

UNIVERSITY OF  
**CALGARY**  
FACULTY OF NURSING

# **UNIT 14**

## **(OPTION)**

### **Alterations in Endocrine Function**

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# UNIT 16

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## Alterations in Endocrine Function

The regulation of homeostasis by the endocrine system is both complex and subtle. During your nursing career you will encounter patients with mild to significant alterations in endocrine regulation. Your role as patient educator is of particular importance in the successful long-term management of these disorders. It is hoped that by increasing your own understanding of endocrine pathophysiology, your skills in assessment and patient education will be improved.

This module will contain two basic components: the mechanisms of hormonal regulation and alterations of hormonal regulation. Each component is further subdivided into hypothalamic-pituitary, thyroid and adrenal sections.

## Overview

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### Aim

The general aim of this module is to facilitate your understanding of the mechanisms underlying hormone regulation and hormonal alterations. Upon completion of this unit, you will have a thorough understanding of the regulation of hormone release and transport. You will be introduced to the cellular mechanisms of hormone release and you will review the structure and function of the endocrine glands. You will have a good understanding of the pathophysiology, clinical manifestations and evaluation and treatment for most of the endocrine diseases you will encounter during your nursing career.

### Objectives

On completion of this unit, you will be able to:

1. Name the five general functions of the endocrine system.
2. Be able to identify the following hormones as proteins, polypeptides, amines or lipids: growth hormone, prolactin, adrenocorticotrophic hormone, antidiuretic hormone, oxytocin, epinephrine, thyroid hormone, glucocorticoids, aldosterone, estrogen and testosterone.
3. Name the 3 basic patterns of hormone release.
4. Define signal transduction, first and second messengers and identify at least two second messengers.
5. Describe the structural relationship between the hypothalamus and the anterior pituitary and posterior pituitary.
6. Name the two posterior pituitary hormones and describe their actions.
7. Describe the synthesis, regulation of release and actions of thyroid hormone, cortisol, aldosterone, adrenal estrogens/androgens and epinephrine.
8. Describe the various mechanisms of hormone alteration.
9. Describe the pathophysiology, clinical manifestations, evaluation and treatment of the following disorders:
  - a. Syndrome of inappropriate antidiuretic hormone (SIADH)
  - b. Diabetes insipidus
  - c. Hypopituitary disorder
  - d. Hyperpituitarism (primary adenoma and acromegaly)
  - e. Thyrotoxicosis
  - f. Hypothyroidism (primary, congenital)
  - g. Disorders of the Adrenal Cortex (Cushing's Disease, hypersecretion of adrenal androgens/estrogens and hypocortical functioning)
  - h. Disorders of the adrenal medulla

## Resources



### *Required*

McCance, K., & Huether, S. (2001). *Pathophysiology: The biologic basis for disease in adults and children* (4<sup>th</sup> ed.). St. Louis: Mosby. Pages: 597-610; 614-618; 24-638, (including Table 20-2) and 655-666.

Print Companion: Alterations in Endocrine Function

### *Learning Activities*

Student Activities One to Eight

### *Supplemental Materials*

Student Activity Nine

## Web Links

All web links in this unit can be accessed through the Web CT system.

## Section 1: Mechanisms of Hormone Regulation



**Read:** McCance, K., & Huether, S. (2001). Pages: 597-598.

Hormones may be classified by structure. Review Table 19-1 on page 626 of McCance and Huether. Recall that thyroid hormone, epinephrine and norepinephrine (amines) are derived from a single amino acid – tyrosine.

For an overview of the interactions between hormones and their receptors, follow this link:

Colorado State University

<http://arbl.cvmb.colostate.edu/hbooks/pathphys/endocrine/moaction/>



### Learning Activity #1

Match each of the hormones with its structural category.

- |                |  |
|----------------|--|
| a. protein     | _____ 1. Mineralcorticoids – Aldosterone |
| b. polypeptide | _____ 2. Growth Hormone                  |
| c. amine       | _____ 3. Thyroid Hormone                 |
| d. lipid       | _____ 4. Glucocorticoids – Cortisol      |
|                | _____ 5. Estrogens                       |
|                | _____ 6. Antidiuretic Hormone            |
|                | _____ 7. Oxytocin                        |



**Note:** 'Polypeptide' as used in the McCance and Huether text refers to a short amino acid chain. Other texts may simply refer to these hormones as peptides.

### Regulation of Hormone Action

Hormone release is controlled by other hormones (hormones that regulate the release of other hormones are known as **tropic hormones**), chemical factors and neural control. Negative feedback is commonly found in the human endocrine system.

Recall that negative feedback loops consist of several components including a receptor, an integrating center with a set point and an effector(s). Information regarding the levels of a variable are sent to the control center and compared to the set point value. If the level of the variable is too high or too low then the effectors are implemented to change the variable level **in the direction opposite to the initial change**. This is **negative** feedback. An example of a negative feedback loop is the reduction of corticotropin releasing hormone (CRH) secretion by the hypothalamus, and of



adrenocorticotrophic hormone (ACTH) secretion from the anterior pituitary by cortisol, an adrenal cortical hormone. Positive feedback mechanisms are rare in humans.

Steroid hormones are generally not stored. They are synthesized and released. The control of steroid release is accomplished by controlling their synthesis.



