome any diffusion defect which at that time was thought to be the main cause of hypoxia in hyaline membrane disease (Craig, Fenton, and Gitlin, 1958). However, it has subsequently been shown that adherence to this limit may lead to an increased mortality in premature babies with respiratory distress (Avery and Oppenheimer, 1960) and to a higher incidence of neurological abnormality of the type associated with hypoxia (McDonald, 1963). Warley and Gairdner (1962) and Gupta (personal communication) have shown that individual babies with the respiratory distress syndrome may be grossly hypoxic in concentrations of inspired O₂ as high as 40%, and that increasing the O₂ concentration to levels previously considered dangerous may be necessary to achieve anything like a satisfactory Po₂ in the baby's arterial blood. There is now evidence from kittens that it is high O₂ tensions in the blood, rather than high concentrations of ambient O₂, that lead to retrolental fibroplasia (Ashton, 1964). The clinician is therefore in a dilemma: individual babies are liable to retrolental fibroplasia if exposed to high O₂ concentrations, while other babies will

suffer death or neurological damage if high O₂ concentrations are withheld. What is more, both situations may occur at different times in the same baby.

Assessing a baby's O₂ requirements is made very difficult by lack of data on which to base a judgement. The most reliable way of determining O₂ requirements is by direct measurement of Po₂ in arterial blood. This carries with it the disadvantage of having to obtain arterial blood from a sick infant and of needing special facilities and equipment. The clinician has to depend on signs such as the presence or absence of cyanosis, respiratory irregularity, restlessness (rarely present in hypoxic premature babies), and frequency of apnoeic spells. Any or all of these signs may be useful but none can be said to be entirely reliable. For instance, babies who are grossly hypoxic are not always obviously cyanosed. Additional information would undoubtedly be valuable, and we believe that in certain circumstances this is provided by monitoring the rectal temperature, which can then be used as a guide to O₂ requirements (Davis and Tizard, 1961).

Theoretical Background

It has been shown in babies (Oliver and Karlberg, 1963) as in many newborn mammals, e.g. cat (Hill, 1959), rabbit (Adamsons, 1959), monkey (Dawes, Jacobson, Mott, and Shelley, 1960), guinea-pig (Dawes and Mestyán, 1963), that moderate hypoxia impairs the ability to increase heat production in response to a cool environment, resulting in a fall in deep body temperature. Fig. 1 shows a model of the situation in homeothermic newborn animals. In the temperature range known as the neutral range, O₂ consumption and, therefore, heat production is minimal. The environmental temperature below which the oxygen consumption starts to

FIG. 1.—A diagram of the relation between metabolic rate, rectal temperature, and environmental temperature in the newborn mammal.

rise is known as the critical temperature. Below this environmental temperature, O_2 consumption must be increased if the deep body temperature is to be maintained. It follows that if the ability to increase O_2 consumption is impaired by hypoxia the deep body temperature will fall. If the hypoxia is corrected, the O_2 consumption, and later the deep body temperature, will rise. Conversely, the ability to maintain the deep body temperature at environmental temperatures below the neutral range implies that the baby is not seriously hypoxic. This argument is known to apply to a normal newborn baby breathing air and low oxygen mixtures, respectively (Oliver and Karlberg, 1963). It is reasonable to suppose that in babies with inadequate respiratory exchange (respiratory distress, pneumonia, etc.) the argument will also apply as between oxygen-enriched mixtures and air, and that information so obtained might be of use clinically. Given a constant thermal environment the necessary information can be obtained by measuring the deep body temperature or, more certainly, by measuring the O_2 consumption directly (see Tizard, 1964).

Methods

The observations reported here were made on babies in the Neonatal Ward of the Hammersmith Hospital. This ward admits newborn babies needing special care, the majority of whom are premature, but a substantial minority are term babies with a wide variety of clinical conditions. Over the past 18 months, more than 300 oxygen consumption studies have been made on over 100 of these babies in an investigation into temperature control in newborn babies, under various conditions of sickness and health. The babies reported in this paper were studied in the course of this investigation, and all showed clinical or laboratory evidence of hypoxia. In many of them, hypoxaemia was confirmed by measurement of arterial PO_2 . The commonest clinical condition was that of 'hyaline membrane disease' (the respiratory distress syndrome of the newborn). This group of hypoxic babies includes those with birth weights varying between 780 and 3,000 g. and gestations between 28 weeks and 41 weeks.

