These data suggest that transport to and from the intensive care unit may influence transport to and from a follow-up head computed tomography (transport head computed tomography [tHCT]) scan.

METHODS: Forty-five patients (24 men, 21 women; Glasgow Coma Scale score ≤8; mean age, 47.3 ± 19.0 years) who had a traumatic brain injury (n = 26) or subarachnoid hemorrhage (n = 19) were retrospectively identified from a prospective observational cohort of PbtO2 monitoring in a neurosurgical intensive care unit at a university-based level I trauma center. PbtO2, intracranial pressure, and cerebral perfusion pressure were monitored continuously and compared during the 3 hours before and after 100 tHCT scans.

RESULTS: The mean PbtO2 before and after the tHCT scans for all 100 scans was 37.9 ± 19.8 mm Hg and 33.9 ± 17.2 mm Hg, respectively (P = .0001). A decrease in PbtO2 (>5%) occurred after 54 tHCTs (54%) and in 36 patients (80%). In instances in which a decrease occurred, the average decrease in mean, minimum, and maximum PbtO2 was 23.6%, 29%, and 18.1%, respectively. This decrease was greater when PbtO2 was compromised (<25 mm Hg) before tHCT. An episode of brain hypoxia (<15 mm Hg) was identified in the 3 hours before tHCT in 9 and after tHCT in 19 instances. On average, an episode of brain hypoxia was 46.6 ± 16.0 (standard error) minutes longer after tHCT than before tHCT (P = .008). Multivariate analysis suggests that changes in lung function (PaO2/fraction of inspired oxygen [FiO2] ratio) may account for the reduced PbtO2 after tHCT (parameter estimate 0.45, 95% confidence interval: 0.024-0.871; P = .04).

CONCLUSION: These data suggest that transport to and from the intensive care unit may adversely affect PbtO2. This deleterious effect is greater when PbtO2 is already compromised and may be associated with lung function.

KEY WORDS: Brain oxygen, Computed tomography, Intracranial pressure; Subarachnoid hemorrhage, Transport, Traumatic brain injury

Patient outcome after severe traumatic brain injury (TBI) or subarachnoid hemorrhage (SAH) often is poor. Clinical and laboratory research demonstrates that much of the poor outcome is associated with delayed cerebral injury that develops after the initial evaluation and neuroimaging (eg, head computed tomography [HCT]). This concept of secondary neuronal injury and its prevention is central to modern TBI and SAH management. Since the introduction of HCT in the 1970s, it has become a critical follow-up study to evaluate patients with severe brain injury. Follow-up HCT scans may be obtained routinely or when there is neurological deterioration or development of intracranial hypertension. This requires transport of severely ill patients from the intensive care unit (ICU) to the computed tomography (CT) suite in the radiology department. Evidence suggests that intrahospital transport (IHT), including even the shortest transports,
Brain tissue oxygen partial pressure (PbtO₂) and intracranial pressure (ICP) can be assessed continuously in brain-injured patients using intraparenchymal sensors, thereby detecting events that may contribute to secondary neuronal injury. Obser-
tional clinical studies demonstrate that compromised PbtO₂ is common in severe brain injury and associated with a poor outcome. Furthermore, the cumulative duration of reduced PbtO₂ is associated with a poor outcome. We examined how IHT, specifically between the ICU and the radiology department for HCT, affects PbtO₂, ICP, and cerebral perfusion pressure (CPP) in critically ill patients.

MATERIALS AND METHODS

Patient Population

Patients with TBI or SAH admitted to the Hospital of the University of Pennsylvania, a level I trauma center, and who had a PbtO₂ monitor in the neurointensive care unit (NICU) were considered for this study. Patients were retrospectively identified from a prospective observational database (the Brain Oxygen Monitoring Outcome study) with institutional review board approval. Patients who (1) had an admission Glasgow Coma Scale (GCS) score of 8 or less immediately after initial resuscitation or deteriorated to a GCS score of 8 or less within 24 hours of admission, (2) underwent follow-up HCT scans that required transport between the NICU and the radiology department (tHCT), (3) were volume resuscitated and hemodynamically stable for at least 12 hours before observation, (4) were intubated and mechanically ventilated, and (5) had at least 3 hours of PbtO₂ monitoring in the NICU before and after tHCT were included in this study.

