2

Homeostasis

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Chapter 2 Learning Objectives

- To gain knowledge and insight into the physiology related to the systems involved in maintaining homeostasis
- To gain insight into conditions related to dysfunction of these systems
- To apply theory to practice by understanding the clinical presentation when homeostatic failure occurs in patients
- To gain insight into pharmacological preparations to either prevent or treat relevant conditions
- To follow a structured approach to patient assessment thereby identifying homeostatic failure at a treatable stage
- To demonstrate evidence of professional development within your portfolio in relation to understanding homeostasis and homeostatic mechanisms
- To continue to develop the ability to question and critique the evidence base and effectiveness of plans of care for patients with a variety of homeostasis problems

Introduction

Homeostasis refers to the state of functional equilibrium within the body's internal environment, namely the cells, tissues, organs and fluids (Clancy & McVicar 2002).

Maintenance of homeostasis depends primarily on providing an internal environment suitable for facilitating normal cellular function. Certain stimuli can result in alterations to the internal physical environment referred to as stressors, all of which have the ability to affect cellular function pathologically. Insults range from compromise to a specific cellular function, through to multi-organ failure and death. As a result the internal environment is constantly changing or adapting; this is a direct result of physical, psychological and environmental stressors.

This chapter will define homeostasis and the homeostatic mechanisms essential to the normal functioning of healthy cells, which preserve the fundamental processes necessary to maintain cell, tissue and organ function i.e. life. One such example is the regulation of carbon dioxide, an acid and waste product of cellular metabolism which, if it accumulates, can lead to respiratory depression, hypoventilation or a low respiratory rate (<10 breaths/minute). Healthy lungs excrete carbon dioxide in water, which we exhale. Any diseases that affect the lung tissue or reduce the diffusion of gases across the alveolar–capillary membrane may affect the ability of the lungs to excrete carbon dioxide (Chapter 3).

The human body has many homeostatic mechanisms: thermal, chemical, neural and hormonal, which govern a multitude of cellular processes. Some processes are more familiar to us and more common than others, such as the production of insulin, the regulation of food intake, water and electrolyte balance, the hypothalamic control of body temperature, the maintenance of cardiac output, oxygen and carbon dioxide levels, and the balance between acidity and alkalinity as a consequence of metabolism. Others are less known but are of no less importance, such as calcium homeostasis for the remodelling and repair of bones (Chapter 8). A myriad of hormones released by the thyroid gland control growth, the sleep–wake cycle, and the body's response to stress and illness to maintain health.

Throughout the 24-hour cycle the body continuously adjusts all of these mechanisms, and many others, to maintain equilibrium and optimum cell function. During illness or excessive or prolonged exposure to stressors, the ability of the body to self-regulate and maintain an optimum internal environment by manipulating these finely tuned mechanisms is exceeded and cells and organs dysfunction; this can manifest as acute or chronic disease.

There are many different symptoms and clinical presentations associated with homeostatic failure; in fact most of the clinical conditions presented by patients are as a result of a failure on some level. This chapter will

demonstrate how homeostatic failure is related to specific diseases, such as diabetes, case examples will illustrate the complexity of such clinical presentations and relevant investigations, and the clinical care required will be reviewed. The challenge for nurses is to be able to link the symptoms to the altered pathophysiology of the many diseases associated with homeostasis, and to understand the role of the nurse in supporting patients with a variety of clinical problems as a result of homeostatic failure. Questions should focus on understanding what assessments and investigations the patient may undergo. What observations should be performed in the acute stages of homeostatic failure? How should treatment and management plans be constructed and evaluated? What information is required by the patient from healthcare professionals to adjust life styles such as those with newly diagnosed diabetes or renal failure?

Applying Theory to Practice: Exercise 2.1

Before commencing this chapter:

- Identify which organs or glands you already know that are involved in maintaining homeostasis
- Compose a list of diseases that you already know that affect the body's ability to maintain homeostatic function in relation to temperature, blood pressure, water balance, glucose control and acid base
- Add this list to your developing portfolio and on completion of this chapter expand your portfolio with the new disorders you have gained knowledge on and elaborate on how these disorders affect physiological function and how to recognise homeostatic failure
- This might include the patient's history, clinical presentation and tests such as blood analysis

Homeostatic feedback mechanisms: a matter of balance

In the role of the nurse you need to understand how the different hormones and chemicals are 'switched' on and off, or how they are regulated up or down in response to a stimulus. These mechanisms operate on feedback systems that either result in a positive or negative response. For a system to operate there must be detectors and effectors, which are ultimately coordinated by nerve centres, most often the hypothalamus in the brain. A feedback mechanism is one in which a positive effect is seen, with the release of chemicals or hormones which target specific cells or organs to achieve the desired effect.

Detectors are mostly nerve receptors that detect changes and monitor the variable that needs to be controlled. An example of a detector would be baroreceptors or stretch receptors that detect changes in blood volume by the amount of 'stretch' or volume of blood in the carotid sinus and arch of the aorta. When the circulating blood volume falls, as in haemorrhage, the baroreceptors send impulses back to the brain, or coordinating centre. In turn, via the autonomic nervous system, several mechanisms are stimulated, all aimed at increasing and redirecting the circulating volume and restoring blood pressure (Figure 2.1).

An effector is a gland, organ or muscle that will bring about or 'effect' the change (Figure 2.1). The release of catecholamines, such as noradrenaline and adrenaline, from sympathetic nerve endings causes an increase in heart rate in an attempt to raise cardiac output. When we review the mechanisms of hypotension there are many effectors that come into play to try to raise the blood pressure; these will be discussed later in the chapter.

A negative feedback system is designed to respond in the opposite direction of the deviation so that the deviation is reduced in potency or shut off, causing the opposite effect to occur i.e. a rise in blood pressure. The deviation feeds back to the cardiovascular system, in this case via the brain, to reverse or alter the direction of the deviation or reduce its potency, in other words to halt the hypotension. Equilibrium is achieved as the blood pressure is restored to normal, achieving homeostasis.

A positive feedback system responds in the same direction as the initial stimulus, enhancing the potency of and amplifying the stimulus rather than controlling, reducing or shutting off the deviation. An example would be the activation of the clotting cascade in response to haemorrhage. The up-regulation and production of clotting factors are amplified to control bleeding and achieve haemostasis.

Failures in homeostatic mechanisms can lead to life-threatening clinical emergencies such as severe hypotension, hypo- or hyperglycaemia, haemorrhage, and acid-base imbalances, which can cause patients to

