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Modelling the mechanical effects of tracheal tubes in normal subjects

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ABSTRACT: The addition of tracheal tubes to the respiratory system contributes an extra mechanical burden in terms of pressure necessary to overcome its own resistance.

On the basis of experimental data from the literature and on a previously reported mathematical model of the inspirogram, we wished to study predictions of pressures, volume, flow and work of breathing during the use of tracheal tubes.

The present investigation indicates that: 1) the loss in volume is greater at the beginning of inspiration and with narrower tubes; 2) in order to preserve tidal volume the inspiratory drive must be increased at any time during inspiration with the use of diminishing internal diameter of the tubes; 3) alternatively, tidal volume can be maintained by increasing inspiratory duration; and 4) the addition of tubes with internal diameter of 9 mm (no. 9) and 8 mm (no. 8) increases the total resistive work by 115 and 154%, respectively, whilst total elastic work decreases 9 and 16% in relation to the nonintubated patient.

These findings are consequent to the turbulent flow pattern that normally occurs within the tracheal tubes and connectors themselves. We believe they are relevant to the physician confronted with a patient needing tracheal intubation.

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The addition of tracheal tubes to the airways contributes an extra mechanical burden to the respiratory system (rs) [1–5]. Their resistance is highly flow-dependent, augmenting disproportionately with increasing flow rates [6–8]. Only a few studies have dealt with the mechanical effects on the respiratory system (other than the increased resistive work of breathing) originating from the use of tracheal tubes [8, 9]. Probably, the scanty data on respiratory mechanical effects of intubation could be ascribed to the complex and sometimes dangerous experiments that would have to be performed in human beings. In order to avoid these difficulties, mathematical and mechanical models of the intubated respiratory system are very useful. Unfortunately, in general, other accepted models that employ actual human data do not deal with intubation [10–12], and those that take into account tracheal tubes do not address the human respiratory system [1, 2, 5, 13].

In the present investigation, a model originally proposed and validated by ZIN and co-workers [14–18] has been used. They presented model predictions of the immediate (first loaded breath) response to inspiratory resistive loading, based on inspiratory driving pressure waves and on active respiratory mechanics data [15, 17]. Their model predictions were compared with experimental results and closely fitted the latter.

Hence, the aims of the present study are threefold. Firstly, using real human mechanical data, we wanted to calculate the time profiles of driving pressure, volume and flow throughout inspiration for control driving pressure, and also when driving pressure is increased, thus simulating commonly found pathophysiological situations. From these data respiratory system elastic, intrinsic resistive, tracheal tube resistive, and total resistive pressures, and respiratory system elastic, tracheal tube resistive and total resistive work of breathing are calculated. Secondly, we wanted to compute the loss in volume secondary to the addition of tracheal tubes to the respiratory system. Thirdly, we wanted to calculate the increases in respiratory driving pressure and inspiratory duration (TI) necessary to maintain tidal volume (VT). We believe such a comprehensive analysis of the effects of tracheal tubes on the human respiratory system has not hitherto been performed.

Material and methods

Model definition

In making numerical predictions of inspiratory parameters it is necessary: 1) to define the inspiratory driving pressure waveform; 2) to characterize the impedance offered by the respiratory system; 3) to describe the equation of motion of the active respiratory system; and 4) to re-evaluate it with the tracheal tubes added.

Driving pressure

Pressure developed at the airway opening during an inspiratory effort against occluded airways at relaxed volume (Vr) of the respiratory system (P°ao) allows quantification of the neuromuscular inspiratory drive potentially available to produce breathing movements [19, 20]. In anaesthetized subjects the occlusion pressure wave was [17]:

$$P°ao(t) = 16.5t^4 - 44.3t^3 + 3.3t^2 + 5.8t \qquad (1)$$

where t is time (in seconds) from the onset of the occluded effort.

Active impedance

In the same subjects from whom P°ao(t) was obtained, average active elastance of the respiratory system (E'rs) and intrinsic resistance of the respiratory system (R'rs) amounted to, 31.2 $cmH_2O \cdot l^{-1}$ and 2.15 $cmH_2O \cdot l^{-1} \cdot s$, respectively [21].

