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Extradural Hematoma: Analysis of Factors Influencing the Courses of 161 Patients

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The clinical and computed tomographic (CT) findings in a series of 161 consecutive patients operated upon for postraumatic extradural hematoma are analyzed. Thirteen (8%) patients had delayed epidural hematoma formation. The overall mortality for the series was 12%, significantly lower than that observed during the prior "angiographic" period at the same unit (30%). Because all but 1 of the deaths occurred among the 66 patients unconscious at the time of operation (27% mortality in this subgroup), the authors sought differential factors between comatose and noncomatose patients at operation. There were no significant differences between these groups in age, sex, mechanism of injury, preoperative course of consciousness (lucid interval or not), or epidural hematoma location and shape. In contrast, significant differences were seen between the two subgroups in trauma-to-operation interval, hematoma volume, CT hematoma density (mixed low-high CT density vs. homogeneous hyperdensity), midline displacement, severity of associated intracranial lesions, and postoperative intracranial pressure (ICP). Patients comatose at operation usually evidenced a more rapid clinical deterioration (a shorter trauma-to-operation interval) and tended to have a large hematoma volume, a higher incidence of mixed CT density clot (hyperacute bleeding), more marked shift of midline structures, more severe associated lesions, and higher postoperative ICP levels. (*Neurosurgery* 23:44–51, 1988)

Key words: Computed tomography, Extradural hematoma, Glasgow coma scale, Head injury, Intracranial pressure

The introduction of computed tomography (CT) was a definitive step forward in the diagnostic work-up of postraumatic extradural hematoma. Aside from the improved patient management that results from more rapid identification of the clot and more accurate definition of coexistent intracranial lesions, CT allows anatomoclinical correlation that was not feasible during the angiographic era (1, 3–10, 12, 14–19, 21, 23, 26, 29–31, 33, 35, 39–41, 43–45, 47, 51–53, 55–57, 60, 62–68, 70–82).

In this report, we analyze the correlations among different clinical and pathological variables in a series of 161 patients with postraumatic epidural hematoma treated during the CT era. Because the mortality of patients undergoing operation for epidural hematoma is virtually limited to those undergoing operation in coma, emphasis was placed on the factors that differentiate comatose and noncomatose patients.

CLINICAL MATERIAL AND METHODS

This series includes 161 consecutive patients with postraumatic epidural hematoma operated upon between November 1977 and July 1986. They represent 3% of the total 5124 head injury patients admitted to our department during this period. We excluded from the study patients showing a small extradural hematoma beneath a depressed skull fracture, as well as 5 patients operated upon without having CT and 4 managed nonsurgically. Nearly two-thirds of the patients were admitted to our unit directly from the scene of the accident, and the rest were referred by local hospitals unequipped for CT. Transportation was always by road.

Patients' ages ranged from 3 days to 78 years (average 27.7 years). The cause of injury was a traffic accident in 74 cases, a fall in 60, a blow in 15, and birth trauma in 1 and was unknown in 11 cases. All patients were evaluated upon arrival, and the Glasgow coma scale (GCS) scores were recorded. Twenty-seven (17%) patients had relevant associated extracranial lesions. CT was performed immediately after the admission of comatose patients. CT consisted of four-slice (80% of cases) or five-slice (20%) unenhanced scans performed with an EMI brain scanner (160 × 160 matrix) (EMI Ltd., X-ray Systems Division, Hayes, Middlesex, England). Some patients, among them those with chronic epidural hematoma, also had postcontrast CT. Patients conscious at admission were scanned when they developed alterations of consciousness, severe headache, or motor or pupillary changes. In 10 patients, the epidural hematoma was not evident on initial CT, and another 3 who were operated upon for epidural hematoma immediately after admission developed delayed epidural hematomas at different locations. The hematomas were classified as homogeneously hyperdense, heterogeneous (mixed high-low density), or primarily hypodense with a contrast-enhancing membrane (Zimmerman Type III) (8, 9, 14, 26, 30, 43, 55, 57, 79, 80). Hematoma volume was calculated from CT slices using a method reported by other authors (17, 58). Seventy (43%) patients had associated intracranial lesions apparent on initial or follow-up CT, but in all patients the epidural hematoma was considered the predominant lesion. Four patients with posterior fossa or occipital-posterior fossa epidural hematoma developed secondary hydrocephalus.

