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PRECURSORS TO RAPID ELEVATIONS IN INTRACRANIAL PRESSURE

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Abstract: **Intracranial pressure (ICP) monitoring and management have substantially improved the outcome of patients with traumatic brain injury (TBI). However, rapid elevations in ICP remain a significant problem as they may lead to secondary brain injury and worse outcome due to cerebral ischemia. Current therapy is targeted towards treating rapid ICP elevations after they occur. Ideally, anticipatory treatment to obviate any elevation in ICP could occur if reliable precursors to ICP elevation were determined. In this paper, we report evidence for a physiologic transition zone prior to rapid elevations in ICP. We found that in thirty-three episodes of ICP elevation recorded from eleven patients, there was a statistically consistent decrease in the cardiac component of the ICP signal and the coefficient of correlation between the ICP trend and the pulse amplitude. We conclude that specific ICP signal metrics may serve as precursors that characterize the transition zone prior to a rapid elevation and may enable prediction of these elevations up to thirty seconds ahead.**

Keywords: **Intracranial pressure, Intracranial hypertension, Cerebral Perfusion Pressure, Autoregulation, Time series Prediction, Traumatic brain injury, Head Injury**

I. Introduction

Traumatic brain injury (TBI) is the leading cause of death and disability in children in the United States [1]. Elevated intracranial pressure (ICP) following TBI may result in secondary injury due to decreased cerebral perfusion pressure[1] (CPP) and cerebral ischemia[2]. ICP monitoring and therapeutic interventions to control elevated ICP ($>$ 20 mmHg) have resulted in improved outcomes [2–4]. However, current therapy is targeted towards treating rapid ICP elevations *after* they occur. Ideally, anticipatory treatment based on reliable detection of precursors could obviate elevations in ICP.

In many patients, including those with TBI, there is a natural progression of physiologic states from the time of injury, or onset of disease, through recovery or death [5]. The physiologic state of the patient may shift rapidly from from a compensated physiologic state to an uncompensated disease state. We hypothesize that there exist physiologic "transition zones" between compensated and uncompensated disease states, and that these transition zones may be detected by a careful analysis of physiologic signals. Accurate characterization of the transition zone prior to an uncompensated TBI state with an elevated ICP could lead to prophylactic therapy and could further improve outcome.

Other investigators have described a number of precursors to ICP elevations. Szewczykowski *et al.* described a "warning zone" in which the amplitude of ICP variations is strongly related to the mean ICP [6]. They hypothesized that this was caused by impaired compensating ability. Turner *et al.* observed that four patients who developed elevated ICP had increased variance over periods of thirty-three minutes prior to an ICP increase [7]. Several groups have found that an increase in the cardiac component[3] of the ICP signal precedes elevations [2,8]. Czosnyka *et al.* observed a short spontaneous decrease in ABP at the beginning of plateau waves in eleven of sixteen cases [9].

A few investigators have also attempted time series prediction of the ICP signal [10–12]. These attempts employed wavelet decompositions to separate the signal into different frequency bands followed by neural networks to predict the wavelet coefficients, which were finally used to construct the predicted signal segments. Although these methods were able to reproduce the cardiac component of the intracranial pressure, they were not able to accurately track trends, and the ability of these methods to predict rapid elevations was not reported.

Based on preliminary analysis of heart rate, ABP, and ICP spectrograms from four TBI patients, we noticed a significant change in the cardiac component of the ICP signal 10-25 seconds prior to an elevation in ICP. The following is a report of our detailed findings from eleven pediatric patients with severe TBI.

II. Methodology

A. Patient Population and Management

This study included eleven patients with head injuries who had a mean Glasgow Coma Scale (GCS) score of 6 (range 3–14) and were admitted to the pediatric intensive care unit at Doernbecher Children's Hospital. The study was reviewed and approved by the Institutional Review Board of Oregon Health and Science University, and the requirement for informed consent was waived. There were seven female and four male patients with an age range of 3 to 18 years (mean age 7.2 years). Nine of the patients

[1]The CPP is defined as the difference between the systemic arterial blood pressure and the intracranial pressure, CPP = ABP − ICP.

[2]Ischemia is a decrease in blood supply caused by constriction or obstruction of the blood vessels or decreased blood volume.

[3]The cardiac component of the ICP signal is defined as the frequency components that are near the heart rate.