Equation of motion

The equation of motion of the respiratory system can be adequately represented by a single first-order differential equation:

$$P°ao(t) = E'rs \cdot V(t) + R'rs \cdot \dot{V}(t) \qquad (2)$$

where at any instant (t), P°ao generates instantaneous flow ($\dot{V}$) and volume (V) displacement from Vr. During quiet breathing, inertial forces are negligible [22].

The present model predictions in nonintubated subjects were made using equation (2), and assuming that R'rs and E'rs did not vary within the range of volumes and flows calculated.

Addition of tracheal tubes

The pressure-flow relationships of the three tracheal tubes (8.0, 8.5 and 9 mm ID) can be described by Rohrer's equation ($P = K_1 \cdot \dot{V} + K_2\dot{V}^2$). Rohrer's constants ($K_1$ and K_2) pertaining to the two tracheal tubes used in the present model were provided previously [21]. Under these circumstances, the model predictions were based on:

$$P°ao(t) = E'rs \cdot V(t) + (R'rs + K_1) \cdot \dot{V}(t) + K_2\dot{V}^2(t) \qquad (3)$$

In this model, it has been assumed that tracheal tubes do not affect the intensity (rate of rise) and shape of inspiratory neural drive.

Limitations

The present results apply to the first loaded breath in anaesthetized humans. Naturally, in pathophysiological situations the P°ao(t) waveform and the timing of breathing could change, thus modifying the equations and parameters in the model. Notably, under steady-state conditions resistive loading may generate dynamic hyperinflation and, hence, intrinsic positive end-expiratory pressure (PEEPi); thus, equation (1) would become: $P(t) = E \cdot V(t) + R \cdot \dot{V}(t) + PEEPi$. Nevertheless, the data gathered from this theoretical study can shed some light on otherwise unknown aspects of respiratory behaviour in the presence of added loads.

Data production

Using the model, P(t), $\dot{V}$(t), and V(t) were calculated at 1 ms intervals by means of the iterative computational procedure used previously [11, 15–18]. These functions allow the computation of elastic pressure (Pel,rs(t) =E'rs·V(t)), intrinsic respiratory system resistive pressure (Pres,rs(t)=R'rs·$\dot{V}$(t)), tracheal tube resistive pressure (Pres,t(t)=$K_1\dot{V}(t) + K_2\dot{V}^2(t)$), total resistive pressure (Pres,tot(t)=Pres,rs(t)+Pres,t(t)), respiratory system elastic work (Wel,rs(t)=Σ(Pel,rs(t)·ΔV(t))) (where ΔV is the difference between two consecutive volume points), tracheal tube resistive work (Wres,t(t)=Σ(Pres,t(t)·ΔV(t))), total resistive work (Wres,tot(t)=Σ(Pres,tot(t)·ΔV(t))) and total work (Wtot(t)=Wres,tot(t) + Wel,rs(t)) throughout inspiratory duration (1.2 s). In addition, the variables were also calculated for doubled (2P) and quadrupled (4P) values of P°ao(t) (P).

Results

The time courses of V throughout inspiration are depicted in figure 1. It can be seen that with the addition of tracheal tubes V(t) decreases progressively, the loss in V being proportionately greater early in inspiration. Indeed, at end-inspiration the three curves converge. In figures 1b and c, P°ao(t) is doubled and quadrupled, respectively. Qualitatively, the same pattern found in figure 1a can be observed.

