Intracranial pressure response after pharmacologic treatment of intracranial hypertension

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BACKGROUND: The accepted treatment of increased intracranial pressure (ICP) in patients experiencing severe traumatic brain injury is multimodal and algorithmic, obscuring individual effects of treatment. Using continuous vital signs monitoring, we sought to measure treatment effect and ascertain the accuracy of manual data recording.

METHODS: Patients older than 17 years, admitted and requiring ICP monitoring between 2008 and 2010 at a high-volume urban trauma center, were retrospectively evaluated. Timing and dose of ICP-directed therapy were recorded from paper and electronic medical records. ICP data were collected automatically at 6-second intervals and from manual charts. A statistical mixed model was applied to all data to account for multiple sampling.

RESULTS: A total of 117 patients met inclusion criteria; 450 treatments were administered when nursing records indicate an ICP greater than 20 mm Hg, while 968 treatments were given when ICP was greater than 20 mm Hg by automated data. Pharmacologic treatments identified include hypertonic saline (HTS), mannitol, barbiturates, and dose escalations of propofol or fentanyl infusions. Treatment with HTS resulted in the largest ICP decrease of the treatments examined, with a 1-hour ICP reduction of 8.8/9.9 mm Hg (for a small/large dose) according to manual data and a reduction of 3.0/2.4 mm Hg according to automated data. Propofol and fentanyl escalations resulted in smaller but significant ICP reductions. Mannitol (n = 8) resulted in statistically insignificant trends down in the first hour but rebounded by the second hour after administration. The average ICP in the hour before medication administration was higher for barbiturates (27 mm Hg) and mannitol (32 mm Hg) than for the other interventions (18–19 mm Hg).

CONCLUSION: ICP fell after administration of HTS, mannitol, or barbiturates and showed continued improvement after 2 hours. ICP fell initially after treatment with short-acting propofol and fentanyl but trended back up after 2 hours. Manually recorded data consistently overestimated treatment effectiveness. Automated data collection gives a more accurate assessment of patient status and responsiveness to treatment. (*J Trauma Acute Care Surg*. 2014;77: 47–53. Copyright © 2014 by Lippincott Williams & Wilkins)

LEVEL OF EVIDENCE: Therapeutic study, level IV.

KEY WORDS: Traumatic brain injury; secondary insults; intracranial pressure; critical care documentation; automated documentation.

Severe traumatic brain injury (TBI) is the leading cause of death after injury, despite advances in treatment and monitoring.[1] Physiologic vital signs, including intracranial pressure (ICP) and cerebral perfusion pressure (CPP), are monitored continuously in the critical care setting. Automated, high-frequency monitoring systems are capable of capturing and recording massive amounts of these valuable physiologic data. However, vital information is typically still collected manually at predetermined intervals that may not reflect a rapidly changing clinical situation.

Treatment of patients with severe TBI aims to prevent or mitigate secondary injury consisting of cerebral edema, inflammation, and ischemia. Treatment administrations represent periods of particular clinical interest when documentation is integral to effective evaluation. In most centers, treatment for intracranial hypertension is tiered, with "first-tier" interventions (hyperosmolar therapy, increased sedation, external ventricular drainage, short-term hyperventilation) and more aggressive "second-tier" interventions (hyperventilation to a $Paco_2$ of less than 30 mm Hg, high-dose barbiturates, surgical decompression).[2] Clinical practices to achieve these goals vary between centers, and there are limited data on the short-term effects of commonly used pharmacologic agents.

Hyperosmolar therapy with mannitol[3–6] or hypertonic saline (HTS)[7–11] is well recognized to aid in ICP control. While some studies show similar effects of equimolar doses of mannitol and HTS on ICP control,[12,13] others suggest that HTS offers potential superiority.[9,14–16] In our practice, these agents are commonly administered as boluses, with HTS less commonly administered as a continuous infusion.

A variety of sedatives and analgesics are used to ameliorate pain and agitation in patients with severe TBI. The Brain Trauma Foundation found only one study fulfilling inclusion criteria for their management guidelines, performed by Kelly et al.[17] This study not only compares end points (mortality, Glasgow Outcome Scale [GOS]) after the use of propofol or

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morphine sulfate but also measures clinical end points; the study found ICP to be lower by Day 3 in the propofol group. There have been no systematic studies of the short-term effects of propofol on ICP, although it is commonly used clinically to aid in ICP control. Similarly, the few existing randomized trials that examine the effects of opioids on ICP or CPP are conflicting and at best suggest minor improvements in ICP control.[18–22]

We hypothesized that the short-term effects of pharmacologic interventions for increased ICP could be measured with continuous, automated vital signs data and that this is a more accurate representation of the clinical picture than manually recorded measurements.

