The patient with acute endocrine problems

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~~The patient with acute endocrine problems~~Acute Managing acute endocrine disorders~~endocrine problems~~

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Aims

The aim of this chapter is to identify key roles and functions of the endocrine system~~,~~ to appreciate how altered physiology can disrupt homeostasis and cause a medical emergency. The chapter will discuss the more commonly experienced endocrine disorders and use case studies to explore the nurse's role in recognising and responding appropriately to patients with acute endocrine problems.

Objectives

After reading this chapter you will be able to:

- Recognise the major organs involved in endocrine function.
- Identify the roles of endocrine organs in maintaining homeostasis.
- Identify the common endocrine disorders and differentiate between patient presentations.

- Understand the use of assessment and therapeutic interventions to maximise the health status of patients with endocrine disorders.
- Describe the responsibilities of the nurse in assessing, monitoring and managing patients with acute endocrine disorders.

Introduction

Although most endocrine emergencies occur rarely, diabetic emergencies are witnessed by most nurses caring for patients within the hospital setting. It is important, therefore, that nurses have insight into glucose metabolism and control and into their responsibilities in monitoring and managing this aspect of patient care. These principles are paramount not only with regards to an emergency, but routinely, as the monitoring and control of blood glucose is frequently part of patient management. Uncommon presentations of endocrine malfunction that can occur acutely include thyrotoxicosis and adrenal insufficiency. All these can present with severe patient deterioration, so a basic understanding of the endocrine system and associated hormones is required.

Applied physiology

Overview of the endocrine system

The endocrine, nervous and immune system work in harmony to regulate the internal and external environment of human beings. The endocrine system is composed of various glands that are widely dispersed throughout the body, and they secrete chemical messengers called hormones (Brashers and Huether 2017). The major endocrine glands are the pituitary, thyroid, parathyroid, thymus, pancreas, adrenal, ovaries and testes. The endocrine structure and function witness some changes due to the ageing process (Brashers and Huether 2017). In contrast to the exocrine glands,

which discharge their products via ducts into the external environment, e.g. pancreatic juice, the endocrine glands, which are ductless, synthesise and release hormones directly into the circulation. Figure 11.1 identifies the position of the major endocrine glands and the hormones they produce.

Hormones have a wide variety of functions, and their effects can last for minutes, hours or even days. Hormones are composed predominantly from amino acids or sometimes cholesterol, as with the steroid hormones. They affect organs that have hormone-specific receptors, and these are known as target cells for that hormone. Circulating hormones activate the target cells directly, and the degree of response is related to total blood concentration. The more hormone circulating, the greater the target cell activity, though some hormones can have powerful effects at a very low concentration. Hormones are broken down rapidly either within their target cells or by the liver and kidney. The functions of the endocrine organs that may be involved in a medical emergency are summarised in Table 11.1.

The hormones have a wide influence on body processes, such as reproduction, maintenance of fluid and electrolyte balance, cellular growth and metabolism (Dixon and Salamanson 2006). The hormonal regulation helps to initiate adaptive responses to emergency demands and therefore assist to maintain an optimum internal environment. The endocrine glands respond to specific signals, and hormone production by each endocrine gland is finely balanced. The regulation of hormone release is controlled by negative and positive feedback. Both feedback systems help to maintain balance between physiological production and bodily demand (Lawal 2008). The negative feedback control loops are inhibitory (oppose the change) while positive feedbacks are stimulatory (potentiate the change). Most homeostatic feedback systems are negative feedback, e.g. blood glucose control, and an example of a positive feedback mechanism is uterine contraction during childbirth. Endocrine disorder is characterised by either underproduction or overproduction, and common endocrine imbalance

are discussed below.

Structure and function of the endocrine glands

The hypothalamic-pituitary axis (HPA) forms the structural and functional basis to integrate endocrine and neurologic systems, which is referred to the neuroendocrine system. The hypothalamus is connected to the pituitary via the pituitary stalk and regulates the two lobes by neural and hormonal pathways. The HPA produces several hormones that affect other body functions, such as the thyroid and adrenal gland.

The hypothalamus

The hypothalamus, located in the diencephalon, is an important link between the nervous system and the endocrine system. It is involved in many of the normal physiological mechanisms that contribute to homeostasis, such as temperature control, thirst and hunger reflexes. The hypothalamus controls the pituitary gland, which, in its turn, regulates most of the other glands in the endocrine system. Figure 11.2 demonstrates the extensive influence of the hypothalamus, the anterior and posterior pituitary gland and their target organs.

The pituitary

The pituitary gland has an important role in the control of other glands. It has two lobes, anterior and posterior, producing eight hormones in total: these are summarised in Table 11.1.

The **anterior pituitary** is under the control of the hypothalamus, but also acts independently. The hormones most likely to be related to a medical emergency are:

- Adrenocorticotrophic hormone (ACTH).
- Thyroid stimulating hormone (TSH).

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The anterior pituitary gland produces adrenocorticotrophic hormone (ACTH) in response to corticotrophin-releasing hormone (CRH) released by the hypothalamus under the neural influence of the sympathetic nervous system. ACTH causes the adrenal cortex to secrete glucocorticoid hormones such as cortisol (hydrocortisone). Glucocorticoid receptors are widely present in most body tissues. The major functions of cortisol are:

- Increased gluconeogenesis.
- Inhibition of glucose utilisation.
- Fatty acid mobilisation and catabolism by muscle cells.
- Modification of the body's response to injury.

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These represent a metabolic response to stress and oppose the action of insulin.

The anterior pituitary produces thyroid-stimulating hormone (TSH) in response to thyrotropin-releasing hormone (TRH) from the hypothalamus. TSH targets the thyroid gland, resulting in rising levels of thyroid hormones (T4 thyroxine, T3 triiodothyronine). Thyroid hormones control cell metabolism and growth. The posterior lobe of the pituitary is an extension of the hypothalamus and contains hypothalamic neurones that are specialised to secrete hormones rather than neuro-transmitters. The hormone most likely to be related to a medical emergency is antidiuretic hormone (ADH). Antidiuretic hormone (ADH), also known as vasopressin, is made by the hypothalamus, but is actually secreted from the posterior pituitary. It is released in response to several different stimuli, but most importantly to a change in the solute concentration of the blood or a change in blood pressure. Osmoreceptors are situated in the hypothalamus and respond to the changes in tonicity (effective osmolality) of extracellular fluid. If the tonicity rises, then ADH release is stimulated, and the kidneys will increase reabsorption of water, resulting in concentrated urine. If the extracellular fluid tonicity falls, then ADH release is inhibited, leading to reduced water reabsorption resulting in dilute urine. Osmoreceptors will respond to a change in

tonicity of around 2%. Baroreceptors are situated in the right atrium and carotid sinus. They respond to changes in intravascular volume. A reduced circulating volume will result in increased ADH release, and an increased circulating volume will inhibit ADH release. Baroreceptors require a 10% change in circulating volume before they respond. In some circumstances, baroreceptors can override osmoreceptors because the control mechanism will attempt to maintain intravascular volume at the expense of normal extracellular fluid osmolality.

The thyroid gland

The thyroid gland is composed of two lobes joined by an isthmus. It is located in the lower part of the neck anterior to the trachea, inferior to the thyroid cartilage (see Figure 11.3) and has an extensive arterial blood supply. When viewed under the microscope, the thyroid gland is composed of closely packed follicles which comprise epithelial cells enclosing a colloid-filled space. These functional units synthesise, store and secrete thyroid hormones. Thyroid cells form the wall of each follicle, and these cells enlarge as their metabolic activity increases. This accounts for the gland becoming visible (goitre) in certain thyroid disorders. In addition to supporting cells, the thyroid also contains C cells which synthesise calcitonin.

Two pairs of parathyroid glands are embedded in the posterior surface of the thyroid gland (see Figure 11.3). The thyroid makes and stores thyroid hormones (T3 and T4), and it is able to hold up to a 100 days' supply. Iodine is necessary for the production of thyroid hormones. Thyroid hormones affect virtually every organ in the body and increase metabolic rate. The parathyroid glands produce parathyroid hormone (PTH), which regulates serum calcium levels. PTH secretion is stimulated when ionised serum calcium falls. This hormone influences the bones and the kidneys, leading to restoration of normal calcium levels (bone resorption and increased renal tubular calcium reabsorption, respectively).

Effects of thyroxine include:

- Stimulates basal metabolic rate, resulting in increased oxygen consumption and heat production (thermogenesis).
- CNS and cardiovascular sensitivity to catecholamines, e.g. increased heart rate and contractility.
- Enzyme synthesis, which promotes protein, fat and carbohydrate metabolism.
- Growth and development, e.g. of the nervous system.

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PTH is one of the principal hormones that control mineral metabolism, the others being vitamin D and calcitonin. It is important that a constant ionised calcium concentration is maintained in the extracellular fluid, as key physiological functions, e.g. bone mineralisation, neuromuscular excitability, blood coagulation and cell membrane integrity, are reliant upon this state.

Most hormone production is affected by negative feedback loops, whereby the initial hormone producer reacts to subsequent levels of hormones produced by the target organ. The regulation of the production of thyroid hormone is a good example of a negative feedback loop (see Figure 11.4).

Adrenal glands

The adrenal glands are a pair of small glands situated on top of the kidneys. They are split into two regions:

- Adrenal cortex.
- Adrenal medulla.

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The adrenal cortex secretes many steroid hormones, collectively known as corticosteroids. In addition to the glucocorticoids (e.g. cortisol), the other major groups of adrenocortical hormones

include mineralocorticoids (e.g. aldosterone) and androgenic hormones (e.g. dehydro epiandrosterone – DHEA). Production of cortisol and androgen precursors is controlled by ACTH. The androgen precursors are converted in peripheral tissues to oestrogen and testosterone. Aldosterone production is regulated by angiotensin and potassium. This hormone is the principal sodium-retaining steroid hormone and maintains normal fluid balance and circulating volume.

The adrenal medulla is both a part of the autonomic nervous system and endocrine system. On response to sympathetic stimulation, two hormones, adrenaline and noradrenaline, are released. Adrenal medulla cells can be considered as neuronal cells that function as an endocrine gland.

