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Individualised continuous distending pressure applied within 6 hours of delivery in infants with respiratory distress syndrome

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SUMMARY A preliminary study was performed in which a simple clinical technique for estimating appropriate levels of continuous distending pressure (CDP) in infants with respiratory distress syndrome (RDS) was used to compare two groups of infants; one group had CDP started very early in life (3·1 ± 0·3 hours) while in the other treatment was started at a more conventional age (23 ± 5·4 hours). Appropriate CDP was identified as the point at which transpulmonary transmission of airways pressure to the oesophagus was seen suddenly to increase, while serial measurements allowed CDP levels to be instituted and varied according to physiological signs during the course of each infant's disease. Oxygen requirements fell to <35% more rapidly in the early-treated group (10·6 ± 1·2 v. 67·4 ± 5·6 hours; $P<0·001$), as did the requirement for a CDP >4 cmH_2O (28·9 ± 5·3 v. 87·6 ± 14·2 hours; $P<0·001$). Better ($P<0·01$) values for pH, $Paco_2$, and $A\text{-}aDo_2$ were observed in the early-treated group. We believe that the use of this simple technique has numerous advantages and that very early introduction of CDP can be realised in a manner selective enough for it only to be used in those infants in whom intervention is justified.

A report by Gregory *et al.*[1] led to widespread use of continuous distending pressure (CDP) in the management of the respiratory distress syndrome (RDS). Most studies have shown its use to be associated with improvement of arterial oxygenation, reduced severity of disease, and lessened exposure to high oxygen concentrations. There have been few large and controlled trials of CDP, and advances in sick infant care have led to reductions in mortality from RDS,[2] making it difficult to assess the contribution of any one component in management.[3] Roberton,[4] in a review of reports, was unable to find unquestionable evidence of an effect on survival of infants with RDS as a result of treatment with CDP. If CDP is to have a beneficial effect on mortality it seems likely that application earlier in the disease is required[2 5–7] but at a pressure low enough to avoid complications. Allen *et al.*[8] combined their data with those from similar studies and demonstrated an improvement in mortality by the earlier use of CDP.

While the earlier use of CDP is desirable, the associated morbidity prohibits over-liberal use.[9] If an acceptable risk/benefit ratio is to be preserved it is essential to select only those infants in whom its use would be beneficial, and subsequently to titrate CDP according to the infant's changing needs.

This paper reports our early experience with a simple clinical technique which we believe allows these criteria to be fulfilled.

Patients and methods

After the initial report of a simple method for measuring 'optimal' continuous distending pressure[10] we applied the technique, later described in greater detail,[11] to the first 9 premature infants that became available for study which had RDS (Table 1) according to our modification[12] of the criteria of Baden *et al.*[13] These 9 infants are called the *late-treated* group. A fluid-filled No. 5 FG feeding tube with an end hole was passed through one nostril into the stomach, and attached to a microdot

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transducer. Pressure fluctuations with respiration were displayed* visually and graphically, and the catheter withdrawn until the wave-form changed to an oesophageal pattern, establishing that the catheter tip was in the lower third of the oesophagus. Nasal CDP was applied through a shortened nasotracheal tube inserted through the other nostril into the posterior nasopharynx, and the applied pressure increased from zero in 2 cmH_2O increments. Optimal CDP (OCDP) was defined as the least pressure at which increased transpulmonary transmission of the applied pressure could be detected by a shift of the recorded wave pattern (Fig. 1). As this study was designed to test the applicability of a simple clinical tool, actual measurement of oesophageal pressures was not performed, nor was it necessary to identify the changes sought. This procedure was repeated at frequent intervals, and the pressure at which a wave-form shift occurred was maintained until the next OCDP estimation. There were no difficulties in identifying the point at which shift occurred. In the smallest infants this shift was small but clearly defined and reproducible. We observed no wave-form shift below OCDP and no further upwards shift above OCDP. Occasionally measurement was deferred if an infant was too active, but this was not a problem. Between estimations the feeding tube was repositioned in the stomach and left open to prevent gastric distension from nasal CDP. The tube was flushed with a small volume of saline before the next recording, and replaced if wave damping occurred. In this late-treated group 5 infants were receiving conventional CDP, at pressures below OCDP, at the time OCDP was first measured.

