Distending pressure in infants with respiratory distress syndrome

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The application of distending pressure to 40 babies with the respiratory distress syndrome (RDS) is described. Pressures greater than 10 cm H₂O were rarely used. On starting distending pressure, considerable improvements in blood gases occurred in all but 4 babies, 2 of whom had pneumothoraces and the other rapidly deteriorated and died from an intraventricular haemorrhage shortly afterwards.

In 27 babies the distending pressure was applied for hypoxaemia during the course of the disease. 16 survived without further intervention, though one case eventually required long-term continuous distending pressure (CDP) for pulmonary oedema. 11 out of the 27 required intermittent positive pressure ventilation (IPPV), with 6 survivors.

In 13 other babies who had not received CDP previously, the technique was used to assist weaning from IPPV. It sustained oxygenation in each case, and 12 of these babies survived.

Seven babies developed pneumothoraces and 2 babies intrapulmonary cysts. 6 babies died, 3 with pneumothoraces. The overall incidence of intraventricular haemorrhage was 3 proven and 2 suspected cases. There was no evidence that distending pressure in any form increased the incidence of intracranial haemorrhage.

The various techniques for administering continuous positive airways pressure (CPAP) and continuous negative pressure have been usefully grouped together by Chernick (1973) under the term continuous distending pressure (CDP). CDP has been shown to give significantly better results than conventional therapy in the management of infants with respiratory distress syndrome due to hyaline membrane distress (Fanaroff *et al.*, 1973; Rhodes and Hall, 1973). In addition, CDP has proved useful in weaning infants off ventilators (Cumarasamy *et al.*, 1973) and in situations where pulmonary oedema proves difficult to control in infants breathing spontaneously (Hatch *et al.*, 1973; Robertson, 1974).

We describe our experiences over a 20-month period with the use of CDP administered to 40 babies, of whom 34 survived.

Materials and methods

All babies were on the Special Care Baby Unit of our hospital. 36 were born in the hospital and 4 were

referred from outside centres. All babies were suffering from RDS according to the standard criteria (Davies *et al.*, 1972). They were nursed naked in a thermal environment to keep their abdominal skin temperature in the range of 36–36.5 °C.

Umbilical arterial and venous catheters were inserted in all cases as soon as the diagnosis was made. The arterial catheter was used for blood pressure monitoring as well as blood sampling, and was left *in situ* for as long as required. Sufficient oxygen was given to keep the arterial Po₂ in the range of 60–90 torr, and base was given to correct pH to 7.25. When possible, hourly tube feeds of expressed breast milk were given. When CDP was administered by head box or when feeds accumulated in the stomach, 10% dextrose with appropriate electrolyte supplements was given through one of the umbilical catheters.

Blood gases were measured on an IL 313 Blood Gas analyser. In most infants early assessment of severity was carried out using the hyperoxia test (nitrogen washout test). The inability to achieve an arterial Po₂ of >100 torr while breathing pure oxygen, within the first 6 hours, used to carry a poor prognosis (Boston, Geller, and Smith, 1966; Robertson *et al.*, 1968). To allow comparisons between arterial oxygen tensions at different alveolar oxygen concentrations the alveolar-arterial

oxygen difference ($A-aDO_2$) was calculated, using the alveolar air equation and assuming a respiratory quotient of 0.8.

$$PAO_2 = FIO_2 - PACO_2 \left[FIO_2 + \frac{1 - FIO_2}{R} \right]$$

where PAO_2 is alveolar oxygen tension; FIO_2 is inspired oxygen concentration; $PACO_2$ is alveolar CO_2 tension assumed to be the same as arterial CO_2 tension; R is respiratory quotient. Thus a decrease in $A-aDO_2$ represents improving pulmonary function and oxygenation and vice versa.

CDP was applied in three ways. (1) CPAP was applied by means of a head box. The system originally described by Gregory *et al.* (1971) was used; initially using a home-made box and more recently a Vickers head box. (2) Alternatively, CPAP was applied by means of an endotracheal tube; Etches, Houghton, and Moore (1973) have described our technique of administering CPAP using the Bennett PR2 special ventilator with the rate control turned down to zero. (3) Continuous negative pressure was applied using the Pulmarca 1400 chamber (Draeger Ltd). Whichever technique of CDP was applied, the pressure rarely exceeded 10 cm H_2O .

