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High Frequency CT Findings within 24 Hours after Cerebral Infarction

Susan D. Wall[1]
Michael Brant-Zawadzki[1]
R. Brooke Jeffrey[1]
Barbara Barnes[2]

Twenty-six patients with clinically documented acute cerebral infarction were evaluated by computed tomography within the first 24 hr. In 21 patients, subtle mass effects and/or focal areas of decreased attenuation corresponding to areas of clinical deficit were demonstrated. Enhancement occurred in only five of 15 infarctions rescanned after contrast administration. In three of these, the region of infarction became isodense after contrast administration. In a patient with multifocal infarcts, enhancement was the only clue to infarction in one focus. The improved spatial and contrast resolution of current generation scanners appears to have significantly increased the sensitivity of computed tomography in demonstrating acute cerebral infarction and has important clinical application.

Although prior authors have described the computed tomographic (CT) evolution of cerebral infarction [1–4], little is available regarding its appearance within the first 24 hr. Most investigators state that CT scanning is unrevealing within the first 48 hr in the majority of cases [2, 5–8]. Follow-up CT scans are often needed after an initial "negative" study to document the site and extent of infarction. Despite reportedly low diagnostic sensitivity in the first days, early CT scanning in patients with acute stroke has been of clinical value in excluding intracranial hemorrhage due to aneurysm or other lesion, thereby allowing the institution of anticoagulant therapy. Such therapy has found its place in the setting of ischemic neurologic events [9]. An early scan has been helpful also in excluding other pathology in those patients with infarction who present a diagnostic dilemma by their atypical clinical appearance.

Because of the improved spatial and contrast resolution of the current scanners, we undertook a retrospective study to reevaluate the CT findings within 24 hr of cerebral infarction. Early accurate CT diagnosis could direct the appropriate therapy and reduce the number of follow-up studies, thus improving the care of these patients and reducing the cost.

Materials and Methods

The medical records were reviewed of all patients with clinically documented acute cerebral infarction over an 8 month period. Our selection criteria excluded those patients with transient ischemic events. Twenty-six patients had CT scans within 24 hr of ictus. Because time of ictus may be difficult to establish with precision, age of infarct at the time of CT (routinely recorded) was determined by the number of hours since the most recent reliable report of the patient being well. Chart review and, when possible, interview with the admitting physician established the time when the patient was last observed by a responsible adult to have been symptom-free (and/or a report of same by the patient himself). The selection criteria were strict and in some cases exaggerated the age of infarct. For instance, ictus at times occurred after bedtime but before waking in the morning, in which case the infarct was aged from bedtime to hour of CT scan. If firm historical data of well-being within 24 hr before CT could not be established, the patient was excluded from the study.

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[1] Department of Radiology, University of California School of Medicine, San Francisco, and San Francisco General Hospital, 1001 Potrero Ave., San Francisco, CA 94110. Address reprint requests to M. Brant-Zawadzki.

[2] Department of Neurology, University of California School of Medicine, San Francisco, and San Francisco General Hospital, San Francisco, CA 94110.

