RESPIRATORY DISTRESS SYNDROME OF THE NEWBORN: A CRITIQUE OF CURRENT MANAGEMENT OF THE VETILATION-OXYGENATION PROBLEM*

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IN THE MANAGEMENT of the respiratory distress syndrome (R.D.S.) of the newborn, the anaesthetist is usually a somewhat unhappy consultant. As a consultant he must accept certain limitations: an anaesthetist telling the "neonatologist" how to treat this syndrome is rather like an internist, consulted on a difficult cardiac arrhythmia or electrolyte problem in the intensive care unit, responding with a lecture on anaesthesia for open heart surgery or for a pyloromyotomy.

For two good reasons, however, both neonatologist and anaesthetist are missing golden opportunities here. Firstly, the recent literature of anaestheisa[1-4] abounds in works on respiratory physiology relevant to the problem in R.D.S. The emphasis is on new facts about oxygenation of patients during and after anaesthesia, on the effects of artificial ventilation, on problems of venous admixture from shunting, and on the importance of matching ventilation to perfusion in the lungs. By striking coincidence, if it be one, the neonatologist is primarily concerned with difficulties of oxygenation in severe R.D.S. and with venous admixture when shunting is a prominent factor, while the part played by the mismatching of perfusion to ventilation is probably far more significant than has yet been realized.

Secondly, the anaesthetist should know the facts of normal and abnormal lung function and of acid-base balance in the newborn.[5] This will make him not only a better consultant but a better anaesthetist too.

NORMAL RESPIRATORY PHYSIOLOGY IN THE NEWBORN

Avery's book[6] and references are standard reading. Certain selected ideas only will be considered here.

Table I gives an idea of the typical course for arterial blood gases in normal infants during the first 24 hours. The normal infant is born both hypoxic and hypercarbic by adult standards. Within 24 hours of birth, arterial carbon dioxide tension has fallen markedly, pH has risen to about normal adult levels, and oxygen tension has also come up, but only to figures which would be considered low for an adult. Let us consider two aspects only of these findings.

It is curious that arterial carbon dioxide tension is not apparently linked to the control of respiration in the newborn in the same obvious way as in adult. Using the ventilation equivalent (the ratio of physiologic alveolar ventilation

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TABLE I

NORMAL ARTERIAL BLOOD GASES, FIRST 24 HOURS OF LIFE

Age	pH	P_{aCO_2} (mm. Hg)	P_{aO_2} (mm. Hg)
birth	7.26	55	—
+20 minutes	—	47–49	59
1 hour	7.30	40	61
3–4 hours	7.33	40–44	72
24 hours	7.38–7.40	35–36	71

Date abstracted from references 5 and 7.

to oxygen consumption) as an indicator, Prod'hom *et al.*[5] found no evidence of hyperventilation in spite of low arterial carbon dioxide tensions at 24 hours of life. There is a fascinating, but unhelpful, resemblance between the arterial carbon dioxide tension of the newborn and that of the parturient mother at this period.[8] The answer may only be known when a way is found to measure intracellular carbon dioxide tension and total body stores. Meantime, plausible theories to fit the incomplete facts[9,10] are acceptable, so long as they are recognized as open to revision as more facts come to light.

Arterial oxygen tensions are even more interesting. The limits of normal tensions in adults are much wider than was thought five years or so ago; there is a downward slope of these normal limits with advancing years. It is now certain that there is also an upward slope from birth to some undetermined point, probably in early childhood.

Venous admixture of arterial blood from true right-to-left shunting is much higher in the normal 24-hour-old infant than in the adult. Uneven ventilation-perfusion ratios contribute something to venous admixture immediately after birth, but this "virtual" shunt has disappeared by four hours of life, and at 24 hours only a true right-to-left shunt remains, about 20 to 25 per cent of cardiac output. Up to 5 per cent might be considered normal in the adult.

Where is this increased shunting taking place? The contribution of bronchial and Thesbian veins may be greater than in the adult, but there is no evidence for this. The evidence is against the persistence of a patent foramen ovale or ductus arteriosus at 24 hours of life.[11] The possibility of direct pulmonary arteriovenous communications exists,[12] but these have not yet been demonstrated to be very much larger in the newborn lung. Perfusion of completely unventilated alevoli remains the most acceptable explanation.[13]

It is interesting to compare and contrast the lung of the newborn with that of the adult. The most striking difference is in the greater alveolar-arterial oxygen gradient in the newborn; this factor becomes much more serious in severe R.D.S. Apart from this, there are certain similarities, and the normal newborn's lung is perhaps not nearly as inefficient as we may once have thought. The work of Prod'hom *et al.*[5] was unfortunately done on infants of diabetic mothers, delivered by caesarian section, but has been largely confirmed for normal infants of non-diabetic mothers. The ratio of tidal volume to functional residual capacity is about 0.20 compared with 0.21 in the adult, and the ratio of alveolar ventilation to functional residual capacity is around 0.14 in both cases. On a body-weight

basis, functional residual capacity is somewhat lower, being about 24 ml./kg. body weight in the 24-hour-old infant compared with 30–40 ml./kg. in the adult.

Two observations are not in line with what has been commonly believed by anaesthetists. The figures for tidal volume and anatomical dead space at 24 hours of life, viz. approximately 14 ml. and 3 ml. respectively, are lower than we had generally thought: certainly figures closer to 20 ml. for tidal volume and 7 ml. (or 1 ml. per lb. body weight) for dead space have now been commonly accepted.[14] Secondly, Prod'hom *et al.*[5] have pointed out that frequency of respiration may reach 60/min. even in normal infants at this age.

Normal Blood Gases in the First Week of Life

A number of difficulties have to be overcome. These are (1) technical problems of arterial sampling in the newborn and the necessity to do this without upsetting the infant, if values are to have true meaning; (2) the question of validity of the usual nomograms, such as the Siggaard-Andersen, for blood containing foetal haemoglobin: this is a serious and unsettled point. Prod'hom, Levison, *et al.*[5] considered it unsafe to use adult diagrams, and instead employed a modified Davenport diagram with some assumptions of their own. We have used the ordinary Siggaard-Andersen nomogram.

