Archives of Disease in Childhood, 1981, **56**, 326-330

Effect of tracheal suction on oxygenation, circulation, and lung mechanics in newborn infants

G SIMBRUNER, H CORADELLO, M FODOR, L HAVELEC, G LUBEC, AND A POLLAK

University Children's Hospital, Division of Neonatology, Vienna, and Institute of Medical Statistics and Documentation, Vienna

SUMMARY Transcutaneous Po_2, heart rate, and aortic blood pressure were measured in 10 mechanically-ventilated newborn infants to assess the degree and course of hypoxaemia, and to monitor the cardiovascular and respiratory changes during tracheal toilet. Five infants weighed <1250 (mean 994) g, and 5 infants weighed >1750 (mean 2216) g. During tracheal suction the $TcPo_2$ fell from 68 ± 27 ($\bar{x}$ ± SD) to 43 ± 23 mmHg, and the heart rate from 144 ± 8 to 123 ± 25 beats/minute, but the blood pressure increased from 44 ± 24 to 49 ± 24 mmHg. Hypoxaemia ($TcPo_2$ <50 mmHg) occurred in 7 of 8 initially well-oxygenated infants when suctioned. The decrease in $TcPo_2$ was similar for both groups of infants. It was greater in infants with controlled ventilation and an F_IO_2 ≥0·8 than in infants with intermittent mandatory ventilation and an F_IO_2 <0·8. The $TcPo_2$ fall correlated well with the $TcPo_2$ during the control period but not during the time that the infants were disconnected from the respirator. A critical re-evaluation of routine tracheal toilet is needed.

Newborn infants who are intubated in order to be ventilated by a respirator have to be routinely suctioned to free them from lung secretions and to prevent the endotracheal tube from becoming blocked. For this tracheal toilet, respirators that have a closed air supply system have to be disconnected from the patient. This interruption of oxygen supply and ventilation often leads to transient hypoxaemia, bradycardia,[1 2] arrhythmias,[3] decreased lung compliance,[4] and negative intrapulmonary pressure.[5] Even death after tracheal suction has been reported.[6 7] But the extent and course of hypoxaemia have not been studied, and nor have the cardiovascular and respiratory changes that take place during tracheal suction. We therefore investigated the effects of tracheal toilet on transcutaneous Po_2 ($TcPo_2$), heart rate, and blood pressure in newborn infants in an intensive care nursery. We paid special attention to the cardiovascular response of each infant to the sudden hypoxia induced by tracheal suction.

Material and methods

We studied 10 newborn infants with respiratory distress taken at random from our intensive care nursery. Each was being ventilated via a tracheal tube with a Dräger Spiromat* or a Bourns LS 104† respirator. Each infant was placed in one of two groups according to his weight on the day of the study. There were 5 infants in each group: group A <1250 g, group B >1750 g. The groups differed significantly (P<0·01) in gestational age. Details of the 10 infants, the duration that each was disconnected from the respirator, and the duration of bag ventilation are shown in Table 1. One infant in group A and 3 in group B were ventilated by intermittent mandatory ventilation, but the others received controlled ventilation. Three infants in group A and 2 in group B needed inspired oxygen concentrations of at least 80%. Both groups were similar in respect to the severity of the disease, the duration that they were disconnected from the respirator, and the period of bag ventilation.

Tracheal toilet was performed routinely every 2 hours by nurses. Some nurses were experienced in this procedure and others were not, and the time that it took to suction the infants varied. The infants did not have pulmonary haemorrhage or purulent pneumonia, and only small amounts of secretion were obtained. No additional oxygen was contained within the incubator and the entire respirator circuit was disconnected from the endotracheal tube, so that if the baby did breathe during suctioning he inhaled room air.

The respirator was disconnected, 0·5 ml 0·9% saline instilled, and the respirator reconnected for a few breaths only. Then the infant was suctioned via

*Drägerwerk AG., Lübeck, West Germany.
†Bourns, Inc., life systems division, Riverside, California, USA.

