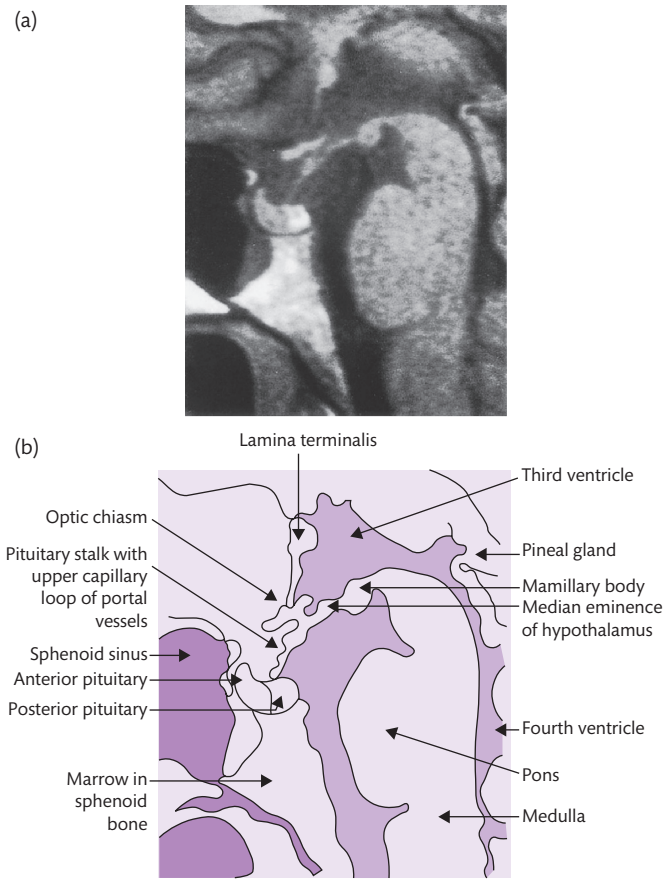

Fig. 13.2.1.1 Sagittal MRI scan of normal pituitary gland and an anatomical line drawing.

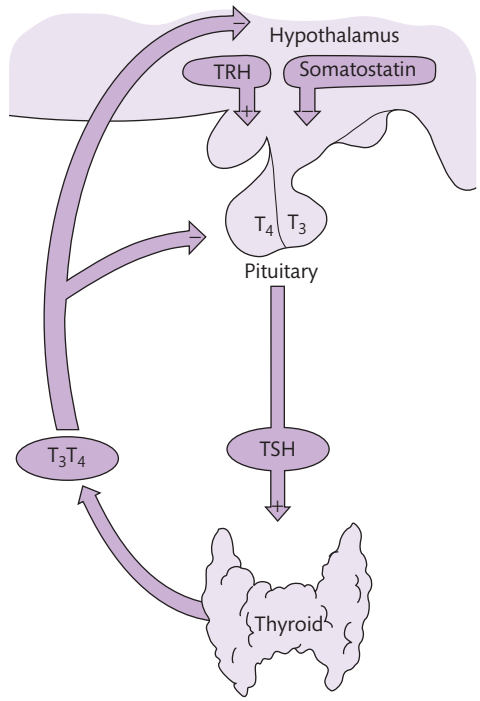

Fig. 13.2.1.2 Diagram of the hypothalamo-pituitary–thyroid axis showing negative feedback loops. TRH, thyrotropin-releasing hormone; TSH, thyroid-stimulating hormone.

the lateral aspects of the cavernous sinuses and constitutes the sellar diaphragm. The cavernous sinuses are on either side of the sella, lateral and superior to the sphenoid sinuses, and contain important neurovascular structures including the cavernous segments of the internal carotid arteries and the cranial nerves III, IV, V, and VI. The optic chiasm is located superiorly and is separated from the pituitary by the suprasellar cistern and the sellar diaphragm (Figs. 13.2.1.1 and 13.2.1.2).

