

# Immediate and Delayed Traumatic Intracranial Hemorrhage in Patients With Head Trauma and Preinjury Warfarin or Clopidogrel Use

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**Study objective:** Patients receiving warfarin or clopidogrel are considered at increased risk for traumatic intracranial hemorrhage after blunt head trauma. The prevalence of immediate traumatic intracranial hemorrhage and the cumulative incidence of delayed traumatic intracranial hemorrhage in these patients, however, are unknown. The objective of this study is to address these gaps in knowledge.

**Methods:** A prospective, observational study at 2 trauma centers and 4 community hospitals enrolled emergency department (ED) patients with blunt head trauma and preinjury warfarin or clopidogrel use from April 2009 through January 2011. Patients were followed for 2 weeks. The prevalence of immediate traumatic intracranial hemorrhage and the cumulative incidence of delayed traumatic intracranial hemorrhage were calculated from patients who received initial cranial computed tomography (CT) in the ED. Delayed traumatic intracranial hemorrhage was defined as traumatic intracranial hemorrhage within 2 weeks after an initially normal CT scan result and in the absence of repeated head trauma.

**Results:** A total of 1,064 patients were enrolled (768 warfarin patients [72.2%] and 296 clopidogrel patients [27.8%]). There were 364 patients (34.2%) from Level I or II trauma centers and 700 patients (65.8%) from community hospitals. One thousand patients received a cranial CT scan in the ED. Both warfarin and clopidogrel groups had similar demographic and clinical characteristics, although concomitant aspirin use was more prevalent among patients receiving clopidogrel. The prevalence of immediate traumatic intracranial hemorrhage was higher in patients receiving clopidogrel (33/276, 12.0%; 95% confidence interval [CI] 8.4% to 16.4%) than patients receiving warfarin (37/724, 5.1%; 95% CI 3.6% to 7.0%), relative risk 2.31 (95% CI 1.48 to 3.63). Delayed traumatic intracranial hemorrhage was identified in 4 of 687 (0.6%; 95% CI 0.2% to 1.5%) patients receiving warfarin and 0 of 243 (0%; 95% CI 0% to 1.5%) patients receiving clopidogrel.

**Conclusion:** Although there may be unmeasured confounders that limit intergroup comparison, patients receiving clopidogrel have a significantly higher prevalence of immediate traumatic intracranial hemorrhage compared with patients receiving warfarin. Delayed traumatic intracranial hemorrhage is rare and occurred only in patients receiving warfarin. Discharging patients receiving anticoagulant or antiplatelet medications from the ED after a normal cranial CT scan result is reasonable, but appropriate instructions are required because delayed traumatic intracranial hemorrhage may occur. [Ann Emerg Med. 2012;59:460-468.]

Please see page 461 for the Editor's Capsule Summary of this article.

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**Editor's Capsule Summary***What is already known on this topic*

Anticoagulant and antiplatelet drugs increase the risk for traumatic intracranial hemorrhage after blunt head trauma.

*What question this study addressed*

What is the incidence and prevalence of immediate and delayed traumatic intracranial hemorrhage in patients with blunt head trauma who are receiving clopidogrel and warfarin?

*What this study adds to our knowledge*

In this prospective observational multicenter study of 1,064 patients, the prevalence of immediate traumatic intracranial hemorrhage was 12% for patients receiving clopidogrel and 5.1% for those receiving warfarin. Delayed traumatic intracranial hemorrhage was 0% and 0.6%, respectively.

*How this is relevant to clinical practice*

In blunt head trauma, patients receiving clopidogrel may be at greater risk of immediate traumatic intracranial hemorrhage than those receiving warfarin. Delayed traumatic intracranial hemorrhage is rare and it may be reasonable to discharge a patient after a normal head CT scan result.

recommending routine cranial CT imaging and hospital admission for neurologic observation in head-injured patients receiving warfarin.<sup>11-14</sup> These recommendations, however, are not informed by rigorous, prospective, multicenter studies identifying the prevalence and incidence of immediate traumatic intracranial hemorrhage and delayed traumatic intracranial hemorrhage in patients receiving warfarin.

The evidence supporting an increased risk of traumatic intracranial hemorrhage in patients receiving clopidogrel is more limited,<sup>11</sup> despite this drug being one of the most commonly prescribed worldwide.<sup>15</sup> Although small retrospective studies suggest an increased risk of traumatic intracranial hemorrhage and mortality in head trauma patients receiving clopidogrel,<sup>6,16,17</sup> current guidelines do not explicitly recommend routine CT imaging for these patients after blunt head trauma.<sup>11-13</sup> In addition, the risk of delayed traumatic intracranial hemorrhage in patients receiving clopidogrel is entirely unknown.

**Goals of This Investigation**

Knowledge of the true prevalence and incidence of immediate and delayed traumatic intracranial hemorrhage in patients receiving warfarin or clopidogrel would allow clinicians to make evidence-based decisions about their initial patient evaluation and disposition. Therefore, we assessed the prevalence and incidence of immediate and delayed traumatic intracranial hemorrhage in patients with blunt head trauma who were receiving either warfarin or clopidogrel. Warfarin and clopidogrel cohorts were compared. We hypothesized that the prevalence for immediate traumatic intracranial hemorrhage was similar between patients receiving clopidogrel and those receiving warfarin and that the cumulative incidence of delayed traumatic intracranial hemorrhage in both groups was less than 1%.

**SEE EDITORIAL, P. 469.****INTRODUCTION****Background**

The use of anticoagulant and antiplatelet medications, specifically warfarin and clopidogrel, is steadily increasing.<sup>1-3</sup> Previous studies suggest that patients receiving either of these medications are at increased risk for traumatic intracranial hemorrhage after blunt head trauma, but the risk in a large, generalizable cohort is unknown.<sup>4-6</sup>

**Importance**

The majority of patients with traumatic intracranial hemorrhage are identified on initial cranial computed tomographic (CT) scan. Limited data, however, suggest that patients receiving warfarin are at increased risk for delayed traumatic intracranial hemorrhage (traumatic intracranial hemorrhage diagnosed within 2 weeks of injury after an initially normal cranial CT scan result).<sup>7-9</sup> The concern for delayed traumatic intracranial hemorrhage is highlighted by the not uncommon practice of reversing warfarin anticoagulation in patients with head trauma and a normal cranial CT scan result.<sup>10</sup> The potential risk for both immediate and delayed traumatic intracranial hemorrhage has generated guidelines

**MATERIALS AND METHODS****Study Design**

This was a prospective, observational, multicenter study conducted at 2 trauma centers and 4 community hospitals in Northern California. The study was approved by the institutional review boards at all sites.

**Setting and Selection of Participants**

Adult (aged  $\geq 18$  years) emergency department (ED) patients with blunt head trauma and preinjury warfarin or clopidogrel use (within the previous 7 days) were enrolled. We defined blunt head trauma as any blunt head injury regardless of loss of consciousness or amnesia. We excluded patients with known injuries who were transferred from outside facilities because their inclusion would falsely inflate the prevalence of traumatic intracranial hemorrhage. Additionally, patients with concomitant warfarin and clopidogrel use were excluded.