## Learning Activity #2

1. Hormones can affect any cell they contact.
  - a. True
  - b. False
2. Hormones may be released in response to:
  - a. neural mechanisms
  - b. other hormones
  - c. chemical factors
  - d. a, b and c
3. Hormone release can be decreased in a negative feedback loop by
  - a. the hormone released
  - b. the trophic hormone
  - c. the physiological effect
  - d. a, b and c
4. Water soluble hormones cross cell membranes and bind to their receptors in:
  - a. the nucleus
  - b. the cytosol
  - c. on the cell membrane
  - d. a and b

## Hormone Transport



**Read:** McCance, K., & Huether, S. (2001). p. 599.

Water soluble hormones generally circulate in their free form. Lipid soluble hormones are transported by plasma proteins. Recall that only the free hormone, and not the bound hormone, is available for transport.



## Cellular Mechanisms of Hormone Release

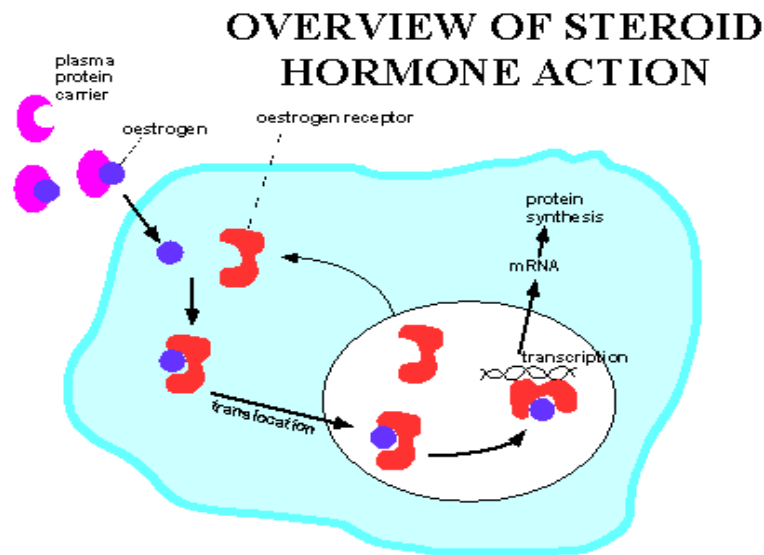
**Read:** McCance, K., & Huether, S. (2001). pp. 599-602.

Hormones and neurotransmitters (ligands) are known as first messengers, chemicals which carry a chemical 'message' from one cell to the receptors on or in another cell. **Second messengers** are intracellular molecules generated by the binding of a hormone or neurotransmitter to a plasma membrane

receptor. These molecules are the first link between the ligand and the biochemical cascade which they activate. Common second messengers include: cyclic adenosine monophosphate (cAMP) cyclic guanosine monophosphate (cGMP), calcium ions, inositol trisphosphate and diacylglycerol. Signal transduction is the series of steps from the binding of the ligand to its receptor to the production of the physiological response.

Steroid hormones do not bind with plasma membrane receptors or initiate a biochemical cascade. They bind to INTRAcellular or INTRANuclear receptors, as they can easily diffuse across cell membranes. After binding to their receptors, the resulting hormone-receptor complex moves into the nucleus, binds directly with the DNA and alters the transcription of RNA (gene expression).

The following is a schema of the mechanism of action of any steroid hormone, in this case estrogen:



You will not be required to demonstrate knowledge beyond the stated objective for this section. (Define signal transduction, first and second messengers and identify at least two second messengers.) Familiarity with the basic concepts of signal transduction is an asset as this a primary field of research. Many types of hormonal dysfunction operate at this level in the category generally known as 'postreceptor deficits'. These biochemical cascades are also a major site targeted by pharmaceutical intervention.

## Structure and Function of Endocrine Glands



### *Hypothalamic-Pituitary System*

**Read:** McCance, K., & Huether, S. (2001). pp. 603-608

The relationship between the hypothalamus and the pituitary is complex. The hypothalamus regulates the release of pituitary hormones, but the mechanisms of this regulation vary with the anterior versus posterior pituitary. The anterior and posterior pituitary arise from different embryonic origins and may be viewed as two separate glands fused together. The hypothalamus and anterior pituitary are joined by a portal system. The hypothalamus releases hormones into this system, which then regulates the release of anterior pituitary hormones. Some stimulate release whereas others inhibit this release. The relationship between the hypothalamus and posterior pituitary is very different. The cell bodies of neurons in the hypothalamus synthesize oxytocin and ADH. These hormones are transported to the posterior pituitary down the axons of these neurons in a tract called the hypothalamohypophyseal tract (recall that the pituitary is also known as the hypophysis). Action potentials travelling along these tracts from the hypothalamus then stimulate the release of oxytocin or ADH.



**Note (a contradiction):** The McCance text states that some hypothalamic releasing hormones are known as factors such as growth hormone releasing factor. This is no longer the case and hypothalamic releasing hormones are known as hormones not factors (e.g. growth hormone releasing hormone).



