Conventional and CT Metrizamide Myelography in Arnold-Chiari I Malformation and Syringomyelia

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Four normal controls and 26 cases of Arnold-Chiari I malformations and/or syringomyelia were reviewed. The pathologic cases included five isolated Arnold-Chiari I malformations, nine communicating syringomyelia, five idiopathic syringomyelia, four posttraumatic syringomyelia, one syringomyelia with hemangioblastoma, and two post-shunt syringomyelia. The objectives of this study were to compare the accuracy of conventional metrizamide myelography with CT metrizamide myelography and to study indirectly the hydrodynamics of CSF flow in syringomyelia by comparing the sequential enhancement patterns of the spinal cords and cord cavities in the different groups of patients. Twenty-five patients underwent conventional metrizamide myelography immediately before CT metrizamide myelography, and one patient underwent CT metrizamide myelography only. Scans were obtained 1–2 hr, 4–8 hr, and 12–24 hr after injection of metrizamide, but not all patients were scanned during all three intervals. CT metrizamide myelography was found to be more sensitive than conventional metrizamide myelography in the diagnosis of both Arnold-Chiari I malformation and syringomyelia. Performing just an immediate and a delayed scan was found to be more cost-effective than doing all three scans. Contrary to previous reports, it was found that delayed (12–24 hr) scans demonstrated more syrinx cavities than intermediate ones. In studying the sequential enhancement patterns of the spinal cords and cord cavities, some interesting trends were observed that tend to support the theories of Aboulker and of Ball and Dayan of transneural passage of CSF into cord cavities in syringomyelia.

Almost a century ago, Chiari described two kinds of caudal ectopia of the hindbrain [1, 2]. In this study, we examined only the Arnold-Chiari I malformation, which involves caudal displacement of the cerebellar tonsils [1, 3].

Syringomyelia, first coined by C. P. Ollivier in 1827, derives from two Greek words meaning "a cavity" and "the spinal marrow" [4]. Syringomyelia has been divided into five groups [5, 6]: (1) communicating syringomyelia or syringomyelia associated with developmental anomalies at the foramen magnum and posterior fossa or acquired abnormalities such as basal arachnoiditis; (2) posttraumatic syringomyelia; (3) syringomyelia as a sequela of arachnoiditis confined to the spinal canal; (4) syringomyelia associated with spinal cord tumors; and (5) idiopathic syringomyelia unrelated to any of the above categories.

Numerous theories have been proposed by different authors to explain the pathogenesis of syringomyelia, but none of these is universally accepted. The Gardner theory [7] hypothesized that impaired outflow of CSF from the fourth ventricle, caused by failure of the foramina to open between the sixth and eighth weeks of embryonic life, leads to the transmission of arterial pulsations of CSF to the central canal, thereby creating a syrinx cavity. Hence, in the "communicating" syringomyelia (of Gardner) there is a communication between the syrinx and the fourth ventricle via an open obex. However, the pathophysiologic mechanism suggested by Gardner has been the subject of a prolonged and as yet unresolved controversy, and this has led to the idea of "noncommunicating" syringomyelia. This variety usually develops as a late sequela of spinal cord trauma, arachnoiditis,

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and neoplasms. The Aboulker theory [8] postulated that the syrinx cavity is caused by a transmedullary passage of CSF, which is in turn caused by obstruction at cisterna magna plus high venous pressures.

Williams [9] hypothesized that a craniospinal pressure dissociation is created by a defective intracranial drainage that causes partial obstruction to the free flow of CSF from the cranium into the spinal subarachnoid spaces. This craniospinal pressure dissociation "sucks" fluid from the fourth ventricle through the central canal into the syrinx cavity. Ball and Dayan [10] proposed that the CSF is forced into the syrinx by CSF pressure waves along the Virchow-Robin spaces. These various theories of pathogenesis have led to the development of various surgical treatments that sometimes are not very effective [5, 11–13]. Better understanding of the pathogenesis will likely lead to better treatment in the future.

Arnold-Chiari I malformation and/or syringomyelia are variable in onset, symptoms, and course. The patients usually present with myelopathy and a dissociated sensory loss, sometimes accompanied by brainstem signs. However, there are no reliable clinical clues to the diagnosis, and misdiagnoses are common [3, 5, 11–13]. This had led to great reliance on imaging examinations for diagnosis. The advent of CT metrizamide myelography (CTMM) afforded a highly sensitive diagnostic test. Also, the use of sequential scans provides useful information about the hydrodynamics of CSF flow in syringomyelia [14–16]. This type of information provides an opportunity to test the validity of the different theories of pathogenesis of syringomyelia in vivo.