Noradrenaline is both a neurotransmitter of the sympathetic nervous system and a hormone when released from the adrenal medulla. As noradrenaline and adrenaline enter the bloodstream, heart rate, blood pressure and respiratory rate increase, sweat glands become more active, the mouth becomes dry, glycogen breakdown increases, and blood glucose levels increase.

If the adrenal glands are destroyed or removed, appropriate steroid replacement will be required, including glucocorticoids and mineralocorticoids, as these are essential for life. Examples of common steroids that are commonly administered are prednisolone, hydrocortisone and dexamethasone. Steroids are used to treat inflammation, immune system disorders and prevent rejection of transplanted organs, and the side effects are listed in box 1. Steroids must not be withdrawn suddenly, as the adrenal glands need time to increase their own production of corticosteroids. Interestingly, medullary catecholamine replacement is not required, because the sympathetic nervous system can independently produce noradrenaline, which acts on adrenergic receptors, producing the characteristic sympathetic effects.

side effects of steroids

- Weight gain (truncal obesity).
- Hair growth (hirsutism).

- Delayed wound healing.
- Diabetes.
- Muscle weakness and wasting.
- Bone thinning and osteoporosis.
- Fluid retention (oedema).
- Gastric ulceration.
- Skin thinning, acne and stretch marks.
- Suppression of all inflammatory processes and generalised reactions of inflammation (can mask signs of underlying pathology, e.g. acute abdomen).
- Increased susceptibility to infections.

The pancreas and glucose homeostasis

The pancreas is an elongated, flattened organ situated in the epigastric and left hypochondriac regions of the abdominal cavity. It is both an exocrine and endocrine gland; the exocrine portion secretes pancreatic juice that aids the digestion of carbohydrates, proteins and fats through a duct (Waugh and Grant 2018). The endocrine component of the pancreas is a ductless gland that consists of the islets of Langerhans, which contain hormone-secreting cells. The islets of Langerhans produce three types of cells, termed alpha, beta and delta cells. The alpha cells secrete glucagon, the beta cells secrete insulin, and the delta cells secrete somatostatin. The plasma glucose level is the most important determinant of the rate of insulin release from the beta cells or glucagon from the alpha cells. High levels of glucose trigger insulin secretion and low plasma glucose leads to glucagon secretion, which in turn promotes glycogenolysis by the liver. Thus, insulin may be considered an anabolic hormone, whereas glucagon is catabolic in nature; insulin will lower blood glucose and glucagon will raise blood glucose. Insulin and glucagon have an inhibitory effect on each other, and both can be inhibited by somatostatin; thus, there is complex interplay

between the three hormones. Figure 11.5 gives a summary of the control of blood glucose by pancreatic enzymes and the liver. Insulin secretion can also be triggered by an increase in amino acids (e.g. a high protein meal) and ingestion of food as a result of hormones released by the gastrointestinal mucosa. There are, however, many mechanisms in place that operate both to increase and decrease circulating glucose as necessary to maintain homeostasis (Figure 11.5).

The role of the pancreas in glucose homeostasis

The specialised cells in the islet of Langerhans secret hormones which are finely balanced through sympathetic and parasympathetic stimulation to prevent the development of endocrine disorders (Waugh and Grant 2018; VanMeter and Hubert 2014). The opposing actions of glucagon and insulin regulate the blood glucose level, with glucagon raising the blood glucose level while insulin lowers the blood glucose level (Table 11.2). Insulin performs many important functions, including facilitating the transport of glucose into cells and promoting glycogen storage in the liver and muscles by activating enzymes to enable the storage of glucose. The cells preferentially use glucose for energy, facilitated by insulin. The presence of the insulin inhibits fat metabolism while glucose is utilised. In the absence of insulin (either due to low glucose levels or inadequate circulating levels of insulin), fat metabolism is increased, and free fatty acids are released into the circulation. These are used as an alternative energy source, as the cells are unable to take up glucose due to the lack of insulin. It is this mechanism that accounts for the weight loss exhibited by newly diagnosed patients with type 1 diabetes. Insulin also promotes *protein deposition in cells and tissue growth* – a lack of insulin will also lead to protein being used as alternative energy by cells. It is the utilisation by the cells of amino acids (protein) and free fatty acids that results in ketoacidosis.

The role of the liver in glucose homeostasis

The greatest amount of glucose input during the periods between meals and during the overnight fast comes from the contribution of the liver (Figure 11.6). The liver plays such an important role

in glucose production, that a total hepatectomy will result in death within 24 hours due to hypo glycaemia. The liver helps to maintain blood glucose homeostasis through two general mechanisms:

- The breakdown of stored glycogen (glycogenolysis).
- The formation of glucose from non-glucose precursors (gluconeogenesis).

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If dietary glucose is available, the liver increases its store of glycogen, but this is limited to 75–100g. The minimum required by the body daily is 125–150g in order to supply the brain, for which glucose is a mandatory requirement. During fasting, the initial source of glucose is from stored glycogen, but as this is restricted, amino acids (released from tissue protein) contribute about 75g of glucose daily via gluconeogenesis. This is mostly muscle protein, but there is a degree of protein breakdown from most organs. Glycerol (released from adipose tissue) can be converted to glucose by the liver, providing a further source of glucose, but can only contribute about 20g. Finally, lactate (produced from muscle) can also be metabolised by the liver for gluconeogenesis and thus provide additional glucose.

The role of the kidney in glucose homeostasis

No glucose is excreted by the kidney under normal conditions. Glucose is filtered into the glomerular fluid and is reabsorbed, but in hyperglycaemia, some of the glucose will be present in the urine (glycosuria). The presence of glucose in the distal tubules raises the osmotic pressure of the urine and reduces the amount of water reabsorbed by the proximal tubule, thus resulting in an increase in urine output (polyuria) and subsequent hypovolaemia (triggering thirst – polydipsia). This accounts for these symptoms being predominant in the presentation of diabetes mellitus. Sustained hyperglycaemia will result in dehydration, with fluids and electrolytes lost from the body as a result of an osmotic diuresis.

Catecholamines in the acutely ill

Extreme physiological stressors such as infection represent a stimulus for stress response. An increase in adrenaline or noradrenaline levels due to stress of critical illness (sympathetic nervous system stimulation) or via medication infusion to manage hypotension, can result in hyperglycaemia. Catecholamines stimulate glycogen breakdown into glucose by the liver (glycogenolysis) and inhibit insulin secretion by the pancreas. A patient may therefore require an insulin infusion *temporarily* to control the high blood glucose level. The liver is also responsive to plasma glucose levels, with low levels resulting in a release of glucose (by glycogenolysis) and high levels leading to glucose uptake. However, the liver is also very sensitive to levels of insulin (and glucagon), as glycogenolysis will continue to occur even in the face of a high plasma glucose level (as in the diabetic without insulin), identifying the importance of the hormonal influence. In the presence of insulin, however, glycogenolysis is inhibited, whereas glucagon will have the opposite effect. The physiological effects of catecholamines on glucose metabolism is summarised in Table 11.3.

Food with a low glycaemic index include:

- Oats.
- Lentils.
- Chickpeas.
- Carrots.
- Chocolate.

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Food with a high glycaemic index include:

- Ripe fruit.
- French fries.

- White rice.
- Sugary drinks.

Sources of glucose

The two main sources of glucose are:

- Intestinal absorption of dietary glucose and its precursors.
- Release of glucose from the liver.

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Plasma glucose levels increase with meals, rising slightly and then returning to basal levels after around two hours; therefore, both of these mechanisms are necessary for normal daily functioning. The most direct pathway for the formation of glucose is as a result of carbohydrate metabolism. As there is very little pure glucose in the diet, this results predominantly from the breakdown of the larger molecules, disaccharides and polysaccharides. The former are hydrolysed (broken down, using water to split the molecule) and absorbed rapidly, causing a prompt increase in plasma glucose concentration.

The rate of rise in blood glucose after the ingestion of foods are ranked in a glycaemic index. Carbohydrate foods that have an immediate effect on blood glucose levels are regarded as high glycaemic index, while those that have a slow effect on the blood glucose level are called low glycaemic index food. Foods containing disaccharides have a high glycaemic index. Polysaccharides enter the bloodstream more slowly, therefore having a low glycaemic index. Foods with a low glycaemic index release their energy slowly and may help those with type 2 diabetes with blood glucose control. However, a low glycaemic index diet is not recommended for children and young people with type 1 diabetes, due to limited evidence to support its effectiveness (National Institute for Health and Care Excellence [NICE] 2015).

Proteins are metabolised to amino acids, but some are able to donate their carbon atoms for glucose formation. Glucose generation from protein or from any other non-glucose source is called gluconeogenesis. With regard to fat metabolism, triglycerides release glycerol, which can be converted readily to glucose by the liver, but this accounts only for approximately 10% of the carbon atoms available from triglycerides, and thus has only a minor role in gluconeogenesis.

Neurological consequences of hypoglycaemia:

- Irritability, shakiness, confusion.
- Abnormal behaviour.
- Seizures.
- Loss of consciousness.

Utilisation of glucose

All tissues utilise plasma glucose, with some being obligatory users that cannot mobilise alternative substances when glucose is unavailable. Nervous tissue is unable to utilise free fatty acids (FFA), which are a major circulating fuel, hence the serious neurological sequelae of hypo-glycaemia. Other tissues that require exclusively glucose include red blood cells, the intestinal mucosa and the renal medulla. When FFA levels are high, and glucose and insulin levels are low, they will switch to use FFA as their primary metabolic fuel.

Alterations in glucose homeostasis

Maintenance of plasma glucose levels within certain limits is of vital importance, because the central nervous system, including the retina, depends upon an uninterrupted supply for normal functioning, the brain being an obligate glucose user (exclusively utilises glucose, as opposed to free fatty acids or amino acids). Insulin facilitates the entry of glucose into cells; therefore, any variation of blood glucose from the normal range (both excess and deficit) that is caused by a deficiency of insulin leads to a type of diabetes, depending on the aetiology of the condition:

- **Type 1.**

- **Type 2.**
- **Gestational.**
- **Impaired glucose tolerance and impaired fasting glycaemia.**

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Type 1 diabetes – insulin-dependents diabetes mellitus (IDDM) – is the result of a primary defect in the ability of the pancreatic islet tissue to respond to glucose with a release of insulin.

