therefore when using sedation to concentrate on the adequacy of sedation and analgesia and also to ensure the patient is getting sufficient REM sleep.

Sedation of patients needs to be adequately assessed at regular intervals. To undertake this, the Richmond Agitation Sedation Scale (RASS) has been devised (Table 2.7). The procedure for RASS assessment can be viewed in Table 2.8.

The higher the score, the more agitated or restless the patient is and is in need of intervention. Positive scores require an increase in sedation and a negative score requires a reduction in sedation. Ideally, the patient should score 0 with the aid of nursing and possibly pharmacological intervention.

It is important to note that frequent objective sedation assessments are vital within the critical care setting, using a valid and reliable sedation assessment tool. This is vital in ensuring that the critical care nurse correctly assesses the patient's sedation depth and titrates the sedation infusion dose to avoid severe cardiorespiratory depression.

### 2.3 Haemodynamic monitoring

#### Non-invasive Cardiovascular Haemodynamic Monitoring

#### Temperature

Nursing and medical interventions are commonly based on temperature recordings which, if erroneously made, can lead in extreme instances to an elevated temperature being unrecognised (Edwards 1997a). A sound understanding of temperature measurement and influencing factors is essential for nurses who care for patients in critical care.

When taking the temperature, it is the temperature set by the hypothalamus which is attempted to be determined. The pulmonary artery temperature measurement is suggested to be the most accurate way of measuring hypothalamic set point temperature. This form of temperature measurement is available in critical care settings and if inserted this temperature is generally recorded.

Other temperature sites which are in close proximity to the brain (axilla, oral, eardrum) and tympanic membranes also reflect the brain's thermal environment. The rectal temperature is proposed to be the most accurate tympanic membrane measure of hypothalamic temperature, and is used in critical care when the patient is unconscious. In this instance rectal temperatures are useful as long as the probe is placed correctly. As it is uncomfortable and embarrassing for conscious patients it is promptly removed when the patient regains consciousness. The measured rectal temperature is consistently higher than oral or axilla temperatures, a difference usually in the region of 0.3–1°C.

The axilla temperature is more convenient, but is generally not commonly used. This is because the axilla temperature is considered to be a skin temperature and not adequate as an indicator of core temperature. However, peripheral skin temperatures are used for determining vasoconstriction or vasodilatation to help assess the adult patient's circulation status.
The sublingual route is rarely used as patients are intubated orally or nasally and as such are unable to comply with this type of measurement. The electronic thermometer is becoming increasingly popular to replace the traditional mercury-filled device. In addition, single-use chemical thermometers are also available, which work by using a chemical that changes colour with increasing temperature.

A tympanic membrane closer to the brain is the ear, a temperature site that is becoming increasing popular in critical care. It uses tympanic membrane thermometry and is known as the infrared light reflectance thermometer. It detects the temperature within the eardrum. This site of measurement has clear advantages: the close proximity of the measurement site to the hypothalamus, convenience, comfort, rapidity and acceptance by the patient. It registers in a matter of seconds with little inconvenience and no discomfort to the patient. Inaccurate readings usually occur due to inconsistent measurement techniques by clinicians. A temperature is recorded to determine if it is normal, high or low. It is often assumed that, when it is high, the person has an infection, but this is not always the case. (Edwards 2003a). This will be reflected in the peripheral skin temperature, as it gives a good indication of the presence and severity of a circulatory defect. The toe temperature gradient provides a valuable, inexpensive and non-invasive monitor of tissue perfusion.

Skin temperature can be used to determine the severity of shock:

- During hypovolaemia circulation to the major organs and central temperature needs to be maintained.
- Under ANS control improves the circulation through:
  - Baroreceptor activity – vasoconstriction
  - Noradrenaline – receptors causes further vasoconstriction.

The end result is:

- heat conservation
- cool extremities that feel cool to touch
- an increase in BP
- improved circulation to the body’s major organs.

**Pulse**

The rhythmic contraction of the left ventricle of the heart results in a transmission of a pressure impulse through the arteries. This pulse is customarily palpated at the radial artery in the wrist. The important factors to consider in relation to the radial pulse are:

- Rate
- Rhythm
- Pressure (volume)
- Deficits with apex rate.

The pulse rate is an important component of cardiac output. Fluctuations of pulse rate in the well individual normally occur together with fluctuations in stroke volume.
to maintain optimum cardiac output for the activity being performed, for example, rest or exercise. In the resting adult, the pulse rate would normally be about 70 beats per minute. A rate greater than 100 beats per minute is termed a tachycardia, and a rate less than 60 beats per minute is termed a bradycardia.

If an altered pulse does not produce signs of haemodynamic changes it is not necessary to treat it, but if the patient does show such signs, e.g. volume depletion, immediate treatment is indicated. This may include drug or intravenous infusion therapy or non-pharmacological measures can be used, such as the Valsalva manoeuvre or the physician may perform carotid sinus massage.

The rhythm of the pulse may vary normally with respiration, especially in young adults, so that the pulse is irregular, speeding up at the peak of inspiration and slowing down with expiration; this is termed sinus arrhythmia. An irregular pulse is commonly categorized into the following rhythms:

- Regularly irregular
- Irregularly irregular.

A regularly irregular pulse is most likely to be caused by ectopic beats (a beat originating from a site other than the sino-atrial node) which occur prematurely. If they persist in an acutely ill person, the medical staff will require notification, as they can be indicative of increased cardiac irritability. This may be due to ischaemia or drugs (such as digoxin), increased sympathetic activity as a result of stress (for example hypoxia), or they may be related to potassium imbalance, all of which require further investigation. An irregularly irregular pulse usually indicates atrial fibrillation where atrial behaviour is chaotic and disorganized and the transmission of impulses to the ventricles is irregular.

The importance of using the pulse as an early reliable indicator of physiological change is often overlooked and a greater significance put on the blood pressure (BP). Yet, the pulse rate is less invasive and less time-consuming and the pulse is measured more accurately.

**Pulse pressure**

This is a wave of pressure caused by a sequence of distension and elastic recoil in the wall of the aorta, which forces blood rapidly down the systemic arterial system. It determines the strength of force of the pulse and it can be defined as the difference between the systolic and diastolic blood pressures.

When the pulse pressure is low, the strength of the pulse may be feeble and thready, e.g. in hypovolaemia. When the pulse pressure is high, the pulse strength may be bounding and the person experiencing this may feel palpitations or a pounding heart:

- increased by increased stroke volume (SV) during exertion
- increased by arteriosclerosis (loss of elasticity).

The pulse deficit is the difference between the heart rate counted at the apex of the heart using a stethoscope and the pulse rate counted simultaneously at the wrist. For the majority of patients the heart rate and pulse rate will be the same, but a deficit will occur in:

- atrial fibrillation
- multiple ectopic beats.

**Peripheral pulses**

There are many pulses in the body where an artery surfaces over a bony
protrusion. The main pulses are: apical; radial; carotid; femoral; brachial; aortic; popliteal; dorsalis pedis. The femoral and carotid pulses are important when establishing the adequacy of cardiac output, for example in someone who has suddenly lost consciousness due to possible cardiac arrest. The brachial pulse is used to measure blood pressure; and the pulses of the lower limbs, the popliteal pulse located behind the knee and the dorsalis pedis and posterior tibial pulses in the feet are important in determining adequacy of perfusion to the lower limbs.

By feeling these pulses a critical care nurse can determine if a pulse is present, absent, strong and equal, faint and equal, any weakness or a bounding feeling as if there is a great pressure within the artery, whether it is fast or slow or irregular. These will all give the nurse indications as to whether perfusion is inadequate or over-supplied, each giving the nurse clues to the overall circulation of each individual area of the body.