The objectives of this study were fourfold: (1) to compare the accuracy of conventional metrizamide myelography with CTMM in the diagnosis of Arnold-Chiari I malformation and syringomyelia; (2) to compare the sequential enhancement patterns of the spinal cord in isolated Arnold-Chiari I malformation, in different types of syringomyelia, and in normal controls; (3) to compare the sequential enhancement patterns of the cord cavities in different types of syringomyelia; and (4) to compare the data obtained with different theories of pathogenesis of syringomyelia.

Subjects and Methods

We reviewed the records of 26 patients with Arnold-Chiari I malformation and/or syringomyelia who were referred to us between January 1982 and January 1985. Twenty-five patients underwent conventional myelography with 8–10 ml of metrizamide at 240–280 mg/ml concentration, injected via the lumbar approach in 23 patients and via C1–C2 puncture in two patients, before CTMM. One patient underwent only CTMM with 7 ml of metrizamide at 170 mg/ml concentration. Immediate scans were obtained within 1–2 hr after injection of metrizamide. Intermediate and delayed scans were obtained at 4–8 hr and 12–24 hr, respectively, after injection. Scans were obtained in five patients at all three time intervals, in 13 patients at two of the intervals, and in eight patients at only one time interval. The raw scan data on 19 patients could be retrieved for analysis. The scans were obtained with either a GE 8800 or a GE 9800 scanner. The sections were usually 5 mm thick, but thicknesses up to 10 mm were sometimes used.

A control group of four patients also underwent serial CTMM scanning. The patients were referred for cervical myelography because of clinical problems other than Arnold-Chiari I malformation or syringomyelia. In two control patients, scans were obtained at all three time intervals; in the other two patients, only two serial scans were obtained.

The concentration of metrizamide in the subarachnoid space at the cervical spine level after cervical myelography depends on several factors. First, it depends on the volume and concentration of the metrizamide injected. Second, the amount of mixing between the metrizamide and the CSF in the subarachnoid space has definite effects. This in turn depends on the amount of movement produced during the procedure, which depends on the anatomy and cooperativeness of the patient. Third, the individual variation of CSF volume in the subarachnoid space should also be considered. Fourth, some metrizamide may be spilled inadvertently into the head.

Since the concentration of metrizamide in the cervical subarachnoid space presumably has a direct effect on the enhancement of the cervical cord and cord cavity and this concentration is virtually impossible to standardize, attenuation measurements have to be made independent of it so that valid interpatient comparisons can be made. To achieve this, the following scheme was used in this study. All attenuation (CT number) measurements in a patient were divided by the measured CT number of the cervical subarachnoid space of the patient in the initial scan. These corrected attenuation values were then used for comparison with other patients. If a patient had two serial scans but no initial scan, then the CT number of the cervical subarachnoid space at time zero was extrapolated from the other two points assuming a uniform rate of decrease in the CT number with time. Patients with only one scan were excluded from the semiquantitative analysis. All CT number measurements were made in regions 16 pixels in size. Attempts were made to determine the CT number at approximately the same levels in the different scans of the same patient so that the beam-hardening effect caused by the bony structures would be about the same. Scanning techniques, including kilovoltage pressure and filters, were also constant; therefore, beam-hardening artifacts should not have been a major problem. To decrease partial-volume artifacts, all CT number measurements were done carefully so as not to include adjacent structures.

For standardization purposes, analysis was done with corrected attenuation values interpolated to 6 hr and 15 hr after myelography. Only scans obtained within the intervals of 4–8 hr and 12–18 hr after myelography were included in the analysis. All the patients included in the analysis had undergone two or more serial scans.

Finally, the longitudinal extent of the imaged syrinx cavities as well as the frequency of filling of the fourth ventricle by metrizamide were evaluated in the patients with idiopathic and communicating syringomyelia.

Results

The 26 patients comprised 15 women and 11 men aged 18–69 years (mean, 43.3). Seventeen of the patients underwent surgical treatments. The final clinical diagnoses included five isolated Arnold-Chiari I malformations, nine communicating syringomyelia, one syringomyelia with hemangioblastoma, and two postshunt syringomyelia.