**Note:** A good schematic overview of the HPA axis can be viewed at:

Southwest Fisheries Science Center

[http://swfsc.nmfs.noaa.gov/mmd/congress/Curry%20Lit%20Review/  
Figs.html](http://swfsc.nmfs.noaa.gov/mmd/congress/Curry%20Lit%20Review/Figs.html)

## Hypothalamic Trophic Hormones

Hypothalamic trophic hormones are secreted into the portal system, and either increase or decrease the release of anterior pituitary hormones.

<b>Hypothalamic Hormone</b>	<b>Anterior Pituitary</b>
Growth Hormone Releasing Hormone (GHRH)	Growth Hormone (GH)
Growth Hormone Inhibiting Hormone (GHIH)	
Thyrotropin Releasing Hormone (TRH)	Thyroid Stimulating Hormone (TSH)
Corticotropin Releasing Hormone (CRH)	Adrenocorticotrophic Hormone (ACTH)
Prolactin Releasing Hormone (PRH)	Prolactin
Prolactin Inhibiting Hormone (PIH) (note this is actually dopamine)	
Gonadotropin Releasing Hormone (GnRH)	Follicle Stimulating Hormone (FSH) and Lutenizing Hormone (LH)

## Anterior Pituitary Trophic Hormones

The tropic hormones from the anterior pituitary include: FSH, LH, ACTH and TSH.

## Other Anterior Pituitary Hormones

### *Growth Hormone*

Growth Hormone stimulates cells to increase in size and to divide. Its major targets are bone and muscle cells. The effects of GH are mediated by insulin like growth factors or IGFs, also known as somatomedins. The metabolic effects of GH are to: increase blood sugar, lower blood amino acid levels (due to increased protein synthesis) and increased blood fatty acid levels.

### *Prolactin*

Prolactin promotes milk production by the breasts. Secretion depends on the dominance of PRH versus PIH. PIH is dominant in males. In females, estrogen stimulates PRH release. This may account for premenstrual breast swelling and tenderness. At the end of pregnancy, PRH levels are high. Suckling stimulates PRH release and inhibits PIH release.

Marieb, E.N. (1998). *Human Anatomy and Physiology* (4<sup>th</sup> ed.). Menlo Park, California: Benjamin/Cummings. p. 595-601.

## Posterior Pituitary Hormones

### *Oxytocin*

Oxytocin primarily affects uterine smooth muscle. It initiates contractions in a quiescent uterus, and increases the strength and frequency of contractions in an active uterus. It is also involved in milk **ejection**. It can also be used to stimulate labor or to reduce postpartum bleeding. The stimulus for the release of oxytocin is the distension of the uterus by the fetal head. This is a rare example of a positive feedback loop in humans. The more the distension, the more the release, the stronger the contractions and more the distension.

For an overview of oxytocin, follow this link:

Colorado State University

<http://arbl.cvmbs.colostate.edu/hbooks/pathphys/endocrine/hypopit/oxytocin.html>

The three-dimensional structure and folding of oxytocin can be seen at:

University of Wisconsin—Eau Claire

<http://cgu.cs.uwec.edu/Oxytocin/anim.gif>



**Future Possibilities:** It is known that oxytocin stimulates maternal behavior and child-mother bonding in most mammalian species. Now, recent scientific research suggests that oxytocin may be responsible for partner pair-bonding or “love” in both males and females! You can view some of this controversial research at:

Student Advantage.com

<http://www.studentadvantage.com/lycos/article/1,1534,c3-i63-t287-a23831,00.html>

Oxytocin.org

<http://www.oxytocin.org/oxytoc/>

### *ADH*

The major function of ADH or vasopressin is to control plasma osmolality. It acts to increase the permeability of the distal renal tubules and collecting ducts. This results in increased water reabsorption and the production of more concentrated urine. At pharmacological doses, it will increase blood pressure (hence vasopressin). It may be given in cases of severe hemorrhage.

## Learning Activity #3



1. T F The hypothalamus is connected to the anterior pituitary via the hypothalmohypophysial tract.
2. T F Hormones are synthesized and stored in the anterior pituitary, but only stored (not synthesized) in the posterior pituitary.
3. T F Emotional stress can influence the release of hypothalamic hormones.
4. T F Vasopressin is another name for aldosterone.
5. T F ADH acts by decreasing the permeability of the distal renal tubules and collecting ducts.
6. T F Oxytocin stimulates milk production.
7. T F Growth hormone increases blood amino acid levels.
8. T F Growth hormone decreases blood glucose levels.

## Thyroid Gland



**Read:** McCance and Huether. Pages 608-610.

The thyroid gland secretes two related hormones: triiodothyronine ( $T_3$ ) and tetraiodothyronine ( $T_4$ ).  $T_4$  is converted to  $T_3$  in the periphery. TH has a variety of physiological effects. It increases the basal metabolic rate as measured by increased oxygen consumption and increased heat production. It increases the number of beta adrenergic receptors in the heart, skeletal muscle, adipose tissue and in lymphocytes. It also increases gut motility, raises blood glucose and lowers blood cholesterol levels. The thyroid gland is the only organ which stores iodine in our body, which is used in the synthesis of  $T_3$  and  $T_4$  (Greenspan & Strewler, 1997).



