Cranial computed tomographic findings in a large group of children with drowning: Diagnostic, prognostic, and forensic implications*

Karim T. Rafaat, MD; Robert M. Spear, MD; Cynthia Kuelbs, MD; Kourosh Parsapour, MD; Bradley Peterson, MD

Objectives: **The primary aim of this study is to better define both the type and incidence of cranial computed tomography (CT) abnormalities in children following submersion injury.**

Design: **This is a retrospective chart review; patients were selected from a drowning registry that extends from January 1989 to April 2006.**

Setting: **Children's Hospital, San Diego.**

Patients: **Patients were included if they were admitted to the hospital with a diagnosis of drowning and had a cranial CT within 24 hrs of submersion. Of 961 patients in the registry, 156 were included.**

Interventions: **None.**

Measurements and Main Results: **Eighteen percent (28 of 156) of children had an abnormal initial head CT, 82% (128 of 156) had a normal CT. Fifteen percent (24 of 156) of patients initially had a normal head CT and later had an abnormal CT. Abnormal CT findings were remarkable for diffuse loss of gray-white differentiation (75% on presentation) and bilateral basal ganglia edema/infarct (50% on presentation). There was no evidence of intra- or extra-axial blood nor were there any unilateral findings in any of the abnormal CTs. Presenting Glasgow Coma Scale was significantly lower in those who presented with an abnormal versus a normal head CT ($p < 0.001$). All patients with an abnormal initial CT presented with a Glasgow Coma Scale of 3, and all eventually died. Outcome was also very poor in those with a normal first CT and an abnormal second CT; 54% died and 42% remained in a persistent vegetative state.**

Conclusions: **These data from the largest study of CT findings in pediatric drowning clearly illustrate that following submersion injury, intra- or extra-axial bleeding is not seen on cranial CT. Furthermore, an abnormal CT scan at any time was associated with a poor outcome (death or persistent vegetative state). The CT findings and the presenting Glasgow Coma Scale of patients with drowning differ from those of patients who have suffered abusive head trauma. (Pediatr Crit Care Med 2008; 9:567–572)**

Key Words: near drowning; tomography; x-ray computed; Glasgow Outcome Scale; Glasgow Coma Scale; child abuse; hypoxia-ischemia; brain

The World Health Organization estimates that nearly 400,000 people die annually from drowning (1). Drowning is the second most common cause of accidental death in infancy and childhood, second only to motor vehicle accidents (2), and in survivors is associated with significant long-term neurologic morbidity (3–5).

No large studies exist that describe the cranial computed tomography (CT) findings in children with drowning; only a few, small retrospective reviews examine this subject (6, 7). The primary purpose of this study is to better define both the patterns and incidence of cranial CT abnormalities found in children following submersion injury. The secondary objective is to examine the relationship between injury variables, CT findings, and outcome. Additionally, a large, detailed study of CT findings after severe hypoxic-ischemic injury, such as occurs following drowning, may be useful to differentiate these patients from those who have suffered abusive head trauma (AHT).

MATERIALS AND METHODS

Patients. A database of 961 children evaluated at Rady's Children's Hospital, San Diego, with the diagnosis of drowning between January 1989 and April 2006, was screened for possible enrollment in this study. The study protocol was approved by the internal review board of Children's Hospital, San Diego. A waiver of consent was granted for this retrospective review of an internal registry.

Inclusion and Exclusion Criteria. Included were all patients admitted to the hospital with a diagnosis of drowning who had a cranial CT within 24 hrs of submersion injury. The diagnosis of drowning was based on an internally derived definition and includes patients with mild or clinically insignificant submersion times. Patients were excluded if they had known pre-existing CT abnormalities or if they presented with evidence of trauma. A total of 156 patients were included in the study.