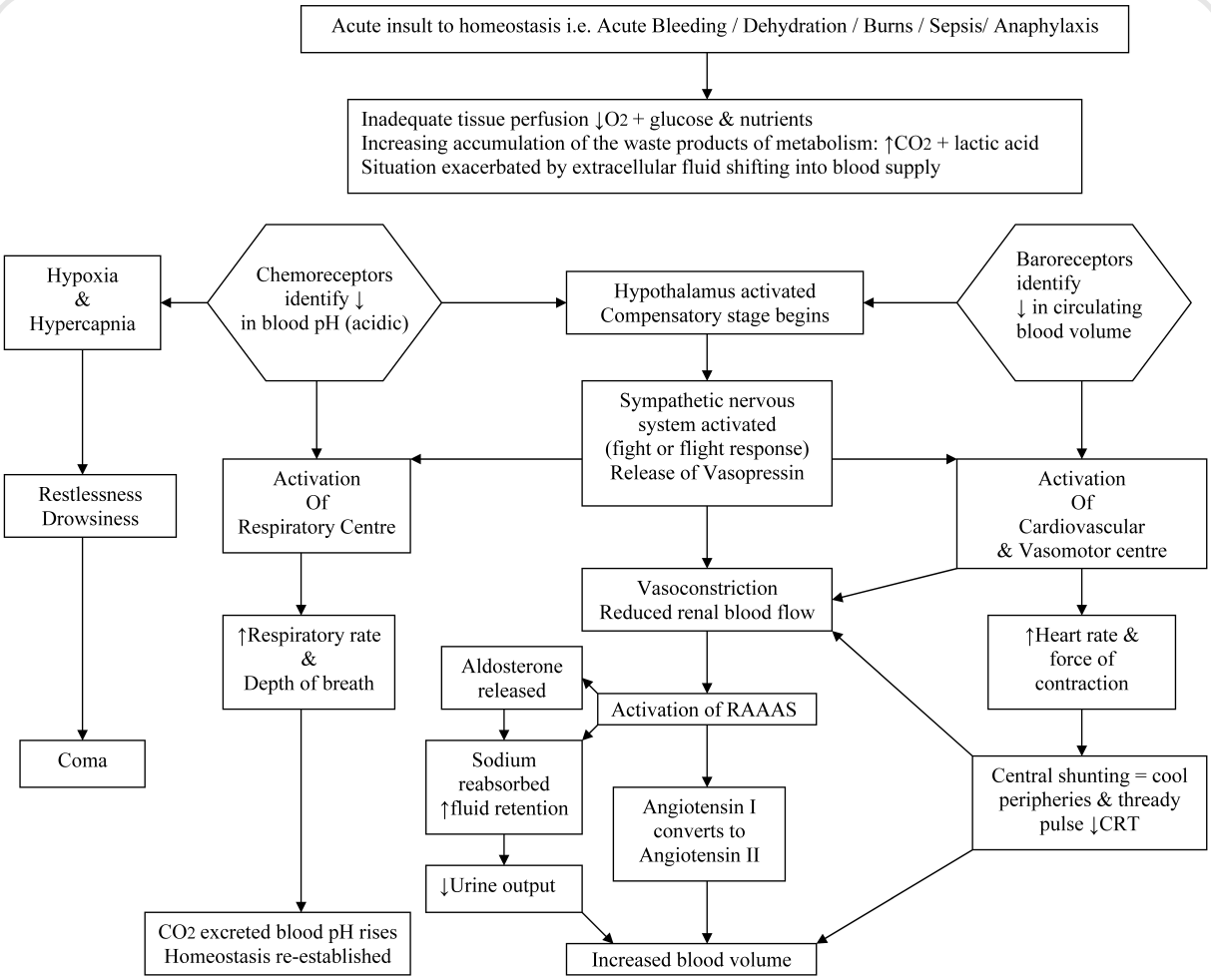

Figure 2.1 Maintaining homeostasis

develop renal failure and ultimately cardiac arrest or death. Extreme homeostatic failure can lead to the development of multi-organ dysfunction syndrome (MODS) necessitating admission to intensive care units.

The hypothalamus

One of the most important homeostatic regions of the body is the hypothalamus, situated below the thalamus on top of the brain stem and comprising the base of the third ventricle. It is the nerve control centre for many autonomic responses, which include respiratory rate and depth, heart rate and force of contraction, blood pressure and gut motility. Metabolic control includes body temperature regulation, water balance and thirst control, hunger and fullness mechanisms, which are linked to glucose and insulin levels, and regulation of the sleep–wake cycle or 'biological clock'. Emotional responses such as fear, rage and pleasure are also under hypothalamic control as the hypothalamus sits within the limbic centre, an area responsible for emotional responses, which are relayed via the autonomic nerve pathways of the hypothalamus (Figure 2.2).

The hypothalamus exerts its effects by two main mechanisms, the release of hormones and activation of nervous pathways to achieve the desired effect. The hypothalamus is in close contact with other important centres, the pituitary gland and the mammillary bodies or nuclei that are involved in the sense of smell (olfactory pathways)

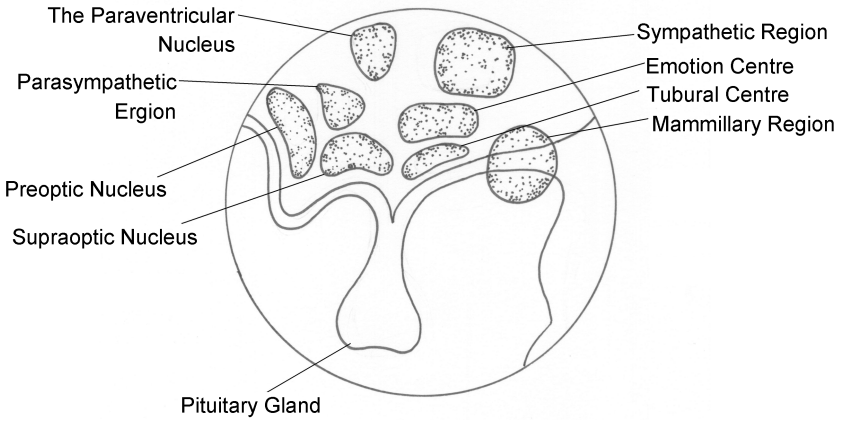

Figure 2.2 Structure of the hypothalamus (Source: http://library thinkquest.org)

Regulation and control of temperature

The main thermoregulation centre in the hypothalamus is the preoptic region (Figure 2.2), which regulates temperature via the autonomic nervous system (ANS). This area comprises of a heat-loss and heat-promoting centre or the thermoregulatory centre. Humans maintain a constant core temperature about 37°C which is independent of the surrounding environment. We are capable of living in extreme climates with the external temperature ranging from –52°C to +49°C (Hinchliffe et al. 1999).

The core temperature is shared between the organs within the skull, thoracic and abdominal cavities; these deep-seated organs have the highest temperature. The shell or periphery is the heat loss surface; the organs and deeper vessels are separated from the skin by a layer of fatty tissue, which acts as an insulating layer during cold weather. The skin has the lowest temperature and this can range from 27°C on the toes to 34°C on the forehead (Hinchliffe et al. 1999). A temperature gradient therefore exists between the core and the periphery. Sometimes patients who have severe infections can have a high core temperature of >39°C but peripherally they can be cold demonstrating peripheral shutdown, often a sign of severe sepsis and circulatory collapse. Exercising skeletal muscles can generate 30–40 times more heat compared to the rest of the body (Marieb 1998). Heat or cold is detected by peripheral receptors (afferents) located in the skin and central thermoreceptors that detect blood temperature are located in the core.

The hypothalamus responds to input via autonomic efferent pathways. The nature of the stimulus will determine the character of the physiological response; whether to gain heat by shivering and vasoconstriction of the periphery or to promote heat loss by sweating and vasodilation of the periphery. See Figure 2.3.

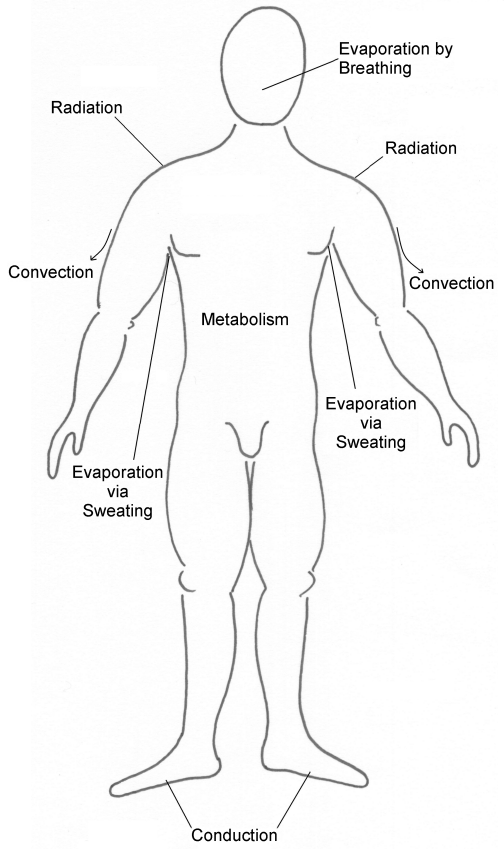

Figure 2.3 Heat loss and heat generating mechanisms

Anatomy & Physiology in Action: 2.1

Maintaining Homeostatic Balance of Heat Loss and Heat Gain		
Radiation: heat is lost as infrared waves from the body to the air. A dense object like the body will transfer heat to the environment. Energy flow is always from warmer to cooler air/ objects (Marieb 1998).	Conduction or convection: the transfer of heat between objects in contact with each other. It involves molecule-to-molecule contact. Thermal energy moves through a medium such as fluid. Convection is the transfer of heat to the air, causing warm air to expand and rise. The cooler air becomes denser and falls: the warmed air is continually replaced by cool air. This mechanism enhances the exchange of heat from the body surface to air – as the cooler air absorbs heat by conduction. That is how a summer breeze on a hot day cools the skin.	Evaporation: the conversion of water from a liquid into a gas or water vapour. Water molecules absorb heat from the environment – called the heat of vaporisation. The kinetic energy or temperature of the liquid will determine how much energy is generated and hence water vapour produced. An example is demonstrated by huskies in the Antarctic – as the huskies exhale, heat is lost as water vapour and because the surrounding air temperature is –28°C the water vapour is visible.
	Conduction and convection account for 15–20% of heat loss (Marieb 1998). Think about this statement – what might that mean for patients? Patients who experience fever are often fanned: this is called forced convection – fanning enhances the heat loss process. Patients should not be directly fanned as this can cause shivering and generate heat to inadvertently raise the temperature. Try the experiment on yourself or a colleague. Keep the fan on one part of the body for 10 minutes or more and see the effects.	Insensible water loss. Water vapour is lost via respiration, the mucosa of the mouth and skin. A patient with a chest infection on oxygen with a respiratory rate of 40 breaths/minute can lose water vapour due to the high respiratory rate and pulmonary secretions. This is exacerbated by the drying effects of oxygen and enhances the feeling of dehydration. During vigorous exercise the body can perspire and evaporate up to 2L of water just through sweat. In normal resting conditions and in environmental temperatures of 31°C–32°C the sweat glands produce 0.5L sweat/day. Sweat output increases up to 12L of body water/day depending on the external temperature and degree of humidity.