The technique of getting arterial blood from the newborn without making him cry or hyperventilate is not easy, and required in fact several weeks exploratory work on our part. Since these were normal infants the use of umbilical catheterization seemed unjustified. The femoral artery has a totally undeserved reputation as the vessel of choice: we had very little success with it. The infant maintains a persistent attitude of flexion while at rest; it is difficult to palpate the femoral artery, and the vein is frequently punctured by mistake. Reports of vascular complications of the lower limb made us unenthusiastic. As far as we are concerned, the superficial temporal artery has also enjoyed a false popularity, being both unreliable and quite upsetting to the infant. We were also disappointed in the brachial artery in the arm and at the elbow.

Using the radial artery, we eventually evolved a technique which met our requirements fairly consistently. All punctures were done by the same doctor and nurse. The infants were removed briefly from their cribs but were kept wrapped in their blankets; the puncture was done with an absolute minimum of handling and disturbance (Fig. 1). By continuous practice the process was reduced to the greatest possible simplicity. The baby was kept warm and comfortable and was frequently given a glucose bottle. If the first attempt was unsuccessful the infant often became restless or cried if further efforts were made, and the procedure was then abandoned immediately. Time, patience, and perseverance are essential.

It is important to use a 2 ml. syringe and a number 25 needle: entry is made at approximately the same angle as for venepuncture, never at a right angle, and it is often necessary to aspirate gently. A similar technique has recently been described by others,[15] but we did not use special needles, nor did we use the flexor carpi radials as a landmark. The ulnar artery must be palpated before the radial is assaulted. There is no "feel" to an artery, as opposed to a vein, but

there are characteristic sensations of resistance when one is not in the proper place, e.g. in a tendon or impinging on bone.

The importance of having the baby in a quiet state is clearly illustrated in Figure 2. In the early exploratory part of our work, samples were accepted as probably representative, even when some minimal disturbance had occurred. We were forced to this approach by the belief that it was probably impossible to do any better as far as arterial sampling at this period of life is concerned. Many weeks' further persistence led us to achieve successful puncture of the radial artery without the slightest upset to the infant, and the results of some thirty such analyses were used for what we believe are reliable figures. The differences between these sets of results are quite marked, particularly as far as oxygen tensions are concerned.

These were all normal, full-term infants with high Apgar ratings (7–10) at birth. Blood was drawn anaerobically, and the sealed syringes were placed in ice and analysed within 30 minutes. The arterial pH and P_{CO_2} were determined by the method of Astrup, and the standard bicarbonate derived from the nomogram of Astrup. A Clark oxygen electrode (Beckman Instruments) was used to measure the arterial oxygen tensions.

Some details of these infants are shown in Table II; the distribution of age in hours and of arterial oxygen tensions are shown in Figures 3 and 4; while the final figures for these infants are shown in Table III.

TABLE II

DATA ON 31 NORMAL INFANTS

Weights (kg.)		Ages (hours)		Apgar score (number of infants)			
range	mean	range	mean	7	8	9	10
2.4–4.5	3.3	36–108	72	1	5	11	14

TABLE III

BLOOD GASES: INFANTS AND OLDER CHILDREN COMPARED

Total number	Mean age	Range	pH (units)	P_{CO_2} (mm. Hg)	P_{O_2} (mm. Hg)	Standard bicarbonate (mEq./L.)
13	12 yrs.	9–16 yrs.	7.40 ±0.05	34.0 ±6.0	93.5 ±8.2	22.2 ±0.6
31	72 hrs.	36–108 hrs.	7.40 ±0.03	38.8 ±4.0	82.8 ±12.5	23.5 ±1.7
Significance				$p < 0.3$	$p < 0.3$	$p < 0.05$

Note absence of statistical difference in arterial oxygen tensions between 3-day-old infants and 12-year-old children.

It is interesting to compare these results with those found in older children[16] whose average age was 12 years (Table III). These older children were free from pulmonary and cardiac disease, and were sampled at rest and without upset, in bed before breakfast.

Perhaps surprisingly, there is no significant statistical difference between

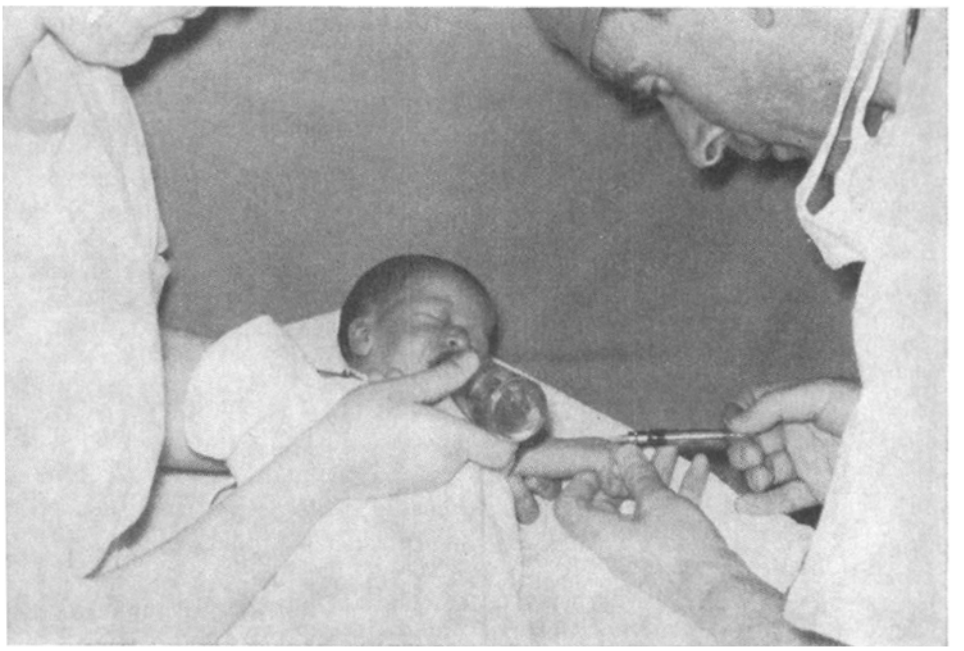

FIGURE 1. Radial artery puncture in the newborn.

