Automated Pupillometry and Detection of Clinical Transtentorial Brain Herniation: A Case Series

Alexander Papangelou, MD; Elizabeth K. Zink, MS, RN†; Wan-Tsu W. Chang, MD‡§; Anthony Frattalone, MD‖¶; Daniel Gergen, BA**; Allan Gottschalk, MD, PhD**††; Romergryko G. Geocadin, MD**†††‡*

ABSTRACT Introduction: Transtentorial herniation (TTH) is a life-threatening neurologic condition that typically results from expansion of supratentorial mass lesions. A change in bedside pupillary examination is central to the clinical diagnosis of TTH. Materials and Methods: To quantify the changes in the pupillary examination that precede and accompany TTH and its treatment, we evaluated 12 episodes of herniation in three patients with supratentorial mass lesions using automated pupillometry (NeurOptics, Inc., Irvine, CA). Herniation was defined clinically by the onset of fixed and dilated pupils in association with decreased levels of consciousness. Automated pupillometry was measured simultaneously with the bedside clinical examination, but the clinical team was blinded to these results and could not act on the data. Data from the pupillometer were downloaded 1–2 times per week onto a secured laptop, and data processing was facilitated by the use of Mathematica 8.0. Results: Neurologic Pupil Index measurements, values generated by the pupillometer based on an algorithm that incorporates pupillary size and reactivity in a normal population, were found to be abnormal before 73% of TTHs. This abnormality occurred at a median of 7.4 h before TTH. All episodes of TTH were reversed after clinical intervention at a median of 43 min after the event. The value did not fall to 0 in 42% of clinical herniations, but it did decrease to very abnormal values of 0.5–0.8. Conclusions: The potential of automated pupillometry to guide the management of severely injured neurologic patients is intriguing and warrants further study in the critical care unit and beyond. The utility of a portable device in the combat setting may allow for triage of patients with severe neurologic injury.

INTRODUCTION

Monitoring of pupillary size and reactivity is a cornerstone of the bedside neurologic examination in comatose patients with brain injury from multiple etiologies. During transtentorial herniation (TTH), the pupil size and reactivity are the most sensitive and easily identifiable findings.[1] The prognostic and clinical value of pupillary examinations has been established for traumatic and nontraumatic injuries[1–15] and has been featured in benchmark studies as far back as the early 1980s.[2,7] Invasive measures such as intracranial pressure (ICP) monitors and cerebral oximetry are useful supplements to the neurologic examination, but fail to reliably predict those at risk for TTH.[16–20]

Clinically, TTH is heralded by pupillary dilation and deterioration of consciousness through injury to the third cranial nerve and the ascending arousal system.[21] Traditionally, it is believed that the medial temporal lobe protrudes downward between the midbrain and edge of the tentorium, causing third nerve compression and ipsilateral pupillary dilation. Of note, although TTH has long been considered a compartmental phenomenon,[22–24] Miller Fisher and Ropper postulated that clinical symptoms are produced through passive torqueing of the mesencephalon by the expanding mass.[25] If TTH progresses to compress the contralateral cerebral peduncle against the tentorium cerebelli, termed Kernohan's notch syndrome, contralateral pupillary dilation and ipsilateral weakness would result leading to potential false localization. If this process goes undetected and untreated, progression to coma and death are possible.[26]

The pupillary examination traditionally includes assessment of pupil size, shape, symmetry, and reactivity to light. Current practice includes estimation of pupil size in millimeters or the use of other descriptive terms such as pinpoint, small, moderate, or large. Common subjective terms such as brisk, sluggish, or fixed are used to describe pupil reactivity. On the other hand, the pupillary response to light can be quantified by its

*Department of Anesthesiology, Emory University Hospital, 1364 Clifton Road NE, Atlanta GA 30322.

†The Johns Hopkins Hospital Department of Neuroscience Nursing, 600N Wolfe Street, Baltimore MD 21287.

‡Department of Neurology, University of Maryland Medical Systems, 22S Greene Street, G7K55, Baltimore MD 21201.

§Department of Emergency Medicine, University of Maryland Medical Systems, 22S Greene Street, G7K55, Baltimore MD 21201.

‖Department of Neurology, San Antonio Military Medical, Center, 3551 Roger Brooke Drive, San Antonio TX 78219.

¶Department of Trauma Critical Care, San Antonio Military Medical Center, 3551 Roger Brooke Drive, San Antonio TX 78219.

**Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, 600N Wolfe Street, Baltimore MD 21287.

††Department of Neurosurgery, Johns Hopkins University School of Medicine, 600N Wolfe Street, Baltimore MD 21287.

