Archives of Disease in Childhood, 1976, **51**, 13.

Continuous positive airways pressure treatment by a face chamber in idiopathic respiratory distress syndrome

HANS AHLSTRÖM, BJÖRN JONSON, and NILS W. SVENNINGSEN

From the Departments of Paediatrics and Clinical Physiology, University of Lund, Sweden

Ahlström, H., Jonson, B., and Svenningsen, N. W. (1976). *Archives of Disease in Childhood*, **51**, 13. **Continuous positive airways pressure treatment by a face chamber in idiopathic respiratory distress syndrome.** During a 3-year period 45 infants with idiopathic respiratory distress syndrome (IRDS) requiring ventilatory support were treated in the neonatal unit. Continuous positive airways pressure (CPAP) via the face chamber was applied as initial therapy in 39 infants and during weaning from initial intermittent positive pressure ventilation (IPPV) treatment in 5 infants, whereas 1 infant received IPPV only. Among the 39 infants initially treated with CPAP 9 required IPPV as well. The overall survival rate was 37/45 or 82%.

Incapacity to hyperoxygenate while breathing 100% oxygen was the indication for CPAP while occurrence of apnoeic attacks was the indication for IPPV. Pao_2 during the hyperoxia test before ventilatory support was less than 50 mmHg in 10 infants and between 50 and 105 mmHg in 35 infants.

Surviving infants were followed up with neurological and developmental control examinations as well as chest *x*-ray, and in several infants pulmonary function tests. 3/37 infants had moderate neurological sequelae and only 1/37 infants developed bronchopulmonary dysplasia. No deleterious effects of the face chamber were seen. As the face chamber is a noninvasive and easily applied technique for CPAP therapy without hazards, it is proposed that it should be used at a still earlier stage of IRDS in order to lesson the need for IPPV treatment and to increase the neurological and lung functional quality of survival.

In a previous report face chamber treatment was suggested as a feasible method for establishing continuous positive airways pressure (CPAP) in infants with idiopathic respiratory distress syndrome (IRDS) (Ahlström, Jonson, and Svenningsen, 1973). Ever since Gregory *et al.* (1971) showed the beneficial effect of CPAP application in IRDS several devices have been described mainly to avoid the complications of tracheal tubes (Ackerman *et al.*, 1974; Agostino *et al.*, 1973; Barrie, 1973; Baum and Roberton, 1974; Caliumi-Pellegrini *et al.*, 1974; Chernick and Vidyasagar, 1972; Dunn, 1974; Fanaroff *et al.*, 1973; Harris, 1972; Kattwinkel *et al.*, 1973; Olinsky, MacMurray and Swyer, 1973; Rhodes and Hall, 1973; Salle *et al.*, 1974).

The present investigation was undertaken to develop further and evaluate the results of face chamber treatment with regard to the immediate effects on survival and to later pulmonary disease and neurological and developmental sequelae.

Received 21 April 1975.

Methods

Equipment and procedures. The face chamber equipment (FC 100, Siemens-Elema AB, Solna, Sweden) consists of the face chamber itself, the air-oxygen unit, and the intensive care crib (Fig.). The face chamber is an aluminium cylinder with a tight fitting, easily removable macrolone lid in one end and a sealing diaphragm in the other. The sealing diaphragm is a latex ring, available in 5 sizes, filled with styrene particles, and is disposable since latex is not very durable and cannot be adequately disinfected in the ward.

The air-oxygen unit consists of an air-oxygen mixer, a flow meter, a humidifier, and a vacuum unit. The