*Datascope Physiological Monitor, Datascope Corporation, New Jersey, USA.

Table 1 *Criteria used to determine the two study groups*

Late-treated group	*Early-treated group*
1. Negative gastric aspirate shake test	1. Negative gastric aspirate shake test
2. At least 5 of the following criteria with disease progression at 24 hours of age.*	2. Chest *x*-ray compatible RDS
Respiratory rate >60/min	3. A-aDO_2 in hyperoxia test >525 mmHg or F_IO_2 >0·75
Intercostal recession and/or substernal retraction	4. Clinical signs compatible with RDS
Grunting	5. OCDP >4 cmH_2O
Recorded Pa_{O_2} <60 mmHg	
Recorded Pa_{CO_2} >50 mmHg	
Chest *x*-ray compatible with RDS	
Cyanosis in room air	

*From Baden *et al.*[13]

Having found that this technique was simple and clinically applicable, we measured OCDP within 6 hours of delivery in the next 17 infants that became available for study; we believed these babies would develop significant RDS (Table 1, early-treated group). All infants had a negative gastric aspirate shake test, which we found in a large group of premature infants indicated a 66% chance of developing RDS and a 100% chance of developing respiratory symptoms,[12 14] and an *x*-ray pattern compatible with RDS. With both these criteria satisfied, an umbilical artery catheter was inserted and a 'hyperoxia test' performed in 100% oxygen,[15] unless the fractional inspiratory oxygen (F_IO_2) concentration was already

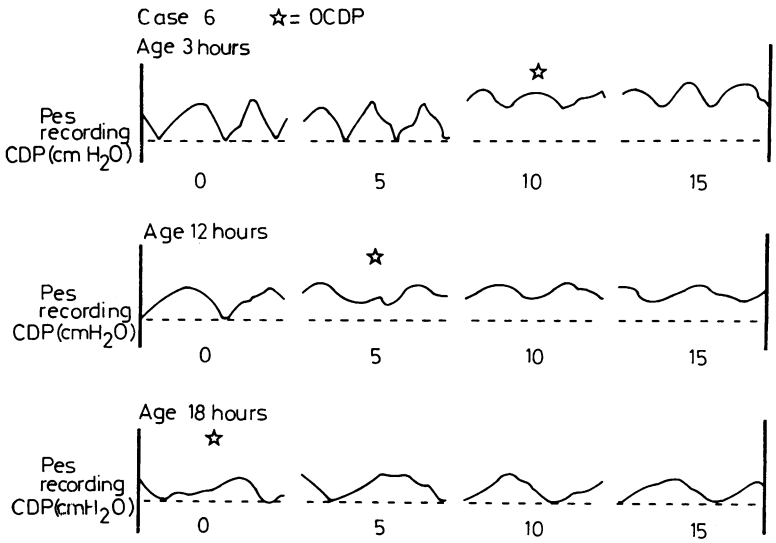

Fig. 1 *Oesophageal pressure (Pes) wave-forms observed over a range of distending pressures in one infant at 3, 12, and 18 hours of age. The pressure at which wave-form shift occurred (OCDP) is indicated, and is seen to fall with resolution of RDS.*

$>0{\cdot}75$. All 17 infants had either an F_{IO_2} $>0{\cdot}75$ or an alveolar-arterial oxygen pressure difference ($A\text{-}aDo_2$) >525 mmHg and, having clinical signs compatible with RDS, had OCDP estimations performed. In only 13 infants (subsequently called the *early-treated* group) in whom the initial OCDP value was >4 cmH_2O, was OCDP maintained, since we felt that a lesser value reflected absent or mild disease. Of the 4 infants excluded by this last criterion one developed RDS, one transient tachypnoea, one congenital heart disease, and the other had early group B β-haemolytic streptococcal infection.

