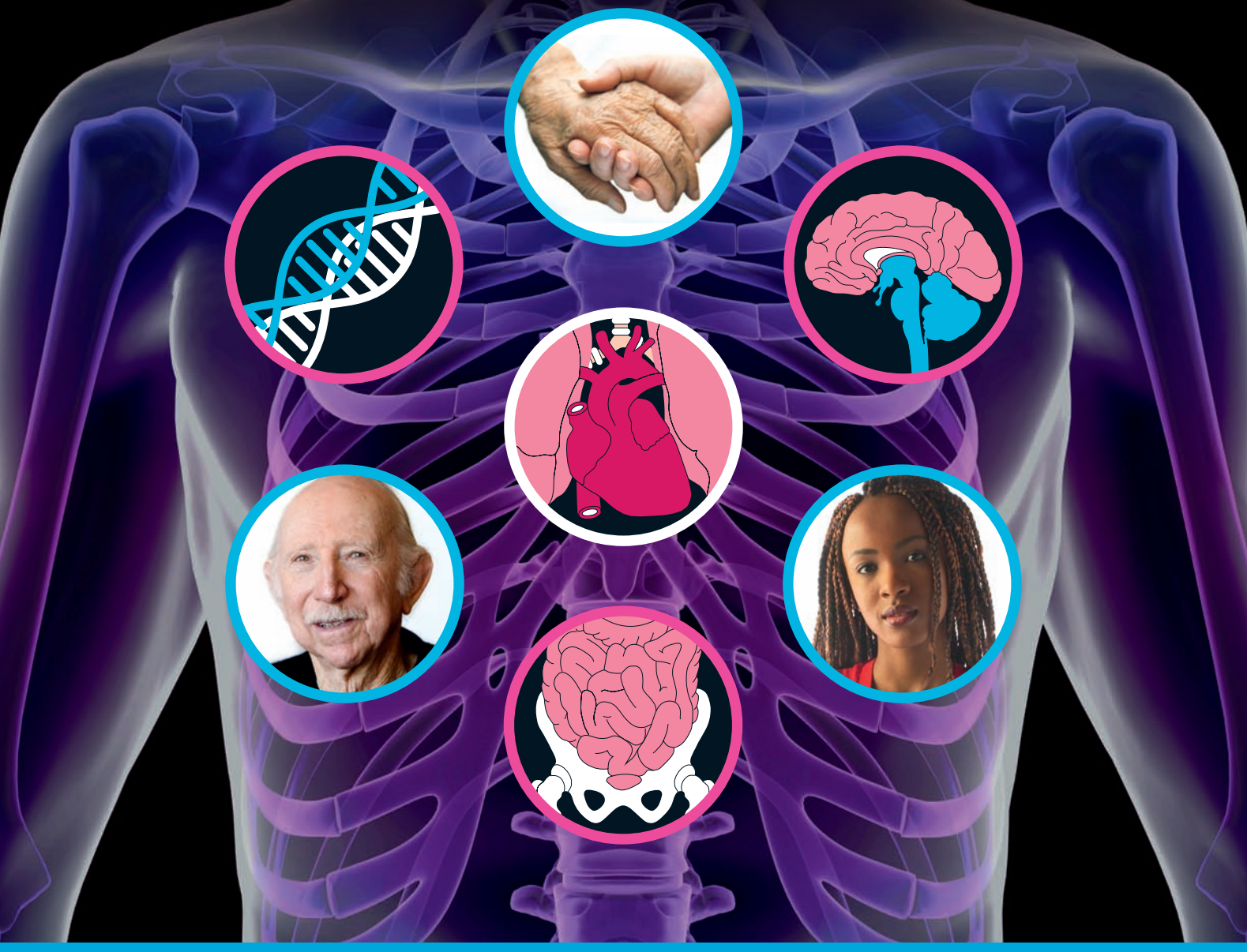
LeMone & Burke's

# Adult Nursing

## Acute and Ongoing Care

Ian Peate • Muralitharan Nair • Laureen Hemming • Karen Wild



ALWAYS LEARNING

PEARSON

LeMone & Burke's

# Adult Nursing

Visit the *Adult Nursing* Companion Website at **www.pearsoned.co.uk/lemone** to find valuable **student** learning material including:

