

SCINTIGRAPHIC DEMONSTRATION OF CEREBRAL INFARCTON IN A "WATERSHED" DISTRIBUTION

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A 24-year-old woman who had a history of intermittent leg vein thrombophlebitis suffered sudden onset of chest pain and shortness of breath shortly after arising from a nap in the evening of Nov. 10, 1970. She collapsed and was taken to Parkland Memorial Hospital by ambulance. On arrival her blood pressure was 104/70 and her pulse was 140/min and regular. She exhibited pallor and tachypnea, as well as cyanosis of the nail beds. Massive pulmonary embolism was suspected, and a pulmonary arteriogram was performed at once. A large central saddle clot was found to have obstructed the entire right pulmonary circulation as well as the vessels of

the lingula and left lower lobe. Only upper segments of the left upper lobe circulation were preserved. A pulmonary embolectomy was performed, followed by ligation of the inferior vena cava. As the patient recovered from anesthesia early on Nov. 11, 1970, left extremity weakness was noted which progressed rapidly to left hemiplegia. Although only a residual effect of anesthesia was at first suspected, she made a

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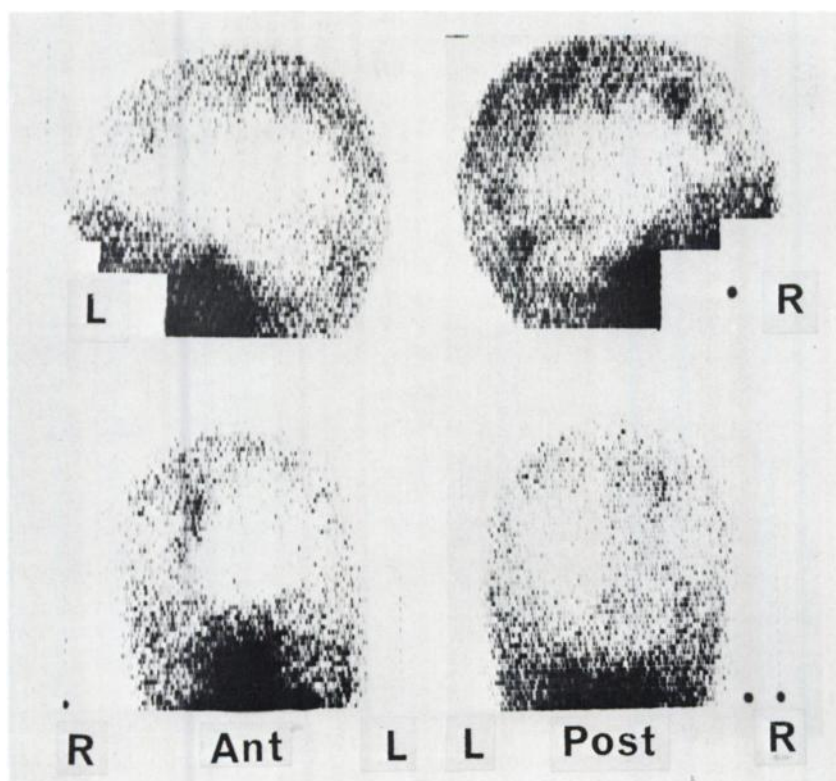


FIG. 1. ^{99m}Tc -pertechnetate brain scan in four projections shows right-sided, paramedian, strip-like abnormal tracer localization which exhibits nodular characteristics on right lateral view. Localization is in "watershed" region between anterior and middle cerebral circulations as well as posterior and middle cerebral arteries.

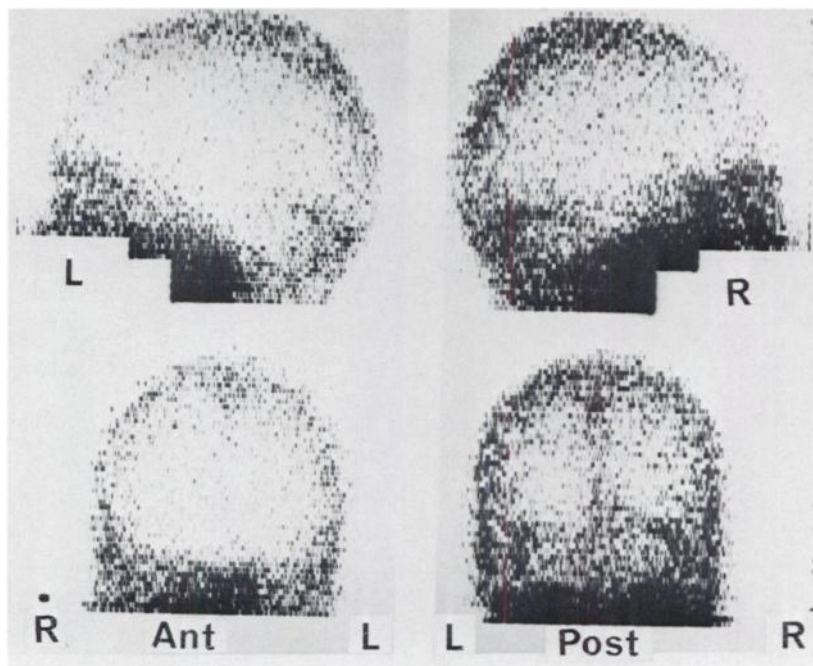


FIG. 2. Brain scan of same patient done 3 months after scan in Fig. 1 shows disappearance of watershed localization which accompanied clinical recovery of patient.

slow and unsatisfactory recovery. By Nov. 19, 1970 the possibility of a cerebral vascular accident was seriously considered, and a brain scan was requested.

Thirty minutes after the administration of 200 mg of potassium perchlorate by mouth the patient was given an intravenous dose of 10 mCi of ^{99m}Tc as sodium pertechnetate. One hour thereafter a rectilinear scan (Fig. 1) was carried out with a dual 5-in. crystal unit. The anterior view showed a right paramedian, strip-like lesion which, on the right lateral view, extended posteriorly in a nodular configuration through the frontal and parietal regions into the occipital region. Because the area of tracer localization seemed to lie along the border between the anterior and middle cerebral circulations, a scan diagnosis of cerebral infarction in a "watershed" distribution was made.

Thereafter the patient began a rapid clinical recovery and was normal by Feb. 10, 1971, except for a minimal neurological deficit in the left extremities. A brain scan was performed with the same technique employed above, and the results were normal, as Fig. 2 shows. The strip-like localization of confluent nodules in the watershed region was no longer apparent, confirming the diagnosis of a cerebral vascular accident in a watershed distribution.

DISCUSSION

The size and distribution of infarction that occurs with occlusion or stenosis of a major cerebral artery varies considerably from patient to patient. Usually the spectrum of findings may be explained by physio-

logical and anatomical variations in collateral channels. At times, infarction may be limited to, or be more pronounced in, a zone *between* the expected distribution of two major cerebral arteries. The term "watershed" (1) has been applied to these borders zones between two arterial systems. This type of infarction is most frequently observed on the cortical surface of the lateral convexity of the brain between the areas of the anterior and middle cerebral arteries.

Since the watershed region is in the periphery of a vascular territory, infarction may be seen in this area with proximal occlusive disease, but bilateral and symmetrical infarction of the watershed area has also been observed in patients with acute hypotensive or anoxic episodes. Asymmetrical involvement can occur with such episodes if there is unilateral occlusive disease. A shower of tiny emboli reaching the periphery of a vascular territory can also result in this distribution of infarction. Either of the latter two mechanisms could have been present in our patient.

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