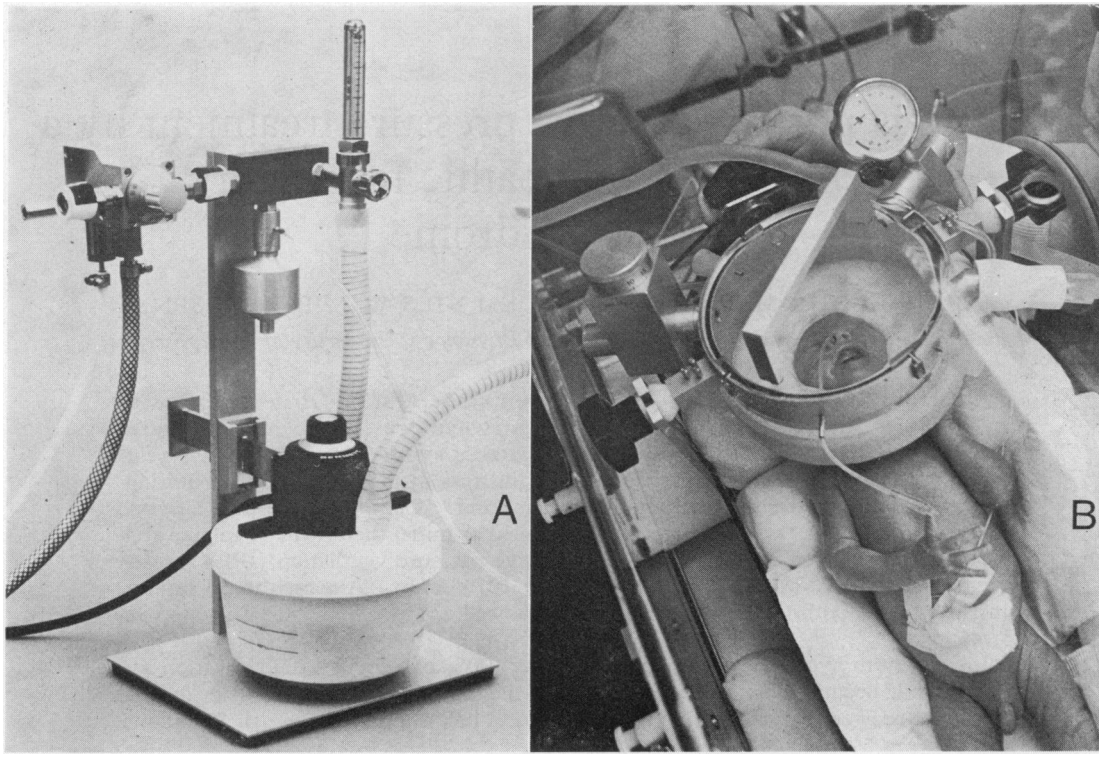

FIG.—*CPAP face chamber equipment (FC 100). (A) Air-oxygen unit. (B) Face chamber and intensive care crib.*

mixer allows adjustment of the oxygen concentration from 21%, i.e. pure air, to 100%, the flow meter adjustment of gas flow through the face chamber. Air conditioning is achieved by a Bennett cascade humidifier that can be adjusted to appropriate temperature of the outgoing gas checked by a thermometer. The vacuum unit is used to evacuate the interior of the latex ring to make it inflexible. The crib is a neonatal intensive care crib (Cooke, 1970) which can be tilted in the vertical plane, and has stages to support the face chamber.

The infant is placed in the crib in an incubator. The face chamber without the lid is placed on the stages over the infant's head. The latex ring of suitable size is gently adapted around the infant's face to fit snugly. In this position the ring is fixed by the evacuation of air from its interior. From the air-oxygen unit humidified air and oxygen mixture of suitable composition and temperature (about 35°C) is circulated through the face chamber with the lid attached. A flow rate of 12–15 l/min is recommended to eliminate any dead space. The pressure, monitored on the manometer, is adjusted by a pressure regulator valve attached at a port of the face chamber. The manometer has a safety outlet to avoid accidental pressures of more than 20 cm H_2O.

Treatment with the face chamber was started as soon as the diagnostic criteria were fulfilled (see below). An initial chamber pressure of 6 cm H_2O and an oxygen concentration of 40% was used in most cases. If the response was inadequate, i.e. if grunting did not stop, pressure was increased in steps of about 2 cm H_2O, and if arterial partial pressure of oxygen (Pao_2) was below 50 mmHg the oxygen concentration was raised usually in steps of 10%. When adequate clinical and laboratory (*p*H and blood gases) improvement was noted a slow tailing off of treatment was begun with a reduction of 5% oxygen concentration and/or 2 cm H_2O of chamber pressure every 4th hour. Changes of treatment were followed up by blood gas analyses. The crib was tilted from left lateral to right lateral position ½-hourly according to routine practice to promote airway drainage in patients with severe lung disease. A ½-hourly record was kept of breathing and heart frequency, grunting, cyanosis, oxygen concentration, and pressure in the chamber, and of other significant clinical data. The infants were given routine intensive care with regard to calorie intake, fluid and electrolyte balance, and acid-base balance.

Patients

From a total population of 19 500 newborn infants 63 were classified as having IRDS according to the

clinical criteria described below. In 45 infants ventilation was started as soon as the following criteria were fulfilled.

Clinical criteria. Grunting, tachypnoea (>60/min), intercostal retractions, and cyanosis observed within 8 hours of age, and a chest *x*-ray compatible with hyaline membrane disease (HMD). The *x*-ray patterns were scored according to Prod'hom *et al.* (1974) as HMD grade I (reticulogranular pattern) and HMD grade II (air bronchogram and opaque lung fields).

Laboratory criteria. Pa_{O_2} <100 mmHg during 100% oxygen breathing for 10 minutes (hyperoxia test).