Patient Management

Each patient was managed according to a locally written algorithm based on published SAH and TBI recommendations, that included aggressive resuscitation, surgical removal of mass lesions, ICP control, and management of secondary cerebral insults. Ventilator management was tailored to maintain PaO₂ at more than 100 mm Hg and PaCO₂ between 30 and 35 mm Hg. Albumin and crystalloid boluses and phenylephrine were used to keep systolic blood pressure at more than 100 mm Hg and central venous pressure at approximately 8 cm H₂O. ICP more than 20 mm Hg was treated using head elevation, sedation (lorazepam), analgesia (fentanyl), muscle paralysis (vecuronium), cerebrospinal fluid drainage, and mannitol. Optimized hyperventilation was used selectively to control ICP provided that the PbtO₂ or jugular oxygen saturation was not adversely affected. Pentobarbital was administered or a decompress hem- icraniectomy performed if ICP remained elevated despite these measures. SAH patients were kept euvoletic, underwent early aneurysm occlusion using surgical or endovascular techniques based on aneurysm morphology, and received nimodipine. If symptomatic vasospasm developed, hypertension was induced, and, if necessary, angioplasty was performed.

Follow-up HCT Scans

Follow-up HCT scans were routinely performed 1 and 3 days after TBI or SAH or when there was neurologic deterioration or an unexplained increase in ICP. All follow-up HCTs were performed in the Department of Radiology. This required transport in an elevator from the fifth to the first floor. The transport team consisted of 1 or 2 ICU nurses, a respiratory therapist, and a hospital transport aid. Each patient had a portable monitor for on-line continuous heart rate, blood pressure, and arterial oxygen saturation (SaO₂) monitoring and was manually ventilated by a respiratory therapist using an Ambu bag. In the radiology department, patients were mechanically ventilated at their previous NICU settings. PbtO₂ was not monitored during transport because the Licox monitor (Integra Lifesciences Corp., Plainsboro, New Jersey) has a separate display monitor, and thus, to facilitate transport, it was not moved with the patient.

ICP Monitoring

ICP (Camino; Integra Neurosciences Corp.), brain temperature, and PbtO₂ (Licox monitor; Integra Lifesciences Corp.) were monitored continuously. Intraparenchymal probes (ICP, brain temperature, and PbtO₂) were inserted at the bedside in the NICU through a burr hole into the frontal lobe and secured with a triple-lumen bolt. The monitors were placed in white matter that appeared normal on admission HCT and on the side of maximal pathology. The function was checked according to published techniques (fraction of inspired oxygen [FiO₂] challenge and follow-up CT scan).

Physiological Measurements and Data Collection

The following parameters were continuously monitored before and after tHCT in each patient: (1) heart rate using 12-lead electrocardiography, (2) mean arterial blood pressure by radial artery catheter, (3) SaO₂ by pulse oximetry, (4) ICP, (5) brain temperature using an intraparenchymal temperature probe (Licox monitor; Integra Lifesciences Corp.), and (6) local PbtO₂. CPP was estimated from the measured parameters (CPP = mean arterial pressure − ICP). Physiological parameters were recorded continuously using a bedside monitor (Component Monitoring System M1046-9090C; Hewlett Packard, Palo Alto, California) linked to a computerized, multimodality data acquisition system (MF’100; Biopac, Goleta, California). In addition, these physiological variables and FiO₂ and central venous pressure were recorded every 15 minutes on the ICU flowsheet. PaO₂, and hemoglobin (Hgb) were measured between 1 and 3 hours before and after a tHCT scan.

Statistical Analysis

Each tHCT was considered a separate event, and each patient at each tHCT scan served as his or her own internal control. Physiological measurements and outcome PbtO₂ were characterized using the mean, minimum, and maximum values measured during the 3 hours before and after a tHCT scan. The ICP, CPP, and PbtO₂ values recorded each minute were averaged every 15 minutes and the means calculated from each 15-minute period. The means and standard deviations were given for each of these variables. Differences between mean values 3 hours before and after a tHCT scan were evaluated using a paired t test. To explain the PbtO₂ change before and after a tHCT scan, a multivariable linear regression model was fit to explore the association between the PbtO₂ changes with candidate predictors. These included age, sex, CPP, Hgb, and the FiO₂/PaO₂ ratio. Robust variance estimator was used to account for the clustering effect of multiple CT scans in the same patient. Data are
expressed as the mean ± standard deviation or as the median where the data are not normally distributed unless otherwise stated. A \( P \) value of <.05 was considered statistically significant. All analyses were performed using the commercially available software package SAS Version 9.1 (SAS, Inc., Cary, North Carolina).

