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# Controlled Ventilation with Intermittent Positive-Pressure Breathing in the Management of Acute Ventilatory Failure Associated with Chronic Obstructive Pulmonary Disease

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CONTROLLED MECHANICAL VENTILATION may be employed for ventilatory support in patients with respiratory failure when assisted ventilation fails. The purpose of controlled ventilation is to reduce patient respiratory control and effort and to substitute adequate artificial ventilation, thereby providing time for appropriate medical therapy. With improvement the patient's own ventilatory effort may be capable of maintaining normal or clinically stable balance. These principles have been adequately demonstrated in acute ventilatory failure with volume-cycled respirators (1, 2). It was the purpose of this prospective study to evaluate in patients with chronic obstructive lung disease, a group usually difficult to manage during acute ventilatory failure, the following: [1] whether conventional pressure-cycled intermittent positive-pressure breathing (IPPB) could achieve effective controlled ventilation under bedside conditions; [2] at what point this procedure should be substituted for conventional assisted IPPB; and [3] the sequential steps necessary to achieve this goal.

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## MATERIALS AND METHODS

This report is based on 21 ventilatory studies performed at the bedside on 19 patients with chronic obstructive lung disease in acute ventilatory failure. Major pulmonary diagnoses (Table 1) included asthmatic bronchitis, chronic bronchitis, cor pulmonale, obstructive emphysema, and tuberculosis. In addition, medical complications of pneumonia, cerebrovascular problems, cardiac disease, obesity, bacteremia, gastrointestinal abnormalities, and shock characterized the type of patient studied.

The presumptive causes for ventilatory failure are included in Table 1. Acute respiratory insufficiency was judged clinically and confirmed by arterial blood gas measurements ($Paco_2 > 55$ mm Hg, $pH < 7.25$, and $Pao_2 < 60$ mm Hg, as acute changes). Constant conditions were maintained during the trial period. Tracheal suctioning was permitted, but the addition of mucolytic, bronchodilator, or wetting agents was deferred until the best control cycles were established. Periodic brief deep breathing was permitted.

Serial arterial blood gases were drawn via a Cournand needle at 15- to 30-min intervals until optimal ventilation was established and frequently thereafter to prevent hyperventilatory alkalosis. Once relative stabilization of

$P_{CO_2}$ and pH occurred, serial gases were drawn at 1- to 2-hr intervals to facilitate adjustment of the respirator. All tidal volumes were measured at the exhalation port with a Monaghan Ventilation Meter. Initial tidal volumes were arbitrarily selected to be 500 cc to 700 cc.

Arterial blood was obtained anaerobically and immediately analyzed in the IL instrument (Model 102) for $P_{O_2}$, $P_{CO_2}$, and pH at 37 C. Blood exposed to known gas concentrations (Scholander) was tonometered in a water bath at 37 C for $P_{O_2}$ calibration. Dry gas was used for the $P_{CO_2}$ calibration slope. All duplicate samples were required to check within 5 mm for $P_{O_2}$ (50- to 100-mm range), 2 mm for $P_{CO_2}$, and 0.005 units for pH. Oxygen saturation was calculated from the $P_{O_2}$ and the pH using the standard dissociation curve for oxyhemoglobin at 37 C and 7.40 pH. Plasma $CO_2$ content and bicarbonate were calculated from the measured $P_{CO_2}$ and pH by means of a standard nomogram based on the Henderson-Hasselbalch equation.

The sequence of approach is presented below. A period of 1 hr in which $P_{CO_2}$ failed to fall more than 5 mm Hg was accepted as ventilatory failure. Failure of one method was the indication to move to the next.

#### IMPROVED ASSISTED VENTILATION

Every attempt was made to improve conventional assisted respiration. In all cases, a tracheostomy or an endotracheal tube was established and ventilation performed by the Bird Mark 7 or 8 intermittent positive-pressure respirator with 40% oxygen setting and, occasionally, 100% oxygen. It should be noted that the 40% dial value represents the mixture dial on the IPPB unit only and does not represent such concentrations. Actual concentrations of inspired oxygen were not measured. The reasons for failure of assisted IPPB were judged to be [1] progressive clinical obtundation despite assisted IPPB therapy; [2] failure to reduce or to prevent a rise in $P_{aCO_2}$ on assisted IPPB; [3] patient uncooperative or agitated, refusing to accept or phase with respirator; [4] persistent tachypnea, physical exhaustion, obvious excessive work of breathing; and [5] patient becoming obtunded on oxygen therapy.

