Commentary

MR of the Pituitary Gland: Functional Imaging?

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Remarkable progress has been made over the past two decades in pituitary gland imaging. Whereas plain radiographs and pluridirectional tomography provided only an indirect assessment of the pituitary gland by disclosing changes in the adjacent bone, CT and MR allowed direct multiplanar visualization of the gland itself. MR, more than any other of its imaging counterparts, demonstrated the gland and its surrounding structures in elegant detail. Yet despite many remarkable improvements, all imaging methods remained tools for morphologic assessment; they gave no indication of pituitary gland function. The only "radiologic" technique that could assess function was selective sampling of the inferior petrosal veins for hormonal assays. In reality, this was a biochemical test in which the radiologist participated rather than an imaging method unto itself.

This issue of the AJNR contains two papers that describe pituitary gland appearance in various disease states (1, 2). I have been asked by the Editor to comment on the current status of MR of the pituitary gland as a functional imaging technique based on these reports. I was specifically asked, "Are these papers describing morphology of the gland, or gland function?" Each paper needs to be examined individually to answer this question.

Moses et al analyzed the posterior lobe in three disorders: neurogenic diabetes insipidus (DI), primary polydipsia, and nephrogenic diabetes insipidus. The MR was justified on the basis that these groups of patients are difficult to separate on clinical grounds or on the basis of standard water deprivation tests. Six patients with primary polydipsia and eight patients with central DI were evaluated. They were able to use MR to differentiate primary polydipsia from central DI. They found the normal hyperintense signal of the posterior lobe to be present in all the primary polydipsia patients and in none of those with central DI. They concluded that the presence or absence of the high signal intensity is a useful and clinically important differential diagnostic tool in patients with hypotonic polyuria. This conclusion is partially justified. It is justified in that the presence of the high signal indicates normal vasopressin stores and, therefore, an intact neurohypophyseal system; it eliminates central DI as a diagnostic consideration. On the other hand, the absence of the signal is not as straightforward. In Moses' series, three of four patients with nephrogenic DI lack the signal which patients that are thought to synthesize and secrete vasopressin normally possess. Therefore, in nephrogenic DI, the absence of the signal does not indicate absence of neurohypophyseal function, only a depletion of neurosecretory reserves. Furthermore, two of 92 normal patients did not have the signal. Other authors have found that anywhere from 0 to over 20% of normal people do not have this signal (3–5). What can be concluded about *their* neurohypophyseal function?

In large part, the inability to demonstrate the normal high signal is likely due to imaging technique (head tilt, thick sections, etc) or an anatomic variation in the gland, such as small size or eccentric position. Yet, in clinical practice, these same anatomic variations and technical limitations are encountered every day. They are problematic. Therefore, attaching significance to the lack of posterior lobe high signal must be made cautiously; lack of the signal should only be interpreted as abnormal if the examiner *is absolutely certain that a normal posterior lobe cannot be identified anywhere in the sella turcica.* With that proviso, Moses' paper does demonstrate that MR can assess neurohypophyseal reserves of neurosecretory material, and qualifies MR as a functional imaging method for the posterior pituitary gland. Similarly, Fujisawa, Colombo, Tien (and others) had previously demonstrated the absence of the high intensity signal in the diabetes

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insipidus (3–5). Furthermore, Fujisawa was able to show depletion of the high intensity signal in animals subjected to long-term hypertonic feeding (6).

Lundin et al describe serial changes in macroprolactinomas of the anterior pituitary gland in patients undergoing long-term bromocriptine therapy. They found that in a follow-up period spanning several years bromocriptine reduced the size of all tumors. The size reduction was significant within 1 week of initiating therapy and, in some cases, shrinkage continued for several years. They further describe continuous intratumoral signal intensity pattern changes that they feel represent internal transitions between intratumoral hemorrhage, cysts, and necrosis. Finally, they found that there was a significant trend towards increasing T2 values over time. They ascribe this to an increase in water content in the tumor.

Lundin's paper is fundamentally different from Moses' work in that it follows along traditional radiologic descriptions of morphologic assessment, albeit in greater detail than previously possible. Lundin confirms previous observations that bromocriptine visibly reduces tumor size and causes intratumoral tissue changes (7–9). In contrast to Moses, Lundin's paper does not assess pituitary function. Even the lengthening of T2 is a morphologic demonstration; its clinical significance and functional implication is still uncertain. Perhaps at some point in the future it might be shown that long T2 adenomas are less hormonally active, but this is purely speculative.

Medical "imagers" are continually experiencing an improvement in the capabilities of imaging methods to assess morphology. This is progressively being supplemented by newly discovered and welcome abilities to assess tissue function. We are now witnessing interesting and valuable adaptations of these new capabilities to clinical practice.

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