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Continuing Professional Development

Assessing cardiovascular status: a guide for acute care nurses

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Learning Objectives
1. perform a cardiovascular assessment in order to determine the cardiovascular status of an adult acute care patient;
2. recognise cardiovascular dysfunction; and
3. take appropriate actions to prevent further deterioration in cardiovascular status.

Introduction
The function of the cardiovascular system is to transport oxygen, nutrients and hormones to the body’s cells and to remove waste products from the cells (Darovic 2002). The delivery of oxygen and nutrients will not be adequate in all regions of the body if there is insufficient blood flow at insufficient pressure. Cardiac output is the principal determinant of the adequacy of cardiovascular function. Adequate oxygen and nutritional levels in the blood also contribute to the effectiveness of cardiovascular function.

Many patients admitted to acute care areas of a hospital experience cardiovascular compromise due to conditions such as acute myocardial infarction (AMI), acute coronary syndrome or exacerbations of chronic heart failure. Additionally, patients can experience cardiovascular collapse due to bleeding or cardiac dysrhythmias postoperatively. As a consequence, nurses in acute care settings need to be competent in assessing the cardiovascular status of adult patients. The aim of this paper is to provide a framework for assessing the cardiovascular status of patients in acute care settings using the determinants of cardiac output. We provide a brief review of the determinants of cardiac output before discussing both the aims of cardiovascular assessment and how to perform such an assessment.

Physiological determinants of cardiac output
Cardiac output is the amount of blood ejected by the heart in litres per minute. The average cardiac output in the resting state is approximately 5 litres per minute (L/min), but may vary in the healthy individual from 4 L/min to 8 L/min (Daily and Schroeder 1994). Cardiac output is determined by heart rate and stroke volume (see Figure 1). In turn, stroke volume is determined by preload, afterload and contractility (see Figure 1). The aforementioned terms are explained briefly in the following text.

Figure 1: Determinants of cardiac output

Stroke volume is the amount of blood ejected with each heart beat, and is the difference between the volume of blood in the ventricle just prior to systole and the volume of blood remaining in the ventricle after systole. The normal range of stroke volume is 60 to 130 mL (Daily and Schroeder 1994).

Preload refers to the amount of blood in the ventricles just prior to systole. Right heart preload is clinically estimated by assessing jugular venous pressure (JVP). Right heart pressure can also be measured as a central venous pressure (CVP) via a central venous catheter. Left heart preload can be measured as a pulmonary artery wedge pressure via a pulmonary artery catheter within critical care areas.

Afterload refers to the resistance in the arterial circuit that the heart has to pump against. Cold, shutdown peripheries suggest a high resistance or increased afterload. An increase in afterload means that the ventricle has to pump more forcefully than usual to eject blood. Resistance in the systemic arterial circuit can be quantified by calculating a systemic vascular resistance in critically ill patients with a pulmonary artery catheter.
Contractility refers to the effectiveness of the heart’s pumping action. In critical care areas, cardiac output can be measured and quantified via a pulmonary artery catheter using the thermodilution method. The cardiac index can then be calculated from the cardiac output and body surface area, and used as a guide to assess the integrity of contractility. In addition, the left and right ventricular stroke work indices can be calculated to provide a specific measure of contractility; however, such values are rarely used. In acute care areas, there is no way for nurses to clinically measure or quantify contractility. Inferences about the state of cardiac contractility are made according to pathologies known to cause myocardial damage such as an AMI. For example, contractility is likely to be decreased in a patient with an ischaemic myocardium. Additionally, inferences regarding contractility are made based on the state of the other determinants of cardiac output. That is, if cardiac output is decreased in the presence of normal heart rate, heart rhythm, preload and afterload, then contractility is assumed to be reduced.

Aims of assessing cardiovascular status
The aims of assessing cardiovascular status are to collect, verify and communicate data related to the patient’s cardiovascular status; respond to such data with therapeutic interventions in the most appropriate manner according to the patient’s individualised needs; evaluate patient responses to therapeutic interventions; and establish a therapeutic relationship with the patient.

Assessment of the cardiovascular system involves the integration of clinical, laboratory and radiological assessments to provide an overall impression of the patient’s cardiovascular status. Cardiovascular assessment is reliant on the clinical assessment techniques available to nurses in any acute setting rather than on data obtained from invasive monitoring devices. In major centres where invasive monitoring devices may be used, data from such devices need to be confirmed by and integrated with clinical assessment findings. Cardiovascular assessment findings should be used to assess the impact of current therapy and determine the most appropriate course of management. Importantly, assessment of the cardiovascular status of patients should occur on an ongoing basis, not as an isolated event.