**Blood pressure (BP)**

By definition, blood pressure is the force exerted by the blood on the walls of the vessels in which it is contained (Edwards 1997b). It varies with age, gender, weight, stress level, mood, posture, physical activity. BP also varies through the heart and vascular system. It is highest and most variable in the aorta and other elastic arteries, decreasing through arterioles and capillaries. A number of factors, most significantly cardiac output, peripheral resistance, elasticity of vessels and hormonal and chemical control mechanisms determine it. Maintenance of an adequate blood pressure is essential to permit perfusion of the brain, and the coronary arteries, and the production of urine by the kidneys.

However, in the person admitted to critical care, the homeostatic mechanisms, responsible for maintaining optimum blood pressure (Table 2.9) may be stretched to their limit, fail to function, or be interfered with by drugs. The consequences of not being able to maintain an adequate blood pressure may lead ultimately to cerebral hypoxia, cardiac failure, acute renal failure and multi-system failure. These states occur as a result of prolonged hypotension (a low BP) or hypertension (a high BP).

**Hypotension** will only occur when all of the homeostatic mechanisms are exhausted. It may occur in hypovolaemia where there is a diminished circulatory fluid volume. Hypovolaemic shock is the state that results from hypovolaemia and is a further decrease in the circulating fluid volume so large that the body’s metabolic needs cannot be met.

**Hypertension** is consistent elevation of systemic arterial BP. This can be equally harmful to the patient in the acute setting, especially if it results in the breakdown of a recent surgical anastomosis or increases the work of a damaged myocardium. The generally agreed values for the upper limits of a normal BP is 140 systolic and 90 diastolic. Hypertension can affect the circulation by damaging the wall of the systemic blood vessels, stimulating the vessels to thicken and strengthen to withstand the stress, this gradually narrows the lumen of the blood vessels, and can lead to heart disease or intra-cerebral haemorrhage (stroke).

Use a Doppler if difficult to palpate a limb pulse

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Patient assessment and investigations

Haemodynamic monitoring

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Blood pressure (BP)

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Increasing hypertension can also be indicative of raised intracranial pressure (when combined with a simultaneous decrease in pulse rate). The increasing blood pressure in this instance is a protective measure to maintain cerebral perfusion if the intracranial pressure increases following head injury, anoxia or space occupying lesions.

Monitoring BP is an important facet of the critical care nurse’s role as systolic pressure reflects the adequacy of cardiac output, and diastolic pressure reflects the peripheral resistance exerted by the arterioles, measured in millimetres of mercury. Measuring the BP remains one of the most important and widely used assessment tools in hospital, as from this one test much information can be gleaned about the patient’s state of health.

In critical care areas the standard mercury blood pressure machines are not generally used. The majority of patients have an arterial pressure monitor inserted, whereby the patient has a needle inserted into an artery (the radial) which is attached to a transducer and plugged into the monitor. The consequence is an arterial pressure waveform and a continuous read out of BP (see later). This line is attached to a pressure bag and is also used as access for blood analysis.

### Respiration

The respiratory rate in adults is normally between 8 and 18 breaths per minute. Counting should be over a minute and take place when the patient is resting and unaware of the observation, since conscious awareness of breathing can lead to alteration in rate and pattern. This is because breathing is under the control of both the involuntary and voluntary nervous system.

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**Table 2.9** Summary of homeostatic mechanisms that govern BP

<table>
<thead>
<tr>
<th>Control</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control of resistance via the sympathetic nervous system, maintains vasomotor tone in all vessels</td>
<td>Directly via baroreceptors</td>
</tr>
<tr>
<td>Indirectly via chemoreceptor</td>
<td></td>
</tr>
<tr>
<td>Chemical control</td>
<td>Adrenaline and noradrenaline</td>
</tr>
<tr>
<td>ADH</td>
<td></td>
</tr>
<tr>
<td>Angiotensin II</td>
<td></td>
</tr>
<tr>
<td>Atrial natriuretic peptide (ANP)</td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
</tr>
<tr>
<td>Inflammatory mediators</td>
<td></td>
</tr>
<tr>
<td>Renal autoregulation</td>
<td>Renin</td>
</tr>
<tr>
<td>Aldosterone</td>
<td></td>
</tr>
<tr>
<td>Capillary dynamics</td>
<td>Pressures exerted within the capillaries:</td>
</tr>
<tr>
<td>Filtration</td>
<td></td>
</tr>
<tr>
<td>Absorption</td>
<td></td>
</tr>
</tbody>
</table>
Control of respiration

Neural mechanisms and generation of breathing rhythm:

- Medullary respiratory centres
  - medulla oblongata
    - dorsal respiratory group (DRG) inspiration
    - ventral respiratory group (VRG) inspiration and expiration
  - pace setting centre normal respiratory rate and rhythm of 15–20 breaths per minute – eupnoea.

- Pons respiratory centre
  - influence and modify activity of medullary neurons.

- Pneumotaxic centre
  - inhibitory impulses to inspiratory centre of medulla
  - fine tune breathing rhythm.

- Apneustic centre
  - inspiratory drive
  - prolongs inspiration
  - breath holding in inspiratory phase
  - breathing deep and slow
  - apneustic centre inhibited by pneumotaxic centre.

Factors influencing the rate and depth of breathing:

- Changes in response to body demands.

- Inspiratory depth is determined by the respiratory centre.

- The greater the frequency the greater the force of respiratory muscle contraction.

- The rate of respiration is determined by how long the inspiratory centre is active or how quickly it is switched off.

- Irritating reflexes
  - vagal nerve – respiratory centres
    - constriction of air passages
    - mucus, debris, cigarette smoke, noxious fumes
  - same irritants stimulate cough in trachea, bronchi; sneeze in nasal cavity.

- Inflation reflex
  - Hering–Breuer reflex ends inspiration so expiration can begin.

- Influence of higher brain centres
  - hypothalamic controls
    - emotions and pain can modify respiratory rate and depth
    - an ↑ in temperature ↑ respiratory rate
    - a ↓ in temperature ↓ respiratory rate
  - cortical controls
    - conscious control over rate and depth of breathing – medullary centres bypassed
    - this process limited – why?
  - chemical controls
    - central chemoreceptors in the medulla
    - peripheral chemoreceptors in vessels in the neck – mildly sensitive

- Chemoreceptors
  - carbon dioxide:
    - hypercapnia → hyper-ventilation
    - hypocapnia → hypo-ventilation → apnoea
  - oxygen:
    - found in the peripheral chemoreceptors
    - arterial oxygen must reduce substantially before oxygen levels increase ventilation
  - arterial pH
    - effect on central chemoreceptors is insignificant compared to the effect of hydrogen ions generated by elevations in carbon dioxide
    - mediated through the peripheral chemoreceptors
changes in carbon dioxide and hydrogen concentration are interrelated but distinct stimuli.

**Interactions of carbon dioxide, oxygen and arterial pH**

- Every cell in the body must have oxygen to live; the body's need to rid itself of carbon dioxide is the most important stimulus for breathing in a healthy person.
- Carbon dioxide does not act in isolation, and various chemical factors enforce or inhibit one another's effects:
  - rising carbon dioxide levels are the most powerful respiratory stimulant; low partial pressure of carbon dioxide levels depress respiration
  - low oxygen tensions augment partial pressure of carbon dioxide effects; high partial pressure of oxygen levels diminish the effectiveness of carbon dioxide stimulation
  - when arterial partial pressure of oxygen falls below 60 mmHg, it becomes the major stimulus for respiration, and ventilation is increased via reflexes initiated by the peripheral chemoreceptors. This may increase oxygen loading into the blood, but it also causes hypocapnia and an increase in blood pH, both of which inhibit respiration
  - arterial pH does not influence the central chemoreceptors directly.

