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A COMPUTER MODEL OF INTRACRANIAL DYNAMICS

William James Thoman, Samsun Lampotang, Dietrich Gravenstein, Jan van der Aa

Department of Anesthesiology
University of Florida Brain Institute
University of Florida College of Medicine
Trailer 237 Annex 13
Mowry Road
Gainesville, FL 32608

ABSTRACT

A software model of intracranial dynamics was developed using existing relationships between patient physiology and brain dynamics. The relationship between the physiologic parameters arterial partial pressure of oxygen (PaO_2), arterial partial pressure of carbon dioxide ($PaCO_2$), mean arterial blood pressure (MABP), temperature (T) and brain dynamics were obtained from literature and were coded into software using Borland C. This computer model was integrated with a full scale patient simulator which generated the physiological inputs to the brain model. This integration allows users of the patient simulator to see the effects of their clinical interventions on brain dynamics through a real-time display. The display outputs the following intracranial variables: cerebral blood flow (CBF), cerebral blood volume (CBV), cerebral metabolic rate ($CMRO_2$), cerebral perfusion pressure (CPP), and intracranial pressure (ICP). The ability to visualize intracranial events as they are affected by physiology is a useful tool for teaching and training students.

INTRODUCTION

The ability to influence intracranial dynamics is of importance to clinicians in order to minimize or prevent brain injury. Anesthesiologists make assumptions about intracranial pressure, neurophysiological events and the effects of different treatments to brain dynamics. They control brain dynamics by adjusting physiologic parameters ($CMRO_2$(T), PaO_2, $PaCO_2$, MABP). The present brain model provides a technique, using physiologic data from the literature, to generate a dynamic display of intracranial events (ICP, CBF, CBV) as they are affected or altered by all modeled variables simultaneously. Because intracranial events are normally not evident to physicians, and because this model incorporates influences of all variables concurrently in real time, this effort might be a useful tool to teach neurophysiology. Currently the brain model receives its physiological data from a full-scale patient simulator (Human Patient Simulator v. B, Medical Education Technologies, Inc., Sarasota, FL).[1]

METHODS

In order to model ICP, the intracranial space is broken down into four volumes: brain (1150 mL), blood (75 mL), cerebrospinal fluid (CSF) (75 mL), and a "mass" (0 to 75 mL).[2,3,4] A mass of zero represents a normal brain while a non-zero mass represents an abnormality (e.g. a tumor or hematoma). Acute changes in ICP are determined by changes in CBV, and the elastance. This is based on the study by Riseberg et al.[5] which showed a linear relationship between CBV and CBF and that ICP is affected by acute changes in cerebral blood volume. The relationships between the physiologic parameters ($CMRO_2$, PaO_2, $PaCO_2$, MABP) and CBF are used to calculate CBF following the flow chart on Fig. 1. An average CBF value of 52.5 mL/100g brain/min is used based on literature[6,7,8], and the % change in CBF due to the physiologic parameters ($CMRO_2$, PaO_2, $PaCO_2$, MABP) are either added or subtracted to this average value. All the equations are derived from literature data and follow the path of the flow chart in Fig. 1.

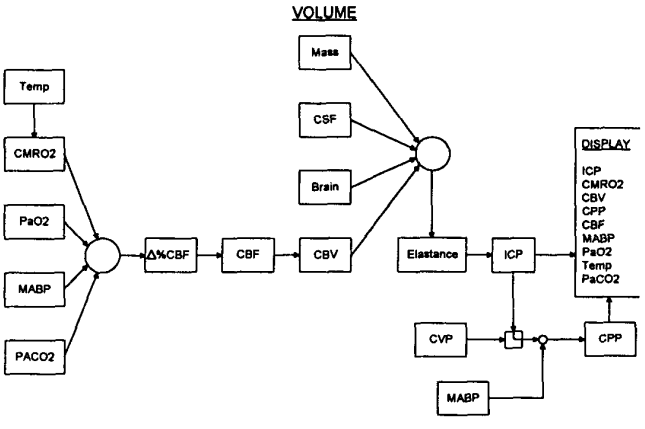

FIGURE 1. Flow chart of brain model

Temperature, PaO_2, $PaCO_2$, systolic blood pressure, diastolic blood pressure, and the central venous pressure are all physiologic parameters which are modeled by the full patient simulator (HPS). The brain model is interfaced with the HPS and updates the physiologic parameters at a sampling frequency of 10 Hertz. These parameters are then used in the brain model software to determine the CBF.