RESULTS

Patient Characteristics

Forty-five patients met all inclusion criteria. There were 24 men and 21 women, mean age 47.3 ± 19.1 years. Twenty-six patients had TBI and 19 patients had aneurysmal SAH. All patients had an admission GCS score of 8 or less except for 2 SAH patients who were admitted with a good grade but deteriorated shortly thereafter to a GCS score of 8 or less.

Follow-up HCT Scans

One hundred HCT scans that required transport between the NICU and the radiology department were performed. Each patient received at least 1 tHCT scan; 76 of these were routine follow-up tHCTs and 24 were clinically indicated (ie, a deterioration in neurological condition or significant increase in ICP). Among the 45 patients, 16 patients had 1 tHCT scan, 12 patients had 2 tHCT scans, 11 patients had 3 tHCT scans, and 6 patients had more than 3 tHCT scans. At the time of tHCT scan, all patients were intubated and mechanically ventilated. Mean total transport time including preparation for transport, transport to and from the radiology department, time in the CT suite, and time to reconnect all monitors and reinstitute therapy in the NICU was 1.46 hours (95% confidence interval: 1.34-1.59). A significant change in management occurred after 19 (79%) of “indicated” tHCTs and 4 (5.3%) of the “routine” follow-up tHCTs.

Physiological and Clinical Variables

The \( \text{PaO}_2/\text{FiO}_2 \) ratio, \( \text{Hgb} \), ICP, and CPP were averaged 3 hours before and after a tHCT scan and the differences calculated (Table 1). The mean ICP before and after the 100 tHCT scans was 12.9 ± 6.6 mm Hg and 13.2 ± 8.5 mm Hg, respectively. The mean CPP before and after the 100 tHCT scans was 83.9 ± 15.7 mm Hg and 84.5 ± 14.5 mm Hg, respectively. The mean minimum and maximum ICP and CPP are listed in Table 1. To examine which variables other than ICP and CPP may affect \( \text{PbtO}_2 \), we compared \( \text{FiO}_2, \text{SaO}_2, \text{PaO}_2, \) and \( \text{Hgb} \) before and after tHCTs. To estimate lung function, we used the \( \text{FiO}_2/\text{PaO}_2 \) ratio. These variables were similar before and after tHCTs (Table 1).

Was IHT Associated With Compromise of Brain Oxygen?

A decrease in \( \text{PbtO}_2 \) defined as reduction more than 5% occurred after 54 tHCTs (54%) and in 36 patients (80%). In instances in which a decrease occurred, the average decrease in mean, minimum, and maximum \( \text{PbtO}_2 \) was 23.6%, 29%, and 18.1%, respectively (or absolute decreases of 10.9 ± 4.4 mm Hg, 16.5 ± 30.7 mm Hg, and 5.8 ± 9.5 mm Hg). If \( \text{PbtO}_2 \) was compromised (<25 mm Hg) before tHCT, the average decrease in mean, minimum, and maximum \( \text{PbtO}_2 \) after tHCT was 45.1%, 55.2%, and 19.4%, respectively, a clinically significant decrease. The relative reduction was even greater and also clinically significant when \( \text{PbtO}_2 \) was less than 15 mm Hg before the tHCT (Figure 1). During the 3 hours before the tHCT scan, \( \text{PbtO}_2 \) was normal (>25 mm Hg) in 50 instances. One episode of compromised \( \text{PbtO}_2 \) (<25 mm Hg) was observed before 38 tHCTs and 2 episodes before 12 tHCTs. Before all tHCTs, the average number of episodes of compromised \( \text{PbtO}_2 \) was 0.62. When ≥1 episodes of compromised \( \text{PbtO}_2 \) were observed, the mean duration was 74.34 ± 58.60 (standard error of the mean [SEM]) minutes. After 54 tHCT scans, \( \text{PbtO}_2 \) was normal during the first 3 hours. Episodes of compromised \( \text{PbtO}_2 \) were observed once after 40 tHCTs and twice after