Blood–gas analyses were performed after the institution of OCDP, and thereafter as clinically indicated. Aortic blood pressure (BP) was recorded without CDP, at OCDP, and hourly during the course of treatment. Subsequent retrospective analysis of the case notes was performed to record serial changes in arterial oxygen tension (Pao_2), pH, F_{IO_2}, and $A\text{-}aDo_2$. In this analysis we elected to examine the data in the time frames 0–6, 7–12, 13–24, 25–48, and 49–72 hours of age. Results were not available for every infant in every time frame, and if more than one result was available for a single infant in a time frame the mean value for these results was used in analysis. Calculations for F_{IO_2} in each time frame were made from the case notes record of oxygen concentrations rather than from the F_{IO_2} values noted at the time blood-gases were taken. A mean and standard error was calculated for each group in each time frame (Fig. 2) and the results compared.

The $A\text{-}aDo_2$ was calculated according to the formula:

$$A\text{-}aDo_2 = F_{IO_2}(AP\text{–}WVP) - (Paco_2 + Pao_2)$$

where AP is the atmospheric pressure and WVP is water vapour pressure. All measurements were expressed as mmHg. If CDP was being applied, the CDP (in mmHg) was added to AP.

Comparisons were made between the two groups using Student's *t* test. Three infants died and were included in the study until acute deterioration, at which point they received terminal positive pressure ventilation.

Results

OCDP was applied very early in life [means ± SE] ($3{\cdot}1 \pm 0{\cdot}3$ hours) to 13 patients and at a more conventional time ($23{\cdot}5 \pm 4$ hours) to 9 others. Table 2 compares several parameters in our two

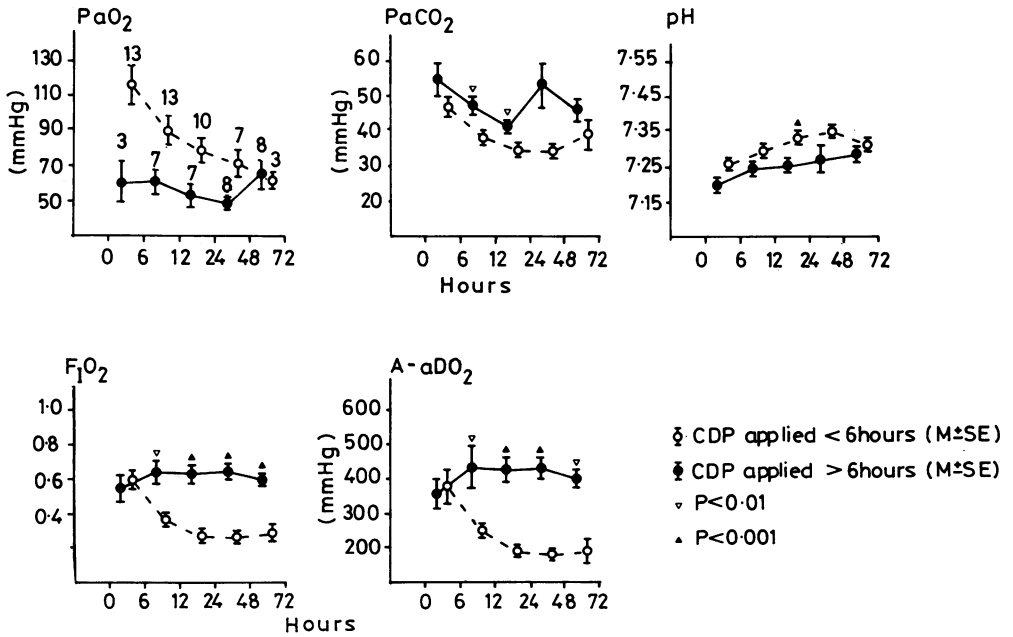

Fig. 2 *Results of serial blood–gas analyses (means ± SE) are compared for early-treated and late-treated groups (see text). Only statistical differences with P values <0·01 or <0·001 are shown, less statistically significant differences being given in the text. The number of infants included for each group for each time frame is shown in the Pao₂ graph.*

Table 2 *Comparison of several parameters between groups early- and late-treated with OCDP*