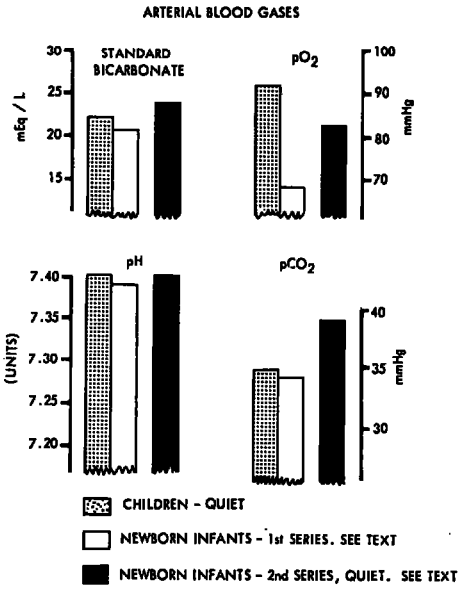

FIGURE 2. Differences between blood gases in normal infants and those in older children. Infants in the first series were sampled with some difficulty and were upset; those in the second series were sampled in truly basal circumstances. Note the striking differences in P_{aO_2} and P_{aCO_2} values.

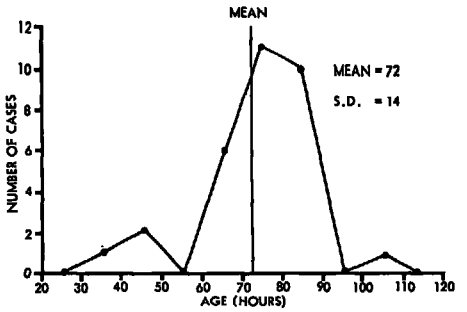

FIGURE 3. Distribution of ages (in hours) of 31 normal newborn infants used for arterial sampling.

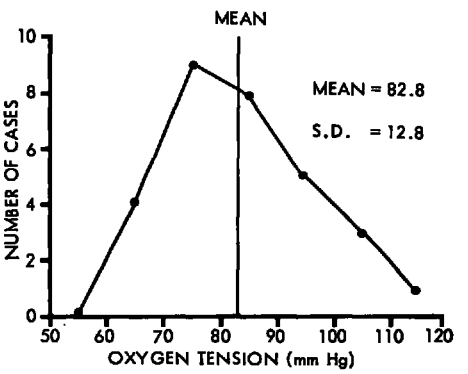

FIGURE 4. Distribution of arterial oxygen tension values found in 31 normal newborn infants.

oxygen tensions in the normal three-day-old infant and in the older child. Possibly the level in our sample of children around 12 years of life is close to the peak in life, but many more data are needed. How, for example, does the line of oxygen tensions slope upward in early infancy and childhood—smoothly, or with one or more characteristic steps; Our data suggest that the slope may be a steep one in the first few days after birth. Certainly the first months of life need further exploration.

A clue might be taken from the patterns of arterial oxygen tension during anaesthesia in normal infants in the first months of life (Fig. 5).[17] There is a considerable scatter in the findings when breathing is spontaneous, but a distinct upward trend when breathing is controlled, the inspired oxygen percentage being 50 per cent in all cases. Allowing that it may be dangerous to assume that the scatter in the first case could be due to the presence of a variable amount of atelectasis, and that this variable factor can be rendered insignificant by controlled respiration, we could advance the working hypothesis that this upward trend indicates diminishing right-to-left shunting. Direct measurements in normal infants are needed here.

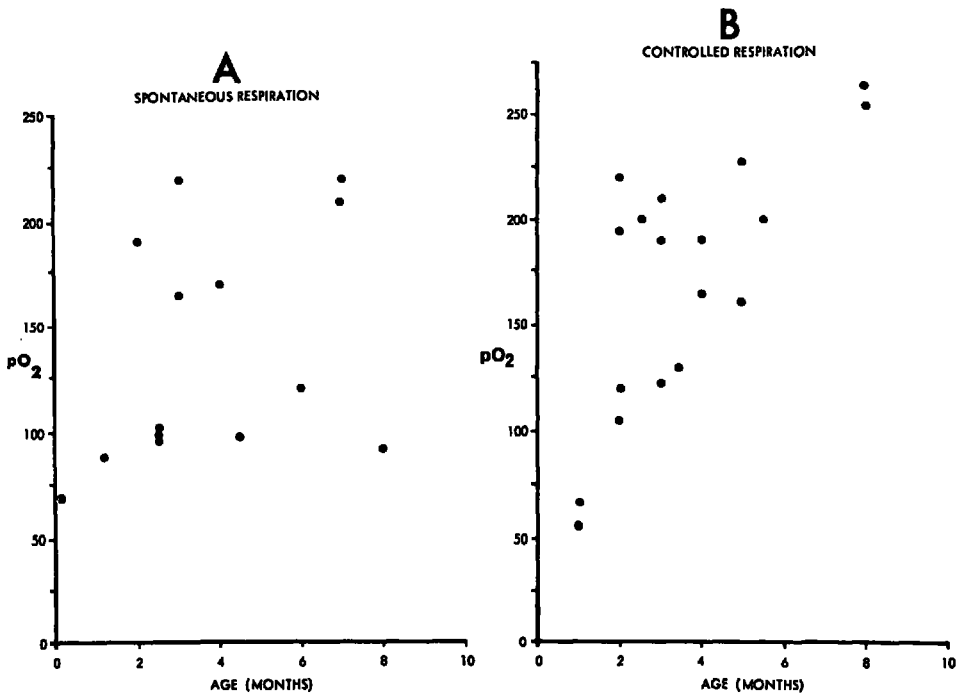

FIGURE 5. Arterial oxygen tension values in the first few months of life in normal infants under general anaesthesia (inspired oxygen 50%), with (A) spontaneous and (B) controlled respiration. Note the tendency toward increasing arterial oxygen tensions when respiration was controlled. This tendency was not seen with spontaneous respiration.