**History:** The incredible ability of the thyroid gland to uptake iodine resulted in disaster for the children of Chernobyl. The explosion released radioisotopes including radioactive iodine. The children took up this iodine and now significantly higher rates of thyroid cancer can be seen in these children. For more information please go to:

<http://www.ratical.org/radiation/inetSeries/ChernyThyrd.html>

## Learning Activity #4

1. The hypothalamus releases Thyroid Stimulating Hormone (TSH)
  - a. true
  - b. false
2. TSH does all of the following except:
  - a. stimulates the release of thyroid hormone
  - b. increases potassium uptake
  - c. increases the synthesis of thyroid hormone
  - d. increases prostaglandin synthesis
3. The thyroid gland normally produces thyroid hormone in the following ratio: 10% T<sub>4</sub> and 90% T<sub>3</sub>.
  - a. true
  - b. false



## Adrenal Glands

**Read:** McCance and Huether. Pages 614-619 (To Tests of Endocrine Function).

**Note:** The functional anatomy of the adrenal gland, and the difference between the cortex and medulla, can be reviewed at:

The University of Colorado

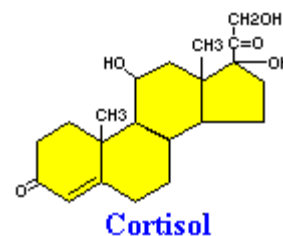
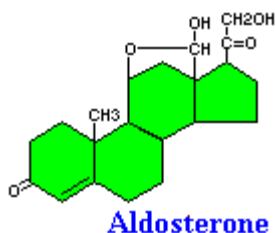
<http://arbl.cvmbs.colostate.edu/hbooks/pathphys/endocrine/adrenal/anatomy.html>



### *Hormones of the Adrenal Cortex*

#### **Mineralcorticoids**

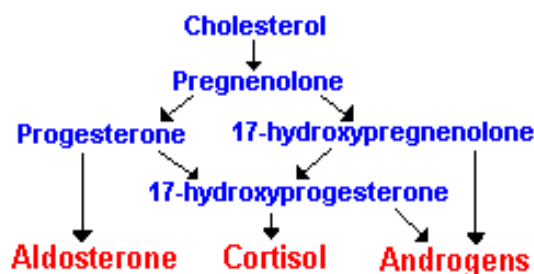
The adrenal cortex synthesizes several mineralcorticoids, but aldosterone is the most common and the most potent. The main action of aldosterone is to stimulate the reabsorption of sodium from the kidneys, perspiration, saliva and gastric juices. The net effect is to increase plasma sodium levels (plasma potassium and hydrogen ion levels decrease). Water is also retained as the sodium is reabsorbed. The major regulator of aldosterone release is the renin-angiotensin mechanism. Decreased blood pressure or plasma osmolality stimulate the release of renin. Renin converts angiotensinogen to angiotensin I. Angiotensin I is converted to the active form, angiotensin II, by angiotensin-converting enzyme (ACE). Note ACE inhibitors will decrease angiotensin and therefore aldosterone release, resulting in reduced blood volume and pressure.



### Glucocorticoids

Glucocorticoids include cortisol, cortisone and corticosterone, but only cortisol is secreted in significant amounts. Cortisol is our chronic stress hormone (epinephrine and glucagon are our acute stress hormones). Stress stimulates the secretion of cortisol. Note that this stress need not involve emotional arousal; a bacterial infection may serve as a physiological stressor. Cortisol enhances the vasoconstrictive effects of epinephrine. It also raises blood glucose, amino acids and triglyceride levels. Over the long term, the action of cortisol in producing high blood triglycerides and cholesterol levels may contribute to premature atherosclerosis (Brindley, 1996)

Both cortisol and aldosterone are steroid hormones that are synthesized from cholesterol:



### Adrenal Estrogens and Androgens

Adrenal estrogens are secreted in such small quantities that they are not considered physiologically significant. The major adrenal androgen is dehydroepiandrosterone or DHEA. In adult males, the effects of adrenal androgens are masked by testis hormone production. In adult females, the physiological function of the adrenal androgens may be important for the maintenance of libido. Adrenal androgens also stimulate the growth of axillary and pubic hair in boys and girls, and contributes to their growth spurt (Tortora & Grabowski, 2000).

### Hormones of the Adrenal Medulla

Chromaffin cells are actually specialized sympathetic postganglionic neurons that secrete the catecholamine hormones epinephrine and norepinephrine. The major hormone is epinephrine. ACTH and cortisol increase the secretion of epinephrine. Stresses such as hypoxia, hypercapnia,



hypoglycemia and hemorrhage will also increase its release. The hormone is involved in the 'flight or fight' response.



