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*AJNR Am J Neuroradiol* 1989, 10 (1) 65-76
http://www.ajnr.org/content/10/1/65

# Herniation of the Suprasellar Visual System and Third Ventricle into Empty Sellae: Morphologic and Clinical Considerations

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**Intrasellar herniation of the optic nerve, optic chiasm, optic tract (suprasellar visual system, SVS), and anteroinferior third ventricle can occur into primary or secondary empty sellae. The anatomic part of this study evaluated the appearance of the SVS in subjects with normal sellae ($n$ = 52), the patterns and prevalence of SVS herniation in enlarged primary empty sellae ($n$ = 24), and the patterns of intrasellar herniation of the SVS in secondary empty sellae ($n$ = 8). The clinical part of this study was to correlate the visual status with the anatomic patterns of the intrasellar herniated SVS. High-resolution MR and CT were used to define the anatomy. MR was superior to CT in all groups in defining accurately the SVS relationship to the sella turcica.**

**In the normal group, the SVS invariably had a straight-line appearance formed by the optic nerve, optic chiasm, and floor of the third ventricle and was above the sella. The SVS was herniated in three of 24 enlarged primary empty sellae. A difference in the appearance of the hypothalamic and infundibular recesses in the primary empty sella group with SVS herniation (dilated recesses and formation of an obtuse angle) and in the secondary empty sella group with SVS herniation (nondilated recesses and formation of an acute angle) was observed. Visual disturbances in primary empty sellae with SVS herniation were present in two of three subjects. Visual disturbances may be absent or minimal in primary empty sellae and secondary empty sellae with herniation of the SVS. Progression of the symptoms—visual field defects, optic atrophy, and loss of vision—is not inevitable. There was no correlation between the severity of visual symptoms and the degree of herniation of the SVS in either the primary or secondary sellae.**

**We found that intrasellar herniation of the SVS into a primary or secondary empty sella is well delineated with MR, and MR should facilitate decisions concerning surgery or therapy. Visual disturbances proved to be an unreliable indicator of herniation.**

**This article appears in the January/February 1989 issue of *AJNR* and the March 1989 issue of *AJR*.**

Received January 27, 1988; accepted after revision June 2, 1988.

Presented at the annual meeting of the American Society of Neuroradiology, New York City, May 1987.

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***AJNR* 10:65–76, January/February 1989**
0195–6108/89/1001–0065


Herniation of the suprasellar visual system (SVS) (i.e., suprasellar portions of the optic nerves, chiasm, and tracts and the anteroinferior third ventricle) into an empty sella can occur in the presence of either primary or secondary empty sellae [1–5].

The prevalence of herniation of the SVS and associated visual disturbances in primary empty sellae is not well established. A small number of cases suggesting herniation of the SVS have been reported [6–15], and only rarely has herniation been documented radiologically [1–4, 6, 10, 12, 15]. Herniation of the SVS into a secondary empty sella is more frequent and is associated with multiple causes resulting in inflammation and adhesions; associated visual symptoms are variable [1–3, 5, 9, 13, 16–18].

This study was undertaken (1) to determine the normal appearance of the SVS with normal sellae, (2) to determine the appearance and prevalence of SVS herniation into primary empty sellae, (3) to determine the appearance of the intrasellar herniation of the SVS in secondary sellae, and (4) to correlate visual disturbances with the observed patterns of herniation. Surgical procedures are available for therapy of visual disturbances associated with SVS herniation, and MR visualization of the SVS anatomy should be helpful in selecting and planning therapy.

**TABLE 1: Intrasellar Herniation of the Suprasellar Visual System in Secondary Empty Sellae (ES)**

| Case No. | Age | Gender | Clinical Diagnosis or Therapy/ Follow-up Interval (months)[a] | Endocrine | Visual Fields |
|---|---|---|---|---|---|
| 1 | 49 | F | Prolactinoma coexisting in ES/36 | Hyperprolactinemia | Right upper temporal defect |
| | | | Bromocriptine therapy/34 | Normal at 34 & 54 months | No change in defect at 34 & 54 months |
| 2 | 32 | F | Postpartum necrosis/96 | Hypopituitarism | Normal (to confrontation testing) |
| 3 | 51 | F | Large nonfunctional adenoma, radiation therapy/132 | Normal | Bitemporal hemianopsia; normal at 60 months; right temporal field defect at 96 months |
| | | | | Normal | Incongruous right homonymous hemianopia at 132 months |
| 4 | 42 | F | Pituitary adenoma coexisting in ES/132 | Hyperprolactinemia | Normal |
| | | | Spontaneous apoplexy/24 | Hypopituitarism | Normal |
| 5 | 56 | M | Large LH-secreting adenoma, surgically treated/72 | Partial hypopituitarism; elevated LH level | Bitemporal hemianopia |
| | | | Bromocriptine therapy/60 | Partial hypopituitarism; normal LH level | Unchanged |
| | | | Bromocriptine therapy/0 | Partial hypopituitarism; low LH level | Bitemporal hemianopia; worsened, then stabilized |
| 6 | 67 | M | Surgery for large, nonfunctioning adenoma/84 | Hypopituitarism | Normal |
| | | | Pituitary radiation/80 | Hypopituitarism | Minimal bitemporal constriction (equivocal) |
| 7 | 66 | F | Cushing disease; surgery for microadenoma/48 | Hypercortisolism | Normal |
| | | | Pituitary radiation/36 | Recurrent hypercortisolism | Normal OD; optic atrophy OS |
| | | | | Hypopituitarism | Progressive loss of vision OD to central scotoma; temporal field defect 24 months after irradiation |
| 8 | 52 | F | Large, nonfunctioning adenoma, surgery/24 | Partial hypopituitarism | Bitemporal hemianopia |
| | | | Radiation for residual tumor/24 | Hypopituitarism | Defects worsened, then stabilized |

Note.—LH = luteinizing hormone; OD = right eye; OS = left eye.

[a] Interval between date of diagnosis or therapy and follow-up examination or MR study.

## Materials and Methods

With MR we evaluated the appearance and relationships of the anteroinferior third ventricle and SVS to the sella turcica in three groups of human subjects. Herniation was defined as the anteroinferior third ventricle and part of, or all of, the SVS being within the sella turcica; that is, below the theoretical plane of the diaphragma sellae. The recess angle, that angle formed by the posterior wall of the optic recess and the anterior wall of the infundibular recess, was evaluated in all three groups. A primary empty sella was defined radiographically as an intrasellar CSF cistern either with or without enlargement of the sella and with or without remodeling of the pituitary gland. The primary empty sella has no known etiologic factors other than congenital deficiency of the diaphragma sellae and the resultant interaction with the CSF. A secondary empty sella is an empty sella resulting from a specific cause, either iatrogenic, such as surgery and irradiation, or noniatrogenic, such as infarction and infection.

