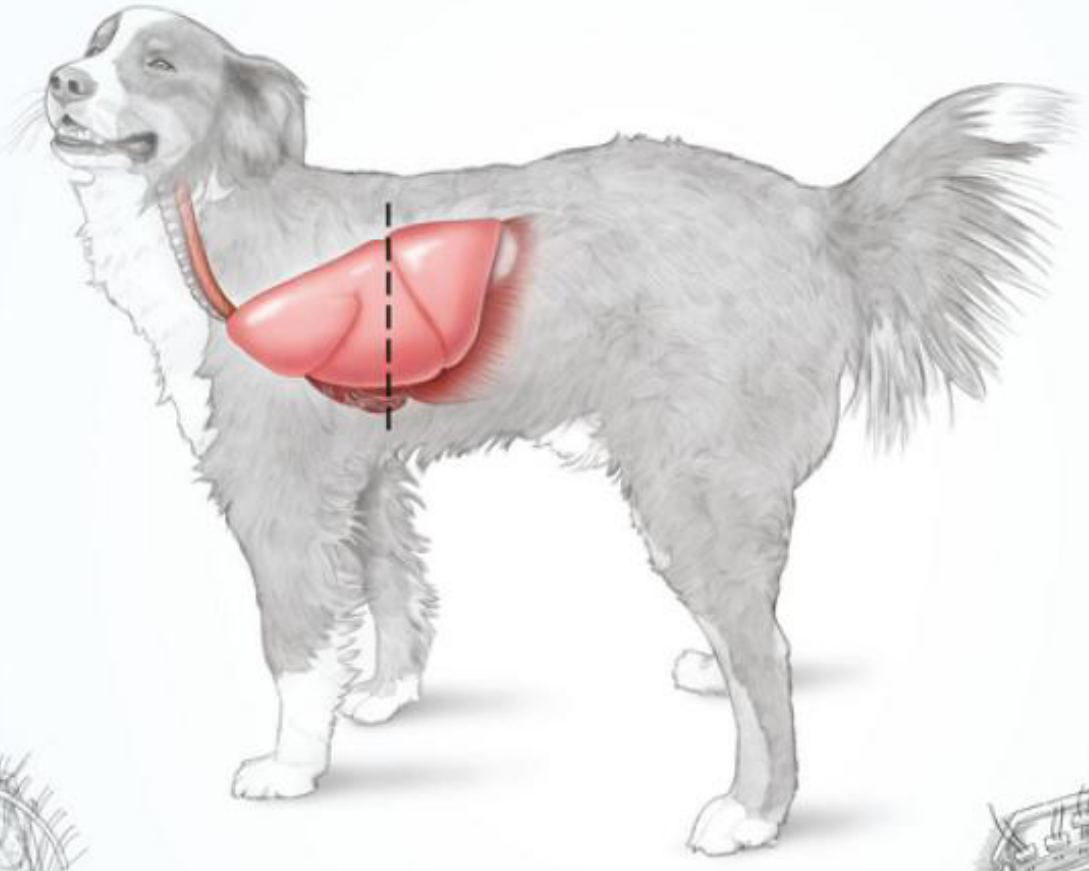
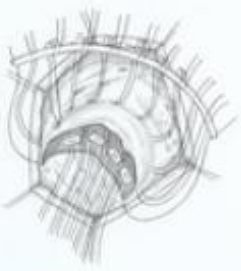
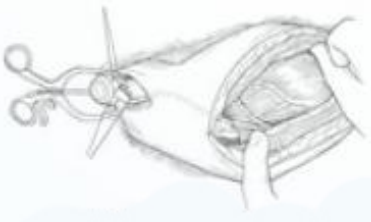
Small Animal Thoracic Surgery

E. Christopher Orton · Eric Monnet







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This book is dedicated to our patients, who have been our greatest teachers, and their families, who have shown us the depth of the bond between species.

Christopher Orton
Eric Monnet

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Preface

Thoracic surgery in small animals has grown in importance and prominence since the earlier publication of a book on the same subject. Several important advances in understanding of thoracic diseases and surgical technique have occurred, none more important than the development of minimally invasive techniques such as video-assisted thoracoscopic surgery.

In this first edition of a new and updated book on small animal thoracic surgery, emphasis has been placed on surgical technique, decision making, and perioperative care. We have endeavored to provide detailed descriptions of techniques that we have experience with and that have worked best in our hands. To this end, several new illustrations have been added to this new book, all beautifully drawn by Molly Borman. Her work blends seamlessly with the original illustrations by Thomas McCracken from the first book on the subject of small animal thoracic surgery. We are eternally grateful for the contributions of both of these talented individuals. Where alternate techniques and approaches exist, we have made every effort to reference the experience and techniques of others.

It is our hope that this textbook will prove useful to those seeking information from basic to advanced cardiothoracic surgery. As always, we welcome feedback from our colleagues who share our interest and passion for care of our small animal companions.

E. Christopher Orton
Eric Monnet

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/orton/thoracic

The website includes:

- videos of the procedures described in the book.

Section I

General Principles

1

Cardiopulmonary Function

E. Christopher Orton

A major function of the cardiopulmonary system is to deliver oxygen to tissues and eliminate carbon dioxide generated by tissue metabolism. To accomplish these functions, the respiratory and cardiovascular systems must act in close concert. Compromise of either system—or both systems—can adversely affect the outcome of animals undergoing thoracic surgery. The ability to quickly assess cardiopulmonary function and pinpoint the cause and severity of problems is firmly grounded in an understanding of cardiopulmonary physiology and pathophysiology. This ability is a core skill for those who undertake interventions in the thorax.

The Oxygen Pathway

The oxygen pathway is a clinically useful concept that provides a logical framework for evaluation and correction of disturbances in the cardiopulmonary system (Figure 1.1). It considers the transport of oxygen as a sequential, step-by-step process beginning with atmospheric oxygen and ending with oxygen delivery to tissues. Each step in the pathway is critically important and must be assessed independently to assure adequate overall cardiopulmonary function. The steps of the oxygen pathway can be viewed as a clinical checklist for monitoring cardiopulmonary function in animals before, during, and after thoracic surgery. Steps in the pathway include ventilation, pulmonary gas exchange, hemoglobin saturation, hemoglobin concentration, oxygen content, cardiac output, and oxygen delivery.

Ventilation

Ventilation is the mechanical process that causes air (a mixture of gases) to flow into and out of the lungs. Not all gas flow (L/min) into the respiratory system reaches areas of gas exchange; consequently, *total ventilation* or *minute volume* (V_T) is divided between *alveolar ventilation* (V_A), where gas exchange occurs, and *dead space ventilation* (V_D).

$$V_T = V_A + V_D \tag{1.1}$$

Anatomic dead space ventilation includes gas flow to anatomic areas not normally involved in gas exchange. Physiologic dead space includes anatomic dead space, as well as flow to alveoli that are ventilated but not receiving pulmonary blood flow. While anatomic dead space remains constant, physiologic dead space changes depending on the number of functioning alveoli. Furthermore, the ratio of V_D to V_A changes with the respiratory rate and *tidal volume* and cannot be easily determined clinically. For example, an animal that is panting increases V_T and V_D several-fold without necessarily changing V_A. Thus, the adequacy of V_A cannot be determined by just measuring V_T.

Carbon Dioxide Tension

The primary drive for alveolar ventilation is *arterial carbon dioxide tension* (P_aCO_2). Under physiologic conditions, the central respiratory center drives V_A to keep P_aCO_2 at about 40 mm Hg, regardless of the total amount of carbon dioxide produced (V_{CO_2}) based on the size, metabolism, and activity level of the patient. This relationship of P_aCO_2, V_A, and V_{CO_2} is described by Equation 1.2, where K is a conversion constant:

$$P_aCO_2 = \frac{V_{CO_2} \times K}{V_A} \tag{1.2}$$

By definition, *hypoventilation* is present when V_A fails to match V_{CO_2}, and as a result, P_aCO_2 increases (i.e., > 40 mm Hg for animals at sea level). Conversely, *hyperventilation* is present when V_A exceeds what is necessary to eliminate V_{CO_2} causing P_aCO_2 to decrease (i.e., < 40 mm Hg at sea level). Thus, in the

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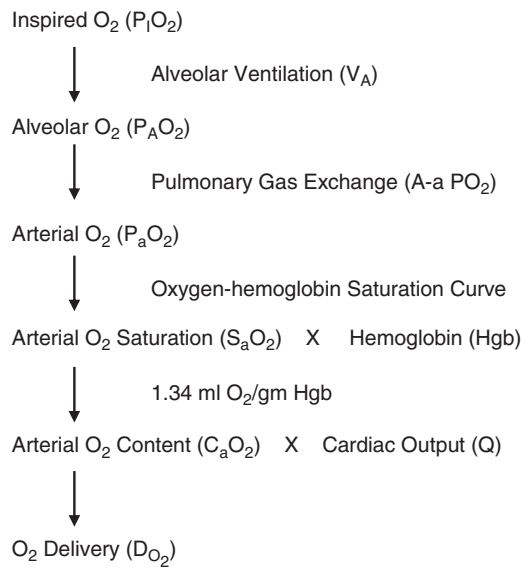


Figure 1.1 Oxygen Pathway

clinical setting, adequacy of ventilation is determined by P_aCO_2 from a blood gas analysis. If P_aCO_2 is normal based on the regional normal value, then ventilation to the gas exchange regions of the lung is considered adequate. (Note: The regional normal value of P_aCO_2 is altitude dependent because animals that reside at higher altitudes increase relative V_A to compensate for lower inspired oxygen tension.)

Alveolar Gas Equation

Because *arterial oxygen tension* (P_aO_2) cannot be higher than *alveolar oxygen tension* (P_AO_2), P_AO_2 is critically important to all subsequent steps in the oxygen pathway. P_AO_2 is not measured clinically, but can be estimated from the *alveolar gas equation:*

$$P_AO_2 = P_IO_2 - P_aCO_2/R \quad (1.3)$$

From above equation, it is apparent that P_AO_2 is a function of the *inspired oxygen tension* (P_IO_2), P_aCO_2 (and thereby V_A), and the *respiratory exchange ratio* (R). The respiratory exchange ratio is the ratio of *oxygen consumption* (V_{O_2}) to V_{CO_2}. The respiratory exchange ratio can be determined by indirect calorimetry, but this is not routinely done in the clinical setting. In a study of dogs evaluated by indirect calorimetry, R was found to be 0.76 in postoperative or post-trauma dogs compared to an R of 0.84 in normal dogs [1]. For purposes of the above calculation, R is generally assumed to be 0.8. The P_IO_2 is determined by the *fraction of inspired oxygen* (F_IO_2, 0.21 in ambient air), *barometric pressure* (P_B, 760 mm Hg at sea level), and the *vapor pressure of water* (P_{H_2O}, 47 mm Hg at 100% saturation and body temperature):

$$\begin{aligned} P_IO_2 &= F_IO_2 (P_B - P_{H_2O}) \\ &= 0.21 (760 \text{ mm Hg} - 47 \text{ mm Hg}) \\ &= 150 \text{ mm Hg (at sea level)} \end{aligned} \quad (1.4)$$

Thus, the P_IO_2 of room air at sea level is approximately 150 mm of Hg. From the above equation, it can be seen that either barometric pressure or F_IO_2 can alter P_IO_2, and, in turn, the P_AO_2. Substantial change in barometric pressure is most likely to result from residence at altitude, whereas F_IO_2 is altered clinically by administration of supplemental oxygen. Increasing F_IO_2 to 40% nearly doubles P_IO_2 and increases P_AO_2 without changing V_A.

Alveolar ventilation is the other major determinant of P_AO_2. The alveolar gas equation predicts that an animal breathing room air at sea level with a P_aCO_2 of 40 mm Hg would have a P_AO_2 of approximately 100 mm Hg:

$$\begin{aligned} P_AO_2 &= F_IO_2(P_B - P_{H_2O}) - P_aCO_2/R \\ &= 0.21(760 \text{ mm Hg} - 47 \text{ mm Hg}) \\ &\quad - 40 \text{ mm Hg} / 0.8 \\ &= 150 \text{ mm Hg} - 50 \text{ mm Hg} \\ &= 100 \text{ mm Hg} \end{aligned} \quad (1.5)$$

A rule of thumb, for every 1 mm Hg elevation in P_aCO_2, there will be approximately a 1.25 mm Hg decrease in P_AO_2 (and P_aO_2).

Hypoventilation

Adequate ventilation requires central respiratory centers, spinal pathways, peripheral respiratory nerves, primary respiratory muscles, pleural-pulmonary coupling, and pulmonary mechanics to be intact or normal. Hypoventilation occurs when any component of this pathway is disrupted or abnormal. Important causes of hypoventilation include depression or injury of the central respiratory center, injury or disease of the neuromuscular apparatus of ventilation, disruption of pleural-pulmonary coupling (e.g., pneumothorax), and/or abnormal pulmonary mechanics that increase the work of respiration to levels that cannot be sustained by the patient. The major determinants of respiratory work are airway resistance and lung compliance. Obstructive airway disorders or restrictive lung conditions, or both, increase respiratory work leading to hypoventilation when they are severe.

Breathing Patterns

Clinical assessment of ventilation should include observation of breathing. The first indication that a patient is hypoventilating may come from the simple observation that ventilatory excursions are poor. Information about abnormal pulmonary mechanics is gained from observation of the pattern of breathing. Animals adopt a respiratory rate and pattern that minimizes respiratory work. Normal breathing balances the major elastic force of *lung compliance* with the major viscous force of *airway resistance*. Elastic forces in the lung are minimized by a *rapid and shallow* breathing pattern, whereas resistance forces in the lung are minimized by a *slow and deep* breathing pattern (Figure 1.2). Thus, animals with restrictive lung diseases (e.g., pulmonary edema, interstitial pneumonia, pulmonary fibrosis, pleural effusion) will adopt a rapid and shallow breathing pattern, whereas animals with airway obstruction (e.g., laryngeal paralysis, bronchoconstriction) will tend to adopt a slow and deep pattern of breathing. Obstructive breathing patterns can be further assessed by observing of the phase of respiration that produces the most ventilatory effort. Upper airway obstruction causes an exaggerated effort during inspiration, whereas lower airway obstruction causes an exaggerated effort during expiration.

Tidal Volume and Minute Volume

Total ventilation can be measured directly with a respirometer attached to an endotracheal tube or tight-fitting mask. *Tidal volume* is the volume (mL) of gas expired during each breath and is normally at least 10 mL/kg of body weight. *Minute volume* (V_T) is the total volume of gas expired each minute (L/min). If tidal volume or minute volume are low, there is a good possibility that ventilation is inadequate. However, because V_T includes both V_D and V_A, measurement of a normal tidal volume or minute volume does not assure that V_A is adequate.

Arterial Carbon Dioxide Tension

Ultimately, clinical assessment of alveolar ventilation is based on the P_aCO_2. By definition, a patient is hypoventilating when *hypercapnia* (increased P_aCO_2) present. The most direct method of assessing P_aCO_2 is by arterial blood gas analysis. Alveolar ventilation should be considered inadequate when the P_aCO_2 is > 45 mm Hg for patients at or near sea level. Hypoventilation causes both hypoxemia and respiratory acidosis. Administration of supplemental oxygen

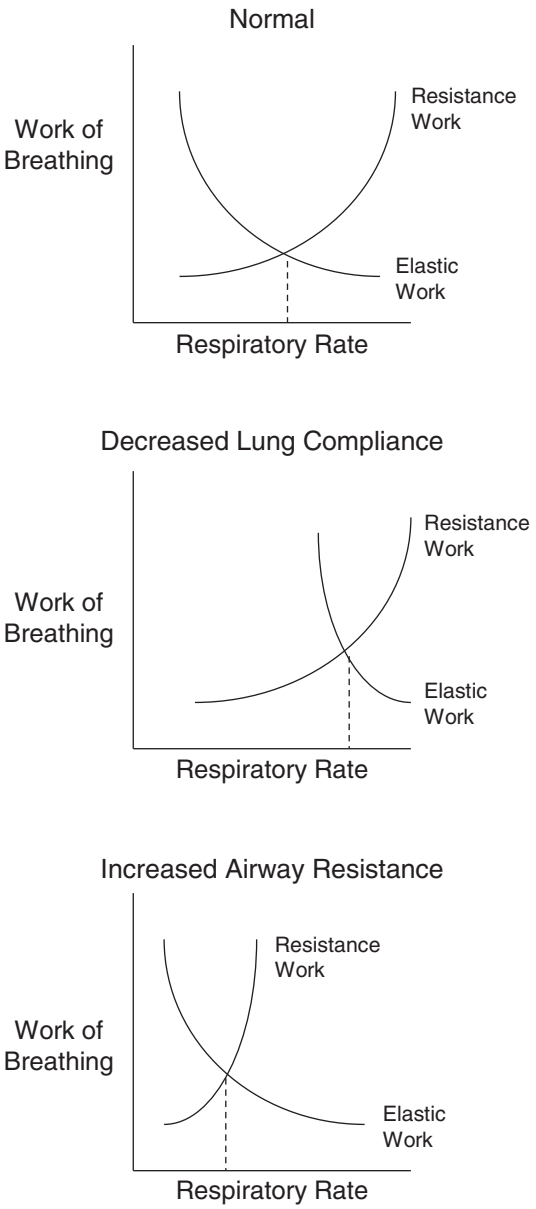


Figure 1.2 Work of Breathing

(i.e., increasing the F_IO_2) corrects hypoxemia caused by hypoventilation by increasing the P_IO_2 and P_AO_2 (see the alveolar gas equation). Of course, administration of supplement oxygen does not correct the respiratory acidosis associated with elevated P_aCO_2, so it is important to correct the underlying cause(s) of hypoventilation even when animals are receiving supplemental oxygen.

End-Tidal Carbon Dioxide Tension

Because diffusion of carbon dioxide in the lung is highly efficient, P_aCO_2 and *alveolar carbon*

dioxide tension (P_ACO_2) are close to equal. The carbon dioxide tension of expired gas at the end of expiration closely approximates P_ACO_2 and is termed *end tidal carbon dioxide tension* ($P_{ET}CO_2$). The $P_{ET}CO_2$ is measured clinically with a capnograph that samples expired gas continuously and reports the peak carbon dioxide tension at the end of expiration. Measurement of $P_{ET}CO_2$ provides a clinical estimate of P_aCO_2, and therefore V_A.

Pulmonary Gas Exchange

Pulmonary gas exchange is the collective process by which oxygen and carbon dioxide are exchanged between the alveolus and blood. Exchange of oxygen is complex and dependent on diffusion across the alveolar-capillary membrane, matching of alveolar ventilation and perfusion, and the amount of venous admixture to arterial blood. Ideally, P_aO_2 should be nearly equal to P_AO_2 predicted by the alveolar gas equation (i.e., 100 mm of Hg under physiologic conditions at sea level). *Impaired pulmonary gas exchange* is present when P_aO_2 becomes substantially less than the predicted P_AO_2. Because P_aO_2 can be measured directly and P_AO_2 can be calculated from measurable values, the degree of gas exchange impairment can be quantified by the *alveolar-arterial oxygen difference* (A-a PO_2):

$$\begin{aligned} \text{A-a } PO_2 &= P_AO_2 - P_aO_2 \\ &= [F_IO_2(P_B - P_{H_2O}) - P_aCO_2/R] \\ &\quad - P_aO_2 \\ &= [0.21(760 - 47) - 40/0.8] - 98 \\ &= 100 - 98 \end{aligned} \tag{1.6}$$

The A-a PO_2 should be < 10 mm Hg for animals breathing room air. The normal A-a PO_2 gradient increases 5 to 7 mm Hg for every 10% increase in F_IO_2. There are three basic mechanisms of gas exchange impairment: *diffusion impairment*, *shunt*, and *ventilation-perfusion (V_A/Q) mismatch*.

Diffusion Impairment

Diffusion of oxygen across the alveolar-capillary membrane is directly proportional to the concentration gradient of oxygen across the membrane and the total membrane area; and inversely proportional to the membrane thickness. Adequate diffusion of oxygen is also a function of the time available to accomplish complete equilibration between the alveoli and blood. Under normal conditions, diffusion of oxygen in the lung is highly efficient and generally is complete by the time blood has traversed about one-fourth of the alveolar capillary bed. Thus, pulmonary disease must be severe before diffusion limits gas exchange. *Diffusion impairment* can result from diseases that affect the alveolar-capillary membrane such as pulmonary edema, interstitial pneumonia, or pulmonary fibrosis. However, because of the efficiency of gas diffusion, these conditions rarely cause hypoxemia by diffusion impairment in animals at rest.

The most important clinical cause of severe diffusion impairment is pulmonary thromboembolism (PTE), which impairs diffusion by decreasing the total membrane area available for oxygen diffusion. Because the cardiac output must be redirected to unobstructed pulmonary capillaries, the transit time available for diffusion is decreased, and this further contributes to diffusion impairment. A reciprocal consequence of pulmonary thromboembolism is an increase in dead space ventilation (V_D/V_T) resulting from the ventilation of unperfused alveoli. Dead space ventilation can be quantified by measuring the P_aCO_2 and mixed exhaled carbon dioxide tension (P_ECO_2):

$$\frac{V_D}{V_T} = \frac{P_aCO_2 - P_ECO_2}{P_aCO_2} \tag{1.7}$$

Determination of P_ECO_2 requires collection of expired gases into a collection bag and analysis of carbon dioxide tension with an infrared analyzer. This determination is rarely performed in clinical patients. In theory, the increase in dead space ventilation could lead to an increase in P_aCO_2. However, carbon dioxide diffuses across the alveolar-arterial membrane about 20 times more rapidly than oxygen and is rarely if ever limited by diffusion.

Administration of supplemental oxygen can be expected to correct hypoxemia caused by diffusion impairment. It does so by increasing P_AO_2, and therefore the concentration gradient of oxygen across the alveolar-capillary membrane that drives diffusion. This explains why hypoxemia due to diffusion impairment, including pulmonary thromboembolism, is so responsive to administration of supplemental oxygen. This response to supplemental oxygen serves as a useful clinical observation that supports an assessment that hypoxemia is the result of this mechanism (e.g., PTE).

Shunt

Shunt occurs when unoxygenated venous blood bypasses viable gas exchange areas of the lung and mixes with oxygenated arterial blood. The resultant *venous admixture* produces hypoxemia. Shunt results from either a *right-to-left cardiac shunt* or *pulmonary shunt*. Examples of right-to-left cardiac shunt

include ventricular septal defect with suprasystemic pulmonary hypertension and Tetralogy of Fallot. Pulmonary shunt results from perfusion of completely collapsed or fluid-filled alveoli. Shunt is an important cause of clinically significant hypoxemia. The magnitude of hypoxemia caused by shunt is a function of the ratio of shunt flow to total cardiac output, termed the *shunt fraction* (Q_s/Q). Because venous admixture has no opportunity for gas exchange, hypoxemia arising purely from shunt is unresponsive to administration of supplemental oxygen. This physiologic reality distinguishes shunt from other causes of hypoxemia and serves as a useful clinical finding for diagnosing shunt as a contributing or sole mechanism of hypoxemia.

Shunt does not affect the P_aCO_2 until it becomes very severe. Thus, shunt usually does not result in hypercapnia. In fact, animals with shunt often have a low P_aCO_2 as a result of hypoxia-driven hyperventilation.

Ventilation-Perfusion Mismatch

Ventilation-perfusion (V_A/Q) mismatch occurs when ventilation and blood flow are not closely matched in gas exchange units. The result is inefficient gas exchange and hypoxemia. If regions of the lung are ventilated but poorly perfused (i.e., high V_A/Q), the functional result is wasted ventilatory effort that does not benefit gas exchange. Because regions of high V_A/Q are associated with complete gas exchange, regions of high V_A/Q do not directly contribute to impaired pulmonary gas exchange and hypoxemia. It does make gas exchange inefficient. In regions of the lung that are perfused but poorly ventilated (i.e., low V_A/Q), the functional result is inadequate bulk flow of oxygen to alveoli to fully oxygenate blood as it flows through these regions. This results in admixture of poorly oxygenated blood from these exchange areas with blood that is more fully oxygenated from normal regions. The net result is overall hypoxemia. *Low V_A/Q mismatch* is an important cause of hypoxemia in animals with pulmonary disease. Any pulmonary condition that disrupts ventilation but maintains blood flow to alveoli will result in low V_A/Q mismatch and global arterial hypoxemia. Because alveoli are still at least partially ventilated in the setting of low V_A/Q mismatch, the resultant hypoxemia is responsive to the administration of supplemental oxygen, depending on the magnitude of low V_A/Q. In reality, pulmonary diseases that cause low V_A/Q mismatch are usually accompanied by an increase in pulmonary shunt, explaining why many pulmonary conditions are only partially or poorly responsive to supplemental oxygen.

Alveolar-Arterial PO_2 Gradient

The A-a PO_2 can be calculated by measuring P_aO_2 and P_aCO_2 by blood gas analysis and inserting these values into Equation 1.6. The calculated A-a PO_2 for animals breathing room air is normally < 10 mm of Hg. A calculated A-a PO_2 of 30 mm of Hg or greater in animals breathing room air suggests significant impairment of gas exchange. Because the normal valve of A-a PO_2 is affected by F_IO_2, the above normal valves do not apply to animals breathing supplemental oxygen. While normal values of A-a PO_2 are reported for various levels of F_IO_2, it is often difficult to determine an accurate F_IO_2 in the clinical setting. Thus, blood gas analysis and calculation of A-a PO_2 is most revealing when performed in animals breathing room air. The A-a PO_2 has been shown to be an important predictor of survival in critically ill dogs [2].

Shunt Fraction (Q_s/Q)

The magnitude of shunt can be determined by calculation of the shunt fraction from the oxygen saturation of arterial blood (S_aO_2), mixed venous blood (S_vO_2), and pulmonary capillary blood (S_cO_2) during breathing of pure oxygen:

$$\frac{Q_s}{Q} = \frac{S_cO_2 - S_aO_2}{S_cO_2 - S_vO_2} \quad (1.8)$$

The S_cO_2 is not measured directly, but is assumed to be 100% during breathing of pure oxygen. Ideally, the S_vO_2 sample should be obtained from a catheter in the pulmonary artery. Alternatively, the S_vO_2 can be approximated from a sample obtained from a central venous catheter. Shunt is the only mechanism of impaired gas exchange that persists during administration of 100% supplemental oxygen. A calculated $Q_s/Q > 10\%$ is abnormal and indicates clinically important gas exchange impairment in animals breathing supplemental oxygen.

Oxygen Saturation and Oxygen Content

The P_aO_2 reflects the amount of oxygen dissolved in plasma. Dissolved oxygen is of course insufficient to meet metabolic demand for oxygen. Hemoglobin greatly increases the oxygen carrying capacity of blood. *Arterial oxygen saturation* (S_aO_2) is defined as the fraction or percent of total hemoglobin binding sites that are bond to oxygen in the arterial blood. The P_aO_2 is the principal determinant of S_aO_2.

The relationship between P_aO_2 and S_aO_2 is described by the *oxygen-hemoglobin saturation curve*

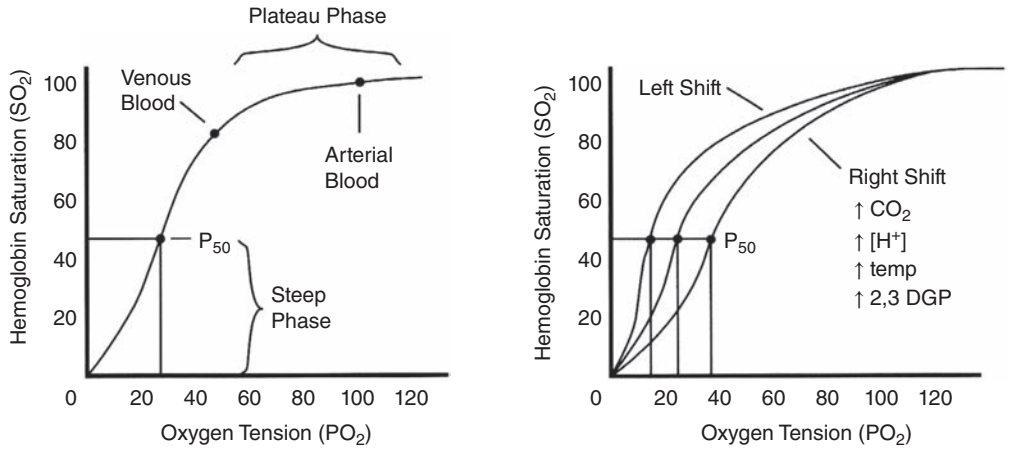


Figure 1.3 Oxygen-Hemoglobin Saturation Curve

(Figure 1.3). The affinity of hemoglobin for oxygen increases as more oxygen binds to it which gives the oxygen-hemoglobin curve its sigmoid shape. The shape of the oxygen-hemoglobin curve has important physiologic and pathophysiologic implications. The plateau phase of the curve causes hemoglobin to remain saturated over a wide of range oxygen tensions. The S_aO_2 is approximately 97% when the P_aO_2 is 97 mm of Hg. The S_aO_2 cannot be increased substantially by higher than normal P_aO_2 values. The steep phase of the oxygen-hemoglobin curve allows for efficient oxygen release in the peripheral tissues where oxygen tension normally decreases. A pathophysiologic implication of the steep phase of the curve is that small changes in P_aO_2 can have profound changes on S_aO_2 when arterial hypoxemia is present.

The oxygen-hemoglobin curve can "shift" to the right or left reflecting changes in the overall affinity of hemoglobin for oxygen. A shift of the curve to the right decreases overall oxygen affinity of hemoglobin, whereas a shift to the left increases hemoglobin oxygen affinity. Conditions that shift the curve to the right include increased CO_2 (Haldane effect), increased hydrogen ion concentration (Bohr effect), increased temperature, and increased 2,3-diphosphoglyerate. Interestingly, conditions that decrease hemoglobin affinity prevail in the peripheral tissues where unloading of oxygen is desirable. Because shifted curves converge in the plateau phase, a shift in the oxygen-hemoglobin curve has a more profound effect on the steep phase than on the plateau phase of the curve. For this reason, shifts in the curve have a greater physiologic effect on unloading of oxygen in the peripheral tissues than on loading of oxygen in the lung. Shifts in the oxygen-hemoglobin curve are quantified by measurement of the oxygen tension at which hemoglobin is 50% saturated (P_{50}). Even though shifts in the oxygen-hemoglobin curve can have an important effect on pulmonary function, they are generally not assessed clinically. Nevertheless, it is useful for clinicians to be mindful of the possibility for such effects in their patients.

Arterial oxygen content (C_aO_2) is the total oxygen present in arterial blood measured in units of mL O_2/100 mL (dL). Each gram of hemoglobin (Hgb) is capable of carrying 1.34 mL of molecular oxygen when fully saturated. Thus, the amount of oxygen bound to hemoglobin can be calculated by multiplying 1.34 (mL O_2/gm), the hemoglobin concentration of blood (gm/dL), and S_aO_2 (%). Dissolved oxygen can be calculated from the P_aO_2. At sea level, dissolved oxygen is equal to 0.003 mL O_2/dL blood/mm Hg P_aO_2. Thus, C_aO_2 is calculated as shown in Equation 1.9:

$$\begin{aligned} C_aO_2\,(\text{mL } O_2/\text{dL}) = {} & S_aO_2(\%) \times \text{Hgb (gm/dL)} \\ & \times 1.34\,(\text{mL } O_2/\text{gm}) \\ & + P_aO_2(\text{mm Hg}) \\ & \times 0.003\,(\text{mL } O_2/\text{dL/mm Hg}) \end{aligned} \tag{1.9}$$

For an animal at sea level with a P_aO_2 of 97 mm of Hg, a S_aO_2 of 97%, and a hemoglobin concentration of 15 gm/dL, the C_aO_2 would be:

$$\begin{aligned} C_aO_2(\text{mL } O_2/\text{dL}) = {} & 0.97 \times 15\,\text{gm/dL} \\ & \times 1.34\,\text{mL } O_2/\text{gm} \\ & + 97\,\text{mm Hg} \times 0.003\,\text{mL } O_2/\text{dL/mm Hg} \\ = {} & 19.5\,\text{mL } O_2/\text{dL} + 0.3\,\text{mL } O_2/\text{dL} \\ = {} & 19.8\,\text{mL } O_2/\text{dL} \end{aligned} \tag{1.10}$$

From this calculation, it is apparent that the contribution of dissolved oxygen to overall C_aO_2 is negligible and for clinical purposes can largely be ignored. Thus, the principal clinical determinants of C_aO_2 are S_aO_2 and hemoglobin concentration. Polycythemia

and anemia can have an important impact on C_aO_2. Within limits, polycythemia is an important adaptive mechanism for physiologic (e.g., altitude) or pathophysiologic causes of chronic hypoxemia. Conversely, anemia substantially decreases C_aO_2. The effect that anemia has on C_aO_2 is sometimes under appreciated. In animals with a relatively normal cardiovascular system, deficits in C_aO_2 caused by anemia can be compensated for by an increase in cardiac output. However, if the cardiovascular system is compromised, as is often the case in critical patients, anemia can have an important adverse effect on O_2 delivery. Thus, as a general rule, it is important to keep the hematocrit ≥ 30% in patients undergoing thoracic interventions.

Oxygen Delivery, Oxygen Consumption, and Oxygen Extraction

Oxygen delivery (D_{O_2}) is the mL O_2 delivered to the peripheral tissues each minute and is the product of C_aO_2 and cardiac output (Q):

$$D_{O_2}(\text{mL } O_2/\text{min}) = C_aO_2(\text{mL } O_2/\text{dL}) \times Q(\text{dL/min}) \quad (1.11)$$

Thus, maintenance of adequate D_{O2} requires adequate pulmonary function (P_aO_2), hemoglobin concentration, and cardiovascular function (Q). When hypoxemia (low P_aO_2) or low hemoglobin concentration cause low C_aO_2, oxygen delivery can be maintained by increasing cardiac output assuming that the cardiovascular system is capable. When D_{O_2} is limited by low cardiac output, compensation is more difficult. Within limits, D_{O_2} can be increased by increasing the hemoglobin concentration (e.g., *polycythemia*) or by increasing oxygen extraction.

Oxygen consumption (V_{O_2}) is the mL O_2 consumed by tissues each minute and can be calculated by multiplying the difference between C_aO_2 and mixed venous oxygen content (C_vO_2) with cardiac output:

$$V_{O_2}(\text{mL } O_2/\text{min}) = [C_aO_2 - C_vO_2(\text{mL } O_2/\text{dL})] \times Q\ (\text{dL/min}) \quad (1.12)$$

From the relationship in Equation 1.12, it can be seen that, in the setting of a low cardiac output, V_{O_2} can be maintained by increasing the $C_aO_2 - C_vO_2$ difference (i.e., increasing oxygen extraction).

The *oxygen extraction ratio* is the proportion of oxygen consumed (V_{O_2}) to oxygen delivered (D_{O_2}):

$$O_2 \text{ extraction} = \frac{V_{O_2}}{D_{O_2}} = \frac{(C_aO_2 - C_vO_2) \times Q}{C_aO_2 \times Q} \quad (1.13)$$

Because cardiac output is in both the numerator and denominator, it cancels out. Thus, determination of O_2 extraction ratio does not require actual measurement of cardiac output. Also, hemoglobin concentration can be assumed to be the same in arterial and mixed venous blood. As a result, calculation of O_2 extraction can be simplified to:

$$O_2 \text{ extraction} = \frac{S_aO_2 - S_VO_2}{S_aO_2} \quad (1.14)$$

Because the O_2 extraction accounts for any deficits in SaO_2 in the delivery of O_2 to tissues, it becomes primarily an index of the adequacy of cardiac output. The utility of the O_2 extraction is that it is independent of the patient size and does not require actual measurement of cardiac output. As such, the oxygen extraction ratio is a clinically useful method of assessing the adequacy of cardiac output. Under physiologic conditions at rest, oxygen extraction is about 0.25. When cardiac output becomes inadequate to meet the demands of the patient, O_2 extraction increases. An O_2 extraction ratio of > 0.4 suggests that cardiac output is inadequate.

Cardiac Output

The principal function of the cardiovascular system is the delivery of blood to the pulmonary and systemic circulations. This function is accomplished by pumping an adequate volume of blood to the pulmonary and systemic circulations (i.e., pulmonary and systemic cardiac output) and maintaining adequate pulmonary and systemic perfusion pressures.

Cardiac Output, Blood Pressure, and Vascular Resistance

The relationship of cardiac output (Q), mean arterial pressure (MAP), atrial pressure (AP), and vascular resistance (R) are described by:

$$Q = MAP - AP/R \quad (1.15)$$

This relationship shows that cardiac output is a direct function of the pressure difference that drives flow. In the pulmonary circulation this is the difference between mean pulmonary arterial pressure and left atrial pressure. In the systemic circulation this is the difference between mean systemic pressure and right atrial pressure. Cardiac output is inhibited by pulmonary and systemic vascular resistances in the pulmonary and systemic circulations, respectively.

Rearranging this relationship demonstrates the determinants of mean arterial pressures for the pulmonary and systemic circulations

$$MAP = (Q \times R) + AP \quad (1.16)$$

Vascular resistances are not measured directly, but can be calculated from Q, MAP, and AP:

$$R = (MAP - AP)/Q \quad (1.17)$$

As predicted by the Law of Poiseuille, vascular resistance is determined by the collective total cross-sectional vascular radius of resistance arteries (i.e., degree of vasoconstriction and vasodilation) and the viscosity of blood (i.e., hematocrit). The pulse pressure (P_p) is the difference between the systolic and diastolic blood pressures around the mean arterial pressure. The P_p is the principle determinant of the "strength" of a patient's peripheral pulse on palpation. The P_p is a direct function of the SV and inverse function of the collective compliance of the large elastic arteries (C_A):

$$P_p = SV/C_A \quad (1.18)$$

The most common cause for a diminished P_p or a "weak" pulse is a poor SV. Poor compliance or "stiffening" of the elastic conducting arteries can have the effect of elevating the P_p. Conditions such as patent ductus arteriosus or aortic insufficiency that allow rapid diastolic "run off" of blood dramatically lower diastolic blood pressure and thereby elevate the P_p. Together, the mean arterial pressure and pulse pressure determine the systolic arterial pressure, which is the major determinant of cardiac afterload.

Pressure-Volume Relationship

The cardiac cycle encompasses the electrical, pressure, volume, flow, and valve motion events that occur during one complete cardiac systole and diastole. During each cardiac cycle, the heart accomplishes two fundamental kinds of external work. It generates pressure (i.e., potential energy) and it ejects volume (i.e., kinetic energy). The relationship of these two events is illustrated by plotting instantaneous ventricular pressure and volume against each other to generate a ventricular pressure-volume plot (Figure 1.4). Pressure-volume plots form the basis of current understanding of cardiac physiology. Each loop of a pressure-volume plot represents one complete cardiac cycle and consists of the rapid diastolic and atrial filling phases, isovolumetric contraction phase, ejection phase, and isovolumetric relaxation phase. The important pressure endpoints are *ventricular end-diastolic pressure* (EDP) and *ventricular systolic pressure* (P_s). The principal volume endpoints are *end-diastolic volume* (EDV) and *end-systolic volume* (ESV). The difference between EDV and ESV is the *stroke volume* (SV). The area inside in pressure-volume loop represents the external work done by the heart in one cardiac cycle. The *ejection fraction* is the SV divided by EDV.

Stroke Volume (Preload, Afterload, Contractility)

Cardiac output is the product of stroke volume and heart rate. Stroke volume is critically important to the maintenance of adequate cardiac output. Stroke volume, in turn, is determined by three important

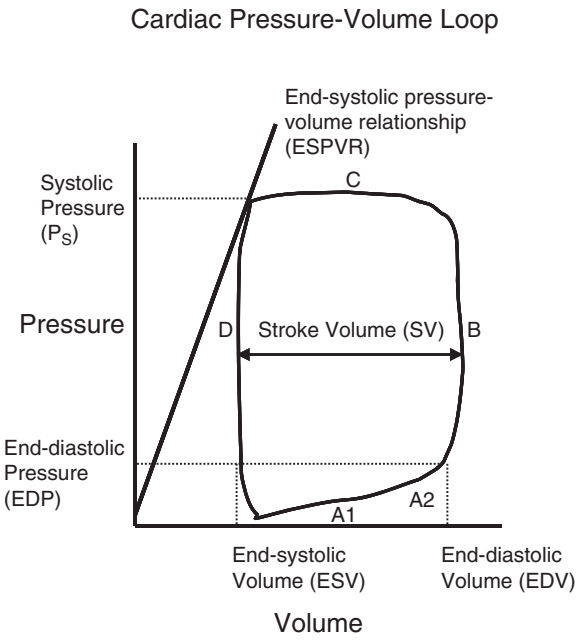
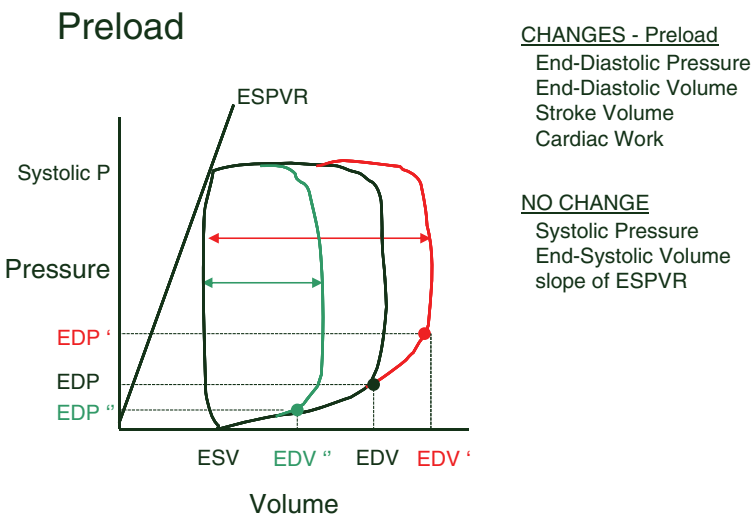


Figure 1.4 Cardiac Pressure-Volume Relationship

Figure 1.5 Cardiac Preload



independent variables: preload, afterload, and contractility. Preload encompasses the Frank-Starling principle of the heart. On a cellular basis, preload is determined by the amount of diastolic strain on each cardiomyocyte. Within limits, the greater the diastolic strain, the more forceful the cardiac contraction. On a whole heart basis, preload is reflected by the EDV and EDP (Figure 1.5). On a beat-to-beat basis, the greater the EDV and EDP, the greater the preload. Since ESV does not change with preload, the net result of an increase in preload is an increase in stroke volume, and vice versa. Factors that determine preload are the *mean filling pressure* of the circulation and the vascular resistance. The mean filling pressure of the circulation is the pressure in the cardiovascular system at zero flow and the theoretical pressure that drives flow of venous blood back to the heart. Mean filling pressure is largely determined by blood volume and venous vascular tone. The mean filling pressure has a direct relationship with preload. Vascular resistance has an inverse relationship with preload. Increases in vascular resistance decrease venous return to the heart and therefore decrease preload and stroke volume. Thus, the determinants of preload reside within the circulation, not in the heart. Afterload is the systolic stress that the ventricular wall must overcome before it can eject volume. The determinants of ventricular systolic wall stress, and therefore cardiac afterload, are predicted by the LaPlace relationship:

$$\text{Wall stress}_s = \frac{\text{Systolic pressure } (P_s) \times \text{Ventricular radius (r)}}{\text{Ventricular wall thickness (h)}} \quad (1.19)$$

On a beat-to-beat basis, afterload is a function of P_s. On a chronic basis, cardiac remodeling (e.g., ventricular dilation and/or wall thickening) also affects afterload. Thus, beat-to-beat afterload is determined by events outside of the heart (i.e., mean arterial pressure and pulse pressure). Afterload has an inverse relationship with stroke volume (Figure 1.6). As afterload increases, stroke volume deceases, and vice versa. In theory, changes in afterload have a minimal effect on external cardiac work (area within the pressure-volume loop). A way to think about afterload is that it reflects the distribution of external cardiac work between generation of pressure and ejection of volume. As the heart is required to generate a higher systolic pressure (i.e., higher afterload), less work is leftover for the ejection of stroke volume.

Contractility, also known as inotropic state, represents the intrinsic contractile state of the heart independent of preload and afterload. On a beat-to-beat basis, contractility is largely a function of the amount of sympathetic (β) influence on the heart. Contractility is also affected by the diseases of the myocardium, cardiac drugs, and cardiac mass. Changes in cardiac mass through cardiac hypertrophy have a direct effect on the global contractility of the heart. Contractility has a direct relationship with stroke volume (Figure 1.7). The greater the contractility or inotropic state, the greater the stroke volume. On a pressure-volume loop, contractility is reflected by changes in the slope of the end-systolic pressure-volume relationship (ESPVR). The net result is a change in the ESV for given loading conditions.

Through the effects of preload, afterload, and contractility, maintenance of cardiac output is a complex interaction of acute and chronic physiologic and pathophysiologic changes in the heart and circulation (Figure 1.8). Understanding these interactive relationships is important for troubleshooting and managing cardiovascular function in the clinical setting.

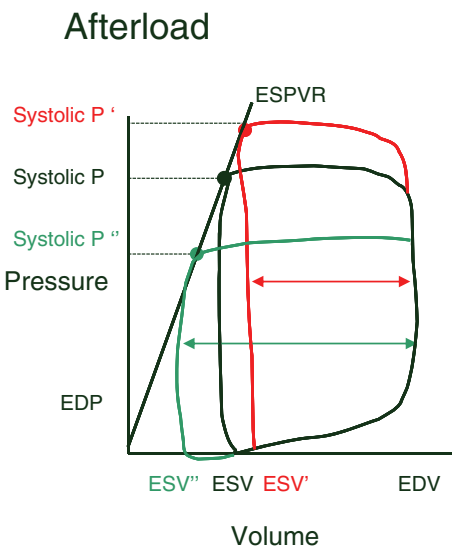


Figure 1.6 Cardiac Afterload

Heart Failure

Heart failure is present when cardiac output is inadequate despite adequate ventricular end-diastolic pressures or when adequate cardiac output can only be maintained at the expense of elevated end-diastolic pressures. Heart failure results from the combined effects of acute or chronic cardiac insufficiency and compensatory neuroendocrine mechanisms. Heart failure manifests as either organ dysfunction secondary to low cardiac output (termed *low output heart failure* or *forward heart failure*) or congestion of organs behind the heart (termed *congestive heart failure* or *backward heart failure*), or both (Figure 1.9). Congestion is manifested behind the left heart by pulmonary edema or pleural effusion; or behind the right heart as ascites, peripheral edema, or pleural effusion.

The stretch or load on myocardial fibers just prior to contraction profoundly influences the degree of myocardial fiber shortening. This load or stretch prior to contraction is the cellular basis of preload. *End-diastolic pressure* in the heart reflects the amount of stretch or preload on the ventricle prior to contraction and in turn are an important determinant of cardiac output. The *Frank-Starling curve* describes the direct relationship between cardiac output and end-diastolic pressures in the heart.

