Cardiovascular Essentials
Sample

Anatomy and Physiology
Cardiovascular Assessment
Pulmonary Practice Principles
Hemodynamics

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HEMODYNAMICS

HEMODYNAMIC CONCEPTS

The primary function of the heart is to move blood forward. As blood is propelled forward, it carries oxygen to the tissues to support cell function. Hemodynamics refers to the movement or the circulation of blood. This complex physiologic process involves not only the heart but also the arteries, veins, and intravascular (circulating) volume. Clinical assessment provides many insights into a patient’s hemodynamic status. A good understanding of basic hemodynamics provides a background for clinical assessment and clinical decision making.

Cardiac Output

Cardiac output (CO) is necessary for the delivery of oxygen to the tissues. The “driver” of oxygen to the tissue is the oxygenated blood ejected from the ventricles. CO is the amount of blood ejected by the ventricle every minute. Each ventricle has its own CO. Unless specifically identified, the term CO is referring to the CO of the left ventricle. CO is used to assess the adequacy of blood flow. Normally, the left ventricle ejects 4–8 L/minute. The calculation of “index” with any hemodynamic parameter or calculation provides an adjustment of the parameter or calculation taking into consideration the patients height and weight (body surface area - BSA) and therefore, individualizes the parameter or calculation to that specific patient. The cardiac index (CI) is used to individualize the CO to the individuals body surface area. The normal CI is 2.5–4 L/min/m².

CO is comprised of two components: stroke volume (SV) and heart rate (HR). SV is the volume of blood ejected by the ventricle with each beat, therefore CO = SV × HR. The volume at the end of the diastole or the end diastolic volume varies from patient to patient and is generally greater in males than females. The EDV of the right ventricle is greater than the left ventricle. The EDV of the left ventricle in males is roughly 160 ml and in females 135 ml (Hudsmith et al., 2005). The ventricles never fully empty and will have an end systolic volume (ESV) that reflects the volume remaining after ejection occurs. The percentage of volume ejected with each beat is referred to as the ejection fraction (EF). Normal EF is about 50% to 60% of the EDV (Dennison, 2019). There are three mechanism that regulate SV: preload, afterload, and contractility. Changes in HR, preload, afterload, or contractility are the determinants of CO (Figure 1). Therefore, when evaluating CO all four components must be considered.

Stroke Volume

SV is the amount of blood ejected from the ventricle with each beat; normal SV is around 60-100 ml/beat. There is no way to directly measure SV, but it can be calculated. SV can be calculated by subtracting the measured volume in the ventricle at the end of diastole when the ventricle is full (EDV) from the measured volume in the ventricle at the end of systole after ejection is complete (ESV): SV = EDV-ESV. It can also

Figure 1: Components of Cardiac Output: Heart Rate, Preload, Afterload, and Contractility
be calculated with the following formula: \( SV = CO - HR \). However, this calculation does not consider the impact of various heart rates. Normal stroke volume index (SVI) is 35-60 ml/beat/m\(^2\). SV provides a more specific assessment of ventricular function than CO because tachycardia has the potential to increase CO to normal or near normal levels in the presence of low SV. Each ventricle has its own SV, however, in most discussions about SV the left ventricular SV is referenced.

The three primary mechanisms that regulate SV are preload, afterload, and contractility. Each ventricle has its own preload, afterload, and contractility. Low SV can be due to low preload, decreased contractility, or high afterload. Valvular dysfunction, such as mitral regurgitation, can also cause a decreased forward SV since some volume is directed back to the left atria. High SV can be caused by increased preload, low afterload, or increased contractility.

It is important to remember that while each ventricle is individually impacted by preload, afterload, and contractility respective to that ventricle, neither ventricle functions in a vacuum and each is dependent on the other. When one fails to work properly, the other will ultimately fail as well. For example, the ventricles share the septum and when dyssynchrony between ventricles occur in situations such as bundle branch block this alters cardiac function. Additionally, the left ventricle is 100% dependent on preload from the right ventricle.

