American Journal of Emergency Medicine 34 (2016) 1102–1107

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Brief Report

Cerebral oximetry with blood volume index and capnography in intubated and hyperventilated patients☆,☆☆,★,★★

Taylor A. Bagwell, MD [a,*], Thomas J. Abramo, MD [a,1], Gregory W. Albert, MD [b], Jonathan W. Orsborn, MD [a], Elizabeth A. Storm, MD [a], Nicolas W. Hobart-Porter, DO [a], Tonya M. Thompson, MD [a], Eylem Ocal, MD [b], Zhuopei Hu [c], Todd Nick, PhD [c,†]

[a] Department of Pediatrics, Division of Pediatric Emergency Medicine, University of Arkansas for Medical Sciences, Little Rock, AR
[b] Department of Pediatrics, Division of Neurosurgery, University of Arkansas for Medical Sciences, Little Rock, AR
[c] Department of Pediatrics, Biostatistics Section, University of Arkansas for Medical Sciences, Little Rock, AR

ARTICLE INFO

Article history:
Received 9 January 2016
Received in revised form 29 February 2016
Accepted 1 March 2016

ABSTRACT

Objective: Hyperventilation-induced hypocapnia leads to cerebral vasoconstriction and hypoperfusion. Intubated patients are often inadvertently hyperventilated during resuscitations, causing theoretical risk for ischemic brain injury. Current emergency department monitoring systems do not detect these changes. The purpose of this study was to determine if cerebral oximetry (r_cSo_2) with blood volume index (CBVI) would detect hypocapnia-induced cerebral tissue hypoxia and hypoperfusion.

Methods: Patients requiring mechanical ventilation underwent end-tidal CO_2 ($ETco_2$), r_cSo_2, and CBVI monitoring. Baseline data was analyzed and then the effect of varying $ETco_2$ on r_cSo_2 and CBVI readings was analyzed. Median r_cSo_2 and CBVI values were compared when above and below the $ETco_2$ 30 mmHg threshold. Subgroup analysis and descriptive statistics were also calculated.

Results: Thirty-two patients with neurologic emergencies and potential increased intracranial pressure were included. Age ranged from 6 days to 15 years (mean age, 3.1 years; SD, 3.9 years; median age, 1.5 years: 0.46-4.94 years). Diagnoses included bacterial meningitis, viral meningitis, and seizures. $ETco_2$ crossed 30 mm Hg 80 times. Median left and right r_cSO_2 when $ETCO_2$ was below 30 mmhg was 40.98 (35.3, 45.04) and 39.84 (34.64, 41) respectively. Median left and right CBVI when $ETCO_2$ was below 30 mmhg was -24.86 (-29.92, -19.71) and -22.74 (-27.23, - 13.55) respectively. Median left and right CBVI when $ETCO_2$ was below 30 mmHg was −24.86 (−29.92, −19.71) and −22.74 (−27.23, −13.55) respectively. Median left and right r_cSO_2 when $ETCO_2$ was above 30 mmHg was 63.53 (61.41, 66.92) and 63.95 (60.23, 67.58) respectively. Median left and right CBVI when $ETCO_2$ was above 30 mmHg was 12.26 (0.97, 20.16) and 8.11 (−0.2, 21.09) respectively. Median duration $ETco_2$ was below 30 mmHg was 17.9 minutes (11.4, 26.59). Each time $ETco_2$ fell below the threshold, there was a significant decrease in r_cSo_2 and CBVI consistent with decreased cerebral blood flow. While left and right r_cSO_2 and CBVI decreased quickly once $ETCO_2$ was below 30 mmHg, increase once $ETCO_2$ was above 30 mmHg was much slower.

Conclusion: This preliminary study has demonstrated the ability of r_cSo_2 with CBVI to noninvasively detect the real-time effects of excessive hyperventilation producing $ETco_2 < 30$ mmHg on cerebral physiology in an emergency department. We have demonstrated in patients with suspected increased intracranial pressure that $ETco_2 < 30$ mmHg causes a significant decrease in cerebral blood flow and regional tissue oxygenation.