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before, Mean ± SD</th>
<th>After, Mean ± SD</th>
<th>Change, Mean ± SE</th>
<th>( P ) Value (For Change)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ICP</td>
<td>12.87 ± 6.58</td>
<td>13.15 ± 8.53</td>
<td>0.28 ± 0.7</td>
<td>.7</td>
</tr>
<tr>
<td>Minimum ICP</td>
<td>9.31 ± 5.90</td>
<td>8.75 ± 6.07</td>
<td>−0.55 ± 0.5</td>
<td>.3</td>
</tr>
<tr>
<td>Maximum ICP</td>
<td>17.09 ± 9.29</td>
<td>18.08 ± 13.94</td>
<td>0.99 ± 1.2</td>
<td>.4</td>
</tr>
<tr>
<td>Mean CPP</td>
<td>83.93 ± 15.72</td>
<td>84.50 ± 14.54</td>
<td>0.69 ± 1.3</td>
<td>.6</td>
</tr>
<tr>
<td>Minimum CPP</td>
<td>71.68 ± 19.78</td>
<td>72.51 ± 18.57</td>
<td>0.99 ± 2.0</td>
<td>.6</td>
</tr>
<tr>
<td>Maximum CPP</td>
<td>95.06 ± 19.09</td>
<td>95.53 ± 15.87</td>
<td>0.53 ± 1.9</td>
<td>.8</td>
</tr>
<tr>
<td>Mean ( \text{Hgb} )</td>
<td>10.37 ± 1.51</td>
<td>10.23 ± 1.39</td>
<td>−0.14 ± 0.13</td>
<td>.3</td>
</tr>
<tr>
<td>Mean ( \text{PaO}_2/\text{FiO}_2 ) ratio</td>
<td>0.59 ± 0.20</td>
<td>0.59 ± 0.21</td>
<td>0.00 ± 0.02</td>
<td>.9</td>
</tr>
</tbody>
</table>

a SD, standard deviation; SE, standard error; ICP, intracranial pressure; CPP, cerebral perfusion pressure; \( \text{Hgb} \), hemoglobin; \( \text{FiO}_2 \), fraction of inspired oxygen.
6 tHCTs. The average number of episodes of compromised PbtO2 that occurred in the 3 hours after a tHCT scan was 0.52. When PbtO2 was compromised after a tHCT, the mean duration of each episode was $111.0 \pm 66.30$ (SEM) minutes. On average, an episode of compromised PbtO2 was $25.26 \pm 8.8$ (SEM) minutes longer after a tHCT scan than before ($P = .006$). An episode of brain hypoxia (<15 mm Hg) was identified during the 3 hours before a tHCT in 9 instances. During the 3 hours after tHCT, 1 and 2 episodes of brain hypoxia were observed after 16 and 3 tHCT scans, respectively. After tHCT, the mean duration of brain hypoxia was $102.39 \pm 67.49$ (SEM) minutes. On average, an episode of brain hypoxia was $46.6 \pm 16.0$ (SEM) minutes longer after tHCT than before tHCT ($P = .008$).

Why Is Brain Oxygen Reduced?

To examine why transport was associated with reduced PbtO2, we examined the following variables in multivariate analysis: age, sex, CPP, Hgb, and the PaO2/FiO2 ratio because each may influence cerebral blood flow or arterial oxygen content. This result is illustrated in Table 3. These data suggested that changes in lung function may be associated with the reduction in PbtO2 associated with transport (Figure 2).

DISCUSSION

Follow-up HCT scans are an important component in the care of severely brain-injured patients. We examined 45 patients with continuous PbtO2 monitoring during the 3 hours before and after 100 HCTs that required IHT. Our findings indicate that (1) ICP and CPP remain stable; (2) mean, minimum, and maximum PbtO2 are reduced; (3) brain hypoxia (PbtO2 <15 mm Hg) is more frequent after IHT; (4) the duration of compromised brain oxygen (PbtO2 <25 mm Hg) or brain hypoxia is significantly longer after IHT; and (5) changes in lung function may account for the reduced PbtO2 associated with IHT. These results, although preliminary, suggest that the need for IHT for an HCT should be carefully considered or alternatively that mobile HCTs in the ICU could play a larger role in the care of critically ill patients with brain injury.