In group 1, the MR studies of the head in 52 consecutive patients examined at University Hospitals of Cleveland for nonendocrine and nonvisual indications were evaluated retrospectively to determine the normal pattern of the SVS in subjects with normal sellae. Special endocrine or visual examinations were not done. There were 22 men 19–73 years old (mean, 44.5; median, 41.5) and 30 women 20–87 years old (mean, 46.0; median, 49.5).

Group 2 consisted of subjects with markedly enlarged primary empty sellae associated with remodeling of the pituitary gland ($n$ = 24). These were identified on MR studies from routine consecutive clinical examinations done at our institution over a period of 6 months. The 15 women and nine men in this group were 26–86 years old (mean, 54.7; median, 53). The detection of an enlarged empty sella was an incidental finding since patients were referred for evaluation of problems not related to pituitary or visual difficulties. The referring physicians were surveyed by questionnaire and asked directly if any visual or endocrine abnormalities were present. One subject had a history of visual field abnormalities for 28 years but had MR for nonvisual and nonendocrine reasons. One subject had had a pneumoencephalographic/tomographic study 19 years before, which demonstrated an enlarged primary empty sella with a normal location of the SVS.

Group 3 consisted of eight patients selected on the basis of identification of the intrasellar herniation of the SVS and third ventricle. The six women and two men in this group were 32–67 years old (average, 51.9; median, 51.5). The clinical characteristics of the eight subjects in this group are illustrated in Table 1. All but the one with

postpartum necrosis had pituitary adenomas. One patient had spontaneous pituitary apoplexy in an empty sella with a coexisting microadenoma. One had radiation therapy only. Three subjects had combined surgical therapy and radiation therapy. One had surgical therapy plus dopamine agonist therapy. One patient with a prolactinoma had a CT study that was nonconclusive for SVS herniation and had received dopamine agonist therapy at the time of MR documentation of intrasellar SVS herniation.

All subjects had MR with a Magnetom 1.0-T, Picker International 0.5-T, or Magnetom 1.5-T system. Those with secondary empty sellae had multiple detailed CT studies with either a Siemens Somatom DRH or a GE 9800 unit that included a bolus of IV contrast material and 4-mm coronal sections at 4-mm intervals.

MR pulse sequences with the 1- or 1.5-T Magnetom unit used coronal ($n = 19$) and sagittal ($n = 51$) T1-weighted spin-echo images, 400/17/4 (TR/TE/excitations), with a $256^2$ matrix, 3-mm slice thickness, and 1-mm gap. T2-weighted sequences were 2000/35,90/1 (TR/first-echo TE, second-echo TE/excitations), with a $256^2$ matrix, 4-mm slice thickness, and 2-mm gap in the coronal plane.

Examinations performed on the 0.5-T Picker unit ($n = 1$) included coronal and sagittal T1-weighted spin-echo images, 500/40/8, with a $128 \times 256$ matrix, 4-mm slice thickness, and 2-mm gap. T2-weighted

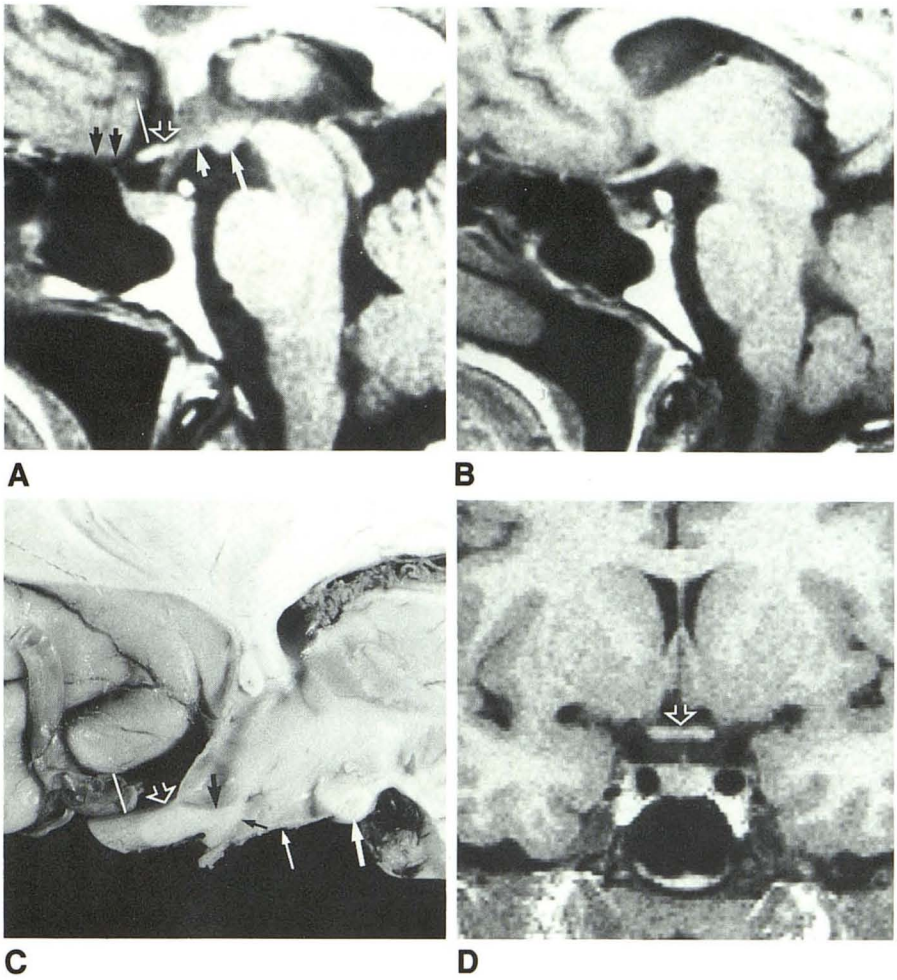



**Fig. 1.—Normal MR appearance of suprasellar visual system (SVS) and anterior third ventricle in adult woman and normal anatomic specimen.**

***A*, T1-weighted sagittal view shows mammillary body (*long solid white arrow*), floor of third ventricle (*short solid white arrow*), optic chiasm (*open arrow*), and optic nerve (*white line*) forming straight line. Optic nerve is directed at optic foramen, whose roof is formed by planum sphenoidale (*black arrows*). Hypophyseal stalk extends from hypophyseal recess of third ventricle to normal pituitary gland. Small amount of CSF is present in proximal hypophyseal stalk, a normal variant.**

***B*, T1-weighted parasagittal view (6 mm lateral to midline) shows optic tract, chiasm, and optic nerve as continuous structure in nearly straight line terminating at optic foramen. Small upward curve or angulation of optic tract posterior to optic chiasm may occur normally. This curve or angulation occurs posterior to dorsum sellae and does not reflect any degree of herniation of SVS.**

***C*, Sagittal anatomic section of anteroinferior third ventricle and SVS shows optic nerve (*white line*), optic chiasm (*open arrow*), straight-line relationship to floor of anteroinferior third ventricle (*small solid white arrow*), and mammillary body (*large solid white arrow*). Optic tract is not normally in this plane of sectioning. Posterior wall of optic recess (*large black arrow*) and anterior wall of hypophyseal recess (*small black arrow*) form recess angle.**

***D*, T1-weighted coronal section shows optic chiasm as straight horizontal bar (*arrow*).**

sequences used 2000/40,50/2 images with a 128 × 256 matrix, 4-mm thickness, and 2-mm gap in the coronal plane. The field of view was 20 cm for all studies. Fifty-two subjects had sagittal examinations; 20 of these 52 had coronal studies also.