Indications for CDP. We have three indications for starting distending pressure during the evolution of RDS. (1) When the infant is unable to maintain an arterial PO_2 of >60 torr in FIO_2 0.6 or greater. (2) In the presence of less severe blood gas abnormalities when the infant is 'working hard' as assessed clinically, and if the signs are present early, the infant is of very low

birthweight, and radiological changes are severe. (3) In attempting to wean from intermittent positive pressure ventilation (IPPV) when the infant is unable to sustain an adequate Pao_2 breathing spontaneously at atmospheric pressure.

Results

The clinical details of the infants studied are shown in Tables I to IV. 16 babies required CDP only. All of these survived (Table I). 11 babies started with CDP but their condition subsequently deteriorated and IPPV was required. 5 of these died, and 6 survived (Table II), CDP having been used to wean them off IPPV. A further 12 babies, who had started IPPV without previous CDP improved sufficiently for them to sustain adequate gas exchange when CDP was substituted for the IPPV. All but one of these survived (Table III). 2 further cases required very long-term CDP for the control of pulmonary oedema associated with a persistent ductus arteriosus (Table IV). One of these (Case 11) had already been treated in the head box. Several babies received distending pressure by more than one technique (Table V).

Thirty-two of the babies had hyperoxia tests before 6 hours of age. 18 were in the 'good prognosis' range; 15 of these survived. 14 were in the 'bad prognosis' range; 12 of these survived. 8 in this latter group required assisted ventilation within 6 hours of birth, and of the other 6, 4 required only CDP and the other 2 died on IPPV. Of the 15

TABLE
Babies receiving distending

Case no.	Sex	Birthweight (g)	Gestational age (wk)	Type of distending pressure	Pressure used (cmH ₂ O)
1	F	2420	34	Box	6-8
2	M	2300	35	ETT	3-12
3	M	1340	29	Box	4-10
4	F	2200	34	Box	2-10
5	M	1400	33	Box	3-9
6	M	2040	33	Box*	3-8
7	M	1150	29	Box	4-9
8	F	1330	31	Box*	2-6
9	M	2970	37	Box	4-12
10	M	2180	34	Box	4-10
11	M	1650	29	Box	4-5
12	M	2150	32	Box	?
13	F	1740	31	CNP	5-8
14	F	1300	28	CNP	4-12
15	F	3140	38	ETT	3-10
16	M	2770	40	Box	2-12

Box, Gregory Box. ETT, endotracheal tube. CNP, continuous negative pressure. *Required two periods of CDP.

'good prognosis' survivors, 7 required periods of IPPV in addition to CDP.

Effects on oxygenation (Fig. 1-3). We have blood gas data covering the period when distending pressure was begun on 31 occasions in 28 babies (Fig. 1). On 27 occasions the A- aDO_2 decreased, indicating improved oxygenation. Of the 4 cases in whom the A- aDO_2 deteriorated on starting distending pressure, 2 (Cases 20, 24) had pneumothoraces, the third (Case 27) was deteriorating rapidly and died shortly afterwards, but the fourth (Case 9, Table VI) made satisfactory progress. There was no apparent correlation between the degree of improvement in oxygenation (assessed by a decreasing A- aDO_2) and the length of time distending pressure was necessary, nor the need for subsequent positive pressure ventilation.

CDP was reduced gradually in all cases. Despite this precaution in the 33 cases where blood gas analyses were taken just before and just after discontinuing distending pressure, the A- aDO_2 increased in all but 4 cases (Fig. 2). This indicated persisting pulmonary disease but also showed the effectiveness of even very low pressure (1.5-2 cm H_2O) in improving arterial oxygenation. In general, the greater the rise in the A- aDO_2 on discontinuing distending pressure, the greater the likelihood of further ventilatory assistance being required. Babies whose A- aDO_2 did not increase to more than 240 torr were unlikely to deteriorate again

FIG. 1.—A- aDO_2 before and after starting CDP. A fall in A- aDO_2 means improving oxygenation. x, died; ●, pneumothorax when CDP started.