Conventional Myelography vs. CTMM of Arnold-Chiari I Malformation and Syringomyelia

Of 11 cases of Arnold-Chiari I malformation (five isolated, six with syringomyelia), only one case was not diagnosed by conventional metrizamide myelography alone, giving an over-

all sensitivity of 90% (Fig. 1A). However, no supine cross-table lateral film was obtained in the case missed by conventional myelography. Therefore, the sensitivity of conventional myelography potentially could be higher. All 11 cases were diagnosed by CTMM (Fig. 1B).

The comparison of the two methods in the diagnosis of syringomyelia is summarized in Table 1. In the diagnosis of syringomyelia, conventional metrizamide myelography was unable to demonstrate cord-size abnormalities in one of nine cases of communicating syringomyelia, one of five cases of idiopathic syringomyelia, and two of four cases of posttraumatic syringomyelia (Fig. 2). The overall sensitivity, therefore, was only 79%. A widened spinal cord was seen in 63% of cases and an atrophic cord in 16%. All cases were diagnosed by CTMM at some point during the study, so the overall detection rate was 100%.

Only 13 patients had an initial scan, 12 had an intermediate scan (4–8 hr), and 16 had a delayed scan (12–24 hr). The immediate scans demonstrated cord-size abnormalities in 100% of the patients. However, contrast material was seen within the cord in only 8% (Fig. 3A). The intermediate scans demonstrated cord-size abnormalities in 100% of the cases, but contrast material was seen within the cord in only 67% (Fig. 3B). Delayed scans demonstrated contrast material within the cord in 100% of the cases, but only 69% of these also showed cord-size abnormalities. In the other 31%, the contrast material in the subarachnoid space was not dense enough for good delineation of the spinal cords. Eight patients

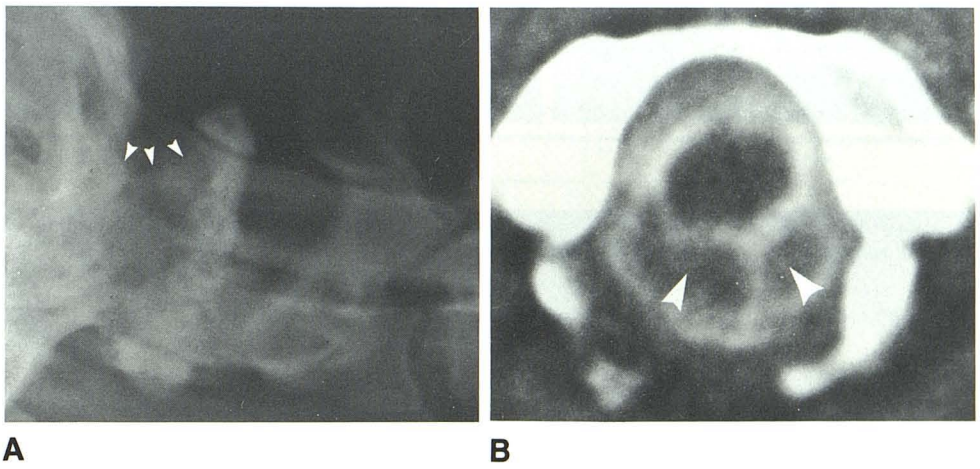

Fig. 1.—Arnold-Chiari I malformation.

***A*, Conventional metrizamide myelogram. Posterior indentation of subarachnoid space down to C1 level (*arrowheads*).**

***B*, CTMM at C1 level clearly shows inferiorly displaced cerebellar tonsils posterior to cord (*arrowheads*).**

TABLE 1: **Comparison of Conventional Metrizamide Myelography and CTMM in the Diagnosis of Syringomyelia**