Methodological Limitations

Our study has several potential limitations. First, the data were obtained from only 45 patients, and therefore the results should be considered preliminary. However, physiological data from the 3 hours before and after 100 tHCTs were examined and a robust variance estimator was used to adjust for any potential clustering effect caused by multiple CT scans in the same patient. Second, the data were examined retrospectively may bias our results, and because the study was conducted with patients treated at a single institution, it may lack

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before, Mean ± SD</th>
<th>After, Mean ± SD</th>
<th>Change, Mean ± SE</th>
<th>P Value (for change)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean PbtO2</td>
<td>37.93 ± 19.79</td>
<td>33.95 ± 17.21</td>
<td>−4.0 ± 1.0</td>
<td>.0001</td>
</tr>
<tr>
<td>Minimum PbtO2</td>
<td>30.10 ± 16.48</td>
<td>27.56 ± 15.73</td>
<td>−2.5 ± 0.92</td>
<td>.007</td>
</tr>
<tr>
<td>Maximum PbtO2</td>
<td>48.31 ± 32.89</td>
<td>41.92 ± 22.96</td>
<td>−6.4 ± 2.7</td>
<td>.02</td>
</tr>
</tbody>
</table>

*PbtO2, brain tissue partial pressure; SD, standard deviation; SE, standard error.

FIGURE 1. Histograms illustrating the change (%) in brain oxygen before and after transport head computed tomography (CT) scan. PbtO2, brain tissue oxygen partial pressure.
testing, it was not possible to monitor these parameters, so we considered IHT as a secondary cerebral insult. We also did not use a continuous end-tidal CO2 monitor; CO2 may have changed during transport, thereby influencing PbtO2. Our results tell us about how the transport affected the patient but not what happened during transport. Sixth, cardiac and pulmonary dysfunction is common after severe TBI or SAH, and this could influence how well IHT is tolerated. However, patients in this study were volume resuscitated and stabilized for at least 12 hours before observation. Seventh, we only examined transport to and from the HCT. Transport to and from other diagnostic or therapeutic procedures may have yielded other findings. However, it is the transport and its associated change in patient position and ventilation, rather than the destination, that are the potential insult. Our results also do not describe what happens if modern transport ventilators are used because we relied on Ambu bag hand ventilation. Finally, this was not a pure observational study in that interventions were directed at low PbtO2, low CPP, and high ICP; this may bias the results. Despite these limitations, our data at least confirm and extend previous studies and recommendations that an organized efficient process that is supported by appropriate personnel and equipment is required for IHT of the critically ill patient.

**Follow-up HCT**

HCT scans and follow-up HCTs have improved our ability to manage patients with severe brain injury. In addition, serial HCTs have contributed to our understanding of severe TBI and SAH pathophysiology and can be useful in outcome prediction outcomes.20–22 However, the timing and number of follow-up HCTs remain poorly defined. In addition, the impact of serial HCTs on patient outcome has not been evaluated in a randomized study, and there is little evidence that links improved clinical outcome to follow-up HCTs. Despite this, there is a general consensus that follow-up HCTs be obtained within 24 hours of admission, particularly when patients are first imaged within 3 to 6 hours of their injury or ictus, when intracerebral and epidural hematomas are managed without surgery, when there is coagulopathy, after a craniotomy or placement of a ventricular catheter, or when there is neurological deterioration.23–28 In addition, a repeat HCT should be considered when the initial GCS score is less than 8 because the imaging results in these patients may lead to intervention without neurological change.29

Our data do not allow us to comment on the clinical value of follow-up HCTs and specifically do not address the role of HCT in moderate or mild TBI or in patients with good-grade SAH. However, management changes were very infrequent unless there was deterioration in neurological condition or an increase in ICP. This is consistent with observations by Roberson et al20 who obtained serial HCTs in 107 comatose patients 1, 3, 5, 7, and 14 days and 3 months after TBI. They observed that management was not changed if findings on the initial HCT were normal and the patient had improved. Similarly, Smith et al27 in a more recent study that included 116 patients observed that no patient underwent a new

