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## USING PHYSIOLOGICAL MODELS AND DECISION THEORY FOR SELECTING APPROPRIATE VENTILATOR SETTINGS

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**ABSTRACT. Objective.** To present a decision support system for optimising mechanical ventilation in patients residing in the intensive care unit. **Methods.** Mathematical models of oxygen transport, carbon dioxide transport and lung mechanics are combined with penalty functions describing clinical preference toward the goals and side-effects of mechanical ventilation in a decision theoretic approach. Penalties are quantified for risk of lung barotrauma, acidosis or alkalosis, oxygen toxicity or absorption atelectasis, and hypoxaemia. **Results.** The system is presented with an example of its use in a post-surgical patient. The mathematical models describe the patient's data, and the system suggests an optimal ventilator strategy in line with clinical practice. **Conclusions.** The system illustrates how mathematical models combined with decision theory can aid in the difficult compromises necessary when deciding on ventilator settings.

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**KEY WORDS.** mechanical ventilation, decision theory, decision support systems

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## INTRODUCTION

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Several systems have been developed to aid in the process of selecting appropriate ventilator settings in mechanically ventilated patients [1–6]. Those finding their way into clinical practice [3, 4] have focussed on using rules either to automate clinical protocols for particular ventilator modes [3], or to keep the patient within a “zone of comfort” during pressure support ventilation [4, 5].

Key to these rule-based systems is that they automate the heuristics of the clinician, controlling patient ventilation without providing a deep physiological picture of the patient's state. This may be appropriate in situations where the system controls ventilation every 5 minutes [4, 5] i.e., when the clinician is not at the bedside. However, when the clinician is present at the bedside a different sort of system might be required. One which, in addition to controlling patient ventilation, supports the clinician in understanding the patient's state from numerous clinical measurements, before a ventilation strategy is selected.

Decision support systems (DSS) based on physiological models may provide this deeper understanding. Using such models patient state is described by the parameters of the models. If the models are tuned to a particular patient, by estimating the parameters, they might be used to answer “what if” questions predicting the outcomes of different ventilation strategies. In addition, when

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physiological models are combined with utility theory [7], the expected utility of outcomes can be quantified and ventilator settings selected so as to maximize expected utility.

A few DSS have previously been developed for selecting ventilator settings based upon models and utility theory [8, 9]. However, the recent advances in this field have been in the application of rule-based systems, which do not exploit the large wealth of physiological knowledge in this field [4, 5]. In this paper a new DSS for optimising ventilator settings is presented. This system includes physiological models and utility functions in a decision theoretic approach to optimising ventilation. The purpose of this paper is to present the structure and function of the system and illustrate its use in a single clinical example. In the first section the DSS is described, including the mathematical models and utility functions used in the system. The second section illustrates how the system is used with an example of a patient ventilated following coronary artery bypass grafting surgery (CABG).

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## THE DSS

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Figure 1 illustrates the structure of the DSS for suggestion of optimal ventilator settings. Physiological models are used to simulate the effects of changes in ventilator settings on pressures and volumes in the lung, and the oxygenation and acid-base status of the blood.

Included in these physiological models are parameters describing lung function, metabolic, circulatory, and blood state. Parameters give a description of the patient's physiological status, which can be assumed to be relatively constant for model predictions. For example when describing gas exchanges abnormalities in the lung, pulmonary shunt and ventilation/perfusion (V/Q) mismatch might be considered better parameters than arterial oxygen pressure, having deeper physiological meaning and remaining relatively constant on changes in inspired oxygen fraction. Values of parameters are either directly measured or estimated from clinical data by using the models. Parameter estimation requires known values of the specific patient's ventilator settings and physiological model variables listed in the legend of Figure 1. The parameter estimation process is described later in the text. Values of parameters provide a clinical picture of the patient's state describing all clinical measurements, from which patient specific simulations of changes in ventilator settings can be performed.

Also represented in the system are mathematical functions quantifying clinical preference to the goals and side effects of ventilator therapy. These are described later

in the text, but include: sufficient oxygenation; minimising the risk of acidosis and alkalosis, and minimising the risk of ventilator induced lung injury due to barotrauma or oxygen toxicity. These functions are expressed as penalties. For any model simulated ventilatory strategy the total preference is expressed as the sum of the penalties for each of the simulated goals or side effects. A high total penalty indicates a reduced clinical preference toward the strategy.

