

# Frequency and Variation of the Posterior Pituitary Bright Signal on MR Images

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Sagittal T1-weighted series with 3-mm sections have routinely been used for all cranial MR studies at our institution. It was apparent from examining these studies that the rate of occurrence of a normal posterior pituitary bright signal was lower than has been previously reported, particularly in older patients. This prompted both a retrospective and a prospective review and analysis of the posterior lobe bright signal in three patient categories. The overall frequency of posterior pituitary bright signal and the influence of sex and age were evaluated in one category. An age-related statistically significant decline in the frequency of posterior pituitary bright signal was found, with a decline rate of approximately 1% per year. An evaluation of the occurrence of anatomic variation in the location of posterior lobe bright signal was made in a second group of 1500 patients. Aberrant location of the posterior lobe was found to be uncommon and was seen most frequently in patients with a sellar fossula. Temporal variation in the presence or absence and size of the posterior lobe bright signal was evaluated in a third group of 36 patients who had at least two MR examinations available for review. Follow-up MR study showed an obvious posterior lobe bright signal in 8% of these patients for whom no bright signal was apparent at the time of initial examination. Loss of the posterior lobe bright signal was apparent in another 25% of patients. A significant change in size of the bright signal was apparent in 19% of patients within this category.

Our results indicate that variation in the bright signal of the posterior pituitary lobe should be expected as a normal physiological occurrence.

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The MR scans from three categories of patients were reviewed to study the rate of occurrence of the posterior pituitary bright signal in normal patients, the rate of occurrence of anatomic variation in location of the bright signal, and the variation in presence of the bright signal and its size in patients who had more than one MR examination.

## Subjects and Methods

The MR studies were obtained on a GE 1.5 T superconducting magnet using multislice spin-echo pulse sequences. Sagittal T1-weighted images were acquired with parameters of 800/20/4 (TR/TE/excitations), 3-mm slice thickness with 0.6-mm interslice gap, 256 × 256 acquisition matrix, and a 24-cm field of view. Coronal T1-weighted images were also obtained in a few patients, with a field of view of 20 cm. The other imaging parameters remained the same. Axial and coronal cardiac gated intermediate and T2-weighted images were acquired with 2000/20, 70/1, 5-mm slice thickness, 2.5-mm interslice gap, 256 × 256 acquisition matrix, and 24- and 20-cm field of view, respectively. The corresponding pixel dimensions were approximately 1 mm × 1 mm for both spin-echo sequences.

Three different categories of patients were examined; category 1 contained two subgroups of 200 patients each. The patients ranged in age from 7 months to 85 years, and the MR diagnosis in every case was either normal study or mild cerebral atrophy. In general, these examinations were performed for clinical indications such as headache and seizure disorder.

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Patients noted to have a moderately or markedly empty sella were excluded. The MR image intensity of the pituitary gland was evaluated retrospectively and prospectively using two different strategies. The first strategy involved analysis of T1-weighted images in 94 females and 106 males. The second strategy involved analysis of T1- and T2-weighted images in 97 females and 103 males. The data were examined for the influence of sex and age on the frequency of occurrence of posterior pituitary bright signal. A case was considered "positive" for posterior pituitary bright signal only if the posterior lobe signal could be delineated as definitely separate from the signal of the dorsum sellae marrow fat. A bright signal was diagnosed when the signal intensity of the posterior lobe exceeded that of the corpus callosum and was of approximately the same intensity as that of the bone marrow fat of the dorsum sellae. Patients without a bright signal were assigned a frequency of 0. Patients with a bright signal were assigned a frequency of one. A linear least squares regression analysis of frequency versus age was performed for the total population and for males and females separately. Although frequency is a dichotomous categorical variable and thus not normally distributed, regression analysis still provides useful data in agreement with the central limit theorem.

The second category of patients was evaluated for anatomic variation in the location of the posterior pituitary bright signal. This category included all cranial MR studies with images judged to be of adequate diagnostic quality obtained over a 6-month interval between September 1987 and March 1988. This group of 1500 cranial MR examinations therefore included both normal and abnormal scans, although patients with sellar and/or parasellar disease were excluded.

