## Measurement of Spontaneous Ventilation with the Engstrom Respirator

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Drs. Saklad and Yamada remark that if the patient is being ventilated by an Engstrom apparatus and inasmuch as this apparatus has a spirometer incorporated into it, the patient's ability to breathe spontaneously may be determined in the following fashion:

(1) Stop the ventilator.

(2) Remove the inspiratory tube at a point close to the chimney.

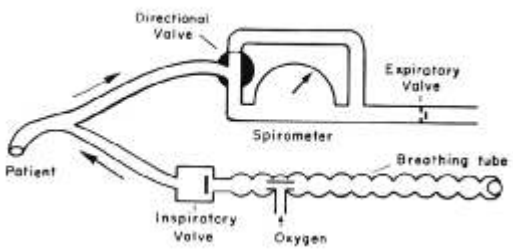
(3) Insert a unidirectional valve, as the Collins "J" valve, on the inspiratory side of the chimney.

The patient will then inhale room air and exhale down the expiratory tube. With the spirometer valve in the ON position, the exhaled gases are directed through the spirometer and the patient's tidal exchange and minute volume can be thus determined.

There are two disadvantages to the above plan: (1) a resistance of approximately 0.5 to 0.7 mm. of mercury is reached when exhaling 500 ml. rather rapidly; (2) the patient is exposed to room air.

The resistance is an undesirable feature. This might make it unwise to employ this means to measure the respiratory ability of certain types of patients.

Should it be desirable to meter the respiratory ability of a patient who is being ventilated with oxygen-enriched air, increased oxygen tension can be maintained during the spirometer study by attaching a breathing tube to the inspiratory valve, and allowing oxygen to run into it as in the accompanying figure.

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# CASE REPORTS

## Respiratory Obstruction with Oxygenation Apnea

This report, presented by Dr. Thomas F. Horbein to the Committee on Clinical Anesthesia Study Commissions, describes a case of respiratory obstruction with postoxygenation apnea. It is of interest in regard to the problem of diagnosis of the cause of apnea and as a documented example of a clinically occurring extreme degree of respiratory acidosis. It illustrates several physiologically interesting aspects of response to hypoxia and hypercapnia.

### Case Report

This two and one-half year old Caucasian male was admitted with progressive respiratory distress, beginning with a cough the day prior to admission and accompanied by a low grade fever. He had had surgery at twenty months of age for a Meckel's diverticulum, and five months prior to the present illness, repair of a large interatrial septal defect and pulmonic valvular stenosis was performed. He had experienced several episodes of bronchiolitis during the first year of life and was treated subsequently for pneumonia and frequent attacks of asthma. Between such episodes he was reported to be normally active but always possessed blueness of his nail beds and required two pillows in order to sleep at night.

Physical examination on admission revealed a well-developed and nourished child, slightly

smaller than expected for his age. He was pale, cyanotic with retraction of his chest on inspiration and audible expiratory wheeze. Pulse was 100/minute; temperature 98.6° F. He had grade II systolic and diastolic murmurs, loudest over the pulmonic area. The liver was palpable two fingersbreadth below the right costal margin. White blood cell count was 17,800 with 87 per cent polymorphonuclear leukocytes, hematocrit, 46 per cent. Chest roentgenogram revealed possible early pneumonitis in the right lower lung field.

The child was placed in an oxygen tent with cold mist and started on penicillin. The following morning he suddenly stopped breathing and was immediately resuscitated with mouth-to-mouth respiration. Immediately thereafter he had a heart rate of 120 per minute with regular rhythm, marked expiratory difficulty, and his liver was noted to be three fingersbreadth below the costal margin. He was digitalized, placed on steroids by intravenous infusion, and penicillin was increased to 4,000,000 units per day. An electrocardiogram showed no evidence of strain. A repeat chest film revealed extension of the pneumonitic process with infiltrate in both bases. The patient remained afebrile but respiratory difficulty became progressively greater. On the morning of the third hospital day he was quite limp and only weakly responsive to painful stimulation. He was extremely cyanotic. Respiration was rapid and very labored with marked inspiratory retraction of the entire rib cage and prolonged expiration associated with coarse, easily audible wheezing.

Forty-eight hours following admission bronchoscopy and tracheostomy were performed. Although the child was almost totally unresponsive to stimulation, tightness of the jaw muscles necessitated the use of anesthesia. This was begun with halothane in oxygen by mask, which resulted in almost immediate apnea. Respiration was controlled until sufficient depth of anesthesia had been obtained to permit introduction of the bronchoscope.