**Linking Knowledge to Practice**

✅ It is important to utilize the “index” when available to accurately assess hemodynamic parameters. This provides an evaluation based on the patient’s body surface area and is a more accurate reflection of the functioning of the heart. For example, a female who is 5’3” tall and weighs 130 pounds has a body surface area of 1.61 m\(^2\). If she has a measured cardiac output of 5.0 L/min, which is in the normal range of 4-8 L/min, her calculated cardiac index is 3.1 L/min/m\(^2\). This cardiac index falls in the normal range of 2.5–4 L/min/m\(^2\). Compare this to male patient who is 6’3” tall and weighs 250 pounds with a body surface area of 2.413 m\(^2\). With a “normal” measured cardiac output of 5.0 L/min his calculated cardiac index is 2.07 L/min/m\(^2\), which is below the normal parameters for cardiac index of 2.5–4 L/min/m\(^2\). While both patients have a normal cardiac output of 5 L/min, the cardiac index evaluation demonstrates why a “normal” cardiac output may not be normal for all and supports using the cardiac index which is adjusted for body surface area to obtain a more accurate understanding of cardiac output in relation to the patient.

**Preload**

Preload (Figure 1) is defined as the stretch on the ventricular myocardial fibers at the end of ventricular diastole. The volume of blood filling the ventricles determines myocardial fiber length or stretch. According to Starling’s law, within physiological limits the greater the myocardial fibers are stretched during ventricular filling, the stronger the force of the subsequent contraction and the greater the SV (Figure 2) (Opie, 2004). However, when the myocardial fibers are overstretched from too much volume, SV will no longer increase and in instances of continuous overstretching SV may begin to decrease.

![Figure 2: Preload](image)
Venous return to the heart determines the amount of blood entering the right ventricle. Venous blood returned to the right atrium (RA) enters the right ventricle (RV) and is subsequently transferred to the left ventricle (LV). It is this volume of blood that produces the stretch of the myocardial muscle fibers during diastole. If venous return decreases below normal, as with hypovolemia, preload decreases as does SV. Conversely, excessive preload results in congestion and overstretching of the ventricle and ultimately loss of contractility from the overstretched myocardial fibers.

**Right Ventricular Preload**

- Measured invasively by right atrial pressure (RAP) or central venous pressure (CVP) (Figure 3).
  - Normal: 2-6 mm Hg.
- Volumetric catheters allow for the measurement of right ventricular end diastolic volume (RVEDV).
  - Normal: 100-160 ml.
- **Noninvasive** findings with *increased* right ventricular preload include
  - Jugular venous distention
  - Hepatojugular reflux
  - Pitting peripheral edema* See Linking Knowledge to Practice
  - Increased weight * See Linking Knowledge to Practice
- **Noninvasive** findings with *decreased* right ventricular preload include:
  - Flat neck veins

![Diagram of heart with pulmonary artery catheter](image)

**Figure 3:** Assessment of preload with a pulmonary artery catheter. The section of the PA catheter that passes through the right atrium has a port that measures RA pressure or CVP and assesses RV preload. With the balloon inflated in the pulmonary artery, the distal port indirectly measures LA pressure and assesses LV preload.

**Linking Knowledge Practice**

✔ *Please note that pitting edema and increased weight may reflect increased right ventricular preload. However, there are other causes for peripheral edema and weight gain that are not related to increased preload so these two assessment findings should not be used as the only assessment tools. Jugular venous distention is a much more reliable indicator of increased right ventricular preload than weight gain and peripheral edema.*
Left Ventricular Preload

- Measured invasively by the mean pulmonary artery occlusive pressure (PAOP) (Figure 3) which is also referred to as the pulmonary capillary wedge pressure (PCWP), the pulmonary artery wedge pressure (PAWP), the pulmonary wedge pressure (PWP) or simply the “wedge”.
  - When the balloon on the tip of the pulmonary artery catheter is inflated, forward flow of blood is occluded distal to the tip of the catheter. This creates a static column of blood that reflects pressure back from the left atrium (Figure 3) providing a measure of left atrial pressure.
  - Normal: 5-12 mm Hg.
  - PAOP pressures are not an accurate reflection of left ventricular filling volume in the presence of mitral valve disease or decreased LV compliance.
  - The pulmonary artery diastolic (PAd) pressure can be used in place of the PAOP to assess left ventricular filling pressure if no pulmonary hypertension is present. The mean left atrial pressure can also be used if a left atrial catheter is in place.

