



Oxygen transport and the use of pulse oximetry 46-53

Multiple-choice questions and submission instructions 54

Practice profile assessment guide 55

Practice profile 26

By reading this article and writing a practice profile, you can gain ten continuing education points (CEPs). You have up to a year to send in your practice profile and guidelines on how to write and submit a profile are featured immediately after the continuing professional development article every week.

## Oxygen transport and the use of pulse oximetry

### In brief

#### Author

Georgina Casey RGN, BSc, PGDipSci, MPhil is a freelance author.  
Email: gmlcasey@hotmail.com

#### Summary

Pulse oximetry is a useful way to monitor oxygen saturation in patients in a variety of clinical settings. Nurses need to understand the principles of oxygen transport and delivery to use pulse oximetry effectively in clinical practice.

#### Keywords

- Nursing care
- Oxygen therapy
- Respiratory system and disorders

These key words are based on subject headings from the British Nursing Index. This article has been subject to double-blind review.

NS102 Casey G (2001) Oxygen transport and the use of pulse oximetry. *Nursing Standard*. 15, 47, 46-53. Date of acceptance: June 6 2001.

### Aims and intended learning outcomes

The aim of this article is to examine the oxygen transport system and the use of pulse oximetry in clinical practice. This article explains the physiological processes in oxygen transport and delivery to body tissues. The need for in-depth knowledge about the factors associated with oxygenation, and how these relate to pulse oximetry, is emphasised. After reading this article you should be able to:

- Outline the normal requirements for oxygen and the cellular and systemic effects of hypoxia.
- State the three requirements for effective delivery of oxygen to body tissues.
- Explain the processes by which saturation of haemoglobin occurs.
- Discuss the effect of haemoglobin levels on the transport of oxygen.
- Describe the effect of variations in cardiac output on tissue oxygenation.
- Relate these factors to the evaluation of tissue oxygenation in the clinical setting.

### Introduction

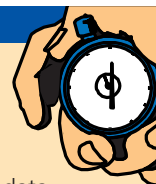
The pulse oximeter is a useful monitoring device for evaluating the oxygen status of patients in a variety of clinical settings. While initially used in theatres and critical care units, pulse oximeters are currently found in many areas of practice, including midwifery, general medical and surgical units and the community (Carroll 1997, Simpson 1998). In future, the pulse oximeter could become as commonly used as the thermometer. However, it can be difficult for nurses to critically evaluate information given by the pulse oximeter if they do not have sufficient

knowledge of the physiology associated with oxygen transport and delivery in the body. The need for nurses to understand the various processes associated with oxygenation and how to relate this information to pulse oximetry is discussed.

When taking a person's temperature, nurses also evaluate related clinical signs that indicate whether or not the thermometer is giving an accurate reading. Signs such as shivering and skin that is hot to the touch would indicate a fever, despite a normal temperature reading on the thermometer. In this case, we would question the accuracy of the thermometer and retake the patient's temperature. On using the pulse oximeter, however, there is a tendency to believe what the equipment is telling us. This is partly due to the complexity of the machine, but it is also because of a lack of understanding of the physiology underlying the processes of oxygenation that determine the pulse oximeter's readings.

### TIME OUT 1

Table 1 introduces three fictional patients who will be referred to throughout the article. As the physiology of oxygenation is explained, more data will be given about these patients. This will help you to predict their oxygen status with increasing accuracy and confidence. The initial information given in Table 1 is incomplete and a nurse would not be able to base his or her assessment solely on this information. However, for this exercise you are asked to look at the data and decide which of these three patients has the most oxygen being delivered to the tissues.



### Online archive

For related articles visit our online archive at:  
[www.nursing-standard.co.uk](http://www.nursing-standard.co.uk)  
and search using the key words above.



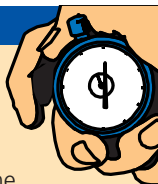
The Time Out exercise specifically asked about oxygen delivery to the tissues. When using pulse oximetry it is easy to forget that, while these machines measure the amount of oxygen being carried by haemoglobin in the arterial blood, the ultimate purpose of evaluating a patient's oxygen status is to determine how much oxygen is reaching the cells of the body. It is also important to note that the amount of oxygen delivered to the tissues cannot be determined using pulse oximetry.

