Automatic Control of Arterial Carbon Dioxide Tension in Mechanically Ventilated Patients

Tyrone Fernando, John Cade, and John Packer

Abstract—This paper presents a method of controlling the arterial carbon dioxide tension of patients receiving mechanical ventilation. Controlling of the CO_2 tension is achieved by regulating the ventilator initiated breath frequency and also volume per breath.

Index Terms—Arterial carbon dioxide tension, automatic control, mechanical ventilation.

I. INTRODUCTION

MODERN lung ventilators are equipped with a variety of support techniques that provide the clinician various ways of artificially ventilating patients. Mandatory minute ventilation (MMV) is one such ventilation technique that interact with the patient in such a way that the delivery of a preset minimum minute volume (inhaled volume per minute) is guaranteed. The number of volume control breaths[1] per minute automatically increases or decreases to ensure the delivery of the requested mandatory or minimum minute volume. The concept of MMV appears attractive in that it provides the ability to increase or decrease the number of volume control breaths in a given time span. Thus, if the patient is apneic, an appropriate number of volume control breaths are delivered, and as the patient resumes spontaneous ventilation, the number of volume control breaths decreases gradually. This support scheme provides flexibility for a patient to automatically take up the work load of breathing and for the machine to gradually wind down at the same time.

For a patient ventilated in MMV, the clinician selects the level of minimum minute ventilation and this setting remains unaltered until a change is requested by the clinician. In the event of an increase or decrease in arterial CO_2 tension, the MMV setting may be decreased or increased respectively by the clinician to correct the disturbed arterial CO_2 tension. Regulating the MMV setting to maintain an acceptable arterial CO_2 tension could be performed automatically. This paper deals with such an automatic ventilation scheme that maintains the arterial CO_2 tension less than a specified level determined by a clinician.

T. Fernando is with the Department of Electrical and Electronic Engineering, University of Western Australia, Crawley, WA 6009, Australia (e-mail: tyrone@ee.uwa.edu.au).

J. Cade is with the Intensive Care Unit, Royal Melbourne Hospital, Parkville 3052, Australia.

J. Packer was with the Department of Electrical and Electronic Engineering, University of Melbourne, Parkville 3052, Australia (e-mail: jsp@ee.mu.oz.au).

[1]A volume control breath delivers a fixed volume specified by an operator.
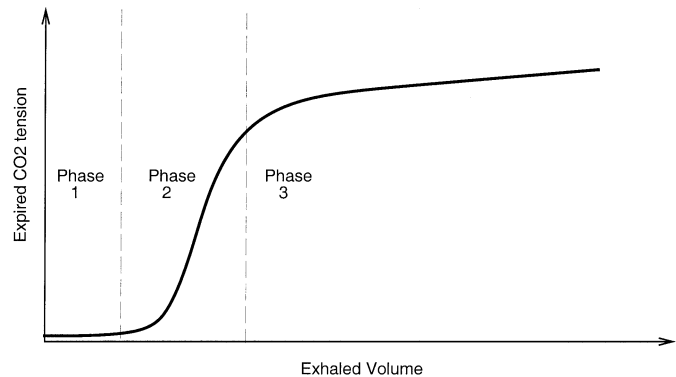

Fig. 1. Three phases of SBT-CO_2 plot.

II. RESPIRATORY DEAD SPACE

During respiration not all inhaled air reaches the alveoli, as part of it remains in the conducting portion of the airway. The region of the respiratory system where gas exchange does not take place is termed the respiratory dead space. By examining the CO_2 content of the exhaled air, Bohr formulated the following relationship [1]:

$$\frac{V_T - V_D}{V_T} = \frac{P_{\overline{E}_{CO_2}}}{P_{a_{CO_2}}} \tag{1}$$

where V_D is the dead space, V_T is the tidal volume (i.e., volume inhaled per breath), $P_{a_{CO_2}}$ is the arterial CO_2 tension, and $P_{\overline{E}_{CO_2}}$ is the average CO_2 tension in exhaled air. A typical trace of exhaled CO_2 tension against exhaled volume (i.e., SBT-CO_2 plot[2]) consists of three phases [1]: phase 1 comprising the CO_2-free gas from the airways, phase 2 a transition phase characterized by a S-shaped up-swing in the tracing representing the washout of airway with alveolar gas, and phase 3 comprising CO_2-bearing gas from the alveoli (see Fig. 1). The initial volume of expirate which includes the volume of fresh air down to the alveolar/fresh air interface represents the airway dead space. Airway dead space is, therefore, defined as the volume of air that does not reach the sites of gas exchange (i.e., alveolus). On the SBT-CO_2 plot this corresponds to an exhaled volume in phase 2. The exact location of this position on the SBT-CO_2 plot is reported in [2] and [3], and here it is taken as the exhaled volume corresponding to the point of inflection

[2]Single breath test of carbon dioxide.

on the SBT-CO$_2$ curve. Alveolar tidal volume is the part of inhaled volume that reach the alveoli

$$V_T^{alv} = V_T - V_D^{aw} \qquad (2)$$

where V_T^{alv} is alveolar tidal volume and V_D^{aw} is airway dead space. However, not all inhaled air that reaches the gas exchanging zone takes part in the mixing process. This fractional volume in alveoli that does not contribute to gas exchange is termed alveolar dead space. Combination of alveolar dead space and airway dead space defines the total dead space of the respiratory system

$$V_D = V_D^{alv} + V_D^{aw} \qquad (3)$$

where V_D^{alv} is alveolar dead space. The effective alveolar ventilation is the difference between alveolar tidal volume and alveolar dead space

$$V_A^{eff} = V_T^{alv} - V_D^{alv} = V_T - V_D \qquad (4)$$

where V_A^{eff} is the effective alveolar ventilation.

