Acute clinical grading in pediatric severe traumatic brain injury and its association with subsequent intracranial pressure, cerebral perfusion pressure, and brain oxygenation

ANTHONY A. FIGAJI, F.C.S.,^{1,2} EUGENE ZWANE, Ph.D.,³ A. GRAHAM FIEGGEN, F.C.S.,^{1,2} JONATHAN C. PETER, F.C.S.,¹ AND PETER D. LEROUX, M.D.⁴

Divisions of ¹Neurosurgery and ²Pediatric Neuroscience, School of Child and Adolescent Health, Institute for Child Health, University of Cape Town, Red Cross Childrens Hospital, Rondebosch, Cape Town, South Africa; ³Infectious Disease Epidemiology Unit (Biostatistics), School of Public Health and Family Medicine, University of Cape Town; ⁴Department of Neurosurgery, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania

Object. The goal of this paper was to examine the relationship between methods of acute clinical assessment and measures of secondary cerebral insults in severe traumatic brain injury in children.

Methods. Patients who underwent intracranial pressure (ICP), cerebral perfusion pressure (CPP), and brain oxygenation (PbtO₂) monitoring and who had an initial Glasgow Coma Scale score, Pediatric Trauma Score, Pediatric Index of Mortality 2 score, and CT classification were evaluated. The relationship between these acute clinical scores and secondary cerebral insult measures, including ICP, CPP, PbtO₂, and systemic hypoxia were evaluated using univariate and multivariate analysis.

Results. The authors found significant associations between individual acute clinical scores and select physiological markers of secondary injury. However, there was a large amount of variability in these results, and none of the scores evaluated predicted each and every insult. Furthermore, a number of physiological measures were not predicted by any of the scores.

Conclusions. Although they may guide initial treatment, grading systems used to classify initial injury severity appear to have a limited value in predicting who is at risk for secondary cerebral insults.

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KEY WORDS • brain oxygenation • cerebral perfusion pressure • clinical assessment • Glasgow Coma Scale • secondary insult • traumatic brain injury

IN pediatric patients who have experienced trauma, clinical assessment and clinical grading at admission can be used to guide treatment protocols and to predict outcome.³ In large part, these various grading systems, for example, the GCS,¹³ PTS,²⁸ PIM2,²⁶ pupillary reactivity,³⁰ and brain CT classification,¹⁴ are associated with the severity of the patient's primary injury. However, outcome after severe TBI is affected also by secondary cerebral injury that evolves during the days after the initial primary insult. Therefore, current treatment of a child with severe TBI in the PICU attempts to avoid or ameliorate secondary cerebral injury.

Common secondary cerebral insults in TBI that are associated with adverse outcomes include elevated ICP,

reduced CPP, hypotension, systemic hypoxia, and brain hypoxia.^{2,4–6,8,10,11,18,21,22,24,27,29,31} These and other secondary insults, although potentially avoidable, are common and may contribute to death in as many as 42% of children who die after admission for head injury.²³ The relationship between outcome and the various grading or scoring systems used to classify pediatric patients who have suffered trauma is well described. However, the relationship between these grading systems of injury and the likelihood of secondary insults is less well defined.

In this study we sought to examine the relationship between acute measures of injury severity on admission and the subsequent development of secondary cerebral insults in children with severe TBI after admission to the PICU.

Methods

Ethics approval for the study was obtained from the institutional review boards of the Red Cross Children's Hospital and the University of Cape Town.

Abbreviations used in this paper: CPP = cerebral perfusion pressure; FiO₂ = fraction of inspired O₂; GCS = Glasgow Coma Scale; ICP = intracranial pressure; MABP = mean arterial blood pressure; PbtO₂ = brain O₂ tension; PICU = pediatric intensive care unit; PIM2 = Pediatric Index of Mortality, revised version; PTS = Pediatric Trauma Score; SaO₂ = saturation of O₂; TBI = traumatic brain injury.

TABLE 1: Admission demographic and clinical variables

Category	No. of Patients*
age (range)	6.5 ± 3.4 yrs (9 mos–14 yrs)
initial GCS score	
3	5
4	9
5	12
6	10
7	11
8	5
median GCS motor component score (range)	3 (1–5)
pupil reaction	
bilat reactive	41
unilat nonreactive	5
bilat nonreactive	6
PTS	3 ± 1.4
PIM2 score	0.16 ± 0.2
CT classification	
I	3
II	31
III	13
IV	3
evacuated mass lesion	2
nonevacuated mass lesion	0

* Unless stated otherwise.