Oxygen is necessary for the function of all body cells. Glucose and oxygen are used by the cells to create adenosine triphosphate (ATP), the molecule that provides energy for most cell activities. In the absence of oxygen, some body tissues, for example skeletal muscle, are able to continue making ATP, but only for a limited time. This process is referred to as anaerobic metabolism and leads to a build up of lactic acid that, if prolonged, will result in cell death (ischaemia and necrosis). The brain and heart muscle are more vulnerable to hypoxia because the cells in these tissues cannot rely on anaerobic pathways and, therefore, require a constant supply of oxygen.

When we assess a person's oxygenation status, using a pulse oximeter, we are trying to determine how much, and how well, oxygen is being transported to the tissues and cells of the body. However, the pulse oximeter only identifies how much oxygen is being carried by individual haemoglobin (Hb) molecules; it cannot give information about how much Hb is present in the blood and how well that Hb is being transported around the body. To acquire an accurate reflection of a patient's oxygenation status, it is necessary to evaluate a number of other factors.

#### TIME OUT 2

Using this knowledge and your clinical experience, list four signs or symptoms that might be evident in a person experiencing hypoxia. Discuss the reasons why these signs or symptoms have occurred.



Signs that indicate hypoxia include fatigue and central nervous system disturbances, for example, lethargy, restlessness and confusion. These arise because reduced oxygen delivery or uptake means less ATP production, which results in impaired cell function (Porth 1994). Other signs, such as increased respiratory rate and depth, and increased heart rate, indicate the body's effort to compensate for hypoxia. One of the more commonly described signs of hypoxia is

Table 1. Pulse oximetry readings for three patients

| Name   | Mr Adams | Mrs Bryant | Mr Charles |
|--|----------|------------|------------|
| Age  | 56       | 42         | 78         |
| Pulse oximeter reading<br>(percentage saturation of haemoglobin) | 84       | 94         | 99         |

Fig. 1 Signs of cyanosis



cyanosis (Fig. 1). This occurs when saturation of haemoglobin in the blood falls below 70 per cent (Bray *et al* 1999, Woodrow 1999). However, this is subject to a number of variables and is an indication of severe hypoxia. Pulse oximetry provides a much earlier indicator of falling oxygen saturation levels.

Cyanosis is a blue-purple colour visible in the nail beds, skin and mucosal membranes (gums, tongue, under the eyelids). Cyanosis occurs when a large amount of haemoglobin in the blood is poorly saturated. Saturated haemoglobin is a bright red colour, while desaturated molecules are darker. Central cyanosis is a more reliable indicator of poor respiratory or cardiac function than peripheral cyanosis. Peripheral cyanosis (fingers and toes) is more likely to result from poor circulation to these regions. Poor blood flow through a region means the cells extract more oxygen from the blood, so haemoglobin becomes more desaturated. When checking for signs of cyanosis, always check a region of the body that remains warm (mouth, eyes), or warm an extremity to increase the circulation. Detecting cyanosis in people with dark skins, jaundice, or in poor or fluorescent light, can be problematic. A person with severe anaemia might not have enough desaturated haemoglobin to appear cyanotic, even with low blood oxygen levels. Thus the pulse oximeter has