Heat generating mechanisms

The peripheral receptors send impulses when the blood is cooler than the hypothalamic set point to the posterior heat-promoting centre.

Applying Theory to Practice: Exercise 2.2

- How does the body generate heat in response to cold external temperatures or following immersion in cold water or an infusion of cold fluids during a blood transfusion or prolonged resuscitation?

Administering cold intravenous fluids can quickly cause hypothermia in an already compromised patient.

- How can this be prevented?

Under the control of the sympathetic nervous system, vasoconstriction of the periphery is activated diverting blood flow away from the skin surface to deeper tissues and vessels by closing precapillary sphincters and reducing blood flow to the skin. Patients who are very cold may have white, marble-like skin which may be cyanosed; this is caused by the trapping of blood in the periphery with loss of oxygen from the blood to the tissues, hence the blue tinge of the skin (Hinchliffe et al. 1999). The hairs or pili on our skin play a part in trapping heat; each hair follicle is attached to erector muscles which help to lift the hair follicle – the trapped heat acts as an insulting layer. This makes our hairs stand on end and also gives us goose pimples or bumps.

Another effective way of raising the core body temperature is shivering. Any muscle activity will increase the body temperature; shivering is activated by the hypothalamus when the shell temperature falls below 35°C. Shivering is much more effective as a mechanism at higher temperatures as the muscles become weak and uncoordinated at lower temperatures. In the elderly shivering is much reduced due to an ineffective response to sympathetic activity. The elderly can lose heat over a period of time and therefore may not take effective measures to raise the body temperature.

Thyroxine (T_4) is an important hormone secreted by the thyroid gland that stimulates metabolically active cells to consume oxygen; this is called the calorigenic effect (Ganong 1995) and is effective in raising the basal metabolic rate (BMR). Chemicals such as adrenaline and noradrenaline also raise the body temperature by increasing the BMR and heat is gained. Another provider of heat is the powerhouse of the body, the liver. Through its enzymatic functions and processes it provides a constant source of heat.

Applying Theory to Practice: Exercise 2.3

Review the warming devices in your clinical areas that are available to warm patients and fluids

- Which mechanisms of heat production are employed?
- Which is the most effective?

Anatomy & Physiology in Action: Exercise 2.4

Thyroxine is an important hormone released by the thyroid gland. Identify the anatomical position of the thyroid gland and make a list of the functions of the different hormones released by the gland.

Removal of the thyroid is called a thyroidectomy

- For what reasons may this occur?

Heat loss mechanisms

During warmer weather the increased air temperature causes vasodilation of dermal blood vessels bringing the vessels close to the skin surface so that heat can be lost via conduction, radiation and convection. In addition stimulation of the sweat glands via sympathetic fibres helps to cool the body by the process of evaporation. This negative feedback system is finely tuned to maintain the hypothalamic set point; during warmer weather the heat promoting centre is inhibited and the heat loss centre is activated.

Anatomy & Physiology in Action: Exercise 2.5

- Using Figure 2.1 apply the feedback pathway to the regulation of temperature. Draw the pathway and insert the mechanisms that are related to the homeostatic control of temperature.
- What and where would the receptors and effectors be located?
- What effectors are found within the skin that result in sweating and shivering?
- Make a list of the factors that affect heat gain and heat loss.

Developing and Delivering Expert Care: 2.1

Normal Body Temperature

The body can only function within a narrow temperature range between 35.6ºC–37.8°C despite changes in air temperature.

Body temperature fluctuates about 1 degree in 24 hrs. It is lowest in the early morning and highest in the early evening.

Temperature helps maintain optimal enzymatic function for effective cell metabolism.

Increases in the temperature will increase enzymatic activity or catalysis – therefore each rise of 1°C results in a 10% increase in chemical reactions (Marieb 1998).

Temperatures above 41°C can cause convulsions. The upper limit for life is 43°C although there are case reports of patients surviving beyond this upper limit. At this temperature proteins begin to denature and cell function is destroyed.

Hypothermia is a core temperature below 35°C. Heat generating mechanisms are activated such as shivering and erector pili muscles.

At 34ºC intense shivering occurs, there is difficultly in the movement of digits, changes in skin colour – cyanosis and some confusion or behavioural changes may occur.

At 30ºC–32ºC shivering progressively ceases. There is increased sleepiness, bradycardia or slowed heartbeat, shallow respirations and moderate confusion.

At 28ºC the patient may appear dead. If uncorrected, coma, cardiac arrest from severe rhythm disturbances or respiratory arrest and death will occur when the body temperature approaches 21ºC.

Pathology of fever

Fever is also known as pyrexia or controlled hyperthermia. It is derived from the Latin word *febris* or febrile. Fever occurs when the body temporarily fails to maintain the temperature within normal limits. The set-point is elevated by 1–2º C and is a symptom of many medical conditions. White blood cells and macrophages release endogenous (internal) pyrogens (fire starters) or cytokines into the bloodstream. These chemicals act directly on the thermostat in the hypothalamus causing the release of prostaglandins and elevating or re-setting the set-point. Fever also increases the basal metabolic rate; it inhibits the growth of bacteria so assisting the body's defence mechanisms to fight invading pathogens, eating is inhibited (anorexia) and proteins are denatured which can result in irreversible brain damage.

Figure 2.4 Mercury thermometer

Developing and Delivering Expert Care: Exercise 2.6

Classification of Fever

Pyrexia (fever) can be classed as

- Low-grade: 38–39°C (100.4–102.2°F)
- Moderate: 39–40°C (102.2–104°F)
- High-grade: >40°C (>104°F)
- Hyperpyrexia: >42°C (>107.6°F) (a medical emergency)

During clinical placements identify patients who have changes in temperature.

- Can you identify the causes?
- What treatments are required to manage patients with hypothermia and hyperpyrexia?

Applying Theory to Practice: Exercise 2.7

Causes of Fever

Under the following three headings identify some of the micro-organisms, diseases or therapies that might cause a fever:

- Infections:
- Inflammation:
- Allergic reactions:

Cancer and chemotherapy treatments cam also induce a fever due to tumour necrosis. Brain injuries can cause fever that is unresponsive to cooling measures and anti-pyretics.

Why might that be?

Review the location of the hypothalamus.

Immunological diseases like lupus erythematosus, sarcoidosis and inflammatory bowel diseases can also cause fever.

The destruction of tissues which can occur in **haemolysis**, surgery, myocardial infarction, crush syndrome, **rhabdomyolysis** and cerebral hemorrhage may induce fever.

- In clinical practice see if you can identify patients who have experienced some of the diseases or treatments mentioned.

Drugs can also cause a 'drug fever' either as a direct consequence of the drug or as an adverse reaction to the drug (e.g. antibiotics). Discontinuation of some drugs like heroin withdrawal can induce a fever.

The homeostatic control of blood pressure

Before discussing the compensatory mechanisms deployed to control blood pressure there is a need to review some commonly used definitions. Blood pressure is defined as the force or pressure exerted on blood vessel walls by the circulating volume of blood in a closed system. Blood pressure is a hydrostatic pressure as the blood is confined within a closed system; confined liquids exert a pressure against the walls of the container and therefore any changes in flow, viscosity, resistance and structure will influence the pressure. See Chapter 4 for a more in-depth review of the cardiac cycle.