In figure 2, losses in volume caused by tube no. 9 at the beginning of inspiration (t=0.05 s) amount to 33.2, 37.3 and 43.9% when P, 2P and 4P were used, respectively. For tube no. 8 the corresponding values are 43, 47, and 52.5%. At TI, 4.2, 7.9, and 15.8% of VT are lost when tube no. 9 is used (P, 2P and 4P, respectively),

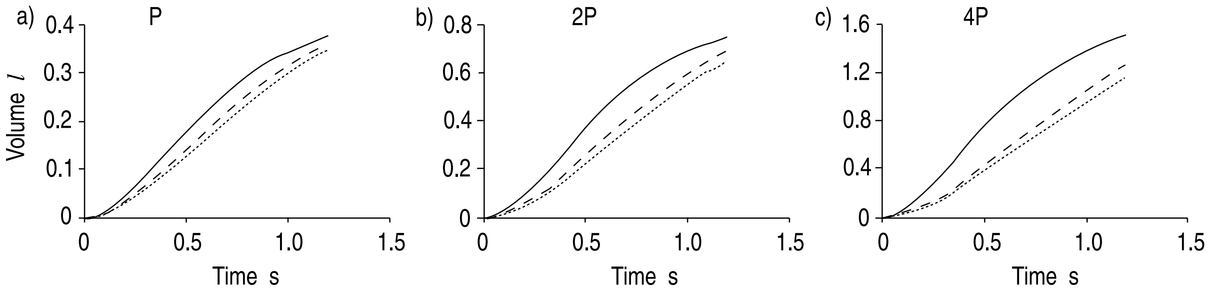

Fig. 1. – Volume plotted against time during inspiration. In the panels each line represents time course of volume for: ——— : total resistance (R) equals lower active intrinsic respiratory system resistance (R'rs); – – – : R equals R'rs plus the addition of the resistance pertaining to tube no. 9 (plus equipment); ········· : R equals R'rs plus the addition of the resistance pertaining to tube no. 8 (plus equipment). a) average control driving pressure was used in computations (P); b) driving pressure was doubled (2P); c) driving pressure was quadrupled (4P). Note that ordinate scale is expanded two fold from a to b, and four fold from a to c. For the sake of clarity the curves pertaining to tube no. 8.5 are not shown, but they lay between those of tubes no. 9 and no. 8.

and tube no. 8 yields VT losses of 7.2, 13.1, and 23%, respectively.

The amount of change in inspiratory drive required to compensate the V(t) losses shown in figure 2 is obtained as the percentage ratio of control V(t) to V(t) found with added tracheal tubes [15] (fig. 3). At t=0.05 s, the required increases in inspiratory drive caused by tube no. 9 amount to 50.3, 60.6, and 78.2% when P, 2P, and 4P were used, respectively; and for tube no. 8 the corresponding values are 73.8, 88.5, and 110.2%. At TI, the corresponding values are 4.4, 8.6, and 18.8% when tube no. 9 was used; and 7.7, 14.1, and 29.9% for tube no. 8.

Tidal volume can also be maintained by increasing TI. For tube no. 9 TI should increase 0.07, 0.12, and 0.23 s when driving pressure equals P, 2P, and 4P, respectively. For tube no. 8 the corresponding values are 0.14, 0.19, and 0.32 s.

The addition of tracheal tubes also modifies the distribution of pressures within the system. In figure 4a, at the beginning of inspiration Pres,rs is greater than Pel,rs; as inspiration continues, Pres,rs decreases and Pel,rs increases. In figures 4b and c, Pres,rs, Pel,rs and Pres,t are plotted (as percentage of total driving pressure) against time throughout inspiration for tubes no. 9 and no. 8, respectively. Due to the presence of Pres,t at the onset of inspiration, Pel,rs and Pres,rs decrease similarly (33.2 and 33.6% (tube no. 9), and 42.9 and 42.8% (tube no. 8), respectively). At the end of inspiration, Pel,rs decreases