Data. Data collected included patient age, location of drowning, and estimated submersion duration. Submersion duration was estimated from first responders' logs; if this was not available, the submersion duration was based on family interview. Cardiopulmonary resuscitation (CPR) was defined as having received chest compressions either in the field or upon presentation to the emergency department (ED). In the case of bystander CPR, duration was considered to be that reported by first responders, and does not necessarily imply asystole, a nonperfusing rhythm or difficulty with ventilation. The patient's initial Glasgow Coma Scale (GCS) was recorded upon the

***See also p. 653.**

From the Departments of Pediatric Critical Care (KTR, RMS, BP) and Pediatrics (CK), Rady Children's Hospital, San Diego, CA; and Division of Pediatric Critical Care (KP), University of California, Davis Children's Hospital, Sacramento, CA.

Supported, in part, by Children's Hospital Department of Critical Care Research Fund.

The authors have not disclosed any potential conflicts of interest.

For information regarding this article, E-mail: krafaat@hotmail.com

DOI: 10.1097/PCC.0b013e31818c8955

Table 1. Age distribution of study patients

Age (yrs)	No. Patients (n = 156)
0–1	18 (12%)
1–2	44 (28%)
2–3	32 (21%)
3–4	22 (14%)
4–5	5 (3%)
5–10	24 (15%)
10–16	11 (7%)

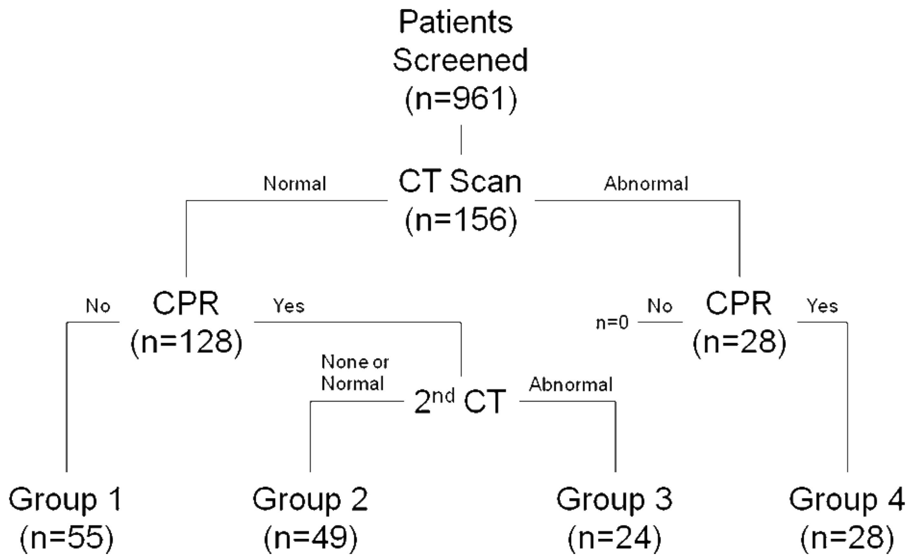

Figure 1. Distribution of patients within computed tomography (CT) groups.

Table 2. Presenting Glasgow Coma score

	Patient Group	GCS 3	GCS 4–7	GCS 8–11	GCS 12–15
1	Normal CT, no CPR (n = 55)	10 (18%)[a]	19 (35%)	11 (20%)	15 (27%)
2	Normal CT, CPR (n = 49)	25 (51%)[b]	12 (24%)	7 (14%)	5 (10%)
3	Normal first CT, abnormal second CT, CPR (n = 24)	23 (96%)	1 (4%)		
4	Abnormal first CT, CPR (n = 28)	28 (100%)			

[a]Ten (100%) patients arrived in the emergency department intubated; [b]Twenty-three (92%) patients arrived in the emergency department intubated.

