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Minimisation of dissipated energy in the airways during mechanical ventilation by using constant inspiratory and expiratory flows – Flow-controlled ventilation (FCV)

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ABSTRACT

It has been suggested that energy dissipation in the airways during mechanical ventilation is associated with an increased probability of ventilator induced lung injury (VILI). We hypothesise that energy dissipation in the airways may be minimised by ventilating with constant flow during both the inspiration and expiration phases of the respiratory cycle. We present a simple analysis and numerical calculations that support our hypothesis and show that for ventilation with minimum dissipated energy not only should the flows during inspiration and expiration be controlled to be constant and continuous, but the ventilation should also be undertaken with an I:E ratio that is close to 1:1.

Introduction

For many years substantial effort has been made to find methods of ventilation which minimise the probability of ventilator induced lung injury (VILI) with energy being most recently suggested as a key factor [1]. As the field has developed, possible contributions from many different phenomena have been investigated, including barotrauma arising from high plateau pressures [2,3], volutrauma arising from large tidal volumes [4–6], atelectrauma caused by cyclic collapse and re-inflation of alveoli [7–10], and various combinations of barotrauma and flow induced by high driving pressure [11,12].

All of these variables are associated with lung stress and/or strain, but no single variable or phenomenon is solely responsible for the onset of VILI. It now appears probable that a combination of mechanical effects, many of which are linked to ventilation strategy, contribute to the problem. In many cases the mechanical effects are exacerbated by conditions such as lung heterogenity and vascular pressure [13–15].

As the lungs expand and contract during mechanical ventilation, both stress and strain change with time. As this happens energy is applied to the lung system by the ventilator. Some of this energy is dissipated in the airways and lung tissue.

It has been suggested that an important contributor to VILI may be non-rupturing damage occurring to the lung tissues at a rate faster than the body is able to repair, as a result of the energy dissipation in the tissues [16]. Additional factors may be local stress amplification arising from lung heterogenity, leading to the initiation of significant damage despite the fact that global stress levels may appear to be well within the tolerance of the tissue [17].

For any material, the area under the stress-strain curve is the energy per unit volume applied in stretching the material [18]. If the material is subject to cyclic stress, the stress-strain curve traces out a loop and the energy dissipated in the material is proportional to the area within the loop (arising from the hysteresis) [19]. This is analogous to the pressure-volume (PV) loop during respiration, where for spontaneous breathing the work of breathing is simply the area within the PV loop [20,21]. For a ventilated patient, when the measured pressure is corrected for pressure drop in the ventilation system to give the intratracheal pressure, the area within the PV loop (when intratracheal pressure is used) is the work done by the ventilator on the patient's respiratory system. It is the energy dissipated in the patient during one breath (=respiratory cycle). This energy comes from the ventilator.

In recent years, there has been increasing interest in the potential contribution to VILI from energy applied to the patient by the ventilator during the inspiration phase of ventilation [1,22], with some evidence from work in animals to suggest that the rate at which energy is applied is indeed related to the onset of damage [23–26].

Part of the energy applied to the patient by the ventilator during inspiration is dissipated in the patient's airways and lung tissue and part

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is stored as potential energy by the stretching of the elastic components of the lung parenchyma and the chest. On expiration, this potential energy is released – and, now, part of this stored energy is dissipated in the patient during expiration and part is recovered, provokes the egress of gas and is finally dissipated outside the patient in the ventilator, it's associated tubing, and the atmosphere. It seems most plausible that it is not the energy applied by the ventilator (which is stored and recovered in part) but the energy dissipated in the patient's airways and lung tissue (during both inspiration and expiration) that is related to lung injury.

The hypothesis

Because energy is also dissipated in the patient during expiration, it is almost certainly worth controlling the overall energy dissipated in the lungs during *both* inspiration and expiration phases of the respiratory cycle in order to minimise the energy dissipation that can potentially contribute to lung damage. We hypothesise that this can be achieved by controlling the flow to be constant, continuous and equal during inspiration and expiration phases, with an I:E ratio which is close to 1:1.

In the following, we use a simple model to analyse the energy dissipation during respiration. The analysis supports our hypothesis above. We illustrate this with numerical calculations. Finally, we discuss further work necessary to determine if this hypothesis is valid in patients.

Theory

In this section, we derive mathematical expressions for the energy dissipated in a simple model of a lung unit during respiration, and show that – for a given tidal volume the energy dissipated during inspiration and expiration phases is minimised when the flows during inspiration and expiration are constant and continuous with time. We then go on to find the I:E ratio necessary to minimise energy dissipation when constant, continuous flows are used, for a given minute volume. This is very close to 1:1, rather than the values of 1:1.7–1:2 typically used in conventional ventilation methods.

