



Paramedic Resource Manual

ENDOCRINE SYSTEM SECTION NINE

2014 Update by
Ontario Base Hospital Group Education Subcommittee

OBJECTIVES: ENDOCRINE SYSTEM

The objectives indicate what you should know, understand and be prepared to explain upon completion of this module. The self assessment questions and answers will enable you to judge your understanding of the material.

Upon completion of this module, the student should be able to:

1. define and give an example of:
 - a) Endocrine gland
 - b) Exocrine gland
 - c) Mixed gland
 - d) Double gland.
2. name and locate the principal endocrine glands.
3. list the major functions and secretions of each principal gland.
4. describe the action of each secretion listed in 3.
5. describe the mechanism for maintenance of normal blood glucose levels.
6. define gluconeogenesis and glycogenolysis.
7. describe the pathophysiology of diabetes, differentiating between:
 - Type I and Type II diabetes
 - Hypoglycemia and hyperglycemia
 - DKA and HHNK.
8. differentiate clinically between hypoglycemic and hyperglycemic emergencies.

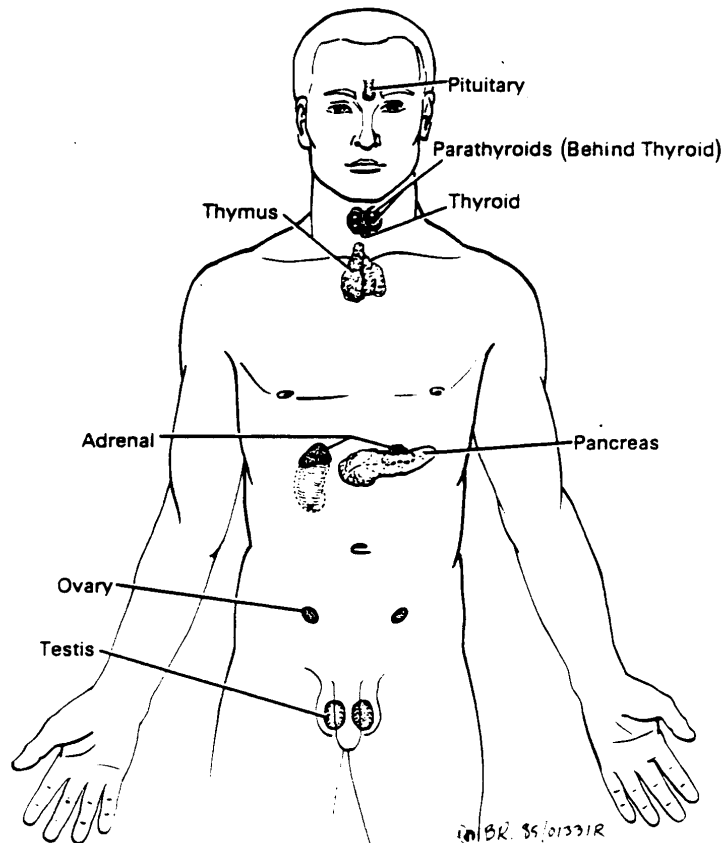
If you have studied this subject previously, you may test your ability using the self assessment questions. If you are able to obtain 90% or greater, you may choose not to do the unit and merely review the sections, or parts of sections, where weakness may exist. If you obtain less than 90%, it is recommended that the module be done in its entirety, stressing areas where more review is needed.

MAJOR ENDOCRINE GLANDS

The endocrine system, which exerts control over development, growth and metabolic activities, is comprised of a number of glands distributed throughout the body (Figure 1). The eight major glands of the system are:

1. Pituitary
2. Thyroid
3. Parathyroid
4. Thymus
5. Islets of Langerhans in the pancreas
6. Adrenal (suprarenal)
7. Testis (in the male)
8. Ovary (in the female).

FIGURE 1: MAJOR ENDOCRINE GLANDS



This module confines itself primarily to a discussion of the anatomy and physiology of the major endocrine glands. There are, however, other hormonal organs, tissue or cells in the body which also have a secretory function. These include the kidney, pineal gland, placenta and cells in the gastrointestinal tract.

Although dysfunction may occur in any gland, the pancreas is the one most commonly involved in pathologies seen by the Paramedic. Therefore, a more detailed discussion of the pancreas is presented in the second section of this module.

TYPES OF GLANDS

ENDOCRINE GLAND

Endocrine glands are ductless glands, although not all of the ductless glands in the body are endocrine glands, e.g. spleen. Their secretions are always hormones which pass directly into the bloodstream. Endocrine glands are made up of clusters of glandular epithelium.

Hormones are the secretions produced by the endocrine glands, and may be a form of protein, amine or steroid (lipid or fat). The function of the hormones is to maintain homeostasis (a constant internal body environment) by changing the physiological activities of cells. A hormone may stimulate changes in the cells of an organ or groups of organs, called target organs; a hormone may also directly affect the activity of all the cells in the body.

MIXED GLAND

A mixed gland is a gland which has both endocrine and exocrine functions. The exocrine component has ducts, through which its secretions pass. The pancreas is a gland with both endocrine (insulin production) and exocrine (production of digestive enzymes) functions.

DOUBLE GLAND

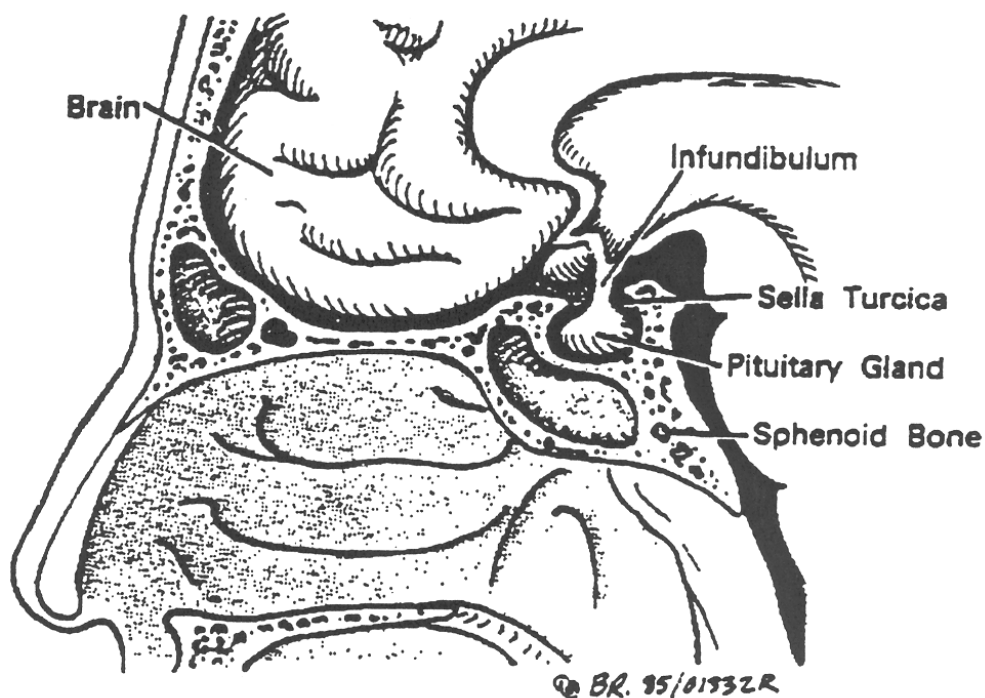
A double gland consists of two main parts in which the two components differ in their embryological derivation, nature of hormones released and the mechanism of neural control. The pituitary and adrenal glands are examples of double glands.

