Rhinorrhea, Ventricular Radiopharmaceutical Stasis and Communicating Hydrocephalus: Evaluation by Serial Cisternography

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Spontaneous cerebrospinal fluid rhinorrhea has been known to occur in association with hydrocephalus. The specific pathophysiology which results in a potential communication between the cerebrospinal fluid space (subarachnoid space) and the nasopharynx is unknown. The relationship of CSF movement and rhinorrhea was evaluated in ten random source mongrel dogs. These data suggest that spontaneous CSF rhinorrhea may occur during the early developmental phase of communicating hydrocephalus in dogs. At this time radiopharmaceutical movement showed ventricular entry and clearing. When the lateral ventricles enlarged, ventricular radiopharmaceutical stasis was seen and the rhinorrhea disappeared. This suggests that CSF rhinorrhea may act as a compensatory mechanism which partially protects the CSF compartment to withstand the extra CSF during the early development of communicating hydrocephalus.

Key words: Cisternography, rhinorrhea, communicating hydrocephalus.

Cerebrospinal fluid rhinorrhea is a well documented sequel to craniofacial trauma. A less common but clinically important cause of cerebrospinal fluid rhinorrhea is hydrocephalus.17 When the normal pathways of CSF absorption are obstructed, CSF is probably absorbed by alternative pathways.2,11,13 In the early development of hydrocephalus, CSF rhinorrhea may act as a compensatory mechanism to limit CSF pressure but as chronic communicating hydrocephalus develops, changes occur in the ependymal lining of the ventricular walls which may provide an alternate pathway from which CSF absorption could occur.

Utilizing an experimental model for chronic communicating hydrocephalus, we have documented the serial changes of abnormal CSF movement and its relation to spontaneous rhinorrhea on cisternography. Correlation with the clinical status of the animal, the stage in development of hydrocephalus, cisternographic images and pathological findings form the basis of this communication.

Materials and Methods

Ten random source, male and female adult mongrel dogs (10–20 kg) were utilized for this study. Each animal served as its own control by having a baseline cisternographic study performed.28 After determining that each animal had a normal cisternographic pattern, communicating hydrocephalus was experimentally induced by a method of subarachnoid injection of a silicone mixture.7–9 The development of chronic communicating hydrocephalus as well as confirmation of rhinorrhea was documented by obtaining serial cisternograms at five to ten day intervals following injection of the silicone mixture.

Baseline and follow-up radioisotope cisternograms were performed by the following method:

Anesthesia was induced by intravenously administered sodium pentobarbital (25 mg/kg) and the animal placed in the left lateral recumbent position. The trachea was visually intubated with a cuffed endotracheal tube to provide for a patent airway and to prevent aspiration of stomach contents. The area overlying the cisterna magna was shaved and scrubbed with an antiseptic solution. With the neck flexed and the head firmly immobilized, the cisterna magna was
punctured at midline with a standard 20 gauge spinal needle using the external occipital protuberance and the lateral borders (wings) of the atlas (first cervical vertebrae) as landmarks. When clear flowing CSF was observed at the needle hub the radiopharmaceutical (serum albumin labeled with 500 μCi 99mTc) was injected (1.0 cc total dose) into the cisternal subarachnoid space.

Cisternographic images (lateral and vertex) were obtained at thirty minutes, four hours and at 24 hours on a Picker 5 inch Magnascanner.°

Experimental Production of Communicating Hydrocephalus

The animal was anesthetized, positioned, intubated and prepped as for cisternography. Cisternal punctures were made with a Bardic intracath (17 gauge needle with 19 gauge polyethylene catheter) utilizing sterile technique. Under fluoroscopic control, the needle was introduced; angled laterally to avoid traumatizing the brain stem and to allow passage of the catheter into the basal cisterns. When clear flowing CSF appeared at the needle hub, the catheter (tip made radiopaque by coating with sterile powdered tantalum‡) was advanced along the skull base anterior to the ventricular outlets (in the region of the pontine and interpeduncular cisterns). Proper position of the catheter was monitored by dorsoventral and lateral radiographs of the skull. One to three milliliters of the silicone material was injected into the subarachnoid space.

