# Association of Traumatic Brain Injuries With Vomiting in Children With Blunt Head Trauma

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**Study objective:** We aimed to determine the prevalence of traumatic brain injuries in children who vomit after minor blunt head trauma, particularly when the vomiting occurs without other findings suggestive of traumatic brain injury (ie, isolated vomiting). We also aimed to determine the relationship between the timing and degree of vomiting and traumatic brain injury prevalence.

**Methods:** This was a secondary analysis of children younger than 18 years with minor blunt head trauma. Clinicians assessed for history and characteristics of vomiting at the initial evaluation. We assessed for the prevalence of clinically important traumatic brain injury and traumatic brain injury on computed tomography (CT).

**Results:** Of 42,112 children enrolled, 5,557 (13.2%) had a history of vomiting, of whom 815 of 5,392 (15.1%) with complete data had isolated vomiting. Clinically important traumatic brain injury occurred in 2 of 815 patients (0.2%; 95% confidence interval [CI] 0% to 0.9%) with isolated vomiting compared with 114 of 4,577 (2.5%; 95% CI 2.1% to 3.0%) with nonisolated vomiting (difference –2.3%, 95% CI –2.8% to –1.5%). Of patients with isolated vomiting for whom CT was performed, traumatic brain injury on CT occurred in 5 of 298 (1.7%; 95% CI 0.5% to 3.9%) compared with 211 of 3,284 (6.4%; 95% CI 5.6% to 7.3%) with nonisolated vomiting (difference –4.7%; 95% CI –6.0% to –2.4%). We found no significant independent associations between prevalence of clinically important traumatic brain injury and traumatic brain injury on CT with either the timing of onset or time since the last episode of vomiting.

**Conclusion:** Traumatic brain injury on CT is uncommon and clinically important traumatic brain injury is very uncommon in children with minor blunt head trauma when vomiting is their only sign or symptom. Observation in the emergency department before determining the need for CT appears appropriate for many of these children. [Ann Emerg Med. 2014;63:657-665.]

Please see page 658 for the Editor's Capsule Summary of this article.

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0196-0644/$-see front matter

http://dx.doi.org/10.1016/j.annemergmed.2014.01.009

## INTRODUCTION

### Background and Importance

Blunt head trauma in children results in more than 450,000 emergency department (ED) visits annually in the United States.[1] Most blunt head trauma is minor and is associated with a very low prevalence of clinically important traumatic brain injuries.[2,3] Recently, there has been substantial attention in the medical literature and lay press about the use of computed tomography (CT) scanning for children with minor blunt head trauma. Recent research has in great part focused on the risks of radiation-induced malignancy and therefore the need to use CT judiciously.[4,5]

[†]All participants are listed in the Appendix.

Children with minor blunt head trauma frequently present to the ED with a history of vomiting.[2,3] Although vomiting is common in children with traumatic brain injuries (both clinically important traumatic brain injuries and traumatic brain injuries on CT), it also occurs in those with head trauma without traumatic brain injury, and therefore controversy exists about whether vomiting by itself discriminates between those who do and do not have traumatic brain injuries. In a previous meta-analysis, the presence of vomiting in children after head trauma, regardless of other symptoms or signs of traumatic brain injury, did not increase the overall prevalence of intracranial hemorrhage on CT, although it did increase the prevalence of neurosurgery.[6] Pooled estimates and previous studies, however, have not provided the prevalence of traumatic brain injury when vomiting is the only sign or symptom.

**Editor's Capsule Summary**

*What is already known on this topic*

Prediction rules for evaluation of pediatric blunt head trauma patients often include vomiting as a risk factor, but the meaning of vomiting in the absence of other signs and symptoms is unknown.

*What question this study addressed*

This secondary analysis of a multicenter study of 42,112 children compared the prevalence of clinically important traumatic brain injury after minor blunt head trauma in children with isolated vomiting with that of children with nonisolated vomiting.

*What this study adds to our knowledge*

Five thousand three hundred ninety-two children had vomiting, and 0.2% of the 815 with isolated vomiting had clinically important traumatic brain injury versus 2.5% of the 4,577 with nonisolated vomiting.

*How this is relevant to clinical practice*

Clinicians may consider observation in place of imaging studies in children with vomiting as the sole risk factor after minor blunt head trauma.

Vomiting has variably been included in prediction rules of traumatic brain injury in children with blunt head trauma.[7] In several prediction models of traumatic brain injury that do not include vomiting, children misclassified as not having traumatic brain injury (clinically important traumatic brain injury or traumatic brain injury on CT) frequently had a history of vomiting.[7] Our Pediatric Emergency Care Applied Research Network (PECARN) group derived and validated prediction rules separately for children younger than 2 years and those aged 2 to 18 years to identify children at very low risk of clinically important traumatic brain injury for whom CT scans can typically be obviated. For patients aged 2 to 18 years, those with a history of vomiting are classified as not being at very low risk of clinically important traumatic brain injury.[2] A history of vomiting, however, does not necessarily indicate that a patient is at high risk of clinically important traumatic brain injury, particularly if the history of vomiting is present in the absence of other signs or symptoms of traumatic brain injury (ie, isolated vomiting).