All software has been written in JAVA including the mathematical models and penalty functions, optimisation code, and user interfaces. The system runs on a standard PC and is linked to a research database system built in our research group [10, 11], so that almost all data required to run the DSS can be automatically collected from routinely available intensive care equipment. Currently data can be collected from intensive care ventilators (Servo 300 and Servo i, Maquet, Solna, Sweden), an intensive care monitor (Siemens SC 9000; Siemens AG, Erlangen, Germany), and several capnographs and expiratory O<sub>2</sub> sensors (Oxigraf monitor; Oxigraf, CA, USA, COSMO; Respironics, CA, USA). Currently only values of blood gasses need to be entered into the system by hand in order to run the DSS. Data collection is performed via RS232 connection using the JAVA communications class. Model simulations run within one second.

The DSS may now be used in 3 ways: (1) to estimate patient specific parameters providing a clinical picture of the patient's state describing all measurements; (2) to answer "what if" questions, simulating the effects of different ventilator strategies; and (3) to find the optimal ventilatory strategy, a process which occurs automatically in the DSS using repeated simulations to find the ventilatory strategy which gives the minimum total penalty.

### *Mathematical models in the DSS*

This section describes the physiological models and mathematical functions describing clinical preference. For the physiological models a detailed description of the mathematics will be omitted, as these have been published previously [12–14]. The models describing clinical preference will be described, as these have not been published previously.

#### *Mathematical model of oxygen transport*

This model describes transport of oxygen in the lungs, blood and to the tissues, and has previously been published [12]. In the model the lung is divided into two compartments involved in gas exchange each with different ventilation and perfusion. The ventilation of each compartment is described as a fraction of the total alveolar ventilation (V<sub>A</sub>), e.g., fA<sub>2</sub> to the compartment labelled 2

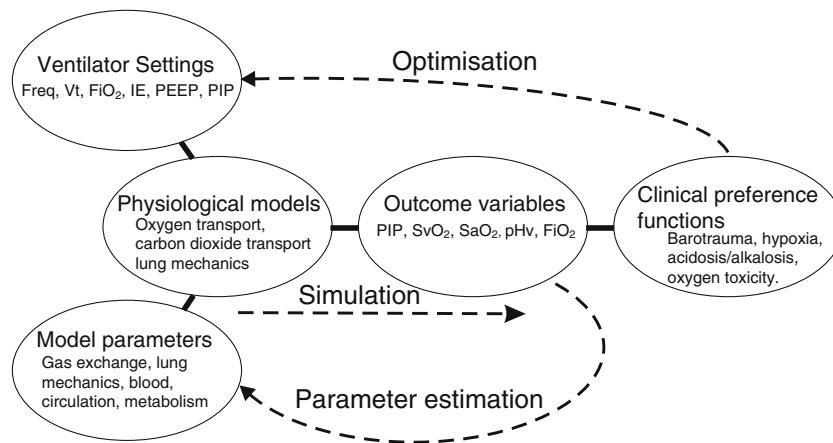


Fig. 1. The structure of the decision support system. Ovals illustrates components of the system, which includes ventilator settings ( $f$ ,  $V_t$ ,  $FiO_2$ , I:E-ratio, PEEP and PIP), model parameters (shunt,  $fA_2$ ,  $V_d$ , compliance, DPG, Hb, COHb, MetHb, temp,  $Q$ ,  $VO_2$  and  $VCO_2$ ), physiological models and their variables ( $F_{et}CO_2$ ,  $F_{et}O_2$ ,  $SaO_2$ ,  $PaO_2$ ,  $PaCO_2$ ,  $pHa$ ,  $SvO_2$ ,  $PvO_2$ ,  $PvCO_2$ , and  $pHv$ ), those variables selected as surrogate outcomes (PIP,  $SvO_2$ ,  $SaO_2$ ,  $pHv$  and  $FiO_2$ ) and functions describing clinical preference (barotrauma, hypoxia, acidosis–alkalosis, oxygen toxicity). The system can be used to perform parameter estimation, simulate the effect of a change in the ventilator settings and to generate a suggestion of “optimal” ventilator settings, as described in the text.

in Figure 2, where  $V_A$  is the total ventilation ( $V_T$ ) excluding anatomical dead space ventilation ( $V_D$ ). The perfusion of lung compartments is described as a fraction of the total perfusion ( $Q$ ) excluding the fraction of flow due to shunting of pulmonary blood ( $f_s$ ), e.g.,  $f_2$  to the compartment labelled 2 in Figure 2.  $f_s$  represents the fraction of blood flowing passed the lungs without being involved in gas exchange. Gas exchange abnormalities are represented using parameters describing  $V_D$ ,  $V/Q$  mismatch and  $f_s$ . By setting  $f_2 = 0.9$ ,  $V/Q$  mismatch is described by values of  $fA_2$  which describes the fraction of ventilation ( $fA_2$ ) to a compartment receiving 90% of the non-shunted blood flow. A value of  $fA_2 = 0.9$  would then be consistent with 90% of the alveolar ventilation going to 90% of the non-shunted perfusion, values of  $fA_2$  lower than 0.9 indicating a  $V/Q$  mismatch.