Clinical assessments are open to inaccuracies and/or misinterpretation. Such misinterpretations may occur as a result of patient-related variables such as chronic disease, clinician-related variables such as inadequate hearing or lack of knowledge, or technology-related variables such as a faulty stethoscope or an incorrectly sized blood pressure cuff. In order to minimise the impact of any possible inaccuracies, care should be taken not to assume that individual findings provide more information than they actually do. The totality of assessment findings should make the strongest impression, not an individual finding. For example, an assessment of peripheral perfusion only provides an indication of overall limb perfusion.

If the temperature, pulses and capillary return are adequate, one cannot assume that there is adequate perfusion to all areas of the limbs. Similarly, findings that suggest limb temperature, pulses and capillary return are inadequate may indicate chronic vascular disease rather than an acute deterioration in cardiac output.

Assessing cardiovascular status using the cardiac output framework
In this paper, the determinants of cardiac output are used as a framework to assess cardiovascular status. Such a framework is useful because it can be used to perform an extensive cardiac assessment – for example, on admission – or a rapid assessment during a critical incident. In the following text, we explain how to perform a rapid assessment for a patient with an acute change in their cardiovascular status that may have been caused by a life-threatening condition such as an AMI or a post-operative haemorrhage. An assessment of cardiovascular status is reliant upon nurses asking pertinent questions and using observation, palpation and auscultation skills.

Global assessment of cardiovascular function
The first stage of assessing cardiovascular function is to assess global perfusion. Assessing cerebral, renal, peripheral and respiratory perfusion allows nurses to rapidly establish an overall impression of cardiovascular status. The adequacy of each of the aforementioned areas of perfusion can be determined as follows:

- cerebral perfusion: alert and orientated;
- renal perfusion: ≥ 0.5 mL/kg/hour of urine;
- peripheral perfusion pressure: warm, dry, pink limbs; palpable peripheral pulses; capillary refill less than 2 seconds; and
- respiratory perfusion: oxygen saturation via pulse oximetry (SpO₂) ≥ 94%, absence of peripheral and central cyanosis.

Blood pressure can also be taken to provide important information about perfusion. In order to ensure adequate perfusion to organs, mean arterial pressure (MAP) usually needs to be greater than 70 mmHg (Higgins, Yared and Ryan, 1996). An estimation of MAP can be made by adding one-third of the systolic blood pressure value to two thirds of the diastolic blood pressure value. A MAP of 70 mmHg tends to correspond with a systolic blood pressure of 90 mmHg. Although blood pressure recordings should never be taken in isolation, a systolic value of less than 80 mm Hg usually warrants immediate additional assessments and responses. If adverse perfusion findings are found, clinicians are directed to a rapid cardiovascular assessment, as outlined in Figure 2 (see page 36).

Heart rate
Assessment of heart rate involves an assessment of rate, rhythm and pulse. Heart rate is readily assessed either by counting a radial pulse or looking at the displayed rate on a cardiac monitor. If using a cardiac monitor, it is essential that the equipment is set-up and applied to the patient appropriately.
output may remain stable for short periods if the tachycardia is neither sustained nor too rapid. Hence, ongoing assessments of cardiovascular function, including blood pressure, are important to determine the level of cardiovascular compromise caused by a tachycardia.

On finding a patient tachycardic, nursing responses must include attempts to identify likely causes of tachycardia. Common causes of tachycardia in the acute care setting include pain, fever, anxiety and hypovolaemia (see Table 1). Pain may be alleviated through comfortable positioning and administration of analgesics; fever may respond to antipyretics and cooling measures; anxiety may be reduced through open communication, explanations and administration of sedatives; and hypovolaemia may be treated by administering intravenous fluid therapy. Tachycardias caused by myocardial dysfunction, such as ventricular tachycardia, may need long-term treatment with anti-arrhythmic agents or an internal cardiac defibrillator.

Cardiac output may remain satisfactory when patients have a bradycardia because the increase in diastolic filling time allows

<table>
<thead>
<tr>
<th>Table 1: Factors that may cause a low cardiac output</th>
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<tbody>
<tr>
<td>Heart rate</td>
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<tr>
<td>Tachycardia</td>
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<tr>
<td>Fever</td>
</tr>
<tr>
<td>Chronic heart failure</td>
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<tr>
<td>Anxiety</td>
</tr>
<tr>
<td>Hypovolaemia</td>
</tr>
<tr>
<td>Pain</td>
</tr>
<tr>
<td>Bradycardia</td>
</tr>
<tr>
<td>Medications such as</td>
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<tr>
<td>B-blockers, calcium</td>
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<tr>
<td>channel blockers</td>
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<tr>
<td>Dysrhythmia such as</td>
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<tr>
<td>atrial fibrillation</td>
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<tr>
<td>Hypoxia</td>
</tr>
<tr>
<td>Hypokalaemia</td>
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<tr>
<td>Hypomagnesia</td>
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</tbody>
</table>