The 45 infants requiring ventilatory support were divided into three groups. *Group A*, 30 infants treated with CPAP only (Table, A); *Group B*, 9 infants initially treated with CPAP but later requiring IPPV as well (Table, B); *Group C*, infants who received IPPV as initial treatment (Table, C); 3 of these (Cases 40, 41, 42) participated in a randomized comparison between CPAP and IPPV. (The randomized study had to be given up at an early stage because of the obvious advantages of CPAP (Ahlström *et al.*, 1973)). The other 3 infants (Cases 43, 44, 45) had apnoea on admission to the neonatal unit and required immediate intubation and IPPV.

Status before ventilatory treatment. Hyperoxia tests performed within one hour of the start of CPAP or IPPV showed a moderate to severe incapacity to hyperoxygenate as indicated by low Pa_{O_2} during oxygen breathing. Pa_{O_2} was 103 and 105 mmHg in 2 infants, between 50 and 100 mmHg in 33 infants, and below 50 mmHg in 10 infants. 8 of the latter 10 infants belong to groups B and C.

Chest *x*-ray findings before starting treatment showed HMD grade II in 14 out of 30 infants in group A and in 14 out of 15 infants in groups B and C. Infants belonging to groups B and C in general had lower birthweights, lower gestational age, and more pronounced signs of IRDS than those in group A.

CPAP and/or IPPV treatment. In infants of groups A and B initial treatment with CPAP was started between 3 and 31 hours (average 12 hours) after birth. Initial chamber pressure ranged between 4 and 10 (average 7) cm H_2O. Maximum pressure applied was between 4 and 10 cm H_2O in all infants except for Cases 2, 11, 32, and 36, who for some time required a pressure of 12 cm H_2O. The duration of CPAP treatment varied in group A between 7 and 110 hours (average 56 hours). In infants requiring IPPV as well, CPAP was interrupted after 5 to 24 hours (average 15 hours). During IPPV positive end-expiratory pressures between 3 and 6 cm H_2O were applied.

Weaning from IPPV was started with a 10-minute period hourly of CPAP applied via the endotracheal tube. When the infants had shown adequate breathing during such a period for 6 to 8 hours, they were extubated and put on CPAP via the face chamber for further weaning as indicated in B and C in the Table.

Follow-up studies. The following control examinations were performed in the follow-up study of the surviving infants. Physical, neurological, and developmental examinations were performed at 2, 4, 6, 10, and 14 months of age with ophthalmoscopy and auditory tests at 6 and 14 months of age. Sonoencephalogram with A-scanning evaluating the size of cerebral ventricles was performed at 2–6 and 10 months of age. Cerebrospinal fluid (CSF) examination at 1–2 months of age included evaluation of cytological signs of intracranial haemorrhage, i.e. the occurrence of siderophages which has been considered indicative of such intracranial haemorrhage (Blennow *et al.*, 1975; Sörnäs, 1967). Lung mechanics were studied in some infants according to the method described by Ahlström and Jonson (1974). Control chest *x*-rays were taken at 2, 6, and 10 months of age.

Results

Survival. In group A all 30 infants survived IRDS. One infant (Case 21) died later from septicaemia unrelated to the ventilatory treatment. No hyaline membranes were found at necropsy.

In group B 6 of 9 infants died, all having hyaline membranes at necropsy. 2 infants could not have survived because of tentorial rupture with intracranial haemorrhage after birth injury (Cases 31 and 39). Another 2 infants (Cases 34 and 37) had intracranial vein haemorrhage, and 1 infant (Case 38) had septicaemia and pneumonia (streptococcus type B) in addition to hyaline membranes. In 1 infant (Case 36) hyaline membranes and massive pulmonary atelectases were the only significant findings at necropsy.

In group C 1 infant died (Case 40) who had been treated primarily by IPPV, having been included in the randomized study (Ahlström *et al.*, 1973). This is the only infant who never received CPAP treatment, but she has nevertheless been included in this report of all infants observed during the period of this study.

If only infants treated with IPPV because of apnoea are considered (i.e. excluding Cases 40, 41, 42) 36 of 42 survived IRDS, i.e. an overall survival rate of 86%. If we take the total number of infants and ignore the cause of death and treatment given, the survival rate would be 37 out of 45, or 82%.

