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## **Acute Isodense Subdural Hematomas: A Problem in Anemic Patients**

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# Acute Isodense Subdural Hematomas: A Problem in Anemic Patients

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Although numerous articles have appeared in the literature on chronic isodense subdural hematomas, acute isodense subdural hematomas have received little attention. An experimental model was developed that demonstrated that blood with reduced hemoglobin concentration, 8–10 g/dl, is isodense with the brain. Two anemic patients with acute isodense subdural hematomas are reported.

Isodense or isoattenuating subdural hematomas have proven to be a difficult diagnostic problem and have been the subject of numerous reports [1–13]. The time that a chronic subdural collection may become isodense on computed tomography (CT) varies from 1 week to 90 days after trauma [3, 5, 8]. In most instances, it appears isodense in 2–6 weeks [6]. There has been little mention of isodense subdural hematomas in the acute clinical setting. In patients with low hemoglobin levels, an acute subdural hematoma may be isodense from the outset. We developed an experimental model to define the range of hemoglobin concentration that would be isodense with the brain in the acute setting. We also encountered two anemic patients whose hemoglobin levels fell within the experimentally derived limits of the isodense range and who had acute isodense subdural hematomas.

## Materials and Methods

We initially evaluated 25 CT head scans. The average CT number of normal cortex adjacent to the inner table of the skull was 32–48 Hounsfield units. Gelatin was mixed with water in various concentrations, with ultimate density approximating this normal cortical density. This gelatinous mixture was then poured into a specially constructed cylindrical plastic phantom with nylon sampling chambers (fig. 1). The mixture was allowed to harden, thus providing a standard for visual comparison. By mixing a constant volume of whole blood with progressively increasing amounts of normal saline, blood samples of different hemoglobin concentrations were obtained. The hemoglobin concentration was evaluated using standard capillary tube and centrifuge techniques. Hemoglobin concentrations of 8–18 g/dl were placed in the sampling chambers in the phantom and scanned.

Scans were obtained at 124 kV, 200 mA, and 9.8 sec scan time. The samples were immediately scanned and measured directly. When clot retraction occurred, all measurements were obtained through the clot itself rather than through the serum. All scans including the 25 normal CT scans were made on a G.E.CT/T 8800 scanner.

## Results

The CT unit values observed for the different concentrations of hemoglobin are shown in figure 2, where the CT number is plotted as a function of time. The dark region represents the range of normal cortical density. Each curve represents the values obtained for particular hemoglobin concentrations. Initially at 0 hr, hemoglobin values of 8–11 g/dl were in the isodense range. Hemoglobin

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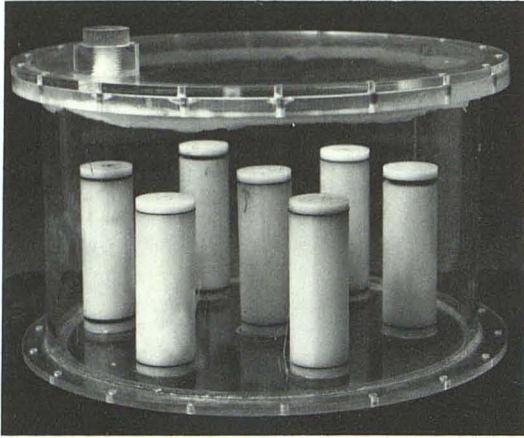
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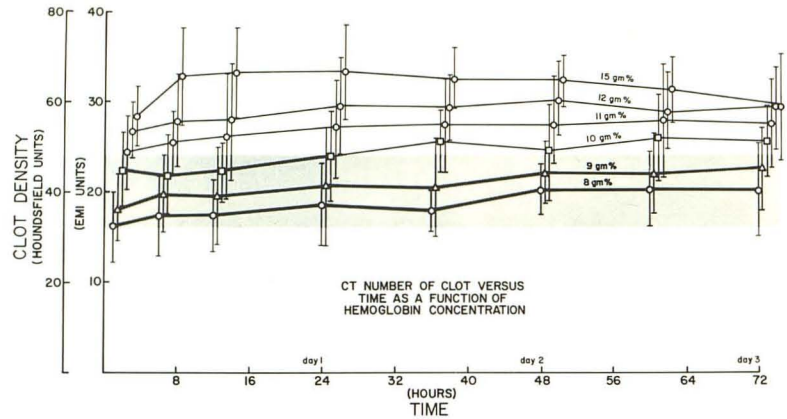
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Fig. 1.—Plastic phantom has seven nylon sampling chambers into which blood with various hemoglobin concentrations was placed. Gelatinous mixture with same density as normal brain cortex surrounds chambers.