Table 1 *Details of the 10 infants*

Cases	*Weight at study (g)*	*Gestational age (weeks)*	*Age at study* (days)*	*Diagnosis*	*F_{IO_2}* at study*	*Type of ventilation*	*Duration of disconnection* (seconds)*	*Duration of bag ventilation* (seconds)*
Group A								
1	1020	28	2	RDS	1·00	Controlled	22	18
2	980	27	0·6	Aspiration	0·81	IMV	41	14
3	1000	27	0·5	RDS	0·80	Controlled	90	10
4	1000	30	0·8	Apnoea	0·45	Controlled	60	27
5	970	28	11	Aspiration R-L shunt	0·76	Controlled	55	88
Mean±1 SD	994±19	28±1·2	2·8†		0·76±0·2		56±22	35±32
Group B								
6	2040	34	5	RDS	0·39	Controlled	57	22
7	2150	36	2	RDS	0·86	IMV	46	40
8	2700	40	0·9	Aspiration	0·84	Controlled	44	26
9	1900	33	22	Congenital heart disease	0·65	IMV	35	17
10	2290	34	3	RDS	0·53	IMV	24	13
Mean±1 SD	2216±306	35·4±2·9	3†		0·64±0·24		41±12	24±11
Total mean±SD	1605±675	31·7±4·4	1·8†(0·6–22)		0·70±0·22		47·5±19·7	28±23

*Mean values of 3 studies in one patient, †median.
RDS = respiratory distress syndrome, F_{IO_2} = flow of inspired oxygen, IMV = intermittent mandatory ventilation.

the open endotracheal tube and ventilated by means of an Ambu-bag supplied with oxygen. For suctioning a negative pressure of about 200 cm water and catheters of 1·0–1·5 mm (inside diameter) and 1·5–2·0 mm (outside diameter) were used. Three measurements were obtained for each infant with a mean time of 11·8 hours (range 4 to 36). When the infant was in a clinically stable condition we recorded the $TcPo_2$ and the aortic blood pressure, and then we did the same during the tracheal toilet, and during the 5-minute period after bag ventilation. The $TcPo_2$ was measured at a skin temperature of 44°C with a Dräger-Hellige Oxypart with the electrode fixed to the right side of the chest. The heart rate was determined either from an electrocardiogram or from blood pressure tracing. The pressures in the aorta and tracheal tube were measured with pressure transducers (Statham Db 23). The tidal volume was either read from the volume-constant respirator or was determined by pneumotachography (using a Fleisch pneumotachograph 00, a differential pressure transducer (Statham P 15), and electronic integrating circuits). A Beckman R 611 polygraph registered all variables simultaneously. We checked the calibrations and scale linearity before and after each measurement.

The time immediately before the respirator was disconnected, the time at the end of suctioning, and at 1, 2, and 5 minutes after bag ventilation were chosen for the analysis. The time that the respirator was disconnected was chosen as representing the time of suctioning because the precise time could be read from the tracings. Heart rate and blood pressure were calculated from a 10-second record. The paired Student's *t* test was used for statistical analysis.

Results

The average changes of $TcPo_2$, heart rate, and blood pressure in the 10 infants are shown in Table 2. During tracheal suction the $TcPo_2$ and heart rate decreased but the blood pressure increased. Bag ventilation with pure oxygen reversed all these changes. Five minutes after bag ventilation both the $TcPo_2$ and blood pressure were higher than during the control period. Each patient reacted to tracheal toilet in a similar manner in all 3 studies done on him.

The course of the $TcPo_2$ in each patient is shown in

Table 2 *Transcutaneous Po_2, heart rate, blood pressure, and dynamic respiratory compliance during tracheal toilet (mean±1 SD) in 10 infants*

	Control	*Suctioning*	*Bag ventilation*	*Minutes after bag ventilation*		
				1	*2*	*5*
$TcPo_2$ (mmHg)	68±27	43±23*	56±27	64±34	69±38	77±34
Heart rate (beats/min)	144±8	123±24*	145±16	148±11	148±8	143±8
Blood pressure (mmHg)	44±24	49±24*	46±24	46±24	45±24	47±24
Compliance (ml/cm water)	0·9±0·5	NA	NA	0·9±0·4	0·8±0·3	0·8±0·4

NA = not applicable. *Statistically significant differences ($P<0·05$) compared with control values.
Conversion: traditional to SI units—7·5 mmHg ≈ 1kPa, 98·07 cmH_2O ≈ 1Pa.