**Data Collection and Processing**

The treating ED faculty physicians recorded patient history and medication use, injury mechanism, and clinical examination,

including initial Glasgow Coma Scale score (GCS) and evidence of trauma above the clavicles (defined as trauma to the face, neck, or scalp) on a standardized data form (Appendix E1-E4, available online at <http://www.annemergmed.com>) before cranial CT (if obtained). Imaging studies were obtained at the discretion of the treating physician and not dictated by study protocol. At each site, approximately 10% of patients (nonrandomly selected) had a separate, independent faculty physician assessment that was masked and completed within 60 minutes of the initial assessment to evaluate the reliability of preselected clinical variables. Data on patients eligible but not enrolled (failures of the study screening process) during ED evaluation were abstracted from their medical records to assess for enrollment bias.

### Outcome Measures

Immediate traumatic intracranial hemorrhage was defined as the presence of any intracranial hemorrhage or contusion as interpreted by the faculty radiologist on the initial cranial CT scan. Patients without a cranial CT scan during initial ED evaluation were excluded from the immediate traumatic intracranial hemorrhage calculation. Delayed traumatic intracranial hemorrhage was defined as traumatic intracranial hemorrhage on cranial CT scan, occurring within 14 days after an initial normal CT scan result and in the absence of repeated head trauma. Neurosurgical intervention was defined as the use of intracranial pressure monitor or brain tissue oxygen probe, placement of a burr hole, craniotomy/craniectomy, intraventricular catheter or subdural drain, or the use of mannitol or hypertonic saline solution.

Patients were admitted to the hospital at the discretion of the emergency physician. Patients with normal cranial CT scan results and therapeutic international normalized ratio levels are not reversed at the participating centers. Electronic medical records were reviewed in a standardized fashion by research coordinators and site investigators to assess international normalized ratio results, CT scan results, ED disposition, and hospital course. Patients admitted to the hospital for at least 14 days were evaluated for the presence of delayed traumatic intracranial hemorrhage through review of the electronic medical record. Patients discharged from the ED or admitted to the hospital for fewer than 14 days received a consented, standardized telephone survey at least 14 days after the index ED visit. The 14-day follow-up was deemed sufficient to identify clinically important delayed traumatic intracranial hemorrhage.<sup>8,18,19</sup> Repeated cranial imaging was obtained at the discretion of the patients' treating physicians. If patients were unable to be contacted by telephone survey or the electronic medical record, the Social Security Death Index was reviewed to evaluate for death.<sup>20</sup>

### Primary Data Analysis

Data were compared with Stata for Windows (version 10.0; StataCorp, College Station, TX). Normally distributed continuous data were reported as the mean (SD), and ordinal or non-normally distributed continuous data were described as the median with interquartile ranges (25% to 75%). For primary, stratified, and sensitivity analyses, proportions and relative risks were presented with 95% confidence intervals (CIs). Categorical data were compared with

$\chi^2$  test or Fisher's exact test in cases of small cell size. Interrater reliability of independent variables recorded by initial and second physicians was reported as percentage of agreement.

To ensure that differences in outcome between cohorts were not a result of differences in injury severity, we performed both stratified and sensitivity analyses. We compared the following strata: patients aged 65 years or older, patients with minor head injury (GCS scores 13 to 15), patients with an initial GCS score of 15, patients with a ground-level fall, patients with physical evidence of trauma above the clavicles, patients without concomitant aspirin use, and patients evaluated at a community hospital. In addition, we stratified the analyses by degree of anticoagulation (international normalized ratio  $\geq 1.3$  and  $\geq 2.0$ ). Sensitivity analyses were conducted assuming those patients without an initial cranial CT had immediate traumatic intracranial hemorrhage and did not have traumatic intracranial hemorrhage. Finally, we compared the cumulative incidence of delayed traumatic intracranial hemorrhage, assuming all patients lost to follow-up had a delayed traumatic intracranial hemorrhage.