PATIENTS AND METHODS

Patients

Study subjects were admitted to the R Adams Cowley Shock Trauma Center, a high-volume academic urban trauma center, between 2008 and 2010. The institutional review board approved the collection of retrospective data on patients older than 17 years admitted to the neurotrauma critical care unit with severe TBI who required invasive ICP monitoring. Severe TBI was defined as postresuscitation Glasgow Coma Scale (GCS) score of less than 9 with TBI confirmed by computed tomography (CT).

Management Protocol

Patients with severe TBI admitted to the R Adams Cowley Shock Trauma Center are admitted to a dedicated neurotrauma critical care unit and managed according to a standardized tiered protocol in accordance with the Brain Trauma Foundation guidelines.[23] Treatment targets the maintenance of ICP less than 20 mm Hg and CPP greater than 60 mm Hg, as described previously and shown in Table 1.[24] All patients included in the study had placement of a clinically indicated intraparenchymal monitor (Camino, Integra NeuroSciences Plainsboro, NJ) or intraventricular catheter (Codman, Raynham, MA).

Data Collection

Patient demographics, mechanism of injury, routine vital signs, method of ICP monitoring, and need for surgical intervention with cranial decompression were recorded. Admission head CT was assigned a Marshall Classification score according to the presence of basal cistern compression, midline shift greater than 5 mm, and lesions greater than 25 cm^3 by a blinded reviewer.[25] Outcomes measured included in-hospital mortality, overall length of stay (LOS), and LOS in the intensive care unit.

All drug treatments for increased ICP that could be identified were recorded from paper and electronic charts. These included hyperosmolar therapy, analgesia, and sedation. Analgesia was overwhelmingly provided in this patient population with a continuous infusion of fentanyl with doses between 25 μg/h and 550 μg/h. Sedation agents included propofol, lorazepam, midazolam, and dexmedetomidine. The vast majority of patients received propofol in doses of 20 μg/kg per minute to 100 μg/kg per minute for sedation. Other agents, when used, were almost exclusively introduced after prolonged sedation with propofol. We chose to focus on fentanyl and propofol because the use of other agents was relatively infrequent in this population.

TABLE 1. Patient and Injury Characteristics

	n = 117
Age, mean ± SD, y	40.0 ± 17.7
Male, n (%)	93 (79.4)
Mechanism of injury, n (%)	
Motor vehicle/motorcycle crash	51 (44)
Fall	32 (27)
Pedestrian struck	13 (11)
Assault	16 (14)
Other	5 (4)
Blunt injury, n (%)	103 (88.0)
GCS score, postresuscitation, median (IQR)	6 (5–7.5)
Marshall CT score, median (IQR)	2 (2–3)
Injury Severity Score (ISS), median (IQR)	29 (25–38)
Polytrauma, n (%)*	46 (39.7)
In-hospital mortality, n (%)	22 (18.8)
LOS, median (IQR), d	14.0 (10.7–18.7)
Intensive care unit LOS, median (IQR), d	11.6 (8.5–16.7)
Craniotomy/craniectomy, n (%)	35 (35.7)

*Defined as nonhead ISS greater than 15.

All instances of treatment with HTS given as a bolus, mannitol, a discrete dose of a barbiturate, or an increased dose of continuously administered propofol (for sedation) or fentanyl (for analgesia) were recorded from paper and electronic records. To account for varying doses in HTS, the volume and concentration were multiplied, and doses were defined as "small" (≤750 or the equivalent of 250-mL 3% NaCl solution) or "large"(>750 or >50-L 3% NaCl solution or >100 mL of 7.5% NaCl). All but one dose of mannitol were 25 g; the one remaining dose was 50 mg. Barbiturates included were thiopental (125, 150, or 250 mg), methohexital (50, 70, 75, or 90 mg), and pentobarbital (50 mg or 100 mg). Treatments were correlated with recorded vital signs and included for analysis when the 5-minute mean ICP value was greater than 20 mm Hg or nursing records indicated an ICP greater than 20 mm Hg. This was done to exclude treatments given for reasons other than intracranial hypertension (ICH).