Type 2 diabetes, which was previously referred to as non-insulin-dependent diabetes mellitus (NIDDM), manifests as a relative insensitivity of the tissues to plasma insulin. Gestational diabetes is a pregnancy-induced diabetes, while impaired glucose tolerance and impaired fasting glycaemia is referred to as a pre-diabetes state, characterised by an elevated body glucose level, but not one high enough to prompt a diagnosis of diabetes. A summary of the characteristics of diabetes based on WHO (World Health Organisation) (2018) classification is shown in Table 11.4.

Impaired glucose tolerance and impaired fasting glycaemia

Impaired glucose tolerance (IGT) and impaired fasting glycaemia (IFG) are intermediate conditions, and people with IGT or IFG are at high risk of progressing to type 2 diabetes, although this is not inevitable.

Hyperthyroidism symptoms:

- Tachycardia and tachyarrhythmia.
- BP ↑.
- Nausea.
- Hyperactivity.
- Diarrhoea.
- Agitation.

- Heat intolerance.
- Goitre.
- Exophthalmos (prominent bulging eyes).

Acute endocrine problems and emergencies

Thyrotoxicosis, thyroid storm/crisis

Hyperthyroidism results from an excess of thyroid hormones (T3 and T4), with an exaggerated form being a thyroid crisis. A thyroid crisis (or storm) can be triggered by infection, surgery, trauma or any other acute episode (e.g. myocardial infarction, stroke and eclampsia), but fortunately, is very rare. An over-secretion of thyroid hormones will lead to a hypermetabolic state, resulting in hyperpyrexia, tachycardia, hypertension, agitation and tremors. The management is aimed essentially at reducing the effects of these hormones until the patient is stable. Drug therapy will include the use of:

- Beta-blockers such as metoprolol, to reduce sympathetic activity.
- Sedatives, such as chlorpromazine or haloperidol.
- Corticosteroids such as hydrocortisone, to inhibit the conversion of T3 to T4.
- Carbimazole, a specific anti-thyroid drug, inhibits enzymes that play a role in T3 and T4 production.
- Iodine is a specific antithyroid therapy used to inhibit thyroxine release and treat some forms of hyperthyroidism.

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Hypothyroidism symptoms:

- Hypothermia.
- Slow movements and thoughts.

- Fatigue.
- Excessive sleep.
- Constipation.
- Weight increase.
- Dry skin.
- Course hair, brittle nails.
- Goitre.
- Hoarse voice.
- Bradycardia.
- Puffy face.

Myxoedema and myxoedema coma

Myxoedema results from decreased thyroid hormone secretion (hypothyroidism), with signs such as bradycardia and slow mental functioning being representative of the hypometabolic state. The management strategies are focused on restoring thyroid levels, such as levothyroxine, which can take some time to stabilise and will usually be taken for life, as this is a long-term condition.

A myxoedema coma (rare, but with a high mortality) can occur in a patient with a chronic condition who is challenged by additional stress such as trauma. They may have had previous thyroid surgery or inappropriate doses of anti-thyroid medication such as carbimazole. The extreme hypometabolic condition will result in bradycardia, leading to hypotension and poor tissue perfusion, which, in turn, can result in a metabolic acidosis. Hypoventilation may result from the decreased conscious level, leading to hypercarbia and hypoxia and risking airway integrity. Other manifestations that may occur include fatigue, reduced cardiac output, an increased sensitivity to drugs, e.g. opiates, and paralytic ileus. The coma itself is likely to have been caused by multiple contributory factors, including the primary hypometabolism and secondary hypothermia, hypoxia, hypercarbia and hypoglycaemia. Management strategies are focused upon IV thyroxine replacement, support of

airway, breathing and circulation requiring invasive monitoring and intensive care support.

Adrenal insufficiency

Addison's disease is a chronic deficiency of cortical hormones, with symptoms reflecting the lack of cortisol (muscle weakness, fatigue, hypoglycaemia, ileus, reduced immunity to infection, low cardiac output), aldosterone (polyuria, dehydration, thirst, hypovolaemia, hyponatraemia, hyperkalaemia, postural hypotension, arrhythmias) and androgens (loss of libido and body hair). The condition is controlled by lifelong hormone replacement therapy. In times of stress (e.g. surgery, trauma, infection) where the increased demand for cortisol cannot be met, a patient may develop an Addisonian crisis or acute adrenal insufficiency. The immediate management of this life-threatening emergency will include urgent rehydration and correction of the hypoglycaemia. Cortisol will need to be administered, e.g. in the form of intravenous hydro-cortisone, and the trigger for the crisis (e.g. infection) addressed.

Causes of acute adrenal insufficiency:

- Abrupt withdrawal of steroid therapy.
- Stress, trauma, infection, surgery.
- Addison's disease.
- Pituitary or hypothalamic damage.

Pheochromocytoma or catecholamine crisis

A pheochromocytoma is a (very rare) tumour of the adrenal medulla, whereby abnormally high levels of adrenaline and noradrenaline are secreted into the systemic circulation. The release of these catecholamines is intermittent, but results in headaches, tachycardia, hyperglycaemia, blurred vision, bowel disturbances and very severe hypertension. Following blood

pressure stabilisation (usually with alpha- and beta-blockers), a pheochromocytoma will require surgical removal.

Diabetes insipidus (DI)

This is due to an insufficiency of ADH (antidiuretic hormone) being produced by the posterior pituitary. This can be precipitated by neuropathology affecting the hypothalamus or pituitary, or, rarely, by an insensitivity of the kidney to ADH (nephrogenic diabetes insipidus). It results in excessive water loss (polyuria), and if untreated, the patient will become profoundly hypovolaemic and hypernatraemic (as water is lost in excess of sodium). This situation is managed by the administration of desmopressin (DDAVP).

Syndrome of inappropriate antidiuretic hormone hypersecretion (SIADH)

This is characterised by excessive antidiuretic hormone (ADH) being secreted by the posterior pituitary gland. Its aetiology includes neuropathology (e.g. head injury, subarachnoid haemorrhage) or some carcinomas or infections. SIADH causes water retention (potential fluid overload) and haemodilution of solutes resulting in hyponatraemia. Initial management necessitates fluid restriction, with some situations including the administration of normal saline, but extreme care must be taken not to increase the plasma sodium by more than 12mmol/L per 24 hours (i.e. 0.5mmol per hour). It is important that SIADH is differentiated from other conditions causing hyponatraemia (e.g. cerebral salt-wasting syndrome) in which there is an accompanying hypovolaemia, as opposed to hypervolaemia, with its corresponding implications for fluid management strategies. Electrolyte imbalance is discussed in more detail in Chapter 4.

Diabetic emergencies

Endocrine emergencies account for about 1.3% of medical emergency admissions to critical care

(Kerr, Wen ham and Newell-Price 2017), and the majority are related to diabetes. Early recognition and treatment of possible problems, with an understanding of glucose management, is essential for the nurse caring for patients with diabetes. However, a small-scale study by Lange and Pearce (2017)that evaluates diabetes knowledge of registered nurses found a significant gap in diabetes knowledge among some staff in the hospital trust. Similarly, National Diabetes Inpatient Audit (2018) found that 20% of hospitals in England and Wales have no specialist inpatient diabetes nurses (DISNs).

Diabetes emergencies are either due to hypoglycaemic or hyperglycaemic episodes, and the most common problems are:

- Hypoglycaemia.
- Hyperglycaemia:
  - Diabetic ketoacidosis (DKA).
  - Hyperglycaemic hyperosmolar syndrome (HHS).
  - Euglycemic diabetic ketoacidosis.

Diabetic emergencies: hypoglycaemia

Hypoglycaemia is a biochemical diagnosis based on a blood glucose of less than 4mmol/L (JBDS 2018). It may be caused by insulin overdosage, a delayed meal following administration of insulin or increased metabolic rate due to exercise or acute febrile illness. The patient who is hypo-glycaemic will exhibit autonomic symptoms such as sweating, warmth sensation, anxiety, nausea and palpitations as a result of sympathetic nervous system stimulation. Other symptoms (e.g. tiredness, poor coordination, visual disturbances, drowsiness, confusion, seizures) are due to the effects of low glucose levels upon the nervous system (neuroglycopenia). Prolonged low blood glucose levels may starve the brain of glucose, resulting in coma and possibly death. The autonomic symptoms usually occur first, with neuroglycopenia more evident with a blood glucose of less than 2.5mmol/L.

Hypoglycaemia is a serious medical condition which can either be mild (self-treated), or severe hypoglycaemia, if assistance with care is required from another person (JBDS 2018). Once a patient has had a severe episode of hypoglycaemia, they may have impaired recognition of hypoglycaemia symptoms over the subsequent 24 hours. Other factors that can contribute to hypo unawareness include having type 1 diabetes for a number of years, use of certain drugs such as beta blockers, stress, depression and alcohol consumption (Diabetes UK 2018). The risk factors for hypoglycaemia include patients on insulin or sulfonylurea treatment, strict glycaemic control, impaired awareness of hypoglycaemia, severe hepatic and renal dysfunction, sepsis, increasing age and cognitive dysfunction (JBDS 2018). It is a requirement for a patient who is unable to notice hypoglycaemia to inform DVLA if they are driving. Some studies have indicated that the annual prevalence of hypoglycaemia is between 30–40% of people with diabetes (Strachan 2014). Zaccardi et al. (2016) found an increase of 39% in hospital admissions for hypoglycaemia over a 10-year period in England.

The potential causes of inpatient hypoglycaemia include medical issues such as inappropriate administration of insulin, mobilisation after illness, major amputation of limb and nutritional issues such as missed or delayed meal, vomiting or prolonged starvation due to being nil by mouth (JBDS 2018). However, there are also occasional healthcare professional-associated hypoglycaemic induced by error.