PITUITARY GLAND (HYPOPHYSIS CEREBRI)

The pituitary was formerly referred to as the master endocrine gland, because of its controlling influence on many of the other endocrine glands. This reddish-brown gland, the size of a garden pea, lies in the sella turcica (pituitary fossa, hypophyseal fossa), a fossa on the superior aspect of the body of the sphenoid bone in the base of the skull.

The pituitary is attached to the hypothalamus at the base of the brain by a stalk called the infundibulum.

FIGURE 2: LOCATION OF THE PITUITARY GLAND



The anterior lobe (adenohypophysis) of this double gland is responsible for the secretion of:

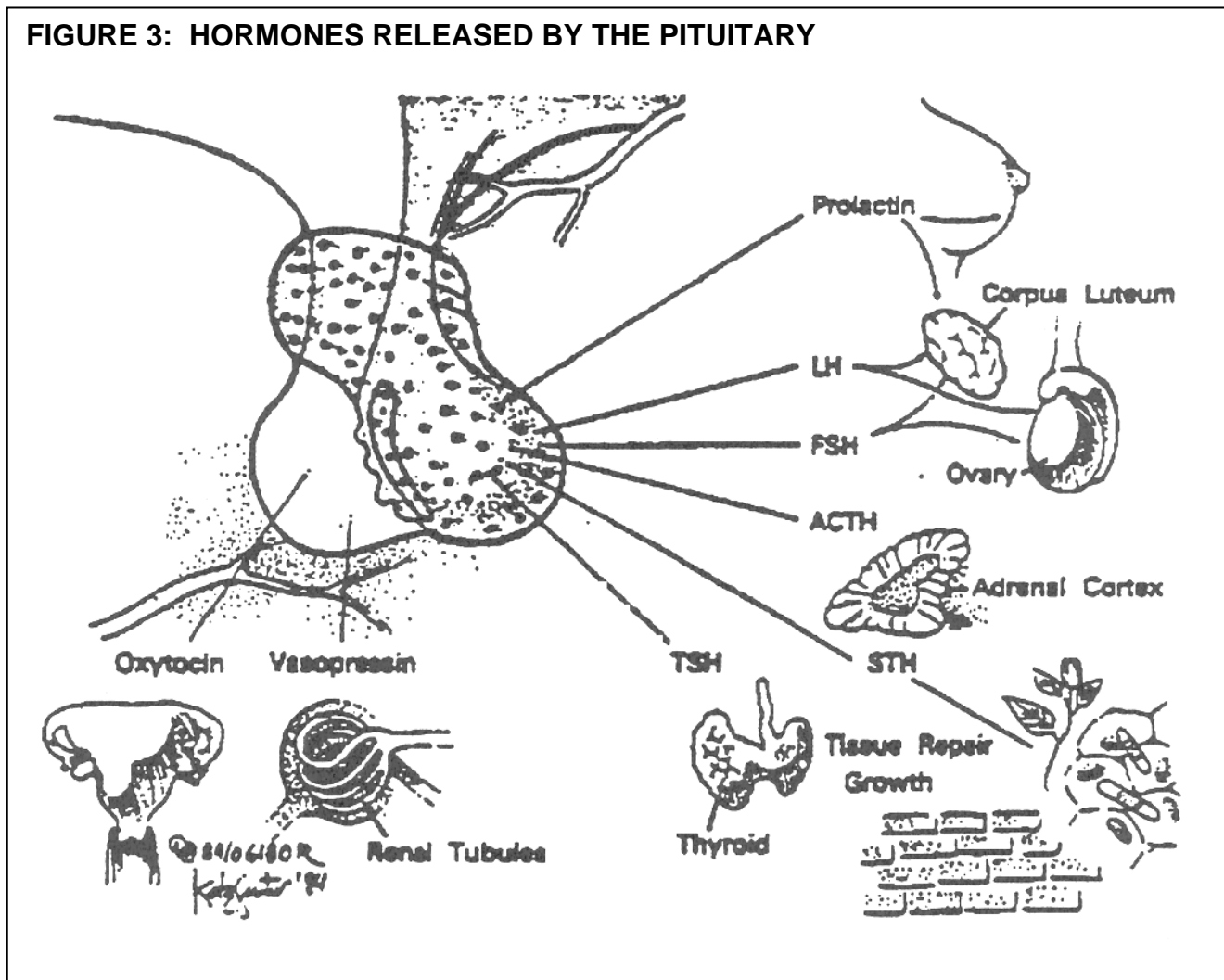
1. Growth hormone - somatotropic (STH)
2. Thyroid stimulating hormone - thyrotropic (TSH)
3. Adrenal cortex stimulating hormone - adrenocorticotropic (ACTH)
4. Sex gland stimulating hormones - gonadotropic (LH and FSE)
5. Mammary gland stimulating hormones - prolactin.

The posterior lobe secretes:

1. Oxytocin
2. Vasopressin (antidiuretic hormone - ADH).

Both of these cause the contraction of the smooth muscle in various organs.

FIGURE 3: HORMONES RELEASED BY THE PITUITARY



HYPOTHALAMUS

Most of the hormones secreted by the anterior pituitary stimulate the production of hormones by other endocrine glands (target glands).

