Atelectasis Following Tracheal Suction in Infants

BERNARD BRANDSTATER, F.F.A.R.C.S.,* AND MUSA MUALEM, M.D.†

Although tracheobronchial suction is a commonplace maneuver it occasionally has serious consequences. Cardiovascular collapse, and even death, have been reported,1,2 indicating that negative pressure in the lungs can produce serious disorders of cardiac and pulmonary function. Less dramatic disturbances presumably occur often. Boba and others3 observed an acute fall in arterial oxygen saturation following tracheal suction in human subjects. Schmidt4 reported an increase in elastic lung resistance and mild arterial hypoxemia following tracheal suction in anesthetized patients. Similar changes have been observed when negative pressure was applied to the tracheas of anesthetized dogs.5,6 There is a fall in pulmonary compliance and an increase in venous admixture, both of which can be reversed by positive-pressure inflation. The shunting which follows atelectasis is less than might be expected from the extent of lung collapse, but changes in pulmonary compliance are closely related to the amount of induced atelectasis.8

The effects of tracheal suction presumably are similar in dog and man, and are related quantitatively to the force and duration of negative pressure. Especially vulnerable is the infant, in whom a suction catheter may occupy a large part of the lumen of a small nastrotracheal or tracheostomy tube. The studies reported here were designed to show the extent of atelectasis, as represented by compliance change, occurring in the lungs of infants subjected to tracheobronchial suction, and the subsequent behavior of collapsed areas.

METHOD

Subjects of this study were six newborn infants being treated for severe tetanus neonatorum with a regimen which included paralysis with muscle relaxants and mechanical ventilation through a nasotracheal tube. Their ages ranged from 7 to 34 days, and their lungs were judged to be normal by clinical and radiologic examination. In each case the regimen had to be maintained for three or four weeks; during this period many tests and observations were made.

Tests were carried out with the subjects lying supine. Pressures at the airway and in an esophageal balloon were compared by the use of a Statham differential strain gauge. The balloon, of thin rubber latex, 5 cm in length, was inserted into the lower esophagus at a level where there was minimum cardiac artifact. The difference between airway and esophageal pressure was considered to approximate transpulmonary pressure. Respired air passed through a heated Fleisch pneumotachograph, and the flow signal was integrated electrically to yield a record of expired tidal volume. The integration system was volume-calibrated using a 100-ml syringe and an airflow pattern similar to a typical exhalation; the system was close to linear within the range of volumes used in these studies. Tidal volume and transpulmonary pressure were recorded continuously on a Grass Polygraph. The experimental system is shown in figure 1.

Two different ventilators were used. One was a Bird respirator, in most instances driven by pressurized air, or with oxygen when extended periods of atelectasis were expected. When this ventilator was in use the peak limit of inflation pressure was kept constant, and changes in dynamic pulmonary compliance were seen on the record as changes in tidal volume.

* Associate Professor and Chairman, Department of Anesthesiology. Present address: Department of Anesthesiology, Loma Linda University, Loma Linda, California 92354.
† Associate Professor.

Received from the Department of Anesthesiology, American University of Beirut, School of Medicine, Beirut, Lebanon. Supported in part by research grant HD 00780 from the National Institutes of Health, United States Public Health Service.
The second ventilator was a Harvard small-animal respirator, model 671, which is a piston pump with a stroke volume continuously variable from 0 to 100 ml. For most studies air was used for ventilation, oxygen enrichment being resorted to when indicated by extensive atelectasis. Ventilatory rate was usually set at 40/min, but could be varied at will. Using this pump, tidal volume could be kept at a predetermined constant level, and in these studies it varied between 20 and 40 ml. Changes in pulmonary compliance were seen as changes in the recorded transpulmonary pressure.

Before the beginning of each study the subject was given a reinforcing dose of muscle relaxant, either dallylnortoxiferine, \(2\) mg, or \(d\)-tubocurarine, \(3\) mg, administered intramuscularly. These doses abolish tetanic spasms for more than an hour in newborn infants, and were considered sufficient to produce optimal flaccidity for these studies. The patient was examined to exclude any leak around the tube in the larynx, and the tracheobronchial tree was subjected to a routine irrigation with 2 ml of sterile saline solution, followed by suction. At the beginning of each test the expansion history of the infant’s lungs was standardized by a series of deep inflations at a pressure of approximately 25 cm H\(_2\)O, administered either by connecting a bag and T piece to the nasotracheal tube or by manipulating the inflation pressure control on the Bird ventilator. For purposes of study, tracheal suction was performed in a manner similar to that commonly practiced by skilled nurses, with equipment and tubes identical to those used routinely in our hospital for respiratory care of infants.