The effects of PaO_2 on CBF

A curve showing the relationship between PaO_2 and %CBF was obtained from literature.[7] Equations [1], [2] and [3] describe this relationship.

Eq. [1]: $0 <= PaO_2 < 40$

$$\%CBF_{PaO_2} = 241.20908 - (2.90778 * PaO_2)$$

Eq. [2]: $40 <= PaO_2 < 61$

$$\%CBF_{PaO_2} = 1174.3974 - 70.115107 * PaO_2 + 1.7436586 * (PaO_2)^2 - 0.01948396 * (PaO_2)^3 + 8.232781E-5 * (PaO_2)^4$$

Eq. [3]: $61 <= PaO_2 < 750$

$$\%CBF_{PaO_2} = 104.47978 - 0.02543 * PaO_2$$

Units: $\%CBF_{PaO_2}$ (%CBF), PaO_2 (mmHg)

The effects of $PaCO_2$ on CBF

A curve showing the relationship between $PaCO_2$ and %CBF was obtained from literature.[7] For the model, the lower portion of this curve was redrawn for $PaCO_2$ values below 20 mmHg to account for cases of hyperventilation. This is based on clinical observations during aggressive hyperventilation where CBF values as low as 10 mL/100g brain/min have been observed [personal communication, Dietrich Gravenstein, M.D., June 13, 1996]. Equations [4], [5] and [6] describe this relationship.

Eq. [4]: $0 <= PaCO_2 < 20$

$$\%CBF_{PaCO_2} = 2.6 * PaCO_2$$

Eq. [5]: $20 <= PaCO_2 < 80$

$$\%CBF_{PaCO_2} = 1.76562 + 2.50347 * PaCO_2$$

Eq. [6]: $80 <= PaCO_2 < 100$

$$\%CBF_{PaCO_2} = 158.0634 + 0.55461 * PaCO_2$$

Units: $\%CBF_{PaCO_2}$ (%CBF), $PaCO_2$ (mmHg)

The effects of $CMRO_2$ on CBF

$CMRO_2$ is affected by temperature. The relationship between temperature and the cerebral metabolic rate was obtained using equation [7].[6,9] From this equation, the $CMRO_2$ is determined based on the body temperature (esophageal).

Eq. [7]: $CMRO_2$ as function of temperature

$$CMRO_2 = \exp(a + b * T)$$

(a = -2.7579, b = 0.1089)

The $CMRO_2$ value is then used to determine the change in %CBF. Equation [8] describes the linear relationship between %CBF and the metabolic rate.[8]

Eq. [8]

$$\%CBF_{CMRO_2} = -9.88769 + 30.87319 * CMRO_2$$

Units: $\%CBF_{CMRO_2}$ (%CBF), $CMRO_2$ (mL O2/100 mL brain/min), T(°C)

The effects of MABP on CBF

MABP is calculated from the systolic and diastolic blood pressures. Using the relationship obtained in literature, the %CBF is determined based on the MABP. Equations [9], [10] and [11] describe this relationship.

Eq. [9]: $0 <= MABP < 60$

$$\%CBF_{MABP} = -9.3627273 + 3.8025758 * MABP - 6.6594872E-2 * (MABP)^2 + 1.0904429E-3 * (MABP)^3 + 8.839161E-6 * (MABP)^4$$

Eq. [10]: $60 <= MABP <= 140$

$$\%CBF_{MABP} = 100$$

Eq. [11]: $140 < MABP < 185$

$$\%CBF_{MABP} = -9824.923 + 255.15379 * MABP - 2.43203203 * (MABP)^2 + 1.0147878E-2 * (MABP)^3 - 1.555245E-5 * (MABP)^4$$

Units: $\%CBF_{MABP}$ (%CBF), MABP (mmHg)

Cerebral Blood Flow (CBF)

The cerebral blood flow is calculated by adding or subtracting the changes in CBF caused by PaO_2, $PaCO_2$, $CMRO_2(T)$, and MABP to the normal value of 52.5 mL/100g brain/min for CBF. The %CBF from the previous equations [1, 2, 3, 4, 5, 6, 8, 9 , 10 and 11] are used to calculate the new CBF value. Following equation [12], the %CBF is subtracted by 100% in order to find the change in CBF. Depending on whether the change in CBF is positive or negative, the CBF will either increase or decrease.