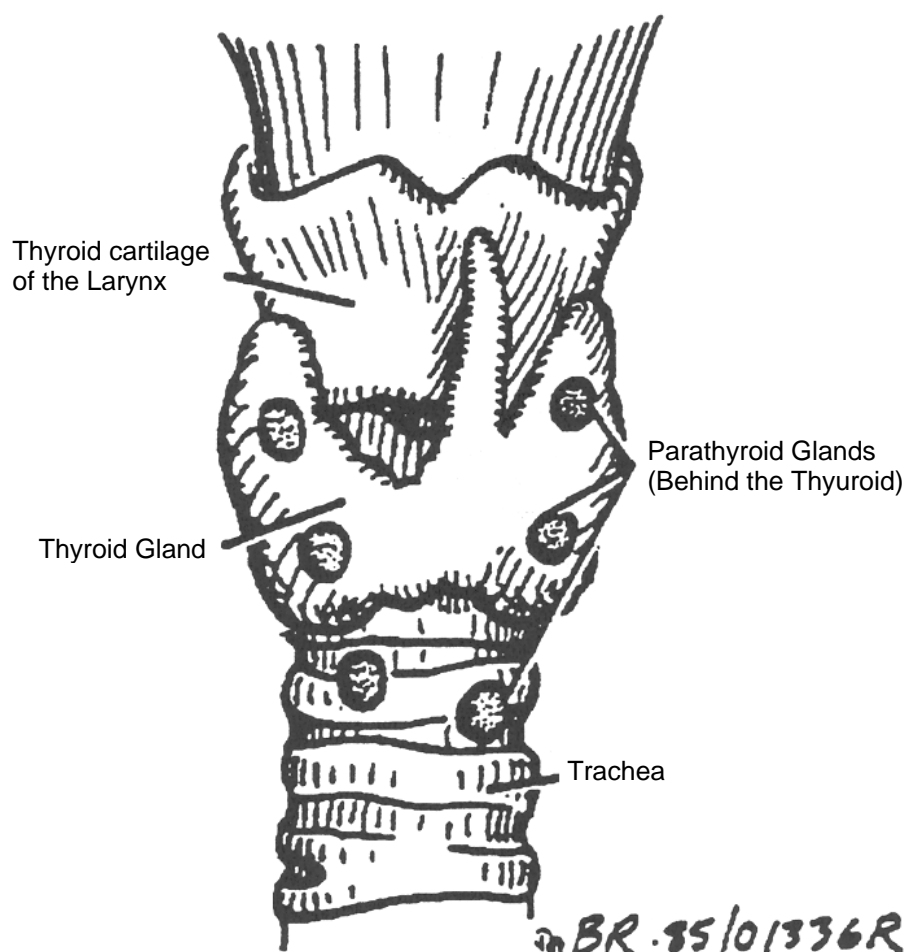
Control of the rate of secretions in these target glands is accomplished by the hypothalamus through cells sensitive to changes in hormone levels in the blood. Since the hypothalamus

responds to an absence of pituitary hormones by producing substances (releasing factors) which enhance hormone secretion, this is referred to as a negative feedback mechanism.

THYROID GLAND

This gland is situated below the larynx at the upper part of the trachea. it consists of two lateral pear-shaped lobes united at the lower part by the isthmus, which lies in front of the second, third and fourth tracheal rings.

FIGURE 4: THYROID AND PARATHYROID GLAND



Inadvertent penetration of this very vascular gland during needle or surgical cricothyroidotomy may result in severe hemorrhage.

The function of the thyroid gland is to:

1. Take up the inorganic iodine (derived from food) present in the blood.
2. Produce the hormone thyroxine.
3. Store thyroxine as thyroglobin.
4. Release thyroxine when stimulated to do so by the thyroid stimulating hormone (TSH) secreted by the adenohypophysis (pituitary gland).

THYROXINE

1. Thyroxine controls the metabolic rate of the body (the rate at which the body uses oxygen and metabolizes food products).
2. Thyroxine is necessary for normal physical and mental development to occur.

PARATHYROID GLANDS

These, the smallest of the endocrine glands, may be variable in number and position. Normally there are four - two situated on the posterior surface of each of the lateral lobes of the thyroid gland. They lie beneath the capsule of the thyroid and may in fact be embedded in its tissue.

The parathyroid glands secrete a hormone, parathyroid hormone (parathormone) (PTH), which is concerned with calcium metabolism. The secretion of this hormone is regulated by blood calcium levels, and is not under the control of the adenohypophysis.

