

## A THERMODYNAMIC MODEL TO PREDICT HYPOTHERMIA THAT RESULTS FROM PNEUMOPERITONEUM PROCEDURES

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***Abstract.** In this work, hypothermia associated with pneumoperitoneum procedures is studied. A thermodynamic model is developed to allow for the computational simulation of the thermal body response to pneumoperitoneum procedures, which are required by laparoscopic surgery. The numerical results predict the body temperature decay (or loss of energy) in time when the pneumoperitoneum procedure is conducted in a patient. The influence of several operating parameters (e.g., inlet air mass flow rate and temperature) on the resulting hypothermia level is analyzed. The model therefore allows for the search of mechanisms to reduce the loss of energy, and consequently, the hypothermia level due to pneumoperitoneum procedures.*

**Keywords:** Balance of energy, Intra-abdominal hypertension, Body temperature regulation

### 1. INTRODUCTION

Increased intra-abdominal pressure as used in laparoscopic surgery produces various changes in the human organism (Rosenthal et al., 1997). For example, the top five complications for laparoscopic low anterior resections are bleeding, hypothermia, small bowel injuries recognized and unrecognized, disorientation accompanied by ureteral injury, and anastomotic leaks (MacFadyen and Ponsky, 1996).

Intra-abdominal hypertension is commonly present in patients with multiple trauma that have received a great deal of reposition fluids, causing an increase in the interstitial fluid volume. Significant elevations of intra-abdominal pressure are observed in a variety of situations, such as: post-surgery intra-abdominal hemorrhage (Fietsam et al., 1989; Cullen et al., 1989; Jacques and Lee, 1988; Shelly et al., 1987; Platell et al., 1990; O’Leary and Park, 1991), complex abdominal vascular procedures such as hepatic transplants (Cullen et al., 1989; Shelly et al., 1987; O’Leary and Park, 1991), severe abdominal trauma (Cullen et al., 1989; Jacques and Lee, 1988, O’Leary and Park, 1991), utilization of pneumatic anti-shock garment (Mcswain, 1988), peritoneal gas insufflation during laparoscopic procedures (Motev

et al., 1973; Westerband et al., 1992), etc. The effects of increased intra-abdominal pressure can be associated to multiple co-existent factors and individual clinical repercussions in each patient. Besides hypovolemia, acidosis and coagulopathy, hypothermia is a serious complication associated with these patients. Hypothermia is very common in severely traumatized patients (Jurkovich et al., 1987).

Hypothermia is defined as a core body temperature below 36 °C. It is an unavoidable pathophysiologic consequence of the serious injury and the follow up reanimation (Bernabei et al., 1992; Gregory et al., 1991; Jurkovich et al., 1987; Steinemann et al., 1990). Hypothermia in patients with intra-abdominal hypertension is present in most cases (Ivatury et al., 1998; Eddy et al., 1997). Brandon Bravo (1999) introduced a simple mathematical model in an attempt to describe thermal steady state in trauma patients, concluding that pre-heating of intravenous fluids is mandatory in patients with high fluid requirements. In the literature, no specific studies were found demonstrating a direct relation between the increase in intra-abdominal pressure and body temperature. Although hypothermia has been observed in most cases where intra-abdominal hypertension is present, the degree of hypothermia due to it solely has been impossible to determine precisely because, in general, there are other factors involved, such as: multiple trauma, hypotension, politransfusion, laparo-toracotomies with humid surfaces exposure, aortic or inferior cava vein clamping associated with coagulopathy, and acidosis.

The intra-abdominal hypertension and hypothermia have similar triggering factors. The question to be answered is whether or not it is possible to establish a direct correlation between the increase in intra-abdominal pressure and hypothermia, not considering other factors, normally present in trauma patients, for example. The objective of this study is to introduce a transient mathematical model based on a balance of energy between the body and the surroundings, aiming to investigate the sole effect of the increase in the intra-abdominal pressure on the body temperature, such as in a pneumoperitoneum procedure.

## 2. THEORY

The physical model is described by the schematic diagram shown in Fig. 1. The human (or animal) body is divided in three control volumes interacting energetically with one another and the ambient. Control volume 1 (CV 1) is the space being filled by the insufflating fluid during the pneumoperitoneum procedure, initially with a mass of insufflating fluid,  $m_1$ , equal to zero, which is mathematically modeled as a sphere with its radius increasing in time. Control volume 2 (CV 2) is composed by the abdominal tissues (peritoneum, muscles, bowels and abdominal organs) that surround CV 1, and it is modeled mathematically as a spherical shell. Control volume 3 (CV 3) models the rest of the body (head, torax, arms and legs). The thermodynamic analysis consists of writing the conservation equations for each control volume in transient regime.