GCS, Glasgow Coma Scale; CT, computed tomography; CPR, cardiopulmonary resuscitation.

patient's arrival to the ED. The CT results were obtained from final dictations made by an attending radiologist. Before August 2005, CTs were performed using a General Electric Single Slice CTI scanner (Chalfont St. Giles, UK). After August 2005, scans were performed using a General Electric 64 slice VCT scanner. The Glasgow Outcome Scale (GOS) was used to measure outcomes (1 = dead, 2 = vegetative state, 3 = severe disability, 4 = moderate disability, or 5 = good outcome) and was determined from review of medical records of most recent follow-up visits (minimum 6 months after initial injury) (8).

For the purpose of data analyses, patients were divided into four groups: group 1, those who did not receive CPR and had a normal CT upon presentation; group 2, those who received CPR, had a normal CT upon presentation and had no abnormal follow-up scans; group 3, those who received CPR, had a normal first CT and an abnormal second CT; and group 4, those who received CPR and had an abnormal first CT.

All data were entered into a Microsoft Excel database (Redmond, WA). Preliminary statistical analyses were performed using Excel's embedded statistical functions. Further analyses were performed using Stata Corporation's Intercooled STATA version 9.2 (StataCorp, College Station, TX). The Kruskal-Wallis test was used when mean submersion duration and mean total, in-field and in-hospital CPR duration were compared across the four groups. Fisher's exact test was used to examine the significance of the association between presenting GCS and the presence of an abnormal cranial CT. All data are presented as mean ± SD (range).

RESULTS

A total of 961 patients were screened for inclusion into the study; 281 were excluded as they were not admitted to the hospital. Five hundred seven patients were excluded because no CT scan was performed within 24 hrs and 17 were excluded due to coincidental trauma. One hundred fifty-six patients (58% male, 42% female) who were admitted to the pediatric intensive care unit under the care of the critical care service were included in the study. The mean patient age was 3.8 ± 3.4 yrs (range: 0.3–15.5 yrs). The distribution of patients' ages can be seen in Table 1.

Seventy-three percent (113 of 156) of submersion accidents occurred in outdoor pools, 15% (23 of 156) in bathtubs, 9% (15 of 156) in lakes, ponds, and rivers, 2% (3 of 156) in buckets, and 1% (2 of 156) in the Pacific ocean. See Figure 1 for details of the four groups in this study.

The presenting GCS of all patients is shown in Table 2. All patients in groups 1 and 2 had an initially normal cranial CT and either had no further CTs or had subsequent CTs that were normal. This set of patients is broken down into those who did not receive CPR (group 1) and those who did (group 2). Forty-nine of 104 patients with a normal initial CT received CPR either in the field or upon presentation to the ED (group 2). Those patients who presented with a GCS of 8 or higher all received CPR in the field for less than 5 mins. The duration of submersion and CPR, both in the field and in-hospital, are detailed in Table 3. For those who received CPR, the mean time from submersion to first CT was 8.7 ± 7.9 hrs (range: 0.9–23.7 hrs); for those who did not receive CPR, time from submersion to first CT was 3 ± 2.6 hrs (range: 0.5–3.8 hrs).

Fifteen percent (24 of 156) of patients presented with a normal cranial CT but later had an abnormal scan (group 3). The distribution of their presenting GCSs is also detailed in Table 2. All 24 patients received CPR. Submersion and CPR duration for group 3 are detailed in Table 3. Mean time from injury to first CT was 8.7 ± 7.8 hrs (range: 1.5–23.3 hrs). Mean time interval between first normal and second abnormal CT was 2.8 ± 1.6 days. Abnormal cranial CT findings for group 3 are detailed in Table 4 (see also Figs. 2 and 3). On no scan was there evidence of either intra- or extra-axial blood or unilateral findings.

