Imaging of Pediatric Pituitary Abnormalities

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- Pituitary imaging
- Pituitary malformations
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The normal pituitary is a bean-shaped gland that resides in the sella turcica, a fossa in the superior sphenoid bone within the central skull base. In humans, the gland has two dominant lobes, the posterior lobe (neurohypophysis, neural lobe) and the anterior lobe (adenohypophysis). The adenohypophysis is further comprised of: the pars distalis, which forms the most the anterior lobe; the pars tuberalis, which partially envelops the inferior stalk; and the pars intermedia, which correlates with the intermediate lobe in lower animals and which may secrete pro-opiomelanocortin (POMC) in the younger population, but is felt to involute by adulthood.1 Anterior and posterior lobes are distinct embryologically and functionally. The hypothalamus, which is situated directly above, along the inferolateral aspect of the third ventricle, directs activity to each lobe by a different mechanism.

**Magnocellular pathway.** Hormonal supply to the posterior pituitary (neurohypophysis) is mediated through the magnocellular neurosecretory pathway. Because the posterior gland develops in contiguity with the hypothalamus, axons run continuously from the hypothalamic supraoptic and paraventricular nuclei into the neurohypophysis. The secretory mechanism relies on preserved axonal structure to transport oxytocin and vasopressin-containing vesicles from the cell bodies into the neurohypophysis.

**Parvocellular pathway.** The anterior pituitary (adenohypophysis) develops from an embryologic anlage that is distinct from the hypothalamus. As a result, no axonal contiguity exists between the hypothalamus and the anterior lobe. This mechanism of hypothalamic-pituitary communication relies instead on the hypothalamic-hypophyseal portal system. The portal system transmits parvocellular messengers from the hypothalamic median eminence into the capillary bed of the anterior gland, where the messenger hormones can effect their influence to regulate adenohypophyseal secretion. Hormones secreted from the anterior gland include those that are derived from the basophilic population (follicle-stimulating hormone), chromophobe (prolactin), and gonadotropes (follicle-stimulating hormone, luteinizing hormone).
hormone, luteinizing hormone, corticotropin [ACTH], and thyroid-stimulating hormone) and those that are derived from the eosinophilic population (prolactin and growth hormone). In addition to these classic hormones, it is clear that the adenohypophysis also secretes many additional growth factors, cytokines, neurotransmitters, and other peptides.2

Both the hypothalamic axons and the hypophyseal portal system course through the hypothalamic infundibulum into the pituitary stalk. The stalk then inserts on the gland at or near its midline, at approximately the junction of the mid and posterior thirds. This position correlates roughly with the midpoint of the junction of the larger anterior and smaller posterior lobes. In practice, there can be significant variability even within the normal population in the overall size of the gland, the relative proportions of anterior and posterior lobes, the laterality of stalk insertion, and the overall bilateral symmetry of the gland.

Because of their differing embryologic origins, the anterior and posterior lobes have distinct arterial supplies.3,4

Posterior pituitary supply. The posterior pituitary derives its supply primarily from the inferior hypophyseal arteries, which arise bilaterally from the meningohypophyseal trunks. These meningohypophyseal trunks, which branch from the dorsal precavernous internal arteries, also give rise to local tentorial and meningeal branches. The inferior hypophyseal arteries may also supply portions of the superficial (peripheral) anterior pituitary lobe.

Anterior pituitary supply. Supply to the anterior gland is predominantly through the hypophyseal portal system on the surface of the pituitary stalk, which drains from the median eminence. Arterial supply to the median eminence itself is derived from the superior hypophyseal arteries, which may arise from either the posterior communicating arteries or from the supraclinoid internal carotid arteries directly. The superior hypophyseal arteries also supply portions of the superior stalk and optic chiasm.5 The anterior lobe also receives venous blood from short portal vessels draining from the posterior lobe, accounting for up to one-third of the anterior pituitary supply.

Venous drainage. Venous blood drains from the anterior and posterior lobes into the cavernous sinuses. Blood draining from these sinuses may be sampled via simultaneous bilateral inferior petrosal sinus catheters to suggest laterality of secretory tumors when imaging is unable to resolve tumor location.

Although the sella itself is formed as a fossa within the central sphenoid bone, sellar margins are largely comprised of dura. Thus, the superior margin of the sella is defined by a dural plane, the diaphragma sella, with the infundibulum penetrating through the diaphragmatic hiatus to meet the gland below. The osseous anterior sellar wall, sellar floor, and posterior sellar wall are also lined by dura, which may consist of a thin sheet of fairly uniform thickness or it may be somewhat thicker, particularly around the anterosuperior and posterosuperior portions of the gland. Venous intercavernous sinuses may run within a thickened dural margin to communicate with one or both cavernous sinuses.5 A dural plane also separates the sella from the cavernous sinuses laterally. Sinusoids within the cavernous sinuses contain venous drainage from not only the pituitary but also inflow from the ophthalmic veins, middle cerebral veins, and sphenoparietal sinuses. The sinuses further drain into the superior and inferior petrosal sinuses, pterygoid plexuses, and ophthalmic and angular veins of the face. It is the drainage into the inferior petrosal sinus that enables venous sampling via jugular catheters to determine the side of a hormone-secreting lesion.
Knowledge of surrounding structures is crucial for the surgical transsphenoidal endoscopic approach that has become widely used for exploration to the gland. Thus the floor and anterior wall of the sella may both abut the sphenoid sinus, offering a potential surgical window for resection of intrasellar pituitary tumors. Patients in whom tumors extend significantly into the suprasellar compartment may, in addition, require a subfrontal craniotomy for more complete tumor resection.

**EMBRYOLOGY**

The pituitary gland, and indeed the entire hypothalamic-infundibular-pituitary axis, develop through a complex interplay between the developing diencephalon and the primitive oral stomodeum.¹⁻⁸ The process has been studied extensively in lower animals, and is believed to parallel the development in humans albeit at a different rate. Development and tropism proceed through opposing gradients of growth factors, transcription factors, and signaling proteins to encourage or discourage differentiation of specific cell types along what will become the infundibulum, stalk, and pituitary gland.

Development of the hypothalamic-pituitary axis is felt to proceed through 4 stages¹

1. **Pituitary placode.** At approximately the fifth week of gestation, the anterior ridge of the neural plate displaces ventrally to form the roof of the developing oral cavity. The roof of the oral cavity thickens to compose the pituitary placode, which marks the onset of pituitary organogenesis.