## Results

### *Group 1*

The normal appearance of the SVS was determined in 52 subjects with normal pituitary glands and sellae turcica on MR examination. On sagittal sections the optic chiasm and optic nerves formed a straight line from the optic canal (Fig. 1). The floor of the third ventricle continued the straight line to the mammillary bodies. The optic tracts, which pass obliquely posterolaterally from the optic chiasm, continued posteriorly as a straight line in most cases, although in some there was a slight elevation, either curved or angulated, of the more posterior portion of the optic tracts (Fig. 1B). A parasagittal section can image the optic nerve, optic chiasm, and optic tract in the same section, thus accounting for the straight-line appearance continuing from the optic foramen to the region of the geniculate body in those cases in which there was no curve or angulation of the optic tract. Normal variation in the location of the anterior third ventricle relative to the sella did not alter the straight-line appearance of the floor of the third ventricle, optic chiasm, and optic nerve. Figure 1C shows a human gross anatomic specimen in the sagittal view, illustrating the normal straight-line appearance of the optic nerve and chiasm and floor of the third ventricle and recess angle.

In the coronal plane the optic chiasm was seen as a thin uniform horizontal band (Fig. 1D) without any downward angulation. Portions of the optic nerves and optic tracts were appreciated on other sections and obviously did not have any intrasellar representation when there was no intrasellar position on sagittal sections. The anatomy of the SVS was best seen on T1-weighted sequences. On T2-weighted sequences the SVS was isointense relative to other white-matter tracts in the brain and was identified less readily.

The recess angle was acute (less than 90°) in all subjects.

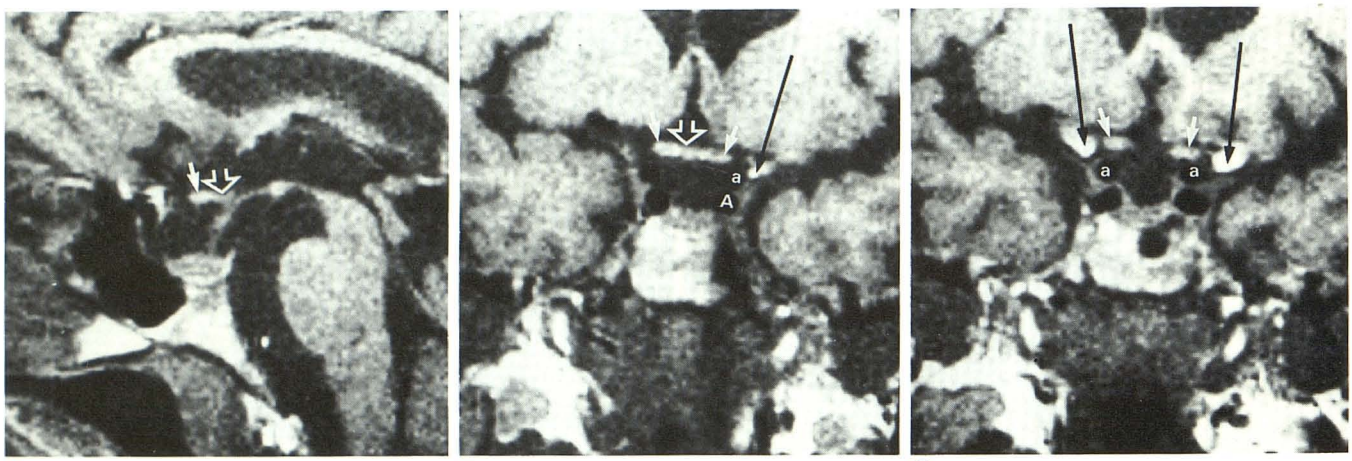



**Fig. 2.—MR appearance of enlarged primary empty sella with no suprasellar visual system or third ventricle herniation. Comparison with CT showed distinctive imaging advantage of MR over CT. A 19-year follow-up comparison with pneumoencephalogram with tomography documented no interval change.**

***A*, T1-weighted sagittal section shows optic chiasm (*open arrow*) and optic nerve (*solid arrow*) to be horizontal in position and in same plane as planum sphenoidale. Mammillary body, floor of third ventricle, and optic chiasm/optic nerve form straight line directed at optic foramen. Other sections showed optic nerve through optic foramen. Hypophyseal stalk traverses intrasellar cistern to normal-sized nonremodeled pituitary gland.**

***B*, T1-weighted coronal section 3 mm in front of plane of hypophyseal stalk shows optic chiasm (*open arrow*) and optic nerves (*solid white arrows*) as nearly horizontal bar. Intracavernous internal carotid artery (A) and intradural infraclinoid internal carotid arteries (a) are low-signal black circles. Left anterior clinoid process (*black arrow*).**

***C*, T1-weighted coronal section 3 mm in front of *B* in plane through anterior clinoid processes (*black arrows*). Optic nerves (*white arrows*) are directly above intradural internal carotid arteries (a) and medial to anterior clinoid processes. Pituitary gland is at bottom of partially empty sella.**

**TABLE 2: Intrasellar Herniation of the Suprasellar Visual System in Primary Empty Sellae**

| Case No. | Age | Gender | Endocrine | Visual Fields | Herniation |
|---|---|---|---|---|---|
| 9 | 75 | M | Normal | Nonprogessive upper bitemporal field defects for 28 years | Marked |
| 10 | 46 | M | Normal | Normal for 20 months | Moderate |
| 11 | 37 | F | Normal | Upper temporal depression in left eye | Minimal |

*Group 2*

In the group of enlarged primary empty sellae, a normal SVS was defined in a way similar to that of group 1. The normal appearance of the SVS was present in 21 of 24 cases (Fig. 2). Any angulation downward of the SVS into the enlarged empty sella, as seen on sagittal and/or coronal sections, was considered to be herniation of the SVS. By using this definition, herniation of the SVS occurred in only three of the 24 cases of enlarged primary empty sellae found incidentally (Table 2; Figs. 3–5). One patient (Fig. 3) showed dilated lateral ventricles but no evidence of obstructive hydrocephalus. Another patient (Fig. 4) had enlarged lateral ventricles and the MR appearance of aqueductal stenosis of the distal aqueduct with dilatation of the proximal aqueduct. There were no clinical signs of hydrocephalus, but CSF pressures were not measured. The subject with minimal SVS herniation (Fig. 5) had a monocular upper temporal depression in the left visual field without any intraocular abnormalities.

The recess angle was acute in 21 subjects, slightly less than 90° in one subject (Fig. 3), nearly 90° in one subject (Fig. 5), and obtuse in one subject (Fig. 4).