The analysis starts with CV 1, by combining the equation of state for ideal gases, the mass and energy conservation equations, with the following final result:

$$h_{int}(T_2 - T_1) + \frac{\dot{m}_{in} c_{p,f} T_{in}}{4\pi r^2} = \frac{c_{v,f}}{R} \left[ p_1 \frac{dr}{dt} + \frac{r}{3} \frac{dp_1}{dt} \right] + p_1 \frac{dr}{dt} \quad (1)$$

where  $h_{int}$  - intra-abdominal heat transfer coefficient,  $W/(m^2.K)$ ;  $r$  - sphere inner radius,  $m$ ;  $T_2$  - CV 2 temperature,  $K$ ;  $T_1$  - CV 1 temperature,  $K$ ;  $\dot{m}_{in}$  - insufflating fluid mass flow rate,  $kg/s$ ;  $c_{p,f}$  - insufflating fluid specific heat at constant pressure,  $J/(kg.K)$ ;  $T_{in}$  - insufflating fluid temperature,  $K$ ;  $c_{v,f}$  - insufflating fluid specific heat at constant volume,  $J/(kg.K)$ ;  $R$  - constant of the insufflating gas,  $J/kg.K$ ;  $p_1$  - intra-abdominal pressure,  $N/m^2$  and  $t$  - simulation time,  $s$ .

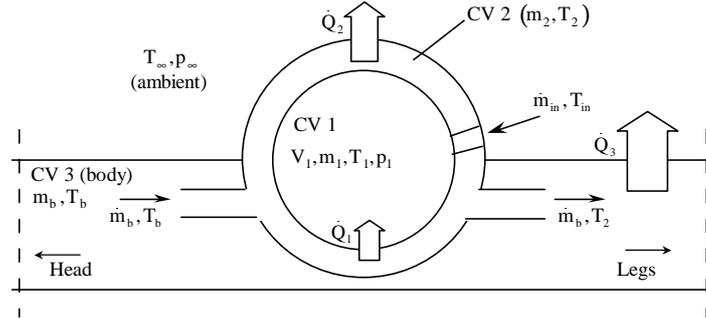


Figure 1 – Schematic diagram of a human or animal body divided in three control volumes.

Next, assuming the forces in the radial direction during the abdominal expansion are in equilibrium, a relation between  $p_1$  and  $r$  is written as follows:

$$\frac{dp_1}{dt} = \frac{1}{2\pi r^2} [k_b - 4\pi r(p_1 - p_\infty)] \frac{dr}{dt} \quad (2)$$

where  $k_b$  - abdomen elastic constant,  $N/m$ , and  $p_\infty$  - external ambient pressure,  $N/m^2$ . In the equilibrium of forces, it has also been assumed that the sphere inner and outer surface areas are approximately equal to  $2\pi r^2$  (i.e., sphere superior half).

The combination of Eq. (2) with Eq. (1) allows for the determination of the increasing rate of the sphere inner radius during the insufflating process. The final result in dimensionless form is given by:

$$\frac{d\tilde{r}}{d\theta} = \frac{(k-1) \left[ H_{int} (\tau_2 - \tau_1) + \dot{M}_{in} \frac{\tau_{in}}{4\pi \tilde{r}^2} \right]}{P_1 + \frac{C}{\tilde{r}} - \frac{2}{3} (P_1 - 1) + (k-1)P_1} \quad (3)$$

where dimensionless variables were defined as follows:  $P_1 = \frac{p_1}{p_\infty}$ ;  $\tilde{r} = \frac{r}{D}$ ;  $C = \frac{k_b/D}{p_\infty}$ ;

$\theta = t \frac{\dot{m}_{b0} c_{p,f} T_\infty}{p_\infty D^3}$ ;  $H_i = \frac{h_i D^2}{\dot{m}_{b0} c_{p,f}}$ ;  $\tau_i = \frac{T_i}{T_\infty}$ ;  $\dot{M}_i = \frac{\dot{m}_i}{\dot{m}_{b0}}$ ;  $k = \frac{c_{p,f}}{c_{v,f}}$ , i.e., CV 1 pressure, sphere

inner radius, abdomen elastic coefficient, time, heat transfer coefficients, temperatures, mass flow rates and ratio of insufflating fluid specific heats, respectively, taking  $D$  – reference

diameter, m and  $\dot{m}_{b0}$  - initial blood mass flow rate that flows around the inflated space in the abdomen, kg/s (Yang, 1989). The index ‘‘i’’ refers to the variable location in the domain.