2. **Rudimentary Rathke pouch.** As the oral cavity roof invaginates, it forms the rudimentary Rathke pouch. Because of a lack of overlying mesenchyme, when the roof invaginates it soon comes into contact with the evaginated diencephalon that will ultimately develop into the posterior pituitary. The contacting portion of the pouch develops into the pars intermedia.

3. **Definitive Rathke pouch.** Rathke pouch further deepens and folds on itself until it closes and the communication of the Rathke pouch cavity with the primitive oral cavity is lost. During this phase, the continued evagination of diencephalon results in development of the infundibulum.

4. **Adult Gland.** There is no further communication of the pouch with the oral cavity, and the specific cell types have developed in a specific spatial and temporal sequence.

Inherent in the development of the pituitary and associated structures is a well-orchestrated signaling system that allows cell differentiation and migration in specific orders. Thus, early in development the ventral diencephalon secretes several factors, including bone morphogenetic proteins (BMPs), Wnt5a, and fibroblastic growth factor (FGF) 10. Likewise, the oral ectoderm expresses Sonic Hedgehog (Shh) and Lhx3, ultimately thickening to form the pituitary placode. Gradations in signaling hormones such as BMP2 and FGFs provide for differentiation of specific pituitary cell types in their characteristic spatial and temporal arrangement. For example, there is a greater tendency for gonadotropes, thyrotropes, lactotropes, and somatotropes to develop ventrally (in this order, with gonadotropes most ventral), and melanotropes and corticotropes to develop dorsally.⁹ Several factors are necessary to develop the posterior lobe, including Pit-1, Oct-1 and Oct-2, and Pit-Oct-Unc domain factors of the Brn family. Defects in specific genes may cause precise malformations. For example, a mutated or absent Brn-2 does not affect initial hypothalamic development, but it is associated with failed differentiation of the hypothalamic supraoptic and paraventricular nuclei; without these nuclei, the axons normally leading from them do not
form and therefore the posterior gland will not form normally either. The reader is
referred to several excellent reviews of the genetics of pituitary development and
genetic basis of anomalies.\textsuperscript{1,6,10–12}

**IMAGING TECHNIQUE**

Preliminary evaluation of the sella is almost always performed using magnetic reso-
nance imaging (MRI). Although an MRI examination takes considerably longer than
computed tomography (CT) (at least 20 minutes for MRI and perhaps up to 35–40
minutes depending on the sequences obtained, compared with 5 minutes for CT),
MRI offers markedly better parenchymal definition than CT, uses no ionizing radiation,
and may be acquired in any orthogonal or oblique plane. MRI is not prone to the beam-
hardening artifact from the surrounding sphenoid bone that is present on CT, but other
artifacts can influence image quality, particularly metallic artifacts in dental braces or
prostheses.

Sequences obtained for MRI vary significantly by institution, and choice of
sequences depend on the specific scanner hardware available, field strength, and
radiologist preference. In general, scans are obtained to survey the gland and
surrounding tissue adequately, with coverage in at least 2 planes. Thus examinations
might include sagittal and coronal T1-weighted and T2-weighted sequences, and
gadolinium-enhanced sagittal and coronal T1-weighted sequences. Because sellar
structures are so fine it is imperative to obtain thin-section images to limit the effects
of partial volume averaging, a condition in which 2 adjacent structures cannot be
clearly resolved because they lie within the same slice. Traditional imaging at 1.5 T em-
ployed 2 or 3 mm scan thickness with little or no gap (10% or less) between sections.
With advances in scanner technology, and with the growing popularity and prevalence
of 3 T scanners, many centers now obtain 1- or 1.5-mm sections or even volumetric
scans to assess the sella. However, because increasing magnetic fields are associ-
ated with greater susceptibility artifacts, further advances in field strength may not
offer similar gains in imaging resolution. Artifacts are of particular concern when scan-
ning the sella, particularly in the peripubertal population in whom dental braces are
common; technologists may be able to modify scanning parameters to minimize the
effect of such metallic artifacts.

In addition to high-resolution sellar imaging, many institutions also obtain one or
more survey sequences of the entire brain. As certain pituitary abnormalities can be
associated with other midline defects, these larger field images should be scrutinized
for congenital or developmental anomalies.

Some pituitary abnormalities, most notably microadenomas, are best or most reli-
ably seen using T1-weighted sequences obtained with gadolinium chelate administra-
tion. Gadolinium is a lanthanide element that has 7 unpaired electrons, giving it
paramagnetic properties.\textsuperscript{13} This atom shortens the so-called T1 and T2 relaxation
times of water. As a result, gadolinium that either remains in the blood pool or extra-
vasates from the blood pool through a damaged blood-brain barrier causes the con-
taining structure to appear bright, or hyperintense. This property distinguishes
enhancing tissues from nonenhancing tissues.

It has long been known that free gadolinium is highly toxic to humans. However, when
gadolinium is chelated to specific ligands it has a favorable safety profile. Ligands in
clinical use include agents such as diethylene triamine pentaacetic acid (DTPA), which
bind strongly to the gadolinium ion thereby minimizing release of free gadolinium into
serum. Until about 2006, the approved gadolinium chelates were believed to have
minimal potential side effects apart from the occasional allergic reaction. However, it
has become clear that patients in dialysis-dependent kidney failure are at significant risk for a recently described and remarkably debilitating scleroderma-like disorder known as nephrogenic systemic fibrosis (NSF, formerly known as nephrogenic fibrosing dermopathy).\textsuperscript{14} Although there is evidence that patients with a glomerular filtration rate <30 mL/min/1.73 m\textsuperscript{2} are at greatest risk for NSF, particularly patients dependent on dialysis, many centers exercise caution in patients with even moderate renal failure (GFR \(\geq 30–59\) mL/min/1.73 m\textsuperscript{2}). Depending on the degree of renal function remaining, these patients may be offered a half-dose preparation of a newer cyclic agent with greater T1-relaxivity, conferring improved contrast enhancement even at a lower dose. Alternatively, the contrast may be withheld entirely with recognition of the limitation on the diagnostic yield of the study.