Continuous, automated real-time vital sign data were captured through a vital sign data recorder (VSDR) from bedside monitors (GE-Marquette-Solar-7000/8000) as previously described.[24] In short, the VSDR captures data (systolic and diastolic blood pressure, ICP, CPP, heart rate, etc.) every 6 seconds. Data are then transferred via a secure server and processed; 5-minute means are calculated. Artifacts are filtered by removing outliers, defined as ICP less than 0 mm Hg, ICP greater than 100 mm Hg, CPP less than 0 mm Hg, and CPP greater than 250 mm Hg. Vital signs were also transcribed from paper charts; ICP, CPP, and mean arterial pressure are noted each hour, except when ICP is greater than 20 mm Hg and when ICP is recorded every 15 minutes per protocol. Nursing staff see real-time, continuous data on the clinical monitors at the bedside.

TABLE 2. Instances of Treatment When Intracranial Pressure is Greater Than 20 mm Hg, as Defined by the Presence of a Manually Recorded Value Greater Than 20 mm Hg (Nursing) or at Least 5 Minutes of Consecutive ICP Greater Than 20 mm Hg Recorded by the Automatic Vital Signs Data Recording System (Automated)

Treatment	Nursing	Automated	Both
Small HTS	86	165	79
Large HTS	89	117	72
Mannitol	8	7	7
Propofol	139	343	115
Fentanyl	75	219	68
Propofol and fentanyl	33	93	30
Barbiturate	20	24	15

Statistical Analysis

Statistical analyses were performed in Excel (Microsoft; Redmond, WA), SAS (Cary, NC), and Matlab Student R2012, version 8.0 (Natick, MA). Demographic data were summarized as percentages or means with SD and medians with interquartile range (IQR). The Student's *t* test was used to compare mean ICP values. A statistical mixed model was applied to ICP values after treatment administration to account for multiple sampling. $p < 0.05$ for results was considered statistically significant.

RESULTS

A total of 117 patients met inclusion criteria. Patient and injury characteristics can be seen in Table 2. Briefly, patients were primarily male (79.4%), with a mean (SD) age of 40.0 (17.7) years and a median postresuscitation GCS score of 6. The most common mechanism of injury was motor vehicle or motorcycle crash, affecting 51 patients (44% of the sample), followed by 32 patients experiencing a fall. Overall in-hospital mortality was 18.8%.

A total of 450 treatments were administered in this patient population when the nursing record indicates an ICP greater than 20 mm Hg, as detailed in Table 2. A total of 968 treatments were administered during an hour in which automated, continuous data registered at least five consecutive minutes of ICP greater than 20 mm Hg. To ascertain whether comparisons could be made between these sets of treatment administrations, 386 instances of treatment were identified when both the manual record indicated an ICP greater than 20 mm Hg and the automated data included an ICP greater than 20 mm Hg for at least 5 minutes. ICP changes as calculated from manual or continuous data were compared within this matched set. Figure 1 shows a representative 12-hour period of ICP monitoring in one patient. Manually recorded and automated ICP are shown in thick and thin lines, respectively. Manual recordings are made every 15 minutes when ICP is greater than 20 mm Hg. A dose of HTS is indicated in the bottom bar by a circle (beginning of transfusion) and line (duration).

Table 3 shows the mean ICP in the hour before treatment with each of the analyzed interventions. Mean ICP before HTS, an increase in dose of propofol or fentanyl, or a combination of increase in dose of both propofol and fentanyl ranged from 18.51 mm Hg to 19.99 mm Hg according to automatic data and from 20.46 mm Hg to 22.63 mm Hg in the nursing records. Mannitol and barbiturates were given after average ICP values ranged from 26.13 mm Hg to 33.45 mm Hg in the hour before treatment. Dose increases varied between 5 μg/kg per minute and 75 μg/kg per minute, with relative increases of 5% to 650%. Fentanyl dosing ranged from 25 μg/h to 550 μg/h, with dose increases of 20 μg/h to 300 μg/h (16–650% relative increase). No correlation was seen between absolute or relative dose increase and ICP decrease after treatment.