The alveolar dead space can be related to areas of SBT-CO$_2$ plot (see Fig. 2). In Fig. 2, the dashed vertical line falls through the point of inflection of the SBT-CO$_2$ curve. The areas X, Y and Z represents the following.

Area-X	Efficient part of ventilation.
Area-Y	A defect in CO$_2$ elimination that represents wasted ventilation due to alveolar dead space.
Area-Z	A defect in CO$_2$ elimination that represents wasted ventilation due to airway dead space.

The alveolar dead space can be defined using Area-X and Area-Y [1], [4]

$$V_D^{alv} = \frac{\text{Area-}Y}{\text{Area-}Y + \text{Area-}X} V_T^{alv}. \qquad (5)$$

Refer to the Appendix for a clinical trial on the measurement of airway deadspace and alveolar deadspace of a mechanically ventilated patient in the Intensive Care Unit (ICU) at the Royal Melbourne Hospital (RMH).

III. ARTERIAL CO$_2$ TENSION ESTIMATION

This section describes two algorithms [5] that can estimate arterial CO$_2$ tension of mechanically ventilated patients. The continuous estimation of arterial CO$_2$ tension is based on instantaneous, correlated measurements of ventilatory flow, airway pressure, and CO$_2$ tension of exhaled air measured by a capnograph. Although end tidal CO$_2$ tension may approximately follow the variations in arterial CO$_2$ tension, various studies have shown that utilization of end tidal CO$_2$ as a noninvasive monitoring substitute for trends in arterial CO$_2$ tension in critically ill patients may be misleading [6], [7]. The
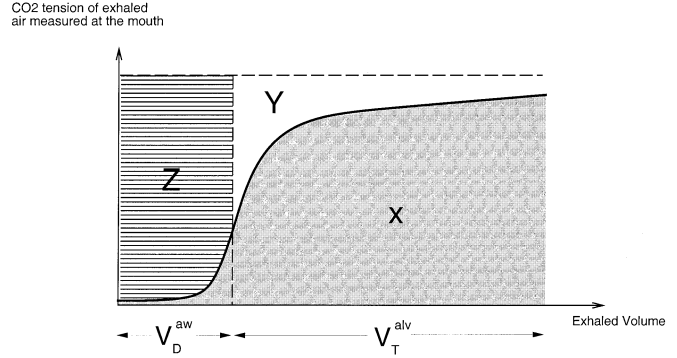

Fig. 2. SBT-CO$_2$ plot and the partition of regions X, Y, and Z.

algorithms described here are based on the efficiency of gas mixing in the lung as oppose to an algorithm based on end tidal CO$_2$ tension. The efficiency of gas exchange may be defined using Bohr's equation

$$\frac{V_D}{V_T} = 1 - \frac{P_{\overline{E}_{CO_2}}}{P_{a_{CO_2}}} = 1 - \text{efficiency} \qquad (6)$$

where V_D is the dead space, V_T is the tidal volume, $P_{a_{CO_2}}$ is the arterial CO$_2$ tension, and $P_{\overline{E}_{CO_2}}$ is the average CO$_2$ tension of exhaled air. The term $P_{\overline{E}_{CO_2}}/P_{a_{CO_2}}$ represents how well the inhaled tidal volume is used in the lungs for the purpose of gas exchange, and hence the term may be referred to as the efficiency of gas mixing

$$\text{efficiency} = \frac{P_{\overline{E}_{CO_2}}}{P_{a_{CO_2}}}. \qquad (7)$$

Experiments carried out on human subjects [5], [8] have found that the efficiency of gas mixing stays relatively constant for various levels of ventilation. Hence, if the efficiency of gas mixing is calculated upon performing a blood test and assumed constant in an individual patient for subsequent breaths following the blood test, the only unknown in (7) is the blood CO$_2$ tension, provided that the average partial pressure of CO$_2$ in exhaled air is known. Using basic gas laws, $P_{\overline{E}_{CO_2}}$ can be expressed as follows:

$$P_{\overline{E}_{CO_2}} = \frac{1}{V_T} \int_0^T \left(\frac{P_{CO_2}(t)(P_B - P_{H_2O})}{P(t) + P_B - P_{H_2O}} \right) \dot{V}(t) \, dt \qquad (8)$$

where $P_{CO_2}(t)$ is the partial pressure of CO$_2$ in exhaled air, $P(t)$ is mouth pressure, $\dot{V}(t)$ is the flow, V_T is the tidal volume, T is the exhalation time, P_B is the barometric pressure, and P_{H_2O} is the saturated water vapor pressure.

It should be noted that the accuracy of estimation can be affected by possible variations in efficiency of gas mixing that may occur. The problem of changes in efficiency can be overcome by incorporating a monitoring module to detect such changes and to request the operator to perform a new blood test to calculate the new efficiency of gas mixing thereby improving the accuracy of CO$_2$ estimation. The efficiency of gas mixing is considered to change if any of the following events occur three times.