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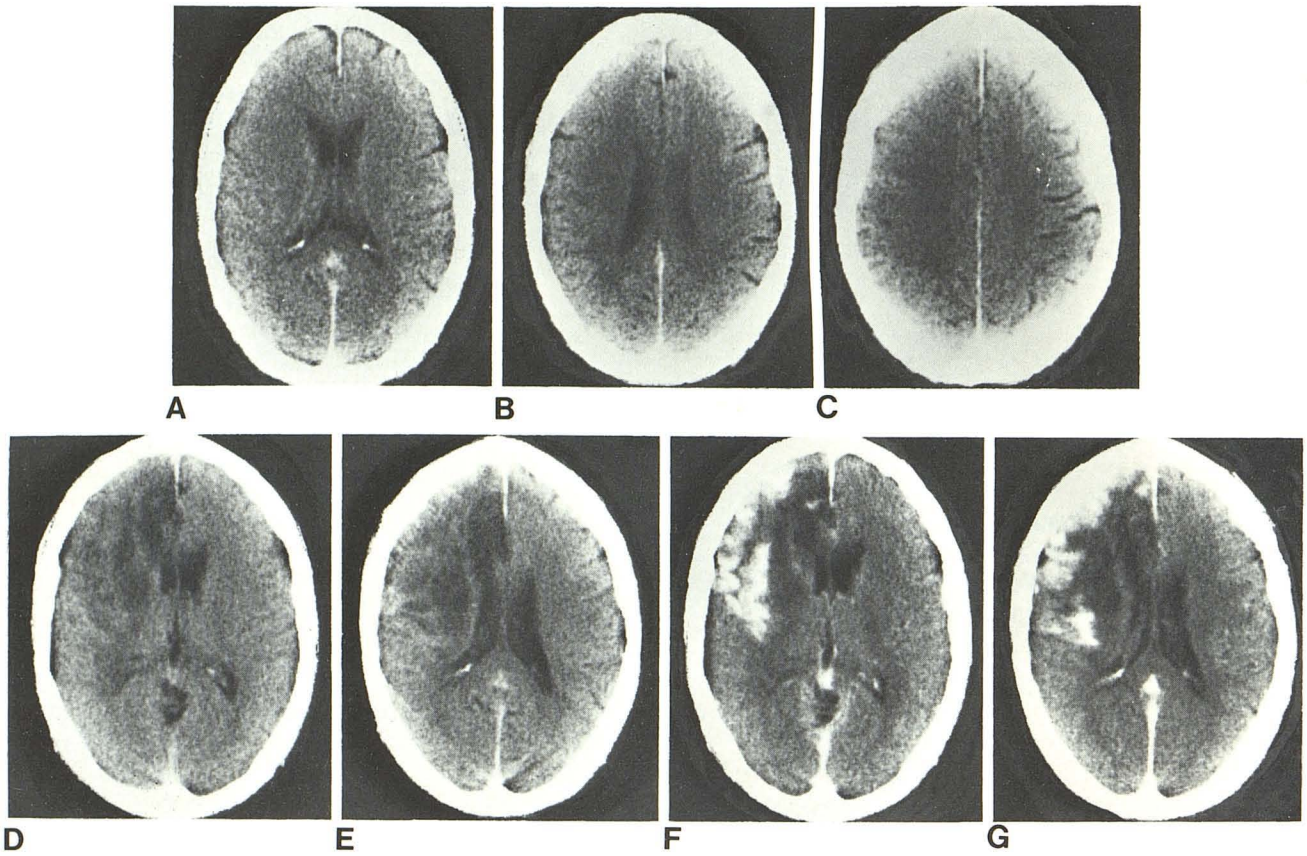


Fig. 1.—Infarct with mass effect only, case 1, 55-year-old man with 12 hr of left hemiparesis. **A–C,** Effacement of right convexity sulci on three contiguous levels. Opercular sulci were effaced on lower cut (not shown). Scans 12 days later without (**D** and **E**) and with (**F** and **G**) contrast confirm anterior and middle cerebral infarction. Mass effect has progressed; hypodensity and marked contrast enhancement has developed.

All scans were obtained with a G.E. 8800 scanner using 9.6 sec scan time. All patients had noncontrast scans and 20 patients had scans following intravenous contrast administration. Contiguous 10 mm thick axial cuts were obtained throughout the entire cranial vault. Hypaque 30% was administered via 150 ml intravenous bolus followed by 15 ml drip infusion. Infusion generally required 10 min for completion and repeat scanning was initiated 5 min after bolus. Eight patients had follow-up scans before discharge. Four patients died and autopsy findings were available in three.

The CT findings were reviewed retrospectively without knowledge of the initial report and with history limited to "possible stroke." The spectrum of findings was analyzed and categorized according to a grading system established for communication between the authors. Mass effect of increasing severity was graded by effacement of sulci only, sulci and nearby cistern, or sulci, cistern, and ventricle. Decreased attenuation (hypodensity) was graded as none, mild, moderate, or marked. Contrast enhancement was coded as contrast not given, none, isodense (relative to normal brain parenchyma), or hyperdense.

The 26 patients were 50–93 years old. Ten were women and 16 were men. Hemiparesis was the major clinical feature in 23 of the 26. Twelve had a history of cardiac arrhythmias or recent myocardial infarction. Proof of acute cerebral infarction was based on compelling clinical evolution or autopsy.