Eq. [12]

$$CBF = 52.5 + \left(52.5*\left(\%CBF_{PaO_2} - 100\%\right) \div 100\%\right) + \left(52.5*\left(\%CBF_{PaCO_2} - 100\%\right) \div 100\%\right) + \left(52.5*\left(\%CBF_{CMRO_2} - 100\%\right) \div 100\%\right) + \left(52.5*\left(\%CBF_{MABP} - 100\%\right) \div 100\%\right)$$

Units: CBF (mL/100g brain/min), $\%CBF_{CMRO2}$ (%CBF), $\%CBF_{MABP}$ (%CBF), $\%CBF_{PaO2}$ (%CBF), $\%CBF_{PaCO2}$ (%CBF)

Cerebral Blood Volume (CBV)

From the CBF value, the CBV is determined. The relationship between CBF and CBV was determined using data extrapolated from the literature.[8] A linear curve was fitted so that it would go through the origin and is described by Eq. [13].

Eq. [13]

$$CBV = \frac{15*CBF}{10.23476}$$

Units: CBV (mL/100mg brain/min), CBF (mL/100 mL brain/min)

Intracranial Pressure (ICP)

The elastance curve, ICP vs. Change in Total Intracranial Volume is shown in Fig. 2. The total change in intracranial volume can represent changes in CBV, CSF volume, brain volume and mass volume. The mass volume can represent an acute change in intracranial volume such as a hematoma or a gradual change such as a tumor. By entering a mass volume at the beginning of the simulation, the compensation zone of the elastance curve (flat portion) can be diminished and the ICP will respond to smaller changes in volume. Equations [14], [15] and [16] describe the response of ICP to changes in total intracranial volume due to CBV and mass volume in accordance to the elastance curve.

Eq. [14]: CBV <= 39

$$ICP = 12$$

Eq. [15]: 39 < CBV <= 104

$$ICP = -21895944 + 14.990125*CBV - 0.34429666*(CBV)^2 + 3.2218647E-2*(CBV)^3 - 9.380431E-6*(CBV)^4$$

Eq. [16]: CBV > 104

$$ICP = -479 + 6.0*CBV$$

Units: ICP (mmHg), CBV (mL)

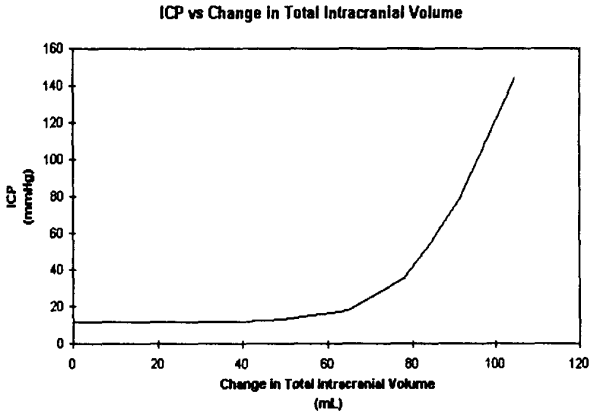

FIGURE 2. Brain model elastance curve

Cerebral Perfusion Pressure (CPP)

Using the ICP value or the central venous pressure (CVP), the cerebral perfusion pressure is calculated using equations [17] or [18]. The CPP is determined by subtracting CVP or ICP from MABP. Maintaining an adequate CPP is important to clinicians and, in a clinical setting, it is common practice to use CVP instead of ICP since the latter is not monitored on a regular basis.