Iatrogenic hypoglycaemia

Iatrogenic hypoglycaemia (i.e. caused by health care professionals) is preventable. Examples of iatrogenic hypoglycaemia include stopping artificial feeding (enteral or total parenteral nutrition) for procedures such as CT scan, but leaving an insulin infusion running, or giving an incorrect higher dose of insulin.

Management of hypoglycaemia

Mild episodes can be treated by consuming refined carbohydrates, such as dextrose tablets, followed by long-acting carbohydrates, e.g. biscuits. Otherwise, patients may be treated with 'Glucogel' (30% glucose) which is a gel that is applied to the buccal mucosa. *It is contra-indicated to give anything by mouth to an unconscious patient.* In the unconscious patients, 1mg of glucagon can be given intramuscularly (IM) or intravenously (IV). Hospitalised patients may be treated with 250mL of 10% dextrose (IV) administered over a few minutes, followed by a continuous infusion depending upon local hospital guidelines. If no IV access is available, glucagon may be given intramuscularly. The administration of high concentrations of dextrose intravenously carries the risk of thrombophlebitis, therefore cannula insertion sites should be monitored closely. Whilst it is important to consult local guidance, JBDS (2018) have summarised the management of hypoglycaemia in different categories of patients, as seen below:

Management for conscious orientated, conscious but confused, unable to cooperate but able to swallow:

1. Give 15–20g quick acting carbohydrate such as:
    - 5–7 dextrosol tablets. Or
    - 150–200mL pure fruit juice e.g. orange. Or
    - 3–4 teaspoons of sugar in water.
2. Repeat capillary BG 10–15 minutes. If <4mmol/L, then repeat (up to 3 times in total, if required).
3. If remains <4mmol/L after 30 – 45 minutes or 3 cycles, then contact medical staff and consider glucagon IM or IV, 150–200mL of 10% glucose over 15 minutes.

4. Once >4mmol/L and the person has recovered, consider long-acting carbohydrates, e.g. 2 biscuits, slice of toast or 200–300mL of milk.
5. Treat hypoglycaemia, but do not omit insulin injection if due – however, regimen may need to be reviewed.
6. Ensure appropriate documentation, continue regular BG monitoring for 24–48 hours, consider cause of hypoglycaemia, provide hypoglycaemia education or refer to Diabetes team.

Management of unconscious and/or having seizures and/or very aggressive:

1. Check ABCDE assessment, institute appropriate interventions and request immediate medical help.
2. If on insulin infusion, stop immediately.
3. Consider one of the following 3 appropriate options:
    - If there is IV access, give 75–100mL 20% glucose over 15 minutes, check BG after 10 minutes and if less than 4.0mmol/L, repeat or give
    - 150–200mL of 10% glucose IV over 15 minutes, use an infusion pump if available and check BG after 10 minutes, if less than 4.0mmol/L, repeat
    - In case of no intravenous access, give glucagon, 1mg intramuscularly, but this may take up to 15 minutes to take effect.
4. Once >4mmol/L and the person has recovered, consider long-acting carbohydrate, such as 2 biscuits, a slice of toast or 200–300mL of milk (not soya).
5. Treat hypoglycaemia, but do not omit insulin injection if due – however, regimen may

need to be reviewed.

6. Consider possible cause of hypoglycaemia, document in notes and continue BG monitoring or refer to Diabetes team.

Management of adults who are nil by mouth:

1. For patients on a variable rate intravenous insulin infusion, adjust according to the prescribed regimen and seek medical attention. Most variable rate insulin infusions should re-commence once blood glucose level is over 4mmol/L, though adjustment of the rate may be indicated.

2. Consider one of the following options

    a) 75–100mL 20% glucose over 15 minutes, check BG after 10 minutes, and if less than 4mmol/L, give 150–200mL of 10% glucose over 15 minutes and repeat BG after 10 minutes until the patient is reviewed by the clinical nurse specialist or doctor.

    b) Hypoglycaemia may persist for up to 24–36 hours after the last dose, if the hypoglycaemia was due to long-acting insulin treatment or sulfonylurea, especially if there is a renal impairment.

3. Once >4mmol/L and the patient has recovered, consider intravenous infusion of 10% glucose at a rate of 100mL/hour.

4. Ensure appropriate documentation, continue regular BG monitoring for 24–48 hours, provide hypoglycaemia education or refer to diabetes team.

Diabetic emergencies: hyperglycaemia

Hyperglycaemia: diabetic ketoacidosis (DKA)

Diabetes ketoacidosis is a life-threatening complication of diabetes and accounts for a significant amount of all diabetes emergency-related hospital admissions. Most cases of DKA occur in patients with type 1 diabetes, but occasionally occur in type 2 diabetes. It is due to an increased demand for insulin, inadequate adjustment of insulin injection to meet the required needs of the body, severe physical or psychological stress or physical trauma without compensatory insulin and increased resistance to insulin due to various factors such as pregnancy or infection (Brashers, Jones and Huether 2017). The resultant lack of adequate insulin required to drag glucose into the cells leads to the body burning fatty acids to provide energy and thereby producing ketone bodies.

DKA is a triad of hyperglycaemia, hyperketonaemia and metabolic acidosis, thus, the diagnostic criteria are:

- Ketonaemia ≥3mmol/L or significant ketonuria.
- Blood glucose >11mmol/L or known diabetes mellitus.
- Bicarbonate (HCO_3) <15mmol/L **and/or** venous pH <7.3.

noindent

In addition to elevated blood glucose, acidosis, ketonaemia and ketonuria, the signs and symptoms of DKA include vomiting, dehydration, abdominal discomfort, unusual smell on the breath, tachycardia, alteration in potassium level, confusion and coma. Both DKA and HHS are triggered commonly by infection, but may be secondary to trauma, myocardial infarction and non-compliance with diabetes management. These emergencies may occur also in previously undiagnosed diabetics, thus being a first presentation of diabetes mellitus. Other precipitating factors include inappropriate insulin dosage or omission, acute illness, hyperthyroidism, alcohol abuse, other co-morbidities such as pancreatitis and drugs such as corticosteroids.

Management of DKA

For management of a deteriorating patient with DKA, use the ABCDE assessment approach and respond appropriately. Early recognition is essential to promote positive patient outcome. The management of DKA in adults include rehydration, insulin therapy and potassium replacement if needed, because insulin is a potent stimulus for potassium cellular uptake (Joint British Diabetes Societies Inpatient Care Group 2013). Therefore, fluid status, blood glucose, blood ketones and vital signs need to be closely monitored.

Fluid administration (most important initial therapy)

Assess fluid status, establish intravenous access, send blood for Urea and Electrolyte, bicarbonate, plasma glucose, blood culture and venous PH. Administer 0.9% sodium, 1 litre with no potassium over 1 hour, repeat over 2 hours and check potassium level, give another 1 litre over the next 2 hours and re-check potassium level and continue close monitoring of blood glucose level and cardio vascular status. Dextrose 10% is commenced to run at 125mL/hour when capillary blood glucose is less than 14mmol/L (NICE 2015).

Insulin therapy

The aim of insulin therapy is to suppress ketogenesis, reduce blood glucose level and correct electrolyte imbalance. Commence a fixed rate intravenous insulin infusion (FRII) based on 0.1 units/kg body weight and monitor blood ketones and capillary blood glucose level to determine whether adjustment is required. Avoid hypoglycaemia, and introduce dextrose regimen if blood glucose <14mmol (JBDS 2018).

Potassium replacement

Potassium is an essential mineral which helps with fluid balance regulation and electrical impulse transmission. The normal potassium level is 3.5–5mEq/L, and hypokalaemia is a life-threatening

condition, but is not common in DKA. 40mmol of potassium is added per litre of normal saline if the value is between 3.5–5.5mmol/L, and a medical review is required if below 3.5mmol/L.

Resolution of DKA

Resolution of DKA is achieved when there is a rapid improvement marked with pH >7.3, bicarbonate >15mmol/L and blood ketone level <0.6mmol/L.

Hyperglycaemic hyperosmolar syndrome (HHS)

HHS is also referred to as hyperosmolar hyperglycaemic nonketotic syndrome (HHNS), and it is a rare metabolic emergency requiring prompt management. Although it can affect both patients with type 1 and type 2 diabetes, it is common among people with type 2 diabetes. Like DKA, the precipitating factors include illness or infection. Other contributory factors are undiagnosed diabetes, poorly controlled type 2 diabetes, drugs such as long-term diuretics, beta-blockers (block the release of insulin) and corticosteroids. In HHS, there is some endogenous insulin secretion that is enough to extinguish excessive lipolysis and ketogenesis, but insufficient to facilitate adequate glucose utilization. It is characterised by:

- Hypovolaemia.
- Marked hyperglycaemia (>30mmol/L) without significant hyperketonaemia (<3.0 mmol/L) or acidosis (pH >7.3, bicarbonate >15 mmol/L); and
- Osmolality >320mOsmol/Kg.

noindent

The signs and symptoms include marked dehydration, elevated blood glucose level, dry mouth, skin and mucous membranes, polydipsia, polyuria and impaired consciousness levels. The goals of management are to treat the underlying cause, replace fluid and electrolyte losses and normalise the osmolality and blood glucose level. Similar to DKA, intravenous fluid management and potassium

replacement are commenced, but insulin therapy is only initiated if there is a significant ketonaemia >1mmol/L or ketonuria +++ Some of the similarities and differences between DKA and HHS are presented in Table 11.5.

Euglycemic diabetic ketoacidosis (EDKA)

This is a serious medical condition characterised by increased blood ketone, metabolic acidosis, but with blood glucose level less than 11.1mmol (200mg/dL). EDKA can present a diagnostic challenge because of normoglycaemia (blood glucose level is within the normal range). The causes include infection, decrease calorie intake/fasting, pancreatitis, prolonged vomiting, diarrhoea and patient on insulin pump (Rawla et al. 2017). It is important to monitor metabolic profile, check blood glucose level, arterial blood gas, ketone level, anion gap, bicarbonate and pH value. The management is to correct dehydration with intravenous normal saline of 4–5 litres and insulin drip with dextrose containing solution to correct anion gap and bicarbonate level.