I
pressure only. All survived

Age (hr) distending started	Duration (hr) of distending pressure	Duration of exposure to $\text{FIO}_2 \geq 0.6$		Complications
		In distending pressure (hr)	Total (CDP + spontaneous)	
10	82	2	2	—
13	65	11	11	1 ETT change; <i>Esch. coli</i> in ETT aspirate
38	17	0	17	—
34	24	1	31	—
11	58	0	3	Pulmonary cysts
6	60	3	8	—
8	35	0	16	<i>Esch. coli</i> in blood culture
8	65	3	11	—
31	61	25	41	—
10	33	0	4	Anal atresia; transferred for surgery 45 hr
5	31	0	3	See Table IV and text
28	44	19	94	Pneumothorax
9	16	0	2	—
3	28	2	5	—
58	64	21	77	1 ETT change; <i>Esch. coli</i> in ETT aspirate
36	48	6	10	—

TABLE

Babies starting on distending pressure and

Case no.	Sex	Birthweight (g)	Gestational age (wk)	Type of distending pressure	Age (hr) distending pressure applied	Distending pressure used (cmH ₂ O)	Distending
							Duration (hr)
17	M	2750	38	ETT	7	4-15	106
18	M	3200	40	Box	35	3-14	51
19	F	2260	34	ETT	8	5-10	112
20	M	2030	34	Box	16	?	68
21	F	2040	34	ETT	4	2-7	91
22	F	990	29	CNP	3	4-10	78
23	M	1710	33	Box	42	6-10	44
24	M	2290	34	ETT	19	9-10	46
25	F	1280	29	Box	6	9	8
26		1260	29	Box	17	9	21
27	F	1930	34	ETT	7	7	6

HMD, hyaline membrane disease; BPD, bronchopulmonary dysplasia; IVH, intraventricular haemorrhage.

TABLE

Infants starting with IPPV and who were

Case no.	Sex	Birthweight (g)	Gestational age (wk)	Type of distending pressure	Pressure used (cmH ₂ O)	Age (hr) distending pressure applied	Distending
							Duration (hr)
28	M	1200	30	ETT	5-8	3	63
29	F	1790	30	ETT, Box	4-10	63	85
30	F	3440	42	ETT	3-5	5½	7
31	F	1670	33	ETT, Box	4-13	41	26
32	F	2270	34	ETT, Box	3-8	28	107
33	F	3870	37½	ETT	7-8	15½	60
34	F	2660	37	Box	?	26	66
35	F	1290	28	ETT	3-10	2½	99
36	F	1430	29	ETT	7	10	66
37	F	2730	40	ETT	2-7	17	95
38	F	3510	39	ETT	4-7	55	39
39	M	1860	39	ETT	4-7	2	35

TABLE

Babies requiring long-term distending

Case no.	Sex	Birthweight (g)	Gestational age (wk)	Pressure used (both ETT)	Age started (hr)	Distending pressure	
						Duration (hr)	No. of periods
40	F	1190	28	2-10	2½	746	10
11	M	1650	29	4-7	170	292	5

II

requiring subsequent ventilation. 5 died

pressure		IPPV		Duration of exposure to $\text{Fio}_2 \geq 0.6$		Complications
No. of periods	Duration (hr)	No. of periods	Distending pressure (hr)	IPPV (hr)	Total (IPPV + CDP + spontaneous)	
4	8	1	72	8	87	4 ETT changes; <i>Esch. coli</i> in ETT aspirate
2	12	1	27	12	60	2 ETT changes
4	41	3	33	29	71	5 ETT changes
2	33	1	42	32	76	—
2	31	1	42	31	77	2 ETT changes; <i>Esch. coli</i> in ETT aspirate
3	84	1	5	73	100	Pneumothorax; 2 ETT changes
2	44	3	26	43	116	Died; HMD, BPD; 3 ETT changes
2	12	1	12	46	64	Died; HMD, early BPD; 1 ETT change; pneumothorax
1	13	1	8	13	27	Died; necropsy refused; pneumothorax
1	7	1	21	7	46	Died; necropsy refused; pneumothorax
1	18	1	6	18	31	Died; HMD, IVH