One hundred nineteen (74%) patients with acute epidural hematomas were operated upon within 24 hours of injury, and 42 (26%) underwent operation 2 to 23 days after trauma. Sixty-six (41%) patients underwent operation in coma (GCS score ≤8), 66 (41%) noncomatose patients showed some impairment in level of consciousness, and 29 (18%) were fully conscious at the time of operation. The clot was removed by osteoplastic craniotomy in 140 patients. The others underwent craniectomy and/or excision of bone fragments. Eighteen patients required a second operation to remove contused brain (6 cases), newly formed epidural hematoma (3 cases), reaccumulated or incompletely evacuated epidural clot (5 cases), brain hematoma (2 cases), subdural empyema (1 case), or tension pneumocephalus (1 case). In 61 patients, intracranial pressure (ICP) was monitored postoperatively using an intraventricular catheter or an epidural fiber optic sensor. Raised ICP was managed with hyperventilation, aggressive medical therapy, or surgical evacuation of associated lesions when indicated. Control CT was performed when patients did not improve after operation or showed subsequent deterioration or adverse ICP changes. The final result was graded at least 6 months after injury according to the outcome scale of Jennett and Bond (38): good recovery, moderate disability, severe disability, vegetative state, or death.

RESULTS

The overall mortality for this series was 12%. The difference in mortality between patients undergoing operation in coma (27%) or in a conscious state (1%) was highly significant ($\chi^2 = 25.7$; $P < 0.0005$). Twenty-five (38%) comatose and 8 (8.4%) noncomatose patients developed some degree of disability, and this difference was also highly significant ($\chi^2 = 33.8$; $P < 0.0005$). In fact, neurological status at operation was the clearest determinant of outcome.

No differences were seen in neurological status at operation or final outcome of the 103 (64%) patients admitted directly from the scene of the accident and the 58 (36%) patients referred from local hospitals.

The distribution of cases by age decade is shown in Figure 1. The distribution was almost identical in both subgroups, and age per se did not seem to influence final outcome. Mortality in the pediatric population (under 15 years old), consisting of 15 comatose and 28 noncomatose patients, did not differ from that of the adults. The difference in the mortality rate of comatose patients under and over 20 years old (15% vs. 36%) was not statistically significant.

The proportions of males (84%) and females (16%) were identical in the comatose and noncomatose subgroups, and sex did not influence the final results.

The mechanisms of injury were similar in comatose and noncomatose patients. Falls were more frequent during the 1st decade of life (61%), and traffic accidents predominated from the 2nd to the 4th decades, with high speed trauma significantly more frequent than other mechanisms ($\chi^2 = 22.9$; $P < 0.0005$). The occurrence of high speed trauma was slightly more frequent in comatose than in noncomatose patients, but the difference is not significant. Falls carried a higher mortality rate than traffic accidents in the subgroup of comatose patients ($\chi^2 = 6.1$; $P < 0.05$), but were not associated with higher morbidity in either subgroup.

The distribution of cases related to the trauma-to-operation interval is shown in Figure 2. The later the patient came to operation, the better the final result for both comatose and noncomatose patients. The proportion of comatose patients operated upon within 12 hours of injury was significantly higher than that of noncomatose patients ($\chi^2 = 9$; $P < 0.005$). The mortality of comatose patients was restricted to those undergoing operation within 12 hours of trauma. Every noncomatose patient who developed some degree of disability and the single death were operated upon within this time period. There were 18 (19%) noncomatose patients operated upon more than 48 hours after trauma (subacute epidural hematoma cases) and 4 (4%) operated upon later than 13 days (chronic epidural hematoma cases). The correlation between the age of the patient and the interval from trauma to operation shows that older patients in both comatose and noncomatose subgroups were operated upon earlier. The percentages of comatose patients <20 and >20 years old who underwent operation within 12 hours of injury were 59% and 92%, respectively ($\chi^2 = 10.4$; $P < 0.005$), and those of noncomatose patients were 42% and 65%, respectively ($\chi^2 = 4.7$; $P < 0.05$).

In Table 1, the preoperative course of consciousness is correlated with final outcome in comatose and noncomatose

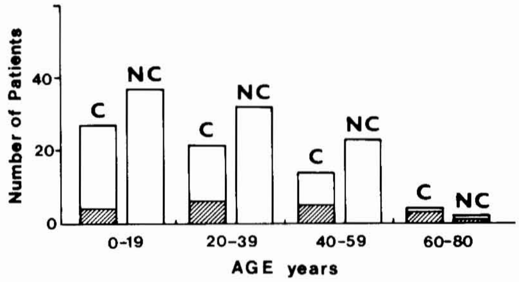

FIG. 1. Distribution of epidural hematoma cases by age decades. *C* and *NC symbols* over *bars* represent patients comatose and noncomatose at operation, respectively. *Striped areas* within *bars* represent fatal cases.