Included in the model of oxygen transport is an implementation of the oxygen dissociation curve (ODC) [15], which describes the relationship between oxygen pressure ( $PO_2$ ) and haemoglobin oxygen saturation ( $SO_2$ ) in the blood. The position of the ODC is shifted depending on changes in  $PCO_2$ , temperature, carboxyhaemoglobin (COHb), methaemoglobin (metHb), pH and the concentration of 2,3 diphosphoglycerate (DPG). All values except DPG can be measured in a blood sample. DPG can therefore be used as a parameter describing the position of the ODC. Values of the DPG parameter can be estimated from a blood sample with sufficiently low oxygen saturation. Also included in the oxygen model are parameters describing oxygen consumption ( $VO_2$ ) and cardiac output ( $Q$ ).

To estimate values of shunt and  $V/Q$  mismatch requires fitting this model to measurements of ventilation and arterial oxygenation at 4–6 different levels of inspired oxygen fraction, in a procedure taking 10–15 min [16]. When values of shunt and  $V/Q$  mismatch have been estimated the model can be used to predict arterial oxygenation for different levels of  $FiO_2$ .

The model of oxygen transport described here has been shown to fit the data of numerous patient groups [12, 17–21] and has been shown to have a structure complex enough to reproduce results of the reference experimental technique for measuring gas exchange [22]. In addition it has been shown that reasonable estimates of  $f_s$  and  $fA_2$  can be obtained quickly from routinely available clinical data [18].

The oxygen model can be used in two ways: as a diagnostic tool, estimating patient specific values of shunt and  $V/Q$  mismatch; and to predict the effect on oxygenation of adjusting ventilator settings.

#### Mathematical model of carbon dioxide transport

This model describes carbon dioxide storage and transport in the lungs, blood, interstitial fluid and tissues, and has been published previously [13, 14]. In the lungs carbon dioxide transport is described, like oxygen transport, by a two compartmental model with the same values of parameters describing gas exchange abnormalities, i.e., anatomical dead space,  $V/Q$  mismatch and shunt (Figure 2). In the blood  $CO_2$  storage is represented as a set of reaction equations describing the acid–base chemistry of the blood [13]. Significant stores of  $CO_2$  are present in

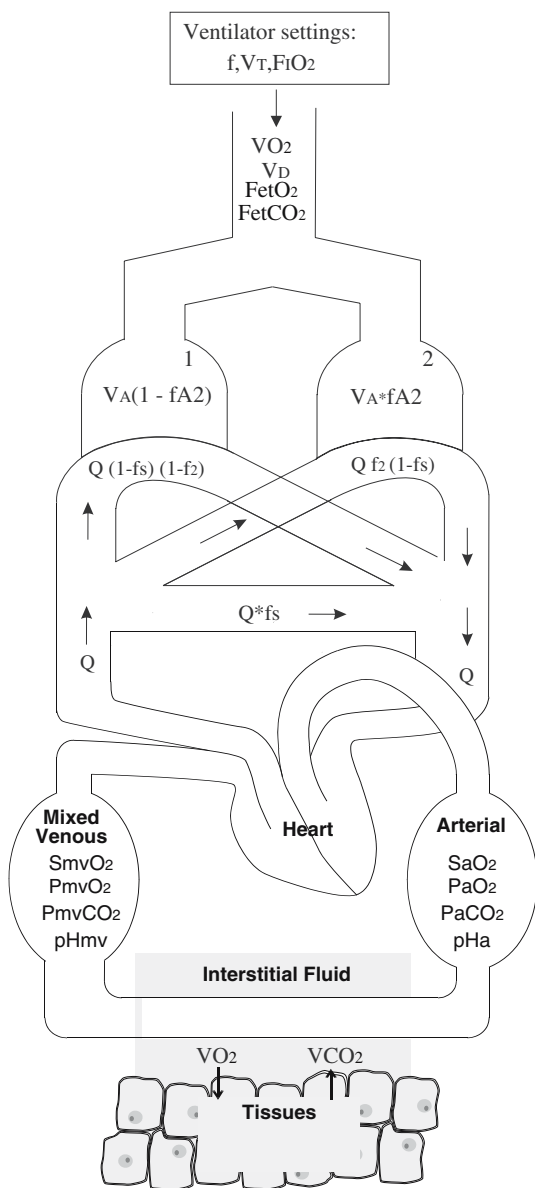


Fig. 2. The structure of the mathematical models of oxygen and carbon dioxide transport.

the interstitial fluids and tissues. The model therefore includes equations describing the acid base chemistry of the interstitial fluid and tissues [14]. Also included in the CO<sub>2</sub> model are parameters describing CO<sub>2</sub> production (VCO<sub>2</sub>) and Q.