**Appearance of the Normal Sella**

The pituitary stalk extends inferiorly as a direct continuation of the hypothalamic infundibulum, projecting into the gland through a hiatus in the diaphragma sella. The stalk is broadest superiorly and tapers inferiorly to its insertion in the gland (\textbf{Figs. 1 and 2}). Although the upper limits of each segment of stalk have been defined in adults (3.5 mm at the median eminence, 2.9 mm at the midpoint and 1.9 mm at its insertion\textsuperscript{15}) corresponding standards are scarce in children and assessment of pediatric stalk size tends to be based on experience rather than reference to age-appropriate measurement ranges. In children, as in adults, the stalk should taper from infundibulum to insertion on the gland, and caliber significantly less than 1 mm at any point should be regarded with suspicion.

In general the stalk is best seen on postgadolinium T1-weighted imaging because of the strong enhancement of the stalk. The stalk is moderately well seen on T2-weighted imaging because cerebrospinal fluid (CSF) acts as a natural source of contrast against the lower signal stalk. It is seen poorest on T1-weighted imaging because CSF and stalk are relatively close in inherent signal.

\textbf{Fig. 1.} A coronal section through the mid-sella. Note the tapering stalk that penetrates through the hiatus of diaphragma sella (not labeled) to insert on the central pituitary gland. The third (III), fourth (IV), ophthalmic trigeminal (V\textsubscript{1}) and maxillary trigeminal (V\textsubscript{2}) nerves sit in the lateral dural wall of the cavernous sinus; the sixth cranial nerve (VI) is the only nerve actually within the sinus, usually near the inferolateral margin of the internal carotid artery (ICA). The ICA continues anterior to the depicted plane before projecting intracranially, superiorly, and posteriorly back into plane, where it bifurcates into the middle cerebral artery (MCA) and anterior cerebral artery (ACA).
CSF surrounding the stalk follows the properties of simple fluid, specifically low signal on T1-weighted imaging and high signal on T2-weighted imaging. Sometimes CSF pulsation artifacts reduce T2 signal in areas of greatest fluid motion, that is, displaced from other turbulence-causing structures such as the stalk and supraclinoid arterial structures.

Fig. 2. Normal MR imaging through the sella. (A) Coronal T2-weighted image showing the stalk inserting on the central gland. Portions of the cavernous internal carotid artery (Cav Int Car Art) are shown, with the abducens nerve (VI) near the inferolateral vessel margin. The optic chiasm, supraclinoid internal carotid artery, and anterior cerebral arteries are also labeled. (B) Sagittal noncontrast T1-weighted midline image through the sella. The hypothalamus (Hypo) and infundibulum (Infun) continue into the stalk, which inserts just anterior to the junction of the “bright spot” laden neurohypophysis (NH) and adenohypophysis (AH). The chiasm sits directly superior to the pituitary. The CSF-filled third ventricle (3V) is seen as a low signal fluid-filled structure. Portions of the brain stem including midbrain (MB) and pons are also labeled. (C) Sagittal postcontrast T1-weighted image. Distinction between AH and NH is lost, because the anterior gland is now enhanced. The infundibulum and stalk also enhance well. Portions of the sphenoid bone (Sph) and clivus (Cl) are also labeled. (D) Coronal postcontrast T1-weighted image shows many of the structures in (A). Note how well and homogeneously the gland enhances. In addition, with the benefit of contrast cranial nerves can be seen as nonenhancing foci in the lateral cavernous sinus wall: third (III), fourth (IV), ophthalmic trigeminal (V1), and maxillary trigeminal (V2).
internal carotid arteries. Ordinarily the diaphragma sella is not well seen on routine sequences, however heavily T2-weighted sequences obtained at higher resolution (such as fast imaging employing steady-state acquisition [FIESTA] or constructive interference in steady state [CISS] imaging) may clarify the diaphragma in some patients by intensifying signal in the adjacent CSF.

Although the stalk must communicate with both the posterior and anterior lobes of the pituitary, it is frequently seen entering the gland near the midline above the posterior aspect of the anterior lobe, rather than at the junction of the anterior and posterior lobes. This apparently discordant configuration is likely related to the frequent anterior protrusion of the posterior lobe tissue in the midline that meets the stalk at its insertion. This lobule of posterior lobe, which is almost too thin to resolve by MRI but is clearly seen on gross specimens, is partially surrounded by the pars tuberalis of the anterior lobe.5

The gland is traditionally felt to be bilaterally symmetric, but within the normal population there exists considerable asymmetry even within normally functioning glands. Thus the pituitary stalk may normally insert slightly off midline, the configuration of the posterior pituitary bright spot may lie off-center, and even the gross contour of the gland may vary from side to side. In addition, the sella turcica itself may not be symmetric, either on a developmental basis or related to acquired factors such as tortuosity of the cavernous internal carotid artery.

Almost directly superior to the stalk’s insertion on the gland sits the optic chiasm. On coronal images this may appear ovoid or slightly indented, such as the cross section of an erythrocyte. Signal tends to be isointense to white matter on T1-weighted imaging and T2-weighted imaging. It is ordinarily symmetric, and does not exhibit significant enhancement following gadolinium administration.

Lateral to the sella are the cavernous sinuses. Although they collect blood from discrete veins, instead of hollow central cavities the cavernous sinuses contain countless sinusoids, which together return venous blood to the petrosal sinuses. The sinusoids are isointense on T1-weighted imaging, slightly hyperintense on T2-weighted imaging, and enhance significantly with gadolinium because of the relatively large concentration of blood that fills them. The most conspicuous structure within each cavernous sinus is the cavernous segment of the internal carotid artery. The rapid flow of blood in these arteries leaves little magnetic signal, and thus normally flowing arterial blood appears dark. The cavernous carotid arteries loop anteriorly before continuing intracranially, ultimately bifurcating into the middle and anterior cerebral arteries.

Poorly seen about the sinuses, but of great clinical and surgical importance, are the third to sixth cranial nerves. Of these, only the abducens nerve (CN VI) actually sits within the sinusoids, usually near the inferolateral margin of the cavernous internal carotid artery. The remaining cranial nerves in this region, the oculomotor nerve (CN III), trochlear nerve (CN IV), ophthalmic trigeminal nerve (CN V1), and maxillary trigeminal nerve (CN V2), actually reside in the lateral cavernous sinus wall. Although the entire trigeminal nerve complex leaves the pons and enters Meckel cave together, the fibers of the mandibular portion (CN V3) are the most inferiorly placed among all the trigeminal fibers, and are best seen on sella scans as they exit the foramen ovale.