1. Introduction

There is ample evidence to support the physiologic premise that hyperventilation leads to decreased arterial blood carbon dioxide partial pressure ($Paco_2$) with subsequent decreased cerebral blood flow [1–3]. Hypocapnia-induced vasoconstriction and associated decreased cerebral blood flow lead quickly to subtle clinical signs and symptoms of compromised cerebral perfusion. If severe or prolonged, this hypoperfusion may increase a patient's risk for ischemic brain injury [2–4]. Variations in $Paco_2$ and corresponding capnography or end-tidal CO_2

☆ Work previously presented at the 22nd Pediatric Critical Care Colloquium; poster session; September 11-13, 2015; Little Rock, AR.
☆☆ Grants/funding: none obtained for this study.
★ Conflicts of interest: The authors declare that they have no conflict of interest.
★★ Author contributions: TAB and TJA conceived the study, designed the trial, ensured data collection, and drafted figures/tables and manuscript. TJA, GWA, JWO, EAS, NWHP, TMT, and EO assisted with conduction of the study and data collection. ZH and TN provided statistical planning/design and data analysis. All authors contributed significantly to manuscript revision and editing.

* Corresponding author at: University of Arkansas for Medical Sciences, 1 Children's Way, Slot 512-16, Little Rock, AR 72202. Tel.: +1 501 364 1089.
E-mail address: tabagwell@uams.edu (T.A. Bagwell).

[1] Previously worked at: Department of Pediatrics, Division of Pediatric Emergency Medicine, Vanderbilt School of Medicine, Nashville TN.
[†] The author has passed away last year after his contribution to this work.

http://dx.doi.org/10.1016/j.ajem.2016.03.005
0735-6757/

($ETco_2$) are frequently encountered during trauma and medical resuscitations and postresuscitative care; the etiologies are numerous [2]. For example, intubated pediatric patients are often inadvertently hyperventilated while being manually ventilated or bagged during resuscitations [5]. In addition, therapeutic hyperventilation has been used in the acute management of known or suspected increased intracranial pressure (ICP) for more than 40 years [4]. Standard prehospital and emergency department (ED) monitoring systems include cardiac telemetry, pulse oximetry, and capnography. Capnography or $ETco_2$ trends can be influenced by changes in ventilation, pulmonary and systemic perfusion, and/or pulmonary gas diffusion. Standard resuscitation goals aim to keep $Paco_2$ at normal values between 35 and 46 mm Hg or between 30 and 35 mm Hg if instituting therapeutic hyperventilation. Unfortunately, $ETco_2$ does not always correlate with $Paco_2$, and without confirmation via blood gas, $Paco_2$ may not be well controlled during resuscitation. Current monitoring systems are unable to detect hypocapnia-induced changes in regional cerebral perfusion and/or physiology, and there is no validated noninvasive tool for monitoring changes in cerebral perfusion during resuscitation and postresuscitative care. However, the noninvasive use of cerebral oximetry or regional cerebral tissue oxygen saturation (r_cSo_2) with cerebral blood volume index (CBVI) is capable of detecting real-time physiologic changes and providing feedback to clinicians regarding local cerebral tissue perfusion, oxygenation, and metabolism/oxygen extraction [6–7,9–11]. Cerebral oximetry readings are obtained using near infrared spectroscopy probes placed over the forehead [7,8]. There are standardized normal values, and acquired readings/trends reflect dynamic changes in local tissue perfusion, oxygenation, and metabolism/oxygen extraction referred to as *regional cerebral tissue oxygen saturation* [6–7,9–11]. Values are expressed as a ratio of venous oxyhemoglobin to deoxyhemoglobin (r_cSo_2), and pediatric normal values range from 60% to 80% [6,9,10–11]. Abnormally low values ($r_cSo_2 < 50\%$), abnormally high values ($r_cSo_2 > 80\%$), and interhemispheric discordance (difference in left and right of >10%) reflect pathology [11]. Studies have shown that r_cSo_2 values less than 50% or a change (in either direction) of 20% or greater from a patient's baseline may be indicative of increased risk for hypoxic brain injury [11]. From these data, information on regional blood flow can also be extrapolated, and this is referred to as *cerebral blood volume index* with readings ranging from −50 to +50 [9]. Negative CBVI readings are interpreted as low-flow states, and readings are not pulse dependent, which is especially useful in resuscitations [9].

We present information regarding a convenience sampling of 32 intubated and manually ventilated patients with viral meningitis, bacterial meningitis, and/or seizures at risk for increased ICP. Data will be presented detailing $ETco_2$, r_cSo_2, and CBVI during resuscitation including rapid sequence intubation (RSI) and postresuscitative care. All patients experienced nontherapeutic hyperventilation at some point during their care. Data analysis will be reviewed detailing a distinct correlation between falling $ETco_2$ values and falling r_cSo_2 and CBVI values. These findings illustrate the role cerebral oximetry can play during resuscitation as a noninvasive and real-time cerebral physiology assessment tool. Findings also suggest that the utilization of this additional information may allow clinicians to more accurately tailor neurocardiovascular resuscitative and postresuscitative efforts in the pediatric ED (PED).