The models of acid–base chemistry and CO<sub>2</sub> transport used here have been verified against experimental data reported in the literature [13, 14]. When simulating normal ventilation using the model, it has been possible to reproduce normal conditions in the lungs, arterial and venous blood, interstitial fluid and tissue. For use in the DSS it is necessary to simulate the effects of varying

ventilation volume on acid–base and CO<sub>2</sub> levels. In doing so it has been possible to reproduce changes in changes in end tidal CO<sub>2</sub> seen in the experimental literature on varying ventilation volume [14].

For known values of gas exchange parameters the CO<sub>2</sub> model can be used to simulate steady-state conditions of the acid–base status of blood for different ventilatory minute volumes [14]. Since tissue stores of CO<sub>2</sub> are large [14] the effects of changing ventilation on acid–base chemistry do not reach a steady state until 0.5–1 h after the change in ventilation. The simulations performed as part of the DSS, therefore, reflect the values of acid–base chemistry and CO<sub>2</sub> levels 0.5–1 h following a change in ventilatory minute volume.

*Mathematical model of lung mechanics*

The current model of lung mechanics included in the system is very simple. This model describes the relationship between peak inspiratory pressure (PIP) and tidal volume as a one compartmental model with a constant value of compliance representing the whole lung. The compliance (ml/cm H<sub>2</sub>O) is described as the change in volume, i.e., VT, divided by the corresponding change in pressure, i.e., PIP minus positive end expiratory pressure (PEEP).

Assuming a constant value of compliance, the model can be used to predict the effect on PIP of changing VT assuming a volume controlled ventilator strategy. In some patients this assumption may be inappropriate due to, amongst other factors, volume dependent compliance and the effects of airway resistance, this assumption is considered further in the discussion.

*Mathematical functions describing clinical preference*

The system includes mathematical functions, which describe clinical preference towards the goals and side effects (outcomes) of mechanical ventilation (Figure 3). In classical decision theory [7] these functions are called utility functions, where the function describes a preference towards a specific outcome; or penalty functions, where the function describes a dislike towards a specific outcome. Penalty functions are associated with hypoxaemia, acidosis and alkalosis, risk of oxygen toxicity and absorption atelectasis, and barotrauma. Each of these goals and side effects is associated with a surrogate outcome variable, values of which are obtained from either the result of model simulations, or the ventilator. In this system the risk of acidosis/alkalosis is represented by venous pH level, and risk of oxygen toxicity and absorption atelectasis is represented by FiO<sub>2</sub> level. PIP level represents risk of barotrauma, as this has shown to correlate positively with mortality [23]. The penalty associated with the risk of barotrauma is scaled with