Figure 2 shows ICP changes according to the nursing and automated records after treatment with hyperosmolar therapy. Figure 3 shows ICP changes after escalation of sedation or analgesia. All treatments resulted in significant ICP changes after 1 hour or 2 hours except for mannitol and barbiturate administration. Notably, there were only eight (in the automated data) or seven (in the nursing records) instances of mannitol administration in this patient population. There was no significant change in mean ICP after mannitol administration. Barbiturates were also used relatively sparingly in this patient population, with 20 instances recorded when manual records indicate an ICP greater than 20 mm Hg and 24 when continuous data show an ICP greater than 20 mm Hg for 5 minutes. No statistical difference was seen between different doses of mannitol (25 g or 50 g) or different drugs and doses of barbiturates.

ICP fell after administration of a "small" dose of HTS by 8.83 mm Hg in the first hour and 9.76 mm Hg in the second hour according to the manual data. Automated data indicated a decrease of 3.04 mm Hg and 5.48 mm Hg in the first and second hours, respectively. Large doses of HTS showed similar

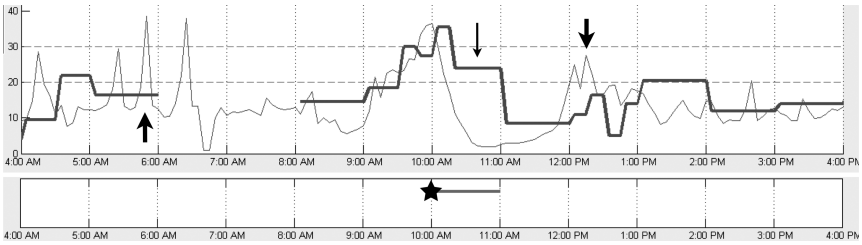

Figure 1. A visualization of manually recorded (*thick line*) and continuously monitored (*thin line*) ICP over 12 hours. ICP (mm Hg) is on the vertical axis. The bottom bar shows administration of HTS at 10:00 AM (*star*). The *thin arrow* indicates the time lag of manual data. The *thick arrows* show periods of intracranial hypertension not captured in the manual record that in this case likely contribute to overestimation of treatment effect in the manual documentation.

TABLE 3. Mean ICP in the Hour Before Treatment With HTS, Mannitol, Barbiturates, or an Increased Dose of Continuously Infused Propofol and/or Fentanyl

Treatment	Mean ICP (Manual Records), mm Hg	Mean ICP (Automated Data), mm Hg
Small HTS	20.91	19.35
Large HTS	21.50	19.99
Mannitol	26.22	33.45
Propofol	20.46	19.10
Fentanyl	21.43	18.98
Propofol and fentanyl	22.63	18.51
Barbiturate	26.14	26.61

trends. Propofol escalations resulted in an ICP decrease of 7.3 mm Hg (manual) or 1.58 mm Hg (automated) in the first hour. Similarly, fentanyl resulted in ICP decreases of 8.22 mm Hg (manual) and 3.77 mm Hg (automated) in the first hour of treatment. A simultaneous increase in fentanyl and propofol resulted in a change of 7.09 mm Hg (nursing) or 2.15 mm Hg (automated). While ICP continued to fall in the second hour after administration of HTS, mannitol, or barbiturates, it rose insignificantly in the same period after administration of propofol or fentanyl.

The ICP changes shown are based on overlapping but not identical sets of treatment instances. To validate the similarity of the sets of treatment instances, a nested set analysis was performed on the set of 386 treatments that were administered when both the nursing and automated records indicated that ICP was greater than 20 mm Hg. There were no significant differences in ICP changes after treatment between the full set and subset for any treatment type. ICP change after treatment with small or large doses of HTS or elevations in propofol and fentanyl were significantly different when calculated with manual or continuous data within this matched set. There is no significant difference between manual and continuous data when looking at matched instances of barbiturate (n = 15) and mannitol (n = 7) administration.

DISCUSSION

The availability of high-frequency automated, continuous ICP data allows for a detailed examination of ICP changes after treatment with common pharmacologic agents used to treat patients with severe TBI. Limited current data exist on the effects of these therapies on ICP, despite widespread clinical use.