**Urine Output**

The process of passing urine or emptying the bladder is called micturition also known as voiding or urination. Occurs generally when about 200 ml of urine has collected in the bladder activating stretch receptors. The average urinary output should be between 30 and 70 ml or more per hour. This should be regularly monitored by urine output, either from collecting the patient's urine in a bedpan or urinal or by a catheter inserted into the bladder and collected in a bag, and charting it on a fluid chart. The minimum urine output is \( \leq 30\) ml/hour; some practitioners prefer 50–70 ml/hour. Fluid balance charts measure a patient's:

- fluid intake (IV, oral, EF)
- fluid output (urine, wound/chest drains, vomiting, diarrhoea, insensible loss)

UO reduces during:
- stress to increase BP
- loss of circulating volume
- renal failure, hypoxic injury to the kidney (ATN), retention
- heart failure (LVEF, CHD, CHF, MI)

UO increases:
- in diabetes insipidus
- diuretic phase of renal failure
- following the administration of diuretics
- in hypothermia (massive diuresis, due to extreme cold).

If there are concerns about the patient's kidney function, overall fluid and electrolyte balance, quality of urine and circulatory status, then urinary output should be measured at regular intervals, and accurately recorded. If urine output falls below 0.5 ml/kg/hour for more than 2 hours, the medical team should be informed as fluid administration may need to be increased or diuretics prescribed. Urine output is measured at hourly intervals and accurately recorded. Interpretation of urine output is always considered as an overall fluid balance over a 24-hour period.
**Urine Testing**

The kidney has a prime role in maintaining normal healthy life and many early changes that occur in the body may be reflected in the urine well before they become clinically obvious. A critical care nurse is usually the first person to deal with a patient admitted to the unit, and has the most opportunity of contact with, and a chance to observe, the patient. Thus, nurses are well placed to aid in the detection and diagnosis of disease, as they may be the first to be aware of the patient’s clinical condition. Often there are some clues (Table 2.10), which can suggest a few simple preliminary tests that may easily show whether to pursue a particular line of investigation and these tests can be performed by the critical care nurse on a urine sample.

Urine examination can yield important information about the early signs of disease, as many life-threatening conditions of insidious onset such as diabetes, cancer of the bladder or renal disease may be revealed by the analysis of the constituents of the urine (Table 2.11). Taking notice of some of the areas measured in routine urine tests helps to provide valuable clues to the patient’s condition or the effectiveness of treatment. It is unfortunate that urine testing is described as ‘routine’ and generally undervalued. Urine testing is a simple and cost-effective procedure. It is fast, easy to interpret and non-intrusive to patients.

### Appearance of urine and cause

- **Colour**
  - yellow-orange to brownish green – bilirubin from obstructive jaundice
  - red to red-brown – haemoglobinuria
  - smoky red – unhaemolysed red blood cells (RBC)
  - dark wine colour – haemolytic jaundice

### Table 2.10 Clues suggesting preliminary urine tests are required

<table>
<thead>
<tr>
<th>Symptom or sign</th>
<th>Possible diagnosis</th>
<th>Tests to consider</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>? Malnutrition</td>
<td>Look for ketones</td>
</tr>
<tr>
<td>Weight loss, perhaps with an increase in thirst</td>
<td>? Diabetes</td>
<td>Look for glucose and ketones</td>
</tr>
<tr>
<td>Frequency of micturition</td>
<td>? Infection</td>
<td>Test for bacteria (i.e. nitrites) or protein and blood</td>
</tr>
<tr>
<td></td>
<td>? Renal disease</td>
<td>Test for specific gravity, protein, blood</td>
</tr>
<tr>
<td>Yellow tinge to skin</td>
<td>? Jaundice</td>
<td>Test for increases in urobilinogen and urine bilirubin</td>
</tr>
</tbody>
</table>
Table 2.11  Urine testing: significance of results

<table>
<thead>
<tr>
<th>Measure</th>
<th>Interpretation</th>
<th>Significance in disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific gravity</td>
<td>SG gives a good indication of the net fluid balance and is of particular value in patients where there is an unquantifiable loss, such as in burns cases, breathing difficulties, diarrhoea or fever. In healthy adults, SG varies between 1005 and 1035 (pure water is the standard, with a SG of 1000).</td>
<td>Urine with a persistently low SG is suggestive of diabetes insipidus or renal damage. An increase in specific gravity will indicate dehydration, perhaps due to bleeding, vomiting, diarrhoea, reduction in fluid intake or fever.</td>
</tr>
<tr>
<td>The pH</td>
<td>Under normal circumstances, the urine has a pH of around 6 but it can range from about 5 to 8.5.</td>
<td>Metabolic acidosis from starvation, high protein diets or diabetic ketoacidosis will lead to an acid urine but diets including a lot of vegetable, mild or even bicarbonate-based antacids can cause an alkaline urine, when the pH will rise.</td>
</tr>
<tr>
<td>Blood</td>
<td>Positive results must be followed up to determine where the blood is coming from. False positive results may occur, from containers contaminated with bleach, skin preparation with povidone iodine, or from the use of stale urine.</td>
<td>Asymptomatic haematuria is usually the earliest sign of cancer of the bladder. It can also be due to trauma, infection or stones. The blood will disappear with resolution of the infection, or stone.</td>
</tr>
</tbody>
</table>

(Continued)
### Table 2.11 (Continued)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Interpretation</th>
<th>Significance in disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Protein</strong></td>
<td>As renal disease progresses, detectable levels of protein will be found in the urine.</td>
<td>A number of diseases are associated with proteinuria including renal disease, urinary tract infection, hypertension, pre-eclampsia and congestive heart failure. When testing for urinary protein, a morning specimen of urine is recommended to ensure sufficient concentration.</td>
</tr>
</tbody>
</table>

In early renal disease, the glomerulus and tubules may leak small amounts of protein into the urine.**

| **Bilirubin and urobilinogen** | When the liver is diseased or there is obstruction to the flow of bile into the gut, bilirubin or its metabolites are likely to be found in significant quantities in the urine. | Urobilinogen is normally present in urine, but elevated levels may indicate liver abnormalities or excessive destruction of red blood cells, such as in haemolytic anaemia. |

In normal health, bilirubin is not found in the urine as it is excreted via the bile duct into the gut.**

| **Nitrites**             | Nitrites are not normally present in urine, but are produced in increasing numbers when Gram-negative bacteria such as *E. coli* convert dietary nitrates (found in the preservatives in meat products and cheese and smoked food) to nitrites. It would be appropriate to send the specimen to the laboratory for culture and sensitivity and refer the patient to the doctor for treatment. | As *E. coli* is responsible for 80% of urine infection the presence of nitrites is strongly suggestive of urinary tract infection. Visible signs may also be present – for example, is the specimen clear or cloudy? Cloudiness should be noted. If the specimen is turbid and one or more of the four tests are positive, there is a 50% chance that the urine is infected. |

Urine normally contain nitrates from dietary metabolites, and some of the common bacteria responsible for urinary infections will convert these nitrates to nitrites.
<table>
<thead>
<tr>
<th><strong>Glucose</strong></th>
<th>There are two categories of urine tests for glucose, the Clinitest and impregnated test strips.</th>
<th>The presence of glucose may be due to raised blood glucose levels (hyperglycaemia). It can be associated with many medical conditions such as diabetes mellitus, stress, Cushing’s syndrome and acute pancreatitis.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ketones</strong></td>
<td>There are two tests available for ketones: Acetest, which is a tablet test, and a strip test.</td>
<td>Usually ketones may be found in people who are fasting, but can also be present in excessive amounts in people with uncontrolled diabetes. Ketones are acidic substances and when present in excess can lead to metabolic acidosis, which, if untreated, can cause death.</td>
</tr>
<tr>
<td><strong>Odour</strong></td>
<td>Normal, freshly voided urine has very little smell, but develops an ammoniacal smell on standing. Infected urine smells foul and may have a characteristic fishy smell on voiding and the smell worsens on standing.</td>
<td>Ketoacidosis in patients who have been starving or suffering from anorexia or diabetes gives urine a characteristic smell. Eating fish, curry or other strongly flavoured foodstuffs can also make the urine smell.</td>
</tr>
</tbody>
</table>

**Glucose**
Not normally found in urine.

**Ketones**
When the body metabolizes fat waste, the breakdown products are the ketones – excreted in the urine. In good health they are not detectable in urine.

