TITLE: HYPERGLYCEMIA AND NEUROLOGIC OUTCOME IN PATIENTS WITH CLOSED HEAD INJURY

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Introduction. In humans, closed head injury (CHI) frequently is associated with evidence of ischemic injury,[1] despite well-preserved blood pressure and PaO_2. In animal models of global and focal cerebral ischemia, hyperglycemia increases both morphologic brain damage and neurologic deficit.[2,3] Hyperglycemia also increases the size of infarction in collaterally perfused territories but not in end-arterial infarctions.[4] Because of the relationship between hyperglycemia, varying degrees of cerebral ischemia, and poor neurologic outcome, we studied the effect of serum glucose on long-term neurologic function in patients with severe CHI (Glasgow Coma Scale $\leq$8) intracranial pressure.

Methods. We retrospectively reviewed data from 70 consecutive patients, all of whom had been entered in a prospective study of neurologic outcome following CHI. All required continuous monitoring of intracranial pressure (ICP). Excluded were: 1) patients <12 years, 2) those who expired or were termed "brain dead" within 48 hours of CHI, 3) diabetics. Each patient was evaluated on admission using the Glasgow Coma Scale (GCS) and Glasgow Trauma Scale. Blood glucose, age, sex, weight, ICP, blood pressure, and cranial computed tomographic (CT) scan results were recorded on admission. All patients were intubated and hyperventilated to maintain therapeutic hypocapnia. Treatment with mannitol, barbiturates, paralytic agents, steroids, and vasopressors was recorded for the first 7 days post-admission as were the BP, ICP, serum glucose, and serum osmolarity. Delayed elevations in ICP (beyond 7 days) and complications that could interfere with the patient's progress (e.g., sepsis and adult respiratory distress syndrome (ARDS)) were noted. Each patient's clinical course was followed and each was ranked 3 to 6 months post-CHI using the Glasgow Outcome Scale. Data were analyzed using non-parametric analysis of variance to answer the following questions: 1) Does the admission blood glucose correlate with the neurologic outcome at 6 months using the Glasgow Outcome Scale? 2) Does the admission blood glucose correlate with the admission Glasgow Coma Scale? 3) Does the admission blood glucose level correlate with the admission CT scan? To avoid statistical difficulties posed by the small numbers of patients in some individual categories, the following categories were combined for analysis. Outcome data were analyzed in terms of death, poor outcome (persistent vegetative state or severely disabled), and favorable outcome (moderately disabled or functional). Glasgow Coma Scales 3, 4, and 5 were combined as were 6, 7, and 8. CT scans were grouped according to absence of intracranial hemorrhage (normal or edema only) or presence of intracranial hemorrhage (subdural, epidural, intracerebral, or intraventricular).

Results. The admission blood glucose predicted outcome ($P<0.03$), primarily because of the high correlation between high blood glucose on admission and death (Table 1). The admission blood glucose did not correlate with GCS ($P>0.9$), but correlated strongly with the presence or absence of intracranial hemorrhage ($P<0.01$; Table 2).

Conclusions. These data demonstrate that a high admission blood glucose level following CHI is associated with a poorer neurologic outcome. This difference, which apparently is not associated with clinical status on admission as reflected in the GCS, is associated with the appearance of intracranial bleeding on CT scan. Further studies should determine if the high blood glucose level simply represents a physiologic response to more severe injury or independently worsens ischemic tissue injury following central nervous system trauma.

References.

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Table 1. **Admission Blood Glucose vs Outcome**

	N	Blood Glucose (means ± SD)
Dead	20	221 ± 94
Poor outcome	19	186 ± 46
Favorable outcome	31	173 ± 36

Table 2. **Admission Blood Glucose vs CT Scan**

		Results
	N	Blood Glucose
CT Scan		(means ± SD)
No Intracranial Hemorrhage	**29**	**166 ± 42**
(Normal)	16	163 ± 46
(Edema)	13	169 ± 38
Intracranial Hemorrhage	**41**	**207 ± 71**
(Subdural or epidural)	20	201 ± 48
(Intracerebral)	7	179 ± 54
(Intraventricular)	14	231 ± 99