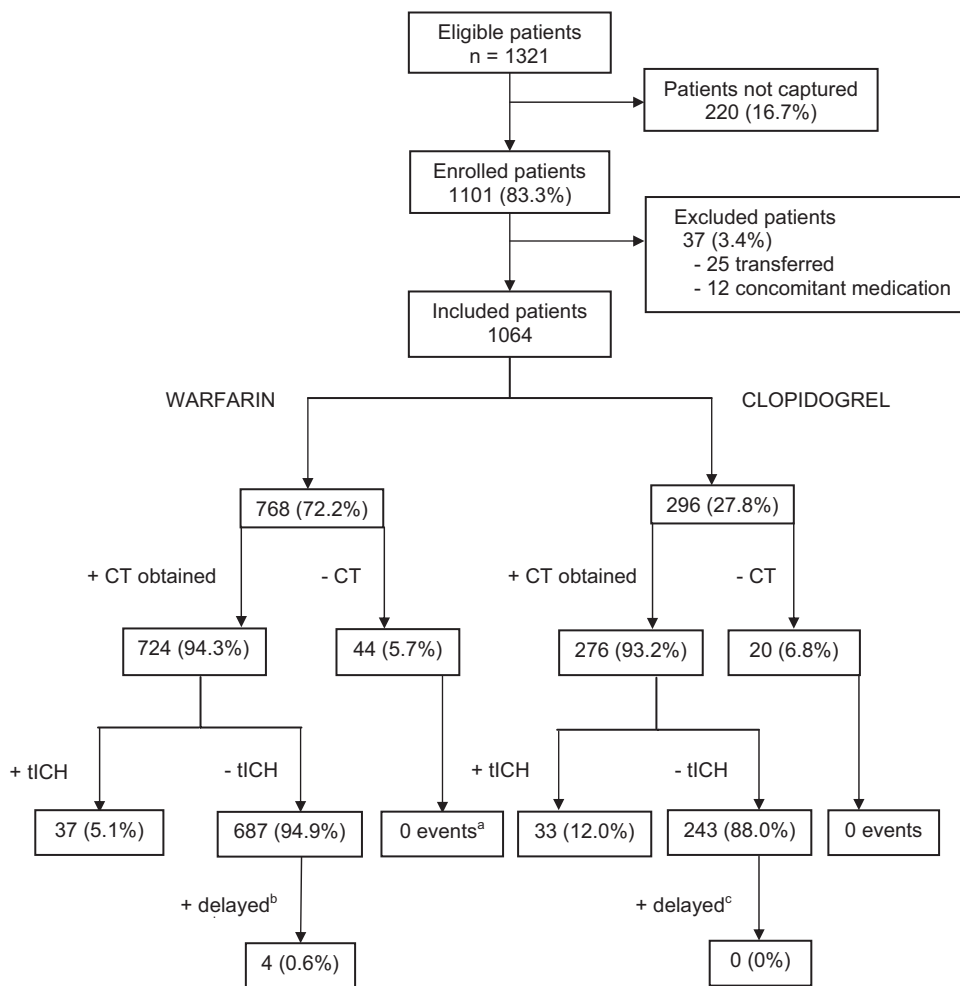
## RESULTS

### Characteristics of Study Subjects

Between April 2009 and January 2011, 1,101 patients were enrolled (83.3% of all eligible patients) (Figure). Comparison of patients enrolled and those eligible but not enrolled demonstrated similar characteristics (age, sex, medication use, ED cranial CT, and hospital admission) and outcomes (immediate traumatic intracranial hemorrhage, neurosurgical intervention, and inhospital mortality). Reasons for failures of the study screening process were unknown. Thirty-seven patients were excluded (25 transferred patients and 12 patients with concomitant clopidogrel and warfarin use), leaving 1,064 patients for data analysis. Of the 1,064 patients, 768 patients (72.2%) were receiving warfarin and 296 patients (27.8%) were receiving clopidogrel. There were 364 patients (34.2%) from 2 designated Level I or II trauma centers and 700 patients (65.8%) from 4 community hospitals. The most common mechanism of injury was a ground-level fall ( $n=887$ ; 83.3%) followed by direct blow ( $n=59$ ; 5.6%) and motor vehicle crash ( $n=51$ ; 4.8%).

The majority ( $n=932$ ; 87.6%) of patients had a GCS score of 15, and 752 (70.7%) patients had physical examination findings of head trauma above the clavicles. The primary indication for warfarin and clopidogrel use was atrial fibrillation (543/768; 70.7%) and coronary artery disease (158/296; 53.4%), respectively. Most patients reported receiving their medication less than 24 hours before injury (warfarin group 660/768, 85.9%; clopidogrel group 252/296, 85.1%). In patients receiving warfarin, 603 of 768 (78.5%) had an international normalized ratio measurement on initial evaluation in the ED (median international normalized ratio 2.5; interquartile range 2.0 to 3.3). The majority of these patients (576/603; 95.5%) had an elevated international normalized ratio ( $\geq 1.3$ ), and 458 of 603 (76.0%) had an international normalized ratio ( $\geq 2.0$ ).

One thousand of the 1,064 (94.0%; 95% CI 92.4% to 95.3%) received a cranial CT during initial ED evaluation. Hospitalization rates were similar for patients receiving warfarin (271/768; 35.3%) and clopidogrel (93/296; 31.4%). Patient clinical characteristics were similar in both groups, except for headache, concomitant aspirin use,



Abbreviations: CT, computed tomography; tICH, traumatic intracranial hemorrhage

<sup>a</sup> One patient lost to follow-up

<sup>b</sup> Two patients lost to follow-up

<sup>c</sup> Two patients lost to follow-up and one patient died after discharge from the emergency department

**Figure.** Flow of patients in the study.

and evidence of trauma to the neck and scalp laceration, which were more common in the clopidogrel group (Table 1).

### Main Results

Seventy of the 1,000 patients had immediate traumatic intracranial hemorrhage on ED CT scan. The prevalence of immediate traumatic intracranial hemorrhage was higher in patients receiving clopidogrel (33/276; 12.0%; 95% CI 8.4% to 16.4%) than warfarin (37/724, 5.1%, 95% CI 3.6% to 7.0%; relative risk=2.31, 95% CI 1.48 to 3.63;  $P<.001$ ) (Table 2). Follow-up was obtained for 63 of 64 of patients not undergoing cranial CT during initial ED evaluation, and none subsequently received a diagnosis of traumatic intracranial hemorrhage. Mortality and neurosurgical intervention rates after immediate traumatic intracranial hemorrhage were not statistically different between cohorts (Table 2).

The majority of patients with immediate traumatic intracranial hemorrhage (45/70; 64.3%) had a normal mental status (GCS score=15), with similar proportions between the warfarin (23/37; 62.2%) and clopidogrel (22/33; 66.7%) cohorts. Furthermore, in patients with immediate traumatic intracranial hemorrhage, 4 of 37 (10.8%) in the warfarin cohort and 6 of 33 (18.2%) in the clopidogrel cohort had no loss of consciousness, a normal mental status, and no evidence of trauma above the clavicles.

The prevalence of immediate traumatic intracranial hemorrhage varied by participating center. The prevalence of traumatic intracranial hemorrhage was highest at the Level I trauma center (12.6%; 95% CI 8.1% to 18.3%) compared with the Level II trauma center (5.0%; 95% CI 2.3% to 9.2%) and the 4 community centers (5.4%; 95% CI 3.9% to 7.4%). All clinical

**Table 1.** Demographic and clinical characteristics of the study population.

Characteristic	Patients, No. (%)		
	Total (n=1,064)	Warfarin (n=768)	Clopidogrel (n=296)
<b>Demographics</b>			
Age, mean (SD), y	75.4 (12.7)	75.3 (13.0)	75.7 (11.9)
Male sex	502 (47.1)	362 (47.1)	140 (47.3)
<b>Mechanism of injury</b>			
Ground-level fall	887 (83.3)	644 (83.9)	243 (82.1)
Fall from height	37 (3.5)	23 (3.0)	14 (4.7)
MVC, <35 miles/h	18 (1.7)	12 (1.6)	6 (2.0)
MVC, ≥35 miles/h	24 (2.3)	16 (2.1)	8 (2.7)
MVC, unknown speed	9 (0.8)	4 (0.5)	5 (1.7)
Pedestrian struck by automobile	4 (0.4)	4 (0.5)	0
Bicyclist struck by automobile	4 (0.4)	3 (0.4)	1 (0.3)
Direct blow	59 (5.6)	45 (5.9)	14 (4.7)
Unknown mechanism	16 (1.5)	13 (1.7)	3 (1.0)
Other mechanism	6 (0.5)	4 (0.5)	2 (0.7)
<b>Clinical history</b>			
Vomiting	45 (4.2)	34 (4.4)	11 (3.7)
Headache	357 (33.6)	239 (31.1)	118 (39.9)
Loss of consciousness or amnesia	196 (18.4)	136 (17.7)	60 (20.3)
Concomitant aspirin use	43 (4.0)	19 (2.5)	24 (8.1)
<b>Physical examination</b>			
Alcohol intoxication	33 (3.1)	26 (3.4)	7 (2.4)
<b>Any evidence of trauma above the clavicles</b>			
Trauma to face	406 (38.2)	296 (38.5)	110 (37.2)
Trauma to neck	36 (3.4)	20 (2.6)	16 (5.4)
Basilar skull fracture	2 (0.2)	1 (0.1)	1 (0.3)
Scalp abrasion	157 (14.8)	110 (14.3)	47 (15.9)
Scalp contusion	309 (29.0)	221 (28.8)	88 (29.7)
Scalp laceration	182 (17.1)	117 (15.2)	65 (22.0)
Normal mental status (GCS score 15)	932 (87.6)	674 (87.8)	258 (87.2)
Mild head injury (GCS score 13–15)	1035 (97.3)	747 (97.3)	288 (97.3)
Moderate head injury (GCS score 9–12)	18 (1.7)	13 (1.7)	5 (1.7)
Severe head injury (GCS score 3–8)	11 (1.0)	8 (1.0)	3 (1.0)
<b>ED course</b>			
Initial cranial CT	1000 (94.0)	724 (94.3)	276 (93.3)
Admitted to hospital	364 (34.2)	271 (35.3)	93 (31.4)

MVC, Motor vehicle crash.

variables measured for interrater reliability had substantial agreement (range 87% to 100%).<sup>21</sup>

The cumulative incidence of delayed traumatic intracranial hemorrhage was assessed in the 930 patients with an initial normal cranial CT scan by telephone survey (843; 90.6%) or electronic medical record review (83; 8.9%). Of the 4 patients lost to follow-up, none was identified in the Social Security Death Index.