*Group 3*

In all eight cases of secondary empty sellae with herniation of the SVS and anterior third ventricle, MR was unequivocally superior to CT in establishing the diagnosis (Figs. 6–10). Three patients with metal artifacts within the sella turcica showed the signal void and displaced signal artifact caused by the intrasellar metal [19]. An abnormal orientation of the floor of the third ventricle was present in these three patients, and the appearance was the same as the abnormal orientation of the floor seen in patients with SVS without signal void artifacts from metal (Table 3). The recess angle was acute in five subjects and not identified in three subjects.

Visual findings included normal vision, visual field defects, optic atrophy, and loss of vision (Table 1).

*Appearances of Herniation*

The detailed anatomic features of intrasellar SVS herniation as seen on MR are given in Table 3 for primary (group 2) and secondary (group 3) empty sellae. Figure 2 is representative of the nonherniated SVS appearance in enlarged primary empty sellae and Figure 6 illustrates the features of intrasellar herniation of the SVS.

## Discussion

The definition of intrasellar herniation of the SVS based solely on spatial relationships is the translocation of the anteroinferior third ventricle and the chiasm with optic nerves and optic tracts into the sella turcica. We found that MR was superior to CT in imaging the SVS, and our experience is consistent with a previous report [20].

The MR appearance of the SVS, optic and infundibular recesses, and anterior third ventricle and their relationships to the sella turcica in normal subjects (group 1) were consist-

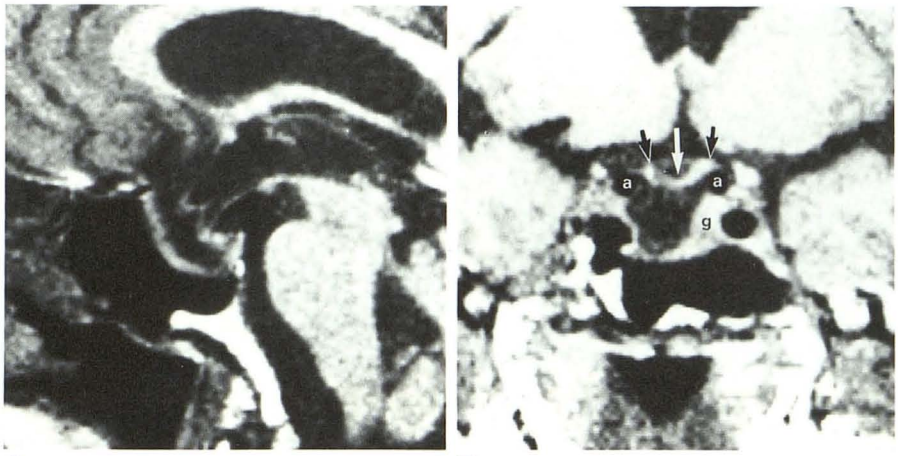
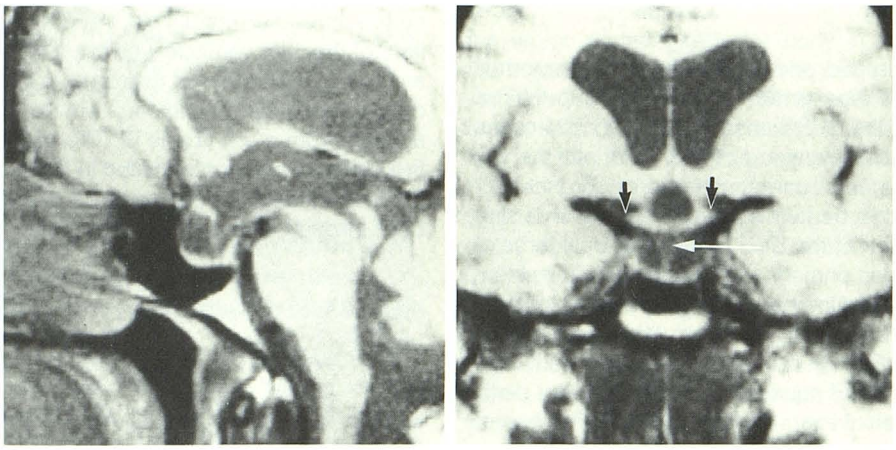



**Fig. 3.—Case 9: 75-year-old man. Pronounced herniation of suprasellar visual system (SVS) and anteroinferior third ventricle into asymmetrically enlarged primary empty sella.**

***A*, Sagittal T1-weighted section shows definite herniation of anteroinferior third ventricle and SVS into enlarged empty sella. Location of optic chiasm, as represented by posterior wall of optic recess (Fig. 1C), had a horizontal orientation. Dilatation of hypothalamic recess is present. Recess angle (formed by posterior wall of optic recess and anterior wall of hypothalamic recess) is slightly less than 90°.**

***B*, Coronal T1-weighted section in plane of anterior clinoid processes shows optic nerves (*short arrows*) diverging upward from inferiorly bowed optic chiasm (*long arrow*) and going superiorly and laterally coursing normally over internal carotid arteries (a) as they are in contact with anterior clinoid processes. Pituitary gland (g) is to left and inferior; on other sections hypophyseal stalk was seen from hypothalamic region to displaced gland.**

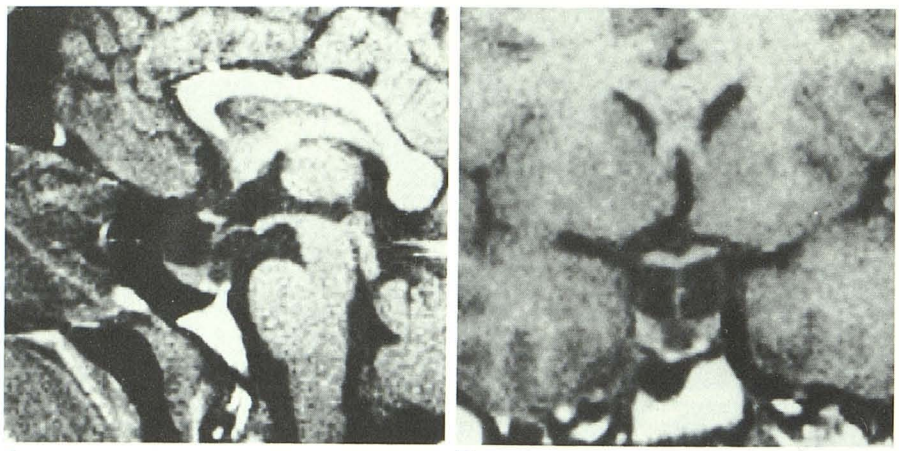


A B

Fig. 4.—Case 10: 46-year-old man. Herniation of infundibular recess of third ventricle and minimal herniation of optic chiasm into asymmetrically enlarged primary empty sella.