The third category of patients was evaluated for temporal variation in the posterior pituitary bright signal. This category included 36 patients examined during the same 6-month interval who also had at least two MR studies of adequate diagnostic quality available for review. The patients ranged in age from 1 to 67 years, with an average age of 31 years, and included 16 males and 20 females. The interval between initial examination and reexamination ranged from 2 to 24 months, with an average of 10.8 months.

**Results**

The frequency of occurrence of posterior pituitary bright signal for patients in category 1 is given in Table 1. The correlation between frequency of posterior pituitary bright signal and age was significant for the total population, and for males and females considered separately using both strategies of T1-weighted image analysis and combined T1- and T2-weighted analysis. The age-related decline in the rate of occurrence of posterior pituitary bright signal is summarized in Tables 2 and 3. As indicated in Table 2, extension of the regression line to age 0 yields an overall frequency at birth of 84.4% using T1-weighted analysis, and 95.7% with T1- and T2-weighted analyses. As shown in Table 3, there is an annual

**TABLE 1: Rate of Occurrence of Posterior Pituitary Bright Signal**

| Images Analyzed     | Total Rate of Occurrence (%) | Female Rate of Occurrence (%) | Male Rate of Occurrence (%) |
|---------------------|------------------------------|-------------------------------|-----------------------------|
| T1-weighted         | 52.0                         | 59.6                          | 45.3                        |
| T1- and T2-weighted | 63.0                         | 67.0                          | 59.2                        |

**TABLE 2: Rate of Occurrence of Posterior Pituitary Bright Signal at Age Zero\***

| Images Analyzed     | Total Rate of Occurrence (%) | Female Rate of Occurrence (%) | Male Rate of Occurrence (%) |
|---------------------|------------------------------|-------------------------------|-----------------------------|
| T1-weighted         | 84.4 ± 13.5                  | 90.8 ± 20.5                   | 80.4 ± 17.6                 |
| T1- and T2-weighted | 95.7 ± 13.2                  | 102.1 ± 19.5                  | 90.6 ± 18.1                 |

\* 95% confidence.

**TABLE 3: Annual Decline of Posterior Pituitary Bright Signal\***

| Images Analyzed     | Total Rate of Occurrence (%) | Female Rate of Occurrence (%) | Male Rate of Occurrence (%) |
|---------------------|------------------------------|-------------------------------|-----------------------------|
| T1-weighted         | 0.956 ± 0.347                | 0.884 ± 0.515                 | 1.078 ± 0.467               |
| T1- and T2-weighted | 0.955 ± 0.338                | 1.000 ± 0.494                 | 0.939 ± 0.470               |

\* 95% confidence.

decline in the rate of occurrence of posterior pituitary bright signal of approximately 1% per year with both strategies of image analysis.

A total of 1500 patients was evaluated in category 2, and in nine cases (0.006%) an anatomic variant in location of the posterior pituitary bright signal was identified. In three patients, the bright signal was located in a sellar fossula inferiorly placed at the root of the dorsum sellae. In three other patients, the bright signal was located inside the pituitary gland in the middle of the pituitary fossa directly in line with the pituitary infundibulum. Location contiguous to the superior surface of the pituitary gland in relation to the distal infundibulum was noted in two patients. In one patient with no clinical evidence of pituitary dwarfism and an otherwise normal MR, the bright signal was found in the median eminence of the hypothalamus.

Patients in category 3 had multiple MR studies available for review and were examined for temporal variation of the posterior lobe bright signal. Nineteen (53%) of the 36 cases available for review showed a significant change in signal intensity. In three cases the initial MR study did not demonstrate a posterior pituitary bright signal while the follow-up examination showed an obvious bright signal. Nine patients had a posterior pituitary bright signal on the initial MR, which was not present on the follow-up MR. In five patients a small bright signal became much larger on the follow-up examination, and in two patients an initially large bright signal became much smaller on the repeat study. Six of the 19 patients had otherwise normal MR examinations and the signal intensity change was an increase in size in three of these and a decrease in the other three. The other 13 patients all had intracranial disease, including two patients with changes of atrophy, two with multiple cerebral infarcts, and nine with tumors in various locations, including one small ganglioglioma of the temporal lobe, one small tentorial meningioma, one recurrent sphenoid wing meningioma, one frontal glioma, three brainstem gliomas, one esthesioneuroblastoma of the