‡‡Department of Neurology, Johns Hopkins University School of Medicine, 600N Wolfe Street, Baltimore MD 21287.

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maximal diameter, latency, constriction velocity (CV), minimal diameter, and dilation velocity (DV).[27] Previous studies have shown that accuracy of both pupillary size and reactivity improves with automated pupillometry when compared with traditional assessments by nurses and physicians.[27–30] Inter-rater reliability also increases with automated measurement.[31–32] One prior study evaluated automated pupillometry in the context of a case series of three TTHs,[33] but presented only qualitative data. Interestingly, others have reported abnormalities in the Neurologic Pupil Index (NPi), a composite of quantitative pupillary parameters, before severe elevations of ICP.[34] In neurocritical care, there are few evidence-based therapies that improve outcome. Historically, herniation has been associated with poor outcomes, including brain death. Research indicates that reversal of TTH is possible with rapid and aggressive interventions.[35–37] Frequently, when treatment is initiated, extensive secondary brain injury has already occurred and surviving patients will suffer severe long-term disabilities. This concept underscores the need for subclinical detection of rampant mass effect, as waiting for full clinical herniation (decrease in consciousness and fully dilated pupils) may very well be regarded as poor surveillance rendering it difficult to meaningfully reverse TTH.[38]

A device such as a pupillometer has great promise in the combat setting given ease of use, portability, and accuracy, even with limited training and neurologic expertise. Patients who have herniated and need aggressive management can easily be identified. Promise beyond this basic utility will need to be clearly established.

In the context of a case series, this study aims to more fully characterize the quantitative pupillary response in patients with supratentorial herniation.

METHODS

This study utilized a dynamic infrared pupillometer (NPi-100; NeurOptics, Inc., Irvine, CA, USA). The pupillometer emits a flash of white light to stimulate pupillary response and then digitally captures the response as a video and displays numeric results on an liquid crystal display screen.[28] It

TABLE I. Patient Demographics and Admission Overview

Patient	Age (yr)	Sex	Race	Primary Diagnosis	Number of Herniations	ICU Days	LOS	Disposition
1	34	M	W	Cerebral abscesses	10	9	9	Death (care withdrawn)
2	23	F	W	AVM (ruptured)	1	33	71	Death (care withdrawn)
3	29	F	B	SDH/SAH/ICH	1	30	37	Rehabilitation facility

AVM, arteriovenous malformation; F, female; M, male; ICH, intracerebral hemorrhage; LOS, length of stay; SAH, subarachnoid hemorrhage; SDH, subdural hematoma.

TABLE II. The 12 Reported TTH Events and the Subsequent Clinically Driven Interventions. The Correlation Between Conventional Pupillary Examination and Pupillometer-Derived NPi is Displayed

Clinical Herniation No.	Left Clinical Examination	Right Clinical Examination	Left NPi	Right NPi	Interventions
Patient 1					
1	Large, round, fixed	Large, ovoid, fixed	0	0	Mannitol 75 g, HV, 1 L of 2% HS, 30 cc 23.4% HS × 2, propofol infusion increased
2	Large, round, fixed	Moderate, ovoid, fixed	0	0	30 cc 23.4% HS × 2, HV
3	Large, round, fixed	Large, round, fixed	0	0	30 cc 23.4% HS × 2, HV, propofol infusion increased
4	Large, irregular, fixed	Large, irregular, fixed	0	0	30 cc 23.4% HS × 2, HV
5	Large, round, fixed	Large, round, fixed	0	0	30 cc 23.4% HS, mannitol
6	Large, round, fixed	Large, round, fixed	0	0	Mannitol 75 g, 30 cc 23.4% HS, HV, propofol bolus ×2 (150 mg total)
7	Large, irregular, fixed	Small, round, fixed	0.5	3.6	30 cc 23.4% HS
8	Large, round, fixed	Small, round, brisk	0.7	4.2	30 cc 23.4% HS
9	Large, round, fixed	Moderate, ovoid, fixed	0.7	0.8	30 cc 23.4% HS
10	Large, fixed	Moderate, round, sluggish	0.8	3.9	30 cc 23.4% HS
Patient 2					
1	Large, round, fixed	Large, round, fixed	0.8	0.9	3% HS bolus, HV
Patient 3					
1	Large, round, fixed	Large, round, fixed	0	0	30 cc 23.4% HS, 250 cc 2% HS, 50 cc/h of 3% HS

HS, hypertonic saline; HV, hyperventilation.