Patient Selection

Clinical and physiological data were retrospectively obtained from a larger prospective observational study of severe TBI in children at Red Cross Children's Hospital. Patients were included in this study if they met the following criteria: 1) they underwent continuous ICP, CPP, and PbtO₂ monitoring; and 2) the initial injury was classified according to the GCS, motor component of the GCS, PTS, PIM2, pupil reactivity, and the Marshall CT classification of head injury. Each of these grades had to be recorded for a patient to be included in the study.

Grading of TBI Severity at Patient Admission

Glasgow Coma Scale. Postresuscitation GCS score (pediatric coma score for preverbal children) was recorded in all patients. The motor component of the GCS was separately documented.

Pediatric Trauma Score. The PTS includes 6 variables: weight, systolic blood pressure, mental status, airway maintenance, skeletal injury, and open wounds.²⁸

Pediatric Index of Mortality. The PIM2 uses the following PICU admission variables to predict outcome:²⁶ systolic blood pressure, pupillary reaction (fixed or reactive), PaO₂/FiO₂ ratio, base excess, elective admission (yes/no), mechanical ventilation (yes/no), recovery from

TABLE 2: Summary of physiological variables for all patients*

Category	Value
mICP ₂₄	16.5 ± 11 mm Hg
mICP _{total}	14.8 ± 9.3 mm Hg
ICP _{peak}	34.6 ± 17.4 mm Hg
ICP >20 mm Hg	17.9 ± 24.8 hrs
mICP >20 mm Hg	22.3 ± 13.4 mm Hg
CPP _{low}	40.2 ± 15.9 mm Hg
median (IQR) no. of hrs at CPP <40 mm Hg	0 (0–2)
median (IQR) no. of hrs at CPP <50 mm Hg	3 (0–12)
median (range) no. of episodes of PaO ₂ <60 mm Hg	0 (0–16)
lowest PaO ₂	87 ± 44 mm Hg
median (range) no. of episodes SaO ₂ <90%	0 (0–9)
lowest PbtO ₂	11.2 ± 7.4 mm Hg
mPbtO ₂ 24	28.4 ± 11.5 mm Hg
median (range) no. of hrs at PbtO ₂ 5 mm Hg	0 (0–20)
median (range) no. of hrs at PbtO ₂ <10 mm Hg	1 (0–22)

* IQR = interquartile range. See *Secondary Cerebral Insults in Methods* for definitions of other abbreviations.

surgery (yes/no), cardiac bypass (yes/no), and high risk/low risk diagnosis.

Pupil Reactivity. In this study, postresuscitation pupillary reactions were classified as bilaterally reactive (1), unilaterally nonreactive (2), or bilaterally nonreactive (3). The influence of medications was excluded.

Head CT Classification. The severity of TBI was classified according to the Marshall system¹⁴ based on head CT scan findings at admission. The grades include the following: diffuse injury Score I (normal scan), II (abnormal scan with open cisterns), III (effaced or obliterated cisterns), or IV (midline shift > 5 mm); evacuated mass lesion; and lesion > 25 cm³ not surgically evacuated.

Patient Age. Age also was used in this study to classify patient risk given that age may influence outcome^{9,17} and physiological thresholds.⁴

Physiological Monitoring

The PbtO₂, ICP, CPP, and pulse oximetry (SaO₂) were monitored continuously and recorded hourly. The CPP was calculated as the difference between MABP and ICP (CPP = MABP–ICP). The PbtO₂ catheter (LICOX, Integra Neuroscience) was placed 2.5 cm below the cortical surface into right frontal lobe white matter that appeared normal on the head CT scan. The PbtO₂ monitor function and stability were confirmed by an appropriate PbtO₂ increase following an O₂ challenge. A follow-up

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head CT scan was obtained in each patient to confirm correct placement of the monitor.