- If minute volume increases by 800 ml/min or more and end-tidal CO_2 tension[3] does not drop by at least 2 mmHg.
- If minute volume decreases by 800 ml/min or more and end-tidal CO_2 tension does not increase by at least 2 mmHg.
- If minute volume increases by 800 ml/min or more and estimated CO_2 tension does not drop by at least 2 mmHg.
- If minute volume decreases by 800 ml/min or more and estimated CO_2 tension does not increase by at least 2 mmHg.
- If inspiratory time changes by more than 0.5 s.
- If peak airway pressure changes more than 10 cm H_2O.
- If previous PEEP[4] is less than 10 cm H_2O and changes by more than 5 cm H_2O.
- If previous PEEP is greater than or equal to 10 cm H_2O and changes by more than 2 cm H_2O.

Equations (7) and (8) together with a monitoring module for detecting changes in efficiency of gas mixing provide a framework for estimating arterial CO_2 tension. Algorithm 1 outlines a technique for estimating arterial CO_2 tension.

Algorithm 1

1.0   Step one: Based on initial simultaneous $P_{a_{CO_2}}$ and SBT-CO_2 trace

    1.1 Do a blood test and measure $P_{a_{CO_2}}$
    1.2 Use (8) to calculate $P_{\overline{E}_{CO_2}}$
    1.3 Calculate efficiency using Bohr's equation

$$\text{efficiency} = \frac{P_{\overline{E}_{CO_2}}}{P_{a_{CO_2}}}$$

2.0   Step two: Based on subsequent SBT-CO_2 trace without $P_{a_{CO_2}}$ measurement

    2.1 Use (8) to calculate $P_{\overline{E}_{CO_2}}$
    2.2 Estimate CO_2 tension using Bohr's equation

$$\text{Estimated } P_{a_{CO_2}} = \frac{P_{\overline{E}_{CO_2}}}{\text{efficiency}}$$

3.0   Step three: Applicable to measurement in Step two

    3.1 Has the efficiency changed?
        Yes: Go to step one
        No: Go to step two

Efficiency of gas mixing can also be defined using the areas of SBT-CO_2 curve [9]

$$\text{Efficiency} = 1 - \frac{V_D^{alv}}{V_T^{alv}} = \frac{\text{Area-}X}{\text{Area-}X + \text{Area-}Y}. \qquad (9)$$

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[3]End-tidal CO_2 tension is the partial pressure of CO_2 in a sample of exhaled air taken at the end of exhalation.

[4]PEEP stands for positive end expiratory pressure.

The algorithm proposed by [5] for CO_2 estimation is based on the efficiency expression derived by Fletcher. The method of estimation is shown in Algorithm 2.

Algorithm 2

1.0   Step one: Based on initial simultaneous $P_{a_{CO_2}}$ and SBT-CO_2 trace

    1.1 Do a blood test and measure $P_{a_{CO_2}}$
    1.2 Calculate Area-X and Area-Y using SBT-CO_2 plot
    1.3 Calculate efficiency using

$$\text{efficiency} = \frac{\text{Area-}X}{\text{Area-}X + \text{Area-}Y}$$

2.0   Step two: Based on subsequent SBT-CO_2 trace without $P_{a_{CO_2}}$ measurement

    2.1 Calculate Area-X from SBT-CO_2 plot
    2.2 Calculate V_T^{alv} from SBT-CO_2 plot
    2.3 Estimate Area-Y using

$$\text{Estimated Area-}Y = \left(\frac{1 - \text{Efficiency}}{\text{Efficiency}} \right) \text{Area-}X$$

    2.4 Estimate Arterial CO_2 tension using the following equation

$$\text{Estimated } P_{a_{CO_2}} = \frac{\text{Area-}X + \text{Estimated Area-}Y}{V_T^{alv}}$$

3.0   Step three: Applicable to measurement in Step two

    3.1 Has the efficiency changed?
        Yes: Go to step one
        No: Go to step two

It should be noted that the efficiency expression resulting from Fletcher's equation includes only alveolar dead space and alveolar tidal volume as opposed to total dead space and total tidal volume incorporated in the efficiency expression resulting from Bohr's equation. It has been reported that efficiency defined by Fletcher's equation is less sensitive to changes in ventilation than the efficiency defined using Bohr's equation [5]. Hence, it may be expected that incorporating the efficiency expression derived using Fletcher's equation for arterial CO_2 estimation produces more accurate readings of arterial CO_2 than incorporating the efficiency expression derived using Bohr's equation. The algorithm implemented by [5] claims an accuracy of ± 4 mmHg.