TABLE III. Relationship Between Conventional Pupillary Examination and Abnormal NPi Before Clinical Herniation

Herniation # by patient	Time before herniation (h)	Left clinical exam	Right clinical exam	Left NPi™	Right NPi™
Patient 1					
1	**69.1***	Small, round, brisk	Small, round, brisk	1.2	4.8
	21.3	Large, round, sluggish	Small, round, sluggish	1.8	4.3
	17.8	Moderate, round, brisk	Large, round, sluggish	4.3	2.4
3	**5.1***	Small, round, sluggish	Small, round, sluggish	4.1	1.9
4	**30.9***	Large, ovoid, fixed	Moderate, round, sluggish	1.6	4.2
	11	Large, ovoid, fixed. Left larger than right	Small, round, sluggish	0.6	3.6
	9.9	Small, round, brisk	Small, round, sluggish	4.5	3.6
	5.9	Small, round, sluggish	Small, round, sluggish	4.9	3.8
	2.9	Small, round, sluggish	Moderate, round, sluggish	4.6	3.7
	2.1	Moderate, round, sluggish	Large, round, sluggish. Right larger than left	3.2	1.4
	1.2	Moderate, round, brisk	Moderate, round, sluggish. Right larger than left	4.1	2.3
	0.6	Moderate, round, brisk	Moderate, round, sluggish. Right larger than left	3.8	2.8
	0.4	Moderate, round, brisk	Moderate, round, sluggish. Right larger than left	3.9	2.7
5	**9.8***	Moderate, round, brisk	Moderate, round, sluggish	4	2.8
	8	Moderate, ovoid, sluggish. Left larger than right	Moderate, round, sluggish, minimally reactive	3.6	2.6
	7.4	Small, round, brisk	Small, round, sluggish. Right larger than left	4.5	3.8
	6.8	Moderate, round, brisk	Moderate, round, sluggish. Right larger than left	4.4	3.1
	6.4	Moderate, round, brisk	Moderate, round, sluggish. Right larger than left	4.2	2.7
	2.4	Small, round, brisk	Small, round, brisk. Right larger than left	4.5	3.5
	1.9	Small, round, brisk	Small, round, brisk. Right larger than left	4.3	2.3
	0.8	Small, ovoid, brisk	Small, round, brisk	4.6	3.7
6	**1.1***	Large, ovoid, minimally reactive, brisk	Small, round, brisk	3.2	2.4[a]
	1	Large, round, minimally reactive. Left larger than right	Moderate, round, minimally reactive	0.6	0[a]
7	**3.9***	Small, round, brisk	Small, round, sluggish	4.3	3.5
9	**1.2***	Small, round, brisk	Moderate, round, brisk	4.6	3.4
Patient 2[b]					
1	**84.6***	Small, round, fixed	Small, round, fixed. Right larger than left.	0	3.6
	81.5	Small, round, nearly fixed, reaction questionable.	Small, round, nearly fixed, reaction questionable. Right larger than left.	0	4.3
	80.5	Small, round, fixed	Small, round, fixed	0	4.5
	76.8	Small, round, nearly fixed, reaction questionable.	Small, round, nearly fixed, reaction questionable.	0	4.4
	75.7	Small, round, fixed. Left greater than right.	Small, round, fixed	3.7	4.6
	73.7	Small, round, fixed	Small, round, sluggish	3.7	4.5
	58.8	Moderate, round, sluggish	Moderate, round, sluggish	2.4	4.5
	54.9	Small, round, sluggish	Small, round, sluggish	0	3.8
	51.5	Small, round, sluggish	Small, round, sluggish	3.3	4.5
	49.9	Small, round, sluggish	Small, round, sluggish	3.6	2.4
	47.7	Moderate, round, sluggish	Moderate, round, sluggish	1.1	3.1
	45.6	Moderate, round, sluggish	Moderate, round, sluggish	3.9	2.6

(continued)

TABLE III. Continued

Herniation # by patient	Time before herniation (h)	Left clinical exam	Right clinical exam	Left NPi™	Right NPi™
Patient 3					
1	**54.8***	Moderate, round, sluggish	Moderate, round, sluggish	2	3.5
	42.3	Moderate, round, sluggish	Moderate, round, sluggish	1.2	4
	38	Moderate, round, sluggish	Moderate, round, sluggish	3.6	4.6
	14.4	Moderate, round, brisk	Moderate, round, brisk	3.4	4.2
	8.2	Moderate, round, brisk	Moderate, round, brisk	3.5	4.3
	5.4	Moderate, round, brisk	Moderate, round, brisk. Right larger than left	2.9	2.7
	3.6	Moderate, round, brisk	Moderate, round, brisk Right larger than left	2.5	3.5

*First abnormal NPi measurement before indicated herniation. [a]Denoted measurements and correlating examinations were taken within 4 min of each other, immediately before clinical herniation no. 6. [b]Abnormal NPi values documented before the institution of pharmacologic coma. Coma induced for intractable intracranial hypertension. These values were not included in the statistical analysis because of the confounder of severe intracranial hypertension.