Next, applying the first law of thermodynamics to CV 2, the evolution of the temperature of the abdominal tissues in time is given in dimensionless form as follows:

$$\frac{d\tau_2}{d\theta} = \frac{1}{M_2} \left\{ 2\pi\tilde{r}^2 [H_{out}(1-\tau_2) - H_{in}(\tau_2 - \tau_1)] + \dot{M}_b \tilde{c}_b (\tau_b - \tau_2) \right\} \quad (4)$$

where nondimensional variables were defined as  $M_i = \frac{m_i c_{body} T_\infty}{p_\infty D^3}$  and  $\tilde{c}_b = \frac{c_b}{c_{p,f}}$ , with  $c_b$  - blood specific heat, J/kg.K and  $c_{body}$  - body specific heat, J/kg.K. Bailey and Flowers (1995) reported that at a maximum intra-abdominal pressure of 80 mm Hg, the blood mass flow rate around the abdominal tissues drops to zero. Therefore, in this model, it has been assumed a linear model for computing the blood flow rate in the abdomen with respect to intra-abdominal pressure, from two known points, i.e.,  $\dot{m}_b = \dot{m}_{b0}$  when  $p_1 - p_\infty = 0$  mm Hg, and  $\dot{m}_b = 0$  when  $p_1 - p_\infty = 80$  mm Hg. Taking  $p_\infty = 760$  mm Hg, the resulting dimensionless blood mass flow rate as a function of intra-abdominal pressure is given by:

$$\dot{M}_b = \frac{1.105 - P_1}{0.105} \quad (5)$$

Analyzing the rest of the body, CV 3, by applying the first law of thermodynamics, the behavior of the body temperature in time is calculated by the following nondimensional equation:

$$\frac{d\tau_b}{d\theta} = \frac{\dot{M}_b \tilde{c}_b}{M_b} (\tau_2 - \tau_b) + (1 - \alpha) H_{out} (1 - \tau_3) \tilde{A}_3 \quad (6)$$

where a dimensionless body surface area was defined as  $\tilde{A}_3 = \frac{A_3}{D^2}$ , with  $A_3$  - exposed body surface area, m<sup>2</sup> and a dimensionless body mass,  $M_b$ , with  $m_b$  - body mass, kg. Equation (6)

introduces a body metabolic heat ratio, defined as  $\alpha = \frac{\dot{Q}_{gen}}{\dot{Q}_3}$ , where  $\dot{Q}_{gen}$  - body heat generation, W and  $\dot{Q}_3 = h_{out} A_3 (T_\infty - T_3)$  - heat loss to the ambient through the exposed body surface, W. This way, the model allows for the investigation of the effect on the body temperature, of the body capacity to respond to heat loss.

The insufflated mass of gas in CV1 is computed by accounting for mass conservation as follows:

$$\frac{dM_1}{d\theta} = \tilde{c}_{body} \dot{M}_{in} \quad (7)$$

where  $\tilde{c}_{\text{body}} = \frac{c_{\text{body}}}{c_{p,f}}$ .

The temperature of the insufflated mass of gas varies in time according to a balance of energy in CV 1. It is calculated, in dimensionless form, as a function of the increasing rate of the sphere radius as the abdomen inflates, as follows:

$$\frac{d\tau_1}{d\theta} = \frac{1}{M_1 \tilde{c}_1} \left[ \dot{M}_1 \left( \tau_{\text{in}} - \frac{\tau_1}{k} \right) + H_{\text{int}} (2\pi \tilde{r}^2) (\tau_2 - \tau_1) - P_1 (4\pi \tilde{r}^2) \frac{d\tilde{r}}{d\theta} \right] \quad (8)$$

where  $\tilde{c}_1 = \frac{c_{v,f}}{c_{\text{body}}}$ .