Cardiac output and ventricular end-diastolic pressure are not only functionally related, but are the physiologic parameters directly responsible for the two adverse manifestations of heart failure; namely,

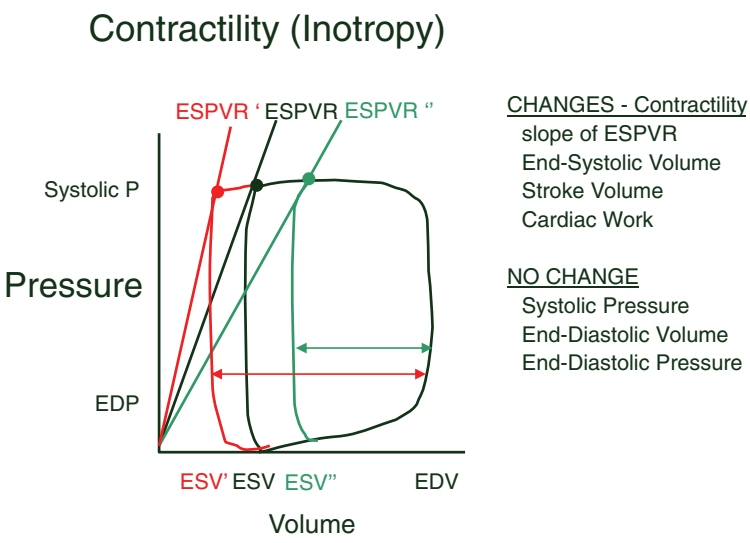


Figure 1.7 Cardiac Contractility

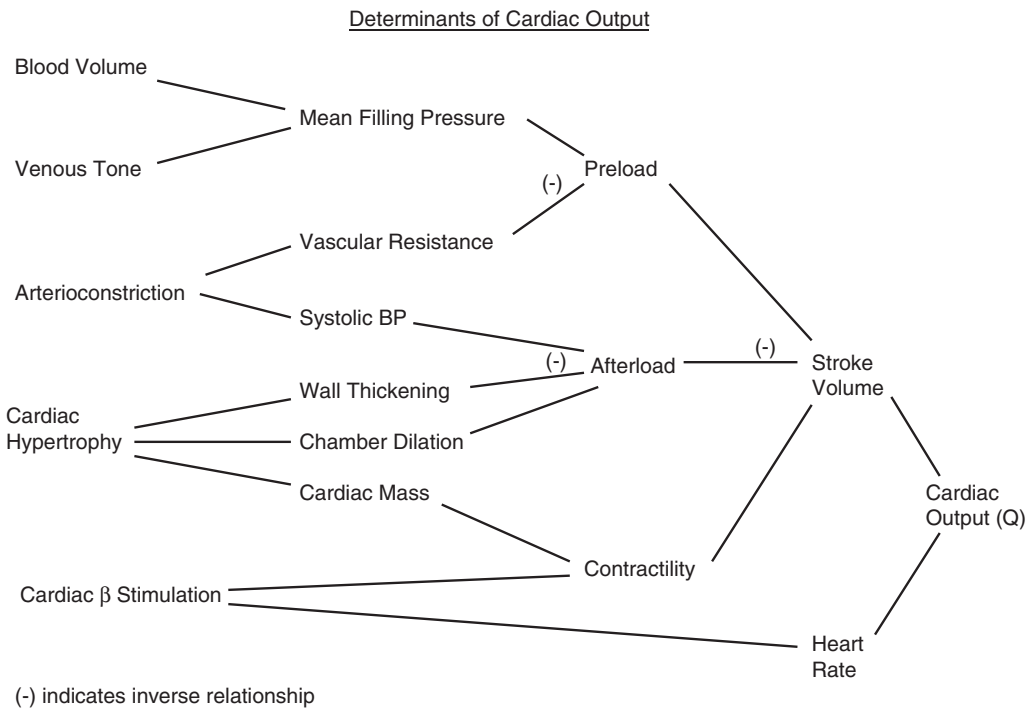
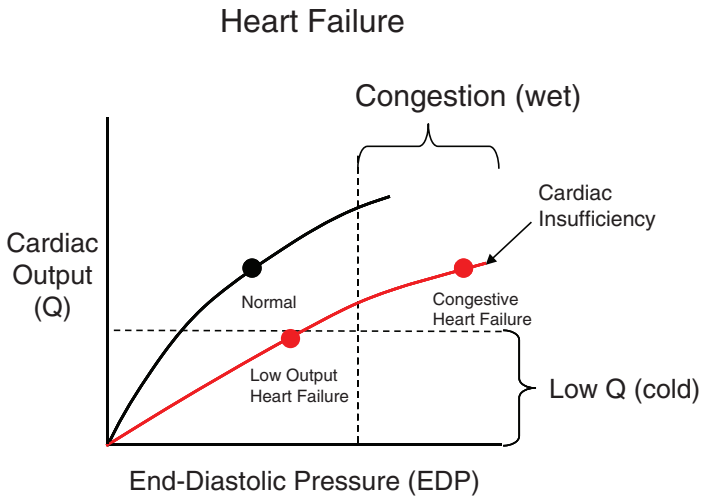


Figure 1.8 Determinates of Cardiac Output

inadequate perfusion and congestion. Initially, impaired cardiac function narrows the cardiac reserve (i.e., the ability to increase cardiac output during activity or exercise). The clinical manifestation is exercise or activity intolerance. Eventually, cardiac output can become low enough that it fails to meet metabolic needs of organ systems and tissues even at rest. If this happens acutely, the patient may manifest low output heart failure. Organ and tissue dysfunction become apparent. The patient is "cold" rather than "warm." While ventricular end-diastolic pressure exerts a positive influence on cardiac output, it also is the effective downstream pressure that resists venous return to the heart. Congestion occurs when end-diastolic pressure elevates capillary hydrostatic pressure to the point where a net efflux of water from capillaries to the interstitial space occurs. The result is edema of the organs and tissues behind the failing heart. The patient is "wet" rather than "dry."

Cardiac insufficiency is caused by one or a combination of four basic mechanisms: *primary myocardial failure*, *hemodynamic overload*, *diastolic dysfunction*,

Figure 1.9 Heart Failure



or *cardiac arrhythmias*. Progression of heart disease can be arbitrarily divided into three phases. The first phase of heart disease occurs when an initiating cardiac injury or insufficiency is present. If the initiating cardiac insufficiency is acute and overwhelming, then *low output heart failure* may ensue. More often in animals the cardiac insufficiency is not initially overwhelming or lethal, but rather slowly progressive. In this case, the presence of heart disease may be signaled only by the presence of physical findings such as abnormal heart sounds or murmurs, and not be associated with overt symptoms of heart failure other than possible activity or exercise intolerance.

The second phase of heart disease is hallmarked by activation of the *neuroendocrine response* to cardiac insufficiency. This neuroendocrine response ensures that blood pressure and cardiac output are maintained principally through the retention of vascular blood volume and the constriction of arteries and veins. *Cardiac hypertrophy* generally begins during this phase, particularly when the initiating cardiac insufficiency results from hemodynamic overload. The type of cardiac hypertrophy depends on the nature of the cardiac insufficiency. During this phase, clinical evidence of cardiac insufficiency in the form of cardiomegaly occurs, although overt signs of heart failure still may not present. Symptoms would still be mostly associated with reduced activity or exercise capacity.

Although the neuroendocrine response is initially adaptive, ultimately this response becomes maladaptive. This is the third phase of heart failure. During this phase, the neuroendocrine response "overcompensates," producing high end-diastolic pressures primarily through the retention of blood volume. The result is congestion in the form of tissue and organ edema. Inappropriate arterioconstriction is also present during this phase, contributing to poor tissue perfusion. This state is termed *congestive heart failure*. It is possible in advanced cases of cardiac insufficiency for both congestive heart failure and low output heart failure to be present.

Summary

Adequate cardiopulmonary function is dependent on sequential physiologic processes to achieve the ultimate goal of delivering oxygen to tissues. Derangement of any of these physiologic processes can result in inadequate oxygen delivery. Success in thoracic surgery often depends on quickly pinpointing and correcting disruptions in oxygen delivery. Fortunately, techniques for determining the cause(s) and magnitude of pathophysiologic disruptions in the cardiopulmonary system are readily available to the clinician with an understanding of these processes.

References

1 Walton RS, Wingfield WE, Ogilvie GK, Fettman MJ, Matteson VL. 1996. Energy expenditure in 104 postoperative and traumatically injured dogs with indirect calorimetry. *Journal of Veterinary Emergency and Critical Care* 6:71–79.

2 Van Pelt DR, Wingfield WE, Wheeler SL, Salman MD. 1991. Oxygen-tension based indices as predictors of survival in critically ill dogs: clinical observations and review. *Journal of Veterinary Emergency and Critical Care* 1:19–25.

2

Cardiopulmonary Monitoring and Supportive Care

E. Christopher Orton

Monitoring and supportive care before, during, and after surgery are keys to the success of thoracic surgery. Successful cardiopulmonary monitoring and supportive care is grounded in a solid working knowledge of cardiopulmonary physiology and pathophysiology reviewed in Chapter 1. While the emphasis of this chapter is on the cardiopulmonary systems, monitoring and support of other organ systems including blood, renal, hepatic, and neurological systems are also crucially important. Levels of monitoring and support range from basic to advanced, depending on the underlying condition and the type of surgery undertaken. Certainly advanced monitoring and support are not required for all animals undergoing thoracic surgery. Supportive care is tailored to what is necessary based on anticipation of problems, vigilant observation, and effective response to problems.

Ventilation

Ventilation is the movement of air into and out of the lungs. It is dependent on adequate function of the neuromuscular apparatus that supports breathing, coupling of the thoracic wall and diaphragm to the lungs, and tolerable limits on the work of breathing. Thus, ventilation can be impaired by neuromuscular injury or depression, conditions within the pleural space, or increases in the work of breathing due to pulmonary disease. Clinical evaluation begins with the simple observation of breathing. Ventilatory effort may be observed to be poor, particularly in the immediate post-operative period when the influence of anesthetic drugs is still present. Information is also gained from observation of the pattern of breathing. Animals that are not thermoregulating adopt a pattern of breathing that minimizes respiratory work. Normal breathing balances the work necessary to overcome lung compliance (elastic forces) and airway resistance (viscous forces) by varying the depth and rate of breathing. Animals with restrictive lung conditions (e.g., pulmonary edema, pulmonary fibrosis, pleural effusion, pneumothorax) adopt a rapid and shallow breathing pattern, whereas animals with obstructive lung conditions tend to adopt a slow and forced breathing pattern. Further, animals with upper airway obstruction (e.g., laryngeal paralysis) exhibit effort on inspiration, whereas animals with lower airway obstruction (e.g., bronchoconstriction, intrathoracic airway collapse) exhibit effort on expiration.

Tidal volume (mL) and minute volume (mL/min) can be measured directly in animals with a Wright's respirometer. Tidal volume (mL) is the volume of gas expired with each breath and is normally 10 mL per kg of body weight. Ventilation is likely inadequate in animals with low tidal and minute volumes. However, because the distribution of total ventilation between dead space ventilation and alveolar ventilation varies, measurement of normal total ventilation does not ensure that alveolar ventilation is adequate. For this reason, assessment of the adequacy of alveolar ventilation is based on measurement of P_aCO_2.

By definition, hypoventilation is present when P_aCO_2 is increased above normal and hyperventilation is present when P_aCO_2 levels are lower than normal. P_aCO_2 is measured directly by arterial blood gas analysis. Significant hypoventilation is present whenever the P_aCO_2 is >45 mm Hg. Hypoventilation contributes to hypoxemia and causes respiratory acidosis. As a rule of thumb, for every 1 mm Hg elevation of P_aCO_2, P_aO_2 will be decreased by 1.2 mm Hg. Administration of supplemental oxygen easily corrects hypoxemia caused by hypoventilation, but does not correct the associated respiratory acidosis. Animals with persistent elevations in P_aCO_2 >60 mm Hg will generally require intubation and ventilatory support with positive pressure ventilation until the underlying cause can be relieved.

Adequacy of ventilation can also be assessed by measurement of end-tidal CO_2 (Figure 2.1). Because

Small Animal Thoracic Surgery, First Edition. E. Christopher Orton and Eric Monnet.

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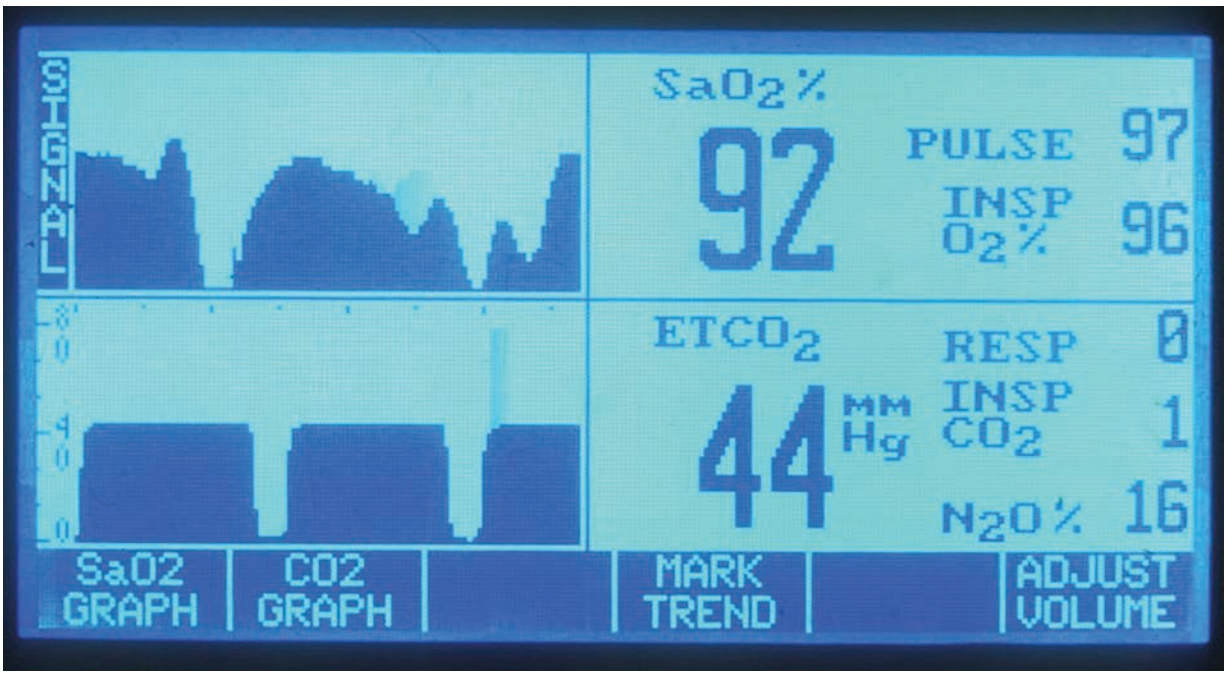

Figure 2.1 Pulse Oximetry and Capnography

diffusion of CO_2 in the lung is highly efficient, P_aCO_2 and alveolar carbon dioxide tension (P_ACO_2) are essentially equal. The carbon dioxide tension of expired gas at the end of expiration closely approximates P_aCO_2 and is termed end tidal carbon dioxide tension ($P_{ET}CO_2$). The $P_{ET}CO_2$ is measured clinically with a sampling port on the end of an endotracheal tube and capnograph that measures expired gas continuously and reports the peak carbon dioxide tension at the end of expiration. Measurement of $P_{ET}CO_2$ provides a good clinical estimate of P_aCO_2, and therefore of the adequacy of alveolar ventilation. A disadvantage of capnography is that measurement of $P_{ET}CO_2$ is only available in animals that are intubated. Thus, its principal utility is to evaluate ventilation is during anesthesia or in animals placed on ventilatory support.

There are two basic strategies for maintaining animals on ventilatory therapy for extended periods. Each has advantages and disadvantages. If ventilatory support is needed for <24 hours, then animals can be maintained with an endotracheal tube. A disadvantage of this approach that heavy sedation is typically required to maintain intubation. This further depresses ventilation, inhibits mucociliary clearance, promotes hydrostatic pulmonary congestion, and increases the risk for pneumonia. For animals needing longer periods of ventilatory support, placement of a cuffed tracheostomy tube is generally preferred to allow the patient to be awake and not recumbent. Weaning from ventilatory support is generally easier in animals that are not sedated. The major disadvantage of this approach is the risk of nosocomial infection at the tracheostomy site.

Gas Exchange

Pulmonary gas exchange is the collective process by which oxygen and carbon dioxide are exchanged between the alveolus and pulmonary venous blood. Exchange of oxygen is complex and dependent on diffusion across the alveolar-capillary membrane, matching of alveolar ventilation and perfusion, and avoidance of shunting of blood around the area of gas exchange. Ideally, arterial oxygen tension (P_aO_2) should be nearly equal to the oxygen tension in the alveoli (P_AO_2), which, in turn, is determined by alveolar ventilation. Gas exchange becomes impaired when P_aO_2 is substantially less than the calculated P_AO_2. The result is hypoxemia (low P_aO_2). Hypoxemia due to impaired gas exchange is additive to hypoxemia caused by hypoventilation. The causes of impaired gas exchange include diffusion impairment, ventilation-perfusion mismatch, and shunt. These are reviewed in Chapter 1. With the exception of right-to-left cardiac shunt, impairment of gas exchange results from primary pulmonary disorders.

Hypoxemia is assessed by direct measurement of P_aO_2 by arterial blood gas analysis. Gas exchange is assessed by calculation of the alveolar-arterial oxygen

difference (A-aPO_2), typically while animals are breathing room air. Calculation of A-aPO_2 is reviewed in Chapter 1. The A-a PO_2 should be <10 mm Hg for animals that are breathing room air. Animals with A-a PO_2 gradient of >30 mm Hg on room air have significant impairment of gas exchange and likely require administration of supplemental oxygen (40% to 50% F_IO_2). The response to supplemental oxygen is variable, depending on the underlying mechanism(s) of impaired gas exchange. Pulmonary shunt and right-to-left cardiac shunt are typically unresponsive to supplemental oxygen. Animals with severe hypoxemia secondary to pulmonary shunt may require additional respiratory support strategies such as positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP).

The adequacy of arterial oxygenation can also be assessed by monitoring arterial hemoglobin saturation (S_aO_2). The relationship of S_aO_2 to P_aO_2 is described by the oxygen-hemoglobin saturation curve. The sigmoid shape of the curve means that S_aO_2 will be relatively maintained until P_aO_2 falls <60 mm Hg. After that, S_aO_2 will tend to fall precipitously with further decreases in P_aO_2. A calculated S_aO_2 is typically provided as part of a blood gas analysis. S_aO_2 can also be monitored by pulse oximetry. Pulse oximetry uses colorimetry to estimate in hemoglobin saturation in capillary beds. It relies on detection of pulsatile changes in hemoglobin saturation to estimate S_aO_2. Its advantages are that it provides continuous data and it is noninvasive. It tends to work best when pulse pressures are normal to strong and less well when pulses are weak.

Ultimately, the oxygen content arterial blood (C_aO_2) is primarily determined by S_aO_2 and the concentration of hemoglobin in blood (gm/dL). Each gram of hemoglobin carries 1.34 mL of O_2 when fully saturated. While C_aO_2 is typically not measured directly in the clinical setting, it does serve to illustrate the importance of maintaining both pulmonary function and adequate hemoglobin levels in animals before and after surgery. Little is accomplished by efforts to maintain P_aO_2 and S_aO_2, when hemoglobin is allowed to fall to inappropriate levels. Ideally, the goal in surgery patients should be to maintain hematocrit >30% and/or hemoglobin >10 gm/dL.

Blood Pressure

Monitoring blood pressures provides critical information about the cardiopulmonary status of the patient. Pressure monitoring can include systemic arterial pressure, central venous pressure, pulmonary arterial pressure, and pulmonary wedge pressure. Each gives information about a different aspect of cardiovascular function.

Systemic Blood Pressure

Systemic arterial pressure is the most important pressure, reflecting overall cardiovascular function. It is described by systolic, diastolic, and mean pressure. The mean systemic blood pressure is the primary driver of tissue perfusion and is a direct function of cardiac output and systemic vascular resistance. Systemic vascular resistance is determined by the cumulative cross-sectional radius of the systemic arterioles (i.e., degree of arterioconstriction and arteriodilation) and the viscosity of blood (i.e., packed cell volume) Pulse pressure is the difference between the systolic and diastolic pressures. It determines the quality or strength of the pulse on palpation. Systemic pulse pressure is a direct function of stroke volume and an inverse function of compliance of the large elastic arteries. On a beat-to-beat basis, pulse pressure or pulse strength does provide information about the adequacy of stroke volume and thereby cardiac output. However, it does not provide information about the mean systemic pressure or the adequacy of tissue perfusion pressures. Therefore, measurement of systemic blood pressure is necessary to assure adequate cardiovascular function patients undergoing surgery.

Systemic blood pressure can be measured indirectly or directly. Indirect measurement includes Doppler and oscillometric methods. The Doppler method involves the Doppler detection of blood flow in a peripheral artery and a proximal cuff with a manometer (Figure 2.2). This method reliably measures only systolic pressure. That, combined with qualitative assessments of pulse pressure, allows for inferences about the adequacy of mean systemic pressure. Oscillometric blood pressure measurement is based on detection of oscillations in pressure during cuff inflation. This method provides estimates of systolic, diastolic, and mean systemic pressures. Indirect blood pressure measurement is least reliable when blood pressure and/or flow are low, precisely when accuracy is needed most. Direct measurement of systemic blood pressure is the most accurate and most reliable method for animals that need advanced monitoring. It also provides access for collection of samples for arterial blood gas analysis. Direct arterial catheterization can be done on most animals by percutaneous placement of an over-the-needle catheter into a dorsal pedal artery (Figure 2.3). Percutaneous access to a femoral artery can also be gained using a

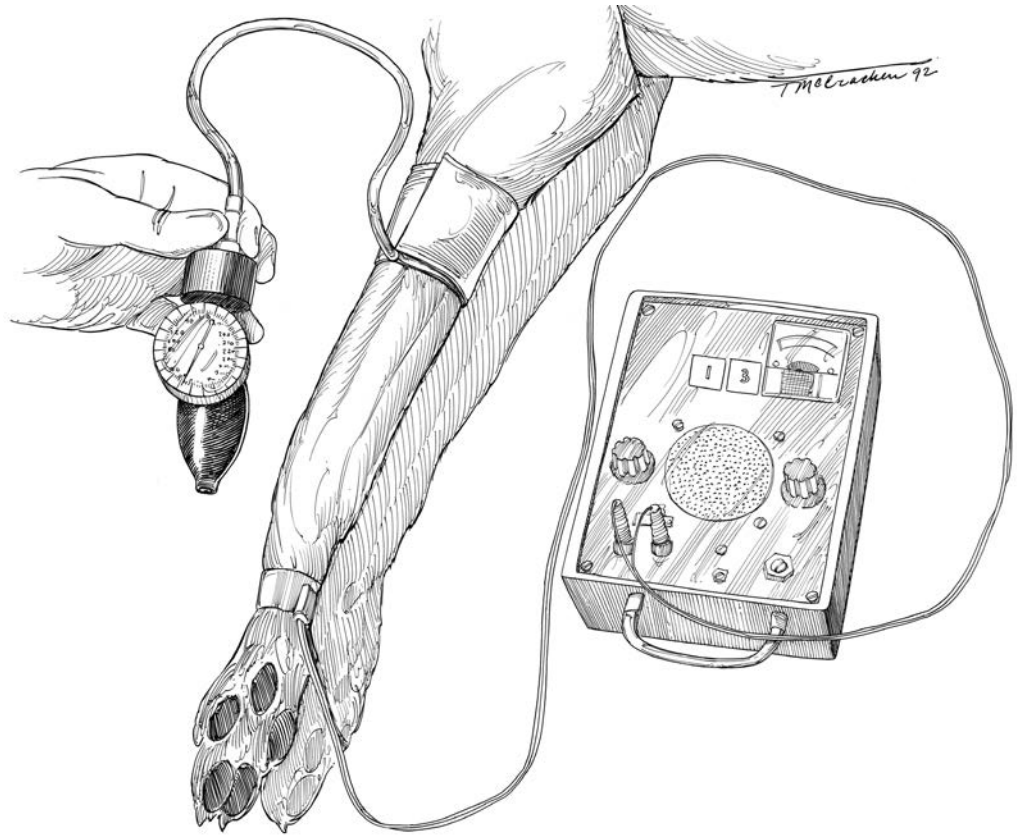

Figure 2.2 Systemic Blood Pressure—Indirect Doppler

Seldinger technique (Figure 2.4). The femoral artery is first punctured with a needle. A guidewire is passed through the needle into the artery. The catheter is then passed over the guidewire into the artery.

A general goal of supportive care in the perioperative setting is to maintain mean systemic pressure >60 mm Hg. Hypotension in the perioperative setting is caused by low cardiac output or inappropriate

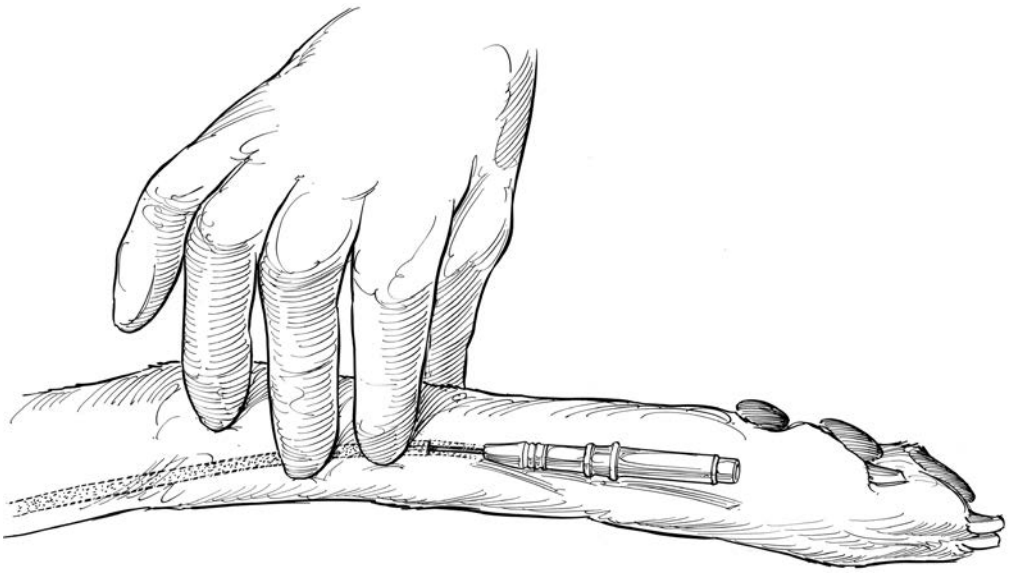

Figure 2.3 Systemic Blood Pressure—Direct Percutaneous

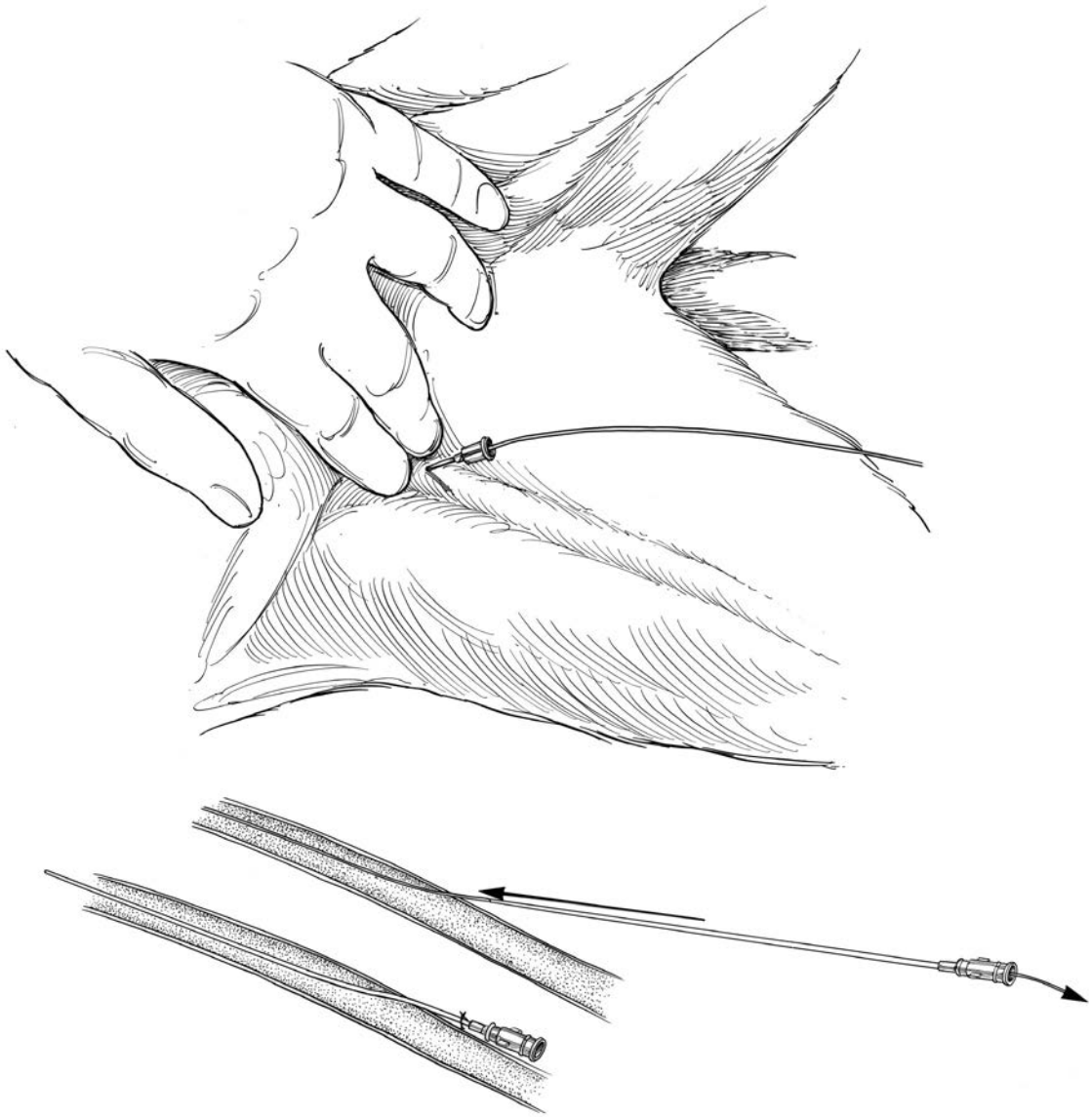

Figure 2.4 Systemic Blood Pressure—Direct Seldinger

vasodilation, or both. Attention is first directed at correcting any deficits in cardiac output through volume support and/or administration of inotropic drugs. Animals that remain hypotensive after cardiac output support has been optimized may have inappropriate vasodilation commonly associated with the systemic inflammatory response. These animals may benefit from judicious administration of vasoconstrictor drugs such as alpha agonists or vasopressin.

Central Venous Pressure

Central venous pressure (CVP) is measured by direct catheterization of a jugular vein and passage of a catheter into a central intrathoracic vein (Figure 2.5). Commercially available jugular catheters with up to 3 lumens are available. Placement of these catheters utilizes the Seldinger method. While the cardiac and respiratory cycles create fluctuations in venous pressures, it is the mean central venous pressure that is of primary interest for patient monitoring. Mean CVP provides information about the adequacy of vascular blood volume and right heart preload. Mean CVP of about 3 to 5 cm H_2O (2 to 3.5 mm Hg) suggest that vascular blood volume and right heart preload are adequate in surgery patients. Lower CVP suggests the need for more volume support of the patient. High CVP suggests the possibility of vascular volume overload, right heart dysfunction, or cardiac tamponade. Animals with right heart dysfunction may require a higher CVP to maintain adequate cardiac output. The pitfalls of monitoring CVP have been the subject of

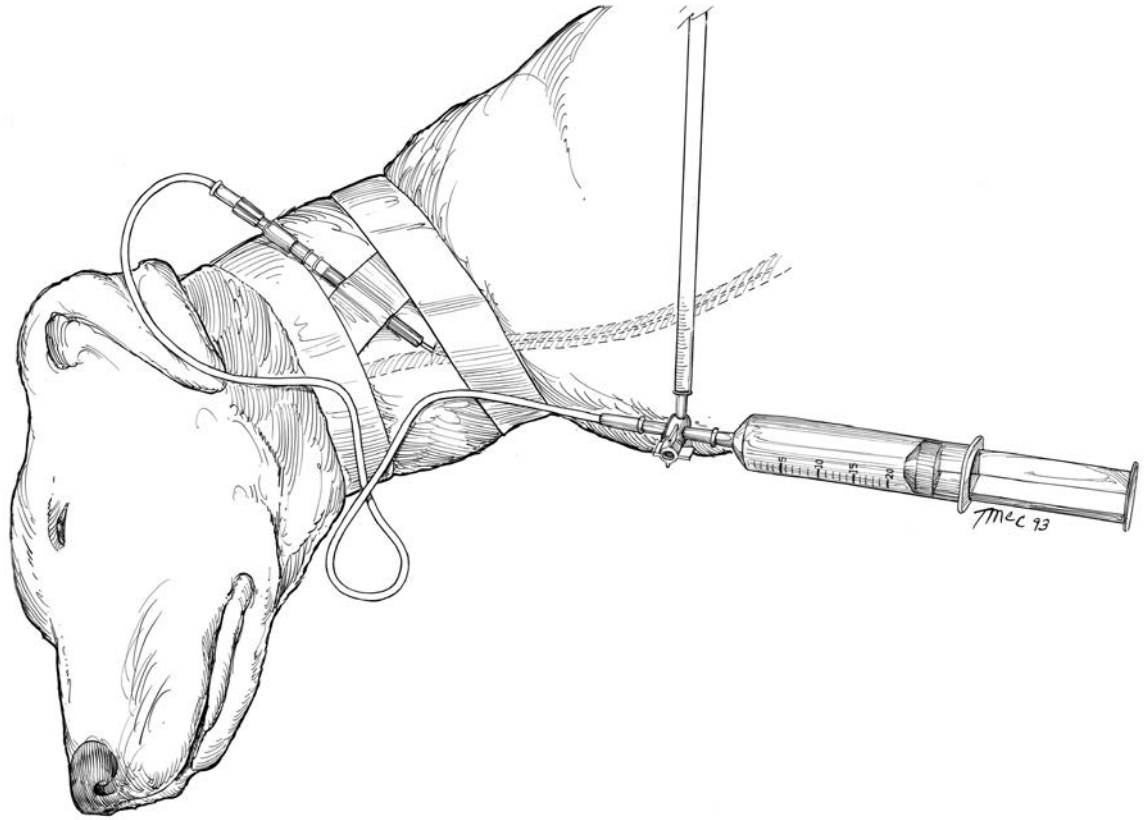

Figure 2.5 Central Venous Pressure

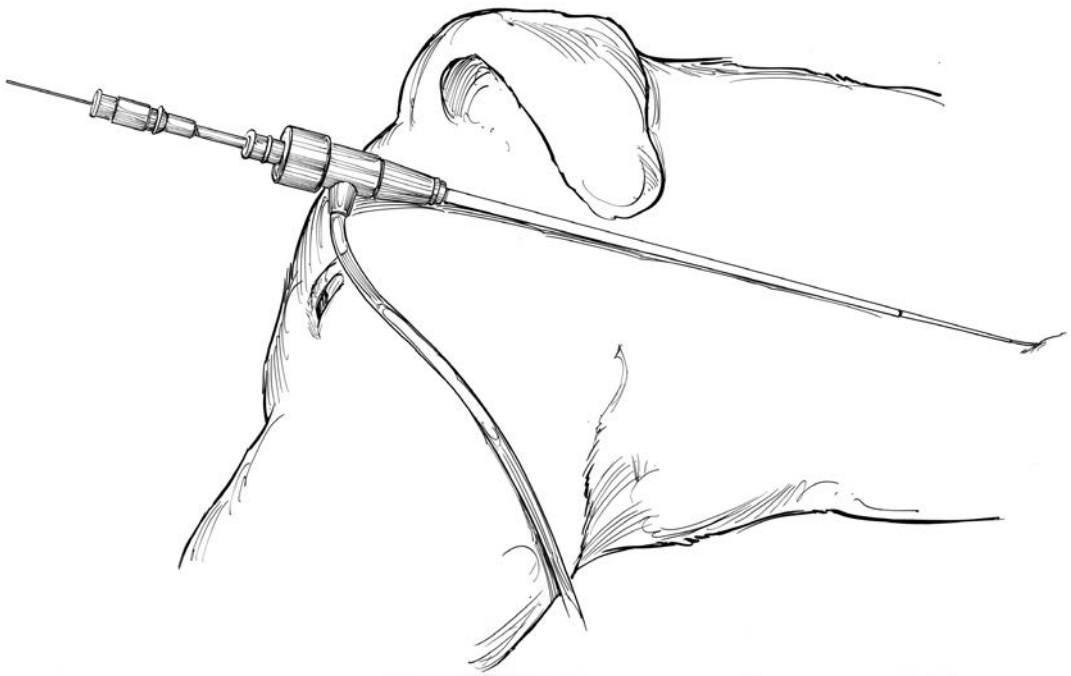

Figure 2.6 Jugular Introducer Catheter

many studies. Nevertheless, monitoring tends in CVP is one of the best ways to understand the adequacy of volume support in surgery patients.

Pulmonary Arterial and Wedge Pressures

While systemic arterial pressure and CVP form the basis of pressure monitoring for most patients, additional information can be gained in certain patients by introduction of a balloon catheter into the pulmonary artery. Balloon catheters are introduced via a specialized introducer catheter placed into a jugular vein (Figure 2.6). Jugular introducer catheters are place using a Seldinger technique. They are equipped with a diaphragm on the end to allow introduction of the balloon catheter and side injection port for flushing. The balloon catheter is passed through the right heart while monitoring characteristic pressure waveforms as the catheter passes through the right atrium, right ventricle, and pulmonary artery (Figure 2.7).

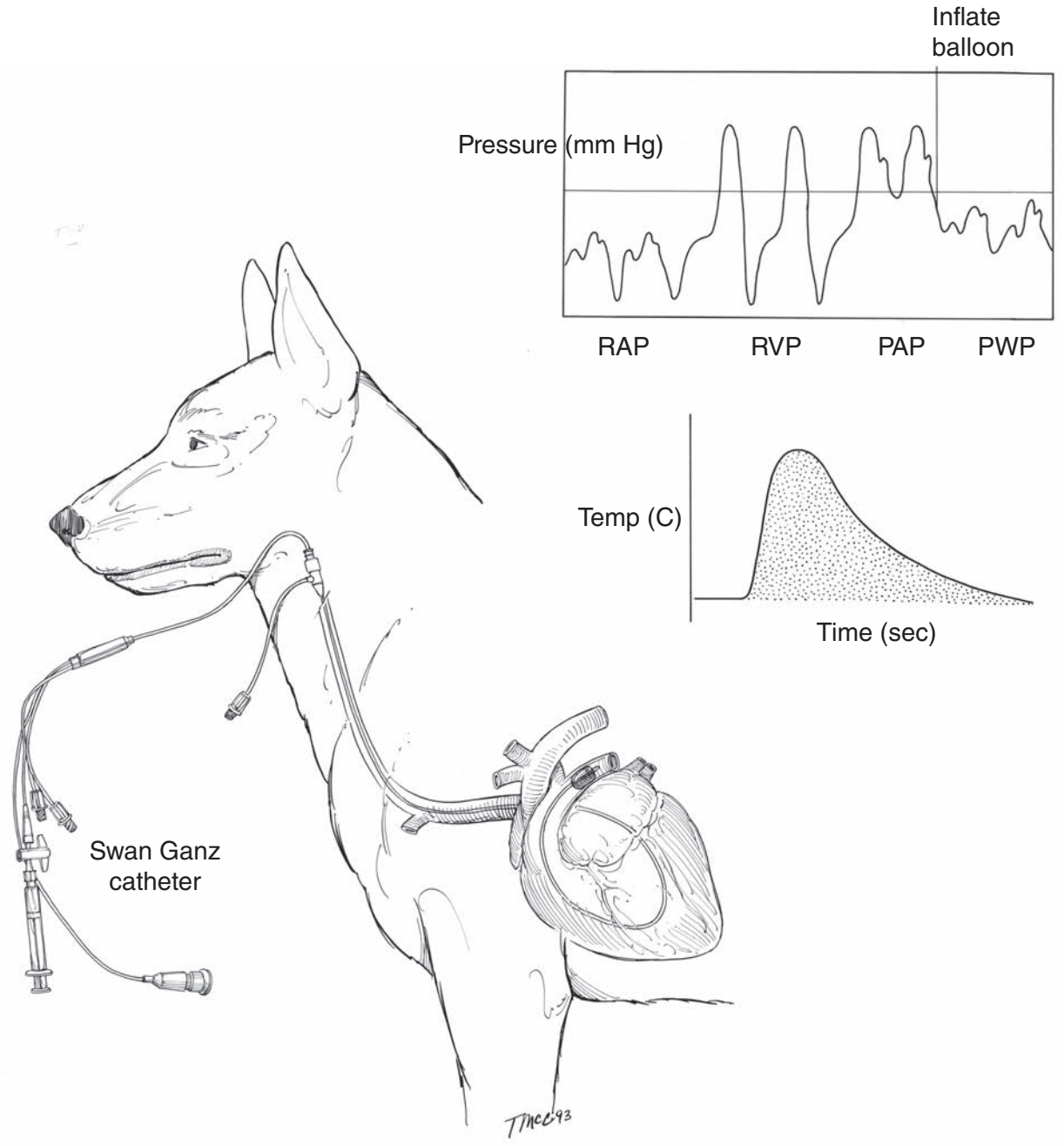

Figure 2.7 Swan-Ganz Pulmonary Catheter

Placement is aided by inflating a small balloon at the catheter tip, which helps "flow direct" the catheter through the right heart. A lumen at the catheter tip allows continuous measurement of pulmonary artery pressures (PAP). A more proximal lumen allows continuous measurement of right atrial pressure (RAP), or CVP. Pulmonary wedge pressure (PWP) is obtained by intermittent inflation of the balloon at the catheter tip. This momentarily stops flow to a segment of the pulmonary vascular bed, allowing it to become an extension of the fluid-filled catheter tip. Thus, PWP becomes an estimate of left atrial pressure, which, in turn, provides information about preload to the left heart.

Balloon catheters are particularly useful for managing animals during and after cardiac surgery and in animals with pulmonary hypertension. A specialized type of balloon catheter known as a Swan-Ganz catheter also has a small thermistor at the catheter tip that measures blood temperature. This is used to measure cardiac output by the thermodilution method.

Cardiac Output

Cardiac output is the volume of blood pumped by the heart per unit of time typically expressed in mL/min or L/min. Cardiac output converted to dL/min and multiplied by oxygen content (ml O_2/dL) gives the oxygen delivery (VO_2) to the tissues in mL O_2/min. As reviewed in Chapter 1, oxygen content (C_aO_2) is primarily a function of the adequacy of saturation of hemoglobin (S_aO_2) and the concentration of hemoglobin in the blood. From these relationships, it can be seen that adequate delivery of O_2 to tissues is ultimately dependent on maintaining pulmonary function, hemoglobin concentration, and cardiac function. Evaluation and support of cardiac output represents the last critical step to providing cardiopulmonary supportive care.

Cardiac output assessment can be accomplished by measurement of absolute cardiac output or by evaluating indices that reflect the adequacy of cardiac output. In the clinical setting, cardiac output can be measured by the thermodilution method using a Swan-Ganz pulmonary catheter. This is a type of *indicator dilution* method that uses injections of cold saline at known volume and temperature to generate a thermodilution (blood temperature versus time) curve. The area under the thermodilution curve is inversely proportional to cardiac output and can be used to calculate an absolute cardiac output. Typically, the calculation is done by a hemodynamic monitor capable of supporting thermodilution. Absolute cardiac output is, of course, a function of the size and metabolic requirements of the patient. Typically, cardiac output is normalized to body surface area to determine the cardiac index (mL/min/M^2). Although values of cardiac index are well established for human patients, they are less well established for animals. In addition, there are a number of clinical situations, such as the presence of intracardiac or peripheral arteriovenous shunt where absolute measures of cardiac output become less meaningful and do not assure adequate tissue perfusion.

In small animals, assessment of cardiac output is typically focused on indices that reflect adequacy of cardiac output rather than its absolute measurement. Two indices most often used in the clinical setting to evaluate the adequacy of cardiac output are resting blood lactate concentration and calculation of oxygen extraction. Blood lactate concentration is typically included as a measured index by most blood gas analyzers. Persistently elevated resting blood lactate levels >2 suggest that cardiac output is not adequate. As reviewed in Chapter 1, the oxygen extraction ratio can be calculated by measuring arterial and venous oxygen saturations (Equation 1.14). Normally, the peripheral tissues only extract about 25% of the oxygen that is delivered to tissues. When oxygen delivery is inadequate because of low cardiac output, the only available compensation is for the peripheral tissues to extract a higher percentage of what is delivered. Calculated oxygen extraction ratios >0.4 provide strong evidence that cardiac output is inadequate.

Support of cardiac output is directed at maintaining adequate stroke volume and appropriate heart rate and rhythm. As reviewed in Chapter 1, stroke volume is a function of cardiac preload, afterload, and contractility. On a beat-to-beat basis, cardiac afterload is an inverse function of systolic blood pressure in the absence of aortic stenosis. Because systolic hypertension is uncommon in the perioperative period in animals, increased afterload is rarely an acute cause of decreased cardiac output in the perioperative setting. In the rare instances where systolic hypertension is present, appropriate vasodilator therapy to lower systolic blood pressure would be indicated. Maintenance of cardiac output in the perioperative setting is generally focused on maintaining appropriate cardiac preload and contractility. Assuring adequate preload in the perioperative period is primarily directed at maintaining adequate blood volume.

Historically, assessment of blood volume and venous return has been based on CVP. Preoperative CVP can serve as a useful guide for intraoperative and postoperative volume administration, especially in animals with preexisting cardiac disease. Serial

echocardiographic monitoring of cardiac dimensions or volume has emerged as a more reliable way to evaluate the adequacy of cardiac preload. Deficits in cardiac preload are addressed by administration of volume. The type of volume support is guided by the total protein and hemoglobin concentrations. Overreliance on crystalloid fluids to correct volume deficits in the perioperative can lead to hemodilation and should be avoided. Correction of low cardiac output after preload and afterload have been optimized is generally directed at increasing cardiac contractility through administration of inotropic drugs such as beta-adrenergic agonists (dobutamine) and phosphodiesterase-3 inhibitors (milrinone), or both. Prior to administering inotropic therapies, acute mechanical causes of low cardiac output such as thromboembolism (cardiac, systemic, or pulmonary), cardiac tamponade, or constrictive pericarditis (pericardiotomy syndrome) should be ruled out by echocardiography.

Heart Rate and Rhythm

Assessment of heart rate and rhythm in the perioperative period is typically accomplished by continuous monitoring of the electrocardiogram. Abnormalities in heart rate and/or rhythm can have profound effects on cardiac output as well as place the patient at risk for sudden cardiac arrest. Sinus tachycardia is recognized as an increase in the resting heart rate of the normal rhythm with sinus origin P-waves in front of narrow (supraventricular origin) QRS-complexes (Figure 2.8a). As with any supraventricular origin rhythm, QRS complexes can take on a wide-complex morphology if there is aberrancy of conduction. Treatment is directed at correcting its underlying cause(s) including low cardiac output, pain, and anxiety.