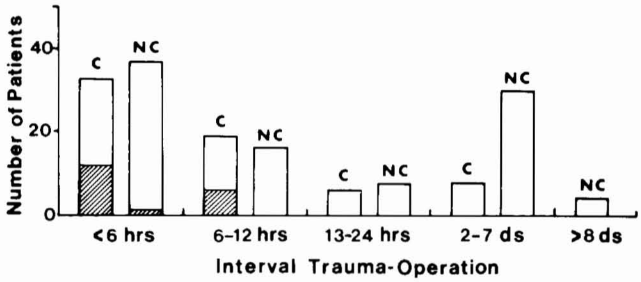

FIG. 2. Distribution of epidural hematoma cases as related to the interval from trauma to operation. *C* and *NC symbols* over *bars* represent patients comatose and noncomatose at operation, respectively. *Striped areas* within *bars* represent fatal cases. (*hrs* = hours; *ds* = days)

TABLE 1
Course of Consciousness before Operation as Related to the Final Outcome in 66 Comatose and 95 Noncomatose Patients Operated upon for Epidural Hematoma[a]

Course	Total No. (%)	No. Cases with Each Outcome (%) GR	MD	SD	D
		Comatose			
C-U	22 (33)	9 (41)	7 (32)	—	6 (27)
U-U	33 (50)	9 (27)	15 (45)	1 (3)	8 (24)
U-C-U	9 (14)	4 (44)	1 (11)	—	4 (44)
?-U	2 (3)	1 (50)	—	1 (50)	—
Total	66 (100)	23 (35)	23 (35)	2 (3)	18 (27)
		Noncomatose			
C-S	8 (8)	7 (87.5)	1 (12.5)	—	—
S-S	7 (7)	7 (100)	—	—	—
U-C-S	28 (29)	26 (93)	1 (3)	—	1 (3)
U-S	16 (17)	10 (62.5)	6 (37.5)	—	—
U-C	23 (24)	23 (100)	—	—	—
C-C	6 (6)	6 (100)	—	—	—
?-S	7 (7)	7 (100)	—	—	—
Total	95 (100)	86 (91)	8 (8)	—	1 (1)

[a] Abbreviations: GR, good recovery; MD, moderate disability; SD, severe disability; D, death; C, conscious; U, unconscious; S, somnolent.

patients. Forty-two (64%) comatose patients were rendered unconscious by impact, and 31 (47%) had a lucid interval at some point. The mortality of comatose patients was not significantly influenced by the preoperative course of consciousness, but patients who were unconscious throughout the preoperative period had nearly twice the disability rate (48%) of those who were conscious at some moment (26%) (χ^2 = 3.5; P > 0.05). Seventy-four (78%) patients in the noncomatose group were rendered unconscious by impact, and 66 (69%) underwent operation somnolent or semicomatose, with mortality (1 case) and morbidity being restricted to the latter group.

In Table 2, pupillary changes and motor score at operation are correlated with the final outcome in comatose patients. Forty-one (62%) patients showed uni- or bilateral mydriasis, but only those with bilateral mydriasis (11 cases) or mydriasis contralateral to the hematoma (2 cases) had a significantly poorer outcome than patients with normal pupils (χ^2 = 22.6; P < 0.0005). The motor score narrowly correlated with the final result. Only 5% of the patients with a motor score of 4 or 5 died, whereas 80% of those with a motor score of 1 had a fatal outcome (χ^2 = 17.1; P < 0.0005). The mortality rate of patients with a motor score of 2 or 3 was 24%, but the difference from the mortality of patients having a motor score of 4 or 5 was not significant. The occurrence of hemiparesis, present in 24 (36%) comatose patients, did not correlate with the final result. The final outcome of noncomatose patients was not influenced by the presence of clinical signs of ipsilateral mydriasis (3 cases), hemiparesis (12 cases), cranial nerve abnormalities (7 cases), papilledema (6 cases), mental deterioration (3 cases), or severe bradycardia (5 cases).

Skull fracture was evidenced in x-ray films of the skull or at operation in 53 (80%) comatose patients and in 81 (85%) noncomatose patients. Seventeen patients had depressed fractures. The mortality rate was significantly higher in comatose patients without evident skull fracture than in those with fracture (χ^2 = 5.7; P < 0.05). Moderate disability was also higher in noncomatose patients without skull fracture, but the difference was not statistically significant. The location of the epidural clot (frontal—26 (16%) cases, temporal—31 (19%) cases, temporoparietal—50 (31%) cases, parietal—43 (27%) cases, occipital-posterior fossa—11 (7%) cases) or its shape (131 (81%) biconvex, 15 (9%) monoconvex, 10 (6%) irregular, and 5 (3%) bilenticular biconvex) did not differ in the comatose and noncomatose groups and thus did not correlate with final outcome. By contrast, hematoma volume correlated with the severity of the clinical presentation. The larger the clot, the higher the incidence of preoperative coma and the worse the outcome (Fig. 3). Hematomas larger than 150 cc in volume occurred in 23% of comatose and in 3% of noncomatose patients (χ^2 = 15.8; P < 0.0005), and hematomas smaller than 90 cc were seen in 56% of comatose and 86% of noncomatose patients (χ^2 = 18; P < 0.0005). Hematoma size influenced the final outcome of comatose patients. Mortality was significantly higher in patients with clots over 150 cc in volume than in those with smaller clots (χ^2 = 16.1; P < 0.0005). In noncomatose patients (except for the patient who died) who had a hematoma of more than 150 cc, the final outcome did not correlate with hematoma size. Six of 8 noncomatose patients who developed moderate disability had hematomas smaller than 60 cc in volume.