Results

Among the 26 patients, three had bilateral areas of infarction (detected by CT in two and at autopsy in one). Thus, a total of 29 infarcts occurred in the 26 patients. Positive CT findings in an area corresponding to the neurologic symptoms were seen in 23 (79%) of the 29 infarcts. One patient had acute CT findings of infarction in a clinically silent area in addition to CT abnormalities in the region of clinical deficit (autopsy proven). Both cortical and deep hemispheric infarcts were demonstrated within the 24 hr period.

At the time of CT, the interval from onset in the 23 detected lesions was 2–24 hr. Of the 29 infarcts, there were positive CT findings within the first 12 hr in 15. Three of these were under 6 hr old.

Middle cerebral artery distribution infarctions predominated in this series, although posterior cerebral and anterior cerebral infarcts also were demonstrated. The most common CT findings were areas of decreased attenuation within the gray matter, effacement of opercular and/or convexity sulci, and subtle mass effect on adjacent cisterns (figs. 1–6). Since asymmetry of sulci or sylvian cistern can be seen on a single CT section in normal patients, an infarct was diagnosed on this finding alone only if at least three contiguous sections showed effacement (fig. 1). Of the 23 CT proven infarcts, 18 had signs of mass effect. Effacement of sulci only was seen in four cases, sulci and adjacent cisterns in seven, and sulci, cistern, and ventricle in seven. Seventeen infarcts showed low density areas. In 13 cases the two findings of mass effect and diminished attenuation were associated. The only finding suggesting infarction was effacement of sulci in five cases and diminished density in four.

Contrast enhancement occurred in five lesions within 24 hr, becoming isodense and obscurring the low-density region in three. However, in one patient with multifocal infarcts, it was the only clue to infarction in one of the foci (fig. 3). Table 1 demonstrates the

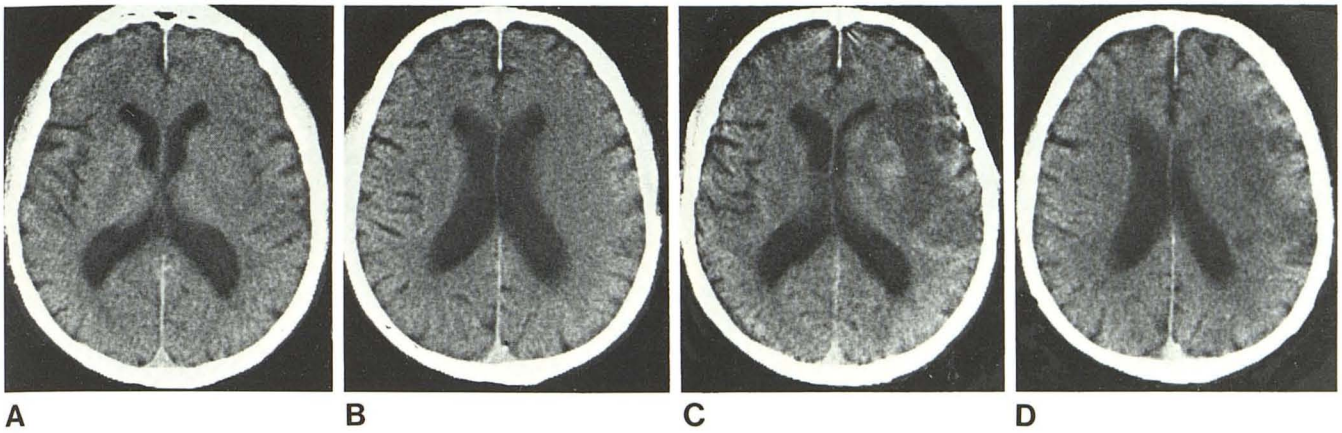


Fig. 2.—Infarct with mass effect only, case 4, 72-year-old man with atrial fibrillation and sudden right hemiparesis. **A** and **B**, 3 yr after ictus. Subtle effacement of convexity and opercular sulci on left. Coumadin was begun immediately. **C** and **D**, Follow-up study without contrast 4 days later. Hemorrhagic component within infarcted region, now hypodense and exhibiting markedly increased mass effect.