Applying Theory to Practice: Exercise 2.8

Thermometers

The first thermometers were used nearly 300 years ago based on the principles of a liquid (mercury) expanding in a sealed glass bulb. The temperature is recorded along a scale on the thermometer. There are two temperature scales in use today, Fahrenheit and Celsius. In the UK Celsius is commonly used.

Mercury thermometers were used to take the temperature orally: from the axilla, mouth and rectum. There was also a low-grade thermometer, which had a blue coloured bulb for easy identification. Today there are many alternatives to thermometry.

- Identify which thermometers are used within your practice areas.
- How are they cleaned? Does this affect there reliability?
- Why do they use this system? Are they research based?

Developing and Delivering Expert Care: Exercise 2.9

Recording the Temperature

Normal Temperature Readings:

Rectal temperature 0.5°C higher than the oral temperature

Otic temperature or tympanic (ear) is at, or higher than 38°C (100.4°F)

Oral temperature is at, or higher than 36.5°C–37.5°C (99.5°F)

Axillary temperature is at, or higher than 37.2°C (99°F)

- Identify in your clinical area which methods are utilised to measure the temperature (Chemical, digital, tympanic or temporal).
- How is temperature monitored during surgery?
- Identify what alternatives may be used to monitor unconscious patients who cannot shiver or change their body temperature due to anaesthesia.

The 'gold standard' of temperature measurement is either the pulmonary artery catheter (PAC) or any device that can measure the blood temperature directly. The problem with these devices is that they are highly invasive and are only available in high dependency and intensive care settings.

Pulse pressure

The pulse pressure is the difference between the maximal systolic pressure and minimal diastolic pressure (Klabunde 2005). The pulse pressure is affected by the stroke volume and the elasticity of the arteries. If the 'normal' blood pressure in healthy adults is 120/80mmHg then the 'normal' pulse pressure is roughly 40mmHg. Increases in heart rate and contractility can widen the pulse pressure as can head injuries associated with raised intracranial pressure. A narrowed pulse pressure is seen in hypovolaemia, septic shock, patients on glycerine trinitrate (GTN) infusions (a potent vasodilator), or during major haemorrhage when the systolic pressure falls due to reduced arterial stretch; the diastolic pressure may drop slightly but often remains fairly constant. If you refer to the case study of Mark Bradbury below, his blood pressure has dropped to 85/55mmHg, he has a narrowed pulse pressure of 30mmHg. The clinical significance of severe blood loss resulting in a reduced or narrowed pulse pressure is that oxygen delivery to the tissues and subsequently the cell is compromised resulting in cellular dysfunction.

Scenario 2.1

Major Haemorrhage

Mr Mark Bradbury, a 35-year-old Caucasian man is admitted to the emergency department following a road traffic accident. He sustained an open fractured right femur, ruptured spleen and a fractured pelvis, resulting in internal bleeding, and lacerations to his face and hands.

On examination he is pale, cold and clammy

His vital signs are:

Airway – Clear

Breathing – Respiratory rate 36, SpO2 95% on 12L/minute O_2 via non-rebreathe system

Circulation – Pulse 120, weak, regular. Blood pressure 85/45mmHg. Capillary refill time >4 seconds. Blood glucose 4.8mmols/L.

Disability – Patient responds only to voice on the AVPU scale = V. Pupils – Pupils equal and reactive to light (PEARL)

Exposure – Open/compound fracture right leg. Distended abdomen, tender with evidence of guarding in left upper quadrant. Lacerations to face and hands Temperature 35.6°C.

Past medical history (PMH): Nil of note

Social – Smokes 30/day. Alcohol: 20 units/week. Lives alone.

Mean arterial pressure (MAP)

Another important pressure recording is the mean or average arterial blood pressure (MAP). It is often recorded in brackets beside the blood pressure reading on monitors. The MAP reflects the perfusing pressure needed to maintain arterial blood flow and hence oxygen delivery to tissues and organs during each cardiac cycle. A MAP of 60mmHg is necessary to supply the coronary and cerebral arteries and the kidneys, which are more sensitive to pressure changes than other organs. The normal mean arterial pressure range is 70–110mmHg. At normal resting heart rates it can be calculated simply as the following equation in Anatomy & Physiology in Action: 2.2.

Anatomy & Physiology in Action: 2.2

MAP = systolic + (diastolic x 2)/3

Normal diastolic filling time is twice as long as the systolic time and is therefore measured twice. In patients who are unstable this is a less accurate method but trends are helpful in assessing how the organs are being perfused guiding clinical judgement and interventions.

In Scenario 2.1 Mr Bradbury's current cardiovascular status is reduced; this is identified through the reduced capillary refill time and the MAP, which is calculated at 56mmHg. In a young and fit person this MAP is relatively normal and is well tolerated, but even the young and fit can experience renal compromise if the insult is sustained or the MAP is reduced even further over a number of hours causing renal impairment and failure of other organs.

Developing and Delivering Expert Care: Exercise 2.10

Calculate the pulse pressure and MAP from the following blood pressure recordings

120/80 mmHg =
90/50 mmHg =
160/90 mmHg =
70/40 mmHg =

Anatomy & Physiology in Action: 2.3

Mean Arterial Pressure – Why is this so important?

Essentially there is no 'normal blood pressure' for many patients with cardiovascular disease. Healthcare professionals can get caught out if a 'normal' blood pressure is recorded which might not be 'normal' for that patient. Another way of defining 'normal' blood pressure is whatever pressure is required to maintain organ function. As we age that will certainly rise as arteries lose their elasticity and resistance increases. If the BP drops more than 30mmHg below 'normal' or premorbid blood pressure it should be considered as hypotension for that patient.

For example, if the BP is 180/90 –
MAP = 180 + (90 x 2) = 360/3 = 120mmHg

If the BP drops to 120/80 this would not be recognised as significant by healthcare professionals as it lies within the 'normal' range. Attempts to restore the BP are not undertaken and this can result in organ failure.

MAP = 120 + (80 x 2) = 280/3 = 89mmHg

As you can see there is a difference of about 30mmHg between the two MAPs; for some patients this may be the difference between maintaining the fine balance between adequate blood flow to the tissues and ischaemia and MODS.

Many patients can maintain perfusion even if the blood pressure is lower than normal; the important determining factor is whether the patient can maintain sufficient cardiac output and oxygen delivery to the vital organs over a period of time. Cardiac output is very difficult to measure outside of critical care areas. One much cheaper and simpler method is urine output. The kidneys are a very good indicator of adequate perfusion; you can measure the volume hourly, observe the colour and concentration, analyse the blood urea and electrolytes in relation to the history, clinical information and diagnosis.

Renal blood flow

Why are the kidneys so sensitive to blood pressure changes? Despite their innate ability to auto-regulate blood flow over a wide and variable blood pressure range (80–180mmHg), during periods of hypotension they cannot maintain this state for too long without ischaemia occurring. It's because they receive approximately 20–25% of the cardiac output per minute, which they are dependent upon. Normal cardiac output is roughly 5L/minute or 5000ml/minute. Renal blood flow is approximately 400ml/min/100g at rest. The kidneys require a high blood flow, as the primary function of the kidney is to filter blood and produce urine. Most of the filtration of the blood occurs in the cortex region of the kidney.

The hydrostatic pressure within the glomerular capillaries is much higher than in normal capillaries; this is important in maintaining the glomerular filtration rate within the Bowman's capsule. The kidney is richly innervated by sympathetic noradrenergic nerve fibres, which maintain the glomerular filtration rate (GFR) by altering the diameter of the afferent arterioles if arterial pressure falls. The afferent arteriole dilates to maintain blood flow and pressure within the glomerular capillary. Prostaglandin E_2 (PGE_2) is synthesised in the renal medulla cells and is a potent vasodilator. The kidney is able to maintain function and GFR by a combination of autoregulatory mechanisms across a wide range of pressures from 80–180mmHg and renal vasodilation.

In acutely unwell patients the urine output should be calculated using the following formula: 0.5ml/kg/hr.

It is essential that an accurate weight assessment is made to guide the amount of urine expected per hour.

Calculate the following:

Weight is 80kg. How much urine should the patient pass/hour?