The nasotracheal tube was a soft Portex plastic tube with an internal diameter of \(3\) mm (external diameter equivalent to \(13\) Fr.). The suction catheter was a soft vinyl feeding tube, size 8 or \(5\) Fr. (external diameter 2.7 mm or \(1.7\) mm), with a smooth, closed, rounded tip and two lateral openings. With suction off, the catheter was inserted as far as it would easily pass, and suction was then opened for three or four seconds while the catheter was gently moved, then quickly withdrawn. The suction source was a standard Gomco mobile suction machine whose gauge registered pressure levels which fluctuated during suction but did not exceed \(-46\) cm H\(_2\)O.

**RESULTS**

When performed as described above, tracheal suction consistently produced a sharp fall in pulmonary compliance. Effects of suction on the lungs were greater when suction was prolonged and when the larger suction catheter was used. In 26 observations made during use of the Bird respirator, tidal volume fell by amounts that ranged from 25 to 70 per cent of control; falls of 40 to 50 per cent were common (fig. 2). Infants ventilated at constant volumes with the Harvard pump had increases in transpulmonary pressure following tracheal suction, ranging from 15 to 60 per cent of control in 19 observations (fig. 3).
Pulmonary compliance could be restored to normal by applying an inflation pressure of 25 to 30 cm H$_2$O across the lungs. A cycle of lung collapse and re-expansion could be repeated as often as desired, with full reversibility in both directions (figs. 4 and 5).

When an infant was connected to the same respirator following tracheal suction, with inflating pressure or stroke volume at the original setting, the lungs remained partially collapsed until a high pressure or a large tidal volume was applied. With the Bird respirator, failure of tidal volume to return to its presuction level was observed for periods as long as half an hour (fig. 5). With the Harvard pump, transpulmonary pressure often came down from its immediate peak following suction, but stabilized at a level well above control.

Partial re-expansion of the lungs occurred commonly, and was more complete when initial inflation pressure or stroke volume was high. Figure 6 shows a test run in which transpulmonary pressure was increased step-

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**Fig. 2.** Effect of tracheal suction on tidal volume in an infant ventilated with a Bird respirator set to produce a peak transpulmonary inflation pressure limit of 11 cm H$_2$O.

**Fig. 3.** Effect of tracheal suction on transpulmonary pressure in an infant ventilated at a constant tidal volume of 20 ml.

**Fig. 4.** Effect of repeated inflation and suction in an infant ventilated at a constant stroke volume of 33 ml.

**Fig. 5.** Tidal volume record in an infant ventilated with a Bird respirator at a transpulmonary pressure of 14 cm H$_2$O. Normal compliance is restored by several deep inflations. Without deep inflation the lungs remain in partial collapse, and show no likelihood of spontaneous reopening.
wise; convincing evidence of progressive re-expansion did not occur until a transpulmonary pressure of 16.5 cm H$_2$O was applied. In other infants full re-expansion failed to occur at a transpulmonary pressure of 17 cm H$_2$O. At pressures below 15 cm H$_2$O it was common to see little or no re-expansion. When the Harvard respirator was used some lungs failed to re-expand fully even when the stroke volume was twice the estimated normal tidal volume. Full re-inflation could be achieved consistently by giving three deep breaths, each at 25 cm H$_2$O pressure maintained for two seconds.

**DISCUSSION**

That tracheobronchial suction can have deleterious effects upon the lungs has long been recognized. Modern suction techniques usually provide for a pressure-control device, such as a lateral opening in the suction tubing, to shorten the period of negative pressure. We have the impression that, in spite of occasional warnings in the literature, the effects of suction are usually considered to be of small magnitude and to cease when the catheter is withdrawn. Our data from babies point to an extent of lung collapse which is greater than commonly believed, certainly greater than we were expecting to find.

One may question whether the changes in dynamic pulmonary compliance observed in our subjects were wholly the result of atelectasis. Negative pressure may produce changes in air passages and still-ventilated lung units that will be seen as compliance changes. In anesthetized dogs Collier and Mead invoked time-dependent surface-tension changes as a partial explanation of fall in compliance, but these phenomena are not likely to be a factor in events which occur instantaneously in response to suction. Colgan, Whang and Gillies induced atelectasis in dogs, and found that recorded changes in compliance were closely related to the amount of lung collapse seen later at postmortem examination. We have failed to find a convincing mechanism for compliance fall other than collapse of air spaces, and believe that the changes seen in our subjects were a quantitative index of atelectasis.

We have no data to indicate the amount of venous admixture which accompanied atelectasis in our subjects. In a few instances there
was obvious cyanosis when infants ventilated with air incurred decreases in compliance of more than 50 per cent. From observations in dogs venous admixture is considerably less than might be expected from the extent of atelectasis, owing to a shift of perfusion away from nonventilated areas. Nevertheless, this compensatory shift may take time to become fully operative; the immediate result of tracheal suction may be a peak of hypoxemia that could endanger a critically-ill patient.