determines the following parameters separately: pupil size at rest and at the peak of constriction, percentage change in pupil size, time between delivery of light stimulus and onset of pupillary constriction (latency), average CV, maximum CV, and DV.[27, 34] The device utilizes a proprietary algorithm, incorporating all of the above parameters, to determine the NPi, which has a range of 0–5. Normative population modeling established a normal range of 3–5 for NPi. Values below 3 are considered abnormal and those closer to 0 even more so. The NPi can be abnormal in one or both pupils, and it is also considered abnormal when the NPi difference between pupils is greater than or equal to 0.7.[34] This prospective observational case study was approved by the Johns Hopkins Institutional Review Board and undertaken in a large academic medical center with a 24-bed neuroscience critical care unit (NCCU). Critically ill patients admitted to the NCCU with a supratentorial lesion and clinician-determined neurologic examination frequency of every 1–4 h were monitored with the pupillometer. Measurements were obtained at the same time as nurse's routine neurologic examination. The entire care team was blinded to the result of the pupillometer and as such could not act on these data. Those with a preexisting pupillary abnormality that prevented changes in pupil size (e.g., nonreactive pupils and iridectomy); bilateral facial, ocular, or periorbital injuries or edema (conditions that limit pupillary assessment); or a "comfort measures only" order were not monitored. A TTH was determined by the primary neuro intensive care unit (ICU) team based on the following standard clinical criteria: (1) perceived fixed and dilated pupil (s) and (2) decreased level of consciousness.[2] After herniation was confirmed, we continued to perform pupillometer measurements concurrent with bedside pupillary examinations until the patient returned to baseline or established a new neurologic baseline. Each TTH was considered an independent event as long as the patient exhibited at least partial pupillary examination recovery before a subsequent TTH. Potential Kernohan's notch and central herniation syndromes were treated in the same manner, and data were marked for analysis. Data from the pupillometer were downloaded 1–2 times per week onto a secured laptop and translated via software provided by Neuroptics. Data extracted from the patient's chart included demographics, diagnoses, cerebral perfusion pressure (CPP), ICP, Glasgow Coma Scale (GCS, range 3–15), FOUR score (FS, range 0–16), neurologic examination, and interventions around the time of herniation. The FS is an alternate coma scale that incorporates best response of eye opening, motor function, brain stem reflex, and respiratory pattern and has comparable predictability on outcome to the GCS.[39]

The primary research team performed the data analyses. Neuroptics did not have any direct input into the design, data collection, processing analysis, or interpretation. Data processing was facilitated by the use of Mathematica 8.0 (Wolfram Research, Inc., Champaign, IL, USA).

RESULTS

Data were collected as described and evaluated *post hoc* from three patients (Table I) who collectively experienced 12 herniation events described in Table II. Abnormal NPi values were collected before clinical herniation and the associated quantitative and clinical pupillary examination results are presented in Table III. The quantitative pupillary findings, level of consciousness, and measures of ICP and cerebral perfusion are illustrated for each patient in Figures 1–3.

Patient 1 was a 34-yr-old man who suffered from flu-like symptoms and headaches 1 wk before presentation. He was transported to an outside hospital with spontaneous eye opening, global aphasia, and right hemiplegia and was subsequently transferred to the NCCU at Johns Hopkins Hospital. He underwent an emergent craniotomy for drainage of a lesion that revealed angioinvasive fungus on initial pathologic examination. He experienced 10 herniation events, all treated successfully as described in Table II. The abnormal pupillary examinations observed before each herniation are summarized in Table III. However, his neurologic examination continued to decline and comfort care only was implemented on the ninth hospital day. His quantitative pupillary findings and related neurologic and physiologic data are detailed in Figure 1.