IV. CONTROL OF $P_{a_{CO_2}}$ BY REGULATING V_A^{eff}

The level of arterial CO_2 tension is dependent on the CO_2 production in cells and the magnitude of effective alveolar ventilation. A disturbance in CO_2 production or the magnitude of effective alveolar ventilation will alter the level of arterial CO_2
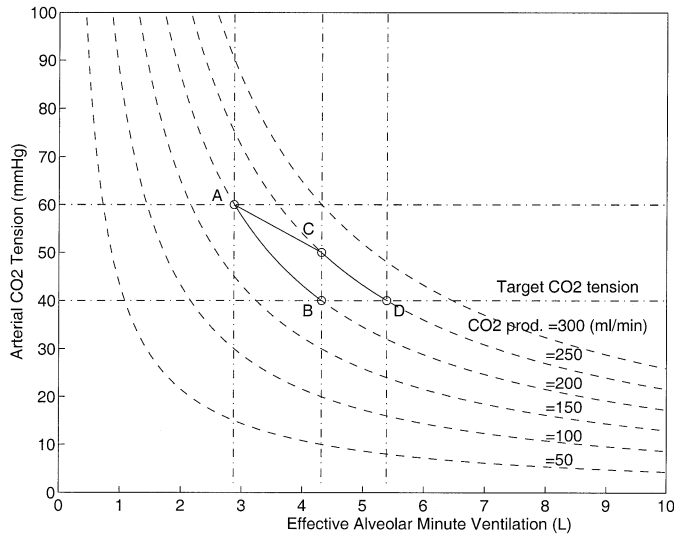
Fig. 3. One-step-ahead control performance.

Fig. 4. Oscillations of CO_2 production, arterial CO_2, and effective alveolar minute ventilation.

tension. The relationship between these variables are given by the alveolar air equation [10]

$$P_{a_{CO_2}} = \frac{0.863 * \dot{V}_{CO_2}}{\dot{V}_A^{eff}}. \tag{10}$$

Fig. 3 shows the inverse relationship between the blood carbon dioxide tension and the effective alveolar minute ventilation for various CO_2 production values.

A one-step-ahead control strategy may be adopted in the regulation of blood CO_2 tension. A one-step-ahead controller by definition attempts to push the control variable (i.e., $P_{a_{CO_2}}$) from the current value to the desired value in one step. The CO_2 controller takes corrective action every five minutes, which is also the sampling time of the controller. The performance of the controller can be explained by referring to the details of Fig. 3.

A patient with an unacceptable blood CO_2 tension of 60 mmHg with an alveolar minute ventilation of 2.8 l/min and CO_2 production of 200 ml/min can be represented at point A on the diagram. The controller in an attempt to push the patient's blood CO_2 tension from the present value to the desired value of 40 mmHg calculates a desired alveolar ventilation of 4.3 l. If the CO_2 production remains constant until the next sample (i.e., 5 min into the future), the patient descends down line AB and reaches the desired CO_2 level in one step. The one-step-ahead control law can be written as

$$^{new}V_A^{eff} = {}^{current}V_A^{eff} \frac{^{current}P_{a_{CO_2}}}{^{target}P_{a_{CO_2}}} \tag{11}$$

where $^{current}V_A^{eff}$ is the current effective alveolar ventilation, $^{current}P_{a_{CO_2}}$ is the current arterial CO_2 tension, $^{target}P_{a_{CO_2}}$ is the desired arterial CO_2 tension, and $^{new}V_A^{eff}$ is the calculated new effective alveolar ventilation to correct the current arterial CO_2 tension. The above control strategy is derived using (10) and assuming that $\dot{V}_{CO_2}$ is constant between sampling periods. However, if the CO_2 production changes (e.g., it increases from 200 mL/min to 250 mL/min), the patient instead of descending from point A to point B moves from point A to point C. In
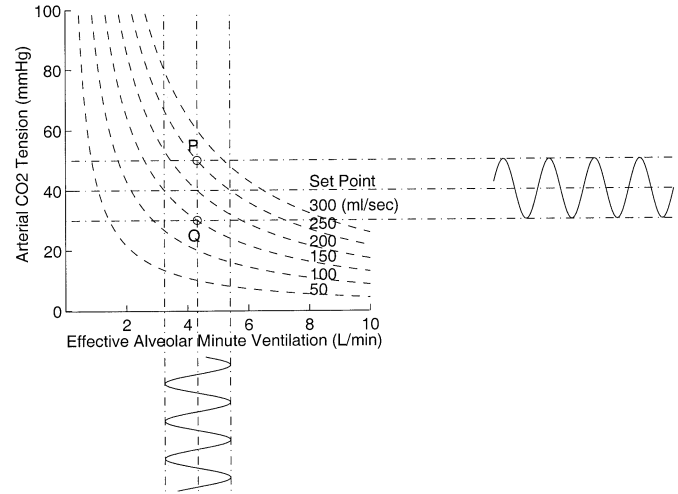
this situation, the target value is not reached in one step due to the disturbance in the CO_2 production; the controller again attempts to push the patient from point C to the new desired point D by calculating a new alveolar ventilation of 5.4 L. It is clear that desired performance of the controller can be guaranteed even under sudden changes in CO_2 production, provided it remains stable after the change. However, if the CO_2 production is unstable and oscillates about a mean CO_2 production value of $\dot{V}_{CO_2}$ with an amplitude of $\Delta\dot{V}_{CO_2}$, then from (10) it is clear that blood CO_2 tension oscillates about the desired CO_2 tension with an amplitude of $\Delta P_{a_{CO_2}}$ where

$$\Delta P_{a_{CO_2}} = 0.863 \frac{\Delta\dot{V}_{CO_2}}{\dot{V}_A^{eff}} \tag{12}$$

provided that alveolar ventilation is unchanged. Fig. 4 shows this scenario, as the CO_2 production oscillates between 150 to 250 mL/min for a constant effective alveolar minute ventilation of 4.3 L represents an oscillatory movement of a point between P and Q. It is clear that arterial CO_2 tension will oscillate between 30 to 50 mmHg as shown in the horizontal oscillations. If the magnitude and direction of CO_2 production oscillations are known, the controller can then suppress the oscillations in CO_2 tension by oscillating the alveolar minute ventilation with an amplitude of $\Delta\dot{V}_A^{eff}$ where

$$\Delta\dot{V}_A^{eff} = \dot{V} \frac{\Delta\dot{V}_{CO_2}}{\dot{V}_A^{eff}}. \tag{13}$$