The pressure increase in CV 1 is calculated by using the equation of state for ideal gases, in dimensionless form as follows:

$$P_1 = \frac{3}{4\pi} \frac{R}{c_{\text{body}}} \tau_1 \frac{M_1}{\tilde{r}^3} \quad (9)$$

### 3. RESULTS AND DISCUSSION

The mathematical model developed in the previous section allows for the simulation in time of the behavior of a human or animal body when submitted to a pneumoperitoneum procedure. For that, Eqs. (3), (4), (6), (7) and (8) are integrated in time, starting from a known initial condition  $(\tilde{r}, \tau_2, \tau_b, \tau_1, M_1)_0$  at  $\theta = 0$ . A time step adaptive, 4<sup>th</sup> – 5<sup>th</sup> order Runge-Kutta-Fehlberg method (Kincaid and Cheney, 1991) was utilized to integrate numerically the set of ordinary differential equations.

The set of initial conditions utilized to obtain results in this study was  $(\tilde{r}, \tau_2, \tau_b, \tau_1, M_1)_0 = (0.001, 1.03857, 1.03857, 1., 10^{-5})$ , where  $\tilde{r}_0$  was not taken equal to zero, to avoid a numerical indetermination at  $\theta = 0$ . The model also requires the specification of several other parameters related to the operating conditions and to the body to start the simulations, which were selected as follows:  $\dot{m}_{b0} = 0.1342$  kg/s (Yang, 1989),  $c_{\text{body}} = 3.475$  kJ/kg.K (Yang, 1989),  $R = 287$  J/kg.K (air),  $c_{p,f} = 1.005$  kJ/kg.K (air),  $k = 1.4$  (air),  $m_2 = 10$  kg,  $D = 1$  m,  $m_b = 70$  kg,  $T_\infty = 298.15$  K,  $p_\infty = 0.1$  MPa,  $T_{\text{in}} = 298.15$  K,  $\dot{m}_{\text{in}} = 0.01$  kg/s,  $c_{v,f} = 0.716$  kJ/kg.K,  $c_b = 3.889$  kJ/kg.K (Yang, 1989),  $k_b = 100$  N/m,  $h_{\text{out}} = 5$  W/m<sup>2</sup>.K,  $h_{\text{in}} = 50$  W/m<sup>2</sup>.K and  $\alpha = 0.5$ . The case simulated numerically by the present model, corresponds to a 1.68 m tall subject, weighing 80 kg, that was submitted to an exploratory laparoscopy. The total body surface was estimated at 1.9 m<sup>2</sup> using the DuBois formula (DuBois and Dubois, 1916):  $A = 0.007184 m^{0.425} l^{0.725}$  ( $l$  – body length, cm and  $m$  – total body mass, kg). In the numerical simulations, the exposed body surface area was reduced to  $A_3 = 1$  m<sup>2</sup>, to account for the non-exposed part of the body which was in contact with the surgical table. Additionally, a maximum dimensionless abdomen inner sphere radius was established,  $\tilde{r}_{\text{max}} = 0.2$ , such that the inflated space does not increase beyond that point, and if fluid continues to be insufflated, the pressure will increase. Therefore, to simulate the

pressure control in actual laparoscopic surgeries, an intra-abdominal pressure control was adopted in the model, by setting a high pressure set point,  $P_{1H} = 1.04$ , and a low pressure set point,  $P_{1L} = 1.03$ , such that the intra-abdominal pressure stabilizes between these limits at some point in time, during the simulation, by turning on and off the insufflating fluid mass flow rate.

The influence of the variation of several of the model parameters is investigated, by varying one at a time, and analyzing the effect on the resulting degree of hypothermia. Figure 2 shows the effect of the variation of the insufflating fluid mass flow rate on the time that it is necessary to reach the maximum allowed abdomen distension, i.e., to reach  $\tilde{r}_{max}$ . The time to reach  $\tilde{r}_{max}$  decreases as  $\dot{M}_{in}$  increases, thus the model captures the expected physical trend.

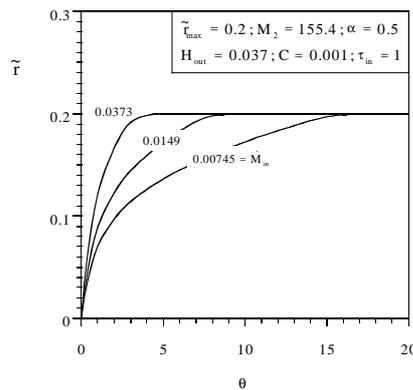


Figure 2 – The effect of insufflating fluid mass flow rate on the time to achieve maximum abdominal distension.