80 x 0.5 = ml/hr

Ageing, arterial wall changes and other factors can affect the MAP especially in the elderly; this makes it more difficult for blood to flow through the arteries due to changes in the luminal wall of the artery. As the pressure is increased along the length of the arteriole, the MAP will also increase; over time a higher MAP is required to maintain arterial circulation to the organs. A sustained fall in MAP as a result of hypovolaemia, dehydration, sepsis or myocardial injury can lead to ischaemia resulting in organ failure.

Before discussing the homeostatic control of blood pressure it is important to include a brief overview of oxygen supply and demand. Outside of intensive care units it is difficult to monitor whether the oxygen supply to the tissues is adequate or not. When patients experience an inadequate oxygen supply to metabolically active cells an oxygen debt is accrued, which if not corrected can lead to chest pain and myocardial infarction, rhythm disturbances, confusion, gut ischaemia, organ failure and many other clinical manifestations associated with hypoxia and lactic acidosis. It is important as nurses that we appreciate and understand our role when recording observations, particularly the respiration rate and oxygen saturations which may be the first clinical warning signs that there is a problem; we need to act on these early warning signs by communicating this in a timely fashion to medical colleagues and to ensure that appropriate interventions are commenced to correct the problem and to restore homeostasis as quickly as possible.

Oxygen delivery

The primary function of the cardiovascular system is to maintain adequate blood supply, nutrients and oxygen essential for aerobic respiration and to remove the waste products of cellular functions. The delivery of oxygen to tissues is dependent on availability in the atmosphere, diffusion of the gas across the alveolar–capillary membrane, haemoglobin carrying capacity and finally the diffusion of oxygen down its concentration gradient across the cell membrane to be utilised by the mitochondria for energy or adenosine tri-phosphate (ATP) production.

Oxygen delivery can be represented by the equation outlined in Exercise 2.11.

The amount of oxygen delivered to the tissues in a healthy adult is about 1000ml/minute or 550ml/min/m^2. Normal oxygen consumption (VO_2) is 250ml/minute or 140ml/min/m^2 (Hinds & Watson 1996).

Anatomy & Physiology in Action: Exercise 2.4

$DO_2 = Q$ (Hb x 1.34 x SaO_2

DO2 = delivery of oxygen, Q = cardiac output

- Why is this important to maintain particularly in patients with homeostatic failure?
- What simple blood test is performed to analyse oxygen content in the blood?

Controlling and maintaining acid base balance

Let us now look at the acid base balance of fluids which is essentially the regulation of hydrogen (H^+) ion concentration of extracellular fluid (ECF). All cells are very sensitive to changes in H^+ ion concentration. You may have heard of the pH scale which is used to denote the H^+ ion concentration in solution or alternatively the number of moles per litre of molarity. The scale ranges from 0–14. It is logarithmic, that is each change of one pH unit equals a tenfold increase in H^+ concentration: this is an important point to remember that can have profound clinical implications. The pH of a solution is defined as the negative logarithm of the hydrogen ion concentration [H^+] in moles/litre or –log [H^+] (Marieb 1998).

Anatomy & Physiology in Action: 2.4

The normal or optimal pH range for arterial blood = 7.4
Venous and interstitial fluid pH = 7.35
Intracellular fluid pH = 7.0
Arterial blood gases actually measure the true plasma pH. Any reading below 7.40 is acidosis and above 7.40 is alkalosis.

Normal Arterial Blood Gas Values:
pH = 7.35–7.45
$PaCO_2$ = 4.5–6.0 kPa (kilopascals is the unit of measurement)
PaO_2 = 10.6–13.3 kPa
HCO_3 = 22–26mmols
Base excess or deficit (BE) = –2 to +2

Anatomy & Physiology in Action: 2.5

Understanding the Terminology: Acids and Bases

Acids are proton donors

Bases are proton acceptors

Strong acids e.g. hydrogen chloride (HCl) completely dissociate in H_2O which binds to H^+

Strong bases dissociate quickly

Weak acids e.g. carbonic acid (H_2CO_3) do not completely dissociate

Weak bases e.g. sodium bicarbonate ($NaHCO_3$) ionise incompletely

Bicarbonate is the most important inorganic buffer acting as an alkaline reserve

The equation below summarises the homeostatic balance between acid and base and the role of the main buffering systems of the body the lungs and kidneys. The arrows denote that the process is dynamic and bi-directional.

$CO_2 + H_2O \leftrightarrow H_2CO_3^- \leftrightarrow HCO_3^- + H^+$

Carbon dioxide production, transport and excretion

A by-product of respiration is carbon dioxide (CO_2) produced in large amounts by cellular processes; it is an important influence on the pH of the blood. We produce approximately 15,000 to 20,000 mmol of CO_2 each day or 200ml/minute (Marieb 1998). The body is constantly balancing the pH of the blood and ECF to maintain equilibrium.

CO_2 is transported by three mechanisms; 60% is converted into bicarbonate (HCO_3^-) ions formed by the reaction of CO_2 and H_2O at tissue level, this forms a weak acidic solution – carbonic acid (H_2CO_3). CO_2 is transported in solution in the blood, as carbonic acid rapidly dissociates into bicarbonate (HCO_3^-) and H^+ ions in the presence of an enzyme carbonic anhydrase found mainly in red blood cells. As the blood passes across the alveolar capillary membrane CO_2 diffuses from the capillary into the alveolar sac and is excreted by the lungs.

The body is very sensitive to minute changes in pH, CO_2 and O_2 levels. Excretion of CO_2 via the respiratory system is regulated by peripheral chemoreceptors found in the carotid bodies and aortic arch and central receptors in the medulla which are sensitive to the pH of the cerebrospinal fluid (CSF). CO_2 diffuses easily across the blood–brain barrier whereas H^+ and HCO_3^- do not. The response is mediated by the medulla, increasing impulses to the phrenic nerve which innervates the diaphragm and the intercostals nerves which innervate the intercostal muscles to increase the rate and depth of respirations within 1–3 minutes to rebalance CO_2 concentration.

Carbonate–bicarbonate buffering system

The kidneys are the main regulators of acid base along with proteins, blood (Hb) and acids which act as buffers to control sudden changes in pH. When the pH rises, H^+ ions (act as acids) are released to mop up the excess base. When the pH drops (acidosis) H^+ ions bind to raise pH back to normal. This results in shifting the blood pH within normal limits as H+ is removed or added to blood. Why is the H^+ ion concentration so important? Essentially cells can become denatured and destroyed within a few hours if the pH is <6.8 or >8.0 even for a few hours. The kidneys may take a few hours or even days to alter the pH. What might happen to the delicate balance between acid and base if the patient has renal failure?

The phosphate buffer system is present in low concentrations in the ECF. The buffering systems include sodium salts of dihydrogen phosphate ($H_2PO_4^-$) and monohydrogen phosphate (HPO_4^{2-}) which are effective intracellular fluid buffers when phosphate levels are high. 75% of all buffering occurs within the cells and plasma proteins. Amino acids are also involved in buffering; groups of atoms carboxyl (-COOH) dissociate and release H^+ ions as the pH rises, amino acids act as bases and accept protons (H^+) to reduce the acidity of solutions. The

kidneys have a number of functions maintaining acid-base homeostasis. They excrete other acids generated by cellular metabolism e.g. phosphoric, uric and lactic acid and ketone bodies. They regenerate new bicarbonate ions and replenish the stores as HCO_3^- is excreted from the lungs.

Developing and Delivering Expert Practice: Exercise 2.12

There are four main blood gas derangements:

- Respiratory acidosis
- Respiratory alkalosis
- Metabolic acidosis
- Metabolic alkalosis

Can you identify the causes of each of the above acid base disturbances and also determine whether the pH will be acidic or alkaline, whether the CO_2 and HCO_3^- will be elevated or lowered?
Interpreting blood gases is complicated and takes time, in your clinical areas collect some examples and see if you can work them out. Get a colleague to check your answers.

In summary

Acid base balance is a dynamic and constantly changing process. It is dependent on functioning lungs and kidneys, adequate haemoglobin and steady homeostatic states. When patients experience an insult such as Mark Bradbury has, this fine balance is disrupted and compensatory mechanisms are activated. If the insult is overwhelming despite the activation of the above mechanisms, decompensation will occur which may result in respiratory and/or cardiac arrest if uncorrected.

Control of insulin and glucose release

The pancreas is both an endocrine (consists of ductless glands that empty hormones directly into the bloodstream) and exocrine gland releasing pancreatic juice into the small intestine for digestive purposes. Millions of pancreatic islets – Islets of Langerhans – produce pancreatic hormones from the following cells:

- Alpha cells – Involved in glucagons synthesis
- Beta cells – Insulin production
- Delta cells – Growth hormone inhibiting factor (GHIF) somatostatin.

