CHANGES IN THE RADIOISOTOPE CISTERNOGRAM IN CEREBROVASCULAR-OCCLUSIVE DISEASE

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Radioisotopes have been used in the study of the cerebrospinal fluid (CSF) since Sweet and Locksley (*1*) first used ^{131}I-albumin to determine the formation, flow, and reabsorption of CSF in man. Since that time, cisternographic techniques using radionuclides have been used to evaluate CSF dynamics in conditions ranging from the detection of CSF rhinorrhea (*2*) to communicating hydrocephalus (*3*). Recent work has shown that abnormalities in the flow of CSF occur following subarachnoid hemorrhage (*4*). These usually take the form of diminished movement of ^{131}I-albumin around the cerebral hemispheres and, in some cases, the occurrence of a communicating low-pressure hydrocephalus. The etiology of the diminution in flow following the subarachnoid hemorrhage has not been well studied although one might suspect it to be due to adhesions from an accompanying arachnoiditis. The purpose of this paper is to further (*5*) describe changes seen on the radioisotope cisternogram following an acute cerebrovascular occlusive (CVA) episode.

MATERIALS AND METHODS

A total of 11 patients with acute CVAs were studied with cisternography. The diagnosis of a CVA was made on the basis of clinical evidence and the finding of a normal spinal fluid cell count. Cisternography was performed according to the method described by Ashburn and DiChiro (*6*). In some patients, brain scans were performed 2 hr after the intravenous injection of 15 mCi of $^{99m}TcO_4^-$, either simultaneously with the cisternogram, or at some time during the course of the patient's hospitalization. In some patients, the $^{99m}TcO_4^-$ was injected as a bolus into a large vein in the antecubital fossa, and rapid sequence imaging was performed over the head. This is referred to in the Results section as a cerebral perfusion study.

RESULTS

Table 1 gives the clinical and laboratory data pertinent to the 11 patients on whom cisternography was performed. Seven of the eleven patients had abnormalities in the flow pattern of the tracer. In three, these changes were marked, i.e., no tracer movement around the affected cerebral hemisphere was detected. In three cases, the flow was decreased as shown by an easily distinguished difference in the counting rate over the two hemispheres, yet some counts were discernible on the affected side. In one patient, minimal changes were observed in which a true abnormality could be seen in the flow of the tracer over both hemispheres, yet the difference in counting rate was less than in the group described as having decreased flow. Four patients had normal cisternograms. All three of the patients with marked changes on the cisternogram had abnormal brain scintiphotos, and two of these had abnormal cerebral perfusion studies. The three patients classified as having decreased CSF tracer flow also had abnormal brain scintiphotos, and one of these three had a normal perfusion study. The single patient with minimal changes on cisternography had a normal brain scintiphoto and an abnormal perfusion study which became normal on restudy 6 days later. Three of the four patients with normal cisternograms had normal brain scintiphotos, and the remaining patient had a technically unsatisfactory study. Two of these patients had normal perfusion studies; the other two patients were not studied by this method.

Figure 1 shows a comparison of a normal cisternogram and the abnormal study obtained in Patient 1. The abnormal areas are marked with arrows.

Figure 2 (Patient 11) shows diminished flow of tracer material around the left hemisphere at 4 hr. These changes are still visible at 24 hr. The brain scan was markedly positive in a middle cerebral distribution.

Figure 3 (Patient 10) shows markedly diminished

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TABLE 1. RAW DATA ON ALL 11 CVA