- Interactive Case Studies
- Multiple Choice Questions
- Weblinks
- Searchable Glossary
- Flashcards

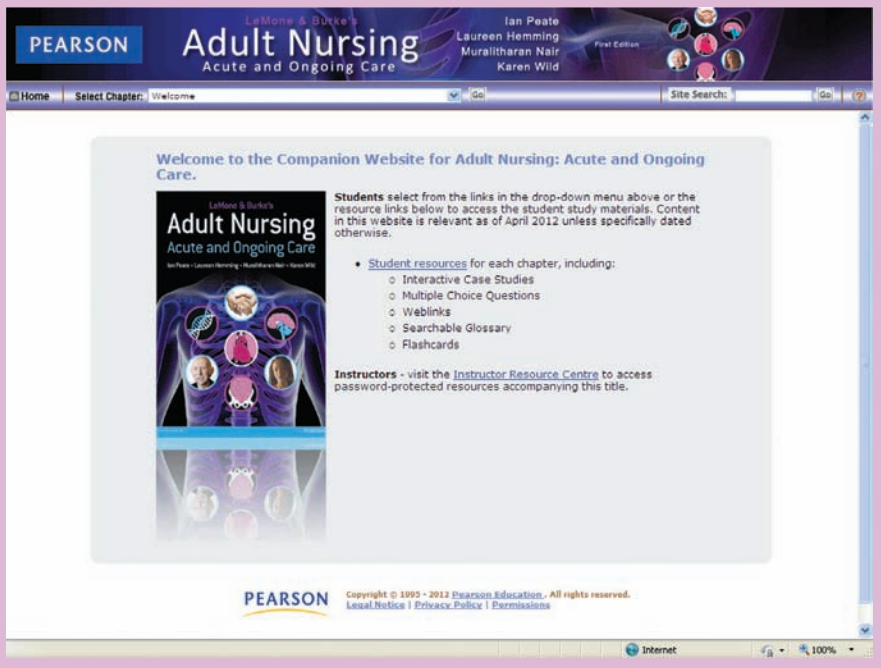


**Table 6.1** Organs, hormones, functions and feedback mechanisms of the endocrine system

| Endocrine organ | Hormone secreted | Target organ and their functions |
|---|---|---|
| Thyroid gland | Thyroid hormone (TH): thyroxin ($T_4$) is the major hormone secreted by the thyroid gland. It is converted to tri-iodothyronine ($T_3$) at the target tissues. | Maintains metabolic rate and growth and development of all tissues. $T_3$ and $T_4$ are secreted in response to thyroid-stimulating hormone (TSH). |
| | Calcitonin | Maintains serum calcium levels by decreasing bone resorption (the loss of bone or substance by disease) and decreasing resorption of calcium in the kidneys whenever levels of plasma calcium are elevated. |
| Parathyroid gland | Parathyroid hormone (PTH) | Maintains serum calcium levels by stimulating bone resorption and formation and by stimulating kidney resorption of calcium in response to falling levels of plasma calcium. |
| Adrenal cortex | Mineralocorticoids (e.g., aldosterone) | Promote kidney tubule reabsorption of sodium and water and excretion of potassium in response to elevated levels of potassium and low levels of sodium, thereby increasing blood pressure and blood volume. |
| | Glucocorticoids (e.g., cortisol) | Help regulate metabolism of carbohydrates, fats and proteins. Activate anti-inflammatory responses to stressors. Low cortisol levels stimulate hypothalamic secretion of corticotropin-releasing hormone (CRH), which stimulates the anterior pituitary gland to release ACTH, which in turn stimulates the adrenal cortex to secrete cortisol. |
| | Gonadocorticoids (androgens and small amounts of oestrogen and progesterone) | The quantity of sex hormones produced here is small, and the mechanism is not well understood. |
| Adrenal medulla | Catecholamine (epinephrine and norepinephrine) | Stimulate the heart, constrict blood vessels, inhibit visceral muscles, dilate bronchioles, increase respiration and metabolism and promote hyperglycaemia. Secreted in response to physical or psychological stress. |
| Anterior pituitary (adenohypophysis) | Growth hormone (GH) | Promotes growth of body tissues by enhancing protein synthesis and promoting use of fat for energy and thus conserving glucose. Release is stimulated by growth hormone releasing hormone (GHRH) in response to low GH levels, **hypoglycaemia**, increased amino acids, low fatty acids and stress. |