Delayed traumatic intracranial hemorrhage was identified in 4 of 687 (0.6%; 95% CI 0.2% to 1.5%) patients receiving warfarin and 0 of 243 (0%; 95% CI 0% to 1.5%) patients receiving

clopidogrel (Figure). Two of these 4 patients were deemed nonoperable and died from extensive traumatic intracranial hemorrhage. The characteristics of the 4 patients who experienced a delayed traumatic intracranial hemorrhage are represented in Table 3. One additional patient receiving clopidogrel died at home from unknown causes 8 days after initial ED visit and did not present to hospital at time of death.

**Sensitivity Analyses**

We performed both stratified and sensitivity analyses to assess the validity of our results (Table 4). The stratified analyses confirm an increased risk of immediate traumatic intracranial hemorrhage in those patients receiving clopidogrel compared with warfarin across all strata. Likewise, the sensitivity analyses also confirm the increased risk of traumatic intracranial hemorrhage in patients receiving clopidogrel.

The final sensitivity analysis assessed the 4 patients lost to follow-up and the 1 death from unknown causes. Assuming all patients had a delayed traumatic intracranial hemorrhage, its cumulative incidence would increase to 6 of 687 patients (0.9%; 95% CI 0.3% to 1.9%) in the warfarin group and 3 of 243 (1.2%; 95% CI 0.3% to 3.6%) in the clopidogrel group.

**LIMITATIONS**

Our results should be interpreted in the context of several limitations. This was an observational study; thus, CT scans were not obtained for all patients and ethical considerations prevented CT scanning solely for study purposes. Some patients not undergoing CT scan during initial ED visit potentially had an undiagnosed traumatic intracranial hemorrhage, although none was identified in follow-up. Furthermore, some patients with a negative initial CT scan result may have eventually developed an undiagnosed delayed traumatic intracranial hemorrhage. We did, however, obtain clinical follow-up, which is a reasonable method to evaluate for clinically important outcomes when the definitive test is not ethical or feasible.<sup>22</sup> The increased risk of immediate traumatic intracranial hemorrhage in the clopidogrel cohort may be attributed to the higher prevalence of concomitant aspirin use compared with the warfarin cohort (8.1% versus 2.5%). However, we conducted a subgroup analysis excluding patients with concomitant aspirin use, and the clopidogrel cohort maintained a significant increased risk for immediate traumatic intracranial hemorrhage compared with the warfarin cohort. We did not collect data on patients with isolated preinjury aspirin use<sup>23</sup> or patients without preinjury antiplatelet or anticoagulation use. Finally, patients receiving warfarin may be more acutely aware of the bleeding risks associated with their medication than those receiving clopidogrel. Therefore, patients receiving warfarin may be more apt to seek emergency care, even with trivial head trauma, and thus have less severe mechanisms of injury compared with patients receiving clopidogrel. We were unable, however, to identify such behavior because the clinical characteristics, mechanism of injury, and CT scan rate were similar overall between the warfarin and clopidogrel groups.

**Table 2.** Prevalence of traumatic intracranial hemorrhage, neurosurgical intervention, and mortality.

Outcome Measures	Patients, No. (%) [95% CI]			Differences in Proportions, % (95% CI)
	Total (n=1,064)	Warfarin (n=768)	Clopidogrel (n=296)	
Immediate tICH* <sup>†</sup>	70/1,000 (7.0) [5.5 to 8.8]	37/724 (5.1) [3.6 to 7.0]	33/276 (12.0) [8.4 to 16.4]	6.8 (2.7 to 11.0)
Inhospital mortality after immediate tICH	15/70 (21.4) [12.5 to 32.9]	8/37 (21.6) [9.8 to 38.2]	7/33 (21.2) [9.0 to 38.9]	-0.4 (-19.7 to 18.8)
Neurosurgical intervention after immediate tICH	12/70 (17.1) [9.2 to 28.0]	5/37 (13.5) [4.5 to 28.8]	7/33 (21.2) [9.0 to 38.9]	7.6 (-10.1 to 25.5)
Delayed tICH <sup>‡§</sup>	4/930 (0.4) [0.1 to 1.1]	4/687 (0.6) [0.2 to 1.5]	0/243 (0.0) [0.0 to 1.5]	-0.6 (-1.1 to 0.0)

\*Immediate tICH is defined as the presence of tICH on initial cranial CT.  
<sup>†</sup>Sixty-four patients did not receive initial cranial CT.  
<sup>‡</sup>Delayed tICH is defined as the presence of tICH on cranial CT or autopsy after negative initial cranial CT result without new head trauma.  
<sup>§</sup>Four patients were lost to follow-up (2 warfarin and 2 clopidogrel) and 1 patient died after discharge from the ED (clopidogrel).

**Table 3.** Patients with delayed traumatic intracranial hemorrhage (all with preinjury warfarin use).

Patient Sex and Age, Years	Mechanism of Injury	Initial GCS Score	Initial INR	Repeated Cranial CT Findings (Days After Initial Cranial CT)	Neurosurgical Intervention/ Inhospital Mortality (Days After Initial Cranial CT)		Comments
Woman, 63	Ground-level fall, isolated head injury	15	1.15	Massive subdural hematoma with uncal herniation (3)	Mannitol/died (3)		Patient was discharged home from initial ED visit. She was found obtunded at home 3 days later. She was taken immediately to the ED and died in the hospital the same day.
Man, 63	Ground-level fall, isolated head injury	15	1.50	Small intraparenchymal contusion and subarachnoid hemorrhage (1)	No/no		Patient was admitted to the hospital. Routine repeated cranial CT showed a small tICH. Discharged home HD 4.
Man, 79	Ground-level fall, isolated head injury	15	4.95	Small intraventricular hemorrhage (7)	No/no		Patient was admitted to the hospital. Repeated cranial CT obtained for a change in mental status on HD 7. Patient improved and was discharged home on HD 8.
Man, 91	Ground-level fall, isolated head injury	15	1.90	Large intraparenchymal, subarachnoid, and intraventricular hemorrhage with midline shift of 9.3 mm (3)	No/died (7)		Patient was admitted to the hospital. On HD 3, repeated cranial CT obtained for a change in mental status demonstrated a large tICH, and patient was made DNR. Died on HD 7.