Figure 3 shows the effect of the variation of the insufflating fluid mass flow rate on the time evolution of the intra-abdominal pressure. For the three mass flow rate sets, the desired pressure level is eventually achieved, where the higher the flow rate, the faster the pressure set point is reached. The pressure peak in the beginning of the simulation is due to the very small available space for the incoming gas, and as the abdomen expands the pressure decreases, remaining above ambient pressure until maximum abdominal distension is reached, when the pressure starts to rise again up to pressure set point.

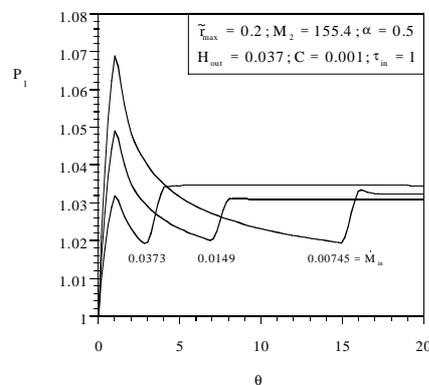


Figure 3 – The effect of insufflating fluid mass flow rate on intra-abdominal pressure.

Next, the effect of the body metabolic heat ratio,  $\alpha$ , on the evolution in time of the body temperature, is investigated. Figure 4 depicts the body temperature response in time for three different values of  $\alpha$ , 0.1, 0.5 and 0.8, i.e., with an increasing capacity of the body heat loss compensation. As expected, the degree of hypothermia is more severe as the metabolic ratio decreases, which shows the model captures what is expected physically. The body temperature, for  $\alpha = 0.5$ , after 4 hours of laparoscopic surgery, predicted by the numerical simulation was  $\tau_b = 1.0252$ , which corresponds to  $32.51^\circ\text{C}$ .

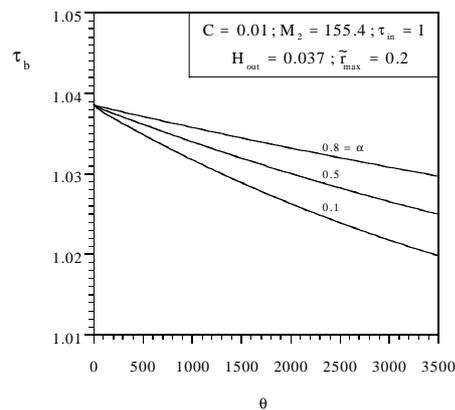


Figure 4 – The effect of the body metabolic heat ratio on the degree of hypothermia.

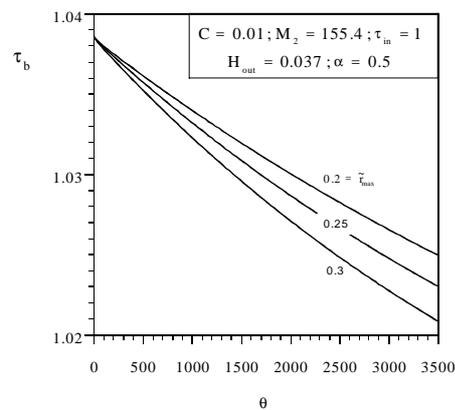


Figure 5 – The effect of the maximum abdomen distension radius on the degree of hypothermia.

Another important parameter in the degree of hypothermia is the maximum abdominal distension radius,  $\tilde{r}_{\max}$ . Figure 5 shows that the body temperature drops faster if a larger amount of insufflating fluid enters the abdominal space. Therefore, the results stress the importance of somehow accessing the particular value of  $\tilde{r}_{\max}$ , for an individual who is going to be submitted to a pneumoperitoneum, such that the physician could take the necessary measures to avoid hypothermia, e.g., make a decision on the expected surgery duration according to each individual.

The effect of two other major surgery variables is investigated in Fig. 6, i.e., the insufflating gas temperature and the blood mass flow rate that initially flows through the relaxed abdomen (no intra-abdominal hypertension). The simulation results show that,

between 20 and 30 °C, the fluid temperature had no influence on the final body temperature,  $\tau_{b,f}$ , after 4 hours of surgery. The insufflating fluid low temperature has been considered the primary cause of the hypothermia in prolonged procedures (MacFadyen and Ponsky, 1996). The present results are at least an indication that such effect has to be investigated further. On the other hand, the initial blood mass flow rate has a significant effect on the final body temperature. The lower the initial abdominal blood flow rate, the lower the final temperature will be, thus it is important for the physician to access the specific value of this parameter for each individual, prior to surgery, such that decisions could be made on the expected surgery duration and other necessary preventive measures.