Figure 2: Rapid cardiovascular assessment

Rapid Cardiovascular Assessment
When confronted with a patient who appears to be deteriorating rapidly in relation to their cardiovascular status, a systematic rapid assessment must be undertaken. Such an assessment should focus on the identified problem and encompass the following principles:

General
General appearance of the patient, particularly in regard to level of consciousness, patient position, central colour, diaphoresis

Airway
Does the patient have a patent airway?

Breathing
Respiratory rate and rhythm, effort of breathing

Circulation
Presence of central pulse – femoral or carotid
Pulse rate, rhythm and volume
Peripheral colour and temperature, is skin dry or clammy?

Additional
Presence of chest pain, or neck, arm or central back pain
Depth of breathing, air entry to lung fields, auscultation of adventitious sounds, SpO2
Blood pressure
Presence of nausea or vomiting
Jugular venous pressure
Physical appearance of any wounds
Pain surrounding any wounds
12 lead electrocardiogram

Figure 3: Frank-Starling Law

Frank-Starling Law

The Frank-Starling Law describes the response of the cardiac output to increases in preload. The principle of this law is that there is an increasing cardiac output in response to the increasing myocardial fibre length that occurs with increases in preload. This increase only occurs to a certain point, beyond which the myocardial fibres are considered to be ‘overstretched’ and any further increases in preload lead to reduced cardiac output. The optimal preload differs for each individual person and is determined by the level of dysfunction of the myocardial fibres. Clinical states such as myocardial infarction or heart failure cause such dysfunction. (Guyton & Hall, 2001)
for an increase in stroke volume, thereby compensating for a low heart rate. According to the Frank-Starling Law (see Figure 3), an increase in ventricular filling results in greater shortening of ventricular muscle fibres, which, in turn, leads to increased muscle shortening, an increased stroke volume and more effective ejection. However, sinus bradycardia may also be a late sign that the heart is failing in its ability to respond to a reduced blood pressure with an increase in heart rate. In such a situation, compensatory mechanisms may be failing (see Figure 4, page 37 for details about compensatory mechanisms). A sinus bradycardia may also be deliberately induced by drug therapies to reduce the heart’s workload after an AMI. Other common clinical conditions that can influence heart rate and cardiac output are listed in Table 1 (see page 36).

Cardiac monitoring has the advantage of showing the heart rhythm as well as the heart rate, and may provide immediate direction to clinicians regarding the most appropriate responses and response times. For example, ventricular tachycardia displayed on a cardiac monitor would indicate to clinicians that rapid responses in terms of further clinical assessments, calls for assistance and pharmacological therapies may be required. Care must be taken, however, not to assess the cardiac monitor in isolation from the patient. Electromechanical dissociation or pulseless electrical activity is a condition in which the patient is asymptomatic when the rate is less than 100 bpm, blood pressure may still be compromised because loss of atrial contribution to ventricular filling (i.e. loss of atrial kick) can lead to a decrease in ventricular volume of up to 20 percent (Davidson et al. 2004). An acute episode of rapid atrial fibrillation warrants immediate assessment of blood pressure, and investigations to detect possible antecedents such as hypoxia and hypokalaemia.

In addition to vital information about heart rate and cardiac output, palpation of pulses provides information about perfusion quality, particularly in peripheral limbs. As illustrated in Table 2, documentation of findings may include a grading scale. Weak, thready pulses suggest cardiac output may be reduced. Full, bounding pulses may indicate fever and a very high cardiac output that could lead to cardiac compromise.

Nurses tend to assess heart rate by palpating a peripheral pulse such as the radial artery. If the patient’s conscious state is an issue, the carotid pulse should always be palpated. Clearly, an absent carotid pulse and loss of consciousness requires nurses to instigate full cardiopulmonary resuscitation.