- Oxytocin induces contraction of the smooth muscles in the reproductive organs. In women, oxytocin stimulates the myometrium of the uterus to contract during labour. It also induces milk ejection from the breasts.

## THYROID GLAND

The thyroid gland is anterior (in the front) to the upper part of the trachea and just inferior (below) to the larynx. This butterfly-shaped gland has two lobes connected by a structure called the isthmus.

The glandular tissue consists of follicles filled with a jelly-like colloid substance called thyroglobin, a glycoprotein–iodine complex. Cells within the follicles secrete thyroid hormone (TH), a general name for two similar hormones: thyroxin ($T_4$) and tri-iodothyronine ($T_3$). The primary role of thyroid hormones in adults is to increase metabolism. TH secretion is initiated by the release of TSH by the pituitary gland and is dependent on an adequate supply of iodine. The thyroid gland also secretes calcitonin, a hormone that decreases excessive levels of calcium in the blood by slowing the calcium-releasing activity of bone cells, serves as a marker for sepsis (infection) and is believed to be a mediator of inflammatory responses.

## PARATHYROID GLANDS

The parathyroid glands (usually four to six in number) are embedded on the posterior surface of the lobes of the thyroid gland. They secrete parathyroid hormone (PTH), or parathormone. When calcium levels in the plasma fall, PTH secretion increases. PTH also controls phosphate metabolism. It acts

primarily by increasing renal excretion of phosphate in the urine, by decreasing the excretion of calcium, and by increasing bone reabsorption to cause the release of calcium from bones. Normal levels of vitamin D are necessary for PTH to exert these effects on bone and kidneys.

## ADRENAL GLANDS

The two adrenal glands are pyramid-shaped organs that sit on top of the kidneys. Each gland consists of two parts, which are distinct organs: an inner medulla and an outer cortex.

The adrenal medulla produces two hormones (also called catecholamine): epinephrine (adrenaline) and norepinephrine (noradrenaline). These hormones are similar to substances released by the sympathetic nervous system and thus are not essential to life. Epinephrine increases blood glucose levels and stimulates the release of ACTH from the pituitary; ACTH, in turn, stimulates the adrenal cortex to release glucocorticoids. Epinephrine also increases the rate and force of cardiac contractions; constricts blood vessels in the skin, mucous membranes and kidneys; and dilates blood vessels in the skeletal muscles, coronary arteries and pulmonary arteries. Norepinephrine increases both heart rate and the force of cardiac contractions and vasoconstricts throughout the body.

The adrenal cortex secretes several hormones, all corticosteroids. They are classified into two groups: mineralocorticoids and glucocorticoids. These hormones are essential to life. The release of the mineralocorticoids is controlled primarily by an enzyme called renin. When a decrease in blood pressure or sodium is detected, specialised kidney cells release renin to act on a substance called angiotensinogen (plasma protein produced by the liver). Angiotensinogen is modified by renin and other enzymes to become angiotensin II, which stimulates the release of aldosterone from the adrenal cortex. Aldosterone prompts the distal tubules and the collecting ducts of the nephron to reabsorb increased amounts of water and sodium back into the circulating blood to increase circulating blood volume and pressure.

The glucocorticoids include cortisol and cortisone. These hormones affect carbohydrate metabolism by regulating glucose use in body tissues, mobilising fatty acids from fatty tissue and shifting the source of energy for muscle cells from glucose to fatty acids. Glucocorticoids are released in times of stress. An excess of glucocorticoids in the body depresses the inflammatory response and inhibits the effectiveness of the immune system.