#### CONTROLLED VENTILATION

Controlled ventilation was instituted in the following sequence when assisted ventilation failed.

*Machine Cycle:* With the sensitivity at 40 +, the IPPB was set for automatic cycling of 10 to 15/min, a flow dial setting of 15, and pressure of 15 cm $H_2O$. A period of 5 to 7 min was permitted for synchronization to occur. If successful, the pressure, flow, and rate dials were reset for effective ventilation, confirmed by arterial blood.

*100% Oxygen Suppression with Machine Automatic Cycle:* Since ventilation in patients with chronic hypercapnia may be depressed by oxygen administration, 100% oxygen was given by assisted IPPB or tracheostomy box until hypoventilation ensued. A limit of 5 to 7 min was set for this to occur. If successful, preset automatic machine cycle was then instituted as in Machine Cycle, using only air mixtures.

*Drug Suppression and Machine Automatic Cycle:* Before drug use the airway was checked, the IPPB was preset and ready to ventilate, and a bag respirator was at the bedside in case of mechanical failure. Either morphine or succinylcholine or both were used arbitrarily: *morphine sulfate*—3 to 5 mg, intravenously, with total dose up to 10 to 20 mg as needed to reduce agitation and produce muscle relaxation; then machine cycle as described above, with 2 to 4 mg at intervals as required; *succinylcholine*—20 to 40 mg, intravenously, as a stat dose, repeated as needed to desired level and followed immediately with machine cycle.

Two phases of controlled ventilation after synchronization were delineated.

*Hyperventilation Period:* This was the phase of acute hyperventilation required to lower $P_{aCO_2}$ and $[H^+]$ and improve $P_{aO_2}$. The criteria for improvement were [1] patient no longer agitated or uncooperative and able to cycle with ventilator; [2] adequate tidal volume on the respirator, even with oxygen administration; [3] improvement in blood gas parameters; [4] reduction in gross ventilatory effort in those with severe obstruction and obvious excessive work of breathing; and [5] disappearance of cyanosis.

*Maintenance Period:* Herein was that phase administered supportively to maintain adequate alveolar ventilation (as defined by $P_{CO_2} = 40$ to 60 mm Hg; pH = 7.30 to 7.45; $P_{O_2} \geq 70$ mm Hg) while associated medical therapy was continued.

## RESULTS

#### ANALYSIS OF CONTROLLED VENTILATION APPROACH

Of the 21 instances requiring ventilatory support 4 were managed by assisted venti-

TABLE 1. Clinical Data*

| Patient | Age | Sex | Diagnosis | Cause of Acute Ventilatory Failure | $Paco_2$–pH–$Pao_2$† | |
|---|---|---|---|---|---|---|
| | | | | | Initial | End |
| | *yr* | | | | | |
| Assisted ventilation | | | | | | |
| M. H. | 47 | M | Asthmatic bronchitis, bronchiectasis, $CO_2$ narcosis, alcoholism | $CO_2$ retention, bronchospasm, and secretions | 110–7.18–283 | 67–7.42–142 |
| M. K. | 80 | F | CB, pulmonary fibrosis, pleural effusion, pneumococcal pneumonia, CHF secondary to cor pulmonale, CVA, hypertension, CAD | Pneumonia, pleural effusion, CHF | 90–7.24–64 | 68–7.36–67 |
| F. Bu. | 62 | M | CB, CPE, TBC (inactive), pneumococcal pneumonia, $CO_2$ narcosis, cirrhosis, duodenal ulcer, CAD | Pneumonia, secretions, Demerol® | 60–7.30–99 | 46–7.39–97 |
| E. $P._2$ | 56 | F | CB, CPE, bronchiectasis, cor pulmonale, atelectasis, pneumonitis (*Klebsiella-Aerobacter*), $CO_2$ narcosis, CVA, acute myocardial infarction, septicemia | Pneumonia, secretions | 129–7.12–178 | 65–7.33–310 |
| Controlled ventilation | | | | | | |
| A. Machine cycle | | | | | | |
| F. Ca. | 65 | M | CB, pneumonia, obesity, $CO_2$ narcosis, cor pulmonale, CAD, CHF and PVCs, gastric dilatation, ? pulmonary emboli | Secretions, poor cough, obtunded, tachypnea, failure to cycle with IPPB | 110–7.18–154 | 78–7.31–180 |
| E. $P._1$ | 56 | F | See Patient E. $P._2$, acute bronchitis, $CO_2$ narcosis, CAD and AMI, cardiac arrest, shock, CVA | Cardiac arrest, apnea, secretions, exhaustion | 63–7.34–90 (received $NaHCO_3$) | 43–7.52–288 |
| P. Ha. | 87 | M | CB and pulmonary fibrosis, acute bronchitis, CAD and CHF, old CVA, ? emboli, hypotension, cardiac arrest | Cardiac arrest, secretions, shallow and rapid respiration | 98–7.05–81 | 37–7.34–311 |
| F. C. | 65 | M | Pulmonary TBC, CB, CPE, cor pulmonale, pneumonitis, $CO_2$ narcosis | Tachypnea, secretions, somnolent | 86–7.24–76 | 49–7.48–212 |
| P. Hu. | 65 | M | CB–CPE–staphylococcal pneumonia, sedation (Librium®, barbiturates), $CO_2$ narcosis, cor pulmonale, dehydration | Secretion, drugs | Not available | 56–7.41–500 |
| A. $M._1$ | 59 | M | Asthmatic bronchitis, CPE, TBC (?activity), $CO_2$ narcosis, adrenal insufficiency, osteoporotic fracture T-6 and T-7, chronic pyelonephritis, hypertension | Pseudomonas septicemia and pneumonia, secretions, tachypnea, exhaustion | 92–7.24–50 | 54–7.46–260 |