**Preload**

Preload is influenced by factors that alter the overall fluid status of the body. When assessing preload, consideration needs to be given to the amount of fluid in the body and the distribution of that fluid. Assessments of overall fluid status involves auscultating breath sounds; monitoring the volume and type of oral, intravenous and enteral fluid administration; measuring urine output; and considering the fluid balance chart and patient’s level of thirst. In addition, assessments of overall fluid status include weighing patients at the same time each day, assessing the presence, severity, and distribution of oedema; checking tissue turgor; monitoring gastrointestinal integrity and activity; and measuring central or jugular venous pressures. As an adequate circulating volume is required for a satisfactory cardiac output, a low preload or hypovolaemia requires therapeutic interventions. Blood losses need to be replaced with fluids such as normal saline or blood products (synthetic or natural) that will boost circulating volume. Such fluid replacement is likely to moderate any concurrent compensatory tachycardia. Fluid therapy, however, should not be administered until hypovolaemia is established.
A raised preload may not necessarily indicate that cardiac output is satisfactory. Indeed, according to the Frank-Starling Law, patients with long-standing cardiac failure can develop a dilated myocardium that pumps ineffectively in response to sustained stretching of the ventricular muscle fibres. Clinical signs that may support suspicion of a high preload and inadequate cardiac output include shortness of breath, fine crackles in the lower-to mid-lung fields on auscultation, urine output less than 0.5 mL/kg/hr, weight gain of more than 2 kg in one day, swelling of ankles, lethargy, ascites, and raised JVP / CVP. The aforementioned clinical signs are suggestive of congestive heart failure and will require therapeutic interventions. Such interventions may include a restricted intravenous and alimentary fluid intake to 1.5 L/day, a dietary sodium restriction and diuretic therapy. Oral or intravenous potassium supplements may also be required depending on the type of diuretic used and serum levels measured.

If the patient develops shortness of breath, a reduced oxygen saturation on pulse oximetry or a decrease in mentation suddenly or over a few hours, acute pulmonary oedema should be suspected. Other signs that may be evident during acute pulmonary oedema include diaphoresis, cool clammy peripheries, use of accessory respiratory muscles, patients trying to sit up to catch their breath and oliguria. Such clinical findings warrant immediate reporting to appropriate personnel or a call for urgent medical assistance. In situations that develop suddenly, such clinical findings can lead to respiratory and cardiac arrest within 3 to 5 minutes. In the acute setting, adverse events such as acute pulmonary oedema may be averted by ongoing assessment of cardiovascular status in patients known to be at risk and some institution of appropriate management according to clinical findings.

**Afterload**

To assess afterload, that is, the degree of arteriolar resistance that the heart must pump against, nurses palpate peripheral circulation and measure blood pressure. A higher systolic blood pressure infers a higher peripheral resistance to ejection of an adequate stroke volume. In patients with a poorly contracting ventricular muscle, such as those with AMI or cardiomyopathy, increased afterload can significantly reduce cardiac output. In such patients, peripheral resistance may be reduced with drugs such as calcium channel blockers.

For patients without heart disease, a high peripheral resistance can be a compensatory mechanism to increase blood pressure for losses in circulating volume. For example, a patient who presents to the emergency department with pelvic injuries and internal bleeding sustained in a vehicle accident may have a cold, shutdown peripheral circulation caused by the body's need to maintain adequate perfusion to vital organs. In such patients, immediate treatments include fluid resuscitation to increase preload, stroke volume and blood pressure. Once circulating volume is re-established, peripheral resistance usually decreases.

A low afterload in the form of warm, dilated peripheral circulation and hypotension may be evident in patients with high fevers or sepsis. Patients with sepsis can present with full bounding pulses, warm peripheries, hypotension and a very high cardiac output. The heart, however, cannot sustain a high cardiac output for weeks and antibiotics to treat the sepsis are required along with drugs, such as intravenous noradrenaline, to vasoconstrict the peripheral circulation. Such therapeutic treatments are usually carried out in critical care units.

**Contractility**

There is no clinical assessment to measure myocardial contractility. Following assessments of heart rate, preload and afterload, inferences are made about contractility based on the patient’s overall clinical state. For instance, if the aforementioned parameters are found to be satisfactory and the patient has clinical signs of low cardiac output such as low urine output, reduced mental status or hypotension, it is likely that contractility will be deemed to be inadequate. Thus, inferences of contractility are made after clinical assessments have excluded other potential causes for deviations in cardiac output. The changes in contractility, preload, afterload and heart rate expected for clinical conditions commonly encountered in acute care settings are outlined in Table 3.

### Table 3: Clinical findings in common clinical conditions

<table>
<thead>
<tr>
<th>Parameter</th>
<th>AMI</th>
<th>Septic shock</th>
<th>Hypovolaemia</th>
<th>Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Tachycardia or bradycardia</td>
<td>Tachycardia</td>
<td>Tachycardia</td>
<td>Tachycardia or bradycardia</td>
</tr>
<tr>
<td>Preload</td>
<td>Increased or decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Increased</td>
</tr>
<tr>
<td>Afterload</td>
<td>Increased or decreased</td>
<td>Decreased</td>
<td>Increased</td>
<td>Normal or increased</td>
</tr>
<tr>
<td>Contractility</td>
<td>Decreased</td>
<td>Increased</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
</tbody>
</table>

**AMI** = acute myocardial infarction
A note about blood pressure

The individual value of blood pressure is frequently misinterpreted to be a direct indication of cardiac output, but this is not the case. Blood pressure is the amount of pressure exerted by blood on the walls of the arteries. It is created by the amount of blood flow or cardiac output flowing through a patient’s arterial system and the degree of arteriolar constriction.