Fig. 1. During the control period 8 infants had a $TcPo_2$ $\geqslant$50 mmHg and 2 infants a $TcPo_2$ <50 mmHg. During the suctioning period all 8 initially well-oxygenated infants showed a similar decrease in the $TcPo_2$ (27·8 $\pm$ 8·3 mmHg; mean $\pm$ SD) (range 17·0–43·6). Seven of these 8 infants became hypoxaemic ($TcPo_2$ <50 mmHg) at least once during tracheal toilet (3 infants once, 2 infants twice, and 2 infants three times). Thus in 8 infants, initially well oxygenated, hypoxaemia took place 13 times in 24 studies of tracheal toilet. Their $TcPo_2$ rise after suctioning and bag ventilation varied considerably. Some infants took 1 to 2 minutes after bag ventilation to reach control values. The other 2 infants, who had been hypoxaemic from the beginning, showed little change in $TcPo_2$ values.

The amount that $TcPo_2$ decreased was not related to body weight or gestational age and was similar for groups A and B (27·6 v 22·4 mmHg). The decrease in $TcPo_2$ was greater in the 6 infants with controlled ventilation than in the 4 infants with intermittent mandatory ventilation (28·2 v 14·4 mmHg). It was also greater in the 5 infants with F_IO_2 $\geqslant$0·8 than in the 5 infants with F_IO_2 <0·8 (30·1 v 19·9 mmHg). If the 2 newborn infants with initial hypoxaemia and cardiovascular disease were excluded the results would not be greatly different. The $TcPo_2$ decrease correlated well with the $TcPo_2$ levels during the control period ($r = 0·60$, $P<0·01$), but not with the duration of disconnection from the respirator ($r = 0·18$, NS). The $TcPo_2$ fall was independent of the disconnection time, irrespective of the method of ventilation, weight, gestational age, or F_IO_2 levels in these patients (Fig. 2). A fairly large $TcPo_2$ drop took

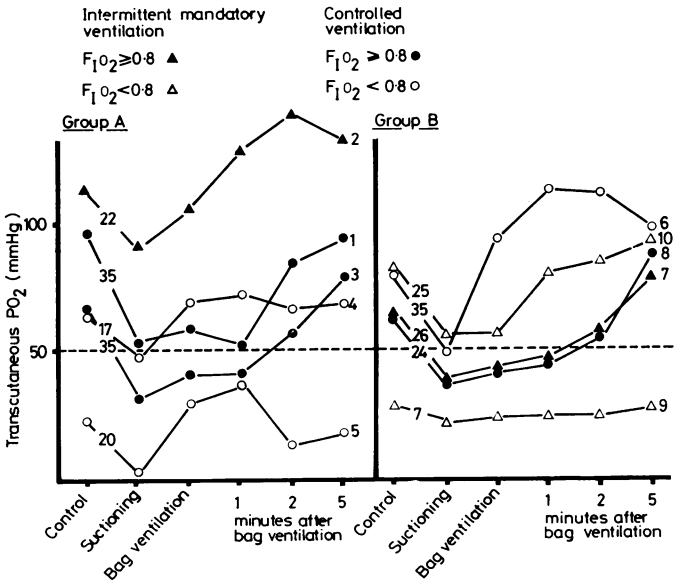

Fig. 1 *$TcPo_2$ changes in both groups of infants. Each symbol represents a mean value of 3 studies in one infant. $TcPo_2$ values below dotted line indicate hypoxaemia. The lowest $TcPo_2$ values are given for the suctioning period. The numbers on left are the amounts by which $TcPo_2$ was decreased (in mmHg), those on right are case numbers.*

Conversion: traditional units to SI— 7·5 mmHg $\approx$ 1 kPa.