**Odour**
A urine specimen should be noted before further testing.
Patient assessment and investigations

Haemodynamic monitoring

- brown-black – melanoma
- dark-brown – liver infection
- green – bacterial infection
- Odour – infection, diabetes, anorexia

The significance of the urine test strip results can be found in the:
- specific gravity 1005–1035 (state of hydration)
- pH 4.5–8 (acid–base balance)
- blood (cancer of the bladder, stones, infection, trauma)
- protein (renal disease, UTI, hypertension, pre-eclampsia, CHF)
- bilirubin and urobilinogen (liver disease, haemolytic anaemia)
- nitrates (UTI)
- glucose (diabetes mellitus, stress, Cushing’s syndrome, acute pancreatitis)
- ketones (fasting, uncontrolled diabetes mellitus).

Taking note of some of the areas measured in a routine urine test may:
- Allow evaluation of a person’s fluid balance
- Aid diagnosis
- Assist in monitoring circulatory status
- Help to provide valuable clues to the effectiveness of treatment.

The results of a urine test should be recorded accurately in the critical care patient’s records, as soon as possible after testing. A negative test result may not only point to an alternative diagnosis, but it is also a valuable baseline indicator to be referred to later in evaluating the progress of a patient during the course of his or her illness. A negative result should always be recorded even if at the time it appears unimportant, or irrelevant.

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**Oxygen Saturation**

Adequate tissue oxygenation depends on a balance between oxygen supply and delivery, and the tissue demand for oxygen. When oxygen demands exceed oxygen supply, hypoxia occurs. Hypoxia can cause vasoconstriction of blood vessels and thus redistribute the circulating volume. Most cells require oxygen to survive, function correctly, and maintain tissues.

Hypoxia can occur from:
- a blockage whereby the tissues become hypoxic due to a reduced blood flow, as in arteriosclerosis
- the loss of red blood cells which carries oxygen to the cells, often observed in haemorrhage
- the inability to get oxygen into the circulation, seen in patients with impaired respiratory function.

---

**Always check the oxygen saturation site for tissue perfusion – especially after prolonged usage**

The critical care nurse is frequently the first to observe the presence of hypoxia and the one who can intervene to correct the problem with oxygenation. Hypoxia may be observed in a number of ways. There may be changes in behaviour and level of consciousness:
- the inability to think abstractly or perform complex mental tasks
- restlessness

---
Hypoxia can cause vasoconstriction of blood vessels and thus redistribute the circulating volume. Most cells require oxygen to survive, function correctly, and maintain tissues.

There may be changes in BP, pulse, and colour of mucous membranes. This may lead the nurse to extend the assessment for hypoxia, by obtaining an oxygen saturation measurement, or by the doctor obtaining arterial blood for blood gas analysis (see later). Very early signs of cerebral under-perfusion are the inability to think abstractly or perform complex mental tasks, restlessness, apprehension, uncooperativeness and irritability. Short-term memory may also be impaired. This is because the brain continuously needs a steady supply of oxygenated blood flow. A family member may need to be called upon for documentation of the patient’s normal personality and intellectual status.

**Oxygen saturation monitoring**

Oxygen saturation is the measure of molecules of oxygen attached to haemoglobin, and is widely used in many patient care settings. There are four molecules of oxygen attached to each haemoglobin; when this occurs blood is fully saturated at the normal percentage of 98%. O₂ monitoring uses pulse oximetry, which is a non-invasive technique to measure the saturation of blood in the arterial capillaries. It is a spectrophotometric measurement of the proportion of oxygenated haemoglobin in the arteries. The absorption of light by de-saturated and fully saturated haemoglobin is different.

This light absorption is measured by a special light detector and appears as the percentage oxygen saturation of the haemoglobin in the arteries. The light detector of the oximeter is attached to a tissue that is reasonably transparent to these wavelengths of light. This may be the:

- finger
- toe
- ear lobe.

As such, it is very useful in following changes in arterial oxygenation. There must be:

- A good flow of blood to the area (not effective if severe vasoconstriction is present)
- No mechanical movement of the probe – will cause interference
- No nail varnish – this will affect the normal haemoglobin saturation measured.

Pulse oximetry is used:

- To estimate arterial oxygen saturation (SpaO₂)
- To monitor changes in arterial oxygen saturation.

O₂ monitoring uses pulse oximetry and the nurse is frequently the first to observe the presence of hypoxia and the one who can intervene to correct the problem. Most cells require oxygen to survive, function correctly, and to maintain organ function. Hypoxia can occur from a blockage and reduced blood flow, as in arteriosclerosis; or from the loss of red blood cells which carry oxygen to the cells, often observed in haemorrhage;
or from the inability to get oxygen into the circulation, seen in patients with impaired respiratory function.

Adequate tissue oxygenation depends on a balance between oxygen supply and delivery, and the tissue demand for oxygen. When oxygen demands exceed oxygen supply, hypoxia occurs. Hypoxia may be observed in a number of ways. There may be changes in behaviour and level of consciousness. This is because the brain continuously needs a steady supply of oxygenated blood flow, and this is why the brain is a sensitive indicator of a patient's perfusion status.

**Limitations of O$_2$ monitoring**

It is important for practitioners to note that the oxygen saturation monitor can give misleading information regarding the true nature of the patient's oxygen status. This is due to how oxygen is loaded onto haemoglobin:

- Each haemoglobin molecule can combine with four molecules of O$_2$
- After the first molecule binds the haemoglobin molecule changes shape
- Haemoglobin more readily takes up two more molecules – uptake of fourth is further facilitated
- All four are bound – fully saturated
- One, two, three – partially saturated
- The unloading of one oxygen molecule enhances the unloading of the next, and so on.

Critical care nurses need to be made aware that:

- An oxygen saturation of 90% may not indicate to the nurse that there is a low oxygen supply in the blood (determined by partial pressure of oxygen).
- If the oxygen saturation falls below 85% the pulse oximeter may become progressively less accurate.
- Pulse oximetry cannot be used in any form of carbon monoxide inhalation because the carboxyhaemoglobin will result in the oximeter over-reading the saturation level.
- In anaemia there is a reduction in haemoglobin; those available will be fully saturated.

When using pulse oximetry in practice other observations should be undertaken with it:

- colour
- pulse rate
- breathing pattern and rate
- arterial blood gases (which gives partial pressure of oxygen).

An awareness of these principles will ensure that oxygen saturation monitoring is safe, and minimize the potential for unrecognised hypoxaemic episodes.