Both chronic and spontaneous atrial fibrillation may be present or occur in perioperative setting. It is more likely in animals with underlying structural heart disease, however can occur as a lone rhythm disturbance without apparent cardiac disease. Atrial fibrillation is recognized as a fast to very fast narrow QRS-complex tachycardia without recognizable P-waves (Figure 2.8b). The key diagnostic finding is its irregularity even at very fast rates. Atrial fibrillation adversely affects cardiac output by causing very fast heart rates and through the loss of atrial contraction. Management of atrial fibrillation in the perioperative setting is typically directed at rate control by administration of diltiazem. Spontaneous atrial fibrillation associated in the perioperative is often self-limiting and can be managed by rate control. Cardiosynchronous cardioversion of atrial fibrillation is generally not attempted in the perioperative setting unless the animal cannot be stabilized. A variety of other supraventricular tachyarrhythmias can occur in the perioperative period, including atrial flutter (Figure 2.8c), focal atrial tachycardia (Figure 2.8d), and atrioventricular nodal tachycardia (Figure 2.8e). These are generally recognized as fast to very fast regular tachycardias with narrow (supraventricular origin) QRS-complexes. P-wave morphology and timing depend on the specific rhythm disturbance. Management depends on specific rhythm diagnosis.

Ventricular premature complexes (Figure 2.8f) and ventricular tachycardia (Figure 2.8g) are common in the perioperative setting. The later rhythm may be paroxysmal or sustained. The hallmark diagnostic finding is a wide (ventricular origin) QRS-complex. P-waves are dissociated from the wide QRS-complexes, which helps distinguish ventricular tachycardia from supraventricular tachycardias with aberrant conduction. Management of ventricular tachycardia depends on the degree of associated hemodynamic impact and the perceived risk for sudden cardiac arrest. Paroxysmal ventricular rhythms with normal to mildly elevated rates and uniform QRS complexes (idioventricular rhythms) are usually self-limiting and generally do not need to be suppressed. Fast ventricular tachycardia that adversely affects hemodynamic status should be treated with intravenous lidocaine. Ventricular tachycardias with tight coupling intervals, suspected multifocal origin, or polymeric QRS complexes (torsade de point) carry a risk for sudden cardiac arrest and should also be treated.

Bradyarrhythmias occasionally are seen the perioperative period. Sinus bradycardia is recognized as a slowing of the normal sinus origin rhythm caused by inappropriate parasympathetic tone to the heart. It is commonly observed during surgery due to the effects of anesthetic drugs, but is unusual in the postoperative setting. Treatment with parasympatholytic drugs is undertaken if it adversely affects cardiac output. Atrioventricular (AV) block is an uncommon postoperative complication of certain cardiac surgeries such as septal defect repairs. Second-degree AV block is recognized as an intermittent dropping of the QRS complex after a sinus origin P-wave. Third-degree AV block is recognized as complete dissociation between the P-waves and a typically wide QRS complex ventricular escape rhythm. High-grade second-degree and third-degree AV block will generally adversely affect cardiac output and require temporary cardiac pacing in the immediate postoperative period until a

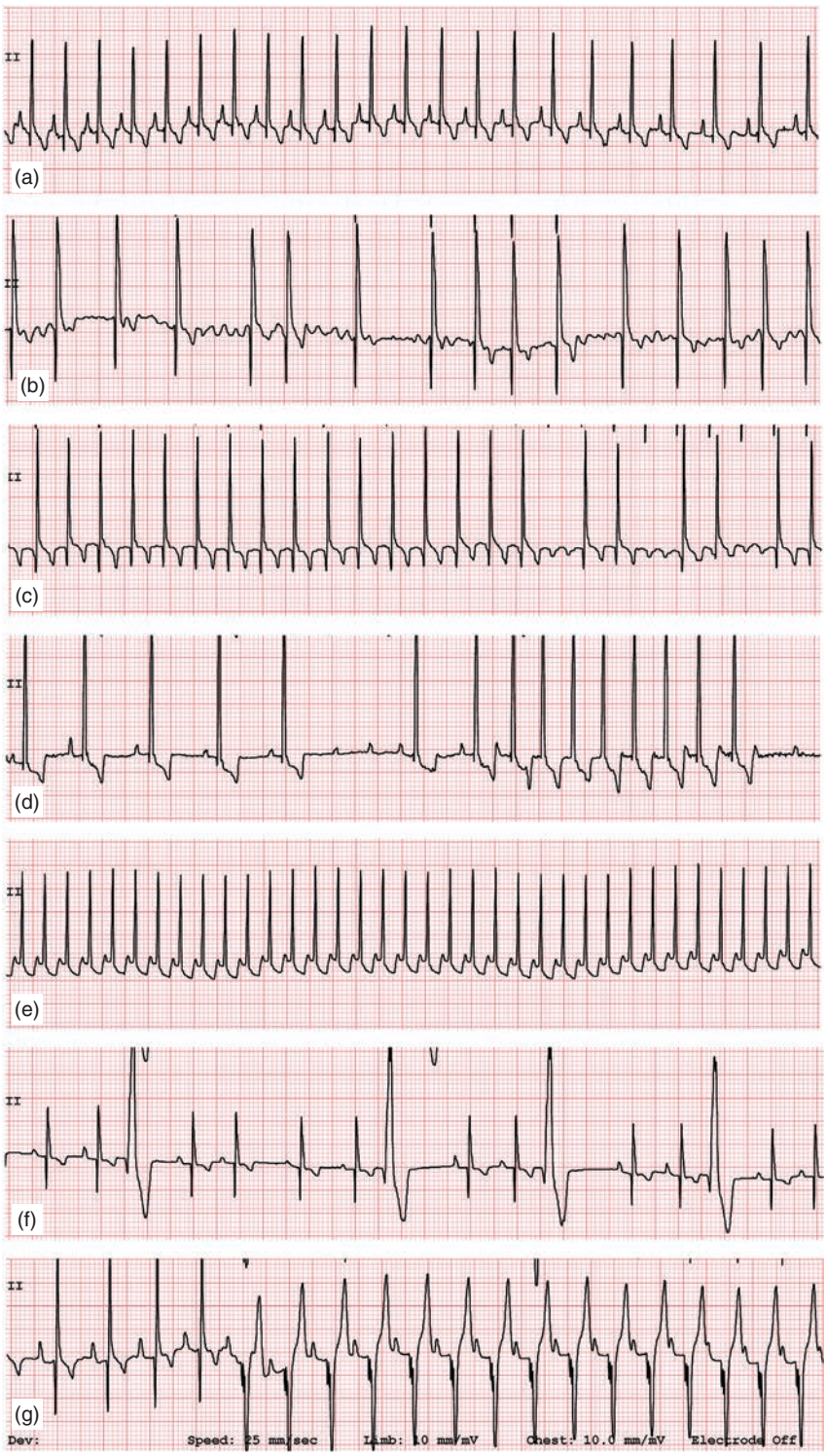

Figure 2.8 Tachyarrhythmias

permanent pacemaker can be implanted. Atrial standstill is a slow cardiac rhythm recognized by an absence of P-waves. The rhythm may be preceded by a period of gradual diminishment of the P-wave voltage. This rhythm occasionally occurs in the perioperative as a result of hyperkalemia, usually caused by oversupplementation of potassium. Urgent treatment is indicated to lower the serum potassium.

3

Instrumentation

E. Christopher Orton

Proper instrumentation is an important element of successful thoracic surgery. In addition to basic instruments used in general surgery, several specialized instruments are needed for thoracic surgery. The standard retractor for thoracic approaches is a *Finochietto retractor* (Figure 3.1), which should be available in at least four sizes to accommodate different size animals.

A general design characteristic of thoracic instruments is length. An optimal working length for most surgeries is 17 to 23 cm. Both short and long *scalpel handles* with #11, #10, and #15 *scalpel blades* are needed (Figure 3.2). The standard tissue forceps for thoracic surgery is the *DeBakey tissue forceps* (Figure 3.3a), which allows for atraumatic handling of thoracic tissues. At least one pair of DeBakey forceps with carbide inlays for grasping needles facilitates suturing within the deep thoracic cavity (Figure 3.3b). These are typically demarcated by a gold handle. Standard long handled rat-tooth tissue forceps

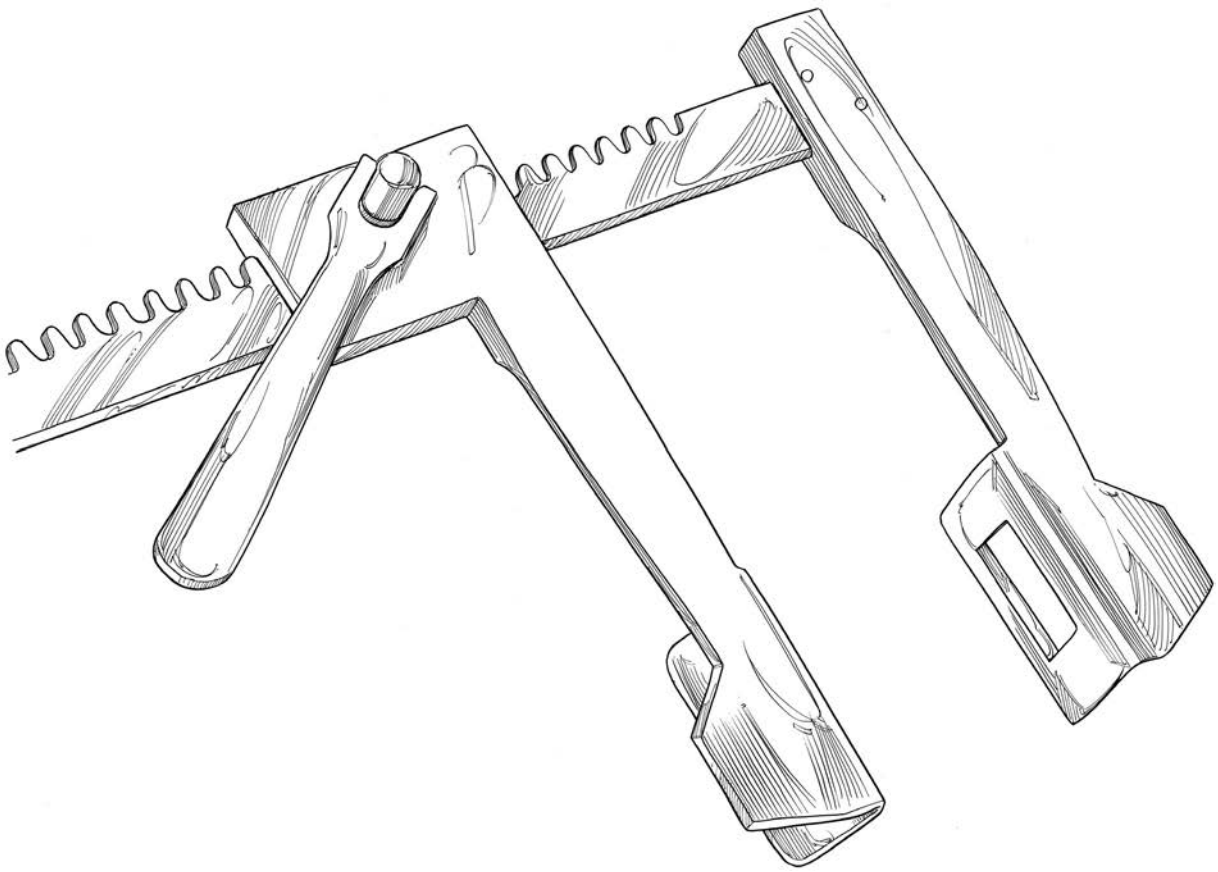

Figure 3.1 Finochietto Retractors

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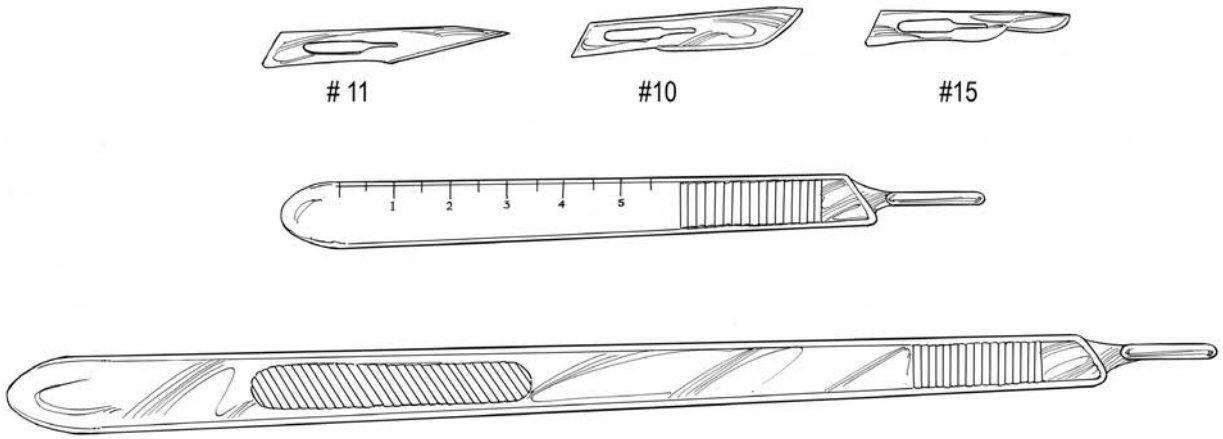

Figure 3.2 Scalpel Handles and Blades

are also useful for working with muscular tissues such as the diaphragm (Figure 3.3c). A selection of scissors should be available, including *Mayo* (Figure 3.4a), *Metzenbaum* (Figure 3.4b), *delicate Metzenbaum* (Figure 3.4c), and *45° Potts scissors* (Figure 3.4d).

Curved scissors are generally more versatile than straight designs. *Reverse Potts scissors* are useful for certain cardiac and vascular surgeries but are not absolutely necessary. Needle holders should be long and available in a selection of sizes to accommodate a variety of needle sizes, including *Mayo-Hager* (Figure 3.5a), *Crile-Wood* (Figure 3.5b), and *Castroviejo needle holders* (Figure 3.5c).

Angled thoracic forceps (Figure 3.6) are one of the most versatile and important instruments used in thoracic surgery. They should be available in 10 to 12 different sizes and angles, ranging from very large to very fine and 60° to 90° to accommodate different types of surgery and patient sizes. Various types of hand-held retractors are useful for working in the depths of the thoracic cavity. One of the most versatile retractors is the *malleable retractor* (Figure 3.7a), which comes in a variety of lengths and widths. *Cardiovascular retractors* (Figure 3.7b) also come in a selection of widths and are particularly useful for cardiac surgeries. Standard *Army-Navy retractors* (Figure 3.7c) are helpful in performing thoracic approaches.

Lastly, various atraumatic vascular clamps are critically important for cardiac surgeries. Basic types include straight, angled (Figure 3.8), and tangential vascular clamps (Figure 3.9). Vascular clamps come in a variety of sizes, handle angles, and tooth patterns to accommodate various procedures and patient sizes. A well-equipped thoracic pack would typically include at least 12 vascular clamps of different sizes and shapes.

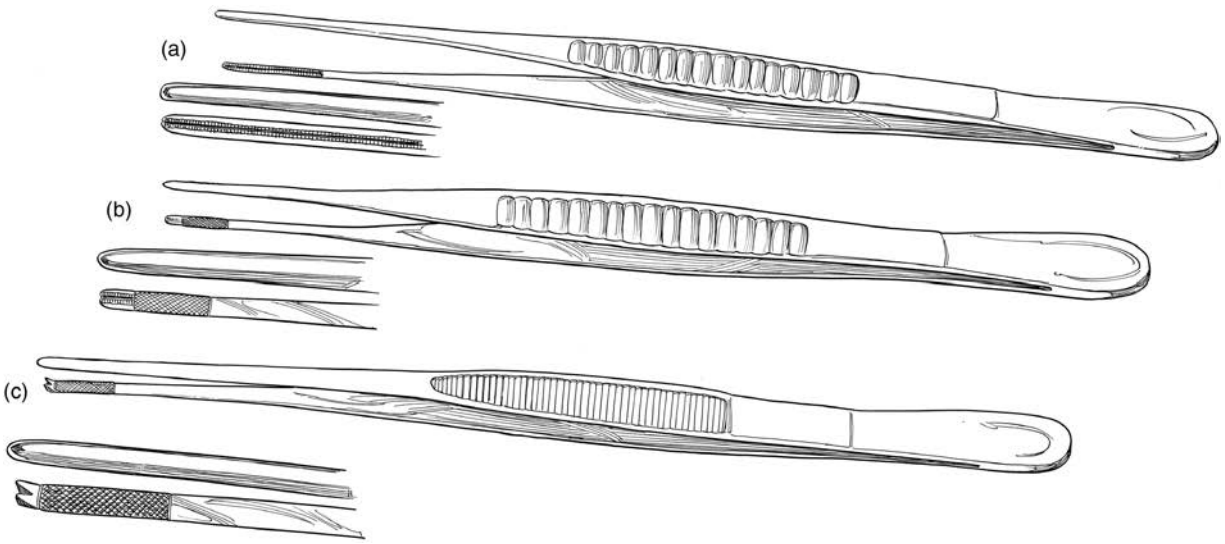

Figure 3.3 DeBakey Tissue Forceps

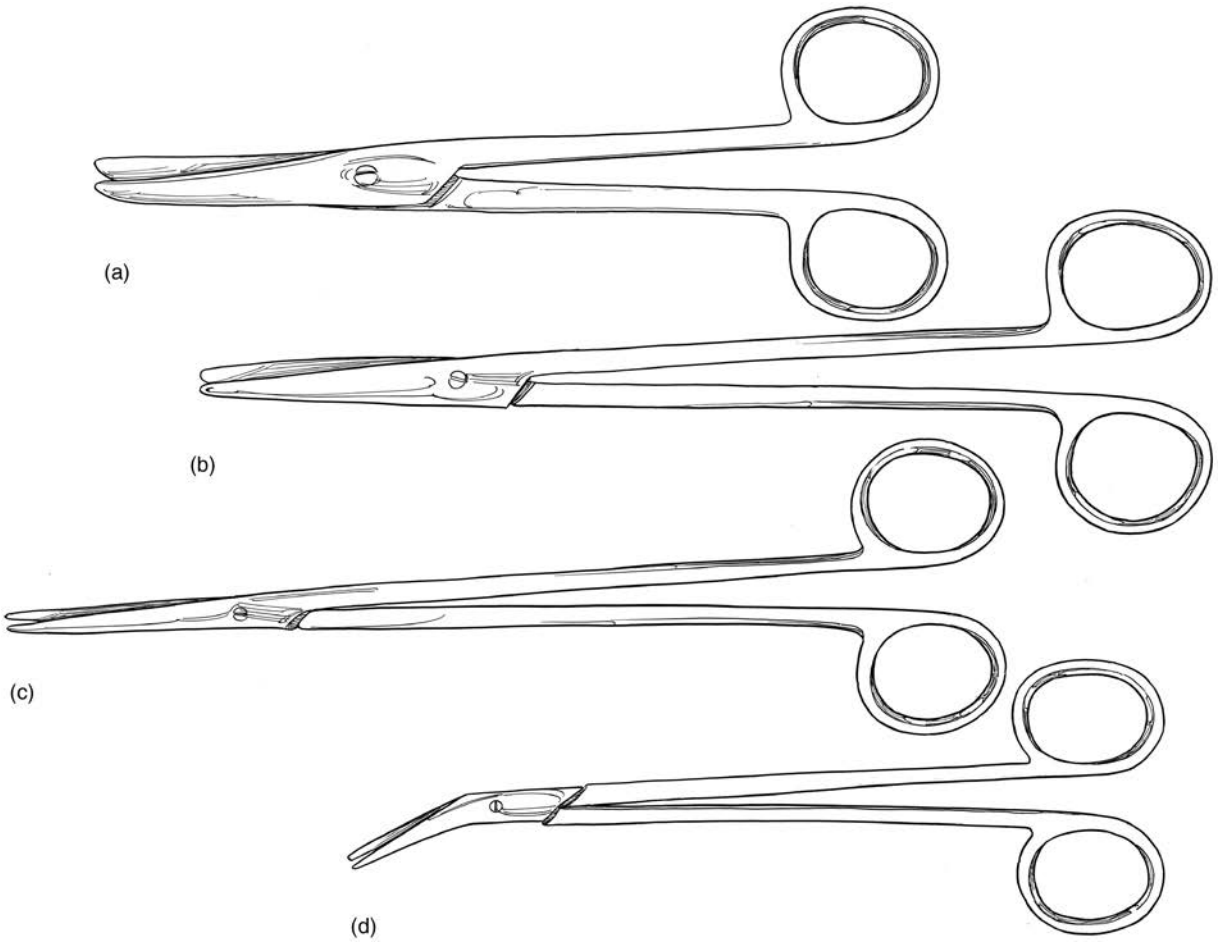

Figure 3.4 Scissors

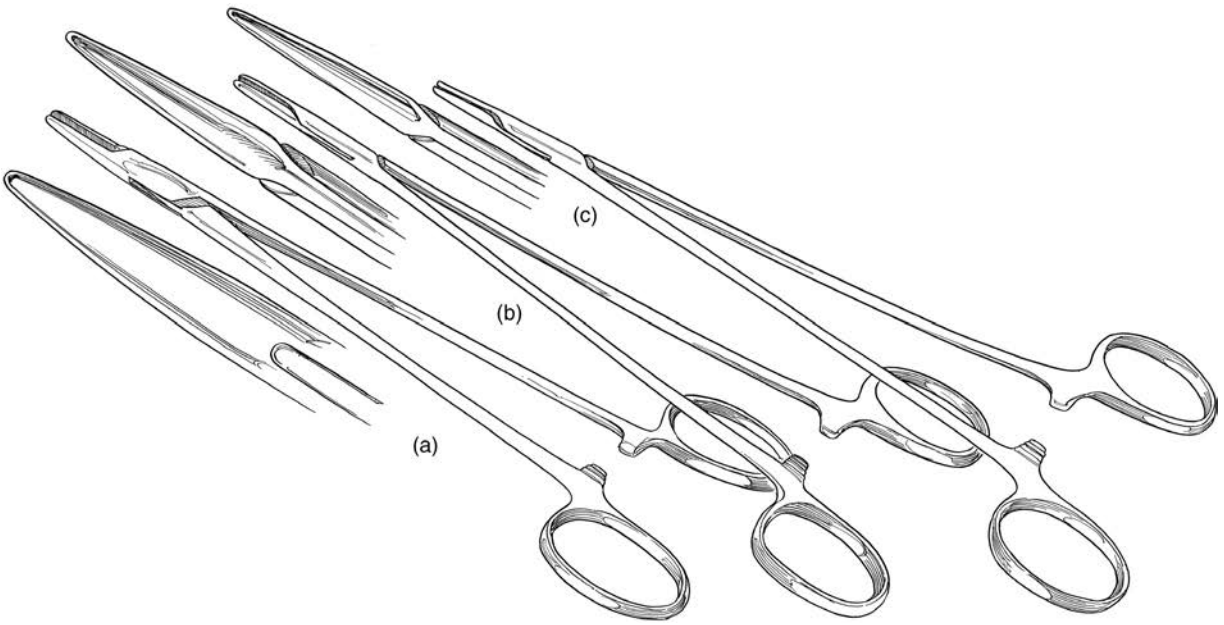

Figure 3.5 Needle Holders

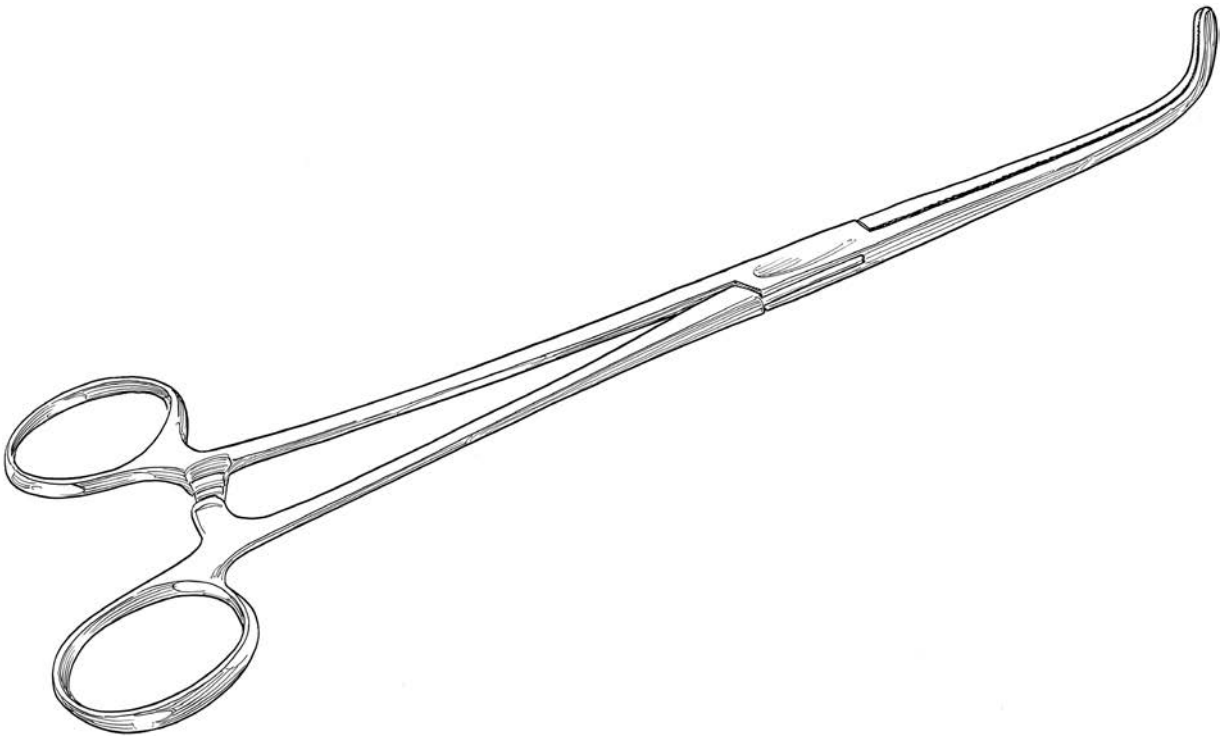

Figure 3.6 Angled Thoracic Forceps

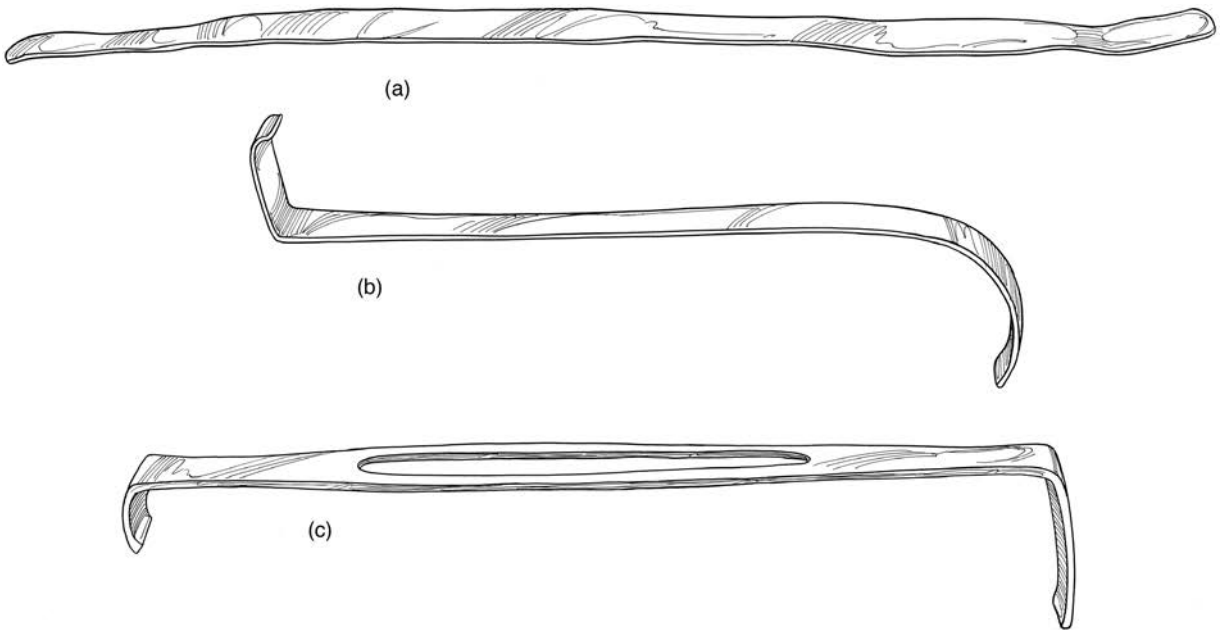

Figure 3.7 Hand Retractors

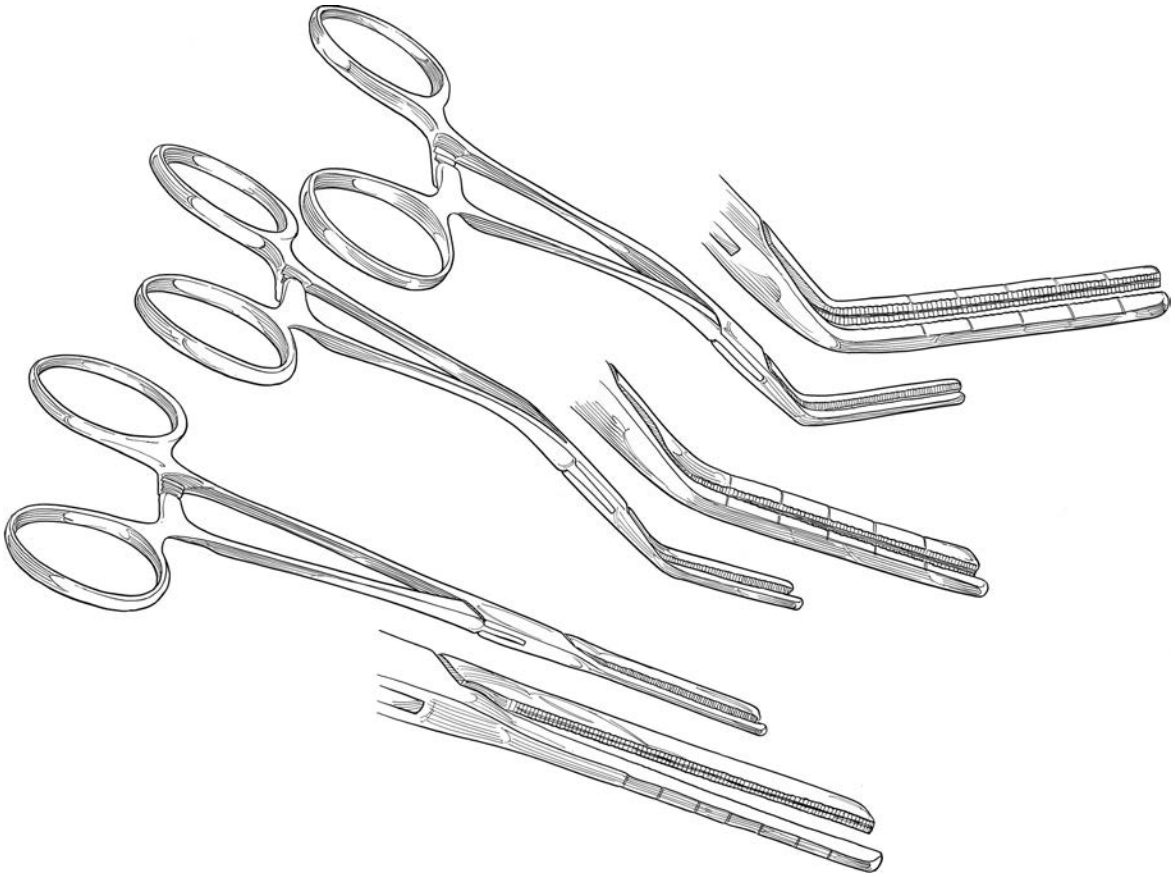

Figure 3.8 Vascular Clamps

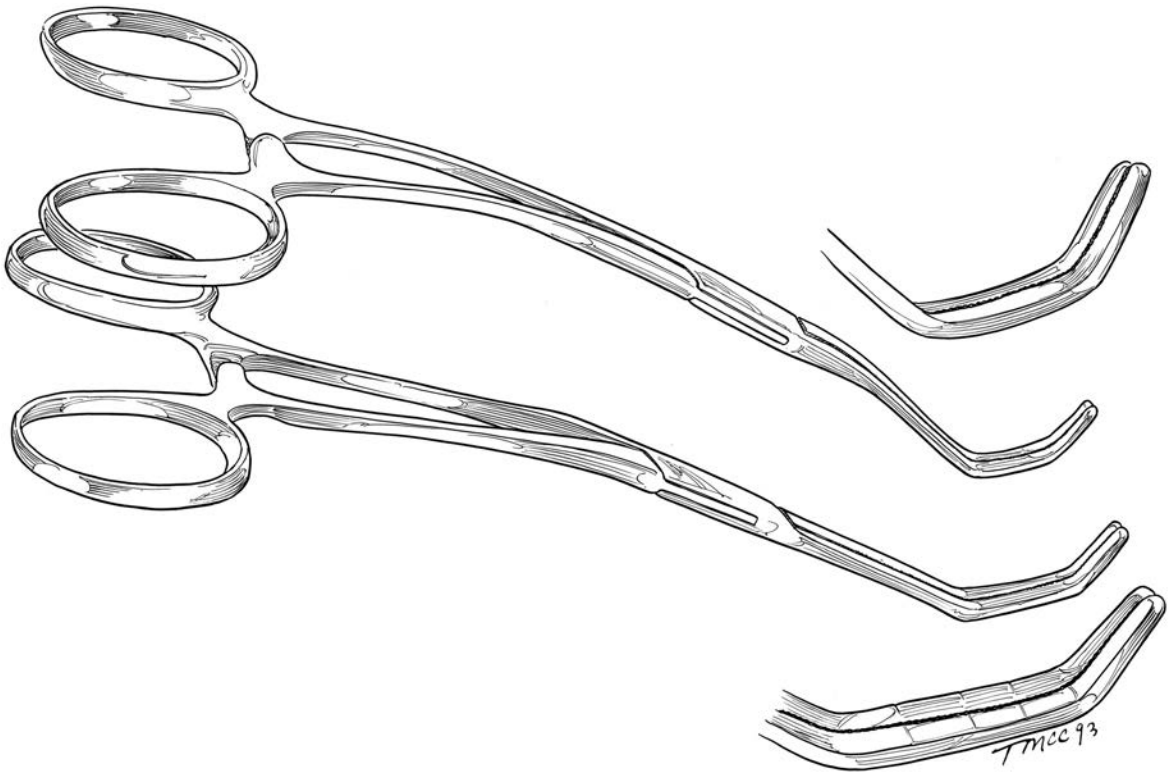

Figure 3.9 Tangential Vascular Clamps

Section II

Thoracic Approaches

4

Thoracotomy

E. Christopher Orton

Intercostal thoracotomy is the most commonly employed thoracic approach used for animals. The shape of the thoracic cavity in companion animals makes thoracotomy the more conducive approach, both from the standpoint of access to thoracic structures and from the fact that lateral recumbency is a more physiologically natural position in these species. While thoracotomy usually provides the most optional access to regional thoracic structures, its chief limitation is that it does not provide access to the entire thoracic cavity. Structures outside of the immediate vicinity of the approach, particularly structures in the contralateral side of thorax, can be difficult or impossible to access. Thus, planning both the side and intercostal space for a thoracotomy is critical to a successful surgery. Planning should specifically include consideration of which thoracic structures will be assessable for surgery, as well as which structures will not be available should surgery reveal something unexpected.

Intercostal Thoracotomy

The site of an intercostal thoracotomy may be the third through the tenth intercostal space on either side of the thorax depending on the thoracic structures to be exposed. A lateral thoracic radiograph or other imaging modalities (ultrasound, CT, MRI) are helpful in determining the intercostal space that best exposes a desired thoracic structures or pathology. Organs in the cranial abdomen can be accessed by combining a caudal intercostal thoracotomy and diaphragmatic incision.

The skin and cutaneous trunci muscle are incised over the desired intercostal space (Figure 4.1a). The incision is extended from the costovertebral junction toward the sternum. For a thoracotomy in the fourth through sixth intercostal space, the latissimus dorsi muscle is incised parallel to the skin incision. Once the latissimus dorsi muscle in incised, the correct intercostal space is confirmed by counting from the first intercostal space. The fifth rib is an important landmark recognized by the insertion of the scalenus muscle and the origin of the external abdominal oblique muscle. Depending on the desired intercostal space, either the scalenus or external abdominal oblique muscle is incised (Figure 4.1b). The serratus ventralis muscle should be separated between its muscle bellies to expose the intercostal space (Figure 4.1c). The intercostal muscles are incised in the middle of the intercostal space to avoid damage to the intercostal vessels and nerves (Figure 4.1d). The thoracotomy is completed by bluntly puncturing the pleura with scissors, allowing the lungs to fall away, and then extending the incision with scissors. The intercostal incision may be extended dorsally to the tubercle of the rib and ventrally past the costochondral arch to the level of the internal thoracic vessels to achieve full exposure. The internal thoracic artery courses parallel and on either side of sternum beneath the transversus thoracis muscle. Awareness of its position is the key to avoid incising it. The ribs are slowly spread with a Finochietto retractor to gain the needed exposure to desired thoracic structures (Figure 4.2). Exposure can be increased if necessary by cutting the costal cartilage just caudal to the thoracotomy (Figure 4.2 inset), although this is rarely necessary.

A thoracostomy tube is always placed and secured prior to closure of the thoracotomy. The safest method of placing the thoracostomy tube is by passing a large right-angle forceps from inside to outside of the thoracic cavity and then using the forceps to guide the tube into the thoracic cavity. This minimizes the risk of puncturing thoracic structures during placement. The tube is secured to the outside thoracic wall with a *finger trap* type suture. The end of the thoracostomy tube should be positioned in the cranial ventral thoracic cavity close to the sternum. During closure of the thoracotomy, the thoracostomy tube is left open to

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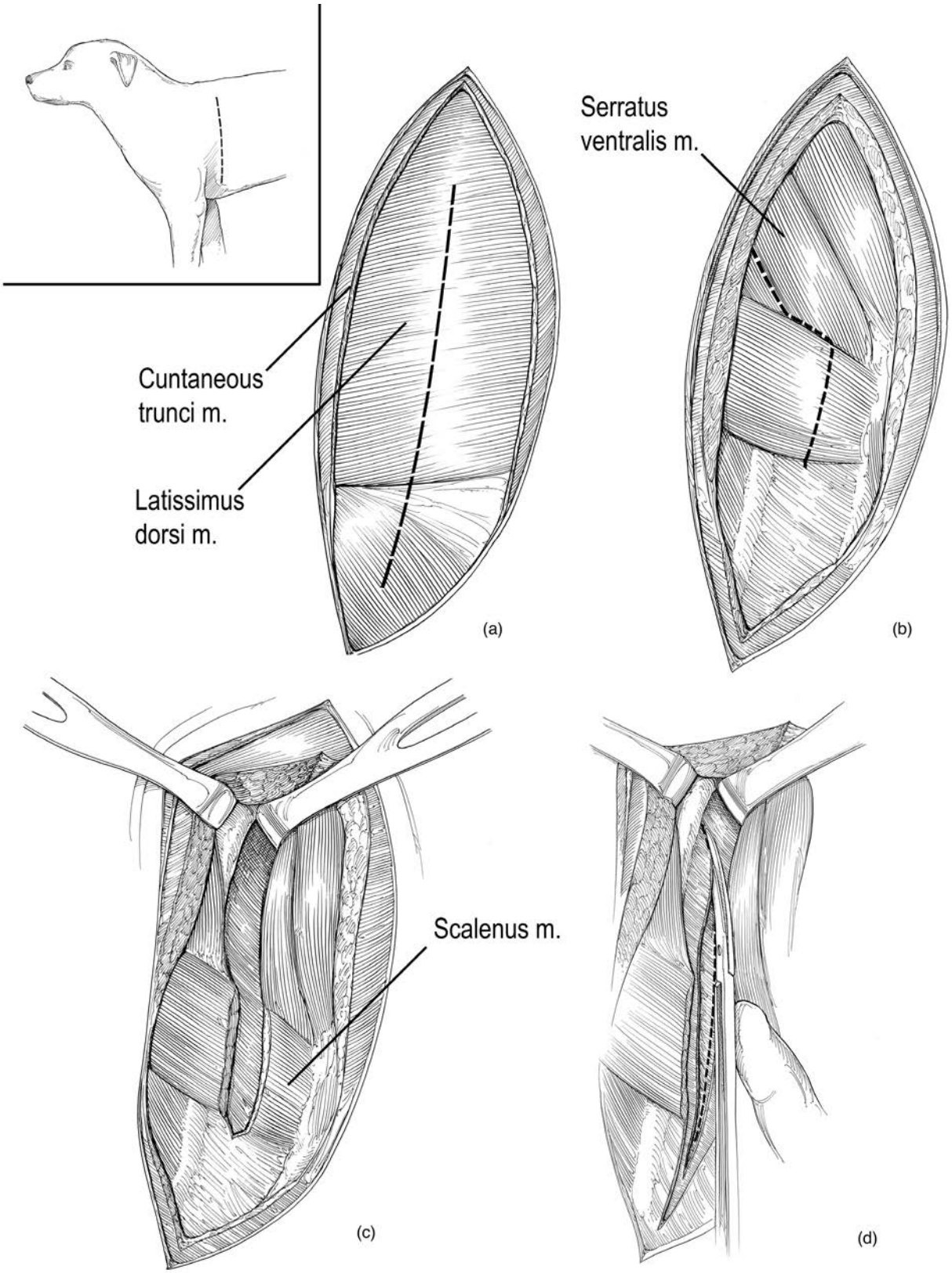

Figure 4.1 Intercostal Thoracotomy

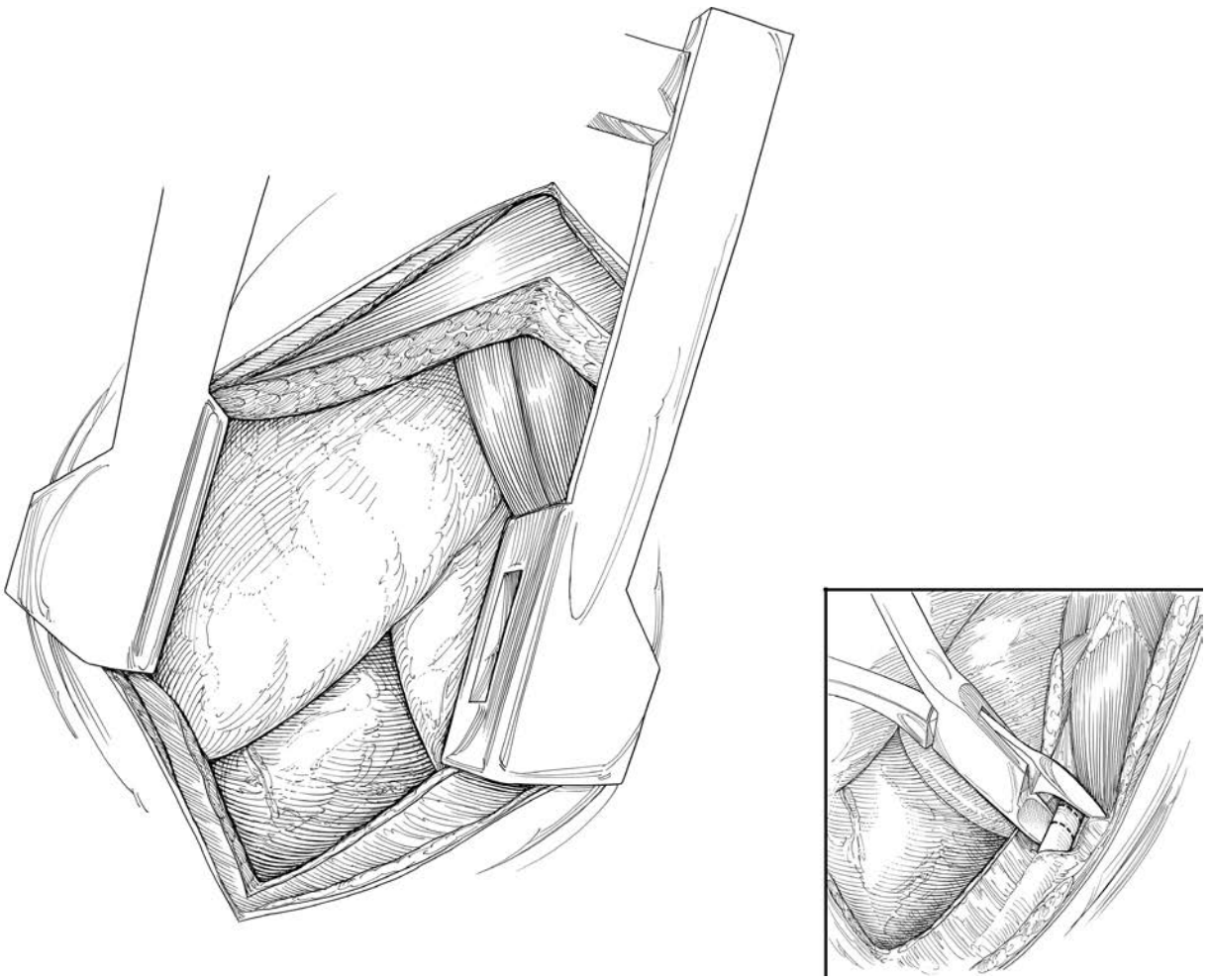

Figure 4.2 Thoracotomy Retraction

atmosphere to avoid any possibility of creating a tension pneumothorax.

Closure of the thoracotomy is accomplished by preplacing heavy interrupted circumcostal sutures around the ribs immediately adjacent to the thoracotomy (Figure 4.3a). These sutures are passed bluntly through the intercostal spaces by reversing the large needle to avoid damaging the intercostal vessels and internal thoracic structures (Figure 4.3 inset). Once the circumcostal sutures are all placed, an assistant approximates the ribs while the surgeon ties the adjacent suture. The serratus ventralis, scalenus or external abdominal oblique, and pectoralis muscles are closed in a single layer with a continuous suture pattern (Figure 4.3b). The latissimus dorsi muscle and associated tissues are then closed with a second continuous suture pattern. The subcutaneous tissues and cutaneous trunci muscle are closed together in a separate layer. Lastly, the skin is closed in standard fashion. After the thoracotomy is closed, the thoracostomy tube is used to evacuate air and fluid from the thoracic cavity.

Postoperative Management

The immediate recovery period is the most critical period after thoracic surgery. Monitoring and management of the pleural space is the most important issue after thoracotomy. The pleural space should be evacuated at least hourly for the first few hours after surgery. If the thoracostomy tube is nonproductive for air or fluid, then the tube can be pulled a few hours after surgery. If pneumothorax or fluid production persists after surgery, then it is prudent not to remove the tube until the thoracic space becomes nonproductive.