The silicone mixture consisted of 3 ml sterilized silastic 382§ (polysiloxane polymer) mixed with 3.5 ml sterilized dilution fluid 360§ (dimethylpolysiloxane) warmed to 37°C in a water bath. Two drops of sterilized catalyst§ (stannous octoate) were added to the silicone mixture just prior to injection. The mixture is injected slowly with a sterile 1 ml plastic syringe, cleaning the catheter and syringe hubs of excess silicone material with sterile gauze pads. When the injection was completed the catheter and needle was removed and any bleeding at the injection site was controlled by a hemostatic pad. The animal was then placed supine with the head hanging dependent for 20–30 minutes to aid flow of the injected silicone mixture anteriorly and superiorly. The mixture polymerizes into a firm mass that obstructs CSF flow.7 Artificial ventilation and observance of the animal’s vital signs during and after the injection procedure was necessary to reduce morbidity and mortality of the experimental animals.

Results

The control cisternograms show a normal CSF flow pattern with a lack of spontaneously occurring hydrocephalus or CSF rhinorrhea (Fig. 1A–D). Of the ten animals in our experimental group, eight (80%) developed communicating hydrocephalus with five (63%) of the hydrocephalic animals demonstrating some degree of CSF rhinorrhea.

The animals were separated into three groups on the basis of their serial cisternographic patterns (Table 1).

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* Picker Corporation, 1275 Mamaroneck Avenue, White Plains, N. Y. 10605.
† C. R. Bard Corporation, Murray Hill, New Jersey.
‡ Powdered tantalum: Fansteel Corporation, Baltimore, Maryland.
§ Dow Corning Corporation, Midland, Michigan.
Group I consisted of two animals which did not develop communicating hydrocephalus. In Group II, four animals developed communicating hydrocephalus with a cisternographic image showing ventricular radiopharmaceutical entry with clearing at 24 hours (Fig. 2A). The four hour vertex image demonstrated radiopharmaceutical in the area of the nasopharynx consistent with CSF rhinorrhea (Fig. 2B). One animal in Group II demonstrated a serial cisternographic image pattern consisting of ventricular radiopharmaceutical entry with clearing and CSF rhinorrhea but after two months the cisternographic image showed ventricular entry with radiopharmaceutical stasis at 24 hours and no detectable activity in the nasopharynx (Fig. 3). Group III consisted of three animals that developed communicating hydrocephalus with ventricular entry and clearing but did not show CSF rhinorrhea.

At necropsy the animals in Group II had moderate communicating hydrocephalus. The animal with radiopharmaceutical stasis (Group II, Animal #7) had markedly enlarged ventricles (Fig. 4). Necropsy performed on the animals in Group III demonstrated minimal enlargement of the lateral ventricles, somewhat more marked in the posterior horns. The aqueduct of Sylvius was patent and without obstruction in all animals. The lamina cribrosa was unobstructed with silicone in all animals.

**Discussion**

The leakage of cerebrospinal fluid through the cribriform plate into the nasopharynx in both animal

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**TABLE 1.** Experimental Results: Animals Classified By Cisternographic Pattern.

<table>
<thead>
<tr>
<th>Animal #</th>
<th>Communicating Hydrocephalus</th>
<th>CSF Rhinorrhea</th>
<th>Cisternographic Pattern Ventricular Clearing</th>
<th>Ventricular Stasis</th>
<th>Rhinorrhea Disappeared</th>
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- No  + Yes
and man has been documented by cisternographic studies. Schechter et al described the association of hydrocephalus and rhinorrhea and proposed theoretical mechanism by which the two phenomena coexist.\(^\text{17}\) Di Chiro has documented the presence of CSF rhinorrhea in dogs with spontaneously occurring hydrocephalus.\(^\text{6}\) In both series clinical evidence suggests that this might provide a mechanism by which inappropriate production of cerebrospinal fluid is partially compensated.