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**End-tidal carbon dioxide ($P_{ET}$CO$_2$) monitoring**

The $P_{ET}$CO$_2$ monitors exhaled carbon dioxide on both intubated and non-intubated patients. The normal range for expired $P_{ET}$CO$_2$ is generally between 4.5 and 5.7 kPa.
This method of expiratory gas analysis can be undertaken through a nasal cannula which simultaneously delivers supplemental oxygen. In lungs where ventilation is uniformly distributed and evenly matched to perfusion end-tidal CO$_2$ ($P_{ET}$CO$_2$) reasonably reflects partial pressure of arterial CO$_2$:

- Pulmonary embolism or decreased cardiac output is associated with a decrease in $P_{ET}$CO$_2$, because of decreased alveolar blood flow.

- An increase in $P_{ET}$CO$_2$ reflects the presence of airway narrowing or other lung disease associated with respiratory changes in the mechanical properties of the lungs.

This method of expiratory gas analysis is not commonly used in all clinical practice areas, but studies are showing its benefits and value in respiratory management of patients. In the future in may be as normal as attaching an oxygen saturation monitor.

**Arterial blood pressure monitoring**

This involves a needle being put into a patient artery, generally the radial or brachial. This measure of BP determines BP in a different way (BP is measured by the pressure exerted on the sides of the blood vessels) from manual BP and therefore the two measures cannot be compared. It depends on:

- compliance (distensibility) of elastic arteries
- stroke volume
- rises during ventricular systole, decreases during diastole
- systolic pressure ($P_S$) – pressure in arteries during ventricular systole (cardiac contraction)
- diastolic pressure ($P_D$) – pressure in arteries during ventricular diastole (resting period)

**Invasive Cardiovascular Haemodynamic Monitoring**

Invasive cardiac monitoring gives a much clearer picture of a patient’s haemodynamic state; however, it is invasive and therefore has numerous complications attached.

**Systolic and diastolic pressures**

- Systolic pressure ($P_S$) ~ 110–120 mmHg
  - semilunar valves open and blood is ejected
  - compliance decreases pressure needed to eject blood into arteries
  - increased stroke volume (amount ejected) → increased pressure
- Diastolic pressure ($P_D$) ~ 70–80 mmHg
  - semilunar valves closed
elastıc recoil of arteries contributes to continued pressure → movement of blood.

Mean arterial pressure (MAP)
- average pressure in main arteries
- heart spends more time in diastole
  ● therefore, MAP = diastolic pressure ($P_D$) + (pulse pressure [$P_p$] divided by 3)
  ● $MAP = P_D + (P_p/3)$

Capillary blood pressure
- Pressure in capillaries
- Pressure drops from ~35–40 mmHg (at arterial end) to ~15–20 mmHg (at venous end)
- Lower pressure helps prevent breakage of capillary walls and decreases fluid loss to tissues

Venous blood pressures
- Low, steady pressure
- Venous return supported by:
  ● valves – prevent backflow: varicose veins – failure of valves allows blood to accumulate in veins; especially common in legs
  ● respiratory pump – changes in thoracic and abdominal pressures during breathing
  ● thoracic pressure decreases and abdominal pressure increases during inspiration, decreases and abdominal pressure increases during inspiration
  ● muscular pump ‘milking’ by skeletal muscle promotes return prolonged inactivity or prolonged contraction causes blood to pool (may allow clots to form)

Maintaining blood pressure
Blood pressure (BP) varies directly with:
- Cardiac output (CO)
- controlled by cardiac centers in medulla oblongata
- sympathetic outflow
- parasympathetic outflow

Blood volume (BV)
- Peripheral resistance (PR).
Based on controlling blood vessel diameter. The mechanisms include neural and chemical controls alter distribution to meet demands of various organs/tissues to maintain overall MAP through vasomotor tone.

1. Neural control of resistance
- Vasomotor centre (VMC) controls vasomotor tone
  ● located in medulla oblongata (as part of cardiovascular centre)
  ● maintains vasomotor tone in all vessels
- Reflexes initiated by baroreceptors or chemoreceptors integrated in medulla oblongata (reticular formation)
- Vasomotor tone
  ● vasomotor fibres (sympathetic outflow)
    - most fibres use noradrenaline
    - increased sympathetic activity → vasoconstriction → increased BP
  ● fibres to vessels serving skeletal muscle use ACh
    - increased sympathetic activity → vasodilation → increased flow to skeletal muscle (generally little importance to overall BP).

2. Baroreceptors
- Baroreceptors (pressoreceptors) present in carotid sinus, aortic arch, most other elastic arteries of neck and thorax
1. Increased BP stimulates baroreceptors
   ● increases impulses to inhibit vasomotor centre
   ● decreased sympathetic outflow
   ● vasodilation
   ● decreased BP

2. Concurrent impulses from baroreceptors (stimulated by increased BP) to CIC increase parasympathetic outflow to heart also decrease sympathetic outflow

3. Prolonged hypertension causes baroreceptors to ‘reset’ to higher pressure.

3. Chemoreceptors
   ● Chemoreceptors located in aortic arch and large arteries of neck
   ● Connected to CAC and vasomotor centre by afferent fibres
   ● Respond to oxygen (O₂), pH (hydrogen ion), carbon dioxide (CO₂) levels
     ● decreased O₂ or pH, or increased CO₂ → increases impulses to CAC and vasomotor centre → increased sympathetic outflow → increased heart rate and vasoconstriction → increased BP → helps move blood through system faster → gets blood to lungs faster.

4. Chemical control of resistance
   Chemicals that act on vessels, heart or blood volume:
   ● Noradrenaline (norepinephrine) (NE; from adrenal medulla) → vasoconstriction
   ● Adrenaline (epinephrine) (epi; from adrenal medulla):
     ● vasoconstriction, except in skeletal and cardiac muscle
   ● nicotine (in tobacco) – stimulates sympathetic ganglionic neurons and adrenal medulla
   ● Antidiuretic hormone (ADH; a.k.a. vasopressin; released from neurohypophysis)
     ● stimulates water reabsorption
     ● at high levels, causes vasoconstriction
   ● Angiotensin II
     ● produced from angiotensinogen in response to renin from kidney
     ● causes intense vasoconstriction
     ● stimulates secretion of ADH and aldosterone (long-term control)
   ● Atrial natriuretic peptide (ANP; from atria of heart) – antagonizes aldosterone and causes general vasodilation
   ● Alcohol
     ● inhibits ADH secretion
     ● depresses vasomotor centre
   ● Endothelium-derived factors – affect vascular smooth muscle
     ● inflammatory chemicals – vasodilation
       – histamine, prostacyclins, kinins and others
       – released during inflammatory response
     ● endothelin – potent vasoconstrictor, released in response to low blood flow
     ● nitric oxide (NO) – vasodilator released in response to high blood flow; causes systemic and local vasodilation.

5. Regulates blood volume (BV)
   ● Blood volume important to: venous pressure, venous return, EDV, SV, CO control:
     ● direct renal control – responds to both increased and decreased blood pressure
- Increased BP → increased filtration → increased water loss → decreased BV
- Decreased BP → decreased filtration → decreased water loss → increased BV

 indirect renal control – renin-angiotensin pathway – responds to decreased blood pressure (Figure 2.1)
- decreased BP → juxtaglomerular cells of kidney tubules secrete

Fig 2.1: The renin–angiotensin–aldosterone system. ADH = anti-diuretic hormone; ACE = angiotensin-converting enzyme; BP = blood pressure.
renin → converts angiotensinogen to angiotensin I → II
– kidney also releases renin in response to sympathetic impulses

● Angiotensin II
– stimulates aldosterone secretion
– stimulates ADH secretion
– causes vasoconstriction.