**GLAND SIZE AND SIGNAL**

Several large series of pediatric subjects have showed considerable variability in the size, shape, and signal of normal pediatric pituitary glands. Even among endocrinologically normal subjects of identical age and gender, gland measurements may vary
significantly. Therefore the size of the gland must be considered within the clinical scenario, and assessment of gland function may be normal across a wide range of pituitary sizes and shapes.

The normal gland is hyperintense on T1-weighted imaging at birth, so that the anterior and posterior lobes are almost indistinguishable. The gland is also physiologically enlarged, with a corresponding concave superior margin. The high gland signal and prominent size are usually attributed to the lingering effects of maternal hormones. In the next 8 to 10 weeks the gland assumes the infant configuration: (1) the volume declines to a normal infant size; and (2) T1 signal in the anterior lobe drops to near isointensity with the posterior pons, the structure that is ordinarily used as an internal reference on pituitary studies (Fig. 3).

Signal in the posterior gland remains high on T1-weighted imaging in most patients. The source of this neurohypophyseal bright spot has been the subject of spirited debate. It is well known that fatty substances appear bright on T1-weighted imaging, so the lipid-rich secretory vesicle bilayer wall had historically been believed to account for the high T1 signal. However, it is also well known that concentrated protein solutions may also give a high T1 signal, and many investigators now believe that the neurophysin-vasopressin complex itself accounts for much of the high signal in the

![Fig. 3. Normal postnatal progression of the pituitary gland. All sagittal T1 noncontrast midline images. (A) At birth and through the early days of life the anterior lobe is hyperintense, and the gland is prominent with a convex superior margin. (B) After approximately 2 weeks the gland has begun to shrink and the anterior lobe is not quite as bright. (C) By approximately 2 months the gland assumes its normal infant state, with lower signal in the anterior gland and slight concavity of the superior gland margin.](image-url)
posterior gland. The relevance lies not in the source of the signal but its very presence. High signal in the posterior gland suggests a functioning hypothalamic-infundibular-pituitary axis, whereas the ectopic location of this signal (ie, hypothalamus, infundibulum, or stalk) suggests disruption of the normal axonal pathway leading into the posterior gland. This signal is characteristically absent in patients with central diabetes insipidus.

The normal pituitary gland enhances symmetrically following administration of intravenous gadolinium. Intravenous gadolinium is used to characterize blood flow and tissue types within the sella. Dynamic contrast-enhanced sella scans, which require special preparation and are thus not included in the routine scanning protocol of most centers, have revealed the timing and pattern with which the hypothalamic-pituitary axis enhances. In general, the gland enhancement may take well over a minute, and with modern scanners many sets of images may be obtained in that time to chronicle temporal progression. The normal hypothalamus does not enhance well but the infundibulum does, reflecting the concentration of gadolinium in the blood filling the portal plexus that supplies the anterior gland. Enhancement then spreads down the pituitary stalk, with contrast especially intense at the diamond-shaped pituitary tuft at the stalk insertion. From the tuft, the contrast spreads centrifugally, first filling the central adenohypophysis and then incrementally enhancing to the outer margin. Anterior gland enhancement is considerably more robust than posterior gland enhancement, but because the posterior gland is intense even at baseline it also appears bright after contrast. Indeed, reference to unenhanced T1-weighted images is necessary for interpretation of postcontrast scans, because one must be sure that high signal on those scans actually reflects enhancement and not baseline signal. Many gland lesions enhance less than the anterior gland, but some sellar lesions (such as a diaphragma sella meningioma) may enhance quite prominently.

The simplest and earliest method used for measuring gland size was measurement of the maximum height in the midline. Gland height may indeed be the single most useful measurement in gland size determination, and it is still used for quick approximation of gland size. However, there is significant variability in the shapes of normal glands, and a gland that appears shallow may have a large width (transverse measurement) and/or depth (anterior-posterior measurement) resulting in normal volume overall. A more advanced technique uses the product of the maximal 3 orthogonal dimensions and a correction factor (0.524, or \( \pi/6 \)), which is derived from the formula for the volume of a sphere (\( 4\pi r^3/3 \)); because glands are usually aspherical this formula is inaccurate in most situations. The most accurate formula based on current imaging technology adds the volume of the gland on each slice, either from a single plane (usually sagittal) or by averaging results from 2 or 3 planes. Indeed, investigators have found high correlation between measurements obtained in different planes, confirming the accuracy of both measurements. From these studies, representative normal pituitary growth curves can be generated (Fig. 4). However, ranges of normal do vary among investigators, emphasizing the difficulty of relying on so-called normal ranges obtained using differing scanning techniques.

Glands of preterm infants are taller than those of normal term infants, even when correcting for gestational age. Preterm glands are so tall that preterm infants with a corrected postnatal age of up to 6 months have larger glands than even 2-year-old normal term children. The large gland size may relate to reduced insulin-like growth factor 1 and increased growth hormone in premature infants.

In normal term children the pituitary height is fairly stable for the first 2 years of life, with some investigators measuring a modest decline in this period and others showing modest growth. Much of the gland growth in the first 2 years is derived from...
Fig. 4. Relative size of the pituitary gland in the first 16 years and contribution of posterior lobe (Data from figures adapted from Takano K, Utsunomiya H, Ono H et-al. Normal development of the pituitary gland: assessment with three-dimensional MR volumetry. AJNR Am J Neuroradiol 1999;20(2):312–5). (A) Diagram illustrating relative growth of the total pituitary. Dashed lines indicating approximate means, with solid lines representing +1 and −1 standard deviation. Black lines indicate boys; gray lines and gray background indicate girls. Boys exhibit a relatively linear growth of the pituitary from birth to the mid-second decade, perhaps exhibiting a slight increase in slope at and beyond puberty. Girls have similar rate of growth in the first half decade, but from that point through puberty the rate increases at a faster rate compared with boys. (B) Relative growth of posterior lobe. Line and background shades parallel (A). In boys the rate of growth of the posterior gland is most rapid in the first half decade, before resuming a shallower slope through the mid-second decade. In girls the slopes and total volumes tend to be slightly lower, but the total posterior lobe volume shows greater variability after 16 years.
increases in gland width and depth, and the rate of gland growth is similar in boys and girls.