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**TABLE 3. Variables Examined in a Multivariable Analysis for an Association Between Intrahospital Transport and Brain Oxygen Change (N = 100, Transport to Head Computed Tomography)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>0.055</td>
<td>0.090-0.2400</td>
<td>.56</td>
</tr>
<tr>
<td>Age</td>
<td>0.0004</td>
<td>−0.0037-0.0045</td>
<td>.86</td>
</tr>
<tr>
<td>CPP (change in average)</td>
<td>0.0042</td>
<td>−0.0015-0.0099</td>
<td>.15</td>
</tr>
<tr>
<td>Hemoglobin (change)</td>
<td>−0.0058</td>
<td>−0.1150-0.0008</td>
<td>.05</td>
</tr>
<tr>
<td>FiO2/PaO2 (change)</td>
<td>0.447</td>
<td>0.0240-0.8710</td>
<td>.04</td>
</tr>
</tbody>
</table>

CI, confidence interval; CPP, cerebral perfusion pressure; FiO2, fraction of inspired oxygen.

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**FIGURE 2.** Regression plot illustrating the relationship between brain oxygen (y-axis, log corrected) and change in lung function (FiO2/PaO2 ratio) before and after transport head CT scan. (FiO2, fraction of inspired oxygen; PaO2, brain tissue oxygen partial pressure.)

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HRP external validity. However, the patients were all treated according to a standard protocol, the data were entered prospectively, and each patient met clearly delineated inclusion criteria. Third, the patients were examined at different time points after TBI and SAH. This can influence cerebral blood flow and PbtO2. Each patient, however, was his or her own internal control, and time was factored into our statistical model, which should reduce the potential influence of time after TBI or SAH. Fourth, the reason that patients underwent a tHCT scan may vary, and this may influence our results. The data associated with scans performed for a clinical reason rather than on a routine basis may account for the change in PbtO2. Three fourths of the scans were performed for routine reasons, so this and our statistical methods should limit potential bias. Fifth, we did not collect ICP and PbtO2 data during tHCT or during HCT. Although these data would have been very inter-
intervention when clinical findings were stable, even if the follow-up HCT findings were worse. Our findings that PbtO_2 is decreased and the duration of brain hypoxia increased after tHCT suggest that careful thought be given to the practice of routine follow-up tHCTs. The need for a follow-up HCT after severe TBI may be better guided by the severity of the initial brain imaging findings, serial clinical examinations, and neuromonitoring. Alternatively, portable HCTs may need to be more widely available and used.

IHT

Today, IHT of critically ill patients for diagnostic or therapeutic procedures is regarded as a continuum of intensive care. However, IHT carries an inherent risk, and complications or mishaps are common during transport, with estimates between 5% and 70% depending on how complications are defined. Our findings extend these observations and demonstrate that PbtO_2 is reduced after IHT, specifically for tHCT, and that these reductions often are greater when PbtO_2 already is compromised. When interpreting our data, it is difficult to answer whether IHT and tHCT per se increase the risk of a PbtO_2 decrease or whether the critical condition of the patient or the reason for the tHCT contributed to the PbtO_2 change. The respective roles of IHT and cause of IHT on PbtO_2 may only be resolved in a randomized study, a study that likely will be difficult to justify or conduct. Since analyzing the results described in this study, we have purchased a portable HCT. We have begun an analysis of patients who undergo portable HCT (CereTom; Neurologica, Danvers, Massachusetts) in our unit. In a preliminary review of 300 portable HCTs, deleterious effects on PbtO_2 are very rare, suggesting that IHT is the most likely contributing factor. Finally, an unanswered question is whether the reduced PbtO_2 associated with IHT contributed to worse outcome. This may be difficult to answer (ie, how did the information from the HCT vs a change in PbtO_2 influence outcome?).