We are going to focus on the endocrine function of the pancreas; the release of insulin in response to raised blood glucose levels.

What is insulin?

Insulin is comprised of a 51 chain of amino acid synthesised by the beta cells. It is synthesised as a polypeptide chain proinsulin. Insulin has a very short half-life of 5 minutes. Eighty per cent of insulin is degraded by the liver and kidneys. Raised blood levels of glucose stimulate the release of insulin. The activation of insulin causes glucose to enter target cells. Insulin triggers enzyme activity which catalyses the oxidation of glucose for ATP production, enhances glycogenesis and converts glucose to fat (lipogenesis). Insulin stimulates amino acid uptake, protein synthesis and fat storage.

Developing and Delivering Expert Care: 2.1

Physiological effects of insulin

- Lowers blood sugar levels – (accelerates glycogenesis)
- Increases the membrane transport of glucose into cells
- Influences protein and fat metabolism[

Insulin inhibits

- Glycogen breakdown to glucose (glycogenolysis)
- Amino acid and fatty acid conversion to glucose (gluconeogenesis)

Hyperglycaemia – defined as beta cells secreting insulin in response to

- Catecholamines (epinephrine)
- Growth hormone
- Glucagon
- Thyroxine

Gluconeogenesis 'new glucose'

Gluconeogenesis is the synthesis of glucose from lactic acid and non-carbohydrate molecules e.g. fatty acids, glycerol and amino acids. The release of glucose by liver cells into the blood causes the blood glucose level to rise.

Developing and Delivering Expert Care: 2.2

Different Types of Shock

Hypovolaemic shock may result from a number of reasons, from haemorrhage as in the clinical case or from other factors as listed below:

- Exogenous – burns, trauma, sepsis
- Endogenous – interstitial loss or third space fluid shifts
- Increased capillary leakage

The main clinical characteristics are a reduction in circulating volume, reduced venous return, reduced stroke volume and cardiac output and ultimately blood pressure. The only simple bedside measurement available is blood pressure. However as we will discover later on when we review the compensatory mechanisms for maintaining blood pressure, hypotension is often a late sign and can be misleading, as the body may have experienced a significant loss of circulating volume before the blood pressure changes.

Cardiogenic shock is defined as a systolic arterial pressure <90mmHg, and in a patient with known hypertension a fall of more than 30mmHg from their premorbid reading. Other features include reduced renal perfusion, urine output <20ml/hour, cold and peripherally vasoconstricted, changes to mental state – i.e. new onset confusion and signs of myocardial failure such as raised jugular venous pressure, pulmonary oedema and basal crepitations.

Causes:

- Acute myocardial infarction
- Cardiomyopathy
- Myocardial dysfunction post cardiac surgery
- Mitral and aortic regurgitation

Obstructive shock is defined as a fall in cardiac output due to mechanical obstruction to the circulation or by restriction of ventricular filling and ejection

- Cardiac tamponade – is the accumulation of fluid or blood in the pericardial sac, it acts like a tourniquet around the heart or myocardium reducing the force and contractility of the ventricular contraction and so less blood is ejected /beat.
- Pulmonary embolism – can completely occlude the pulmonary capillary circulation casing catastrophic circulatory collapse.

In both scenarios severe and refractory hypotension is the main clinical feature associated with distended neck veins, cyanosis, tachycardia, severe respiratory distress (in PE) and if untreated both situations can lead to cardio-respiratory arrest and death.

Distributive shock – is essentially related to the mal-distribution of blood flow caused by severe and prolonged vasodilation and changes in vascular resistance as a result of anaphylactic reaction to antibiotics, blood transfusions or allergies. Other factors such as sepsis or spinal cord injury where there is a loss of vascular tone and capacitance.

The rise in blood glucose inhibits glucagon release and stimulates the secretion of insulin. Glucagon is released by alpha cells in response to a fall in blood glucose levels and is another example of a negative feedback system. Secretion is inhibited by GHIF (somatostatin). Glucose enters cells by facilitated diffusion. Normal venous glucose levels are 3.6–6.1mmol/L.

Figure 2.5 summarises the example of a negative feedback system. Patients who are experiencing high levels of stress may have a stress induced hyperglycaemia

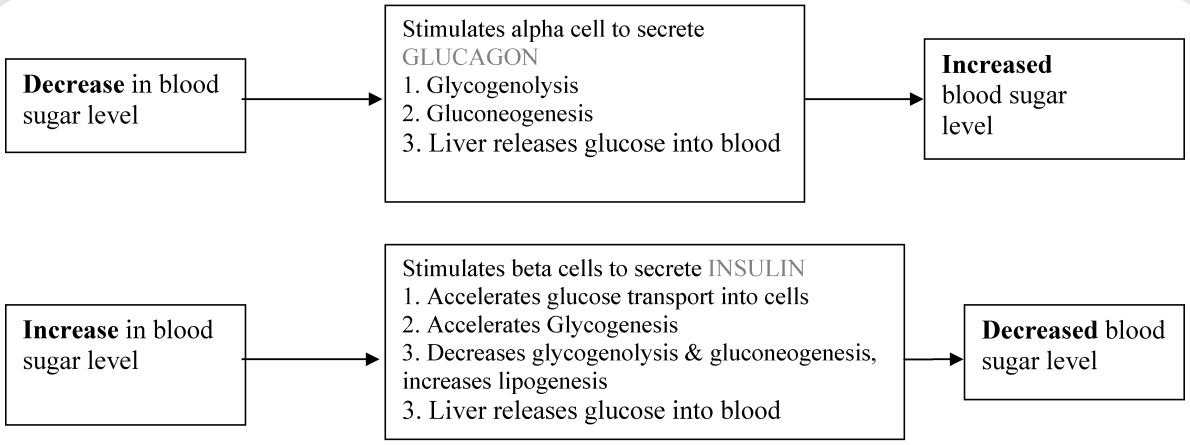

Figure 2.5 Negative feedback system: blood glucose and control

as a result of sepsis, catecholamines, glucagons, cortisol and growth hormone; this is due to excessive glucose release.

Defining shock/haemodynamic collapse

Patients may experience circulatory collapse for a variety of reasons: this is commonly known as shock. Physiological shock can be defined as an inadequate circulatory volume to facilitate the oxygenation and nutritional needs of the body's cells; this internal insult is intensified by the insufficient removal of the waste products of cellular metabolism (leading to metabolic acidosis). Initially this can result in compensatory homeostatic responses, which aim to counteract the internally identified deficit or surplus of waste products. If homeostasis is not restored, the internal environment will become increasingly hypoxic and acidotic. This acidotic environment is not compatible with cellular function, initially resulting in cellular injury. Depending on the insult this process can be instantaneous or have a gradual accumulative affect escalating from cell to tissue damage, and progressing into multi-organ failure.

In clinical practice all serious illnesses have the potential to result in reduced cellular perfusion producing physiological shock.

Now that you have gained insight into the different types of shock the chapter will progress into discussing the protective and compensatory mechanisms the body can deploy to avert the problems associated with shock such as tissue necrosis and the initial patient assessment process will be discussed, providing the reader with a

Developing and Delivering Expert Care: 2.3

Stages of Shock

There are essentially four stages of shock:

- **Initial stage**: tissues are poorly perfused; there is a decreased cardiac output (CO), increased anaerobic metabolism and lactic acid formation
- **Compensatory stage**: is still reversible. Low CO activates sympathetic nervous system and compensatory mechanisms are deployed to improve tissue perfusion
- **Progressive stage**: compensatory mechanisms are inadequate and failing. There is profound vasoconstriction, with severe metabolic acidosis, CNS and myocardial depression.
- **Irreversible or refractory stage**: cellular necrosis and multiple organ failure are evident and non-retractable.

As healthcare professionals it is important that we can recognise the different signs and stages of shock to prevent and anticipate further problems, to communicate effectively to other professional groups and to proactively intervene with appropriate and life saving treatments.

structured approach to patient care that can be applied throughout their developing career.

Blood pressure

The blood pressure is normally regulated by several complex and inter-related neural, chemical and hormonal mechanisms. It is an example of a negative feedback system.