Eq. [17]: CVP >= ICP

CPP = MABP - CVP

Eq. [18]: ICP > CVP

CPP = MABP - ICP

Units: CPP (mmHg), MABP (mmHg), CVP (mmHg), ICP (mmHg)

RESULTS

The purpose of the brain model is to show how intracranial dynamics are affected by patient care. To demonstrate the effects of patient management on intracranial dynamics, we ran a scenario on the human patient simulator which depicted a trauma case (Fig. 3). We started with a patient who had suffered trauma that resulted in an elevated ICP which was set at approximately 20-22 millimeters of mercury. The brain model allows the user to enter a volume mass or the desired ICP at the beginning of each simulation. If an ICP value is entered, the brain model will calculate the approximate mass volume needed to elevate ICP in accordance to the elastance curve (Fig. 2). The patient was pre-oxygenated prior to arriving in the operating room. This can be seen at the top of Fig. 3 where the PaO_2 curve is plotted.

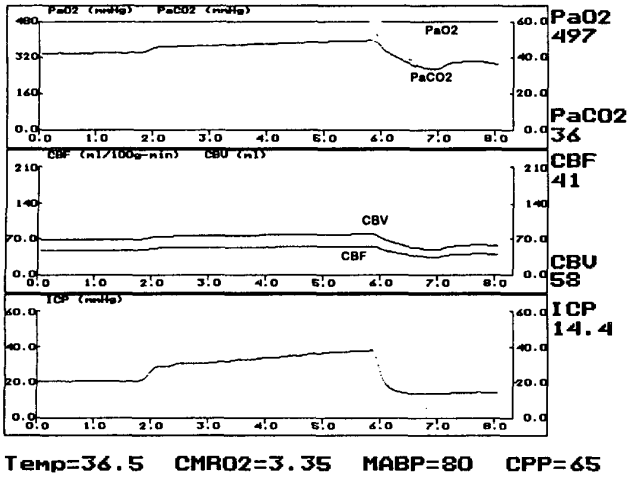

FIGURE 3. Brain model display

For the scenario (Fig. 3), an unconscious spontaneously breathing pre-oxygenated patient with an elevated ICP of 22 mmHg (bottom display) comes into the operating room. One and a half minutes into the procedure (X-axis), the anesthesiologist administers anesthesia with 400 mg of thiopental and 10 mg of vecuronium. After administration of the drugs, the patient's blood pressure starts to drop and the patient becomes apneic at approximately two minutes. The $PaCO_2$ curve (top display) reveals a sharp small increment. The ICP (lower display) increases sharply as a response to the change in carbon dioxide. This sharp response to CO_2 demonstrates the linear relationship between CBF and $PaCO_2$ (Eqs. 4,5 and 6) in which the brain's response is to increase CBF in order to maintain the oxygen supply. As the CBF increases (middle display) so does the CBV (middle display) which then results in a change in total intracranial volume causing the ICP to go up. Since the patient started with an elevated ICP, small changes in blood volume can cause significant increases in ICP. This is explained by the fact that we are located at the shoulder of the elastance curve (Fig. 2) which shows an exponential relationship. Looking at the $PaCO_2$, CBF, CBV and the ICP curves we can see that they all increase together.

Four minutes into the procedure, the anesthesiologist attempts to ventilate the patient but cannot. The anesthesiologist administers 80 mg of esmolol in order to reduce the effects of the laryngoscopy which causes increases in blood pressure and heart rate. As seen in Fig. 3, the $PaCO_2$ continues to increase which results in increases of the CBF, CBV, and ICP. Approximately, six minutes into the procedure, the clinician successfully intubates the patient and starts hyperventilating the patient. Hyperventilation causes the $PaCO_2$ to drop sharply inducing a decrease in CBF and CBV. The decrease in CBV is then reflected on the ICP which also shows a sharp decrease. Once the patient's is intubated and under mechanical ventilation, the ICP can be managed by adjusting physiological parameters of the patient.

DISCUSSION AND CONCLUSION

From inspecting at the display of the Model (Fig. 3), it is clear that a real time display of intracranial dynamics can be a useful tool in teaching the effects of changing physiology during patient care management. The scenario demonstrated how patient management affected ICP. The brain model offers a straightforward approach of bringing together existing relationships between physiology and the brain found in the literature. When interfaced with a full scale patient simulator, brain response to changes in physiology was demonstrated in a real time fashion. Individuals using the simulator can see the response to their actions immediately. Because most clinicians usually rely on assumptions based on the physiologic relationships used in this model, we expect this model to be a useful tool.

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