While ICP is monitored continuously at the bedside, busy clinical staff are unable to observe all changes in real time. To this end, vital signs (ICP, CPP, and mean arterial pressure) are recorded hourly by trained nursing staff in the bedside record, with ICP recorded every 15 minutes when greater than 20 mm Hg as per our institutional protocol. If an intraparenchymal monitor is present, continuous monitoring is visible at the bedside, while an intraventricular catheter requires clamping before an accurate measurement can be observed. Whether this manual documentation is clinically adequate has been explored previously by several groups, including our own. An early study found the nurse "end-hour" ICP value to be a reasonable estimate for mean ICP over the past hour.[26] Venkatesh et al.[27] found a strong correlation between end-hour and 15-minute ICP values in 16 patients. All measurements in this study were manually recorded, however. Zanier et al.[28] showed that while computer-generated end-hour data accurately reflect manually recorded values, ICH tended to be underestimated in patients showing ICP instability. Most recently, Kahraman et al.[24] compared the area under the curve of ICP when ICP is greater than 20 mm Hg between automated and manual measurements, found poor agreement, and also found that the automated values were better predictive of eventual functional neurologic outcome.[24] The studies of Zanier et al. and Kahraman et al. suggest that automated monitoring captures elements of patient physiologic status that are lost in manual documentation.

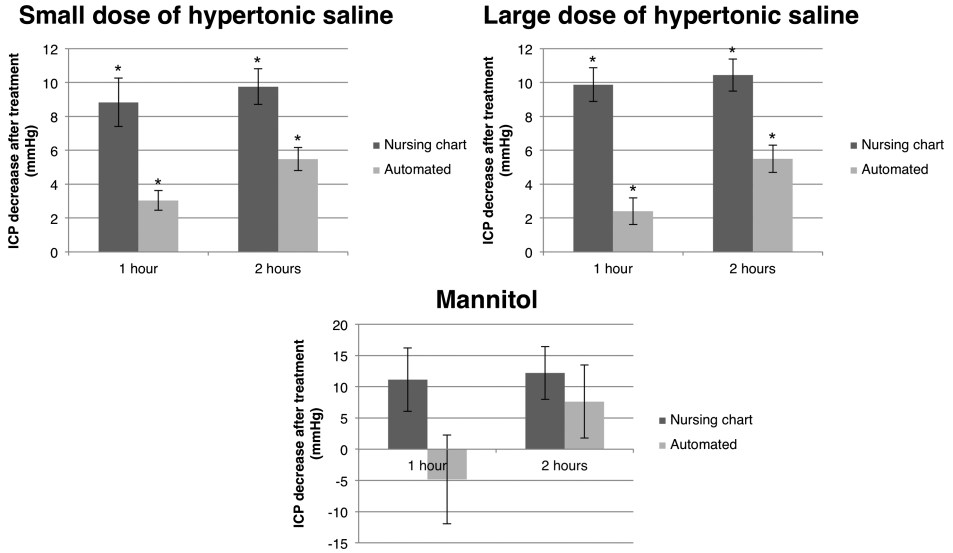

Figure 2. ICP decrease after treatment with hyperosmolar agents. HTS doses are designated as ''small'' (≤250-mL 3% NaCl solution) or ''large'' (>250-mL 3% NaCl solution). *Bars* indicate SE. *$p < 0.05$ when compared with baseline ICP value.