It was also surprising to find that arterial carbon dioxide tensions in the three-day-old infant were higher, though not significantly so, than those in the resting 12-year-old. Our findings were higher than those of Stahlman,[9] who reported that levels of 35 mm. Hg were only achieved by the eighteenth day of life, thus raising the possibility of hyperventilation in her series.

In summary, then, with the exception of slightly low oxygen tensions, the values for blood gases in our three-day-old infants could well have passed for those in adults.

PATHOPHYSIOLOGY OF R.D.S.

Respiratory Distress Syndrome is not clinically difficult to diagnose. Characteristically, infants with the syndrome are premature, have low Apgar scores at birth, and develop signs of respiratory distress within a few hours. Properly speaking, we should adhere to a definition of this syndrome which would exclude specific causes of respiratory distress such as aspiration pneumonitis, but this is not always easy. Rapid, laboured, grunting respiration; rib-retraction; cyanosis even in oxygen, with deepening respiratory and metabolic acidosis do not sound unfamiliar to any anaesthetist who runs an intensive care unit; but the

R.D.S. of the newborn differs in the mystery of its causation, in the fact that spontaneous cure occurs with minimal treatment in some patients, and in that attempts to manage the fully developed condition in severe cases are generally unsatisfactory. In addition, a peculiar alveolar lining membrane is often found at autopsy, which has resulted in the name "hyaline membrane disease." Some idea of the complexity of the factors at work in these cases may be obtained from Figure 6, modified from Swyer and Levison.[19]

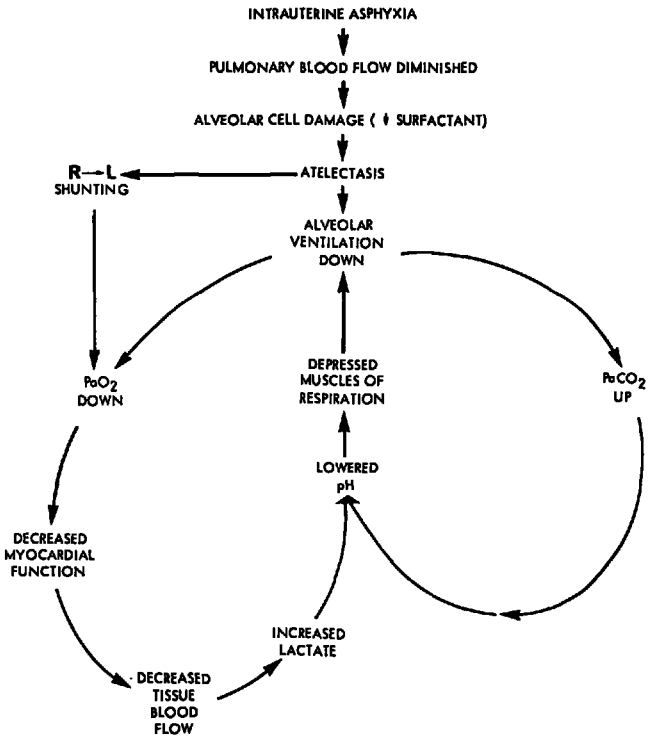

FIGURE 6. Factors at work in R.D.S., modified from Swyer and Levison.[19]

It would be useful to be able to classify degrees of severity of the condition, so that various methods of management could then be assessed against expected mortality. This has been done[20] on the basis of the degree of true right-to-left shunting present, and its change with passing hours. Three different types are thus described, which are clinically quite indistinguishable. There is the possibility of error from certain assumptions which have to be made in calculating these shunts, but it is the direction of change rather than the absolute value which counts. Tedious calculations can be avoided and a good idea of severity and prognosis can be got from serial measurements of oxygen tensions while the patient is breathing 100 per cent oxygen,[21] as shown in Figure 7.

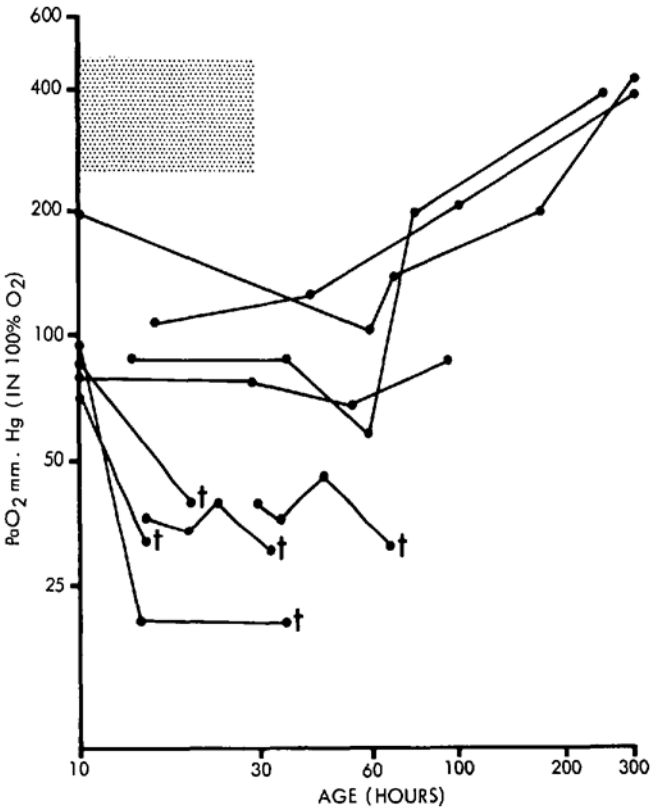

FIGURE 7. Serial measurement of arterial oxygen tensions in infants with R.D.S. gives a good indication of prognosis.