Noninvasive findings with increased left ventricular preload include:
- Lung sounds are the physical assessment finding that reflect increased LV preload. Elevated LV filling pressures resulting in a backup of fluid in the lungs can manifest in the physical assessment with:
  - Inspiratory crackles
  - Dyspnea
  - Orthopnea
  - Decreased oxygen saturation
- Chest radiograph findings consistent with congestion

Noninvasive findings with decreased left ventricular preload include:
- Findings consistent with decreased LV preload are reflected in signs of deceased CO and perfusion to the tissues. Some of the earliest signs are:
  - Tachycardia with narrowed pulse pressure leading to hypotension
  - Cool skin
  - Decreased urine output

Preload and Ventricular Compliance

It is important to understand that when assessing preload using a pulmonary artery catheter, pressure is used to reflect end diastolic volume. In a normal ventricle, there is an expected relationship between volume and pressure (Figure 4). There are several situations however where pressure will not accurately reflect end diastolic volume.

- A stiff or noncompliant ventricle that does not expand easily while filling during diastole can produce a higher pressure with a normal volume status. Conversely, a large, dilated ventricle (more compliant) stretches easily but requires large volumes to fill the ventricle. It is difficult to produce a high pressure, even when the volume in the ventricle is higher than normal. Therefore, a change in pressure accurately reflects a change in volume only in the presence of a normally compliant ventricle.
• Increased compliance is seen in dilated cardiomyopathy and mild to moderate aortic regurgitation.
• A decrease in ventricular compliance (stiff / noncompliant) is seen with ventricular hypertrophy that occurs with moderate to severe aortic stenosis, untreated hypertension, or hypertrophic cardiomyopathy.
• Myocardial ischemia and restrictive cardiomyopathy also result in a stiff noncompliant ventricle.
• Elevated preload and increases in intrathoracic (positive pressure ventilation) and intra-pericardial pressures (pericardial effusion) will also decrease the compliance of the ventricle.

**Factors Influencing Preload**

There are many factors that can impact preload. Preload can be impacted not only by volume but also the location of the volume within the circulatory system and the ability of the blood to return to the heart and cause the myocytes to stretch. Some of the most common factors that influence preload include:

♦ Amount of volume / blood in the venous bed

♦ Venous tone
  • Venous vasoconstriction will increase venous return and ultimately increased preload.
  • Venous vasodilatation will decrease venous return and ultimately decreased preload.

♦ Body position
  • When a patient is hypotensive the act of lying the patient down and elevating their feet will passively increase venous return to the right side of the heart.
  • This maneuver can “bolus” the patient with 300-500 ml of volume depending on the size of the patient and the beginning position of the patient.

♦ Intrathoracic pressure and intra-pericardial pressure
  • An increase in these pressures limits the heart’s ability to expand during filling.
  • Positive pressure ventilation results in decreased preload and subsequently a lower CO.
  • Pericardial effusion results in decreased RV preload as the volume in the pericardial space can limit the expansion of the RV during diastole.

♦ Atrial kick
  • Atrial kick provides approximately 25% of ventricular filling. Loss of atrial kick with rhythms such as atrial fibrillation can result in decreased preload.

♦ Diastolic relaxation (ventricular compliance)
  • A stiff, noncompliant ventricle may slow passive ventricular filling and impact preload.

♦ Diastolic filling time (heart rate)
- When HR increases, the diastolic time shortens, decreasing the time available to fill the ventricle.
- Lower HRs are generally preferred to allow for adequate filling.

Left ventricular function (contractility)
- When left ventricular contractility is reduced the ESV is increased resulting in increased preload.
- With hypercontractile function preload can decrease as there is a lower ESV.

Causes of Decreased Preload
There are many factors that can decrease preload. Some of the most common reasons for a decrease in preload include:

- Decrease in circulating volume
  - Hemorrhage
  - Dehydration
  - Burns
  - Excessive diuresis
  - Third space shifting of fluids out of the vascular beds
- Changes in size of venous vascular bed (excessive venous vasodilation producing relative hypovolemia)
  - Sepsis
  - Anaphylaxis
  - Venous vasodilating medications (Examples include: NTG, ACE Inhibitors, Angiotensin Receptor Blockers, Calcium Channel Blockers -CCB, morphine)