In a minority of studies a standard ward incubator (Oxygenaire) was used. To prevent undue fluctuations in the environmental temperature, the heat control was checked over the period of observation and adjusted manually where necessary. Rectal temperature was measured by an electrical thermometer (thermistor or thermocouple) inserted at least 10 cm. into the child's colon (Karlberg, 1949) and strapped in place with adhesive tape. O_2 concentrations in the incubator were frequently checked, using a paramagnetic O_2 analyser (Beckman). Throughout the period of observation, nursing procedures were reduced to a minimum to prevent fluctuations in environmental temperature and O_2 concentration.

In the majority of the studies, the baby was placed in a specially constructed incubator in which minute by minute O_2 consumption is measured and recorded. With this incubator, one can select any environmental temperature and any O_2 concentration which then stay constant to within $0.1^\circ C.$ or $0.1\% O_2$, over the period of observation. Heat exchange by radiation is controlled by opaque walls at the temperature of the ambient air. Rectal temperature was monitored as before and the child's clinical condition (colour, respiration, activity) was constantly watched. Where arterial PO_2 was measured, blood was taken from an indwelling umbilical artery catheter into a heparinized syringe, the PO_2 being measured immediately on a Clark's electrode (Beckman macro electrode, sample size 0.2 ml. blood).

Results

On numerous occasions in the course of the wider investigation, reducing the environmental temperature to below the critical temperature produced a negligible or only a small increase in O_2 consumption, with the result that rectal temperature began to fall. On at least 21 occasions, increasing the concentration of O_2 in the atmosphere by 20% or more, without changing other environmental

conditions, caused a substantial increase in the rate of oxygen consumption. The normal baby's metabolic response in these circumstances is not affected by increasing the atmospheric O_2 concentration (Oliver and Karlberg, 1963; Scopes, unpublished data), so that this substantial rise suggested that the babies concerned were hypoxic before the added O_2 was given. In 11 of the babies concerned, the fall in body temperature was prevented or the rate of fall was reduced during the period concerned, usually 10 to 20 minutes. In the other 10 no change in the rate of fall could be seen in this short period, despite the increase in oxygen consumption. The lower environmental temperature chosen was usually between 26 and 29° C., and the increase in oxygen consumption effected by adding O_2 was from 10 to 50%. In babies in whom there was undoubted severe anoxia (see individual cases), the increase was between 36 and 50%. These observations are seen in better perspective when individual cases are described in detail. In some of these cases oxygen consumption together with rectal temperature was being measured, in others the rectal temperature alone was recorded while the baby was in the nursing incubator.

Case 1 (Fig. 2). This coloured baby was born at 41 weeks' gestation weighing 2,980 g. The mother was given pethidine, 100 mg. intramuscularly, one hour before delivery. During labour the foetal heart rate fell to 100 and delivery was assisted by use of the vacuum extractor. At birth the child breathed at once. At 1 minute the child was in good condition with a heart rate of 148 per minute, crying, pink, and with good limb tone. Having been born at 11 p.m. she spent the night in the nursery and appeared quite well, but at 8 a.m. next morning she was found to have a rectal temperature of 31.1° C. There was no distress or cyanosis and apart from her low temperature no abnormality was found on clinical examination.