The anterior lobe comprises nearly 80% of the gland and includes the pars distalis, pars intermedia, and pars tuberalis. Staining characteristics divide the pars distalis into a central 'mucoid wedge' and two 'lateral wings'. On light microscopy the cells of the anterior lobe show variation in size, shape, and histochemical staining features. They are organized in nests and cords, and are separated by a complex capillary network. The pars intermedia is poorly developed in humans and lies between the pars distalis and the posterior pituitary. Large numbers of cells in the central zone are basophilic and produce adrenocorticotropic hormone (ACTH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), and thyrotropic hormone (TSH). Most of the cells in the lateral wings are acidophilic and produce growth hormone (GH) and, less frequently, prolactin (PRL) (Table 13.2.1.1).

The pars tuberalis is an extension of the anterior lobe along the pituitary stalk. It is formed by normal acini of pituitary cells distributed around surface portal vessels. The anterior lobe also includes follicular cells, derived from secretory cells and constituting follicles within the gland, and folliculostellate cells (less than 5% of the adenohypophyseal cells), which have a physiological role that is not clear.

The anterior pituitary receives most of its blood supply from the hypothalamo-hypophyseal portal system (primary plexus, long portal venous system, and secondary plexus), which originates from the capillary plexus of the median eminence and superior stalk derived from the terminal ramifications of the superior and inferior hypophyseal arteries. This system carries blood and hypophysiotropic hormones down to the stalk. The remainder of

Table 13.2.1.1 Hormone-producing cells in anterior pituitary gland

Type of cell	Approximate percentage of adenohypophyseal cells	Distribution
Somatotrophs or GH cells	50%	Greatest density in lateral wings
Lactotrophs or PRL cells	20%	Mainly in posterior portions of lateral wings
Corticotrophs or ACTH cells	15–20%	Mainly middle and posterior portions of mucoid edge
Gonadotrophs or FSH and LH cells (produce FSH and LH in isolation or by the same cell)	10%	Evenly distributed throughout anterior lobe
Thyrotropes or TSH cells	5%	Mainly in anterior part of mucoid edge

ACTH, adrenocorticotropic hormone; GH, growth hormone; FSH, follicle-stimulating hormone; LH, luteinizing hormone; PRL, prolactin; TSH, thyrotropic hormone.

the blood supply is through the pituitary capsular vessels originating from the superior hypophyseal arteries. The venous drainage from the anterior pituitary is through the cavernous sinuses into the petrosal sinuses and the internal jugular veins.

The anterior lobe has no direct innervation, apart from a few sympathetic nerve fibres spreading to the anterior lobe along blood vessels. The hypothalamic regulation is exerted via the neurohormonal link with the hypothalamic regulatory peptides reaching the pituitary via the portal vessels.

General physiology

The secretion of the anterior pituitary hormones is under elegant regulation exerted by hypothalamic peptides and, with the exception of prolactin, by the negative feedback (at both the hypothalamic and pituitary level) of hormones from the target glands (Fig. 13.2.1.3). The hypothalamic peptides are secreted in the median eminence and are transferred to the anterior pituitary gland via the hypothalamic–pituitary portal system. They integrate environmental and neural information and bind to specific high affinity cell membrane receptors of the particular pituitary cell type. Failure of the target gland results in decreased negative feedback and increased hypothalamic and pituitary secretion. Primary overactivity of the target gland results in increased negative feedback and decreased hypothalamic and pituitary secretion. Additional 'short-loop' feedback, in which pituitary hormones affect the secretory activity of the hypothalamus, is also implicated in the network contributing to the meaningful function of the pituitary gland. Finally, the anterior pituitary synthesizes several peptides, growth factors, and cytokines that play an important part in autocrine and/or paracrine control of pituitary secretion and/or cell proliferation.