During the forty-five minutes of bronchoscopy a 10-liter per minute flow of oxygen with 0.5 to 0.8 per cent halothane was insufflated down the bronchoscope, ventilation being achieved by rhythmic occlusion of the end of the bronchoscope between periods of visualization. Chest movement was noted to be minimal, particularly on the right side. Following piecemeal aspiration of a spongy white mass in the right main stem bronchus aeration of the right lung was improved. The bronchi appeared open but markedly inflamed and edematous. During this time the child made no effort to breathe but coughed weakly upon manipulation of the bronchoscope. The tracheostomy was performed with the bronchoscope in place, ventilation being achieved as before, but on a regular, rhythmical basis. Since there was no response to the skin incision, halothane was discontinued. Upon completion of the tracheostomy forty minutes later the child was apneic and totally unresponsive to pain. Aeration of the lungs was improved, but attempts to restore spontaneous respiration by permitting carbon dioxide accumulation were ineffectual, even when apnea was carried to the point of beginning gross cyanosis and cardiac slowing. The child was taken to the recovery room with respiration manually controlled.

At this time his blood pressure was 125/60, pulse 110, temperature 96.6° F. He was not cyanotic while being ventilated with 100 per cent oxygen but was totally unresponsive. An arterial blood sample was obtained anaerobically in a heparinized syringe and was analyzed for $P_{O_2}$, $P_{CO_2}$, and *p*H on an Instrumentation Laboratories research model blood gas electrode unit. During this time the patient began to show weak twitching movements of the right arm and leg and irregular diaphragmatic gasps at a rate of one to four per minute. The initial blood sample while ventilated with 100 per cent oxygen showed:

$Pa_{O_2}$—107 mm. of mercury; $Pa_{CO_2}$—235 mm. of mercury; *p*H—6.74; BB—8.9.

As a result of these findings the patient was ventilated more vigorously while heart rhythm and blood pressure were carefully monitored. Within five minutes he developed twitching about the lips and eyelids which did not disappear on decreasing the magnitude of ventilation. Soon thereafter weak but rhythmical respiration appeared and progressively increased until the patient could be maintained unassisted on 100 per cent oxygen. Depth

of respiration and movement of all extremities improved rapidly over the next half hour, at the end of which time the child was opening his eyes and appeared more responsive than at any time preoperatively. A second arterial sample, one hour after the first with the child breathing spontaneously on 100 per cent oxygen, showed:

$Pa_{O_2}$—114 mm. of mercury; $Pa_{CO_2}$—88 mm. of mercury; *p*H—7.00; acid excess—8 m*M*/liter.

Minute volume at this time was 1,700 ml. at a rate of 24 per minute. Administration of isoproterenol-Neo-Synephrine aerosol by IPPB resulted in an increase to 2,100 ml. minute volume at 25 breaths per minute. Sixteen milliequivalents of sodium bicarbonate were administered intravenously. A blood sample obtained two hours after entering the recovery room while spontaneously breathing room air revealed:

$Pa_{O_2}$—50 mm. of mercury; $Pa_{CO_2}$—57 mm. of mercury; *p*H—7.27; acid excess—2.0–0 m*M*/liter.

Over the next hour respiration continued to become easier with only minimal retraction of the lower intercostals on inspiration and definite decrease in expiratory time. Blood studies at the end of three hours, while breathing spontaneously a mixture of air and nebulized oxygen mist, showed:

$Pa_{O_2}$—214 mm. of mercury; $Pa_{CO_2}$—48.3 mm. of mercury; *p*H—7.37; acid excess—0 m*M*/liter.

He continued to do well during the night and respiration was further improved the following morning. Minute volume was 2,700 ml. at 28 respirations per minute. Chloromycetin was added to the regimen. He was maintained on continuous heated aerosol via the tracheostomy with intermittent bronchodilator delivered by IPPB. Careful attention to aseptic tracheal care was maintained. His improvement was continuous and remarkably rapid. Within a week his tracheostomy was closed. He began to play about the ward. Intelligence and development seemed normal, and there was no evidence of cyanosis of lips or nail beds. He was discharged two weeks after admission. His subsequent course has been uneventful.