When the baby was placed in the oxygen consumption incubator she had already been warmed to 33° C. At an environmental temperature of 33° C., breathing air, her rectal temperature was rising steadily (Fig. 2). When the environmental temperature was lowered to 28° C., the expected increase in O_2 consumption did not occur, and the rate of rise of rectal temperature fell. The environmental temperature was then raised first to 33° C. and then to 35° C., and the atmosphere was enriched to 50% oxygen, without causing any rise in oxygen consumption. When the environmental temperature was again reduced to 28° C., there was a substantial increase in O_2 consumption. Replacing the atmosphere with air at 28° C. resulted in a fall in O_2 consumption to near basal level, and a fall in rectal temperature. Enriching the inspired air once more to 50% O_2 again enabled the child to increase her O_2 consumption and to maintain the body temperature. It

FIG. 2.—(Case 1 in text.) Rectal temperature, ambient oxygen concentration, rate of oxygen consumption, and environmental temperature, over a 2½-hour period, in a 2,980 g. baby.

was concluded that the baby, who showed no cyanosis or respiratory difficulty, was hypoxic in air but not in 50% O_2 . Her subsequent progress was uneventful and at 1 day of age she was able to maintain her rectal temperature spontaneously in air at 28° C. It is noteworthy that all these changes could be inferred by observation of the rectal temperature, but more rapid and definite information was obtained by measuring oxygen consumption.

Case 2 (Fig. 3). This baby was born prematurely (34 weeks' gestation) and weighed 2,070 g. At birth she breathed at once and at 1 minute had a heart rate of 120, good limb tone, and was pink in colour. At 2 minutes, however, there was a generalized tonic convulsion followed by apnoea, necessitating resuscitation. Immediately after spontaneous respiration had restarted at 5 minutes there was mild sternal recession. At 4 hours the respiratory rate had risen to 64, and there was mild recession and grunting. No cause was found for the convulsion—the CSF was not blood stained.

At 22 hours, when the baby was placed in the O_2 consumption incubator, she still had mild respiratory distress. At an environmental temperature of 34.5° C.,

FIG. 3.—(Case 2 in text.) The effect on metabolic rate and rectal temperature of variation in incubator temperature and ambient oxygen concentration. Each measurement was made over a 10-minute period. P_{O_2} measured while the child breathed air was 47 mm. Hg.

breathing 41% O₂, the rectal temperature was rising slowly. On changing the environmental temperature to 29° C., while the child was breathing 41% O₂, there was an increase in O₂ consumption (Fig. 3), but at this relatively cool temperature the rectal temperature fell slightly. While breathing air at the same cool temperature, the O₂ consumption rate fell to near basal levels, and the rate of fall of rectal temperature was accelerated. It was concluded that this child was hypoxic in air and in fact the arterial P_{O_2} estimated at the end of the observation, when the child was breathing air, was 47 mm. Hg. She was kept in 40% oxygen for the next 24 hours and subsequently had an uneventful recovery.

Case 3 (Fig. 4). This baby was born after 35 weeks' gestation weighing 1,960 g. There had been a small ante-partum haemorrhage at 27 weeks. At 35 weeks the mother was examined under anaesthesia and the membranes were ruptured. The cord prolapsed and the baby was, therefore, delivered at once by lower segment caesarean section. At 1 minute, the baby was apnoeic, limp, and blue, with a heart rate of 66. His airways were aspirated and his trachea intubated, oxygen being given by intermittent positive pressure. By 1 hour of age there was obvious subcostal recession on inspiration and he had developed severe respiratory distress, which lasted 5 days. Over this period his P_{O_2} was consistently low (see Fig. 4) unless very high concentrations of oxygen were given (70–80%).

At 4 days of age he was tested in the oxygen consumption incubator. At an environmental temperature of 34.9° C., breathing 77% O₂, his basal metabolic rate was measured; at this time the rectal temperature was rising slowly. On reducing the environmental temperature to 28.2° C., while he was breathing 77% O₂, there was a rise in O₂ consumption, though the rectal temperature fell slightly. When tested in 54% O₂ and 42% O₂ for

FIG. 4.—(Case 3 in text.) The effect on metabolic rate and rectal temperature of variation in incubator temperature and ambient oxygen concentration. Each measurement was made over a 10-minute period. P_{O_2} while breathing 70% O₂ was 90 mm. Hg; P_{O_2} while breathing 54% O₂ was 54 mm. Hg; and P_{O_2} while breathing 34% O₂ was 22 mm. Hg.