Ventilation may be depressed after thoracic surgery by anesthetic drugs, air or fluid in the pleural space, tight thoracic bandages, or somatic pain arising

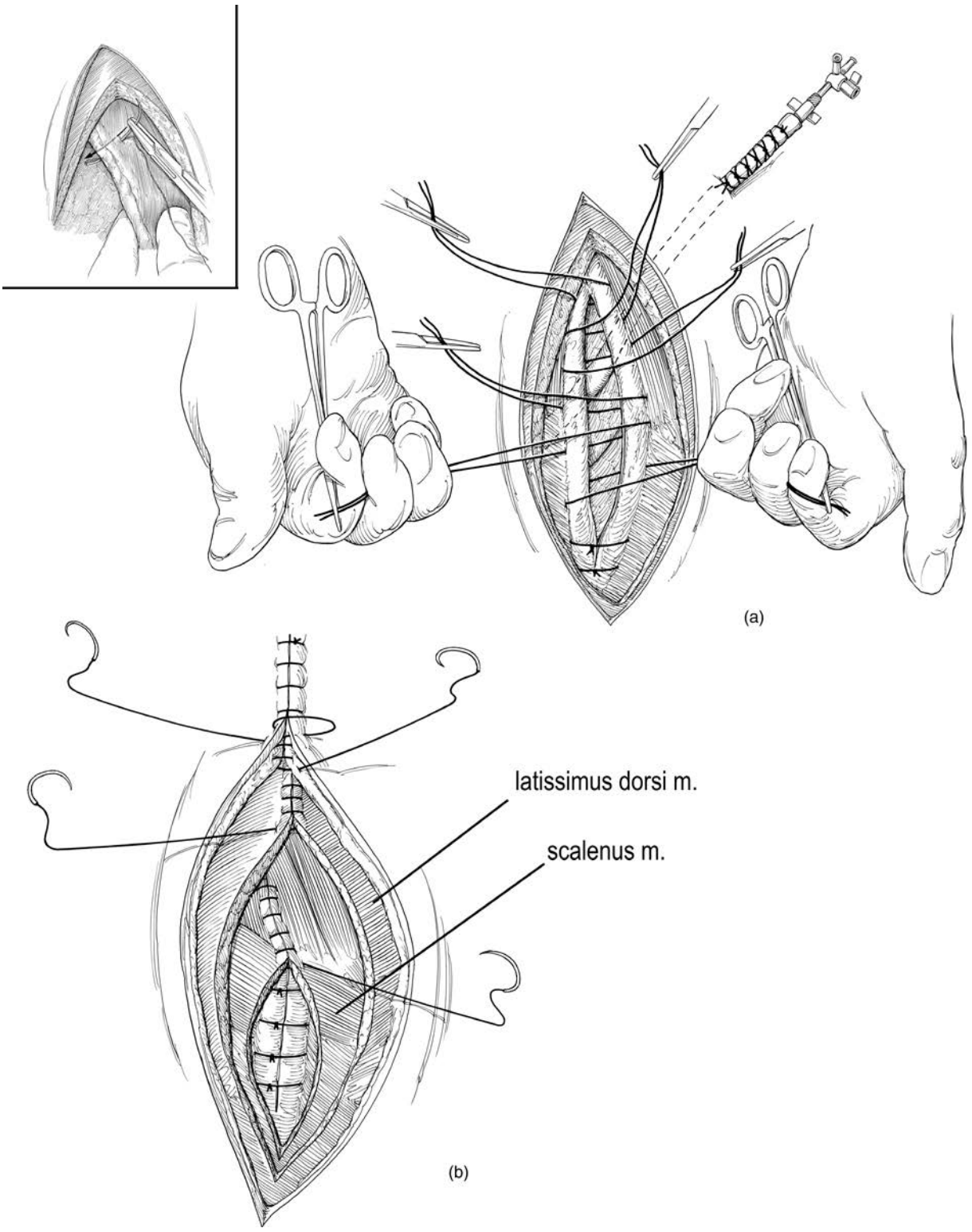

Figure 4.3 Thoracotomy Closure

from the thorax wall. Hypoventilation contributes to hypoxemia and acidosis, neither of which is well tolerated by the postoperative patient. Tidal volumes less than 10 mL/kg measured with a Wright's respirometer suggest that inadequate ventilation may be present. Hypoventilation in the recovery period is confirmed by a $P_aCO_2 > 50$ mm Hg. Hypoventilation should be managed by correcting its underlying cause(s) rather than administration of supplemental oxygen.

Hypothermic animals should be slowly surface-warmed with warm-water bottles or circulating water blankets during and after surgery. Hypovolemia, myocardial depression, and residual anesthetic drugs are the most common causes of circulatory compromise during the recovery period. Appropriate supportive care to correct hypovolemia is essential. The choice of fluid therapy should be based on the underlying cause of hypovolemia. Whole blood or packed RBCs should be administered if necessary to keep the hematocrit above 25%. The patient should be evaluated for acid-base and electrolyte disorders, and these should be corrected. Urine production should be monitored in critical cases to assure adequate renal function after surgery. Thoracic bandages may aid in sealing the thoracotomy incision, but should be placed loosely so as not to restrict ventilation.

Analgesia is indicated in all thoracic surgery patients. Ideally, an analgesic protocol after thoracic surgery should provide effective analgesia while minimizing effects on ventilation. A variety of analgesic drugs and administration techniques have been advocated for small animals after thoracic surgery, including parenteral opioids, epidural opioids, selective local anesthetic nerve blocks, or intrapleural local anesthesia. Usually combinations of the above techniques provide the most effective analgesia while minimizing the adverse effects on ventilation. Continuous rate infusion of fentanyl (1 to 5 μg/kg/h) provides effective analgesia and has the advantage of allowing rapid titration to balance the analgesic effect and minimize excessive hypoventilation. Epidural administration of morphine or oxymorphone in dogs after thoracotomy has been shown to both prolong the drug effect for up to 10 hours and decrease the risk for hypoventilation [1–3]. Selective intercostal nerve blocks with bupivacaine provide up to 12 hours of analgesia and avoid some of the undesirable side effects of high-dose parenteral opioids [1, 4–6]. The nerve block is performed prior to closure of an intercostal thoracotomy and should include the intercostal space used for thoracotomy and two intercostal spaces cranial and caudal to the thoracotomy site. The total amount of bupivacaine used to perform the block at all sites should not exceed 5 mg/kg. A disadvantage of nerve blocks is that it is difficult to repeat the block after its effect has worn off. While intercostal nerve blocks have been advocated as a sole method for analgesia after thoracotomy, its best use is probably as an adjutant to parenteral opioid therapy. Intrapleural administration of bupivacaine has been advocated as an alternative to intercostal nerve block, particularly after median sternotomy [6–9]. Its advantages include ease of administration, ability to readminister, minimal effects on ventilation, and 6- to 12-hour duration. The mechanism of action is thought to be blockage of the spinal nerve roots as they enter the spinal cord. As a result, placement of the animal in dorsal recumbency for 5 minutes after administration is thought to enhance its effectiveness. Bupivacaine (1.5 to 3 mg/kg) can be administered intrapleurally through the thoracostomy tube prior to recovery from anesthesia. Intrapleural administration of bupivacaine is painful and should be preceded by administration of intrapleural administration of lidocaine (1mg/kg) in awake animals. Thoracotomy per se is associated with a low complication rate, with severe complications being more likely to arise from the underlying thoracic pathology [10].

References

1 Pascoe PJ, Dyson DH. 1993. Analgesia after lateral thoracotomy in dogs: Epidural morphine versus intercostal bupivacaine. *Veterinary Surgery* 22: 141–47.

2 Popilskis S, Kohn D, Sanchez JA, Gorman P. 1991. Epidural vs. intramuscular oxymorphone analgesia after thoracotomy in dogs. *Veterinary Surgery* 20: 462–67.

3 Carregaro AB, Freitas GC, Lopes C, Lukarsewski R, Tamiozzo FS and Santos RR. 2014. Evaluation of analgesic and physiologic effects of epidural morphine administered at a thoracic or lumbar level in dogs undergoing thoracotomy. *Veterinary Anaesthesia and Analgelsia* 41(2): 205–11.

4 Berg RJ, Orton EC. 1986. Pulmonary function in dogs following intercostal thoracotomy: a comparison of morphine, oxymorphone and selective intercostal nerve block. *American Journal of Veterinary Research* 47:471–74.

5 Flecknell PA, Kirk AJ, Liles JH, Hayes PH, Dark JH. 1991. Post-operative analgesia following thoracotomy in the dog: an evaluation of the effects

of bupivacaine intercostal nerve block and nalbuphine on respiratory function. *Laboratory Animal* 25:319–24.

6 Thompson SE, Johnson JM (1991). Analgesia in dogs after thoracotomy: a comparison of morphine, selective intercostal nerve block, and intrapleural regional analgesia with bupivacaine. *Veterinary Surgery* 20:73–77.

7 Conzemius MG, Brockman DJ, King LG, Perkowski SZ. 1994. Analgesia in dogs after intercostal thoracotomy: A clinical trial comparing intravenous buprenorphine and intrapleural bupivacaine. *Veterinary Surgery* 23:291–98.

8 Dhokarikar P, Caywood DD, Stobie D, Raffe MR, Albrecht M, Kannan M, King V, Hegstad B, Randall D. 1996. Effects of intramuscular or intrapleural administration of morphine and intrapleural administration of bupivacaine in dogs that have undergone median sternotomy. *American Journal of Veterinary Research* 57:375–80.

9 Stobie D, Caywood DD, Rozanski EA, Bing DR, Dhokarikar P, Raffe MR, Kannan MS, King VL, Hegstad RL, Randall DA. 1995. Evaluation of pulmonary function and analgesia in dogs after intercostal thoracotomy and use of morphine administered intramuscularly or intrapleurally and bupivacaine administered intrapleurally. *American Journal of Veterinary Research* 56:1098–1109.

10 Moores, AL, Halfacree ZJ, Baines SJ and Lipscomb VJ. 2007. Indications, outcomes and complications following lateral thoracotomy in dogs and cats. *Journal of Small Animal Practice* 48(12): 695–98.

5

Sternotomy

E. Christopher Orton

Median sternotomy is the only thoracic approach that provides access to the entire thoracic cavity, and is therefore the approach of choice when exploratory surgery of the thorax is indicated. Surgeons should not avoid this approach in small animals because of a perception that it is associated with excessive postoperative pain or complications [1]. Structures in the dorsal thoracic cavity such as the great vessels, bronchial hilus, and thoracic duct are more difficult, but not impossible, to access in large, deep-chested dogs when using this approach.

Median sternotomy is performed with the patient in dorsal recumbency. The skin and subcutaneous tissues are incised on the ventral midline over the sternum (Figure 5.1a). The pectoral musculature is incised and slightly elevated to expose the midline of the sternebrae. The sternum is opened on its midline with an oscillating bone saw, taking care not to injure underlying thoracic structures (Figure 5.1b). Exposure is gained by placement of a Finochietto retractor (Figure 5.1c). Depending on the type and degree of exposure of the thoracic cavity needed, it is highly desirable to leave either the manubrium or xiphoid, or both, intact to increase stability of the sternotomy after closure. Median sternotomy may be combined with a ventral midline celiotomy if a combined approach to the thoracic and abdominal cavities is needed (Figure 5.2a). Similarly, median sternotomy may be extended into the cervical region by combining a sternotomy with a ventral midline cervical approach (Figure 5.2b).

Prior to closure of a sternotomy, a thoracostomy tube is introduced into the thoracic cavity from a subcostal position lateral to midline (Figure 5.3a). Subcostal placement is considered less painful than intercostal placement. Stable closure of the sternotomy is important to decrease postoperative pain, risk for pneumothorax, or delayed union of the sternum. Figure-of-eight orthopedic wires are passed around each sternebrae, incorporating a costosternal junction within the figure-of-eight (Figure 5.3b). In small dogs and cats, heavy-gauge monofilament suture can be used in lieu of wire to close the sternotomy. The pectoral muscles, subcutaneous tissues, and skin are closed in separate layers, using separate continuous suture patterns.

Postoperative Care

Postoperative care is essentially the same as outlined for a thoracotomy in Chapter 4. Analgesia is an important aspect of management after sternotomy. The wound and thoracostomy site should be protected from contamination by placement of self-adhering bandages.

Reference

1 Ringwald RJ, Birchard SJ. 1989. Complications of median sternotomy in the dog and literature review. *Journal American Animal Hospital Association* 25:430.

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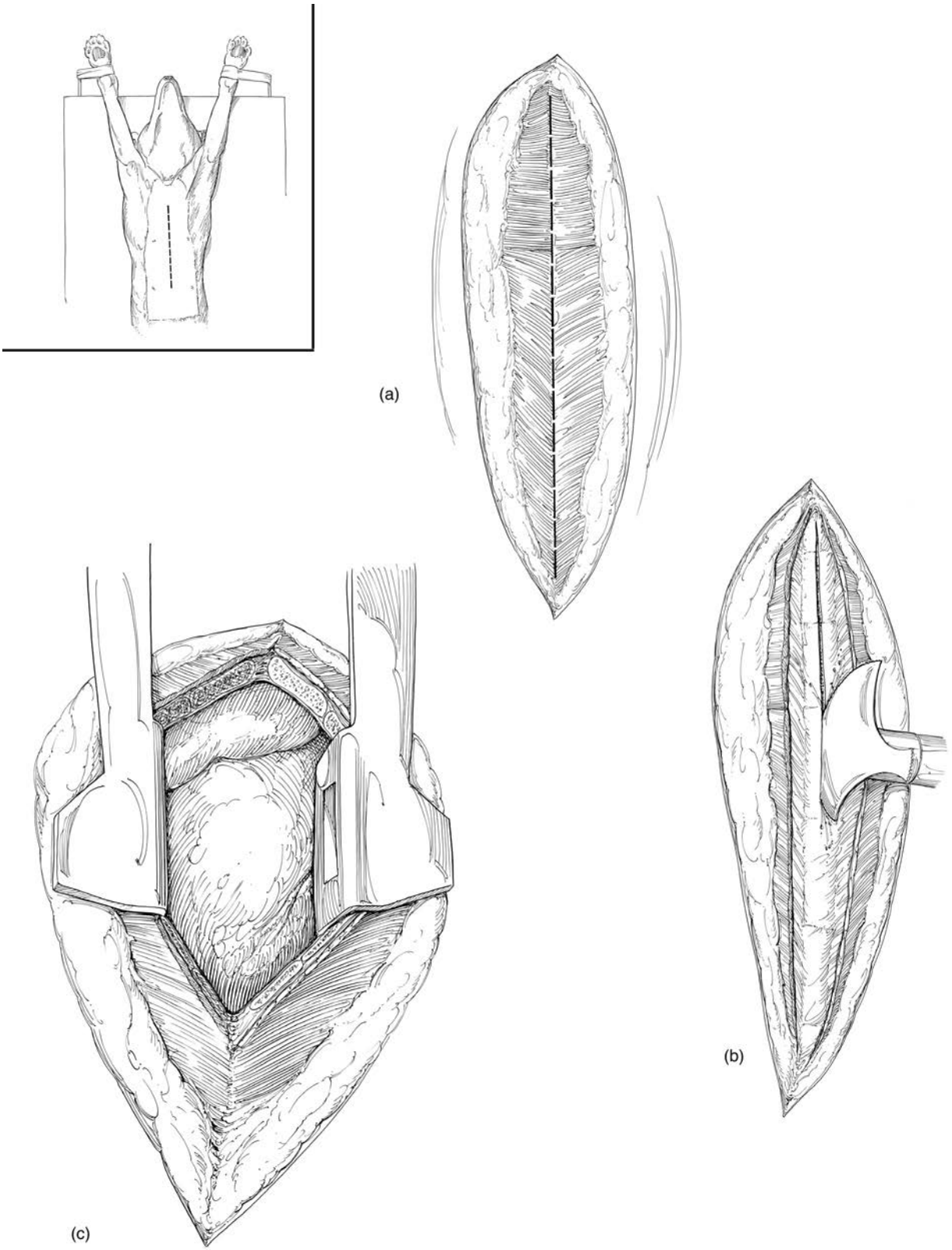

Figure 5.1 Sternotomy

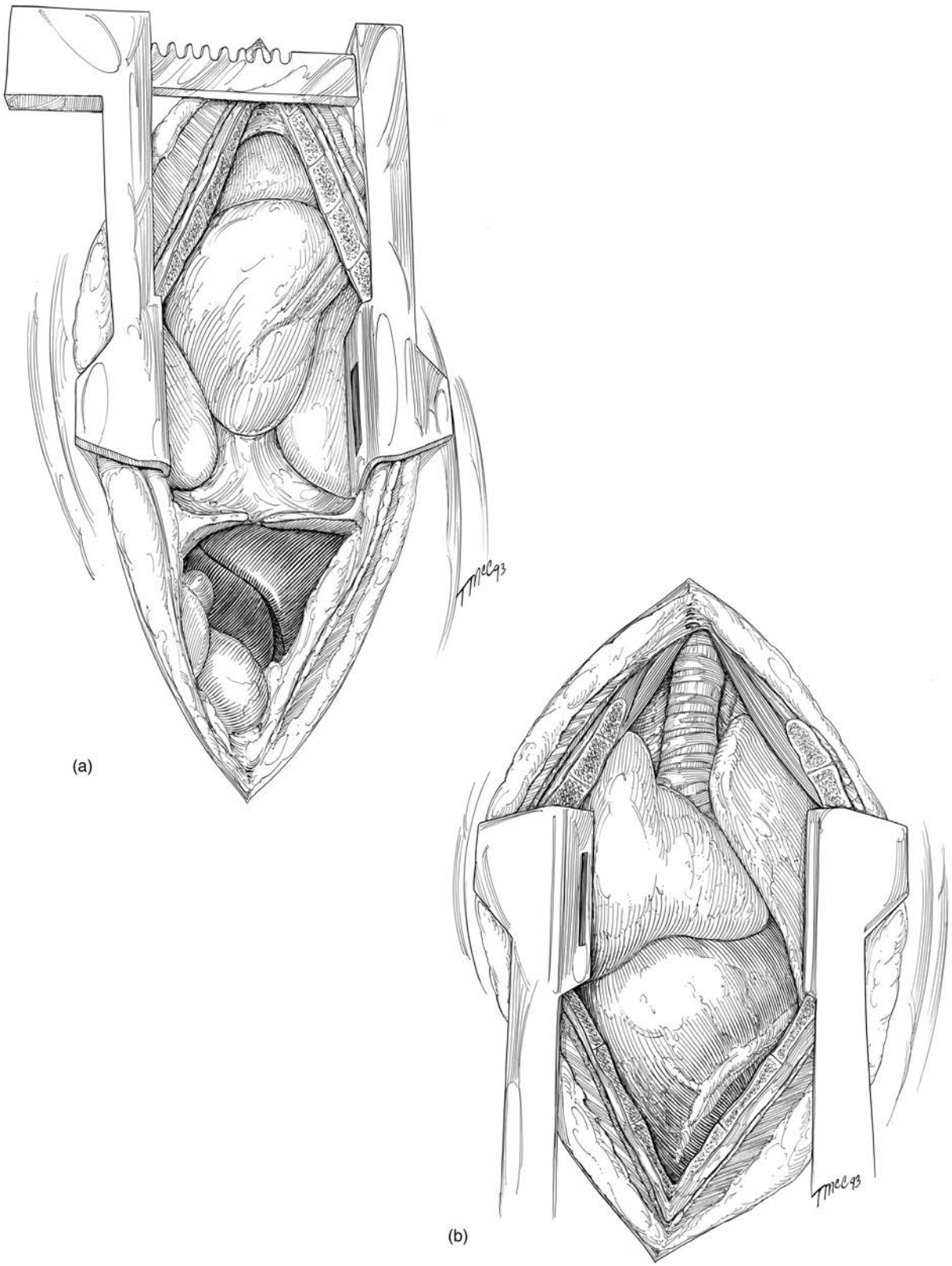

Figure 5.2 Extension of Sternotomy Approach

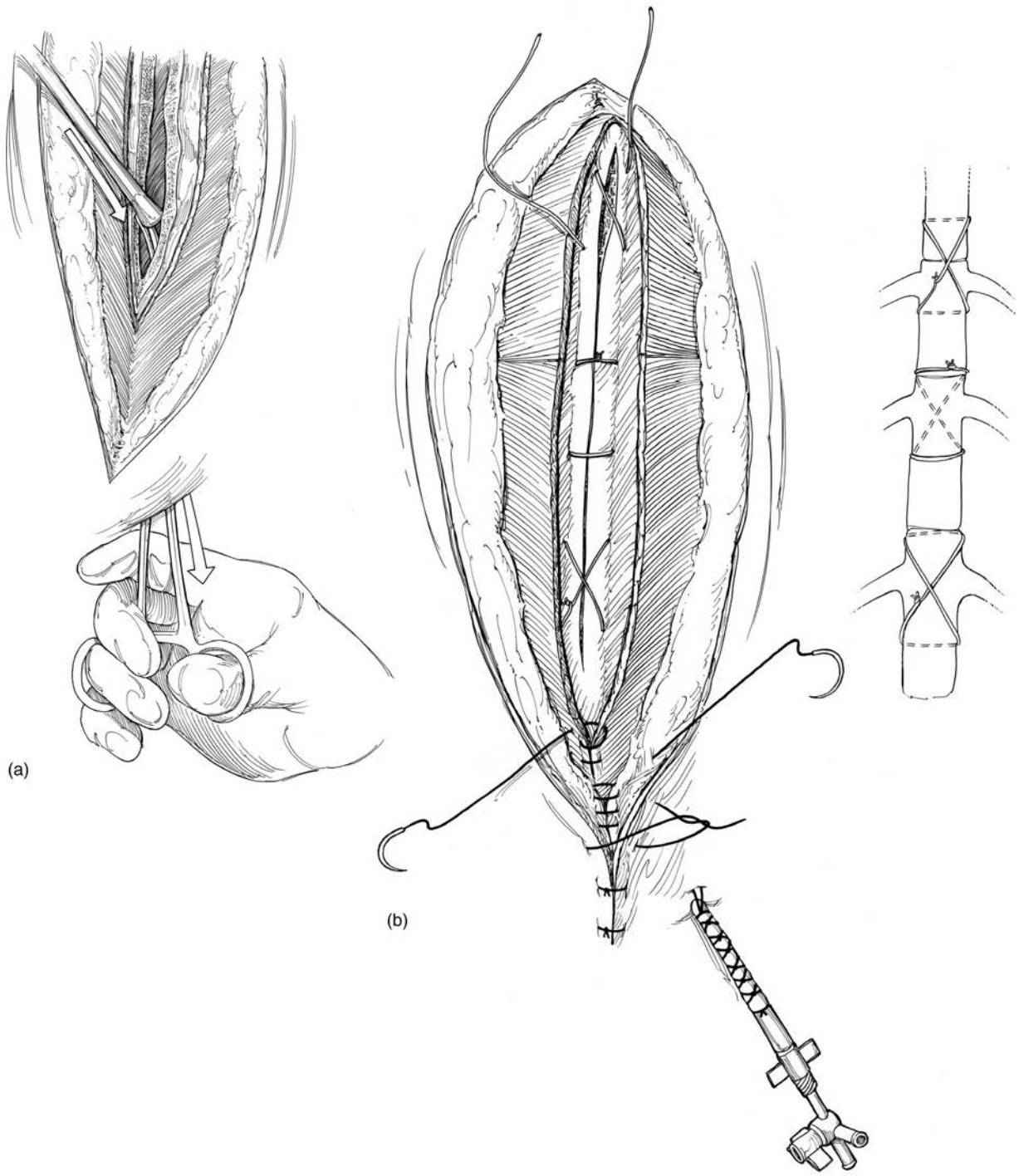

Figure 5.3 Closure of Sternotomy

6

Minimally Invasive Thoracic Surgery

Eric Monnet and E. Christopher Orton

Minimally invasive surgery (MIS) encompasses a variety of surgical techniques, all intended to minimize the size of surgical incisions in an effort to reduce surgical trauma, decrease post-operative pain, and hasten recovery of the patient. MIS strategies include limited- or minimal-incision surgery, endoscopic or video-assisted surgery, and image-guided surgery/interventions. Each utilizes specialized equipment and techniques in an effort to reduce the invasiveness of surgery. All of these strategies, or combinations of these, have established or emerging applications in veterinary thoracic surgery.

Minimal-Incision Thoracotomy

Limited- or minimal-incision surgery limits incisional size and retraction to the minimum necessary to accomplish focal access to a defined region or structure. This can be a stand-alone approach to accomplish procedures like pericardial window, or it can be combined with other MIS strategies such as video-assisted surgery or image-guided interventions. An example is a left minimal-incision thoracotomy over the cardiac apex. This approach can be used to accomplish defined surgical procedures such as pericardial window or implantation of an epicardial pacing lead. Another application is to provide an access point for image-guided interventions to the left side of the heart. In this application controlling purse-string sutures are placed in the cardiac apex to allow introduction catheter access for intracardiac interventions under fluoroscopic and/or echocardiographic guidance. This approach is an example in the rapidly emerging field of cardiac surgery known as hybrid cardiac surgery. These procedures are performed in an imaging-capable operating room known as a hybrid OR.

Minimal-incision thoracotomy to the cardiac apex is accomplished on the left side in the ventral third of the eighth to tenth intercostal space, depending on cardiac size. The location of the cardiac apex is confirmed by thoracic radiography or fluoroscopy before surgery. An incision is made in the skin, subcutaneous tissues, and external abdominal oblique muscle (Figure 6.1a). The intercostal muscles are incised and a small retractor is placed to expose the apical pericardium (Figure 6.1b). The retractor is applied from the ventral direction to avoid interference with cardiac imaging. The pericardium is opened and sutured to the incision to expose the cardiac apex.

Video-Assisted Thoracoscopic Surgery (VATS)

Endoscopic surgery in the thorax is termed *video-assisted thoracic surgery* or *video-assisted thoracoscopic surgery*. Video-assisted thoracic surgery combines endoscopic visualization with a limited- or minimal-incision thoracotomy to perform the surgery, whereas video-assisted thoracoscopic surgery utilizes multiple specialized portals to gain access to the thoracic space and specialized instruments to perform the surgery. The acronym *VATS* has been applied to both approaches. Currently, the latter approach is used more often in veterinary surgery. Video-assisted thoracoscopic surgery has been used to explore the pleural space, collect biopsies of different organs, and perform advanced surgical procedures in dogs and cats [1–9].

Instrumentation

Video-assisted thoracoscopic surgery utilizes a rigid endoscope, video camera, and cable (Figure 6.2). A tower with light source, high-definition monitors, and radiofrequency energy source are also required (Figure 6.3). An insufflator is not required since

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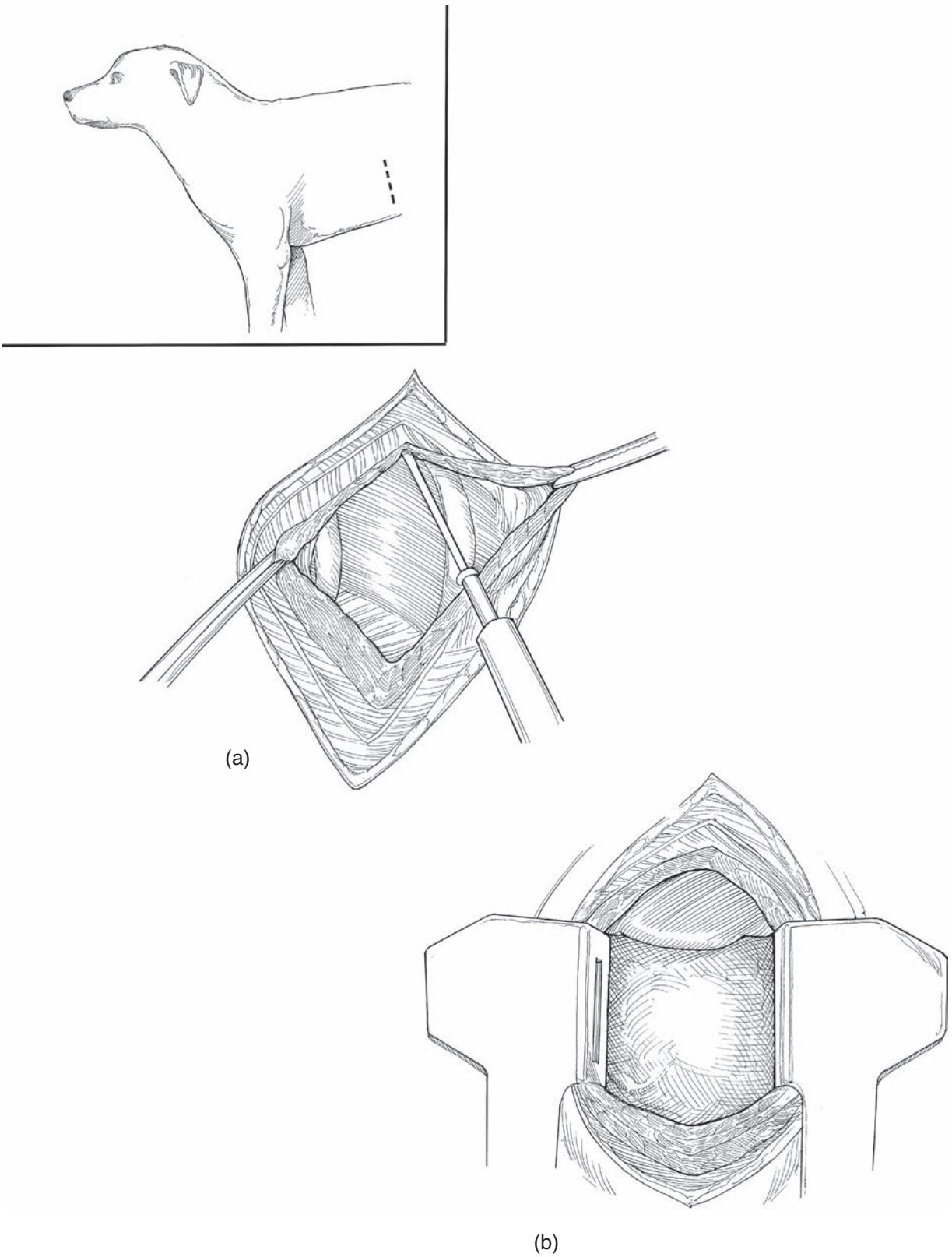

Figure 6.1 Minimal-Incision Thoracotomy

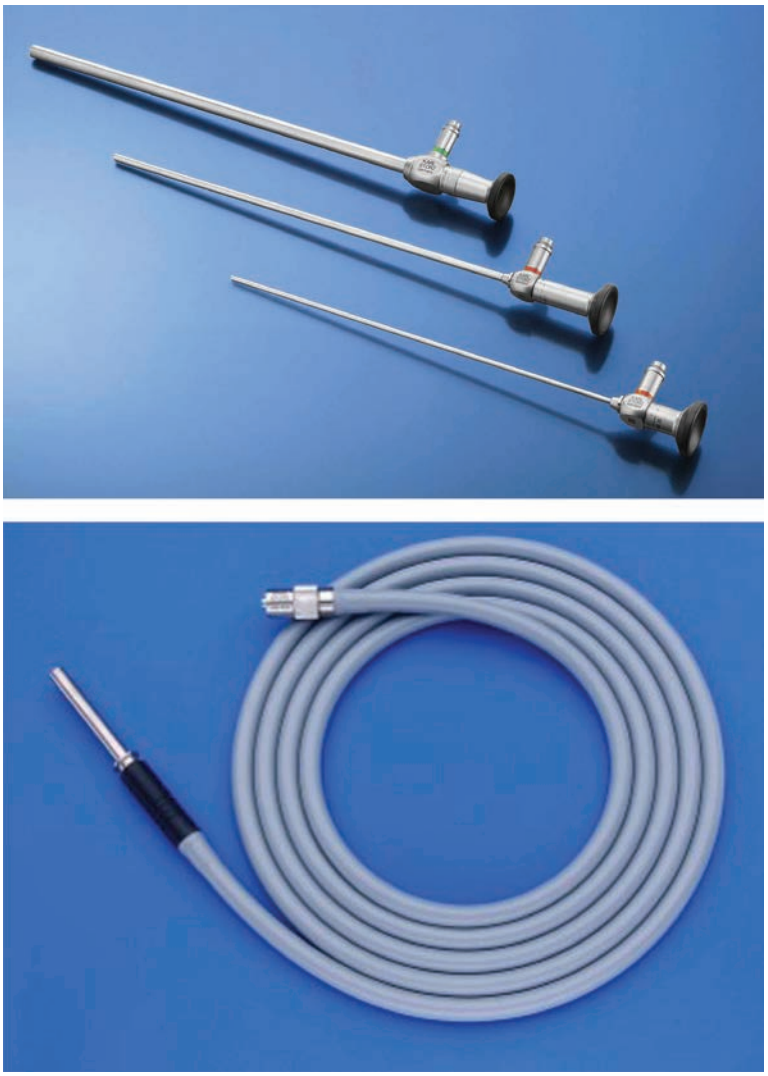

Figure 6.2 Endoscopes and Cable (© KARL STORZ GmbH & Co. KG)

insufflation of carbon dioxide into the pleural space is not recommended or required in animals.

Endoscopic cameras exist in different configurations. High-definition cameras are currently used by most surgeons and are becoming standard. They provide images in a 1080p compatible with high-definition monitors. This technology provides better depth perception, better recognition of anatomical landmarks and instruments, and less surgeon fatigue compared to the previous 16:9 format. Digital cameras also provide very good quality images. Flat-screen high-definition monitors are required to visualize images from the camera. Two monitors are ideal because they allow the surgeon and the assistant to have a forward view on each side of the surgery table.

Current systems use xenon light sources, which offer excellent color reproduction. High-definition cameras require abundant light; therefore, a 300 W light source is needed, especially when thoracoscopy is performed in a deep-chested dog. Blood in the surgical field absorbs light, and this is another reason why a 300 W light source is recommended. Light-emitting diode (LED) lights are increasingly used in video-assisted endoscopic surgery. They provide good color reproduction and do not generate heat. Light sources are connected to the endoscope by a light cable. Light transmitting cables exist in different diameters. The larger the diameter, the more light transmitted to the endoscope. Light cables are made of multiple fibers. When more than one-third of the fibers are broken, the quantity of light transmitted is significantly affected.

Rigid endoscopes are used in thoracoscopic surgery. They conduct light to the pleural space and images from the pleural space to the camera head. Endoscopes come in diameters from 2.4 to 10 mm. Larger endoscopes transmit a wider field image and more light to the pleural space. A 5 mm rigid endoscope is

Figure 6.3 Endoscopic Tower (© KARL STORZ GmbH & Co. KG)

adequate to perform thoracoscopy in dogs. An endoscope that is 30 cm in length can be used with any size of patient. Pediatric endoscopes with a 2.4 mm diameter have application in small breed dogs, young dogs, and cats. Rigid endoscopes also come with different angles of view. Angled-view endoscopes allow visualization around structures and masses. Endoscopes with 0° and 30° angles are most commonly used during thoracoscopy (Figure 6.4). Higher angles of view are difficult to use because they look backward. Endoscopes with variable angles are available, but only in 10 mm diameter.

Video-assisted thoracoscopic surgery utilizes ports or cannulae to gain access to the pleural space without damaging the intercostal neurovascular bundle (Figure 6.5). Thoracoscopic ports do not require valves since insufflation is not used. Access ports can be soft or rigid. Soft or compressible ports place less pressure on intercostal tissues. Ports are usually threaded to prevent displacement during the procedure. Ports exist in different diameters from 5.5 mm to 15 mm. The size used depends on the size of the patient and the equipment that will be used. A 5.5 mm and 11.5 mm port will be most appropriate for dogs. Stapling equipment usually requires an 11.5 mm port.

Specialized instruments for thoracoscopic surgery include scissors, fine-tooth grasping forceps, atraumatic forceps, and needle holders (Figure 6.6). A fan retractor to retract lung lobes from the field of view is useful and may help avoid the need for one lung

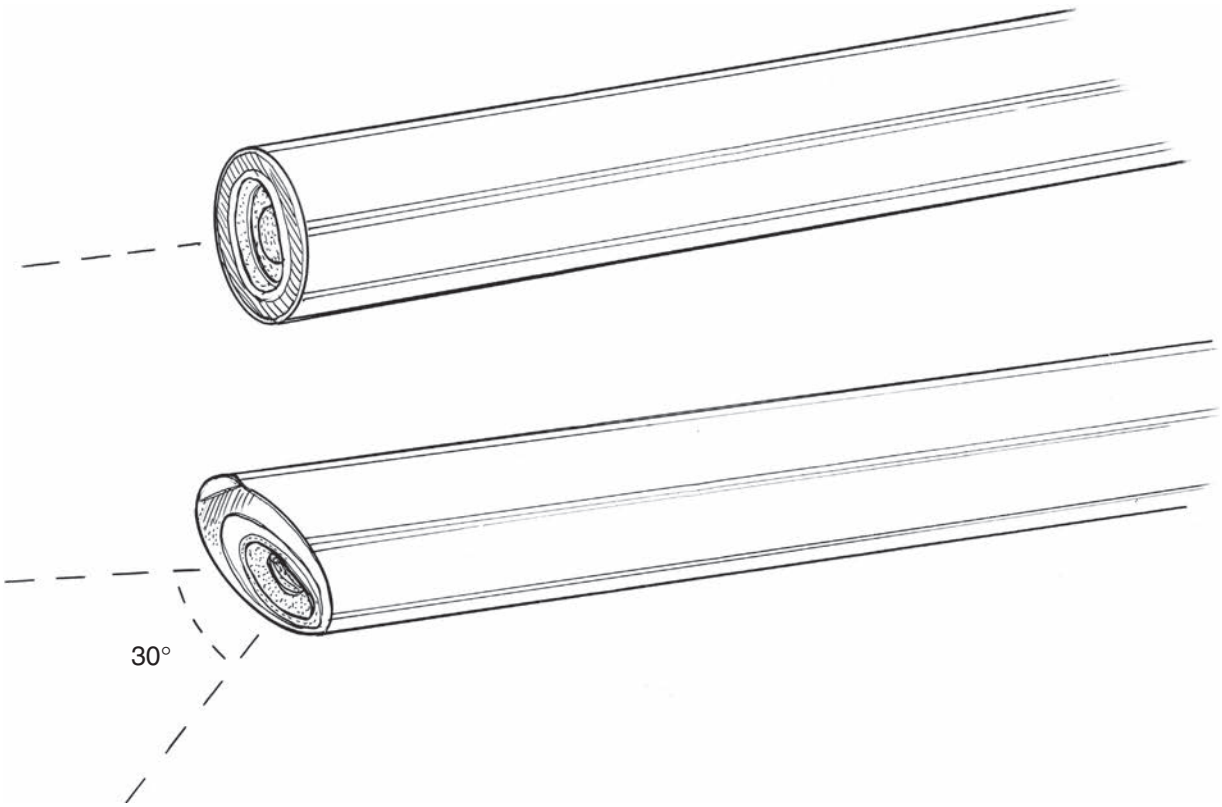

Figure 6.4 0° and 30° Endoscopes

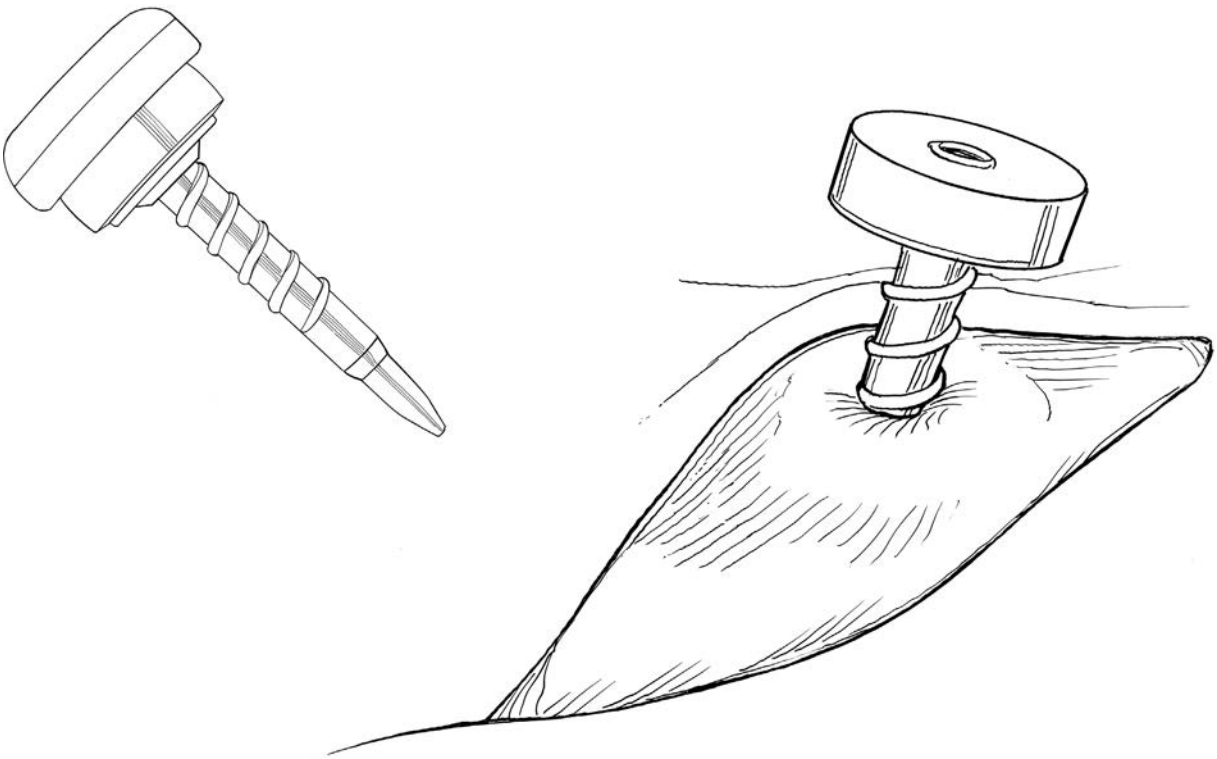

Figure 6.5 Thoracoscopic Ports

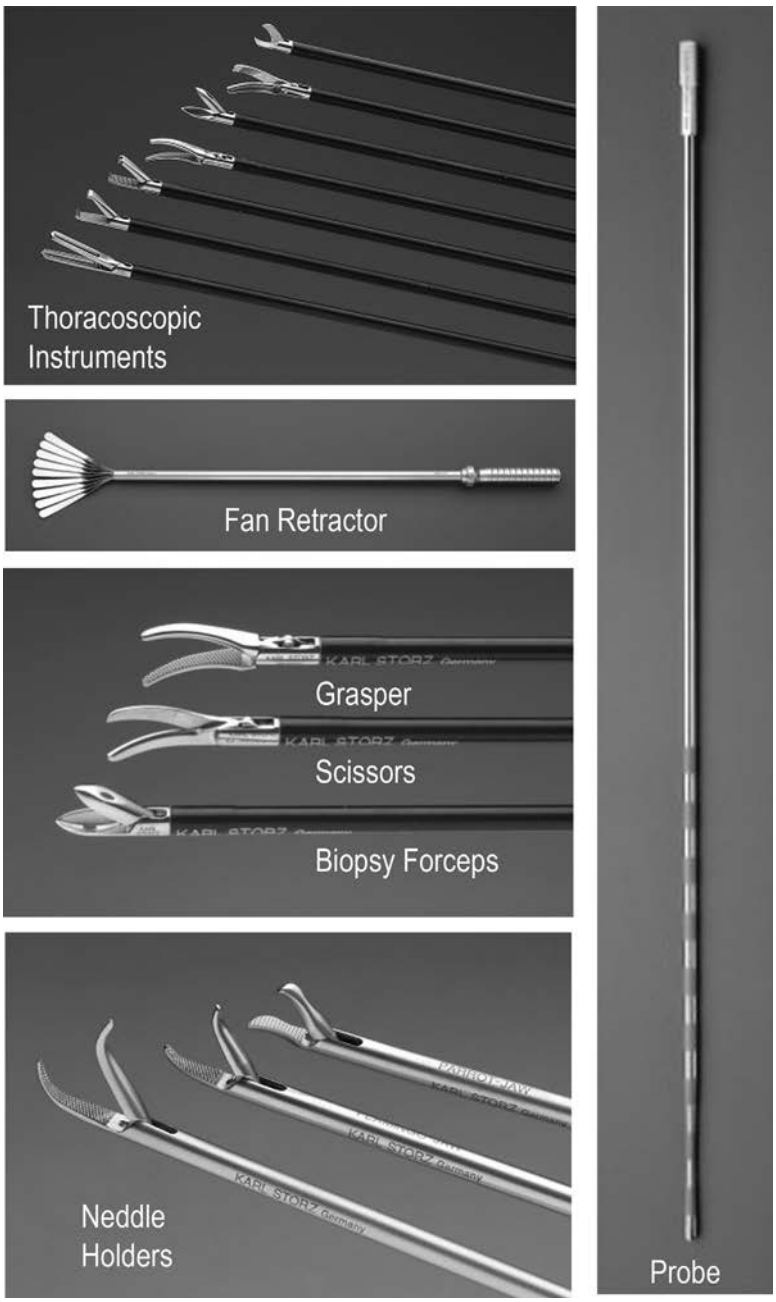

Figure 6.6 Instruments for Thoracoscopic Surgery

ventilation for certain procedures. Suction and irrigation devices to keep the surgical field clear are helpful for procedures such as dissection of cranial mediastinal masses. These instruments can be connected to an electrocautery unit to perform hemostasis. Retrieval bags are used to prevent seeding of tumors to the thoracic wall during retrieval of lung or mediastinal masses through a portal site [10, 11].