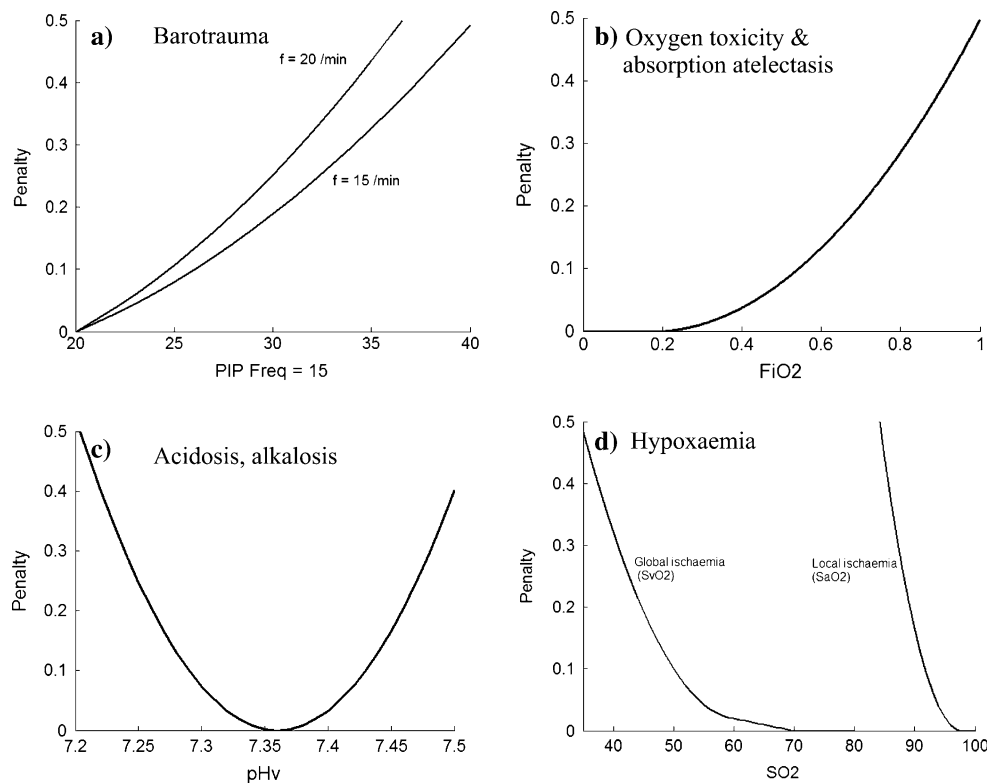


Fig. 3. The penalty functions included in the DSS. The penalty function for barotrauma is based on PIP (Figure 3a). Individual penalty curves are representing penalties incurred for different respiratory frequency ( $f$ ) with two example curves  $f=15$  breaths/min and 20 breaths/min illustrated here. For all curves PIP below or equal to 20 cmH<sub>2</sub>O has a penalty of 0. In addition a small penalty is added to the barotraumas penalty for respiratory frequencies greater than 15. The penalty function for oxygen toxicity and absorption atelectasis is defined as a function of  $FiO_2$  (Figure 3b).  $FiO_2$  equal to 0.21 has been defined to have penalty of 0. The penalty function for insufficient oxygenation (hypoxaemia) is represented as the sum of two functions where  $SvO_2$  represent global ischaemia and  $SaO_2$  local ischaemia:  $SvO_2$  equal to 70% has been defined as a global ischaemia penalty of zero, and  $SaO_2$  equal to 98% has been defined as a local ischaemia penalty of zero. The penalty function for acidosis and alkalosis is based on mixed venous pH (Figure 3c). Mixed venous pH equal to 7.36 has been defined to have a penalty of 0.

respiratory frequency ( $f$ ), such that higher frequencies at the same pressure incur a greater penalty. In addition a small penalty is added for respiratory frequencies greater than 15. This is necessary to enable an increasing barotrauma penalty with increasing respiratory frequency even if PIP is less than 20 cmH<sub>2</sub>O. Hypoxaemia is represented as the sum of two functions:  $SvO_2$  is used to represent the risk of global ischaemia as it represents the net effects on the whole body of oxygen delivery and consumption;  $SaO_2$  is used to represent two factors: local ischaemia, which may occur with low arterial oxygenation even if whole body venous oxygen levels appear normal; and to represent the increased load on the heart, whereby an increased cardiac output may be required to maintain oxygen delivery if arterial oxygen saturation is reduced.

The shape of each penalty function defines the relationship between an outcome and the associated penalty. In the DSS these shapes have been derived from input provided by a domain expert. These functions are then scaled, such that the subjective preference toward a par-

ticular outcome is the same on all graphs. This means that the total penalty can be represented as a sum of the individual functions illustrated in Figure 3. In the current version of the DSS the scaling procedure was based on the input from a domain expert. Scaling was performed by generating 20 test case patients. The domain expert was then asked to suggest a ventilation strategy for these test patients. The utility functions were then scaled such that the decision system behaved as the expert, suggesting similar settings for the ventilator. It is important to note that these functions are extremely subjective and that different functions may be defined for different clinicians or intensive care specialities. This point will be addressed further in the discussion.