CRITICISMS BY ANAESTHETISTS OF THE USUAL MANAGEMENT OF R.D.S.

The difficulty of the problem and the many possible lines of treatment are obvious from Table IV; they should be enough to discourage a busy department

TABLE IV
MANAGEMENT OF R.D.S.
Modified from Swyer and Levison[19]

Prevention	Ventilatory management	Metabolic management
↓	↓	
Surfactant Replacement	Assisted ventilation ? Thoracic fixation	1. Buffers
↓		2. Extracorporeal circulation
Atelectasis	? Pharmacological stimulation	↓
↓	↓	
Increased R. to L. shunting	CO_2 retention → Hypoxia	→ Respiratory acidosis → Metabolic acidosis
	↑	
	Hyperbaric oxygenation	

of anaesthesia from getting too deeply involved. But even as a consultant, the anaesthetist will soon find three main criticisms to offer:

1. The criteria for the use of artificial ventilation generally horrify anaesthetists, particularly the degree of hypoxia and hypercarbia (Table V). So much of the practice of anaesthesia is to prevent complications or to arrest them as early as possible that we ask "Why wait so long?"

TABLE V

INDICATIONS FOR ASSISTED VENTILATION ADVOCATED BY DELIVORIA-PAPADOPOULOS, LEVISON AND SWYER[23]

Clinical signs			Blood gases	
respiratory rate	heart rate	colour	P_{aCO_2}	P_{aO_2}
< 30/min or > 120/min	< 80/min or >160/min	Cyanosed in 100% O_2	> 80 mm. Hg	< 40 mm. Hg while breathing 100% O_2

An explanation, if not a complete justification, is to be found in the complications which have occurred with artificial ventilation (Table VI), again reported from an excellent centre.[22] These are almost all of a mechanical or infective nature; it is interesting to observe how frequently the tube was inserted into the right main bronchus. One could respect the paediatricians' fear, in such circumstances, that the cure could be worse than the disease, particularly since the condition is self-limiting anyway in some cases. The use of the classification by degree of right-to-left shunt would help in making a decision to begin artificial ventilation sooner.[20]

2. The second main criticism of current practice is that not only are ventilators apparently reserved for moribund patients, but they are badly selected and used poorly even then. The emphasis on assisted rather than controlled respiration is strange to the anaesthetist's way of thinking. With the above criteria surely it would seem essential to rest the exhausted baby by doing all his breathing for him. Most ventilators are incapable of fulfilling the requirements here. The need for high pressures, constant flow, rapid cycling, and small, adjustable tidal volumes makes the Engstrom about the only machine which approaches the ideal. One finds respirators which have the advantage of smaller size and lesser complexity, such as the Bird or Bennett, being used far below their capabilities. It is often disheartening to intubate a patient and come by the ward some time later when adjustments have been made by someone in attendance. Frequently the respirator is making reassuring sounds, but a moment's inspection and ausculation of the chest will show that it is not in phase, that pressures are too low, and that the only guide is apparently the last figures for blood gases, already obviously out of date. There is often an inordinate fear of lowering arterial carbon dioxide tension.

Experience with the use of ventilators in neonatal anaesthesia has shown that cycling pressures of 40 cm. H_2O are by no means excessive. Such pressures are enormously reduced by the resistance of an endotracheal tube whose internal diameter is often around 3.0 mm.[17]

TABLE VI

MEDICAL AND MECHANICAL COMPLICATIONS IN 20 INFANTS HAVING RESPIRATOR CARE[22]

Complications in 13 fatal cases				Complications in 7 survivors			
mechanical	no. of infants	medical	no. of infants	mechanical	no. of infants	medical	no. of infants
Tube in right main bronchus	12	Klebsiella pneumonia	7	Tube in right main bronchus	6	Klebsiella pneumonia	4
Tube in left main bronchus	3	Erythroblastosis foetalis	2	Tube in left main bronchus	1	Unknown pneumonia	1
Subcutaneous tube displacement	4	Intracerebral haemorrhage	3	Obstructive secretions	7	Pneumothorax	1
Leak past tracheostomy tube	6	Haemorrhagic disease of newborn	1	Subcutaneous tube displacement	3	Anaemia	5
Obstructive secretions	13	Pneumothorax	3	Leak past tracheostomy tube	7	Hyperbilirubinaemia	1

In neonatal anaesthesia one finds a wide spectrum of arterial blood gases with spontaneous and controlled respiration, and the range of acceptable carbon dioxide tensions may be as wide as 20–60 mm. Hg (Fig. 8). Similarly, in the management of R.D.S., it is often easy to lower arterial carbon dioxide tensions, but the main problem is rather to raise oxygen tensions and improve tissue oxygenation, and this is often difficult.

Retrolental Fibroplasia

The fear of causing retrolental fibroplasia lingers on in some uninformed minds. Prematurity and prolonged exposure to high inspired oxygen concentrations are only two of the necessary predisposing factors. Actual pathology depends on the oxygen tension reached in the infant's arterial blood, and with severe R.D.S. this is always less than 100 mm. Hg, even when the infant is ventilated with 100 per cent oxygen. It is believed[21,23] that levels of at least 150 mm. Hg are necessary to cause retrolental fibroplasia.