As for the correlation between patient age and clot volume, the average hematoma volume in comatose patients <20 and >20 years old was 68 cc and 115 cc, respectively. The average hematoma volume was 50 and 51 cc in noncomatose patients <20 and >20 years old, respectively.

TABLE 2
Correlation between the Motor Score (GCS) or Pupillary Changes at Operation and the Final Outcome in 66 Comatose Patients Operated upon for Epidural Hematoma

	No. Cases (%)	No. Cases with Each Outcome (%)			
		GR	MD	SD	D
Motor score					
1	10 (15)	1 (10)	1 (10)	—	8 (80)
2–3	37 (56)	15 (41)	12 (32)	1 (3)	9 (24)
4–5	19 (29)	7 (37)	10 (53)	1 (5)	1 (5)
Pupils at operation					
Equal	25 (38)	11 (44)	10 (40)	1 (4)	3 (12)
Ipsilateral mydriasis	28 (42)	11 (39)	12 (43)	1 (4)	4 (14)
Contralateral mydriasis	2 (3)	—	—	—	2 (100)
Bilateral mydriasis	11 (17)	1 (9)	1 (9)	—	9 (82)
Total	66 (100)	23 (35)	23 (35)	2 (3)	18 (27)

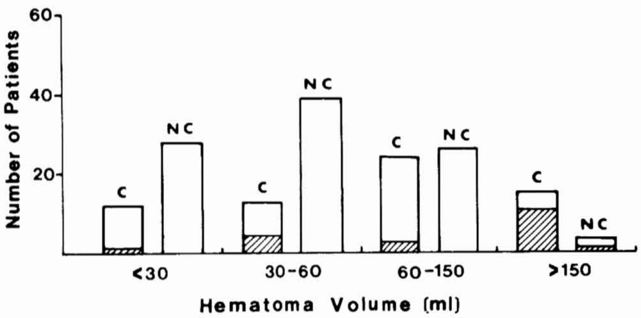

FIG. 3. Hematoma volume in comatose (*C*) and in noncomatose (*NC*) patients. *Striped areas* within *bars* represent fatal cases.

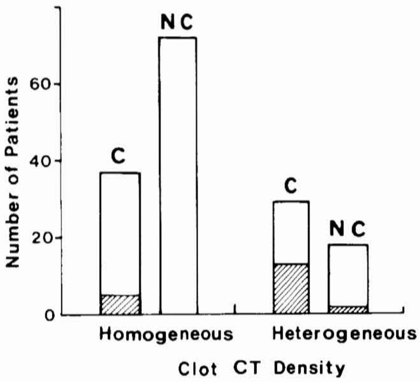

FIG. 4. Distribution of epidural hematomas showing a homogeneous hyperdense CT density or heterogeneous (mixed high-low) CT density in patients comatose (*C*) and noncomatose (*NC*) at operation. *Striped areas* within *bars* represent fatal cases.

The incidence of heterogeneous (mixed high-low CT density) hematoma was significantly higher in comatose (44%) than in noncomatose (18%) patients (χ^2 = 11.1; P < 0.001 (Fig. 4). Comatose patients with heterogenous hematomas had a significantly poorer outcome than those with homogeneous clots (χ^2 = 7.9; P < 0.01). The proportion of noncomatose patients with heterogeneous clot who had some impairment of consciousness at operation was twice (21%) that in those who were alert at operation (10%), but the difference was not significant.

CT showed black dots trapped inside the epidural clot in 11 comatose and in 10 noncomatose patients. Whether they corresponded to air bubbles is uncertain as CT density was not measured. All of these patients had skull fractures that in most instances extended into the sinuses or the mastoid region. Four comatose patients had pneumocephalus, and all of these had skull fractures.