Fig. 2a. Normal gradient for diffusion of oxygen

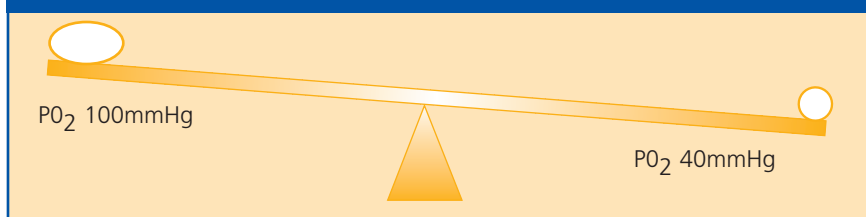


Fig. 2b. Gradient for diffusion of oxygen at 5,000 feet

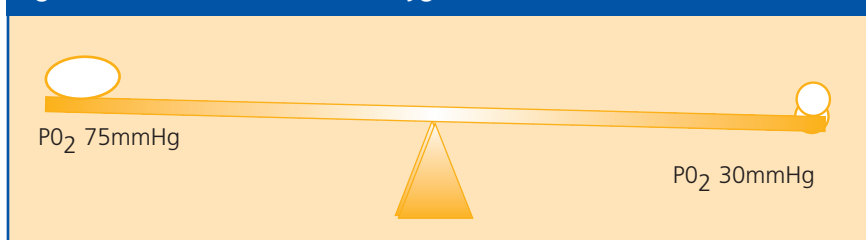
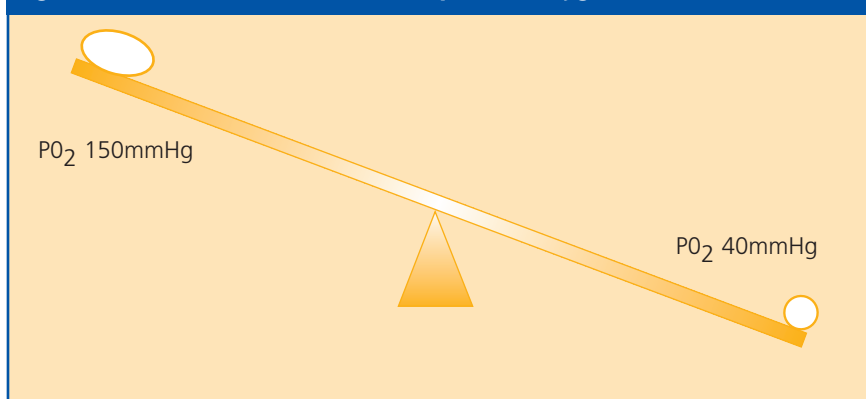


Fig. 2c. Gradient for diffusion with 28 per cent oxygen



an important role in the assessment of adequate oxygenation of the tissues.

#### Oxygen delivery

Delivery of oxygen to the tissues is determined by three factors:

- The oxygen saturation, or amount of oxygen being carried by the haemoglobin.
- The amount of haemoglobin present in the blood.
- How well the haemoglobin is being transported around the body.

Of these factors, only oxygen saturation is demonstrated by the pulse oximeter. Therefore, on examining the pulse oximetry readings given for the patients in Table 1, while Mr Charles has the best reading (99 per cent), it is not possible to determine which of these patients has the best tissue oxygenation. This article explains the processes by which oxygen saturation of haemoglobin occurs. The effect of haemoglobin content and transportation on oxygen delivery, and their

effect on the ability of the pulse oximeter to provide an accurate reflection of the patient's oxygenation status, are also discussed.

#### Haemoglobin

Haemoglobin is contained in red blood cells. A haemoglobin (Hb) molecule is composed of four protein chains (globin), each of which contains a molecule of haem. Haem is the iron-containing portion of Hb. Hb molecules contain four iron atoms, each of which is capable of combining with an oxygen molecule. A single red blood cell contains over 600 million Hb molecules (Higgins 1995).

The binding of oxygen to Hb molecules is determined by the concentration of oxygen in the plasma ( $PO_2$ ), and it is this figure that is measured when taking arterial blood gases. The concentration of a gas such as oxygen is measured as a pressure in millimetres of mercury (mmHg). The concentration of oxygen in the arterial blood is mainly determined by the amount of oxygen that enters the plasma as the blood passes through the lungs.

When we inhale we draw air into the lungs. Oxygen enters the alveoli – the gas exchanging portions of the lungs – by the process of external respiration. At sea level the pressure of the air in the atmosphere is about 760mmHg. This air contains 21 per cent oxygen. As we inhale, the air becomes humidified and warmed. Water vapour contributes about 47mmHg of the barometric pressure to inhaled air as it enters the lungs (Bray *et al* 1999). This alters the pressure of available oxygen so that the concentration of oxygen inhaled is actually 21 per cent of 713mmHg (that is 760mmHg minus 47mmHg) or 150mmHg. By the time the air enters the alveoli, this figure has fallen to 100mmHg of oxygen because the inhaled air has mixed with 'dead space' (air in the trachea and bronchi where gas exchange cannot occur), and residual air as it travels to the lungs.

Blood coming into the lungs via the pulmonary arteries has an oxygen concentration or  $PO_2$  of about 40mmHg; the same as venous blood (Bray *et al* 1999). The steep concentration gradient from the alveoli (100mmHg) to the blood (40mmHg) allows diffusion of oxygen to occur. In the healthy, resting, adult lung, blood passes through the pulmonary capillaries in about three-quarters of a second (Bray *et al* 1999). During this short time, an equilibrium is reached by diffusion and the concentration of oxygen in the blood and alveoli are equalised.