sphenoid sinus and nasopharynx, and one acoustic neuroma with hydrocephalus. None of the patients with tumors had increased intracranial pressure or hydrocephalus except the single person with acoustic neuroma, who had a non-shunted hydrocephalus at the time of both the initial and the follow-up MR studies. This patient was not operated on and had an increase in size of the posterior lobe bright signal on follow-up MR. In the patients with intracranial disease, eight had a decrease in size of the posterior pituitary bright signal on the repeat MR, and five had a bright signal size increase. Overall, in the combined group of 19 patients with normal and abnormal MR studies, 11 patients had a decrease in size of the bright signal and eight had an increase on the follow-up examinations. In the other 17 patients with no change of the pituitary signal on follow-up study, seven had no detectable posterior pituitary bright signal initially.

### Discussion

The posterior pituitary develops from a downward growth and descent of neuroepithelium from the diencephalon. The axons of cells in the supraoptic and paraventricular hypothalamic nuclei, comprising the infundibular stem, transport their polypeptide hormone products (vasopressin and oxytocin), bound to the carrier protein neurophysin, to terminals in the posterior lobe for storage or release. These axon terminals constitute the main bulk of posterior lobe pituitary tissue, with the denser parts related to the perivascular zones, and less dense portions within the intermediate zones [1]. The pituicytes are protoplasmic astrocytes representing the neurohypophyseal glial cell component. The hyperintensity of the posterior pituitary bright signal may be related to its function of storage of the neurophysin-peptide complex, since a bright signal is also seen in the hypothalamic median eminence when it subserves the function of storage, developing into a "miniature posterior lobe" in patients with tumoral compression or destruction of the infundibulum, or after traumatic transection or hypophysectomy [2]. A role for the pituicytes in the storage process of the neurosecretory material with a resultant relationship to the MR bright signal has been postulated [3, 4], but a detailed biochemical explanation at the cellular level has not been elucidated. It has been shown, however, that the neurophysin-peptide complex is stored within vesicles of the neurohypophyseal axon terminals [5] and is released into the perivascular spaces. Pituicyte processes enclose the axon terminals with interposition between them and the capillary perivascular space, and in response to physiologic stimuli for hormonal release, such as dehydration, the number of such enclosed axon terminals diminishes and the number of axon terminals exposed in the perivascular space is increased so that neurohypophyseal hormone release is facilitated [5].

As first reported by Mark et al. [6], the posterior pituitary bright signal on MR was thought to be caused by fat within the sella turcica. Subsequent reports, however, established the origin of the bright signal within the posterior lobe [7, 8]. The MR images of patients with sickle cell anemia sometimes offer further interesting ancillary evidence of the posterior pituitary location of the bright signal. The bone marrow of

these patients is extremely deficient in fat and even a very small posterior pituitary bright signal can be discerned separately from the dorsum sellae in these patients (Fig. 1A). An oblique view, showing to best advantage the direct continuation of the infundibulum into the posterior lobe, may also confirm this observation (Fig. 1B). In the evaluation of sagittal T1-weighted images, patients with a large obvious posterior lobe bright signal present no difficulty in the distinction between pituitary gland and osseous margins of the sella (Fig. 2). In cases in which there is also a very high signal due to high fat content in the sellar bone marrow, examination of the parasagittal sections adjacent to the midsagittal will often resolve the difficulty; sellar bone marrow fat is expected to be evident on more than just one midsagittal section. In some other cases, a thin crescent of low signal intensity may be delineated representing the cortical margin, which shows the separation of the bright signal in the posterior lobe from that of the dorsum sellae marrow fat (Fig. 3).

The rate of occurrence of posterior pituitary bright signal in normal patients has been reported to be as high as 90% and even 100% in some previous publications [7, 8]. In one of these reports [8], no correlation was found between rate of occurrence of the bright signal and patient age. Our analysis of posterior pituitary bright signal occurrence, however, does not confirm the high frequency previously reported and shows a definite and statistically significant relationship to patient age. In two groups of 200 patients studied, the overall frequency of occurrence of the bright signal was 52% when sagittal T1-weighted image analysis was used alone, and 63% when both T1- and T2-weighted images were used for evaluation. Inclusion of the T2-weighted images means that a very small posterior pituitary bright signal, which cannot be identified separately from a high signal of dorsum sellae marrow fat on T1-weighted images, will be apparent owing to change to low signal of the marrow fat on the T2-weighted views. The posterior pituitary bright signal remains hyperintense in both the intermediate and T2-weighted images. Relationship of bright signal occurrence to age when studied with statistical methods also indicated an age decline rate of approximately 1% per year (Table 3).