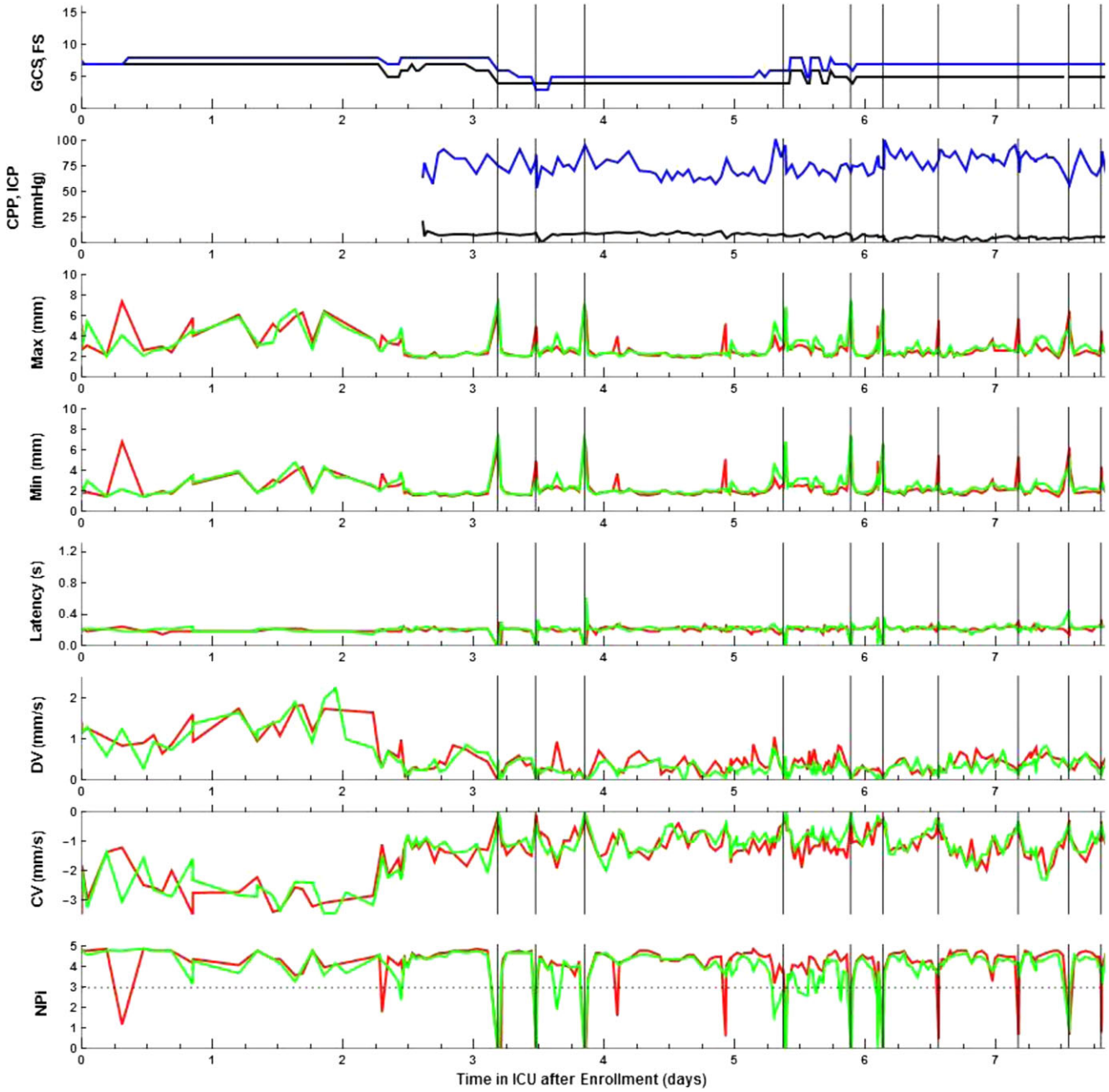

FIGURE 1. Quantitative pupillary findings, level of consciousness, and measures of cerebral perfusion and ICP for patient 1. Vertical black lines indicate the onset of clinical herniation, which are described in Table II. Level of consciousness is reflected by the GCS (3–15; black) and FS (0–16; blue). GCS and FS declined before the first clinical herniation. CPP is illustrated by the blue line and ICP by the black line. The ICP monitor was placed during ICU day 3. ICP did not rise above 20 mm Hg, and CPP (blue) was maintained above 60 mm Hg at all times. Quantitative pupillometry is shown for both eyes. The left eye is represented with red lines and the right eye by green tracings. Maximal pupillary dilation is noted at times of clinical herniation. The latency fell to 0 with each clinical herniation, indicating the lack of any pupil reaction. DV and CV fell to 0 with each clinical herniation. A mirrored slowing of DV and CV and slight reduction in minimum (Min) and maximal (Max) pupillary size occurred approximately 24 h before the first clinical herniation only. NPi fell to 0 (or close to 0) with each clinical herniation.