As could be expected from the differences in hematoma volume, the midline shift seen on initial CT was more marked in comatose than in noncomatose patients. A shift of more than 12 mm occurred in 20% of the comatose vs. 4% of the noncomatose patients. There was less than 8 mm of displacement in 59% of the comatose vs. 84% of the noncomatose ($\chi^2 = 12.7$; $P < 0.001$). Half of the comatose and 37% of the noncomatose patients with midline shift of <4 mm had contralateral associated lesions (20 cases), a posterior fossa or occipital-posterior fossa hematoma (8 cases), or a bilateral epidural hematoma (1 case). The correlation between midline shift and mortality in comatose patients was significant only when extreme values (<4 mm vs. >12 mm) were considered ($\chi^2 = 6.3$; $P < 0.05$). The correlation between preoperative midline shift and patient age is shown in Figure 5. The incidence of >8-mm shift was similar in comatose and noncomatose patients <20 years. In patients over 20 years old, midline displacement of >8 mm occurred significantly more often in comatose (54%) than in noncomatose (9%) patients ($\chi^2 = 23.8$; $P < 0.0005$). In the subgroup of comatose patients, midline shift was significantly greater in elderly patients ($\chi^2 = 6.6$; $P < 0.05$), and the reverse was true in the subgroup of noncomatose patients ($\chi^2 = 5.3$; $P < 0.05$).

As shown in Table 3, 76% of comatose and 21% of noncomatose patients had some type of associated intracranial lesion ($\chi^2 = 47.5$; $P < 0.0005$). The incidence of associated lesions was significantly higher in comatose patients, and these lesions were more severe. For instance, cerebral hemispheric swelling and multifocal brain contusion, which carried a significantly higher mortality rate than other types of associated lesions ($\chi^2 = 10$; $P < 0.002$), were never seen in noncomatose patients. The morbidity rates in noncomatose patients with and without associated lesions were significantly different ($\chi^2 = 15.3$; $P < 0.0005$).

Table 4 summarizes the clinical factors and CT findings that may have conditioned the timing of operation in this series. The average age of both comatose and noncomatose patients decreased as the trauma-to-operation interval increased, so that older patients were operated upon sooner. The occurrence of a lucid interval was unrelated to the timing of operation. The rhythm and severity of neurological deterioration influenced the timing of surgery so that patients with more rapidly developing symptoms, more pupillary changes, and lower GCS scores tended to be operated upon sooner. Noncomatose patients with poorer GCS scores, focal deficits, or some impairment in the level of consciousness underwent operation sooner than other patients in the subgroup; if we exclude those operated upon within 12 hours because of a depressed skull fracture, 94% of the patients operated upon within this time interval were somnolent. Clot volume and the incidence of heterogeneous (mixed CT density) clot were greater in patients operated earlier.

ICP was measured after hematoma evacuation in 55 (83%) of the comatose patients. Pressure was within normal limits in 19 patients, 6 of whom died from medical complications. Twenty-one patients had moderately raised ICP (15–35 mm

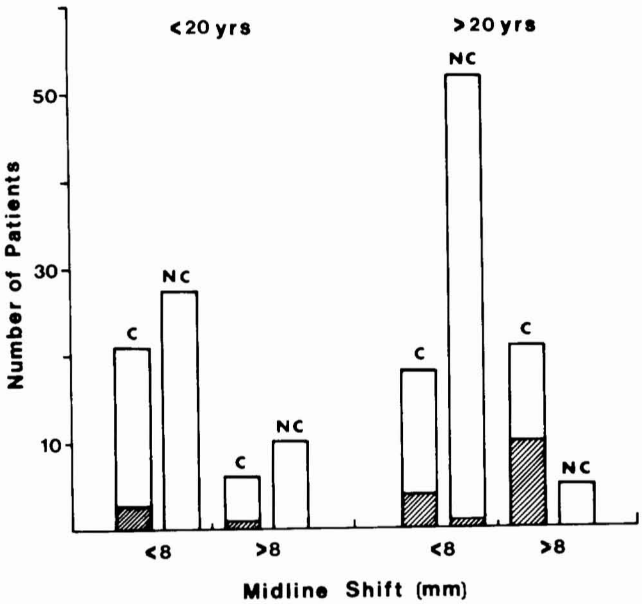

FIG. 5. Correlation between preoperative midline displacement and the age (<20 years and >20 years) of patients with epidural hematomas who were comatose (*C*) and noncomatose (*NC*) at operation. *Striped areas* within *bars* represent fatal cases.

Hg), which was controlled by nonsurgical treatment except in 8 cases in which an expanding brain contusion was removed. ICP over 35 mm Hg was seen in 15 patients with associated hemispheric or diffuse brain swelling or multifocal brain contusion. Half of these patients were unresponsive to high dose thiopental, and they had a mortality rate of 73%, significantly higher than in patients with normal (32%) or moderately raised (5%) ICP ($\chi^2 = 15.4$; $P < 0.0005$). Six noncomatose patients underwent postoperative ICP measurement because of coexistent brain contusion seen on initial CT; pressure was within normal limits except in 1 patient who required a partial lobectomy.