Anatomic variation in the location of the bright signal is infrequent, and was found in only nine cases of the 1500 cranial MR studies reviewed over a 6-month period (Figs. 4–6). Four of these cases appeared to represent aberrant location of the posterior lobe, three of which had a sellar fossula, with a bright signal seen within it (Fig. 4A). In a single case (Fig. 6) the bright signal was noted in the hypothalamic median eminence in association with a sella turcica, which was unusually small. Hypothalamic median eminence location of the bright signal has been reported in patients with pituitary dwarfism [4, 9]. The patient in this case, however, had no clinical findings of pituitary dwarfism and was therefore thought to have the uncommon anatomic variant of an undescended posterior lobe. In two other patients, a posterior pituitary signal was noted along the superior surface of the remainder of the pituitary gland at the termination of the pituitary stalk. This could represent incomplete descent of all or part of the posterior lobe. There appeared to be CSF within the posteroinferior part of the sella in these patients (Fig. 5A).

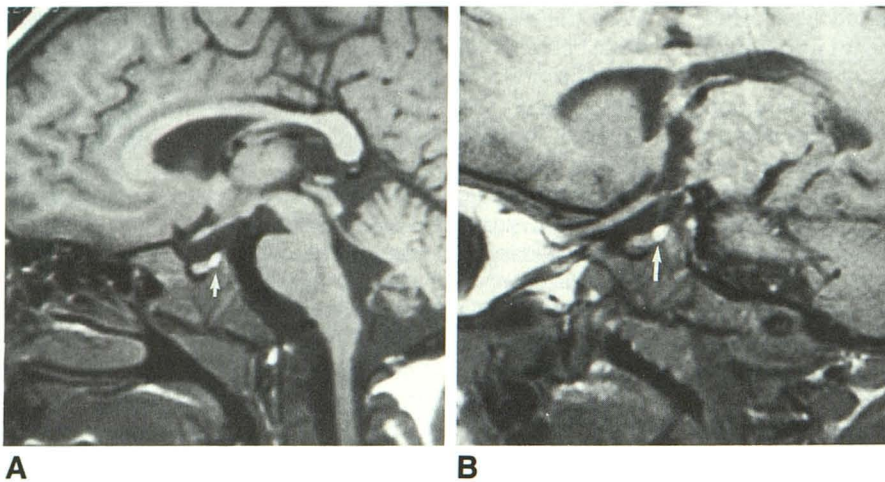


Fig. 1.—Small normal posterior pituitary bright signal in 5-year-old with sickle cell anemia. Sellar bone marrow is devoid of fat.

A and B, Midsagittal (A) and oblique (B) MR images (800/20/4) show bright signal adjacent to dorsum sellae (arrow). Direct relation of infundibulum to posterior lobe is shown on oblique view, which also demonstrates intracranial portion of optic nerve to advantage.

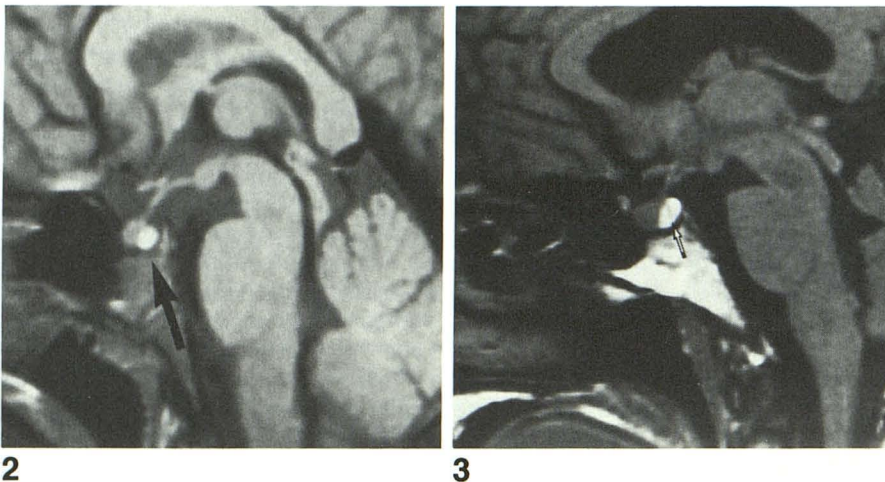


Fig. 2.—Midsagittal MR image (800/20) shows normal large posterior pituitary bright signal (arrow) in 7-year-old girl.