The rate of diffusion of oxygen from the alveoli





into the blood in the pulmonary arteries is determined by three main factors:

- Concentration gradient.
- Surface area available for diffusion.
- Thickness of the alveolar-capillary membrane.

These factors are subject to alteration through disease processes and can also be changed by clinical intervention.

**Concentration gradient** A concentration gradient is said to exist when diffusing substances (gas or liquid) move from an area where they are in high concentration to a region of low concentration. It sometimes helps to think of these substances as rolling down a slope. The bigger the difference in concentration between one area and another, the steeper the slope and the faster the substance rolls down it (Fig. 2a).

There is a steep concentration gradient for oxygen between the alveoli ( $PO_2=100\text{mmHg}$ ) and the blood in the pulmonary arteries and capillaries ( $PO_2=40\text{mmHg}$ ). This gradient remains the same for any person at sea level, breathing normal air. However, the concentration gradient changes with a change in altitude.

#### TIME OUT 3

To calculate the concentration ( $PO_2$ ) of inspired oxygen at sea level, we take the barometric pressure (760mmHg) and subtract 47mmHg (pressure of water vapour added as the air travels into the lungs). We then multiply the result by 21 per cent. Imagine that you are working in Denver, Colorado. This city is at an altitude of over five thousand feet and here the atmospheric pressure is only 576mmHg. Can you calculate the pressure of inspired oxygen for people living in this city?

Now imagine yourself climbing Mount Everest – a peak of over eight thousand feet. Here the barometric pressure is 250mmHg. Calculate the pressure of inspired oxygen at this altitude.

For each of these exercises the calculation is the same: barometric pressure, less 47mmHg, and then multiply by 21 per cent. The amount of oxygen in the air remains the same, but the pressure of oxygen changes with changes in barometric pressure. In Denver the pressure of inspired oxygen is about 111mmHg and this would fall to about 75mmHg in the alveoli (Fig. 2b). At the top of Mount Everest the pressure of inspired oxygen is 42mmHg, falling to 30mmHg in the alveoli (Bray *et al* 1999). At this altitude

there is no concentration gradient for diffusion. Most climbers use oxygen at this altitude, and those who do not spend considerable time acclimatising at slightly lower altitudes.

Another way to alter the pressure or concentration gradient for the diffusion of oxygen is to increase the percentage of inspired oxygen, and we do this when we supply oxygen therapy. For example, if a patient was prescribed oxygen at 28 per cent, the pressure of inspired oxygen becomes (760mmHg-47mmHg) multiplied by 28 per cent or 199mmHg. This would fall to about 150mmHg in the alveoli, but it still increases the concentration gradient for the diffusion of oxygen significantly (Fig. 2c).

#### TIME OUT 4

Calculate the pressure of inspired oxygen for a person receiving:

- 40 per cent oxygen.
- 24 per cent oxygen.

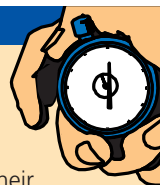


The answers to Time Out 4 are (a) 285mmHg and (b) 171mmHg. By increasing the pressure or concentration of inspired oxygen, we can significantly increase the gradient for diffusion of the gas across the alveolar-capillary membrane. This can compensate for changes in other factors that affect diffusion rates, and thus provides the most basic treatment for hypoxia.

**Surface area** The surface area available for diffusion of oxygen is determined by the number of alveoli that are receiving both air and blood flow. In the adult male this area is between 50 and 90 square metres (Bray *et al* 1999). Surface area is affected by normal physiological factors, such as posture, and pathological conditions. When the lungs are in an upright position, the upper alveoli receive good oxygenation, but the blood flow might not be great, whereas the alveoli at the base of the lungs receive less oxygen, but better blood flow. Blood flow and oxygen delivery to the alveoli alter with changes in body position. Lying down, or increasing the pulmonary arterial blood pressure (as, for example, during exercise) will increase blood flow to the upper pulmonary capillaries and, therefore, increase the surface area available for diffusion. Any disease or condition that decreases air or blood flow in the lungs will reduce the surface area for diffusion and thereby reduce the amount of oxygen crossing into the blood and binding to Hb.