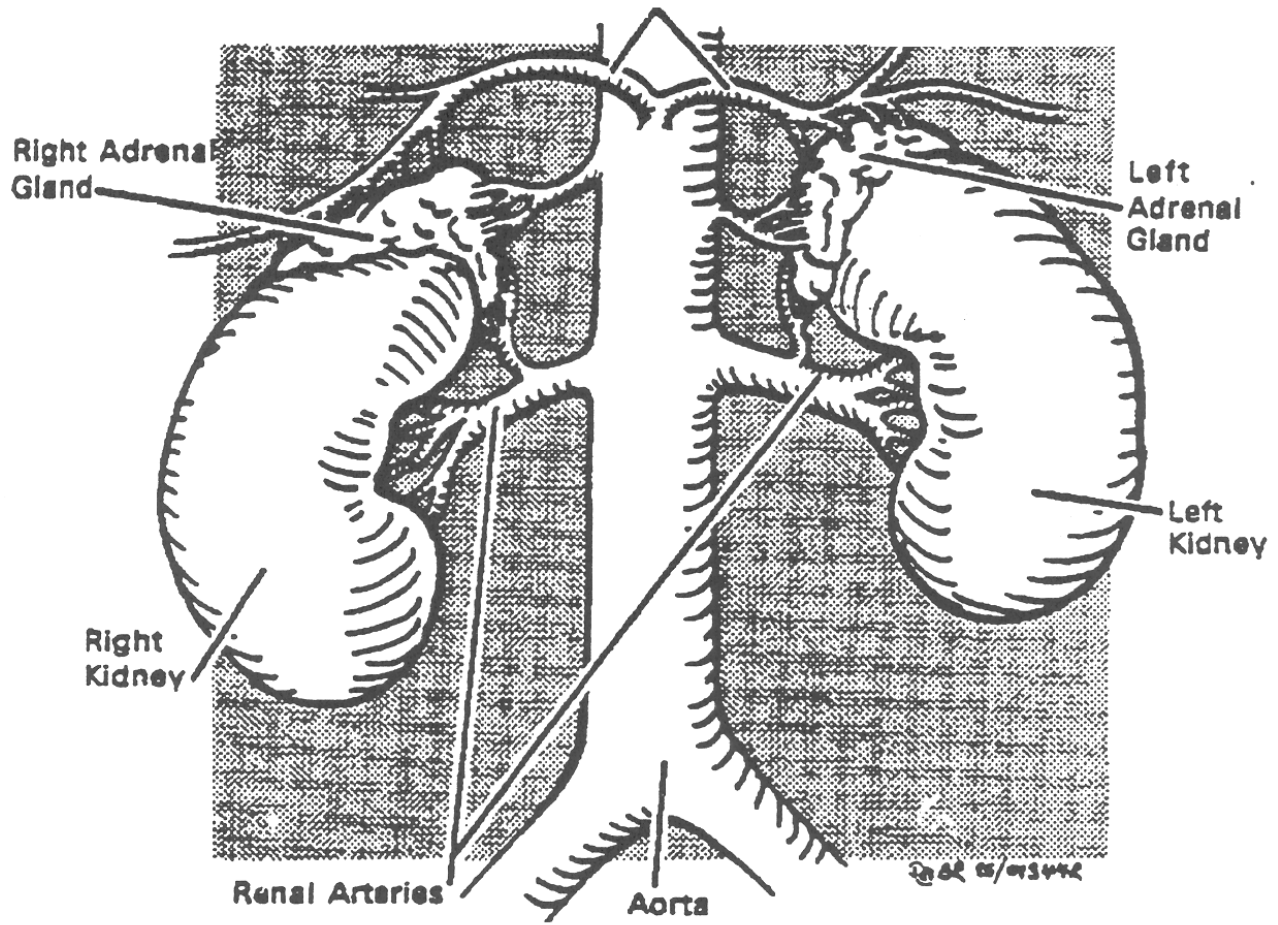
THYMUS

The thymus lies in the anterior and superior parts of the thorax. It is a relatively large structure at birth and continues to grow until puberty. Although the thymus decreases in size during adult life its physiological importance does not. It provides the mold, or model, for other organs, such as the liver and spleen, to produce T-lymphocytes (white blood cells concerned with the defense against infection and disease).

ADRENAL (SUPRARENAL) GLANDS

These two asymmetrical glands are found in close proximity to the upper poles of the kidneys. The right one is pyramidal and embraces the upper pole of the right kidney; while the left is crescentic and is located on the suprarenal aspect of the left kidney above the hilum. Each measures about 5 cm long and 3 cm thick. Each gland is supplied by three relatively large arterial branches, indicating its relative importance to the body.

FIGURE 5: ADRENAL GLAND



The adrenal glands are comprised of two functionally separate parts, the cortex and medulla.

The adrenal cortex secretes three main groups of hormones:

1. Glucocorticoids which are concerned with helping the body to cope with stressful situations.
2. Mineralcorticoids which have a similar function to that of the glucocorticoids, as well as affecting sodium and water balance in the body.
3. Androgens, the male sex hormones, which are only produced in small amounts.

The action of the hormones secreted by the adrenal medulla is to prepare the body for sudden action at a time of pain or fear (fight or flight situations). These hormones are:

- Adrenalin (epinephrine)
- Noradrenalin (norepinephrine).



Clinical vignette

Pheochromocytoma is a rare tumor of the adrenal gland that causes excess secretion of adrenalin. This may lead to hypoglycemia in non-diabetics. Administration of Glucagon may affect the adrenal gland/tumor resulting in even greater release of adrenalin and this may, paradoxically, worsen hypoglycemia.

GONADS

At the time of puberty, the adenohypophysis starts to secrete progressively more gonadotropic hormones. There are two in particular which affect both the ovaries and the testes. These are:

- Follicle stimulating hormone (FSH)
- Luteinizing hormone (LH) or interstitial cell stimulating hormone (ICSH) as it is called in the male.

OVARIES

The FSH and LH secreted by the adenohypophysis stimulate the development of all the female reproductive organs and initiate the menstrual cycle. The menstrual cycle is a series of rhythmic changes which occur throughout the childbearing years.

Each cycle lasts about 28 days and during this time three major hormone related events occur.

1. The FSH stimulates the maturation of the follicle containing an ovum which in turn produces the hormone estrogen.
2. The FSH and LH together bring about ovulation (the release of the ovum from the follicle and ovary).
3. The LH causes the lining of the uterus, the endometrium, to thicken in preparation for the implantation of a fertilized ovum. LH also causes the follicular cells left behind in the ovary to change their characteristics and colour to become the corpus luteum. The corpus luteum in turn produces large quantities of the hormone progesterone and small quantities of estrogen.

If fertilization and implantation do not take place, the corpus luteum will degenerate, the production of progesterone and estrogen will decrease and menstruation (bleeding) will occur.

(Estrogen production will also decrease after ovulation has taken place). At this time the negative feedback mechanism will be initiated.



Clinical vignette

The ovaries and corpus luteum are sites where the formation of cysts is relatively common. These cysts can cause torsion of the ovary resulting in pain, or they can bleed or rupture.

TESTES

The seminiferous tubules of the testes are stimulated by the secretion of FSH to produce spermatozoa and bring them to maturation.

The secretion of the ICSH stimulates the interstitial cells, the cells of Leydig, to produce the male hormone testosterone. The action of testosterone is to stimulate development and activity of male sex glands, as well as bringing about the changes that produce the secondary sexual characteristics (growth of body hair, and voice change). Testosterone levels are controlled by a negative feedback loop.

“FRINGES” OF THE ENDOCRINE SYSTEM

There are body structures whose tissue have a glandular appearance and have been suspected, despite the lack of convincing evidence, of having endocrine properties.

PINEAL GLAND

Located in the forebrain, the pineal gland was the subject of speculation for years. In fact, as recently as 1985, when this pre-course manual was first written, understanding of the pineal gland's function was very poorly understood. Today, a little bit more is understood about the Pineal gland, however, Physiologists and Endocrinologists still have a long way to go.

The pineal gland produces Melatonin which plays a role in the body's circadian rhythm or "biological time clock". In the dark, the pineal gland is stimulated to produce and release melatonin and in the daylight, melatonin production is reduced. It's recently been suggested that treatment with melatonin may prevent and/or reduce jetlag, reduce the risk of developing certain tumors and may slow the aging process. The Pineal Gland also regulates our patterns of eating (hunger), sleeping, reproduction (female reproductive cycle), and behaviour.

Seasonal affective disorder (SAD) has been linked to the Pineal Gland, as there is a reduction in Melatonin during the winter months when daylight is shortened. In some occult religions, it is sometimes referred to as the "third eye" and is believed to be a dormant organ that can be awakened to give mystical powers.



Clinical vignette

The pineal gland has a midline position within the brain and in some adults it is calcified, making it a visible landmark on skull x-rays. If it has shifted out of the midline, it may indicate the presence of a pathological process such as an expanding lesion.

INTESTINAL TRACT GLANDS

Intestinal tract glands that secrete gastrin, secretin and cholecystokinin are endocrine glands because their secretions pass directly into the circulatory system. The significance of these hormones are not discussed in this precourse material.

THE KIDNEY

The peptide angiotensin, found in the bloodstream, is effective in causing high blood pressure and its presence is primarily the result of kidney activity. This "hormone" is not produced in the kidney, but an enzyme from this organ, renin, is responsible for initiating chemical changes in a protein in the blood stream which cause eventual production of the "hormone". Also, evidence indicates that red blood cell formation is influenced by a hormonal agent, erythropoietin and again the kidney is involved.