Using a case study the compensatory mechanisms initiated during major haemorrhage will be discussed. In cardiogenic shock the problem is not hypovolaemia induced hypotension but the lack of response by the heart muscle to the stimulatory effects of adrenaline and noradrenaline. Compensatory mechanisms are activated but are ineffective. The same is seen in septic shock when the peripheral vasculature cannot vasoconstrict the arteries in response to sympathetic stimulation, compensation is ineffective resulting in a high cardiac output, tachycardia, hypotension and vasodilation or reduced systemic vascular resistance (SVR).

Hypovolaemia induced hypotension

Changes in blood pressure are detected by baroreceptors found in the arch of the aorta, carotid sinus and all large arteries of the neck and thorax. The first part of the reflex to be activated is the baroreceptors or 'stretch' receptors, which are sensitive to changes in the stretch or pressure exerted by fluctuations in the volume of blood. When blood pressure falls the receptors detect a change in the amount of 'stretch', there is an increased rate of impulses firing via afferent pathways back to the vasomotor or cardio-acceleratory centre within the medulla oblongata. Cardiac output, as discussed in Chapter 4, is the heart rate multiplied by the stroke volume, stroke volume being the amount of blood ejected per ventricle per beat. The stroke volume falls during haemorrhage as does the cardiac output, what is seen clinically is a fall in systolic blood pressure, and a narrowing of the pulse pressure. To compensate for the reduction in stroke volume, the heart rate is increased to distribute less blood faster around the body to the major organs to maintain oxygen delivery, perfusion and ultimately organ function.

How is the heart rate increased?

The heart rate is usually under the autonomic control of the parasympathetic or vagal nerve fibres (see Chapter 4 for more detail). The medulla receives information from baroreceptors and chemoreceptors via afferent nerve pathways on the volume and pressure status within the cardiovascular system. During times of emotional or physical stress and illness the sympathetic nervous system, via efferent vagal fibres, induces the 'fight or flight response' by releasing noradrenaline from nerve fibres which bind to beta one (β_1) adrenoreceptors within the heart muscle to increasing the force of contraction (inotropy) and the heart rate (chronotropy). The adrenal medulla synthesises adrenaline and noradrenaline, which circulate in the blood stream. Adrenaline binds to β_1 adrenoreceptors also increasing heart rate and contractility. At higher concentrations of adrenaline it binds to alpha (α_1) adrenoreceptors causing peripheral smooth muscle vasoconstriction. As the rate of impulses from the SA node is increased, the conductivity of the impulse through the AV node also has to increase. In other words the gate (AV node) is opened to let more impulses pass through so that the ventricles can contract faster to raise the cardiac output.

Noradrenaline also exerts a vasoconstricting effect on the vasculature by binding to a_1 adrenoreceptors within the tunica media (middle arterial wall lumen) to bring about vasoconstriction of the vascular smooth muscle and increased systemic vascular resistance. Vasoconstriction shunts blood away from the peripheral circulation and the gut to the core organs; the clinical signs exhibited are cold, shutdown peripheries with an increased capillary refill time, as demonstrated by the clinical case in Scenario 2.1. The redirection of blood flow away from the mesenteric circulation of the gut is very

Developing and Delivering Expert Practice: Exercise 2.13

In the elderly due to SA node disease and conduction problems this may not be evident.

- Why might this be a problem if a patient is bleeding and the heart rate did not increase?

Patients on beta blockers will not be able to increase their heart rate as a natural response why not?

What effects does beta blockade have on sympathetic stimulation?

Refer to the *British National Formulary* (BNF) for your answer.

difficult to measure and quantify, but the patient may exhibit signs such as nausea and vomiting.

Overall the cardiovascular effects of noradrenaline on the blood pressure are increased blood pressure, MAP and pulse pressure. The collective term for noradrenaline and adrenaline is catecholamine; high catecholamine levels in the blood also stimulate the release of renin which is an important polypeptide released in low perfusion states by the kidney as part of the compensatory mechanisms for blood pressure control. While the body is attempting to maximise and increase available circulating blood volume it also attempts to reduce urine output and water loss by stimulating other hormones such as aldosterone and anti-diuretic hormone to conserve water.

Renin-angiotensin-aldosterone mechanisms

In response to a significant drop in renal artery pressure below 80mmHg, such as in Scenario 2.1, the low blood pressure and MAP would stimulate the kidney's own tubuloglomerular feedback mechanism which is coordinated by the macula densa cells of the juxtamedullary apparatus (JMA). The reduced stretch on the juxtaglomerular cells is similar to the effects of baroreceptors. Other mechanisms such as increased B_1 adrenoreceptor stimulation brought about by sympathetic activity from the renal nerve triggered by shock and the kidney detects haemorrhage. The vasodilation of the afferent arteriole is also another indicator that something is wrong with blood flow. When the GFR, arterial blood pressure and circulating volume fall as in our case scenario due to haemorrhage, there is a decrease in the delivery of sodium and chloride ions to the distal convoluted tubule which is detected by the macula densa cells. Remember they control and initiate the kidney's own feedback mechanism. A fall in the plasma concentrations of sodium and potassium ions also stimulates the secretion of renin. The combination of all these mechanisms causes renin to be released from the juxtaglomerular cells. Renin has a direct action on and causes afferent arteriole constriction.

Renin's main purpose is to bring about the release of the enzyme angiotensinogen from the liver, which undergoes a catalytic change to form angiotensin I. It circulates throughout the blood; certain endothelial membranes, particularly within the lining of the lung, produce an enzyme called angiotensin-converting enzyme (ACE). When the angiotensin comes into contact with the enzyme it converts angiotensin I to angiotensin II, a powerful vasoconstrictor. Further peripheral vasoconstriction occurs by increasing the systemic vascular resistance and raising the blood pressure. Angiotensin II also causes vasoconstriction of the efferent arterioles increasing the glomerular capillary pressure in the Bowman's capsule to maintain GFR. The glomerular filtration rate is the amount of filtrate produced per minute by the kidneys. Normal GFR is 120–125ml/minute or about 7.5L/hour. Ninety-nine per cent of filtrate is normally reabsorbed; normal urine production per day is 1–1.5L. The reason why maintaining the GFR is so crucial is that the GFR is directly proportional to the net filtration pressure, this means that even small changes in the hydrostatic pressure of the glomerular capillary or the large surface area available for filtration will affect the rate at which solutes are removed from the filtrate and the reabsorption of water and other substances. Filtration can cease completely with relatively small changes in blood pressure. It is also important to remember that the kidneys have a high demand for oxygen and are very sensitive to changes in oxygen delivery which can result in pre-renal failure, ischaemia or acute tubular necrosis (ATN).

Angiotensin II also acts on the adrenal cortex to release aldosterone, a mineralocorticoid. The function of aldosterone is to increase the reabsorption of sodium and water in the distal convoluted tubules in the kidney in response to low blood pressure and low circulating blood volume. As a result of this the urine becomes dark and concentrated as water is removed.

Anti-diuretic hormone

Angiotensin II also stimulates the release of anti-diuretic hormone (ADH), or vasopressin, from the posterior pituitary. Anti-diuretic hormone inhibits or affects the production of urine. The hormone is released when water needs to be conserved. ADH exerts its effect on the lining of the distal convoluted tubule (DCT) and collecting ducts by increasing water permeability. Water moves out of the DCT and collecting ducts by osmosis and is reabsorbed by the peritubular capillaries and transported into the circulation to raise the circulating volume. The removal of water also causes sodium to move across and so both water and sodium are reabsorbed. ADH is also triggered by pain and emotional stress, hypoxia, severe exercise and surgery. In the absence of ADH dilute urine is formed, this is called diabetes insipidus. Excessive ingestion of alcohol inhibits ADH production causing the loss of large volumes of urine, intense thirst and a dry mouth.

Developing and Delivering Expert Practice: Exercise 2.14

Diabetes insipidus is a syndrome of ADH deficiency. It is characterised by the production of large amounts of dilute urine leading to dehydration and unrelieved thirst. It can be caused by damage to the hypothalamus or the posterior pituitary gland as a result of raised intracranial pressure from head injuries.

ADH is also released by the hypothalamus in response to signals from osmoreceptors located in the hypothalamus, which constantly detect the concentration of solutes in the blood. When the solute concentration is increased the osmoreceptors send impulses to the hypothalamus to secrete ADH. The thirst mechanism is also stimulated so that more water is consumed to balance the concentration of the solutes.