Eighteen percent (28 of 156) of patients had abnormal findings on their initial cranial CT (group 4). The distribution of their presenting GCSs is detailed in Table 2. All 28 patients received CPR. Submersion and CPR duration, both in

Table 3. Duration of submersion and CPR

	Patient Group	Mean Duration of Submersion[a]	Mean CPR in Field[a]	Mean CPR in Hospital[a]	Mean Total CPR Time[a]
1	Normal first CT, no CPR (n = 55)	4 ± 2, (1–10)			
2	Normal first CT, CPR (n = 49)	9 ± 7, (1–40)	11 ± 13, (1–60), n = 49	18 ± 11, (0–40), n = 13	16 ± 20, (1–70)
3	Normal first CT, abnormal second CT, CPR (n = 24)	10 ± 6, (2–20)	16 ± 10, (5–31), n = 24	15 ± 11, (0–35), n = 11	23 ± 18, (5–60)
4	Abnormal first CT, CPR (n = 28)	13 ± 8, (3–30)	26 ± 12, (8–60), n = 28	25 ± 19, (0–70), n = 21	46 ± 20, (8–100)
	Significant intergroup difference?[b] (p)	<0.001	<0.001	<0.001	<0.001

[a]Data are presented as mean ± SD, (range). All times in minutes; [b]Kruskal-Wallis test.
CT, computed tomography; CPR, cardiopulmonary resuscitation.

Table 4. Findings on second CT for group 3

CT Findings[a]	Incidence (n = 24)
Diffuse loss of GWD	20 (83%)
Loss of basal ganglia GWD	2 (8%)
Loss of thalamic GWD	1 (4%)
Loss of cortical GWD	1 (4%)
LDC in basal ganglia	7 (29%)
LDC in cortex	2 (8%)
LDC in brainstem	1 (4%)
Effaced lateral ventricles	4 (17%)
Effaced basal cisterns	3 (13%)
Effaced third ventricle	1 (4%)

[a]All findings are bilateral.
CT, computed tomography; GWD, gray-white differentiation; LDC, low density changes.

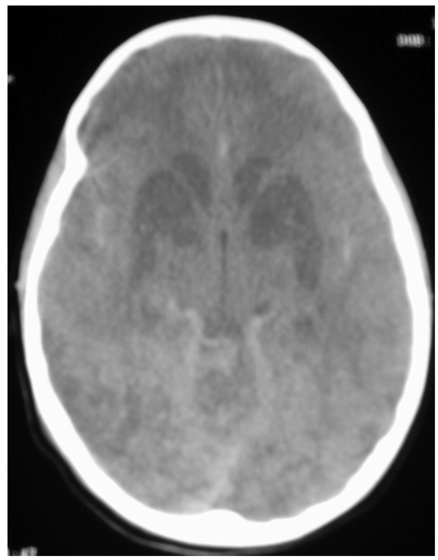

Figure 2. Second computed tomography from group 3 showing evidence of both bilateral low density changes in the basal ganglia and diffuse loss of gray-white differentiation.

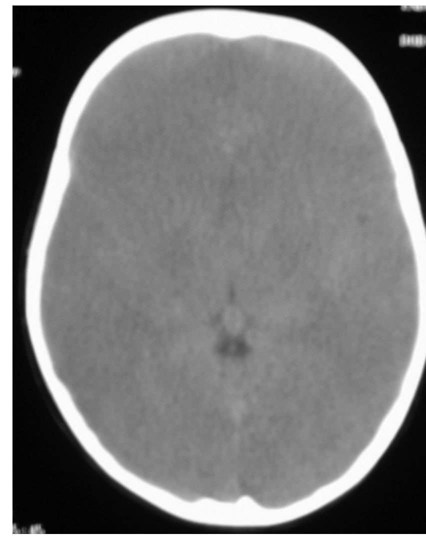

Figure 3. Second computed tomography from group 3 showing diffuse loss of gray-white differentiation and effaced basal cisterns.

the field and in-hospital, are detailed in Table 3. Mean time from injury to CT was 9.6 ± 7.5 hrs (range: 1–22.3 hrs). Abnormal findings for group 4 are detailed in Table 5 (see also Figs. 4 and 5). On these scans, there was no evidence of either intra- or extra-axial blood nor were there any unilateral findings. Six of these patients had repeat head CTs, 100% of which showed evidence of diffuse cerebral edema, and 50% showed evidence of bilateral basal ganglia infarcts. On none of the repeat scans was there any evidence of either intra- or extra-axial blood or unilateral abnormalities.