## Learning Activity #5

1. Glucocorticoids do all of the following except:
  - a. increase T lymphocyte proliferation
  - b. suppress interleukin action
  - c. promote fat deposits in the cervical area
  - d. increase gluconeogenesis
2. Factors involved in the secretion of adrenocorticotrophic hormone include:
  - a. diurnal rhythms
  - b. stress
  - c. plasma osmolarity
  - d. circulating cortisol levels
3. The main function of aldosterone is to conserve calcium.
  - a. True
  - b. False
4. Aldosterone secretion is stimulated by angiotensin II.
  - a. True
  - b. False
5. Renin converts angiotensin II to angiotensin I.
  - a. True
  - b. False
6. Weak androgens secreted by the adrenal cortex are converted to stronger androgens in the periphery.
  - a. True
  - b. False
7. The major catecholamine secreted by the adrenal medulla is epinephrine.
  - a. True
  - b. False
8. Bacterial invasion can activate the stress response without emotional arousal.
  - a. True
  - b. False

## Section 2: Alterations of Hormonal Regulation

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**Note:** the Alberta Medical Association's (AMA) Clinical Practice Guidelines for the Laboratory Testing and Diagnosis of endocrine diseases, including all of the pathologies described in this unit, can be viewed at this link:

The Alberta Medical Association

<http://www.amda.ab.ca/cpg/index.html>

### Mechanism of Hormonal Alterations



**Read:** McCance and Huether. Pages 624-625.

The pathways for hormone delivery to the cells can be seen in Figure 20-1. Due to the complexity of these pathways, pathology may result from dysfunction in any one (or more) of these numerous phases. This complexity contributes to the slow nature of research in this research.

### Alterations of the Hypothalamic-Pituitary System



**Read:** McCance and Huether. Pages 625-633.

**Note:** this is a good site to refer your patients with pituitary dysfunction to:

Pituitary Network Association

<http://www.pituitary.com/>

#### *Diseases of the Posterior Pituitary*

##### **Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)**

**SIADH** is characterized by high levels of ADH in the absence of normal physiologic stimuli. The most common cause is ectopic production. Clinical manifestations include: serum hypoosmolality and hyponatremia, and urine hyperosmolality. Symptoms are due to the hyponatremia and include: thirst, fatigue, and gastrointestinal symptoms. If the serum sodium levels drop below 115 mEq/L, muscle twitching and convulsions may result.

If you want to read an overview of SIADH from a nursing continuing education perspective, follow this link:

RNWEB

<http://www.rnweb.com/ce/799/adh2.html>**Diabetes Insipidus**

Diabetes Insipidus or aldosterone insufficiency leads to polyuria and polydipsia. There are 3 main forms: neurogenic, nephrogenic and a psychogenic form. The neurogenic form occurs when there is insufficient ADH. This may be due to a lesion to the hypothalamus, infundibular stem or posterior pituitary, resulting in decreased ADH production. The nephrogenic form is usually associated with end-organ failure of the kidneys, resulting in nonresponsiveness to ADH. The psychogenic form results from abnormally large fluid intake. Treatment of the neurogenic form involves replacement of ADH (vasopressin).

If you want to read an overview of diabetes insipidus from a nursing continuing education perspective, follow this link:

RNWEB

<http://www.rnweb.com/ce/799/adh2.html>

**History:** A brief history of the research on nephrogenic diabetes insipidus is summarized at:

The NDI Foundation

<http://www.ndif.org/fs-hist.html>



## Learning Activity #6

List 4 possible receptor-associated disorders of the water-soluble hormones:

1. \_\_\_\_\_
2. \_\_\_\_\_
3. \_\_\_\_\_
4. \_\_\_\_\_

Answer the following questions true or false.

1.    T    F    Pathogenic mechanisms affecting target cell responses are more common in lipid-soluble hormones.
2.    T    F    Documenting the abnormal release of hypothalamic releasing hormones is difficult due to the short half-life and low concentrations of hypothalamic hormones.
3.    T    F    The most common cause of increased ADH levels is ectopic production.
4.    T    F    Treatment with barbiturates may result in SIADH.
5.    T    F    Phenytoin may be effectively used to treat ectopically produced ADH.
6.    T    F    In patients with diabetes insipidus, water restriction results in decreased urine volume.
7.    T    F    SIADH is characterized by low levels of ADH in the absence of normal physiologic stimuli for its release.

### *Diseases of the Anterior Pituitary*

#### **Hypopituitarism**

Hypopituitarism may be primary and due to the destruction of the anterior pituitary, or secondary due to inadequate secretion of releasing hormones. The pituitary gland is well vascularized and therefore vulnerable to ischemia. Pituitary infarction is one cause of primary hypopituitarism. Postpartum infarction following severe hemorrhage and vascular collapse is one example (Sheehan syndrome). Up to 32% of women with severe postpartum hemorrhage have some degree of hypopituitarism. No clinical manifestations are evident unless at least 75% of the gland is destroyed.

Greenspan, F.S. & Strewler, G.J. (1997) *Basic Clinical Endocrinology*. (5<sup>th</sup> ed.) Stamford, CT: Appleton and Lange. (pp. 125-130).

The hyposecretion of growth hormone is very rare. Progeria is a very severe form of growth hormone hyposecretion resulting in body tissue atrophy and the clinical signs of premature aging. A GH deficiency in children results in slow growth of the long bones and a condition known as pituitary dwarfism.

This condition is usually accompanied by other anterior pituitary deficiencies.

Marieb, E.N. (1998). *Human Anatomy and Physiology*. (4<sup>th</sup> ed.). Menlo Park, California: Benjamin/Cummings. (p. 597)

### Hyperpituitarism

Hyperpituitarism is due to primary adenomas. These tumors are usually benign and slow growing. They usually cause the hypersecretion of one or more pituitary hormones. Prolactinomas are the most common (60%). GH hypersecretion occurs in 20% of these cases, and ACTH hypersecretion in 10% of the cases.