Management of ICP, CPP, and PbtO₂

All patients were resuscitated and underwent endotracheal intubation and mechanical ventilation. Each patient was treated in the PICU according to a local algorithm consistent with the Guidelines for the Management of Severe Pediatric TBI.¹ In general, a minimum CPP target of 50 mm Hg was set for patients and test elevation of CPP was performed if the PbtO₂ was < 20 mm Hg. Compromised (low) PbtO₂ was defined as < 20 mm Hg and was treated using a hierarchical treatment algorithm in a cause-directed fashion. In general, this meant attempting to identify a possible cause for the reduction in PbtO₂ and more aggressively treating abnormal or borderline values for ICP, CPP, low PaCO₂, low arterial saturation/low PaO₂, and low hemoglobin. The following specific measures were used, depending on ICP, MABP, PaO₂, hemoglobin, transcranial Doppler flow velocities and status of autoregulation (when known), and PaCO₂: 1) elevated ICP was treated more aggressively if present; 2) the patient's blood pressure was elevated to test PbtO₂ at a higher CPP with volume infusion and/or inotropic support unless impaired autoregulation caused concomitant increases in ICP with elevated blood pressure; 3) higher PaCO₂ was tolerated to induce cerebral vasodilation if ICP was not elevated; 4) a lower threshold for blood transfusion was used to increase hemoglobin to 10 g/dl or more; and 5) the FiO₂ was increased as an emergency temporary measure or if PbtO₂ remained low despite optimization of the above parameters.

Secondary Cerebral Insults

The following secondary cerebral insults were evaluated in this study: elevated ICP, reduced CPP, compromised PbtO₂, and systemic hypoxia. Physiological data were collated from hourly observations of continuously monitored ICP, CPP, PbtO₂, and peripheral SaO₂. In addition, all arterial blood gas samples were evaluated. Values considered abnormal were in keeping with recently published guidelines.¹

Intracranial Pressure. The following ICP values were calculated and recorded for each patient as: mean ICP during the first 24 hours (mICP₂₄), mean ICP for the full duration of monitoring (mICP_{total}), number of episodes of ICP > 20 mm Hg, mean value for all ICP readings > 20 mm Hg (mICP > 20 mm Hg), and highest ICP (ICP_{peak}).

Cerebral Perfusion Pressure. The CPP insults were calculated for each patient as the lowest CPP (CPP_{low}) and the number of episodes of CPP < 40 and CPP < 50 mm Hg.

Brain O₂ Tension. The following PbtO₂ values were calculated for each patient: minimum PbtO₂ recorded during the patient's hospital course, mean PbtO₂ during the first 24 hours (mPbtO₂24), and the number of episodes of PbtO₂ < 10 or < 5 mm Hg.

Systemic Hypoxia. Systemic hypoxia was defined as

PaO₂ < 60 mm Hg on arterial blood gas measurement, or arterial SaO₂ < 90% on peripheral oximetry or arterial blood gas. In addition the lowest PaO₂ observed also was recorded.

Treatment Variables

To supplement our evaluation of the relationship between initial grading and secondary cerebral insults we also examined whether a patient received one of the following treatments: hypertonic saline, use of inotropes, blood transfusion, and decompressive craniectomy.

Statistical Analysis

The individual acute clinical scores and head CT classification recorded in each patient were evaluated as independent variables and subsequent physiological parameters as dependent variables. Spearman correlation coefficients were used to determine relationships between scores and physiological variables. For categorical scores, the Pearson chi-square test was used for relationships with categorical variables and Kruskal–Wallis for continuous variables. The Kruskal–Wallis test evaluates equivalence between 2 medians while ignoring the ordering in the categorical covariates. The Pearson chi-square test was used to test the frequency distribution of categories within the individual scores. Scores that were significant in univariate analysis were entered into a multivariate analysis model. Significance was set at a probability level = 0.05. Data are reported as means ± SD or medians (interquartile range and/or range).

Results

Clinical Characteristics

Fifty-two pediatric patients with severe TBI who were monitored for ICP, CPP, and PbtO₂ between June 2006 and May 2008 were included in the study. Demographic, clinical, and physiological data are summarized in Table 1. All children were < 15 years old. The distribution within age groups was as follows: 0–3 years (27.9%), 4–7 years (38.5%), 8–11 years (30.8%), and 12–14 years (3.8%). The mechanism of injury was motor vehicle accident–related in 40 (76.9%), crush injury in 3 (5.8%), gunshot wound in 4 (7.7%), blunt assault in 2 (3.8%), fall from a height in 1 (1.9%), stab to the head in 1 (1.9%), and nonaccidental injury (shaken baby syndrome) in 1 (1.9%). The initial systolic blood pressure was < 90 mm Hg in 13 patients (25%), and initial systemic hypoxia (SaO₂ < 90% or PaO₂ < 60 mm Hg) was documented in 14 (26.9%). Physiological variables for all patients after admission to the PICU are summarized in Table 2.