Complications during treatment. Pneumothorax occurred in 3 out of 30 infants (10%) during CPAP treatment. In another of these 30 infants pneumothorax was observed on chest *x*-ray before CPAP was applied. All these 4 infants had success-

TABLE

Clinical and laboratory data of 45 infants

Case no. and sex	Birthweight (g)	Gestational age (w)	Hyperoxia test (PaO_2 mmHg) 40%–100% O_2	Chest x-ray (HMD)*	Treatment	
					Age at start (h)	Duration (h)
Group A: 30 infants treated with CPAP face chamber only						
1 F	1860	34	19–65	I	CPAP 5	13
2 „	1830	34	31–69	II	„ 12	15
3 „	2250	35	38–67	I	„ 5	7
4 „	2370	33	36–49	II	„ 24	97
5 M	2500	35	35–65	II	„ 11	44
6 „	2500	34	53–98	I	„ 12	27
7 „	2870	36	49–76	I	„ 7	31
8 „	2300	34	35–65	II	„ 11	44
9 „	2420	35	26–53	I	„ 10	20
10 „	1900	33	35–58	I	„ 8	37
11 „	2210	35	21–60	II	„ 13	110
12 „	1840	32	38–85	II	„ 18	73
13 „	1610	32	22–75	II	„ 11	68
14 „	1900	34	35–105	I	„ 14	98
15 „	2300	35	35–85	I	„ 16	68
16 F	1810	32	35–65	II	„ 16	65
17 M	2930	36	48–100	I	„ 10	23
18 M	2450	36	30–57	II	„ 8	85
19 F	3000	36	28–65	II	„ 10	46
20 M	3550	36	36–91	II	„ 24	50
21 „	1710	32	33–66	II	„ 8	73
22 „	2780	35	35–87	II	„ 7	94
23 „	1700	33	37–78	I	„ 31	70
24 „	3510	36	40–103	I	„ 30	75
25 „	2850	35	27–97	I	„ 12	86
26 „	2450	35	45–87	I	„ 7	88
27 F	3800	36	42–97	I	„ 6	12
28 „	1500	33	40–95	I	„ 10	54
29 „	2200	35	33–47	II	„ 8	69
30 M	1890	33	40–100	I	„ 6	48
Mean	2326	34	35–77		12	56
SD	554	1·3	8 18		7	29
Group B: 9 infants treated with IPPV after initial CPAP						
31 M	1160	29	42–52	I	CPAP 5	24
					IPPV 29	7
32 M	1930	32	18–26	II	CPAP 3	5
					IPPV 8	96
					CPAP 104	44
33 F	1500	31	25–60	II	CPAP 6	18
					IPPV 24	196
					CPAP 220	48
34 M	2580	35	35–87	II	CPAP 22	20
					IPPV 42	8
35 F	1020	29	18–40	II	CPAP 6	3
					IPPV 9	108
					CPAP 117	60
36 M	2990	35	24–75	II	CPAP 11	30
					IPPV 41	13
37 „	1340	30	35–85	II	CPAP 18	24
					IPPV 42	30
38 „	2130	35	16–36	II	CPAP 12	5
					IPPV 17	18
39 „	1720	33	20–48	II	CPAP 14	6
					IPPV 20	11
Mean	1818	32	26–56		11	
SD	659	2·5	9 21		6	
Group C: 6 infants treated initially with IPPV but weaned off via CPAP face chamber						
40 F	1550	30	25–49	II	IPPV 11	25
41 „	1290	29	35–70	II	IPPV 18	66
					CPAP 84	52
42 „	2100	33	28–55	II	IPPV 16	102
					CPAP 118	48
43 M	1500	30	17–30	II	IPPV 6	6w
					CPAP 6 w	8w
44 „	1800	33	24–43	II	IPPV 6	120
					CPAP 126	72
45 „	2650	35	21–47	II	IPPV 16	144
					CPAP 160	48
Mean	1815	31	25–49			
SD	494	2·3	6 13			

*HMD I and HMD II (see patients). †0, normal; +, pathological; – not examined. HM, hyaline membranes;