DKA and HHS are caused by an absolute or relative deficiency of effective circulating insulin with associated increased levels of glucagon, catecholamines, cortisol and growth hormone. These result in increased glycogenolysis by the liver, generating hyperglycaemia. In DKA, the deficiency of insulin and increased counter-regulatory hormones lead to increased lipolysis and production of ketone bodies, with a resulting metabolic acidosis. The disturbed acid-base balance due to the metabolic acidosis is due to the dissociation of the H^+ ion from the ketone body acetoacetic acid. Patients with HSS do not develop ketoacidosis, but the mechanism for this is unclear.

Ketoacidosis

A metabolic condition associated with an accumulation of ketone bodies. Ketones (acetoacetic acid and beta hydroxybutyrate) result from the breakdown of free fatty acids and deamination of amino acids. Ketones can be smelt on the breath (like fruit or nail polish remover) due to acetone (from acetoacetic acid).

Differential diagnosis:

- *Diabetic ketoacidosis*: known history of diabetes.
- *Alcoholic ketoacidosis*: usually known history of alcohol abuse (no hyperglycaemia).

noindent

Starvation: ketosis (rather than ketoacidosis) (no hyperglycaemia).

Both DKA and HHS present with vomiting and a history of polyuria, polydipsia and weight loss. Patients with DKA can additionally have abdominal pain, although the underlying pathophysiology for this is unclear. Confusion is more common in HHS and believed to be related to the increase in serum osmolality rather than the hyperglycaemia. Physical signs include those associated with hypovolaemia (see patient assessment) resulting from the polyuria. This has been estimated to reach 5–8 litres in DKA and 8–10 litres in HHS (Kearney and Dang 2007). The excessive urine output leads to depletion in sodium, potassium, magnesium and phosphates. The main aims for treating both DKA and HHS are to correct dehydration, decrease the blood glucose level, correct electrolyte abnormalities and treat any precipitating causes, such as infection. Some patients with DKA or HHS will require initial management in the intensive care unit.

Hypovolaemia and urine output

Although patients with DKA or HHS will develop hypovolaemia (as a result of polyuria) they will still be passing urine. This is in contrast to patients who are hypovolaemic from other (non-diabetic) causes (e.g. haemorrhage) who will be oliguric or even anuric.

Assessment and physical examination

A systematic ABCDE approach to patient assessment is essential in order to identify problems and initiate appropriate and timely interventions(Resuscitation Council 2015) . Patients with DKA may have airway problems due to deteriorating levels of consciousness. Respiratory assessment may reveal signs of hypoxaemia and respiratory distress if a chest infection is the cause of the disruption in glucose homeostasis. Tachypnoea, with deep sighing respirations, can occur as a result of respiratory compensation for the metabolic acidosis arising from their ketoacidosis. Those with DKA or HHS may be cardiovascularly unstable as a result of hyperglycaemia, because their urine output may be inappropriately high (due to excreting excess glucose accompanied by water), resulting in severe hypovolaemia and dehydration with associated tachycardia, hypotension and hypothermia. The hypoglycaemic patient is often clammy or sweaty, whereas the patient with hyperglycaemia typically appears warm and dry, hence touching the patient may be informative. Disability assessment (including blood glucose measurement) will reveal hypo- or hyperglycaemia and may demonstrate altered neurological function (confusion, weakness and reduced level of consciousness) caused by altered glucose levels. Abnormal observations should always be reported immediately, particularly adverse changes in neurological status. Finally, measurement of the blood glucose, biochemistry and electrolytes will assist in identifying the underlying cause of the endocrine dysfunction (Resuscitation Council 2015). Table 11.6 gives an account of assessment findings of people with abnormal blood glucose levels.

CASE STUDY 11.1 Mr Shah: Part 1

Initial assessment

Mr Shah, a 65-year-old man was brought to the Emergency department this morning with a worsening abdominal pain. He has a past medical history of type I diabetes (aged 15), which is

normally well-controlled. Mr Shah is on steroid, glargine insulin 20 units daily (long-acting insulin) and Humalog (fast-acting insulin) 4–6 units three times daily. He was a lorry driver before retiring. He has been feeling very tired, lethargic and complaining of abdominal upset and loss of appetite. He recently had a flu-like illness and had been making a good recovery, but now he feels worse. He had been reducing his insulin, as he was not eating properly, and monitoring his blood glucose accordingly. The previous evening he had felt hot and unwell; early in the morning, the abdominal pain started, and by 10:00 he then came to the emergency department. He last assessed his blood glucose at midnight, and as it was raised at 13.2mmol/L (normal range normal range 4–10mmol/L). He did not want to administer his insulin as he had not eaten supper. He weighs 80Kg.

Assessment at 10:30

Airway: on assessment, you note his airway is patent, with no stridor or gurgling sound. He is able to speak, though he feels unwell. Therefore, there is no immediate airway risk, so the assessment is continued.

Breathing: his lips and oral mucosa appear dry, as he is mouth breathing, but show no evidence of central cyanosis. He is able to speak in full sentences. His breathing pattern appears a little laboured. His respiratory rate is 18/minute, with oxygen saturations of 95% and deep, equal, bilateral chest movements. He is using his accessory muscles to breath. On lung auscultation, no adventitious breath sounds are heard. His SpO_2 is within the target saturation of 94–98% set by medical staff, recommended by the British Thoracic Society guidance. There is no need to institute oxygen administration, though it is prescribed, if required. You notice a strange smell on his breath and, in light of his past medical history, are concerned this might be a sign of raised ketones.

Breathing NEWS (0 + 1 + 0) = 1

Circulation: on admission to the emergency department, his skin feels warm and dry. His capillary refill time is 2 seconds. His BP is 99/74mmHg, with a heart rate of 105, and his pulse feels regular. He reports passing urine frequently overnight and is happy to use the bottle so an accurate assessment of fluid balance can be maintained using a fluid balance chart. He feels thirsty and is drinking freely, but he also feels nauseated. You explain that you would like a sample of urine to test for glucose, ketones and infection as soon as he feels the need to pass urine. You establish IV access and take blood for venous gasses, electrolytes, glucose, ketones, renal function and infection markers.

Circulation NEWS (2 + 1) = 3

Disability: He is assessed as alert, using ACVPU (RCP 2017). He understands where he is and who you are, so no new confusion is evident. Hyperglycaemia (and hypoglycaemia) is a common cause of a reduced level of consciousness and can present with drowsiness, confusion or coma due to its effect on the nervous system (neuroglycopenia). Always check a blood glucose if a patient presents with new confusion or their level of consciousness drops.

His blood glucose is 23.5mmol/L (**normal range 4–10mmol/L**). You are concerned, as he is hyperglycaemic and reporting abdominal pain. Those with DKA can present with abdominal pain, which may be due to a primary problem causing the DKA (that requires treatment), or a symptom of the acidosis associated with DKA (Umpierrez and Freire 2002).

Disability NEWS = 0 However, the high blood glucose is a cause for concern. Bedside blood ketone testing was deemed necessary.

Exposure: Mr Shah looks flushed and uncomfortable. His temperature is recorded at 38.5°C. You are unsure of the reason for this and decide to collect sputum and urine samples when possible. The notes identify his BMI is 40, confirming obesity. His abdomen appears distended, he has regular bowel sounds on auscultation and is not constipated. However, when gently palpating the abdomen you notice guarding, and Mr Shah says 'ooh that hurts'. You do not want to continue with any further abdominal assessment, but will ask your medical colleagues to examine him, to minimise discomfort for Mr Shah.

Exposure NEWS = 1

Total NEWS = 5

noindent

Mr Shah has a high blood glucose level and ketones are present. Your concern increases when urinalysis reveals:

- +++ glucose.
- ++ ketones.
- Positive to protein and leucocytes.

noindent

You are aware that he has the potential to deteriorate rapidly. His NEWS of 5 requires urgent escalation.

Action: escalate to doctor and outreach team

SBAR used to articulate concerns regarding NEWS score of 5.

As a result of the SBAR handover, it is recommended that a second IV cannula is sited and blood cultures are taken, as per the Sepsis Trust's protocol.

Medical Review: 11:00

Given his history and presentation, a provisional diagnosis is made of diabetes ketoacidosis. A urinary tract infection is likely, and abdominal problems cannot be discounted. An arterial blood gas sample is taken for analysis also.

Initial blood results confirm suspicions.

The team discuss the blood results and patient presentation/potential problems.

No **airway** problems are noted, but if the blood glucose continues to rise, an altered level of consciousness may threaten airway integrity.

No specific **breathing** problems are identified; the low $PaCO_2$ combined with the low pH suggests a partial respiratory compensation for a metabolic acidosis. The pH of 7.22 (normal range 7.35–7.45) confirms acidosis. The lowered bicarbonate of 16mmol/L (normal range 22–26mmol/L), along with the negative BE of 6 (BE -6, normal range +/- 2), is consistent with a metabolic acidosis. The high blood glucose, the presence of ketones in the blood and urine suggests DKA. The criteria to diagnose DKA is a triad of hyperglycaemia, metabolic acidosis and hyperketonuria, which are all clinical manifestations presented by Mr Shah. The team discuss how this is caused by a deficiency of insulin, reducing the ability of the glucose to enter the cell to be broken down into energy. The liver responds by breaking down fat (lipolysis) into ketones for energy. The presence of ketones lowers the blood pH causing acidosis. This will be treated under circulation.

As SpO_2 remains within the normal range, and the extra work of breathing will reduce as the acidosis resolves, no further action is taken for breathing. The Hb is within normal range, suggesting oxygen transport is good.

Action to be taken to support **circulation and disability** include:

- Insertion of a second IV canula.
- 500mL NaCl 0.9% to be given over 15 minutes. It is decided not to include K^+ supplements in this first bag, as his serum K^+ is in the high normal range. This will be checked again in 1 hour.
- An infusion of soluble insulin, diluted with 0.9% normal saline for a concentration of 1 unit /mL, is prepared. This needs to be given at 0.1units/kg/hr. Mr Shah weighs 80kg, so the infusion is prescribed to run at (0.1 × 80 = 8) 8mL/hr. This is given via the second IV line.
- Serum glucose, ketones and K^+ to be checked in 1 hour.
- Urinary catheter inserted to monitor urine output accurately, residual of 300mL obtained, sample sent for microscopy.
- A stat dose of cefuroxime 250mg IV is given, prescribed 12 hourly.
- The usual dose of long acting insulin is administered when due.

noindent

Continuous cardiac monitoring is commenced and reveals sinus rhythm. Serum K^+ is on the high end of normal, so this needs to be closely monitored. The insulin infusion will reduce serum K^+. Urea is higher than the normal range, suggesting (as the creatinine is normal) that Mr Shah does not have renal dysfunction, but may be dehydrated. The raised WCC and CRP is consistent with infection; the source needs to be found.