Why did a reduction in PbtO_2 occur? There are many possibilities, but our results suggest that a compromise in pulmonary function may be associated with our findings, ie, a change in FiO_2/PaO_2 ratio was associated with reduced PbtO_2. The exact impact of this is uncertain because for all tHCTs, the FiO_2/PaO_2 ratio was similar before and after the scan. It is well-known that there may be wide variations in PaCO_2 associated with hand ventilation using an Ambu bag. Increases in PaCO_2, however, often increase PbtO_2 when ICP remains normal. A change in pulmonary function is consistent with previous studies that show a deterioration of gas exchange and respiratory mechanics may occur during IHT and that IHT may increase the risk of ventilator-associated pneumonia. Waydhas et al observed that a decrease in arterial oxygenation occurred in 84% of patients and a long-term deterioration of respiratory function was observed in 12% of cases after IHT. Similarly, Evans and Winslow in a study of 36 patients observed that 19 patients (53%) had clinically important changes in arterial oxygen saturation, heart rate, and/or systolic blood pressure associated with IHT. In part, this change in pulmonary function may be related to the ventilator techniques (eg, manual ventilation or small transport respirators) used during transport that adversely affect tidal volume. In addition, changes in patient position may alter pulmonary function. Our database does not include patient position during transport, although patients routinely were transported on their ICU bed in the same position as when in the ICU. They were only flat for the short duration of the HCT. We have found in a prospective, observational, repeated-measures study of 33 consecutive comatose patients who were exposed to 12 different body positions in random order while ICP, CPP, and PbtO_2 were monitored that the effects of position vary considerably among patients. Some studies suggest that the risk of IHT complications may be associated with the urgency of IHT, the number of intravenous solutions and infusion pumps, the time spent outside the ICU, disease severity, therapeutic intensity, and the need for mechanical ventilation, particularly with positive end-expiratory pressure. Our database was not designed to predict when PbtO_2 would decrease, however, our results suggest that the PbtO_2 decrease was greater when it already was compromised before tHCT. Consistent with this, Andrews et al observed that pretransfer secondary cerebral insults were predictive of further insults associated with IHT in head-injured patients.

CONCLUSION

Experimental and clinical evidence demonstrates that there is a significant association between poor patient outcome and the number, duration, and intensity of episodes of low PbtO_2 after severe brain injury. Therapy to improve PbtO_2 may improve TBI outcome or limit cerebral ischemic injury after stroke. Our results show that tHCT scan can compromise PbtO_2, perhaps through a change in lung function. Although we cannot comment on how IHT affects patient outcome after severe brain injury, we recommend that (1) careful consideration be given to when a follow-up HCT is needed; (2) when manual ventilation is used during IHT, it is accurately approximated to pretransfer ventilation; (3) appropriate equipment and personnel are available; and (4) a formalized plan for IHT be used. The results also suggest that portable HCT may play an important role in neurointensive care.

Disclosure

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REFERENCES


The authors demonstrated that intrahospital transportation from the intensive care unit to the CT scanner and back may produce a decrease in brain oxygenation as measured by a focal probe. The decreases in oxygenation did not occur in all patients but were more pronounced when brain oxygenation was already suboptimal before transport. Neurological outcomes of the patients were not studied, but the decrease in oxygenation certainly could not have been helpful. The exact reasons for decreases in brain oxygen in this setting provide fertile ground for further study; the authors’ analysis suggested a role for lung function. Conceivably, PbtO$_2$ was affected by methods of ventilation during transport or by changes in position for CT scanning. Although appropriate physiological monitoring was performed during transport in this study, these data were not reported to have been recorded for retrospective analysis and correlation with the brain oxygenation changes. Continuous recording of heart rate, arterial pressure, peripheral oxygen saturation, and expired CO$_2$ levels, along with correlation with specific events during transport, might shed light on the mechanisms involved in producing relative brain hypoxia. Whatever the mechanism, transporting a patient with a serious brain injury who requires ventilatory support offers significant risk of secondary insult to the brain from respiratory and/or hemodynamic fluctuations. The authors have demonstrated that risk. This should spur investigation into methods of reducing that risk. Understanding the exact reasons for the decrease in brain oxygenation would, of course, be helpful in attempting to avoid the problem. Provision of mobile CT scanners that can operate at the bedside in the intensive care unit can obviate the need for assuming the risks of transport. The patient would still require brief supine positioning for imaging, and further investigation of the effects of that positioning would be appropriate. The addition of mobile CT scanner to the intensive care unit may actually turn out to be cost-effective if that prevents deterioration of brain oxygenation and neurological status, thus potentially decreasing the duration and expense of ICU care. However, even if transport for CT is unnecessary, intrahospital transport of such patients is necessary for other destinations, such as the operating room, so research aimed at maximizing the safety of intrahospital transport would be worthwhile.