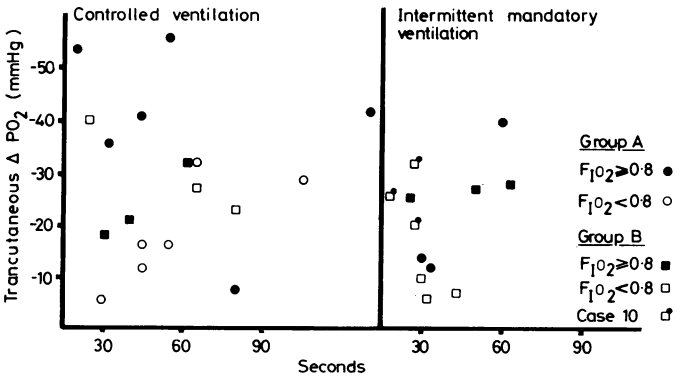

Fig. 2 *The period that infants were disconnected from the respirator and the decrease in $TcPo_2$ in the two groups of infants. Each symbol represents the result of one study in one patient. Case 10 had a large decrease in $TcPo_2$ despite the shortest suctioning time. (Heart rate and blood pressure changes are shown in Fig. 3).*

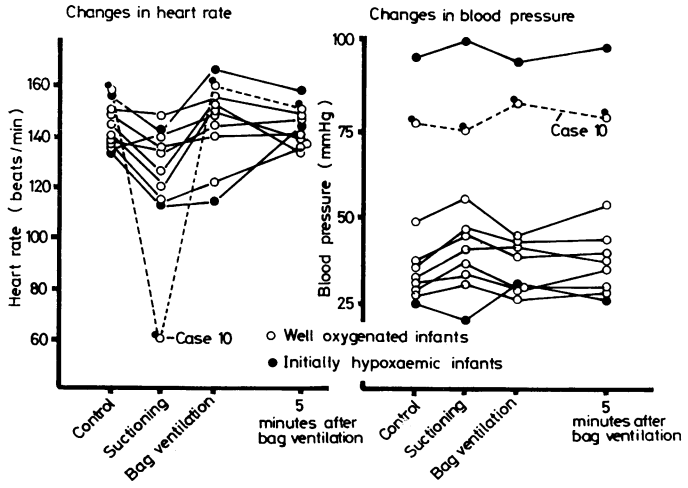

Fig. 3 *Changes in heart rate and blood pressure during tracheal toilet in 8 babies who had initially been well oxygenated and 2 babies who had initially been hypoxaemic. Each symbol represents the mean value of 3 studies in one infant.*

place in one infant (Case 10) who was disconnected from the respirator for the shortest time. It is of note that the $TcPo_2$ did not always reach its lowest value ($TcPo_2$ min) during suctioning. The $TcPo_2$ min was recorded 13 times during and 17 times after the suctioning period.

In each patient the heart rate and the blood pressure (Fig. 3) reacted uniformly to tracheal suction. The heart rate fell in 9 and the blood pressure rose in 8 of the 10 infants. Two infants showed a fall in blood pressure each time they were suctioned; one was initially hypoxaemic, the other (Case 10) developed severe bradycardia when suctioned. While the heart rate returned to control values, the blood pressure in 8 infants was still slightly raised 5 minutes after bag ventilation. The aortic pulse pressure remained unchanged during the entire study period in 6 infants in whom we had obtained an undampened blood pressure tracing. In one infant we observed cardiac arrhythmias.

Discussion

It is common for hypoxaemia to occur during tracheal suctioning and the $TcPo_2$ can fall to hypoxic levels.[2 8 9] Our study shows that tracheal suctioning considerably disturbs the oxygenation and causes hypoxaemia on 1 occasion out of 2 in nearly all infants even though they may initially have been well oxygenated. The hypoxaemia is not immediately reversed by bag ventilation with oxygen, but can last as long as 2 minutes in some patients (Fig. 1). We believe this procedure may hinder recovery from the initial disease.