III

weaned off using distending pressure. 1 died

pressure		IPPV		Duration of exposure to $\text{Fio}_2 \geq 0.6$		Complications
No. of periods	Duration (hr)	No. of periods	Distending pressure (hr)	IPPV (hr)	Total (CDP + IPPV + spontaneous)	
4	9	2	9	9	57	<i>Esch. coli</i> in ETT aspirate
5	112	4	54	101	190	2 ETT changes; <i>Pseudomonas</i> in ETT aspirate
1	1	1	7	1	17	—
2	46	2	10	36	52	1 ETT change
3	38	2	36	37	84	1 ETT change; <i>Staphylococcus albus</i> in ETT aspirate; pulmonary cysts
1	1	1	49	1	68	1 ETT change; sacral agenesis
2	21	2	28	15	49	Pneumothorax
4	35	5	65	14	208	1 ETT change; <i>Staph. albus</i> in blood; late pulmonary haemorrhage
5	26	5	65	24	187	3 ETT changes; pneumothorax
4	32	4	49	25	88	1 ETT change; <i>Esch. coli</i> in ETT aspirate
1	1	1	26	1	62	—
2	33	3	35	34	69	Died; HMD, IVH

IV

pressure for pulmonary oedema

IPPV		$\text{Fio}_2 \geq 0.6$			Complications
Duration (hr)	No. of periods	Distending pressure (hr)	IPPV (hr)	Total (hr)	
292	9	8½	66	87	9 ETT changes; <i>Esch. coli</i> in ETT aspirate
165	5	—	—	—	5 ETT changes; <i>Esch. coli</i> and <i>Proteus</i> in ETT aspirate; see Table I; died 6 wk old; IVH

TABLE V
Routes by which CDP was administered to 40 babies

ETT only	17
Head box only	14
Negative pressure (CNP) only	2
Head box & ETT	6
Head Box & CNP & ETT	1

FIG. 2.— $A-aDO_2$ when CDP was discontinued. A rise in $A-aDO_2$ means deteriorating oxygenation. $\times$, died; $\bullet$, required subsequent ventilatory assistance.

(Fig. 2). There did not seem to be a greater likelihood of deterioration after stopping CDP if it had been administered by head box or tube, particularly when allowance is made for the fact that babies receiving CDP by tube had often been ventilated previously.

In 26 cases blood gases were analysed before and after converting from IPPV to CDP. There was a marked improvement in $A-aDO_2$ in 13 cases (Fig. 3) and in 13 cases it deteriorated. Improvement was most often found in those babies who had been very active and restless while ventilated, yet were unable to sustain an adequate Pao_2 when breathing spontaneously in high oxygen concentrations at atmospheric pressure. There was no association between change in $A-aDO_2$ and the need for subsequent IPPV.

Pco_2 changes. We did not set any limit of $Paco_2$ above which CDP would not be attempted. 9

FIG. 3.—Change in $A-aDO_2$ when CDP was substituted for IPPV. $\bullet$, IPPV subsequently; $\times$, died.

babies had $Paco_2 > 60$ torr on starting CDP and 7 of these survived without additional IPPV. The $Paco_2$ increased on starting CDP in 11 babies, decreased in 16, and in 3 it remained constant. The most dramatic was a fall from $Paco_2$ 115 to $Paco_2$ 60 in Case 9. A rise in $Paco_2$ of more than 10 torr occurred in 4 cases when CDP was started. Two subsequently needed IPPV. On stopping distending pressure, $Paco_2$ increased in 17, fell in 9, and remained constant in 7. The degree or direction of change bore no relation to the severity of previous illness nor to the need for further distending pressure or IPPV. When distending pressure replaced IPPV, there was a rise in $Paco_2$ in 16 infants, a fall in 8, and in 2 it remained constant. Of the 8 cases where the $Paco_2$ fell when distending pressure replaced IPPV, only 2 infants needed subsequent ventilation, compared with 10 out of the 16 who had a rise in $Paco_2$ ($\chi^2 = 1.7$, not significant).

Duration of oxygen therapy. In Table I it can be seen that those babies who required CDP alone only had transient subsequent exposure to greater than 60% oxygen. Even the babies who required periods of positive pressure ventilation

(Table II) had relatively short exposures to high inspired oxygen tensions, rarely exceeding 100 hours. The 2 infants who died after 64 and 116 hours exposure, however, did have mild histological changes of pulmonary oxygen toxicity at necropsy examination. Babies who started on positive pressure ventilation had longer overall exposure to $\text{FIO}_2 \geq 0.6$ (Table III) and also longer exposure to distending pressure. Only one of these latter infants died and none of the survivors had signs of persisting chronic lung disease at the time of discharge.