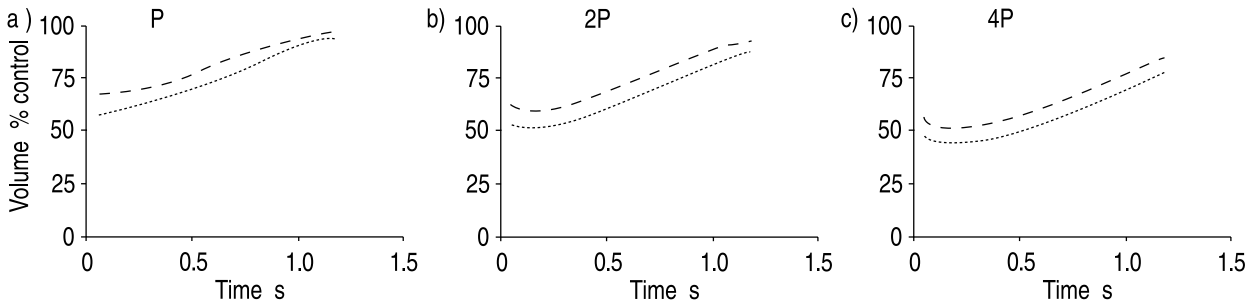

Fig. 2. – Inspired volume, expressed as percentage of control value, *i.e.* without tracheal tube (V(t) (%control)=V(t) (with tracheal tube)/V(t) (control)), plotted against time during inspiration. Lines indicate the addition of tracheal tubes: – – – : no. 9 (plus equipment); ········ : no. 8 (plus equipment). a) average control driving pressure was used in computations (P); b) driving pressure was doubled (2P); c) driving pressure was quadrupled (4P).

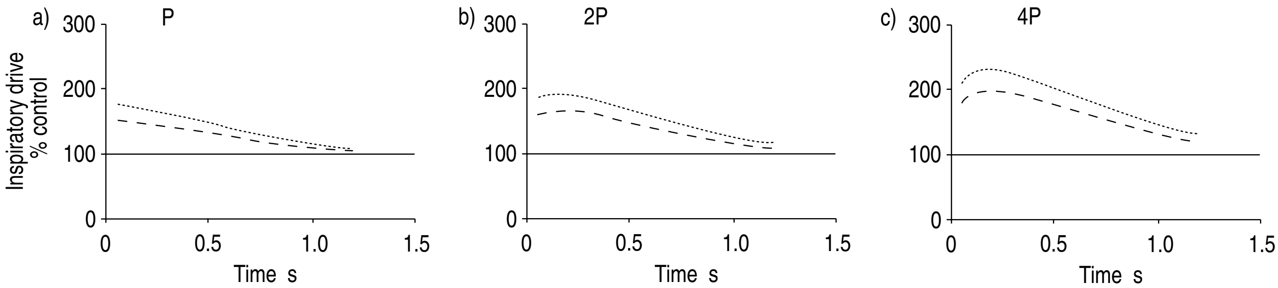

Fig. 3. – Changes in intensity of inspiratory drive, expressed as percentage control (without tracheal tube), required to maintain tidal volume constant in the face of added tracheal tubes (no. 8 and no. 9) plotted against time during inspiration. a) average control driving pressure was used in computations (P); b) driving pressure was doubled (2P); c) driving pressure was quadrupled (4P). – – – : tube no. 9; ········· : tube no. 8.

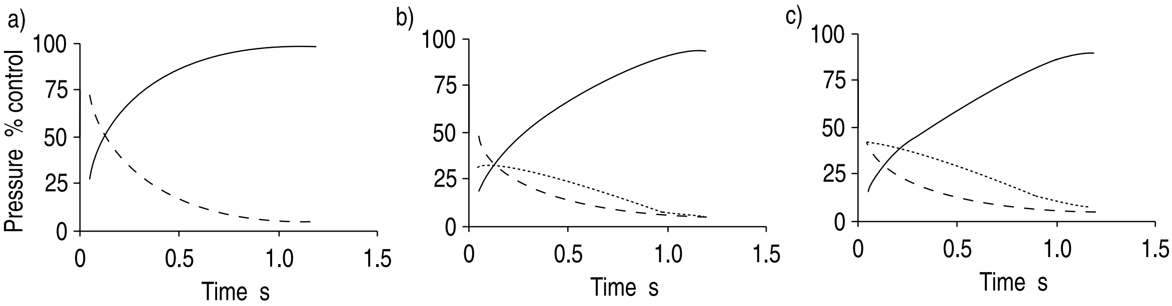

Fig. 4. – Pressure, expressed as percentage of control driving pressure, plotted against time during inspiration. a) total resistance (R) equals lower active intrinsic respiratory system resistance (R'rs); b) R equals R'rs plus resistance pertaining to tube no. 9 (plus equipment); c) R equals R'rs plus resistance pertaining to tube no. 8 (plus equipment). ——— : respiratory system elastic pressure (Pel, rs); – – – : intrinsic respiratory system resistive pressure (Pres,rs); ·········· : tracheal tube resistive pressure (Pres,t).