Pt	Date of CVA	Clinical observations	Neurological clinical course	Date and results of cisternogram	Date and results of brain scan and perfusion study	Date and results of echo-enceph-alogram	Date and results of EEG and arteriogram
(1) 69 F	2/25/71	L. hemiparesis, mitral stenosis, atrial fibrillation, congestive heart failure, probable embolism to R. middle cerebral artery	Deepening L. hemiparesis and coma	3/1/71 Markedly decreased flow over R. hemisphere	3/1/71 Focal increased uptake R. parietal region	3/2/71 No shift	3/2/71 Delta II, focal R. hemisphere
(2) 80 F	5/7/71	Mild L. hemiparesis, R. middle cerebral artery distribution, L. carotid bruit	Pt. discharged within 24 hr, no followup	5/13/71 Normal	5/13/71 Scans technically unsatisfactory	None	5/7/71 Intermittent slowing L. temporal region
(3) 96 M	5/20/71	L. hemiparesis, cardiomegaly, and pulmonary edema on 5/25/71, responding to treatment	No improvement of hemiparesis by time of discharge 6/8/71	6/2/71 Decreased flow over R. hemisphere	5/28/71 Focal increased uptake R. parietal region, and decreased perfusion on R.	None	5/26/71 G II dysrhythmia, generalized, delta I generalized
(4) 53 F	12/28/71	L. hemiparesis with R. middle cerebral artery syndrome, past hx of L. middle cerebral artery occlusion 4 yr prior to present illness	No improvement at time of discharge on 1/27/71	1/4/71 Decreased flow over R. hemisphere	12/27/70 Normal 1/7/71 Focal increased uptake R. parietal region	12/28/70 No shift	12/28/70 Delta II activity R. temporo-parietal region
(5) 76 F	6/2/71	Mild L. hemiparesis, transient ischemic episode noted in same distribution one month prior to hospitalization	Marked improvement within 24 hr	6/3/71 Normal	6/3/71 Normal	None	G II focal dysrhythmia, temporoparietal region
(6)	5/18/71	Sudden L. hemiparesis	Marked improvement by 5/21/71	5/21/71 Decreased flow over R. hemisphere	5/21/71 Equivocal increased uptake in R. parietal region. Normal perfusion study	None	5/27/71 Normal EEG

* Reference number to case is in parenthesis in extreme left column.

flow of the tracer around the left cerebral hemisphere at 8, 18, 44, and, to a slight extent, even 68 hr. The cerebral perfusion study revealed diminished tracer activity on the left in both arterial and venous phase. The brain scan shows an area of increased uptake also on the left; however, the changes seen are not as striking as those on the cisternogram and cerebral perfusion studies.

Figure 4 shows the results of cisternography, cerebral perfusion study, and brain scan in Patient 8. The patient was having Jacksonian seizures involving the left side of her body at the time of the first perfusion study, brain scan, and cisternogram. Minimal changes in the cisternogram were noted over the right hemisphere on both the 6- and 22-hr study. The cerebral perfusion study performed on May 20, 1971, showed diminished perfusion of the left cerebral hemisphere which returned to normal by May 26. The brain scan was normal. The patient's clinical course indicated a right cerebral lesion, and the significance of the diminished perfusion to the left hemisphere on the pertechnetate perfusion study is unknown.

DISCUSSION

The classical description of the propagation of CSF (*7*) has been that it is forced from the ventricles by a filtration pressure, swept along by the ciliary movements of ependymal cells, and reabsorbed at the venous sinuses by the relatively lower venous pressure. The role played by these forces is poorly understood; however, it does appear that arterial pulsation within the intracranial blood vessels exerts a force in propagation of the CSF (*8*). Since the

PATIENTS WHO HAD CISTERNOGRAPHY*

Pt	Date of CVA	Clinical observations	Neurological clinical course	Date and results of cisternogram	Date and results of brain scan and perfusion study	Date and results of echo-enceph-alogram	Date and results of EEG and arteriogram
(7) 54 M	5/25/71	Prontomedullary stroke on R. with L. hemiparesis	Marked recovery within 24 hr almost complete by 6/2/71	5/27/71 Normal	5/28/71 Normal brain scan and perfusion study	None	5/27/71 Normal EEG and arch aortagram
(8) 50 F	5/19/71	Jacksonian seizures on L. and L. hemiparesis, minimal clearing of hemiparesis by 6/5/71	Marked improvement in hemiparesis by 7/8/71	5/26/71 Minimal decreased flow over posterior hemisphere on R.	5/20/71 Normal brain scan. Perfusion study abnormal on L. 5/26/71 Normal perfusion study	5/19/71 Normal	5/19/71 G III Dysrhythmia R. hemisphere, 5/19/71, normal bilateral carotid arteriograms
(9) 58 M	6/10/71	Dense L. hemiparesis in the distribution of R. posterior cerebral artery	No followup	6/14/71 Normal	6/11/71 Normal brain scan and cerebral perfusion study	None	6/11/71 Delta I activity R. parietotemporal, 6/23/71, arteriogram shows posterior cerebral artery occlusion on R.
(10) 80 F	7/30/71	R. hemiparesis (flaccid), congestive heart failure, bilateral plantar extension response, spasticity of L. side of body indeterminant in past date	No change in hemiparesis	8/9/71 Markedly decreased flow over L. hemisphere	8/4/71 Increased uptake in parietal region 8/4/71 Decreased perfusion on L.	8/2/71 4-mm shift toward R.	8/2/71 G II generalized dysrhythmia and delta I activity L. frontocentral and temporal hemispheres
(11) 71 F	8/7/71	Sudden coma with R. hemiparesis, questionable congestive heart failure	No improvement in hemiparesis in one week followup	8/18/71 Decreased flow over L. hemisphere	8/10/71 Focal increased uptake L. frontoparietal region. Normal cerebral perfusion study	8/7/71 Normal	8/9/71 Dysrhythmia G II generalized, delta II L. hemisphere, maximum frontotemporal region