In this section, we have primarily examined the anatomy of the organs other than the pancreas that have been conveniently classified as endocrine glands. However, we should remain aware there are certain organs, tissues or cells, whose primary role is not endocrine, but may emit secretions to the circulation and assume an endocrine function.

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SELF-ASSESSMENT: MAJOR ENDOCRINE GLANDS

MARKS

[2] 1. Give one example of a:

a) mixed gland

b) double gland

[2] 2. What is a mixed gland?

[2] 3. What is a double gland?

[2] 4. The (a) _____ gland is attached to the base of the brain and its body rests in the b) _____ of the sphenoid bone.

[1] 5. The secretions of endocrine glands pass directly into the blood stream. True or False.

[2] 6. Name two different endocrine glands that are located in the abdomen.

[2] 7. What is the anatomical location of the thyroid gland?

[1] 8. Where are the parathyroid glands found?

[1] 9. What chemical substances are released by the adrenal medulla?

[2] 10. Which of the following hormones are produced by the adenohypophysis?

- a) Oxytocin
- b) Estrogen
- c) Growth hormone
- d) Thyroxine
- e) Adrenalin

[9] 11. Complete the following chart. -

GLAND	HORMONE (S) PRODUCED	FUNCTION (S) OF HORMONE (S) PRODUCED
adrenal cortex	thyroxine parathromone ADH 1. glucocorticoids 2. mineralocorticods 3.	similar to gluco-corticoids sex hormones
adrenal medulla	1. epinephrine 2. norepinephrine	

[2] 12. The kidneys secrete hormones that control
(a) _____ and regulate (b) _____ production.

28 TOTAL

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SELF-ASSESSMENT ANSWERS: MAJOR ENDOCRINE GLANDS

1. a) Pancreas
 b) Pituitary gland (hypophysis)
2. A gland with both endocrine and exocrine function.
3. A gland consisting of at least two major components which differ in their hormone production.
4. (a) Pituitary, (b) Sella Turcica
5. True.
6. Adrenals (suprarenals) and Pancreas
7. Below the larynx, anterior to the upper end of the trachea.
8. One pair on the posterior aspect of each of the lobes of the thyroid gland.
9. Adrenalin (epinephrine), and Noradrenalin (norepinephrine)
10. c) Growth Hormone

11.

GLAND	HORMONE(S) PRODUCED	FUNCTION OF HORMONE(S)
thyroid	thyroxine	control of metabolic rate of tissues
parathyroids	parathormone	calcium metabolism
pituitary- posterior lobe	ADH	conserves water
adrenal cortex	1. glucocorticoids 2. mineralocorticoids	stress response similar to glucocorticoids
adrenal medulla	3. androgens 1. epinephrine 2. norepinephrine	sex hormones "fight or flight response"

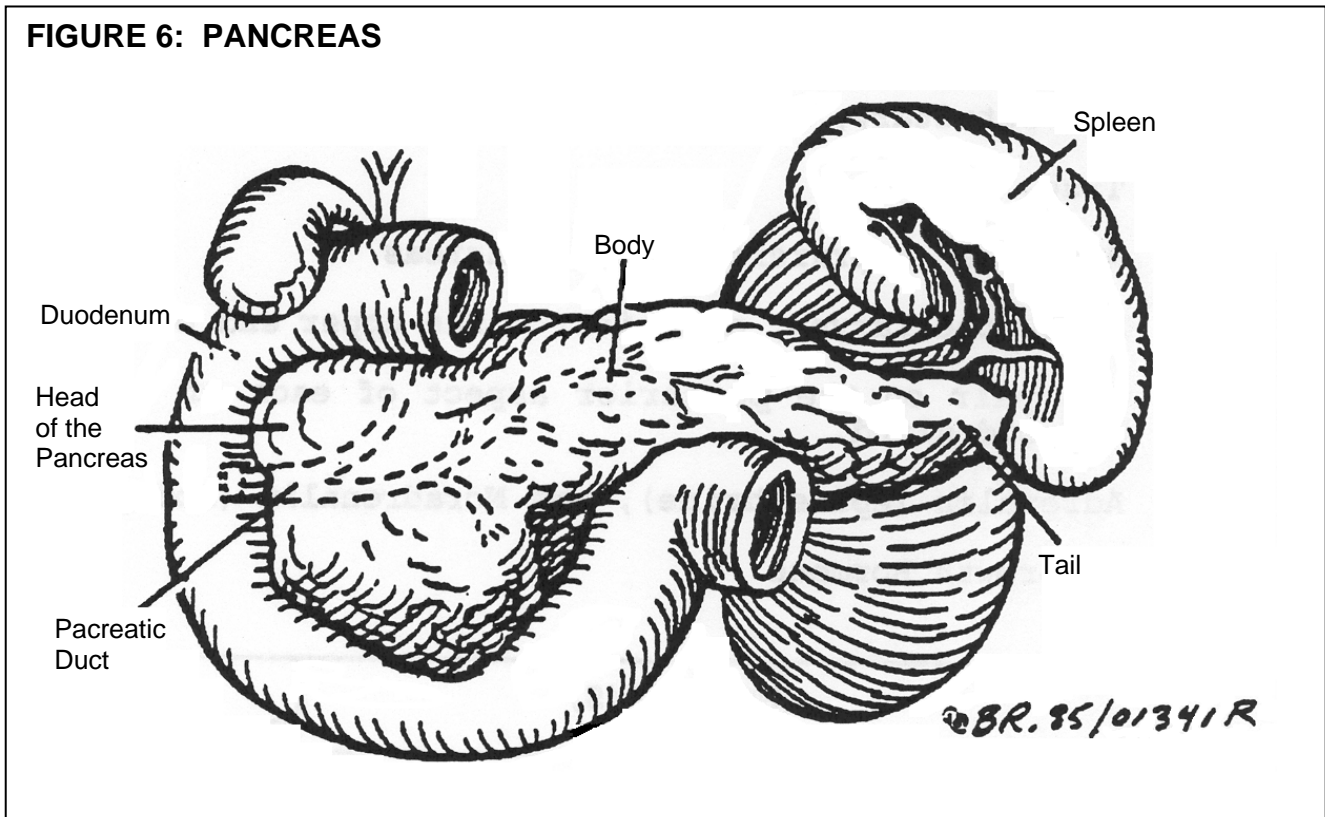
12. (a) Blood pressure; (b) Red blood cell

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THE PANCREAS

The pancreas consists of a head, body and tail. It lies behind the stomach, lying transversely on the posterior abdominal wall. Its head lies in the loop of the duodenum and its tail reaches the hilum (point of entry or exit of ducts, nerves, vessels) of the spleen.