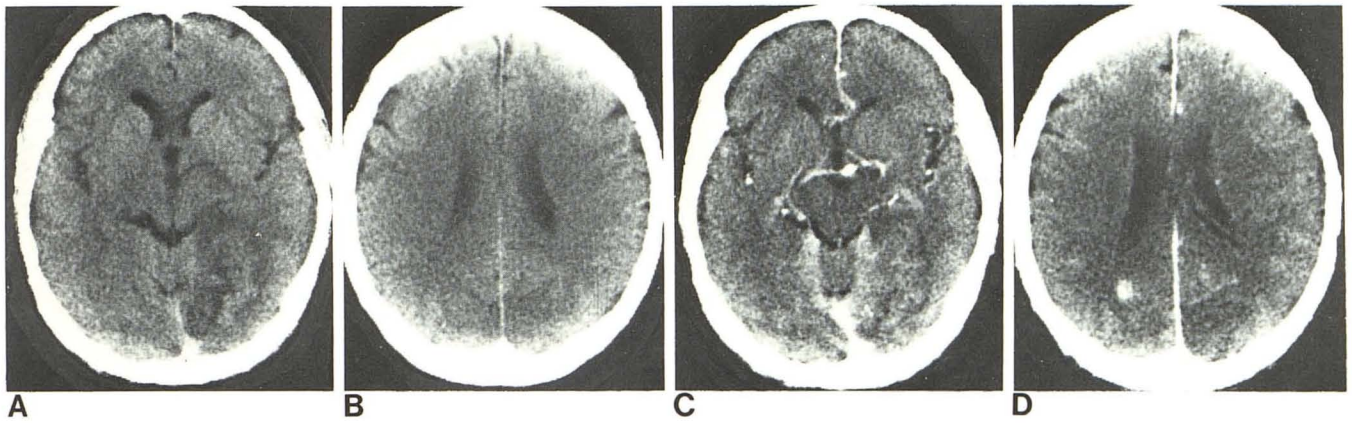
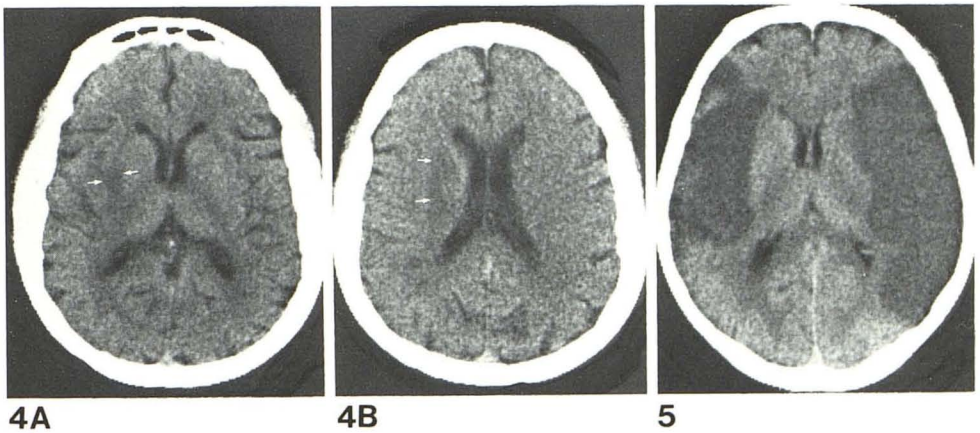


Fig. 3.—Infarct as lucency obscured by contrast material, case 3, 61-year-old man with bilateral blindness and right hemiparesis. **A** and **B**, After 8 hr, without contrast. Vague lucency in left occipitotemporal region; no obvious abnormality on right. **C** and **D**, Contrast infusion obscures left-sided lesion, but reveals abnormal enhancement in right occipital lobe on high cut (see text).

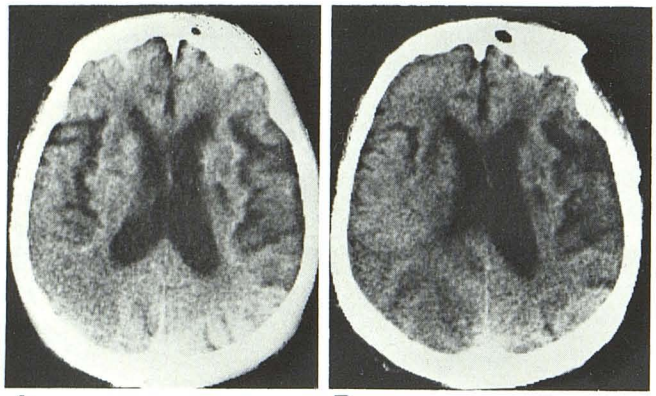
Fig. 4.—Infarct as lucency, case 19, 75-year-old woman with left hemiparesis of less than 24 hr. Adjacent, noncontrast scans. Subtle lucency in right basal ganglia (*arrows*). Minimal effacement of opercular sulci.