INR, International normalized ratio; HD, hospital day; DNR, do not resuscitate.

## DISCUSSION

Contrary to our hypothesis, the prevalence of immediate traumatic intracranial hemorrhage in patients with clopidogrel was significantly higher compared with those receiving warfarin despite the cohorts' having similar characteristics. Additionally, we determined in a large and generalizable cohort of patients receiving warfarin or clopidogrel that the development of a delayed traumatic intracranial hemorrhage after a negative initial cranial CT scan result is rare and does not warrant routine hospitalization for observation or immediate anticoagulation reversal with blood products.

To our knowledge, this is the first large, prospective study of head-injured patients with preinjury warfarin or clopidogrel use. We identified 10 warfarin and 3 clopidogrel studies that reported a prevalence of immediate traumatic intracranial

hemorrhage.<sup>4,6,24-32</sup> The prevalence for immediate traumatic intracranial hemorrhage in patients with preinjury warfarin use ranged from 0% to 65%.<sup>4,24-32</sup> The 3 studies evaluating immediate traumatic intracranial hemorrhage in patients with preinjury clopidogrel use demonstrated a prevalence of traumatic intracranial hemorrhage ranging from 36% to 71%.<sup>6,25,26</sup> The overall quality of these studies, however, was limited because the majority were small (<100 patients), retrospective registry studies. These studies suffered from significant inclusion bias because the sampled population originated from a trauma registry (patients admitted to a trauma center) and excluded not only patients evaluated and discharged from the trauma center ED but also all patients evaluated at community hospitals. In addition, the prevalence of traumatic intracranial hemorrhage was likely falsely elevated because of the

**Table 4.** Stratified and sensitivity analyses for immediate traumatic intracranial hemorrhage.

Analyses*	Patients, No. (%) [95% CI]		Differences in Proportions, % (95% CI)	Relative Risk (95% CI)
	Warfarin (n=768)	Clopidogrel (n=296)		
Primary analysis	37/724 (5.1) [3.6 to 7.0]	33/276 (12.0) [8.4 to 16.3]	6.8 (2.7 to 11.0)	2.31 (1.48 to 3.63)
Patients 65 y or older	33/594 (5.6) [3.9 to 7.7]	24/217 (11.1) [7.2 to 16.0]	5.5 (3.7 to 7.4)	1.99 (1.20 to 3.29)
Patients with GCS score 13–15	30/703 (4.3) [2.9 to 6.0]	29/268 (10.8) [7.4 to 15.2]	6.6 (2.5 to 10.6)	2.54 (1.55 to 4.14)
Patients with GCS score 15	23/631 (3.6) [2.3 to 5.4]	22/239 (9.2) [5.9 to 13.6]	5.6 (2.2 to 9.5)	2.53 (1.44 to 4.44)
Patients with ground-level fall	30/608 (4.9) [3.4 to 7.0]	27/225 (12.0) [8.1 to 17.0]	7.1 (2.5 to 11.6)	2.43 (1.48 to 4.00)
Patients with evidence of trauma above the clavicles	29/502 (5.7) [3.9 to 8.2]	21/205 (10.2) [6.4 to 15.2]	4.5 (–0.2 to 9.1)	1.77 (1.04 to 3.04)
Patients without concomitant aspirin use	36/705 (5.1) [3.6 to 7.0]	29/252 (11.5) [7.8 to 16.1]	6.4 (2.1 to 10.7)	2.25 (1.41 to 3.60)
Patients evaluated at community hospitals	21/485 (4.3) [2.7 to 6.5]	17/161 (10.6) [6.3 to 16.4]	6.2 (1.1 to 11.3)	2.44 (1.32 to 4.51)
Warfarin patients with INR $\geq$ 1.3	35/556 (6.3) [4.4 to 8.6]	33/276 (12.0) [8.4 to 16.3]	5.7 (1.3 to 10.0)	1.90 (1.21 to 2.99)
Warfarin patients with INR $\geq$ 2.0	31/441 (7.0) [4.8 to 9.8]	33/276 (12.0) [8.4 to 16.3]	4.9 (0.4 to 9.4)	1.70 (1.07 to 2.71)
Assume patients without cranial CT imaging did not have immediate tICH	37/768 (4.8) [3.4 to 6.6]	33/296 (11.1) [7.8 to 15.3]	6.3 (2.4 to 10.2)	2.31 (1.48 to 3.63)
Assume patients without cranial CT imaging had immediate tICH	81/768 (10.5) [8.5 to 12.9]	53/296 (17.9) [13.7 to 22.8]	7.4 (2.5 to 12.2)	1.70 (1.23 to 2.34)

\*Based on patients who received a cranial CT scan on initial evaluation after head injury.

inclusion of patients transferred to a trauma center. Our study is unique in that a majority of patients were evaluated at community hospitals. Furthermore, we included all patients with any degree of head trauma. Thus, the current study identifies the prevalence of traumatic intracranial hemorrhage in a more generalizable population than those sampled from trauma registries. Numerous case reports and case series described delayed traumatic intracranial hemorrhage, though to our knowledge no previous study evaluated the cumulative incidence of delayed traumatic intracranial hemorrhage.<sup>7-9</sup>

Current guidelines recommend that patients with head trauma and preinjury warfarin undergo routine cranial CT imaging.<sup>11-14</sup> These recommendations are based on theoretical risk and retrospective data because large, prospective studies excluded anticoagulated patients or did not specifically study patients receiving warfarin.<sup>33-36</sup> Despite the lower prevalence of traumatic intracranial hemorrhage in this study, the results confirm the substantial risk of traumatic intracranial hemorrhage in patients with blunt head trauma who are receiving warfarin and the benefit of routine cranial CT imaging, even in community hospitals. Previous guidelines, however, do not consider preinjury clopidogrel an indication for cranial imaging,<sup>11-13</sup> despite retrospective data suggesting an increased risk for traumatic intracranial hemorrhage.<sup>6,16,17</sup> The current results indicate that the approach to the head-injured patient with preinjury clopidogrel should be similar to that for the head-injured patient with preinjury warfarin use: liberal cranial imaging. Because delayed diagnosis of traumatic intracranial hemorrhage increases morbidity and mortality, early diagnosis of traumatic intracranial hemorrhage is important to initiate treatment, including coagulopathy reversal or neurosurgical intervention.<sup>37-39</sup>

The prevalence of immediate traumatic intracranial hemorrhage in well-appearing patients is also very concerning. More than 60% of patients with immediate traumatic intracranial hemorrhage in both warfarin and clopidogrel cohorts had a normal mental status (GCS score=15). Additionally, a significant proportion of patients (11% in the warfarin cohort and 18% in the clopidogrel cohort) had no loss of consciousness, a normal mental status, and no physical evidence of trauma above the clavicles. Current National Institute for Health and Clinical Excellence head injury guidelines (updated 2007) recommend urgent (<1 hour) CT imaging in patients with head injury and preinjury warfarin use, provided they sustain loss of consciousness or amnesia.<sup>12</sup> In our study, 49 of 70 (70%) patients with immediate traumatic intracranial hemorrhage did not sustain loss of consciousness or amnesia. We recommend routine urgent CT imaging in head-injured patients with previous warfarin or clopidogrel use, even in well-appearing patients without a history of loss of consciousness or amnesia.