As a result of the compensatory mechanisms that occur in response to changes in cardiac function (see Figure 4), it is most common for changes in blood pressure to be due to a combination of changes in both cardiac output and arterial constriction. For example, reduced cardiac output due to hypovolaemia as a result of postoperative bleeding is likely to be associated with a compensatory increase in arterial constriction that limits the reduction in blood pressure in the early stages of bleeding. Hence, although the patient may be normotensive, other clinical signs that would indicate to you that cardiovascular function was compromised in this situation might include cool peripheries, tachycardia and decreased urine output. As a result of a complex interplay between cardiac output and arterial resistance, measured blood pressure should never be taken as an isolated value, but, rather, as part of a total clinical assessment.

It is also important to note that changes in blood pressure do not necessarily indicate that cardiac output has changed in the same way. For example, a postoperative patient may be hypertensive due pain. In such an instance, the increase in blood pressure is not necessarily an indication that cardiac output has increased. In a similar manner, arterial vasodilation due to hyperthermia might result in a decreased blood pressure with no change in cardiac output.

Conclusions

The aim of this paper was to provide acute care clinicians with a framework for assessing cardiovascular status using the determinants of cardiac output. As many patients admitted to the acute care areas of hospitals experience acute cardiovascular compromise, clinicians need to be competent at assessing cardiovascular status. Cardiovascular deterioration may be due to changes in heart rate, heart rhythm, preload, afterload or contractility. An understanding of the determinants of cardiac output can assist acute care nurses to systematically assess cardiovascular function in order to optimise patient outcomes.

Review Comments

Nursing work has changed over the last decade so that we must care for sicker patients, often with complex illnesses and treatments and often outside our specialty knowledge. A nurse working in oncology may have a patient who is developing sepsicaemia and hypotension. A nurse working in orthopaedics may care for a patient with chronic heart failure. This article assists nurses in these situations by providing a framework for assessing and understanding cardiovascular status so that appropriate treatment can be planned. The authors use the framework of heart rate, preload, afterload and contractility to determine what assessment is needed and what the results mean in terms of the patient’s cardiovascular state. This framework is particularly successful because it is simple enough that the nurse does not have to be an expert, but complete enough to determine what actions are required.

A rapid assessment (figure 2) is provided to assess patients who are getting sicker fast and a quick reference table (3) to determine possible causes. Both are useful to the nurse in acute situations. The authors provide concise but thorough explanations of the physiology of the cardiovascular system and compensatory mechanisms. Although the authors refer to assessments provided in critical care, the main focus is on what can be done in acute care where less technology or support is available. This article would be particularly successful if it is followed by another describing treatments, particularly medications with the changes in parameters described. A section on the effect that aging might have on cardiovascular parameters would have been beneficial as so many of our patients are older and compensatory mechanisms may not be as efficient. Finally, a case study demonstrating the subtle changes in assessment, which contribute to a complete cardiovascular picture of the patient, would have been useful. It is often these patients, with small changes in heart rate, blood pressure and dyspnea, who we need to recognise and treat before more serious changes occur.

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Continuing education questions

1. Discuss the determinants of cardiac output
2. Describe your initial responses on finding a patient:
   a. short of breath
   b. tachycardic
3. Explain why the compensatory mechanisms activated in response to hypotension can be detrimental in the long term for patients with established cardiac failure
4. Describe the causes and associated management strategies of low preload in the acute care patient.

Review Comments

With the increasing acuity and complexity of patients on the general wards of most hospitals, nurses are now more than ever required to have an advanced knowledge of clinical assessment which can be incorporated into the care of their patients. The ageing population also means many patients in our hospitals have co-morbidities, which impact on their acute admission to hospital. This article provides advanced concepts of cardiovascular assessment in a framework that makes it easy to understand and apply to patient scenarios.

The authors provide clear explanations cardiac output, afterload, preload and stroke volume and directly relate these to assessment findings that may be evident in patients. The figures and tables included provide an easy guide for assessment in an acute situation. This article would be relevant to any nurse working in the acute setting who would often be faced with challenging clinical scenarios.

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References