Gender-associated differences in rate of gland growth tend to manifest themselves after 5 years of age, with boys and girls experiencing similar linear growth of their glands up to that point. Boys continue the relatively linear rate of growth into puberty, with slight increase in growth rate beyond this into the late second decade. After the first half decade, overall growth rate in girls is greater than that in boys, until about puberty when the growth slope declines to a rate similar to that seen in the first half decade. A significant portion of gland growth in the first half decade in boys is related to enlargement of the posterior gland, with more balanced gland growth beyond this. Posterior gland growth is less prominent in girls in the first half decade, and anterior lobe growth becomes even more dominant into puberty. By adulthood, female glands are slightly larger than male glands and remain so into old age.

The characteristic neurohypophyseal bright spot on T1-weighted imaging may be absent even in normal patients, but such a pattern is more common in adult populations than in children. When pathologic, absence of this posterior pituitary hyperintensity reflects either a central secretory deficit, disruption of the integrity of axons projecting from hypothalamus to neurohypophysis, or hypoplasia of the gland (Figs. 5–7). Fujisawa has defined 4 typical scenarios in which posterior gland intensity is abnormally low:

1. A subset of the normal population does not accumulate significant neurophysin-vasopressin (NP-VP) complex, but these patients are asymptomatic and clinically normal. These patients likely secrete VP soon after it is formed, and therefore do not accumulate enough to confer high signal to the posterior gland.

2. Mass effect within the sella from tumor or even granulomatous disease may, by virtue of its bulk, functionally prevent normal axonal transport of vesicles, resulting in their accumulation just superior to the mass. This is referred to as the “damming phenomenon.”

3. When the stalk is transected, either on a congenital basis or from prior trauma, a high signal focus of variable size may be identified along the stalk trajectory.

![Image](https://example.com/image.png)

**Fig. 5.** Ectopic posterior pituitary gland. (A) Sagittal T1-weighted noncontrast image showing a focal hyperintensity along the inferior hypothalamic margin (arrow). No stalk is seen and the pituitary gland is hypoplastic (arrowhead). (B) Coronal T1-weighted noncontrast image revealing the focal hyperintensity representing nontransported neurophysin-vasopressin vesicles stalled at the inferior hypothalamic margin.
but ectopically (ie, above the level of the normal neurohypophysis). Thus, even though vesicles containing NP-VP may form normally in the hypothalamus, they will not be completely transported into the gland.

4. Patients with central diabetes insipidus may not show any hyperintensity from hypothalamus to pituitary, reflecting an inability to manufacture or package any appreciable NP-VP.

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Fig. 6. Markedly hypoplastic pituitary with absent bright spot. (A) Sagittal noncontrast T1-weighted and (B) contrast-enhanced T1-weighted imaging shows almost complete absence of the gland with an associated shallow sella (arrowheads). The upper infundibulum is present but the characteristic high T1-weighted signal is not seen on noncontrast imaging. After contrast administration (B) the location of the infundibulum and the faint band of sellar pituitary (arrowheads) are defined. The chiasm appears thin but is present (hatched arrow).

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Fig. 7. Anterior pituitary aplasia and posterior pituitary ectopia. (A) Coronal T2-weighted image reveals a thin stalk terminating in the bulbous, incompletely formed posterior pituitary tissue (arrowhead). (B) Sagittal T1-weighted noncontrast image showing hypothalamic hypoplasia, a thin infundibulum, and the bulbous neurohypophysis (arrowhead) dipping into a shallow, malformed sella. No anterior pituitary was identified.
In the past, anecdotal evidence had suggested that a breech delivery was associated with an ectopic neurohypophysis. Breech delivery was believed to predispose to extreme shearing influences on the stalk, possibly in part related to a consequence of the sharp edge of the diaphragm sella against the stalk. Research has shown that birth trauma may explain only perhaps a third of the cases of pituitary dwarfism, indicating that abnormal mediobasal brain induction may underlie both a pituitary axis abnormality and breech positioning. However, postnatal trauma has more clearly been associated with pituitary dysfunction, with clinically detected endocrine dysfunction in 10% of pediatric patients subject to mild to severe traumatic brain injury. Deficiency of growth hormone is the most prevalent feature. Additional consequences from head trauma include precocious puberty relating to the inability of damaged extrahypothalamic loci to inhibit gonadotropin secretion.

PITUITARY ADENOMAS

Adenomas arise almost exclusively in the anterior lobe. These tumors seem to behave differently in children than in adults. In adults, tumors that are functional tend to present when small (microadenomas, measuring <10 mm); tumors that are larger (macroadenomas) tend to present because of consequences of mass effect (such as the classic bitemporal hemianopsia seen with chiasmal compression). In children many of the secreting tumors are quite large at diagnosis. In one pediatric series, at least 70% of tumors had suprasellar extension at diagnosis, and almost 10% were further characterized as giant tumors measuring more than 4 cm.

Pituitary adenomas are rare in childhood, accounting for less than 3% of all supratentorial tumors. Approximately 4% of pituitary adenomas are found in children. However, adenomas are more likely to be functioning in children than in adults. Indeed, some investigators report that up to 95% of pediatric adenomas are hormone-secreting, compared with perhaps one-third in the adult population. Multiple series have shown that prolactinomas are the most common adenoma in the pediatric age group overall, accounting for 48%–52% of tumors. However, prolactinomas are not uniformly prominent throughout the pediatric age range, because the relative proportion of prolactinomas increases steadily through the first decade. In fact, ACTH-releasing tumors with Cushing disease and Nelson syndrome are much more common in the first decade than prolactinomas (71% vs 16%). There is subsequent decrease in ACTH-secreting tumors after the age of 11 years, with these tumors representing 18% to 29% of adenomas for the rest of the second decade. Growth hormone-secreting tumors represent about 8% of lesions, with the proportion slightly higher into later pediatric years.

Detection and conspicuity of microadenomas on imaging depends on lesion size, scan technique, and scan timing. Lesions larger than 3 to 4 mm are usually well seen, regardless of technique employed; resolution of smaller lesions may rely on a higher signal technique to generate thinner sections, higher in-plane resolution, and less imaging noise overall. Typical adenoma signal on routine imaging can vary even within adenoma subtypes, but in general adenomas are isointense or slightly hypointense relative to gray matter on T1-weighted imaging, and isointense to slightly hyperintense on T2-weighted imaging. Hemorrhage may confer susceptibility within the lesion, often seen as diminished T2 signal (and possibly increased T1 signal). Lesions larger than 3 to 4 mm are usually easily seen as areas of diminished signal on postgadolinium imaging, with sensitivity diminishing in smaller lesions (Fig. 8). Contrast-enhanced images must be compared with nonenhanced images to confirm parenchymal enhancement, lest an area of high signal be interpreted as enhancement...
when in fact it was hyperintense on noncontrast imaging to start with (Fig. 9). Cystic change may appear hyperintense on T2-weighted imaging, although hemorrhage into a cyst may also be associated with a contradicting reduction in signal.

