CHAPTER 184
CARDIAC OUTPUT MONITORING

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KEY POINTS

- Cardiac output is the volume of blood transferred by the heart to the systemic circulation over time.
- It is a key determinant of oxygen delivery and an early indicator of hemodynamic instability.
- Cardiac output should be measured in any patient for which appropriate clinical decisions cannot be made without this information.
- Both invasive and minimally invasive methods of cardiac output measurement are available for clinical use in dogs and cats.
- Disease states can have a profound and complex impact on cardiac output.
- Complications of pulmonary artery catheters are rare, but placement should be done either by, or under the supervision of, experienced personnel.

Delivery of oxygen to the body and the removal of cellular metabolic waste are the fundamental roles of the cardiovascular and pulmonary systems. To accomplish these vital functions the pulmonary and cardiovascular systems must work in concert in a complex yet deeply integrated fashion. Each system relies on a pumping mechanism to accomplish the transport of blood or respiratory gases to the sites where the exchange of substrates and waste occurs.

In the case of the cardiovascular system, the heart provides the pumping force and the blood vessels serve to conduct and distribute the pumped blood to the tissues. The elastic properties of the vascular tree allow the force generated by the heart to be stored and applied to the column of flowing blood throughout the cardiac cycle. The volume of blood transferred to the systemic circulation over time is termed *cardiac output.* Cardiac output in humans is typically measured in liters per minute (L/min). Veterinary patients come in a broad range of shapes and sizes, and for this reason, cardiac output is often referenced in terms of milliliters of blood per kilogram of body weight per minute (ml/kg/min). Technically, this is a form of cardiac index because the values are being normalized (or indexed) to body mass; however, the term *cardiac output* is more generally applied to this parameter. Normal values for dogs and cats typically range from 120 to 200 ml/kg/min.[1,2] A related measure is formally called *cardiac index* and relates the volume of blood pumped over time to the animal's body surface area rather than body mass because the former is thought to correlate with metabolic rate (a principal determinant of cardiac output). The cardiac index is expressed in liters per minute per square meters ($L/min/m^2$).[2] The term *combined cardiac output* is used to describe the total volume of blood ejected into the systemic circulation over time when both the right and left ventricles can directly transfer blood to the arterial tree (e.g., fetal circulation, right-to-left patent ductus arteriosus).

Cardiac output is an important measure of cardiovascular function. It provides insights into bulk blood delivery to the body as a whole. When taken together with measurements of the oxygen content of blood, it allows for the determination of whole body oxygen delivery.[1,2] If one knows the patient's heart rate, then knowledge of cardiac output allows the clinician to determine stroke volume. Cardiac output measurements also make it possible for the caregiver to determine important physiologic indicators such as intrapulmonary shunt, systemic and pulmonary vascular resistance, and oxygen consumption. This large array of additional parameters that can be derived once cardiac output is known allow the clinician to potentially make better informed decisions about the need for, or adequacy of, therapeutic interventions and provides a more detailed picture of the patient's cardiovascular status.

INDICATIONS FOR CARDIAC OUTPUT MEASUREMENT

When performed by an experienced and attentive clinician, physical examination of the patient can reveal a great deal about the adequacy of oxygen delivery and cardiac output. Many of the findings of the physical examination relate directly to regional or organ-specific blood flow (e.g., capillary refill time, pulse pressure, mentation). Although these physical examination parameters are invaluable in the repeated assessment of patients and require little more equipment than a wristwatch, some are subjective measures and correlate poorly with an individual patient's actual cardiovascular status.[3] However, it must be noted that although an individual value for capillary refill time, for example, may correlate poorly with more direct measures of cardiac output, the trends in serial physical examination findings in an individual patient typically provide the best and most reliable measure of alterations in that patient's cardiovascular status. Unfortunately, the converse is not true: a patient whose physical examination findings are not changing may be experiencing a decline in cardiac performance that will not be detectable until compensatory mechanisms are exhausted or overcome.