## PANCREAS

The pancreas, located behind the stomach between the spleen and the duodenum, is both an endocrine gland (producing hormones) and an **exocrine** gland (producing digestive enzymes). The endocrine cells of the pancreas produce hormones that regulate carbohydrate metabolism. They are clustered in bodies called pancreatic islets (or islets of Langerhans) scattered throughout the gland. Pancreatic islets have at least four different cell types:

- Alpha cells produce glucagon.
- Beta cells produce insulin.
- Delta cells secrete somatostatin.
- F cells secrete pancreatic polypeptide.

## GONADS

The gonads are the testes in men and the ovaries in women. These organs are the primary source of steroid sex hormones in the body. The hormones of the gonads are important in regulating body growth and promoting the onset of puberty. In men, androgens (primarily testosterone) produced by the testes maintain reproductive functioning and secondary sex characteristics and promote the production of sperm. In women, the ovaries secrete oestrogens and progesterone to maintain reproductive functioning and secondary sex characteristics. Progesterone also promotes the growth of the lining of the uterus to prepare for implantation of a fertilised ovum.

### An overview of hormones

Although we rarely think about the glands of the endocrine system, the hormones they release influence almost every cell, organ and function of our bodies. The endocrine system is instrumental in regulating mood, growth and development, tissue function and metabolism, as well as sexual function and reproductive processes. So what are hormones?

Hormones are chemical messengers secreted by the endocrine organs and transported throughout the body, where they exert their action on specific cells called target cells. Hormones do not cause reactions directly but rather regulate tissue responses. They may produce either generalised effects or local effects.

Hormones are transported from endocrine gland cells to target cells in the body in one of four ways:

- Endocrine glands release most hormones, including TH and insulin, into the bloodstream.
- Neurons release some hormones, such as epinephrine, into the bloodstream.
- The hypothalamus releases its hormones directly to target cells in the posterior pituitary by nerve cell extension.
- With the paracrine ('para' = near; group of local hormones that work on nearby cells) method, released messengers diffuse through the interstitial fluid. This method of transport involves a number of hormonal peptides that are released throughout various organs and cells and act locally. An example is endorphins, which act to relieve pain.

Hormones that are released into the bloodstream circulate as either free, unbound molecules or as hormones attached to transport carriers. Peptide and protein hormones (such as insulin) circulate unbound, while steroid and thyroid

hormones are carried by specific transport carriers **synthesised** by the liver. Hormone receptors are complex molecular structures, located on or inside target cells. They act by binding to specific receptor sites located on the surfaces of the target cells. These receptors recognise a specific hormone and translate the message into a cellular response. The receptor sites are structured so that they respond only to a specific hormone; for example, receptors in the thyroid gland are responsive to TSH but not to LH.

Hormone levels are controlled by the pituitary gland and by feedback mechanisms. Although most feedback mechanisms are negative, a few are positive. Negative feedback is controlled much as the thermostat in a house regulates temperature. Sensors in the endocrine system detect changes in hormone levels and adjust hormone secretion to maintain normal body levels. When the sensors detect a decrease in hormone levels, they begin actions to cause an increase in hormone levels; when hormone levels rise above normal, the sensors cause a decrease in hormone production and release. For example, when the hypothalamus or anterior pituitary gland senses increased blood levels of TH, it releases hormones, causing a reduction in the secretion of TSH, which in turn prompts a decrease in the output of TH by the thyroid gland (see Figure 6.3).

In positive feedback mechanisms, increasing levels of one hormone cause another gland to release a hormone. For example, the increased production of oestradiol (a female ovarian hormone) during the follicular stage of the menstrual cycle in turn stimulates increased FSH production by the anterior pituitary gland. Oestradiol levels continue to increase until the ovarian follicle disappears, eliminating the source of the stimulation for FSH, which then decreases.

