

encouragement. Definitive answers will only come from a controlled trial, which is now being organized with the assistance of The National Multiple Sclerosis Society.

Peter C. Dau, M.D.

Howard L. Weiner, M.D.

Children's Hospital of San Francisco
San Francisco, CA

Intracranial hemorrhage associated with amphetamine use

To the Editor: The recent case report and thorough review provided by Delaney and Estes¹ suggest that there may be several etiologies to intracranial hemorrhage (ICH) associated with amphetamine use. A case of ICH with amphetamine use provides another example of this rare but devastating complication of amphetamine abuse and demonstrates that the etiology can, to some extent, be determined.

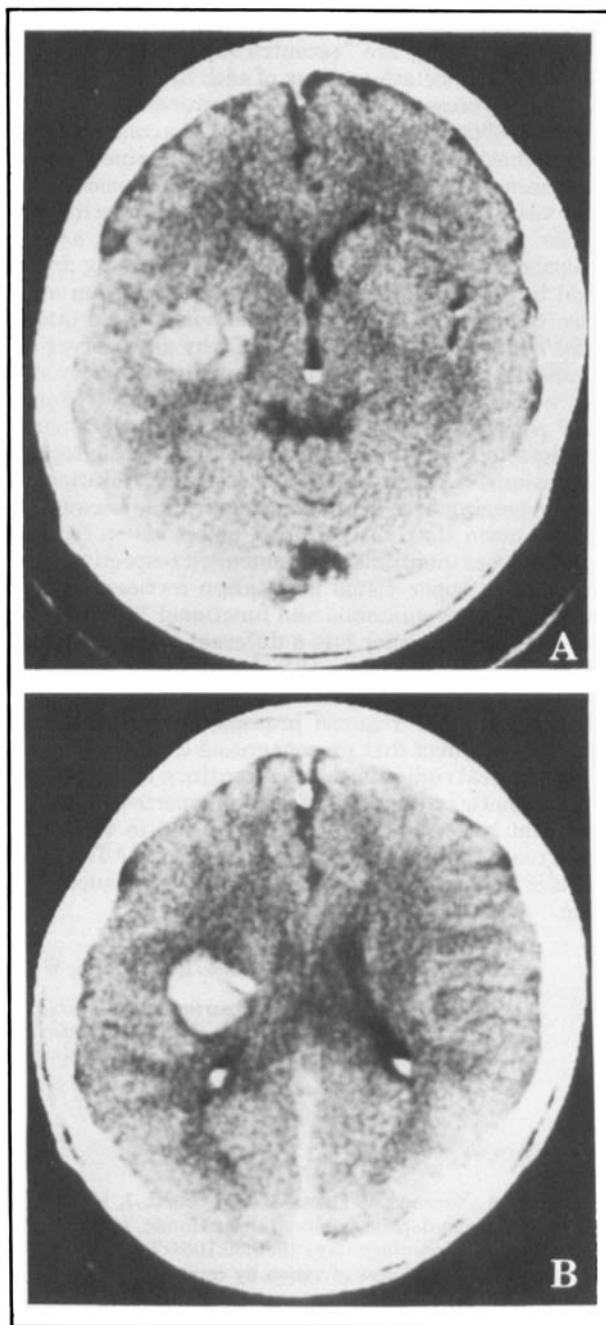
Case report. This 17-year-old single man was admitted with a dense left hemiparesis. According to the patient and witnesses, he was in his usual state of health when 3 hours before admission, he ingested two "black mollys" (40 mg of L- and D-l amphetamine tablets) with a can of beer. Forty minutes later he complained of severe headache and lost consciousness. He was taken home where he was put to bed; on waking 3 hours later, he continued to complain of headache and had a left hemiplegia.

In the emergency room, he had a supine blood pressure of 210/120 and a dense flaccid left hemiplegia. There was minimal nuchal rigidity. There was left hemisensory loss, and an extensor-plantar reflex on the left. He was oriented and alert and the remainder of the examination was negative. Past medical history and review of systems were unremarkable. There was no family history of hypertension, or previous history of headache or hypertension in the patient.

CT on admission, with contrast (figure, A and B), demonstrated a large intracerebral hematoma involving the right insular cortex and adjacent basal ganglia with some compression of the right lateral ventricle. There was no significant midline shift.

Other laboratory evaluations were within normal limits. Drug screen was negative except for the presence of amphetamine-like substances.

The next day, follow-up blood pressures were within normal limits and the patient made a gradual recovery, with minimal hemiparesis 3 weeks later. Two weeks after the first CT a second study showed resolving clot and reduction of edema around the hematoma. Transfemoral four-vessel cerebral angiography was normal except for displacement of vessels in the right middle cerebral distribution, consistent with a resolving clot. No evidence of an aneurysm or arterial-venous malformation was found.



Figure, A and B. CT of brain on admission, with contrast, showing large intracerebral hematoma involving right insular cortex and adjacent basal ganglia, with some compression of right lateral ventricle but no significant midline shift.

Included in the manifestations of amphetamine abuse are those of isolated but documented instances of permanent focal deficits associated with intracranial hematoma.² As our case and others have shown,³ ICH associated with amphetamine abuse need not be related to chronic, intravenous, or an "overdose effect" of the drugs.

As in this case, a relatively small dose of orally ingested amphetamine in an occasional abuser may have devastating effects. In our patient as in others, there may be a sudden hypertensive change in blood pressure and the site of the ICH is similar to that commonly seen in intracerebral hemorrhage due to chronic and poorly controlled hypertension.⁴ Perhaps the etiology in these two groups is the same, although we certainly agree with the authors that in many other instances the etiology may be more complicated.

We believe that it is important, however, to stress that a relatively small dose of amphetamine-like substances in only occasional abusers may nonetheless produce devastating results, including death from sudden lethal hypertensive changes.

Terrence D'Souza, M.D.

David Shraberg, M.D.

*Department of Neurology & Psychiatry
Tulane University School of Medicine
New Orleans, LA*

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Reply from the Author: With amphetamines being a common recreational drug, I suspect that there are many other cases of ICH with amphetamine use, though unrecognized because the history is often incomplete. The additional case provided by Drs. D'Souza and Shraberg conforms to the clinical picture that we described.

Finding elevated blood pressure (BP) in a patient with an acute stroke must be interpreted with caution. A common unexplained observation in stroke patients is an initial elevated BP with subsequent return to normal in 24 to 48 hours without treatment. The initial elevation may be cause or effect, and the relation to amphetamine use is only speculative.

Peyton Delaney, M.D.

*Department of Neurology
University of Texas Medical Branch
Galveston, TX*

Correction

"Skin Ultrastructural Changes in Hallervorden-Spatz Syndrome," April [Suppl] p. 93. Add Margaret L. Grunnet to the names of the authors.

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Skin Ultrastructural Changes in Hallervorden–Spatz Syndrome

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