Other consideration
- Loss of atrial kick
- Tachyarrhythmias (decreased diastolic filling time and / or dyssynchrony between atria and ventricle)
- Standing
- Positive pressure ventilation
- Hyperthermia (tachycardia)

Causes of Increased Preload
There are many factors that can increase preload. Remember preload is related to the stretch on the myocardial muscle fibers. This occurs with volume. However, in the stiff noncompliant ventricle smaller amounts of volume can be expressed as a higher pressure due to the lack of ability of the myocardium to expand during filling. Some of the most common reasons for an increase in preload include:

- Increase in circulating volume
  - Over hydration / hypervolemia.
  - Heart failure (backward failure results in pulmonary and systemic venous congestion).
  - Renal disease.

- Changes in size of vascular space (venous vasoconstriction will increase venous return)
  - Activation of the sympathetic nervous system
  - Activation of the renin angiotensin system
Increased pressure but not volume
  • Pericardial effusion / Tamponade
  • Tension Pneumothorax
  • Stiff, noncompliant ventricle (myocardial hypertrophy, hypothermia, restrictive cardiomyopathies)

Correcting Preload

To increase preload
  • Increase volume
    ▪ Isotonic crystalloids (isotonic solutions keeps the fluid in the vascular bed)
    ▪ Colloids (Hetastarch or Albumin draws fluid into the vascular bed)
    ▪ Blood or blood products when clinically indicated
    ▪ Stop diuretics
  • Change the size of the vascular bed – support venous vasoconstriction
    ▪ Decrease venous vasodilators
    ▪ Treat anaphylaxis or sepsis
  • Other considerations
    ▪ Rhythm control
      o Decrease heart rate
      o Restore atrial kick
      o Control arrhythmias to restore atrial and ventricular synchrony
    ▪ Change position (elevate feet)
    ▪ Treat hyperthermia

To decrease preload
  • Decrease volume
    ▪ Diuretics
    ▪ Ultrafiltration
  • Change the size of the vascular bed – support venous vasodilation
    ▪ Venous vasodilators
      o NTG
      o Morphine Sulfate
      o ACE Inhibitors, Angiotensin Receptor Blockers
      o Calcium Channel Blockers (“INE” CCB such as amlodipine)
  • Other considerations
    ▪ Increase contractility to improve forward flow.
    ▪ ACE inhibitors, angiotensin II receptor blockers, and aldosterone antagonists can also be used to manage preload by countering the effects of renin angiotensin system (vasoconstriction and fluid retention).
    ▪ Lower feet.

Linking Knowledge Practice

✔ Reducing preload reduces myocardial oxygen consumption. Preload reduction is the mechanism of action in low dose intravenous nitroglycerin used to treat ischemic chest pain. However, if preload is reduced to a level that is too low, CO and blood pressure will fall.
ADDITIONAL SAMPLE

Oxyhemoglobin Curve

The oxyhemoglobin curve represents the relationship between oxygen and hemoglobin.

- The partial pressure of the oxygen (PaO₂) in the plasma determines the amount of oxygen that binds with hemoglobin. When the PaO₂ is high, hemoglobin is highly saturated with oxygen, and when the PaO₂ is low, oxygen is released from hemoglobin.
- The relationship between PaO₂ and SaO₂ is displayed in the oxyhemoglobin curve (Figure 9).
  - The curve is S-shaped.
  - The upper part of the curve shows that a PaO₂ above 60 mmHg results in minimal changes in oxygen saturation. This flat part of the curve protects the body by allowing the hemoglobin to remain highly saturated when the PaO₂ ranges from 60 to 100 mmHg.
  - Hemoglobin is about 97% saturated at a PaO₂ of 100 mmHg, so raising the alveolar PaO₂ above 100 can add little more oxygen to the hemoglobin. Hemoglobin becomes fully saturated with the PaO₂ of about 250 mmHg.
  - The lower part of the curve shows that a PaO₂ below 60 results in a significant decrease in oxygen saturation. This allows the tissues to extract large amounts of oxygen with only small changes in PaO₂.
- On a normal oxyhemoglobin curve, a PaO₂ of 60 mmHg = SaO₂ of 90%.
- As the blood passes into the systemic capillaries, it is exposed to lower PaO₂ levels, and oxygen is released from the hemoglobin into the plasma. The high content of CO₂ in the venous capillaries also reduces the affinity that hemoglobin has for oxygen (a shift in the right of the oxyhemoglobin curve) and causes hemoglobin to release oxygen to the tissue. This dissociation of oxygen into the plasma allows for O₂ to diffuse into the tissues for use at the cellular level. The PaO₂ in the capillaries varies from tissue to tissue. The PaO₂ in the myocardium is exceptionally low. The oxyhemoglobin curve is steep so that a small drop in PaO₂ can lead to a substantial dissociation of oxygen from hemoglobin. This unloads more oxygen for use by the tissues.
- Hemoglobin is more willingly accepts CO₂ in the venous system when it is not bound to oxygen. Hemoglobin that binds to CO₂ on the venous side is called carbaminohemoglobin. This allows CO₂ to be returned to the pulmonary capillaries where it be exhaled from the body.