Structured initial assessment

One of the principal skills required by all nurses is the ability to carry out a timely, but comprehensive, patient assessment. The focal point is to ascertain the individual care needs of the patient and prioritise subsequent treatment. To function effectively the practitioner requires excellent communication skills, and a thorough understanding of how pathology and trauma can affect the internal environment and its equilibrium leading to the clinically recognised state of physiological shock.

The initial assessment

There are numerous assessment models, or mnemonics, currently adapted to suit the needs of acutely ill patients and clinical practice. The Resuscitation Council in the United Kingdom (RCUK 2006) recommends the universal adoption of the ABC/DE structure, where each category addresses a separate, but intrinsically linked body component.

Vital signs

Vital signs directly demonstrate the current physiological state of the patient, yet research has clearly demonstrated the inability of healthcare professionals to correctly interpret or act on recorded data. The most common mistake nurses make is to underestimate the seriousness of the clinical data they have recorded; this is of particular importance in relation to the blood pressure due to an over-reliance on this one component. Many clinicians fail to recognise the subtle signs of deterioration associated with shock until there is a dramatic decrease in the blood pressure. The respiratory system is the first to demonstrate compensation therefore the application of the

Developing and Delivering Expert Practice: Exercise 2.15

ABCDE Mnemonic

A = Airway and cervical spine immobilisation (Chapter 15)
When assessing a patient's potential to maintain their own airway, think about why they have been admitted or why they need assessment. This will provide vital clues as to what you might expect. For example if the patient has been involved in an accident they may have sustained a head injury thereby rendering them unconscious, they may have trauma to there mouth and bleeding. The mechanism of injury provides vital clues!

B = Breathing and Ventilation (Chapter 3)
Refer to Chapter 3 which describes the full process

C = Circulation and Haemorrhage control (Chapter 4)
A circulatory assessment should always focus on initially looking at the patient for signs of life and perfusion (Chapter 15). Always feel their pulse to ascertain the Rate, Rhythm and Depth.

D = Disability and neurological assessment (Chapter 5)
Chapter 5 provides in-depth detail on a neurological assessment. In the short term the AVPU tool can be applied.

E = Exposure and environment
No assessment is complete without recording the patient's temperature (an indicator of the internal environment); depending on the type of presentation the nurse may need to see the front and back of the patient to look for signs of injury – remember dignity and respect, in addition a chaperone may be required.

A.B.C. approach will identify deficits early into the assessment process (the reader is referred to Evans & Tippins 2006 for in-depth patient assessment techniques and scenario based demonstrations).

Possible misleading data

Many factors can affect the reliability of vital signs data and clinicians need to take this into consideration when interpreting data, these include any other internal mechanisms that initiate the fight or flight response. These mechanisms mimic the instigation of the compensatory phase of shock i.e. pain, fear, anxiety and stimulants. In contrast several commonly prescribed medications can inhibit the compensatory response, these include beta blockers.

Developing and Delivering Expert Practice: Exercise 2.16

Level of Consciousness
A = Alert
V = Responds to a Verbal Stimulus
P = Responds to a Painful Stimulus
U = Unresponsive

Capillary refill time (CRT)

The recording of a CRT in addition to a radial pulse is an excellent indicator of peripheral perfusion. The patient's nail bed or the pulp of the finger is compressed for five seconds with sufficient pressure to cause blanching. Once pressure is released the nail bed should be engorged with blood and return to normal.

- In adults a CRT of two seconds or less will indicate good peripheral perfusion
- A prolonged CRT should be interpreted in conjunction with other circulatory parameters, such as pulse rate, blood pressure, and conscious level (RCUK 2006).

This figure can be prolonged in the elderly, in a cold environment, and in the presence of poor ambient lighting or circulation-based anomalies such as Raynaud's phenomenon.

Postural blood pressure

Under normal conditions autonomic reflexes stabilise an individual's blood pressure from the lying to sitting/standing positions. This can be seen as the initiation of quick response mediators that result in peripheral vasoconstriction and shunting of blood into the main circulation. When there is an established loss of circulating volume in the peripheries this mechanism fails and a fall in blood pressure results, the recording of a postural blood pressure, therefore, is an excellent eliminative tool to the practitioner suspecting a patient to be in the early stages of shock.

Example of a patient experiencing reduced circulating volume:

- Lying B/P 105/70, H/R 90
- Sitting B/P 85/58, H/R 115

A positive result is achieved if the patient's blood pressure demonstrates a systolic drop of >20mmHg from lying to standing, or >15mmHg from lying to sitting, although the differential may be slightly less, this is offset by dangling the patient's legs over the trolley and the reduction in risk potential. Rises in heart rate exceeding 20 beats per minute (bpm), or a diastolic drop exceeding 10mmHg are also positive indicators.

If circumstances allow lay the patient supine for five minutes, record a blood pressure and heart rate, then assist them to sit up, allow 1–3 minutes then record vital signs with the patient's legs dangling over the side of the trolley. Be aware of the possibility that your patient may become acutely unwell and document any changes to the initial assessment findings.

Defining the cause of shock

Defining the cause of shock will determine the focus of treatment, as the causative agent will need to be dealt with to combat the pathophysiological processes taking place. Shock is a progressive state and, therefore, early identification facilitates early intervention, which improves the patient's prognosis. A dramatic example is demonstrated in the management of meningitis. If left untreated until obvious signs like a haemorrhagic rash are evident, the long-term prognosis is poor.

Conclusion

Maintaining homeostasis is a dynamic process affecting all organs and cells of the body. It comprises complex, inter-related and interdependent systems that respond to the internal and external environment. As nurses our role is to understand the complexities of the chemical, neural and hormonal interplay and how this can manifest as a single disease or as a result of multiple pathologies. Clinical treatments may range from simply correcting hypoxaemia or fluid deficits to the instigation of multi-organ support systems. Our role is to recognise, administer and constantly evaluate prescribed treatment until the fine balance of homeostasis is achieved.

The combined efforts of vasoconstriction of the smooth muscles, the redirection of blood flow, raising of the heart rate, and the complex interplay between the kidneys, adrenal glands and the hypothalamus, all assist in raising blood pressure and maintaining vital oxygen and nutrient supply in potential times of crisis. By applying the structured assessment process discussed, the nurse can identify varying degrees of illness at an early stage and initiate a plan of care based on a working diagnosis which is open to change as the assessment process broadens to encompass specialists and various members of the multidisciplinary team. Complete the following quiz to see direct evidence of your professional development and identify areas in need of further development.

Chapter 2 Summary Quiz

1. What is homeostasis?

A. The brain's ability to speed things up
B. The state of functional equilibrium within the body's internal environment B
C. The state of gases within the body
D. The equilibrium of the venous and arterial blood supplies

2. Catecholamines describe hormones such as:

A. Noradrenaline and adrenaline
B. Glucose and oxygen
C. Blood and lymph
D. Vasopressin and methohaemoglobin

3. Normal body temperature ranges between:

A. 31.6ºC–36.8°C
B. 35.6ºC–37.8°C B
C. 36.6ºC–39.8°C
D. 35.6ºC–36.9°C

4. Which of the following can result in a fever?

A. Infections
B. Inflammation
C. Allergic reactions
D. All of the above

5. A MAP of above what pressure is needed to supply the coronary and cerebral arteries and the kidneys?

A. 90mmHg
B. 10mmHg
C. 60mmHg
D. 120mmHg

6. Body fluids function optimally within a narrow range, at an arterial blood pH ranging between

A. 7.0 and 7.9
B. 0 and 14
C. 7.45 and 7.55
D. 7.35 and 7.45

7. Physiological shock can be defined as:

A. An inadequate circulatory volume to facilitate the oxygenation and nutritional needs of the body's cells
B. Not enough blood to produce urine
C. An inadequate circulatory volume to facilitate the process of haemofiltration
D. A severe fright causing stress

8. If a patient was demonstrating compensatory physiological shock, which of the following blood pressures would be expected?

A. 120/80
B. 50/30
C. 70/60
D.84/59

9. How long should an area of skin be pressed on to identify the capillary refill time

A. 1 minute
B. 10 seconds
C. 20 seconds
D. 5 seconds

10. Which demonstrates an easy to remember tool for both respiratory and cardiovascular assessment?

A. Rate, rhythm and depth
B. Rate, listen and feel
C. Look listen and cross
D. Feel, count and write

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