Linear stapling devices are available to perform partial or complete lung lobectomy [1, 6, 9, 12]. Staplers exist in different lengths and configurations, depending on the size, thickness, and type of the tissue to be resected (Figure 6.7). Linear staplers typically place six rows of staples and cut between the middle rows of stables. The staples are staggered and close in a B-shape fashion to allow blood supply to the edge of the incision. Newer stapling devices apply staples that provide variable compression of the tissue from the edges to the center of the specimen. This new design allows for better distribution of the compression based on tissue thickness—however, it can be associated with more leakage of air after

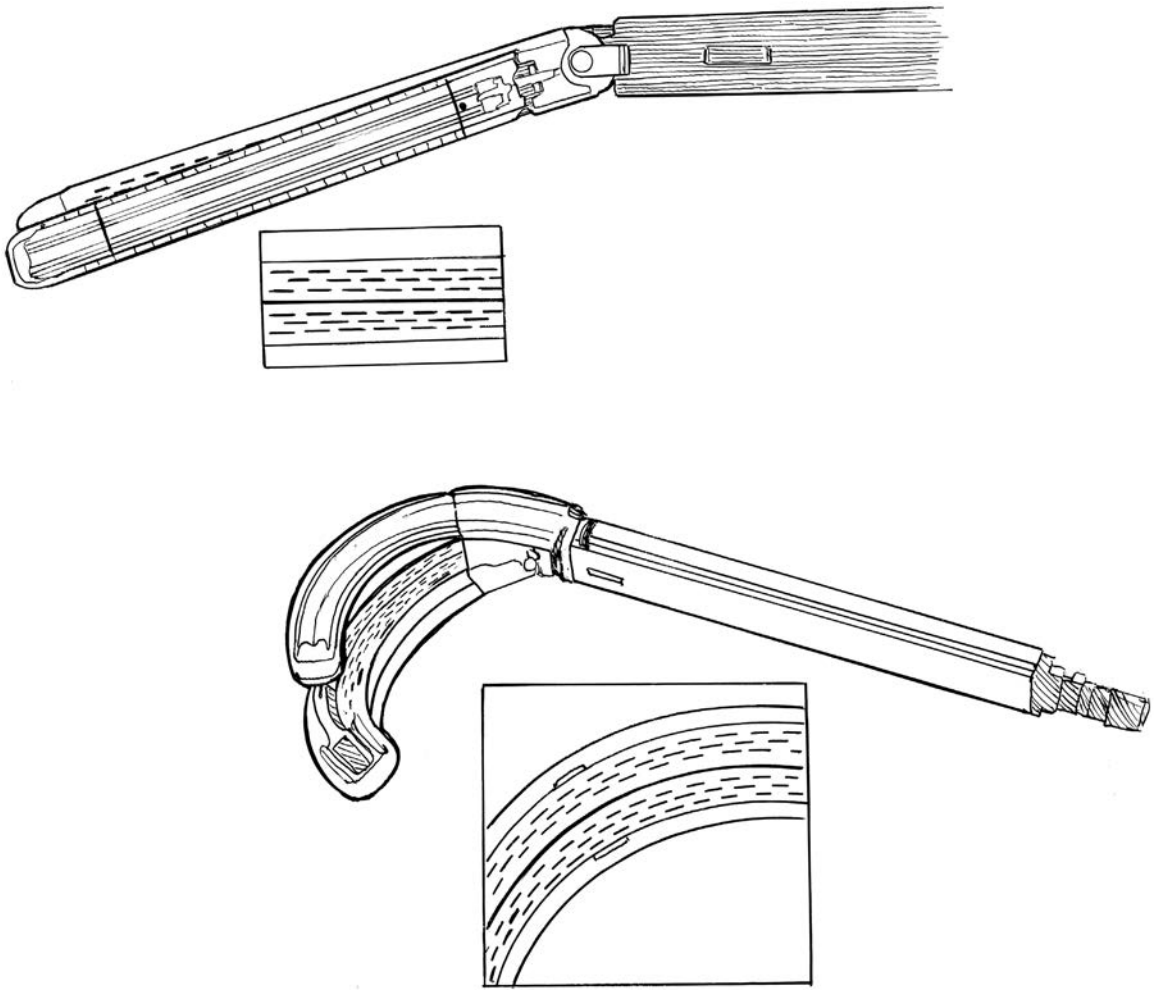

Figure 6.7 Articulating and Curved Endoscopic Stapling Devices

lung lobectomy [12]. Cartridges are usually 30, 45, or 60 mm long. Stable lengths are between 2 and 4 mm. Some staplers have a curved design for application in difficult situations with limited space. Others are capable of articulation to help align placement of the cartridge.

An energy source is needed for thoracoscopic surgery. Modalities can include monopolar electrocautery, energy for tissue fusion devices, and ultrasonic dissectors that provide hemostasis during tissue dissection. Tissue fusion technology or vessel sealant devices utilize bipolar radiofrequency energy to seal blood vessels and lymphatics. These devices measure tissue impedance to deliver the appropriate the amount of energy to achieve a safe seal. Vessel sealant devices are currently approved for sealing vessels up to 7 mm in diameter. Ultrasonic generators deliver sound energy that propagates down the shaft of the instrument causing axial displacement of the instrument tip. Ultrasonic energy devices can seal vessels up to 3 mm in diameter.

General Rules for Thoracoscopic Surgery

Because there is always the possibility for elective or emergent conversion of thoracoscopic to standard surgery, the patient should be clipped and draped for a thoracotomy. Triangulation technique is typically recommended for thoracoscopic surgery. Ports for endoscopic camera and instruments are placed in a triangular configuration around the target organ or lesion. The port for the endoscopic camera should be placed as far as feasible from the surgical field to allow for adjustment of the width of the field of view and magnification if needed. The instrument ports are placed between the camera port and the target organ. Ideally, the camera, target organ, and monitor should be

aligned as close as possible to a straight line to facilitate the surgery.

Thoracoscopic Approaches

An intercostal approach provides good visualization of a limited region of the thorax. Therefore, this approach is used for specific surgical procedures or biopsy of predetermined structures. The transdiaphragmatic subxyphoid approach provides access to both hemithoraces and provides the best opportunity for thorough exploration of the thoracic cavity. However, the dorsal thoracic cavity cannot be well visualized using this approach even with an angled telescope.

Intercostal Approach

Patients are usually placed in lateral recumbency for an intercostal approach. For some procedures, placement in an oblique position with the sternum up allows gravity to shift some organs from the surgical field. Ventral (sternal) recumbency has been used to help exposure for procedures in the dorsal part of the thoracic cavity.

Ports are placed in the third to tenth intercostal space. It is typically best to place ports in the ventral two-thirds of the intercostal space where the ribs are more compliant, facilitating manipulation of the endoscope and instruments (Figure 6.8). A skin incision the diameter of the port is performed with a surgical blade. The subcutaneous tissue and muscle layers are bluntly dissected and the pleural space is penetrated with a curved hemostat forceps. A port is introduced with the blunt trocar. The endoscope is introduced into the first port and then used to visualize placement of additional ports.

After the procedure, a thoracostomy tube is placed through a separate incision. The thoracostomy tube should not be placed through a portal incision because of the risk of air leakage after surgery. Placement of the thoracostomy tube is accomplished under visualization with the endoscope. Ports are removed under visualization with the endoscope to evaluate for bleeding from an intercostal artery, which typically will not bleed until port is removed. If intercostal bleeding occurs, then either electrocautery is applied to the incision or an encircling suture around the rib is placed to control bleeding. Each port site should receive an intercostal block with bupivacaine to assist with analgesia after surgery. Deep muscle layers, subcutaneous tissue, and skin are closed with cruciate sutures in a routine fashion.

Transdiaphragmatic Subxyphoid Approach

The transdiaphragmatic subxyphoid approach allows visualization of both sides of the thoracic cavity (Figure 6.9). Patients are typically placed in dorsal recumbency although it is sometimes useful to place the patient in an oblique position. A long 5 mm port is introduced caudal to the last rib lateral to the xyphoid appendage. The port is directed toward the ipsilateral hemithorax underneath the last rib. The port should not be oriented too vertically to avoid entering the abdominal cavity and directed away from midline to avoid entering the mediastinum.

After the first port is introduced, additional ports are placed in the ninth or tenth right and left intercostal spaces close to the sternum under endoscopic visualization. The mediastinum is dissected from the

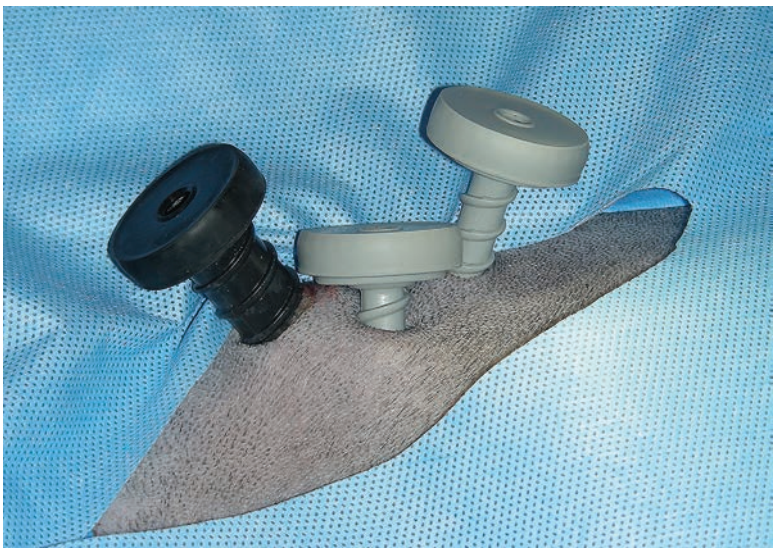

Figure 6.8 Thoracoscopic Approach—Intercostal

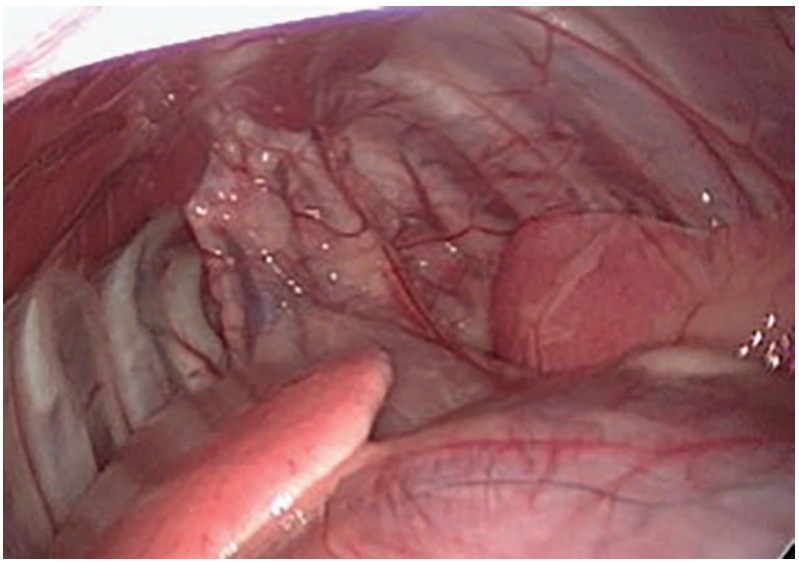

Figure 6.9 Thoracoscopic View—Subxyphoid Approach

sternum with electrocautery or a vessel sealant device. Injury to one or both internal thoracic arteries is a significant hazard during dissection of the mediastinum. A palpation probe can be used to manipulate the mediastinum to help visualize the internal thoracic arteries.

After completion of the surgery, a thoracostomy tube is introduced through a separate incision and ports are removed as described previously. Port sites are closed. The puncture in the diaphragm is not repaired.

Improving Working Space and One-Lung Ventilation

Because access ports are open to atmosphere, patients must be ventilated during thoracoscopic surgery. This can place significant limitations on available working space for surgery. Insufflation with CO_2 is generally avoided because of the risk for tension pneumothorax. If used, insufflation pressures should be < 3 mm Hg to minimize cardiopulmonary compromise. Reduction of ventilator tidal volume improves working space and can be compensated for by increasing the ventilation rate.

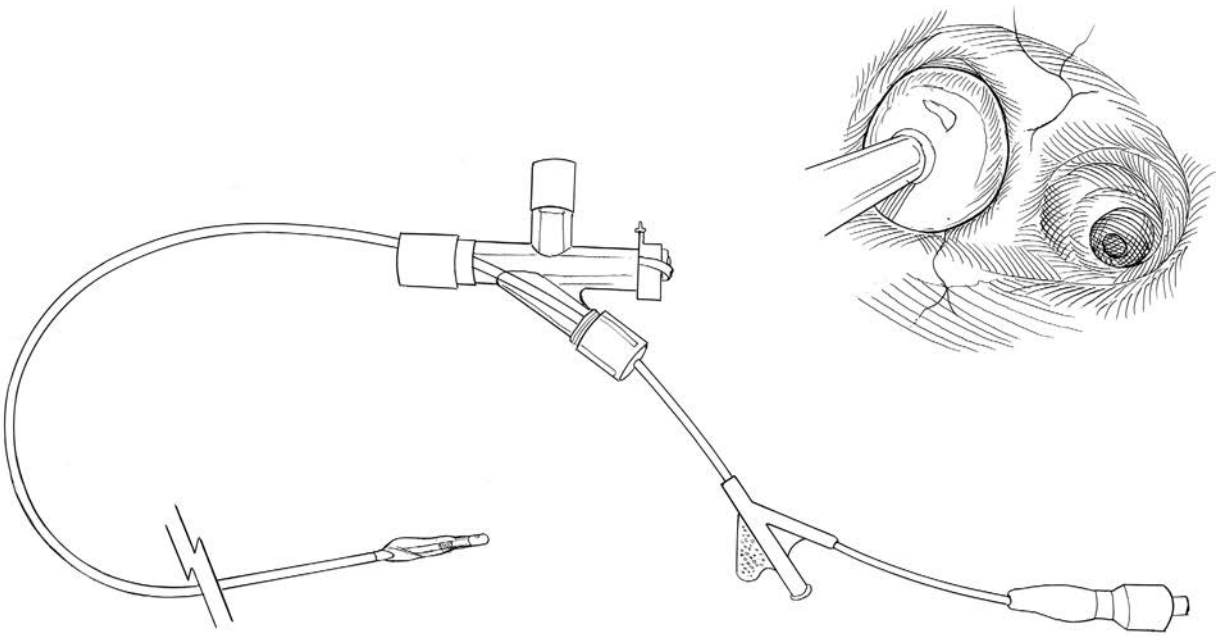

Figure 6.10 Arndt Endobronchial Blocker with Adapter

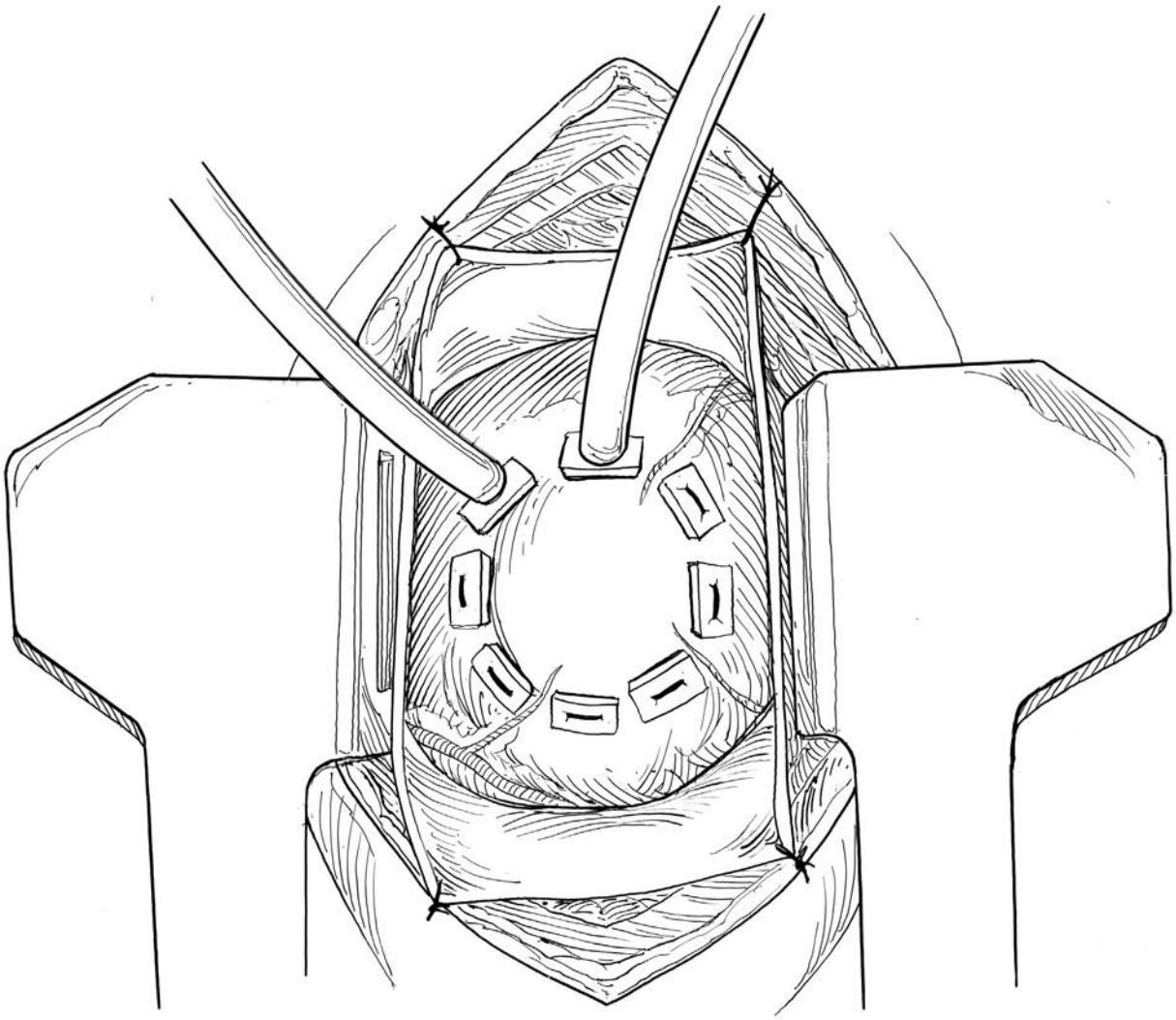

Figure 6.11 Transapical Approach for Hybrid Cardiac Surgery

Certain procedures such as lung lobectomy require a significant amount of working space. In these procedures, one-lung ventilation can tremendously increase available working space and reduce the risk of iatrogenic trauma to the lung parenchyma. One-lung ventilation is accomplished by selective intubation of one bronchus with a long, small endotracheal tube, a double-lumen endotracheal tube, or an endobronchial blocker. Each technique requires bronchoscopy to confirmation correct placement. Selective intubation is not often performed because it requires long, narrow endotracheal tubes that are not readily available. Double-lumen endotracheal tubes are designed for human patients and therefore are not often easy to place in dogs. Double-lumen endotracheal tubes have the advantage of allowing for alternating ventilation from one side to the other. This could be advantageous for procedures such as cranial mediastinal mass removal. Placement of an endobronchial blocker (Arndt endobronchial blocker, Cook, Bloomington, Indiana) is typically the simplest technique and can be applied to different sizes of animals (Figure 6.10). Endobronchial blockers exist in 5, 7, and 9 F sizes with a respective lengths of 50, 65, and 78 cm. Endotracheal tubes cannot be smaller than 4.5, 6, and 7 mm, respectively. Blockers come with an adapter that allows simultaneous delivery of oxygen, and utilization of a flexible endoscope to visualize the placement. The blocker is advanced under bronchoscopic visualization. The bronchoscope is passed within the endotracheal tube into the bronchi that is intended to be blocked. The balloon at the end of the blocker must be well inflated to avoid leak of ventilated gases and resultant overinflation of the lung and barotrauma.

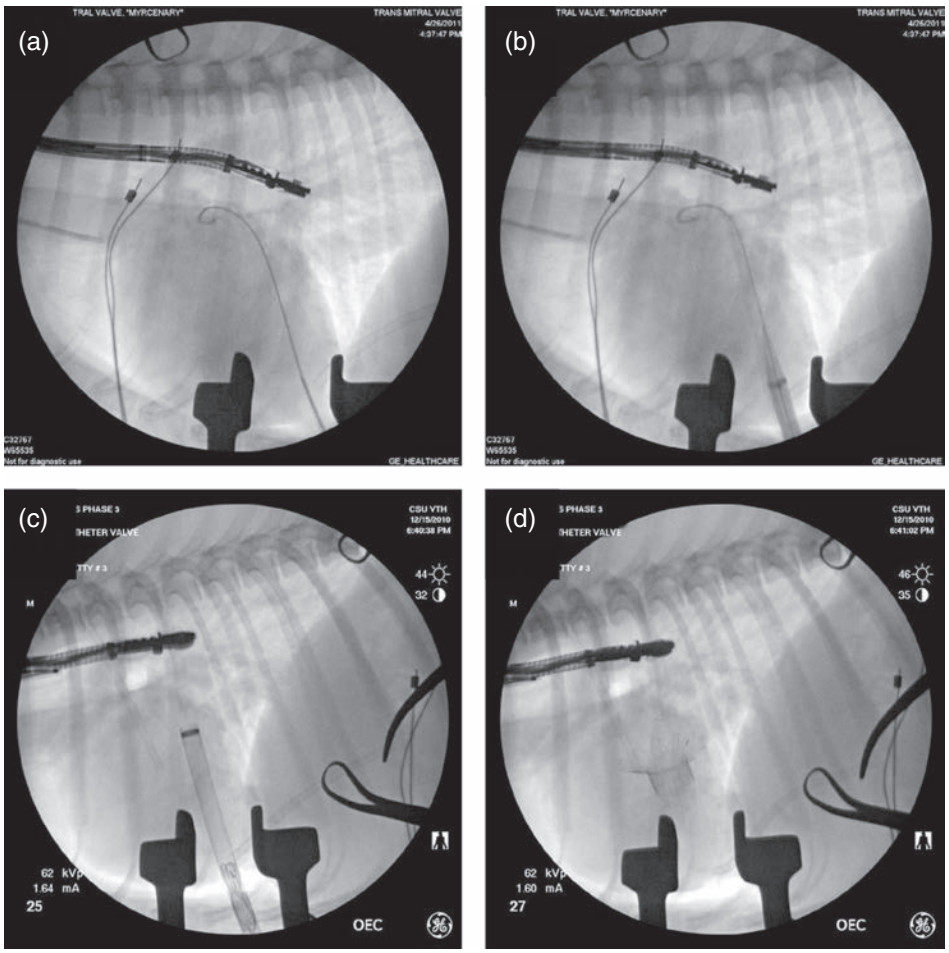

Figure 6.12 Transcatheter Mitral Valve Implantation

One-lung ventilation is initiated at the beginning of the procedure after the patient has been positioned for the surgery. The patient should not be moved after placement of the endobronchial blocker to prevent dislodgment, which can result in complete occlusion of the trachea. A capnograph is used to monitor end-tidal CO_2 ($P_{ET}CO_2$) during one lung ventilation. If the trachea becomes obstructed, $P_{ET}CO_2$ will fall to zero. The blocker should be immediately deflated and returned to the correct position.

Dogs with one-lung ventilation will typically desaturate to a S_aO_2 of 85% to 87% due to induction of pulmonary shunt in the unventilated lung. To mitigate the risk of further desaturation, positive end expiratory pressure (PEEP) is applied to the ventilated lung. PEEP of 2.5 to 5 cm of H_2O supports the ventilated lung with minimal effects on cardiac output [13].

Care after thoracoscopic surgery is the same as for any other thoracic surgery. This includes cardiopulmonary monitoring, management of the thoracostomy tube, and pain control. Animals are usually ambulatory within 24 hours after uncomplicated thoracoscopic surgery.

Image-Guided Interventions

Image-guided interventions are corrective procedures typically performed with catheter-based devices under fluoroscopic and/or ultrasound imaging. Access for these procedures is typically gained through natural orifices or puncture through the wall of hollow organs or vessels. These procedures generally fall under the general categories of *interventional cardiology* for procedures involving the cardiovascular system and *interventional radiology* for procedures on all other systems. Many interventional cardiac procedures can be performed either by "cut down" or "percutaneous" puncture of peripheral vessels such as the femoral artery or vein, jugular vein, or carotid artery. Typically, introducers are placed to gain controlled access to the vessels, and

flow-directed catheters or guidewires are used to direct interventional catheters to the appropriate cardiac site. Current examples of cardiac interventions in small animals include balloon valvuloplasty for pulmonic stenosis and closure of patent ductus arteriosus with a ductal occluder. Examples of thoracic interventional radiology procedures are stent placement for collapsing airway disease or balloon dilation of an esophageal stricture. Because access sites for these procedures are small, these procedures typically can be performed in a catheterization laboratory or procedure room equipped with C-arm fluoroscopy rather than a standard operating room.

Several emerging image-guided cardiac interventions in humans and animals are constrained by either the size or steerability of the interventional catheter or the size of the patent, or both. For these procedures, the interventional catheter can be introduced through a cardiac chamber wall, which provides more direct access to the structure of interest and is less limited by catheter diameter. These procedures are known as *hybrid cardiac surgeries*. Access to the heart for these procedures can be gained through minimally invasive thoracic approaches such as a minimal-incision thoracotomy at the cardiac apex. Access to the left ventricle is gained through two pledget-reinforced pursestring sutures placed in the cardiac apex (Figure 6.11). Introducers, guidewires, and catheters are introduced by direct puncture of the apex. After the procedure the pursestring sutures are tied to control bleeding from the site. An example of a transapical hybrid cardiac procedure is *transcatheter mitral valve implantation*. A guidewire is introduced into the left ventricle and placed across the mitral valve under fluoroscopic and transesophageal echocardiographic guidance (Figure 12a). A delivery catheter is then passed along the guidewire into the left atrium (Figure 12b). A self-expanding valve prosthesis is deployed into the left atrium (Figure 12c) and seated into the mitral annulus (Figure 12d). Examples of other hybrid cardiac procedures in animals have included closure of atrial septal defects, ventricular septal defects, and aorticopulmonary fistula.

The advent of hybrid cardiac and noncardiac interventional surgeries has necessitated the development of *hybrid operating rooms*. These are standard operating suites appropriate for standard and minimally invasive surgery equipped with advanced imaging capabilities to support both video-assisted surgery and image-guided interventions. The hybrid OR can be equipped with sophisticated integration systems that facilitate integration and archiving of the multiple imaging modalities in the room. Many surgeries in humans and animals will likely move increasing toward these minimally invasive modalities in the future.

References

1 Lansdowne JL, Monnet E, Twedt DC, and Dernell WS. 2005. Thoracoscopic lung lobectomy for treatment of lung tumors in dogs. *Veterinary Surgery* 34(5):530–5.

2 Mayhew PD, and Friedberg JS. 2008. Video-assisted thoracoscopic resection of noninvasive thymomas using one-lung ventilation in two dogs. *Veterinary Surgery* 37(8):756–62.

3 Mayhew KN, Mayhew PD, Sorrell-Raschi L, and Brown DC. 2009. Thoracoscopic subphrenic pericardiectomy using double-lumen endobronchial intubation for alternating one-lung ventilation. *Veterinary Surgery* 38(8):961–6.

4 Case JB, Maxwell M, Aman A, and Monnet EL. 2013. Outcome evaluation of a thoracoscopic pericardial window procedure or subtotal pericardectomy via thoracotomy for the treatment of pericardial effusion in dogs. *Journal of the American Veterinary Medical Association* 242(4):493–8.

5 Mayhew PD, Dunn M, and Berent A. 2013. Surgical views: Thoracoscopy: common techniques in small animals. *Compendium Continuing Education for Veterinarians* 35(2):E1.

6 Mayhew PD, Hunt GB, Steffey MA, Culp WT, Mayhew KN, Fuller M, et al. 2013. Evaluation of short-term outcome after lung lobectomy for resection of primary lung tumors via video-assisted thoracoscopic surgery or open thoracotomy in medium- to large-breed dogs. *Journal of the American Veterinary Medical Association* 243(5): 681–8.

7 Skinner OT, Case JB, Ellison GW, and Monnet E. 2014. Pericardioscopic imaging findings in cadaveric dogs: Comparison of an apical pericardial window and sub-phrenic pericardectomy. *Veterinary Surgery* 43(1):45–51.

8 Steffey MA, Daniel L, Mayhew PD, Affolter VK, Soares JH, and Smith A. 2014. Video-assisted thoracoscopic extirpation of the tracheobronchial lymph nodes in dogs. *Veterinary Surgery* 44 (Suppl 1):50–8.

9 Bleakley S, Duncan CG, and Monnet E. 2015. Thoracoscopic lung lobectomy for primary lung tumors in 13 dogs. *Veterinary Surgery* 44(8): 1029–35.

10 Brisson BA, Reggeti F, and Bienzle D. 2006. Portal site metastasis of invasive mesothelioma after diagnostic thoracoscopy in a dog. *Journal of the American Veterinary Medical Association* 229(6): 980–3.

11 Alwen SG, Culp WT, Szivek A, Mayhew PD, and Eckstrand CD. 2015. Portal site metastasis after thoracoscopic resection of a cranial mediastinal mass in a dog. *Journal of the American Veterinary Medical Association* 247(7):793–800.

12 Imhoff DJ, and Monnet E. 2016. Inflation pressures for ex vivo lung biopsies after application of graduated compression staples. *Veterinary Surgery* 45(1):79–82.

13 Kudnig ST, Monnet E, Riquelme M, Gaynor JS, Corliss D, and Salman MD. 2006. Effect of positive end-expiratory pressure on oxygen delivery during 1-lung ventilation for thoracoscopy in normal dogs. *Veterinary Surgery* 35(6):534–42.

Section III

Thoracic Wall and Pleural Space

7

Thoracostomy and Pleural Drainage

Eric Monnet

Drainage of the pleural space is required when air or fluid is present in the pleural space and is interfering with lung expansion. *Thoracocentesis* is performed to provide temporary relief and to collect fluid for diagnostic evaluation. *Thoracostomy tubes* are used for short- to intermediate-term pleural drainage. Drainage systems can implanted under the skin for cases that require chronic pleural drainage.

Analysis of fluid is important to establish a diagnosis and possible etiology for the effusion. Analysis should include bacterial culture and sensitivity, cytology, and biochemical analysis. Pleural effusions can be transudative, nonseptic exudative, septic, chylous, hemorrhagic, or neoplastic effusion. Thoracocentesis improves diagnosis and treatment in 56% of human patients admitted to a critical care unit [1]. Empirical antibiotic therapy has been demonstrated to be inadequate for management of suspected pyothorax; therefore, a culture and sensitivity is required for these cases [2].

Thoracocentesis

Thoracocentesis is typically performed through the lateral thoracic wall with the animal standing or in lateral recumbency. A needle or over-the-needle catheter can be used. The catheter technique is safer but is more susceptible to kinking when larger volumes are removed. The pleural space is entered at an oblique angle with the bevel of the needle facing the lung (Figure 7.1). Once the pleural space is punctured, the needle is angled to be against the thoracic wall to reduce the risk of puncturing the lung. An extension tube should be used to allow the needle or catheter to be manipulated separately from the syringe. A three-way stopcock facilitates removal of large volumes of air or fluid.

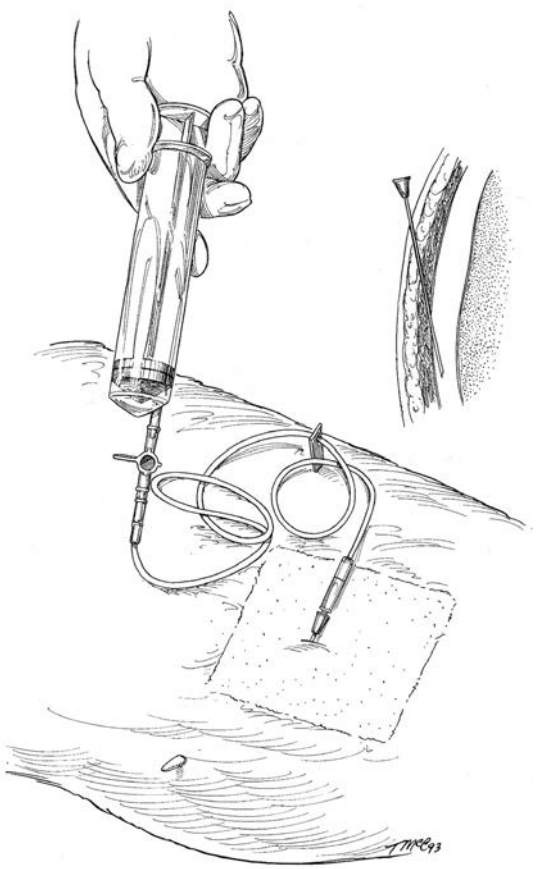

Figure 7.1 Thoracocentesis

Thoracostomy Tubes

Thoracostomy tubes are always placed for short-term management of the pleural space after thoracic surgery. Thoracostomy tubes are also indicated when animals present with large volumes of fluid or air the pleural space or when intermittent thoracocentesis

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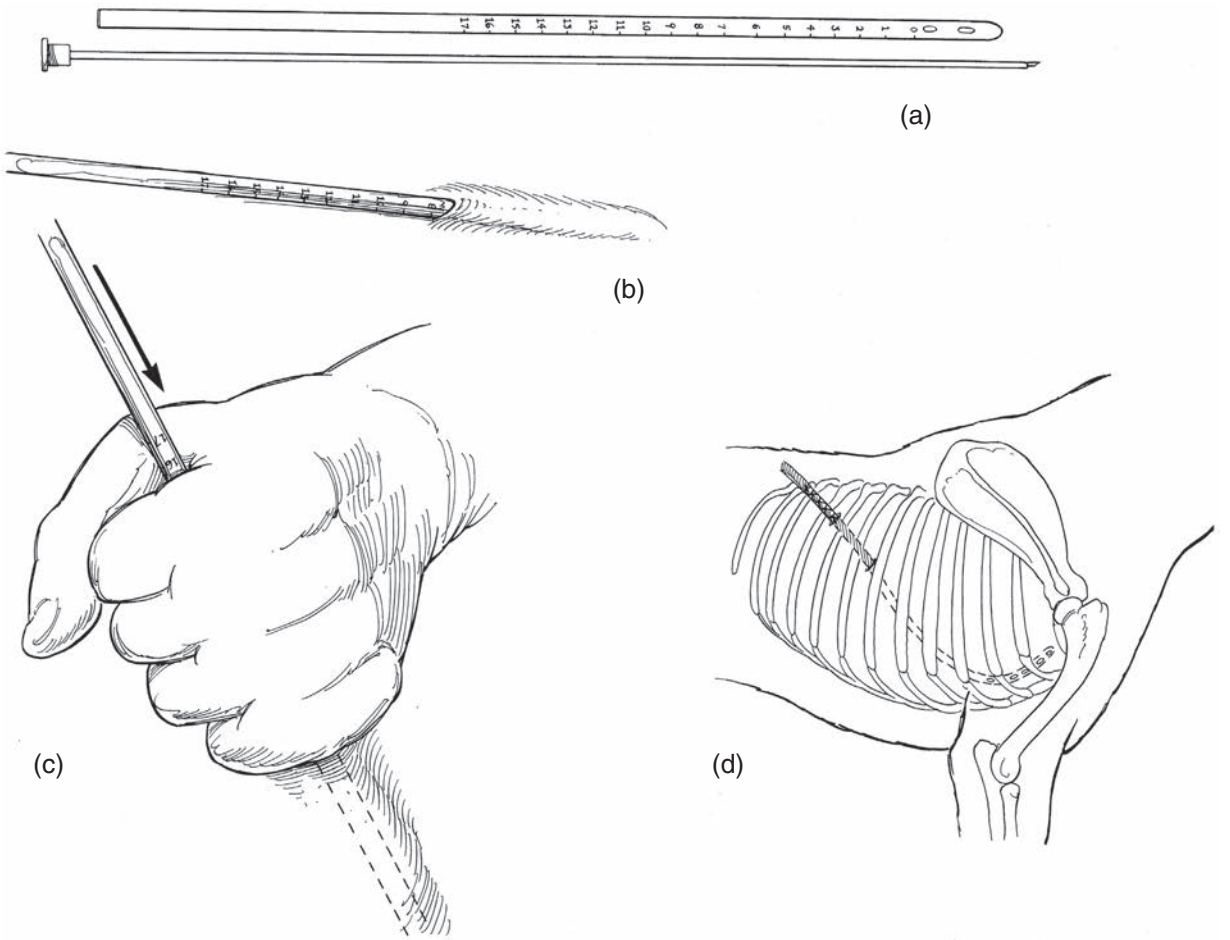

Figure 7.2 Thoracostomy Tube Placement

fails to palliate clinical signs. Thoracostomy tubes also facilitate lavage the pleural space of patients with a pyothorax. Bilateral thoracostomy tubes may sometimes be necessary if the volume of fluid or air produced is very high. Thoracostomy tubes are sometimes placed before thoracic surgery to assure adequate lung expansion and improve arterial oxygen saturation prior to induction of general anesthesia. Relative contraindications for placement of a thoracostomy tube are coagulopathy or presence of pleural adhesions. Adhesions are difficult to predict even with an ultrasound, but fortunately, are rare in dogs and cats.

Different types of thoracostomy tubes are available. Commercially available large-caliber thoracostomy tubes come with a stylet or trocar to aid closed percutaneous placement. Small-caliber thoracostomy tubes are available with a flexible wire that facilitates implantation with a Seldinger technique. The diameter of a thoracostomy tube takes into account both the size of the patient and the amount and character of the fluid. In the case of pyothorax with a viscous exudate, a large-caliber tube will likely be needed (16–20 Fr). For nonviscous fluids, small-caliber tubes may be adequate and will be less painful. These tubes can also be used to instill sclerosing agents. Pneumothorax requires a large caliber drain if the patient is unstable, if there is a tension pneumothorax, or if a pneumothorax looks severe on radiographs [3]. Small-caliber thoracostomy tubes can be placed for initial stabilization and diagnostic evaluation, and changed to a larger caliber tube if necessary.

Thoracostomy tubes should have multiple fenestrations to decrease the likelihood of obstruction by pleural tissues and facilitate efficient drainage of the pleural space. The tube should be rigid enough to enable suction without collapsing. They should be made of a low-reactivity materials such as silicone and have a radiopaque line to allow evaluation of placement on radiographs. Also, it should be rigid enough so that it does not collapse as it traverses the intercostal space. Adapters are used to securely connect the

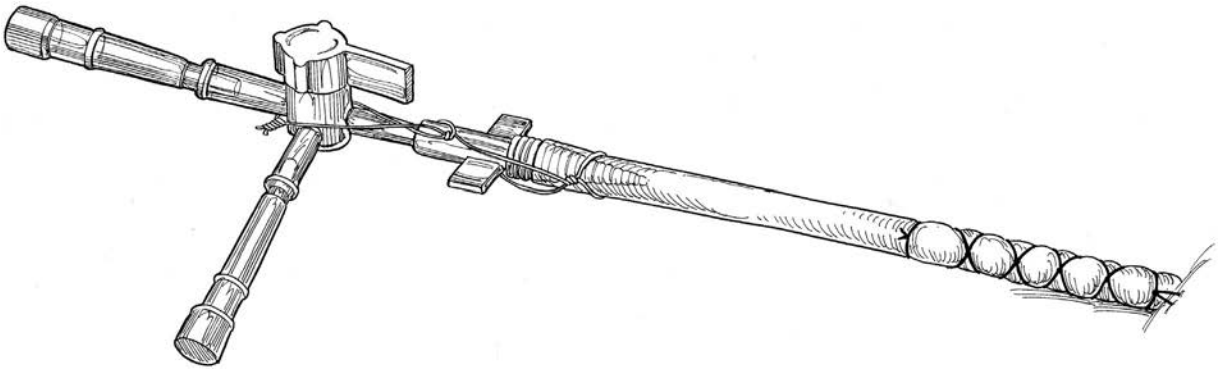

Figure 7.3 Finger-Cuff Suture

thoracostomy tubes to a suction unit or sealing device. The adapter usually has a locking mechanism with a tubing clamp and a three way stopcock to facilitate drainage and prevent air from entering the pleural space. Some adapters have an injection port on the side for injection of medications without disconnecting and contaminating the adapter.

Closed Thoracostomy Tube Placement

Open placement of thoracostomy tubes after thoracic surgery is described in Chapters 4 and 5. Closed placement of a thoracostomy tubes is performed under general anesthesia or heavy sedation with local anesthesia. If general anesthesia is used, all the equipment

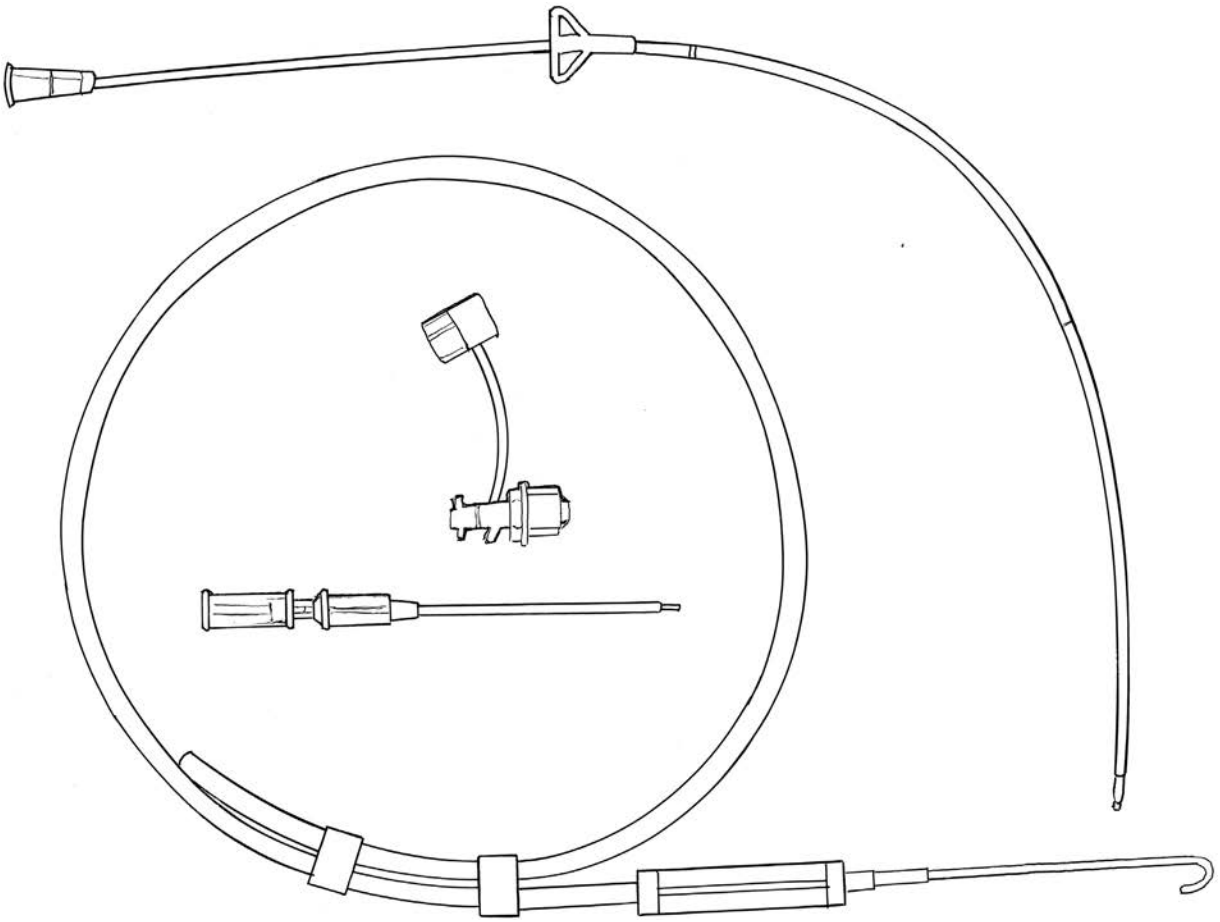

Figure 7.4 Small-Caliber Thoracostomy Tube with Guidewire

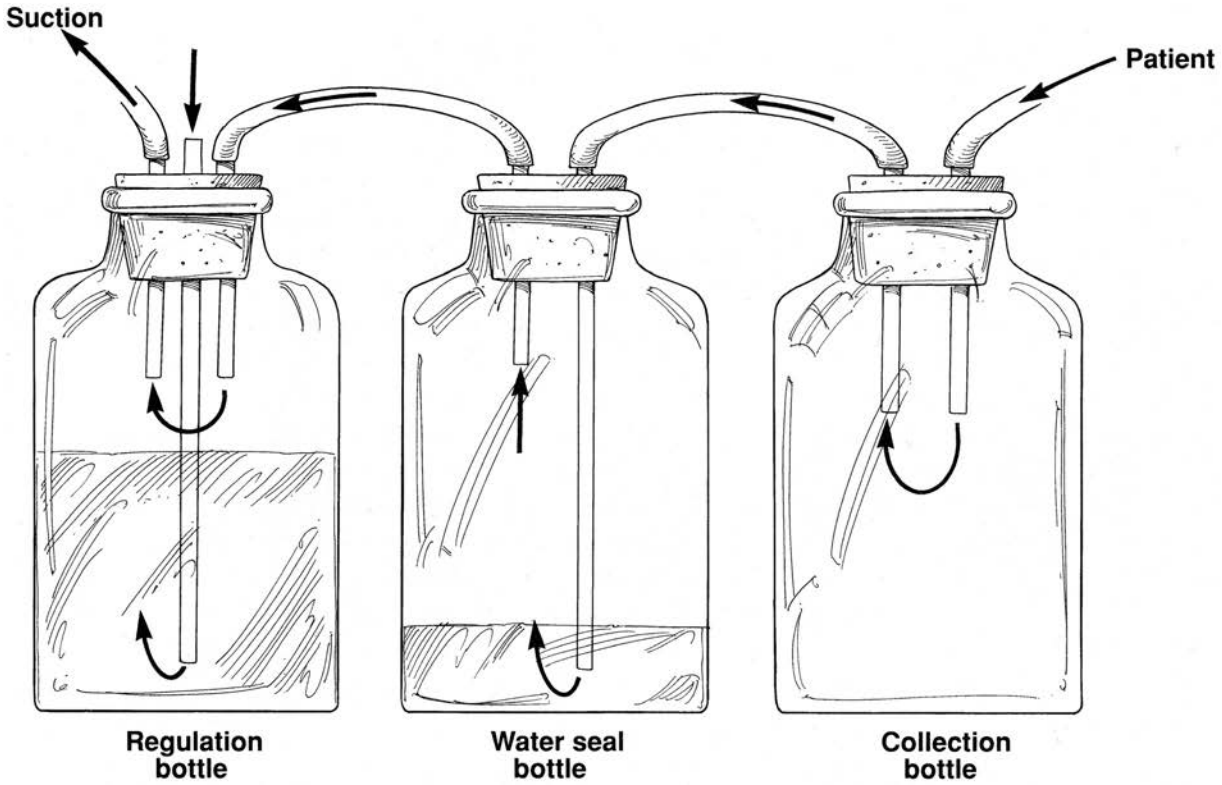

Figure 7.5 Bottle System for Continuous Drainage

should be ready and the thoracic wall should be clipped and ready to be prepared with chlorhexidine scrubs before induction of anesthesia.

Large-caliber thoracostomy tubes are placed with the aid of a stylet with the patient in lateral recumbency (Figure 7.2a). An incision is made in the skin over the caudal dorsal region of the thoracic cavity at the level of the tenth intercostal space. The tube is then tunneled under the skin and latissimus dorsi muscle to reduce the risk of air leakage inside the pleural space (Figure 7.2b) [4]. The tunnel should be over at least three intercostal spaces and directed toward the elbow. The tube and stylet apparatus is lifted perpendicular to the chest wall as one hand firmly grasps the tube to limit the depth of penetration (Figure 7.2c). A brisk but controlled thrust is applied to the end of the stylet and with the tube and style pointed toward the opposite elbow. It is obviously important to limit the depth of penetration to prevent trauma to organs in the thoracic cavity. After the pleural space is entered, the stylet is pulled back to cover its sharp tip and the tube is advanced into the cranial ventral pleural space (Figure 7.2d). The stylet is removed as the tube is advanced. An adapter is attached to the tube and the pleural space is immediately evacuated. The tube is secured to the skin using a finger-trap suture pattern of 2-0 or 3-0 nylon (Figure 7.3). The end of the tube should be secured with a wire or other mechanism to prevent accidental dislodgement. Attachment of a three-way stopcock facilitates intermittent drainage.