Complications. 7 out of the 40 babies developed pneumothoraces. 2 (Cases 34 and 36) had been ventilated previously but developed the pneumothorax while receiving CDP. Case 24 probably developed his pneumothorax before distending pressure was applied. 5 babies developed pneumothoraces while receiving distending pressure in the head box; one baby was in the negative pressure box, and one was receiving distending pressure through an endotracheal tube.

6 out of the 7 pneumothoraces were drained. 3 babies died (Cases 24, 25, and 26) and in all cases the pneumothorax was considered to be an important factor in the cause of death.

Two babies developed cystic changes in the lung fields. In Case 32 the cyst steadily increased in size (Fig. 4) and was eventually removed. Histology suggested congenital lymphatic cystic disease. Case 5 developed midzone cysts on the fourth day which spontaneously cleared (Fig. 5).

Thirty-one endotracheal tube changes were required in the 22 babies in whom CDP was administered by this route (excluding the 2 infants (Table IV) receiving long-term CPAP). Where the tube was used to administer distending pressure an average of one tube change was required for every 40 hours distending pressure. In most cases the tube had to be changed as an emergency due to obstruction or dislodgement. In these 22 babies, 9 significant cultures (*Esch. coli* 6, *Staphylococcus albus* 2, and *Pseudomonas aeruginosa* 1) were obtained from tracheal aspirates or on culturing the tip of the endotracheal tube. All 9 babies were treated with

FIG. 4.—Right apical cysts in Case 32, aged 2 months.

FIG. 5.—Right midzone cysts in Case 5, aged 6 days.

broad spectrum antibiotics and none of them died. Infection was not thought to be an important factor in the 6 deaths in the series.

Cause of death. 6 babies died. In 3 the pneumothorax played a major role in the final deterioration (Cases 24, 25, 26). In Case 24 this was the only post-mortem finding in addition to hyaline membrane disease and early pulmonary oxygen toxicity. Cases 25 and 26 with pneumothoraces were twins who had severe respiratory distress and in whom necropsy was refused, but clinical signs suggested that they had also developed intraventricular haemorrhages. The other 3 infants who died had severe RDS (Cases 23, 27, 39). At necropsy all 3 had classical hyaline membrane disease and 2 (Cases 27, 39) had an associated intraventricular haemorrhage: Case 23 also showed early histological changes of pulmonary oxygen toxicity.

Discussion

Our results confirm the previous satisfactory reports on the use of distending pressure both in

terms of improved oxygenation and decreased mortality rate (Gregory *et al.*, 1971; Chernick and Vidyasagar, 1972; Fanaroff *et al.*, 1973; Rhodes and Hall, 1973; Chernick, 1973). The overall mortality rate in this group of 40 severe cases of RDS was 15%. In only 4 out of the 31 occasions where data are available when distending pressure was applied did the A- aDO_2 fail to improve. 2 of these already had pneumothoraces and the third was rapidly deteriorating and died shortly afterwards while receiving IPPV. The values for the fourth case are shown in Table VI. The PaO_2 improved when CDP was applied, but because of the big fall in the PaCO_2 plus the rise in FIO_2 the A- aDO_2 actually increased. The infant continued to improve and survived without positive pressure ventilation.

We made an arbitrary decision to start CDP at a PaO_2 of 60 torr in $\text{FIO}_2 \geq 0.6$. Others (Dunn, Speidel, and Storrs, 1973) recommend applying CDP at a lower FIO_2 requirement. Our practice has been to start with a pressure of 8–10 cm H_2O . Usually this achieves adequate oxygenation. We have only used pressures in excess of 10 cm H_2O on 7 occasions in attempts to improve oxygenation, the

TABLE VI

Blood gas changes after starting distending pressure in Case 9

Age (hr)	30	31½	34½	38½
FIO ₂	0.8	0.95	0.92	0.90
PaO ₂	24	62	50	150
A-ado ₂	447	614	573	552
Paco ₂	115	60	78	85
Distending pressure (cm H ₂ O)	—	12	12	12

maximum pressure used being 15 cm H₂O. In all cases the increase to >10 cm H₂O was successful and it is worth noting that 5 of the babies treated were large (weight 2.30 kg, Case 2; 2.97 kg, Case 9; 2.77 kg, Case 16; 2.75 kg, Case 17; and 3.20 kg, Case 18).