10-minute periods (Fig. 4), the O₂ consumption rate was reduced in steps to near basal levels, and the rate of fall of rectal temperature was increased. Subsequently, giving him 85% O₂, still at an environmental temperature of 28.2° C., there was a substantial rise in O₂ consumption and the rectal temperature remained stable. Arterial P_{O_2} estimations just before and just after the testing period were 22 mm. Hg in 34% O₂, 54 mm. Hg in 54% O₂, and 90 mm. Hg in 70% O₂. On the sixth day of life he had recovered from his respiratory distress and made good progress.

Case 4 (Fig. 5). This baby was born by breech delivery at 29 weeks' gestation weighing 1,180 g. At 1 minute she was apnoeic, limp, and blue, with a heart rate of 80. She was, therefore, intubated and artificially ventilated by intermittent positive pressure. She developed severe respiratory distress, complicated by repeated apnoeic spells, and needed very high concentrations of environmental oxygen for the first few days of life. At 48 hours an exchange transfusion was performed for jaundice of prematurity. Unlike most babies with respiratory distress, her respiratory difficulties persisted for weeks.

At 48 hours, at the height of her illness, her P_{O_2} in air was only 19 mm. Hg and in 86% O₂ only 54 mm. Hg. She was kept in over 60% O₂ until tested on the eleventh day in the oxygen consumption incubator. She had a satisfactory metabolic response to cooling to 29.7° C. (Fig. 5b) while breathing 62% O₂ and 45% O₂, but in 35% O₂ there was no response. She was maintained at 55% O₂. On the seventeenth day she had a satisfactory metabolic response to cooling to 28.8° C. (Fig. 5c) while breathing 47% O₂ and 37% O₂, but not in 23% O₂. She was now kept in 40% O₂. On the 23rd day (Fig. 5d)

FIG. 5.—(Case 4 in text.) Changes in oxygen requirements over the period of 1 month in a baby with severe and persistent respiratory distress. (a)–(f) stages as stated in the figure.

she had a satisfactory response in 30% O₂ but was cyanosed in air and so was maintained at 30–35% oxygen. At 26 days (Fig. 5e) there seemed to be some response at 23% O₂ but at 31 days (Fig. 5f) she still needed 26% O₂. Her subsequent recovery was gradual. Her eyes have been examined regularly by an ophthalmologist, and at 4½ months of age no evidence of retrolental fibroplasia has been seen.

Case 5 (Fig. 6). This baby was born at home to an unmarried mother who had concealed her pregnancy. Gestation was uncertain but birth weight was 1,640 g. The baby was thought to be dead at birth and was left lying wet and uncovered in a cold room. When he was noted to be gasping he was transferred to the Neonatal Ward, where he was found to have a rectal temperature of 29.5°C. He was a small oedematous immature baby, breathing spontaneously but irregularly, and without any obvious recession or grunting.

In the oxygen consumption incubator at an environmental temperature of 31.8°C. (rectal temperature had by now risen to 31°C.) and breathing 33% O₂, the rectal temperature was rising steadily, but the O₂ consumption rate was very low (3 ml./kg./min.). On replacing with air at the same temperature, the child became slightly 'dusky', the O₂ consumption rate fell, and the rectal

FIG. 6.—(Case 5 in text.) Environmental temperature, rectal temperature, ambient oxygen concentration, and rate of O₂ consumption in the course of rewarming a hypothermic premature baby.

temperature no longer rose. Giving 50% O₂ was associated with a rise in rectal temperature and in rate of O₂ consumption. As the body temperature rose the environmental temperature was increased to 33.2°C. There was at this stage no difference in rate of rise of rectal temperature or of rate of O₂ consumption whether the child was given 50% O₂ or 33% O₂. At 10.15 p.m. the child had a cyanotic episode associated with a fall in O₂ consumption rate and in the rate of rise of rectal temperature. He was subsequently given 50% O₂. It was not considered justifiable to test this baby's O₂ consumption at a lower environmental temperature. He died 4 hours later after an episode of apnoea and was found at necropsy to have a pulmonary haemorrhage—a common finding in cold babies. Again an accurate record of the rectal temperature gave the same information as measurement of O₂ consumption.