## USING THE DSS – AN EXAMPLE

This section illustrates the use of the DSS with the aid of a clinical example of a postoperative coronary artery bypass

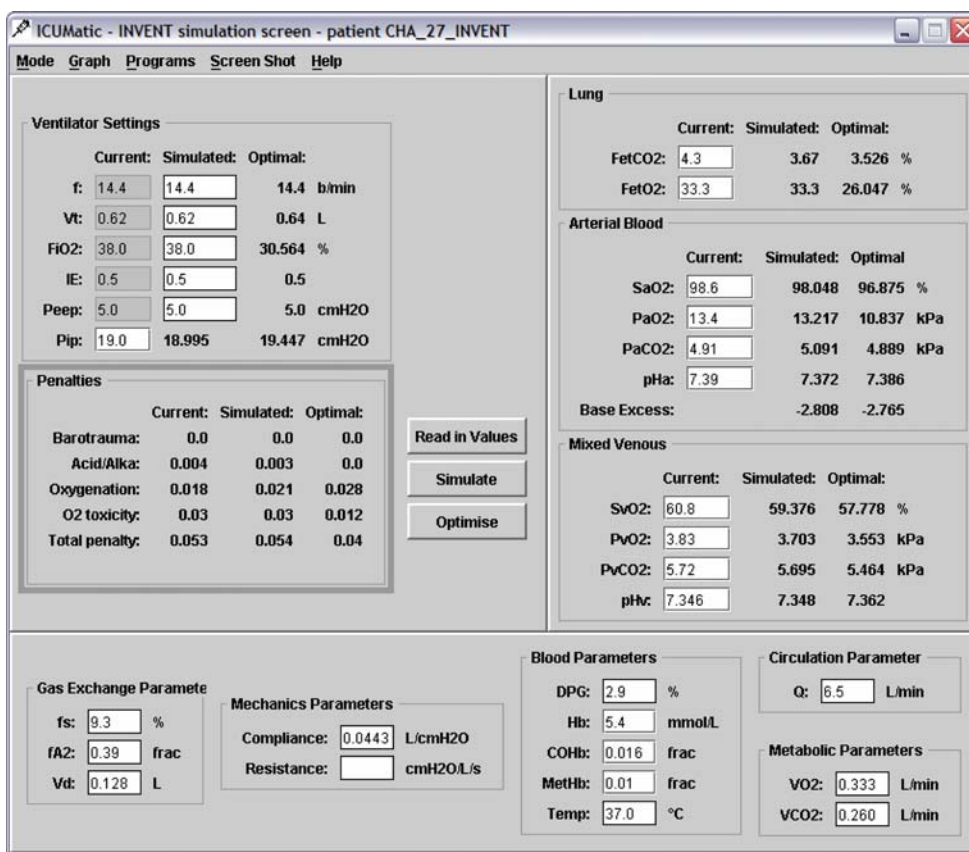


Fig. 4. Illustrates the user interface of the decision support system. This interface is divided into 3 sections. On the left hand side the ventilator settings and the associated penalties are displayed as current, simulated and optimal. The right hand side displays data describing the current, simulated and optimal values of variables describing the lungs, arterial and venous blood. The bottom of the screen displays patient specific parameters organised according to organ systems. The data of a single post-operative cardiac patient is included for illustration.

patient residing in the intensive care following surgery. The application of the system is illustrated by describing the user interface and in doing so it is highlighted how: the system can be used to help interpret patient data in terms of parameters describing lung function, blood state, circulatory state and metabolic state; how the quality of these estimated parameters can be verified; and how the system can be used to suggest ventilator settings which balance the conflicting goals when deciding upon appropriate mechanical ventilation.

Figure 4 illustrates the user interface of the DSS including the data of 1 postoperative CABG patient. The interface is divided up into three sections, the left hand side (LHS), right hand side (RHS) and bottom of the screen. In the LHS and RHS the majority of the variables have three different values in 3 columns, which represent respectively the measured value (Current), inputs or outputs from model simulations (Simulated), and optimal values calculated by the system by minimising the total penalty (Optimal). The LHS contains the ventilator

settings, penalties and function buttons of the system. These function buttons are clicked when the user wishes to perform a simulation, or to find the “optimal” ventilator settings, i.e., those that incur the least predicted penalty. The RHS contains data describing the lung, arterial blood and venous blood. Across the bottom of the screen are patient specific parameters. These are organised according to organ system and include parameters describing lung gas exchange, lung mechanics, blood, circulatory status, and metabolic status. These parameter values are measured or estimated and assumed to be constant for any simulated ventilator setting, an assumption that will be discussed later.

The system is used as follows in three steps. First, the physiological models are fitted to the patient data, so as to estimate patient specific values of the physiological models’ parameters included in the bottom of the interface screen (Figure 4). The quality of the estimated patient specific parameters can then be assessed from how well the mathematical models included in the system fit the clinical

data. These steps are technical by nature and can be run automatically without the clinician being interested in the technicalities of the model fitting. Upon estimation of parameters the clinician can now use the system for decision support. “What if” questions can be asked by inputting values of possible new ventilator settings into the system, which then performs simulations. The computer system can also be requested to automatically find suggestions for appropriate values of ventilator settings for the patient based upon the physiological models and penalty functions. Each of these steps is now described.