* CB = chronic bronchitis; CHF = congestive heart failure; CVA = cerebrovascular accident; CAD = coronary artery disease; CPE = chronic pulmonary emphysema; TBC = tuberculosis; PVCs = premature ventricular contractions; IPPB = intermittent positive-pressure breathing; AMI = acute myocardial infarction; RHD = rheumatic heart disease; MS = mitral stenosis; PAT = paroxysmal auricular tachycardia.

† Hyperventilation phase only.

TABLE 1—(*Continued*)

| Patient | Age | Sex | Diagnosis | Cause of Acute Ventilatory Failure | $Paco_2$–pH–$Pao_2$† | |
|---|---|---|---|---|---|---|
| | | | | | Initial | End |
| | yr | | | | | |
| A. Mu. | 66 | F | CB, pneumococcal pneumonia, cardiac arrest, obesity, shock, renal failure, CHF, CVA, ? lactic acidosis | Cardiac arrest, secretions, hypoxemia | 33–7.44–57 | 40–7.35–220 |
| B. Oxygen depression and machine cycle | | | | | | |
| J. O. | 68 | M | Pneumonia, CB and CPE, TBC (active), pleuritis, bronchiectasis, $CO_2$ narcosis, gastric ulcer, RHD and MS, PAT with block, gastrointestinal bleeding, cirrhosis, bacteremia, dehydration | Pneumonitis, secretions, $O_2$ sensitive, obtunded | 99–7.16–34 | 57–7.28–380 |
| J. G. | 58 | M | CB and CPE, TBC (active), pneumococcal pneumonia, $CO_2$ narcosis, CAD, AMI and CHF, pulmonary emboli, cardiac arrest, alcoholism | Secretions, tachypnea, CHF | 91–7.26–59 | 86–7.31–132 |
| C. S. | 75 | M | Asthmatic bronchitis, pneumococcal pneumonia, cor pulmonale, old TBC, $CO_2$ narcosis, dehydration, oliguria, CHF, alcoholism | Inspissated secretions, agitation, bronchospasm | 67–7.24–33 | 58–7.33–141 |
| C. Morphine sulfate and succinylcholine | | | | | | |
| J. R. | 55 | M | Asthmatic bronchitis, pneumococcal pneumonia, asbestosis, cirrhosis, dehydration | Fatigue, secretions, agitation, bronchospasm, tachypnea | 88–7.12–173 | 58–7.26–101 |
| D. Morphine sulfate | | | | | | |
| J. L. | 70 | M | CB and CPE, pneumococcal pneumonia, cor pulmonale, CAD, CHF, alcoholism, dehydration | Intrinsic disease, secretions, agitation, tachypnea, uncooperative | 77–7.24–93 | 39–7.44–69 |
| M. S. | 60 | F | Asthmatic bronchitis, $CO_2$ narcosis, pseudomonas pneumonia, obesity, pulmonary emboli, chronic pyelonephritis, sigmoid-vesicle fistulae, bacteremia, shock | Pneumonia, secretions, agitation, tachypnea, uncooperative | 118–7.21–62 | 62–7.34–80 |
| A. $M._2$ | 59 | M | See A. $M._1$, lung abscess | Asthma, inspissated secretions, exhaustion, $CO_2$ narcosis, agitation | 76–7.31–71 | 49–7.38–400 |
| R. M. | 49 | F | Asthmatic bronchitis, pneumococcal pneumonia, $CO_2$ narcosis, obesity, CHF, cor pulmonale | Agitated, uncooperative, secretions | 110–7.18–275 | 56–7.35–200 |
| E. Succinylcholine and machine cycle | | | | | | |
| T. W. | 40 | F | Asthmatic bronchitis, pseudomonas pneumonia, $CO_2$ narcosis, duodenal ulcer, cirrhosis, bacteremia, shock, anuria | Exhaustion, secretions, coma, poor synchronization with IPPB | 140–6.99–57 | 62–7.25–57 |
| S. W. | 17 | F | Doriden® ingestion, pneumonia, CB | Coma, tachypnea, poor synchronization with IPPB | 88–7.22–55 | 52–7.38–90 |