Having established the infant on distending pressure our first priority is to lower the FIO₂ to <0.6 because of the possible risk of pulmonary oxygen toxicity, and then gradually to lower the pressure to 1–2 cm H₂O when, if the baby maintains an adequate PaO₂, distending pressure is discontinued. At pressures of 8–10 cm H₂O or above, apnoeic attacks occasionally occurred, particularly in babies of less than 1500 g who were recovering from their respiratory distress. In infants having apnoeic attacks or accumulating CO₂ with an adequate PaO₂

we found that lowering the pressure usually abolished the attacks and brought down the CO₂. The deleterious effects of distending pressure administered to a baby with stable lungs is shown in Fig. 6. This was a premature infant (weight 2030 g, 33 weeks' gestation) who had respiratory difficulties requiring an FIO₂ of 0.9 or greater. However, the chest x-ray looked normal, and a shake test on the mother's liquor had been positive and the illness was subsequently attributed to a haemophilus influenza pneumonia. Application of CDP caused CO₂ accumulation and a marked reduction in the respiratory excursions.

Although our practice has been gradually to reduce distending pressures to 1–2 cm H₂O, we found (Fig. 2) that most infants increase their A-ado₂ markedly, even when this low distending pressure is discontinued. Those infants who had the largest rise in A-ado₂ were those who subsequently needed further CDP or ventilation—(Fig. 2). Large rises in A-ado₂ also occurred in those babies who had received distending pressure by endotracheal tube and in whom the endotracheal tube was left *in situ*. It seemed possible that their greater rise in A-ado₂ was due to inability to grunt; however, all but one of these babies had received IPPV and prolonged oxygen therapy, and though the inability to grunt (Harrison, Heese, and Klein, 1968) may have played a part in increasing their A-ado₂, the severity of their preceding lung disease

FIG. 6.—Changes in respiratory excursion when CDP was applied to an infant with lungs of presumed normal compliance. (Respirations monitored with an impedance pneumograph). Time refers to minutes after the introduction of CDP at time 0. Figures in parentheses indicate the duration of CDP at the pressure shown when the respiratory record was taken.

seems a more likely cause. Moreover, we have found it more satisfactory on reducing pressure to zero to leave the tube *in situ* initially, in case there is rapid deterioration, rather than to remove pressure and tube simultaneously.

One major application of distending pressure which has received comparatively little attention is its use in weaning babies off ventilators. This was used in 21 infants—all the cases shown in Tables III and IV and Cases 17–23 in Table II. Although several cases required further periods of IPPV (Cases 11, 19, 23, 28, 29, 31, 32, 34–40), all but 2 (Cases 23, 29) survived after a further period of distending pressure. Many such infants started spontaneous respirations while being ventilated and yet were unable to sustain an adequate PaO_2 when breathing spontaneously at atmospheric pressure, but did so when distending pressure was applied. In some of those infants the arterial Po_2 increased when CDP replaced IPPV at the same inspired oxygen concentration (Fig. 3). This was noted particularly in babies who were restless and had started to breathe out of phase with the ventilator. The improved oxygenation may be due to improved pulmonary perfusion since the pressure applied to the lung is lower with distending pressure than IPPV and also to the improved oxygenation which occurs when the sick infant lies quietly.

We found a moderately high incidence of pneumothoraces (7 out of 40, 17.5%), but it is within the range reported by other authors using distending pressure (Chernick, 1973). We have no data on the pressure in the head box applied to 2 of the infants, but with the other 5 the pressures were ranging from 8–10 cm and in 1 of these 5 the pneumothorax may have preceded the distending pressure. 5 of the 7 pneumothoraces and both cases with cysts alluded to above were in the head box at the time of diagnosis. There were no sudden rises in pressure and the pressures applied were no greater than those used by many other workers, though higher than those used by Dunn *et al.* (1973). The development of cysts during CDP has not been described before. In Case 32 we believe that the cyst was an example of the condition of intralymphatic air recently described by Stahlman, Cheatham, and Gray (1973) and believed by them to be due to rupture of alveoli into the lymphatics during high pressure IPPV such as used initially in this baby. In Case 5 the lesions may have been similar but they resolved spontaneously and the lung has remained normal since. Furthermore, this infant was not ventilated and the maximum distending pressure used was 8 cm H_2O . We do not think that the cysts were the result of infection

(Avery, 1968; Kuhn and Lee, 1973), though Case 32 did grow *Staphylococcus albus* on one occasion from an endotracheal tube culture.