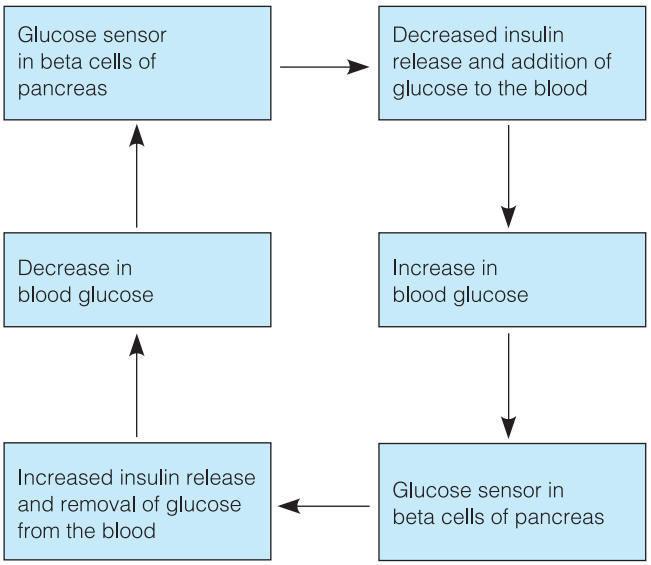



**Figure 6.3** Negative feedback.

Stimuli for hormone release may also be classified as hormonal, **humoral** or neural (Figure 6.4 A, B, C). In hormonal release, hypothalamic hormones stimulate the anterior pituitary to release hormones. Fluctuations in the serum (fluid portion of the blood) level of these hormones in turn prompt other endocrine glands to release hormones. In the case of humoral release, changes in the serum levels of certain electrolytes and nutrients stimulate specific endocrine glands to release hormones to bring these levels back to normal. However, in neural release, nerve fibres stimulate the release of hormones.