6. Capillary dynamics

■ Movement across capillary is based on gradients (Figure 2.2)
  ● Solute gradient (diffusion)
  ● Water gradient (osmosis)
  ● Pressure gradient (hydrostatic pressure)

■ Forces moving fluid out of capillary – moving fluid into the interstitial space
  ● Capillary hydrostatic pressure (HP)

– also called capillary blood pressure (or blood hydrostatic pressure)
– 35 mmHg at arterial end of capillary (average)
– 17 mmHg at venous end of capillary (average)

● Interstitial fluid osmotic pressure = (OP)
– proteins in interstitial fluid exert osmotic pressure on plasma
– pulls fluid out of capillary into tissues
– but normally very little protein present in ISF
– average value is 1 mmHg

■ Forces moving fluid into capillary
  ● Interstitial fluid hydrostatic pressure (HP) – physical pressure pushing interstitial fluid into the capillary

Fig 2.2: Normal capillary dynamics and pressures which allow the normal movement of fluid across a semi-permeable membrane (filtration) to nourish the cell; most of the fluid is absorbed (absorption) back into the circulation. The remaining excess is drained by the lymphatic system. The dynamics can be changed to demonstrate the formation of oedema (see text). HP = hydrostatic pressure; COP = colloidal osmotic pressure; ECF = extracellular fluid; ISF = interstitial fluid; ICF = intracellular fluid; NFP = net filtration pressure.
Central venous pressure (CVP) monitoring

The measurement of the central venous pressure (CVP) provides important haemodynamic information to guide the therapy of patients. Central venous pressure (CVP) is indicated:

- To obtain blood for laboratory estimation
- To administer parenteral nutrition
- Administration of hypertonic or irritating solutions
- Administration of vasoactive or inotropic agents and monitor effect
- As a venous access when all other routes exhausted
- Where massive fluid replacement is required and monitor effect
- Acute circulatory failure.

The CVP normally reflects the volume of blood returning to the heart, which exerts a pressure on the walls of the right atrium and measuring it can provide information about:

- The adequacy of the body’s volume of blood in relation to circulatory capacity
- The effectiveness of the right side of the heart as a pump
- Vascular tone
- Pulmonary vascular resistance.

Measurements on critical care are usually made using a CVP placed within the subclavian vein or internal jugular vein attached to a transducer and then plugged into a monitor. However, a CVP line can also be placed in the external jugular or femoral vein. The insertion of a CVP is a strict aseptic procedure. The patient should be in the supine position. If breathlessness occurs when lying flat, the CVP readings may need to be taken with the patient lying at a greater angle no more than 30°, in which case the angle used should always be indicated alongside the recorded
CVP measurement. The monitor has to be zeroed at regular intervals. The CVP is a dynamic measure and as such differs between individuals; the average is 4–12 cm H$_2$O for water manometers and 2–8 mmHg for mercury transducers.

It is not the single CVP reading that is important but the trend demonstrated by a series of readings over time. Therefore, each time a CVP measurement is made, it is essential that it is made under identical conditions so that all possible variables (such as patient position) remain constant. Patient management should not result from the information received from CVP measurements alone. The wider clinical picture needs to be considered, e.g. blood pressure, cardiac output, heart rate, respiratory characteristics, urine output.

**Complications of a CVP**

- **Air embolism** – the lines used to measure CVP are central venous lines and thus present the inherent danger of air embolism. All intravenous administration equipment should, therefore, possess Luer lock connections to minimize accidental disconnection.
- **Pneumothorax** – damage to the apices of the lungs, leading to pneumothorax.
- **Damage to the ventricular muscles of the heart causing ventricular arrhythmias.**
- **Risk of infection and subsequent sepsis**; maintenance of asepsis is therefore essential.

**Changes in the CVP reading**

Generally, there is an overestimation of the value of the CVP reading. If a fall in CVP occurs this is proposed to indicate a moderate fall; for example, in patients who bleed following surgery, or because of extreme vasodilatation, whereby the capacity of the circulation is increased but the circulating volume remains constant, as in patients with a pyrexia or from the excessive use of vasodilator drugs (Edwards & Manley 1998).

A consequent rise in CVP is proposed to give rise to concerns about fluid overload. This can lead to circulatory collapse, whereby the left side of the heart becomes dysfunctional. The consequences being that the heart is unable to pump blood, leading to a low cardiac output and an increase in right and left ventricular filling pressures. It is presumed, therefore, that the CVP can be used as a guide both to determine severity of fluid loss and measure when too much fluid has been administered, and to ascertain cardiac instability.

This, however, is an overestimation of the value of the CVP reading, as a reduction in CVP will occur in hypovolaemia, during hypervolaemia and left-sided cardiac failure. In the instance of hypervolaemia and the CVP, it may take nearly 24 hours for events occurring in the left side of the heart to reflect through the lungs into the right ventricle, atria, and superior vena cava, and be mirrored as an increased CVP reading.

A common problem caused by hypervolaemia is cardiac failure, and in this instance the left side of the heart generally fails first. This will cause severe systolic dysfunction of the left ventricle and eventually a consequent reduction in stroke volume. As a result there is a decline in the amount of blood returning to the heart (venous return) and hence a reduction in CVP will be
recorded. This implies that CVP levels are not completely reliable in estimating circulatory function. Therefore, a more accurate measure would be that which could determine the pressure in the left side of the heart.

**Pulmonary artery pressure (PAP)**

In the late 1960s the first flow-directed catheter measuring pulmonary right, and left heart pressures was developed. The PAP catheter measures right atrial pressure, right ventricular pressure, pulmonary artery wedge pressure (PAWP), systemic vascular resistance (SVR), cardiac output (CO) and more recently mixed venous oxygen saturation (SvO₂).

A special catheter tip (Swan Ganz or thermodilution pulmonary artery catheter) that sits at the distal port of the pulmonary artery and includes a balloon obtains these measurements. When the balloon is deflated the pressure reflected is the PAP. The PAP indirectly measures the left ventricle’s end-diastolic pressure (LVEDP) and is an invaluable assessment. The normal systolic pressure is 20–30 mmHg and the diastolic is 8–15 mmHg.

When the balloon is inflated or ‘wedged’ the right pressures become blocked by the inflated balloon and the PAWP is recorded, and the tip indirectly reflects left atrial pressure, left ventricular end-diastolic pressure (LVEDP), and left ventricular preload. The PAWP is a much more reliable measurement than the CVP in determining cardiac function. The normal PAWP is 5–12 mmHg, but many patients may require a much higher pressure – 15–20 mmHg – to achieve optimal preload.

If the PAWP is high and CO low, this may indicate hypervolaemia, giving rise to left ventricular insufficiency, and cardiac dysfunction. If hypovolaemia is present both PAWP and CO would be reduced. Hypervolaemia and hypovolaemia require different therapies to maintain adequate cardiac function.

However, this method involves the threading of a catheter from a central vein through the right atrium and right ventricle and into the pulmonary artery. It is a highly invasive technique with a recognized risk of morbidity and mortality. There is an increase in the use of other techniques that adequately measure PAP, PAWP, CO, and SVR, which are becoming more frequently incorporated into critical care haemodynamic monitoring practice.

**Indications for use**

- acute cardiac failure
- shock
- diagnosis of tamponade
- mitral regurgitation
- ruptured ventricular septum
- management of high-risk obstetrical patients
- intraoperative and postoperative management of high-risk patient.
- the CVP fails to give accurate or sufficient detail regarding cardiac function.
The PAP measure

- When the balloon is deflated the pressure reflected is the pulmonary artery pressure (PAP). The normal systolic pressure is 17–32, the diastolic is 7–13, and the mean is 9–19 mmHg (these measures may vary in the literature).