* Reference number to case is in parenthesis in extreme left column.

normal propagating force of CSF flow is not well understood, the etiology of the cisternogram changes noted in our patients with CVA are likewise not well understood. Rudd, et al (*4*) and Williams, et al (*9*) have shown that abnormal flows of CSF can occur following a subarachnoid hemorrhage. Of the nine patients reported by Rudd, all had abnormal flow of CSF with ventricular filling and/or diminished or absent movement of the tracer over the cerebral convexities. It was the feeling of these investigators that the abnormalities observed were possibly related to arachnoiditis and fibrosis secondary to the blood in the subarachnoid space. Further, all of the patients in their study had had craniotomies before the cisternogram studies, and the possibility of an iatrogenic etiology of the cisternographic findings cannot be ruled out.

All of our patients had CVA disease (none had craniotomies); therefore iatrogenic fibrosis or arachnoiditis could not account for the changes noted in our patients' cisternograms. The effect of diminished arterial pulsation on the side of the lesion as an etiology of the cisternographic findings is unknown. The most attractive explanation is diminished regional CSF flow secondary to edema of the brain following the occlusion. This possibility must be strongly considered since brain edema occurs following an occlusion of an intracerebral vessel (*10*). Cerebral edema has been reported to occur within a few hours following the infarct and may last for several days. This being the case, a patient with an acute cerebral infarct might be expected to develop an abnormal cisternogram which would presumably return to normal if performed months later. Further

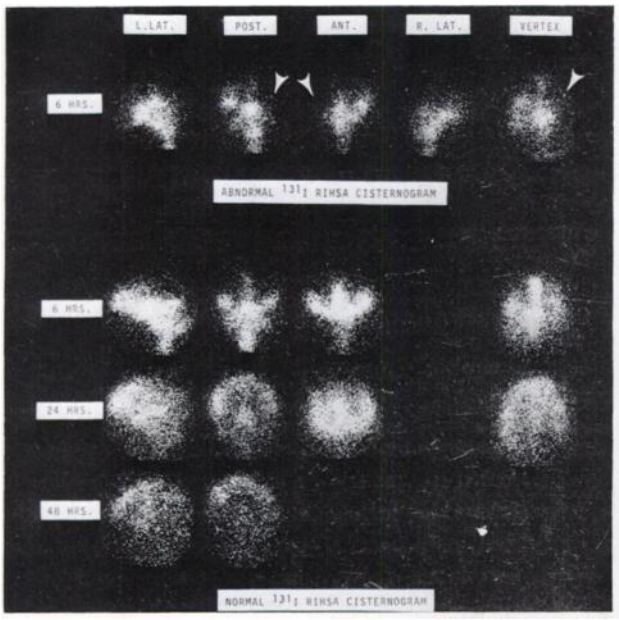

FIG. 1. Comparison of cisternograms in normal patient (bottom) and that of patient with well-documented right hemispheric CVA (top). Patient 1: Note markedly decreased flow around right hemisphere following occlusion (arrows).

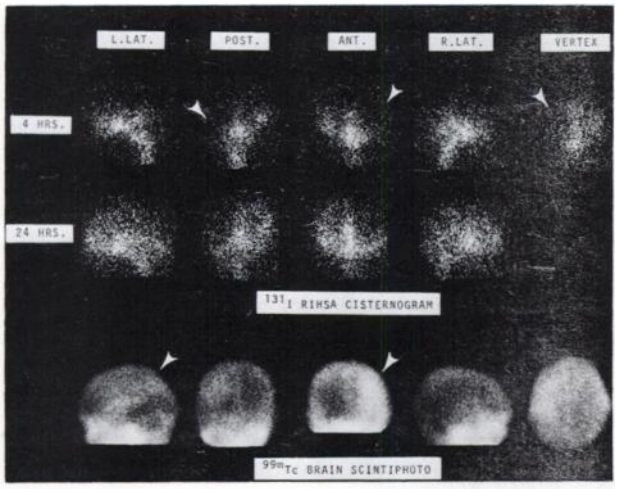

FIG. 2. Abnormal cisternogram (Patient 11) in left middle cerebral artery distribution (arrows). Brain scan is positive in same area.

work must be performed before this information can be ascertained. The relationship between lesions noted on the brain scans, cerebral perfusion studies, and the cisternogram has not become evident yet and will have to await further studies.