2. Methods

We present an observational retrospective case series detailing the r_cSo_2, CBVI, and $ETco_2$ readings of 32 intubated and manually ventilated patients during PED resuscitation and stay. Patients were selected and data was collected at 2 level 1 trauma centers (pediatric EDs) at pediatric tertiary care facilities from 2011 to 2015. Data was pulled from both facilities' PED RSI continuous quality improvement programs database, PED electronic medical record(EMR) and vital signs, and PED cerebral oximetry database files. At both facilities, cerebral oximetry is a standard monitoring tool in pediatric patients with neurologic emergencies at risk for increased ICP, unstable multisystem trauma, respiratory failure, or cardiac arrest [6–10]. When in use, patients have left and right cerebral oximetry probes (INVOS 5100C Somanetics) placed on the forehead, and data (left and right r_cSo_2 and left and right CBVI) are sampled and recorded at 5-second intervals [6–10]. Intubated PED pateints also have inline capnography with $ETCO_2$ recordings every 5 seconds.

PED patients with neurologic emergencies—viral meningitis, bacterial meningitis with positive cerebrospinal fluid (CSF) cultures, and/or seizures requiring active resuscitation and intubation with periods of captured unintentional hyperventilation ($ETco_2 < 30$ mmHg) —were initially selected from the pediatric ED EMR, PED intubation, and cerebral oximetry REDCap (Research Electronic Capture Database) databases. Patients were selected by one of the primary investigators (after questioning the patient's respiratory therapist [RT] or trainee). Episodes of hypocapnia ($ETco_2 < 30$ mmHg greater than 2 minutes) were selected as inclusion criteria because our review was designed to detect the effects of deviation from the standard clinical practice for increased ICP management targeting $ETco_2$ 30-35 mmHg. At both PEDs, there is a dedicated PED RT who supervised accredited RT trainees. Upon patient resuscitation with intubation, the PED RT performs continuous bagging until the patient is transferred to the pediatric intensive care unit. The PED $ETco_2$ protocol was a target range between 35 and 45 mmHg unless the PED attending or fellow suspects increased ICP; then the $ETco_2$ target range is 30-35 mmHg. Only patients with inclusion and exclusion criteria and PED left and right r_cSo_2, CBVI, and $ETco_2$ monitoring with no gaps in data recording were selected by the primary investigator for analysis. Table 1 gives a stepwise process for patient selection and exclusion. Per PED cerebral near-infrared spectroscopy standard protocol, left and right probes were placed, and $ETco_2$ monitoring was used during resuscitation, RSI, intubation, ventilation, and cerebral oximetry and $ETco_2$ data were internally recorded every 5 seconds. The left and right r_cSo_2, CBVI sequence, date, and times were matched to the corresponding $ETco_2$. Correlating continuous bagging ventilation rate by the PED RT (or trainee or non-PED RT) to the $ETco_2$ recording was not possible. Monitoring devices remained in place for the duration of ED care. Health Insurance Portability and Accountability Act compliance for all aspects of patient data was maintained. The institutional review board at each facility granted approval including a waiver of informed consent.

Table 1
Data collection and patient inclusion and exclusion methods

Study inclusion criteria (in stepwise selection process):	Study exclusion criteria:
1. PED patients with meningitis: viral or bacterial with positive CSF cultures, with or without seizures. 2. Medical(nontrauma history) resuscitation with RSI intubation 3. Potential for medically induced increased ICP (nontrauma) as indicated by PED records or by CT scan report indicating possible increased ICP 4. Bag ventilation by RT or trainee (never placed on ventilator) with simultaneous $ETco_2$ monitoring with simultaneous cerebral oximetry 5. Unintentional $ETco_2 < 30$–mm Hg periods (>2 min) due to excessive bagging upon questioning the patient's RT or RT student and not PED attending directed 6. Had continuous (5-s interval) left and right r_cSo_2 with left and right CBVI correlating with $ETco_2$ monitoring with no missing data	1. Episodes of bradycardia after intubation 2. Episodes of hypotension by BP measurements for age during PED stay 3. Requiring pressor support during PED stay especially 10 min before and 20 min after low $ETco_2$ 4. Trauma history 5. CT scan indicative of physical abuse pattern (old or new) 6. Cerebrovascular accidents 7. Hydrocephalus with cerebral spinal fluid shunts 8. Brain tumors new or previously diagnosed 9. Prior neurosurgical interventions 10. Known cerebrovascular anomalies 11. Lapse or lack of corresponding left and right r_cSo_2, left and right CBVI with $ETco_2$

CT, computed tomography; BP, blood pressure.