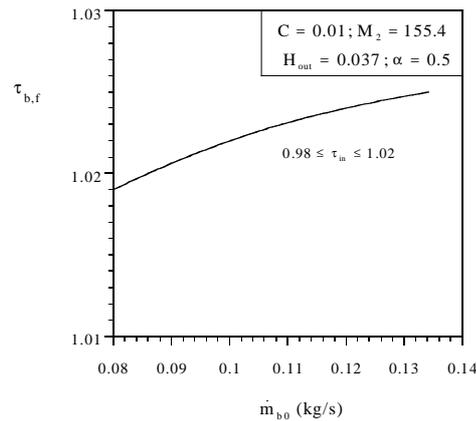


Figure 6 – The effect of the insufflating gas temperature and initial blood mass flow rate on the final hypothermia level.

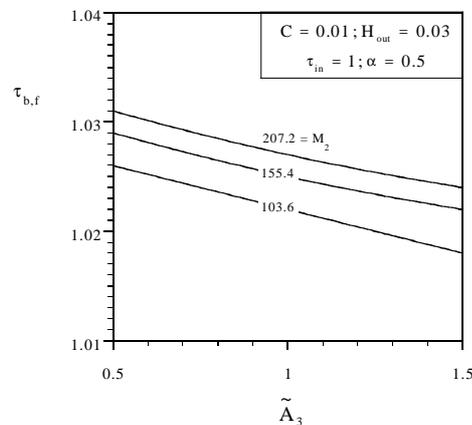


Figure 7 – The effect of the mass of abdomen tissues and exposed body surface area on the final hypothermia level.

Figure 7 shows the effect of the mass of abdomen tissues and exposed body surface area on the final body temperature after 4 hours of surgery ( $\theta \cong 3500$ ). As the exposed body surface increases, the final body temperature decreases, stressing the importance of reducing the exposed surface to a minimum, during surgery. The increase in abdominal tissues mass around the inflated abdominal space results in less hypothermia, stressing the importance of extra-care with thin individuals submitted to pneumoperitoneum procedures.

Finally, Fig. 8 shows that the ambient temperature had a minor effect on the hypothermia level after 4 hours of simulation, where the ambient temperature was varied between 20 and 30 °C. However, the external heat transfer coefficient showed a major influence on the degree of hypothermia, i.e., as  $H_{out}$  increases  $\tau_{b,f}$  decreases. Both findings are important, in the sense that they are complementary, i.e., it appears that not too much concern should be given to the operating room temperature, but to the air circulation in it, that must be kept as minimum as possible.

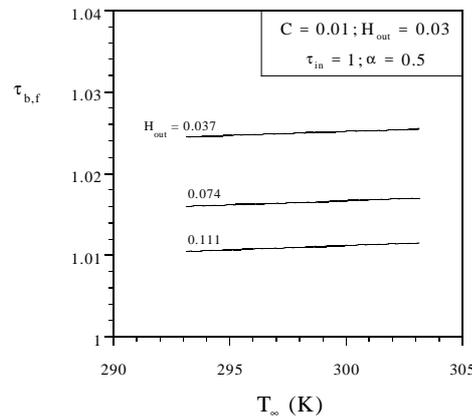


Figure 8 – The effect of the outer heat transfer coefficient and ambient temperature on the final hypothermia level.

#### 4. CONCLUDING REMARKS

In this paper, a simplified thermodynamic model is introduced to simulate the thermal response of a human or animal body to pneumoperitoneum procedures. The numerical results obtained with the model allow for the investigation of the effect on the body temperature, of several physical and operating variables involved in a laparoscopic surgery. The results are general, since appropriate nondimensional groups have been identified and the results presented in dimensionless charts.

The results are shown to capture the expected physical trends according to the variation of several parameters. Furthermore, they bring to attention that some assumed dogmas, such as the need to pre-heat the insufflating fluid and to control the ambient temperature have been shown to have a minor effect on the body temperature decay. The study also identifies some parameters that substantially affect the hypothermia level, such as initial abdominal blood mass flow rate, abdominal maximum distension radius, body metabolic heat ratio, mass of abdomen tissues, exposed body surface and external heat transfer coefficient. In sum, the insight provided by the results motivates the continuation of the present study to an experimental phase, aiming to validate the numerical results, such that the model could eventually be used as an accessory tool for surgeons to predict the thermal body response of their patients prior to surgery, and therefore avoiding unnecessary complications.