Patient 2 was a 23-yr-old woman who was transported to an outside emergency department after collapsing with temporary loss of consciousness. A subsequent computerized tomography (CT) scan of the head revealed diffuse subarachnoid hemorrhage (Hunt and Hess grade V and modified Fisher grade IV) with blood casting the fourth ventricle and diffuse cerebral edema. An extraventricular drain was placed and revealed an ICP in the range of 20–25 mm Hg. The patient was rapidly transported to the NCCU at The Johns Hopkins Hospital, where she presented with fixed and dilated pupils bilaterally. Cerebral resuscitation was undertaken with osmotherapy and hyperventilation that resulted in return of pupillary reactivity

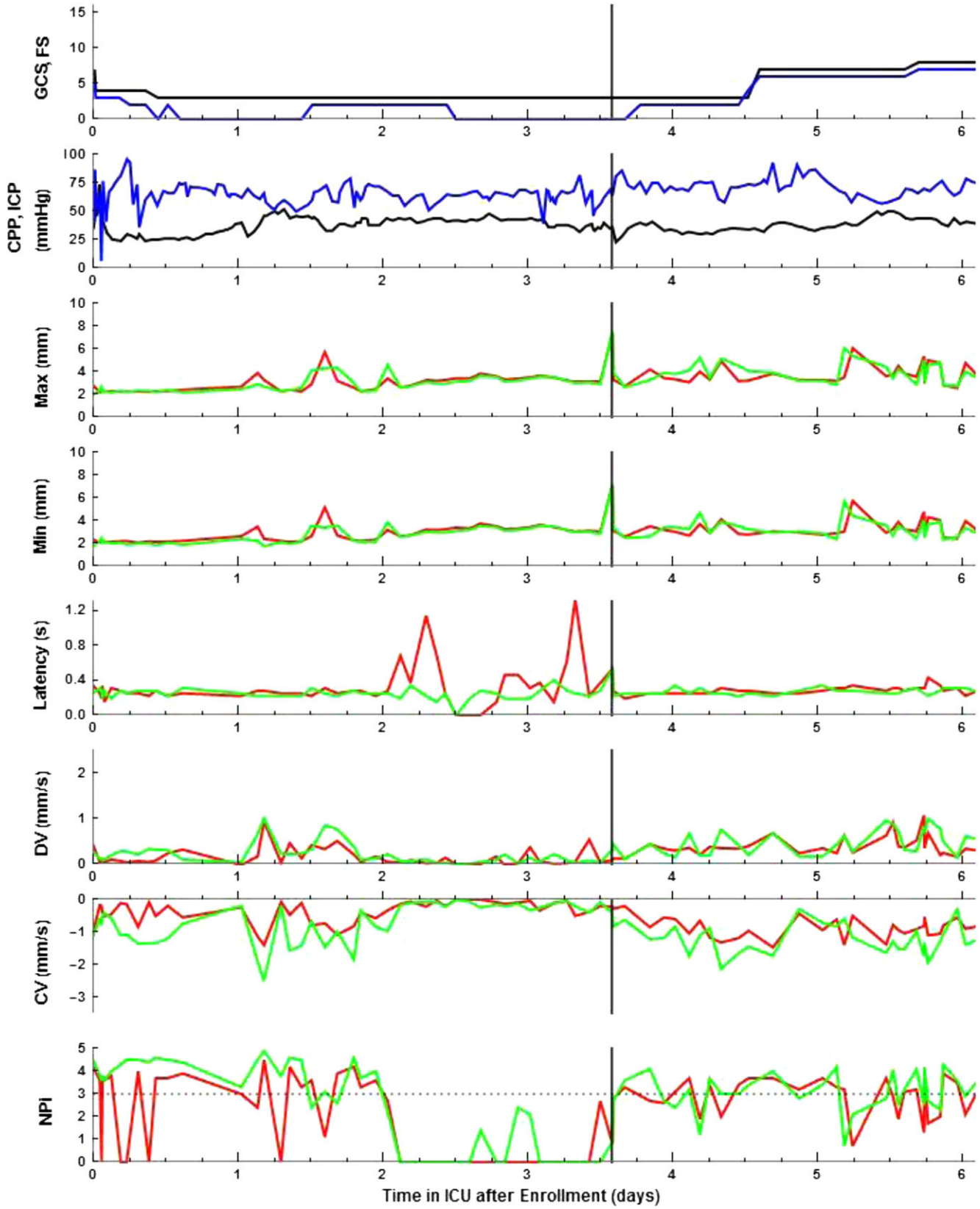

FIGURE 2. Quantitative pupillary findings, level of consciousness, and measures of cerebral perfusion and ICP for patient 2. See legend to Figure 1 for details. The GCS (3–15; black) at the time of TTH was 3T. The patient had intractable elevated ICP (black), with values generally ranging between 25 and 50 mm Hg. CPP (blue) was generally maintained greater than 60 mm Hg but at times fell to 50 mm Hg. The pupil acutely dilated at the time of TTH. The