DELAYED EPIDURAL HEMATOMA FORMATION

Thirteen (8%) patients developed a delayed epidural hematoma that was evidenced by control CT 5 hours to 6 days after injury (average delay = 50.3 hours). At admission, these patients had normal CT (7 cases), epidural hematoma (3 cases), contralateral subdural hematoma, and/or diffuse brain swelling (3 cases). Retrospective examination of the admission CT scans disclosed a thin extracerebral blood collection at the site of the delayed hematoma formation in 3 of these patients.

Ages of the 13 patients ranged from 9 to 70 years (average 27.6 years). The trauma was due to traffic accidents in 9 cases (5 passenger and 4 pedestrian) and falls in 4 cases. Four patients were severely hypotensive at admission because of blood loss due to associated extracranial injuries. All but 1 had a skull fracture at the site of delayed hematoma formation. All were rendered unconscious by impact, but 5 recovered consciousness (2 briefly before falling into coma and 3 definitively). Ten patients were comatose and 3 were conscious when the delayed hematoma was detected. Six patients remained neurologically unchanged between admission and delayed epidural hematoma detection, 5 deteriorated, and 2 improved. ICP was above normal limits in the 7 comatose patients in whom it was measured, indicating the need for control CT, and it decreased after hematoma evacuation.

TABLE 3

Correlations between the Types of Associated Intracranial Lesions and the Final Outcomes in 66 Comatose and 95 Noncomatose Patients Operated upon for Traumatic Epidural Hematoma

Associated Lesion	Comatose					Noncomatose				
	No. (%)	Outcome[a]				No. (%)	Outcome			
		GR	MD	SD	D		GR	MD	D	Total
None	16 (24)	7 (44)	5 (31)	—	4 (25)	75 (79)	73 (97)	2 (3)	—	91 (57)
Diffuse brain swelling	15 (23)	8 (53)	4 (27)	1 (7)	2 (13)	2 (2)	2 (100)	—	—	17 (11)
Hemispheric brain swelling	6 (9)	1 (17)	1 (17)	—	4 (66)	—	—	—	—	6 (4)
Multifocal brain contusion	4 (6)	—	1 (25)	—	3 (75)	—	—	—	—	4 (2)
Focal brain contusion	24 (36)	8 (33)	10 (42)	1 (4)	5 (21)	12 (13)	6 (50)	5 (42)	1 (8)	36 (22)
Other[b]	10 (15)	3 (30)	4 (40)	—	3 (30)	8 (8)	6 (75)	1 (12.5)	1 (12.5)	18 (11)

[a] Abbreviations: GR, good recovery; MD, moderate disability; SD, severe disability; D, death.

[b] Includes subdural hematoma, ischemic area adjacent to the epidural hematoma, massive subarachnoid hemorrhage, intraventricular hemorrhage, and tension pneumocephalus.

TABLE 4

Correlations between Different Clinical and CT Findings and Timing of Operation in 66 Comatose and 95 Noncomatose Patients Operated upon for Traumatic Epidural Hematoma

Time of Operation	No. Cases	Average Age (yr)	% with Lucid Interval	Mydriasis No. (%)	Average GCS Score	Average Clot Volume (ml)	Average Midline Shift (mm)	% with Heterogeneous Clot	% with Associated Lesions
					Comatose				
<6 hr	33	30.62	45%	25 (76%)	4.58	102.38	6.76	61%	77%
6–12 hr	19	27.21	58%	12 (63%)	4.78	127.87	9.11	42%	52%
13–48 hr	14	20.57	50%	4 (29%)	5.71	52.50	4.93	14%	80%
					Noncomatose				
			%Somnolent at Operation	*Focal Signs*					
<6 hr	37	30.88	81%	14 (38%)	11.52	57.64	4.90	32%	32%
6–12 hr	16	29.48	87%	5 (31%)	11.62	66.81	4.74	19%	12%
13–48 hr	20	22.40	65%	6 (30%)	12.20	48.60	4.96	10%	25%
3–13 d	18	24.27	44%	1 (6%)	13.33	40.88	2.73	—	6%
>13 d	4	19.75	—	—	14.75	33.25	1.99	—	—

Three noncomatose and 3 comatose patients in this subgroup made good recoveries, 4 comatose patients became moderately disabled, and 3 comatose patients died.