The findings of a thorough physical examination, particularly when complemented with hemodynamic monitoring (see Chapter 183), are sufficient to guide the clinician in directing the care of most patients. However, there exists a subset of critically ill veterinary patients in which more direct assessment of cardiac output (and its derived parameters) is essential for proper case management. Patients with sepsis, septic shock, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome make up the bulk of veterinary patients for which more invasive measures of cardiac output are likely to be required. In patients with severe compromise of the pulmonary or cardiovascular system cardiac output monitoring may also be required to optimize their care. It is in the care of these patients that clinicians may find themselves unable to make appropriate decisions regarding management without the additional information provided via cardiac output monitoring.

In patients with complex disease states such as those mentioned earlier, the individual's cardiovascular and pulmonary systems may be compromised to such an extent that the typical measures of cardiovascular status and performance give contradictory information and suggest therapies that have opposing mechanisms of action (e.g., expanding or depleting extracellular fluid volume). An all-too-common example is a septic patient that has developed capillary leak syndrome (enhanced permeability of systemic capillaries and venules, promoting tissue edema). This patient typically has a low central venous or mean arterial pressure, or both (which suggests that additional intravenous fluid therapy might be of benefit), while at the same time exhibiting marked peripheral edema (which might lead the clinician to want to be less aggressive with fluid administration). The treatment of such a patient would be enhanced by knowledge of cardiac output and oxygen delivery, which are always of primary importance and can mandate a course of action in the face of conflicting findings. Cardiac output can also be a much earlier indicator of deteriorating cardiovascular status because compensatory mechanisms such as reflex vasoconstriction can maintain other indicators like mean arterial pressure near normal levels in the face of worsening cardiac performance.

MEASUREMENT OF CARDIAC OUTPUT

Invasive Methods of Determining Cardiac Output

Nearly all invasive techniques for measuring cardiac output rely on one of two methods: the Fick oxygen consumption method or the indicator dilution method. The commonly used thermodilution method is, in principle, a modification of the indicator dilution method using thermal energy as the indicator. Both methods are discussed here.[4]

Fick oxygen consumption method

The Fick oxygen consumption method is considered the gold standard and is the oldest method of measuring cardiac output. This technique relies on the Fick principle, which states that the total uptake (or release) of a substance by the peripheral tissues is equal to the product of the blood flow to the peripheral tissues and the arteriovenous concentration difference (gradient) of the substance. For a substance that is taken up by the tissues (such as oxygen), the Fick principle says in effect that "what went in minus what came out must equal what was left behind." The Fick principle when applied to cardiac output and oxygen uptake can be expressed as follows:

$$\text{Cardiac output} = \frac{\text{Oxygen consumption}}{\text{Arteriovenous oxygen content difference}}$$

When one uses the original Fick method to determine cardiac output, oxygen consumption is determined by measuring the oxygen concentration difference in the inhaled air and the exhaled air collected from the patient over time (typically 3 minutes). Alternatively, the arteriovenous oxygen content difference can be determined by measuring the oxygen content of both an arterial and a mixed venous blood sample. Although oxygen content analyzers are available, it is more typical for the clinician to measure the oxygen partial pressure (PO_2), hemoglobin saturation (SO_2), and hemoglobin concentration ([Hb]) with a blood gas analyzer and manually calculate oxygen content using the following formula:

$$\text{Oxygen content} = ([\text{Hb}] \times 1.36 \times SO_2) + (0.003 \times PO_2)$$

The principal drawbacks to this approach in veterinary medicine are that it is not a continuous real-time measure of cardiac output and that reliable collection of respiratory gases requires that the patient be intubated. In addition, use of the Fick method relies on the patient's remaining in a stable hemodynamic and metabolic state throughout the period of gas or blood collection; thus the less stable the patient's condition, the less reliable this method becomes. Lastly, results obtained by the Fick method are largely invalid in the presence of significant intracardiac or intrapulmonary shunting of blood.