#### REFERENCES

Bailey, R. W. & Flowers, J. L., 1995, Complications of Laparoscopic Surgery, Quality Medical Publishing, Inc.

- Bernabei, A., Levison & M., Bender, J., 1992, The effects of hypothermia and injury severity on blood loss during trauma laparotomy, *J Trauma*, vol.33 p.835.
- Brandon Bravo, L. J. C., 1999, Thermodynamic modelling of hypothermia, *Eur. J. of Emergency Medicine*, vol. 6, pp. 123-127.
- Cullen, D. J., Coyle, J. P. & Teplich, R., 1989, Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients, *Crit Care Med.*, vol. 17, pp. 118-121.
- DuBois, D. & DuBois, E. F., 1916, A formula to estimate the approximate surface area if height and weight be known, *Arch. Intern. Med.*, vol. 16, pp. 863-871.
- Eddy, V.; Nunn, C. & Morris, J. A., 1997, Síndrome do Compartimento Abdominal: A Experiência de Nashville. *In: Hirshberg, A. & Mattox, K. L., Cirurgia Emergencial Controlada: Controle do Dano Cirúrgico, Clin Surg North Am*, vol. 4, Interlivros.
- Fietsam, R.; Villalba, M. & Glover, J. L., 1989, Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair, *Am. Surg.*, vol. 55, pp. 396-402.
- Gregory, J., Flancbaum, L. & Townsend, M., 1991, Incidence and timing of hypothermia in trauma patients undergoing operations, *J Trauma*, vol.31, p.795.
- Ivatury, R. R.; Porter, J. M.; Simon, R. J., 1998, Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome, *J. Trauma*, vol. 44, pp. 1016-1023.
- Jacques, T. & Lee, R., 1988, Improvement of renal function after relief of raised intra-abdominal pressure due to traumatic retroperitoneal hematoma, *Anaesth. Intensive Care*, vol. 16, pp. 478-482.
- Jurkovich, G. J.; Greiser, W. B. & Luterman, A., 1987, Hypothermia in trauma victims: an ominous predictor of survivor, *J. Trauma*, vol. 27, pp. 1019-1024.
- Kincaid, D. & Cheney, W., 1991, *Numerical Analysis: Mathematics of Scientific Computing*, Wadsworth, Belmont, CA.
- MacFadyen Jr., B. V. and Ponsky, J. L., 1996, *Operative Laparoscopy and Thoracoscopy*, Lippincott-Raven Publishers.
- Mcswain, N. E., 1988, Pneumatic anti-shock garment: state of the art 1988, *Ann Emerg Med*, vol. 17, pp. 506-525.
- Motev, M., Ivankovich, A. D. & Bieniarz, J., 1973, Cardiovascular effects and acid-base and blood gas changes during laparoscopy, *Am. J. Obstet. Gynecol.*, vol. 115, pp. 1002-1011.
- O'Leary, M. J. & Park, G. R., 1991, Acute renal failure in association with a pneumatic antishock garment and with tense ascites, *Anaesthesia*, vol. 46, pp. 326-327.
- Platell, C., Hall, J. & Dobb, G., 1990, Impaired renal function due to raised intra-abdominal pressure, *Intensive Care Med*, vol. 16, pp. 328-329.
- Rosenthal, R.J., Friedman, R. L. & Phillips, E. H., 1997, *The Pathophysiology of Pneumoperitoneum*, Springer Medicine.
- Shelly, M. P., Robinson, J. W. & Hesford, J. W., 1987, Hemodynamic effects following surgical release of increased intra-abdominal pressure, *Br J Anaesthesiol*, vol. 59, pp. 800-805.
- Steinemann, S., Shackford, S. R. & Davis, J. W., 1990, Implications of admission hypothermia in trauma patients, *J. Trauma*, vol. 30, pp. 200-202.
- Westerband, A., Van De Water, J. M. & Amzallag, M., 1992, Cardiovascular changes during laparoscopic cholecystectomy, *Surg Gynecol Obstet*, vol. 175, pp. 535-538.
- Yang, W., 1989, *Biothermal-Fluid Sciences Principles and Applications*, Hemisphere, USA.