bilaterally. A post-resuscitation CT scan revealed a new right frontoparietal intraparenchymal hematoma with a calculated volume of 80 cc causing 11 mm of right-to-left midline shift at the level of the foramen of Monro and subtle tonsillar herniation. The patient was taken to the operating room emergently for clot evacuation and hemicraniectomy. An intraoperative angiogram revealed an arteriovenous malformation underlying the right frontotemporal hematoma. Pupillometry was initiated postoperatively with the corresponding data presented in Figure 2 starting from this time point. The immediate postoperative course was complicated by cardiac arrest (pulseless ventricular tachycardia), which was successfully managed; intractable intracranial hypertension necessitating pentobarbital infusion titrated to EEG burst suppression; bacteremia caused by newly discovered endocarditis; herniation 10 h after discontinuation of pentobarbital (Fig. 2, day 3.5, Table II); and angioplasty with administration of nicardipine for treatment of vasospasm in the left internal carotid and middle cerebral arteries. The patient did not regain consciousness during her hospital course and died approximately 2 mo later when life support was withdrawn after a catastrophic bowel perforation.

Patient 3 was a 29-yr-old woman with a medical history of hypertension and end-stage renal disease requiring hemodialysis. She presented to a local emergency department with the worst headache of her life following a scheduled hemodialysis session. CT revealed right temporal subdural hematoma/subarachnoid hemorrhage, and the patient was transported to the Johns Hopkins NCCU. A conventional angiogram was negative, but on hospital day 3, she experienced acute right greater than left pupillary dilation and sluggish reactivity (Fig. 3, day 3.5, Table III; patient 3, line 1) with maintenance of arousal and orientation accompanied by an increase in headache intensity. An emergent head CT revealed a new right parietal intraparenchymal hemorrhage. The remainder of the hospital course was complicated by mild disseminated intravascular coagulation, a generalized tonic clonic seizure with subsequent cardiac arrest (pulseless electrical activity) that was successfully resuscitated, TTH (Fig. 3, day 5.3), persistent thrombocytopenia of unclear etiology, and the development of right paramedian parietal and occipital lobe infarcts. She was eventually discharged to an acute in-patient rehabilitation facility.

DISCUSSION

We describe the quantitative pupillary findings and clinical course of three patients who experienced a total of 12 herniation events, 10 of which occurred in the context of normal ICP (≤20 mm Hg, ICP not measured during one event). For all events, the pupils of one or both eyes became subjectively enlarged and fixed, although pupillometry frequently discerned subtle reactivity to light. In conscious patients, pupillary findings of herniation were always associated with a reduction in consciousness. There were several herniations that occurred in NCCU patients who were determined to be "floor status" by the medical team, underscoring the often unpredictability of neurologic decompensation in those with severe brain injury.

In these patients, an abnormal NPi measurement was observed at some prior point in 73% of the herniation events (patient 2 excluded because of the confounder of sustained severely elevated ICP), with the first abnormality occurring at a median of 7.4 h before herniation (Table III, with corresponding clinical pupillary examination). With treatment, in 100% of cases, the NPi returned to a normal value at a median of 43 min (range 5–128 min) after clinical herniation.

Although NPi did not decline to 0 in 42% of clinical herniations, it did decrease to very abnormal values of 0.5–0.8 in this subset of events. We speculate that these situations represented pupillary states that were observed minutes before a completely fixed and dilated pupil would have occurred without intervention. Interestingly, the clinical team perceived the pupil as fixed and dilated in all of these cases, highlighting the fact that quantitative pupillometry can distinguish between clinically indistinguishable states.

Of note, as well demonstrated in patient 1, individual pupillary metrics can be abnormal before herniation. However, because of the interdependence of variables, the value of these findings is limited. For instance, a change in the speed of pupillary constriction cannot be interpreted in isolation, but rather must be considered in the context of the response amplitude.[40–42] The interdependence of the components of the pupillary response is accounted for in the calculation of the NPi. The mechanism(s) underlying changes in the pupillary examination before significant intracranial hypertension or supratentorial herniation remains unclear. Of particular interest in this study were the multiple episodes of bilateral fixed and dilated pupils in one patient (Fig. 1). It is extremely unlikely that these episodes could represent TTH with a simultaneous Kernohan's notch phenomenon or central herniation, as they were quickly and rather easily reversed. This clinical course seems to favor the mechanism of passive torqueing postulated by Miller Fisher and Ropper.[25]

Importantly, it has been established that TTH frequently occurs in the absence of intracranial hypertension,[43–45] although they can certainly coexist. We have illustrated one patient (Fig. 2) who was treated with pentobarbital for sustained elevations in ICP and developed clinical TTH upon emergence from drug-induced coma. The pupillometer may be able to

change in pupillary diameter with the continued lack of reactivity drove the clinical team to suspect TTH as this patient emerged from a drug-induced coma. The latency was reduced but was not 0 at the time of suspected TTH. As with latency, the DV and CV were reduced but not 0 with suspected TTH. Thirty-six hours before TTH, there was a reduction in CV and DV that corresponded to the induction of a pentobarbital coma. NPi values were 0.8 and 0.9 at the time of clinical TTH. Interestingly, these values were actually increased from the values seen in the preceding 36 h. Max: maximal pupillary diameter; Min: minimum pupillary diameter.