Carbon dioxide rebreathing methods

The Fick equation can be used to determine cardiac output using carbon dioxide production rather than oxygen uptake. There are two methods, the complete rebreathing technique and the partial rebreathing technique. The following equation is used to calculate cardiac output using the complete rebreathing technique:

$$\text{Cardiac output} = \frac{CO_2 \text{ elimination by the lungs}}{\text{Arteriovenous } CO_2 \text{ difference}}$$

This technique requires breathholding and does not provide continuous measurements.

A monitor has been developed for the partial rebreathing technique (NICO). The partial rebreathing technique combines measurements obtained during a nonrebreathing period with values obtained during a rebreathing period. The following equation is used:

$$\text{Cardiac output} = \frac{\text{Difference in } CO_2 \text{ elimination and end-tidal } CO_2}{\text{Difference in arterial } CO_2 \text{ between baseline and rebreathing phase}}$$

The monitor is connected to a flow and carbon dioxide sensor and an adjustable dead-space breathing loop in the circuit between the patient and the Y piece. The monitor controls a valve that diverts gas flow through the breathing loop during the rebreathing phase. Values for cardiac output are determined every 3 minutes. This method measures only the pulmonary capillary blood flow that participates in gas exchange and calculates the shunt fraction. Values obtained using the NICO monitor have been shown to compare well with those obtained using the lithium dilution method in dogs; however, lower tidal volumes such as those used in lung-protective ventilation strategies have been shown to promote underestimation of cardiac output by partial rebreathing methods compared with the thermodilution technique.[1] The NICO monitor may not provide an accurate determination of cardiac output in smaller dogs.[2] The size of the rebreathing circuit also limits the use of the device in dogs and cats.

Indicator dilution method (including thermodilution)

In actuality, the indicator dilution method is simply an adaptation of the Fick method using indicators that are more easily collected and measured than elemental oxygen. The basis still lies in the Fick principle and conservation of matter (or thermal energy).

In this method an exogenous indicator is injected into the patient's mixed venous blood via a pulmonary artery catheter[5] (see Chapter 202), and the dilution of the indicator is followed continuously until both the original concentration peak associated with injection and a secondary peak due to recirculation are observed. By plotting the concentration of the indicator against time, one can obtain the area under the curve of the concentration versus time plot. Cardiac output is determined by taking the known amount of indicator and dividing it by the area under the curve. Typically this process is an integrated function of the software packages included with modern cardiac monitoring equipment. In the laboratory setting the indicator maybe a dye such as indocyanine green; however, this method is seldom used in clinical patients.

The indicator of choice is often thermal energy. Modern pulmonary artery catheters can be equipped with a sensitive thermocouple that can give highly accurate continuous measurements of blood temperature. This type of pulmonary artery catheter has been termed a *Swan-Ganz catheter* after the physicians who developed it and introduced it into clinical practice in human medicine.

Although the technology has advanced, the technique still relies on the Fick principle. By injecting a known volume of saline at a known temperature (typically room temperature; chilling is no longer needed with modern catheters) into the right-sided circulation, one can use the thermocouple to follow the dilution of this cool sample in the larger, warmer blood volume of the patient. Integration of this temperature signal can provide the clinician with a reliable measure of cardiac output. Recorded values are usually the average of three measurements taken in a short time, one after another. Good agreement is considered to be values that do not vary by more than 10%.