Case 6 (Fig. 7). This baby was born prematurely (probably 28 weeks' gestation) weighing 1,020 g. At birth, respiration started spontaneously, but within minutes there was rapid respiration with grunting and recession. He went on to develop severe clinical respiratory distress together with episodes of apnoea.

At 24 hours in the oxygen consumption incubator at an environmental temperature of 32°C., breathing 40% O₂, the child's rectal temperature was gradually falling,

FIG. 7.—(Case 6 in text.) Environmental temperature, ambient oxygen concentration, arterial P_{O_2} , rectal temperature, and rate of O_2 consumption in a 1,020 g. premature baby who subsequently died with an intraventricular haemorrhage.

indicating that this temperature was below the neutral range for this child. When breathing air there was a small fall in oxygen consumption rate, and when breathing 70% O_2 the rate returned very nearly to that obtaining in 40%. However, neither of these changes was reflected in the record of the rectal temperature which continued to fall slowly. Estimations of arterial P_{O_2} while the child breathed the respective O_2 concentrations were carried out during or immediately after the test period and are entered in Fig. 7. Although hypoxia was abolished by giving 70% O_2 (in fact a dangerously high P_{O_2} was achieved), this was not reflected in the rate of oxygen consumption. This child's failure to achieve a metabolic response was clearly not due only to hypoxia. He subsequently died, and at necropsy he was found to have pulmonary atelectasis with hyaline membrane and an intraventricular cerebral haemorrhage.

Case 7 (Fig. 8). This baby was born after 33 weeks' gestation weighing 2,100 g. She developed respiratory distress and was treated with added oxygen. At 24 hours she still had rapid respirations and sternal recession. During a test period, the incubator temperature was maintained at 31.7° C. While she was breathing air her rectal temperature was falling. When the O_2 concentration of the atmosphere was increased to 40–50%, the rectal temperature stabilized. Reducing the O_2 concentration once more to 21% was followed by a fall in rectal temperature. Environmental O_2 concentrations of 50% and 32% were associated with a stable rectal temperature. It was concluded that this child needed at least 32% O_2 in her atmosphere. She subsequently made a good recovery.

FIG. 8.—(Case 7 in text.) Variation of rectal temperature with O_2 concentration of inspired air in a premature baby with respiratory distress. Incubator temperature was maintained at 31.7° C.

Case 8 (Fig. 9). This baby, the smaller of twins, was born at 33 weeks' gestation weighing 920 g. She developed respiratory distress within 2 hours of birth. At 24 hours, while breathing 30% O_2 and with the incubator temperature maintained at 33° C. (as it was throughout the period of observation), her rectal

FIG. 9.—(Case 8 in text.) Variation of rectal temperature with O_2 concentration of inspired air in a premature baby with respiratory distress. Incubator temperature was maintained at 33° C.

temperature was falling. Raising the concentration of oxygen in the inspired air to 40% and then 60% was associated with a stabilization and then a rise in rectal temperature: reducing the O₂ concentration to between 35 and 40% was accompanied by a fall in rectal temperature, which was reversed by once more raising the O₂ concentration to 60%. At 39 hours of age when the child was clinically somewhat better, reducing the O₂ concentration to 30% was no longer accompanied by a fall in rectal temperature which in fact continued to rise.

Case 9. This child was born after 32 weeks' gestation and weighed 1,860 g. at birth. At 1 minute respiration was established and the child was in good condition. At 2 hours of age she had several episodes of apnoea, and then developed increasing dyspnoea. At 4 hours of age her respiratory rate was 80, and there was grunting, subcostal recession, and frequent apnoeic episodes. At this time arterial Po₂, while the child breathed 100% oxygen, was 42 mm. Hg, and Pco₂ was 35 mm. Hg.