### Goals of This Investigation

To more fully understand the importance of a history of vomiting, we aimed to determine the prevalence and types of clinically important traumatic brain injuries and traumatic brain injuries on CT in children who vomit after minor blunt head trauma, particularly those who have isolated vomiting. Additionally, we aimed to assess the relationship between the timing and degree of vomiting with the prevalence of clinically important traumatic brain injury and traumatic brain injury on CT. Finally, we sought to provide the prevalence of clinically important traumatic brain injury and traumatic brain injury on CT when patients have vomiting and 1 other important sign or symptom of traumatic brain injury, as is often found in clinical practice.

## MATERIALS AND METHODS

### Study Design and Setting

We performed a planned secondary analysis of data from a large prospective observational cohort study conducted at 25 centers in the PECARN between June 2004 and September 2006. The study was approved by each site's institutional review board. Full details of the study have been published previously.[2] Below, we present details specific to the present analysis.

### Selection of Participants

In the main cohort study, we enrolled children younger than 18 years with Glasgow Coma Scale scores of 14 to 15 after nontrivial blunt head trauma who presented to the ED within 24 hours of the injury. We excluded patients with trivial trauma, defined by that resulting from ground-level falls or running into stationary objects, with no evidence of traumatic brain injury other than scalp abrasions or lacerations. We excluded patients with penetrating head trauma, preexisting neurologic disease impeding clinical assessment, or syncope or seizure preceding the head trauma, as well as patients transferred to the ED with neuroimaging already obtained. For this secondary analysis, we also excluded patients with bleeding disorders or ventricular shunts. We did not exclude patients with trauma to other body regions in association with head trauma or those who were potential victims of abuse.

### Methods of Measurement

Clinicians completed a standardized history and physical examination before cranial CT (if obtained) and documented the findings on a case report form. They evaluated for the presence or absence of a history of vomiting that occurred at any time after the traumatic event, up to the time of their ED evaluation (ie, included vomiting in the ED but only up to the time of evaluation).
If vomiting was present, clinicians documented the number of vomiting episodes (categorized as 1, 2, >2, or unknown), the timing of onset (before head injury, within 1 hour of injury, 1 to 4 hours after the injury, >4 hours after the injury, or unknown), and the time since the last episode (<1 hour before ED evaluation, 1 to 4 hours before ED evaluation, >4 hours before ED evaluation, or unknown). We defined multiple retching/vomiting within a 1-minute period as 1 episode of vomiting.

Two clinicians independently evaluated a convenience sample of 4% of patients to assess interobserver agreement of findings from patient history and physical examination. The presence of a history of vomiting (κ score of 0.91; lower 95% confidence interval [CI] 0.89) had excellent interobserver agreement.[8]

**Table 1.** Definitions of isolated vomiting.

| Extensive definition: No signs or symptoms other than a history of vomiting | PECARN Rule-Based Definition: No Signs or Symptoms Other Than a History of Vomiting Defined by the Age-Specific PECARN Prediction Rule Variables | |
|---|---|---|
| Patient <18 y met all of following: | Patient <2 y met all of the following: | Patient 2–18 y met all the following†: |
| No history of LOC | No LOC greater than 5 s | No history of LOC |
| GCS/Pediatric GCS score of 15 | Pediatric GCS score 15 | GCS score 15 |
| No signs of altered consciousness (eg, sleepiness, agitation) | No signs of altered consciousness (eg, sleepiness, agitation) | No signs of altered consciousness (eg, sleepiness, agitation) |
| No palpable skull fracture or signs of basilar skull fracture | No palpable skull fracture | No signs of basilar skull fracture |
| Acting normally per parent/guardian | No severe mechanism of injury* | No severe mechanism of injury* |
| No scalp hematoma or other traumatic scalp finding (eg, abrasion or laceration) | Acting normally per parent/guardian | No severe headache |
| No headache (for patients 2–18 y) | No temporal, parietal or occipital scalp hematoma | |
| No seizure after the head trauma | | |
| No neurologic deficits (eg, motor or sensory abnormalities) | | |
| No amnesia (for patients 2–18 y) | | |

*LOC*, Loss of consciousness; *GCS*, Glasgow Coma Scale.
*Motor vehicle crash with patient ejection, death of another passenger, or rollover; pedestrian or bicyclist without helmet struck by a motorized vehicle; falls greater than 3 feet (if aged <2 years); falls greater than 5 feet (if aged 2 to 18 years); or head struck by a high-impact object.
†The list does not include one of the rule predictors for children aged 2 to 18 years, namely, vomiting because vomiting is the focus of this article.

We defined isolated vomiting in 2 ways based on the absence of specific other clinical findings on initial ED history and physical examination (see definitions in Table 1). The first definition (termed "extensive" definition of isolated vomiting) was based on an extensive list of variables, and the second definition was based solely on the factors in the PECARN prediction rules. We analyzed the data according to these 2 definitions of isolated vomiting as the medical literature suggests that clinicians often assess children from the vantage point of having either no signs or symptoms other than a single finding of concern (extensive definition) or having no signs or symptoms other than vomiting defined solely by the age-specific PECARN prediction rule variables.[9,10] One particular difference between the 2 definitions is that the extensive definition does not consider the mechanism of injury because it is not in itself a symptom or sign of traumatic brain injury. However, because severe mechanism of injury is one of the factors in the PECARN prediction rules, patients with vomiting and a severe mechanism of injury do not meet the PECARN rule-based definition of isolated vomiting. Finally, we did not assess for headache or amnesia in patients younger than 2 years.