Fig. 5.—Infarcts as lucencies, case 20, 73-year-old woman found comatose, who had been well 12 hr before. Large, low density areas in both middle cerebral artery distributions proved to be infarcts at autopsy. Sulci absent and ventricles small relative to patient's age.



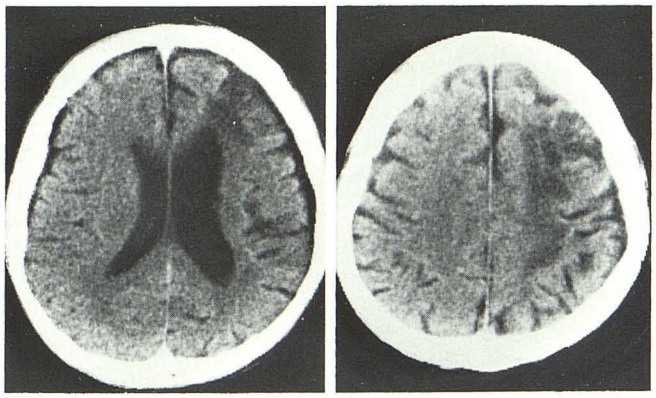
marked variability in combination of findings along each spectrum of mass effect, hypodensity, and enhancement.

Six patients showed false-negative scans. At the time of CT the age of these infarcts was 2–14 hr. One patient had a positive follow-up scan but negative initial scan at 2 hr. Two patients with false-negative scans were again negative when restudied, one on day 5 and one on day 8 postictus. Two other patients had multiple old infarcts, making the CT diagnosis of a clinically acute event more



A B

Fig. 6.—Infarct as mass effect, case 17, 73-year-old woman. **A,** 12 hr after sudden onset of left hemiparesis. Effacement of right posterior sylvian and occipital sulci. **B,** Repeat study 3 days later. Low density and increased mass effect in this region. Old deep hemispheric infarcts show more striking low density, but no mass effect.



A B

Fig. 7.—Previous infarction, recent infarction not detected in 66-year-old man. Recurrent right hemiparesis developed less than 15 hr before CT. Gross low attenuation in left watershed distribution. Ipsilateral enlargement of sulci and ventricles suggested atrophy, indicating chronicity of lesion. Recent acute infarction not identified.

TABLE 1: **CT Findings after Cerebral Infarction**

Case No.	Age of Infarct (hr)	Mass Effect	Hypodensity	Enhancement*
1	12	S, C	None	CNG
2	3	S, C, V	None	Hyperdense
3	18	Left: S, C, V;	Mild	Isodense
		Right: none	None	Hyperdense
4	3	S, C, V	None	CNG
5	12	S only	Moderate	Isodense
6	12	S, C, V	Moderate	CNG
7	8	S, C	Mild	None
8	12	S only	Mild	None
9	12	None	Moderate	Isodense
10	24	S, C	Moderate	CNG
11	2	None	Mild	None
12	12	S, C	Mild	None
13	24	S, C	Mild	None
14	24	None	Moderate	None
15	12	S, C, V	Mild	None
16	24	S, C	Mild	None
17	12	S, C	None	None
18	23	None	Mild	None
19	24	S only	Mild	CNG
20	12	Left: S, C, V;	Marked	CNG
		Right: S, C, V	Marked	CNG
21	10	Left: S only;	None	CNG
		Right: none†	None	CNG

Note.—S = sulci, C = cistern, V = ventricle, CNG = contrast not given.
* Isodense and hyperdense are relative to normal brain parenchyma.
† Right infarct missed on CT, proven at autopsy.

difficult despite comparison with prior studies (fig. 7). One area of infarction (in a patient with positive contralateral CT findings) was missed on CT at 9 hr, but was positive at autopsy.