Under **exposure**, Mr Shah's abdomen is assessed by the medical team. General tenderness is discovered on palpation, but with no evidence of rebound tenderness that may indicate peritonitis. The team decide to re-examine his abdomen as the treatment for DKA progresses and

acidosis resolves. Paracetamol 1gm IV is prescribed and given, to reduce pyrexia and abdominal pain.

Ongoing care

The nurse feels confident to explain to Mr Shah exactly what is going on and asks if there is anybody that should be called. Mr Shah requests his daughter be informed so she can bring him in some pyjamas and toiletries. He is surprised at how quickly his blood glucose has risen. You suggest that both him and his daughter chat to the team when he is feeling better, so he can be confident in managing his diabetes, asking for help should he feel unwell in the future

Mr Shah is in the resuscitation area in the emergency department to facilitate close monitoring of his condition whilst initial interventions are in progress and a bed on the acute medical unit can be found. The initial 500mL of normal saline is complete, and the fixed rate insulin infusion is in progress. He has been given his normal dose of long-acting insulin. His urine output is looking to be good for the following hour. In light of this output, the medical team prescribe normal saline 1L with 40mmol KCL to be infused over 2 hours via a volumetric pump.

12:00

His observations are checked prior to transfer to AMU.

A and B: breathing less laboured, RR 16, SpO_2 95%.

C: BP 109/80, HR 93, urine output 200mL for last hour. K^+ now 4.7mmol/L.

D: alert, blood glucose 18mmol/L, ketones 4.5mmol/L.

E: temp 37.8°C.

noindent

NEWS = 2 (blood glucose and ketones remain raised)

The nurse is reassured but realises that Mr Shah is far from completely well, even though his NEWS is reduced. His BP has risen, and his HR is falling as a result of the IV fluid, helping to restore fluid balance. Hourly monitoring continues.

Maximising endocrine status

Insulin administration

Insulin treatment will increase glucose utilisation in peripheral tissues and also decrease glucose production by the liver. It also decreases the formation of ketones and inhibits the release of free fatty acids (FFAs), thereby correcting the metabolic acidosis. The ultimate aim is to achieve a blood glucose between 10–15mmol/L over 24–48 hours. The use of insulin may lead to hypokalaemia, as it facilitates movement of potassium into cells. Close electrolyte monitoring is therefore essential, and replacement is administered to maintain serum potassium between 4–5mmol/L.

National Patient Safety Agency (NPSA) insulin safety guidance (2010)

Recommendations to reduce the number of wrong dose incidents involving insulin:

- All insulin bolus doses should be measured and administered using an insulin syringe (not intravenous syringe).
- The term ‘units’ should be used at all times (not U or IU).
- Training programmes should be in place for all health care professionals

involved in the administration of insulin.

noindent

The transition from insulin infusion to the subcutaneous route is challenging, but should be attempted once the patient is stable and is able to eat and drink.

Glycaemic control

Normal fasting blood glucose levels are 3.5–5.5mmol/L, fluctuating to 7–9mmol/L following a meal. Insulin infusions are titrated against a sliding scale of blood glucose, aiming usually for a blood glucose within the 'normal range'. It is well recognised that hyperglycaemia is toxic: Falciglia et al. (2009) demonstrated an increase in mortality of ICU patients with a blood glucose of greater than 6.1mmol/L which was unrelated to illness severity. However, such tight control over blood glucose brings logistical challenges with the frequency of monitoring of blood glucose, and more importantly, a risk of iatrogenic hypoglycaemia, which can be harmful. A blood glucose of 2.2mmol/L is associated with a six-fold increase in mortality, and lower levels could be fatal. Hence, local recommendations are to maintain a blood glucose between 4 and 10mmol/L, rather than strictly between 4 and 6mmol/L in the critical care setting (see NICE-SUGAR Study 2009).

NICE-SUGAR Study (2009): a large randomised controlled trial

On admission to intensive care, patients were randomised to:

- Either maintain intensive glucose control of between 4.5–6.0mmol/L;
- Or conventional glucose control of less than 10mmol/L.

noindent

Although there was no difference between the two groups regarding length of stay on intensive care, the conclusion of this large multi-centre international trial was that intensive glucose

control increased mortality among adults in intensive care. There were also significantly more episodes of severe hypoglycaemia (less than 2.2mmol/L) in the group with intensive glycaemic control (6.8% versus 0.5%) and increased morbidity.

Fluid and electrolyte management

Fluid administration for hypovolaemic patients (DKA and HHS) commonly includes using isotonic/normal saline (0.9% NaCl), but hypernatraemic patients may need half normal saline (0.45% NaCl). Correcting the fluid deficit will increase the intravascular volume and lower the plasma osmolality and blood glucose levels by dilution. The infusion rate will depend upon the circumstances, but the initial aim would be to correct the hypovolaemia within 24 hours.

Hyperglycaemia and serum sodium

Measured serum sodium needs to be recalculated in hyperglycaemia to obtain a 'true sodium level'. This is because extracellular osmolality rises in the presence of excess glucose (as it is slower to enter cells if there is a relative lack of insulin), with water accompanying the glucose into the extracellular fluid. As the extracellular fluid is diluted, the sodium concentration falls. This is described as a translational hyponatraemia because there is no change in total body water. The sodium level will not need to be treated, as this will correct itself as the glucose level normalises.

The chronicity of the situation should be considered with acute hyponatraemia (<48 hours) being more amenable to a faster correction than a longstanding condition, however, plasma sodium levels should not be increased faster than 12mmol per 24 hours.

Hypokalaemia will need correction, particularly with respect to patients receiving insulin, to attain levels between 4–5mmol/L. Patients who are hypokalaemic due to polyuria are at risk of having their hypokalaemia exacerbated iatrogenically when large fluid volumes are administered, as they

will be haemodiluted. Fluid administration, in these circumstances, should therefore include a potassium supplement. Phosphate depletion is common in DKA, but replacement is seldom required.

Pharmacological interventions

The pharmacological management (hypoglycaemic agents) options include:

- *Sulphonylureas*: e.g. glibencamide, gliclazide, chlorpropamide, tolbutamide. These drugs lower blood glucose levels by increasing insulin production by the pancreas. They may also increase the sensitivity of the tissues to insulin. Drug interactions may include NSAIDs (including aspirin) enhancing the effect and thiazide diuretics reducing efficacy.
- *Metformin*: this belongs to a group of chemicals called biguanides. The mechanism of action is not understood fully, but it may stimulate uptake of glucose into muscle and reduce glucose release from the liver. It is most commonly prescribed in conjunction with a sulphonylurea if a patient is not responding to the latter alone.
- *Thiazolidinediones*: e.g. pioglitazone, rosiglitazone, which belong to chemicals called glitazones. They appear to reduce tissue resistance to insulin and are usually administered alongside a sulphonylurea or metformin.
- *Prandial (relating to a meal) glucose regulators*: nateglinide and repaglinide have differing mechanisms of action, but both are unique in that they act postprandially (after eating) to stimulate insulin release by the pancreas.
- *GLP-1 agonist (Glucagon like peptide 1 receptor agonist)*: e.g. exenatide, liraglutide, lixisenatide, which stimulate insulin release, suppress glucagon, slow gastric emptying, offer sense of satiety and fullness and lead to reduced food intake.
- *DPP-4 inhibitors or gliptins*: a class of oral hypoglycaemics that block the enzyme dipeptidyl peptidase-4 (DPP-4). Examples of DPP-4 agents are

sitagliptin, vildagliptin, saxagliptin, linagliptin and alogliptin.

- *Sodium-glucose co-transporter-2 (SGLT2) inhibitors*: also called gliflozins, e.g. dapagliflozin, canagliflozin and empagliflozin; they block glucose re-absorption in the kidney and thereby increase urinary glucose excretion (glycosuria) to reduce blood glucose level.

CASE STUDY 11.2 Mr Shah, type I diabetic: Part 2

Mr Shah, with type 1 diabetes, was admitted to the emergency department this morning with abdominal pain and was feeling generally unwell. He was found to have diabetic ketoacidosis and initial treatment with fluids and a fixed insulin infusion were started at 11:00.

Diabetic ketoacidosis is a life-threatening medical condition characterised by hyperglycaemia, osmotic diuresis, metabolic acidosis, glycosuria, ketonuria and dehydration. It is due to an increased demand for insulin, inadequate adjustment of insulin injection to meet the required needs of the body, severe physical or psychological stress or physical trauma without compensatory insulin and increased resistance to insulin due to various factors such as pregnancy or infection (Waugh and Grant 2018). Other precipitating factors include inappropriate insulin dosage or omission, acute illness, hyperthyroidism, alcohol abuse, other co-morbidities such as pancreatitis and drug such as corticosteroids.

Mr Shah had recently been unwell, possibly has a new urinary tract infection and has not stuck to his normal insulin regimen, which could have contributed to his DKA development. Ketoacidosis has resulted in increasing hyperglycaemia, polyuria, dehydration, hypovolaemia

and electrolyte imbalance. If not treated in a timely manner, confusion, coma and death may ensue (Waugh and Grant 2018). Diabetic ketoacidosis accounts for a substantial amount of all diabetes-related hospital admissions.

A key indication for managing type 1 diabetes is the need for exogenous insulin due to progressive destruction of beta cells leading to insulin insufficiency. With lack of circulating insulin in the system, Mr Shah underwent a series of compensatory mechanisms which led to lipolysis and, consequently, ketones in the blood. Hyperglycaemia-induced osmotic diuresis can also lead to dehydration and electrolyte imbalance. Other manifestations of DKA are Kussmaul respiration (deep sighing breaths), acetone breath, nausea, vomiting and coma. Depending on individual compensatory reserve, DKA can develop as early as 24 hours. Noncompliance to insulin therapy accounts for a significant number of DKA cases, and this could be a contributory cause in this case.

