2004

Calibrating an Intracranial Pressure Dynamics Model With Clinical Data - A Progress Report

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Citation Details

Calibrating an Intracranial Pressure Dynamics Model with Annotated Clinical Data--a Progress Report

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This work was supported in part by the Thrasher Research Fund
Background: Intracranial Pressure (ICP)

- Traumatic brain injury often causes ICP to increase
  - Frequently due, at least initially, to internal bleeding (hematoma)
- Persistent elevated ICP $\Rightarrow$ reduced blood flow $\Rightarrow$ insufficient tissue perfusion (ischemia) $\Rightarrow$ secondary injury $\Rightarrow$ poor outcome
- Poor outcomes often occur despite the availability of many treatment options
  - The pathophysiology is complex and only partially understood
Background: ICP Dynamic Modeling

• Many computer models of ICP have been developed
  → Models have sophisticated logic
  → Potentially very helpful in a clinical setting
• However, clinical impact of models has been minimal
  → Complex models are difficult to understand and use
• Another issue is that clinical data often lack the annotations needed to facilitate modeling
  → Exact timing for medications, CSF drainage, ventilator adjustments, etc.
Research Objective

• Use an IRB approved protocol to collect prospective clinical data
  ➔ Carefully annotate the data regarding timing of therapy and mild physiologic challenges
• Use the data to calibrate a computer model of ICP dynamics
• Use the calibrated model to estimate patient response to treatment and challenges
• Compare model response to actual patient response
• Improve the model and the calibration process
Method: Experimental Protocol

• Change the angle of the head of the bed (HOB)
  ➔ From 30° to 0° for example, and vice versa
  ➔ Such changes directly influence ICP

• Change the minute ventilation (VR)
  ➔ Clinician adjusts VR to achieve specified ETCO₂
  ➔ Decreasing ETCO₂ (mild hyperventilation) triggers cerebrovascular autoregulatory (AR) response
    ✓ Intracranial vessels constrict ➔ intracranial blood volume decreases ➔ ICP decreases
  ➔ Increasing ETCO₂ has the opposite effect
Method: ICP Dynamic Model

- Core model logic
  - State variables: fluid volumes and AR status
  - Estimated parameters: compliance, resistance, hematoma volume and rate, control parameters
  - Computed variables: fluid flows and pressures
- Six intracranial volumes (state variables)
  - Arterial blood (ABV), Capillary blood (CBV)
  - Venous blood (VBV), Cerebral spinal fluid (CSF)
  - Brain tissue (BTV), Hematoma (HV)
Method: Diagram showing Volumes & Flows

- Arterial Blood Flow
- Capillary Blood Flow
- Venous Blood Flow
- Blood Outflow
- Subdural Bleeding
- Hematoma
- Brain Volume
- Swelling
- CSF Production
- CSF Volume
- Reabsorption of CSF

Smooth Muscle State
Method: Model Logic for Pressures

- **Total Cranial Volume** = ABV + CBV + VBV + CSF + BTV + HV
- **Intracranial Pressure (ICP)**
  \[
  = \text{Base ICP} \times 10^{\left(\text{Total Cranial Volume} - \text{Base Cranial Volume}\right)/\text{PVI}}
  \]
  - PVI (pressure-volume index) is the amount of added fluid that would cause pressure to increase by a factor of 10
- **Arterial, capillary, and venous pressures**
  \[
  \longrightarrow P_{ab} = \text{ICP} + \left(\frac{\text{ABV}}{\text{Arterial Compliance}}\right)
  \]
  \[
  \longrightarrow P_{cb} = \text{ICP} + \left(\frac{\text{CBV}}{\text{Capillary Compliance}}\right)
  \]
  \[
  \longrightarrow P_{vb} = \text{ICP} + \left(\frac{\text{VBV}}{\text{Venous Compliance}}\right)
  \]
Method: Model Logic for Cerebrovascular AR

• Arteriolar resistance changes in order to maintain needed blood flow rate
  ➔ higher resistance = constriction
  ➔ Lower resistance = dilation
  ➔ Time constant for adjustment process: 2-3 minutes
  ➔ Upper and lower bounds

• Cerebrovasular AR responds to multiple stimuli
  ➔ Changing Metabolic needs (e.g., asleep vs. awake)
  ➔ Changing ICP, arterial blood pressure, HOB, and VR
Results: Clinical Data, HOB Changes

- Clinical data showing changes in ICP (mmHg) over time (seconds).
- Changes in HOB (Head Of Bed) positions indicated.
- Graph illustrates the impact of different HOB positions on ICP.
Results: Clinical Data, ETCO2 Changes

![Graph showing ICP changes over time with VR:12 and VR:15 annotations.](image-url)
Results: Model Response to HOB Decrease

Note: Actual ICP data has been low-pass filtered and decimated to remove the pulsatile component.
Results: Model Response to HOB Increase

Note: Actual ICP data has been low-pass filtered and decimated to remove the pulsatile component.
Results: Model Response to ETCO₂ Increase

Note: Actual ICP data has been low-pass filtered and decimated to remove the pulsatile component.
Results: Model Response to ETCO$_2$ Decrease

Note: Actual ICP data has been low-pass filtered and decimated to remove the pulsatile component.
Discussion: Model vs. Actual Response

- Model response to raising HOB is very similar to actual response
- Model Response to lowering the HOB is less similar
  - This is plausible since lowering HOB increases ICP, and the body has several mechanisms to resist such increases
  - Most of these are not included in the current model
- Response to ETCO$_2$ changes did not fully reflect the patient’s actual response
  - This is not unexpected, for the same reason:
    - Reliance on a single cerebrovascular AR mechanism in the model
Discussion: Summary

- A model of ICP dynamics was calibrated to replicate the ICP recorded from specific patient during an experimental protocol.
- The calculated ICP closely resembles actual ICP.
- The cerebrovascular AR logic in the model only partially captures the patient’s response to respiration change.
- Next steps: (1) refine the AR logic in the model (2) use optimization to automate the calibration process (3) predict response.