Vert, André, and Sibout (1973) reported the occurrence of intraventricular haemorrhage in association with distending pressure and related this to the increased intracranial vascular pressure when distending pressure is applied by a system which involves a tightly fitting neck seal. 3 babies in this series had proven intraventricular haemorrhages (Cases 11, 27, 39). 2 others probably occurred (Cases 25, 26), but permission for necropsy was refused. In the 3 confirmed cases 2 received distending pressure by endotracheal tube only (Cases 27, 39) and the third case (Case 11) developed signs and symptoms of intraventricular haemorrhage after recovering from RDS. After prolonged distending pressure and IPPV (Table IV) he survived but was noted to be hydrocephalic and died aged 6 weeks after an operation to insert a Pudenz valve. Necropsy showed hydrocephalus secondary to obstruction of the ventricular foramina by an old intraventricular haemorrhage. Even if Cases 25 and 26 did develop intraventricular haemorrhages, we would not regard an incidence of 5 intraventricular haemorrhages in 40 severe cases of RDS as excessive, and would not suggest any causal relation between distending pressure with or without neck constriction and IVH.

Cases 11 and 40 (Table IV) have been described in detail elsewhere (Robertson, 1974). Both had severe pulmonary oedema with heart failure due to a widely persistent ductus arteriosus. The use of distending pressure in this situation proved most satisfactory where pulmonary oedema could not be controlled pharmacologically. There are obvious disadvantages to all the methods of applying CDP. We do not feel safe in continuing oral feeds when applying CDP by head box as access to the upper airway is restricted should the child regurgitate. The poor access is also a disadvantage should apnoea occur and intubation be required. However, the trunk is easily accessible for clinical examination and x-rays. The problems are reversed with the negative pressure box, in that the access to the upper airway is good and feeding is possible, but clinical and radiological examination of the chest and abdomen is impossible without discontinuing the negative pressure. We have also had some difficulties in maintaining body temperature in the negative pressure box. Both box methods have the additional problem of maintaining an adequate neck seal without excoriating the baby's skin. We have minimized this by using a protective collar of gamgee wrapped in tube-gauze placed around the

infant's neck underneath the iris diaphragm of the apparatus.

Using the endotracheal tube to administer the distending pressure avoids all the problems of the neck seal but substitutes the manifold problems of endotracheal tubes—obstruction, infection, and laryngeal injury. We had to change the tubes on average every 40 hours in our cases, and frequently this was required because of deterioration in the infant's condition when the tube became dislodged or obstructed. Furthermore, significant pathogens were grown from the airways of 9 of the infants and in several cases (e.g. Case 22) there were difficulties on extubation due to retained secretions and laryngeal oedema.

A trial of distending pressure administered by face mask, endotracheal tube, or CNP to matched groups of infants with RDS (Olinsky, MacMurray, and Swyer, 1973) showed no difference in outcome between the groups. However, the babies receiving treatment via an endotracheal tube received distending pressure for longer than the other groups. A similar comparison is not possible in our series since in only two cases (Cases 2, 15) was endotracheal intubation for the purpose of administering CDP used from the start. In our opinion endotracheal tubes should be used when other alternatives fail or when long-term distending pressure is required as with the 2 babies in Table IV.

The ability to feed such babies over the weeks, while the airway was protected by the endotracheal tube, counter-balanced the other disadvantages of the tube. The use of nasal catheters to administer distending pressure (Novogroder *et al.*, 1973; Kattwinkel *et al.*, 1973) seems to us a logical alternative of which we have had no personal experience.

There are no published data on the optimum indications for starting distending pressure. We have not found the hyperoxia test useful in forecasting either the need for CDP or the ultimate outcome of the disease. While there is no doubt that CDP increases survival (Fanaroff *et al.*, 1973; Rhodes and Hall, 1973), the technique places an extra load on the nursing and medical staff and we would not be in favour of its use liberally if it distracted facilities away from the more critically ill infants in a busy nursery. Moreover, CDP does carry a definite increased risk of pneumothorax—17.5% in this series and up to 24% in other series (Chernick, 1973), a complication of RDS which in our unit doubles the mortality rate. For these reasons we feel the technique should only be used where adequate facilities for monitoring and intensive care are available.

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