**Figure 9: Oxyhemoglobin dissociation curve.**

Linking Knowledge to Practice

- An ECG should be performed in patients with CO poisoning because of the high risk of myocardial injury associated with CO toxicity.
- Co-existing cyanide toxicity can occur with CO poisoning. A high anion gap metabolic acidosis (discussed below) is characteristics of a substantial cyanide toxicity.
- A lactate level of 10 mmol/L in the setting of smoke inhalation points to the co-existence of cyanide poisoning.

*(Clardy, Manaker & Perry, 2020; Leybell, 2020)*
The main factors that alter the normal association between oxygen and hemoglobin are pH, PaCO₂, temperature of the blood, and the concentration of a substance called 2,3-DPG in the erythrocytes. 2,3-Diphosphoglycerate (2,3-DPG) is a substance produced by erythrocytes during their normal glycolysis, which binds to hemoglobin and decreases the affinity of hemoglobin for oxygen.

**Shifts in Oxyhemoglobin Curve**

A shift in the oxyhemoglobin curve simply means there is a change in a physiological condition that has altered the relationship between oxygen and hemoglobin.

The normal relationship between oxygen and hemoglobin occurs at 37 degrees, pH 7.4, and PCO₂ of 40 mmHg. The impact of changes in pH, temperature, and 2,3-DPG on the relationship between oxygen and hemoglobin (oxyhemoglobin curve) are seen in Figure 9.

Shifts in the oxyhemoglobin curve are more pronounced when PaO₂ levels are low rather than high. Therefore, shifts have a more profound effect with unloading of oxygen at the tissue level than with the uptake of oxygen in the lungs.

**Shift of oxyhemoglobin curve to the left.**
- A shift in the curve to the left represents an increased affinity between oxygen and hemoglobin. It is easier for hemoglobin to pick up oxygen at the lung level, and more difficult to drop it off at the tissue level.
- Hemoglobin is more saturated for any given PaO₂.
- Less oxygen is unloaded at the tissue level for a given PaO₂. This results in decreased tissue perfusion despite a higher SaO₂.
- Causes of shift to left.
  - Decreased temperature (hypothermia)
  - Decreased hydrogen ions (hypocapnia, alkalemia)
  - Decreased 2,3-DPG. Conditions with decreased 2,3-DPG include massive transfusion of stored blood (banked blood stored for as little as one week has low levels of 2,3 –DPG), hypophosphatemia, and hypothyroidism.

**Shift of oxyhemoglobin curve to the right**
- A shift in the curve to the right represents decreased affinity between oxygen and hemoglobin. It is more difficult for hemoglobin to pick up oxygen at the lung level, but easier to drop off at the tissue level.
- Hemoglobin is less saturated for a given PaO₂, and more oxygen is unloaded for a given PaO₂.
- Causes of shift to right.
  - Increased temperature (hyperthermia).
  - Increased hydrogen ions (hypercapnia, acidemia).
  - Increased 2,3–DPG Conditions with increased 2,3-DPG include chronic hypoxemia, anemia, and hyperthyroidism.