*A*, Sagittal T1-weighted section shows dilated infundibular recess and downward displacement of floor of third ventricle. Optic nerve/chiasm complex shows slight herniation of optic chiasm portion. Remodeled pituitary gland is inferior. Lateral and third ventricles are dilated, and there is dilatation of proximal portion of aqueduct. Recess angle is obtuse.

*B*, Coronal T1-weighted section in plane that includes optic tracts (*short arrows*) shows that optic chiasm is slightly bowed but not angled inferiorly. Lateral walls of third ventricle are seen. Dilated infundibular recess (*long arrow*) is midline. Axial high-resolution contrast-enhanced CT scan with coronal reconstruction of sella region showed asymmetrically enlarged hypodense sella turcica with poorly defined linear densities in intrasellar region, raising suspicion of herniation of optic chiasm. Equivocal CT led to MR.



A B

Fig. 5.—Case 11: 37-year-old woman. Minimal intrasellar herniation of dilated infundibular recess and suprasellar visual system (SVS) into primary empty sella.

*A*, Sagittal T1-weighted section shows dilated infundibular recess extending into empty sella. There is horizontal to slightly upward orientation of optic chiasm (see Fig. 1C). Location of optic chiasm is below planum sphenoidale and entrance to sella. Line from mammillary body to region of optic foramen (planum sphenoidale forms roof of optic foramen) is very slightly bowed caudally and is considered to represent minimal SVS herniation. Parasagittal T1-weighted section in plane of optic nerve and optic tract showed that optic nerve had an upward and forward course to optic foramen. Recess angle is almost 90°. Anterior pituitary gland is moderately remodeled on superior surface and there is a hypodensity in posterior pituitary gland (anterior pituitary and posterior gland functions were all normal).

*B*, Coronal T1-weighted section shows very slight but definite angulation of optic chiasm and slight upward direction of optic nerves. Hypophyseal stalk is midline and hypointensity of posterior pituitary is again identified.

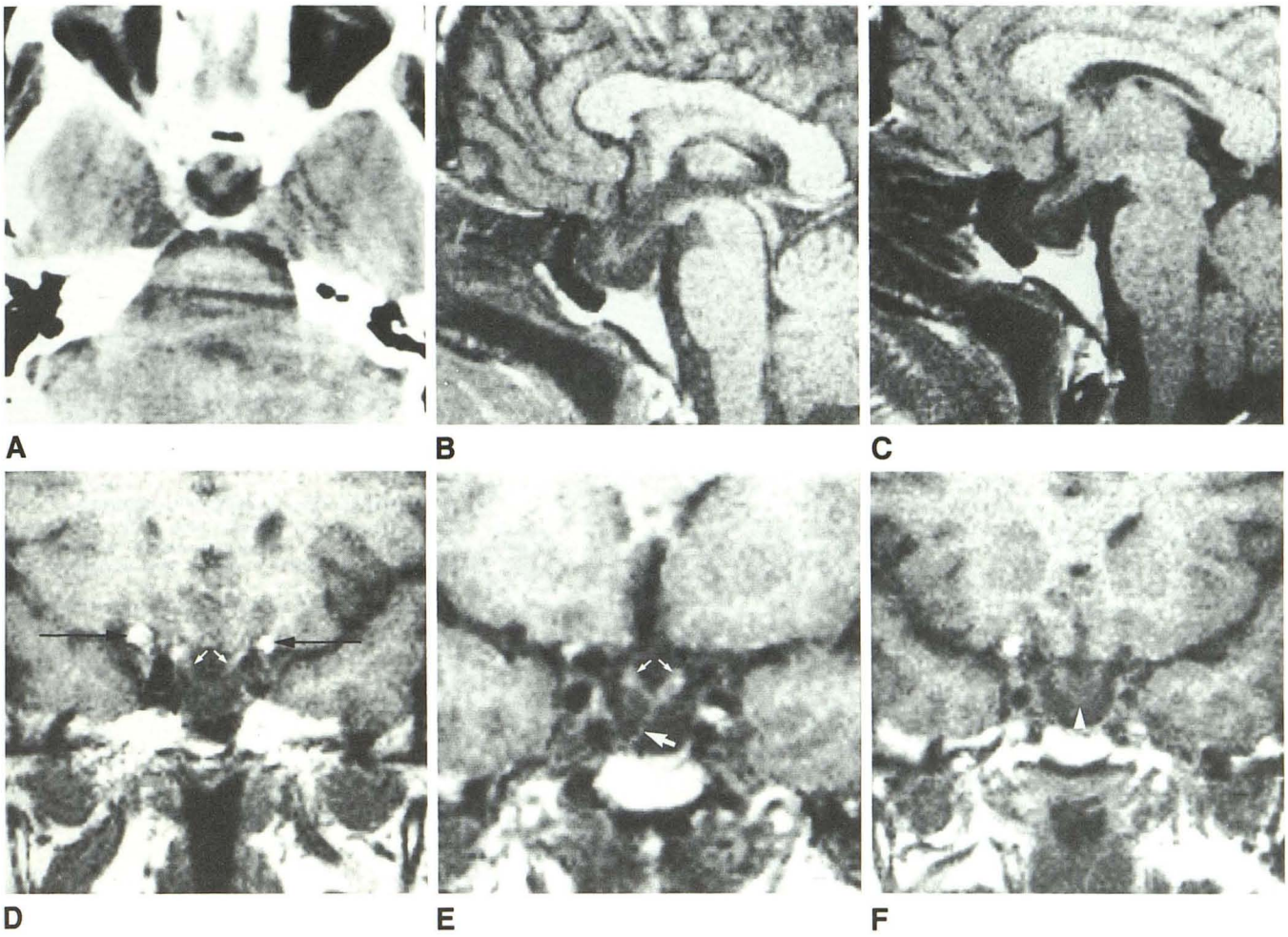



**Fig. 6.—Case 1: 49-year-old woman. Multiple patterns of intrasellar herniation of suprasellar visual system (SVS) and anterior third ventricle into enlarged empty sella in a patient with coexisting prolactin-secreting adenoma.**

***A*, Axial high-resolution nonenhanced CT scan shows enlarged sella with hypodense contrast and diverging tissue densities anteriorly; intrasellar SVS herniation. Original interpretation was "tissue anteriorly" surrounded by CSF or cystic tumor.**

***B*, Sagittal T1-weighted image 1 year later shows herniation of anteroinferior third ventricle and chiasm into enlarged empty sella.**

***C*, Parasagittal T1-weighted image shows composite image of optic tract and chiasm within empty sella.**

***D*, Coronal T1-weighted image in plane of anterior clinoid processes (*black arrows*) shows optic nerves (*white arrows*) going from chiasm in center of sella to optic foramina. Optic nerves are contiguous with intraclinoid portion of internal carotid artery, a normal relationship.**

***E*, Coronal T1-weighted image posteriorly to *D* shows marked angulation of optic nerves (*small arrows*) in plane of hypophyseal stalk (*large arrow*), which deviates to right toward floor of sella. Pituitary gland is presumed to be remodeled inferiorly with component on right side, as indicated by stalk. Pituitary function was normal.**

***F*, Coronal T1-weighted image. Poorly defined density in center of sella is optic chiasm (*arrowhead*).**

ent with the anatomic studies of Schaeffer [21] and with the radiologic appearances as reported by Bull [22] and Rosenbaum et al. [23].