**TIME OUT 5**

Think of three patients you have cared for recently who were monitored using pulse oximetry and had reduced Hb saturations. Can you describe their medical conditions in terms of reduced air or blood flow to the lungs? If you have not nursed anyone recently with such a condition, list five health problems that would result in reduced air or blood flow to the lungs.



Examples of conditions that cause reduced blood flow to the alveoli are pulmonary embolism, pulmonary hypertension tumours compressing blood vessels in a specific region and, in neonates, patent ductus arteriosus. Reduced air flow to the lungs can occur with any obstructive airways disease, for example, asthma, chronic bronchitis, emphysema or bronchiectasis. It also occurs with pneumothorax, atelectasis or surgical partial- or total-lung removal. Cystic fibrosis causes poor alveolar ventilation because of the presence of thick mucus which obstructs the airways. For premature neonates, lack of surfactant means that with every exhalation the alveoli collapse, making subsequent ventilation extremely difficult.

**Thickness of the alveolar-capillary membrane**

The alveolar-capillary membrane, which lies between the alveoli and the pulmonary capillaries, is normally extremely thin (1µm) and allows rapid diffusion of oxygen into the blood. Any condition that causes an increase in the thickness of this barrier, or increases the distance between the air and blood, will reduce the rate of diffusion. One example is pulmonary oedema. In this condition, excess fluid pools in the alveoli and this increases the distance oxygen has to travel across the alveolar-capillary membrane. Thickening of the pulmonary membrane occurs with diseases, such as sarcoidosis and asbestosis that cause pulmonary fibrosis or scarring.

In the healthy, resting lung, by the time blood has flowed through the pulmonary capillaries that surround the alveoli, the pressure of oxygen in the blood will be the same as that in the alveoli – 100mmHg (Bray *et al* 1999). This falls slightly to about 95mmHg as the blood returns to the left side of the heart and mixes with blood from unventilated portions of the lungs (Bray *et al* 1999). At this pressure oxygen will bind to about 97 per cent of the Hb in the red blood cells (RBCs) (Bray *et al* 1999).

The pressure of oxygen in venous blood returning to the heart (and in the pulmonary arteries) is 40mmHg, which gives a Hb saturation of about

70 per cent. There is considerable reserve of oxygen available, so that Hb saturation in the venous blood can fall much lower in exercise or disease. A falling venous Hb saturation would indicate that the supply of oxygen is not meeting the requirements of the tissues (Ahrens 1987). However, because of the difficulty in obtaining venous Hb saturation, it is not readily measured.

The data the pulse oximeter provide relate only to the arterial Hb saturation. It does this using red and infrared light to determine the colour of the Hb passing through arterioles in the area beneath the probe. Fully saturated Hb is a brighter colour than partly or completely desaturated molecules. Because the pulse oximeter uses colour to determine saturation, measurements are subject to error in certain circumstances. The presence of nail polish, tattoos or jaundice can all affect the oximeter reading (Woodrow 1999). Poor blood flow to the area under the probe, incorrect attachment, or a dirty probe, can also cause false readings. More importantly, pulse oximeters can give a falsely high reading in the presence of carbon monoxide.

Carbon monoxide will bind to Hb about 250 times more strongly than oxygen (Bray *et al* 1999), and, once in place, prevents the binding of oxygen. It also turns Hb bright red. The pulse oximeter is unable to distinguish between Hb molecules saturated with oxygen and those carrying carbon monoxide. Because it binds so strongly to Hb, carbon monoxide is slow to be removed from the blood. Falsely high oximeter readings will be obtained from smokers up to four hours after smoking a cigarette (Woodrow 1999). Other sources of carbon monoxide are fires, car exhaust inhalation and prolonged exposure to heavy traffic environments.

**Concentration of haemoglobin**

The concentration of Hb in the blood varies with age and gender (Table 2). The volume of plasma in the blood affects Hb concentration because this is measured relative to plasma. For example, in pregnancy there is a drop in Hb levels because of a normally occurring rise in plasma volume. If a patient is dehydrated, the Hb concentration is increased, not because the patient produces more red blood cells, but because the plasma volume is decreased.