Because signal in adenomas tends to appear similar to adjacent parenchyma, contrast-enhanced sequences are ordinarily obtained to improve adenoma detection. Although adenomas enhance, they typically do so less strongly than normal pituitary parenchyma, and their peak enhancement lags behind that of the normal gland.

Fig. 8. Pituitary adenoma with incidental residual craniopharyngeal canal. (A) Coronal T1-weighted contrast-enhanced image reveals an area of diminished enhancement in the left side of the gland (asterisk), compatible with a microadenoma. There is only slight rightward deviation of the stalk in this instance. (B) Coronal T2-weighted image fails to show the adenoma, but does show the residual craniopharyngeal canal (arrows).

Fig. 9. Pseudo-mass in the pituitary. (A) Congenitally asymmetric appearance of the bright posterior pituitary (PP) on coronal noncontrast T1-weighted imaging, with perhaps 80% of the posterior lobe sitting to the right of midline. The lower signal in the left side of the gland represents asymmetric positioning of the anterior lobe (arrowheads). The stalk (hatched arrow) still inserts near the midline. (B) After contrast is given the anterior lobe on the left side enhances, but does not become as bright as the posterior lobe on the right, causing a pseudo-mass (ψ).
Ordinarily scanning is begun immediately after contrast administration to capture the maximal difference between gland and adenoma. For each minute that scanning is delayed, one would expect to detect approximately 10% to 15% fewer lesions, so that if scanning is delayed for 7 to 10 minutes after gadolinium injection almost all adenomas would be nearly unresolvable. For this reason the dynamic contrast technique, described earlier, may increase sensitivity further in cases in which a lesion is strongly suspected but not detected on preliminary scanning. In instances where scanning is delayed after contrast administration (e.g., mild contrast reaction, nausea, claustrophobia), the examination may still be salvaged by exploiting the differential washout of contrast from the gland (Fig. 10). Because contrast clears faster from normal parenchyma than it does from adenomas, on delayed scans adenomas will appear hyperintense relative to the remaining gland, instead of hypointense as they do in the early postinjection period.25 The crossover point at which enhancement of adenomas begins to surpass normal parenchyma remains poorly defined, but in many cases it may be less than 6 to 8 minutes after contrast injection.

**CYSTIC LESIONS**

Rathke cleft cysts (RCCs) are common incidental pituitary cysts that are found in up to 20% of routine autopsies.26 These cysts arise from the nonobliterated intrasellar lumen of Rathke pouch,27 and most are so small that they are asymptomatic. When cysts are large (on average 16.3 ± 1.2 mm) they become symptomatic, largely because of their mass effect.28 Such cysts may exert enough mass effect to

![Fig. 10. Gadolinium enhancement patterns. The y-axis indicates signal differential between enhanced and baseline images; the x-axis reflects time. The blood pool enhances earliest and strongest, but it also tapers most rapidly with renal clearance of gadolinium. The normal pituitary gland has a shallower increase, somewhat delayed after the blood pool curve, and shows a gradual decrease. Most adenomas have even slower increase and decrease. The greatest difference between parenchyma and adenomas (and therefore greatest conspicuity of adenomas) is in the early gland enhancement period (time point A), when the gland enhances well but the adenoma has only begun to accumulate contrast. If, for some reason, the gland cannot be imaged at that point, the patient may be rescanned, because the adenoma may retain contrast longer and an adenoma will appear as an area of signal that is higher than the gland (time point B).](image-url)
compromise remaining pituitary tissue, or they may grow superiorly to compress the optic chiasm, resulting in a bitemporal hemianopsia. Larger cysts may also be associated with headaches. On MRI, noncomplicated cysts follow fluid intensity, so they are hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging. RCCs may, over time, develop a higher protein concentration, resulting in an increase in T1 signal and, as protein concentration increases, a reduction in T2 signal (Fig. 11). Sludge or intracystic nodules have also been described as areas of layering or nodular signal in the posterior aspect of the gland.29 RCCs do not enhance centrally and most do not enhance about their periphery unless they have suffered recent hemorrhage or infection; enhancement about a cyst should suggest another diagnosis such as hemorrhagic adenoma or craniopharyngioma.

Fig. 11. Rathke cleft cyst. (A) Coronal T1-weighted noncontrast image shows a rounded focus of high signal (asterisk) situated eccentrically toward the left side of the gland. There is associated bowing of the superior gland margin, presumably related to mass effect from the cyst. (B) Axial T2-weighted image, revealing a rounded cavity within the central sella, in which a debris level is present (between arrows). Such levels are among the most reliable imaging features confirming a cyst. (C) Sagittal T1-weighted noncontrast image reveals the relationship of the cyst (asterisk) with the posterior lobe (arrow). A thin band of tissue (not labeled) sits between the posterior lobe and the cyst, which presumably reflects the posterior wall of Rathke cleft and makes this cyst more likely to represent a Rathke cleft cyst than a pars intermedia cyst. (D) Coronal T1-weighted dynamic sella scan proves the lesion is cystic. The y-axis indicates signal and the x-axis reflects time. Normal pituitary tissue enhances well (curve 2), whereas a cyst will not enhance (curve 1).
Pars intermedia cysts form within the pituitary along the posterior margin of Rathke pouch. These are ordinarily asymptomatic lesions measuring less than 3 mm. Configuration varies, but a flattened cystic cavity broadest in the coronal plane, sitting at the junction of anterior and posterior lobes, suggests this diagnosis (Fig. 12). In many instances pars intermedia cysts may be difficult to distinguish from RCCs, which are more common, so many pars intermedia cysts are considered RCCs on imaging.