The suppression only occurs when the CO_2 production oscillations are in phase with the effective alveolar minute ventilation oscillations. The vertical oscillations in Fig. 4 indicates how effective alveolar minute ventilation should oscillate in order to suppress the CO_2 tension oscillations. If the CO_2 production oscillations and the V_A^{eff} oscillations are out of phase then from (10) it is clear that the blood CO_2 tension will oscillate with amplitude

$$\Delta P_{a_{CO_2}} = 0.863 \frac{\Delta\dot{V}_{CO_2}}{\dot{V}_A^{eff}} \frac{2\dot{V}_{CO_2}}{\dot{V}_{CO_2} + \Delta\dot{V}_{CO_2}} \tag{14}$$
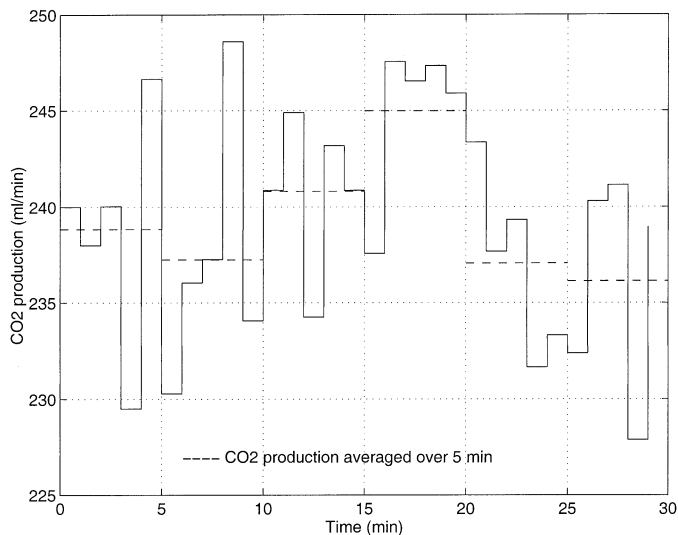
Fig. 5. Variation of carbon dioxide production over time. The online estimation was performed in ICU at RMH.
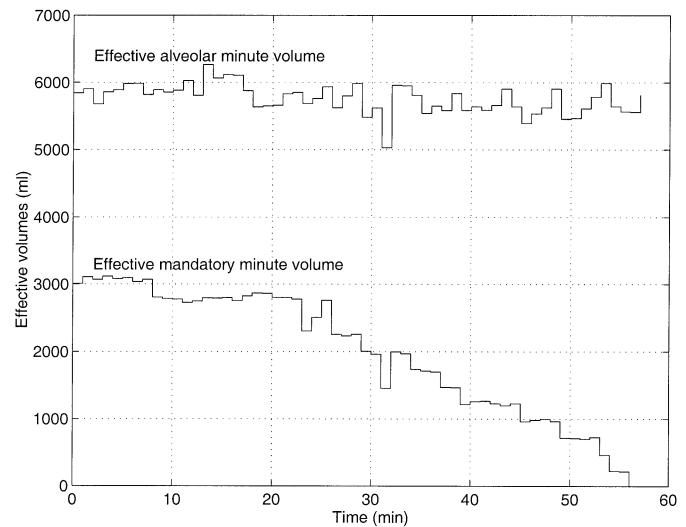

Fig. 6. Pattern of changes in effective mandatory minute volume and the corresponding changes in effective alveolar minute ventilation. All measurements were carried out at RMH.

which is greater than the CO_2 tension oscillations when there is no CO_2 control action present.

Since the disturbance in CO_2 production depends on body metabolism, it cannot be predicted, though it can be continuously measured. Controlling CO_2 tension when oscillatory disturbances are present in CO_2 production may intensify the existing oscillations. When consistent oscillations are present in the CO_2 production, the best performance the controller can achieve is by regulating the CO_2 tension using a running mean CO_2 production value instead of regulating it using the single CO_2 production value at the time of sampling.

From the above discussion it is clear that to control the arterial CO_2 level, it is necessary that the assumption the CO_2 production is relatively constant be valid and regulation of effective alveolar minute ventilation be possible. Fig. 5 shows a typical variation of CO_2 production over a period of 30 min. Here the average CO_2 production varies within a band of 10 mL and from (12) it is clear that this will cause the arterial CO_2 tension to oscillate with an amplitude $4.3/V_A^{\text{eff}}$ provided that effective alveolar minute ventilation is left unaltered. For V_A^{eff} in the range of 4 to 8 L, the expected arterial CO_2 tension oscillations have amplitudes ranging from 1.1 mmHg to 0.5 mmHg. Since the target $P_{a_{CO_2}}$ tension is generally set around 40 mmHg, a control system that can regulate the $P_{a_{CO_2}}$ level with in a band of 1.1 mmHg can be considered entirely acceptable. Such typical variation in CO_2 production is, therefore, not a performance limiting factor of the control system.