Discussion

Previous reports have indicated that CT has a diagnostic sensitivity of up to 84% in the detection of cerebral infarction provided the study is obtained at least 48 hr *after* the ictus [7]. The diagnostic sensitivity of CT within the first 2 days of infarction has been disappointing. Previous authors using earlier generation CT scanners indicate positive findings in only a few cases [2, 5–8] with sensitivities as low as 10% within 48 hr of infarction [7].

Our experience with a current generation scanner indicates that the improved spatial and contrast resolution capabilities allow earlier detection of cerebral infarction. Of the studies within 24 hr after onset of symptoms, 79% showed positive CT findings in a region correlating with the major clinical deficits. Of particular note is the fact that of all the positive scans, 65% were obtained at or less than 12 hr after infarction.

The early CT findings of infarction in our study—focal low attenuation within gray matter and/or subtle mass effect—correlate well with the stroke models of different animal species. These models demonstrate an increased volume of water content of infarcted brain tissue within the first several hours after insult [10]. These experimental data suggest that prolonged anoxia initially disturbs transmembrane ion homeostasis, possibly via abnormalities of the sodium/potassium pump [11]. The altered cell membrane permeability causes an influx of sodium and water from the extracellular to the intracellular compartment resulting in cellular edema [12]. An osmotic gradient is established between the extracellular tissue and the intravascular space, causing transudation of water from capillaries into the interstitium. An overall increase in water content of 3% by weight has been found within the first 4 hr after experimentally produced ischemia in the region affected [11, 13].

It is plausible that subtle mass effects demonstrated by early CT scanning in our patients are the result of edema from these early fluid shifts. The correlation between increased water content and lower CT attenuation number has been established in both experimental models of infarction [1, 14] and autopsy studies [15]. Of note is that the breakdown of the blood-brain barrier generally does not occur within the first 4 hr after infarction in experimental

models. Such disruption is generally seen after the first 12–24 hr of acute infarction [11, 13]. Once the barrier is disrupted, significant macromolecular leakage occurs from the intravascular space. Interstitial edema progresses when hydrostatic pressure is reestablished via reperfusion of the capillary bed [11, 16]. Because of the delay in the breakdown of the blood-brain barrier, it is not surprising that contrast enhancement within the first 24 hr of infarction may be minimal. At times it is sufficient only to mask the subtle low density existing on noncontrast scans (fig. 3). This "masking" effect of contrast material in early infarction has also been seen in an experimental baboon model of infarction [1].

Double-dose contrast studies have been advocated recently for patients following stroke to help identify those with significant blood-brain barrier breakdown [17]. However, we believe that newer generation scanners can identify these changes often enough on the basis of mass effect and lucency on the precontrast scan (fig. 5) that the possible untoward effects of high dose hyperosmolar contrast agents can generally be avoided.

The failure to visualize acute infarction with CT (as in our five patients with negative scans) may be due to several factors. One important factor is infarct size. A punctate infarct may produce striking clinical deficits if located in the internal capsule or brain stem. Yet this lesion may be missed on CT because of its small size. Only one of our patients who had a negative scan initially showed positive findings on subsequent study. In this patient the initial study was obtained within the first 2 hr. (Of interest, we were able to detect deep lacunar infarcts within the internal capsule in three patients studied about 32 hr after ictus. Although these patients did not meet the criteria for inclusion in our study, they do demonstrate the ability of CT to diagnose even small infarcts in the basal ganglia region.) Another difficulty in the CT diagnosis of acute infarction derives from multiple pre-existing infarcts. The subsequent distortion of anatomy and altered brain density may cause a confusing CT appearance, especially if prior scans are unavailable.

Early CT evaluation of suspected cerebral infarction is clinically important. It is helpful not only in establishing a specific diagnosis, but in excluding hemorrhage (fig. 2), neoplasm, and other significant pathologic entities. It precludes inappropriate invasive studies such as angiography. CT is especially helpful in patients who have a diagnostic dilemma because of an atypical clinical presentation. Once a CT diagnosis is made, subsequent studies may be unnecessary, particularly if there is a clear-cut clinical course.

The improved sensitivity of current generation CT scanners shown by our study should positively influence the cost/benefit impact of this imaging method on patients with cerebral infarction, an impact that has been found wanting and criticized in this clinical setting by a previous report [18]. Further, the ability to visualize the earliest changes after critical reduction in blood flow to a brain region may allow the physician to evaluate current or future therapeutic measures that can treat ischemic brain edema or at least lower the threshold for its development.

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