### *Prevalence of SVS Intrasellar Herniation*

Radiographically diagnosed herniation of the SVS in primary empty sellae has been reported rarely [1, 11]. In our cases, the CT demonstration of herniation was difficult, and initial interpretation did not readily identify the findings (Figs. 6 and 7). With secondary empty sellae, the circumstances were the same: Diagnosis of the herniated SVS was difficult to make with CT but could be made readily and accurately with MR. Our experience suggests that intrasellar SVS herniation is more common than previous diagnostic techniques were capable of demonstrating.

### *Patterns of SVS Intrasellar Herniation*

The patterns of herniation are the result of the varying degrees of herniation of the complex of the optic chiasm and anteroinferior third ventricle. The optic nerves and optic tracts have to extend from the herniated optic chiasm to their respective points of fixation, optic foramina and geniculate bodies, respectively, and the abnormal intrasellar course is readily appreciated. The altered orientation of the floor of the

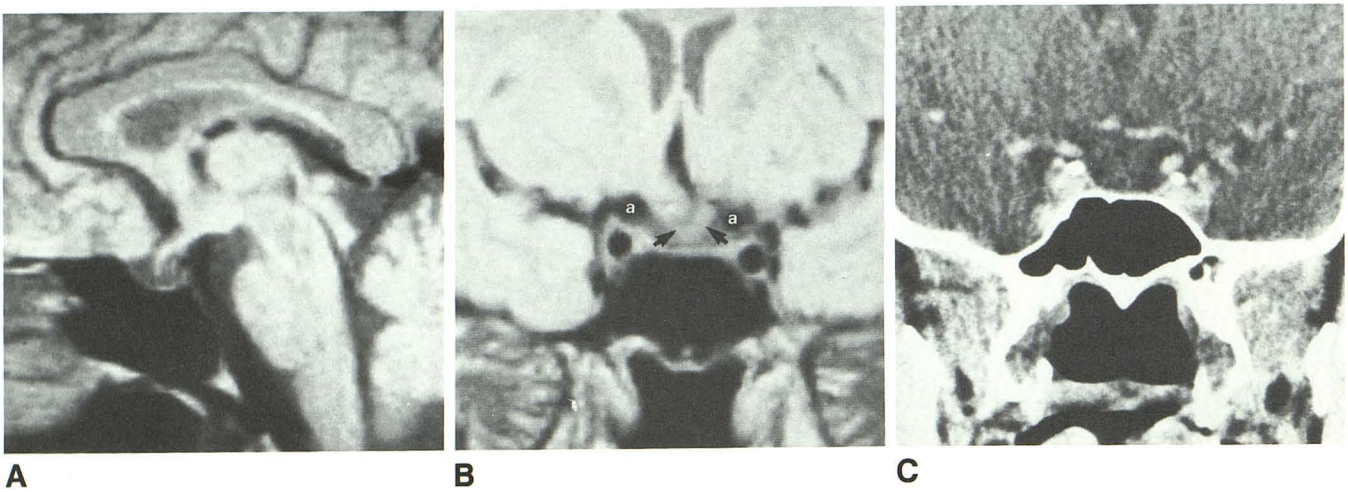



**Fig. 7.—Case 2: 32-year-old woman. Herniation of suprasellar visual system (SVS) into normal small sella turcica in patient with postpartum necrosis of pituitary gland (Sheehan syndrome).**

***A*, Sagittal T1-weighted image shows SVS and opticochiasmal junction herniated into small empty sella.**

***B*, Coronal intermediate-type sequence (2000/40) shows optic nerves (*arrows*) coursing from central inferior portion of empty sella upward to optic foramina and above supraclinoid internal carotid arteries (a).**

***C*, Coronal high-resolution contrast-enhanced CT scan shows vague appearance of optic nerves and optic chiasm within empty sella. This section is anatomically comparable to *B*.**

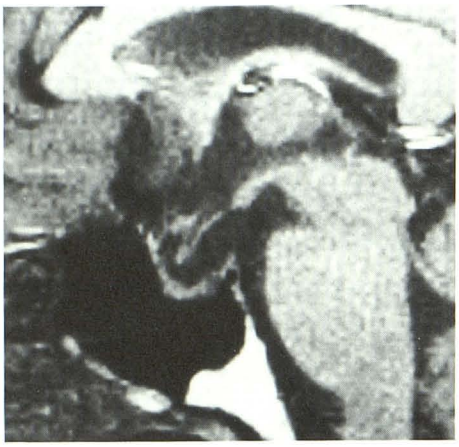


**Fig. 8.—Case 3: 51-year-old woman. Herniation of suprasellar visual system (SVS) and anteroinferior third ventricle into secondary empty sella in patient treated with radiation for pneumoencephalographically/arteriographically proved large pituitary adenoma (chromophobe) with suprasellar extension.**

**Sagittal T1-weighted section shows pronounced herniation of SVS and anteroinferior third ventricle into sella. CSF is within hypophyseal stalk, but there is little dilatation of recess.**

third ventricle is easily identified on sagittal T1-weighted sections and is a diagnostic finding when there is an artifact within the sella such that definition of the optic chiasm and recesses is not accomplished.

The patterns of herniation of the SVS in primary and secondary empty sellae differ somewhat (Table 3). The three cases of primary empty sellae with SVS herniation had dilated hypothalamic recesses and a recess angle of nearly 90° (two subjects) or greater than 90° (one subject) as compared with acute recess angles in the secondary empty sella group. Whether this anatomic difference is meaningful requires further study. The appearance of the dorsum sellae in our cases of enlarged primary empty sellae with SVS herniation was of thinning and intactness consistent with the appearance initially described for primary empty sellae by Kaufman [24] in 1968 and not the truncation of the dorsum sellae seen with third ventricular enlargement secondary to obstructive hydrocephalus.

Herniation of the anterior cerebral arteries was not identified in any of our cases. The anterior cerebral vessels are well seen on MR secondary to flow void and spin dephasing, and demonstration of intrasellar herniation can be expected.

### *Cause of SVS Intrasellar Herniation*

Herniation of the SVS into primary empty sellae is considered uncommon and without a precipitating cause. By contrast, herniation is not uncommon with secondary empty sellae and the precipitating cause is usually evident. Any event that leads to the development of a secondary empty sella with inflammation and adhesions of the SVS (e.g., surgery, radiation, infarction, and infection) can cause herniation. Lee [16] has stressed the role of arachnoiditis with involvement of the optic chiasm resulting in the ischemic chiasma syndrome, but has not described any cases resulting in SVS herniation.