When aged 28 hours she was tested in the O₂ consumption incubator. Breathing 91% oxygen at an environmental temperature of 34.6° C. her O₂ consumption rate was 5.75 ml./kg./min. Reducing the ambient temperature to 27.9° C., while she breathed 91% O₂ was associated with an increase in O₂ consumption rate to 7.3 ml./kg./min., and an arterial Po₂ measured at this time was 44 mm. Hg. At the same cool temperature, the ambient oxygen concentration was reduced to 62%. This was associated with a fall in O₂ consumption rate to 5.9 ml./kg./min. and a fall in rectal temperature. Her arterial Po₂ measured simultaneously was 32 mm. Hg.

She was nursed in 95% oxygen and an environmental temperature of 34° C. but died at 48 hours after an apnoeic episode. Necropsy showed pulmonary atelectasis and hyaline membrane.

Case 10. This child was born by breech delivery after 33 weeks' gestation weighing 1,860 g. At 1 minute he was cyanosed, apnoeic, limp, and unresponsive, with a heart rate of 60. He was intubated and given artificial respiration, and spontaneous respiration started by 4 minutes, but at 10 minutes there was already subcostal recession, and he went on to develop obvious grunting and dyspnoea.

At 13 hours he was tested in the O₂ consumption incubator. At an environmental temperature of 35.4° C. his O₂ consumption rate was 4.7 ml./kg./min. At an environmental temperature of 26.6° C., breathing 35% O₂, his metabolic rate rose to 6.9 ml./kg./min. and a simultaneous arterial Po₂ was 64 mm. Hg. At the same cool temperature, breathing air, the metabolic response was reduced to a rate of 6.2 ml./kg./min., at a time when arterial Po₂ was 46 mm. Hg. In this child the metabolic response to cold was impaired but not abolished at a Po₂ of 46 mm. Hg. He subsequently recovered from his respiratory distress.

Discussion

Our data confirm the thesis that hypoxia in a newborn baby impairs or abolishes his ability to achieve a metabolic response to cooling. In some of the babies (e.g. Cases 3, 4, 9, and 10) hypoxaemia was proved by measurement of arterial Po₂; in all the others there was good circumstantial and clinical evidence of hypoxia. When a baby has a poor or absent metabolic response to cooling, and when the response is restored by enriching the atmosphere with oxygen (all other environmental factors remaining constant), it suggests (1) that he was hypoxic before the added oxygen, and (2) that adding oxygen has corrected the hypoxia. A failure of metabolic response, which is not corrected by enriching the oxygen in his atmosphere, may be explained in one of two ways: first, the enrichment achieved may have been insufficient, or the failure may have been due to other factors. Among the other factors that we have found to inhibit a metabolic response to cooling are symptomatic hypoglycaemia, various drugs, cerebral damage, and severe hypothermia. It follows that if an attempt to restore the metabolic response by putting the baby in a high concentration of oxygen fails, it may be dangerous (in terms of Po₂ levels which, when higher than normal, can cause retrolental fibroplasia) to increase the O₂ concentration still further. If, however, adding O₂ restores the metabolic response, and the ambient O₂ concentration is then kept at not more than 20% above the level at which the response is impaired, probably there is little danger of causing retrolental fibroplasia, because even in babies with normal lungs, the inspired O₂ concentration at which there is danger of causing retrolental fibroplasia (40%) is at least 20% above that at which the metabolic response is impaired (<15% O₂).

In newborn small animals the metabolic response is impaired at inspired O₂ concentrations below 15% and is abolished when the inspired O₂ concentration falls below 8% (Adamsons, 1959; Hill, 1959). These percentages would correspond very roughly with arterial Po₂ of 75 and 30 mm. Hg, respectively. Our data, admittedly inadequate (Cases 3, 4, 9, and 10), suggest that in the human baby the metabolic response is seriously impaired at an arterial Po₂ of 45 to 55 mm. Hg and abolished at a Po₂ of about 30 mm. Hg. One would expect cyanosis at this Po₂, but Gupta (personal communication) has repeatedly observed dark blue arterial blood with Po₂ levels of less than 30 mm. Hg in babies who are clinically pink. In fact, dead babies often remain pink if kept in oxygen (Tizard, 1964). More Po₂ data of this sort are being sought. It is obviously

unethical to test the situation artificially in normal babies and one must wait for cases in which disease has produced the test situation.