Univariate Analysis

Glasgow Coma Score. The postresuscitation GCS score was significantly associated with lowest CPP ($p = 0.004$), CPP < 40 mm Hg ($p = 0.017$), CPP < 50 mm Hg ($p = 0.019$), PbtO₂ < 5 mm Hg ($p = 0.008$) (Fig. 1), and PbtO₂ < 10 mm Hg ($p = 0.045$). There were no associations with

FIG. 1. Graph showing the initial GCS and episodes of $\text{PbtO}_2 < 5$ mm Hg. The GCS scores are grouped in 3 categories: Score 3–4 (14 patients), Score 5–6 (22 patients), and Score 7–8 (16 patients), with the respective frequencies of episodes of $\text{PbtO}_2 < 5$ mm Hg shown for each. Q1 = first quartile; Q3 = third quartile.

any other physiological or treatment variables, including the indices of elevated ICP.

Motor Component of the GCS. The motor response score of the GCS had no significant relationship with any variables when using the Spearman correlation coefficient. However, when the data were examined using the Kruskal–Wallis test a marginal relationship with $\text{PbtO}_2 < 5$ mm Hg ($p = 0.046$) and $\text{PbtO}_2 < 10$ mm Hg ($p = 0.047$) was observed.

Pediatric Index of Mortality 2. The PIM2 score was associated with $\text{CPP} < 40$ mm Hg ($p = 0.008$), $\text{mICP} > 20$ mm Hg ($p = 0.0013$), and ICP_{peak} ($p < 0.0001$).

Pediatric Trauma Score. The PTS had a weak relationship with $\text{PbtO}_2 < 10$ mm Hg ($p = 0.046$), but not with any other variables.

Pupil Reactivity on Admission. Pupil reactivity was associated with lowest CPP ($p = 0.003$), $\text{CPP} < 40$ mm Hg ($p = 0.024$), $\text{CPP} < 50$ mm Hg ($p = 0.032$), $\text{PbtO}_2 < 5$ mm Hg ($p = 0.026$), and $\text{PbtO}_2 < 10$ mm Hg ($p = 0.031$).

Computed Tomography Classification. Individual categories according to admission head CT scan findings demonstrated relationships with $\text{mICP} > 20$ mm Hg ($p = 0.017$), and lowest CPP ($p = 0.006$), $\text{PaO}_2 < 60$ mm Hg ($p = 0.029$), and $\text{SaO}_2 < 90\%$ ($p = 0.023$). Decompressive craniectomy ($p = 0.008$) also was associated with individual Marshall categories. When the various CT categories were dichotomized to Scores I & II and Scores III & IV, the following associations were observed: mICP_{24} ($p = 0.027$), lowest CPP ($p = 0.003$) (Fig. 2), $\text{CPP} < 50$ mm Hg ($p = 0.015$), and $\text{mICP}_{\text{total}}$ ($p = 0.046$). The distribution of observations within the categories of the classification was significantly different; most patients demonstrated Score II and III changes on the initial head CT (44 of 52 patients).

Patient Age. Age was significantly associated with the following: lowest CPP ($p = 0.007$), $\text{CPP} < 40$ mm Hg ($p = 0.001$), and $\text{CPP} < 50$ mm Hg ($p = 0.002$) but with no other variables.

Treatment Variables

Head CT classification was significantly associated

FIG. 2. Graph showing the relationship between CT classification (only diffuse injury Scores I & II compared with Scores III & IV; 50 patients) and lowest CPP and mean ICP for the first 24 hours (mICP_{24}). The 2 patients with evacuated mass lesions were excluded.

with craniectomy ($p = 0.015$) (more patients with diffuse injury Scores III and IV received craniectomy). Pupil reactivity demonstrated a trend toward a significant association with craniectomy ($p = 0.056$) and thiopentone ($p = 0.073$). No other scores demonstrated any other associations with treatment variables.