Craniopharyngiomas (CPs) are benign tumors that, like RCCs, are also derived from embryologic remnants of Rathke pouch. Of the two varieties, the cystic (or adamantinomatous) tumors (ACPs) are more frequent in the pediatric population. These tumors represent about 10% of all childhood brain tumors and account for approximately 90% of pituitary neoplasms in children.\(^\text{30,31}\) The squamous-papillary variety, which is most common in the sixth and seventh decades, has more solid configuration. ACPs typically contain one or more large cysts of varying wall thickness, with central proteinaceous fluid typically of “crank-case oil”-like consistency. Because of the proteinaceous nature of the cavities, these lesions may be quite hyperintense on T1-weighted imaging and only mildly elevated on T2-weighted imaging. Enhancement is seen about the rim but not centrally (Fig. 13). This peripheral enhancement may be thin or nodular, but with the high T1 signal within the cyst at baseline, any contrast enhancement may be difficult to resolve. ACPs arise within the suprasellar cistern but may grow inferior into the sella; purely intrasellar ACPs are rare. These tumors tend to be large when detected, because they present because of mass effect or local effect of tumor on cranial nerves, and are larger at diagnosis than even symptomatic RCCs (36.2 ± 4.7 mm vs 16.3 ± 1.2 mm).\(^\text{28}\) If distinction of RCC from ACP proves challenging by MRI, a CT scan that demonstrates calcification around the lesion margin markedly increases the probability of craniopharyngioma. Although these tumors

![Fig. 12. Presumed pars intermedia cyst. Sagittal contrast-enhanced T1-weighted image reveals a thin band of nonenhancement (arrowhead) corresponding to the posterior aspect of the anterior lobe. A cyst in this location with thin anterior-posterior diameter relative to height and width is suggestive of a pars intermedia cyst rather than a Rathke cleft cyst. Note the artifact from the patient’s dental braces (arrows); technologists may be able to modify scanning parameters to eliminate or at least shift the artifacts if they project through structures of interest.](image-url)
are histologically benign, their insinuation into leptomeninges, cranial nerves, and vessels makes surgical eradication difficult if not impossible.

The term “empty sella” is largely a misnomer, as the sella is rarely truly empty. Underlying this phenomenon is atypical transmission of CSF pulsation through the hiatus of the diaphragm sella. This pulsatility may, over time, flatten the pituitary along the floor of the sella. In adults this may be related to an abnormally wide diaphragmatic hiatus, but in children it most commonly results from prior surgery, chemotherapy, or posttraumatic affects. On imaging, the sella appears largely fluid in signal, with hypo-intense pattern on T1-weighted imaging and hyperintensity on T2-weighted imaging. The gland is typically flattened along the sellar floor and may be most conspicuous after gadolinium administration. In most cases the stalk can be followed to its insertion on the flattened gland along the sellar floor. Little can be inferred from the resulting gland configuration, because gland function may appear clinically normal in many degrees of gland flattening.

Fig. 13. Adamantinomatous (cystic) craniopharyngioma. (A) Coronal T2-weighted image reveals a large cyst filling the sella and suprasellar cistern (asterisks). Coronal (B) and sagittal (C) postcontrast T1-weighted imaging reveals the high signal within the cyst (asterisks) characteristic of craniopharyngiomas. Enhancement is subtle but can be seen around the superior margin of the cyst (arrowheads). In all images the sella is expanded, and mass effect displaces the chiasm superiorly (hatched arrows).
Arachnoid cysts are fluid accumulations within the leaves of arachnoid. These are usually congenital, but they may arise after trauma, infection, or hemorrhage. In children, symptomatic arachnoid cysts cause CSF flow disturbances in 95% of patients, and endocrine abnormalities in 5%. Although these are slow-growing cysts, over time they can cause significant mass effect and even bony remodeling. As such, pediatric patients with suprasellar arachnoid cysts may exhibit deficiencies of growth hormone and thyrotropin, excess stimulation of the hypothalamic-pituitary axis, and possibly hyperinsulinism-related tall stature or overweight status.

SUPRASELLAR TUMORS

Gliomas may arise anywhere along the optic pathway (including optic nerve, chiasm, or tract), or within the adjacent hypothalamus. Hypothalamic gliomas are of particular concern with regard to endocrine function, because these tumors may cause symptoms related to obesity, diabetes insipidus, or other hypothalamic-pituitary dysfunction. Most of these gliomas are graded as juvenile pilocytic astrocytomas. These are particularly common in patients with neurofibromatosis type 1 (NF-1, von Recklinghausen disease), but NF-1 patients tend to fare better with this tumor than patients with sporadic hypothalamic gliomas. These lesions are seen on imaging as thickening of the third ventricular floor and hypothalamus, with margins that are sometimes obscure. On MRI, these tumors tend to show an isointense or perhaps slightly hypointense T1 signal and slightly hyperintense T2 signal. However, unlike other low-grade gliomas, these lesions tend to enhance well. Larger tumors may grow directly into the pituitary stalk.

Tuber cinereum hamartomas are nonneoplastic heterotopias that are comprised of an atypical proliferation of normal cellular elements. Two different clinical scenarios may be seen: smaller pedunculated tumors (<1 cm) tend to be associated with precocious puberty, whereas larger sessile tumors (>1 cm) tend to be associated with gelastic seizures. Because these lesions are comprised of neuronal elements, they tend to resemble brain parenchyma on all modes of imaging (Fig. 14). Some may show a modestly increased T2 signal. The blood-brain barrier is intact, so they do not enhance appreciably.

Among germ cell tumors, germinomas represent just over half; other less frequent germ cell tumors include teratomas, choriocarcinomas, and embryonal cell carcinomas. More than 90% of germinomas are found in males. They most commonly grow in the pineal region but up to one-third are seen in the suprasellar region, and involvement of both sites simultaneously (bifocal disease) is increasingly recognized. A suprasellar germinoma may present as a consequence of its mass effect, for example, through compression of the optic chiasm (producing a bitemporal hemianopsia), or by disruption of the hypothalamic-infundibular-pituitary axis. These tumors tend to be isointense or slightly hyperintense, with strong enhancement of solid portions after gadolinium administration (Fig. 15). Up to half of germinomas contain areas of cystic degeneration, which do not enhance but may appear considerably hyperintense on T2-weighted imaging. Because of the propensity for germinomas to spread within the subarachnoid space, if an intracranial germinoma is suspected additional imaging of the spine is often performed to exclude drop-metastases.