#### *Step 1: Calculation and estimation of patient specific values of the physiological models’ parameters*

Data is processed in order to estimate patient specific values of all the parameters used in the physiological models, i.e., parameters describing lung gas exchange and mechanics (shunt,  $fA_2$ ,  $V_d$ , compliance); blood (2,3 diphosphoglycerate (DPG), haemoglobin (Hb), carboxyhaemoglobin (COHb), methaemoglobin (MetHb), temperature (Temp)), circulatory parameters (cardiac output (Q)), and metabolic parameters (oxygen consumption ( $VO_2$ ), carbon dioxide production ( $VCO_2$ )). The fitting procedure depends to some degree on the type of patient and hence the quantity and quality of data collected. The post-operative CABG patient illustrated in Figure 4 had data collected from a pulmonary arterial (PA) catheter, a capnograph and an end tidal oxygen monitor. The PA catheter enables measurement of mixed venous blood gases and Q. For a large fraction of the ICU patients a PA catheter is not present. As such estimation of many of the patient specific parameters becomes more difficult. In these cases Q might be measured by other techniques [24], or simulated using models [25]. Mixed venous gases might be estimated from central venous [26], although central venous do not always correlate well with mixed venous [27]. Apart from measurements of blood gases, all these data can be collected automatically by the DSS.

#### *Blood parameters*

For blood, values of Hb, COHb and MetHb are measured in the venous blood. For each patient the mixed venous blood sample is used to estimate the position of the ODC. This is performed by estimating a value of DPG, which enabled the ODC to intersect the measured mixed venous values of  $PvO_2$  and  $SvO_2$ .

#### *Metabolic parameters*

$VO_2$  is calculated using the Fick equation, i.e.,  $VO_2 = Q (CaO_2 - CmvO_2)$ , where  $CaO_2$  is the concentration of oxygen in arterial blood and  $CmvO_2$  is the oxygen concentration in mixed venous blood.  $CaO_2$  and  $CmvO_2$  are

calculated respectively from the measured oxygen pressure ( $PO_2$ ) and saturation ( $SO_2$ ) in the arterial and venous blood from  $\alpha_{O_2} PO_2 + eHb SO_2$ , where  $\alpha_{O_2}$  is the solubility coefficient for oxygen in blood at 37°C ( $\alpha_{O_2} = 0.01$  mmol/l/kPa), and eHb is the effective haemoglobin concentration after subtracting the concentrations of COHb and MetHb from the measured concentration (Hb).  $VCO_2$  was estimated to enable the best possible fit of the models to measured arterial and venous blood gasses. To check whether the values of  $VCO_2$  estimated were reasonable a respiratory quotient (RQ) is calculated, i.e.,  $RQ = VCO_2/VO_2$ . Values of RQ should lie within 0.7–1.0.

#### *Gas exchange parameters*

Anatomical dead space ( $V_d$ ), is calculated using the alveolar air equation, i.e.,  $VO_2 = f (V_t - V_d) (FiO_2 - Fe'O_2)$ , using the measured end tidal oxygen fraction ( $Fe'O_2$ ) and the calculated  $VO_2$  from the Fick equation. Values of shunt and  $fA_2$  are estimated using the data obtained from the previously described system [16], together with measured values from the arterial blood sample (Hba, MetHba and COHba), the estimated DPG, and the calculated  $VO_2$  and  $V_d$ .

#### *Lung mechanics parameters*

Dynamic compliance is calculated dividing the ventilatory tidal volume ( $V_t$ ) by the change in pressure, i.e., PIP minus PEEP. The value of resistance is not calculated, as the patient was ventilated using the pressure regulated volume controlled mode of the ventilator and pause pressure was not available.

#### *Step2: Evaluating the quality of the model fit*

Following calculation and estimation of patient specific values of the physiological models’ parameters an evaluation of the quality of the model fit can be performed. Parameter values are displayed at the bottom of the user interface and the current ventilator settings ( $V_t$ ,  $FiO_2$ ,  $f$ ) are displayed in the current column of the LHS. By inputting the same values of ventilator settings into the simulated column and clicking the “Simulate” button a model simulation is performed. The results of the simulation are presented in the simulated column if the RHS.

Current and simulated values (first and second column of RHS) can be compared. Differences between these would indicate errors either in data collection or the parameter estimation procedure. Similar values, as seen for the patient illustrated in Figure 4, indicate that in this case the physiological models provide a good description of the patient’s data.