Multivariate Analysis

Variables found to be significantly associated with secondary cerebral insults were entered into multivariate analysis. These results are summarized in Table 3. The PIM2 score was associated with $\text{mICP} > 20$ mm Hg ($p = 0.001$), ICP_{peak} ($p < 0.0001$), and $\text{CPP} < 40$ mm Hg ($p = 0.001$). Initial GCS was associated with lowest CPP ($p = 0.016$) and $\text{PbtO}_2 < 5$ mm Hg ($p = 0.017$). Pupil reactivity was associated with lowest PbtO_2 ($p = 0.039$) and had a trend toward an association with lowest CPP ($p = 0.057$), $\text{CPP} < 40$ ($p = 0.073$), $\text{PbtO}_2 < 5$ mm Hg ($p = 0.076$), and $\text{PbtO}_2 < 10$ mm Hg ($p = 0.057$). Age was associated with lowest CPP ($p < 0.0001$), $\text{CPP} < 40$ mm Hg ($p = 0.004$), and $\text{CPP} < 50$ mm Hg ($p < 0.0001$). There were no scores that had significant associations with $\text{ICP} > 20$ mm Hg, mICP_{24} , $\text{mICP}_{\text{total}}$, $\text{PaO}_2 < 60$ mm Hg, lowest PaO_2 , and $\text{SaO}_2 < 90\%$. No significant associations between PTS, CT classification, or motor component of the GCS and any of the measured variables were found in multivariate analysis. There were no significant associations between scores and treatment variables in multivariate analysis.

Discussion

In this study we examined 52 children with severe TBI and evaluated the relationship between acute scores of injury severity—including postresuscitation GCS score, PIM2, PTS, CT classification, pupil reactivity and age—and subsequent secondary cerebral insults, in particular elevated ICP, reduced CPP, compromised brain O_2 , and systemic hypoxia. None of the scoring systems had a consistent relationship with all potential secondary insults, and the strength of the association with different measures of secondary cerebral insults differed between the different scores. These results suggest that although acute grading systems may help predict outcome, they

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TABLE 3: Multivariate analysis showing the associations between scores and physiological measures with significant relationships or trends*

Physiological Category	Predictive Score (p Value)	Regression Estimate (95% CI)
ICP		
ICP >20 mm Hg	none	
mICP >20 mm Hg	PIM2 (0.001)	29.2 (12.5-46)†
ICP _{peak}	PIM2 (<0.0001)	43.8 (22.9-64.7)†
mICP ₂₄	none	
mICP _{total}	none	
CPP		
lowest CPP	initial GCS (0.016)	3.6 (0.7-6.5)†
	age (<0.0001)	1.9 (0.8-3)†
	pupils (0.057)	-5.2 (-11.2 to 0.2)
CPP <40 mm Hg	PIM2 (0.001)	11.2 (4.2-17.1)†
	age (0.004)	-0.6 (-0.9 to -0.2)†
	pupils (0.073)	1.8 (-0.167 to 3.7)
CPP <50 mm Hg	age (<0.0001)	-2.4 (-3.7 to -1.1)†
systemic hypoxia		
PaO ₂ <60 mm Hg	none	
lowest PaO ₂	none	
SaO ₂ <90%	none	
PbtO₂		
lowest PbtO ₂	pupils (p = 0.039)	-3.4 (-6.6 to -0.2)†
PbtO ₂ <5 mm Hg	initial GCS (p = 0.017)	-1 (-1.8 to -0.2)†
	pupils (p = 0.076)	1.6 (-0.2 to 3.4)
PbtO ₂ <10 mm Hg	pupils (p = 0.057)	5.1 (-0.2 to 10.3)

* CI = confidence interval.

† Significant result.

may not always predict whether a pediatric patient will develop secondary cerebral insults after severe TBI.

Glasgow Coma Scale

The relationship between postresuscitation GCS score and outcome after TBI has been well described.^{3,6,9,16} Given that sedation in the acute phase may influence 2 components of the GCS, (eye opening and verbal responses), the motor component of the GCS is often considered to represent a more reliable assessment. For preverbal children, the pediatric coma scale is preferred.²⁵ In a large series of pediatric severe TBI, Ducrocq et al.⁹ reported that a threshold GCS score of 5 or less was associated with mortality and poor outcome. In our study, initial GCS demonstrated an association with different measures of CPP and PbtO₂. However, the GCS score was not associated with various measures of elevated ICP. Furthermore, the motor component of the GCS had no consistent relationships with secondary cerebral insults.