DISCUSSION

The classic conception of acute postraumatic epidural hematoma is that of a lesion that develops immediately after trauma, reaches its greatest size within minutes, and threatens the patient's life after exhausting intracranial volume-buffering mechanisms (20). Epidural hematomas occasionally develop more slowly or are well tolerated, however, in which case the surgical prognosis is much more favorable (5, 8–10, 12, 14, 18, 19, 22, 30–33, 36, 37, 39, 44, 48, 49, 51, 55, 60, 61, 65, 70, 80, 81). An increasing number of epidural hematomas characterized by discrete neurological symptoms or no symptoms at all are being detected by CT, which contributes to reducing the mortality rate in series collected during the CT period (6, 15–17, 47, 66, 71, 72, 77, 81). The global mortality rate for epidural hematoma in our unit fell from 29% during the angiographic period to 12% after the advent of CT, and mortality in the subgroup of patients comatose at operation decreased from 37 to 27% during the same period (15). Although decreased mortality during the CT period might be due in part to the more aggressive medical treatment of raised ICP, it seems mainly dependent on the more rapid diagnosis of acute hematomas and the more precise detection of associated intracranial lesions that influence surgical outcome.

Neurological status at operation seems to be an important determinant of the final result in patients undergoing operation for epidural hematoma in both angiographic and CT series (6, 11, 15–17, 22, 27, 32, 34, 37, 42, 47–50, 54, 59, 67, 71, 72, 77, 81). In view of the fact that the mortality of comatose patients in different CT series is 11 to 43%, in contrast with almost zero mortality in noncomatose patients (6, 15, 17, 47, 67, 71, 72, 77), we conceived our study to analyze the factors distinguishing between patients operated in coma and those arriving conscious at the surgical setting.

In some epidural hematoma series, patient age correlated with final outcome, and mortality and morbidity were lower in younger patients (16, 32, 37, 47–49, 54, 72, 81). The lower mortality rate is attributed to factors like the low incidence of severe injuries and associated intracranial lesions and the slower hematoma growth. In our series, however, the final result was not significantly different in patients under and over 20 years, whether operated upon in a comatose or noncomatose state. The only significant pathological difference was that average epidural hematoma volume in comatose patients over 20 years of age was twice as large as that in patients under 20. Factors like sex or direct vs. indirect admission did not influence the final result. The absence of skull fracture was associated with a higher mortality in comatose patients, but the occurrence of fracture per se did not correlate with neurological status at operation.

As in other series, the interval between trauma and operation was shorter in comatose than in noncomatose patients; the longer the interval, the better the final outcome in both subgroups (32, 37, 42, 48, 49, 59, 72, 81). Unfortunately, the time between deterioration of consciousness and operation, which seems to be a more important determinant of final outcome (the longer the delay, the worse the result) (50), was unknown in most of our patients. In any case, hematoma volume was significantly greater in patients who underwent operation in coma, and the more voluminous the clot, the worse the final result in this subgroup. In the series of Ericson and Håkansson (17), every patient with a hematoma volume over 86 cc was in a comatose state. In our series, of the patients with hematoma volumes of >90 cc, 43% were operated in coma and only 14% were conscious at operation. Zimmerman and Bilaniuk pointed out the positive correlation among hematoma CT density, trauma-to-operation interval, and type of bleeding (arterial vs. venous) (79). The comatose patients who had a poorer outcome in our series also had a significantly higher incidence of hematomas with mixed high-low CT density, a finding suggestive of ongoing bleeding. Hyperacute epidural hematomas usually reach a large volume quickly, show a mixed CT density, and are frequently associated with other intracranial lesions. Hematoma growth may be so rapid that in some patients it may seem preferable to operate without awaiting CT (2). Hyperacute hematomas make zero mortality unattainable even in a well-run head injury care system.

The occurrence of associated intracranial lesions has been found to worsen the prognosis of patients with epidural hematomas (15–17, 32, 37, 42, 47, 49, 59, 67, 81). The incidence of associated lesions is as high as 19 to 58% in different recent series, even though some of the authors only consider cerebral contusions (6, 15–17, 67, 71, 77, 81). The incidence and severity of the associated lesions are significantly greater in comatose cases. Seventy-six per cent of our comatose patients had associated lesions, and the most severe abnormality (i.e., cerebral hemisphere swelling and multifocal brain contusion) was associated with a 37% mortality rate. Nonetheless, when all associated lesions are considered, there is no difference in the mortality rate of comatose patients with and without this type of abnormality, as confirmed by the series of comatose patients of Seelig et al. (71).

Associated diffuse axonal injury, which conditions surgical prognosis in patients with epidural hematoma, is difficult to detect because it may elude CT resolution (69, 71). Its incidence, however, may be inferred from the clinical course. Fifty per cent of our comatose patients were unconscious throughout the preoperative course, and 24% of the noncomatose patients had a discrete alteration in the level of consciousness at operation. High speed trauma, which is known to produce shearing injury of the white matter, was more frequent in patients with some degree of alteration of consciousness than in those who were fully conscious at some moment during the preoperative course (including patients operated in coma who had a lucid interval). The duration of postraumatic coma was significantly longer and the morbidity rate was higher in survivors of high speed injuries than in those surviving low speed trauma.