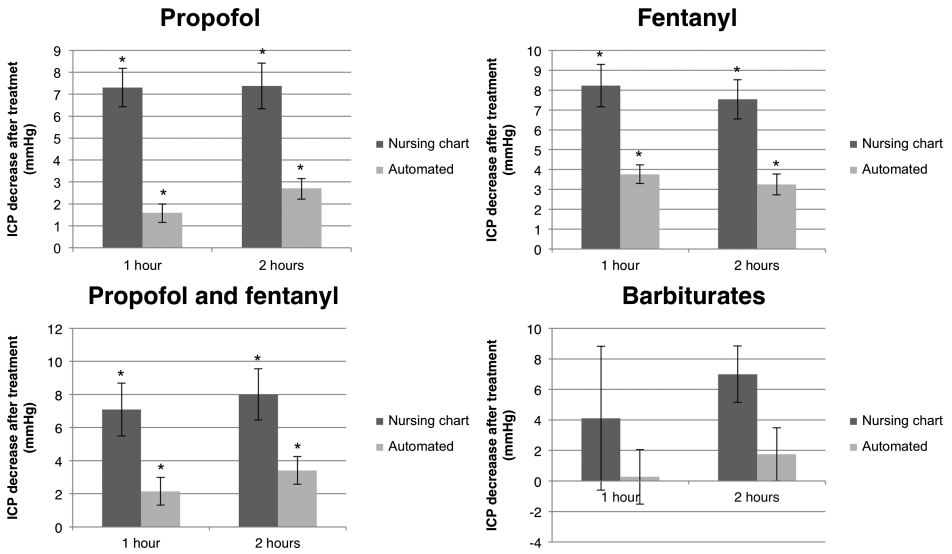

Figure 3. ICP decrease after additional treatment with sedative/analgesic agents. Barbiturates were administered as discrete doses, while propofol and fentanyl were given as dose escalations of continuous infusions. *Bars* indicate SE. *$p < 0.05$ when compared with baseline ICP value.

In this study, we sought to answer first, what ICP changes were seen after common treatments for ICH and, second, whether these changes were accurately characterized by the ICP values in the manual record. In most centers, while continuous ICP monitoring is displayed at the bedside, it is not technologically intuitive to accurately appreciate ICP changes over larger periods. Treatment administration marks a discrete period of time over which ICP changes are of particular interest, as treatment of severe TBI is directed at the maintenance of ICP, CPP, and other physiologic measurements within discrete parameters as outlined by evidence-based guidelines.[23] Judgment of the relative success of an ICP-directed therapy relies, of course, on the methods used to evaluate response.

We found more than twice as many treatment administrations when correlating treatments with continuous data than with those handwritten, and this proportion was markedly higher in instances of propofol escalations, which nursing staff can administer according to a sedation protocol without physician notification. The simplest explanation for this is that there are short periods of ICH that staff are reacting to without documentation—the presence of ICP-related treatment suggests that actions are being taken based on real-time clinical monitoring that does not get recorded manually.

The mean ICP before treatment is indicative of both patient status and previous treatment effectiveness. The average ICP before treatment with HTS or propofol or fentanyl dose escalations was quite similar when looking at either automated or nursing data (no statistic difference between baseline ICPs in each group). The mean ICP in the hour before these treatments were given was marginally less than 20 mm Hg when calculated from continuous data, suggesting ICP fluctuations around that treatment-triggering cutoff. Mannitol and barbiturates were given when patients had significantly higher mean ICP values—reflecting a clinical decision to use these agents for more severe ICH.

Hypertonic saline administration resulted in the largest significant decrease in ICP when compared with the other treatments, and mean ICP continued to decrease in the second hour after therapy. Mannitol, used in acute resuscitation but sparingly in this patient population, resulted in no statistically significant change in ICP, and when automated data were used for analysis, mean ICP trended higher after administration. The disparate mean baseline ICP values before administration of HTS or mannitol make it impossible to compare therapeutic effect directly.

Interestingly, ICP decreased significantly after an increased dose of continuously infused propofol or fentanyl was administered. Propofol is widely used as a sedative agent with rapid onset and short duration of onset. Several older studies found minor ICP decreases after prolonged propofol infusion.[29,30] In a randomized controlled trial, Kelly et al.[17] compared end points for patients sedated with either propofol or morphine sulfate and found improved ICP control and lower therapeutic intensity in the propofol group. The rapidly metabolized narcotic fentanyl is also commonly deployed but with relatively unclear effects on ICP. To our knowledge, this is the first report of the therapeutic effects of propofol or fentanyl using continuous, high-frequency data. Our data show that dose escalations of propofol or fentanyl result modest but significant decreases in ICP over the following 2 hours.

After almost all included treatments, ICP decreases measured with continuous data were approximately half of those calculated from nursing records. However, the discrepancy between manual and continuous data was largest for the treatments administered as elevations in dose of a continuous infusion. The estimated ICP decrease after propofol, for instance, was approximately 4.5 times larger when calculated with handwritten ICP values than when continuous automated data are used. While clinicians are necessarily acting on more evidence than the few numbers recorded by hand, this does suggest that we are

overestimating the effect of these common treatments, especially over periods longer than the minutes one could reasonably expect someone to remain at the bedside watching a monitor. A clinician may observe an initial favorable response in ICP that is obviated by a later, unobserved rebound in ICP. Our data suggest that the current commonly used methods of measuring treatment response do not accurately reflect clinical events.