Study: Finding	No. of Patients (%)				
	Communicating Syringomyelia	Idiopathic Syringomyelia	Posttraumatic Syringomyelia	Syringomyelia with Hemangioblastoma	Overall
Conventional metrizamide myelography:					
Widened cord	6/9 (67)	4/5 (80)	1/4 (25)	1/1 (100)	12/19 (63)
Atrophic cord	2/9 (22)	0/5	1/4 (25)	0/1	3/19 (16)
Normal cord	1/9 (11)	1/5 (20)	2/4 (50)	0/1	4/19 (21)
Immediate CTMM:					
Widened cord	6/8 (75)	2/3 (67)	1/2 (50)	. . .	9/13 (69)
Atrophic cord	2/8 (25)	0/3	1/2 (50)	. . .	3/13 (23)
High-density center + widened cord	0/8	1/3 (33)	0/2	. . .	1/13 (8)
4–8 hr CTMM:					
Widened cord	1/6 (17)	1/4 (25)	1/2 (50)	. . .	3/12 (25)
Atrophic cord	1/6 (17)	0/4	0/2	. . .	1/12 (8)
High-density center + widened cord	4/6 (67)	3/4 (75)	1/2 (50)	. . .	8/12 (67)
12–24 hr CTMM:					
High-density center + widened cord	5/7 (71)	3/5 (60)	2/3 (67)	0/1	10/16 (63)
High-density center	2/7 (29)	2/5 (40)	0/3	1/1 (100)	5/16 (31)
High-density center + atrophic cord	0/7	0/5	1/3 (33)	0/1	1/16 (6)

Note.—CTMM = CT metrizamide myelography.

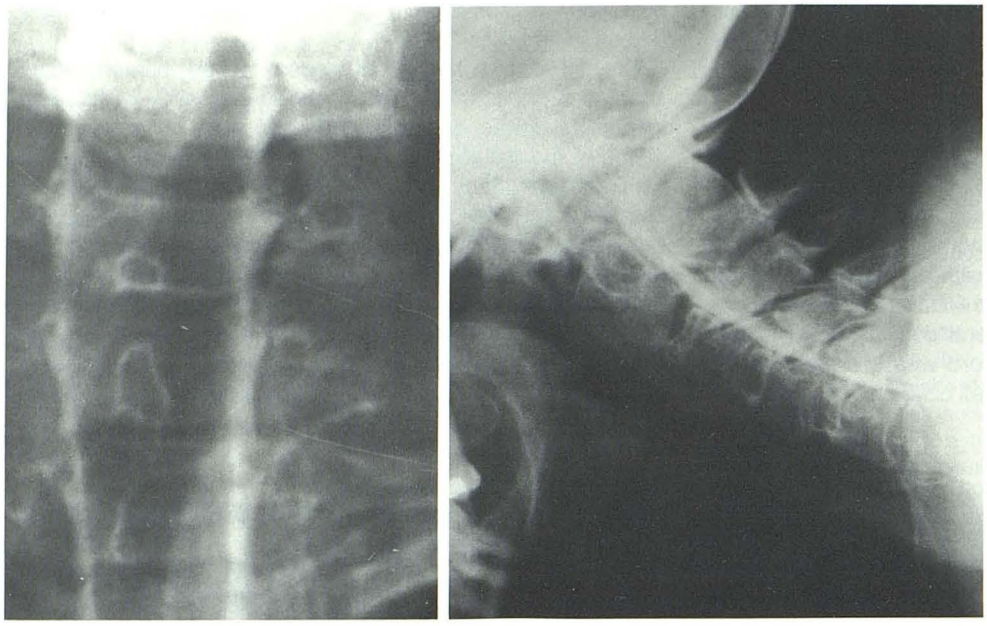

Fig. 2.—Communicating syringomyelia. Conventional metrizamide myelogram shows cord enlargement from C1 to T3.

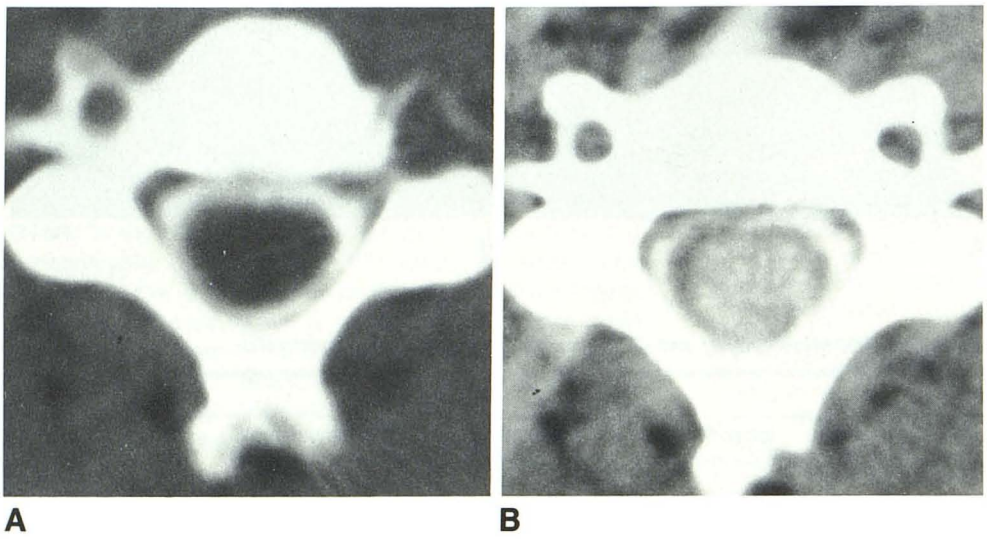

Fig. 3.—Communicating syringomyelia.