Fig. 3.—MR image (800/20) in 41-year-old man with mild atrophy shows bright signal of fat in dorsum sellae (arrow) separated from posterior pituitary bright signal by a thin crescent of low signal representing the osseous cortex.

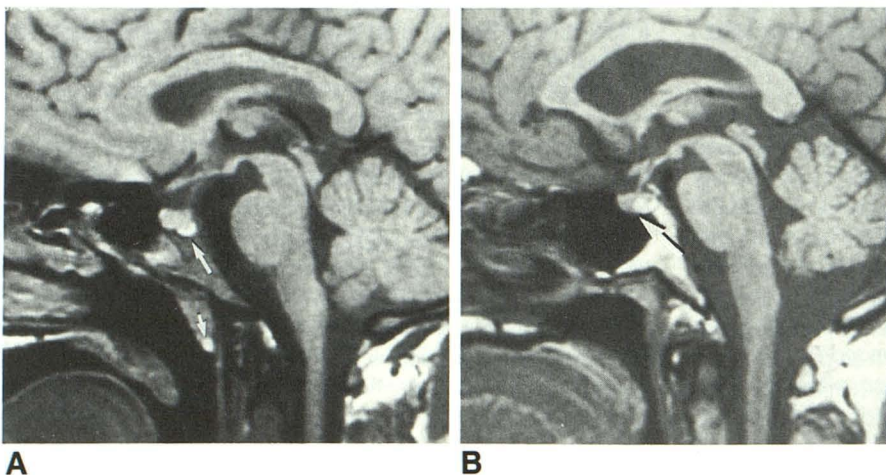


Fig. 4.—MR images (800/20) show normal variants of posterior pituitary bright signal.

A, Inferior location of bright signal inside a fossula of the sella (upper arrow) in 24-year-old man with sickle cell anemia. Incidental Thornwaldt cyst in posterior pharynx (lower arrow) is of same signal intensity as posterior pituitary bright signal.

B, 31-year-old man with 6-month history of dizziness and a normal MR. Posterior pituitary bright signal is partly seen in the middle of the pituitary gland in line with pituitary stalk (arrow).

Differences in appearance of the posterior lobe bright signal are probably due to physiological variation much more often than to variation in anatomic location. The posterior pituitary bright signal may usually correspond to only a part of the posterior lobe. The bright signal, then, can involve the entire posterior lobe (Fig. 2) or may be seen within only a portion of

the posterior lobe (Fig. 1). If the bright signal is most obvious in the anterior part of the posterior lobe, it will appear approximately within the middle of the sella turcica (Fig. 4B). In other cases (Figs. 7A and 8B), the bright signal appears to extend toward or may be seen within the anterior pituitary lobe. This observation has interesting anatomic and physiological cor-

Fig. 5.—MR images (800/20) compare posterior pituitary bright signal variant with a probable Rathke pouch remnant.

A, 23-year-old man with aqueduct obstruction caused by quadrigeminal plate glioma. Posterior pituitary signal is above superior surface of pituitary gland at termination of pituitary stalk (*arrow*). This is thought to represent a normal anatomic variant. Although unlikely, previous hydrocephalus might have had some effect on this anatomic appearance.

B, 32-year-old man with seizure disorder and atrophy. A posterior pituitary bright signal in normal location is contiguous with dorsum sellae. Round isointense signal in relation to termination of infundibulum may represent Rathke pouch remnant or small craniopharyngioma.