### Outcome Measures

We had 2 outcomes: clinically important traumatic brain injury and traumatic brain injury on CT. We defined clinically important traumatic brain injury as death from the traumatic brain injury, neurosurgical procedure for the traumatic brain injury, intubation for at least 24 hours for traumatic brain injury, or hospitalization for 2 or more nights because of the head trauma in association with traumatic brain injury on cranial CT. We considered patients to meet the criterion of hospitalization for greater than or equal to 2 nights for head trauma if they were hospitalized for at least 2 nights for signs or symptoms of head injury (eg, ongoing altered mental status) or the treating physician believed they required ongoing observation for potential acute complications of their traumatic brain injury. This definition excluded children hospitalized greater than or equal to 2 nights solely for suspicion of child abuse, for other social reasons, or for other reasons not related to the traumatic brain injury (such as orthopedic injuries). We defined traumatic brain injury on CT as any acute traumatic intracranial finding or a skull fracture depressed by at least the width of the skull. We did not consider patients with isolated skull fractures that were not depressed by at least the width of the skull as having traumatic brain injury on CT; asymptomatic patients with nondepressed skull fractures but without intracranial abnormalities almost invariably have a favorable prognosis.[11] Cranial CT scans were obtained at the discretion of the treating health care provider. CT scans were interpreted by faculty radiologists at each participating site. Equivocal traumatic findings were interpreted definitively by the study pediatric radiologist, who was masked to both clinical information and previous radiologic interpretation. As described previously, we completed rigorous follow-up procedures for all patients discharged from the ED to determine the 2 outcomes.[2]

### Primary Data Analysis

For all analyses, we removed patients missing any of the PECARN rule predictors. Additionally, for the analysis based on the extensive definition of isolated vomiting (ie, not based solely on the PECARN rule predictors), we removed patients if more than 1 of the other clinical signs or symptoms were missing or marked as unknown (because we could not be sure whether the patient truly had isolated vomiting). Additionally, we analyzed all patients meeting the extensive definition as 1 group rather than 2 age-specific groups because this definition of isolated vomiting does not rely on the PECARN rule predictors (which are somewhat different for children younger than 2 years and for those aged 2 years and older).

We described the data by using counts, percentages, and 95% CIs for categorical variables and the median and interquartile range for continuous variables. We analyzed outcomes in the following groups: (1) all patients with a history of vomiting after the traumatic event who also had other signs or symptoms suggestive of traumatic brain injury (ie, nonisolated vomiting); (2) patients with isolated vomiting (extensive definition); and (3) patients with isolated vomiting according to the PECARN prediction rules (ie, vomiting with no other age-specific PECARN traumatic brain injury rule variable).

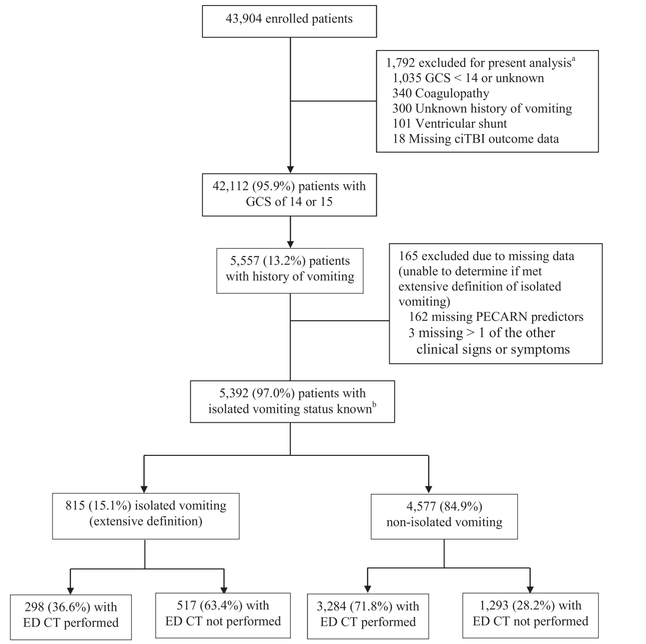
We compared the rates of traumatic brain injury on CT and clinically important traumatic brain injury for children with and without isolated vomiting (extensive definition), using the Newcombe-Wilson continuity-adjusted method because of the low prevalence rates of the outcomes. We conducted 2 multivariable logistic regression analyses to assess the independent associations between traumatic brain injury (clinically important traumatic brain injury and traumatic brain injury on CT) and the number of episodes and timing of the vomiting. We adjusted for all variables included in the extensive definition of isolated vomiting and also included age (categorized as $<2$ years or $\geq 2$ to 18 years), number of vomiting episodes, timing of vomiting onset, and time since last vomiting episode. Age and all variables included in the extensive definition were entered as dichotomous variables in the regression model; vomiting characteristics were entered as categorical variables. Additionally, we adjusted for the time from injury to time of ED evaluation as a continuous variable in hours because this time potentially influences the relationship between the number and timing of vomiting episodes and the outcomes of interest. We were unable to add concomitant abdominal injury to the model because of a low prevalence of this variable in patients with isolated vomiting. We did not include headache and amnesia in the multivariable analyses because they were not assessed in children younger than 2 years. We used the Hosmer-Lemeshow test to assess goodness of fit for both models. Finally, we assessed the prevalence of clinically important traumatic brain injury and traumatic brain injury on CT in patients who had vomiting in addition to 1 other finding in the age-specific PECARN prediction rules to distinguish the prevalence of clinically important traumatic brain injury and traumatic brain injury on CT in patients who have from those who do not have isolated vomiting in a more granular way.

We used SAS/STAT software (version 9.2; SAS Institute, Inc., Cary, NC) for all analyses. Because this was a secondary analysis of the cohort study, we did not estimate sample size needs related to this analysis.