The concern for delayed traumatic intracranial hemorrhage in patients with warfarin use stems from case reports and case series,<sup>7-9</sup> leading guidelines to recommend routine admission for all head-injured patients receiving warfarin despite a normal cranial CT scan result.<sup>14</sup> Moreover, a survey of clinical practices among North American trauma surgeons indicated that 74% of respondents reverse patients receiving warfarin who have blunt head trauma despite a normal cranial CT scan result.<sup>10</sup> Furthermore, 66% of respondents reverse these patients with fresh frozen plasma.<sup>10</sup> Our results indicate that delayed traumatic intracranial hemorrhage occurs infrequently (<1%) in both populations. Thus, patients receiving warfarin or clopidogrel who have a normal cranial CT scan result and no other indications for admission may be discharged home, albeit

with explicit discharge instructions and close follow-up. More important, these patients do not need to have their therapeutic anticoagulation aggressively reversed with blood products. In patients with supratherapeutic international normalized ratio levels, we recommend appropriate medical treatment following current guidelines.<sup>40</sup>

In summary, ED patients with blunt head trauma and preinjury clopidogrel use have a significantly higher prevalence of immediate traumatic intracranial hemorrhage compared with those with preinjury warfarin use. Routine cranial CT imaging is generally indicated in patients with blunt head trauma who are receiving clopidogrel or warfarin, regardless of the clinical findings. The cumulative incidence of delayed traumatic intracranial hemorrhage is very low for both groups, suggesting that in patients with a normal cranial CT scan result, anticoagulation reversal is unnecessary and discharging them home from the ED may be reasonable. Because delayed traumatic intracranial hemorrhage may rarely occur, routine follow-up and appropriate discharge instructions are necessary.

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obtained from <http://nihroadmap.nih.gov/clinicalresearch/overview-translational.asp>.

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## IMAGES IN EMERGENCY MEDICINE

*(continued from p. 456)*

### DIAGNOSIS:

*Traumatic mydriasis with hyphema.* In blunt ocular trauma, the anterior chamber is compressed, forcefully dilating the pupil. This can injure the iris sphincter, dilator muscles, or nerves of the ciliary plexus to form a traumatic mydriasis, which manifests as a moderately dilated pupil with diminished accommodation and reactivity.<sup>1</sup> Visual acuity can be normal or impaired, and intraocular pressure may range from low to high.<sup>2</sup> Treatment with ophthalmic cycloplegics is typically initially used to relax the iris and ciliary body in this often permanent defect.<sup>1,2</sup>

Like traumatic mydriasis, hyphemas may herald significant intraocular injuries. This patient was found to have an associated cyclodialysis—a tear between the uveal tissue and sclera commonly caused by blunt ocular trauma, which allows additional flow of aqueous humor from the anterior segment into the suprachoroidal space.<sup>3</sup>

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APPENDIX E1. Data collection form for the emergency department.

**Coumadin/Plavix Head Trauma Study** v.7.30.09

Patient Stamp Here:

---

If no patient stamp:

NAME: \_\_\_\_\_

MRN: \_\_\_\_\_

Trauma Code:  911  922  933  no code

Date of ED arrival: \_\_/\_\_/\_\_ Time of arrival: \_\_\_\_\_  AM  PM

Date of injury: \_\_/\_\_/\_\_ Time of injury: \_\_\_\_\_  AM  PM  Unknown date and time of injury

Patient contact phone numbers (please check in EMR and patient to confirm contact info):

Verified  Not verified  If no number in EMR → phone no. ( ) \_\_\_\_\_  
 phone no. ( ) \_\_\_\_\_

Mode of arrival:

Private car  Ambulance

Transfer from outside hospital/ED?

Yes  No if yes → date/time arrived at outside ED \_\_/\_\_/\_\_ \_\_:\_\_

Reported Med(s):

warfarin/coumadin  
 clopidogrel/plavix  
 aspirin

Last dose?

within 24 hrs  24-48 hrs  48-72 hrs  3-7 days  >7 days  unknown  
 within 24 hrs  24-48 hrs  48-72 hrs  3-7 days  >7 days  unknown  
 within 24 hrs  24-48 hrs  48-72 hrs  3-7 days  >7 days  unknown

Why is patient taking coumadin/warfarin?

atrial fibrillation (heart arrhythmia)  
 DVT or PE  
 heart valve replacement  
 in-dwelling catheter  
 other \_\_\_\_\_  
 unknown

Why is patient taking plavix/clopidogrel?

coronary artery disease (CAD)  
 stroke (CVA)  
 peripheral artery disease (PAD)  
 other \_\_\_\_\_  
 unknown

Mechanism of injury:

fall from standing height or less  
 fall from greater than standing height (ex. ladder or stairs)  
 direct blow to head (ex. assault, hit head on table, etc)  
 unknown mechanism of injury  
 MVA > 35 MPH  
 MVA ≤ 35 MPH  
 pedestrian vs. auto  
 bicyclist vs. auto  
 other mechanism \_\_\_\_\_

Evidence of head trauma (trauma above the clavicles)?

Yes (complete below)  No (skip to GCS question)

1. Is there trauma to face?  Yes  No

2. Is there trauma to neck?  Yes  No

3. Is there trauma to scalp (from above eyebrows to the occiput)?

depressed skull fracture  signs of basilar skull fracture  abrasion  none  
 contusion/hematoma  laceration  other \_\_\_\_\_

Initial GCS in ED (please fill out)  GCS 15 (If Less than 15, complete below)

Eye	Verbal	Motor
<input type="checkbox"/> 4 Spontaneous	<input type="checkbox"/> 5 Oriented	<input type="checkbox"/> 6 Follow commands
<input type="checkbox"/> 3 Verbal	<input type="checkbox"/> 4 Confused	<input type="checkbox"/> 5 Localizes pain
<input type="checkbox"/> 2 Pain	<input type="checkbox"/> 3 Inappropriate words	<input type="checkbox"/> 4 Withdraws to pain
<input type="checkbox"/> 1 None	<input type="checkbox"/> 2 Incomprehensible sounds	<input type="checkbox"/> 3 Abnormal flexure posturing
	<input type="checkbox"/> 1 None	<input type="checkbox"/> 2 Abnormal extension posturing
		<input type="checkbox"/> 1 None