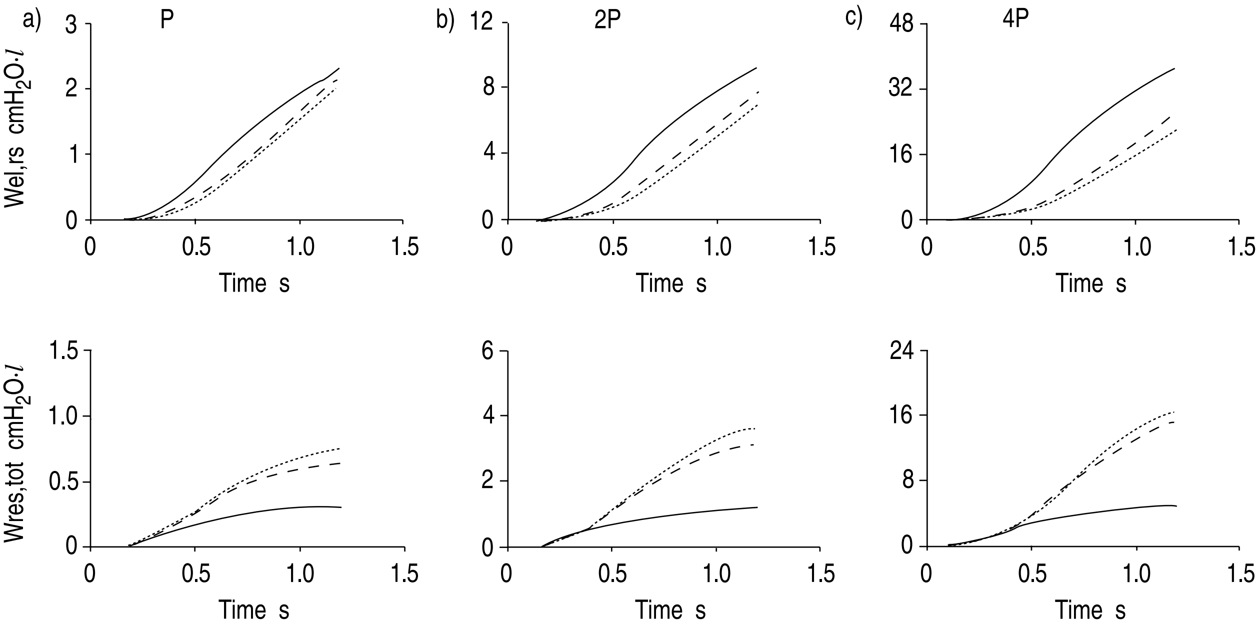

Fig. 5. – Respiratory system elastic work (Wel,rs upper trace) and total resistive work (Wres,tot lower trace) plotted against time during inspiration. ·········· : total resistance (R) equals lower active intrinsic respiratory system resistance (R'rs) plus resistance pertaining to tube no. 8 and equipment; – – – : R equals R'rs plus resistance relative to tube no. 9 and equipment; ——— : R=R'rs. a) average control driving pressure was used in the computations (P); b) driving pressure was doubled (2P); c) driving pressure was quadrupled (4P). Note that ordinate scale is expanded fourfold from a to b, and 16 fold from a to c.

less than at its beginning (4.8 and 8.0% for tubes no. 9 and no.8, respectively). However, Pres,rs increases slightly (3.3 and 10.1% for tubes no. 9 and no. 8, respectively).

Figure 5 shows that the addition of tube no. 9 increases Wres,tot at TI by 115, 162 and 215%, in relation to the control conditions (without tracheal tubes) when P, 2P, and 4P were used, respectively. For tube no. 8, the corresponding values are 154, 201, and 243%. Wel,rs decreases 9, 18 and 41% (tube no. 9) when P, 2P and 4P were used, respectively; for tube no. 8 the corresponding values were 16, 32 and 72%.