lation alone. In the remaining 17 instances 7 (41%) could be cycled simply by machine synchronization. In the remaining 10 cases, where oxygen control was attempted, only 3 (18%) suppressed to the point where machine cycle could be employed. This limited response may be due to the brief time allotted for depression with 100% oxygen. The remaining 7 (41%), after failing to respond to machine cycle or oxygen suppression, required drugs to achieve optimal coordinated respirator effect: 4 with morphine sulfate, 2 with succinylcholine, and 1 employing both.

#### BLOOD GAS DATA—HYPERVENTILATION PHASE

Blood gas data are presented in Table 2. Initial data represent the period where continuous, assisted IPPB ventilation failed and just before controlled ventilation was begun. End values represent the termination of purposeful hyperventilation and the establishment of the maintenance period. During the hyperventilation phase of continuous assisted ventilation, the $Paco_2$ was lowered by a mean value of 35.7 mm Hg, and pH was improved by 0.17 pH units. The mean blood gas changes in each subgroup of controlled ventilation revealed a general reduction in $Paco_2$ and a rise in pH and $Pao_2$ by each modality attempted for each group. In the machine cycle group one patient required correction of hypoxia only. One patient in the oxygen suppression group failed to demonstrate any reduction in $Paco_2$. In cases with severe acidosis, end pH values are influenced by use of $NaHCO_3$. Adequate oxygenation could be provided by all methods. Analysis of mean falls in $Paco_2$ in mm Hg/min for each modality suggests that the method of synchronization was unimportant.

#### FEATURES OF THE IPPB AND VENTILATORY EFFECTS

In Table 3 pressure, flow, and respiratory rate data with the tidal volumes before and during the hyperventilation phase of the particular group are presented. In general, a 67 to 211% increase in tidal volume and a 34 to 50% fall in respiratory rate (maintained at 16 to 20/min) were necessary to obtain effective alveolar hyperventilation. However, minute ventilation changes were variable, despite reduction in $Paco_2$. Mean end inspiratory pressures varied from 24 to 40 cm $H_2O$ and flow settings, from 7.5 to 22.8. The wide range of pressures (18 to 40 cm $H_2O$) and flow settings (5 to 38) (relative dial settings, not absolute values) indicates that great individual variation was present and stresses the need for periodic and personalized attention during the hyperventilation phase of controlled ventilation. The patterns in the assisted group were similar, with less end inspiratory driving pressures required.

#### TIME FACTORS IN VENTILATION

The patients were controlled only until effective spontaneous respirations in conjunction with assisted ventilation were possible. In all groups hyperventilation was terminated within 2 to 6 hr, being briefest with the machine cycle and longest in those suppressed with succinylcholine. Maintenance periods extending from 12 to 30 hr were employed until assisted ventilation could be resumed. The total time on control cycle ranged from 16 hr to 36 hr.