Small-caliber thoracostomy tubes (12 or 14 gauge) have the advantage of being less painful and can be placed with a modified over-the-wire (Seldinger) technique (Figure 7.4). Local anesthesia and light sedation is generally sufficient for placement of small-caliber tubes. A needle catheter is tunneled a short distance under the skin and then across the thoracic wall into the pleural space. A wire is introduced through the catheter into the pleural space. The catheter is then removed and a small incision in made in the skin at the entry point of the wire. The thoracostomy tube advanced over the wire into the pleural space. The tube is then secured to the skin with nylon suture. Thoracic radiographs can be taken to confirm adequate placement of the tube in the pleural space. Small-caliber thoracostomy tubes are generally inadequate in the setting of thick exudates such as pyothorax and may collapse when higher negative pressures are applied to the tube [5].

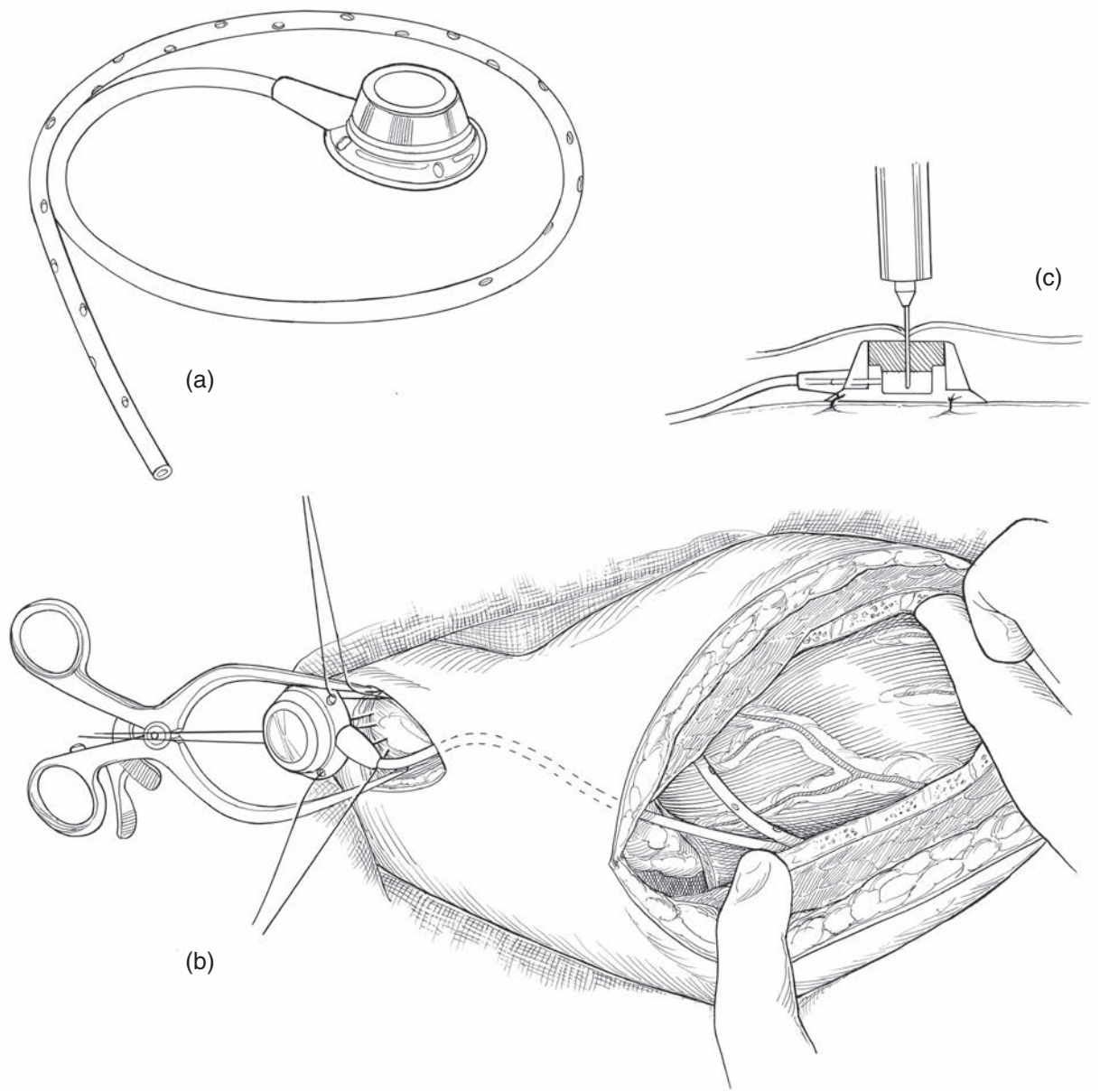

Figure 7.6 Subcutaneous Pleural Drainage

Thoracostomy Tube Management

Intermittent or continuous suction can be applied to large-bore thoracostomy tubes. Small-bore thoracostomy tubes are not able to support continuous suction. Intermittent suction is accomplish by periodic evacuation with a syringe. The time interval depends on the volume and rapidity of fluid or air accumulation. Evacuation is continued until negative pressure is reached. Applied negative pressure should be kept very low to avoid injury to lung parenchyma and prevent inadvertent obstruction the tube by aspiration of mediastinal tissues. No more than 5 mL of negative pressure on the syringe is recommended [6–8].

Continuous suction is mostly required for patients producing a large amounts of fluid or air that cannot be managed by intermittent evacuation. Typically, this is recognized when negative pressures can only be briefly maintained after intermittent suction, particularly air. Continuous drainage is provided by connecting the tube to an apparatus specifically designed for continuous pleural drainage. All of these systems are based on the original three-bottle design (Figure 7.5) consisting of a collection reservoir, water seal,

and mechanism for controlling the amount of applied negative pressure. Active suction drainage allows for more consistent lung reexpansion and function. A negative pressure of 10 cm of water is generally sufficient in most clinical situations.

Long-Term Pleural Drainage

Long-term drainage of the pleural space is sometimes needed for dogs or cats with chronic pleural effusions such as neoplastic effusions, idiopathic chylothorax, and right-sided heart failure [9]. Although the long-term prognosis in these cases tends to be poor, palliative relief can sometimes be gained by long-term pleural drainage. This can be accomplished by implantation of a thoracostomy tube with a subcutaneous access port (PleuralPort, Norfolk Vet Products). This approach reduces the risks of infection and dislodgement by the patient compared to chronically maintaining a standard external thoracostomy tube.

The device consists of fenestrated radio-opaque silicone tube that is placed in the pleural space and a drainage hub that is implanted under the skin (Figure 7.6a). The device is available in 7 and 9 Fr sizes. The silicone tube is placed in the pleural space via minimal-incision thoracotomy, thoracoscopy, or subcostal approach through the diaphragm (Figure 7.6b). Thoracoscopy has the advantage of being able to visualize the silicone tube to confirm ideal placement at the time of implantation. With other approaches positioning can be confirmed by intraoperative fluoroscopy. A J-tip wire can be used to facilitate the placement. After the silicone tube is inserted, it is connected to the hub. A sleeve is used to secure the tube to the hub. The apparatus is flushed with saline to make sure the tube is patent and not kinked. The subcutaneous hub is placed in a pocket created on the lateral thoracic or abdominal wall. The hub is placed in a location that is easy to access when the animal is standing or laying down. The hub is secured with three mattress sutures of nonabsorbable suture.

The hub is accessed by percutaneous puncture with a Huber needle to drain the pleural space or inject medications (Figure 7.6c). A hypodermic needle should not be used to access the hub. Intracavitary chemotherapy can be instilled for the treatment of a mesothelioma. Patency rates for up to 20 days have been reported [9].

References

1 Fartoukh M, Azoulay E, Galliot R, Le Gall JR, Baud F, Chevret S, and Schlemmer B. 2002. Clinically documented pleural effusions in medical ICU patients: how useful is routine thoracentesis? *Chest* 121(1):178–84.

2 Boothe HW, Howe LM, Boothe DM, Reynolds LA, and Carpenter M. 2010. Evaluation of outcomes in dogs treated for pyothorax: 46 cases (1983–2001). *Journal of American Veterinary Medical Association* 236(6):657–63.

3 Baumann MH. 2003. What size chest tube? What drainage system is ideal? And other chest tube management questions. *Current Opinion Pulmonary Medicine* 9(4):276–81.

4 Yoon HY, Mann FA, Lee S, and Branson KR. 2009. Comparison of the amounts of air leakage into the thoracic cavity associated with four thoracostomy tube placement techniques in canine cadavers. *American Journal of Veterinary Research* 70(9): 1161–7.

5 Rozanski EA, and Mooney ET. 2014 In: Bonagura JD, and Twedt DC eds. *Kirk's Current Veterinary Therapy*. St Louis: Elsevier., pp. 700–704.

6 Tillson DM. 1997. Thoracostomy tubes 1. Indications and anesthesia. *Compendium on Continuing Education for the Practicing Veterinarian* 19(11):1258.

7 Tillson DM. 1997. Thoracostomy tubes 2. Placement and maintenance. *Compendium on Continuing Education for the Practicing Veterinarian* 19(12):1331.

8 Sigrist NE. In: Silverstein DC, and Hopper K eds. 2009. *Small Animal Critical Care Medicine*. St Louis: Saunders., pp. 131–137.

9 Brooks AC, and Hardie RJ. 2011. Use of the PleuralPort device for management of pleural effusion in six dogs and four cats. *Veterinary Surgery* 40(8):935–41.

8

Thoracic Wall

Eric Monnet

Structure and Function

The skeletal structure of the thorax of dogs and cats consists of 13 pairs of ribs and costal cartilages, 13 vertebrae, and 9 sternebrae. Each rib forms two synovial costovertebral joints with its corresponding vertebra. The first nine costal cartilages form a synovial articulation with the sternum. Mobility afforded by these synovial joints in conjunction with the curvilinear shape of the ribs and costal cartilages allow expansion of the thoracic cavity when the ribs are moved in a craniodorsal direction. The internal thoracic wall is completed by the internal and external intercostal muscles located between each rib. The remaining thoracic musculature includes the serratus dorsalis, serratus ventralis, scalenus, external abdominal oblique, latissimus dorsi, and pectoralis muscles.

The intercostal arteries and veins originate from the aorta and azygous vein, respectively; course ventrally along the caudal border of each rib; and are contiguous with the internal thoracic artery and vein located lateral to the sternum and internal to the costal cartilages. The intercostal nerves arise from the ventral branches of the thoracic nerves and course with the intercostal vessels. Each rib also has intercostal vessels and a nerve on its cranial border that arises from collateral branches of the main intercostal vessels and nerve.

Functionally, the thoracic wall is composed of passive elastic structures and an active musculature. Together, these passive and active elements produce the "bucket handle" motion of the ribs, allowing for expansion and contraction of the thoracic cavity. The passive elements of the thoracic wall have an intrinsic compliance defined as the change in thoracic volume over the change in pressure across the thoracic wall. The passive elements of the thoracic wall produce either an inward or outward elastic recoil, depending on the thoracic volume. The unstressed volume (V_0) of the thorax is defined as the volume at which the passive elastic structures of the thoracic wall are at rest. When the thoracic volume is less than V_0, a net outward passive recoil of the thoracic wall is created; whereas when thoracic volumes are greater than V_0, a net inward passive recoil of the thoracic wall is created. Because the thoracic wall and lungs are functionally linked by negative pleural pressures, total pulmonary compliance is a function of the additive compliance of the thoracic wall and lungs. Abnormalities in total pulmonary compliance may result from changes in either lung or thoracic wall compliance, or both.

The lung volume at which the passive elastic structures of the thoracic wall and lung are in equilibrium at rest is the functional reserve capacity (FRC). At FRC, the inward elastic recoil of the lungs is exactly balanced by the passive outward elastic recoil of thoracic wall. Pulmonary diseases that decrease lung compliance (i.e., restrictive lung diseases) decrease FRC by increasing the inward elastic recoil of the lung, whereas diseases that decrease thoracic wall compliance increase FRC by increasing outward recoil of the thoracic wall. Loss of functional attachment between the lungs and thoracic wall results in inward recoil and collapse of the lungs and outward recoil of the thoracic wall to its unstressed V_0 position. The latter explains the "sprung" appearance of the rib cage seen in animals with pneumothorax.

The thoracic wall musculature and diaphragm form the active respiratory bellows. Contraction of the diaphragm and inspiratory thoracic muscles generates a negative transthoracic pressure resulting in inspiratory air flow and expansion of the lungs. Transthoracic pressures generated by the inspiratory musculature must be sufficient to overcome both airway resistance and compliance of the lungs and thoracic wall. The passive elastic structures of the thoracic wall assist inspiration until the inspiratory volume exceeds V_0. Pulmonary diseases that increase airway resistance or decrease lung compliance require generation

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of higher transthoracic pressures by the active respiratory bellows and thereby increase the work of respiration. Expiration is passive during quiet breathing and is driven by the elastic recoil of the lungs. The thoracic wall also aids expiration when thoracic volume is greater than V_0. Pulmonary diseases that impair expiratory air flow may require active effort by thoracic muscles that support expiration, resulting in increased work of the respiration.

Paradoxical ventilation results when the thoracic wall and diaphragm do not act in concert to support ventilation. This can result from paralysis of the thoracic wall musculature due to low cervical spinal injury. In this case, negative pleural pressures generated by the contraction of the diaphragm cause passive inward movement of the thoracic wall during inspiration. Conversely, bilateral phrenic nerve injury causes paradoxical ventilation characterized by passive inward movement of the diaphragm and abdomen during inspiration. Paradoxical movement may also occur in segments of the thoracic wall that are unstable as the result of trauma (e.g., flail chest) or congenital anomalies (e.g., pectus excavatum).

Trauma

Trauma to the thoracic wall can result from blunt or penetrating injury [1, 2]. Blunt trauma to the thorax can result in single or multiple rib fractures, hemothorax, pneumothorax, pulmonary contusion, cardiac injury, or combinations of these. Management of thoracic wall trauma must take into consideration the likelihood of injury to internal thoracic structures. Multiple segmental rib fractures result in a flail chest injury that can severely compromise ventilation by inducing paradoxical motion of the flail segment [3, 4]. Sharp or penetrating trauma may or may not penetrate the pleural space. If the pleural space is entered, pneumothorax can result. Penetrating injury of the lung can contribute to pneumothorax. Tension pneumothorax can develop if these injuries form a one-way valve that allows air to enter but not exit the pleural space, causing progressively increased positive pressure in the thoracic cavity. Tension pneumothorax severely compromises pulmonary and cardiovascular function and can be rapidly fatal. Bite injuries to the thoracic wall typically result in a devastating combination of crush and penetrating trauma to the thoracic wall and internal thoracic structures.

Open wounds to the thoracic wall that do not penetrate the pleural space should be treated by standard wound management consisting of wound culture, debridement, irrigation, bandaging, drainage, and appropriately timed wound closure based on amount of soft tissue trauma, contamination, and amount of dead space. If an open thoracic wound has penetrated the pleural space, initial management consists of sealing the wound with a bandage and placing a thoracostomy tube. Continuous suction may be applied to maintain negative pressure in the pleural space while the patient is stabilized. After the patient is stabilized, the wound should be explored and repaired under general anesthesia and positive pressure ventilation. Injury to underlying lungs is managed by direct suturing and conservative partial lung resection, as described in Chapter 15. Severe lacerations to multiple adjacent intercostal spaces are common in bite injuries to the thorax (Figure 8.1a). These are repaired by direct suturing of intercostal muscles reinforced by staggered circumcostal sutures placed in a *basket-weave* pattern (Figure 8.1b). If injury to overlying soft tissues is severe, omentum can be mobilized from the abdomen and placed over the outer thoracic wall to help seal the wound.

Blunt trauma to the thorax is typically managed conservatively. Diagnostic thoracocentesis should be performed and a thoracostomy tube placed if pneumothorax and/or hemothorax are present. Shed blood can be saved and washed for reinfusion, as described in Chapter 9. Autotransfusion of unwashed shed blood is generally avoided unless hemothorax is severe and no alternative exists. Surgical exploration of the thorax should be considered for severe hemothorax that does not subside within a few hours or severe pneumothorax that persists for longer than 24 hours.

Rib fractures are initially managed with an intercostal nerve block with bupivacaine. Lidocaine and bupivacaine (1.5 mg/kg of each drug every 6 hours) can also be infused into the pleural space via the thoracostomy tube to help control pain. Isolated rib fractures resulting from closed blunt trauma usually do not require stabilization unless they are protruding into the pleural space and lacerating the lung. Segmental rib fractures of three or more adjacent ribs will result in a flail chest injury [3, 5]. Flail chest can severely compromise ventilation by paradoxical motion of the flail segment. Animals with a flail chest should initially be placed in lateral recumbency with the flail segment down to decrease paradoxical movement. A loose bandage can also be applied to the chest to limit excursion of the flail chest during expiration. The bandage should not be tight because it can interfere with ventilation. Flail chest is associated with high morbidity because of the likelihood of comorbidities associated with trauma. Early intervention and stabilization of the flail segment is currently recommended to improve pulmonary function [4–6].

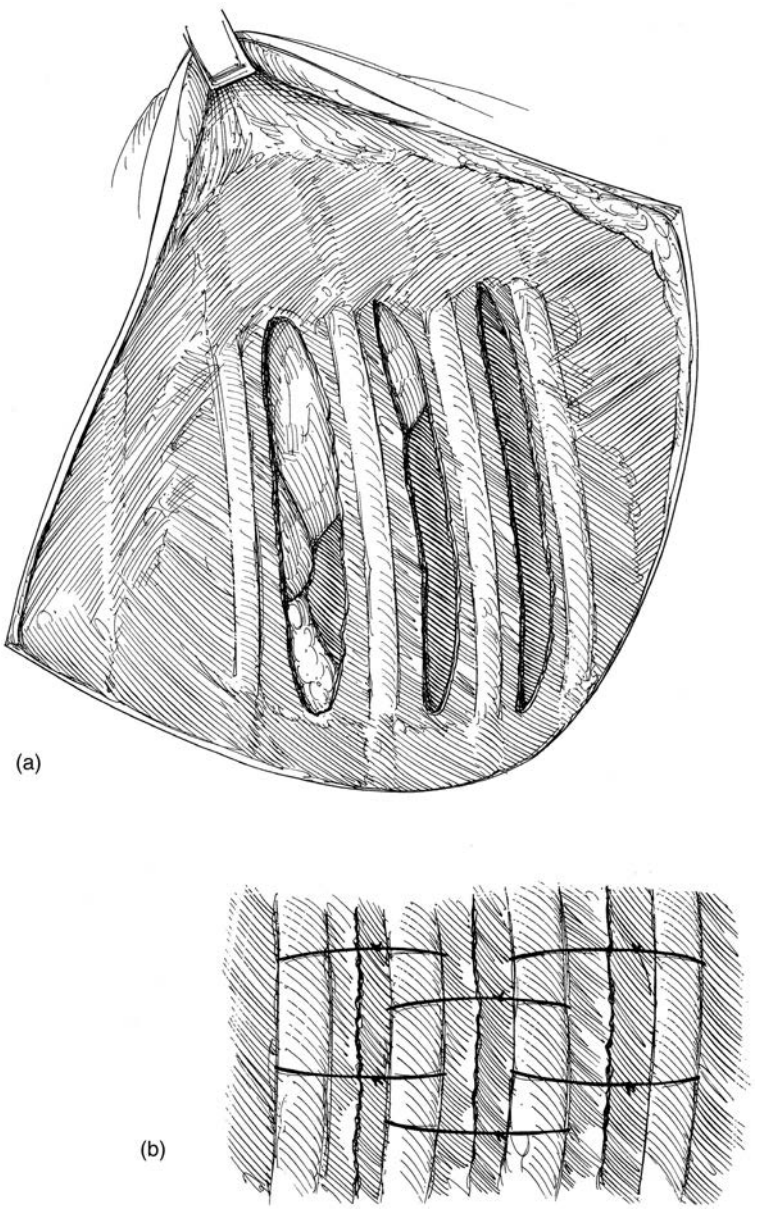


Figure 8.1 Basket-Weave Repair of Intercostal Laceration

Positive pressure ventilation has been recommended to maintain ventilation and reduce hypoxemia associated with pulmonary contusion [7–9]. Flail chest injury in animals can be successfully managed with an external brace that stabilizes and prevents paradoxical movement of the fail segment (Figure 8.2). This is accomplished by fashioning a brace that spans the fail segment and resynchronizes its motion to the adjacent chest wall. The flail segment is fixed to the brace by percutaneous heavy monofilament sutures placed around the ribs and out to the brace.

Occasionally, internal fixation of rib fractures is necessary. This can include animals with severe concurrent soft tissue injury caused by bite trauma or significant instability of the thoracic wall. Several techniques involving pins and/or cerclage wire can be employed (Figure 8.3). Internal fixation can be combined with external splinting to treat flail chest injury [10].

Thoracic Wall Neoplasia

Primary tumors of the ribs and sternum are most often malignant sarcomas. Chondrosarcoma and osteosarcoma are the most common primary rib tumors in dogs [11–15]. Fibrosarcoma and hemangiosarcoma of the ribs have also been reported [12, 16].

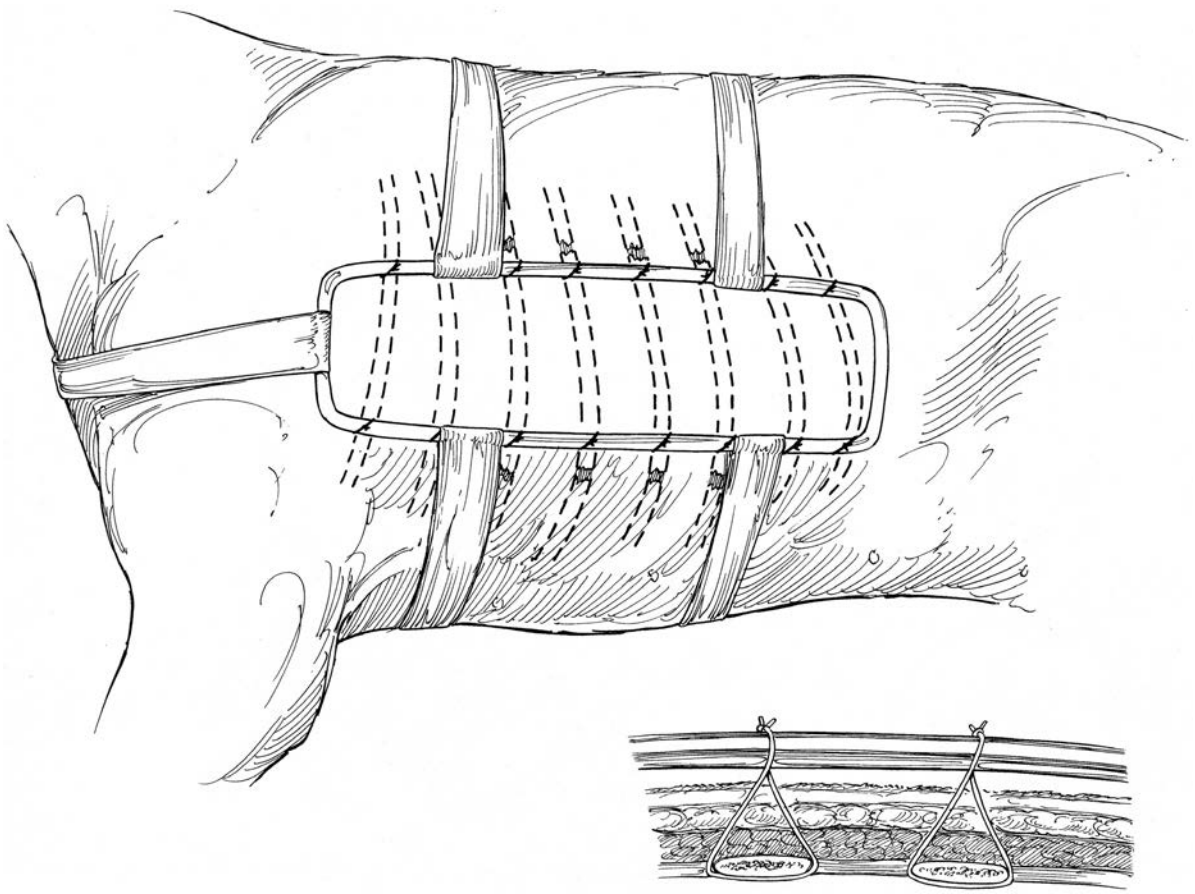


Figure 8.2 External Splint for Flail Chest

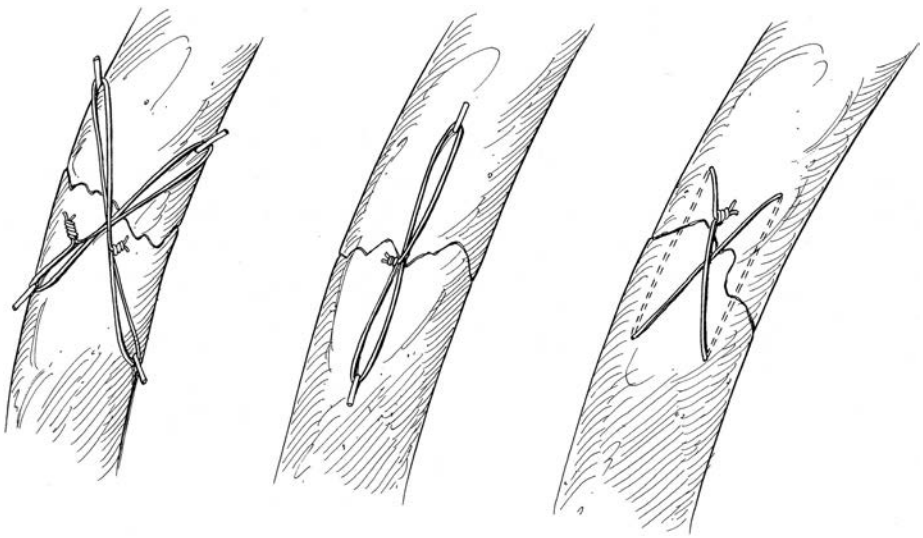


Figure 8.3 Rib Fracture Repair

Soft-tissue sarcomas sometimes invade the thoracic wall and require thoracic wall resection if there is no facial plane between the tumor and the thoracic wall.

Computed tomography (CT) is the imaging technique of choice for surgical planning for thoracic wall neoplasia. Three-dimensional reconstruction provides understanding of the extent of the mass in the pleural space. CT also allows evaluation of the lung parenchyma for metastasis. Ultrasound or CT of the abdominal is indicated to staging, especially when soft-tissue sarcoma is suspected.

Biopsy

Biopsy should be performed prior to surgical resection to establish long-term prognosis and assist with planning the surgery. Chondrosarcoma has a better long-term prognosis compared to osteosarcoma, especially when complete excision is possible [12]. A median survival time of 1080 days has been reported for chondrosarcoma versus 90 days for osteosarcoma. Needle aspiration alone should not be considered reliable. The biopsy can be collected with a True-Cut needle or by incisional wedge biopsy. The biopsy site is chosen in such a way that it can be included in the definitive resection.

Thoracic Wall Resection and Reconstruction

Surgery for neoplasia of the thoracic wall requires en bloc resection and reconstruction of the thoracic wall. Resection of the thoracic wall is also sometimes required for deep-seated infections of the ribs or sternum. Soft-tissue sarcomas require as wide of a resection possible. A 3 cm margin should be the goal. A sterile skin marker and ruler are used to mark a 3 cm margin on the skin (Figure 8.4a). The skin incision is made and extended into the soft tissues keeping the same margin until the thoracic wall is reached (Figure 8.4b). For primary rib tumors, resection should include the rib cranial and caudal to the tumor to increase the chance of obtaining clean margins. CT scan images help with the decision of which intercostal space to enter without compromising the margins. Brief thoracoscopy can be useful to determine which intercostal to perform the initial thoracotomy. After completing cranial and caudal intercostal thoracotomies, osteotomies of the ribs are performed dorsal and ventral to the planned resection. Prior to cutting each rib, encircling sutures are placed around each rib dorsally and ventrally to ligate the intercostal vessels and prevent bleeding. The ribs are cut with a bone cutter. If the first rib is resected, it is important to identify and preserve the axillary artery and vein. If the sternum is involved in the disease process, a partial sternectomy is included. Adhesions to lung lobes, pericardium, diaphragm, or cranial mediastinum are possible. A lung lobectomy or a partial pericardiectomy may be required to complete the resection [16]. The resected tissues are removed en bloc (Figure 8.4c).

Reconstruction of the thoracic wall is required once the resection is completed. The primary goal of reconstruction is to prevent paradoxical motion of the thoracic wall during breathing. If the resection involves less than three ribs, reconstruction with surrounding soft tissues is sufficient to prevent paradoxical movement of the thoracic wall. Latissimus dorsi and the pectorals muscles can be rotated into the repair, depending on the whether the resection is dorsal or ventral (Figure 8.5). Latissimus dorsi myocutaneous flaps have been used to repair the thoracic wall after resection of more than three ribs [17]. However, this technique is possible only if the latissimus dorsi muscle can be preserved without compromising the en bloc resection of the tumor. Tumors generally have to be located ventrally on the thoracic wall for this technique to be feasible. The myocutaneous or muscular flap is completely dissected from its dorsal attachment, keeping the thoracodorsal artery and vein intact. It is then rotated ventrally to close the defect.

If more than three ribs are resected, then some sort of internal rigid support is needed to reconstruct the thoracic wall. Mesh can be used to reinforce the soft tissue reconstruction. Different types of mesh have been used, including woven high-density polyethylene, polypropylene, polytetrafluoroethylene, and polyglactin 910 [11, 18, 19]. Nonabsorbable mesh is most commonly used even with the increased risk of infection. Polypropylene mesh is usually more pliable and easier to use. Mesh induces a significant inflammatory reaction that promotes fibrosis. It is beneficial to cover the mesh with either muscle or omentum that has been brought from the abdominal cavity [18, 19]. Prosthetic reconstruction has been reported to be associated with a 67% rate of complications [19]. The most common complications are seroma and pleural effusion. Mesh has been used also to reconstruct the sternum [19].

Mesh reconstruction is accomplished by securely attaching the mesh with nonabsorbable mattress sutures around the edges of the resected thoracic wall (Figure 8.6a). The mesh is placed under tension during suturing to prevent paradoxical motion postoperatively. Soft tissues are pulled over the mesh to provide coverage of the mesh and seal to the thoracic cavity (Figure 8.6b). Omentum or muscle flaps

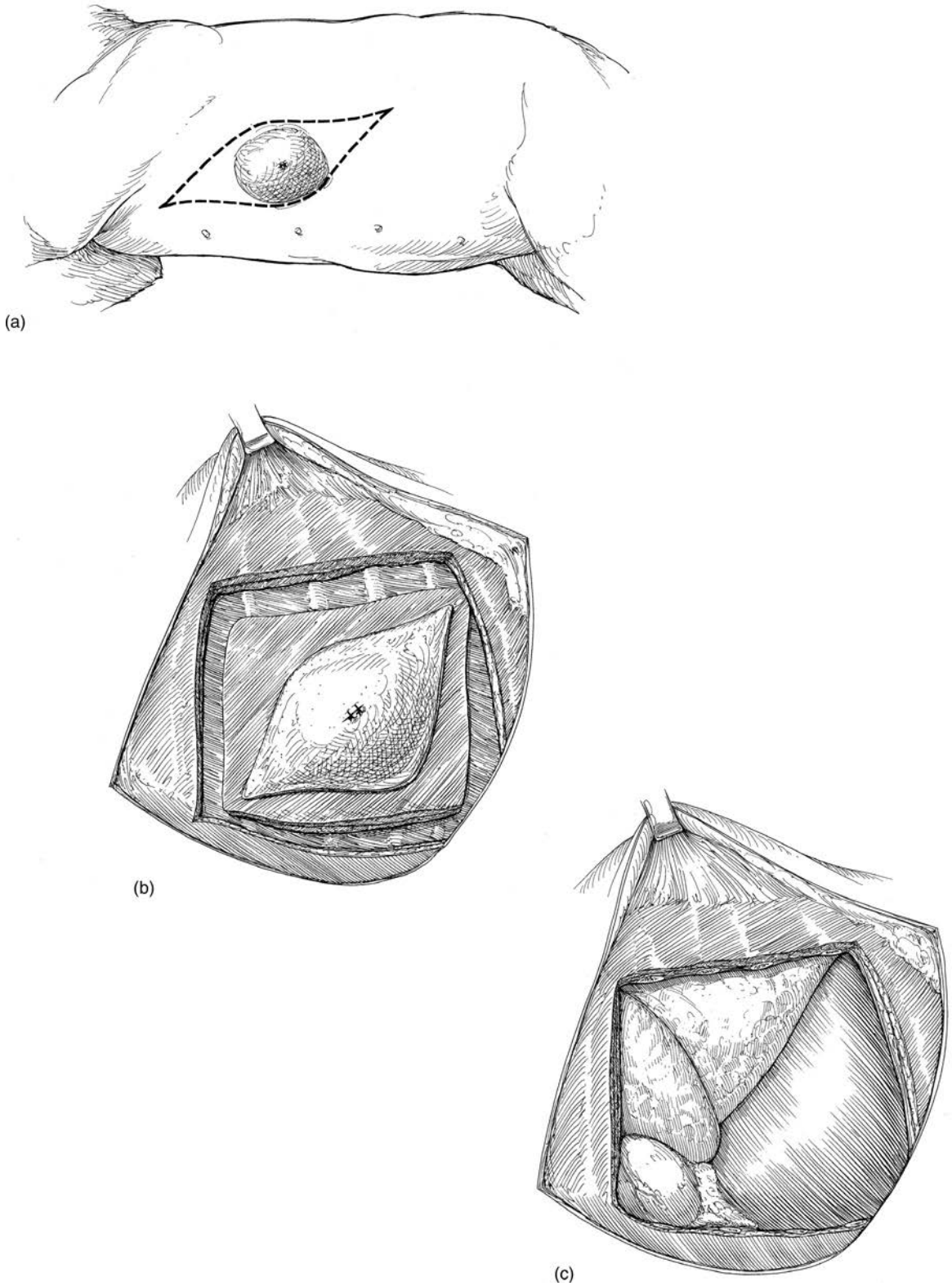


Figure 8.4 Thoracic Wall Resection

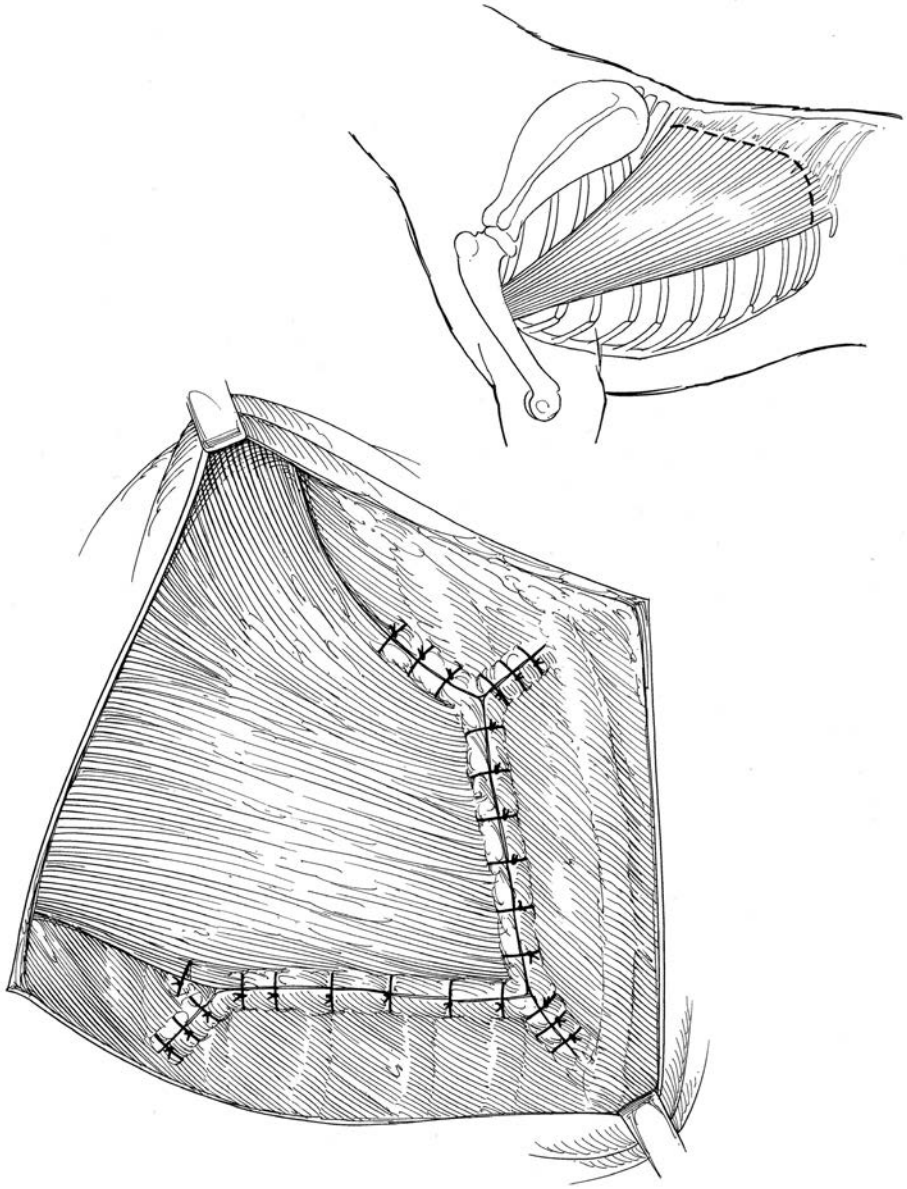


Figure 8.5 Latissimus Dorsi Rotation Flap

can be used to cover the central portion of the mesh if necessary. For very large resections, it may be necessary to reinforce the mesh reconstruction with plates (Figure 8.7). If a section of the sternum has been resected, the unresected ends of the ribs must be stabilized. The opposing ends of the ribs can be brought across the defect and fixed to each other if it does not compromise the expansion of the lungs. However, this technique risks serious compromise of the size of the thoracic cavity and must be undertaken with caution. Larger resections involving the sternum require reconstruction with prosthetic materials.

If the resection involves the last three intercostal spaces, it is possible to advance the diaphragm cranially to the tenth, eleventh, or twelfth rib. The diaphragm is anchored to the cranial aspect of the resection with nonabsorbable suture (Figure 8.8) [11]. Advancement of the diaphragm to the tenth rib may force collapse of the caudal lung lobe, risking pulmonary shunt and hypoxemia. Resection of the caudal lung lobe may be needed to prevent this complication.

The reconstruction is completed by suturing muscle, subcutaneous tissues, and skin over the defect.

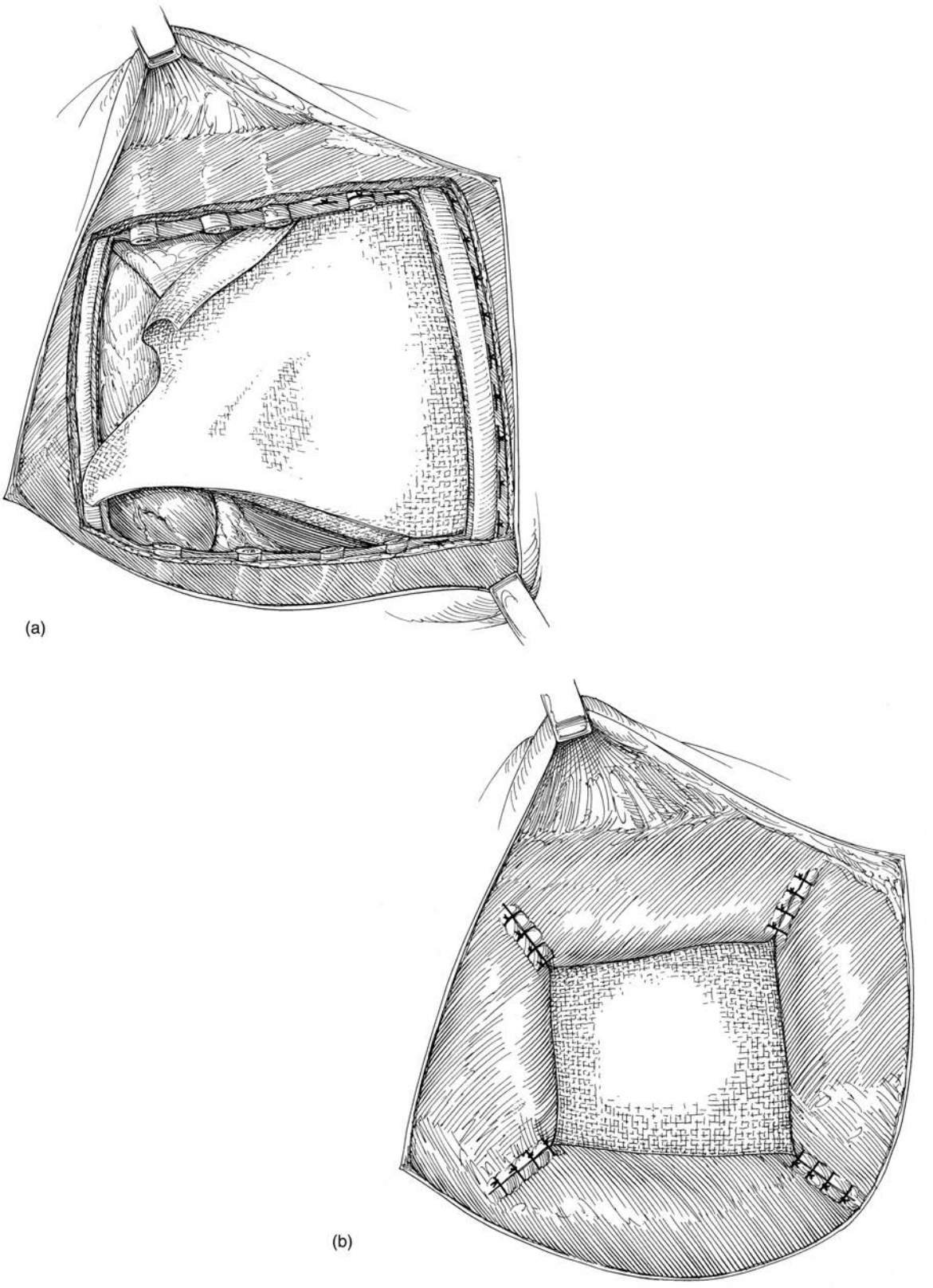
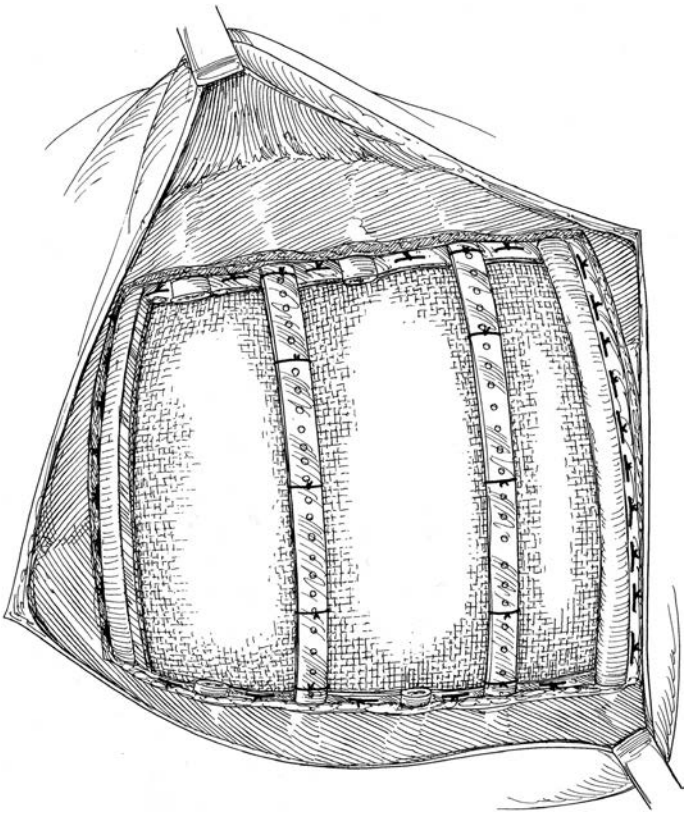


Figure 8.6 Mesh Reconstruction of Thoracic Wall

Figure 8.7 Plate Reconstruction of Thoracic Wall



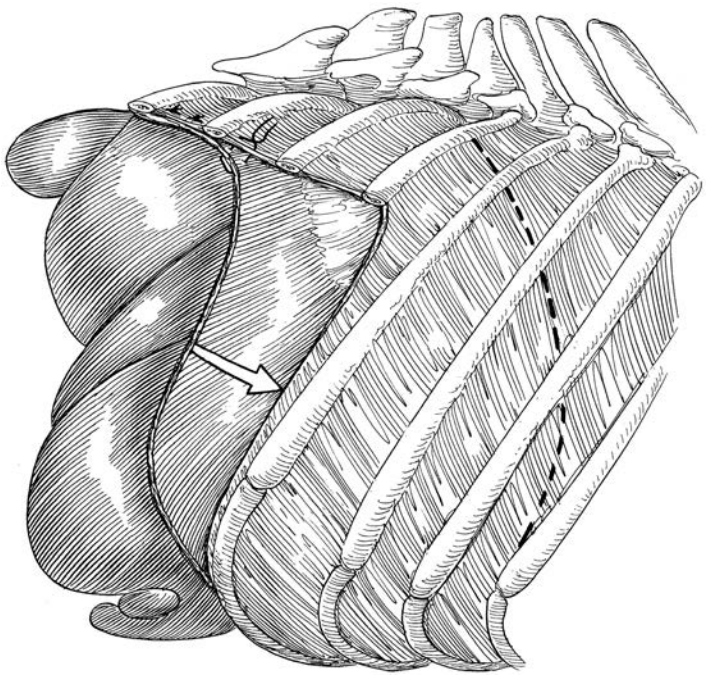


Figure 8.8 Diaphragm Advancement for Thoracic Reconstruction

The primary skin closure can be reinforced with pledgeted tension-releasing sutures in a far-far near-near suture pattern. Tension-releasing sutures are removed three to four days after surgery to prevent necrosis under the pledgets.

Post-Operative Care

Hypoventilation is a serious risk after thoracic wall reconstruction. The animals may require positive-pressure ventilatory support for a period of time after surgery. A temporary tracheostomy facilitates ventilatory support without the need for sedation that can further depress ventilation and make weaning from ventilatory support difficult. During weaning, the patient can be allowed to breathe spontaneously for short periods to test its ability to maintain ventilation without the need to reintubate the patient if ventilatory support needs to be reinitiated. Continuous evacuation of the pleural space with the thoracostomy drain may be needed to maintain negative pressure in the pleural space. Mesh reconstruction may increase the amount of fluid produced in the pleural space. Air leak through the incision is also possible in the first 24 hours after surgery.