1. Has the patient vomited?  Yes  No  Unknown

2. Does the patient have a headache?  Yes  No  Unknown

3. Did (or does) the patient have amnesia or loss of consciousness?  Yes  No  Unknown

4. If GCS is < 15 and the patient has dementia, do you think the dementia is the sole cause of the abnormal GCS?

N/A (GCS 15)  Yes, abnormal GCS is caused by dementia alone  No, abnormal GCS is due to injury  Unknown

5. Is patient clinically intoxicated?  Yes  No  Unknown

**GESTALT BOX (PLEASE HAVE ANY OF FOLLOWING FILL OUT):**  EM Attending  EM R3  EM R2

1. Clinical suspicion for the presence of intracranial hemorrhage on CT (regardless of whether a CT was obtained):

<1%  1-5%  6-10%  11-50%  >50%

2. Clinical suspicion for intracranial hemorrhage requiring neurosurgery (regardless of whether a CT was obtained):

<1%  1-5%  6-10%  11-50%  >50%

3. Were these two questions completed prior to knowledge of CT results?

Yes  No

**PLEASE PLACE COMPLETED FORM IN MARKED FOLDER IN EMR ROOM!!!**

APPENDIX E2. Data collection form for follow-up.

Study ID: \_\_\_\_\_

MRN: \_\_\_\_\_

Name of Patient: \_\_\_\_\_

Disposition from ED:  home/care facility  ICU  floor/telemetry  AMA  Transfer

immtich  delayedich  died2wksdcED  died2wksdcall  all mort

**Directions:**  
 For patients discharged from ED or admitted to hospital: Fill out page 1-2  
 For patients with intracranial hemorrhage: Fill out all pages

\*\*\*ND= not documented\*\*\*

**Patient information:**

Age (at time of arrival): \_\_\_\_\_  ND Gender:  Male  Female

DC date from hospital: \_\_\_ / \_\_\_ / \_\_\_  ND  DC home LOS: \_\_\_\_\_ Days

**Initial CT HEAD: Date:** \_\_\_ / \_\_\_ / \_\_\_\_\_

- may be from outside hospital

initial CT from outside hospital  initial CT not from outside hospital

normal head CT

abnormal head CT

Skull fracture:  unknown or none  yes  
 SDH:  unknown or none  yes  
 EDH:  unknown or none  yes  
 IPH:  unknown or none  yes  
 IVH:  unknown or none  yes  
 SAH:  unknown or none  yes  
 Shift:  unknown or none  yes  
 Herniation:  unknown or none  yes

*1 = normal CT, 2 = no mass > 25 cc, normal cisterns, no shift, 3 = no mass > 25 cc, absent or compressed cisterns, no shift > 5 mm; 4 = same as 3 but with shift > 5 mm, 5 = + mass > 25 cc with surgical evacuation; 6 = + mass > 25 cc without surgical evacuation*

Marshall score (1-6): \_\_\_\_\_

**CT head #2**  unknown or none  yes **Date:** \_\_\_ / \_\_\_ / \_\_\_\_\_

Repeat CT:  same  less (better)  increase (worse)

RepeatCT head:  normal  abnormal

Skull fracture:  unknown or none  yes  
 SDH:  unknown or none  yes  
 EDH:  unknown or none  yes  
 IPH:  unknown or none  yes  
 IVH:  unknown or none  yes  
 SAH:  unknown or none  yes  
 Shift:  unknown or none  yes  
 Herniation:  unknown or none  yes

Marshall score (1-6) \_\_\_\_\_

**LABS:**

Platelet count (initial, may be from outside hospital): \_\_\_\_\_ per microliter  ND

INR level (initial, may be from outside hospital): \_\_\_\_\_ per microliter  ND

Study ID: \_\_\_\_\_

**Follow up phone call***- at least 2 weeks after seen in ED; if has been in-hospital for 2 weeks no need for phone call (fill out "Results")* dc home from ED    dc home from hospital < 14 days    dc home from hospital ≥ 14 days**Consent:**Patient contacted    yes    noFamily contacted    yes    noEMR contact    yes    no

Number of attempts: \_\_\_\_\_

Date of phone call: \_\_\_\_/\_\_\_\_/\_\_\_\_

Patient/family consent    yes    noIf consent declined, reason: \_\_\_\_\_   Unable to contact    yes    no**Symptoms:**post injury problems    yes    noheadache    yes    nonausea/vomiting    yes    nodizziness    yes    noweakness    yes    nopassing out    yes    no

other \_\_\_\_\_

still taking medication (warfarin or plavix)?    yes    no    unknown**Follow up:**seen by PMD    yes    no

date of PMD \_\_\_\_/\_\_\_\_/\_\_\_\_

seen in ED    yes    no

date of ED \_\_\_\_/\_\_\_\_/\_\_\_\_

name of ED \_\_\_\_\_

reason for visit to ED \_\_\_\_\_

admitted to hospital    yes    no

date hosp \_\_\_\_/\_\_\_\_/\_\_\_\_

reason for admission \_\_\_\_\_

name of hospital \_\_\_\_\_

days admitted \_\_\_\_\_

**Results:**CT head repeated    yes    noRepeat CT head:    normal    abnormalSkull fracture:    unknown or none    yesSDH:    unknown or none    yesEDH:    unknown or none    yesIPH:    unknown or none    yesIVH:    unknown or none    yesSAH:    unknown or none    yesShift:    unknown or none    yesHerniation:    unknown or none    yes

Marshall score: \_\_\_\_\_

death of patient    yes    noneurosurgery done    yes    no

type of NS \_\_\_\_\_   Date of NS \_\_\_\_/\_\_\_\_/\_\_\_\_

Study ID: \_\_\_\_\_

**Patients with traumatic ICH: Fill out below**

**Isolated head:**  yes  no

*AIS score: 1 = minor, 2 = moderate, 3 = serious (non-life threatening), 4 = severe (life threatening, survival probable), 5 = critical (survival uncertain), 6 = unsurvivable*

AIS head and neck (0-6): \_\_\_\_\_

AIS face (0-6): \_\_\_\_\_

AIS chest (0-6): \_\_\_\_\_

AIS abdomen (0-6): \_\_\_\_\_

AIS extremities (0-6): \_\_\_\_\_

AIS external (0-6): \_\_\_\_\_

ISS (0-75): \_\_\_\_\_

**LABS:**

INR level (1<sup>st</sup>) even if out of hospital: \_\_\_\_\_ D/T INR level: \_\_\_\_/\_\_\_\_/\_\_\_\_ \_\_\_\_:\_\_\_\_  ND

INR level (2<sup>nd</sup>): \_\_\_\_\_ D/T INR level: \_\_\_\_/\_\_\_\_/\_\_\_\_ \_\_\_\_:\_\_\_\_  ND \_\_\_\_\_ min from initial

INR level (3<sup>rd</sup>): \_\_\_\_\_ D/T INR level: \_\_\_\_/\_\_\_\_/\_\_\_\_ \_\_\_\_:\_\_\_\_  ND \_\_\_\_\_ min from initial