In thermodilution, the indicator is injected into the right atrium and dilution is measured in the pulmonary artery. Dye dilution is performed by injecting dye into the pulmonary artery and measuring the dilution at an arterial site. Transpulmonary thermodilution uses a central venous catheter and a thermistor that is inserted into the femoral artery. This method is potentially as accurate as using a pulmonary artery catheter, and studies in human patients have shown good agreement between the two in the values obtained.[3]

Advances in ion-specific electrode technology have led to novel means of applying indicator dilution principles to determine cardiac output in humans and animals. One such advance is the development of an electrode for lithium ions that can be placed in communication with the patient's arterial bloodstream via an indwelling arterial catheter. Such an electrode can be used to record the dilution of small doses of lithium chloride injected into the venous circulation at either a peripheral or central site. Cardiac output determination by this method has been studied in both dogs and cats, and agreement with cardiac output values obtained via thermodilution methods has generally been good.[6,7] Although the lithium dilution method for determining cardiac output can be termed *minimally invasive,* it is not truly noninvasive because it requires placement of both venous and arterial catheters.

Placement of pulmonary arterial catheters is not a benign procedure, and indications for pulmonary artery catheterization in human patients are controversial. In a large population of critically ill patients, pulmonary artery catheterization was associated with increased 30-day mortality, increased cost of health care, and a longer hospital stay.[4] Another large study found no benefit to therapy directed by pulmonary artery catheter data over standard care.[5] A Cochrane Database systematic review of pulmonary artery catheterization found no difference in mortality or length of stay in critically

ill or surgical patients, but it did find increased health care costs associated with pulmonary artery catheterization.[6]

Noninvasive or Minimally Invasive Methods of Determining Cardiac Output

No consensus for pulmonary artery catheter use exists in veterinary medicine. Noninvasive or minimally invasive methods of measuring cardiac output have been developed due to concerns about complications and reliability of pulmonary artery catheterization. Techniques include transesophageal echocardiography, pulse contour analysis, and thoracic bioimpedance.

Transesophageal echocardiography has been used in humans and a number of animal species as a minimally invasive means of tracking changes in cardiac output and performance. Measurement of blood velocity (using Doppler frequency shifts) and aortic diameter (using echocardiography) allow estimates of stroke volume to be made. To obtain truly reliable and quantifiable measurements of cardiac output, one should initially (and periodically) calibrate transesophageal echocardiography measurements against measurements obtained by one of the more invasive methods discussed earlier. In studies involving anesthetized dogs, results using this method are mixed compared with results using thermodilution.[2,7] The utility of this method is also somewhat limited in small animal practice because of equipment limitations, the time required to obtain acceptable studies, patient tolerance of the probe, and the need for highly trained personnel to be on hand to make the measurements. However, it does hold promise in limited applications (e.g., evaluation and monitoring of anesthetized patients).

Measurement of thoracic electrical bioimpedance is a noninvasive method of evaluating changes in the conductivity of the thorax resulting from the pulsatile flow of blood within the thoracic cavity. Sets of electrodes similar to electrocardiograph electrodes are located superficially on the thorax. Although electrocardiograph electrodes simply measure voltage changes resulting from the intrinsic electrical activity of the heart, the electrodes used in the thoracic electrical bioimpedance method both measure and apply voltage. The principle behind the method is Ohm's law, according to which the conductivity (and impedance) of the thorax to the flow of current can be determined by applying a small known voltage to the patient's thorax and then measuring what portion of that initial voltage reaches a distant sensing electrode. Changes in thoracic blood volume (blood and tissue are good conductors, air-filled lungs are not) can be detected, and estimates of stroke volume and cardiac output can be made using computer algorithms. Although this method holds promise in humans, in whom the size and shape of the thorax are somewhat uniform, the variety of species and breeds seen by the small animal clinician may make any single algorithm of limited utility, and estimates may need to be compared with some frequency with results obtained using invasive methods.