3. The third main criticism is a general one, aimed at the over-all approach. If prevention is the unattainable ideal, surely the next best thing is to treat early, to limit or minimize complications. Efforts to treat fully developed respiratory

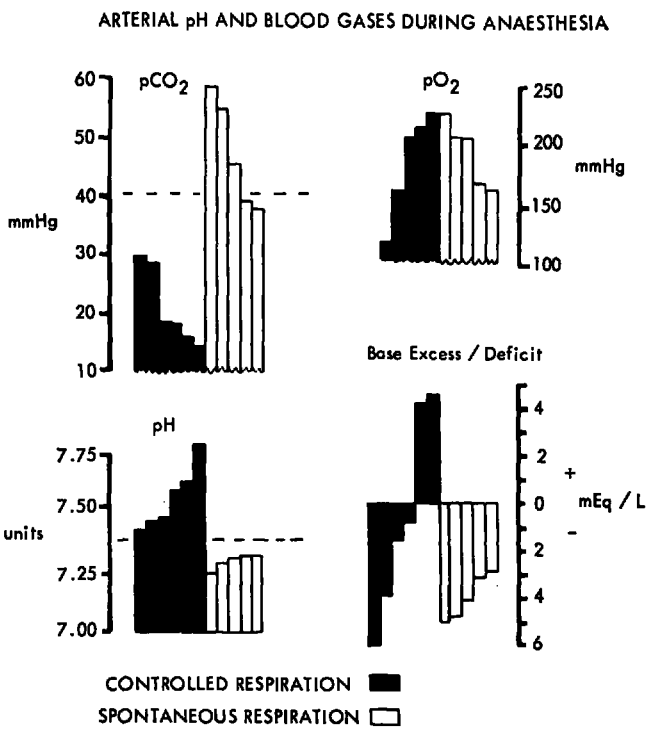

Figure 8. Profile of blood gases found in normal newborn infants during uneventful anaesthesia with spontaneous and controlled respiration.[17]

and metabolic acidosis can never give satisfactory results. The hope has been that each new treatment, bicarbonate or THAM, hyperbaric oxygenation, ventilators, surfactant, would reduce mortality; and in some cases one or a combination of these has helped. But it is immediately more appealing to look for a unified concept of the condition which might eventually lead to one fairly simple line of treatment. Along these lines, certain workers[24] would rename R.D.S. the "pulmonary hypoperfusion syndrome," on the grounds that the precipitating cause of the whole picture is a diminished blood flow in the pulmonary vasculature. Impressive though the theoretical and experimental evidence may be for this, so far the use of drugs such as acetyl-choline or priscoline have not been reported as instant successes. Oxygen, of course, is an excellent dilator of the pulmonary vessels.

It is interesting to speculate that R.D.S. may be only one manifestation of an immature vascular system, which would also involve the microcirculation. The infant in severe R.D.S. soon develops clear signs of poor peripheral tissue oxygenation. Some of this is the effect of pulmonary failure but some of it may be primarily peripheral. Work describing a common peripheral vascular picture in animals "shocked" from a wide variety of causes[25] makes one wonder if treatment should also be directed to the peripheral mechanism, perhaps with massive doses of hydrocortisone.

Use of Negative Pressure vs. Positive Pressure Ventilators

At first sight, to an anaesthetist, the use of a tank type respirator (Fig. 9) seems a retrograde step, but there are reports which suggest certain inherent advantages. One suspects that its acceptance may be due in some measure to the ease with which it can be used; but a patent airway is still essential, there are problems in fitting the cuff around the neck or shoulders, and complications such as pneumothorax or respirator malfunction can occur.[21]

Is this type of respiratory care intrinsically better than positive pressure in R.D.S.? One helpful hint is that better arterial oxygen tensions are achieved even when ventilation, as shown by arterial carbon dioxide tensions, is not as good as that which could be obtained with positive pressure. One immediately thinks of the effect of "ventilation-perfusion scatter"[1] which is important during and after anaesthesia and may also be very important in severe R.D.S. It is possible that there is better matching of ventilation to perfusion with negative pressure ventilation. The point will only be settled by controlled studies which are extremely tedious to do here, or by using the same patient as his own control for both types of respirator, which might give a quicker answer.

Monitoring

The use of continuous E.K.G. monitoring is favoured in some centres (Fig. 9). An auditory signal of bradycardia is of key importance to the nursing staff.[23] Specially designed miniature transitorized monitors have greatly appealed to us here (Fig. 10).

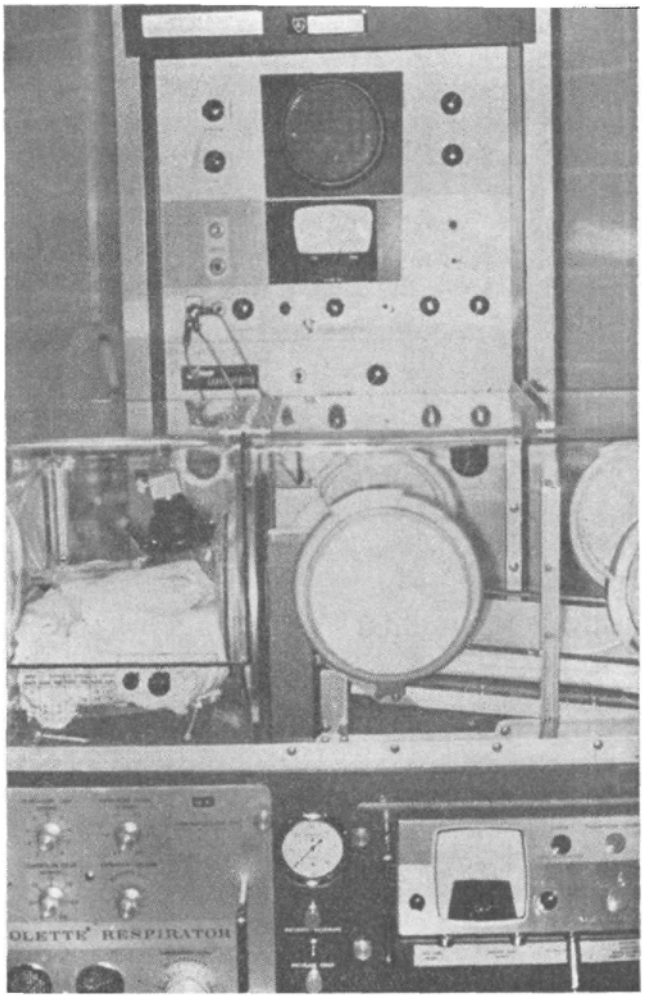

FIGURE 9. Negative pressure respirator with E.K.G. monitor.