Sequential CT during the immediate postraumatic period detects slowly developing hematomas not seen in the initial CT study (true delayed epidural hematomas) (5, 10, 18, 19, 39, 40, 44, 51, 60, 65, 66, 70). These hematomas, which do not meet the classical criteria for hematoma formation described by Ford and McLaurin (20), were only exceptionally documented during the angiographic period (25). In recent CT series, they represent a significant proportion of the total, reaching 23% in one series (70). The mechanism of delayed hematoma formation is controversial, and possibilities that have been postulated are a venous origin, rupture of traumatic meningeal artery pseudoaneurysm, and arteriovenous shunting (10, 19, 24, 28, 66). Concurrent factors may be the presence of skull fracture at the hematoma site, transient intracranial hypotension due to energetic medical treatment (osmotherapy, hyperventilation), cerebrospinal fluid fistula, or surgical decompression, all of which would act to suppress the tamponade effect of ICP (5, 19, 39, 40, 44, 51, 60, 66). Two more contributing factors may be systemic arterial hypotension or increased central venous pressure during the immediate postraumatic period (5, 10, 19). Four of our patients with delayed epidural hematoma underwent evacuation of an epidural hematoma immediately after admission, producing a reduction in the initially raised ICP. Four more patients were admitted with hypovolemic shock secondary to associated extracranial injuries. Liberal use of follow-up CT in patients with severe head injury with one or more of these risk factors is recommended as a means of detecting delayed epidural hematomas.

Nonsurgical management has been proposed for extradural hematomas, acute or not, presenting either with minor neurological symptoms and signs or asymptomatically. With the exception of having a normal level of consciousness, cases suitable for nonsurgical management are not well defined and there is great variability in the criteria (volume, thickness, and location of the hematoma) proposed by different authors (7, 17, 29, 35, 56, 62, 63, 73, 78). Had we established as criteria for nonsurgical management in our noncomatose patients a stable neurological status, a GCS score of ≥13, and midline shift of <4 mm, at least 35% of our patients operated upon 13 to 48 hours after trauma and 50% of those operated later could have been managed nonsurgically. Average age and average hematoma volume were both lower in the patients who fulfilled these criteria. Arguments against nonsurgical management of these cases, apart from the theoretical risk of sudden decompensation and a higher morbidity risk linked to the chronic presence of the clot, are the longer hospitalization time (4.5 weeks on the average) and more frequent follow-up CT (3.5 times on the average) that it entails (13, 46). Prolonged hospitalization is necessary in patients managed conservatively because of the "expansile phase" of the clot, described by Pang et al. (56), which occurs 5 to 16 days after trauma. The average hematoma volume in our noncomatose patients who could have been managed nonsurgically was larger in those operated upon more than 72 hours after trauma than in those operated upon between 12 and 48 hours after trauma. Considering the low operative risks, we propose surgical treatment for these patients.

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COMMENTS

This report represents an extension of the authors' interest in the problem of extradural hematomas. In an earlier publication, the outcome in 82 patients with extradural hematomas before and after the introduction of computed tomography was compared (1). It was concluded that computed tomography significantly improved outcome by enabling the diagnosis of extradural hematoma to be made more rapidly. Now this same group has sought to identify factors that may affect outcome in patients with extradural hematomas demonstrated by computed tomography.

To do this, patients were grouped into those who were comatose and those who were noncomatose. In the former group, a CT scan was obtained immediately on admission; in the latter group, a scan was not done until an alteration in the level of consciousness, severe headaches, motor deficits, or pupillary changes occurred. It is likely that the group of patients who were comatose contained a significant number of patients with diffuse axonal injuries in addition to their extradural hematomas, as the authors acknowledge in their discussion (2). It is not wholly surprising under these circumstances that, even though comatose patients underwent the evacuation of their hematomas sooner after injury, their outcome was poorer.

The authors have succeeded in demonstrating the spectrum of problems posed by extradural hematomas. It is likely that the discussion promoted by this report will contribute to our understanding of those problems.

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1. Cordobes F, Lobato RD, Rivas JJ, Munoz MJ, Chillon D, Portillo JM, Lamas E: Observations on 82 patients with extradural hematomas: Comparison of results before and after the advent of computerized tomography. J Neurosurg 54:179–186, 1981.
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The authors have presented a detailed, exhaustive description of epidural hematomas at their institution. They have characterized the complex clinical behavior of epidural hematomas and separated the clinical course into subgroups. They demonstrated that the groups of patients who were comatose and noncomatose at the time of operation had significantly different outcomes. Their detailed analysis of the CT, clinical course, cranial nerve signs, neurological examination, size of hematoma, and other associated injuries in their large series makes this an excellent characterization of the clinical presentations and outcome of epidural hematomas.

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