- The pulmonary artery pressure (PAP) catheter measures right atrial pressure or CVP, right ventricular pressure (PAP), PAWP, systemic vascular resistance (SVR), and cardiac output (CO).

The PAP is elevated in:

- pulmonary hypertension caused by tension pneumothorax, haemothorax, COPD
- left-sided heart failure
- mitral stenosis/insufficiency
- fluid overload
- tamponade.

The PAP will be decreased in:

- beta-adrenergic stimulation
- hypovolaemic shock.

Pulmonary artery wedge pressure (PAWP)

When the balloon is inflated the right pressures become blocked by the inflated balloon and the PAWP is recorded:

- indirectly reflecting left atrial pressure
- left ventricular end-diastolic pressure (LVEDP)
- left ventricular preload.

The PAWP is a much more reliable measure, in determining the circulating volume. The normal PAWP is 5–12 mmHg, but many patients may require a much higher pressure, 15–25 mmHg.

Changes in PAWP

The PAWP will be elevated in:

- valvular-dependent cardiac dysfunction in mitral stenosis/insufficiency or severe aortic stenosis
- left-sided heart failure
- decreased left ventricular compliance by cardiac tamponade
- PEEP
- myocardial infarction
- vasopressors
- PE, hypoxia, ARDS.

The PAP will be decreased in:

- hypovolaemia
- after load reduction caused by vasodilating agents.

Cardiac output (CO)

This is the amount of blood pumped by the heart per minute (l/min) and determines the function of the heart and cardiovascular system.

The CO changes according to different needs of the body; it is generally about 4–8 l/min. Two variables contribute to CO – stroke volume (SV) and heart rate (HR) – ml/beat. The SV is the volume of blood pumped by the heart in a single systolic contraction of the LV and remains stable at 75 ml/beat. The product of the SV and the HR is equal to the CO, or:

\[
CO \text{ (ml/min)} = SV \text{ (ml/beat)} \times HR \text{ (bpm)}.
\]
Cardiac index

- A CO value is dependent on body size
- To account for changes in CO that occur in patients with different body size, CO is divided by body surface area (units are square metres)
- Body surface area is automatically calculated by the software from the values for height and weight of the patient or nomograms can be used
- Normal range 2.5–4.2 l/min/m²

CO and PAWP

- If the PAWP is high and CO low, this may indicate hypervolaemia giving rise to left ventricular insufficiency, and cardiac dysfunction.
- If hypovolaemia is present both PAWP and CO would be reduced.
- Hypervolaemia and hypovolaemia require different therapies to maintain adequate cardiac functioning.

Systemic vascular resistance (SVR)

The average or total resistance to blood flow in the entire systemic circulation. Lower values indicate vasodilatation (sepsis) while higher values indicate vasoconstriction (stress, hypothermia). Most patients have a range of 9.6–18.8 min/l (770–1500 dyn s/cm²)

\[ \text{SRV} = (\text{Mean arterial pressure} - \text{central venous pressure}) \times 80/\text{cardiac output} \]

Factors that decrease SVR:
- vasodilator therapy
- hyperdynamic septic shock

Factors that increase SVR:
- hypovolaemia
- hypothermia.

CO and SVR

In sepsis the CO may be normal or high due to changes in HR, despite a reduced SV due to the severe vasodilatation from the increase in body temperature. In this instance CO is not a good indicator of circulatory function. The SVR will be reduced but the CO may be normal or high, indications of sepsis and determines treatment, e.g. noradrenaline.

Mixed venous oxygen monitoring (SvO₂)

The SvO₂ or the percentage of saturation of venous haemoglobin, reflects the overall balance between oxygen delivery and oxygen consumption of perfused tissues (Shailer et al 1992). Measurement of SvO₂ is determined by the saturation of haemoglobin in the pulmonary artery. It reflects a mixture of venous saturation from various organ systems and represents oxygen saturation of the body, rather than one organ or area. The normal value for SvO₂ is 75%, with a range of 60–80%.

A decrease in SvO₂ is an early indication that oxygen transport and uptake may be inadequate and
interventions may be necessary. Low values may result from increased oxygen demand or decreased oxygen delivery. Abnormally high $\text{SvO}_2$ (>80%) levels can be caused by high FiO$_2$ rates or decreases in oxygen demand, such as with hypothermic patients or those who are anaesthetized.

Any changes in $\text{SvO}_2$ outside the normal range of 60–80% or a trend of deviation from the baseline that is greater than 10% should be considered for further assessment or intervention. Nevertheless, continuous $\text{SvO}_2$ monitoring can be used as an indicator of the adequacy of oxygen supply to the tissues and as an early warning sign of cardiopulmonary changes and pathophysiological events.

Mixed venous oxygen saturation is used as a measure of adequacy of tissue perfusion. It varies directly with CO, haemoglobin and saturation of arterial blood, and inversely with metabolic rate. Normal is 75%. Decreases occur when oxygen delivery falls or tissue oxygen demand increases:

- all types of shock
- pyrexia, e.g. hyperpyrexia/ hyperthermia
- hypoxia
- when it drops as low as 30%, oxygen delivery is insufficient to meet tissue oxygen demand, with anaerobic metabolism and lactic acidosis.

Increases are more difficult to interpret but are observed in conditions such as:

- arteriovenous fistulae
- cirrhosis
- left to right cardiac shunts
- cyanide poisoning

- hypothermia
- unintentional PA catheter wedging.

Increased readings reflect a failure of cells to take up and utilize oxygen. Can be measured continuously using a fibreoptic PA catheter or from intermittent blood sampling.

**Transoesophageal echocardiography (TOE)**

This uses an ultrasound probe attached to the end of a flexible endoscope, which is introduced into the oesophagus. It is possible to measure cardiac output via an ultrasound probe in this way, as the oesophagus runs parallel with the descending aorta at the level of the fifth and sixth thoracic vertebra. The aorta crosses the oesophagus anteriorly at the arch and it is because of this that the probe placed into the oesophagus requires an angled tip. The probe emits a beam of ultrasound waves of known frequency from the oesophagus across the descending aorta.

The ultrasound waves are reflected off moving objects, e.g. red blood cells, and back to the probe. During diastole when the blood is not moving, the reflected wave is at the same frequency as the emitted wave. During systole, the blood is moving away from the probe and the frequency of the reflected wave is lower. The faster the blood is moving away the greater the drop in frequency. Thus, the Doppler measures the velocity...
Patient assessment and investigations

Haemodynamic monitoring

of the blood flow in the descending aorta.

The oesophageal Doppler technique has the advantage of being continuous and relatively non-invasive – it involves no skin punctures:

- The probes are simple to insert and information on cardiac function is instantly available.
- The ODM has the ability to evaluate cardiac anatomy and performance, together with the facility to accurately estimate volume status.
- The estimation of pulmonary pressures has been shown to have a high degree of correlation with simultaneous invasive measurements.
- The shape of the waveform provides valuable information on circulating blood volume and heart contractility. The probe determines CO and other haemodynamic changes by assessing size, shape, and changes in shape of the velocity waveforms of the descending aortic blood flow, which demonstrates increases in resistance.
- Patients with TOE often show haemodynamic improvement after fluid loading.
- TOE gives a visual display of the heart and blood flow in the descending aorta allowing non-invasive assessment of cardiac output and recognition of overfilling of the left ventricle. Changes in cardiac output can be more accurately followed.