Greenspan, F.S. & Strewler, G.J. (1997) *Basic Clinical Endocrinology*. (5<sup>th</sup> ed.). Stamford, CT: Appleton and Lange. (pp. 125-130).

Prolactinomas produce inappropriate lactation, lack of menses, and infertility in women and impotence in men. Large tumors may result in HYPOSECRETION due to the destruction of the gland, hypothalamic nuclei or the portal system. The clinical manifestations seen as the tumor enlarges include: headaches, fatigue, neck pain, seizures and visual changes.

### Acromegaly and Gigantism

**Acromegaly** is a disease state associated with the excessive secretion of growth hormone in adults. In children the effect of increased GH is called gigantism. The primary cause is a pituitary adenoma. The deleterious effects of excess GH result in increased mortality. The treatment involves removing the tumor surgically, radiotherapy and or treatment with synthetic GHIH.



**History:** one of the most famous persons who was an acromegalic giant was professional wrestler and actor Andre the Giant. For more information go to:

Puresu.com

<http://www.puroresu.com/wrestlers/andre/>

## Alterations of the Thyroid Function



**Read:** McCance and Huether. Pages 633-638 (up to thyroid carcinoma)

Here are some quick facts about thyroid dysfunction:

**Fact:** Two of the most common thyroid diseases, Hashimoto's thyroiditis and Graves's disease, are autoimmune diseases and may run in families.

**Fact:** Hypothyroidism is 10 times more common in women than in men.

**Fact:** One out of five women over the age of 75 has Hashimoto's thyroiditis, the most common cause of hypothyroidism.

**Fact:** Thyroid dysfunction complicates 5%-9% of all pregnancies.

**Fact:** About 15,000 new cases of thyroid cancer are reported each year.

**Fact:** One out of every 4,000 infants is born without a working thyroid gland.

**Fact:** About 100 million people worldwide don't get enough iodine in their diets; this problem has been solved in Canada and most developed countries by adding iodine into table salt ("iodized salt"). For more info on iodine deficiency disorder (IDD) follow this link:

Tulane University

<http://www.tulane.edu/~icec/aboutidd.htm>



**Note:** some good sample photographs of patients with various thyroid conditions can be viewed at:

University of Missouri Healthcare

[http://www.muhealth.org/~daveg/thyroid/thy\\_dis.html](http://www.muhealth.org/~daveg/thyroid/thy_dis.html)



**Note:** these are three good sites to refer your patients to for general information:

The AMA Patient Information Guideline

<http://www.amda.ab.ca/cpg/index.html>

The American Foundation of Thyroid Patients

<http://www.thyroidfoundation.org/>

The Thyroid Society FAQ

<http://www.the-thyroid-society.org/faq/>

To review how thyroid diseases can affect a patient's skin, follow this link:

Thyroid Foundation of Canada

<http://home.ican.net/~thyroid/Articles/EngE9B.html>

**Thyrotoxicosis**

**Thyrotoxicosis** is the clinical syndrome that results when tissues are exposed to high circulating levels of TH. This syndrome is usually due to hyperactivity of the thyroid gland.

**Hyperthyroidism**

Hyperthyroidism is nearly 10 times more frequent in women, and affects about 2% of all women in North America. The most common cause of hyperthyroidism, Graves' disease, affects at least 2.5 million Americans including Olympic athlete Gail Devers who won a gold medal in track after being diagnosed with and treated for Graves' disease. Graves' disease is an autoimmune disease of unknown cause, though in some cases there is a familial predisposition. It occurs when the body develops antibodies to TSH which act on the TSH receptors on the thyroid gland and increase TH production.



**Controversy:** A controversy exists as to the apparent increase in the number of people suffering autoimmune diseases. Some scientists argue that our current 'hygienic' lifestyle may be the cause of this increase. To examine this possibility go to:

Science News Online

[http://www.sciencenews.org/sn\\_arc99/8\\_14\\_99/bob2.htm](http://www.sciencenews.org/sn_arc99/8_14_99/bob2.htm)

**Diffuse Toxic Goiter**

A goiter is an enlargement of the thyroid gland. Once thyrotoxicosis has developed, the condition is known as toxic multinodular goiter. The symptoms are similar to those of Graves' disease.

**Thyrotoxic crisis**

Thyrotoxic crisis or thyroid storm is a life threatening condition that may occur in hyperthyroid patients following excessive stress. The symptoms are due to an increase in catecholamine binding sites in the heart and nervous tissues, making them more sensitive to stress related increases in sympathetic activity.

**Hypothyroidism**

**Note:** A good review on the history and epidemiology of hypothyroidism, especially cretinism and myxedema, can be found at the following link:

Thyroid Disease Manager

[http://www.thyroidmanager.org/Chapter20/20\\_frame.htm](http://www.thyroidmanager.org/Chapter20/20_frame.htm).