A, CTMM immediately after conventional myelography shows cord enlargement clearly, but no intramedullary contrast medium.

B, Intermediate scan 8 hr after conventional myelography shows both cord enlargement and contrast medium within cord cavity (target sign).

had both intermediate and delayed scans, and in three of these patients contrast material was seen within the cord only on delayed scans. In the other five patients, contrast material was seen within the cord on both intermediate and delayed scans (Fig. 4).

Sequential Enhancement Patterns of Spinal Cord in Isolated Arnold-Chiari I Malformation, Different Types of Syringomyelia, and Normal Controls

The results for this section are summarized in Table 2. In the normal control group and in patients with isolated Arnold-Chiari I malformation and communicating syringomyelia, the corrected attenuation values of the spinal cord at 6 hr after myelography were generally higher than those on the initial scans of the corresponding patients. Data available for patients with idiopathic syringomyelia and posttraumatic syringomyelia were insufficient for any statements to be made.

In the normal control group and in patients with isolated Arnold-Chiari I malformation, the corrected attenuation values of the cord 15 hr after myelography were again generally higher than at initial scanning. In communicating syringomyelia, the reverse was seen; that is, the corrected attenuation of the spinal cord was lower at 15 hr than at zero hours. This observation implies that there is enhancement of the spinal cord parenchyma on immediate postmyelography scans, which has been well documented in the literature [14]. This was also shown by three cases in our series in which the

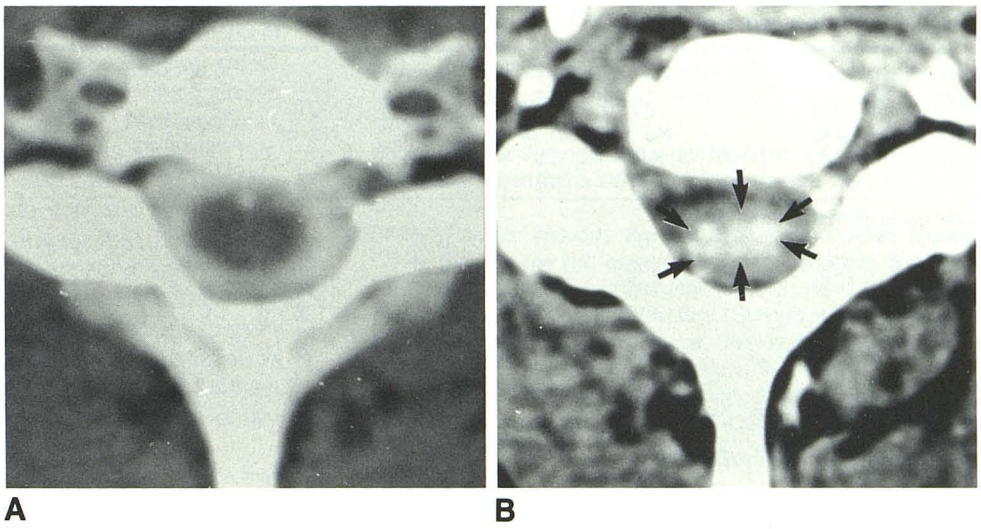

Fig. 4.—Idiopathic syringomyelia.
***A*, CTMM 6 hr after conventional myelography shows cord-size enlargement and small amount of contrast material in cord cavity.**
***B*, Delayed scan at 16 hr shows contrast material within cord cavity better (*arrows*), but outline of cord is not as well defined because of lack of contrast material in subarachnoid space.**

TABLE 2: **Comparison of Sequential Enhancement Patterns of Cord Parenchyma in Different Patient Groups and Normal Controls**