TOE is used to determine:

- Cardiogenic shock – a high diastolic pulmonary venous flow pattern into the left atrium is observed, which is reflected by elevated left atrial pressures. Cardiogenic shock is therefore recognized as a reduction in ejection fraction and the pulmonary venous flow pattern will show predominantly low flow state as evidence of elevated LA pressure.
- Severe fluid overload – there may be evidence of right atrial and ventricular distension with a poorly functioning right ventricle.
- Cardiac tamponade – which is specific for the diagnosis of tamponade.
- Assessment of left ventricular function
- Myocardial ischaemia and infarction
- Complications of cardiac surgery
- Valvular heart disease
- Aortic dissection
- Suspected endocarditis
- Pulmonary embolism
- Thoracic trauma

TOE can also determine:

- Right ventricular (RV) infarction
- RV dilatation
- RV anterior wall hypokinesis
- Paradoxical septal motion and small LV dimensions
- Differential diagnosis as well as diagnosis, where the origin of cardiac dysfunction is uncertain.

This technique holds some obvious advantages over pulmonary artery catheter insertion, namely:

- a reduction in the risk of complications which have been quoted up to 7.2% for the more invasive procedure
ease and minimal expertise required for insertion of the probe and acquisition of signals

- negligible running costs after the initial capital expenditure with the transducer reusable after sterilization in a suitable detergent
- prolonged usage in the same patient
- continuous appreciation of circulatory changes, ventricular function, and the effects of therapies by both medical and nursing staff.

However, a pulmonary artery catheter may still be necessary for absolute measurements of CO, PAWP, SVO$_2$, and SVR, especially in situations such as severe septicaemia. During septicaemia the CO may be normal, despite the reduction in stroke volume, but CO is maintained by the consequent increase in heart rate. Therefore, to obtain an effective diagnosis of septicaemia and to assist in the determination of support therapies, a measure of SVR is necessary.

To determine hypervolaemia does not necessarily require a definite measure of SVR. It could prove useful in patients who it is felt did not warrant the invasive PA technique, and is an attractive proposition for critical care areas possessing neither equipment nor experience in invasive monitoring or concerned about the potential hidden risks.

Haemodynamic information only previously obtainable by pulmonary artery catheterization is now available from this non-invasive technique. Even though, familiarization is needed with this method and a reasonable evaluation period is necessary.

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**Gastric Intramucosal pH (pHi)**

The gut is sensitive to decreases in oxygen delivery. Vasoconstrictors are secreted (angiotensin II and vasopressin) causing redistribution of blood flow to the heart and brain; the gut mucosa is in danger from hypoxia. If oxygen is reduced gut cell respiration switches to anaerobic metabolism, and lactic acid is formed – which is buffered by bicarbonate causing carbon dioxide and water to be released. Therefore, the carbon dioxide levels in the gastric mucosa determine the degree of perfusion.

Gastric tonometry determines gastric intramucosal carbon dioxide and is compared to arterial bicarbonate (assumed to be the same) into a equation to determine pHi. Low intramucosal pHi values are associated with worse prognosis.

**Intracranial pressure monitoring (ICP)**

The skull and meninges contain three major components:

- Brain tissue (80%)
- Cerebral spinal fluid (CSF) (10%)
- Cerebral blood flow (CBF) (10%).

The pressure these three components exert in the rigid skull is termed the intracranial pressure (ICP). The normal range of ICP is between 0 and 15 mmHg; above 15 mmHg is determined a raised ICP. The brain maintains this normal pressure by compensation mechanisms known as autoregulation, which occurs following an insult or injury leading to increased
brain, blood or CSF volume. To compensate the following occurs:

- Displacement of CSF from the cranial subarachnoid space, spinal and lumbar space; CSF production decreases and CSF absorption increases.

- Reduction in CBF, venous blood is shunted away from the affected areas. A widespread reduction in CBF to compensate can lead to further brain insult or ischaemia due to the reduced cerebral perfusion.

These compensatory mechanisms may become exhausted and an increase in ICP above 15 mmHg may occur. This may occur due to:
- Trauma
- Hydrocephalus
- Infection
- Tumours
- Metabolic disorders
- Cerebrovascular accident
- Encephalopathies.

In certain injuries monitoring techniques can be employed to measure the ICP. There are three types of ICP monitoring device – all monitors but only one can drain; these include:

1. Fluid-coupled systems with external transducers:
   - Ventriculostomy (intraventricular catheter, IVC), able to drain excess CSF

2. Fluid-coupled surface devices:
   - Subarachnoid (SA) bolts devised because of the concern for infection with IVCs – unable to drain CSF

- Is only a monitoring instrument
- Can be inaccurate and tend to underestimate the ICP

3. Solid state systems include the fibreoptic system and cable:
   - Can be combined with IVC for simultaneous ICP monitoring and CSF drainage
   - Solid-state systems are not always directly compatible with common critical care bedside monitoring – a separate system for recording and trending is necessary. Can be placed in the:
     - Lateral ventricle
     - Brain parenchyma
     - Epidural space.

Complications of IVC ICP monitors are the risk of infection such as meningitis or ventriculitis, which is related to the duration of catheter insertion. SA bolt infection is rare and generally infections are superficial and rarely involve the brain or the meninges. Injury to the brain due to ICP monitoring includes direct brain puncture, parenchymal or subdural haemorrhage but is uncommon.

Close recording of observations is necessary, especially of mean arterial pressure (MAP). This is necessary to determine adequate CBF. Calculate and record ICP and cerebral perfusion pressure (CPP). CPP is the pressure needed to perfuse the brain, and the normal range is 80–90 mmHg. It is calculated by subtracting the ICP from the MAP:

\[
CPP = MAP - ICP.
\]

CBF is compromised if the CPP is below 60 mmHg. Reduced CPP may result in irreversible brain damage or death. It is thought that the threshold
for mechanical brain injury is an ICP is between 20 and 30 mmHg.

Factors that reduce the ICP:
- Position – head elevated by 30°
- Fluid restriction – slightly dehydrated state
- Temperature control – hypothermia
- Drugs
  - Diuretics
  - Corticosteroids
  - Anticonvulsants
- Hyperventilation – reduced partial pressure of carbon dioxide will cause cerebral vasoconstriction and a reduction in ICP – hypercarbia is important to avoid in patients with increased ICP
- A reduced level of oxygen will increase ICP and hypoxia or cerebral ischaemia should be avoided
- Removal of the cause, e.g. surgery.

2.4 Diagnostic procedures

The Electrocardiogram (ECG) Rhythm Strip

The ECG is a record of the changes in electrical activity occurring within cardiac muscle. The cardiac cells involved in the contraction are specialized and are unlike any other cells in the body, as each individual cell can initiate its own electrical impulse (Edwards 2000b). Although cardiac muscle has this special property, hormones and chemical transmitters are important in producing the finer control of the heart and maintenance of homeostasis.

Bipolar and unipolar electrodes provide an ECG rhythm known as the PQRST waves, which detect the electrical charges within the cardiac cell. The ECG can provide information about the heart rate and rhythm, the effects of electrolytes or drugs on the heart and the electrical orientation of the cardiac muscle. The normal ECG trace should record between 60 and 100 complexes (PQRST) per minute.

What is an ECG?
The action potentials transmitted through the heart during the cardiac cycle can be recorded on the surface of the body. The recording can be obtained by electrodes on the body, connected to an ECG machine. The voltage changes are fed to the machine, amplified and displayed visually on a screen, graphically on ECG paper, or both.

Terminology
- iso-electric line – baseline
- positive – upward deflection
- negative – downward deflection
- voltage – height and depth of a wave
- time – measured along horizontal axis; one second = 5 large squares
- cardiac cycle – represented on ECG by P wave, QRS complex, T wave
- bi-phasic – deflection which is both positive and negative

ECG leads

The ECG leads provide a variety of views of the heart’s electrical activity