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Wayne W. Wakeland  
*Portland State University*, wakeland@pdx.edu

Brahm Goldstein  
*Oregon Health & Science University*

James McNames  
*Portland State University*

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

W. Wakeland\textsuperscript{1} B. Goldstein\textsuperscript{2} J. McNames\textsuperscript{3}

\textsuperscript{1}Systems Science Ph.D. Program, Portland State University
\textsuperscript{2}Complex Systems Laboratory, Oregon Health & Science University
\textsuperscript{3}Biomedical Signal Processing Laboratory, Portland State University

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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
  $\rightarrow$ 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 ETCO$_2$
  - Decreasing ETCO$_2$ (mild hyperventilation) triggers cerebrovascular autoregulatory (AR) response
    - Intracranial vessels constrict $\rightarrow$ intracranial blood volume decreases $\rightarrow$ ICP decreases
  - Increasing ETCO$_2$ 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
Method: Model Logic for Pressures

- **Total Cranial Volume** = \( ABV + CBV + VBV + CSF + BTV + HV \)
- **Intracranial Pressure (ICP)**
  \[
  \text{ICP} = \text{Base ICP} \times 10^{\frac{(\text{Total Cranial Volume} - \text{Base Cranial Volume})}{\text{PVI}}} 
  \]
  - 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**
  - \( P_{ab} = \text{ICP} + \frac{(ABV)}{(Arterial Compliance)} \)
  - \( P_{cb} = \text{ICP} + \frac{(CBV)}{(Capillary Compliance)} \)
  - \( P_{vb} = \text{ICP} + \frac{(VBV)}{(Venous Compliance)} \)
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

- Cerebrovascular 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
Results: Clinical Data, ETCO2 Changes

![Graph showing changes in ICP over time with annotations VR:12 and VR:15](image)

- **ICP (mmHg)**
- **Time (seconds)**
- **VR:12**
- **VR:15**
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 ETCO$_2$ 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.