Analgesia is a very important aspect of the post-operative treatment after thoracic wall resection and reconstruction. Pain can contribute to poor ventilatory effort after surgery. Intercostal nerve block with bupivacaine administered during surgery will help provide analgesia for 6 to 8 hours after surgery if bupivacaine is used [20]. An epidural catheter can be placed at the time of induction [21]. Morphine can then be administered while the ventilation of the patient is monitored. Intrapleural administration of lidocaine and bupivacaine can be administered every 6 hours lidocaine (1.5 mg/kg) is administered first through the thoracostomy tube followed by bupivacaine (1.5 mg/kg) after 5 minutes [22]. Systemic opioids with or without ketamine will typically also be necessary. Oral opioids and nonsteroidal anti-inflammatory drugs are used for long-term analgesia.

Pectus Excavatum

Pectus excavatum is an uncommon congenital deformity characterized by an inward concave deformation of the caudal sternum and associated costal cartilages. The defect has been reported in both dogs and cats [23–28]. Pectus excavatum is inherited in humans, although the inheritance pattern is not simple. Burmese kittens and brachycephalic dogs appear to be predisposed, suggesting a possible heritable basis for the defect in small animals as well [24, 29]. The precise developmental mechanism of pectus excavatum has not been identified. Abnormal traction of the skeletal tissues by underlying soft tissues, including the diaphragm, defective osteogenesis and chondrogenesis resulting in a lack of rigidity of skeletal tissues, or a combination of these, have been postulated. A more recent theory in humans implicates unbalanced overgrowth of costal cartilages arising from the costochondral region. The predisposition for the defect in brachycephalic animals suggests that elevated pleural pressures resulting from chronic elevation of upper airway resistance may contribute to the development of the condition.

Pectus excavatum can cause varying degrees of cardiopulmonary compromise depending on its severity [23, 24]. Clinical signs associated with more severe defects include growth retardation, exercise intolerance, tachypnea, cyanosis, and vomiting. Respiratory distress results from restricted ventilation or paradoxical movement of the deformity during inspiration, or both. Chronic alveolar collapse can contribute to exercise intolerance and hypoxemia by impairing gas exchange (i.e., pulmonary shunt). Cardiac murmurs, arrhythmias, cardiac displacement, and apparent cardiomegaly have been reported in humans and animals with pectus excavatum. Cardiac murmurs are generally attributed to compression of the heart and kinking of the great vessels; however, concurrent congenital heart defects are possible and should be ruled out prior to surgery. Adult humans with pectus excavatum are reported to have an increased incidence of chronic bronchitis and asthma that improves or resolves with surgical correction of the defect [23].

Surgical repair of pectus excavatum is indicated if cardiopulmonary impairment is severe. Both open and closed methods of correction in animals have been described [23, 24, 27]. In very young animals where the tissues are still pliable, it may be possible to treat the defect with external splinting alone. In this technique, several large sutures are placed percutaneously around the sternum and fixed to an external framework fashioned from plastic splinting materials. Older animals usually require surgery on the hard or soft tissues of the thoracic wall to allow correction of the defect. A combination of techniques including: (1) multiple chondrotomy or chondrectomy of malformed costal cartilages, (2) release of underlying soft tissues contributing to sternal displacement including the diaphragm, and (3) internal struts (e.g., K-wires or small plates) and/or external splints to maintain the position of the sternum have been described in humans and small animals. Successful

correction of severe pectus excavatum in a cat by excision of the caudal sternum and costal cartilages has been reported. Fatal re-expansion pulmonary edema has been reported after external splinting for severe pectus excavatum, so clinicians should be alert for this condition after correction of the defect.

References

1 Spackman CJ, Caywood DD, Feeney DA, and Johnston GR. 1984. Thoracic wall and pulmonary trauma in dogs sustaining fractures as a result of motor vehicle accidents. *Journal of the American Veterinary Medical Association* 185(9): 975–7.

2 Spackman CJ, and Caywood DD. 1987. Management of thoracic trauma and chest wall reconstruction. *Veterinary Clinics North America Small Animal Practice* 17(2):431–47.

3 Olsen D, Renberg W, Perrett J, Hauptman JG, Waldron DR, and Monnet E. 2002. Clinical management of flail chest in dogs and cats: a retrospective study of 24 cases (1989–1999). *Journal of American Animal Hospital Association* 38(4): 315–20.

4 Davignon K, Kwo J, and Bigatello LM. 2004. Pathophysiology and management of the flail chest. *Minerva Anestesiology* 70(4):193–9.

5 Poirier WJ, and Vacca VM, Jr. 2013. Flail chest. *Nursing* 43(12):10–11.

6 Dehghan N, de Mestral C, McKee MD, Schemitsch EH, and Nathens A. 2014. Flail chest injuries: a review of outcomes and treatment practices from the National Trauma Data Bank. *Journal of Trauma Acute Care Surgery* 76(2):462–8.

7 Parham AM, Yarbrough DRr, and Redding JS. 1978. Flail chest syndrome and pulmonary contusion. *Archives of Surgery* 113(7):900–3.

8 Pettiford BL, Luketich JD, and Landreneau RJ. 2007. The management of flail chest. *Thoracic Surgery Clinics* 17(1):25–33.

9 Simon B, Ebert J, Bokhari F, Capella J, Emhoff T, Hayward T, 3rd, Rodriguez A, Smith L, and Eastern Association for the Surgery of T. 2012. Management of pulmonary contusion and flail chest: an Eastern Association for the Surgery of Trauma practice management guideline. *Journal of Trauma Acute Care Surgery* 73(5 Suppl 4):S351–61.

10 Lardinois D, Krueger T, Dusmet M, Ghisletta N, Gugger M, and Ris HB. 2001. Pulmonary function testing after operative stabilisation of the chest wall for flail chest. *European Journal Cardiothorac Surgery* 20(3):496–501.

11 Matthiesen DT, Clark GN, Orsher RJ, Pardo AO, Glennon J, and Patnaik AK. 1992. En bloc resection of primary rib tumors in 40 dogs. *Veterinary Surgery* 21(3):201–4.

12 Pirkey-Ehrhart N, Withrow SJ, Straw RC, Ehrhart EJ, Page RL, Hottinger HL, Hahn KA, Morrison WB, Albrecht MR, Hedlund CS, and et al. 1995. Primary rib tumors in 54 dogs. *Journal of the American Animal Hospital Association* 31(1):65–9.

13 Baines SJ, Lewis S, and White RA. 2002. Primary thoracic wall tumours of mesenchymal origin in dogs: a retrospective study of 46 cases. *Veterinary Record* 150(11):335–9.

14 Liptak JM, Kamstock DA, Dernell WS, Monteith GJ, Rizzo SA, and Withrow SJ. 2008. Oncologic outcome after curative-intent treatment in 39 dogs with primary chest wall tumors (1992–2005). *Veterinary Surgery* 37(5):488–96.

15 Liptak JM. 2013. In: Monnet E ed. *Small Animal Soft Tissue Surgery* Chichester; UK: John Wiley & Sons.

16 Liptak JM, Kamstock DA, Dernell WS, Monteith GJ, Rizzo SA, and Withrow SJ. 2008. Oncologic outcome after curative-intent treatment in 39 dogs with primary chest wall tumors (1992–2005). *Veterinary Surgery* 37(5):488–96.

17 Halfacree ZJ, Baines SJ, Lipscomb VJ, Grierson J, Summers BA, and Brockman DJ. 2007. Use of a latissimus dorsi myocutaneous flap for one-stage reconstruction of the thoracic wall after en bloc resection of primary rib chondrosarcoma in five dogs. *Veterinary Surgery* 36(6):587–92.

18 Bright RM. 1981. Reconstruction of thoracic wall defects using Marlex mesh. *Journal of the American Animal Hospital Association* 17(3):415–20.

19 Liptak JM, Dernell WS, Rizzo SA, Monteith GJ, Kamstock DA, and Withrow SJ. 2008. Reconstruction of chest wall defects after rib tumor resection: a comparison of autogenous, prosthetic, and composite techniques in 44 dogs. *Veterinary Surgery* 37(5):479–87.

20 Berg RJ, and Orton EC. 1986. Pulmonary function in dogs after intercostal thoracotomy: comparison of morphine, oxymorphone, and selective intercostal nerve block. *American Journal of Veterinary Research* 47(2):471–4.

21 Carregaro AB, Freitas GC, Lopes C, Lukarsewski R, Tamiozzo FS, and Santos RR. 2014. Evaluation of analgesic and physiologic effects of epidural

morphine administered at a thoracic or lumbar level in dogs undergoing thoracotomy. *Veterinary Anesthesiology Analgesia* 41(2):205–11.

22 Thompson SE, and Johnson JM. 1991. Analgesia in dogs after intercostal thoracotomy. A comparison of morphine, selective intercostal nerve block, and interpleural regional analgesia with bupivacaine. *Veterinary Surgery* 20(1):73–7.

23 Fossum TW, Boudrieau RJ, Hobson HP, and Rudy RL. 1989. Surgical correction of pectus excavatum, using external splintage in two dogs and a cat. *Journal of the American Veterinary Medical Association* 195:91–7.

24 Boudrieau RJ, Fossum TW, Hartsfield SW, Hobson HP, and Rudy RL. 1990. Pectus excavatum in dogs and cats. *Compendium on Continuing Education for the Practicing Veterinarian* 12:341–55.

25 Ellison G, and Halling KB. 2004. Atypical pectus excavatum in two Welsh terrier littermates. *The Journal of Small Animal Practice* 45(6):311–4.

26 Risselada M, de Rooster H, Liuti T, Polis I, and van Bree H. 2006. Use of internal splinting to realign a noncompliant sternum in a cat with pectus excavatum. *Journal of the American Veterinary Medical Association* 228(7):1047–52.

27 Yoon HY, Mann FA, and Jeong SW. 2008. Surgical correction of pectus excavatum in two cats. *Journal of Veteriary Science* 9(3):335–7.

28 Bedu AS, Labruyère JJ, Thibaud JL, Barthélémy I, Leperlier D, Saunders JH, and Blot S. 2012. Age-related thoracic radiographic changes in golden and labrador retriever muscular dystrophy. *Veterinary Radiology and Ultrasound* 53(5): 492–500.

29 Molina-Díaz VM, and Aguirre JC. 2012. Pectus excavatum in an English Bulldog male: A case report. / Pectus excavatum en un Bulldog Inglés: reporte de caso. *Revista CES Medicina Veterinaria y Zootecnia* 7(2):65–70.

9

Pleural Effusions

Eric Monnet

Structure and Function

The thoracic cavity is completely lined by pleura, a serous membrane [1]. The pleura is divided into the parietal and visceral pleura. The parietal pleura covers the thoracic wall, the diaphragm, and the mediastinum, whereas the visceral pleural covers the lungs. The visceral pleura in dogs and cats is thin in comparison to humans and other animals that have a thick visceral pleural. The visceral and parietal pleura meet at the hilus of the lungs and to create the ligaments of the caudal and accessory lung lobes [1]. The pleura consists of a thin connective tissue layer of collagen and elastic fibers covered with a single cell layer of mesothelial cells. Capillaries in the parietal pleura are mostly supplied by the systemic circulation, whereas capillaries in the visceral pleura are supplied by the pulmonary circulation. The pleura is also supported by a rich lymphatic network. Lymphatics of the parietal pleura are composed of flat cisterns called lacunae. These communicate with the pleural space via 2 to 6 micron stomata. These stomata are most abundant on the mediastinum and intercostal spaces. The visceral pleura has a rich network of lymphatics without stomata that drain toward the hilar lymph nodes. Because stomata are not present in the visceral pleura, drainage of particles from the pleural space only occurs via the parietal pleura [1]. The mean thickness of the parietal pleura is 20 to 25 microns and the capillaries are 10 to 12 microns from the surface [2–5]. Capillaries and lymphatics are within the connective tissue of the visceral pleura. The connective tissue or the visceral pleura contributes to the elastic recoil of the lungs and helps prevent over-inflation of the lungs [1]. The visceral and the parietal pleural mesothelial cell are covered with microvilli with a mean density of 300/micron^2. The mesothelial layer is very fragile. Dislodged mesothelial cells in the pleural fluid become round and may transform in macrophages [1]. Mesothelial cells synthesize type I, II, and IV collagen, elastin, and fibronectin similar to fibroblasts and epithelial cells [5, 6]. Deposition of fibrin is increased during inflammation and the presence of effusion.

Pleural fluid is found within the pleural space between the visceral and parietal pleura. It provides lubrication and allows the transmission of respiratory forces between the lung and the thoracic cavity. The normal amount of fluid in the pleural space is 0.04 to 0.2 ml/kg. Pleural fluid production is about 0.01 ml/kg/h and in steady state with absorption [5,7]. In dogs, 2,200 white blood cells/mm^3 are normally present in pleural fluid [1]. A small amount of protein is present in the pleural fluid, which generates a mean oncotic pressure of 3.2 cm of water [1]. Pleural fluid production and absorption are governed by hydrostatic pressure and oncotic pressure. In dogs under normal physiological conditions, the hydrostatic and the oncotic pressure gradients favor formation of pleural fluid from the parietal pleura and absorption from the visceral pleura [7–10]. Pleural fluid is produced by capillaries and filtered through the mesothelial cells. The pleural fluid is cleared by the lymphatic network. Stomas in the parietal pleura contribute to clearance of pleural fluid and especially large particles.

Accumulation of pleural fluid results from a multitude of diseases and conditions that either increase production or decrease clearance of fluid, or both. Increased fluid production results from an increase in interstitial fluid in the lungs, an increase in the hydrostatic pressure gradient, an increase in capillary permeability, or a decrease in the oncotic pressure gradient. Decreased fluid absorption occurs with obstruction of lymphatics or elevation of systemic venous hydrostatic pressure.

Small Animal Thoracic Surgery, First Edition. E. Christopher Orton and Eric Monnet.

Companion website: www.wiley.com/go/orton/thoracic

Diagnosis

Dogs and cats with pleural effusion present with decreased activity levels, difficulty breathing, and coughing. Anorexia and weight loss are common when the condition becomes chronic. Dogs and cats are febrile if an inflammatory component is present. The presence of a pleural effusion is established from auscultation and thoracic radiographs. Reduction of lung sounds and heart sounds on auscultation is commonly reported. Diagnosis of the type of effusion is based on analysis of fluid collected by thoracocentesis. Fluid is classified as transudate, exudate, septic exudate, chylous, hemorrhagic, or neoplastic (Table 9.1). Hemorrhagic pleural effusion is identified by a PCV > 25%. Neoplastic effusions can be a transudate or exudate with neoplastic cells present on cytology. Lymphoma, pulmonary carcinoma, hemangiosarcoma, and mesothelioma are reported causes of neoplastic effusion [11]. Mesothelioma can be difficult to diagnose based on cytology because reactive and neoplastic mesothelial cells look similar. Tissue samples are required to confirm the histological diagnosis of mesothelioma [12, 13].

Mild pleural effusion is recognized on thoracic radiographs by the presence of fissure lines between the lung lobes. As the amount of effusion increases there is loss of detail in the ventral thoracic cavity and the cardiac silhouette is obscured. Ultrasound is useful to identify masses in the cranial mediastinum and to guide thoracocentesis. Computed tomography is often useful to identify and perform CT-guided biopsy of masses in the thoracic cavity.

Pyothorax

Pyothorax is characterized by a septic exudate in the pleural space caused by bacterial or fungal agents. Dogs and cats with a pyothorax are usually anorexic and febrile. They exhibit significant weight loss as the condition becomes chronic. Severe dyspnea is usually the cause for presentation.

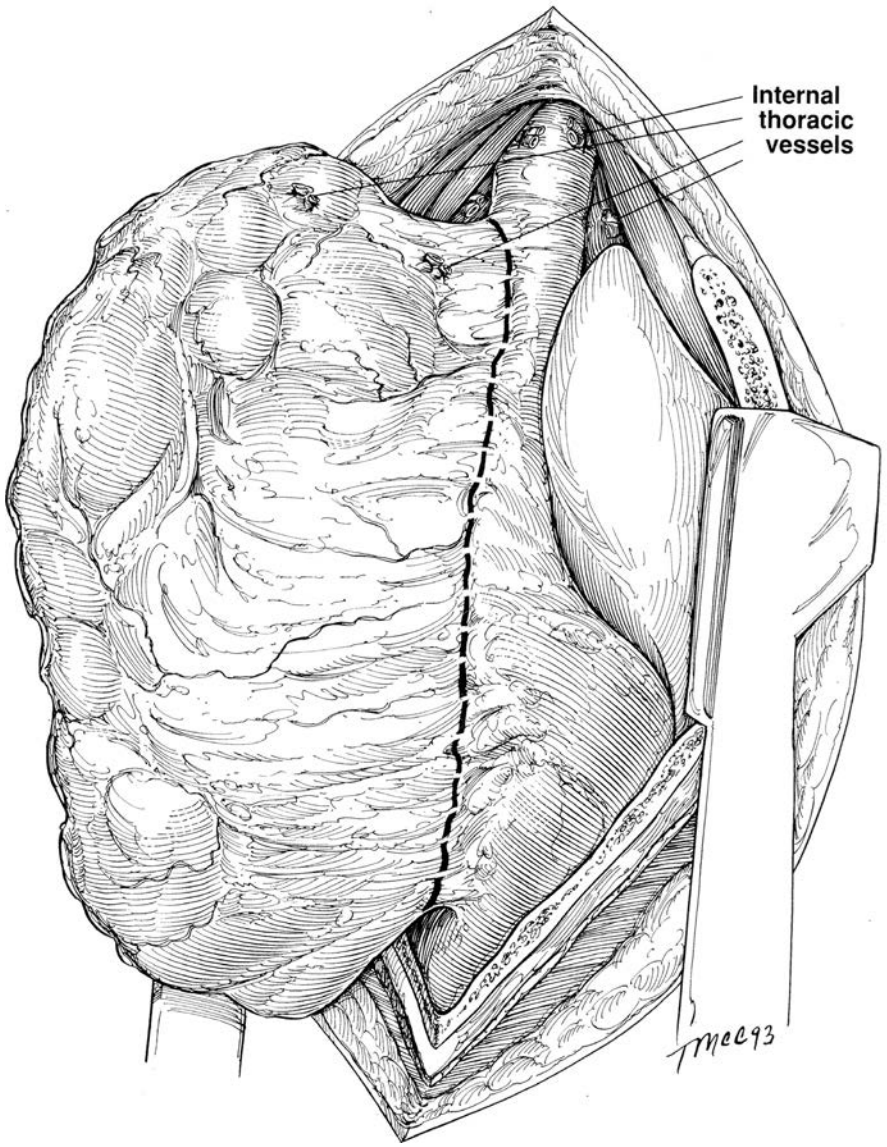
Thoracic radiographs reveal pleural effusion. After drainage of the pleural effusion, radiographs should be repeated to evaluate for presence of masses in the lungs or mediastinum [12]. Fluid analysis demonstrates large numbers of WBC with degenerative neutrophils and possibly microorganisms. Cytology may reveal filamentous organisms, which are more likely to be found on cytology than culture [12]. These organisms are commonly associated with migrating foreign material, especially plant materials. Plant materials may also be identified on cytology. In these cases, antibiotic therapy should be directed at Actinomycetes sp. and Nocardia sp. bacteria. Our antibiotic of choice is clindamycin.

Initial medical treatment consisting of antibiotics based on a culture and sensitivity, thoracic drainage and lavage, and fluid therapy can be attempted for 3 days [14]. If improvement based on blood work and cytology of the pleural fluid is not seen, surgical exploration of the thoracic cavity should be considered. Cats tend to have a better response to medical treatment alone compared to dogs. In one study, 60% of the cats treated medically had a good long-term outcome. The cause of pyothorax in cats is suspected to be the result of contamination from oral flora-caused by bite wounds. In a clinical series in dogs, only 25% of the dogs with medical treatment alone were free of disease at one year [12]. The most common cause of pyothorax in dogs is suspected to be from migrating foreign materials; however, this may depend on the geographic location and activities of the patient. Hunting dogs have a higher incidence of migrating plant material [14–16]. Presence of filamentous organisms in the pleural fluid or a mass/abscess in the cranial mediastinum or lung parenchyma

Table 9.1 Characteristics of Pleural Effusions

	Transudate	Modified transudate	Exudate	Septic Exudate	Chylothorax
Specific gravity	1.018	1.018	1.018		
Nucleated cells (cells/microl)	3×10^3	5×10^3	$> 10 \times 10^3$	30 to 200×10^3	20×10^3
Predominant cell type	mesothelial cells	non degenerate neutrophils	neutrophils and macrophages	degenerate neutrophils	lymphocytes
Protein (g/dL)	1.5		> 3	> 3	3–5
Cytology				Bacteria present	Chylomicron visible Sudan III
Triglycerides					10 to 100 times higher than blood levels

Figure 9.1 Mediastinectomy



should be considered an indication for surgical treatment [12].

Surgical exploration of pyothorax should be undertaken by median sternotomy to allow evaluation of both sides of the thoracic cavity and inspection of all lung lobes and other thoracic organs. The mediastinum, particularly the cranial portion, is typically thickened and abscessed and must be derided or excised (Figure 9.1). The internal thoracic arteries should be identified, ligated, and divided if necessary. Lung lobectomy and pericardiectomy may be required if the infection has extended into these organs. The diaphragm or sternum may need to be debrided if adhesions or osteomyelitis are present. One or two large-caliber thoracostomy drains are placed for drainage and lavage of the pleural space. The sternum is closed in a routine fashion. Freedom from reoccurrence of pyothorax at one year is reported to be 78% in dogs undergoing exploratory surgery [12].

Postoperatively, animals must be aggressively managed for sepsis and possibly septic shock. Continuous hemodynamic monitoring, including electrocardiogram and blood pressure, are typically necessary. Blood gases, electrolytes, lactate, and coagulation profile should be measured periodically, depending on patient stability. A urinary catheter is recommended to evaluate urine production. The goals of the medical treatment after surgery are to maintain the lactate concentration < 2 UI, mean arterial pressure > 60 mmHg, and urine production at least 1 to 2 mL/kg/hr. Preload should be optimized by administration of crystalloid, colloids, and or

whole blood. Administration of plasma is commonly needed to maintain oncotic pressure and replace proteins and coagulation factors lost into the pleural space. Whole blood transfusion is indicated if the PCV drops below 30%. Inotropic support with dopamine or dobutamine is commonly required in the immediate post-operative period to maintain the mean arterial pressure. Pain should be managed with a multimodality approach. Administration of fentanyl by constant rate infusion (2–4 mcg/kg/h) intravenously and intrapleural lidocaine and bupivacaine (1.5 mg/kg of each every 6 to 8 hours) are effective approaches. Arrhythmias are common particularly after pericardiectomy. Ventricular tachycardia is treated if the rate > 140 bpm or it becomes multiform or polymeric. Intravenous lidocaine at 50–100 mcg/kg/min can be administered to control ventricular tachyarrhythmias.

Once the patient is stable, thoracic lavage is performed up to three or four times a day. Usually, 10–20 mL/kg of warm sterile saline is administered into the pleural space via the thoracostomy tube(s). The fluid is left in the pleural space for one hour and then removed. Continuous suction on the thoracostomy drain is recommended in the post-operative period to fully evacuate fluid and air and allow full re-expansion of the lungs. Hypoxemia is a common complication of thoracic surgery. Atelectasis due to fluid or air in the pleural space and hypoventilation due to pain and presence of fluid and air in the pleural space are the most common causes. Supplemental oxygen therapy is recommended if the arterial oxygen saturation is lower than 90% or the alveolar-arterial oxygen gradient on room air is > 20 mmHg. Disseminated intravascular coagulation (DIC) is a possible complication of pyothorax. Coagulation parameters and platelet counts are evaluated daily.

Chylothorax

Chylothorax is due to the accumulation of chyle in the pleural space. Chylomicrons are formed by the absorption of fat from the intestine and are transported to the base of left jugular vein by mesenteric lymphatic vessels, the cisterna chyli, and the thoracic duct. Chylothorax has been associated with heart disease, pericardial disease, trauma, thrombosis of the cranial vena cava, neoplasia in the cranial mediastinum, or heart worm disease [17–19]. Often, chylothorax in dogs is idiopathic because no apparent cause can be identified. Given the poor prognosis associated with idiopathic chylothorax, it is paramount to pursue a complete evaluation, including blood work, thoracic and abdominal radiographs, echocardiography, and fluid analysis in an effort to identify a possible underlying etiology. If a suspected cause is identified, then treatment is directed at correcting the underlying cause while managing the effusion with thoracocentesis and/or thoracostomy drainage.

The surgical treatment of chylothorax due to constrictive pericarditis requires a subtotal pericardiectomy with or without epicardial decortication, as described in Chapter 17. Cranial mediastinal masses are removed via a sternotomy approach. If the mass is invading the cranial vena cava, removing the intravascular mass can be attempted by venotomy after gaining control of proximal and distal flow with tourniquets or vascular clamps. If an obstructing vena caval mass is not resectable, an ePTFE vascular conduit can be placed around the obstruction from cranial vena cava to the right atrium to reestablish flow of venous blood and chyle (Figure 9.2a). A vascular clamp is placed on the cranial vena cava and an end-to-side anastamosis is performed to a large-diameter vascular graft using a continuous pattern of 5-0 monofilament suture (Figure 9.2b). The graft is sutured to the right atrium to complete the bypass graft (Figure 9.2c). Alternatively, a partially obstructed cranial vena cava may be palliated by image-guided placement by a self-expanding or balloon-expandable stent. Chylous effusion caused by recent thrombosis of the cranial vena cave can be treated by placement of a fenestrated thrombosis catheter into the area of thrombosis and infusion of 0.25 to 0.5 U/kg/h of tissue plasminogen activator over a 24-hour period. Chylous effusion secondary to cardiac disease and right-sided congestive heart failure is treated by addressing the underlying cardiac disease and medical therapy for congestive heart failure.

Idiopathic chylous effusion represents a significant management challenge, given how little we understand about its underlying pathogenesis [19]. Several medical and surgical treatments have been attempted in dogs and cats with varying results [20]. Medical treatment consists mostly of low-fat diets and periodic thoracocentesis to palliate the clinical signs. Lipid-soluble vitamins should be administered to compensate for the loss in the chylous effusion. Administration of oral rutin has been advocated for patients with idiopathic chylous effusion with reported improvement chylous volumes in 25% of the cases [21]. Pycnogenol, a pine tree extract with antioxidant and anti-inflammatory properties, has been advocated with a rationale of increasing strength of capillaries [18]. Chyle has been shown not to induce an inflammatory response and to be bacteriostatic

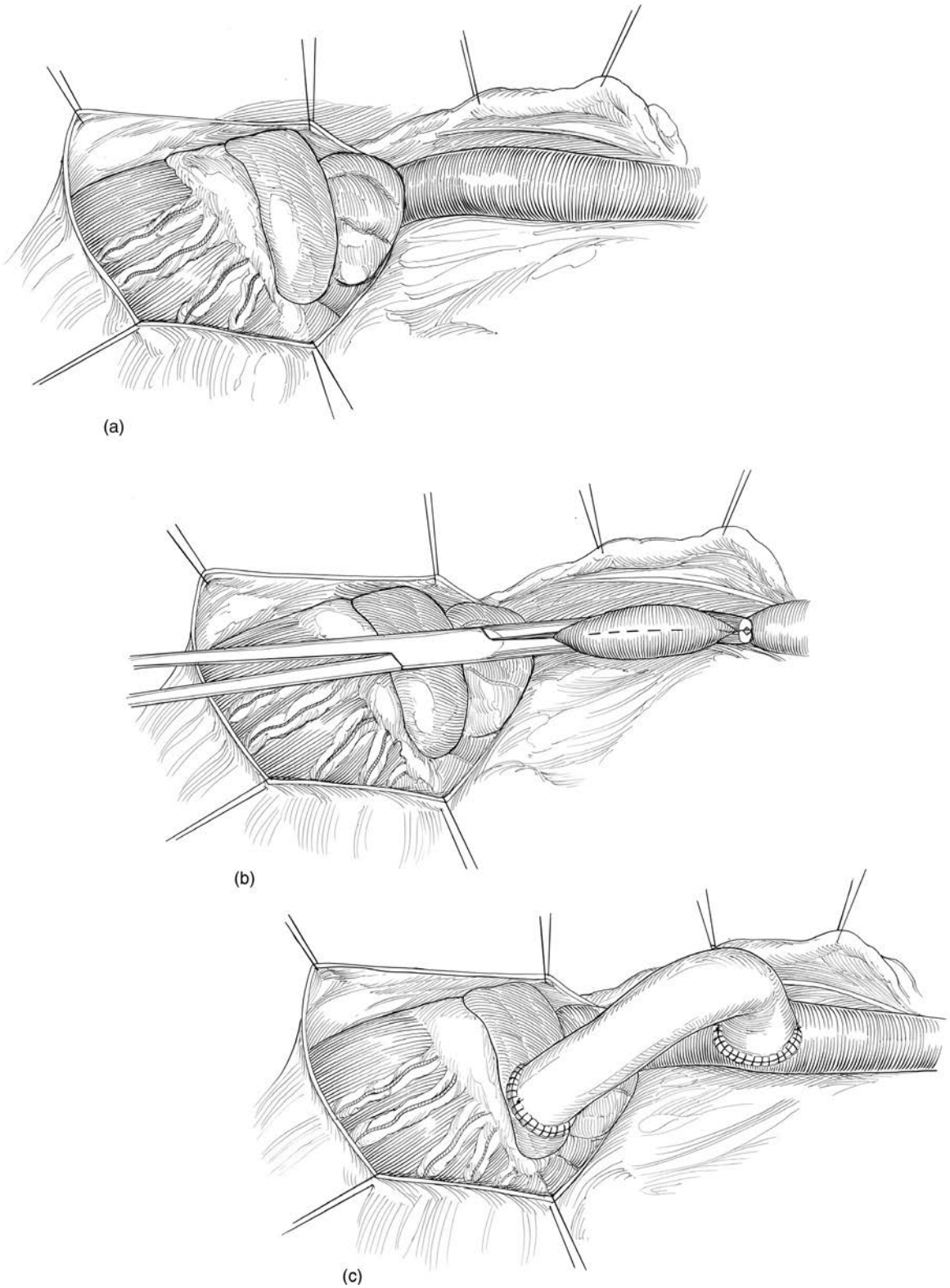


Figure 9.2 Caval-to-Atrial Conduit

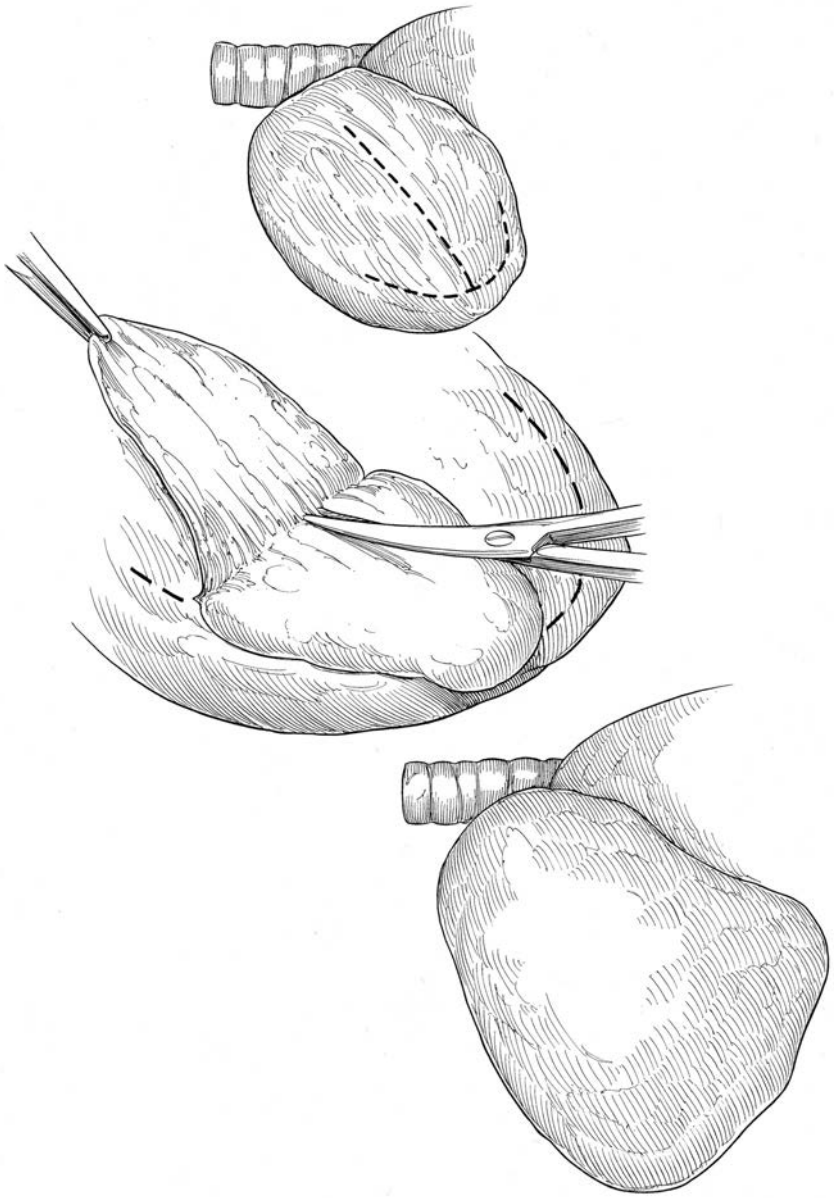


Figure 9.3 Pulmonary Decortication

[22]. However, chronic idiopathic chylous effusion can induce pleural fibrosis that can entrap lungs in a fibrous peel or induce a constrictive pericarditis. Therefore, early surgical intervention is recommended before the onset of fibrosing pleuritis, especially in cats. If the lungs become entrapped in a fibrous peel and cannot be expanded, then surgical exploration and decortication of the lung lobes is necessary to restore pulmonary function (Figure 9.3). This is accomplished by incising the fibrous peel on the surface of the lung and separating it from the underlying parenchyma by careful sharp dissection with scissors.

The surgical treatments for idiopathic chylothorax include thoracic duct ligation or embolization, cisterna chili ablation, subtotal pericardiectomy, omentalization, chronic percutaneous thoracostomy drainage, chronic pleuroperitoneal shunting, or combinations of these. Evidence-based studies supporting any of these treatments in dogs and cats is lacking. The most recent clinical series of treatment of idiopathic chylothorax report success rate between 70% and 85%, regardless of what treatment or combination of treatments is used [23–28]. More effective treatment of idiopathic chylothorax will likely require better understanding of its underlying cause(s).

Currently, surgical treatment of idiopathic chylous effusion can be divided into three strategies: diversion of chyle flow away from the thoracic duct, reduction of resistance to chyle flow, or enhancement of absorption of chylous effusion by redirection to the abdominal cavity.

Diversion of Chylous Flow

The cisterna chyli collects lymphatic flow from the caudal body, including chylous flow from the intestine. The thoracic duct originates from the cistern chyli, carries lymph and chyle through the thoracic cavity, and empties into the left jugular vein close to its junction with cranial vena cava. Diversion of chylous flow away from the pleural space is achieved by either thoracic duct ligation or cisterna chyli ablation. The rationale for thoracic duct ligation or cisterna chyli ablation is to divert the flow of chyle from the thoracic duct toward new lymphaticovenous connections that develop in response to obstruction [24, 29].

Thoracic Duct Ligation

The standard surgical approach for the thoracic duct ligation is a thoracotomy. The thoracic duct is best visualized from a right tenth intercostal thoracotomy in dogs and a left tenth intercostal thoracotomy in cats [29, 30]. Access from a median sternotomy is also possible, but the thoracic duct is more difficult to visualize [27]. After retraction of the diaphragm, the thoracic duct, the aorta, and the azygos vein (left side) are visualized from the dorsal caudal mediastinum. Thoracoscopy has also been used to visualize and ligate the thoracic duct in dogs [28, 31]. Dogs are placed in sternal recumbency, and three access cannulae are placed in the caudodorsal thoracic cavity. The thoracic duct is then isolated and ligated with either clips or sutures. A right flank approach with a transdiaphragmatic exposure of the caudal mediastinum has also been described [32, 33]. This approach allows exposure of the thoracic duct as it enters the thoracic cavity, the cisterna chyli, and a mesenteric lymph node for lymphangiography.

The thoracic duct exhibits a high degree of anatomic variability in dogs and cats. For this reason, techniques to enhance visualization and/or lymphangiograms have been used during surgery in an effort to identify the location and number of branches of the thoracic duct. Feeding heavy table cream to the patient one hour before the surgery enhances visualization of the thoracic duct(s) during surgery. Injection of methylene blue into a mesenteric or caudal peripheral lymph node has also been used to enhance visualization of the thoracic duct and the cisterna chyli [32, 34, 35]. Lastly, injection of indocyanin green and illumination with near-infrared light has been used with success by the author to highlight the thoracic duct.

Lymphangiography is accomplished by injection of low-osmolality nonionic contrast media into the lymphatic system. The contrast agent is administered directly into a mesenteric lymphatic vessel [26, 31, 36–40]. Injection of methylene blue into an ileocecocolic lymph node highlights a lymphatic vessel draining toward the cisterna chyli. A 25 g catheter is placed into the lymphatic vessel and secured with sutures. A water-soluble iodine contrast agent is diluted 1:1 with sterile saline before injection in the lymphatic vessels [31, 37]. The injection is performed while monitoring the flow of contrast agent in the cisterna chyli and the thoracic duct with fluoroscopy. Alternatively, contrast media can be injected directly into a popliteal and mesenteric lymph node [28, 39–42]. Popliteal lymph node is accomplished by injection of 10 to 12 mL of nonionic contrast media (350 mg of iodine/mL) over a five-minute period to outline the thoracic duct on radiographs [37, 41, 42]. Lateral and ventrodorsal radiographs of the thoracic cavity are taken to visualize the thoracic duct. Subtraction radiography can be used to further enhance the thoracic duct and its branches [26]. Computed tomography has also been used to visualize the thoracic duct during lymphangiography and appears to be more effective at outlining branches of the thoracic duct [37, 40]. Magnetic resonance imaging with nano-sized gadolinium has been advocated for lymphangiography [43, 44]. Lymphangiography has also been advocated to document the occlusion of all the branches of the thoracic duct after surgery [28, 35, 40]. That said, efforts to identify and ligate all branches of the thoracic duct have not been shown to correlate with better long-term outcomes.

En-bloc ligation of the caudal mediastinum dorsal the aorta has been advocated as an alternative to thoracic duct ligation with lymphangiography. The rationale for this technique is that, in theory, the thoracic duct(s) must traverse this area regardless of the number of branches or anatomic variation; it therefore decreases the need for intraoperative visualization or lymphangiography [25, 45, 46]. The technique is performed by ligation of all tissue and structures in the caudal mediastinum between the aorta and sympathetic chains. The loose adventitial tissues associated with the aorta are opened (Figure 9.4a). A right-angle forceps is passed to the opposite side of the mediastinum and then back across the base of the vertebra just ventral to the sympathetic chains (Figure 9.4b). At least two ligatures are passed and tied to ligate incorporated tissues, including the azygous

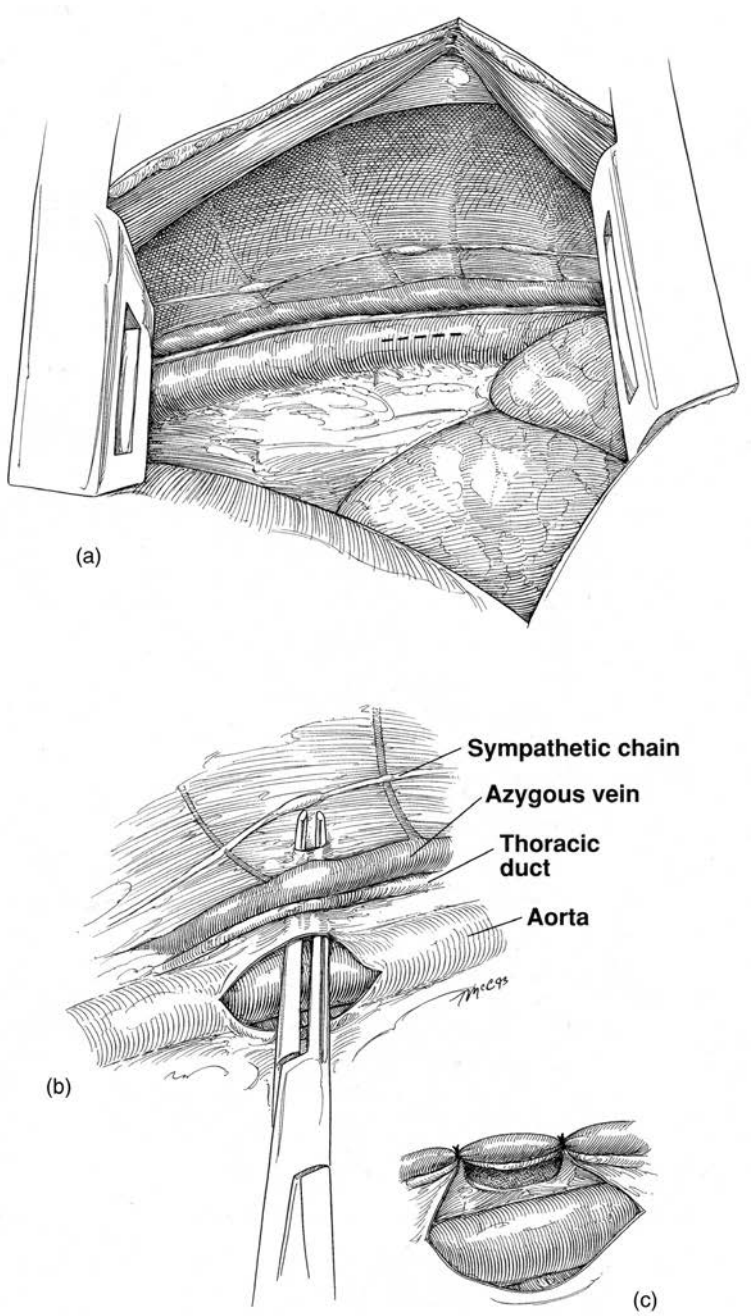


Figure 9.4 *En bloc* Thoracic Duct Ligation

vein (Figure 9.4c). Alternately, the azygous vein can be excluded from the ligation. En-bloc ligation has been reported to incorporate all the branches of the thoracic duct in 93% of the cases [46].

Cisterna Chyli Ablation

Cisterna chyli collects all the lymphatic vessels from the abdominal cavity and pelvic limbs; and serves as the origin of the thoracic duct. Ablation of the cisterna chyli is advocated as an alternative method for diversion of chylous flow away from the thoracic duct and pleural space [24, 26, 38, 47]. The rationale is the same as for thoracic duct ligation, which is that chylous flow is diverted toward new lymphaticovenous connections that form in response to obstruction [24, 26, 38]. The cisterna chyli is located between the aorta and the caudal vena cava at the level of the left renal artery. It is identified by injection of methylene blue in a mesenteric lymph node. The wall of the cisterna chyli is ruptured and omentum is placed

within its lumen [24,26,38]. The cisterna chyli ablation has been performed via laparotomy or video-assisted laparoscopy [48].

Reducing Resistance to Chylous Flow

As previously discussed, disease conditions that resist or obstruct flow of chyle into its venous connection can result in chylothorax. These include caval thrombosis, mediastinal masses, constrictive pericarditis, and right-sided congestive heart failure. When these conditions are recognized in the setting of chylothorax, treatment is directed at relieving the underlying cause. The possible role that high resistance or obstructed flow plays in the pathogenesis of idiopathic chylothorax is poorly understood and largely undocumented. It has been suggested that unrecognized constrictive pericardial disease might play in the pathogenesis of the idiopathic form of the disease. This has resulted in a practice of routinely including subtotal pericardiectomy in the surgical treatment of idiopathic chylothorax without documentation of constrictive physiology. While it can be argued that chylous effusion can induce a fibrous response on the surface of the pericardium, the practice of routinely including pericardiectomy in the surgical treatment of idiopathic chylothorax has been brought into question. Measurements of central venous pressure before and after subtotal pericardiectomy in dogs with chylothorax did not document change before and after pericardiectomy [26]. We currently do not recommend routinely including pericardiectomy in the surgical treatment of idiopathic chylothorax unless constrictive physiology can be documented based on echocardiography and systemic venous pressure measurement.

Enhancing Absorption or Drainage of Chylous Effusion

Several techniques have been explored in an effort to facilitate removal of the chylous effusion from the pleural space. Pleuro-peritoneal shunting with a Denver shunt has been used in an effort to transfer chylous effusion to the peritoneal cavity where it can be resorbed and where its effects are presumably less devastating [49, 50]. This technique has generally proved ineffective in dogs because of limitations on the volume of fluid that can be pumped with the Denver shunt. Pleuro-venous shunts have been used to pump chylous fluid directly into the abdominal caudal vena cava [51]. This approach has physiologic appeal but has been plagued by thrombosis of the catheter. Passive transfer of chylous effusion to the peritoneal cavity by surgically creating a window in the diaphragm has been used with some success. Passing omentum through the window has been used to maintain patency. After making a defect in the diaphragm, the omentum is brought in the thoracic cavity through the defect [25, 27]. Lastly, long-term pleural drainage by implantation of a chronic thoracostomy tube with a subcutaneous access port has been used. The technique is described in Chapter 7. This can provide relief of chylous effusion for a few months, but typically these tubes become obstructed after a period of time [52]. In addition, long-term drainage of chylous effusion generally results in significant malnutrition and weight loss in only a few weeks or months, depending on volume.

Neoplastic Pleural Effusion

When neoplastic effusion is suspected but not conclusive based on cytology, surgical exploration of the pleural space and biopsy of lymph nodes, lungs, and pleura should be considered. Exploratory surgery can be undertaken by sternotomy or video-assisted thoracoscopy with a transdiaphragmatic subxyphoid approach (see Chapter 6). Thoracoscopic lung biopsy can be collected with pre-tied sutures, stapling equipment, or a vessel sealant device. Pre-tied sutures are used to collect biopsy from the peripheral edge of the lung as described in Chapter 15. Thoracoscopic pleural biopsies and lymph node biopsies are collected with 5 mm cup biopsy forceps. Pleural biopsies are collected in the intercostal space. The intercostal neurovascular bundle is identified and avoided during the biopsy. If the bundle is not visible, the biopsies are collected in the caudal part of the intercostal space. Enlarged or reactive lymph nodes are biopsies with a biopsy forceps. Lymph nodes can also be dissected from the cranial mediastinum or the hilus of the lung if needed.

References

1 Light RW. In: Light RW ed. 1982. *Pleural diseases*. pp 1–7 Philadelphia: Wolters Kluwer Health/ Lippincott Williams & Wilkins.