IN DEFENCE OF PRESENT PRACTICES

The essence of success here is teamwork: the best results come from the best organized centres. A full-time director, a neonatologist, is essential, but often the techniques which he may emphasize are tried with much less success in other centres, where nursing organization is poorer.

Unless the anaesthetist has the time, background knowledge, and supporting staff, his role in this milieu had better be that of a consultant. However, a very fruitful exchange of ideas is possible.

The management of the patient must certainly be controlled by one physician, and the anaesthetist's problem is to explain what he believes are the best things to do as far as oxygenation and respirator care are concerned, in an atmosphere where such matters are viewed as desperate measures. Apropos of this, premature

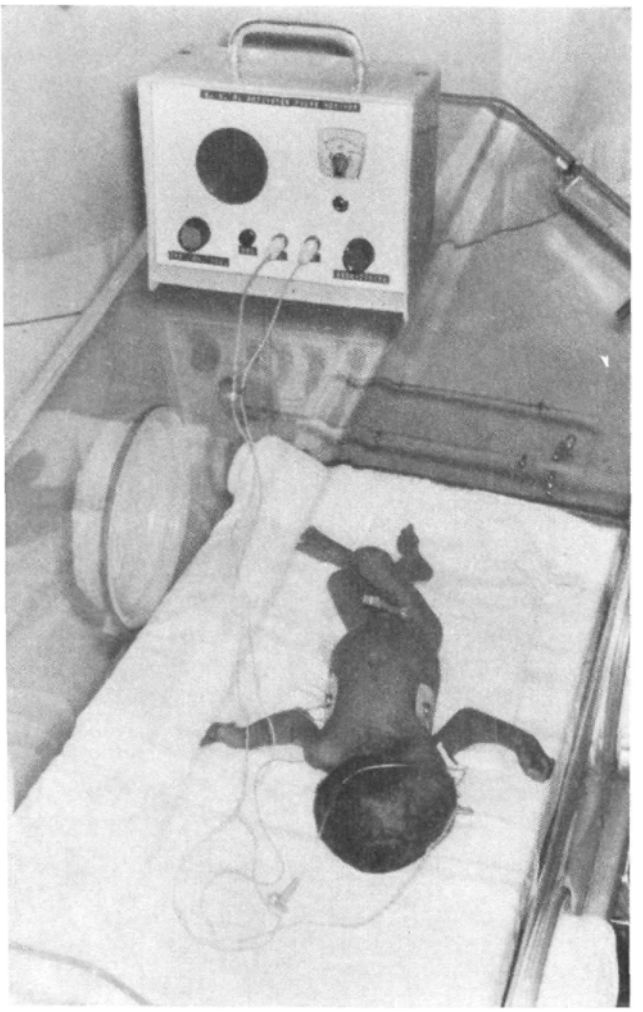

FIGURE 10. Miniature transistorized E.K.G. activated heart rate monitor, attached with special electrodes and leads to a 1 lb. 11 oz. (760 gm.) premature. (Hargrave Research Corporation, Winnipeg.)

wards are excellent for intensive nursing care, but these nurses are often inadequately trained to look after severe respiratory problems. It is strange to see an intensive care unit run by the department of anaesthesia and dealing primarily with respiratory problems and a premature nursery in the same hospital staffed by nurses whose orientation is usually to intensive medical and nursing care, with little or no intercommunication between the two.

SUMMARY

The anaesthetist is usually a consultant in the management of the respiratory distress syndrome of the newborn (R.D.S.). Although he may not tell the

paediatrician how to manage the whole condition, some criticisms of present practices in respiratory care are in order.

Anaesthetic literature is currently dealing in detail with problems relating to alveolar-arterial oxygen differences, and paediatrician and anaesthetist are losing an opportunity to learn from one another.

Some of the pertinent literature and original observations by the authors are described, relevant to changes in normal arterial blood gases in the first few days of life. Most marked changes occur in the first 24 hours, mainly a sharp rise in oxygen tensions and a fall in carbon dioxide tensions. True right-to-left shunting at 24 hours of life in the normal infant is around 20 to 25 per cent, compared with less than 5 per cent in the adult. The authors' findings suggest values at three days of life which are not significantly different from those in healthy older children. Apart from increased alveolar-arterial oxygen difference the lung of the newborn compares more favourably than was previously believed with that of the adult. Details of a preferred technique of arterial puncture are given.

It is easy to diagnose R.D.S. but usually impossible to give a prognosis from clinical observation alone. It is useful to have a classification of degrees of severity. This has been done by measurement of shunting, or more simply by arterial oxygen tensions reached with 100 per cent inspired oxygen. Both of these methods facilitate the assessment of various regimes of management against expected mortality.

The criticisms by anaesthetists of current treatment fall into three groups:

1. The accepted criteria for the use of artificial ventilation are extreme, and can only be explained, if not justified, by the severe complications described as following endotracheal intubation and ventilator care.

2. The requirements of a ventilator in these circumstances, viz. high pressures, constant flow, rapid cycling, and low, controllable tidal volumes, are only approached by the Engstrom; but even the Bird and Bennett tend to be used below their capabilities. Thus one finds them frequently out of phase with spontaneous respiration. There is a dominant fear in the minds of some paediatricians of using too much pressure and of hyperventilation. This often results in totally inadequate function. High pressures, often 40 cm. H_2O or more, and ideally constant high flows during inspiration, are needed to ventilate through infant-size endotracheal tubes. Wide variations in arterial carbon dioxide tensions are tolerable provided that good oxygenation is achieved.

3. It is fundamentally a better approach to look for a unified concept of pathology and management than to treat fully developed respiratory and metabolic acidosis. The answer may lie in promoting better pulmonary blood flow early in the disease. The hope that drugs might be found to do this has not yet been fulfilled; oxygen is a potent pulmonary vasodilator.