SYSTEMIC CONDITIONS

Sarcoidosis is a systemic disease in which the primary feature is formation of noncaseating epithelioid granulomas. The lungs and hilar nodes are most frequently
involved, but sarcoid may also affect the heart, skin, liver, and, infrequently, the central nervous system. When affecting the central nervous system (CNS), sarcoidosis is predominantly extraaxial (ie, it affects brain surfaces rather than brain parenchyma directly). Among extraaxial sites, the sellar and suprasellar regions are preferred, with coarse nodular aggregates typically along the stalk and into the sella that enhance well after contrast. Disease can also involve structures within the subarachnoid spaces, most notably the cranial nerves, in which case they may produce cranial neuropathies. On occasion enhancement from sarcoid may be visualized within parenchyma, deep to the brain surface; in these situations disease is felt to track along perforators from the surface into the parenchyma, mimicking a parenchymal process.

Langerhans cell histiocytosis (LCH) is characterized by pathologic accumulation of Langerhans cells. Typically CNS involvement is seen in patients with systemic disease.
It is believed that infiltration into the hypothalamic-pituitary axis causes CNS-associated symptoms; diabetes insipidus is seen in at least 24% of patients with LCH, although some investigators report an even higher incidence. Anterior pituitary dysfunction is also reported but is seen less commonly; for example, growth hormone deficiency may be seen in between 10 and 20% of patients with LCH, but these patients usually also have diabetes insipidus. Inflammatory thickening, which is best seen on postcontrast T1 images, may occur anywhere along the infundibulum and stalk. The appearance of this infiltration may mimic other inflammatory disorders such as sarcoid or even a lymphocytic hypophysitis.

McCune-Albright syndrome is a rare sporadic genetic disorder characterized by the classic triad: polyostotic fibrous dysplasia, endocrine hyperfunction, and café-au-lait skin lesions. The best known hormonal association, precocious puberty, is seen more frequently in girls. Additional endocrine manifestations include hypercortisolism, hyperthyroidism, and acromegaly. Occasionally a growth hormone–secreting microadenoma is seen on sellar MRI examination. However, on imaging the most prominent findings relate to fibrous dysplasia. Osseous findings relate to proliferation of fibrous tissue in bone marrow by abnormal preosteoblastic cells. Bone lesions typically extend from medulla to cortex, with either a radiolucent or homogeneous “ground-glass” density. Long bones, ribs, and craniofacial structures are most commonly involved. Radiography exposes patients to a relatively low radiation dose, but CT may be necessary to identify subtle areas of involvement (Fig. 16). MRI is a less favorable modality to use for assessment of fibrous dysplasia, although affected areas may occasionally be detected as relatively homogeneous areas of low signal.

ASSOCIATED MIDLINE LESIONS

Callosal abnormalities range from complete aplasia of the gland (callosal agenesis) to callosal hypoplasia, in which the entire callosum forms but is thin or attenuated.
Callosal agenesis. Complete agenesis is characterized by (1) absent cingulate sulcus; (2) colpocephaly, in which there is a parallel arrangement of the lateral ventricles that have teardrop-shaped posterior horns; (3) superior protrusion of the third ventricle, because its upper margin is not as confined as in normal patients; and (4) abnormal longitudinal fascicles that were meant to cross the midline but instead fold back on themselves, representing disordered bundles of Probst.44,45 Midline sagittal imaging reveals radiating bands of gyri and sulci from the ventricular margin, in lieu of the cingulum that is normally present.

Callosal hypogenesis. The callosum forms in a predictable pattern: genu first, followed by body and splenium, and finally the most anteroinferior rostrum. Segments that fail to form appear the latest embryologically, so hypogenesis almost always involves rostrum, and then may involve splenium, then body, then genu. Thus, in hypogenesis the segments of callosum that do form include G-B-S (genu-body-splenium), G-B, or G (Fig. 17). Furthermore, a callosum that possesses an intact rostrum but lacks another segment has likely undergone some destruction of a portion of previously formed segment of the callosum.

Callosal hypoplasia. A hypoplastic callosum forms all of its segments, but is somewhat attenuated. As the callosum experiences considerable growth, with thickening over time, assessment of hypoplasia in the first year may prove challenging.

Chiari hindbrain malformations are comprised of a group of 4 developmental cerebellar anomalies. Type I, by far the most common, has been associated with multiple endocrine deficiencies.46 It is characterized by peglike protrusion of cerebellar tonsils through the foramen magnum. Although numeric thresholds have been used traditionally
to confirm this tonsillar ectopia (extension at least 6 mm below the foramen magnum (basion-opisthion line) in the first decade, at least 5 mm in the second and third decades, and so forth) it is becoming clear that what underlies clinically relevant Chiari malformations is actually the alteration in the normal CSF dynamics that result from such a configuration. Thus investigators have used phase-contrast imaging to show where the CSF flow abnormality exists in symptomatic patients, and may ultimately explain why two different patients with the same measured ectopia may exhibit very different symptoms. Additional imaging features may include syringohydromyelia, the abnormal dilatation of the central canal of the cord related to disordered CSF flow patterns. Symptoms from a Chiari I malformation are nonspecific but include suboccipital headaches (28%–63% of patients), cranial nerve palsies or brain stem symptoms (21%), ataxia (6%–25%), and long tract signs.

Septo-optic dysplasia (SOD, de Morsier Syndrome) comprises a spectrum of disorders that affect midline structures. Although mutations in the HESX1 gene have been implicated, HESX1 causation may be more common in sporadic disease than in familial cases. The most common abnormality is optic nerve hypoplasia (seen in 75%–80% of SOD patients); this is usually bilateral (88%). Microphthalmia or anophthalmia may be seen but is rare. Pituitary symptoms range from isolated growth hormone deficiency to panhypopituitarism; gonadotropin secretion may be preserved in the face of near complete hypopituitarism. Associated brain anomalies include midline forebrain abnormalities such as callosal agenesis, absence of the septum pellucidum and fornix aplasia. Additional features may include schizencephaly and cerebellar hypoplasia. Holoprosencephaly, specifically the lobar variety, may also be seen with SOD.

**SUMMARY**

Evaluation of the sella and surrounding structures in pediatric endocrinopathies is best performed with high-resolution MRI scanning. Adequate assessment relies not only on
determining the size and shape of the gland but also on confirming normal signal characteristics and homogeneous parenchymal enhancement. Surrounding structures, including the hypothalamus-infundibulum-stalk and the skull base and midline structures about the cerebral hemispheres, warrant careful attention to identify any associated abnormalities. Tumors, whether they arise in the gland or affect gland function through mass effect, are usually well resolved on today’s scanners and imaging provides accurate characterization of these lesions.

REFERENCES