The minute ventilation for a mechanically ventilated patient depends on the patient's own respiratory drive and the number of mandatory breaths the operator specifies. The mandatory breaths contribute to a certain mandatory minute ventilation and the patient's respiratory drive contributes to a certain spontaneous minute volume. Both the spontaneous and mandatory minute volumes contain respiratory dead space volume, and subtraction of the two gives the effective minute volume carried by spontaneous and mandatory breaths. The controlling of

$P_{a_{CO_2}}$ assumes that regulating V_A^{eff} is possible. The effective alveolar minute ventilation is composed of the effective minute volume contributed by the spontaneous breaths (i.e., V_{sp}^{eff}) and also the effective minute volume contributed by the mandatory breaths (i.e., $V_{\text{man}}^{\text{eff}}$)

$$V_A^{\text{eff}} = V_{\text{man}}^{\text{eff}} + V_{sp}^{\text{eff}}. \qquad (15)$$

The regulation of V_A^{eff} can only be done by adjusting $V_{\text{man}}^{\text{eff}}$. To be able to set any desired V_A^{eff} by adjusting $V_{\text{man}}^{\text{eff}}$ it is necessary that these two quantities are directly related, any changes to $V_{\text{man}}^{\text{eff}}$ must be reflected in V_A^{eff}. Fig. 6 shows an example of changes in $V_{\text{man}}^{\text{eff}}$ and the corresponding changes in V_A^{eff}. From this it is clear that the variations in V_A^{eff} does not necessarily follow the those in $V_{\text{man}}^{\text{eff}}$. A change in $V_{\text{man}}^{\text{eff}}$ may alter both the V_{sp}^{eff} and V_A^{eff}. There is no direct relationship between $V_{\text{man}}^{\text{eff}}$ and V_A^{eff}, since alterations in $V_{\text{man}}^{\text{eff}}$ does not get reflected in V_A^{eff}. Hence, controlling the $P_{a_{CO_2}}$ level about a target value is not possible in such circumstances.

However, if a patient receives a certain number of mandatory breaths, then this ensures that the patient is guaranteed a certain level of effective alveolar minute ventilation, i.e.,

$$V_A^{\text{eff}} \geq V_{\text{man}}^{\text{eff}}. \qquad (16)$$

From (10) and (16), the following can be written:

$$P_{a_{CO_2}} = \frac{0.832\dot{V}_{CO_2}}{V_A^{\text{eff}}} \leq \frac{0.832\dot{V}_{CO_2}}{V_{\text{man}}^{\text{eff}}} = P_{a_{CO_2}}^{\text{max}}. \qquad (17)$$

From (17), it is clear that using $V_{\text{man}}^{\text{eff}}$ as the control signal and (11) as the control law can only maintain arterial CO_2 level below a certain maximum level (i.e., $P_{a_{CO_2}}^{\text{max}}$). Similarly, (16) and
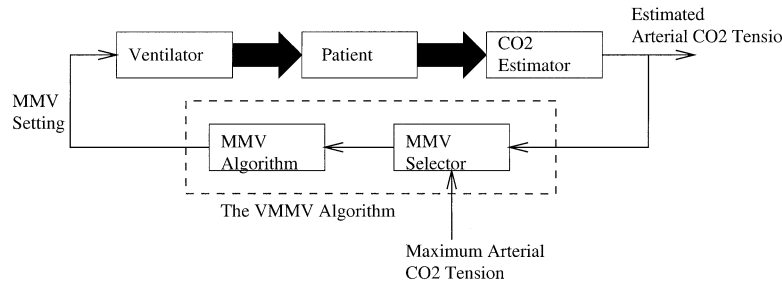
Fig. 7. Implementation of the VMMV algorithm.

(17) can be rewritten for a patient ventilated in the MMV mode as

$$V_A^{\text{eff}} \geq \text{MMV} - \dot{V}_D \qquad (18)$$

where $\dot{V}_D$ is the deadspace volume corresponding to MMV and

$$P_{a_{CO_2}} = \frac{0.832 \dot{V}_{CO_2}}{V_A^{\text{eff}}} \leq \frac{0.832 \dot{V}_{CO_2}}{\text{MMV} - \dot{V}_D} = P_{a_{CO_2}}^{\max}. \qquad (19)$$

It is clear that in this situation using the MMV setting as the control signal and (11) as the control law, again can only maintain arterial CO_2 tension below a certain maximum level (i.e., $P_{a_{CO_2}}^{\max}$). Hence, it may be stated that, while arterial CO_2 tension cannot be controlled about a set target level, it is possible to maintain it below a certain maximum level.

V. VMMV ALGORITHM

To maintain arterial CO_2 tension below $P_{a_{CO_2}}^{\max}$, V_A^{eff} must be regulated according to (11) periodically. The periodic implementation of V_A^{eff} can be done in the MMV mode by periodically selecting a MMV setting as follows:

$$^{\text{new}}\text{MMV} = {}^{\text{new}}V_A^{\text{eff}} + {}^{\text{new}}\dot{V}_D \qquad (20)$$

where $^{\text{new}}\dot{V}_D$ is the deadspace volume corresponding to $^{\text{new}}V_A^{\text{eff}}$. The new minute dead space volume can be estimated by assuming that minute dead space volume varies proportionally to minute volume [8]

$$\frac{^{\text{new}}\dot{V}_D}{^{\text{new}}\dot{V}_D + {}^{\text{new}}V_A^{\text{eff}}} = \frac{^{\text{current}}\dot{V}_D}{^{\text{current}}\dot{V}_D + {}^{\text{current}}V_A^{\text{eff}}}. \qquad (21)$$