	Late-onset OCDP		*Early-onset OCDP*	
	M ± *SE*	(*Range*)	*M* ± *SE*	(*Range*)
Entry into study (hours)	23 ± 5·4	(10–54)	3·1 ± 0·3*	(2–5)
Birthweight (g)	1996 ± 206	(960–2780)	1552 ± 130	(900–2160)
Gestational age (weeks)	32·6 ± 1·1	(27–36)	31·1 ± 0·6	(28–35)
3-minute Apgar score	7·6 ± 0·5	(5–9)	7·3 ± 0·6	(2–9)
A-aD_{O_2} in 100% O_2 (mmHg)	649 ± 10	(625–676)	663 ± 5	(640–689)
Initial OCDP (cmH_2O)	10·9 ± 1	(7–16)	8·4 ± 0·9	(5–16)
F_{IO_2} >0·35 (hours)	97·4 ± 5·6	(85–110)	10·6 ± 1·2*	(5–18)
OCDP >4 cmH_2O (hours)	87·6 ± 14·2	(35–154)	28·9 ± 5·3*	(12–57)

*P<0·001

groups. No significant differences were observed in birthweights (t=1·92; df=20; P=NS), gestational age (t=1·32; df=20; P=NS), or Apgar score (t=0·31; df=20; P=NS). All birthweights were appropriate for gestational age,[16] which was confirmed by postnatal assessment.[17] 'Hyperoxia test' results were available from 7 late-treated and 10 early-treated infants before the onset of OCDP; these were not significantly different (t=1·31; df=15; P=NS). The initial pressures required to achieve OCDP, while not significantly different for the two groups (t=1·78; df=20; P=NS), were slightly higher in the late-treated group, compatible with a later stage of the disease process at the time OCDP was started. A more rapid recovery in the early-treated infants is demonstrated by the more rapid fall in oxygen requirements to an F_{IO_2} <0·35 (t=19·49; df=17; P<0·001) and in the time taken for OCDP to fall below 5 cmH_2O (t=4·36; df=17; P<0·001). In the early-treated group chest x-rays showed rapid improvement after initiation of OCDP, while any radiological improvement in the late-treated group was slow to develop. Our aim was to maintain Pa_{O_2} values between 50 and 80 mmHg in all infants, but we failed to achieve this in the rapidly improving early-treated group, as is evident from

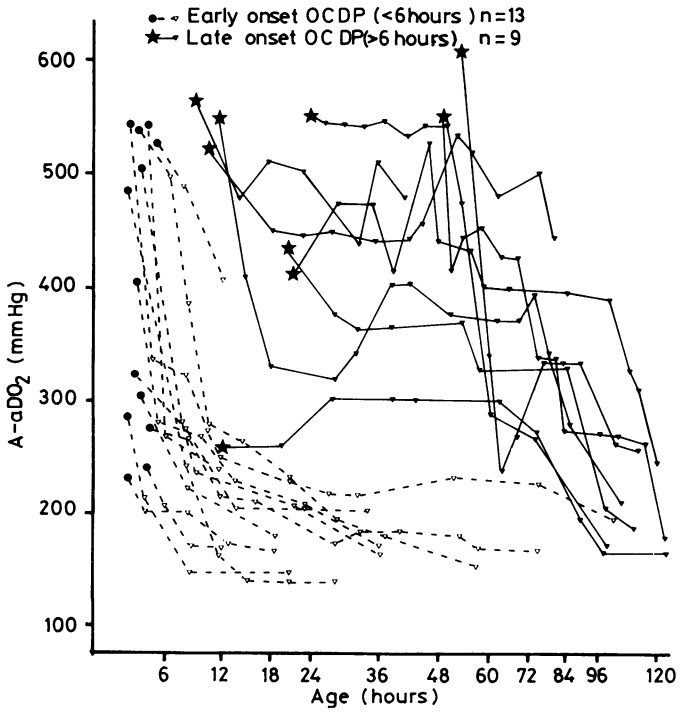

Fig. 3 *Serial values for A-aD_{O_2} from the time OCDP was introduced for all infants.*