## RESULTS

### Characteristics of Study Subjects

We enrolled 43,904 patients in the parent cohort study (76.9% of the 57,030 eligible patients), and 1,792 were excluded




[a] Two patients had more than one exclusion
[b] 4,669/5,392 patients (86.6%) were discharged home from ED, of whom 3,838 (82.2%) completed telephone or mail follow-up; medical records, trauma registries, process improvement reports, and morgue records were reviewed for the remaining 831 (17.8%)

**Figure.** Study population. *ciTBI,* Clinical important traumatic brain injury.

from the present analysis (Figure). Of the remaining 42,112 patients, 5,557 (13.2%) had a history of vomiting. When we categorized these 5,557 patients in regard to whether the vomiting was isolated (extensive definition), 165 (3.0%) were excluded from further analysis because of missing data (ie, unable to determine whether the patient met the definition of isolated vomiting). Of the 5,392 patients with complete data, 4,577 (84.9%) had nonisolated vomiting (had at least 1 other finding per the extensive definition) and 815 (15.1%) had isolated vomiting (extensive definition). The characteristics of patients with nonisolated and isolated vomiting are detailed in Table 2. Clinicians obtained cranial CT scans for 3,284 patients (71.8%) with nonisolated vomiting and on 298 (36.6%) of those with isolated vomiting (extensive definition).

### Main Results

Table 3 demonstrates the prevalence of clinically important traumatic brain injury and traumatic brain injury on CT separately for patients with isolated (extensive definition) and nonisolated vomiting, including the prevalence of traumatic brain injury based on the number and timing of vomiting episodes. All patients with vomiting and clinically important traumatic brain injuries had traumatic brain injuries on CT. Patients with isolated vomiting had a low prevalence of clinically important traumatic brain injury, irrespective of the number or timing of vomiting episodes. Comparatively, clinically important traumatic brain injuries occurred in 2 of 815 patients

**Table 2.** Clinical characteristics of children with isolated (extensive definition) and nonisolated vomiting.*

| Characteristic | Nonisolated Vomiting (N=4,577) | Isolated Vomiting (N=815) |
|---|---|---|
| Median age (interquartile range), y | 5.7 (2.2, 10.7) | 1.6 (0.8, 5.0) |
| Male | 2,799 (61.2) | 419 (51.4) |
| **Mechanism of injury** | | |
| Fall from elevation | 1,432 (31.3) | 401 (49.2) |
| <3 ft | 580 (40.5) | 235 (58.6) |
| 3–5 ft | 664 (46.4) | 154 (38.4) |
| 6–10 ft | 112 (7.8) | 10 (2.5) |
| >10 ft | 34 (2.4) | 0 |
| Unknown height | 42 (2.9) | 2 (0.5) |
| Fall from standing/walking/running | 786 (17.2) | 113 (13.9) |
| Sports | 394 (8.6) | 22 (2.7) |
| Fall down stairs | 281 (6.1) | 56 (6.9) |
| Walked or ran into stationary object | 254 (5.5) | 40 (4.9) |
| Object struck head, accidental | 194 (4.2) | 41 (5.0) |
| Bike collision or fall from bike while riding | 201 (4.4) | 13 (1.6) |
| Assault | 191 (4.2) | 17 (2.1) |
| Motor vehicle crash | 142 (3.1) | 20 (2.5) |
| Other wheeled transport crash | 93 (2.0) | 10 (1.2) |
| Pedestrian struck by moving vehicle | 60 (1.3) | 3 (0.4) |
| Bike rider struck by automobile | 21 (0.5) | 3 (0.4) |
| Other | 481 (10.5) | 74 (9.1) |
| Unknown | 47 (1.0) | 2 (0.2) |
| Concomitant significant injury | 288 (6.3) | 28 (3.4) |
| Abdominal injury only | 41 (14.2) | 1 (3.6) |
| Abdominal injury+other significant concomitant injury | 11 (3.8) | 1 (3.6) |
| No abdominal injury but other significant concomitant injury | 227 (78.8) | 26 (92.9) |
| Unknown | 9 (3.1) | 0 |

*Data are presented as No. (%) unless otherwise indicated.

(0.2%; 95% CI 0% to 0.9%) in the isolated vomiting group versus 114 of 4,577 (2.5%; 95% CI 2.1% to 3.0%) in the nonisolated vomiting group (risk difference –2.3%; 95% CI –2.8% to –1.5%). Traumatic brain injuries on CT occurred in 5 of 298 patients (1.7%; 95% CI 0.5% to 3.9%) in the isolated vomiting group versus 211 of 3,284 (6.4%; 95% CI 5.6% to 7.3%) in the nonisolated vomiting group (risk difference –4.7%; 95% CI –6.0% to –2.4%). For purposes of comparison, in the parent cohort 6,936 patients had neither vomiting nor any extensive definition findings, of whom 3 (0.04%) had clinically important traumatic brain injury.

We present the specific traumatic brain injuries and clinical characteristics in those patients with isolated vomiting in Table 4. None of these patients underwent neurosurgery, although a few had epidural, subdural or extra-axial hematomas. Both patients who had clinically important traumatic brain injuries had severe mechanisms of injury (defined a priori in the parent PECARN study).[2]

Table 5 presents the logistic regression analyses to determine the association of vomiting characteristics with clinically important traumatic brain injury and traumatic brain injury on CT. As noted, the 95% CIs for the adjusted odds ratios cross 1 for all characteristics other than number of episodes of vomiting, for which there is a counterintuitive decrease in risk of traumatic brain injury on CT with increased number of vomiting episodes.