All patients with an initially abnormal CT presented with a GCS of 3. The presenting GCS was higher in those with an initially normal CT (groups 1–3) (mean: 6 ± 4, range: 3–15) compared with those with an initially abnormal CT (group 4) (mean: 3, range: 3) ($p < 0.001$). There was also a significant difference in GCS when comparing groups 1 and 2 (normal cranial CTs) with groups 3 and 4 (abnormal CT, either initially or on follow-up) ($p < 0.001$). Ninety-eight percent of patients with an abnormal first or second CT presented with a GCS of 3, and one patient (2%) presented with a GCS of 4. No patient with a GCS >4 had an abnormal CT.

Statistically significant differences between groups in mean submersion time, mean field CPR time, mean in-hospital CPR time, and mean total CPR time are detailed in Table 3.

All patients who presented with an initially abnormal cranial CT (group 4) had a GOS of 1 (death). Of patients who had a normal first CT, but an abnormal second CT (group 3), 13 of 24 (54%) had a GOS of 1, 10 of 24 (42%) had a GOS of 2 (persistent vegetative state), and 1 of 24 (4%) had a GOS of 5 (good outcome).

DISCUSSION

In 2000, there were over 4000 deaths due to drowning in the United States, representing 1.48 deaths per 100,000 population (9). Nearly half of all drownings occur in children less than 4 yrs old, with the highest rate in toddlers (1–2 yrs old) (10). This study, which included 156 pediatric patients with drowning, is the largest to date. As such, it represents an important examination of the patterns of injury seen on the cranial CTs of pediatric patients with drowning. The observed associations between injury variables, CT abnormalities, and eventual outcome may also serve to support therapeutic decisions.

Cranial CT studies are frequently performed to evaluate the results of an acute, profound episode of hypoxic-ischemic injury. After a global insult of this type, the brain is not uniformly injured. In general, regions of the brain which are metabolically more active are more susceptible to hypoxic injury (11). Gray matter is more sensitive than white matter. The cerebral cortex, thalami, hippocampi, and the basal ganglia are the areas that are preferentially involved (12, 13). These regional differences in vulnerability are reflected in CT findings. Early (within 1–3 days of injury) CT findings may either be normal or show signs of diffuse cerebral edema (5, 6, 14, 15). Later CT imaging may reveal diffuse loss of gray-white differentiation and sym-

Table 5. Initial CT findings of group 4

CT Findings[a]	Incidence (n = 28)
Diffuse loss of GWD	21 (75%)
Loss of basal ganglia GWD	1 (3%)
Loss of cortical GWD	2 (7%)
LDC in basal ganglia	14 (50%)
LDC in cortex	3 (11%)
LDC in cerebellum	1 (3%)
Effaced basal cisterns	4 (14%)
Effaced lateral ventricles	2 (7%)

[a]All findings are bilateral.

CT, computed tomography; GWD, grey-white differentiation; LDC, low density changes.

Figure 4. Initial computed tomography from group 4 showing diffuse loss of gray-white differentiation and effaced lateral ventricles.

metric, low density changes in the basal ganglia which, pathologically, represent a combination of ischemic or infarcted brain and vasogenic edema (5, 11).

Previously, only a few, small retrospective reviews have examined CT findings after a hypoxic-ischemic injury in children. Taylor et al. found that 12 of 17 children (71%) had normal CTs following a drowning event (7). Abnormal findings consisted of either diffuse edema and/or bilateral basal ganglia infarction. Romano et al. looked at cranial CTs in 15 children who presented comatose to the ED following a drowning event and found 80% of the cranial CTs performed within 36 hrs were normal (6). Both studies concluded that an initially abnormal cranial CT is highly predictive of a poor outcome, whereas a normal CT cannot be used to predict outcome.