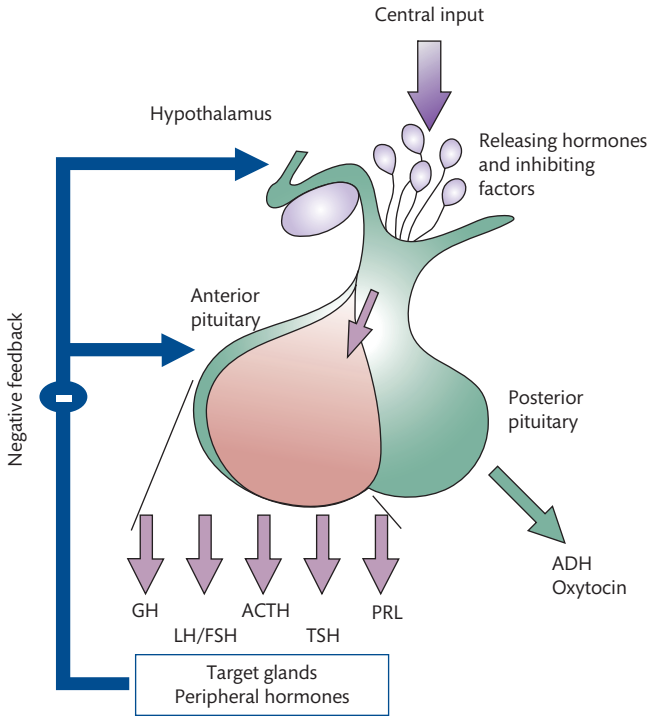

Fig. 13.2.1.3 Regulation of the hypothalamic–pituitary–peripheral function. The anterior pituitary produces GH, LH/FSH, ACTH, TSH, and PRL. The secretion of these hormones is regulated by hypothalamic-releasing and hypothalamic-inhibiting factors and by negative feedback inhibition of their peripheral hormones.

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Clinical features of pituitary disease

The clinical features of pituitary disease, mostly associated with a space-occupying lesion, may result from local mass effects and/or pituitary hormone deficits or hypersecretion.

The local mass effects depend on the size of the tumour and its anatomical position. Headache is usually the consequence of dural stretching. It can be variable (occipital, retro-orbital, bitemporal) and is often nonspecific. The neuro-ophthalmological effects include visual field defects (usually bitemporal hemianopia or upper temporal quadrantanopia or any unilateral or bilateral visual field defect) from compression of the optic chiasm (Fig. 13.2.1.4) and squint, ptosis, or papillary dilatation from ocular nerve palsies caused by lateral tumour extension. Compression of the first or second branch of the trigeminal nerve may rarely result in facial pain. Very large pituitary tumours obstructing the fourth ventricle or the foramen of Monro cause hydrocephalus and expansion of the lateral ventricles. Inferior invasion and erosion of the sellar floor may result in recurrent sinusitis, cerebrovascular fluid rhinorrhoea, and recurrent meningitis. Extension into the temporal lobe may rarely be associated with temporal lobe epilepsy and to the cerebral peduncles with motor and/or sensory disturbances. Superior expansion to the hypothalamus may be associated with hypothalamic dysfunction

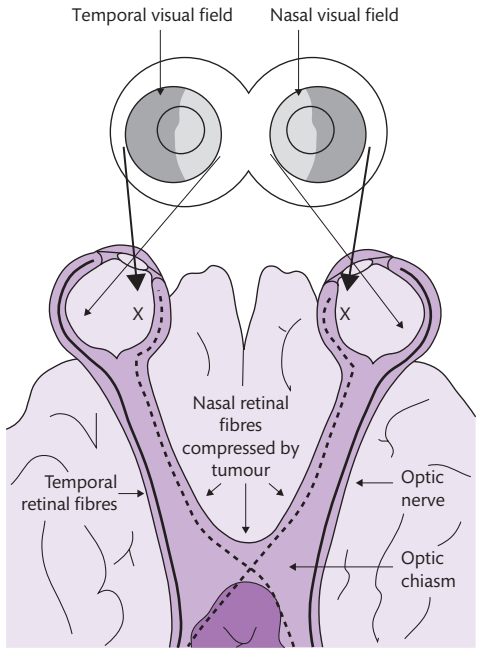

Fig. 13.2.1.4 Neuro-ophthalmological pathways and the classical bitemporal hemianopia that results from compression of the central optic chiasm by a pituitary tumour. However, any degree of unilateral or bilateral visual deficit can occur depending on the anatomical site of the lesion.