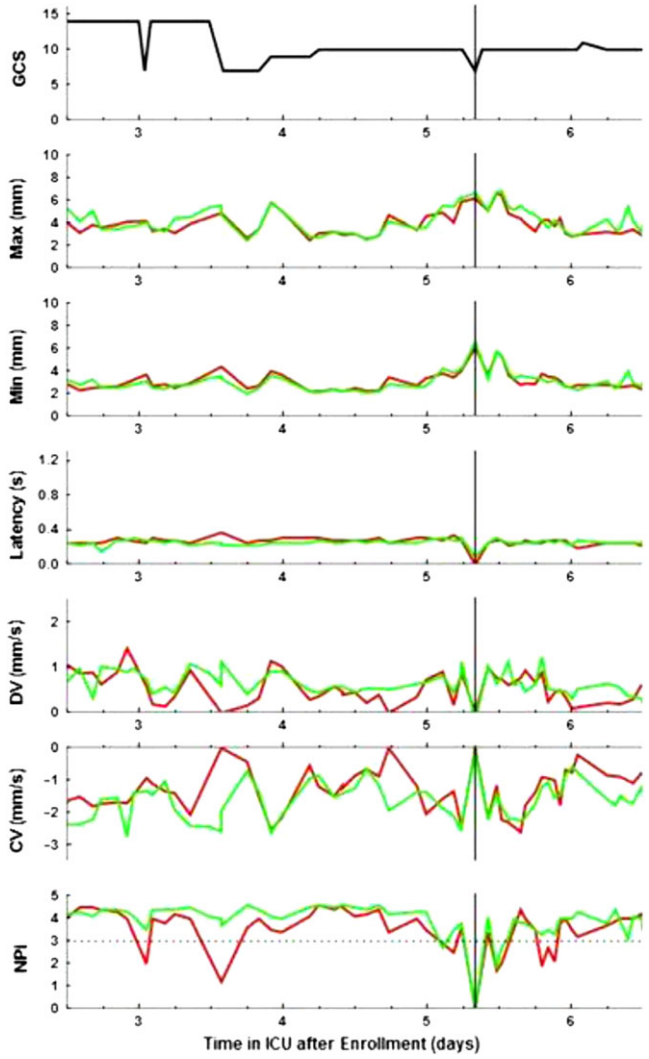

FIGURE 3. Quantitative pupillary findings, level of consciousness, and measures of cerebral perfusion and ICP for patient 3. See legend to Figure 1 for details. No ICP monitor was placed in this patient. The GCS (3–15; black) at the time of TTH fell by 3 points. FS was not calculated. The pupil acutely dilated at the time of TTH. The latency fell to 0 at the time of TTH. As with latency, the DV and CV both fell to 0 with TTH. The NPi was 0 at the time of TTH. Max: maximal pupillary diameter; Min: minimum pupillary diameter.

add insight into these complex situations where multiple processes coexist. A future study could address whether an abnormality in the quantitative pupillary examination is predictive of impending neurologic emergencies, either supratentorial herniation or sustained intracranial hypertension. The value of establishing this association, particularly for the military, could be revolutionary, given the lack of diagnostic equipment and trained personnel in the field.

The limitations of our study include a small sample size in a case series format, greatly limiting any conclusions. Ten of the observed 12 herniation events occurred in one patient. The frequency of neurologic monitoring was determined by the managing team, based on perceived disease severity (Johns Hopkins Hospital NCCU standard practice). This caused some variability in the automated pupillometry measurement frequency across patients, which may have led to an underestimation of the incidence of pre-herniation pupillary abnormalities. A larger prospective study would ideally standardize sampling frequency and would allow determination of the effectiveness of automated pupillometry to identify impending herniation in both the intensive care setting as well as in theater.

CONCLUSION

Changes in automated pupillometry preceded clinical signs of TTH in some instances. Future studies are needed to establish it as a valuable tool for this purpose and whether or not detection of pupillary changes by this method will in fact lead to earlier more aggressive management and improved neurologic outcomes. Portability and ease of use are two factors that make the pupillometer well suited for military health care providers who are often forced to provide care for patients with severe traumatic brain injury in austere environments. In addition to ICU care, utility as a triage device in the combat setting could have a significant impact.

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