A well-defined cause was evident in seven of eight cases with secondary empty sellae; the cause of herniation was not clearly defined in only one subject (Fig. 6). Because of the suspicion of a pituitary adenoma, which may have infarcted with subclinical manifestations, this patient was placed in the

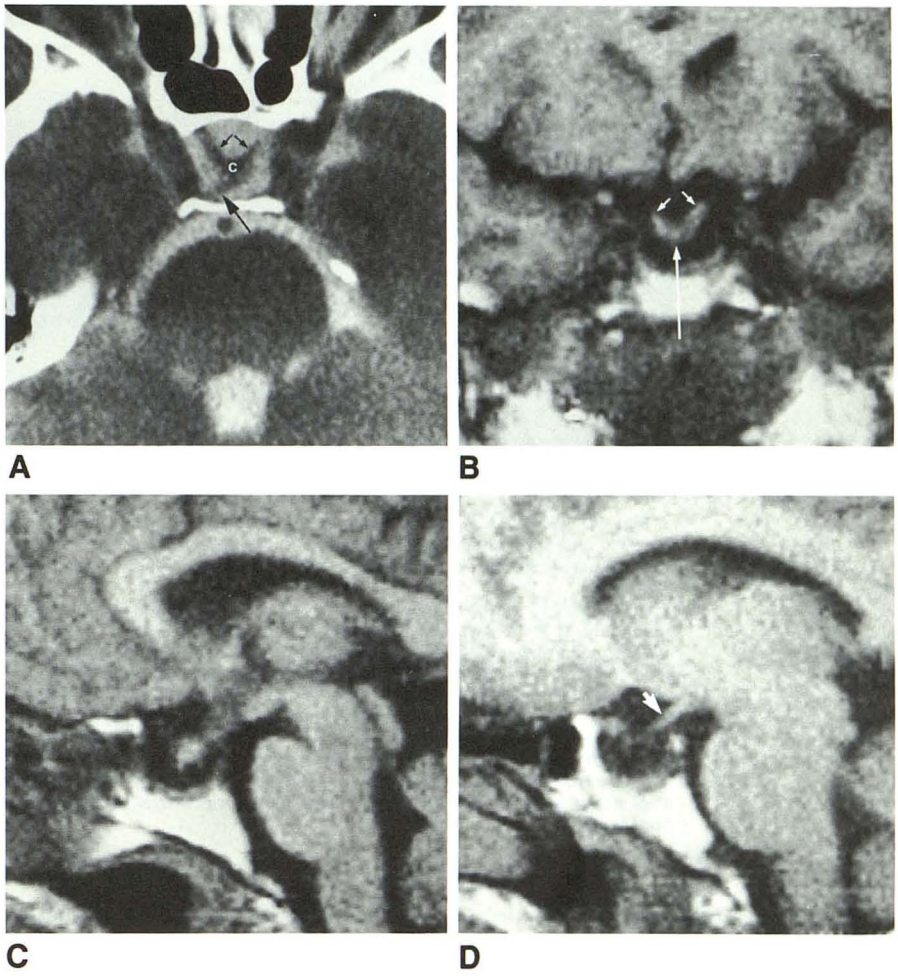



**Fig. 9.—Case 4: 42-year-old woman. Metrizamide-enhanced CT and MR comparison of intrasellar herniation of suprasellar visual system (SVS) into secondary empty sella. Pneumoencephalography with thin-section tomography 11 years earlier showed normal SVS and enlarged primary empty sella with coexisting microadenoma that was suspected to have infarcted spontaneously.**

***A*, Axial metrizamide CT scan after metrizamide cisternography shows unequivocally optic chiasm (c), optic tract posteriorly (*large arrow*), and optic nerves (*small arrows*) diverging anteriorly.**

***B*, Coronal T1-weighted section shows herniated optic chiasm (*long arrow*) and optic nerves (*short arrows*) within empty sella. Pituitary gland tissue is seen posteriorly.**

***C*, Sagittal T1-weighted section shows slight herniation of anteroinferior third ventricle into superior portion of sella turcica and opticochiasmatic junction more inferiorly and centrally within empty sella. Thin rim of pituitary tissue is at bottom of sella.**

***D*, Parasagittal T1-weighted section shows abnormal course of optic tract (*arrow*) directed from intrasellar position of chiasm.**

secondary empty sella group with the understanding that a definitive precipitating cause of herniation was not identified.

This patient (case 6) was treated with a dopamine agonist (Parlodel) after the initial nonconclusive CT scan (Fig. 6A) and before the MR study, which documented SVS herniation. We cannot implicate Parlodel therapy as a factor causing herniation of the SVS in this patient because in all likelihood SVS herniation was present before therapy (Fig. 6A). There is no histologic or autopsy evidence to date of dopamine agonist therapy resulting in adhesions and traction of the SVS [25–31]. If adhesions of the adenoma/gland to the SVS are present before dopamine agonist therapy, it is understandable how herniation of the SVS could occur with reduction in adenoma size and subsequent development of a secondary empty sella. With the increasing use of dopamine agonist therapy, it becomes important to collect data to determine whether dopamine agonist therapy can cause or be associated with intrasellar herniation of the SVS.

Another cause of herniation of the third ventricle into empty sellae occurs with enlargement of the third ventricle resulting from increased CSF pressures with communicating or noncommunicating hydrocephalus. These herniations usually are associated with characteristic bone erosion and truncation of the dorsum sellae, a finding not present in any of our cases. A subtle and not well defined alteration of the CSF pressures and pulsations may exist causing dilatation of the recesses. This possibility has to be considered in the three cases of primary empty sellae with dilated recesses and SVS herniation (Figs. 3–5; Table 3). In case 10 (Fig. 4), the dilated proximal aqueduct is strong evidence for obstructive hydrocephalus, but clinically there was no proof, and diagnostic studies were not believed to be clinically indicated. If the three patients in Figures 3–5 are considered to have an etiologic mechanism, such as an altered CSF pulsation and/or pressure, and are placed in a separate category or in the group of secondary empty sellae, then none of the patients with primary empty sellae ($n$ = 21) had visual abnormalities or herniation of the SVS.