Fig. 2. Differences were significant in the time frames 0–6 hours ($t=2\cdot18$; df=14; $P<0\cdot05$), 7–12 hours ($t=2\cdot10$; df=18; $P<0\cdot05$), and 25–48 hours ($t=2\cdot93$; df=13; $P<0\cdot02$). $Paco_2$ was at a lower level in the early-treated group, which achieved significance at 7–12 hours ($t=3\cdot01$; df=18; $P<0\cdot01$), 13–24 hours ($t=3\cdot18$; df=15; $P<0\cdot01$), and at 25–48 hours ($t=2\cdot65$; df=13; $P<0\cdot02$). The pH was higher in the early-treated group being significant from 0–6 hours ($t=2\cdot97$; df=14; $P<0\cdot02$), 7–12 hours ($t=2\cdot43$; df=18; $P=<0\cdot5$), 13–24 hours ($t=5\cdot44$; df=15; $P<0\cdot001$), and 25–48 hours ($t=2\cdot87$; df=13; $P<0\cdot02$). Oxygen requirements showed no difference in the first 6 hours of life, but thereafter there were pronounced differences at 7–12 hours ($t=3\cdot73$; df=18; $P<0\cdot01$), 13–24 hours ($t=7\cdot37$; df=15; $P<0\cdot001$), 25–48 hours ($t=7\cdot61$; df=13; $P<0\cdot001$), and 49–72 hours ($t=4\cdot81$; df=9; $P<0\cdot001$). Similarly for $A\text{-}aDo_2$, no differences were seen in the first 6 hours, but these were pronounced from 7–12 hours ($t=3\cdot6$; df=18; $P<0\cdot01$), 13–24 hours ($t=7\cdot3$; df=15; $P<0\cdot001$), 25–48 hours ($t=8\cdot09$; df=13; $P<0\cdot001$), and 49–72 hours ($t=3\cdot9$; df=9; $P<0\cdot01$). The most visually evident difference between these two groups is in the $A\text{-}aDo_2$, as seen in Fig. 3. Results before OCDP are omitted to prevent over crowding.

There were 3 deaths in the total study group, one in the early-treated group and 2 in the late-treated one. In all 3 infants there was sudden deterioration with apnoea and circulatory disturbance and a rapid course to death despite positive pressure ventilation. All 3 had intraventricular haemorrhages identified at necropsy. No infant developed a pneumothorax while receiving OCDP, although one infant in the late-treated group had a draining pneumothorax at the initiation of OCDP.

In all cases when OCDP was determined, we observed that a reduction in BP was produced by increasing CDP 2–6 cmH_2O above OCDP. In 3 infants OCDP was reassessed in response to reductions of systolic BP by 10–20 mmHg. The BP in each infant returned to its original value on reduction of CDP to the re-estimated and lower OCDP. Two infants in the early-treated group were hypotensive when OCDP was first applied and became normotensive within a few minutes of OCDP application.

Discussion

The term optimal continuous distending pressure or OCDP has been used in this report in deference to Bonta *et al.*[10] who coined the term in their report of the technique. While we are uncertain if our observations reflect a truly optimal level for CDP, we believe that there is good evidence that this technique offers a means of physiologically evaluating an *appropriate* level of CDP for each infant.

In infants with RDS only 20% transmission of airways pressure to the oesophagus was observed by Gregory *et al.*[1] and 25–34% by Bancalari *et al.*[18] Bonta *et al.*[10–11] confirmed these findings but made the important observation that transmission increased to 64% at OCDP, and fell with any further increase in airways pressure. While there are no studies for infants with RDS in the very early hours of life, Suter *et al.*[19] in adult patients with atelectatic lung disease, found that both under- and overinflation of the lungs can impair lung compliance, and reasoned that positive airways pressure can have both beneficial and detrimental effects as a function of recruiting atelectatic areas for gas exchange (beneficial) thereby increasing functional residual capacity (FRC), compliance, and arterial oxygen tension, or by overdistending alveoli (detrimental) which decreases compliance and eventually obstructs venous return and decreases cardiac output. The influence of CDP, therefore, depends on the balance between recruitment of atelectatic areas and overdistension of air sacs. Bonta *et al.*,[11] and ourselves, have observed that there is a sudden increase in transpulmonary transmission of airways pressure at OCDP in infants with RDS, suggesting that at this point the critical opening pressure of a large number of air sacs has been achieved; according to Suter *et al.*[19] this would result in improved compliance and therefore an increased transmission of airways pressure to the oesophagus. The further observation that any increase in CDP above OCDP (as defined here) results in a fall in transmission, suggests that the balance has swung to overdistension of air sacs with a resultant impairment of compliance and reduced pressure transmission.