Erythropoiesis, the production of RBCs, occurs at a rate similar to their destruction in the normal healthy adult. Because there are so many of these cells – 4 to 5 million per millilitre of blood (Bray *et al* 1999) – and their life span is approximately four months, large numbers of



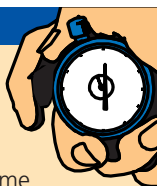
RBCs are synthesised continuously in the bone marrow. Erythropoiesis requires a number of nutritional factors: iron for the production of haem, protein for globin, and folic acid and vitamin B<sub>12</sub> for the manufacture of cell nuclei. Deficiencies in any of these will affect RBC production and might cause anaemia.

Anaemia can also occur as a result of increased destruction of RBCs (for example with sickle cell disease or thalassaemia); increased loss (arising from chronic or acute blood loss); or decreased production as a result of pathophysiological conditions, such as bone marrow pathologies, and decreased production of erythropoietin (in chronic renal failure) (Porth 1994). The manifestations of anaemia relate largely to impaired oxygen delivery to the tissues. Fatigue, pallor, dyspnoea and tachycardia occur because of poor oxygenation and the body's attempts to compensate for this (Porth 1994). It should be remembered that anaemia is a symptom of another condition and, therefore, the underlying causative process should be investigated.

The total amount of oxygen carried by Hb in the blood can be calculated. It is, therefore, possible to determine what effect a reduced Hb count or anaemia has on oxygen transport. Each gram of Hb in the blood is capable of carrying 1.34ml of oxygen (Bray *et al* 1999). This information combined with data on Hb concentration can be used to calculate the total oxygen-carrying capacity of a person's blood.

#### TIME OUT 6

Given a normal Hb of 140g/l, calculate the total oxygen carrying capacity of the blood in ml/l. Three fictional patients are shown in Table 3, but this time their Hb concentrations are given. Calculate each patient's maximum oxygen-carrying capacity.



To calculate the total oxygen carrying capacity of the blood in ml/l, you need to multiply the concentration of Hb by the oxygen-carrying capacity. Thus: 140g/l x 1.34ml/g = 187.6ml/l (grams of Hb per litre of blood) x (millilitres of oxygen per gram of Hb) = (millilitres of oxygen per litre of blood). So, for a person with a Hb of 140g/l, the maximum oxygen-carrying capacity is approximately 188ml of oxygen in every litre of blood.

From Table 3, to calculate each patient's maximum oxygen-carrying capacity you need to take the Hb concentration in grams per litre

Table 2. Normal levels of haemoglobin

|   | Adult males | Adult females | Child (2 to 6 years) | Infant  | Neonate |
|---|-------------|---------------|----------------------|---------|---------|
| Haemoglobin (Hb) (grams per litre of blood) | 140-180     | 120-160       | 110-140              | 100-150 | 140-240 |
| (Bray <i>et al</i> 1999)                    |             |               |                      |         |         |

Table 3. Haemoglobin concentrate

| Name                                 | Mr Adams | Mrs Bryant | Mr Charles |
|--------------------------------------|----------|------------|------------|
| Age (years)                          | 56       | 42         | 78         |
| Pulse oximeter reading (per cent)    | 84       | 94         | 99         |
| Haemoglobin (Hb) concentration (g/l) | 170      | 110        | 100        |

and multiply it by 1.34ml per gram. Mr Adams's maximum oxygen-carrying capacity is 227.8ml/l; Mrs Bryant's is 147.4ml/l and Mr Charles's is 134ml/l. However, not every Hb molecule in the blood will be carrying its maximum of four oxygen molecules. The pulse oximeter is able to detect how much of the Hb in arterial blood is saturated, giving a percentage reading. Oxygen saturations for the three patients are also shown in Table 3. The actual amount of oxygen being carried in the blood can be determined by multiplying the maximum carrying capacity of the Hb by the percentage of molecules carrying oxygen.

#### TIME OUT 7

Look at Table 3 again and, using the figures you calculated above, determine the amount of oxygen in each patient's blood.



The calculations to determine the amount of oxygen in each patient's blood are as follows:

■ Mr Adams: multiply 227.8ml/l by 84/100 = 191.4ml/l.

■ Mrs Bryant: 147.4 ml/l x 94/100 = 138.5ml/l.

■ Mr Charles: 134 ml/l x 99/100 = 132.7 ml/l.

It is apparent from these calculations that even though Mr Charles has the highest pulse oximeter reading, he has the least oxygen in his blood. Mr Adams, in contrast, has a relatively poor oximeter reading, but is carrying more oxygen because he has a higher Hb level.