Several investigators have questioned the use of canine models for the study of hydrocephalus.\(^\text{3,6}\) Reviewing these data the breed of dog must be considered. Purebred beagles and beagle type dogs have a high incidence of spontaneously occurring hydrocephalus.\(^\text{3,6}\) All animals included in our study were

random source, mongrel dogs which were demonstrated by cisternography to have a normal CSF flow pattern without spontaneously occurring hydrocephalus or CSF rhinorrhea.

It is generally recognized that the most common mechanism for the development of communicating hydrocephalus is obliteration of the peripheral subarachnoid pathways which prevents the cerebrospinal fluid that is produced in the ventricles from reaching the arachnoid villi in the parasagittal region for absorption.\(^\text{5,11}\) Measurements of CSF production in communicating hydrocephalus have demonstrated either a normal CSF production rate or one that is only moderately decreased.\(^\text{4,14}\) The enlargement of the cerebrospinal fluid spaces, such as the lateral ventricles, is not sufficient to account for the pathophysiological accommodation that occurs with communicating hydrocephalus.\(^\text{2}\) That is, ventricular enlargement appears to progress more rapidly in the acute phase of communicating hydrocephalus than in the chronic phase. The ventricular increase in volume is never commensurate with that anticipated by calculations of CSF volume. Some method of compensation either through diminished CSF production or alternative CSF absorption pathways must be present.

In the dog, it has been theorized that one method of removal of extra cerebrospinal fluid from the subarachnoid space has been the development of spontaneous CSF rhinorrhea.\(^\text{6,18}\) Another method of alternative CSF absorption has been through transependymal migration into the brain parenchyma and removal by the cerebral veins. Transependymal movement of labeled albumin has been demonstrated in non-communicating hydrocephalus by Bering\(^\text{1}\) and in communicating hydrocephalus by Strecker et al.\(^\text{19}\)

In several series of experiments, James et al have demonstrated that ventricular size, ventricular radiopharmaceutical retention, transependymal radiopharmaceutical movement and changes in CSF pressure are related.\(^\text{11,12}\)

Utilizing this experimental animal model, we were able to document, that in the normal circumstance, radiopharmaceutical injected into the cisterna magna would not appear in the nasopharynx in the first 24 hours of the study. In the acute phase of communicating hydrocephalus in 5 of 8 animals, radiopharmaceutical was seen in the nasopharynx. This is characteristic of the image pattern associated with CSF rhinorrhea. At this time the cisternographic image demonstrated ventricular entry of the radiopharmaceutical with clearing at 24 hours. James et al documented that the CSF pressure is increased at this time.\(^\text{12}\) The development of CSF rhinorrhea may be a compensatory
mechanism to alleviate the relative overproduction or malabsorption of cerebrospinal fluid.

In one animal (7), beginning with a normal cisternogram, ventricular radiopharmaceutical entry with clearing and CSF rhinorrhea was documented by cisternography. As the communicating hydrocephalus progressed to the chronic phase (continued ventricular enlargement) the CSF rhinorrhea disappeared. However, at this time radiopharmaceutical entered the lateral ventricles and remained there for a protracted period of time (stasis) presumably demonstrating CSF absorption by the ventricular ependyma with a decline in CSF pressure into the normal range.11,12

CSF rhinorrhea appears to develop as a compensatory mechanism to increased CSF pressure in the acute stage of communicating hydrocephalus as has been noted by other investigators.16,18 The spontaneous disappearance of CSF rhinorrhea may be related to the fact that CSF pressure has been decreased by the formation of alternative pathways of CSF absorption;11 namely, ventricular transependymal migration leading to penetration of the cerebral circulation in the brain parenchyma.

Traumatic CSF rhinorrhea is a problem which can be corrected by surgical repair of the anatomical defect. In instances of rhinorrhea associated with hydrocephalus, pathological changes in the ventricular ependyma and its mechanical barrier effect may obviate the need for the compensatory effect of CSF rhinorrhea.18 Proper therapy in these patients must be directed toward defining the source of increased pressure, otherwise closure of the fistulous tract may be counter-productive.17

References