Our observation that OCDP occurs at successively lower pressures with time, indicates that the pressure necessary to keep the air sacs inflated decreases with improvement of the disease process, as would be expected. The extremely rapid rate of improvement in the early-treated group suggests that very early application of sufficient CDP to increase the FRC towards normal prevents surfactant destruction by a ventilatory pattern that allows low values for FRC at end-expiration.[20–22] Since surfactant probably has a half-life of the order of 15 hours,[23] and is already deficient in infants with RDS, any reduction of an enhanced rate of destruction would be beneficial. The better prevention of atelectasis in the early-treated rather than the late-treated group is reflected by the difference in $A\text{-}aDo_2$ values, indicating less right-to-left shunting of blood with a more even distribution of pressure over a large number of air sacs. The slow

response in the late-treated group is compatible with the findings of Bancalari *et al.*[18] that application of CDP to a group of infants with RDS at a mean age similar to our late-treated group can reduce lung compliance by predominantly hyperinflating open air sacs rather than by reinflating atelectatic units. That the inferior results of our late-treated group were not simply due to inadequate CDP is suggested by the fall in BP which occurred as pressure increased above OCDP and prohibited any further increase. The fall in $Paco_2$ seen during the first 24 hours in the early-treated group is contrary to the experience of other investigators applying CDP later in life,[18 24–25] and suggests that the decreased saccular ventilation observed in their late-studied infants was prevented in our early-treated group.

Despite the use of very high initial pressures in some early-treated infants we saw no pneumothoraces, probably reflecting a more even distribution of the pressure load without hyperinflation, and a briefer total exposure to increased airways pressure. In the combined study groups the mortality was 14%; the 3 infants who died weighed 900, 900, and 1650 g at birth. While the use of continuous positive pressure with a neck seal has been incriminated in the aetiology of intraventricular haemorrhage,[26] secondary to obstructed venous return to the chest, our BP observations, used as an indirect monitor of cardiac output, which in turn is directly related to mean airways pressure,[27] did not suggest any such occurrence in these infants. Excessive pressure can cause a pronounced increase in intra-[28] and extrapulmonary[29–30] right-to-left shunt, and a reduction in cardiac output[31] and blood pressure.[32] The relationship between lung volume and pulmonary vascular resistance is a U-shaped curve with its nadir at normal FRC,[33] which may partly account for the rapid reduction in $A\text{-}aDo_2$ seen only in the early-treated group. It would appear that our observations of a fall in blood pressure when exceeding OCDP represents the ascending limb of the U-shaped curve, while the two observations of improved BP with introduction of OCDP could represent a shift from the descending limb to the nadir.

No completely satisfactory diagnostic criteria exist for RDS, but most require that clinical and biochemical deterioration continue at least beyond the first 24 hours of life. Any therapeutic modality which prevents the occurrence of these cardinal signs makes comparison of treated and control groups more difficult in terms of equating the initial severity of their lung disease. In this preliminary study we attempted to include only infants we believed would develop severe RDS, this we did by using exclusive entry criteria for the early-treated group. It may be seen that a tendency to lower weights and gestations in the early-treated group, and a preponderance of males (84 v. 44%) would also tend to reduce the likelihood of early recovery in this group.

We believe that this preliminary study supports the observations of Bonta *et al.*,[11] and extends them by showing that measurement of OCDP can not only help to select infants in whom CDP is warranted early in life, but can be used to identify appropriate levels of CDP in each patient throughout the disease process. With this technique we have been able to apply individualised CDP, with confidence, at a much earlier age than in other studies, and demonstrate a striking difference in the clinical course of such infants. Larger numbers are required to demonstrate any significant effect on mortality or morbidity, and a clinical trial of this technique is currently in its early stages.

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