Our results are comparable to those of these prior studies (5, 6); 82% of patients

Figure 5. Initial computed tomography from group 4 showing diffuse loss of gray-white differentiation and bilateral basal ganglia low density changes.

in our study presented with a normal CT scan, and 15% later had an abnormal CT scan, with a mean interval between scans of 2.8 ± 1.6 days. Eighteen percent of our patients presented with an abnormal CT scan, which is also similar to previous studies. A large percentage of our admitted patients did not receive CT scans secondary to short submersion times and normal neurologic evaluation. In these cases, admission was most often secondary to mild pulmonary complications and the consequent need for a period of observation.

In our study, 75% of initially abnormal cranial CTs showed evidence of diffuse edema, as evidenced by loss of gray-white differentiation, and 50% showed evidence of bilateral basal ganglia infarct or edema, findings consistent with those in smaller retrospective reviews. These findings were present on CT within 24 hrs, with 11 patients in group 4 (38%) even showing changes within 4 hrs of submersion injury. These patients in group 4 had submersion and CPR times that were statistically significantly longer than those with initially normal CTs. Of note, none of the 58 abnormal scans in this study showed evidence of either intra- or extra-axial blood or unilateral findings.

All patients with abnormal first CTs died. This is consistent with previous studies of drowning where an initially abnormal CT scan was found to be highly predictive of a poor outcome (5, 6). Dubowitz et al. (16) showed that in children with drowning, magnetic resonance imaging of the brain, performed within 24–48 hrs, is 100% sensitive and 86% specific for a poor outcome (death or persistent vegetative state) if either diffuse edema or T2 weighted changes in the basal ganglia are found. Similarly, as shown by Dean and McComb (17), elevated intracranial pressure following a drowning injury is consistently associated with a bad outcome; of 14 patients with intracranial pressure >20 torr, 10 died and four remained in a persistent vegetative state.

Outcome was also poor in those patients who had a normal initial CT and an abnormal second CT (group 3). Of the 24 patients in group 3, 13 died and 10 had a GOS of 2, indicating a persistent vegetative state. Only one patient had a good outcome (GOS of 5). This patient was submerged in estimated 40°F water, received a total of 15 mins of CPR and the second CT had evidence of diffuse loss of gray-white differentiation that was described as "equivocal." The association between follow-up CT findings and outcome has not been made before this study.

These outcome data may be helpful in guiding direction of therapy after a submersion injury. An abnormal cranial CT following drowning is associated with a poor outcome, which suggests that aggressive therapy may not be warranted. However, a limitation of this study is that specific therapy was neither controlled for nor recorded.

A significant percentage of patients in groups 1 and 2 arrived to the ED intubated. It is likely that artificially low GCSs were assigned secondary to their having received sedation/paralysis to facilitate intubation. In group 2, two patients arrived to the ED with a GCS of 3 without an endotracheal tube in place. In both cases, attempts at intubation were unsuccessful in the field but successful upon arrival to the ED. Although both initial CT scans were normal, both patients were eventually declared brain dead on hospital day number 2, which emphasizes that although a negative CT may be a poor prognostic indicator, no conclusions can be made regarding prognosis after a normal CT (5, 6).

CPR was performed in the field on 101 of 156 patients for a mean duration of 16 mins (range: 1–60 mins). In-hospital CPR was performed on 46 of 156 patients with a mean duration of 21 mins (range: 2–70 mins). The lack of cranial CT findings consistent with traumatic injury to the brain found in this study suggests that CPR, regardless of duration, is not associated with the presence of hemorrhage on the CTs of this large population of children with drowning.

CTs were obtained in 156 of 961 patients with drowning. The clinical judgment used at the time of initial evaluation was not a point of investigation in this study. However, it is important to note that there were no abnormal CTs in patients who did not receive CPR; indeed there were no abnormal findings in the CTs of those patients who presented with a GCS >4, even if they did receive CPR. These data suggest that an initial cranial CT may not be necessary for patients with drowning who did not require CPR.