Group: Case No.	Initial Scan			6 hr Scan			15 hr Scan		
	CT No. of SAS (A)	CT No. of CP (B)	Corrected Attenuation of CP (B/A)	CT No. of CP (C)	Corrected Attenuation of CP (C/A)	Trend (C vs B)	CT No. of CP (D)	Corrected Attenuation of CP (D/A)	Trend (D vs B)
Normal control:									
1	341	50	0.15	54	0.16	↑	59	0.17	↑
2	447	48	0.11	72	0.16	↑	59	0.13	↑
3	329	35	0.11	35	0.11	=	64	0.19	↑
4	282	51	0.18	63	0.22	↑	...	...	...
Isolated Arnold-Chiari I malformation:									
5	394	56	0.14	106	0.27	↑	...	...	...
6	324	46	0.14	48	0.15	↑	51	0.16	↑
7	323	71	0.22	68	0.21	↓	65	0.20	↓
8	387	54	0.14	...	...	...	62	0.16	↑
Communicating syringomyelia:									
9	362	61	0.17	...	...	...	26	0.07	↓
10	453	76	0.17	50	0.11	↓	62	0.14	↓
11	384	46	0.12	104	0.27	↑	75	0.20	↑
12	413	37	0.09	76	0.18	↑	75	0.18	↑
13	472	57	0.12	97	0.21	↑	52	0.11	↓
14	202	65	0.32	73	0.36	↑	...	...	...
15	692	68	0.10	...	...	...	62	0.09	↓
Idiopathic syringomyelia:									
16	209	56	0.27	...	...	...	65	0.31	↑
Posttraumatic syringomyelia:									
17	675	95	0.14	...	...	...	87	0.13	↓
18	960	155	0.16	190	0.20	↑	275	0.29	↑

Note.—SAS = subarachnoid space; CP = cord parenchyma.

patients had undergone premyelography scans, either unenhanced or with IV contrast material only. The spinal cord attenuations showed an average increase of 20 H on immediate postmyelography scans over premyelography scans. The trends for idiopathic syringomyelia and posttraumatic syringomyelia are indeterminate.

Sequential Enhancement Patterns of Cord Cavities in Different Groups

The results are summarized in Table 3. In patients with communicating syringomyelia, the corrected attenuation of the cord cavities at 6 and 15 hr postmyelography were both

TABLE 3: **Comparison of Sequential Enhancement Patterns of Cord Cavities in Different Patient Groups**

Group: Case No.	Initial Scan			6 hr Scan			15 hr Scan		
	CT No. of SAS (A)	CT No. of CC (B)	Corrected Attenuation of CC (B/A)	CT No. of CC (C)	Corrected Attenuation of CC (C/A)	Trend (C vs B)	CT No. of CC (D)	Corrected Attenuation of CC (D/A)	Trend (D vs B)
Communicating syringomyelia:									
9	362	61	0.17	...	...	...	48	0.13	↓
10	453	76	0.17	60	0.13	↓	81	0.18	↑
11	385	35	0.09	91	0.24	↑	67	0.17	↑
12	413	37	0.09	128	0.31	↑	118	0.29	↑
13	473	43	0.09	71	0.15	↑	74	0.16	↑
14	202	64	0.32	111	0.55	↑	...	...	...
15	692	112	0.16	...	...	...	86	0.12	↓
Idiopathic syringomyelia:									
16	209	67	0.32	...	...	...	92	0.44	↑
Posttraumatic syringomyelia:									
17	675	95	0.14	...	...	...	718	1.06	↑
18	960	155	0.16	187	0.19	↑	341	0.36	↑

Note.—SAS = subarachnoid space; CC = cord cavities.

generally higher than at zero hours. The cord cavities of patients with idiopathic syringomyelia and posttraumatic syringomyelia also had higher attenuation values at 15 hr than at zero hours. The status at 6 hr was indeterminate because of lack of data.

Postshunt Syringomyelia

The two patients who were examined after shunting procedures constitute a special group. Both patients had idiopathic syringomyelia originally. One had a syringopleural shunt 5 months before the study and the other had a syringosubarachnoid shunt 11 months before the study. In both patients, conventional metrizamide myelography demonstrated spinal cord enlargement. Both patients subsequently underwent CTMM. In the first case, scans were obtained at 6 and 22 hr after myelography. At 6 hr, no cord cavity was seen. At 22 hr, the cord cavity was denser than the cord substance, which had decreased in density since the earlier scan. In the second patient, scans were obtained immediately and at 24 hr after myelography. On the immediate scan, the cord cavity was essentially as dense as the cervical subarachnoid space and much denser than the cord substance. At 24 hr, the cord cavity became much denser than at 0 hr and also much denser than both the subarachnoid space and the cord substance. The cord substance also increased in density after the immediate scan.