Pediatric Trauma Score

The PTS is a combined trauma and physiological score developed to predict outcome in pediatric trauma

patients.²⁸ Its use has been reported in children with TBI,^{9,12} and PTS < 3 is associated with an increased rate of mortality.^{3,12} Although widely used in the assessment of pediatric trauma patients, in this study the PTS failed to correlate with any of the physiological variables known to be associated with secondary insults.

Pediatric Index of Mortality-2

The PIM2 is used to evaluate patients on admission to the PICU. It is easier to use than Pediatric Risk of Mortality III, which has 17 variables and is used to evaluate patients, not on admission but during the first 24 hours in the PICU.²⁰ Although not specifically designed for the evaluation of trauma patients, the PIM2 has been reported to have a close relationship with outcome in pediatric head injury.¹² We observed that PIM2 was associated with several indices of elevated ICP. However, we did not observe an association between PIM2 and episodes of compromised PbtO₂ or systemic hypoxia.

Pupil Reaction

Pupillary reaction is strongly associated with outcome after TBI.^{15,30} In this study, pupil reaction was associated with PbtO₂ and CPP indices, but not with elevated ICP.

Head CT Classification

There are several classification systems that attempt to relate the initial head CT scan findings to outcome.^{6,7,9,19} The most commonly used system is that developed by Marshall et al.¹⁴ In this study we observed that the Marshall grade was associated with some indices of ICP, presumably reflecting the association of compressed cisterns and midline shift (diffuse injury Scores III-IV) with ICP. Also, Scores III and IV were associated with craniectomy in univariate analysis, which probably reflects both the scores' association with elevated ICP and the tendency to perform craniectomy in the setting of obliterated cisterns and high ICP. However, we did not observe any association between the Marshall grade and PbtO₂ indices, and an association with CPP < 50 mm Hg was found only in univariate analysis.

Patient Age

In the present study age was related to all indices of CPP. This may reflect the lower physiological thresholds in younger children or the tendency to target higher CPP values with increasing age.

A number of physiological measures had no relationships with acute assessment scores (Table 3). There were also no predictors for treatment variables, except for CT classification with decompressive craniectomy (univariate analysis only).

Methodological Limitations

There are a number of possible limitations to our study. First, our sample size is relatively small. It is possible that with a larger sample more consistent relationships between the various scores and physiological variables may have been seen. However, we limited the study to

patients who underwent ICP, CPP, and PbtO₂ monitoring. Often only ICP and CPP or blood pressure are evaluated as secondary insults, yet brain hypoxia may occur even when ICP and CPP are adequately managed or even normal,^{10,27} so the addition of PbtO₂ as an indicator of possible secondary injury can help detect episodes of secondary injury that may be otherwise undetected. Second, we examined some but not all scores used to classify patients who have suffered TBI. For example, we did not obtain the Injury Severity Score, Head Abbreviated Injury Severity Score, Pediatric Risk of Mortality III, and Relative Head Injury Severity Score. Instead the scores chosen for our study are commonly used in pediatric trauma units and PICUs. Third, the secondary insults we evaluated did not include hypo- or hyperglycemia, pyrexia, seizures, or sepsis. However, the secondary insults chosen for analysis have been extensively reported, and each is known to be associated with adverse outcome after TBI. Fourth, we cannot exclude that prompt identification and treatment of potential secondary insults did not confound our results. However, treatment of patients was not directed by patients' clinical scores. Similar treatment was delivered to all patients. Also, secondary insults in this series were frequent (data not shown); therefore, if there were significant associations between the high-risk scores and the frequency of secondary insults we would have expected to demonstrate them in this cohort.

Conclusions

We found that acute assessment scores that describe the primary injury sustained by children with severe TBI have a variable relationship with physiological markers of secondary cerebral injury after admission to the PICU. Although we observed associations between individual scores and select measures of secondary cerebral insults, none of the scores evaluated reliably predicted each and every secondary insult. In our opinion, grading systems used to classify initial injury severity, while they may guide initial treatment, appear to have a limited value in predicting who is at risk for secondary cerebral insults. Therefore, clinicians should remain vigilant for the development of secondary insults in all patients who present with significant head trauma.

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Address correspondence to: Anthony A. Figaji, F.C.S., 617 Institute for Child Health, Red Cross War Memorial Children's Hospital, Klipfontein Road, Rondebosch, 7700 Cape Town, South Africa. email: Anthony.Figaji@uct.ac.za.