Our data describe the absence of both hemorrhage and unilateral findings in hypoxic-ischemic injury. The findings in these cranial CTs differ significantly from those found in children with AHT which often show blood and/or unilateral edema or infarct (18–28). Although this study did not include a group of patients with AHT for comparison, the contrast between the two groups is worthy of discussion in light of its forensic implications. A direct comparison between pediatric patients with drowning and traumatic head injury is presently underway, which will serve to further define the differences that are outlined below. In a prospective study comparing AHT and non-AHT, 81% of children aged 2 yrs and younger who suffered non-AHT had evidence of subdural hemorrhage (SDH) on CT, with 64% of all SDH being secondary to inflicted head injury (26). SDH is a common abnormal finding on cranial CTs of children with AHT, with an incidence of 63%–89% (18, 27, 28). In the absence of SDH, studies of CT findings in AHT frequently show evidence of other intra- and/or extra-axial hemorrhage. Dashti et al. (28) found a 63% incidence of SDH, but in the context of evidence of either intra- or extra-axial hemorrhage on the initial CTs of 31 of 38 (82%) children with AHT.

Furthermore, all patients with an initially abnormal cranial CT presented with a GCS of 3. This differs considerably from the reported GCS on presentation of children with abusive traumatic brain injury. Ewing-Cobbs et al. (29) found evidence of extra-axial hemorrhage on 97% of initial CT scans of children with AHT. Sixty-one percent of these children presented with a GCS of 13–15, 23% presented with a GCS of 9–12, and only 16% presented with a GCS of 3–8. This suggests that after a hypoxic-ischemic injury to the brain, abnormal findings on CT are present only in those patients with severely depressed mental status on initial evaluation (GCS 3 or 4), whereas in abusive head injury, abnormal CT findings can be present in children with a higher or even normal level of consciousness.

CONCLUSIONS

In the largest retrospective study of submersion injury in children to date, cranial CTs performed within 24 hrs of injury were normal in 82%. Abnormal scans, both initially and after an initially normal CT, show evidence of diffuse edema and/or basal ganglia infarct. There was no evidence of either intra- or extra-axial blood or unilateral findings in any of the abnormal head CTs. No patients with a GCS >4 had an abnormal CT at any time. Those patients who presented with an initially abnormal head CT all had a GCS of 3 and all eventually died. Although this retrospective study does not indicate the optimal timing of subsequent cranial CTs after injury, the presence of an abnormal CT scan 2–3 days after injury is also a strong predictor of severe neurologic injury. These data suggest that after a hypoxic-ischemic injury, aggressive therapy in children with an initially abnormal CT scan may not be warranted. The CT findings and the presenting GCS of patients with drowning differ significantly from those patients who have suffered AHT.

ACKNOWLEDGMENTS

We thank Robert Riffenburgh, PhD, for his help with the statistics and Craig Swanson, MD, for help with clarifying figures.