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Fig. 2.—Dark region represents range of normal cortical density. Initially, blood with hemoglobin values of 8, 9, 10 and 11 g/dl was in isodense range. Samples of 8, 9, and 10 g/dl hemoglobin remained in isodense range for first 24 hr. Thereafter, only hemoglobin values of 8 and 9 g/dl were in isodense range.

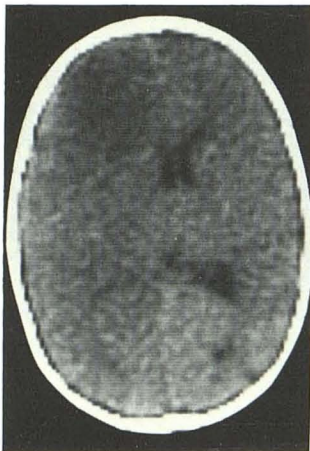


Fig. 3.—Case 1, 9-month-old boy. Noncontrast scan. Poorly defined area of decreased density in right frontal region with associated right-to-left shift of ventricular system. At operation a 30 ml freshly clotted subdural hematoma was found in right frontal region.

concentrations of 8, 9, and 10 g/dl remained isodense during the first 24 hr. Thereafter, only the 8 and 9 g/dl hemoglobin samples were in the isodense range. By 4 hr, discrete clots were noted in all samples. Clot retraction was complete by 48–72 hr. With the exception of the 15 g/dl sample, all samples showed progressive increase in the density through the sampling period, but by 72 hr, these measurements tended to plateau out. The density of the 15 g/dl sample of hemoglobin peaked at 16 hr, following which there was a slight progressive fall.

### Representative Case Reports

#### Case 1

A 9-month-old boy was admitted after he hit his forehead on the dashboard in an automobile accident. Physical examination revealed no lacerations or other external signs of injury. He was unresponsive, and his right pupil was fixed and dilated. The anterior fontanelle was tense. The rest of the physical examination was unremarkable. Noncontrast CT scan 2 hr after the accident dem-

onstrated a poorly defined area of decreased density in the right frontal region with a right-to-left shift of the ventricular system (fig. 3). The patient's admission hemoglobin was 9.2 g/dl.

Because of his deteriorating clinical condition the patient was taken to the operating room for decompressive surgery. A 30 ml freshly clotted subdural hematoma was found and evacuated in the right frontal region. His recovery was uneventful.

#### Case 2

A 46-year-old male alcoholic was admitted in a confused and disoriented state. There were no external signs of head trauma. The pupils were equal but sluggishly reactive to light. He exhibited a mild right hemiparesis. The rest of his physical examination was unremarkable.

Computed tomography demonstrated a large left-to-right shift of the midline structures due to a chronic subdural hematoma on the left side. There was no enhancement after intravenous injection of iodinated contrast material (fig. 4A). He was taken to the operating room and multiple left-sided burr holes were placed, evacuating the chronic subdural hematoma which drained without difficulty. Postoperatively he did well for 3 days; he started talking and his hemiparesis cleared.

However, on postoperative day 4, he became obtunded with right hemiplegia. Computed tomography again demonstrated a left-to-right shift of the ventricular system. However, no apparent cause for the shift was evident on the scan. A postcontrast scan did not provide any more information (fig. 4B). Left carotid angiography demonstrated a large left-sided extracerebral collection (fig. 4C). The patient was taken back to the operating room, where a 110 ml freshly clotted subdural hematoma was found and evacuated from the left frontal parietal region. The patient did well postoperatively, remaining neurologically intact. His hemoglobin prior to the second surgery was 10.0 g/dl.