**Note:** For a sample photograph of what a baby with cretinism looks like, go to:

The University of Illinois at Chicago

[http://www.uic.edu/classes/phyb/phyb402rcj/th\\_pics/thcretin.html](http://www.uic.edu/classes/phyb/phyb402rcj/th_pics/thcretin.html)

For patient with moderate myxedema/hypothyroidism, go to:

The University of Kansas Medical Center

[http://www.kumc.edu/instruction/medicine/pathology/ed/ch\\_21/c21\\_s38.html](http://www.kumc.edu/instruction/medicine/pathology/ed/ch_21/c21_s38.html)

## Learning Activity #7

Fill in the blanks

1. The most frequently studied form of hypopituitarism is \_\_\_\_\_ syndrome.
2. \_\_\_\_\_ is a condition in which all hormones of the anterior pituitary are absent.
3. Most cases of primary adenoma are found with \_\_\_\_\_ examination.
4. An enlarged tongue, interstitial edema and increased size and function of sebaceous glands are clinical manifestations of \_\_\_\_\_.
5. The most common cause of hyperthyroidism is \_\_\_\_\_.
6. Another name for Thyrotoxic crisis is \_\_\_\_\_.





## Alterations of Adrenal Function

**Read:** McCance and Huether, pages 657-666.

### *Diseases of the Adrenal Cortex*

#### **Cushing's Disease**

The most common cause of Cushing's disease is a ACTH secreting pituitary tumor. Other causes of hypercortisolism include ectopic production or the exogenous administration of excess cortisol.



**Note:** Sample photographs of patients with Cushing's disease can be seen at this homepage:

Cushing's Syndrome and Cushing's Disease

<http://cushings.homestead.com/photos.html>

You may want to refer your patients with Cushing's Disease to the Cushing's Support & Research Foundation at:

The Cushing Support and Research Foundation

<http://world.std.com/~csrf/index.html>

#### **Hyperaldosteronism**

Hyperaldosteronism is due to excessive aldosterone secretion by the adrenal cortex. Primary hyperaldosteronism is due to an abnormality of the adrenal cortex. Secondary forms may be due to excessive stimulation of the adrenal cortex by angiotensin II. The important clinical manifestations include hypertension and hypokalemia.

#### **Hypersecretion of Adrenal Androgens/Estrogens**

The effect of androgen hypersecretion is masked in adult males. In prepubescent males, a maturation of the reproductive organs and the development of secondary sex characteristics may occur. In females, virilization may occur including excessive facial and body hair (hirsutism), a deepening of the voice and clitoral enlargement. Amenorrhea infertility and breast atrophy may also occur. Note that another potential cause of hirsutism is polycystic ovarian syndrome.

#### **Hypocortical Functioning**

Hypocortisolism usually involves deficits in both glucocorticoids and mineralcorticoids. Primary adrenal insufficiency is Addison's disease. Hyposecretion may also be due to insufficient ACTH secretion (secondary hypocortisolism). The clinical manifestations include: weakness, fatigue, gastrointestinal disturbances, hypoglycemia, vitiligo, and hypotension. In Addisonian crisis, this severe hypotension leads to vascular collapse.



**Note:** Canadian scientists are actively involved in Addisonian research involving DHEA; follow this link for a summary:

Site van de Patiëntenbeweging

<http://www.spin.nl/nvap0417.htm>

### *Diseases of the Adrenal Medulla*

#### **Adrenal Medulla Hypofunction**

This probably only occurs iatrogenically in individuals following a bilateral adrenalectomy.

#### **Adrenal Medulla Hyperfunction**

This may occur due to pheochromocytomas or tumors of the chromaffin cells of the sympathetic nervous system. The hypersecretion of epinephrine from the adrenal medulla may result in the following clinical manifestations: hypertension, tachycardia, palpitations, headaches, weight loss, and heat intolerance. Note increased secretion may occur in these patients following the ingestion of tyrosine rich foods such as red wine or cheese. Tyrosine is the amino acid precursor of epinephrine.



**Note:** A good site to refer your patients with adrenal diseases to is the NADF homepage at:

National Adrenal Diseases Foundation

<http://www.medhelp.org/nadf/>



### **Learning Activity #8**

Match the following conditions with one or more of the following conditions:

- |  |                                     |
|--|-------------------------------------|
| a. confusion, memory loss and cold intolerance | _____ 1. Grave's disease            |
| b. enlarged facial bones                       | _____ 2. Addison's disease          |
| c. exophthalmos                                | _____ 3. Acromegaly                 |
| d. 'buffalo hump'                              | _____ 4. Cushing's disease          |
| e. hypokalemia                                 | _____ 5. hypothyroidism             |
| f. vitiligo and hypoglycemia                   | _____ 6. hyperaldosterone secretion |
| g. low urine osmolality                        | _____ 7. diabetes insipidus         |

## Final Thoughts

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The endocrine system is a complex web of interconnections that affects virtually every organ and system in the body. Any disruption in this delicate web can have long reaching implications. A thorough knowledge of endocrine physiology and pathology will increase your ability to care for endocrine patients, particularly in the increasingly important nursing role as patient educator.

## References

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Brindley, D. (1996) Metabolism of Triacylglycerols. In D.E. Vance & J. Vance (Eds.), *Biochemistry of Lipids and Lipoproteins and Membranes*. (pp. 171-203). Amsterdam: Elsevier.

Greenspan, F.S. & Strewler, G.J. (1997) *Basic Clinical Endocrinology*. (5<sup>th</sup> ed.). Stamford, CT: Appleton and Lange. (pp. 213-215).