REFERENCES

1. World Health Organization: World Health Report, 2002: Reducing risks, promoting healthy lives, Geneva, 2002
2. Gilchrist J, Gotsch K, Ryan G: Nonfatal and fatal drownings in recreational water settings: United States, 2001–2002. *MMWR Morb Mortal Wkly Rep* 2004; 53:447–452
3. Modell JH: Drown versus near-drown: A discussion of definitions. *Crit Care Med* 1981; 9:351–352
4. Gonzalez-Rothi RJ: Near-drowning: Consensus and controversies in pulmonary and cerebral resuscitation. *Heart Lung* 1987; 16: 474–482
5. Shuman SH: The iceberg phenomenon of near drowning. *Crit Care Med* 1976; 4: 127–128
6. Romano C, Brown T, Frewen TC: Assessment of pediatric near-drowning victims: Is there a role for cranial CT? *Pediatr Radiol* 1993; 23: 261–263
7. Taylor SB, Quencer RM, Holzman BH, et al: Central nervous system anoxic-ischemic insult in children due to near-drowning. *Radiology* 1985; 156:641–646
8. Jennet B, Bond M: Assessment of outcome after severe brain damage. *Lancet* 1975; 1:480–484
9. National Center for Injury Prevention and Control: WISQUARS Leading Causes of Death Reports 2002; 1999–2000
10. Orlowski JP: Drowning, near-drowning, and ice-water submersions. *Pediatr Clin North Am* 1987; 34:75–92
11. Corr P, Gafoor F: Hypoxic encephalopathy due to near-drowning. *S Afr Med J* 1999; 89:1154
12. Marcoux FW, Morawetz RB, Crowell RM, et al: Differential regional vulnerability in transient focal cerebral ischemia. *Stroke* 1982; 13:339–346
13. DeReuck J: The human periventricular arterial blood supply and the anatomy of cerebral infarctions. *Eur Neurol* 1971; 5:321–334
14. Fitch SJ, Gerald B, Magill HL, et al: Central nervous system hypoxia in children due to near drowning. *Radiology* 1985; 156:647–650
15. Schuier FJ, Hossmann KA: Experimental brain infarcts in cats. II. Ischemic brain edema. *Stroke* 1980; 11:593–601
16. Dubowitz DJ, Bluml S, Arcinue E, et al: MR of hypoxic encephalopathy in children after near drowning: Correlation with quantitative proton MR spectroscopy and clinical outcome. *AJNR Am J Neuroradiol* 1998; 19:1617–1627
17. Dean JM, McComb JG: Intracranial pressure monitoring in severe pediatric near-drowning. *Neurosurgery* 1981; 9:627–630
18. Merten DF, Osborne DRS, Radkowski MA, et al: Craniocerebral trauma in the child abuse syndrome: Radiological observations. *Pediatr Radiol* 1984; 14:272–277
19. Zimmerman RA, Bilaniuk LT, Bruce D, et al: Computed tomography of craniocerebral injury in the abused child. *Radiology* 1979; 130:687–690
20. Duhaime AC, Christian C, Moss E, et al: Long-term outcome in infants with the shaking-impact syndrome. *Pediatr Neurosurg* 1996; 24:292–298
21. Ewing-Cobbs L, Prasad M, Kramer L, et al: Inflicted traumatic brain injury: Relationship of developmental outcome to severity of injury. *Pediatr Neurosurg* 1999; 31:251–258
22. Duhaime AC, Christian CW, Rorke LB, et al: Nonaccidental head injury in infants–the "shaken-baby syndrome." *N Engl J Med* 1998; 338:1822–1829
23. Feldman KW, Brewer DK, Shaw DW: Evolution of the cranial computed tomography scan in child abuse. *Child Abuse Negl* 1995; 19:307–314
24. Ghahreman A, Bhasin V, Chaseling R, et al:

Nonaccidental head injuries in children: A Sydney experience. *J Neurosurg* 2005; 103: 213–218
25. Hymel KP, Rumack CM, Hay TC, et al: Comparison of intracranial computed tomographic (CT) findings in pediatric abusive and accidental head trauma. *Pediatr Radiol* 1997; 27:743–747
26. Vinchon M, Defoort-Dhellemmes S, Desurmont M, et al: Accidental and nonaccidental head injuries in infants: A prospective study. *J Neurosurg* 2005; 102:380–384
27. Geddes JF, Hackshaw AK, Vowles GH, et al: Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 2001; 124:1290–1298
28. Dashti SR, Decker DD, Razzaq A, et al: Current patterns of inflicted head injury in children. *Pediatr Neurosurg* 1999; 31:302–306
29. Ewing-Cobbs L, Prasad M, Kramer L, et al: Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Childs Nerv Syst* 2000; 16:25–33