Discussion

In our series, CTMM was found to be significantly more sensitive than conventional metrizamide myelography in the diagnosis of Arnold-Chiari I malformation and various types of syringomyelia. The difference was most marked in posttraumatic syringomyelia, where CTMM made the diagnosis in four of four patients, and conventional metrizamide myelography missed the diagnosis in two of four patients. In both these patients, C1–C2 puncture was required because of extensive arachnoid adhesions in thoracic and lumbar areas.

In terms of the timing of scans in CTMM in the diagnosis of syringomyelia, delayed scans (12–24 hr after myelography) were most useful in our series, demonstrating contrast material within the cords in 100% of cases. The immediate scans were the least useful in terms of demonstrating contrast material within the cord, but they served the purpose of defining the sizes of the spinal cords most accurately. Intermediate scans demonstrated contrast material within the cord in only 67% of the cases, although cord-size abnormalities were shown in 100% of the cases. So, for cost-effectiveness, an intermediate scan can possibly be omitted if the purpose of the CTMM is for diagnosis alone, using the initial scans to detect cord-size abnormalities and the delayed scans for demonstrating contrast material within the cords. However, for understanding the dynamics of fluid flow in syringomyelia, intermediate scans are most helpful. They also demonstrate cord-size abnormality and syrinx in the same image, as well as the cord remnant around the syrinx (Fig. 3B).

In studying the sequential enhancement patterns of the spinal cords and cord cavities, some interesting observations were made. The cord parenchyma of normal controls and patients with isolated Arnold-Chiari I malformation became denser at 6 hr and was still dense at 15 hr, whereas the cord parenchyma of patients with communicating syringomyelia showed an increase in enhancement at 6 hr and a subsequent decrease in enhancement at 15 hr. On the other hand, the cord cavities in communicating syringomyelia showed increasing enhancement from 6 to 15 hr.

These observations strongly favor a transneural passage of metrizamide. The sequential increase in the density of cord parenchyma in normal controls suggests a normal transneural penetration of metrizamide after intrathecal injection, which agrees with the observations made by other authors [14–17]. Presumably, this transneural passage of metriza-

mide is greatly hastened in patients with communicating syringomyelia, as indicated by the decreased enhancement of the cord parenchyma at 15 hr compared with the continued enhancement in normal controls. The continued enhancement of the cord cavities at 15 hr in these patients suggests that the passage of metrizamide into the cord cavities occurs later than the penetration of metrizamide into the cord parenchyma. This gives further indirect evidence that metrizamide enters the cord cavities through the transneural route. These observations agree with and add to the evidence obtained by Aubin et al. [14] and others [15, 16, 18], which tend to support the Aboulker theory [8], as well as the India ink studies in syringomyelia autopsy material reported by Ball and Dayan [10]. The exact route of cord penetration cannot be determined from our study.

The span of syrinx seen in communicating and idiopathic syringomyelia varied between two and 10 spinal segments. Its accuracy on CT, however, cannot be tested, as no other imaging studies (syringography, MR, or sonography) were available on these patients.

Similarly, the effect of the volume of the syrinx (and hence the degree of cord expansion) on the degree of contrast opacification of the syrinx cavity would be difficult to evaluate. Theoretically, larger syrinx cavities will produce greater dilution of entering metrizamide, but they are easier to image than smaller ones. In addition, a larger cavity will usually produce a larger cord, and that presumably will give rise to less dilution in the subarachnoid space and probably less rapid cephalad flow of metrizamide.

The fourth ventricle was filled in 78% of the cases in which the posterior fossa was studied. However, no definite cavity could be identified between the obex and the upper end of the syrinx. Although the possibility of microscopic passage of metrizamide through the obex and an attenuated central canal cannot be excluded, our findings do not support the theory of Gardner [7]. Since no children were included in the present study, our results are applicable only to adult syringomyelia.

In conclusion, our study shows that CTMM is not only a sensitive way of diagnosing Arnold-Chiari I malformation and syringomyelia but it also contributes data that lead to better understanding of the dynamics of fluid flow in syringomyelia. Our results support both the Aboulker theory [8] and the Ball and Dayan theory [10], but prospective studies with a larger number of patients are needed before any definite conclusions can be drawn.

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