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## Glossary

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**acromegaly:** A disease state associated with the excessive secretion of growth hormone in adults.

**Addison's disease:** A disease of the adrenal cortex characterized by the hyposecretion of cortisol.

**adenohypophysis:** The anterior lobe of the pituitary gland. The hormones of the anterior pituitary are: growth hormone, TSH, FSH, LH, prolactin and MSH.

**catecholamine:** A class of peptide neurohormones that includes dopamine, epinephrine, norepinephrine and serotonin.

**cretinism:** A congenital disease of the newborn characterized by hypothyroidism.

**Cushing's disease:** A disease of the adrenal cortex characterized by the hypersecretion of ACTH or cortisol.

**diabetes insipidus:** A disease caused by aldosterone insufficiency that leads to polyuria and polydipsia.

**glucocorticoid:** A steroid hormone synthesized in the adrenal cortex that is secreted under stress, eg. cortisol.

**goiter:** An enlargement or hypertrophy of the thyroid gland.

**Grave's disease:** An autoimmune disease characterized by hyperthyroidism.

**hypophysis:** AKA the pituitary gland (see adenohypophysis and neurohypophysis).

**mineralocorticoid:** A steroid hormone synthesized in the adrenal cortex that is secreted to retain sodium, eg. aldosterone.

**myxedema:** A disease characterized by hypothyroidism in adults.

**neurohypophysis:** The posterior lobe of the pituitary gland. The hormones of the posterior pituitary are: oxytocin, ADH.

**progeria:** A disease caused by hyposecretion of growth hormone that is characterized by body tissue atrophy and premature aging.

**second messengers:** Are intracellular molecules generated by the binding of a hormone or neurotransmitter to a plasma membrane receptor.

**sheehan syndrome:** A syndrome caused by hypopituitarism due to postpartum infarction of the pituitary.

**syndrome of inappropriate antidiuretic hormone:** A syndrome characterized by high levels of ADH in the absence of normal physiologic stimuli.

**thyrotoxicosis:** Is the clinical syndrome that results when tissues are exposed to high circulating levels of TH.

**tropic hormones:** Hormones that regulate the release of other hormones.

## Acronym List

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<b>ACE</b>	Angiotensin-converting enzyme
<b>ACTH</b>	Adrenocorticotrophic hormone
<b>ADH</b>	Antidiuretic hormone
<b>CRH</b>	Corticotropin releasing hormone
<b>cAMP</b>	cyclic adenosine monophosphate
<b>cGMP</b>	cyclic guanosine monophosphate
<b>DHEA</b>	Dehydroepiandrosterone
<b>FSH</b>	Follicle Stimulating Hormone
<b>GH</b>	Growth Hormone
<b>GHIH</b>	Growth Hormone Inhibiting Hormone
<b>GHRH</b>	Growth Hormone Releasing Hormone
<b>GnRH</b>	Gonadotropin Releasing Hormone
<b>LH:</b>	Lutenizing Hormone
<b>PIH</b>	Prolactin Inhibiting Hormone
<b>PRH</b>	Prolactin Releasing Hormone
<b>SIADH</b>	Syndrome of inappropriate antidiuretic hormone
<b>T<sub>4</sub></b>	Tetraiodothyronine
<b>T<sub>3</sub></b>	Triiodothyronine
<b>TRH</b>	Thyrotropin Releasing Hormone
<b>TSH</b>	Thyroid stimulating hormone

## Checklist of Requirements

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### Required Readings

- ☐ McCance, K., & Huether, S. (2001). Pathophysiology: The biologic basis for disease in adults and children (4<sup>th</sup> ed.). St. Louis: Mosby. Pages: 597-609; 613-619; 624-638 (including Table 20-2) and 656-667.
- ☐ Print Companion: Alterations in Endocrine Function

### Required Activities

- ☐ Learning Activity #1
- ☐ Learning Activity #2
- ☐ Learning Activity #3
- ☐ Learning Activity #4
- ☐ Learning Activity #5
- ☐ Learning Activity #6
- ☐ Learning Activity #7
- ☐ Learning Activity #8

## Answers to Learning Activities

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### Learning Activity #1

1. d
2. a
3. c
4. d
5. d
6. b
7. b

### Learning Activity #2

1. b (false)
2. d
3. d
4. c

### Learning Activity #3

1. F (false)
2. T (true)
3. T
4. F
5. F
6. F
7. F
8. F

### Learning Activity #4

1. b (false)
2. b
3. b (false)

### Learning Activity #5

1. b
2. c
3. b (false)
4. a (true)
5. b (false)
6. a (true)
7. a (true)
8. a (true)



## Learning Activity #6

The 4 possible receptor-associated disorders of the water-soluble hormones are:

1. decreased number of receptors
2. impaired receptor function
3. antibodies to the receptor that may mimic hormone or decrease the number of available binding sites
4. unusual receptor expression

Answers to True and False questions

1. F (false)
2. T (true)
3. T
4. T
5. F
6. F

## Learning Activity #7

1. Sheehan syndrome
2. panhypopituitarism
3. post-mortem
4. acromegaly
5. Grave's disease
6. thyroid storm

## Learning Activity #8

1. c
2. f
3. b
4. d
5. a
6. e
7. g