Corticotropin

AKA: Adrenocorticotropic hormone (ACTH)

A 39 amino acid trophic hormone synthesized from POMC. The first 13 amino acids at the amino-terminal of ACTH are identical to α-MSH. The active half-life is ≈ 10 minutes. Produces a diurnal peak in cortisol (the highest peak occurs in the early morning, with a second, lesser peak in the late afternoon) and also increases in response to stress.

The release of cortisol by the adrenal glands is stimulated by the adrenocorticotrophic hormone (ACTH) from the pituitary which in turn is stimulated by corticotropin-releasing hormone (CRH) from the hypothalamus.

Adrenocorticotropic hormone stimulates the adrenal glands to secrete steroid hormones, principally cortisol.

It is a polypeptide tropic hormone produced and secreted by the anterior pituitary gland. It is an important component of the hypothalamic pituitary adrenal axis and is often produced in response to biological stress (along with its precursor corticotropin-releasing hormone from the hypothalamus). Its principal effects are increased production and release of corticosteroids.

In secondary adrenal insufficiency caused by deficient corticotropin (ACTH) release by the pituitary, mineralocorticoid secretion is usually normal and only glucocorticoids need to be replaced.

Cushing’s disease

Nelson syndrome

Primary adrenal insufficiency, also called Addison's disease, occurs when adrenal gland production of cortisol is chronically deficient, resulting in chronically elevated ACTH levels.

When a pituitary tumor is the cause of elevated ACTH (from the anterior pituitary) this is known as Cushing's Disease and the constellation of signs and symptoms of the excess cortisol (hypercortisolism) is known as Cushing's syndrome. A deficiency of ACTH is a cause of secondary adrenal insufficiency. ACTH is also related to the circadian rhythm in many organisms.

Pituitary corticotroph adenomas secrete inappropriate amounts of ACTH, which results in disorderly and excessive production of cortisol by the adrenal gland [1)].

Abellán-Galiana et al. propose an ACTH value <15 pg/mL as a good long-term prognostic marker in

the postoperative period of Cushing's Disease. Reaching the ACTH nadir in less time is associated to a lesser recurrence rate [2].

Adrenocorticotropic hormone deficiency

Adrenocorticotropic hormone deficiency.

Assessment of postoperative ACTH (corticotropin) reserve

Simple assessment protocol for patients who go home on hydrocortisone and were not on it pre-op.

● taper hydrocortisone over 2–3 weeks down to 20 mg PO q AM and 10 mg q 4 PM (a little higher than maintenance to provide for some stress coverage) for several days

● then hold the PM dose and check an 8 AM serum cortisol the next day

● to avoid adrenal insufficiency in patients with incompetent reserve: as soon as the blood is drawn have the patient take their morning cortisol dose and resume regular dosing until the test results are available

● if this 8 AM cortisol shows any significant adrenal function, then taper the patient off hydrocortisone.

Posttraumatic hypopituitarism

see Posttraumatic hypopituitarism

1)
Aron DC, Findling JW, Tyrrell JB: Cushing's disease. Endocrinol Metab Clin North Am 16:705–730, 1987
2)
Abellán-Galiana P, Fajardo C, Riesgo-Suárez P, Pérez-Bermejo M, Ríos-Pérez C, Gómez-Vela J. Prognostic usefulness of ACTH in the postoperative period of Cushing's disease. Endocr Connect. 2019 Aug 1. pii: EC-19-0297.R1. doi: 10.1530/EC-19-0297. [Epub ahead of print] PubMed PMID: 31394502.