In the clinical setting, it is important to consider Hb levels when determining whether a patient is

## Respiratory nursing



Table 4. Patient data: stroke volume

| Name                        | Mr Adams | Mrs Bryant | Mr Charles |
|-----------------------------|----------|------------|------------|
| Age (years)                 | 56       | 42         | 78         |
| Heart rate                  | 80       | 100        | 110        |
| Stroke volume (ml per beat) | 70       | 50         | 35         |

getting sufficient oxygen to the tissues, particularly if performing initial or one-off oximeter readings. If oxygen saturation is monitored over a period of time, it is the alterations in the pattern of readings that are important, not one-off measurements. A drop in the oxygen saturations below 90 per cent in a person with normal Hb levels, is less catastrophic than a similar drop in saturations in a person with a low Hb level.

#### Cardiac output

Even with normal Hb levels and saturations, oxygen will not be delivered to the tissues if blood circulation is inadequate. Cardiac output is the third factor that needs to be assessed to obtain an accurate indication of oxygenation status. In clinical practice, it is not usual to measure cardiac output outside intensive therapy units. However, cardiac output can be estimated by using the patient's blood pressure and pulse readings.

Cardiac output is the volume of blood ejected by the ventricles per minute (Bray *et al* 1999). It is measured in litres per minute and is determined by the volume of blood ejected from the ventricles at each beat (stroke volume) multiplied by the number of beats per minute (heart rate). An average adult male has a cardiac output of about 5 litres per minute at rest. Stroke volume is approximately 70 millilitres per heart beat (Bray *et al* 1999).

While stroke volume is not readily measured in practice, it can be assessed by calculating the difference between systolic and diastolic blood pressure readings. This difference is referred to as the pulse pressure. A person with a blood pressure reading of 120/80mmHg has a pulse pressure of 40mmHg. A smaller pulse pressure indicates a lower stroke volume. Stroke volume can also be evaluated by feeling the radial artery pulse when taking a patient's pulse measurement. A firm or strong pulse indicates a good stroke volume, while a weak or thready pulse indicates low stroke volume (Ahrens 1987). In addition, an elevated pulse can indicate falling

stroke volumes because the heart rate needs to be increased to maintain a normal cardiac output.

#### TIME OUT 8

Table 4 contains information about stroke volume and heart rate for each of the three patients being assessed. Use these figures to calculate the cardiac output for each patient, and complete the table in the spaces provided.



The factors that affect cardiac output can be divided into two main categories: those that involve changes in the amount of blood returning to the heart (venous return), and those that affect the ability of the heart muscle to contract and pump blood back into the circulation. Venous return is affected by changes of posture. In particular, changing position from lying to standing can cause a sudden decrease in venous return and a fall in cardiac output. However, the body rapidly compensates for this by constricting veins and increasing the amount of blood being returned to the heart. The rapid loss of large volumes of blood (greater than 30 per cent of total blood volume) will cause reduced venous return, reduced stroke volume and, therefore, a severe fall in cardiac output. This fluid loss must be replaced immediately to maintain cardiac output and tissue oxygenation.

The ability of the heart muscle to contract is most obviously affected by myocardial infarction. Here the damaged muscle can reduce contractility and thus the stroke volume. Failure of cardiac valves and congestive heart failure can also have this effect. Hypertension can also cause a decrease in stroke volume because the high pressure in the arterial circulation makes it more difficult for the ventricles to pump blood around the body.

#### TIME OUT 9

Recall a patient you have cared for recently who might have experienced reduced cardiac output. Using the equation: cardiac output = stroke volume x heart rate, reflect on how the disease process affected these elements and list the compensatory changes that might have occurred.



A good cardiac output is important to maintain tissue oxygenation. For example, you can see from Table 5 that Mr Charles has a poor cardiac output (3.85 l/min). His stroke volume is reduced because of mitral valve disease. His





heart rate is elevated to 110 beats per minute (Table 4) to compensate for his reduced cardiac output, and this has maintained his cardiac output at a reasonable level. If his heart rate had not increased and had remained at 80 beats per minute, his cardiac output would only be 2.8 l/min. Of the three factors – oxygen saturation, Hb level and cardiac output – cardiac output is the most vital. All the elements involved in tissue oxygenation are presented in Table 5. The last row in the table shows how much oxygen is reaching the tissues for each patient. Based solely on a pulse oximeter reading, Mr Charles has the best reading of 99 per cent, but you can see that he has the worst oxygen delivery of the three patients. Conversely, Mr Adams, who has the worst oxygen saturation, has the best tissue oxygenation.