Analysis of the arterial pressure waveform, or pulse contour analysis, is an additional form of algorithm-dependent monitoring and can allow real-time determination of cardiac output. Some computers using this technology require calibration before use (PiCCO and PiCCO *plus*) and some do not (FloTrac). Transpulmonary thermodilution (PiCCO, PiCCO *plus*) or lithium dilution (PulsCO/LidCO) is used for the initial calibration, and the PulsCO/LidCO system requires calibration every 8 hours. Determination of cardiac output by transpulmonary thermodilution requires a central venous catheter in addition to an arterial catheter. Once the system has been calibrated, heart rate, area under the curve, aortic compliance, and shape of the pressure curve are used to calculate cardiac output for each pulse waveform. There was good correlation between PiCCO *plus* determinations and cardiac output as assessed by an aortic flow probe in a canine model of hemorrhagic shock.[1] In dogs that have anesthesia-induced hypotension or have rapid changes in cardiac output, the PulsCO system does not accurately predict cardiac output compared with the lithium dilution method.[8,9] A potential disadvantage of the pulse contour analysis approach is that the manufacturers of these monitoring devices often advise that central arterial (aortic) waveforms be monitored rather than peripheral arterial waveforms. In a small animal patient this would generally be achieved by advancing a long catheter into the aorta from a femoral artery insertion site. Any device that requires this more labor-intensive form of achieving arterial access is likely to be used less frequently than those for which peripheral arterial access is known to be sufficient for accurate readings.

NORMAL VALUES

The normal values for cardiac output (and related and derived indexes) for dogs and cats are presented in Table 184-1. Values other than cardiac output and cardiac index are given for the reader's consideration but are discussed in greater detail elsewhere (see Chapters 183 and and 202). The normal values listed in Table 184-1 represent composite values obtained from the literature and measurements made on clinical patients and research animals at the School of Veterinary Medicine at the University of California, Davis.[2] These composites include values from animals that were sedated as well as lightly anesthetized animals. Values for fully awake animals might be considered true "normal" values but would not represent normal values for the setting in which clinical measurements are generally obtained.

POTENTIAL CAUSES OF ERROR

Any form of measurement of any parameter carries an intrinsic degree of error. It is the responsibility of the clinician and the nursing staff to avoid compounding this form of uncertainty by introducing additional sources of error (Table 184-2). To this end, clinicians seeking to measure cardiac output using any of the techniques discussed earlier should ensure that they have been trained by

Table 184-1 Normal Cardiopulmonary Values for Dogs and Cats

Parameter (Unit)	Dog	Cat
Heart rate (beats/min)	100-140	110-140
Mean arterial pressure (mm Hg)	80-120	100-150
Cardiac output (ml/kg/min)	125-200	120
Cardiac index (L/min/m^2)	3.5-5.5	—
Stroke volume (ml/beat/kg)	40-60	—
Systemic vascular resistance (mm Hg/ml/kg/min)	0.5-0.8	—
Mean pulmonary artery pressure (mm Hg)	10-20	—
Pulmonary vascular resistance (mm Hg/ml/kg/min)	0.04-0.06	—
Central venous pressure (cm H_2O)	0-10	—
Pulmonary artery wedge pressure (mm Hg)	5-12	—
Oxygen delivery (ml/kg/min)	20-35	—
Oxygen consumption (ml/kg/min)	4-11	3-8
Oxygen extraction (%)	20-30	—

Table 184-2 **Sources of Error in Cardiac Output Measurement (Thermodilution Method)**

Error Source	Brief Description	Adjustments
Respiratory cycle	Pulmonary artery blood cools during inspiration. Venous return varies with intrathoracic pressure.	Make measurements at end expiration.
Arrhythmias	Arrhythmias cause rapid and marked variations in stroke volume.	Treat arrhythmias as indicated.
Altered intracardiac flow	Shunting and regurgitation can cause some of the injectate to bypass the thermistor or delay arrival of some of the bolus volume.	Thermodilution technique may be invalid in patients with significant flow abnormalities.
Low cardiac output	Slow ejection causes warming of the injectate before it reaches the thermistor.	Further therapeutic interventions will be required to increase cardiac output before values will be valid or repeatable.
Injectate factors	Injectate may be the wrong solution, wrong volume, wrong temperature.	Triple-check all aspects of the bolus before injecting.
Thermistor factors	Thrombus may form on the catheter tip. Catheter may migrate. Catheter may be defective.	Check position and reposition or replace catheter as needed.
Additional infusions	Simultaneous infusion of large volumes of crystalloid or colloid solutions can interfere with thermistor detection of the bolus.	Either interrupt the fluid bolus or postpone cardiac output measurements as dictated by patient's needs.

experienced personnel and have suitable hands-on experience with the method before using it in clinical decision making. Misuse of data from Swan-Ganz catheters by insufficiently trained personnel has on occasion led to iatrogenic injury and poor outcomes, and subsequently the devices have fallen out of favor in some segments of human medicine.