In a 2007 report from the Maryland Nursing Workforce Commission, 81% of surveyed nurses indicated that patient care documentation directly affected the amount of time spent in direct patient care.[31] Of those, 54% said that they spent between a quarter and a half of their shift on documentation, and 29% reported spending more than half of their shift on documentation. During the period of this study, eight pages of documentation were produced by routine nursing activities alone per day. The data in the current study suggest that manual vital signs documentation in neurointensive care is inferior to automated data collection for some purposes. In a setting with heavy nursing workload, documentation must be effective and efficient, and when automated monitoring is already present, more intuitive data visualization could ameliorate this redundancy.

Our group has previously made advances in the bedside display of new and intuitive measures of physiologic status in the setting of neurotrauma critical care.[32] These monitors are designed to allow clinicians to quickly assess both current status and historical trends using graphic, colored displays. Groups in cardiovascular,[33–35] hematologic,[36] and emergency medicine[37] have developed "track and trigger" systems that ideally trigger medical responses to patient instability, but efforts have been hampered by concomitant increased nursing workload and calculation and trigger interpretation variability. One group reported on the implementation of an integrated monitoring system that continuously monitors multiple parameters indicative of patient cardiorespiratory stability and found their calculated instability index to correlate well with established instability criteria.[37] Efforts such as these demonstrate the utility of integrating continuous data capture into smarter integrated systems that can tease out trends before they are clinically overt.

Because of the retrospective nature of this study, we are unable to assume intent-to-treat ICH but can only look at ICP changes in patients with evidence of ICH before treatment. This is of course making assumptions about the correlation between treatment and ICH. It is entirely possible, especially with the administration of increased doses of propofol and fentanyl, which are not used solely for ICP control, that these were given for unrelated purposes. We also readily acknowledge that data recorded in the handwritten chart are not comprehensive and does not reflect clinicians' more nuanced understanding of their patients' current physiologic status.

CONCLUSION

In a population of patients with severe TBI, we have shown that ICP changes after routine pharmacologic intervention can be calculated from both handwritten charts and continuous, automated data and that the data from handwritten charts dramatically overestimate treatment effect. Of the therapies examined, administration of HTS resulted in the largest significant decrease in ICP. Improved real-time analysis of treatment effect is possible using continuous vital signs monitoring and could provide valuable and intuitive clinical information.

AUTHORSHIP

K.C. and S.Y. performed the chart review, data processing, and data analysis. P.F.H. performed the data filtration and analysis. H.H.C. performed the statistical analysis. K.C., B.B., and D.M.S. wrote the manuscript. D.M.S. and T.M.S. supervised the project and provided invaluable clinical support.

DISCLOSURE

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EDITORIAL CRITIQUE

The R Adams Cowley Shock Trauma Center has once again provided a unique physiologically focused study in a patient population with severe traumatic brain injury. The study design is a challenging retrospective partial cohort, where patients with intracranial pressure elevations >20 mm Hg are exposed to a variety of pharmacologic ICP control measures. The outcome measure is intracranial pressure change from baseline, using repeated pre-post measurements that are manually charted and automated. They demonstrate the providers are paying attention and intervening on the continuous data being displayed, despite the inherent limitations of manual documentation.

This complex study is limited by the additional potentially complex cross-sectional study design obscuring disease and exposure relationship, as well as the multiple and potentially overlapping ICP treatments that occur in a complex ICU environment. Despite this, the data was treated similarly and one of the important findings is that the response to therapy appears to be truly muted when using all available data, as compared to manual data capture. As the authors allude, providers may be preferentially recording more favorable responses to therapy, related to heuristics and cognitive biases when dealing with high-density ICU data.

The intertwined nature of competing changes in intracranial pressure, blood pressure, heart rate, respiration, volume status, and oxygenation makes it challenging for a clinical team to effectively integrate and react to multiple streams of continuous data display. Providing real-time analytics of continuous data has immense potential for patient-care. For example, the heart rate observation (HeRO) monitor provides a single score reflecting the risk of early sepsis in very low birth weight preterm infants within neonatal intensive care units by integrating real-time, hourly complex heart rate characteristics occurring over the previous 12 hours.

How do we handle the increasing complex dense data in our ICU and hospital environments? Can this data be effectively used for improvement in patient care, or will "big data" analytics overload and impair our ability to deliver quality and cost-effective care? Hopefully, studies like this one will continue to shed light on the benefits of this potentially predictive technology, and ultimately provide longitudinal patient-centered outcomes.

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