with IRDS requiring assisted ventilation

	Neonatal period		Follow-up study	
Maximum CPAP (cmH_2O)	CSF cyto-logy†	Other neonatal complications	Age (m)	Abnormalities
5	0	—	14	None
12	0	—	14	,,
6	0	—	14	,,
10	0	Congenital hypothyroidism	14	,,
10	0	Pneumothorax *during* CPAP	14	,,
6	0	—	14	,,
6	0	—	14	,,
8	0	Pneumothorax *before* CPAP	14	,,
4	0	—	14	,,
6	0	—	14	,,
12	+	Pneumothorax *during* CPAP	14	,,
10	0	—	14	,,
8	0	—	12	,,
6	0	—	12	,,
8	+	Pseudomonas meningitis at 5 d of age	12	Spastic diplegia
10	0	—	12	None
4	0	—	12	,,
10	0	—	10	,,
8	0	—	10	,,
6	0	—	10	,,
8	+	Sepsis (*Staph. aureus*) 3 d after ending CPAP	Died on day 7 (sepsis + general haemorrhages)	
10	0	Pneumothorax *during CPAP*	10	None
6	+	Rh-immunization; 3 exchange transfusions	10	,,
5	0	—	10	,,
6	0	—	6	,,
6	0	—	6	,,
5	0	—	6	,,
6	+	—	6	,,
10	0	—	6	,,
6	0	—	6	
6	+	IPPV because of apnoea	Died day 2 (HM and ICH from tentorial rupture)	
12	+ +	Coliform meningitis with apnoea requiring IPPV	14	Moderate psychomotor retardation
5				
10	+	IPPV because of $PaCO_2$ >90mmHg during CPAP	12	None
4		Pneumothorax *during* IPPV		
6	+	Apnoeic attacks and convulsions	Died day 2 (HM and ICH from terminal vein)	
8	0	IPPV because of $PaCO_2$ >90 mmHg during CPAP	6	None
4				
12	—	Before CPAP apnoea and asystole; grunting during 10–12 cmH_2O CPAP	Died day 2 (HM and massive atelectases in both lungs)	
6	+	IPPV because of apnoea, pneumothorax *during* IPPV	Died day 4 (HM and ICH from terminal vein)	
8	—	IPPV because of $PaCO_2$ above 90mmHg during CPAP	Died day 2 (HM and sepsis (streptococus type B))	
10	—	Breech delivery, twin II; apnoeic attacks during CPAP; asystole during CPAP	Died day 2 (HM and ICH from tentorial rupture)	
	—	Severe hypoxaemia despite IPPV	Died day 3 (HM only)	
5	0	0	18	None
5	0	Pneumothorax and pneumopericardium *during* IPPV	18	None
6	0	Pneumothorax *during* IPPV	10	Bronchitis twice
4	+ +	Bronchopulmonary dysplasia Apnoea requiring IPPV on admission to neonatal unit	8	Slight psychomotor retardation
4	+	Apnoea requiring IPPV on admission to neonatal unit	8	None

ICH, intracranial haemorrhage.

ful pleural drainage from 12 to 24 hours after completed CPAP treatment. In 4 out of 15 infants pneumothorax occurred during IPPV treatment (27%). Pneumothorax was not considered an indication to change CPAP or IPPV treatment in any case.

Hypercapnia was observed during CPAP only in 2 infants (Cases 33 and 35.) Minor complications with skin maceration causing small facial scarring was observed in 2 infants. Gastric distension was no problem in infants on pressures of 10 cm H_2O or below. In 2 infants on higher pressures, gastric distension required interruption of gastric feeding. In all other infants feeding through a nasogastric tube could be continued during the CPAP treatment. Between meals the tube was left open *outside* the face chamber.

Two infants (Cases 15 and 32) developed meningitis requiring intubation and IPPV, in one because of apnoeic attacks. In both infants premature rupture of fetal membranes was a possible causative factor.

Follow-up examinations. Abnormal findings at the latest follow-up examination are indicated individually in the Table A-C. In 8 of 37 survivors CSF examination showed cytological signs of intracranial haemorrhage (ICH). In 3 of these 8 infants neurological sequelae were observed: 2 infants with neonatal meningitis (Cases 15 and 32) had spastic diplegia and moderate psychomotor retardation, respectively, at one year of age, and one infant with severe apnoeic attacks requiring initial IPPV treatment (Case 44) showed slight psychomotor retardation at 8 months of age.

In group A 28 of 29 infants, including 4 infants with CSF cytological signs of ICH, were normal at neurological and developmental follow-up examinations including ophthalmological and auditory tests as well as skull transillumination and sonoencephalogram. In groups B and C 4 of 8 infants showed CSF cytological signs of ICH and 2 of these infants showed moderate and slight cerebral sequelae at follow-up.

None of the 29 infants treated with CPAP by face chamber only (group A) showed any clinical signs of pulmonary abnormalities at follow-up, chest *x*-ray was normal in all these infants. Pulmonary function tests performed at ½–3 and 4–10 months of age in 9 infants in group A showed no abnormality in contrast to the abnormal breathing mechanics of obstructive and restrictive type found in infants after intubation and IPPV treatment previously reported (Ahlström, 1975). In the present study one infant in group C (Case 43) developed severe bronchopulmonary dysplasia with recurrent episodes of bronchitis.

Discussion

The survival rate of the infants reported here must, of course, be evaluated with respect to the severity of IRDS of the infants treated. During the period of this study IRDS of a less severe degree—Pao_2 considerably higher than 100 mmHg at hyperoxia test—was diagnosed in 18 infants who were treated with incubator care and supplemental oxygen only. Thus the 45 infants of the present study represent a high risk group of IRDS. The overall mortality of 8/45 or 18% compares favourably with results obtained by others (Ballard *et al.*, 1973; Baum and Roberton, 1974; Chernick, 1973a, b; Dinwiddie *et al.*, 1974; Reynolds and Taghizadeh, 1974). However, for many reasons such comparisons are very difficult. It appears to be more fruitful to analyse if and how the infants who did not survive might have been saved. All 8 non-survivors had a necropsy performed.