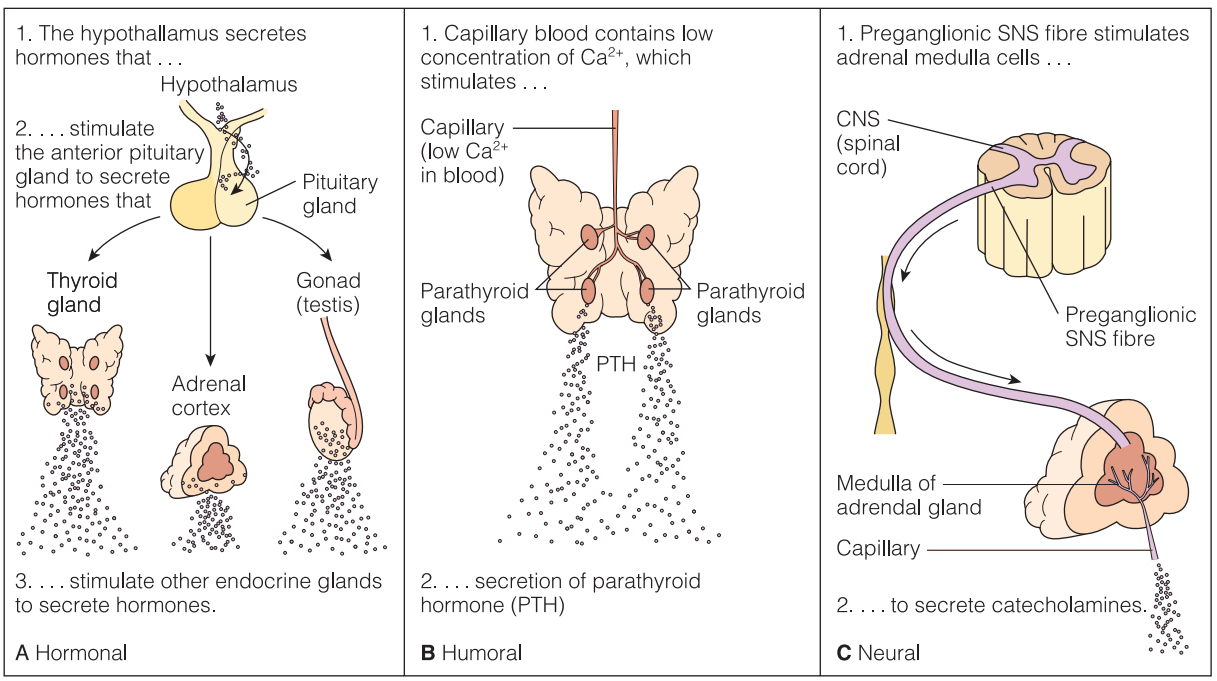



**Figure 6.4** Examples of three mechanisms of hormone release.

# DISORDERS OF THE ENDOCRINE SYSTEM

Now that we have looked at the anatomy and physiology of the endocrine system, let us consider some of the disorders that you might come across in practice such as hyper- and **hypothyroidism**, diabetes mellitus and disorders of the pituitary gland.

## FAST FACTS

### Hyperthyroidism

About 2 in 100 women and 2 in 1000 men develop **hyperthyroidism** at some stage of their life and it can occur at any age.

Source: Patient UK 2010.

## The person with hyperthyroidism

Hyperthyroidism (also called thyrotoxicosis) is a disorder caused by excessive secretion of TH. Because the primary function of TH is to increase metabolism and protein synthesis, hyperthyroidism affects all major organ systems of the body. The increase in metabolic rate and the alterations in cardiac output, peripheral blood flow, oxygen consumption and body temperature are similar to those found in increased sympathetic nervous system activity. The effects of hyperthyroidism are the result of increased circulating levels of TH. This hormonal excess increases the metabolic rate and heightens the sympathetic nervous system's physiologic response to stimulation.

### Pathophysiology of hyperthyroidism

Hyperthyroidism results from many different factors, including autoimmune stimulation (as in Graves' disease), excess secretion of TSH by the pituitary gland, thyroiditis, neoplasm (such as toxic multinodular goitre) and an excessive intake of iodine-containing drugs such as amiodarone. The most common aetiologies of hyperthyroidism are Graves' disease and toxic multinodular **goitre**.

### Signs and symptoms of hyperthyroidism

Signs and symptoms of hyperthyroidism include:

- increased appetite yet with weight loss as a result of increased metabolism;
- increased **peristalsis** and diarrhoea;
- heat intolerance;
- insomnia;
- palpitations;
- increased sweating;
- skin is smooth and warm;
- hair becomes fine;
- hair loss in the scalp, eyebrow, axilla.

For multisystem effects of hyperthyroidism see Figure 6.5.

### Graves' disease

Graves' disease results from overactivity of the thyroid gland. It is thought to be an autoimmune disease, where the immune system produces an antibody that stimulates the cells of the thyroid gland to secrete an excessive amount of thyroid hormones. The disease is seen five times more often in women than in men and occurs most frequently between the ages of 20 and 40. It has a strong hereditary component; when one identical twin has Graves' disease, the other twin will have it 25% of the time. Factors that can trigger the onset of Graves' disease include stress, smoking, radiation to the neck and infectious organisms such as viruses.

The ophthalmopathy (disease of the eye) of Graves' disease is manifested as **proptosis** and visual dysfunction. Proptosis (forward displacement) of the eye occurs in about one-third of cases. The forward protrusion of the eyeballs (exophthalmoses) results from an accumulation of inflammation by-products in the retro-orbital tissues. Often the sclera is visible above the iris. The upper lids are often retracted, and the person has a characteristic unblinking stare. Graves' disease can have an effect on many parts of the body such as the nervous system, eyes, skin, hair/nails, lungs, digestive system, muscles/bones and reproductive system.

### Toxic multinodular goitre

Toxic multinodular goitre is a tumour characterised by small, discrete, independently functioning nodules in the thyroid gland tissue that secrete excessive amounts of TH. It is not known how these nodules grow or become independent, but a genetic mutation of follicle cells is suspected. Elevated TH levels result in signs and symptoms of hyperthyroidism; however, they are slower to develop and neither ophthalmopathy nor dermopathy develop. The person with this type of hyperthyroidism is usually a woman in her 60s or 70s who has had goitre for a number of years.