### *Visual Disturbance in Empty Sellae*

Visual disturbances, especially visual field defects, in patients with primary empty sellae are rare. A review of the literature covering the period from 1968 to 1987 showed only 14 cases [1, 6, 7, 9–11, 14, 32, 33]. Of the 14 cases, only two had radiographic demonstration of intrasellar herniation

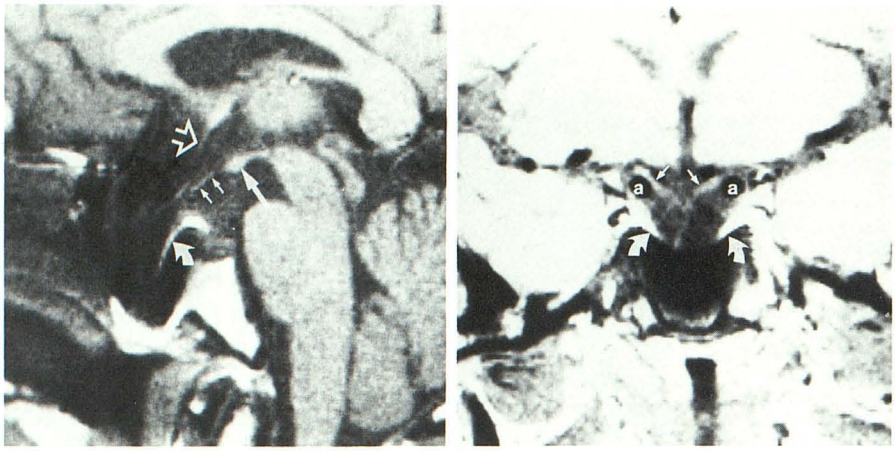



**Fig. 10.—Case 5: 56-year-old man after therapy for intrasellar herniation of anteroinferior third ventricle and optic nerves into secondary empty sella. The patient was treated with transsphenoidal surgical resection and then with dopamine agonist for residual luteinizing hormone–secreting pituitary tumor.**

***A*, Saggital T1-weighted section shows signal-void artifacts and displaced signals (*curved arrow*) secondary to tantalum metal mesh within sella turcica. Floor of anteroinferior third ventricle (*small straight solid arrows*) can be identified, and alignment of floor and mammillary body (*large straight solid arrow*) shows orientation and position that is abnormal and compatible with intrasellar herniation of suprasellar visual system (SVS). Direction of anterior wall of third ventricle and anterior commissure (*open arrow*) supports impression of herniation.**

***B*, Coronal T1-weighted section in plane of anterior clinoid processes shows optic nerves (*straight arrows*) diverging from within sella and coursing upward and laterally, being in contact with intradural internal carotid arteries (a) in pattern consistent with that seen with herniation of SVS. Signal void and displaced signals (*curved arrows*) do not interfere with imaging of herniated optic nerves within sella. Optic chiasm is not identified.**

of the anterior third ventricle [1, 11]. The finding of SVS herniation with visual field defects in two of our three patients (Figs. 3–5) raises the question of herniation being the cause of the visual field defects. Another question that has to be addressed, but is not answered by our material, is whether the altered anatomy reflected in the increased recess angle may mechanically affect the optic chiasm fibers by stretching or angulation.

Visual disturbances are well documented with secondary empty sellae and the causes are multifactorial. Cases of secondary empty sellae with visual loss, visual field defects, and optic atrophy have occurred with operated, irradiated, spontaneously infarcted pituitary tumors, arachnoiditis, increased intracranial pressure with or without hydrocephalus, viral meningitis, and panhypopituitarism [1, 2, 5, 6, 9, 12–14, 34–36]. Our case material shows a spectrum of visual changes consistent with reported findings (Table 1). Visual field defects with secondary empty sellae without SVS herniation occur; therefore, SVS herniation is not a sine qua non for the development of symptoms [13, 16].

The fact that herniation of the SVS can occur in primary empty sellae with absent or minimal visual symptoms and can occur in secondary empty sellae without visual symptoms means that demonstration of herniation of the SVS is not sufficient cause to invoke therapy. In our cases, primary and secondary empty sellae with herniation are characterized by minimal or no visual symptoms or signs, no progression of the visual disturbances over a long follow-up period, and no correlation with the extent of SVS herniation.

With the occasional occurrence of visual symptoms in the general population, and the high incidence of primary empty sellae, it would not be unusual for a coincidental occurrence.

### *Chiasmapexy*

Visual changes associated with intrasellar herniation of the SVS into a secondary empty sella have been treated with chiasmapexy and other surgical procedures [1, 5, 11, 33, 37, 38]. However, chiasmapexy should be considered in light of our case material.

## Conclusions

In summary, diagnosis of intrasellar herniation of the SVS with MR is definitive and readily accomplished with MR. The true frequency of intrasellar SVS herniation has yet to be determined in primary and secondary empty sellae.

Our material shows that visual symptomatology may be absent or minimal, that progression of the visual symptoms is not inevitable, and that there was a lack of correlation between severity of visual symptoms and the marked degrees of herniation of the SVS in the nonsurgical secondary empty sella cases (four of eight) and in the one case of primary empty sella.

**TABLE 3: Anatomic Features of Intrasellar Herniation of the Suprasellar Visual System**

| Feature | Primary Empty Sella | Secondary Empty Sella | | |
|---|---|---|---|---|
| | | Nonsurgical[a] | Surgical | |
| | | | With Metal[b] | Without Metal |
| Optic chiasm (coronal): | | | | |
| Angled | 1 (case 9) | 4 | 0 | 1 |
| Not angled | 1 (case 10) | 0 | 0 | 0 |
| Minimally angled | 1 (case 11) | 0 | 0 | 0 |
| Artifacts | 0 | 0 | 3 | 0 |
| Total | 3 | 4 | 3 | 1 |
| Optic nerves (coronal): | | | | |
| Diverging | 2 (cases 9 & 10) | 4 | 2 | 1 |
| Normal | 1 | 0 | 0 | 0 |
| Not seen | 0 | 0 | 1 | 0 |
| Total | 3 | 4 | 3 | 1 |
| Optic recess: | | | | |
| Enlarged | 3 (cases 9–11) | 0 | 0 | 0 |
| Not enlarged | 0 | 4 | 0 | 1 |
| Artifacts | 0 | 0 | 3 | 0 |
| Total | 3 | 4 | 3 | 1 |
| Hypothalamic recess: | | | | |
| Enlarged | 3 (cases 9–11) | 0 | 0 | 0 |
| Not enlarged | 0 | 4 | 0 | 1 |
| Artifacts | 0 | 0 | 3 | 0 |
| Total | 3 | 4 | 3 | 1 |
| Recess angle:[c] | 1 (case 9) | 0 | 0 | 0 |
| Slightly less than 90° | | | | |
| Almost 90° | 1 (case 11) | 0 | 0 | 0 |
| Obtuse | 1 (case 10) | 0 | 0 | 0 |
| Acute | 0 | 4 | 0 | 1 |
| Not identified | 0 | 0 | 3 | 0 |
| Total | 3 | 4 | 3 | 1 |
| Floor of third ventricle: | | | | |
| Abnormal orientation | 3 | 4 | 3 | 1 |
| Sella: | | | | |
| Intact, enlarged | 3 | 3 | 0 | 0 |
| Intact, not enlarged | 0 | 1 | 0 | 1 |
| Artifacts | 0 | 0 | 3 | 0 |
| Total | 3 | 4 | 3 | 1 |

[a] Example: case 1.
[b] Example: case 5. Signal void and displaced signal artifacts caused by metal obscured features in many instances.
[c] Angle formed by posterior wall of the optic recess and anterior wall of the infundibular recess.

Etiologic factors of SVS herniation in primary empty sellae ($n = 3$) have not been definitively defined, whereas multiple etiologic factors are involved in secondary empty sellae. The significance of the increased recess angle in SVS herniation in primary empty sella has yet to be determined.

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