Therefore

$$^{\text{new}}\dot{V}_D = {}^{\text{current}}\dot{V}_D \left(\frac{^{\text{new}}V_A^{\text{eff}}}{^{\text{current}}V_A^{\text{eff}}} \right)$$

$$= {}^{\text{current}}\dot{V}_D \left(\frac{P_{a_{CO_2}}^{\text{current}}}{P_{a_{CO_2}}^{\max}} \right). \qquad (22)$$

Using (20) and (22) the expression for $^{\text{new}}$MMV can be rewritten as

$$^{\text{new}}\text{MMV} = {}^{\text{new}}V_A^{\text{eff}} + {}^{\text{new}}\dot{V}_D$$

$$= \left(\frac{P_{a_{CO_2}}^{\text{current}}}{P_{a_{CO_2}}^{\max}} \right) \left({}^{\text{current}}V_A^{eff} + {}^{\text{current}}\dot{V}_D \right)$$

$$= \text{MV} \left(\frac{P_{a_{CO_2}}^{\text{current}}}{P_{a_{CO_2}}^{\max}} \right). \qquad (23)$$

Fig. 7 is a schematic of a patient ventilated in VMMV mode. Arterial CO_2 tension (rather than minute ventilation) serves as the input to the VMMV algorithm. The periodic MMV selection is based on (23). Based on the arterial CO_2 tension and minute ventilation, both averaged over 5–min intervals, the MMV selector outputs a new MMV setting every 5 min. The following is a clinical trial of a patient ventilated in the new VMMV mode.

A patient who had been receiving mechanical ventilation for the past 72 hs was chosen to trial on the VMMV mode. The patient was on a rate of 12 volume control breaths per minute, each carrying a tidal volume of 600 mL. A blood test was performed 4 min into the trial, and it revealed an arterial CO_2 tension of 41 mmHg. Based on the arterial CO_2 tension in this minute, a blood gas efficiency of 0.646 was calculated (refer to Section III for details of this calculation). The VMMV mode was not engaged until the 21st minute, until then only the CO_2 estimator was in operation. Within this time span, arterial CO_2 tension varied between 38.9–41.1 mmHg. When the VMMV mode was engaged a maximum arterial CO_2 tension setting of 41 mmHg was chosen as the input to the algorithm.

The minute volume, MMV settings, estimated arterial CO_2 tension, and spontaneous and volume control breaths per minute are shown in Fig. 8. By the 61st minute, the patient was receiving no volume control breaths and was breathing spontaneously. The minute ventilation then remained above the MMV setting constantly for another 20 min at which point the trial was terminated. A blood gas performed in the 78th minute revealed an arterial CO_2 tension of 36.7 mmHg. The estimated arterial CO_2 tension was 1.6 mmHg different from the value obtained from gas analysis. It is noted that throughout the trial, the MMV setting has changed at every 5-min interval, and the arterial CO_2 tension has remained below the set maximum limit.
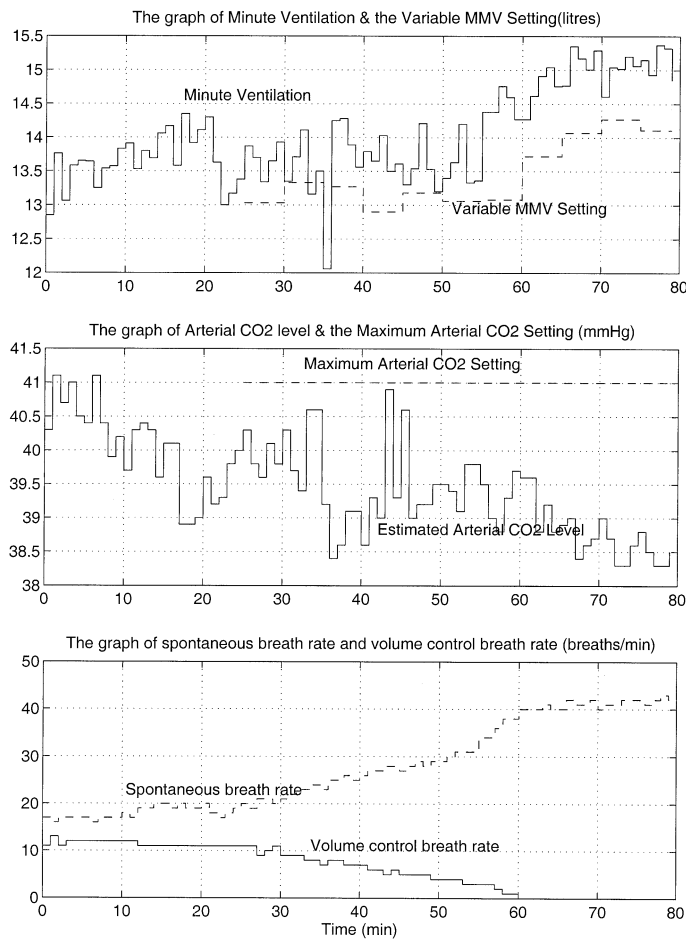
Fig. 8. Variations of minute volume, the MMV setting, the arterial CO_2 tension, spontaneous, and volume control breaths per minute. This VMMV trial was carried out in ICU at RMH.