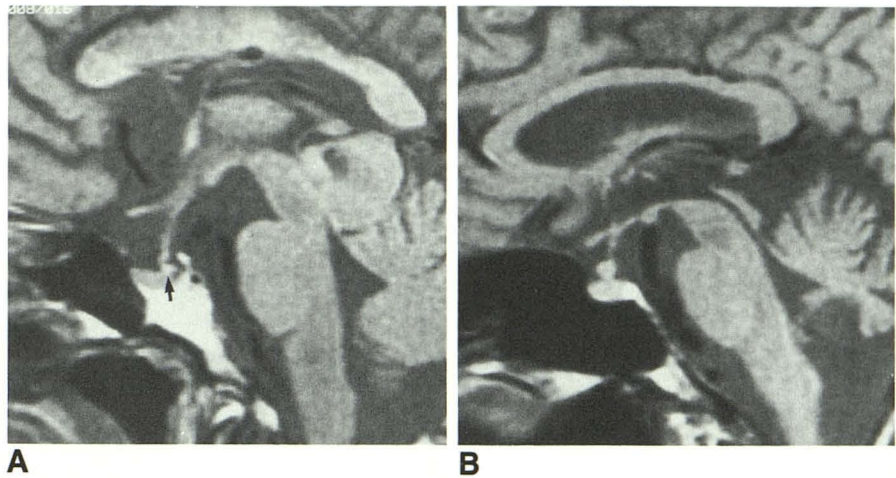


Fig. 6.—Normal variant of posterior pituitary bright signal in median eminence of hypothalamus.

A, Normal MR image (800/20) in 28-year-old woman with complaint of dizziness. There is a large bright signal in hypothalamic median eminence and pituitary stalk with a small pituitary fossa that most likely contains only the anterior pituitary gland. There is a pseudodiverticulum of the lamina terminalis of the third ventricle (*arrow*).

B, 1-year follow-up MR image (800/20) shows reduction in size and intensity of median eminence bright signal (*arrow*). This is consistent with a normal variant rather than disease, such as neoplasm. The presence of an additional developmental anomaly (third ventricle pseudodiverticulum) lends support to this conclusion. The patient had no clinical evidence of pituitary dwarfism.

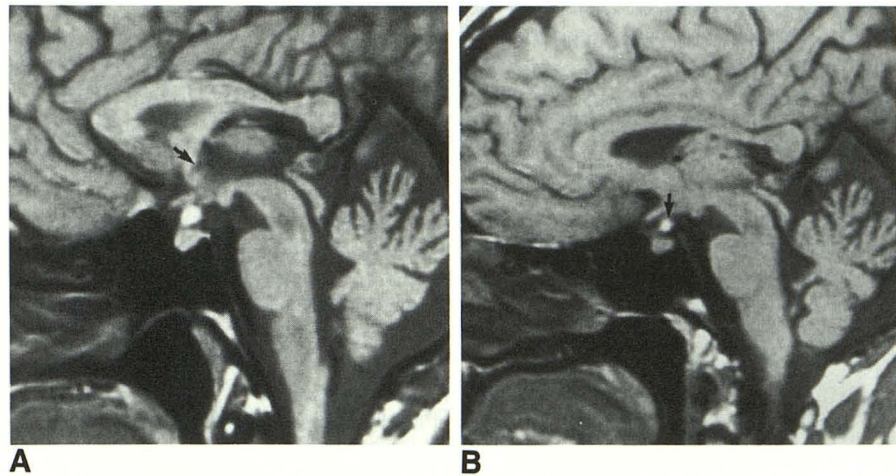
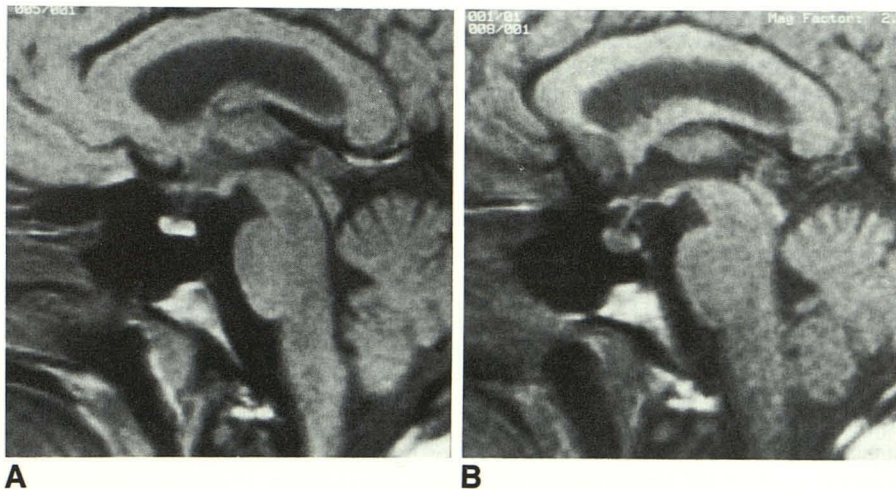


Fig. 7.—Temporal variation in intensity of a normal pituitary signal in 28-year-old man with seizure disorder.