FIGURE 6: PANCREAS



PANCREATIC ISLETS

Although the greater part of the glandular tissue of the pancreas is devoted to the production of digestive enzymes which pass through ducts to the duodenum (exocrine function), cells of another type of tissue can be seen distributed through the gland as isolated islands. These irregular clumps of cells, called the Islets of Langerhans comprise approximately 2 % of the pancreas and constitute the endocrine secretory units of the pancreas. An extensive capillary network is associated with each islet to transport its secretions from the pancreas.

Since the pancreas has both endocrine and exocrine functions, it is therefore a mixed gland. Its endocrine secretions are the hormones insulin and glucagon.

The islet cells may be classified as alpha or beta; the beta cells being most abundant. Insulin is produced by the beta cells in the Islets of Langerhans to lower blood sugar levels. The release of insulin by the pancreas occurs when blood sugar levels are high.

Glucagon, produced by the alpha cells in the Islets of Langerhans, has the opposite effect to insulin. It stimulates the conversion of glycogen (the storage form of glucose) to glucose which is released into the blood, thus raising the level of blood sugar.

There is little nervous influence on insulin hormonal secretion, which appears to be mainly controlled by the level of glucose passing through the islets.

The action of insulin is to facilitate the transport of glucose across the cell membrane, making it available to the cell for oxidation in the Krebs's Cycle. Insulin also facilitates storage of glucose as glycogen in depots primarily in the liver, muscle and kidney.

Glucose is the primary cell fuel. Certain cells can only use glucose. The most important of these are the cells of the CNS. Ninety percent of brain cell metabolism utilizes glucose. In fact, glucose is so important to normal brain cell function that it does not require insulin to facilitate its passage across the cell membrane. To some degree this compensates for lack of a backup stockpile of stored glucose (glycogen) here. The brain is therefore dependent on a constant supply of glucose to maintain its cellular activities. Let us examine how this is achieved.

REGULATION OF BLOOD GLUCOSE LEVELS

About 50% of western world diets consist of carbohydrates which are readily absorbed from the gut after breakdown by digestive enzymes. The bulk of digested carbohydrate is in the form of various sugars, the most plentiful being glucose which is ready for transport by the bloodstream and immediate use by the cell.

Since insulin is the key for the entry of glucose into many cells (brain and liver excluded) it must be produced and transported to the cells, along with the glucose.

Circulating levels of glucose are the trigger for the beta cells in the Islets of Langerhans to release insulin. Promoting the storage of glucose as glycogen (primarily in the liver) is another important role of insulin. In doing so, it inhibits the conversion of glycogen to glucose at this time. However, most of us have long periods, i.e. overnight, where we do not have a food intake.

As glucose from a meal is either used up or stored, insulin levels fall. The hormone glucagon (from the alpha cells of the islets of Langerhans) is then released along with epinephrine from the adrenal medulla. This causes mobilization of glycogen stores, again primarily from the liver. Conversion of glycogen to glucose (glycogenolysis) raises blood sugar levels, ensuring a constant supply to the cells.

However, these stores are limited. In fact, the liver barely stores enough glycogen to meet the metabolic needs of brain tissue for a 24 hour period. As glycogen stores are consumed proteins and fats from other sources (mostly muscle tissue and adipose) are freed for conversion to

glucose by the liver (gluconeogenesis). This process is mediated by glucocorticoids from the adrenal cortex. In times of prolonged starvation the kidneys become involved in gluconeogenesis from fats. This process is facilitated by insulin and enhanced by epinephrine and growth hormone (from the pituitary).



Clinical vignette

Administration of Glucagon will raise the blood glucose level in a patient who has sufficient glycogen stores. A second dose of Glucagon may be indicated for patients whose blood glucose level remains ≤ 4 mmol/L after the first dose, however, if the glycogen stores are inadequate, there will be not effect on BGL.

It should now be apparent that our homeostatic systems are geared to provision of a constant supply of glucose to the CNS, sparing protein and fat for other duties. As with any homeostatic regulatory mechanism, there exists a potential for problems at any one of the stages.

By far the most common endocrine emergency encountered by prehospital personnel will involve the diabetic patient. What follows is a discussion of the more important features of diabetes mellitus, and the implications for you as a care provider. Before proceeding, let's clarify one point ... diabetes insipidus is a different pathology involving primarily the pituitary gland. It will not be discussed in this unit.

DIABETES MELLITUS (DM)

Diabetes is a chronic disorder affecting the way the body uses and stores the foods eaten and is caused by a deficiency or the ineffectiveness of insulin secreted by the pancreas. Over 2 million Canadians live from diabetes (http://www.diabetes.ca/Section_about/index.asp).

Insulin can be unavailable for many reasons:

1. Destruction of the beta cells in the Islets of Langerhans which is believed to involve a genetically transmitted autoimmune problem.

A viral infection either directly destroys beta cells or the resulting autoimmune reaction is responsible.

In either case - no beta cells, means no insulin.

These patients are dependent upon insulin for life, and without it will develop ketosis or ketoacidosis and die. More about this later.

Since most, but not all, of these patients are young at onset, this type of diabetes was formerly referred to as "juvenile diabetes". More appropriately, it is now called insulin dependent diabetes mellitus (IDDM).

2. Resistance to normal or elevated serum insulin levels. Predisposition to this process is also believed to be genetically transmitted. The defect is believed to be related to a decrease in the number of insulin receptors on the cell membrane, which reduces the ability of insulin to act at this level.

There is a correlation between overeating and the onset of this problem. These patients tend to be over 40 and overweight at onset. This accounts for the former term of "age onset diabetes". Currently, it is referred to as non-insulin dependent diabetes (NIDDM) or Type II.

Investigators believe that between 35 and 40 etiologies of this type of disease may be possible.

Management of NIDDM may consist of weight reduction, with or without the use of oral hypoglycemic agents. Insulin may be added temporarily to prevent hyperglycemia, but without it, these individuals can still survive.

3. Other conditions such as surgical removal of the pancreas, pancreatitis, pancreatic injuries and rare endocrine disorders may lead to diabetes.

The goals of management of the diabetic patient are to maintain as nearly normal levels of blood glucose as possible using current therapies. In the non-diabetic person this is achieved by homeostatic feedback mechanisms which constantly monitor and adjust blood glucose levels. Current therapy involving extensive patient education in the regulation of diet and exercise, the monitoring of blood glucose levels and administration of medication sometimes by continuous infusion (insulin pump) is still very inexact, compared with natural mechanisms.

Unfortunately, there is a relatively narrow range of "normal" blood glucose values tolerated by human tissues. Just as glucose deficits within the cell are problematic, glucose excesses are, over time, toxic.