INR level (4<sup>th</sup>): \_\_\_\_\_ D/T INR level: \_\_\_\_/\_\_\_\_/\_\_\_\_ \_\_\_\_:\_\_\_\_  ND \_\_\_\_\_ min from initial

INR level (5<sup>th</sup>): \_\_\_\_\_ D/T INR level: \_\_\_\_/\_\_\_\_/\_\_\_\_ \_\_\_\_:\_\_\_\_  ND \_\_\_\_\_ min from initial

**TREATMENT:**

PRBCs:  no or unknown  received RBC during first 48 hours \_\_\_\_\_ units of PRBCs in 1<sup>st</sup> 48 hours  ND

FFP:  no or unknown  received FFP during first 48 hours \_\_\_\_\_ units of FFP in 1<sup>st</sup> 48 hours  ND

Vit K:  no or unknown  received Vit K during first 48 hours \_\_\_\_\_ mg of Vit K in 1<sup>st</sup> 48 hours  ND

FVIIa:  no or unknown  received FVIIa Weight (kg): \_\_\_\_\_  ND

FVIIa: \_\_\_\_\_ mcg of FVIIa in 1<sup>st</sup> 48 hours  ND

FVIIa: \_\_\_\_\_ mcg/kg of FVIIa in 1<sup>st</sup> 48 hours  ND

Time from arrival at UCDCMC to drug dosing: \_\_\_\_\_ minutes  ND

Study ID: \_\_\_\_\_

Prothrombin complex:  no or unknown  received PTC Weight (kg): \_\_\_\_\_  ND  
 PTC: \_\_\_\_\_ mcg of PTC in 1<sup>st</sup> 48 hours  ND  
 PTC: \_\_\_\_\_ mcg/kg of PTC in 1<sup>st</sup> 48 hours  ND  
 Time from arrival at UCDCM to drug dosing: \_\_\_\_\_ minutes  ND

**Neurosurgical intervention:**  unknown or none

yes

ICP monitor (bolt):  unknown or none  yes  
 Burr hole placed:  unknown or none  yes  
 Craniotomy done:  unknown or none  yes  
 Intraventricular catheter done:  unknown or none  yes  
 Subdural drain done:  unknown or none  yes  
 Use of mannitol for ICP:  unknown or none  yes  
 Use of hypertonic saline for ICP:  unknown or none  yes

Other neurosurgical intervention (describe): \_\_\_\_\_

Date and time of neurosurgical intervention: \_\_\_\_/\_\_\_\_/\_\_\_\_ \_\_\_\_:\_\_\_\_

If no exact date and time documented, give best estimate in minutes: \_\_\_\_\_ minutes

no documentation at all of time of neurosurgical intervention

## OUTCOMES:

Mortality:  yes  no

Mortality in ED:  unknown or none  yes or suspected

Mortality in 48 hours:  unknown or none  yes or suspected

Mortality in 30 days:  unknown or none  yes or suspected

Discharge home:  unknown or none  yes or suspected

Discharge to SNF (Skilled Nursing):  unknown or none  yes or suspected

Transfer to outside hospital:  unknown or none  yes or suspected

Length of ICU stay: \_\_\_\_\_ days  ND

TE (thromboembolism):  unknown or none  yes or suspected

ETT:  yes  no Days on mechanical ventilation (ETT): \_\_\_\_\_ days  ND

Discharge GCS (3-15): \_\_\_\_\_  ND

*1 = death 2 = persistent vegetative, minimal responsiveness, 3 = severe disability, conscious but disable, dependent for daily support, 4 = moderate disability, disabled but independent, can work in sheltered setting, 5 = good recovery, resumption of normal life despite minor deficits*

Glasgow outcome score at discharge, (1-5): \_\_\_\_\_  ND

APPENDIX E3. Data collection form for inter-rater reliability.

**Kappa Datasheet: Head Trauma + Coumadin/Plavix Study v.6.29.09**

Patient Stamp Here:

---

If No Patient Stamp:

NAME: \_\_\_\_\_

MRN: \_\_\_\_\_

**Location:**

UCDMC

Kaiser Sac

Kaiser SSC

Kaiser Ros

Kaiser SRF

Kaiser SSF

Kaiser RWC

**Initial GCS in ED(please fill out)**  GCS 15 (If Less than 15, complete below)

Eye	Verbal	Motor
<input type="checkbox"/> 4 Spontaneous	<input type="checkbox"/> 5 Oriented	<input type="checkbox"/> 6 Follow commands
<input type="checkbox"/> 3 Verbal	<input type="checkbox"/> 4 Confused	<input type="checkbox"/> 5 Localizes pain
<input type="checkbox"/> 2 Pain	<input type="checkbox"/> 3 Inappropriate words	<input type="checkbox"/> 4 Withdraws to pain
<input type="checkbox"/> 1 None	<input type="checkbox"/> 2 Incomprehensible sounds	<input type="checkbox"/> 3 Abnormal flexure posturing
	<input type="checkbox"/> 1 None	<input type="checkbox"/> 2 Abnormal extension posturing
		<input type="checkbox"/> 1 None

**Evidence of head trauma (trauma above the clavicles)?**  Yes (complete below)  No (skip to GCS question)

1. Is there trauma to face?  Yes  No

2. Is there trauma to neck?  Yes  No

3. Is there trauma to scalp (from above eyebrows to the occiput)? (if yes, fill out below)

depressed skull fracture       signs of basilar skull fracture       abrasion       none

contusion/hematoma       laceration       other \_\_\_\_\_

**Has the patient vomited?**  Yes  No  Unknown

**Does the patient have a headache?**  Yes  No  Unknown

**Did (or does) the patient have amnesia or loss of consciousness?**  Yes  No  Unknown

**If GCS is < 15 and the patient has dementia, do you think the dementia is the sole cause of the abnormal GCS?**

N/A (GCS 15)  Yes, abnormal GCS is caused by dementia alone  No, abnormal GCS is due to injury  Unknown

**Is patient clinically intoxicated?**  Yes  No  Unknown

**APPENDIX E4.** Data collection form for missed eligible patients.**Data sheet 4: Missed Eligibles****MRN:** \_\_\_\_\_ **Name of Patient:** \_\_\_\_\_ **MD:** \_\_\_\_\_**Location**

UC Davis   
 Kaiser North Sacramento   
 Kaiser South Sacramento   
 Kaiser Roseville   
 Kaiser San Rafael   
 Kaiser South San Francisco

**1. Patient information (Based on initial ED):**Age (at time of arrival): \_\_\_\_\_  ND Gender:  Male  FemaleDate of ED arrival: \_\_\_/\_\_\_/\_\_\_ Time of arrival: \_\_\_\_\_  AM  PMGCS \_\_\_\_\_  NDHistory of warfarin use:  unknown or none  yes or suspectedHistory of plavix use:  unknown or none  yes or suspectedHistory of aspirin use:  unknown or none  yes or suspected