Discussion

The addition of tracheal tubes to respiratory system decreases V(t) progressively (fig. 1). The loss in V is proportionately greater early in inspiration, and at end-inspiration the three curves converge rather. This reflects decreased rate of rise of P°ao with a consequent decrease in flow. In fact, because of the curvilinear pressure-flow relationships of the tracheal tubes, a greater amount of pressure is spent against the turbulent component of flow-resistance at higher flows. In the present study, these flows occur early in inspiration (0.4 s under control conditions and 0.55–0.6 s in the presence of tracheal tubes); hence, the widest gap between the inspirograms, generated with or without tubes, occur at those times during inspiration. In relation to the control drive, with increased P°ao the tubes cause a greater loss in volume, both in absolute terms and relative to the inspirograms for R=R'rs. Again, this phenomenon is due to the curvilinear pressure-flow relationships of the tubes resulting in larger Pres,t losses with increased inspiratory flow. These data are in agreement with the results obtained by ZIN and co-workers [17] in anaesthetized humans. GAL [3] demonstrated that in awake, healthy, intubated individuals slow vital capacity was unchanged, but forced vital capacity (FVC) decreased significantly to 89% of control.

In addition, Colgan *et al.* [9] reported that in the presence of tubes 7.5–8.5 VT falls 5.6% after extubation, a change in volume in the same order of magnitude as those presently reported (fig. 2).

In order to maintain V(t), the inspiratory drive must be increased with the use of tracheal tubes, and, additionally, with diminishing internal diameter of the tubes (fig. 3). Alternatively, VT could also be maintained by increasing TI in the face of added tracheal tubes [14, 23]. Our study is the first to report this mechanism of VT compensation against added tracheal tubes. However, unless expiratory duration decreased by the same amount, the increase in TI would represent a drop in ventilation [24]. It should be noted that these predictions are based on the assumption that P°ao could be expressed by equation (1) beyond TI=1.2 s.

It can be seen in figure 4 that due to the presence of Pres,t, at the onset of inspiration, Pel,rs and Pres,rs decrease similarly. At the end of inspiration, Pel,rs decreases less than at the beginning, and Pres,rs increases slightly. These results are, in part, due to the increased resistance, which induces a larger lag between driving pressure and attained volume [14, 15, 17]. In other words, if TI were allowed to increase, Pres,rs and Pel,rs would eventually reach their control values. In this connection, in figure 1 it can be seen that the addition of progressively narrower tracheal tubes leads to smaller inspired volumes with identical total driving pressures. In addition, the larger Pres,t at the beginning of inspiration can be ascribed to a higher rate of rise of driving pressure at these moments, with a consequent higher flow [17]. Summarizing, the energy dissipated to overcome Pres,t is related to decreases in Pel,rs and Pres,rs at the first moments of inspiration, whereas at TI it is strongly related to a decrease in Pel,rs.

Figure 5 shows that Wres,tot increases when tracheal tubes are used, and, furthermore, the smaller the tube diameter the larger is Wres,tot, in accordance with previous reports [1, 2, 4, 7, 13, 25]. For R=R'rs, when P°ao is doubled and quadrupled (fig. 5b and c, respectively) Wres,tot shows an increase proportional to those in P°ao and V, *i.e.* for 2P, Wres,tot quadruples (since both P and V are doubled), and for 4P, Wres,tot shows values that are 16 fold those during control condition. However, this is not the case when tracheal tubes are employed; as can be noted in that Wres,tot with tubes is greater than the values expected by simply doubling and quadrupling driving pressure (fig. 5b and c). Again, the turbulent flow pattern generated within the tubes could be responsible for this behaviour. Consequently, V(t) diminishes, yielding a decrease in Wel,rs. Our values of Wel,rs and Wres,tot are in agreement with previous studies on spontaneously breathing anaesthetized subjects maintaining similar VT and frequency (*f*) [2, 25].