#### TRANSITION FROM CONTROLLED TO ASSISTED CYCLE

Controlled ventilation was continued until clinical and blood gas data indicated that the patient could be managed by assisted IPPB. The transition was made abruptly with blood gas monitoring. All survivors returned first to continuous and then to intermittent assisted ventilation until they could ventilate on their own. Generally, most patients could be placed on assisted ventilation on the first trial. Of the 16 patients on controlled ventilation 5 died during the procedure, leaving 11 (69%), who were transferred to assisted

**TABLE 2. Mean Arterial Blood Gas Data—Hyperventilation Phase***

| | Type of Ventilation | | | | | |
|---|---|---|---|---|---|---|
| | Improved Assisted | Controlled | | | | |
| | | Machine Cycle | Oxygen Suppression | Drug Control | | |
| | | | | Morphine Sulfate (MS) | Succinylcholine (SC) | MS and SC |
| Number of cases | 4 | 7 | 3 | 4 | 2 | 1 |
| $Paco_2$, *mm Hg* | | | | | | |
| Initial | 97.2 (60–129) | 80.3 (33–110) | 96.6 (67–99) | 95 (76–118) | 114 (88–140) | 88 |
| End | 61.5 (46–68) | 50.2 (37–78) | 67.0 (57–86) | 51.5 (39–62) | 57 (52–62) | 58 |
| Difference | −35.7 (−14 to −64) | −30.1 (+7 to −61) | −29.6 (−5 to −42) | −43.5 (−27 to −56) | −57.0 (−36 to −78) | −30 |
| pH | | | | | | |
| Initial | 7.21 (7.12–7.30) | 7.25 (7.05–7.44) | 7.22 (7.16–7.26) | 7.23 (7.18–7.31) | 7.11 (6.99–7.22) | 7.12 |
| End | 7.38 (7.33–7.42) | 7.41 (7.31–7.52) | 7.31 (7.28–7.33) | 7.38 (7.34–7.44) | 7.32 (7.25–7.38) | 7.26 |
| Difference | −0.17 (−0.09 to −0.14) | −0.16 (+0.09 to −0.29) | −0.09 (−0.05 to −0.12) | −0.15 (−0.07 to −0.20) | −0.21 (−0.16 to −0.26) | −0.14 |
| $Pao_2$, *mm Hg* | | | | | | |
| Initial | 156 (64–283) | 83.6 (50–154) | 42 (33–59) | 125 (62–275) | 56 (55–57) | 173 |
| End | 154 (67–310) | 282 (180–500) | 218 (132–380) | 187 (69–400) | 73.5 (57–90) | 101 |
| Difference | −2 (−141 to +182) | 198.4 (26 to 230) | 176 (73 to 346) | +62 (−75 to +400) | 17.5 (0 to 35) | −72 |
| Hour to decrease $Paco_2$ | 4:15 | 2:30 | 4:00 | 3:15 | 6:15 | 4:30 |
| Mean fall $Pco_2$/min, *mm Hg/min* | 0.14 | 0.16 | 0.12 | 0.21 | 0.15 | 0.11 |

* Numbers in parentheses indicate the range of values.

TABLE 3. Intermittent Positive-Pressure Breathing (IPPB) Dynamics During Hyperventilation: Tidal Volume, Respiratory Rate, Minute Volume Changes, and IPPB Pressure-Flow Observations (Mean Values)

| Type Ventilation | Tidal Volume | | Mean Increase | Respiratory Rate | | Mean Fall | Change in Minute Volume | Mean $Paco_2$ Change | IPPB Settings | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Initial | End | | Initial | End | | | | Pressure | Flow* |
| | *cc/min* | | % | *breaths/min* | | % | % | *mm Hg* | *cm $H_2O$* | |
| Improved assisted | 300 | 500 | 67 | 33 | 19.3 | 42 | −2.5 | −35.7 | 24.3 | 20 |
| Controlled | | | | | | | | | | |
| Machine cycle | 375 | 658 | 75 | 29 | 16.3 | 44 | −1.3 | −30.1 | 28.5 | 22 |
| Oxygen suppression | 260 | 433 | 67 | 32 | 21.0 | 34 | +9.3 | −29.6 | 30.7 | 21 |
| Drugs: | | | | | | | | | | |
| Morphine | 281 | 687.5 | 144 | 32.8 | 17 | 48 | +26.0 | −43.5 | 28.3 | 7.5 |
| Succinylcholine | 225 | 700 | 211 | 34 | 17 | 50 | +56.0 | −57.0 | 40 | 8 |
| Morphine and succinylcholine | 300 | 500 | 67 | 34 | 18 | 47 | −12.0 | −30.0 | 28 | 20 |

* Relative dial settings only.

ventilation and were maintained thereafter. From this latter group 3 died variable periods later while on a continuous or intermittent assisted schedule, leaving 8 (50%) survivors, who were entirely weaned off the respirator. In the 5 patients who died, no attempt was made to switch to assisted cycle because of the lack of clinical recovery despite improvement of $Po_2$ and $Pco_2$-pH parameters.