## Discussion

This case presented the problem of diagnosis of postanesthetic apnea. The differential diagnosis is simplified in that no muscle relaxants were used. Since halothane had been used in low concentrations and had been discontinued sometime before the end of the procedure, it is unlikely that it played a significant role in the postanesthetic apnea, though a possible contribution cannot be excluded. In retrospect there is little doubt of the underlying cause of apnea, but at the time this was by no means clear. The possibility that apnea might be due to hyperventilation to $P_{CO_2}$ levels below the apneic threshold in an otherwise very depressed child was seriously considered. Complete apnea from carbon dioxide retention alone is thought to be possible, but inspiration of carbon dioxide concentrations as high as 30 per cent has not led to respiratory depression in normal individuals.[1]

The child was in severe respiratory distress prior to the procedure. He was deeply cyanotic, breathing air, and undoubtedly possessed sufficient abnormality of alveolar ventilation and distribution that his arterial $P_{CO_2}$ was well above normal values. On the basis of known estimates of carbon dioxide storage and rate of rise of $P_{CO_2}$ during apnea,[2] it would be difficult to presume an initially normal $P_{CO_2}$ even if the child had remained entirely apneic through the entire period of bronchoscopy.

Though it was not realized at the time, this child's total apnea after a few breaths of 100 per cent oxygen by mask suggests that his respiration was being driven by chemoreceptor drive. The magnitude of this drive is suggested by the occurrence of total apnea, for more commonly oxygen breathing in similar clinical situations in patients with chronic pulmonary emphysema results only in a decrease in ventilation. It has recently been shown that the combination of hypoxia and hypercapnia is a far more potent stimulus to chemoreceptor activity than either alone[3]; in other words these two stimuli interact to produce a more than additive chemoreceptor response. It is likely that this child's ventilatory drive resulted from such a combination of stimuli.

The narcotizing potential of high concen-

trations of carbon dioxide is well known.[1, 4] This patient certainly exhibited rather profound anesthesia at the close of the procedure, being totally unresponsive to painful stimulation and responding with only minimal cough on movement of the tracheostomy tube. It is interesting that allowing the patient to remain apneic to the point of cyanosis did not result in return of respiration even though significant bradycardia occurred. This initial bradycardia with asphyxia is probably on the basis of chemoreceptor stimulation, for it has been shown that chemoreceptor stimulation does result in cardiac slowing.[5]

At the time of initial sampling the child possessed a mild metabolic component to his extreme acidosis. This might have been present on a long standing basis, or it could have been secondary to sympathetic stimulation and release of catecholamines[6] produced by the extreme respiratory acidosis *per se*.[7] Although hypertension, which is also secondary to catecholamine release,[8] classically occurs with carbon dioxide retention, it is interesting that this child's blood pressure was never greatly elevated. This has been observed by others.[9] He was carefully monitored for possible arrhythmias during the period of blowing off of carbon dioxide,[10] but no difficulties were encountered.

The members of the Committee on Clinical Anesthesia Study Commissions believe it should be re-emphasized that this little patient had previously undergone a repair of interatrial septal defect, but he apparently still had some right to left heart shunting since his $Pa_{CO_2}$ was only 50 mm. of mercury on breathing room air. This would result in increased blood flow to the lungs. Children, such as this, are known to develop cardiac failure with only a mild upper respiratory infection. The physical findings on admission, such as the marked cyanosis and respiratory distress (out of proportion to the beginning pneumonic process), the generalized expiratory wheezing and the enlarged liver point to congestive heart failure with incipient pulmonary edema. Congestive heart failure leads to hypoventilation, primarily by reducing pulmonary compliance, and hypoventilation results in hypoxia, hypercarbia and respiratory acidosis. Both prolonged hypoxia and hypercarbia depress the respiratory center so that hypoventilation is increased and respiratory activity becomes dependent upon chemoreceptor stimulation from oxygen-lack and carbon dioxide excess. Thus, a vicious cycle is started.[11]

At the time of bronchoscopy, the child was unresponsive but he exhibited tightness of the jaw muscles. This may have been due to the effects of hypercarbia since carbon dioxide excess is presumed to produce an excessive release of acetylcholine.[12]

The administration of oxygen and elimination of carbon dioxide under such circumstances removes the chemoreceptor stimuli, and in the presence of depressed respiratory center, apnea results.[11] In such situations, as was done in this case, effective artificial ventilation must be continued until the respiratory center can again function normally.

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