## INTERDISCIPLINARY CARE FOR HYPERTHYROIDISM

Hyperthyroidism results from a raised level of thyroid hormone (TH). The treatment of hyperthyroidism focuses on reducing the production of TH by the thyroid gland. Treatment is usually effective. The treatment to reduce the thyroxine level include: medicines, radioiodine and surgery. Long-term follow-up is important, even after successful treatment.

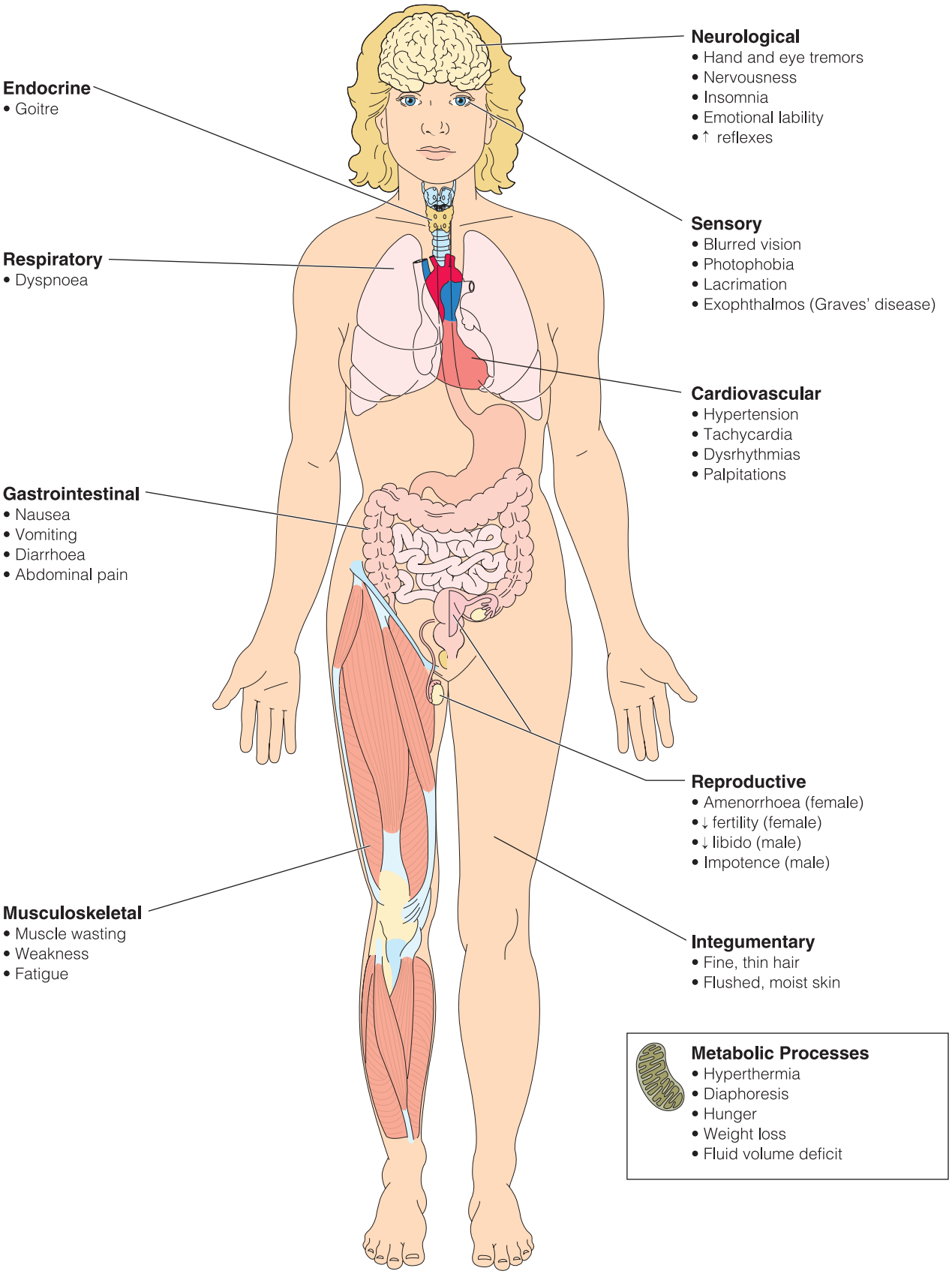



**Figure 6.5** Multisystem effects of hyperthyroidism.

## Medication

Hyperthyroidism is treated by administering antithyroid medications, such as carbimazole 30–40 mg/day, which reduce TH production. Because antithyroid drugs do not affect the release or activity of hormone that is already formed, therapeutic effects may not be seen for several weeks. The person will continue to take the medication until euthyroid state is achieved. To rapidly decrease the cardiovascular symptoms such as **hypertension** associated with hyperthyroidism, propanolol (Inderal), a beta blocker, may form part of initial treatment.

## Surgery

Some hyperthyroid persons have such enlarged thyroid glands that pressure on the oesophagus or trachea causes swallowing or breathing problems respectively. In these cases, removal of all or part of the gland is indicated. In partial **thyroidectomy** enough of the gland is left in place to produce an adequate amount of TH. However, in total thyroidectomy the person then requires lifelong hormone replacement therapy.

## Radioactive therapy

Radioactive iodine-131 ($^{131}I$) is absorbed and is concentrated in the thyroid gland. Iodine-131 damages or destroys thyroid cells resulting in less production of the thyroid hormone. The drug is administered as a single capsule and the patient is advised not to eat for approximately four hours in order for the effective absorption of the iodine. Patients are also advised to drink approximately 2–3 L of fluid over the 24-hour period and to urinate frequently to get rid of the filtrated radioactive iodine promptly. This treatment is offered to patients who may not be suitable for surgery.

## NURSING CARE PLAN The person having a partial thyroidectomy

Review Chapter 1 for routine pre- and post-operative care.

### *Pre-operative care*

- Persons are prepared in accordance with the local policy and guidelines.