Finally, Tables 6 and 7 demonstrate the prevalence and types of traumatic brain injuries in patients with isolated vomiting when defined by the absence of the factors in the age-specific PECARN prediction rules. Overall, the prevalence of clinically important traumatic brain injury in this group was low for both age groups. Of the 2 patients younger than 2 years with vomiting and none of the age-specific PECARN prediction rule factors who had traumatic brain injury on CT, one had an intracranial hemorrhage (extra-axial hematoma). This patient was 2 months old, had fallen less than 3 feet, and had vomited twice. In patients aged 2 to 18 years who had vomiting but none of the other age-specific PECARN rule factors, clinically important traumatic brain injury occurred in 10 of 1,501 (0.7%; 95% CI 0.3% to 1.2%); 5 of the 1,501 (0.3%; 95% CI 0.1% to 0.8%) underwent neurosurgery (Table 7), including 4 patients who had intracranial hematomas drained. Of those 5 undergoing neurosurgery, all had mild to moderate headaches, 3 had nonfrontal scalp hematomas, and all had their vomiting onset within 1 hour of the time of injury.

## LIMITATIONS

The study had certain limitations. Our assessment of vomiting at a single point resulted in an inability to specifically assess for persistence or recurrence of emesis while patients were in the ED, after completion of the case report form. Although we provided training on the definition of a vomiting episode, it is likely that some clinicians had their own definitions in mind. Nevertheless, vomiting is a fairly objective finding, with good interobserver agreement in our study.[8]

Another limitation is that we did not collect data or account for certain potential confounding factors, which may have resulted in vomiting unrelated to the head trauma, such as riding in an ambulance or administration of medications.

Our sample, although the largest prospectively gathered sample of its kind to our knowledge, nevertheless had too few outcomes to provide precise estimates related to the association between traumatic brain injury and the number of episodes and timing of the vomiting. Additionally, we were unable to complete telephone or mail follow-up on the 831 patients (17.8%) for whom isolated vomiting status was known, which could have led to an underestimation of the prevalence of clinically important traumatic brain injury or traumatic brain injury on CT. However, we reviewed the medical records for each patient not contacted by telephone or mail, checked the trauma registries and quality

**Table 3.** Prevalence of traumatic brain injury in patients with isolated (extensive definition) and nonisolated vomiting.

| | Nonisolated Vomiting (N=4,577), n/N (%; 95% CI) | | Isolated Vomiting (N=815), n/N (%; 95% CI) | |
|---|---|---|---|---|
| **Vomiting Characteristic** | **ciTBI*** | **TBI on CT** | **ciTBI*** | **TBI on CT** |
| Any vomiting, irrespective of timing | 114/4,577 (2.5; 2.1–3.0) | 211/3,284 (6.4; 5.6–7.3) | 2/815 (0.2; 0–0.9) | 5/298 (1.7; 0.5–3.9) |
| **Number of vomiting episodes** | | | | |
| 1 | 45/1,707 (2.6; 1.9–3.5) | 93/1,022 (9.1; 7.4–11.0) | 1/326 (0.3; 0–1.7) | 1/74 (1.4; 0–7.3) |
| 2 | 16/893 (1.8; 1.0–2.9) | 26/618 (4.2; 2.8–6.1) | 0/185 (0; 0–2.0) | 2/62 (3.2; 0.4–11.2) |
| >2 | 46/1,772 (2.6; 1.9–3.4) | 82/1,477 (5.6; 4.4–6.8) | 0/267 (0; 0–1.4) | 1/141 (0.7; 0–3.9) |
| Unknown | 7/205 (3.4; 1.4–6.9) | 10/167 (6.0; 2.9–10.7) | 1/37 (2.7; 0.1–14.2) | 1/21 (4.8; 0.1–23.8) |
| **Onset of vomiting** | | | | |
| Before head trauma | 0/39 (0; 0–9.0) | 1/17 (5.9; 0.1–28.7) | 0/23 (0; 0–14.8) | 0/6 (0; 0–45.9) |
| Within 1 h after trauma | 72/2,414 (3.0; 2.3–3.7) | 129/1,694 (7.6; 6.4–9.0) | 0/459 (0; 0–0.8) | 0/157 (0; 0–2.3) |
| 1–4 h after trauma | 30/1,327 (2.3; 1.5–3.2) | 58/987 (5.9; 4.5–7.5) | 1/188 (0.5; 0–2.9) | 3/79 (3.8; 0.8–10.7) |
| >4 h after trauma | 6/481 (1.2; 0.5–2.7) | 13/359 (3.6; 1.9–6.1) | 1/107 (0.9; 0–5.1) | 2/39 (5.1; 0.6–17.3) |
| Unknown | 6/316 (1.9; 0.7–4.1) | 10/227 (4.4; 2.1–8.0) | 0/38 (0; 0–9.3) | 0/17 (0; 0–19.5) |
| **Last episode of vomiting** | | | | |
| <1 h before ED evaluation | 74/2,260 (3.3; 2.6–4.1) | 126/1,857 (6.8; 5.7–8.0) | 0/285 (0; 0–1.3) | 1/148 (0.7; 0–3.7) |
| 1–4 h before ED evaluation | 15/1,304 (1.2; 0.6–1.9) | 37/737 (5.0; 3.6–6.9) | 1/334 (0.3; 0–1.7) | 2/86 (2.3; 0.3–8.1) |
| >4 h before ED evaluation | 1/253 (0.4; 0–2.2) | 3/115 (2.6; 0.5–7.4) | 0/78 (0; 0–4.6) | 0/12 (0; 0–26.5) |
| Unknown | 24/760 (3.2; 2.0–4.7) | 45/575 (7.8; 5.8–10.3) | 1/118 (0.8; 0–4.6) | 2/52 (3.8; 0.5–13.2) |