Even in the absence of a metabolic chamber, useful information may be gained by monitoring the rectal temperature, provided the incubator is equipped to maintain a very stable environment, though some caution and understanding are necessary for the interpretation of the results. Most commercial incubators fail to provide the exacting conditions needed for this test. In particular, babies lose heat by radiation in single-walled incubators, so that an air temperature within the so-called neutral zone does not guarantee a neutral thermal environment. Over the test period the baby must be kept at a constant environmental temperature just below the neutral range which varies in individual babies. In most small premature babies $31-32^{\circ}\text{C}$. is below the neutral range and in most full-term babies $29-30^{\circ}\text{C}$. is below the neutral range (Adams, Fujiwara, Spears, and Hodgman, 1964a, b; Brück, 1961, and personal observations). These environmental temperatures are unacceptably low for nursing some sick babies, so the test period may have to be limited. Monitoring the rectal temperature by repeated insertion of a clinical thermometer is not very satisfactory. The changes sought are small, and factors such as inadequate time for equilibration, and irregular depth of insertion (Karlberg, 1949), may lead to inaccuracies. We prefer a thermistor or thermocouple inserted to about 10 cm. and strapped in place. A device for automatic recording is obviously an advantage. Even with good facilities and equipment one must be careful that large changes in heat lost or gained by evaporation or radiation (Agate and Silverman, 1963) are not affecting temperature change. If, in an environmental temperature below the neutral range, a child's rectal temperature is stable or rising, it suggests he is not seriously anoxic (unless the anoxia is chronic (Brück, Adams, and Brück, 1962).) If a falling rectal temperature is corrected by increasing the ambient O_2 concentration, without other environmental change, it suggests he was previously anoxic and that this has been corrected. If the rectal temperature is falling and that fall is not corrected by increasing ambient oxygen, it may mean that still higher concentrations are needed, or that some factor other than hypoxia is preventing a metabolic response (Case 6, Fig. 7). Caution and thought are needed in deciding the next step in such a case. We have found it safe and convenient to make changes in O_2 concentration in steps of 10-20%.

In many newborn animals heat is produced by non-shivering thermogenesis (Scopes and Tizard,

1963). There is good evidence that in the rabbit a substantial amount of this heat is produced in brown fat (Dawkins and Hull, 1964) and in the human baby there is circumstantial evidence that the same thermogenesis may apply (Dawkins and Scopes, 1965). A 'small for dates' baby with hypothermia, admitted to this hospital recently, had no metabolic response to cold at an age and in a state when he would be expected to have no brown fat (Aherne and Hull, 1964). There was no metabolic response to cooling and no measurable glycerol in his blood. Two weeks later, when he was well covered with fat, he showed a normal metabolic response to cooling, and the cooling was associated on this occasion with a rise in plasma glycerol. Most newborn babies have brown fat in the axilla (Aherne and Hull, 1964), and it is possible that monitoring axillary temperature (near the site of heat production in the brown fat) may be a more sensitive index of metabolic response than monitoring rectal temperature. Observations on these lines are now in progress.

It is clear that monitoring rectal temperature can give valuable information for assessing oxygen requirements in babies. It is especially satisfactory since it depends on the physiological response of the baby rather than on arbitrarily determined 'levels'. The information, if it is to be used clinically, must obviously be assessed in relation to the total clinical situation of the baby including its disease, colour, respiration, and general clinical state.

Conclusion

A baby's need for oxygen enrichment of the inspired air can be assessed by measuring his ability to produce heat. Indirect evidence of this ability can be gained by monitoring the rectal temperature in certain thermal conditions, and this information can be valuable clinically. For a discussion of the limitations of this method and the precautions necessary the reader is referred to the text.

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