Simple lung unit model

The simple lung unit model we use for this demonstration is shown in Fig. 1. It consists of a linear resistor of resistance, R, (Pa/m^3/sec) in series with a linear compliance, C, (Pa/m^3). We have chosen SI units here rather than the more conventionally used units of mbar (pressure), l/min (flow), and ml (volume) so that the energy dissipated is calculated directly in Joules without the need to apply any conversion factors.

During the inspiration phase of respiration a time-varying flow, $Q_i(t)$, is input into the system. During the expiration phase a time varying flow, $Q_e(t)$, is extracted from the system. For convenience, we write these flows as the sum of a constant component in each case (q_i, q_e) and a fluctuating component ($\Delta q_i(t)$, $\Delta q_e(t)$) as follows:

$$Q_i(t) = q_i + \Delta q_i(t) \tag{1}$$

$$Q_e(t) = q_e + \Delta q_e(t) \tag{2}$$

Energy dissipation during inspiration

We first consider the inspiration phase. The tidal volume, V_T, is simply the integral of the input flow over the inspiration time, *tinsp*:

$$V_T = \int_0^{tinsp} Q_i(t)dt = q_i tinsp + \int_0^{tinsp} \Delta q_i(t)dt \tag{3}$$

We wish to ventilate to a constant tidal volume, V_T. In order to achieve this, we arrange that the integral of the fluctuating component of input flow is zero, that is:

$$\int_0^{tinsp} \Delta q_i(t)dt = 0 \tag{4}$$

The tidal volume, V_T, is then:

$$V_T = q_i tinsp \tag{5}$$

At any instant during inspiration, the power dissipated in the resistance, $P_i(t)$, is simply the product of the resistance value, R, and the square of the flow through it, $Q_i(t)^2$:

$$P_i(t) = RQ_i(t)^2 = R(q_i + \Delta q_i(t))^2 \tag{6}$$

The energy dissipated during the whole of the inspiration phase, E_i, is then the integral of this power, $P_i(t)$, over the inspiration time, *tinsp*:

$$E_i = \int_0^{tinsp} R(q_i + \Delta q_i(t))^2 dt \tag{7}$$

When we expand the squared term on the right-hand side of this equation and factor out constant terms, we find:

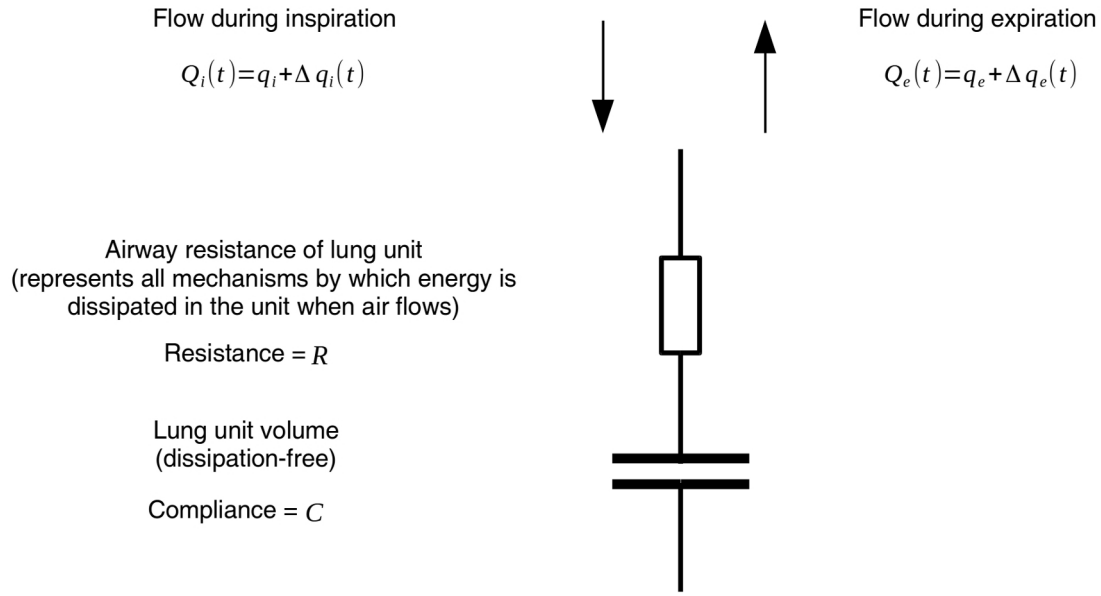

Fig. 1. The simple lung unit model used to develop the flow-controlled ventilation (FCV) theory.