### Discussion

Computed tomography has been established as the diagnostic procedure of choice in the management of head

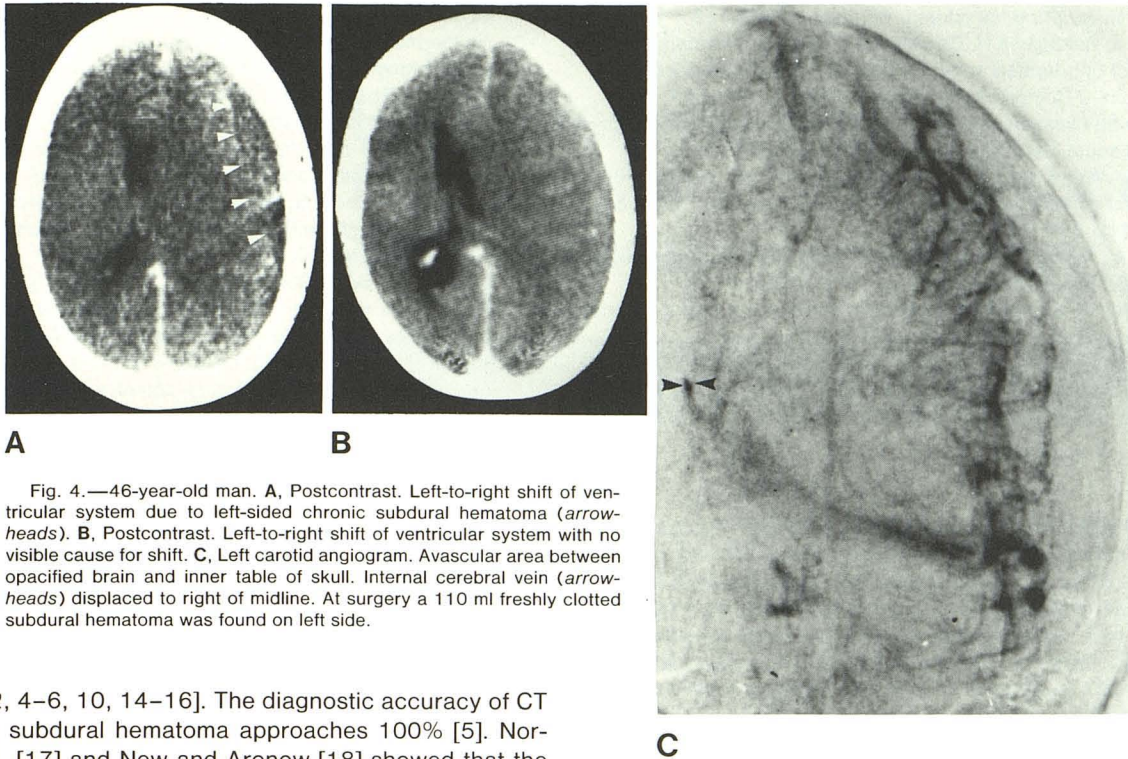


Fig. 4.—46-year-old man. **A**, Postcontrast. Left-to-right shift of ventricular system due to left-sided chronic subdural hematoma (arrowheads). **B**, Postcontrast. Left-to-right shift of ventricular system with no visible cause for shift. **C**, Left carotid angiogram. Avascular area between opacified brain and inner table of skull. Internal cerebral vein (arrowheads) displaced to right of midline. At surgery a 110 ml freshly clotted subdural hematoma was found on left side.

trauma [2, 4–6, 10, 14–16]. The diagnostic accuracy of CT for acute subdural hematoma approaches 100% [5]. Norman et al. [17] and New and Aronow [18] showed that the density of a hematoma is directly related to its hemoglobin concentration. Thus, in the acute stage, a subdural hematoma containing fresh blood has a higher density than normal brain. With increase in clot age and breakdown of the hemoglobin, there is a decline in the attenuation values so that in the chronic phase the density of the subdural hematoma approaches that of cerebrospinal fluid and appears as an area of diminished density. Subdural hematomas pass through an isodense phase in the subacute to chronic period, when the absorption coefficient is the same as that of the adjacent normal brain. The time when they become isodense varies from 1 week to 90 days after the initial trauma, usually 2–6 weeks [3, 5, 8]. These so-called isodense subdural hematomas have received much attention because they pose a particular diagnostic problem in that they are not readily identified on CT.