Diabetics may develop:

- Blindness
- Kidney failure
- Heart disease and stroke: diabetes is the underlying cause of 50% of all heart attacks and strokes
- Peripheral neuropathies
- Peripheral vascular disease.

HYPOGLYCEMIC EMERGENCIES

Decreased levels of blood glucose cause the patient to be symptomatic by two mechanisms:

1. SNS stimulation (beta adrenergic) which results in anxiety, trembling, tachycardia, palpitations, diaphoresis, faintness, dilated pupils, pallor.
2. CNS depression (primarily at the cortical level) resulting in weakness, headache, blurred vision, impaired judgement, confusion, lack of co-ordination, seizures and coma.

The respiratory pattern is usually normal in these patients.

Most often hypoglycemia is seen in the IDDMs who has missed a meal, over-exercised, altered insulin regime, or has an infection. Other causes can include oral hypoglycemic agents, and ethanol abuse over time (primarily due to inadequate glycogen stores and defective gluconeogenesis).

Prolonged or recurrent hypoglycemia can result in brain damage (often in the areas affecting personality) and/or death. The damage is irreversible.

The goals of therapy are directed at rapidly restoring blood glucose levels to the normal range and replacing depleted glycogen stores.

HYPERGLYCEMIC EMERGENCIES

Hyperglycemic states involve increased levels of blood glucose which may be present in some diabetics. However, we will focus on those patients in whom hyperglycemia presents as a life-threatening emergency. These conditions are:

- diabetic ketoacidosis (DKA)
- hyperglycemic, hyperosmolar, nonketotic coma (HHNK).

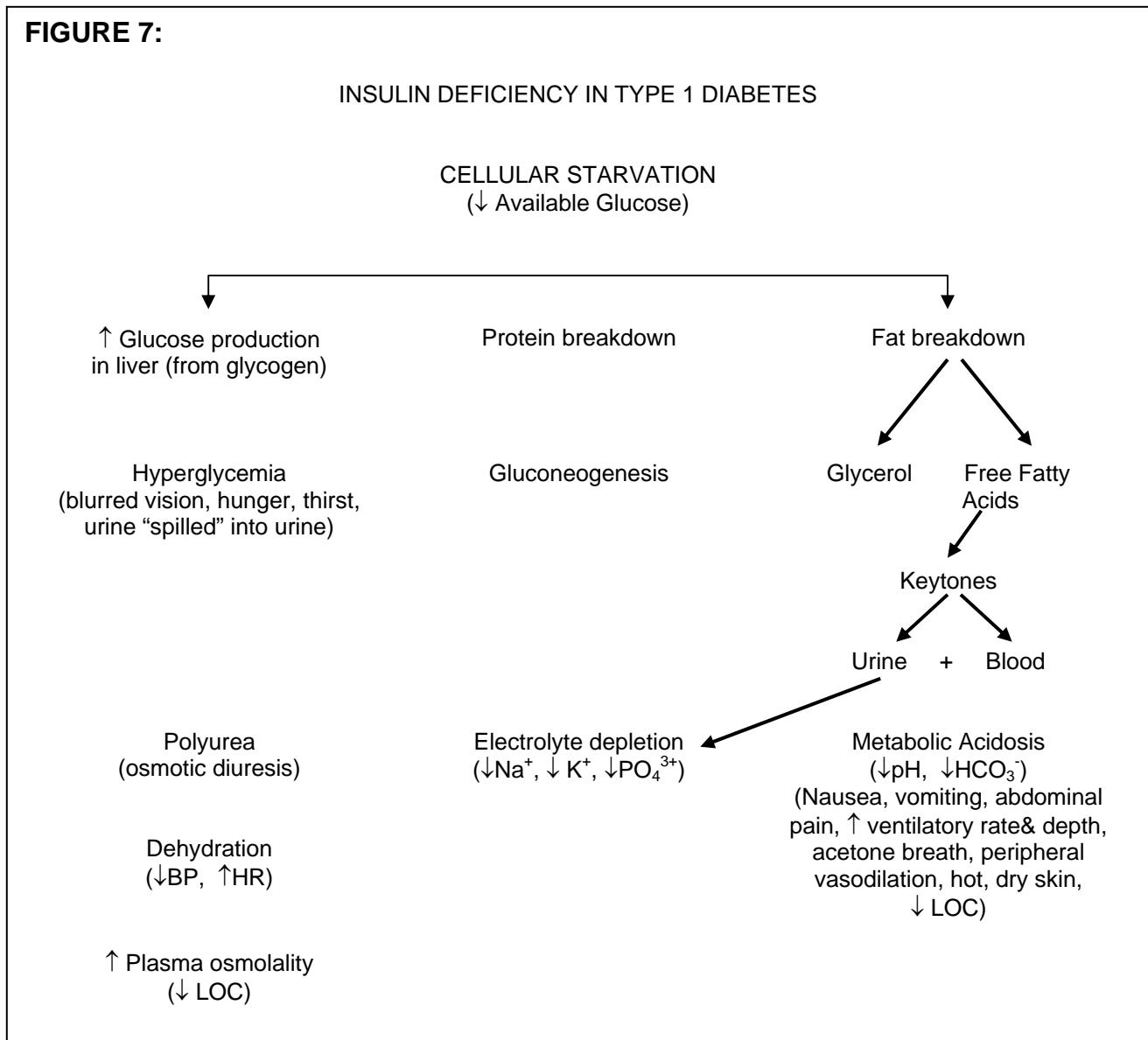
DIABETIC KETOACIDOSIS (DKA)

Increased levels of blood glucose and ketones due to a relative lack of insulin are usually, but not always, seen in IDDM. Symptoms can be attributed to an excess of glucagon, glucocorticoids, growth hormone and catecholamines usually due to stressful events, such as illness or emotional stress.

These hormones, you'll recall, are the ones associated with the body's homeostatic mechanisms in response to decreased blood glucose at the cellular level. They are the hormones of glycogenolysis, and gluconeogenesis in response to cellular starvation.

Schematically, the sequence of events is represented in Figure 7.

FIGURE 7:



It is apparent that a number of vicious cycles operate here:

1. In the absence of insulin, and the cell's inability to utilize glucose, the hyperglycemia will tend to get worse due to the glucose production from protein and fat breakdown.
2. As the serum glucose becomes very high, the kidney is unable to reabsorb the high concentration of glucose in the renal tubules. The glucose trapped in the renal tubules (in urine) results in a large volume of water being extracted (polyuria or osmotic diuresis). This osmotic diuresis also removes electrolytes such as Na⁺ and K⁺.
3. As fats are mobilized for glucose production, the glycerol portion is metabolized to glucose (causing ↑ hyperglycemia). The breakdown of the free fatty acid portion results in ketonuria, again contributing to the diuresis and electrolyte depletion.