#### COMPLICATIONS

Complications (Table 4) caused by the procedure are difficult to evaluate because of associated diseases and therapy. A common problem was the fall in arterial blood pressure, temporarily related to high cycling pressures and often associated with gastrointestinal bleeding or shock. Arrhythmias and gastrointestinal bleeding were common. Falls in urinary output were observed in a few instances; but actual measurements were limited, and the true frequency is unknown.

Comparison of initial $Paco_2$ and pH with mortality rates revealed no significant differences for predicting outcome, and analysis of mortality versus time on controlled cycle indicated that survivors were on controlled ventilation for more prolonged periods.

TABLE 4. Complications During Controlled Ventilation

| Group | Complications* |
|---|---|
| Improved assisted | Reversible hypotension (1), needle dislodged into trachea (1). |
| Controlled: | |
| Machine cycle | Arrhythmia [multifocal PVCs (1), atrial fibrillation (1), cardiac arrest (1), ventricular tachycardia (1)], oliguria (1), gastric dilatation (1), transient cerebrovascular insufficiency (1), hypotension [myocardial damage (2), respirator (2)], gastrointestinal bleeding (2), shock (1). |
| Oxygen suppression | Respirator hypotension (1), shock with ventricular tachycardia (1), acute myocardial infarction (1). |
| Morphine | Reversible respirator hypotension (4), arrhythmia (PAT with block secondary to digitalis administration) (1), gastrointestinal bleeding (2), tracheal bleeding (1), transient oliguria (1), aspiration pneumonia (1), hypokalemia (1). |
| Succinylcholine | Reversible respirator hypotension (1), gastrointestinal bleeding (1). |
| Succinylcholine and morphine sulfate | Reversible respirator hypotension (1), oliguria (1). |

* Numbers in parentheses = numbers of patients; PVCs = premature ventricular contractions; PAT = paroxysmal auricular tachycardia.

MORTALITY DATA

There was no immediate mortality in the assisted group. In the control ventilation group the mortality was 50%, with 31% dying during the procedure and 19% during variable periods after the procedure. Of the 5 who died during controlled ventilation, 4 deaths occurred during the maintenance period and 1 during acute hyperventilation. Thus, of the total 19 patients on ventilatory assistance, the overall mortality was 41%.

## Discussion

Many patients in acute ventilatory failure will require some form of ventilatory assistance. Both intermittent and continuous assisted PPB have been used to improve alveolar ventilation, prevent oxygen respiratory depression, allow delivery of aerosol bronchodilators, improve intrapulmonary air distribution, and reduce the mechanical work of breathing (3). Conversely, IPPB is reported to be of no benefit or detrimental to patients in the chronic stable disease state or to those with superimposed acute ventilatory failure (4–7).

Thus, clinical situations arise where failure of assisted IPPB is associated with detrimental ranges of hypoxemia and acidosis. Possible causes for failure may be [1] inspiratory effort too weak to trigger the respirator; [2] patient too confused or agitated to accept or coordinate his breathing with the rate of the respirator; [3] increases in respiratory work (intrinsic or machine-induced); [4] tachypnea; [5] failure of blood gases to improve, despite what appears to be adequate cycling between patient and respirator; and [6] depression of ventilation secondary to oxygen administration. Controlled ventilation should be considered in such cases since prognosis cannot be determined and immediate support is necessary.

The body tank respirator for controlling ventilation aroused interest in 1951 as a result of the work of Boutourline-Young and Whittenberger (8) in which two moribund patients with emphysema received oxygen and $Paco_2$ was reduced. Favorable reports of the Engström and the Mörch volume-cycled ventilators document that effective hyperventilation and prolonged controlled ventilation are feasible in patients with chronic obstructive lung disease. To our knowledge no large series of patients with chronic obstructive lung disease has been reported in which conventional pressure-cycled respirators (IPPB) were employed for controlled ventilation (2, 9).