As both groups of infants reacted in a similar manner with falls in $TcPo_2$, we disagree that tracheal suction mainly affects small preterm infants. Surprisingly we found no correlation between the duration that they were disconnected from the respirator and the extent of $TcPo_2$ fall. Although the time that it took to suction the infant was not measured, it is reasonable to assume that it was proportional to the period that the infant was disconnected from the respirator. Thus the lack of correlation cannot be ascribed to differences between these two variables. The severity of the disease seems to be the main determinant for the $TcPo_2$ fall, as patients with high F_IO_2 and controlled ventilation were affected to a greater extent than the patients with intermittent mandatory ventilation and lower F_IO_2. However infants receiving intermittent mandatory ventilation also had large decreases in $TcPo_2$ leading to hypoxaemia. In an infant on intermittent mandatory ventilation the oxygen in the alveoli decreases as it is absorbed during suctioning, and as such infants breathe room air the alveolar oxygen concentration is reduced even further. As the rate in the fall of alveolar oxygen is greatly influenced by the rate of $TcPo_2$, these infants are probably equally likely to become hypoxaemic.

The cardiovascular changes noted during tracheal suction were typical of a hypoxaemic response, characterised by a falling heart rate and an increasing blood pressure.[10 11] The heart rate decrease of 21·5 beats/min and the blood pressure increase of 4·4 mmHg are in good agreement with experimental data in fetal lambs.[10] However we cannot exclude the possibility that the baby's activity, in response to this unpleasant respiratory experience, could bring about similar changes.

Even if a baby did not become hypoxaemic ($TcPo_2$ <50 mmHg) a decrease in $TcPo_2$ to levels

between 60 and 50 mmHg would cause the blood to become less saturated with oxygen. At the same time the heart rate would be lower, the pulse pressure the same, and consequently the cardiac output would be decreased. Thus the oxygen availability (cardiac output × 1·34 haemoglobin × saturation) for the body's tissues would be decreased with tracheal suction.

Studies in animals,[12] and in man,[13] indicate that the chemoreceptors are activated when the Po_2 falls below 70 or 100 mmHg respectively. Such a Po_2 fall occurred in our patients; it probably increased the activity of the chemoreceptors and subsequently the pressure area in the brain stem and hence augmented sympathetic outflow. The latter causes vasoconstriction. We therefore think that tracheal suction not only causes hypoxaemia in some infants and diminishes oxygen transport in others, but that it also triggers a hypoxaemic response in nearly all infants which is not immediately reversed by high oxygen levels. This conclusion is supported by our finding cardiovascular responses characteristic of hypoxaemia on nearly all, and yet hypoxaemia ($TcPo_2$ <50 mmHg) on only half of the occasions. Furthermore 8 of these 10 infants had higher blood pressures at 5 minutes after bag ventilation, despite high oxygen levels, than during the control period. The hypoxaemic vasoconstriction is known to affect mainly muscle, splanchnic, renal, and pulmonary vessels. Thus tracheal suctioning and its cardiovascular consequences may well account for many problems in the neonate—such as necrotising enterocolitis, patent ductus arteriosus, or intrapulmonary hypoperfusion.

The fact that hypoxaemic cardiovascular changes took place more often than hypoxaemic $TcPo_2$ levels, might be explained by a dampened transcutaneous Po_2 measurement. The Po_2 probably fell faster and to lower values than suggested by the $TcPo_2$ measurement. The $TcPo_2$ often did not reach its minimum until 2 minutes after bag ventilation. Furthermore, the $TcPo_2$ measurements in steplike changes of inspired oxygen showed a delay compared with direct Po_2 determinations.[14] It can be argued that the $TcPo_2$ measurements show falsely low Po_2 values. During suctioning, heart rate (generally) and blood pressure (occasionally) fell. Then skin perfusion might have been decreased and less oxygen been diffused through the skin to the electrode than with normal perfusion.[15] Po_2 measurements during tracheal toilet should be done using a sensor with an immediate response.