- Teach the person to support the neck by placing both hands behind the neck when sitting up in bed, while moving about and while coughing. *Placing the hands behind the neck provides support for the suture line.*
- Answer questions and allow time for the person to verbalise concerns. *Because the incision is made at the base of the throat, persons (especially women) are often concerned about their appearance after surgery.* Explain that the scar will eventually be only a thin line and that jewellery or scarves may be used to cover the scar.
- Teach the person to expect hoarseness due to generalised swelling at the suture line. *This is expected to diminish with healing and is not caused by laryngeal nerve damage*.
- Provide information about pain control post-operatively *to lessen the person's anxiety about pain control*.
- Check with local protocols with regard to safe preparation of a person for partial thyroidectomy.
- Document all care in accordance with the NMC guidelines for records and record keeping.

### *Post-operative care*

- Nurses should monitor the person's airway half hourly as a result of surgery to the neck area. Ensure that the airway is not obstructed and that the person is sitting in an upright position with the neck supported by pillows. *The support should provide comfort for the person, reduce the strain on the suture line and minimise the pain level*.
- Monitor the wound site for any haemorrhage following surgery. Record blood pressure, respiration and heart rate half hourly and report any changes in the vital signs immediately to facilitate prompt action. *An increase in heart rate and a drop in blood pressure may indicate a haemorrhaging wound which should be reported immediately for prompt action*.
- Observe for signs such as **stridor**, difficulty in swallowing, noisy breathing which could indicate laryngeal nerve damage from surgery. *One of the possible complications of thyroidectomy is damage to the laryngeal nerve during surgery. This nerve damage could result in vocal cord spasm and paralysis of the larynx which could suppress respiration*.
- Observe for signs of tetany resulting from accidental removal of the parathyroid glands: tingling of toes, fingers and lips; muscular twitches; positive **Chvostek's sign** and **Trousseau's sign**. *A sudden decrease in calcium level could result in tetany and if untreated could be fatal for the person.*
- Document all care provided in accordance with the Nursing and Midwifery Guidelines for records and record keeping.