Some reports describing volume-cycled or tank respirators do not stress the problem of synchronization. This phenomenon may be rare, or most patients quickly learn to cycle smoothly. However, Marchand and Van Hasselt (10) described controlled ventilation as "last-resort treatment" for status asthmaticus in advanced ventilatory failure. Despite supportive care their patients remained restless and confused and would not synchronize with the Engström unless morphine (20 to 40 mg) was administered. Others (11, 12) also indicate the problem of synchronization and the need for paralytic or depressant agents with tank or volume-cycled respirators.

Studies with IPPB indicate that simple cycling of the patient with the respirator (machine cycle) may reduce hypercapnia (13). Automatic machine cycle is effective in severe crush thoracic injuries, yet anesthesia or d-tubocurarine was necessary for uncooperative patients (14). Neuromuscular blockade was necessary for successful ventilation in status asthmaticus (15). The use of depressants or sedatives has been discouraged (16). However, it may be physically impossible to manage certain individuals unless such agents are used.

Employing simple machine cycle, the continuous automatic Mark 7 or 8 IPPB unit was most successful in the group of patients who were already somnolent or depressed by intrinsic acidosis, hypoxemia, cerebral insults, drugs, or prior cardiac arrest. The remaining patients (59% of this series)

were agitated, confused, and uncooperative, and either 100% oxygen, succinylcholine, morphine, or all three were given to produce relaxation or muscle paralysis. Once one of these methods was selected, adequate reduction in $Paco_2$ and $[H^+]$ and improvement in $Pao_2$ were possible. Furthermore, the reduction in $Paco_2$ is rate-controllable, depending upon the IPPB features selected. It should be emphasized that, since effective machine synchronization frequently occurs and obviates the need for drugs, it should be attempted first. If synchronization fails, agent selection thereafter will depend upon the existing clinical situation. Succinylcholine in the severely agitated patient will cause rapid paralysis and must be followed immediately by automatic cycle. Those uncooperative patients sensitive to 100% oxygen may depress sufficiently so that no other drug is required. Morphine (or other depressant agents) may be titrated in small doses to reduce agitation. It was our general experience that, once effective control cycle was established, the patient remained coordinated and did not require very frequent or large doses for maintenance.

Care must be employed to avoid reducing the $Paco_2$ too rapidly (17). In only one instance did alkalosis of 7.52 occur, but with no side effects. Rapid correction of acidosis may reduce cerebral blood flow, shift the $O_2$ dissociation curve to the left, or produce hypotension or apnea. Mean times to reduce $Paco_2$ and $[H^+]$ ranged from 2½ hr to 6¼ hr. Whether such intervals are too rapid is difficult to document. Block and Ball (9) reported little difficulty in lowering $Paco_2$ over a 1-hr period. The four deaths in this study during hyperventilation may or may not be related to this phenomenon.

The fairly uniform reduction in $Paco_2$ during the hyperventilation period was accompanied by variable increases in minute ventilation. The mean falls in $Paco_2$ were associated with 34 to 50% reduction in respiratory rate and a 67 to 211% increase in tidal volume. Thus, appropriate management should focus on tidal volumes and respiratory rates and not on the minute volume alone. Factors responsible for the improved ventilation may include improved inspired gas distribution, reduced physiologic dead space, improved ventilation/perfusion relationships, more complete emptying of lung compartments (18), and less metabolic demands related to reduced work of breathing (19).

High inspiratory pressures required for optimal tidal volumes may reduce venous return to the left atrium and cardiac output, particularly in hypovolemia or shock, creating the difficulty of maintaining appropriate $Pao_2$ and $Paco_2$ levels and adequate cardiac output (20). Rapid reversibility of hypotension was noted in our patients once high pressures (40 cm $H_2O$) were reduced, and prolonged periods requiring high inspiratory pressures were not observed. This hypotension was not related to rapid reductions in $Paco_2$ since $Pco_2$ was lowered relatively gradually. Thus, during the procedure temporary reduction in venous return must be balanced by the need for immediate ventilation; once underlying bronchopulmonary and cardiac factors are controlled, reduction in high inspiratory pressures is possible. Similarly, an elevation of central venous pressure and a temporary reduction in urinary output may occur. These changes are reversed with lower IPPB driving pressures, and they should not be interpreted as ischemic acute renal failure.