All the changes brought about by tracheal toilet call for the procedure to be re-evaluated. At first sight, the long time taken to do the suctioning seems at fault. We measured this time accurately. Other authors[8 16] who reported a suctioning time of 15 seconds may have underestimated the time. But even if the time the infant is disconnected from the oxygen supply is kept to less than 30 seconds, hypoxaemia and circulatory changes will take place in some patients. We suggest reducing the frequency of tracheal toilet to a minimum, limiting the duration of suctioning, and using respirators which allow simultaneous ventilation and suction.[17]

References

1 Gregory G A. Respiratory care of newborn infants. *Pediatr Clin North Am* 1972; **19**: 311–24.

2 Dangman B C, Hegyi T, Hiatt M, Indyk K, James L S. The variability of PO_2 in newborn infants in response to routine care (abstract). *Pediatr Res* 1976; **10**: 422.

3 Shim C, Fine N, Fernandez R, Williams M H, Jr. Cardiac arrhythmias resulting from tracheal suctioning. *Ann Intern Med* 1969; **71**: 1149–53.

4 Brandstater B, Muallem M. Atelectasis following tracheal suction in infants. *Anesthesiology* 1969; **31**: 468–73.

5 Rosen M, Hilliard E K. The effects of negative pressure during tracheal suction. *Anesth Analg (Cleve)* 1962; **41**: 50–7.

6 Segal S. Endobronchial pressure as an aid to tracheobronchial aspiration. *Pediatrics* 1965; **35**: 305–12.

7 New York State Society of Anesthesiologists Anesthesia Study Committee. Endotracheal suction and death. *N Y State J Med* 1968; **68**: 565–6.

8 Fox W W, Schwartz J G, Shaffer T H. Pulmonary physiotherapy in neonates: physiologic changes and respiratory management. *J Pediatr* 1978; **92**: 977–81.

9 Emmerich P, Stechele U. Kontinuierliche $tcPO_2$ Messung bei langzeitbeatmeten Kindern. In: Emmerich P, ed. *Pädiatrische Intensivmedizin.* Vol. 3. Stuttgart: Thieme Verlag, 1977: 77–82.

10 Cohn H E, Sacks E J, Heymann M A, Rudolph A M. Cardiovascular responses to hypoxemia and acidemia in fetal lambs. *Am J Obstet Gynecol* 1974; **120**: 817–24.

11 Robinson J S, Jones C T, Thoburn G D. The effect of hypoxaemia in fetal sheep. *J Clin Pathol [Suppl]* 1977; **30**: Supplement (Roy Coll Path) 11, 127–33.

12 Pelletier C L. Circulatory responses to graded stimulation of the carotid chemoreceptors in the dog. *Circ Res* 1972; **31**: 431–43.

13 Biscoe T J, Purves M J, Sampson S R. The frequency of nerve impulses in single carotid body chemoreceptor afferent fibers recorded *in vivo* with intact circulation. *J Physiol (Lond)* 1970; **208**: 121–31.

14 Versmold H T, Linderkamp O, Stuffer K H, Holzmann M, Riegel K P. *In vivo* vs *in vitro* response time of transcutaneous PO_2 electrodes. *Acta Anaesthesiol Scand [Suppl]* 1978; **68**: 40–8.

15 Versmold H T, Linderkamp O, Holzmann M, Strohhacker I, Riegel K P. Limits of $tcPO_2$ monitoring in sick neonates: relation to blood pressure, blood volume, peripheral blood flow, and acid base status. *Acta Anaesthesiol Scand [Suppl]* 1978; **68**: 88–90.

16 Urban J B, Weitzner W S. Avoidance of hypoxemia during endotracheal suction. *Anesthesiology* 1969; **31**: 473–5.

17 Simbruner G, Götz M. A neonatal respirator allowing simultaneous ventilation and suction. *Klin Paediatr* 1976; **188**: 532–8.

Correspondence to Dr G Simbruner, Universitäts Kinderklinik, Währinger Gürtel 74–76, A–1090 Wien IX, Austria.

Received 28 January 1980