The new ventilation mode has demonstrated the ability to encourage patient's spontaneous breathing while ensuring that the arterial CO_2 tension remained below the prescribed maximum settings as determined by the clinician.

The control system cannot raise the arterial CO_2 tension above the level achieved during spontaneous ventilation. In situations where arterial CO_2 tension needs to be increased, patient's spontaneous ventilation needs to be suppressed through sedative medication allowing the mechanical ventilator to "take over" in regulating effective alveolar ventilation and thereby controlling the arterial CO_2 tension. Tighter control of arterial CO_2 tension is possible if spontaneous breathing is suppressed and ventilation is controlled entirely by a ventilator.

VI. CONCLUSION

When a patient is ventilated in the VMMV mode, the clinician decides the maximum arterial CO_2 level. Once this decision is made, the ventilation scheme calculates a new MMV setting every 5 min (hence the name variable MMV) to ensure that arterial CO_2 level stays below the set limit. The ventilation mode while providing all features of the MMV mode can ensure that arterial CO_2 remains below a set limit.
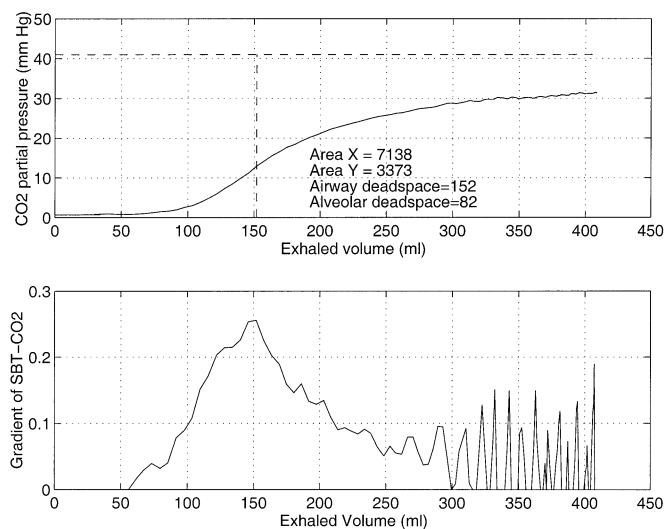

Fig. 9. SBT-CO_2 plot and the gradient of SBT-CO_2. The CO_2 partial pressure signal was obtained from a Hewlett Packard 78 356A capnograph and the flow signal was obtained from a PB-7200a ventilator. The flow signal was integrated to obtain volume. The measurements were made in ICU at RMH.

APPENDIX

To form a SBT-CO_2 trace, it is necessary to access the inspiratory flow waveform and CO_2 partial pressure of exhaled air. The inspiratory flow waveform was accessed from an analog port in the PB-7200a ventilator and the CO_2 partial pressure of exhaled air was accessed from an analog port of the HP-78 356A capnograph. Both analog signals were sampled at a frequency of 100 Hz and processed in an IBM-386 PC. The top graph of Fig. 9 shows a SBT-CO_2 trace. The gradient of this curve is shown in the lower graph of Fig. 9. The maximum peak in the gradient of SBT-CO_2 plot corresponds to the point of inflection of the SBT-CO_2 plot. The maximum peak in the gradient of SBT-CO_2 plot in this patient occurs when the exhaled volume is 152 ml. This point corresponds to the point of inflection of the SBT-CO_2 plot and, therefore, represents the airway dead space. The tidal volume of this breath is 408 ml and hence the alveolar tidal volume is $408 - 152 = 256$ ml. A blood test performed within this time revealed an arterial CO_2 tension of 41 mmHg. The Area-X and Area-Y for this breath was calculated to be 7138 ml mmHg and 3373 ml mmHg, respectively. Using (5) alveolar dead space is $(256 \times 3373)/(7138 + 3377) = 82$ mL. The total dead space of the respiratory system is the sum of alveolar dead space and airway dead space, $82 + 152 = 234$ mL.

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Tyrone Fernando received the B.E. (Hon.) and Ph.D. degrees from the University of Melbourne, Melbourne, Australia, in 1990 and 1996, respectively.

In 1995, he was a Visiting Lecturer in the Department of Electrical and Computer Engineering, Monash University, Melbourne. Since 1996, he has been with the Department of Electrical and Electronic Engineering, University of Western Australia, Crawley, where he is a Senior Lecturer. His main research interests include biomedical engineering, multidimensional systems theory, and robust control.

John Cade received the M.D. degree in 1968 and the Ph.D. degree in 1970, both from the University of Melbourne, Melbourne, Australia.

He has been the Director of Intensive Care at the Royal Melbourne Hospital, a University teaching hospital in Melbourne, Australia, for over 20 years. He is a Professorial Fellow in the Department of Medicine in the University of Melbourne. He has had a long-term interest in clinical, teaching, and research aspects of biomedical engineering, thromboembolism, infections, and ethics.

John Packer received the undergraduate degree in electrical engineering and the M.E. degree from the University of Adelaide, Adelaide, Australia.

He was with the Aeronautical Research Laboratories, Melbourne, Australia, in the field of aircraft and missile control systems. He was with the University of Melbourne until he retired in 1996. He was Chairman of the Department of Electrical and Electronic Engineering from 1978 to 1984 and Deputy Dean of the Faculty of Engineering from 1991 to 1994.