All of the methods for measuring cardiac output that have been discussed rely on the patient's having stable hemodynamics throughout the study period (typically several minutes). In the case of the Fick method, reliable measurements also require that the patient have only small fluctuations in metabolic rate during the study period. With each of the methods discussed, the serial evaluation of measurements is of greater use than any single determination.

DISEASE STATES AND CARDIAC OUTPUT MEASUREMENT

Cardiac output is the product of stroke volume and heart rate. Disease processes that alter either of these factors may alter cardiac output (unless the disease affects both in opposite directions and to equal degrees). Decreasing heart rates may either improve or worsen cardiac output depending on the individual patient. Patients with stiff, noncompliant ventricles or tachyarrhythmias, for example, may benefit from a reduction in heart rate because of greater filling during diastole. Alternatively, a patient with advanced atrioventricular node disease may have reduced cardiac output due to low (ventricular) heart rate. The relationship between heart rate and stroke volume is complex. Moderate increases in heart rate can increase stroke volume via the "staircase effect," whereas greater increases in heart rate may instead reduce stroke volume via impairment of diastolic filling.

Generally, any condition that reduces stroke volume reduces cardiac output if heart rate changes are minimal. Stroke volume is determined by preload, afterload, and contractility. Preload is determined largely by cardiac compliance and filling pressures. Any disease state that reduces filling pressures (e.g., hemorrhage, dehydration) or ventricular compliance (e.g., pericardial tamponade) can reduce preload and cardiac output. Afterload is a complex determinant of stroke volume and is largely dependent on the tone of the vasculature (particularly arterioles) and compliance of the aorta, but in some patients it is influenced by physical abnormalities in the cardiovascular system (e.g., aortic stenosis, arteriovenous fistulas) or the rheology of the blood itself (e.g., hyperviscosity syndromes).

Any process that increases afterload may reduce cardiac output (e.g., α-adrenergic stimulation), and processes that reduce afterload (e.g., reduced blood viscosity, arteriolar dilation) may increase cardiac output. Contractility is a measure of the myocardium's intrinsic ability to generate force and eject blood independent of loading conditions. Contractility may, for example, be depressed by circulating mediators (e.g., sepsis, pancreatitis) or enhanced by β-adrenergic stimulation. Any alteration in a patient's cardiac output should prompt a careful consideration of how disease states may be altering heart rate, preload, afterload, and contractility. Factors known to adversely affect these determinants of cardiac output should be addressed whenever possible.

POTENTIAL COMPLICATIONS

The vast majority of patients in which cardiac output measurements are made experience no direct complications due to the instrumentation or procedures required. However, many complications can occur when hemodynamic data are misinterpreted, and this issue has been discussed earlier in the chapter. A small subset of patients in which Swan-Ganz or other pulmonary artery catheters are placed will experience complications related to the placement, presence, or maintenance of the catheter.[8] These complications include, but are not limited to, the following: catheter-related sepsis, pulmonary artery rupture, damage to cardiac structures, catheter knotting (possibly requiring thoracotomy), hemorrhage, and embolization. For these reasons and others, it is stressed that pulmonary artery catheter placement is not a technique to be learned without the guidance of experienced personnel.

Complications from lithium chloride injection have not been reported in dogs or cats. The other methods of cardiac output determination discussed earlier also are considered to have a very large margin of safety.

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