The foci of management of DKA in adults are: rehydration, insulin therapy and potassium replacement, as insulin is a potent stimulus for potassium cellular uptake (Joint British Diabetes Societies Inpatient Care Group 2013; NICE 2015). Therefore, Mr Shah's fluid status, blood glucose, blood ketones and vital signs were being closely monitored. The aim of treatment is to reduce blood ketones by 0.5mmol/L/hr and blood glucose by at least 3mmol/L/hr.

He was transferred to the higher monitoring bay of the AMU, under the care of diabetologist and the diabetes specialist team. The National Service Framework emphasises the importance of skilful management of diabetes emergencies, DKA inclusive (DH 2001).

13:00: higher monitoring bay, AMU

A and B: breathing, RR 16, SpO_2 95%.

C: BP 110/80, HR 91, urine output 150mL for last hour. K^+ now 4.1mmol/L.

D: alert, blood glucose 13mmol/L, ketones 3.9mmol/L.

E: temp 37.8°C.

NEWS 2 = 2

Mr Shah seems to be progressing well, with a stable NEWS of 2. He is beginning to feel better, and he thinks the paracetamol has helped his abdominal pain. Further blood tests result at 13:00 are below.

Ensuring fluid balance

Fluid replacement is the priority in DKA, as an osmotic diuresis causes fluid depletion with negative cardiovascular effects. Mr Shah was initially tachycardic and hypotensive. 1500mL of IV saline helped replace the volume lost, and his NEWS fell from 5 to 2. NICE (2015) suggest IV fluid therapy should be continued until the deficit is replaced and to maintain fluid balance thereafter. An accurate fluid balance chart is essential, as, if too much fluid is given, the rare, but often fatal, condition of cerebral oedema may develop. This is not common in Mr Shah's age group, being a higher risk for children and young adults.

Ensuring serum potassium balance

The falling serum K^+, despite giving supplemental potassium with the IV fluids, is noted. The litre of normal saline with 40mmol KCL given over 2 hours is nearly complete, and the blood results suggest the potassium supplement should continue with the next bag. Therefore, medical review was sought. There are several factors at play here:

- Initially, when acidosis is present (pH<7.35) the serum K^+ will be raised. The competing H^+ and K^+ ions mean more K^+ leaves the cell (as H^+ enters) into the blood, raising serum K^+. This can be seen in the first blood result with the K^+ of 5.5mmol/L.
- As serum pH moves towards normal, H^+ reduces and potassium enters back into the cell, reducing serum K^+.
- The fixed-rate insulin infusion facilitates entry of glucose into the cell along with K^+, thereby reducing not only blood glucose, but serum K^+ also.

noindent

It can be seen in this case study that the serum potassium is gently falling in spite of IV potassium supplements, but currently remains within a safe range. In short, close monitoring of serum potassium is essential in DKA, as large variations in a short time span can cause fatal cardiac arrhythmias. Cardiac monitoring in the initial management can help early detection of arrhythmia.

Ensuring blood ketone and glucose balance

Mr Shah's blood glucose has now fallen below 14mmol/L. His blood ketones are reducing, but remain raised. Note that the serum pH is also rising to near normal range as the ketones are being excreted. As per NICE (2015) guidance, 10% glucose at 125mL/hr is given in addition to the normal saline infusion. This is to enable insulin infusion to continue, until the blood ketone concentration is <0.6mmol/L and serum pH is >7.3. As this stage is reached, Mr Shah should be able to eat and drink normally, be given a subcutaneous fast-acting insulin and a meal, with the insulin infusion stopping soon after. Blood glucose and ketone monitoring should be performed hourly for the first 6 hours (NICE 2015).

The abdominal pain that prompted his hospital attendance resolved as his DKA resolved. Whilst abdominal pain can be a symptom of the acidosis associated with DKA, further investigation is required, as problems such as pancreatitis, hepatitis and pyelonephritis may be an underlying cause (Bello et al. 2018).

Resolution of DKA has been achieved when:

- pH >7.3.
- Bicarbonate >15mmol/L.
- Blood ketone level <0.6mmol/L.

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Mr Shah and his family will need support in continuing management of his diabetes, both during his hospital stay and on discharge.

This is an example of a patient with DKA who was well-managed on admission. The probable trigger of the event, a UTI, was treated promptly. It is interesting to note that the NEWS score of 2 during his recovery indicated improvement, but close monitoring was still required as he remained at risk of deterioration. NEWS is a guide for assessment of deterioration risk, but should not replace the health care professional's clinical judgement.

Conclusion

The aim of this chapter is to provide an insight to common endocrine disorders, because nurses are often confronted with the challenges of managing this group of patients. A prompt diagnosis

is important in the hospital setting; it is claimed by Kearney and Dang (2007) that, with improved care and early detection, DKA and HHS can be prevented entirely. Certainly, awareness and prompt recognition of these conditions will promote better outcomes in such patients (Kisiel and Marsons 2009). Fortunately, DKA and HHS are relatively uncommon, however, the diabetic emergency of hypoglycaemia is more frequently encountered, so this situation requires particular insight and awareness by nurses into the importance of their role, as many hypoglycaemic episodes are iatrogenic in causation and are associated with increased morbidity and mortality. Nurses have a key role to play in the care of patients with acute endocrine problems, and it is important for nurses to respond appropriately, recognise their limitations, call for assistance and make appropriate referral when necessary.

Glossary <inline>inline1.6</inline>

Endocrine From Greek *endo* inside and *crine* to secrete. The secretion of hormones via ductless glands directly into the bloodstream.

Exocrine *Exo* = outside. Secretion of chemicals via glands with ducts.

Gluconeogenesis Metabolic pathway resulting in the generation of glucose from non-carbohydrate sources such as lactate, glycerol and glucogenic amino acids.

Glycogenolysis Conversion of stored glycogen to glucose by the liver.

Glycosuria Presence of glucose in the urine.

Ketoacidosis A metabolic condition associated with an accumulation of ketone bodies. Ketone bodies are the breakdown product of free fatty acids and the result of deamination of amino acids.

Kussmaul breathing Rapid, deep, laboured breathing due to respiratory compensation for a severe metabolic acidosis. This can arise from either ketoacidosis or renal failure. Named after Adolf Kussmaul, a nineteenth-century German doctor.

Neuroglycopenia A deficiency of glucose in the brain as a result of hypoglycaemia. This adversely affects the functioning of neurones.

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Figure 11.1 The position of the major endocrine glands and the hormones they produce

Figure 11.2 The hypothalamus, anterior and posterior pituitary glands, their targets and associated hormones

Figure 11.3 The thyroid and parathyroid glands

Figure 11.4 Example of a negative feedback loop

Figure 11.5 Control of blood glucose by pancreatic hormones

Figure 11.6 The role of the liver in glucose metabolism

Table 11.1 Summary of the hormones of the endocrine system and their effects

Tissue	Hormones	Target/effect/function
Hypothalamus	TRH (thyrotropin-releasing hormone) DA (dopamine) (or prolactin-inhibiting hormone) GHRH (growth hormone-releasing hormone) SS (somatostatin) (or GHIH growth-hormone inhibiting hormone) GnRH (gonadotropin-releasing hormone) CRH (corticotrophin-releasing hormone)	• Stimulates TSH (thyroid-stimulating hormone) release from the anterior pituitary. • Stimulates prolactin release from the anterior pituitary. • Inhibits prolactin release from anterior pituitary. • Stimulates GH (growth hormone) release from the anterior pituitary. • Inhibits GH release from the anterior pituitary. • Inhibits TSH being released by the anterior pituitary. • Stimulates FSH (follicle-stimulating hormone) release from the anterior pituitary. • Stimulates LH (luteinising hormone) release from the anterior pituitary. • Stimulates ACTH (adrenocorticotrophic

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Tissue	Hormones	Target/effect/function
		hormone) release from the anterior pituitary.
Pituitary gland: anterior lobe	ACTH (adrenocorticotrophic hormone) TSH (thyroid-stimulating hormone) GH (growth hormone) FSH (follicle-stimulating hormone) LH (luteinising hormone) Prolactin	• Stimulates the adrenal cortex – specifically targets cells that produce glucocorticoids, which influence glucose metabolism. • Targets the thyroid gland with subsequent release of thyroid hormones T4 (thyroxine) and T3 (triiodothyronine). • Stimulates cell growth and replication. • Affects follicle development in females – stimulates oestrogens by ovarian cells. • Stimulates cells in tubules where sperm differentiate. • Induces ovulation in females and androgens including testosterone in males. • Stimulates milk production by the mammary glands.
Pituitary gland: posterior lobe	ADH (antidiuretic hormone) Oxytocin	• Increases the absorption of water in the distal tubule and collecting duct in the kidney. • Stimulates smooth muscle in the wall of

Tissue	Hormones	Target/effect/function
		the uterus promoting labour and delivery.
Pineal gland	Melatonin	• Control of 'biological clock'.
Thyroid gland	T4 (thyroxine) T3 (triiodothyronine) Calcitonin	• Directly affect the mitochondria in cells and therefore metabolic rate. • Regulation of calcium concentration in body fluids.
Parathyroid	PTH (parathyroid hormone)	• Regulation of calcium concentration in body fluids.
Thymus	Thymosins	• Role in immunity.
Tissue	**Hormones**	**Target/effect/function**
Adrenal glands	Adrenal medulla: Produce 2 hormones. Adrenaline (epinephrine) and noradrenalin.	• They both potentiate the fight of flight response~~Stimulation of alpha and beta receptors in the blood vessels and myocardium to cause either vasoconstriction or vasodilation, as required~~.
	• ~~Adrenaline~~	
	• Adrenaline • Noradrenaline	• A~~lso a~~ccelerates the utilisation of cellular energy and mobilisation of energy reserves e.g. increase metabolic rate.