At rest, the healthy body uses 250-300ml/min of oxygen. This can rise to between 3-4 l/min during exercise (Bray *et al* 1999). Any condition that causes an increase in metabolic rate will increase the body's demand for oxygen, for example, pregnancy, growth, fever, stress and shock.

The movement of oxygen from the blood to the cells in the tissues occurs by a process of diffusion, similar to that in the lungs. The concentration of oxygen in the body cells is almost non-existent, because it is being used constantly to make ATP. Therefore, a steep gradient exists for rapid diffusion of oxygen out of the capillaries into the tissues. The main factors affecting this process are impaired blood supply to a specific area, for example, in atherosclerosis (Porth 1994), and the distance of individual cells from the nearest capillary. If the distance oxygen has to travel from the capillary to cell exceeds about 50 micrometers, the cell will become hypoxic. This distance is increased in patients with oedema, where the presence of extra fluid in the tissues moves some cells beyond the supply of the nearest capillary (Bray *et al* 1999).


### Conclusion

This article has outlined the processes that stimulate oxygen transport and delivery. These processes determine the saturation reading given by the pulse oximeter. It has also shown that it is important to evaluate several factors when assessing a patient's oxygenation status. Rather than relying

Table 5. Patient data: elements of tissue oxygenation

| Name                                 | Mr Adams | Mrs Bryant | Mr Charles |
|--------------------------------------|----------|------------|------------|
| Age (years)                          | 56       | 42         | 78         |
| Pulse oximeter reading (per cent)    | 84       | 94         | 99         |
| Haemoglobin (Hb) concentration (g/l) | 170      | 110        | 100        |
| Oxygen content of blood (ml/l)       | 191.4    | 138.5      | 132.7      |
| Cardiac output (l/min)               | 5.6      | 5          | 3.85       |
| Oxygen transport to tissues (ml/min) | 1,072    | 693        | 511        |

solely on the reading from a pulse oximeter, it is possible to gain a more accurate picture of the patient's status by incorporating assessment of Hb levels and cardiac output. It is important that you combine the information provided by the pulse oximeter with a physical assessment of the patient, looking for signs that indicate poor oxygenation, for example, increased respiratory and heart rates, and the use of accessory abdominal muscles as compensatory mechanisms in poor oxygenation. Other signs, such as fatigue and central nervous system disturbances (for example lethargy, restlessness and confusion) and central cyanosis, appear as the tissues become deprived of oxygen.

Nurses are using pulse oximetry more frequently in practice. It is important that they understand the underlying physiology of oxygen transport and delivery and the indicators of poor tissue oxygenation to make careful use of pulse oximetry. While pulse oximetry is a useful way to monitor oxygen saturation by measuring the amount of oxygen being carried by haemoglobin in the arterial blood, it cannot be relied on as the sole monitor of oxygen delivery to the body cells. It is important to remember that the ultimate purpose of evaluating the patient's oxygen status is to determine how much oxygen is reaching the cells of the body .

### TIME OUT 10

Now that you have completed the article, you might like to think about writing a practice profile. Guidelines to help you are on page 55.



### REFERENCES

- Ahrens T (1987) Concepts in the assessment of oxygenation. *Focus on Critical Care*. 14, 1, 36-44.
- Bray J *et al* (1999) *Lecture Notes on Human Physiology*. Fourth edition. Oxford, Blackwell Science.
- Carroll P (1997) Using pulse oximetry in the home. *Home Healthcare Nurse*. 15, 2, 88-95.
- Higgins C (1995) Full blood count (RBC, Hb, PCV, MCV, MCH and reticulocytes). *Nursing Times*. 91, 7, 38-40.
- Porth M (1994) *Pathophysiology: Concepts of Altered Health States*. Fourth edition. Philadelphia PA, Lippincott.
- Simpson K (1998) Fetal oxygen saturation monitoring during labor. *Journal of Perinatal and Neonatal Nursing*. 12, 3, 26-37.
- Woodrow P (1999) Pulse oximetry. *Nursing Standard*. 13, 42, 42-46.