*TBI*, Traumatic brain injury.
*ciTBI definition: death, neurosurgical procedure, intubation for at least 24 hours for TBI, or hospitalization for 2 or more nights because of the head trauma in association with TBI on cranial CT.

improvement reports at each site, and reviewed the morgue records for each region to ensure we did not miss any children with clinically important traumatic brain injury; 0 of 831 patients had clinically important traumatic brain injury according to this assessment. In the parent study, of 29,410 patients without known traumatic brain injuries who were discharged from the ED and had telephone follow-up, only 1 had clinically important traumatic brain injury and 4 had traumatic brain injuries on CT (of those who subsequently had CTs).

Finally, clinicians obtained CTs on a minority of children, with selection bias likely toward those with more severe findings and higher prevalence of traumatic brain injury. Because this bias would be expected to inflate the prevalence of traumatic brain injury on CT, the overall prevalence, given isolated vomiting, is likely lower than that reported here.

## DISCUSSION

We evaluated a large sample of children with vomiting after minor blunt head trauma and noted that traumatic brain injury on CT was uncommon and clinically important traumatic brain injury was very uncommon when vomiting was their only sign or symptom of head injury. Traumatic brain injury was more frequent in children with vomiting in the presence of additional signs or symptoms of traumatic brain injury (ie, when vomiting was not "isolated"). The low overall prevalence of clinically important traumatic brain injury and the finding that clinicians obtained CTs in the minority of patients with isolated vomiting suggest that CT is not immediately required in most of these children, and a period of observation before CT decisionmaking should be considered. In children aged 2 to 18 years with isolated vomiting based on the PECARN prediction rule factors, although the overall prevalence of clinically important traumatic brain injury was low, several patients underwent neurosurgery. This finding emphasizes the importance of, at a minimum, several hours of ED observation before CT decisionmaking for these patients to assess for progression of signs and symptoms. Our data also suggest that CT should be strongly considered when a history of vomiting is accompanied by the presence of other concerning clinical findings.

Previous data regarding the potential importance of vomiting in children with minor blunt head trauma are challenging to

**Table 4.** Traumatic brain injuries in children with isolated vomiting (extensive definition).

| Age | Mechanism Specifics | Number of Episodes of Vomiting | TBI on CT | ciTBI |
|---|---|---|---|---|
| 2 mo | Fall from <3 ft | Twice | Extra-axial hematoma | None |
| 3 mo | Fall from 3–5 ft | Twice | Subdural hematoma | None |
| 6 mo | Fall from 3–5 ft | Unknown | Subdural hematoma | Hospitalization ≥2 nights in association with TBI on CT |
| 13 mo | Fall from <3 ft | >2 times | Diastasis of the skull | None |
| 9 y | Hit on forehead by baseball | Once | Cerebral contusion, epidural hematoma, subarachnoid hemorrhage | Hospitalization ≥2 nights in association with TBI on CT |

**Table 5.** Multivariable analyses to identify predictors of TBI according to characteristics of vomiting.*

| | Adjusted OR (95% CI) | |
|---|---|---|
| **Characteristic of Vomiting** | **ciTBI† (n=3,381)** | **TBI on CT† (n=2,092)** |
| **Number of episodes of vomiting** | | |
| 1 | Reference | Reference |
| 2 | 0.8 (0.3–2.0) | 0.5 (0.3–1.0) |
| >2 | 0.9 (0.4–1.8) | 0.5 (0.3–0.9) |
| **Onset of vomiting** | | |
| Before head injury/within 1 h of event | Reference | Reference |
| 1-4 h after event | 0.9 (0.5–1.8) | 0.8 (0.5–1.3) |
| >4 h after event | 0.5 (0.1–2.1) | 0.5 (0.2–1.3) |
| **Last episode of vomiting** | | |
| <1 h before ED evaluation | Reference | Reference |
| ≥1 h before ED evaluation | 0.6 (0.3–1.3) | 0.8 (0.5–1.3) |

*OR*, Odds ratio.
*Variables included in both regression models along with the reference group: altered mental status (ref=no), acting normally per parent (ref=yes), history of loss of consciousness (ref=no), posttraumatic seizure (ref=no), any scalp hematoma (ref=no), other traumatic scalp findings (ref=no), palpable skull fracture (ref=no), clinical signs of basilar skull fracture (ref=no), neurologic deficit (ref=no), number of vomiting episodes (ref=1), onset of vomiting (ref=before head injury combined with vomiting within 1 hour of event), timing of last vomiting episode (ref=<1 hour before ED evaluation) , age group (ref=<2 years), time from injury to ED evaluation in hours.
†Both logistic regression models had an adequate fit (Hosmer-Lemeshow goodness of fit *P*=.68 and *P*=.78 for the ciTBI and TBI on CT models, respectively).