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Tissue	Hormones	Target/effect/function
	Adrenal cortex: Produce 3 groups of steroids. Mineralocorticoids Glucocorticoids and Sex hormones	• Mineralocorticoids, e.g. aldosterone secretion targets cells that regulate the sodium and potassium ions in excreted fluids.
	• ~~Mineralocorticoids~~ • Glucocorticoids	• Glucocorticoids, e.g. cortisol regulates metabolism of fat, protein and carbohydrates.
	• Sex hormones	• Sex hormones: androgens, oestrogens and progesterone influence reproductive functioning.
Pancreatic (Islets of Langerhans)	Insulin	• Lowers blood glucose levels by increasing the rate of glucose uptake and utilisation by cells.
~~**islets**~~		
	Glucagon	• Raises blood glucose levels by increasing rates of glycogen breakdown and glucose release by the liver.
	Somatostatin	• Inhibits insulin and glucagon release by the pancreas, also suppresses exocrine secretion by the pancreas.
	Pancreatic ~~polypetides~~polypeptides (PP)	• Self-regulates endocrine and exocrine pancreatic activity.

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Tissue	Hormones	Target/effect/function
	~~Cholecystokinin (CCK)~~	
	~~Pancreatic polypeptide (PP)~~	
Gonads	Male testes: androgens	• Affects reproductive functioning.
	Female ovaries: oestrogens	• Secondary sexual characteristics.

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Table 11.2 Hormonal secretions of the islets of Langerhans and their roles

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Hormones	Role
Insulin	Decreases high levels of blood nutrients, especially glucose, but also amino acids and fatty acids, by: • Stimulating the uptake and ~~utilization~~ utilisation of glucose by muscle and connective tissue. • Increasing the rate of conversion of glucose to glycogen (glycogenesis), mostly in the liver and skeletal muscles. • Increasing the uptake of amino acids by cells. • Increasing the synthesis of fatty acids and fat storage in adipose tissue (lipogenesis). • Reducing the breakdown of glycogen into glucose

Hormones	Role
	(glycogenolysis). • Preventing the breakdown of protein and fat.
Glucagon	Glucagon increases the blood glucose level by: • Converting glycogen to glucose in the liver and skeletal muscles (glycogenolysis). • Converting non-carbohydrates, such as amino acids, to glucose (gluconeogenesis). Glucagon secretion is stimulated by a low blood glucose level and exercise, and decreased by ~~somastostatin~~somatostatin and insulin.
Somatostatin	Somatostatin: • Inhibits the secretion of insulin and glucagon from the pancreas and the secretion of growth hormone from the anterior pituitary gland.

~~Source:Waugh~~Source: Waugh and Grant (2018)

Table 11.3 Physiologic effects of catecholamines

Organ/t~~T~~issue	Process or result
Brain	•Increase glucose metabolism.

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Organ/t~~T~~issue	Process or result
Skeletal muscle	• Increase glycogenolysis. • Decrease glucose uptake and utilisation (decreases insulin release).
Liver	• Increase glucose production. • Increase glycogenolysis.
Adipose tissue	• Increase lipolysis. • Decrease glucose uptake.

Source: Clayton, McCance and Takahashi (2017)

Table 11.4 Summary of diabetes

Insulin-dependent diabetes mellitus (IDDM)	Non-insulin-dependent diabetes mellitus (NIDDM)
Type ~~I~~1 diabetes	Type ~~II~~ 2 diabetes
• This is less common (10% of diabetics).	• This is becoming increasingly common (90% of diabetics).
• There is a sudden onset that can at any age, but mostly affecting young persons. At diagnosis the person is underweight or normal weight.	• There is a gradual onset, predominantly in adults. The person is usually overweight at diagnosis.
	• Characterised by an insulin resistance (the cause of which is unknown) this may be

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Insulin-dependent diabetes mellitus (IDDM)	Non-insulin-dependent diabetes mellitus (NIDDM)
	combined with a relative reduction in insulin production.
• Characterised by a lack of insulin produced by the beta cells in the islets of Langerhans in the pancreas. This is thought to be immune-induced in the majority of cases.	
	• Managed by diet and oral hypoglycaemic medications.
• Managed by the administration of insulin administration.	
Gestational diabetes	
This ~~is~~ type of diabetes ~~occure~~ occurs during pregnancy (2—5% of pregnancies) and most closely resembles type ~~II~~ 2 diabetes. This type of diabetes will often self-correct~~s~~ after delivery, however, affected mothers have a tendency to develop type ~~II~~ 2 diabetes later on in life.	

Table 11.5 Comparisons and contrasts between DKA and HHS

	Diabetic ketoacidosis (DKA)	Hyperglycaemic hyperosmolar syndrome (HHS)
Definitions		
(American Diabetic Association)	• Blood glucose >-13.8mmol/L. • PH <-7.30. • Bicarbonate <-18mmol/L. • Anion gap >-10. • Ketonaemia.	• Blood glucose >-33.3mmol/L. • Ph <-7.30. • Bicarbonate >-15mmol/L. • Serum osmolality >-320osmol/kg.
Demographics	• Most commonly younger, slimmer	• More commonly older, obese

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	Diabetic ketoacidosis (DKA)	Hyperglycaemic hyperosmolar syndrome (HHS)
	patients with type ~~I~~ 1 diabetes. • Mortality less than 5% – most common cause of death in young people with diabetes.	patients with type ~~II~~ 2 diabetes. • Mortality 15%.
Presentation	• Rapid onset (<-24 hours). • Vomiting, polyuria, polydipsia, weight loss plus abdominal pain.	• Insidious onset (several days—weeks). • Vomiting, polyuria, polydipsia, weight loss.
Physical signs	• As per hypovolaemia: tachycardia, hypotension, low CVP. • Confusion is rare.	• As per hypovolaemia: tachycardia, hypotension, low CVP. • Confusion more common.
Biochemistry	• Blood glucose rarely greater than 40mmol/L. • Ketones in urine.	• Blood glucose often greater than 50mmol/L. • No ketones in the urine.

Table 11.6 Summary of examination and assessment

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	Variable	Hypoglycaemia	DKA	HHS
A/B	• Airway patency. • Respiratory rate pattern. • O_2 saturations.	• Most commonly: normal respiratory rate and pattern.	• Tachypnoea. • **Kussmaul breathing** (rapid, deep laboured) due to respiratory compensation for a	• Most commonly: normal respiratory rate and pattern.

	Variable	Hypoglycaemia	DKA	HHS
			metabolic acidosis.	
C	• BP. • Pulse. • Capillary refill time (CRT). • Skin. • CVP. • Temperature. • Urine output.	• Sweating. • Palpitations due to sympathetic nervous system (SNS) stimulation.	• Tachycardia, hypotension, low CVP due to hypovolaemia. • Increased CRT. • Dysrhythmias due to electrolyte imbalance. • Often hypothermic. • Urine output may be inappropriately high due to hyperglycaemia.	• Tachycardia, hypotension, low CVP due to hypovolaemia. • Increased CRT. • Dysrhythmias due to electrolyte imbalance. • Often hypothermic. • Urine output may be inappropriately high due to hyperglycaemia.
D	• GCS pain assessment.	• Anxiety (due to SNS stimulation). • Tiredness, poor coordination, visual disturbances, drowsiness, confusion, coma, seizures are due to the effects of low glucose levels upon the nervous system (neuroglycopenia).	• Confusion, lethargy, reduced level of consciousness.	• Confusion (thought to be due to increased serum osmolality).

	Variable	Hypoglycaemia	DKA	HHS
		• Reduced level of consciousness due to neuroglycopenia. • Low GCS.		
E1	• Blood results.	• Blood glucose < 3mmol/L.	• Blood glucose > 13.8mmol/L. • PH <-7.30 • Bicarbonate < 18mmol/L. • Anion gap >-10. • Ketonaemia. • Hyponatraemia. • Hypo/hyperkalaemia. • Hypomagnesaemia. • Hypo- or hypophosphatemia.	• Blood glucose >-33.3mmol/L. • Ph <-7.30. • Bicarbonate >-15mmol/L. • Serum osmolality > 320osmols/kg. • Small amount (or no) ketones. • Hyponatraemia. • Hypo/hyperkalaemia. • Hypomagnesaemia. • Hypo- or hypophosphatemia.
E2	• Other.	• Nausea (due to SNS stimulation).	• Nausea, vomiting. • Abdominal pain. • Acetone smell on breath due to ketones.	

Table 11.7 Initial blood results

Arterial blood gas results		normal range		Venous blood results		normal range
pH	7.22	7.35–7.45		Na+	138mmol/L	135–145mmol/L
$PaCO_2$	3.3kPa	4.6–6kPa		K+	5.5mmol/L	3.5–5.5mmol/L
PaO_2	10.8kPa	10.5–13.5kPa		Glucose	22mmol/L	4–10mmol/L
HCO_3^-	16mmol/L	22–26mmol/L		Ketones	5.0mmol/L	<0.6mmol/L
BE	-6.1mmol/L	-2 to + 2		WCC	15.8mmol/L	4–11mmol/L
Lactate	1.8mmol/L	<2mmol		CRP	45mg/L	<10mg/L
				Urea	11mmol/L	3–7mmol/L
				Creatinine	70 mcmol/l	60–110 mcmol/L

Table 11.8 Blood test results at 13:00

Arterial blood gas results		normal range		Venous blood results		normal range
pH	7.29	7.35–7.45		Na^+	142mmol/L	135–145mmol/L
$PaCO_2$	4.3kPa	4.6–6kPa		K^+	4.1 mmol/L	3.5–5.5mmol/L
PaO_2	10.8kPa	10.5–13.5kPa		Glucose	13 mmol/L	4–10mmol/L
HCO_3^-	18mmol/L	22–26mmol/L		Ketones	3.9 mmol/L	<0.6mmol/L
BE	-4.1mmol/L	-2 to + 2		WCC	15.8mmol/L	4–11mmol/L
Lactate	1.6mmol/L	<2mmol		CRP	40mg/L	<10mg/L
				Hb	132g/L	130–160mg/L
				Urea	13mmol/L	3–7mmol/L

				Creatinine	75 mcmol/l	60 – 110 mcmol/L