Ketones in blood are acidic. Compensatory buffering systems (see acid/base unit) become activated and depleted. The respiratory system attempts to compensate by blowing off ketone byproducts (acetone breath) and CO₂ resulting in the hyperventilation (Kussmaul respirations) characteristic of the condition. This may be the presentation of an undiagnosed Type I diabetic.

HYPERGLYCEMIC, HYPEROSMOLAR, NONKETOTIC COMA (HHNK)

Isolated hyperglycemia without ketoacidosis is usually seen in NIDDM. NIDDMs usually have enough endogenous insulin that fats are not mobilized. They do however mobilize proteins in response to precipitating events with the resulting hyperglycemia, polyuria, electrolyte depletion and dehydration without the acidosis component. Therefore a similar sequence of events occurs as illustrated in Figure 7, but without the breakdown of fats.

The history and findings which differentiate diabetic ketoacidosis (DKA) from hyperglycemic, hyperosmolar, nonketotic coma (HHNK) are summarized in Table I.

	DKA	HHNK
Diabetic History	IDDM (TYPE I)	NIDDM (TYPE II)
May be undiagnosed diabetic	Yes	Yes
Respirations	Kussmaul (acetone odour)	Normal
Heart Rate (HR)	Elevated	Elevated
Blood Pressure (BP)	Low	Low
Level of Consciousness (LOC)	Depressed	Depressed
Abdominal Pain	Common	No

Symptoms of hyperglycemia have a slower and less dramatic onset usually over several days, than do those of hypoglycemia. They may in fact go unrecognized by the patient and relatives until they present a life-threatening emergency.

The mortality rate from hyperglycemic emergencies remains significant at 5-10%. Death can result from organ failure subsequent to hypovolemic shock, arrhythmias subsequent to cellular starvation and electrolyte depletion, or embolic events associated with underlying disease and/or increased viscosity (thickness) of plasma.

Therapy is directed at rehydration, restoration of electrolytes and pH levels, reduction of blood glucose levels to within normal limits and identification of underlying precipitating event.

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SELF-ASSESSMENT: THE PANCREAS

MARKS

- [3] 1. Describe the location of the pancreas in relation to the stomach, spleen and duodenum.

- [2] 2. Define:
Gluconeogenesis

Glycogenolysis

- [4] 3. The pancreas produces two hormones; these are:
(a) _____ which (raises/lowers) _____ blood glucose levels and (b)
which (raises/lowers) _____ blood glucose levels.

- [1] 4. Other factors which lower blood glucose are
_____ and _____.

[1] 5. Secretion of insulin is mainly controlled by _____.

6. Compare Type 1 and Type II diabetes by completing this chart.

	TYPE I	TYPE II
[1] Common abbreviation	_____	_____
[1] Usual age of onset	_____	_____
[3.5] Controlled by	1. _____ 2. _____ 3. _____	1. _____ 2. _____ 3. _____ 4. _____
[1] Ketoacidosis prone?	_____	_____
[1] Non-Ketotic Hyperosmolar coma prone?	_____	_____

[1] 7. The mechanism of action of insulin is:

[.5] 8. In the absence of insulin brain cells (are/are not) _____ able to utilize glucose.

[1] 9. Diabetes is a result of _____.

[1] 10. Many diabetic patients have major illnesses secondary to diabetes. Name two of these illnesses that you are likely to encounter in the field.

[1.5] 11. In response to cellular starvation the body breaks down _____, _____ and _____.

[.5]. 12. The cause of ketoacidosis is related to the breakdown of _____.

[2] 13. The conditions resulting from ketoacidosis are primarily (a) _____ acidosis, _____ and (c) _____ depletion. The (d) _____ pain may serve to mislead the examiner.

14. Differentiate between the important features of hypoglycemia and pathologic hyperglycemia below.

	HYPOGLYCEMIA	HYPERGLYCEMIA
[1] Onset of symptoms	_____	_____
[1] Skin condition	_____	_____
[1] Respirations	_____	_____
[1] Onset of coma	_____	_____

30 TOTAL

**ADVANCED LIFE SUPPORT
PRECOURSE
ENDOCRINE SYSTEM**

SELF-ASSESSMENT ANSWERS: THE PANCREAS

1. Behind the stomach, resting on a loop of duodenum on the right, touching the hilum of the spleen on the left.

2. Gluconeogenesis - production of glucose from the breakdown of proteins and fats in the liver.

Glycogenolysis - production of glucose from the breakdown of glycogen in the liver.

3. a) Insulin, lowers
b) Glucagon, raises

4. Exercise, starvation

5. Blood glucose levels (passing through the Islets of Langerhans).

	TYPE I	TYPE II
Common abbreviation	IDDM	NIDDM
Usual age of onset	younger	older
Controlled by	1. insulin 2. diet 3. exercise 4. +/- insulin	1. diet 2. exercise 3. + or - oral hypoglycemics
Ketoacidosis prone?	Yes	not usually
Non-Ketotic Hyper-osmolar coma prone?	Not usually	Yes

7. Facilitating glucose transport across the cell membrane.

8. Are

9. Lack of insulin (either relative or absolute)
10. (Arteriosclerotic) heart disease and stroke Renal disease
11. Glycogen, protein, fats
12. Fat
13.
 - a) Metabolic
 - b) Dehydration (osmotic diuresis)
 - c) Electrolyte
 - d) Abdominal

14.	HYPOGLYCEMIA	HYPERGLYCEMIA
Onset of symptoms	Rapid over minutes to hours	Gradual over hours to days
Skin condition	Pale, wet	Hot, dry
Respirations	Normal	Rapid, deep (Kussmaul) in DKA Normal in HHNK
Onset of coma	Rapid	Much longer,

**ADVANCED LIFE SUPPORT
PRECOURSE
THE NERVOUS SYSTEM**

EVALUATION

Upon completion of this module, please fill in and return this form to your base hospital co-ordinator.

Your comments will help to ensure that this unit is a useful learning module. Please indicate any problems that you may have encountered. All suggestions for improvement are welcomed.

1. How long did it take to complete this module? Please estimate.

Reading	_____	hours
Self assessment	_____	hours
Total time	_____	hours

2. Were the objectives of the module clearly stated?

yes no
If no, please comment.

3. Did you see any of the resource materials?

yes no
If yes, which items

Were they helpful? _____

4. Were the reference notes adequate?

yes no
If no, please comment.

5. Were the reference notes easy to follow?

yes no

If no, please comment.

6. Were the examples provided satisfactory?

yes no

If no, please comment.

7. Were any of the self-assessment questions poorly worded?

yes no

If yes, please specify.

1. Was the level of the module satisfactory for your program of study?

yes no

If no, please comment.

Base Hospital _____

9. General comments or suggested improvements.