**Helpful Hint:** A way to remember the causes for a shift to the right are found in Figure 10. *“Rise in 2, 3-DPG, Rise in Hydrogen ions, and Rise in Temperature” is used to spell “right.”*

**Definitions Related to Oxygen and Hemoglobin**
- Hypoxemia is defined as insufficient oxygenation of the blood
• Mild hypoxemia: $\text{PaO}_2 < 80 \text{ mmHg or SaO}_2 < 95\%$
• Moderate hypoxemia: $\text{PaO}_2 < 60 \text{ mmHg or SaO}_2 < 90\%$
• Severe hypoxemia: $\text{PaO}_2 < 40 \text{ mmHg or SaO}_2 < 75\%$

Hypoxia is insufficient oxygenation of tissues. It is determined by a combination of cardiac index, Hgb, SaO$_2$, patency of the vessels, and cellular demand. Hypoxia is not usually directly measured. End organ function is used to evaluate tissue hypoxia. Common patient signs that may indicate organ dysfunction caused by tissue hypoxia include decreased urine output and hypotension. Lactate levels are also used as an indicator for tissue hypoxia.

Cyanosis is a bluish-purple discoloration of the skin, nail beds, or mucous membranes. This discoloration is best seen in areas when the epidermis is thin and where there is a large quantity of blood vessels. These areas include lips, nose, cheeks, and oral mucous membranes, both buccal and sublingual. The patient’s skin pigment and the lighting in the room can interfere with the ability to detect cyanosis.

Cyanosis usually occurs when there 5 grams/dL of deoxygenated hemoglobin in the capillaries. It is an absolute rather than relative quantity of deoxygenated hemoglobin that causes cyanosis. The absence of cyanosis does not exclude hypoxemia. An anemic patient with hypoxemia may not have enough hemoglobin to express signs of cyanosis. In contrast, patients with polycythemia may have evidence of cyanosis at a higher SaO$_2$ than expected in a patient with cyanosis. The box below provides examples of how the baseline hemoglobin impacts the presence of cyanosis.

Cyanosis is described as:
• Central: Involving lips, tongue, and oral mucous membranes but also includes cyanosis of the periphery. In central cyanosis there reduced SaO$_2$ or there is the presence of abnormal hemoglobin such as methemoglobin.
• Peripheral: Peripheral only cyanosis is caused by decreased peripheral blood from an abnormal physiological condition such as vasoconstriction. This can be seen with high doses of vaso-pressors. The SaO$_2$ can be normal in peripheral cyanosis.
• Pseudo cyanosis: May seen due to drug effect rather than due to deoxygenated hemoglobin. Amiodarone, an antiarrhythmic medication, may produce pseudo cyanosis.

(McMullen & Patrick, 2013; Martin, 2015)

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<tr>
<th>Impact of Baseline Hemoglobin on Manifestation of Cyanosis</th>
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<td>A SaO$_2$ of 79% in a patient with a baseline hemoglobin level of 15 g/dL would generate 5 g/dL of deoxygenated hemoglobin and thus the cyanosis would be present.</td>
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<tr>
<td>However, in a patient with a baseline hemoglobin of 9 g/dL, it would take a SaO$_2$ of 65% to generate 5 g/dL of deoxygenated hemoglobin and for cyanosis to be present.</td>
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THIS ENDS THE ONLINE SAMPLE OF CARDIAC ESSENTIALS

This is just a small sample of CARDIAC ESSENTIALS that is rich in essential information. One of the best ways to advance your knowledge and practice is by making sure your base (not basic) knowledge is strong and that you are fully confident in these foundational concepts you use every day in practice. Do not let the anatomy discussion at the beginning of Cardiac Essentials mislead you into thinking you already “get it” and do not need this information. The information in Cardiac Essentials provides the foundation for your practice but also the base for the next 4 books we are publishing. The pulmonary section is chock-full of vital information that is presented in a manner that makes difficult concepts relatable to practice. In the hemodynamics section we will help you understand how to assess the components of cardiac output both with and without a pulmonary artery catheter to better understand how your patient
is doing. We also provide great information to boost your confidence if you are caring for a patient with a pulmonary artery catheter. Let us guide you on your journey to nursing excellence with a resource that will enhance those things you do every day as well as those things that are less common to you. With the information provided and the Linking Knowledge to Practice tips that help make the information practical you will develop expertise and gain confidence that you never had before. This will result in others recognizing that you are not only confident in your practice but that you are competent and a clinical resource they can count on. Most of all, your patients will be in the best hands! We hope you enjoy this book.

Thank you for your interest,

Carol, Karen, and Cindy