Case 21 recovered from IRDS but died later from septicaemia with no residual signs of IRDS at necropsy. This baby should therefore be included in the group of infants who recovered. Cases 31 and 39 would probably not have survived due to the severe intracranial birth injury.

Three infants (Cases 34, 37, and 38) may have died from IRDS, concomitant disease or both. In Case 38 the occurrence of streptococcus group B in blood cultures and in post-mortem lung examination must be considered a major contributing factor. These 3 infants, and Cases 36 and 40 who probably died from IRDS, might have been saved if we could further improve treatment; consequently they deserve some further comments.

Case 40 who did not receive CPAP treatment should according to our present practice have been treated with the face chamber at a much earlier stage of the IRDS. In Case 36 technical problems with an early prototype of the equipment caused an initial delay in increase of pressure applied. Although the pressure some hours later was increased to 10–12 cm H_2O the infant continued to grunt for several hours. Nowadays we regard grunting as a sign of inadequate treatment that must lead promptly to more adequate treatment and if necessary to IPPV.

Cases 34 and 37 had obvious clinical symptoms of IRDS many hours before the laboratory criteria for initiating CPAP, as used in the present study (see Methods), were met. The delay in treatment of Case 38 was not so long (12 hours as compared to 22 and 18 hours in Cases 34 and 37) but his clinical

state had deteriorated rapidly when the laboratory result (hyperoxia test 36 mmHg) was available 15 minutes after blood sampling. As mentioned above, the streptococcus group B septicaemia probably contributed.

Further improvement of survival can possibly only be accomplished by application of CPAP at a very early stage of IRDS, as has been proposed also by other investigators (Chernick, 1973a, b; Dunn, 1974; Omer, Robson, and Neligan, 1974). This must imply CPAP treatment of infants with only moderate IRDS, who might survive without support of ventilation. To justify such a change of policy the risks of treatment of such infants would have to be very small compared to possible benefits. Therefore, complications, especially those in 'low risk' infants must be carefully evaluated. In the present investigation no infant with an *x*-ray classification of HMD grade I developed pneumothorax. 3 infants with HMD grade II got pneumothorax during face chamber treatment (and 1 before!) and 4 infants during IPPV. In our experience pneumothorax was not a complication significantly contributing to the outcome, as 7 of 8 infants with pneumothorax survived. In addition, the occurrence of pneumothorax during CPAP treatment in the present study (10%) is not higher than pneumothorax occurring naturally in hyaline membrane disease, being 8% according to Cohen *et al.* (1973). In contrast to Baum and Roberton (1974) and others (Pagtakhan, Berg, and Chernick, 1974) we are also not confident that pneumothorax should be regarded as a common complication to CPAP if applied without endotracheal tubes. It may rather be caused by the disease itself.

Intracranial haemorrhage has previously been reported in connexion with continuous distending pressure leading to hydrocephalus (Vert, Andre, and Sibout, 1973). This complication was not observed in any of the infants treated with the face chamber in the present investigation. Nor were there any developmental or neurological sequelae attributable to CPAP treatment.

No deleterious effect of CPAP applied with the face chamber was found in any of the infants after several months of observation with repeated physical examinations including chest *x*-ray. This has been further confirmed by repeated pulmonary function tests in several of these infants (Ahlström, 1975). Case 43, who developed bronchopulmonary dysplasia, required 6 weeks of IPPV treatment because of severe disease.

All techniques providing a continuous distending transpulmonary pressure have some disadvantages, as pointed out by other authors (Chernick, 1973a, b). Nasal catheters for CPAP application have the advantage that the sealing to the airway can be accomplished in a cheap and easy way (Caliumi-Pellegrini *et al.*, 1974; Kattwinkel *et al.*, 1973). However, inserting tubes into the nares causes mucosal irritation and mucopurulent secretions with risk of infection of lower airways and yet does not avoid the risk of pneumothorax, even if taping of the mouth seems unnecessary (Caliumi-Pellegrini *et al.*, 1974). When the infant cries the treatment is interrupted while the infant is breathing through the mouth. In addition, reflexes from the nasal mucosa impose the risk of bronchoconstriction (Holmgren, 1866; James and de Burgh Daly, 1969). Whether or not nasal cannulas of the kind used for CPAP may cause such deformities of the nares as have been reported for nasotracheal tubes (Jung and Thomas, 1974; Rasche and Kuhns, 1972) must also be considered.

The only complications definitely caused by the face chamber are small scars in 2 infants. Such decubital facial scars are now avoided since 5 different sizes of latex rings have become available.