### *Step 3: Performing “what if” questions and finding the optimal ventilator settings*

Following calculation of parameter values and evaluation of the quality of model fit, processes that can occur automatically, the DSS can then be used to provide clinical decision support. The clinician can type possible new ventilator settings into the Simulated column of the LHS of the interface, and by clicking the “Simulate” button, the system will calculate all variables and penalties for these new settings, these being displayed in the Simulated columns of Figure 4. In addition the system can be requested to find the ‘optimal’ ventilator settings of  $\text{FiO}_2$ ,  $f$  and  $V_t$ , i.e. those that incur the least predicted penalty. By clicking the “Optimise” button the DSS automatically searches through combinations of  $\text{FiO}_2$ ,  $f$  and  $V_t$  using a gradient descent method, finding the values resulting in minimum total penalty. Currently the models cannot be used to simulate the effects of changes in PEEP or I:E ratio, and these values are fixed to those selected by the clinician. For the patient illustrated in Figure 4 the DSS suggests optimal ventilator settings such that  $\text{FiO}_2$  was reduced from 38.0% to 30.6%, with a small, almost negligible, increase in tidal volume. These settings gives an ‘optimal’  $\text{SaO}_2$  of 96.7%, and hence reduction in the total penalty from 0.054 to 0.040. This reduction is due to a lower oxygen toxicity penalty, even though the hypoxia penalty is increased. In this case the system suggested that a  $\text{FiO}_2 = 38.0\%$  was not necessary to adequately oxygenate the patient.

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## **DISCUSSION**

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This paper describes a computer system for supporting the clinician in the process of setting the ventilator for patients undergoing mechanical ventilation. The DSS contains models of oxygen and carbon dioxide transport and lung mechanics in combination with mathematical functions describing clinical preference towards the goals and side effects of mechanical ventilation.

To support the clinician the models can be used to estimate patient specific parameters describing lung function, blood, circulation and metabolism. These models and the parameter values enable the clinician to simulate the consequences of different ventilator settings. By combining model simulations and mathematical functions describing clinical preference, the DSS can quantify the utility of different ventilator settings, and make explicit the compromises that exist in setting the ventilator. The DSS can also provide suggestions for optimal ventilator settings. Optimal settings are identified as those which minimise the total penalty.

Previously several DSS have been developed to support the selection of ventilator settings. The majority of these systems have been based on rules which automate the clinician’s heuristic reasoning or some predefined guidelines [3, 4, 6]. Typically these systems focus upon a specific ventilator setting or mode where having a deep understanding of the patient’s state may not be necessary in order to apply the rules. For example, selecting the correct level of pressure has been performed by implementing rules which keeps the patient within “zone of comfort” as defined by the patient’s work of breathing and expired  $\text{CO}_2$  level [4, 5]. This and other systems often act as control systems, for example modifying the level of pressure every 5 min when the clinician is not at the bedside. When the clinician is at the bedside a different type of system might be helpful, i.e., one that provides: a “deeper” understanding of the patient; the ability to simulate the effects of changing ventilator settings; and the ability to make explicit the necessary compromises. The DSS presented here provides this functionality.

When simulating the consequences of different ventilator settings the system has two major limitations. Currently it is not possible to simulate the effects of changing PEEP or I:E ratio. In addition, when performing simulations, parameter values are assumed to be constant. This assumption of constant parameter values may be justified if constraints are placed upon variations in  $\text{F}_{\text{resp}}$ ,  $V_t$  and  $\text{FiO}_2$ . For example: maintaining a respiratory frequency, which does not induce an intrinsic PEEP; or varying  $V_t$  over small enough ranges so that the PV curve can be considered linear and the compliance constant. In practice the ‘optimal’ settings obtained from the system might be seen as targets and small steps in ventilation taken toward these targets with checks for constant parameter values along the way.

The penalty functions included in the system are subjective and reflect the opinion of one clinician. However their explicit formulation means that they can be discussed and modified to reflect the opinion of different clinicians. It has been shown that it is possible to develop computer systems to automate the estimation of utility functions [27], making it possible to describe the preferences of clinicians from different intensive care specialties. Such a system would allow comparison of different clinical opinions and adaptation of the DSS for different clinical environments.

A DSS supporting the process of setting the ventilator based upon models and utility theory has been presented, and illustrated by one patient example. In future, testing of the feasibility of the system is required in patient populations, and further development is needed. The formulation of a DSS using physiological models and utility functions provides a way of making explicit the difficult compromises necessary when setting a ventilator.



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