The consequences of tracheal suction do not end with the appearance of acute lung collapse. Our data show that a collapsed lung will remain collapsed unless high air pressure or volumes are used for inflation. Some lungs remained unexpanded for 30 minutes, and presumably would have remained so for hours, perhaps even days. That collapsed alveoli require a high opening pressure is commonly observed directly when an anesthetist reinflates a collapsed lobe in the open chest. Atelectasis is not different when it occurs in the closed chest, and may remain substantially uncorrected unless deliberate reexpansion is carried out. Such a maneuver has been recommended; our experience suggests that an inflation pressure of about 25 cm H2O is desirable. This finding is comparable to that of Rattenborg and Holladay, who observed in dogs that collapsed alveoli begin to open at an airway pressure between 18 and 23 cm H2O. We have not tested the recommendation of Segal and Boba et al. that positive pressure be applied preventively during the actual performance of suction.

What happens to collapsed lung which is not reinflated is a matter of conjecture. Some areas may open slowly over a period of hours, unless repeated suction adds injury to insult. Other areas may remain collapsed, and the next likely event is infection and pneumonia. Respirator patients are susceptible to pulmonary infection, and postoperative atelectasis, in the past attributed to collection of secretions in the bronchi and absorption of sequestered air, is familiar to anesthetists. The best prevention once lay in coughing and frequent tracheal suction. In recent years it has become clear that the traditional stir-up routine of deep breathing and coughing not only helps to get rid of secretions, but also provides the deep expansion without which microatelectasis may occur even in a normal lung. Our data suggest that, especially in infants, the zealous suction with which we hope to prevent atelectasis may be, in some cases, its principal cause.

The end of every anesthetic provides an opportunity to respect or ignore the sequelae of suction. Before removing a tracheal tube it is customary to aspirate secretions by deep suctioning. Frequently tube and suction catheter are withdrawn together. Without a full reflation of the lungs, for which the tracheal tube should be left in place, the patient may proceed to the recovery room with significant atelectasis and pulmonary shunting. Though arterial unsaturation is to be expected in many recovering patients, anesthetists should avoid making an active contribution to it.

The authors acknowledge with thanks the assistance of Dr. Francis Gabali and Miss Shake Messerlian.

References

Avoidance of Hypoxemia during Endotracheal Suction

BRUNO J. URBAN, M.D.,* AND STANLEY W. WEITZNER, M.D.†

Removal of secretions by endotracheal suction is a frequent and necessary adjunct to ventilator therapy. Unfortunately, this procedure not only interrupts the sequence of ventilation but, in addition, may aspirate intrapulmonic gas. Arterial oxygen tensions thus may be lowered to hazardous levels, and cardiac arrest associated with tracheal suction has been reported.1 Preoxygenation, limiting the duration and magnitude of suction,2,7,8 and the simultaneous insufflation of oxygen through a double-lumen catheter4 have been advocated to avoid this. These measures will prevent desaturation in most cases. However, when the underlying disease process demands high inspiratory oxygen concentrations, preoxygenation will be ineffective, limited suction may not prevent arterial hypoxemia, and the insufflation of additional oxygen may be inadequate.5 Furthermore, the presence of copious secretions makes tracheobronchial toilet without time restriction highly desirable.

Whereas ventilation is the result of an intermittent gas flow into and out of the lung, suction may be thought of as a continuous flow out of the lung, with no inspiratory component. In theory, therefore, removal of intrapulmonic gas could be offset by an increased inspiratory flow. This is easily accomplished by increasing the inspiratory minute volume, thus using the lung as a reservoir for gas removed by continuous suction. Provided the oxygen concentration is kept constant, arterial oxygenation should be maintained regardless of the duration and amount of endotracheal aspiration. We were able to demonstrate this in the laboratory; the results of this study of patients undergoing ventilator treatment confirm the hypothesis.

METHOD

Seven male patients on treatment with a volume-preset ventilator (Engström) were selected at random. Ages, diagnoses, and ventilation requirements, with the corresponding blood gas analyses, are summarized in table 1. The inspired oxygen concentration (FiO2) was computed from the gas input to the ventilator, while exhaled minute volume (Ve), peak airway pressure (P) and respiratory rate (RR) were monitored from the machine.

The amount of gas removed by each suction apparatus with catheter was measured using a Wright Respirometer with the inlet open to room air. These volumes differed according to the particular machine and catheter size utilized (table 2). “Conventional suction”—as used in our institution—was timed and rigidly limited to 15 seconds. If the secretions could not be removed during that period, the ventilator was reconnected and ventilation was resumed before reinstituting tracheal aspiration. “Modified suction” was achieved by introducing the catheter through the suction sidearme

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* Assistant Professor of Anesthesiology.
† Associate Professor of Anesthesiology.

Received from the Department of Anesthesiology, Downstate Medical Center, State University of New York, and Kings County Hospital Center, Brooklyn, New York.