In conclusion the face chamber therapy itself has not involved any significant medical risk. We think the face chamber has the following advantages over other techniques for application of CPAP. It is easily tightened around the face. The seal is brought about with only the slightest pressure on the skin without using any sealants and without pressing on vessels or organs, whereas neck collar sealings for head boxes cause pressure on the neck veins. The ears are outside the chamber, thereby eliminating the risks of inadvertent noise levels (Svenningsen and Blennow, 1973). The face is free for inspection through the lid, which can be removed instantly when access to upper airways is needed. However, in our experience this is seldom required as abnormal secretions from nares or mouth are avoided with this noninvasive technique which allows optimal warming and humidification of air-oxygen mixtures for the mucosa of the nares, mouth, and lower airways. The infant is fed through a gastric tube leading out from the face chamber. No incidence of gastric distension has been observed as long as the pressure is not above 10 cm H_2O. The infant can be nursed, changing position by tilting the crib, and *x*-rays can be taken without interruption of CPAP treatment. This is a major advantage decreasing the risk of deterioration during such procedures. Finally, gas tubings are reduced to a minimum. The crib with infant, face chamber, tubings for gas supply and feeding, valves, manometer, and safety ports are moved as a single piece when the

crib is moved or tilted. This construction also allows transportation of the infant with uninterrupted CPAP treatment.

At the present time we apply the following procedures in the treatment of neonates with IRDS: when clinical signs of IRDS occur control of temperature and acid-base balance, the hyperoxia test, and chest *x*-ray are performed followed by CPAP treatment with the face chamber started as soon as *x*-ray examination has excluded other causes of pulmonary insufficiency. The hyperoxia test is not used as a criterion for CPAP application but only for evaluation of the severity of IRDS before start of treatment, which is usually before 6 hours of age and with an initial CPAP pressure of 4 cm H_2O.

Such management of neonates with clinical signs of IRDS should improve the outcome without causing any harm to infants who might survive without ventilatory support.

We thank our colleagues and the nurses in the neonatal unit for their careful assistance during this study. The study was supported with grants from the Prenatal Research Foundation (Expressens Prenatalforskningsnämnd), Swedish Medical Research Council (grant 14X-2872), and the Swedish National Association against Heart and Chest Disease.

References

Ackerman, B. D., Stein, M. P., Sommer, J. S., and Schumacher, M. (1974). Continuous positive airway pressure applied by means of a tight-fitting face-mask. *Journal of Pediatrics*, **85**, 408.

Agostino, R., Orzalesi, M., Nodari, S., Mendicini, M., Conca, L., Savignoni, P. G., Picece-Bucci, S., Calliumi, G., and Bucci, G. (1973). Continuous positive airway pressure (CPAP) by nasal cannula in the respiratory distress syndrome (RDS) of the newborn. *Pediatric Research*, **7**, 50.

Ahlström, H. (1975). Pulmonary mechanics in infants surviving severe neonatal respiratory insufficiency. *Acta Paediatrica Scandinavica*, **64**, 69.

Ahlström, H., and Jonson, B. (1974). Pulmonary mechanics in infants. Methodological aspects. *Scandinavian Journal of Respiratory Diseases*, **55**, 129.

Ahlström, H., Jonson, B., and Svenningsen, N. W. (1973). Continuous positive airway pressure with a face chamber in early treatment of idiopathic respiratory distress syndrome. *Acta Paediatrica Scandinavica*, **62**, 433.

Ballard, R. A., Kraybill, E. N., Hernandez, J., Renfield, M. L., and Blakenship, W. J. (1973). Idiopathic respiratory distress syndrome. Treatment with continuous negative-pressure ventilation. *American Journal of Diseases of Children*, **125**, 676.

Barrie, H. (1973). Continuous positive airway pressure for respiratory distress syndrome. *Lancet*, **2**, 851.

Baum, J. D., and Roberton, N. R. C. (1974). Distending pressure in infants with respiratory distress syndrome. *Archives of Disease in Childhood*, **49**, 771.

Blennow, G., Aronson, A. S., Svenningsen, N. W., and Sörnäs, R. (1975). Cerebrospinal fluid cytology in normal and asphyxiated neonates. (Submitted for publication).

Caliumi-Pellegrini, G., Agostino, R., Orzalesi, M., Nodari, S., Marzetti, G., Savignoni, P. G., and Bucci, G. (1974). Twin nasal cannula for administration of continuous positive airway pressure to newborn infants. *Archives of Disease in Childhood*, **49**, 228.

Chernick, V. (1973a). Continuous distending pressure in hyaline membrane disease: of devices, disadvantages and a daring study. *Pediatrics*, **52**, 114.