The advantage of maintaining controlled ventilation after the hyperventilation phase has the following rationale: [1] further time for medical therapy and [2] reduction in work of breathing. The improvement in blood gases by mechanical ventilation will not concurrently improve the precipitating and underlying pathophysiologic factors. Controlled cycle may be continued until bronchospasm, secretions, infections, cardiac failure, etc. are controlled, and then

assisted IPPB will be effective. In this study maintenance periods varied with existing medical conditions, with 12 to 30 hr representing the minimum necessary for clinical stablization (total time, 16 to 36 hr). Whether such intervals are safe cannot be defined at present; however, artificial ventilation is possible for prolonged periods with no definable deleterious effects, providing oxygen toxicity is avoided (21, 9).

The total mortality in this series was 41%. The mortality during controlled ventilation was 50%, 31% dying during the procedure and 19% after the controlled cycling was terminated. Complications encountered were bacteremia (*Pseudomonas*—two cases), acute myocardial infarction (two cases), arrhythmia (three cases), gastrointestinal bleeding (one case), shock (four cases), and suspected pulmonary emboli (two cases). The experiences of others vary from complete success (22) to 50% (23) to 30 to 35% mortality (24). Variations in age, extent and type of pulmonary disease, methods of ventilation, and complicating medical conditions must affect such differences, as we have previously emphasized (25). The need for control cycle may influence mortality by [1] selection of more critically ill patients; [2] complications of prolonged control ventilation and immobilization (parenteral therapy, venous stasis with pulmonary emboli, infection, etc.); [3] effects of reduction in $Paco_2$ and $[H^+]$ and exposure to high respirator pressures; and [4] oxygen toxicity.

In the context of the type of patient we have described, this procedure is associated with a significant mortality. While a vigorous attitude is emphasized, controlled ventilation should be employed with care and awareness of the potential dangers and may be performed at the bedside only if constant physiological monitoring and medical and nursing care are available.

## Summary

Controlled mechanical ventilation with pressure-cycled intermittent positive-pressure breathing may be used to maintain adequate ventilation in patients with chronic obstructive lung disease in acute ventilatory insufficiency when optimal assisted intermittent positive-pressure ventilation fails to perform this function. In this series this modality was substituted for assisted ventilation because of poor patient cooperation, inability to reduce $Paco_2$ (or $[H^+]$) or improve $Pao_2$, oxygen depression of respiration, or progressive deterioration despite conventional assisted ventilation.

In 19 patients with 21 episodes of acute ventilatory failure, only 4 trials could be managed by assisted intermittent positive-pressure breathing. The remaining 17 episodes were managed by controlled ventilation in the following sequence, employing either endotracheal intubation or tracheostomy: automatic machine cycle, oxygen depression, and drug suppression. Simple machine cycle was effective in 41% of cases, particularly in the obtunded, comatose patient. With agitated, uncooperative patients oxygen depression in 18% and drug suppression (morphine or succinylcholine or both) in 41% were effective in establishing control.

Proper synchronization with the respirator was a prerequisite for controlled ventilation. Thereafter, effective ventilation could be achieved. Mean falls of 30 to 57 mm Hg in $Paco_2$, improvement in pH of 0.09 to 0.21 units, and adequate $Pao_2$ levels were observed in contrast to the lack of improvement during assisted ventilation. Such improvements were often associated with reduced respiratory rates and increased tidal volumes rather than with gross increases in minute ventilation.

High respirator pressures with moderate inspiratory and slow expiratory flow rates were employed with relative safety except for reversible hypotension encountered with high inspiratory pressures (25 to 40 cm $H_2O$). Complications such as gastrointestinal bleeding, cardiac arrest, arrhythmia, pulmonary emboli, and shock (cardiogenic

or bacteremic) had an adverse effect upon survival and may have been in part a consequence of the procedure. The overall mortality rate was 41%.

A hyperventilation period (2 to 6 hr), directed to eliminate dangerous hypoxemia and acidosis, and a maintenance period (12 to 30 hr), where control ventilation was continued, permitted full medical therapy to correct the acute precipitating factors. Thereafter, conventional assisted intermittent positive-pressure breathing and nonassisted breathing were possible. Whether ventilation is performed at the bedside or in an intensive care unit, constant physiological monitoring is mandatory.

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