summarize systematically because of substantial differences in study populations (eg, ages, illness severity), assessment methods across studies, and outcome definitions.[6,12-16] One systematic review noted that vomiting did not differentiate children with and without intracranial hemorrhage (odds ratio 0.88; 95% CI 0.67 to 1.15).[10] In a separate, recent systematic review, "undefined vomiting" had a pooled negative likelihood ratio of 0.91 (95% CI 0.77 to 1.06) and positive likelihood ratio of 1.29 (95% CI 0.85 to 1.99) for intracranial injury.[6] "Undefined vomiting" had a slightly higher likelihood ratio to predict the need for neurosurgery, with a positive likelihood ratio of 2.36 (95% CI 0.96 to 5.83).[6] However, the systematic reviews compared patients with any vomiting to those without vomiting and did not focus on isolated vomiting. Additionally, the estimates in the systematic reviews provide the test characteristics for children with vomiting who may or may not have had other clinical findings (eg, severe headache, temporal/parietal scalp hematoma). The current study builds on the previous systematic reviews by providing estimates of traumatic brain injury prevalence when vomiting is present without other risk factors of traumatic brain injury.

Although the role of vomiting as an independent discriminator for traumatic brain injury on CT in children appears modest, most but not all previous traumatic brain injury prediction rules for children with minor blunt head trauma include vomiting as either a factor that decreases the prevalence of traumatic brain injury when absent (for rules to identify low-risk patients) or increases the prevalence when present (if the rule was created to identify high-risk patients).[2,3,7,12,17-22] For those rules that include vomiting, however, its definition and categorization differ, with rules using any vomiting, persistent vomiting, or a specific number of episodes of vomiting as predictors.[2,3,18-20,22] The inclusion of vomiting in some rules suggests that its importance may be to identify patients who are not easily captured by other predictors that, overall, may have more discriminatory value. Nevertheless, we found that the prevalence of traumatic brain injury, whether on CT or of clinical importance, was very low if a history of vomiting was the only clinical finding.

Previous investigators have attempted to assess the relationship between traumatic brain injury and the number and timing of vomiting episodes. Our data suggest that the use of these vomiting characteristics is not generally clinically helpful, as evidenced by a counterintuitive increase in traumatic brain injury with fewer episodes of vomiting. Previous data in children suggest a slight increase in the prevalence of intracranial hemorrhage with "persistent vomiting," with a pooled positive likelihood ratio estimate of 3.14 (95% CI 1.30 to 8.05) and a

**Table 6.** Prevalence of traumatic brain injuries in patients with isolated vomiting, and vomiting plus 1 other factor, based on age-specific PECARN prediction rule factors.

| PECARN Prediction Rule Variables | ciTBI, n/N (%; 95% CI) | TBI on CT, n/N (%; 95% CI) |
|---|---|---|
| **Children <2 y** | | |
| Isolated vomiting | 0/567 (0; 0–0.6) | 2/187 (1.1; 0.1–3.8) |
| Vomiting plus altered mental status* | 0/35 (0; 0–10.0) | 1/25 (4.0; 0.1–20.4) |
| Vomiting plus nonfrontal scalp hematoma | 0/76 (0; 0–4.7) | 2/39 (5.1; 0.6–17.3) |
| Vomiting plus LOC ≥5 s | 1/18 (5.6; 0.1–27.3) | 1/14 (7.1; 0.2–33.9) |
| Vomiting plus palpable skull fracture | 0/5 (0; 0–52.2) | 0/3 (0; 0–70.8) |
| Vomiting plus not acting normally per parent | 1/158 (0.6; 0–3.5) | 2/104 (1.9; 0.2–6.8) |
| Vomiting plus severe mechanism of injury | 1/181 (0.6; 0–3.0) | 2/66 (3.0; 0.4–10.5) |
| **Children 2 to <18 y** | | |
| Isolated vomiting | 10/1,501 (0.7; 0.3–1.2) | 26/806 (3.2; 2.1–4.7) |
| Vomiting plus altered mental status* | 9/487 (1.8; 0.8–3.5) | 18/424 (4.2; 2.5–6.6) |
| Vomiting plus any LOC | 3/321 (0.9; 0.2–2.7) | 6/260 (2.3; 0.9–5.0) |
| Vomiting plus clinical signs of basilar skull fracture | 3/16 (18.8; 4.0–45.6) | 6/14 (42.9; 17.7–71.1) |
| Vomiting plus severe headache | 1/69 (1.4; 0–7.8) | 1/60 (1.7; 0–8.9) |
| Vomiting plus severe mechanism of injury | 2/84 (2.4; 0.3–8.3) | 3/59 (5.1; 1.1–14.1) |

*Altered mental status defined as a Glasgow Coma Scale score of 14, agitation, sleepiness, slow to respond, or repetitive questioning.

**Table 7.** Details of traumatic brain injuries in children aged 2 to 18 years with isolated vomiting according to PECARN rule factors.