More recently, Bolder *et al.* [2] and Fiastro *et al.* [13], using mechanical models to simulate spontaneous breathing through different tracheal tubes, showed that Wres,t increases progressively with narrower tracheal tubes and with increasing minute ventilation (VE). Fiastro *et al.* [13] reported that a decrease in tube diameter from no. 9 to no. 8 yields an increase of 42.9% in Wres,t , VT (0.5 *l*) and *f* (15 breaths·min^{-1}) being constant. According to Bolder *et al.* [2], (VT=0.35 *l* and *f*=20 breaths·min^{-1}), when the tube diameter is reduced from no. 9.0 to no. 8.0, Wres,t increases 41.9%. Our results indicate that when tracheal tubes are changed from no. 9 to no. 8 Wres,t will increase 35.9%, (VT=0.35 *l* and *f*=17.9 breaths·min^{-1}). In this context, Bolder *et al.* [2] demonstrated that the higher the VE the larger is the difference in Wres,t between tubes.

Bolder *et al.* [2] also reported that for tube no. 8, Wres,t is 0.43 cmH_2O·*l* at VE=4.2 *l*·min^{-1} and *f*=12 beats·min^{-1}, and it corresponds to 0.85 cmH_2O·*l* when VE=7.0 *l*·min^{-1} and *f*=20 breaths·min^{-1}. For the same tube, the present results show a value of 0.50 cmH_2O·*l* at VE=6.25 *l*·min^{-1} (VT of 0.35 *l*, *f* of 17.9 breaths·min^{-1}), which is intermediate between the data reported by Bolder *et al.* [2]. Hence, the aforementioned findings further support the validity of our model.

In the present computational analysis the von Euler-Cauchy method for "initial value" differential equation was used [26]. We computed P, V and V at 1 ms intervals, and for each value of t (any time during inspiration) three iterations were performed. The option for three in opposition to four or more iterations accounted for a reduction in computational time. Furthermore, at any t, V increased only 0.01% from the third to the fourth passage, and 6×10^{-5}% from the fourth to the fifth.

In the present computations, a model previously developed and reported to be correct was used [14–18, 27]. In agreement with studies on humans anaesthetized with methoxyflurane [19, 20] the occluded TI (infinite load) measured by Behrakis *et al.* [21] was similar to the unoccluded one. Based on this finding, in all instances TI in the present model was limited to the average control TI (1.2 s) [17]. Furthermore, it has been shown in man that the shape of the driving pressure does not depend on the level of inspiratory effort [19]. Since P°ao has been shown to represent the inspiratory driving pressure during control conditions and to remain unaltered during the first loaded breath (see [17] for references), it has been used in our computations. On the basis of the aforementioned premises, the results discussed here pertain to the first loaded breath in spontaneously breathing humans (*i.e.* before a reflex change in P°ao occurs).

The present results indicate that: 1) in relation to the nonintubated condition, the loss in volume is greater early in inspiration, with increasing pressure, and in the presence of narrower tubes; 2) at any time during inspiration, the inspiratory drive must be increased with the use of tracheal tubes and will be greater with the diminishing internal diameter of the tubes; 3) the energy dissipated to overcome the tracheal tube resistance is related to a decrease in Pel,rs and Pres,rs at the first moments of inspiration, whereas at TI equal to 1.2 s it is strongly related to a decrease in Pel,rs; 4) Wres,tot increases proportionately to augmented pressure (2P and 4P) when respiratory system alone is taken into account in the calculations, and it is greater when tracheal tubes are included; and 5) these findings are consequent to the turbulent flow pattern that normally occurs within the tracheal tubes themselves.

Finally, we believe that these results should be instructive for enlightening the physician dealing with a patient needing tracheal intubation. After intubation, the patient himself will necessarily increase his respiratory drive, or the mechanical ventilator setting will need special care in order to provide adequate minute ventilation. In addition, it should be stressed that the behaviour of a tracheal tube placed in the patient may be different from the theoretical behaviour as a result of secretions, head or neck position, and tube deformation [28].

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