

CEREBRAL HEMATOMA WITH INTRAVENTRICULAR BLEEDING

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***A case of intracerebral hematoma is presented showing intraventricular bleeding on the contralateral side. At postmortem examination, a bloody cast of the ventricular system was demonstrated as well as the hematoma.***

The extensive use of brain scanning as an initial diagnostic procedure in evaluating the patient with a cerebrovascular accident has been a source of interesting interpretive exercises and of unusual results arising from the abnormal pathologic physiology. Such an instance is exemplified by the following case report.

CASE REPORT

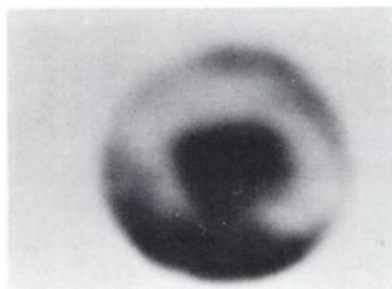
A 55-year-old man was admitted from the Emergency Room at 11:10 pm on September 4, 1973 because of the sudden onset of aphasia and left hemiparesis 2 hr prior to admission. This occurred while standing in front of his home when he felt weak, fell to the left side, and had one episode of vomiting. His past history was significant only in the fact that he had had rheumatic fever in childhood and was on Digoxin for 1 year. On examination his blood pressure was 180/100 and there was a left hemiparesis with aphasia. A positive Babinski reflex was elicited on the left. A lumbar tap yielded fluid containing many red blood cells but no xanthochromia. There was a question as to the possibility of a traumatic tap. At approximately 11:00 am on the following day, a brain scan was performed on a Searle HP scintillation camera after injection of 12 mCi of <sup>99m</sup>Tc-DTPA using the low-energy converging collimator. The image was recorded on 70-mm film using a defocused dot. The right lateral view (Fig. 1) was interpreted as a large temporal lesion, most likely a cerebral

hematoma. The left lateral view (Fig. 2) was unusual in that the ventricular system was well outlined. The anterior and posterior views were not technically adequate for interpretation. An attempt to obtain a repeat scan after 4 hr was prevented by the death of the patient at approximately 1:00 pm.

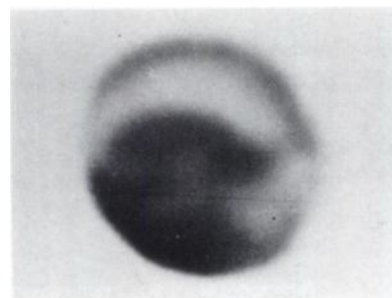
At postmortem examination, there was a large hematoma of the right cerebral hemisphere destroying portions of the basal ganglia, particularly later-

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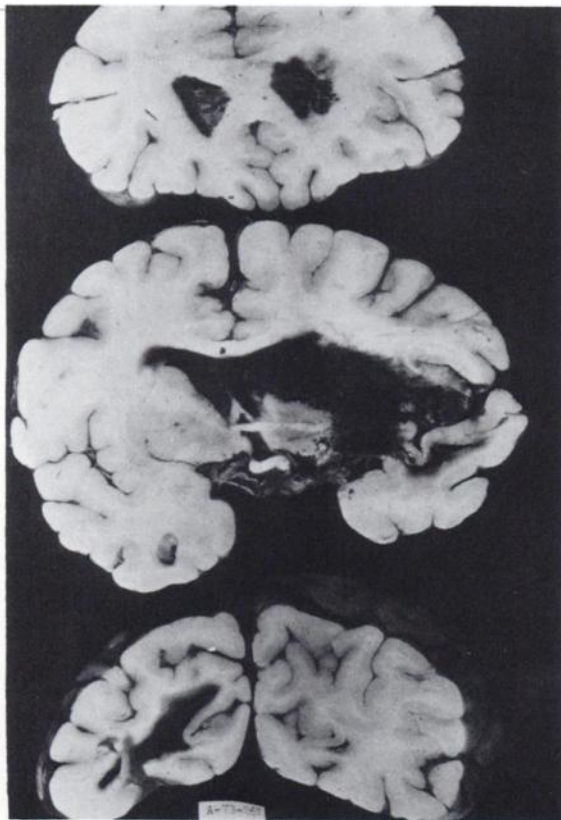
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**FIG. 1.** Right lateral brain scan showing hematoma. Frontal region is to right.



**FIG. 2.** Left lateral brain scan showing ventricular outline. Frontal region is to left.



**FIG. 3.** Coronal sections of brain showing hematoma with rupture into ventricle.

ally. Rupture of the hematoma into the ventricular system had occurred with blood present in both right and left lateral ventricles. There was a shift of the midline from right to left (Fig. 3).

#### DISCUSSION

Primary intracerebral hemorrhage occurs in approximately 10% of all strokes (1). Those that show rapid progression to death are more likely arterial in origin. The common sites for massive cerebral hemorrhage of hypertensive origin are: (A) the lentiform nucleus, (B) the deep white matter of the cerebellar hemispheres, and (C) the pons and midbrain (2). Lentiform nucleus hemorrhage may be divided into the medial ganglionic type which is usually fatal and the lateral ganglionic type which the patient may survive. The medial type arises medial to the putamen and usually destroys the hypothalamus and ruptures into the ventricle. With flooding of the ventricular system, there is usually a marked elevation in temperature.

It is likely that this case fits into the category of the medial type. The estimated time of injection of the radiopharmaceutical was some 12 hr after onset of the hemorrhage, a time when there was still active bleeding. Thus, the isotope traced the course of the blood into the ventricular system. In retrospect then, the lumbar tap was not traumatic with the red cells arising from the rupture into the ventricle. The lack of xanthochromia was due to the short interval between onset of symptoms and death and this would also explain the lack of temperature elevation.

#### REFERENCES

1. TOOLE JF, PATEL AN: *Cerebrovascular Disorders*, New York, McGraw-Hill, 1967, p 246
2. BLACKWOOD W, et al: *Greenfield's Neuropathology*, Baltimore, Williams & Wilkins Co, 1963, p 124