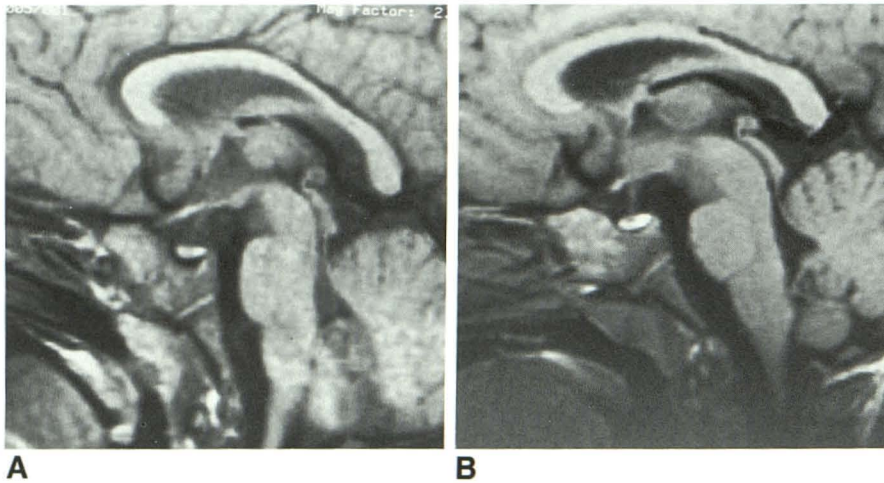
A, Initial MR study (800/20) shows extension of bright signal toward anterior pituitary.

B, Repeat MR examination (800/20) after 13-month interval shows marked regression of pituitary bright signal. There was no change in clinical status of the patient during this interval.



relates. In addition to the pathway of axoplasmic flow down the infundibulum to the neurohypophysis, it has been shown in the monkey that vasopressin-neurophysin is released into the hypophyseal portal system so that it reaches the anterior lobe [10]. Detailed animal pituitary blood flow studies using microcinematographic techniques have also shown continu-

ity of blood flow between the capillary beds of the neurohypophysis and the anterior lobe [11]. In addition to the known effect of vasopressin on the cells of the renal collecting tubules, the vasopressin-neurophysin complex is now believed to exert an influence on the action of ACTH-releasing factor in the anterior pituitary [10].



**Fig. 8.**—2-year-old girl after resection of a right medial temporal lobe glioma that extended into mesencephalon.

**A,** Initial MR study (800/20) shows small bright signal in posterior pituitary.

**B,** 18-month follow-up MR examination (800/20) demonstrates increase in size of tumor involving mesencephalon and hypothalamus. No treatment was given during this interval. The posterior pituitary bright signal shows increase in size. The bright signal is also seen extending into the anterior lobe.

Animal studies have demonstrated the effects on the posterior pituitary of such factors as stress and dehydration [1, 12]. In a small group of patients studied in category 3 who demonstrated significant changes in the bright signal at the time of different MR examinations, no clear pattern emerged either for patients with normal MR studies or for those with intracranial disease (Figs. 7 and 8). The MR observations in this group of patients suggest that the presence or absence of a posterior pituitary bright signal and its variation in size may reflect, at any given time, the endocrine functions of the neurophysin peptide complex, which are undoubtedly determined by numerous factors responding to a dynamic and changing physiological state. This postulate has correlation with studies that have examined the effect of such factors on plasma levels of vasopressin [13–16]. The posterior pituitary bright signal may be a direct reflection of the amount of storage of neurophysin vasopressin complex within the posterior lobe at any given time. On this basis, variation in the bright signal of the posterior pituitary lobe would therefore be expected, and in fact, should represent the physiological norm.

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