Another difficult problem occurs in anemic patients when an acute hemorrhage may be isodense from the outset. Apart from the work of New et al. [18] and a single case report by Kasdon et al. [19] of a nontraumatized, anemic patient with an isodense cerebellar hemorrhage, there has been little mention of acute isodense hematomas in patients with anemia.

Our experimental model showed that hemoglobin values of 8–10 g/dl will be isodense in relation to the adjacent brain. Our two cases had surgically proven acute subdural hematomas. The CT scans in these two patients did not demonstrate the subdural hematoma directly, although a mass effect was evident in each case. The hemoglobin levels in these two patients fell in this experimentally determined range.

A striking feature of our experimental study was the

progressive increase in density of the hematoma during the first 72 hr. Clot retraction, which was also a feature of our experiment, was well documented by Bergstrom et al. [20] and is the major factor in the initial increase in density of an acute subdural hematoma. Dublin et al. [5] noted that acute subdural hematomas may also increase in density during the first 1–3 days after trauma.

An acute subdural hematoma in an anemic patient may be isodense on CT from the outset and pose difficult diagnostic problems. The clinician and radiologist dealing with traumatized patients should be aware of this possibility.

#### REFERENCES

1. Amendola MA, Ostrum BJ. Diagnosis of isodense subdural hematomas by computed tomography. *AJR* 1977;129:693–697
2. Cornell SH, Chiu LC, Christie JH. Diagnosis of extracerebral fluid collections by computed tomography. *AJR* 1978;131:107–110
3. Davis KR, Taveras JM, Roberson GH, et al. Some limitations of computed tomography in the diagnosis of neurological diseases. *AJR* 1976;127:111–123
4. Davis KR, Taveras JM, Roberson GH, et al. Computed tomography in head trauma. *Semin Roentgenol* 1977;12:53–62
5. Dublin AB, French BN, Rennick JM. Computed tomography in head trauma. *Radiology* 1977;122:365–369
6. French BN, Dublin AB. The value of computerized tomography in the management of 1000 consecutive head injuries. *Surg Neurol* 1977;7:171–183
7. Hayman LA, Evans RA, Hinck VC. Rapid-high-dose contrast computed tomography of isodense subdural hematoma and cerebral swelling. *Radiology* 1979;131:381–383
8. Kim KS, Hemmati M, Weinberg PE. Computed tomography in

- isodense subdural hematoma. *Radiology* **1978**;128:71-74
9. Lusins J, Nakagawa H, Bender MB. Computer assisted tomography of unoperated subdural hematoma: short and long term follow-up. *J Comput Assist Tomogr* **1978**;2:460-466
  10. Merino-deVillasante J, Taveras JM. Computerized tomography (CT) in acute head trauma. *AJR* **1976**;126:765-778
  11. Messina AV. Computed tomography: contrast media within subdural hematomas. A preliminary report. *Radiology* **1976**;119:725-726
  12. Moller A., Ericson K. Computed tomography of isoattenuating subdural hematomas. *Radiology* **1979**;130:149-152
  13. Svendsen P. Computer tomography of traumatic extracerebral lesions. *Br J Radiol* **1976**;49:1004-1012
  14. Harwood-Nash DC, Fitz CR. *Neuroradiology in infants and children*. St Louis: Mosby, **1976**;:789-854
  15. Taveras JM, Wood EH. *Diagnostic neuroradiology*. Baltimore, MD: Williams & Wilkins, **1976**;:1047-1090
  16. Zimmerman RA, Bilaniuk LT, Gennarelli T, et al. Cranial computed tomography in diagnosis and management of acute head trauma. *AJR* **1978**;131:27-34
  17. Norman D, Price D, Boyd D, Fishman R, Newton TH. Quantitative aspects of computed tomography of the blood and cerebrospinal fluid. *Radiology* **1977**;123:335-338
  18. New PFJ, Aronow S. Attenuation of measurements of whole blood and blood fractions in computed tomography. *Radiology* **1976**;121:635-640
  19. Kasdon DL, Scott RM, Adelman LS, Wolpert SM. Cerebellar hemorrhage with decreased absorption values on computed tomography: a case report. *Neuroradiology* **1977**;13:265-266
  20. Bergstrom M, Ericson K, Levander B, et al. Variation of the attenuation values of intracranial hematomas. *J Comput Assist Tomogr* **1977**;1:57-63