| Type of TBI | n/N (%; 95% CI) |
|---|---|
| **Clinically important TBI*** | 10/1,501 (0.7; 0.3–1.2) |
| Hospitalization ≥2 nights in association with TBI on CT | 10/1,501 (0.7; 0.3–1.2) |
| Neurosurgery | 5/1,501 (0.3; 0.1–0.8) |
| Intubation for TBI ≥24 h | 0/1,501 (0; 0–0.2) |
| Death because of TBI | 0/1,501 (0; 0–0.2) |
| **TBI on CT*** | 26/806 (3.2; 2.1–4.7) |
| Epidural hematoma | 8/806 (1.0; 0.4–1.9) |
| Cerebral contusion | 6/806 (0.7; 0.3–1.6) |
| Subdural hematoma | 5/806 (0.6; 0.2–1.4) |
| Diastasis of the skull | 4/806 (0.5; 0.1–1.3) |
| Midline shift | 4/806 (0.5; 0.1–1.3) |
| Cerebral hemorrhage/intracerebral hematoma | 3/806 (0.4; 0.1–1.1) |
| Extra-axial hematoma | 3/806 (0.4; 0.1–1.1) |
| Pneumocephalus | 2/806 (0.2; 0–0.9) |
| Subarachnoid hemorrhage | 2/806 (0.2; 0–0.9) |
| Cerebellar hemorrhage | 1/806 (0.1; 0–0.7) |
| Depressed skull fracture width of skull | 1/806 (0.1; 0–0.7) |

*Patients could meet more than 1 criterion for ciTBI or have more than 1 TBI on CT.

negative likelihood ratio estimate of 0.840 (95% CI 0.64 to 0.97).[6]

In conclusion, traumatic brain injury on CT is uncommon and clinically important traumatic brain injury is very uncommon in children with minor blunt head trauma when vomiting is their only sign or symptom, assessed at a single point in the ED. Consequently, CT is generally not required in these children, although a period of clinical observation in the ED before CT decisionmaking is prudent to assess for progression of signs and symptoms. Traumatic brain injury is more frequent in children when the vomiting is accompanied by other signs or symptoms suggestive of traumatic brain injury; CT should be seriously considered in these circumstances.

*The authors acknowledge Rene Enriquez, BS, and Sally Jo Zuspan, RN, MSN, at the PECARN Data Center (University of Utah) for their dedicated and diligent work; the research coordinators in PECARN, without whose dedication and hard work this study would not have been possible; and all the clinicians of the PECARN who enrolled children in this study.*

*Supervising editor:* Kelly D. Young, MD, MS

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*Author contributions:* PSD and NK conceived of and designed the study. NK obtained research funding. PSD, JFL, SA, JH, MGT, RL, EA, and NK supervised the conduct of the study and data collection at participating centers. MM managed the data, including quality control; conducted the statistical analyses; and takes responsibility for the accuracy of the data analysis. PSD and NK drafted the article, and all authors contributed substantially to its revision. MM and NK had full access to all the data in the study and take responsibility for the integrity of the data. PSD takes responsibility for the paper as a whole.

*Funding and support:* By *Annals* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article as per ICMJE conflict of interest guidelines (see www.icmje.org). The authors have stated that no such relationships exist and provided the following details: This work was supported by a grant from the Health Resources and Services Administration/Maternal and Child Health Bureau (HRSA/MCHB), Division of Research, Training, and Education (DRTE) and the Emergency Medical Services of Children (EMSC) Program (R40MC02461). The Pediatric Emergency Care Applied Research Network is supported by cooperative agreements U03MC00001, U03MC00003, U03MC00006, U03MC00007, and U03MC00008 from the EMSC program of the HRSA/MCHB.

*Publication dates:* Received for publication August 21, 2013. Revisions received November 22, 2013, and December 24, 2013. Accepted for publication January 8, 2014. Available online February 19, 2014.

Presented at the Pediatric Academic Societies annual meeting, May 2008, Honolulu, HI; and the Society for Academic Emergency Medicine annual meeting, May 2008, Washington, DC.

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## APPENDIX

Participating centers and site investigators are listed below in alphabetical order: Atlantic Health System/Morristown Memorial Hospital (M. Gerardi); Bellevue Hospital Center (M. Tunik, J. Tsung); Calvert Memorial Hospital (K. Melville); Children's Hospital Boston (L. Lee); Children's Hospital of Michigan (P. Mahajan); Children's Hospital of New York–Presbyterian (P. Dayan); Children's Hospital of Philadelphia (F. Nadel); Children's Memorial Hospital (E. Powell); Children's National Medical Center (S. Atabaki, K. Brown); Cincinnati Children's Hospital Medical Center (T. Glass); DeVos Children's Hospital (J. Hoyle); Harlem Hospital Center (A. Cooper); Holy Cross Hospital (E. Jacobs); Howard County Medical Center (D. Monroe); Hurley Medical Center (D. Borgialli); Medical College of Wisconsin/Children's Hospital of Wisconsin (M. Gorelick, S. Bandyopadhyay); St. Barnabas Health Care System (M. Bachman, N. Schamban); SUNY–Upstate Medical Center (J. Callahan); University of California Davis Medical Center (N. Kuppermann, J. Holmes); University of Maryland (R. Lichenstein); University of Michigan (R. Stanley); University of Rochester (M. Badawy, L. Babcock-Cimpello); University of Utah/Primary Children's Medical Center (J. Schunk); Washington University/St. Louis Children's Hospital (K. Quayle, D. Jaffe); Women and Children's Hospital of Buffalo (K. Lillis).

We acknowledge the efforts of the following individuals participating in PECARN at the time this study was initiated.

PECARN Steering Committee: N. Kuppermann, Chair; E. Alpern, J. Chamberlain, J. M. Dean, M. Gerardi, J. Goepp, M. Gorelick, J. Hoyle, D. Jaffe, C. Johns, N. Levick, P. Mahajan, R. Maio, K. Melville, S. Miller,† D. Monroe, R. Ruddy, R. Stanley, D. Treloar, M. Tunik, A. Walker. MCHB/EMSC liaisons: D. Kavanaugh, H. Park.

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