If R.D.S. is merely one sign of reaction of an immature vascular bed, then some thought might be given to improving tissue blood flow and oxygenation by drugs such as hydrocortisone, which have been extremely effective on the peripheral circulation of shocked animals and of man.

There is some suggestion that negative pressure respirators of the tank type may give better oxygenation than intermittent positive pressure in R.D.S., even

when ventilation is not as good. Some of the success may be due to simpler operation, but there is also the possibility, still unsupported by concrete evidence, that negative pressure results in better matching of ventilation to perfusion.

It is suggested that specifically designed miniature monitors may be more useful for the nurses than cumbersome electrocardioscopes.

The anaesthetist's main problems as a consultant are to establish the best possible respirator care in an atmosphere where ventilators are usually reserved for the moribund; to exchange ideas with the neonatologists, to educate nurses in premature units in respiratory care with ventilators, and to co-ordinate the premature intensive care unit with the general intensive care unit in the hospital.

Résumé

L'anesthésiste est habituellement consulté dans le traitement du syndrôme de détresse respiratoire du nouveau-né. Bien qu'il ne dicte pas au pédiatre la conduite à tenir, il est dans l'ordre de faire des remarques sur les méthodes actuelles de traitement respiratoire.

La littérature en anesthésie traite couramment en détail des problèmes des différences alvéolo-artérielles en oxygène et le pédiatre et l'anesthésiste perdent des chances d'apprendre l'un de l'autre.

Nous résumons la littérature pertinente et les observations originales des auteurs en ce qui concerne les changements des gaz dans le sang artériel normal au cours des premiers jours de la vie. Les changements les plus marqués surviennent au cours des 24 premières heures de la vie; ils consistent en une élévation rapide de la tension en oxygène et une baisse de la tension de CO_2. A 24 heures de vie, la vraie communication de droite à gauche chez l'enfant normal est d'environ 20 à 25 pour cent alors qu'elle est moins de 5 pour cent chez l'adulte. Les résultats des auteurs donnent des chiffres, à 3 jours de vie, qui ne sont pas très différents de ceux d'autres enfants normaux plus agés. A part l'augmentation de la différence alvélo-artérielle de l'oxygène, le poumon de l'enfant est comparable à celui de l'adulte de façon plus favorable qu'on ne le croyait.

Nous mentionnons les détails de la technique de ponction artérielle que nous préférons.

Il est facile de diagnostiquer un syndrome de détresse respiratoire, mais il est habituellement impossible de faire un pronostic d'après l'observation clinique seulement. Il est nécessaire de faire une classification des degrés de sévérité.

L'on a réalisé cela en mesurant la communication ou plus simplement en mesurant les tensions d'oxygène obtenues en faisant inspirer de l'oxygène pur. Ces deux méthodes facilitent l'évaluation des diverses sortes de traitements et de la mortalité à prévoir.

Les critiques des anesthésistes sur le traitement actuel se classent en trois groupes:

1. Les critères acceptés pour l'usage de la ventilation artificielle sont excessifs; ils ne peuvent s'expliquer seulement sinon justifiés par les complications graves décrites à la suite de l'intubation endotrachéale et du traitement par le ventilateur.

2. Les exigences d'un ventilateur dans ces circonstances; exemples: une

pression élevée, un volume constant, un complexe respiratoire rapide et lent un volume d'air courant controlable ne sont réalisés que par l'Engstrom, mais même le Bird et le Bennett sont utilisés là où ils ne le devraient pas. Ainsi on les trouve souvent à contre temps avec la respiration spontanée.

Il existe une crainte dominante dans l'esprit des pédiatres d'employer une pression trop élevée et de l'hyperventilation: il s'ensuit souvent une fonction tout à fait insuffisante. Une pression élevée, souvent 40 cm. H_2O et plus, et un débit rapide durant l'inspiration sont nécessaires pour ventiler à travers un tube endotrachéal pour enfants. L'enfant peut tolérer de grandes variations de tension de CO_2 pourvu que l'on réussisse une bonne oxygènation.

3. Fondamentalement, il est préférable d'essayer de chercher un seul concept de pathologie et de traitement que de traiter une acidose marquée respiratoire et métabolique. La réponse peut se trouver en essayant d'augmenter le débit sanguin pulmonaire plus tôt au début de la maladie. L'espoir que l'on puisse trouver des médicaments pour réaliser cela n'a pas encore été réalisé; l'oxygène est un vasodilatateur pulmonaire puissant.

Si le syndrome de détresse respiratoire est simplement un signe de réaction d'un lit vasculaire immature, alors il faudrait songer a améliorer le débit sanguin tissulaire et l'oxygénation par des médicaments tels l'hydrocortisone qui s'est avérée efficace sur la circulation périphérique des animaux choqués et sur l'homme.

Il est possible que les respirateurs à pression négative du type "tank" puissent donner une meilleure oxygénation que la pression positive intermittente dans le syndrome de détresse respiratoire, même lorsque la ventilation n'est pas aussi bonne. Certains succès peuvent être dus à une opération plus simple, mais il y a aussi la possibilité, que l'on n'a pas démontré cliniquement, que la pression négative entraine une meilleure synchronisation de la ventilation et de la perfusion.

Il est probable que des moniteurs miniatures construits spécifiquement soient plus utiles pour les infirmières que d'imposants cardioscopes.

Les principaux problèmes des anesthésistes, en qualité de consultants, sont d'établir les meilleurs soins possibles avec les respirateurs dans une atmosphère ou les ventilateurs sont habituellement réservés aux moribonds et d'échanger des idées avec les néonatologistes pour enseigner les infirmières dans les unités de soins respiratoires des prématurés avec des ventilateurs et de coordonner l'unité de soins intensifs des prématurés avec l'unité des soins intensifs générale dans l'hôpital.

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