SUMMARY

Eleven patients with acute CVA disease were studied for evidence of abnormal flow of CSF following the intrathecal injection of ^{181}I-albumin. Seven patients had unequivocally abnormal cisternograms consisting of diminished flow of tracer while four had normal cisternograms. The clinical material is presented and possible etiologies discussed.

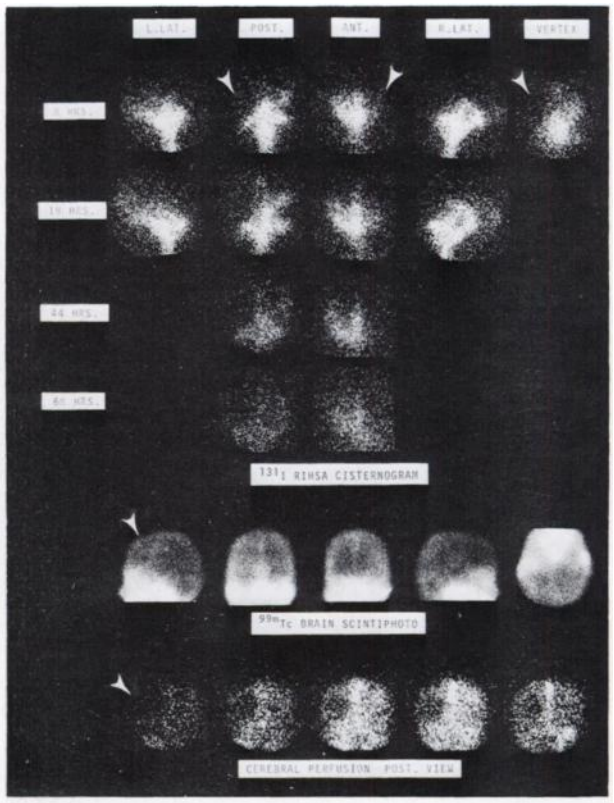

FIG. 3. Abnormal cisternogram involving left cerebral hemisphere (Patient 10). Abnormality is present for 68 hr. Note abnormalities on left (arrows) in perfusion study and brain scintiphotos.

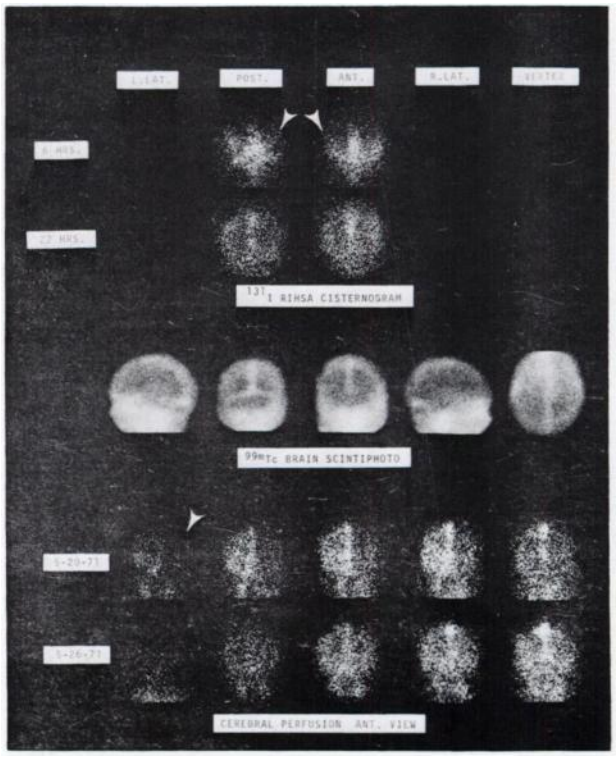

FIG. 4. Minimal changes (arrows) are noted on cisternogram over right cerebral hemisphere (Patient 8). Cerebral perfusion study is initially abnormal (arrow) on left, but reverts to normal 6 days later. Brain scan is normal.

Although the role of the CSF in maintaining normal cerebral metabolism and function has not been established, one might consider the possibility that even temporary alteration in CSF flow in the region of cerebral ischemia may affect the clinical outcome in the patient with acute stroke.

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