Chernick, V. (1973b). Hyaline-membrane disease: therapy with constant lung-distending pressure. *New England Journal of Medicine*, **289**, 302.

Chernick, V., and Vidyasagar, D. (1972). Continuous negative chest wall pressure in hyaline membrane disease: one year experience. *Pediatrics*, **49**, 753.

Cohen, D., Cochran, W., Griscom, T., and Harris, G. B. C. (1973). Naturally occurring pneumothorax in hyaline membrane disease. *Pediatric Research*, **7**, 400.

Cooke, R. (1970). Practical problems associated with artificial ventilation in the newborn infant. *Biology of the Neonate*, **16**, 60.

Dinwiddie, R., Mellor, D. H., Donaldson, S. H. C., Tunstall, M. E., and Russell, G. (1974). Quality of survival after artificial ventilation of the newborn. *Archives of Disease in Childhood*, **49**, 703.

Dunn, P. M. (1974). Continuous positive airway pressure (CPAP) using the Gregory box. *Proceedings of the Royal Society of Medicine*, **67**, 245.

Fanaroff, A. A., Cha, C. C., Sosa, R., Crumrine, R. S., and Klaus, M. H. (1973). Controlled trial of continuous negative external pressure in the treatment of severe respiratory distress syndrome. *Journal of Pediatrics*, **82**, 921.

Gregory, G. A., Kitterman, J. A., Phibbs, R. H., Tooley, W. H., and Hamilton, W. K. (1971). Treatment of the idiopathic respiratory-distress syndrome with continuous positive airway pressure. *New England Journal of Medicine*, **284**, 1333.

Harris, T. R. (1972). Continuous positive airway pressure applied by face mask. *Pediatric Research*, **6**, 410.

Holmgren, F. (1866). Undersökningar öfver verkan af chloroform på kaniner. *Procceedings of Uppsala Society of Medicine*, **2**, 134.

James, J. E. A., and Daly, M. de B. (1969). Nasal reflexes. *Proceedings of the Royal Society of Medicine*, **62**, 1287.

Jung, A. L., and Thomas, G. K. (1974). Stricture of the nasal vestibule. A complication of nasotracheal intubation in newborn infants. *Journal of Pediatrics*, **85**, 412.

Kattwinkel, J., Fleming, D., Cha, C. C., Fanaroff, A. A., and Klaus, M. H. (1973). A device for administration of continuous positive airway pressure by the nasal route. *Pediatrics*, **52**, 131.

Olinsky, A., MacMurray, S. B., and Swyer, P. R. (1973). Continuous pressure breathing in RDS: comparative trial of 3 methods. *Pediatric Research*, **7**, 430.

Omer, M. I. A., Robson, E., and Neligan, G. A. (1974). Can initial resuscitation of preterm babies reduce the death rate from hyaline membrane disease? *Archives of Disease in Childhood*, **49**, 219.

Pagtakhan, R. D., Berg, T., and Chernick, V. (1974). Changing prevalence of alveolar rupture and bronchopulmonary dysplasia in hyaline membrane disease (HMD). *Journal of Pediatrics*, **84**, 919.

Prod'hom, L. S., Choffat, J. M., Frenck, N., Mazoumi, M., Relier, J. P., and Torrado, A. (1974). Care of the seriously ill neonate with hyaline membrane disease and with sepsis (sclerema neonatorum). *Pediatrics*, **53**, 170.

Rasche, R. F. H., and Kuhns, L. R. (1972). Histopathological changes in airway mucosa of infants after endotracheal intubation. *Pediatrics*, **50**, 632.

Reynolds, E. O. R., and Taghizadeh, A. (1974). Improved prognosis of infants mechanically ventilated for hyaline membrane disease. *Archives of Disease in Childhood*, **49**, 505.

Rhodes, P. G., and Hall, R. T. (1973). Continuous positive airway pressure delivered by face mask in infants with the idiopathic respiratory distress syndrome: a controlled study. *Pediatrics*, **52**, 1.

Salle, B., Champin, J.-P., Desebbe, C., and Andre, M. (1974). Le traitement des formes sevères de detresse respiratoire idiopathique du nouveau-né. *Archives Françaises de Pédiatrie*, **31**, 565.

Sörnäs, R. (1967). A new method for the cytological examination of the cerebrospinal fluid. *Journal of Neurology, Neurosurgery and Psychiatry*, **30**, 568.
Svenningsen, N. W., and Blennow, G. (1973). Continuous positive airway pressure and noise level. *Lancet*, **2**, 623.
Vert, P., Andre, M., and Sibout, M. (1973). Continuous positive airway pressure and hydrocephalus. *Lancet*, **2**, 319.

Correspondence to Dr. N. W. Svenningsen, Department of Paediatrics, University Hospital, S–22185 Lund, Sweden.