2 Albertine KH, Wiener-Kronish JP, Roos PJ, and Staub NC. 1982. Structure, blood supply, and lymphatic vessels of the sheep's visceral

pleura. *American Journal of Anatomy* 165(3): 277–94.
3 Albertine KH, Wiener-Kronish JP, and Staub NC. 1984. The structure of the parietal pleura and its relationship to pleural liquid dynamics in sheep. *The Anatomical Record* 208(3):401–9.
4 Albertine KH, Wiener-Kronish JP, and Staub NC. 1985. Blood supply of the caudal mediastinal lymph node in sheep. *The Anatomical Record* 212(2): 129–31, 54–5.
5 Antony VB, Sahn SA, Mossman B, Gail DB, and Kalica A. 1992. NHLBI workshop summaries. Pleural cell biology in health and disease. *The American Review of Respiratory Disease* 145(5): 1236–9.
6 Light RW. In: Light RW ed. *Pleural diseases*. 2013. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.
7 Miserocchi G, and Agostoni E. 1971. Contents of the pleural space. *Journal of Applied Physiology* 30(2):208–13.
8 Miserocchi G, D'Angelo E, and Agostoni E. 1971 Topography of pleural surface pressure after pneumo- or hydrothorax. *Journal of Applied Physiology* 32(3):296–303.
9 Reif JS. 1974. Lung and pleural biopsy. *The Veterinary Clinics of North America* 4(2): 383–94.
10 Miserocchi G. 1997. Physiology and pathophysiology of pleural fluid turnover. *The European Respiratory Journal* 10(1):219–25.
11 Kovak JR, Ludwig LL, Bergman PJ, Baer KE, and Noone KE. 2002. Use of thoracoscopy to determine the etiology of pleural effusion in dogs and cats: 18 cases (1998-2001). *Journal of the American Veterinary Medical Association* 221(7):990–4.
12 Rooney MB, and Monnet E. 2002. Medical and surgical treatment of pyothorax in dogs: 26 cases (1991-2001). *Journal of the American Veterinary Medical Association* 221(1):86–92.
13 Garrett LD. In: Withrow SJ, Vail DM, and Page RL eds. 2013. *Small Animal Clinical Oncology*. St Louis: Elsevier Saunders.
14 Boothe HW, Howe LM, Boothe DM, Reynolds LA, and Carpenter M. 2010. Evaluation of outcomes in dogs treated for pyothorax: 46 cases (1983-2001). *Journal of the American Veterinary Medical Association* 236(6):657–63.
15 Piek CJ, and Robben JH. 2000. Pyothorax in nine dogs. *VetQ* 22(2):107–11.
16 Stillion JR, and Letendre JA. 2015. A clinical review of the pathophysiology, diagnosis, and treatment of pyothorax in dogs and cats. *Journal of veterinary Emergency and Critical Care* 25(1):113–29.
17 Singh A, and Brisson BA. 2010. Chylothorax associated with thrombosis of the cranial vena cava. *Canadian Veterinary Journal* 51(8):847–52.
18 McAnulty JF. 2012. pp 749-59 In: Monnet E ed. *Small Animal Soft Tissue Surgery*. Ames: Wiley-Blackwell.
19 Singh A, Brisson B, and Nykamp S. 2012. Idiopathic chylothorax: pathophysiology, diagnosis, and thoracic duct imaging. *Compendium Continuing Education for Veterinarians* 34(8):E2.
20 Singh A, Brisson B, and Nykamp S. 2012. Idiopathic chylothorax in dogs and cats: nonsurgical and surgical management. *Compendium Continuing Education for Veterinarians* 34(8):E3.
21 Thompson MS, Cohn LA, and Jordan RC. 1999 Use of rutin for medical management of idiopathic chylothorax in four cats. *Journal of the American Veterinary Medical Association* 215(3):345–8, 39.
22 Light RW. In: Light RW ed. 2013 *Pleural Diseases*. pp 412–30. Philadelphia: Wolters Kluwer.
23 Fossum TW, Mertens MM, Miller MW, Peacock JT, Saunders A, Gordon S, et al. 2004. Thoracic duct ligation and pericardectomy for treatment of idiopathic chylothorax. *Journal of Veterinary Internal Medicine* 18(3):307–10.
24 Hayashi K, Sicard G, Gellasch K, Frank JD, Hardie RJ, and McAnulty JF. 2005. Cisterna chyli ablation with thoracic duct ligation for chylothorax: Results in eight dogs. *Veterinary Surgery* 34(5):519–23.
25 Bussadori R, Provera A, Martano M, Morello E, Gonzalo-Orden JM, La Rosa G, et al. 2011 Pleural omentalisation with en bloc ligation of the thoracic duct and pericardiectomy for idiopathic chylothorax in nine dogs and four cats. *Veterinary Journal* 188(2):234–6.
26 McAnulty JF. 2011 Prospective comparison of cisterna chyli ablation to pericardectomy for treatment of spontaneously occurring idiopathic chylothorax in the dog. *Veterinary Surgery* 40(8): 926–34.
27 da Silva CA, and Monnet E. 2011 Long-term outcome of dogs treated surgically for idiopathic chylothorax: 11 cases (1995–2009). *Journal of the American Veterinary Medical Association* 239(1): 107–13.
28 Mayhew PD, Culp WT, Mayhew KN, and Morgan OD. 2012. Minimally invasive treatment of idiopathic chylothorax in dogs by thoracoscopic thoracic duct ligation and subphrenic pericardiectomy: 6 cases (2007–2010). *Journal of the American Veterinary Medical Association* 241(7): 904–9.
29 Birchard SJ, Smeak DD, and Fossum TW. 1988. Results of thoracic duct ligation in dogs with

chylothorax. *Journal of the American Veterinary Medical Association* 193(1):68–71.
30 Birchard SJ, and Fossum TW. 1987. Chylothorax in the dog and cat. *Vet Clin North Am Small Anim Pract*. 17(2):271–83.
31 Radlinsky MG, Mason DE, Biller DS, and Olsen D. 2002. Thoracoscopic visualization and ligation of the thoracic duct in dogs. *Veterinary Surgery* 31(2): 138–46.
32 Martin RA, Richards DL, Barber DL, Cordes DO, and Sufit E. 1988. Transdiaphragmatic approach to thoracic duct ligation in the cat. *Veterinary Surgery* 17(1):22–6.
33 Staiger BA, Stanley BJ, and McAnulty JF. 2011 Single paracostal approach to thoracic duct and cisterna chyli: experimental study and case series. *Veterinary Surgery* 40(7):786–94.
34 Enwiller TM, Radlinsky MG, Mason DE, and Roush JK. 2003. Popliteal and mesenteric lymph node injection with methylene blue for coloration of the thoracic duct in dogs. *Veterinary Surgery* 32(4):359–64.
35 Allman DA, Radlinsky MG, Ralph AG, and Rawlings CA. 2010. Thoracoscopic thoracic duct ligation and thoracoscopic pericardectomy for treatment of chylothorax in dogs. *Veterinary Surgery* 39(1):21–7.
36 Kerpsack SJ, McLoughlin MA, Birchard SJ, Smeak DD, and Biller DS. 1994. Evaluation of mesenteric lymphangiography and thoracic duct ligation in cats with chylothorax: 19 cases (1987–1992). *Journal of the American Veterinary Medical Association* 205(5):711–5.
37 Esterline ML, Radlinsky MG, Biller DS, Mason DE, Roush JK, and Cash WC. 2005. Comparison of radiographic and computed tomography lymphangiography for identification of the canine thoracic duct. *Veterinary Radiology & Ultrasound* 46(5):391–5.
38 Sicard GK, Waller KR, and McAnulty JF. 2005. The effect of cisterna chyli ablation combined with thoracic duct ligation on abdominal lymphatic drainage. *Veterinary Surgery* 34(1):64–70.
39 Brisson BA, Holmberg DL, and House M. 2006. Comparison of mesenteric lymphadenography performed via surgical and laparoscopic approaches in dogs. *American Journal of Veterinary Research* 67(1):168–73.
40 Johnson EG, Wisner ER, Kyles A, Koehler C, and Marks SL. 2009. Computed tomographic lymphography of the thoracic duct by mesenteric lymph node injection. *Veterinary Surgery* 38(3): 361–7.
41 Naganobu K, Ohigashi Y, Akiyoshi T, Hagio M, Miyamoto T, and Yamaguchi R. 2006. Lymphography of the thoracic duct by percutaneous injection of iohexol into the popliteal lymph node of dogs: experimental study and clinical application. *Veterinary Surgery* 35(4):377–81.
42 Singh A, Brisson BA, O'Sullivan ML, Solomon JA, Malek S, Nykamp S, et al. 2011 Feasibility of percutaneous catheterization and embolization of the thoracic duct in dogs. *American Journal of Veterinary Research* 72(11):1527–34.
43 Sena LM, Fishman SJ, Jenkins KJ, Xu H, Brechbiel MW, Regino CA, et al. 2010. Magnetic resonance lymphangiography with a nano-sized gadolinium-labeled dendrimer in small and large animal models. *Nanomedicine (Lond)* 5(8):1183–91.
44 Dori Y, Zviman MM, and Itkin M. 2014. Dynamic contrast-enhanced MR lymphangiography: feasibility study in swine. *Radiology* 273(2):410–6.
45 Viehoff FW, and Stokhof AA. 2003. [En bloc ligation of the thoracic duct in twelve dogs with idiopathic chylothorax]. *Tijdschr Diergeneeskd*. 128(9): 278–83.
46 MacDonald NJ, Noble PJ, and Burrow RD. 2008. Efficacy of en bloc ligation of the thoracic duct: descriptive study in 14 dogs. *Veterinary Surgery* 37(7):696–701.
47 Fingeroth JM. 2005. Effect of cisterna chyli ablation combined with thoracic duct ligation on abdominal lymphatic drainage. *Veterinary Surgery* 34:295.
48 Sakals S, Schmiedt CW, and Radlinsky MG. 2011. Comparison and description of transdiaphragmatic and abdominal minimally invasive cisterna chyli ablation in dogs. *Veterinary Surgery* 40(7):795–801.
49 Smeak DD, Gallagher L, Birchard SJ, and Fossum TW. 1987. Management of intractable pleural effusion in a dog with pleuroperitoneal shunt. *Veterinary Surgery* 16(3):212–6.
50 Smeak DD, Stephenj, Birchard, McLoughlin MA, Lindsey MM, Holt DE, et al. 2001. Treatment of chronic pleural effusion with pleuroperitoneal shunts in dogs: 14 cases (1985-1999). *Journal of the American Veterinary Medical Association* 219(11): 1590–7.
51 Willauer CC, and Breznock EM. 1987. Pleurovenous shunting technique for treatment of chylothorax in three dogs. *Journal of the American Veterinary Medical Association* 191(9):1106–9.
52 Brooks AC, and Hardie RJ. 2011. Use of the PleuralPort device for management of pleural effusion in six dogs and four cats. *Veterinary Surgery* 40(8):935–41.

10

Pneumothorax

Eric Monnet

Pneumothorax results when air enters the pleural space. Entry of air though an open wound on the thoracic wall is termed *open pneumothorax,* whereas air leaking from lesions in the lung parenchyma, trachea, or esophagus is a *closed pneumothorax*. *Tension pneumothorax* is a rapidly fatal form of pneumothorax that occurs when positive pressure develops within the pleural space as a result of air accumulation. Pneumothorax is found in 47% of the dogs involved in motor vehicle accidents and 63% of cats associated with high-rise syndrome [1, 2]. Pneumomediastinum typically accompanies pneumothorax when injury to the trachea is the source of air entry. Spontaneous pneumothorax occurs without history or evidence of trauma and is most often caused by spontaneous rupture of a pulmonary blebs or bulla [3–5]. In a report of 12 dogs with spontaneous pneumothorax, more than one lesion was found in 10 dogs and lesions were bilateral in 7 dogs [4]. Other conditions associated with spontaneous pneumothorax include pulmonary neoplasia, pneumonia, migrating foreign body, heartworm disease, feline asthma, and pulmonary blebs [3–10].

Pneumothorax results in decreased tide volume from partial pulmonary collapse. Minute ventilation is usually maintained by an increase in respiratory rate. Hypoxemia results from combinations of low V/Q mismatch and pulmonary shunt, the latter causing poor response to supplemental oxygen administration. Tension pneumothorax exacerbates pulmonary compromise by interfering with venous return and cardiac output, resulting in rapidly fatal cardiopulmonary collapse. Open thoracic wounds from penetrating trauma allow air to be drawn into the pleural space by negative pleural pressures generated by breathing (sucking chest wounds). These must be temporarily sealed with bandages and the pleural space evacuated of air. Open pneumothorax resulting from biting trauma or closed pneumothorax is associated with blunt trauma and often associated with injury to the thoracic wall such a rib fracture or flail chest, pulmonary contusion, and cardiac injury. Management of concurrent injuries to the thoracic wall is discussed in Chapter 8.

Diagnosis

Clinical signs, auscultation, and thoracic radiographs are the key elements for the diagnosis of a pneumothorax. Pneumothorax causes a restrictive breathing pattern characterized by rapid shallow breathing. Uncoupling of the thoracic wall for the underlying lungs allows outward expansion of the thoracic wall, giving the thoracic cavity a "sprung" appearance. Lung and heart sounds will be muffled on auscultation. Percussion of the thorax produces a resonant sound. Findings on thoracic radiographs include elevation of the cardiac silhouette on the lateral view and absence of pulmonary vasculature in the periphery of the thoracic cavity. CT scanning has been advocated to identify the cause of spontaneous pneumothorax. In one study, 13 of 17 lesions were identified on CT scan compared to 4 by radiographs. However, the sensitivity and positive predictive value of CT scan for diagnosis of bullae associated with spontaneous pneumothorax are less than optimal [9].

Treatment

Treatment of pneumothorax requires urgent evacuation of air from the pleural space. Identification and closure of the source of air entry is necessary in some cases. Management strategies typically are governed by the type and causes of pneumothorax.

Open Pneumothorax

Open pneumothorax is caused by penetrating trauma to the thorax, including gunshot wounds, arrows,

Small Animal Thoracic Surgery, First Edition. E. Christopher Orton and Eric Monnet.

Companion website: www.wiley.com/go/orton/thoracic

stabbing injuries, and bite injury. Because of the high likelihood of injury to internal thoracic structures urgent exploratory surgery is indicated. Early induction of general anesthesia and placement on positive pressure ventilation will often be necessary to stabilize the patient. Exploration of the thoracic cavity is typically performed at the site of the open wound(s). The lacerated intercostal space is opened to gain better access to the pleural space. Repair of injuries to the heart, lungs, or esophagus are completed as needed. The pleural space is copiously lavaged with warm saline and appropriate antibiotic therapy is instituted. A thoracostomy tube is placed. The soft tissues of the thoracic wall are debrided and layers closed meticulously to obtain an airtight seal after surgery.

Closed Traumatic Pneumothorax

Animals with closed pneumothorax caused by blunt trauma to the thorax usually do not require exploratory surgery. Typically, traumatic air leaks will seal with conservative management by pleural evacuation after 24 or 48 hours. Pneumothorax is managed initially by thoracocentesis, followed by placement of a small-caliber thoracostomy tube during stabilization of the patient discussed in Chapter 7. It may become necessary to place a large-caliber thoracostomy tube if the amount of air produced is large. A continuous suction system can also be used to maintain negative pressure in the pleural space and expansion of the lungs. If air leakage does not diminish after 48 hours then exploratory surgery sternotomy should be undertaken to identify and repair the source of the leak. Traumatic rupture of the lung is the most likely cause. All lung lobes should be explored for the presence of traumatic blebs (Figure 10.1) or hematomas (Figure 10.2) that could rupture in the future.

Closed pneumothorax can result from bite wounds to the neck that puncture or lacerate the trachea. In this case, pneumothorax is usually accompanied by subcutaneous emphysema and/or pneumomediastinum. The tracheal injury may spontaneously close within 3 days with thoracostomy tube management alone. However, if the tracheal rupture does not seal within 3 days or the pneumothorax is severe, then a surgical exploration of the neck is indicated. Repair of the tracheal injury is accomplished by primary closure or resection and anastomosis, as described in Chapter 14.

Spontaneous Pneumothorax

Spontaneous pneumothorax is classified as primary when lung pathology is not obvious and secondary when lung pathology is present. Even though the cause may not be obvious, primary pneumothorax is typically the result of rupture of emphysematous bullae (Figure 10.3) or pulmonary blebs (Figure 10.4). Secondary pneumothorax results from an identifiable lung pathology such as neoplasia, pneumonia, migrating foreign body, parasite, feline asthma, and heartworm disease.

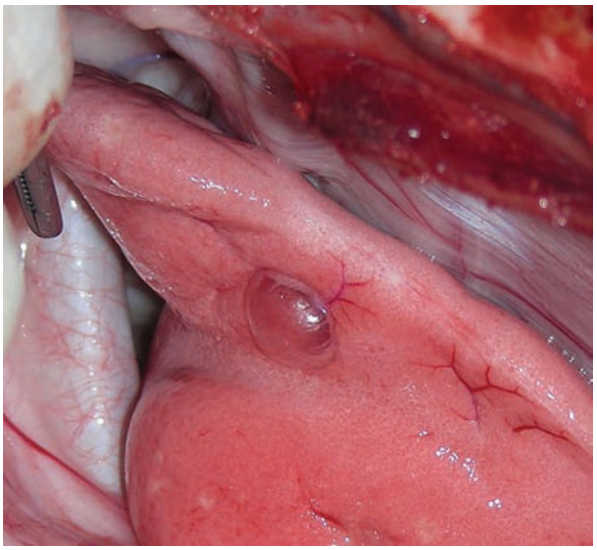


Figure 10.1 Traumatic Pulmonary Bleb

Primary spontaneous pneumothorax is initially managed by urgent thoracocentesis, followed by thoracostomy tube placement. Conservative management alone is associated with a high rate of recurrence. In a study of 64 dogs with primary and secondary spontaneous pneumothorax, exploratory surgery was

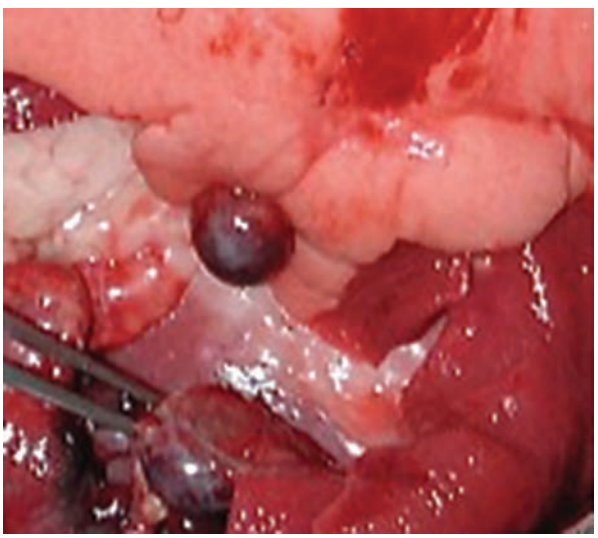


Figure 10.2 Traumatic Pulmonary Hematoma

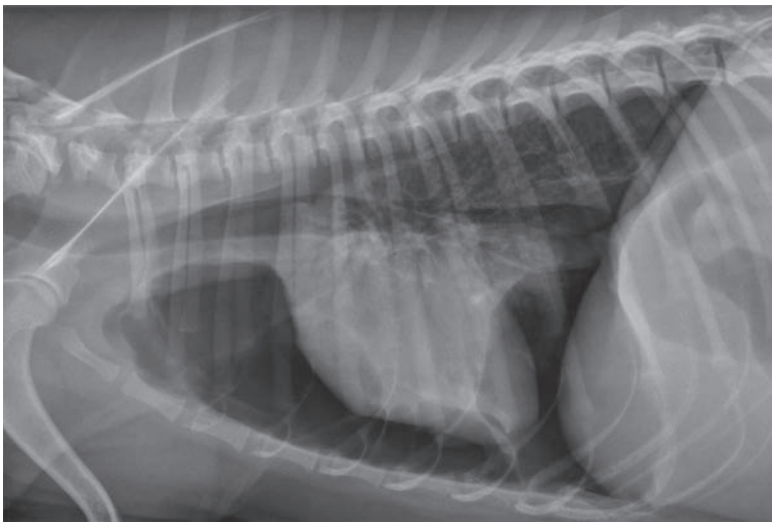
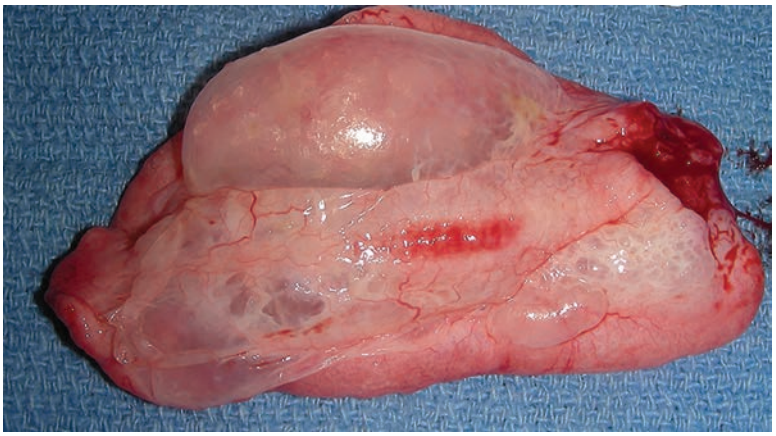


Figure 10.3 Emphysematous Bullae



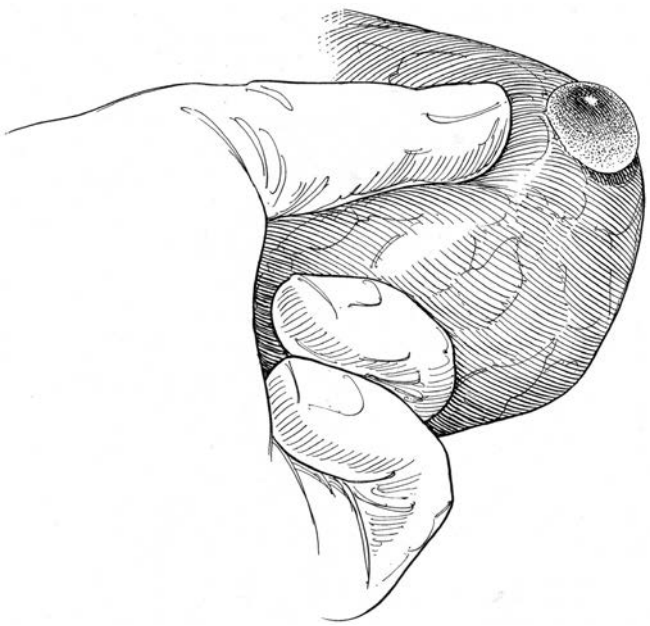


Figure 10.4 Pulmonary Bleb

associated with lower mortality and recurrence rates compared to conservative management with thoracostomy tube alone [3]. In another study of 12 dogs with primary spontaneous pneumothorax secondary to bullous emphysema, surgical resection of the diseased lung resulted in excellent outcome 19 months after surgery [4]. Early surgical exploration and treatment of the underlying pulmonary pathology is the preferred approach for primary and secondary spontaneous pneumothorax. For secondary pneumothorax, the underlying pathology determines the longer-term outcome. Nevertheless, lung lobe resection will typically improve short-term morbidity and mortality.

Exploration of the thorax for spontaneous pneumothorax can be undertaken by surgery or by video-assisted thoracoscopy. Because of the high probability of multiple and bilateral lesions with primary pneumothorax, surgical exploration should be undertaken by sternotomy [3]. Sternotomy allows visualization and palpation of each lung lobe. Video-assisted thoracoscopy has been used successfully to treat primary spontaneous pneumothorax in dogs [11, 12]. A transdiaphragmatic subxyphoid approach with the dog in dorsal recumbency allows exploration of both sides of the thorax, as described in Chapter 6. In a report of three dogs, affected lung lobes were resected during thoracoscopy without the need for one-lung ventilation. Dogs were free of disease 18 to 29 month after surgery [11]. In another report of 12 dogs, conversion to sternotomy was required in 58% of the cases because the lesion could not be identified during thoracoscopy [5]. Successful outcome was recorded in only 50% of the dogs treated by thoracoscopy alone, whereas 83% of dogs treated by sternotomy had a successful long-term outcome [5].

A limitation of thoracoscopic exploration is that it does not allow evaluation of the dorsal aspect of the caudal lung lobes. Further, thoracoscopic lung lobectomy is typically performed with a lateral intercostal approach necessitating a change in the thoracoscopic approach if the lesion cannot be removed by partial lung lobectomy [11].

References

1 Powell L, Rozanski E, and Tidwell A. 1999. A retrospective analysis of pulmonary contusion secondary to motor vehicle accidents in 143 dogs. *Journal of Veterinary Emergency and Critical Care*. 9(1):127–35.

2 Kapatkin AS, and Matthiesen DT. 1991. Feline High-Rise Syndrome. *Compendium on Continuing Education for the Practicing Veterinarian*. 13(9): 1389–94.

3 Puerto DA, Brockman DJ, Lindquist C, and Drobatz K. 2002. Surgical and nonsurgical management of and selected risk factors for spontaneous pneumothorax in dogs: 64 cases (1986-1999). *Journal of the American Veterinary Medical Association*. 220(11):1670–4.

4 Lipscomb VJ, Hardie RJ, and Dubielzig RR. 2003. Spontaneous pneumothorax caused by pulmonary blebs and bullae in 12 dogs. *Journal of the American Animal Hospital Association*. 39(5):435–45.

5 Case JB, Mayhew PD, and Singh A. 2015. Evaluation of Video-Assisted Thoracic Surgery for Treatment of Spontaneous Pneumothorax and Pulmonary Bullae in Dogs. *Veterinary Surgery*. 44(Suppl 1):31–8.

6 Au JJ, Weisman DL, Stefanacci JD, and Palmisano MP. 2006. Use of computed tomography for evaluation of lung lesions associated with spontaneous pneumothorax in dogs: 12 cases (1999-2002). *Journal of the American Veterinary Medical Association*. 228(5):733–7.

7 Oliveira C, Rademacher N, David A, Vasanjee S, and Gaschen L. 2010. Spontaneous pneumothorax in a dog secondary to Dirofilaria immitis infection. *Journal of Veterinary Diagnostic Investigation*. 22(6): 991–4.

8 Mooney ET, Rozanski EA, King RG, and Sharp CR. 2012. Spontaneous pneumothorax in 35 cats (2001-2010). *Journal of Feline Medicine and Surgery*. 14(6):384–91.

9 Reetz JA, Caceres AV, Suran JN, Oura TJ, Zwingenberger AL, and Mai W. 2013. Sensitivity, positive predictive value, and interobserver variability of computed tomography in the diagnosis of bullae associated with spontaneous pneumothorax in dogs: 19 cases (2003-2012). *Journal of the American Veterinary Medical Association*. 243(2):244–51.

10 Thomas EK, and Syring RS. 2013. Pneumomediastinum in cats: 45 cases (2000-2010). *Journal of Veterinary Emergency and Critical Care*. 23(4):429–35.

11 Brissot HN, Dupre GP, Bouvy BM, and Paquet L. 2003. Thoracoscopic treatment of bullous emphysema in 3 dogs. *Veterinary Surgery*. 32(6): 524–9.

12 Case JB, Mayhew PD, and Singh A. 2014. Evaluation of video-assisted thoracic surgery for treatment of spontaneous pneumothorax and pulmonary bullae in dogs. *Veterinary Surgery*. 44 (Suppl 1):31–8.

13 Lansdowne JL, Monnet E, Twedt DC, and Dernell WS. 2005 Thoracoscopic lung lobectomy for treatment of lung tumors in dogs. *Veterinary Surgery*. 34(5):530–5.

14 Mayhew PD, Hunt GB, Steffey MA, Culp WT, Mayhew KN, Fuller M, et al. 2013. Evaluation of short-term outcome after lung lobectomy for resection of primary lung tumors via video-assisted thoracoscopic surgery or open thoracotomy in medium- to large-breed dogs. *Journal of the American Veterinary Medical Association*. 243(5):681–8.

Section IV

General Thoracic Surgery

11

Thymoma and Mediastinal Masses

Eric Monnet

Cranial mediastinal masses include thymoma, ectopic thyroid carcinoma, carotid body tumors, lymphoma, and branchial cyst. Except for lymphoma, these tumors are typically treated surgically via sternotomy or video-assisted thoracoscopy, depending on vascular invasiveness and size. Small well-defined masses that are not invading the cranial vena cava and not wrapping around the major vasculature in the cranial mediastinum can be resected using a thoracoscopic approach.

Thymoma is the most important cranial mediastinal mass. Thymomas are tumors of the epithelial cells of the thymus and are differentiated into epithelial, lymphocyte-rich and clear cell types. Survival is predicted by the degree of invasion by lymphocytes [1]. Cystic thymoma is more common in cats [2]. Thymomas have a low tendency to metastasize, except in cats with cystic thymoma, which have a 20% incidence of metastasis [1–3].

Diagnosis

Clinical signs related to masses in the cranial mediastinum vary depending on the thoracic structures affected. Dogs and cats can present with difficulty breathing and tachypnea due to airway obstruction. Cough is also common clinical sign when the tumor compresses the trachea. Upper respiratory stridor may occur due to injury to the laryngeal recurrent nerves. Cranial caval syndrome characterized by jugular venous distention and swelling of the head is caused by obstruction of the cranial vena cava. Thymoma is most commonly diagnosed in dogs between 9 and 10 years of age. There is no recognized breed predisposition for thymoma in dogs. Paraneoplastic syndromes are common in dogs with thymoma. Myasthenia gravis occurs in 40% of canine cases [1, 3, 4]. Other paraneoplastic syndromes include exfoliating dermatitis, erythema, hypercalcemia, anemia, and polymyositis [1, 3, 4].

Physical examination is usually unremarkable unless obstruction of the cranial vena caval syndrome is present. Auscultation may reveal an absence of lung sounds in the cranial part of the thoracic cavity. Heart sounds may be displaced caudally or dorsally by the mass. Blood work may reveal an anemia, lymphocytosis, and sometimes a thrombocytopenia from immune-mediated destruction. Hypercalcemia has been reported as a paraneoplastic syndrome related to parathyroid-related peptide production. If myasthenia gravis is suspected, a serum titer for acetylcholine receptor antibodies should be conducted.

A mass in the cranial mediastinum may be suspected on thoracic radiographs based on dorsal and lateral deviation of the trachea. The cranial mediastinum appears wide on a ventral-dorsal view with atelectasis of the cranial lung lobes observed when the tumor is large. Megaesophagus may be observed if myasthenia gravis is present. Pleural effusion may be observed, especially if the mass is interfering with the flow of chyle in the thoracic duct. Diagnostic findings for chylous effusion are discussed in Chapter 9.

If a cranial mediastinal mass suspected, ultrasound or image-guided biopsy is undertaken to establish a histologic diagnosis. Fine needle aspiration is preferred because of the risk for profuse bleeding with true cut biopsy. Thyroid carcinoma are typically readily identified based on cytology. Cytology for thymoma reveals a predominance of neoplastic epithelial cells with large numbers of small mature lymphocytes. Flow cytometry is sometimes useful to differentiate lymphoma from thymoma. Thymic lymphocytes are differentiated from circulating lymphocytes by expression of both CD4 and CD8 [5]. Doppler ultrasound is used to evaluate blood flow in the cranial vena cava. Turbulent blood flow and a mass effect visualized in the lumen of the vessel indicate that the tumor has invaded the vena cava.

CT angiogram is a valuable tool for surgical planning. It identifies the extent of the tumor around the

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major structures and vasculature in the cranial mediastinum. CT guided biopsy can also be collected to help differentiate thymoma from lymphoma.

Surgery for Cranial Mediastinal Masses

Cranial mediastinal masses can be treated with surgery, radiation therapy, or chemotherapy depending on tumor type and invasiveness [1, 3, 6]. Median survival of 1,825 days for noninvasive thymoma has been reported in dogs after surgical resection. Median survival falls to 790 days for invasive thymomas treated surgically [1]. Radiation therapy has been associated with a median survival rate of 720 days in dogs and 248 days in cats [6]. The presence of a megaesophagus worsens the prognosis [3].

Sternotomy

Median sternotomy is the approach of choice for surgical excision of cranial mediastinal masses. It provides access to both sides of the cranial mediastinum with a visualization of the brachycephalic trunk and the cranial vena cava. The manubrium is divided leaving the caudal sternebrae and xyphoid intact (Figure 11.1a). After sternotomy careful

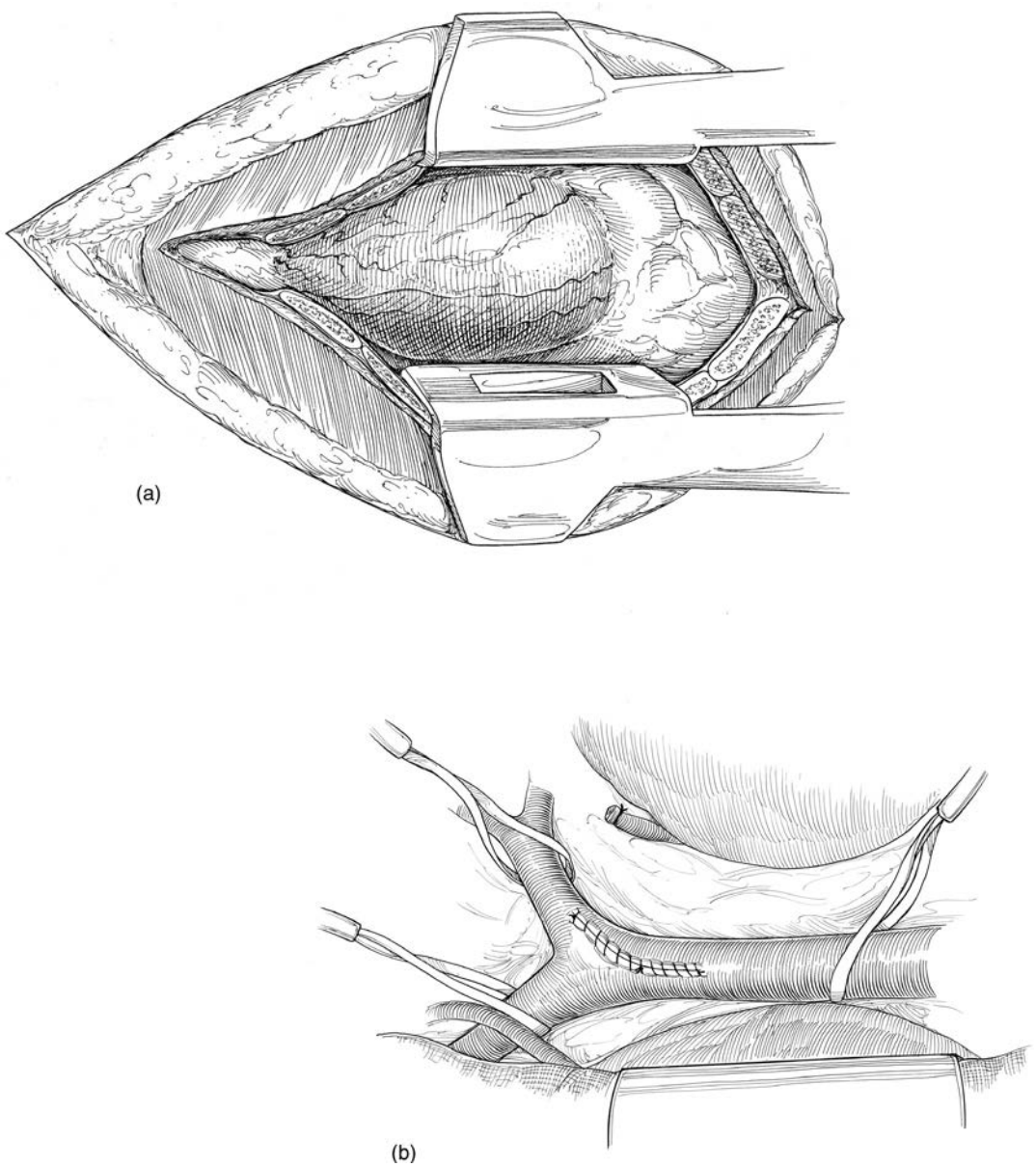

Figure 11.1 Surgical Resection—Thymoma

dissection of the mediastinal tissues are performed to expose the cranial vena cava and its bifurcations. A Finochietto retractor is placed. Both phrenic nerves should be identified and preserved before starting the dissection of the mass. If absolutely necessary, dividing one phrenic nerve will be tolerated in most cases.

Dissection should be conducted as much as possible with electrocautery and/or vessel sealing device to maintain hemostasis. The dissection is performed 360° around the mass. Usually, it is easier to start the dissection cranially and move toward the pericardium. After identifying the major vessels, the dissection is continued along the wall of the vessels. If the internal thoracic arteries are included within the mass, they are ligated and divided. Caudally the mass may be adhered to the pericardium, which will require a partial pericardiectomy.

Thymoma can be either well encapsulated or very diffuse without a well-defined capsule. The encapsulated thymoma usually peels off any major structures and does not envelop the major blood vessels in the cranial mediastinum. Diffuse thymomas have a tendency to envelop major vessels making dissection more difficult and resulting in an incomplete resection. Thymoma and thyroid carcinoma in the cranial mediastinum have a tendency to invade larger blood vessels, specifically the cranial vena cava. In this case, venotomy will be required to remove the intravascular mass. Tourniquets are placed cranial and caudal to the planned site of the venotomy (Figure 11.1b). The tourniquets are occluded and a venotomy is performed with a #11 blade around the suspected point of vascular invasion. Potts scissors are used to extend the incision longitudinally just enough to extract the tumor. Usually, the intravascular portion of the tumor does not attach to the endothelium of the vena cava. If the intravascular tumor extends into the right atrium, then the caudal tourniquet between the mass and the right atrium will not be able to be tightened until the mass has been removed. As soon as the mass is extracted, the tourniquet is tightened to prevent blood loss and air embolism. A tangential clamp can be placed to close the venotomy and the tourniquets are released before suturing. The venotomy is closed with 5-0 monofilament nonabsorbable suture in a simple continuous suture pattern.

Thoracoscopy

Well-defined mediastinal masses that are not invading the cranial vena cava can be resected with by video-assisted thoracoscopy. A CT angiogram is helpful in deciding if the surgery should be attempted by thoracoscopy. A transdiaphragmatic approach is recommended because it allows visualization of both sides of the mass (Figure 11.2). One-lung ventilation has been used, but is not usually necessary [7].

Three or four 5 mm cannulae are required to manipulate and dissect the mass. A 30° telescope is useful to visualize around the base of the tumor and identify the phrenic nerves. The endoscope is placed in the transdiaphragmatic cannula. J hook electrocautery tips are used at the tip of the regular electrocautery handle to facilitate the dissection. Vessel sealant devices (5 mm) are also used during dissection. The cranial mediastinum is incised after visualization of the internal thoracic arteries. A fine-tooth grasping forceps is used to grab the capsule or the mass and the mass is elevated toward the sternum. Dissection is

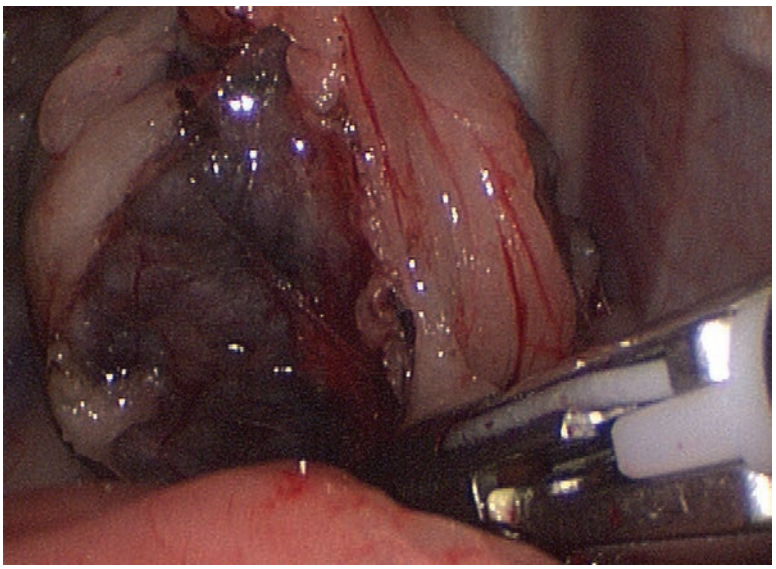

Figure 11.2 Thoracoscopic Resection—Thymoma

performed 360° around the mass while preserving the phrenic nerves. Once the mass is completely freed, it is placed in a retrieval bag to prevent contamination of the thoracic wall [8]. The retrieving cannula incision is enlarged to enable extraction of the tumor. The surgical site is inspected for signs of bleeding.

References

1 Zitz JC, Birchard SJ, Couto GC, Samii VF, Weisbrode SE, and Young GS. 2008. Results of excision of thymoma in cats and dogs: 20 cases (1984–2005). *Journal of American Veterinary Medical Association*. 232(8):1186–92.

2 Patnaik AK, Lieberman PH, Erlandson RA, and Antonescu C. 2003. Feline cystic thymoma: a clinicopathologic, immunohistologic, and electron microscopic study of 14 cases. *Journal of Feline Medicine and Surgery*. 5(1):27–35.

3 Atwater SW, Powers BE, Park RD, Straw RC, Ogilvie GK, and Withrow SJ. 1994. Thymoma in dogs: 23 cases (1980–1991). *Journal of American Veterinary Medical Association*. 205(7):1007–13.

4 Day MJ. 1997. Review of thymic pathology in 30 cats and 36 dogs. *Journal of Small Animal Practice*. 38(9): 393–403.

5 Lana S, Plaza S, Hampe K, Burnett R, and Avery AC. 2006. Diagnosis of mediastinal masses in dogs by flow cytometry. *Journal of Veterinary Internal Medicine*. 20(5):1161–5.

6 Smith AN, Wright JC, Brawner WRJ, LaRue SM, Fineman L, Hogge GS, et al. 2001. Radiation therapy in the treatment of canine and feline thymomas: a retrospective study (1985-1999). *Journal of the American Animal Hospital Association*. 37(5):489–96.

7 Mayhew PD, and Friedberg JS. 2008. Video-assisted thoracoscopic resection of noninvasive thymomas using one-lung ventilation in two dogs. *Veterinary Surgery*. 37(8):756–62.

8 Brisson BA, Reggeti F, and Bienzle D. 2006. Portal site metastasis of invasive mesothelioma after diagnostic thoracoscopy in a dog. *Journal of American Veterinary Medical Association*. 229(6):980–3.

12

Esophagus

E. Christopher Orton

Surgery of the thoracic esophagus in small animals presents some unique challenges based on its anatomical location, unique physiology, and less-than-optimal wound healing. Despite these challenges, surgery of the thoracic esophagus can be successful with careful planning and an understanding of these unique challenges.

Structure and Function

The esophagus consists of cervical, thoracic, and abdominal portions. The thoracic esophagus begins at the thoracic inlet and extends to the esophageal hiatus of the diaphragm. The cranial thoracic esophagus crosses from its position on the left side of the trachea at the thoracic inlet to pass dorsal to the tracheal bifurcation and heart. The esophagus courses over the cardiac base to the right of the aorta and to the left of the vena cavae and azygous vein. If the aorta develops from the right aortic arch, the esophagus passes to the left of the aorta and usually results in a vascular ring anomaly. Vascular ring anomalies are discussed in Chapter 13. The caudal thoracic esophagus travels within the mediastinum and passes into the abdomen with the vagal nerves via the esophageal hiatus.

The esophageal wall is comprised of the adventitia, muscularis, submucosa, and mucosa layers. The adventitia consists of loose connective tissue and is incompletely surrounded by mediastinal pleural as it courses through the thorax. The lack of a complete and intimate serosal layer is cited as one of the reasons for less-than-optimal wound healing. The muscularis is composed of striated muscle over the entire length of the esophagus in the dog. In cats the muscularis changes to smooth muscle over its terminal portion. The muscularis is organized into two indistinct spiral layers at right angles to each other. The muscularis layer thickens at the gastroesophageal junction, which correlates with the high-pressure zone at the gastroesophageal junction. The submucosa loosely connects the mucosa and muscularis layers, allowing them to move independently during the esophageal phase of swallowing. Importantly, the submucosa contains a rich network of blood vessels and innervation that must be preserved as much as possible during esophageal surgery. The submucosa also contains simple mucous glands that lubricate the esophagus. The mucosa is comprised of stratified squamous endothelium that is thrown into numerous longitudinal folds when the esophagus is not distended.

The arterial blood flow to the esophagus is segmental. From cranial to caudal, it is provided by the cranial and caudal thyroid arteries, bronchoesophageal arteries, esophageal branches of the thoracic aorta, and the left gastric artery. Venous drainage is accomplished by veins that largely accompany the arterial blood supply. In the caudal thorax, these veins drain to the azygous vein. These arteries and veins supply a rich plexus of vessels within the submucosal layer that allows a degree of collateralization of blood flow.

Innervation of the esophagus is supplied by branches of the vagus nerve, beginning with the pharyngoesophageal nerves, followed by the recurrent laryngeal nerves and ending with the dorsal and ventral vagal trunks. The primary role of the esophagus is to deliver food and liquid from the oropharynx to the stomach. Primary and secondary peristaltic waves are involuntary and are initiated by distension of the esophagus as the food bolus is delivered to the cranial esophagus. Relaxation of the muscularis just ahead of the food bolus helps propel the bolus. Because esophageal function in small animals is entirely dependent on striated muscle, vagal innervation to the esophagus must be preserved to avoid dysfunction and the devastating complication of megaesophagus. Preserving the vagus nerves is critical for all thoracic surgery.

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Surgical Conditions of the Esophagus

Esophageal Foreign Bodies

Esophageal foreign bodies are the most common problem of the esophagus in dogs, and are also occasionally seen in cat [1, 2]. Bones are the most frequent foreign body in dogs; whereas hooks, needles, and linear foreign bodies are more common in cats. Other types include chew treats, balls, wooden sticks, and toys. Foreign bodies tend to lodge at sites of natural narrowing, which are the thoracic inlet, base of the heart, and the caudal esophagus just cranial to the diaphragm. Clinical signs include regurgitation of solid food soon after eating, retching, ptyalism, restlessness, inappetence, weight loss, or lethargy. Penetration of the esophagus can cause serious complications, including pneumomediastinum, pneumothorax, pleuritis, pyothorax, or bronchoesophageal fistula. Many esophageal foreign bodies can be removed with forceps, repelled orally with a balloon catheter, or advanced into the stomach under fluoroscopic or endoscopic guidance. Very large or sharp foreign bodies, location in the caudal esophageal or at the heart base, chronicity of clinical signs, and/or evidence of esophageal penetration are reasons to consider surgery over more conservative approaches. Surgical options include esophagotomy or removal via a gastrotomy. Esophagotomy has a >90% success rate and similar complication rate compared to endoscopic foreign body removal, so there is little reason to avoid a surgical approach when it is indicated or when conservative removal is not successful [3, 4].

Esophageal Strictures

Esophageal stricture is uncommon in small animals. They can be congenital or acquired. Acquired strictures result from circumferential injury to the mucosa and submucosa with resulting fibrosis and wound contracture. Causes of acquired strictures include esophageal reflux during anesthesia, chronic vomiting, gastric reflux, ingestion of corrosive substances or drugs (e.g., doxycycline), thermal or radiation injury, or foreign bodies [5]. The most important clinical sign is regurgitation of solid food, plus or minus liquids. History of recent anesthesia, administration of a corrosive drug, or chronic vomiting are supportive of the diagnosis. Diagnosis is confirmed by contrast esophagogram and/or esophagoscopy. Contrast esophagography should include fluoroscopic evaluation of esophageal motility. The most common site for esophageal stricture is the caudal thoracic esophagus. Serial esophageal dilation by balloon catheter or bougienage is the preferred initial treatment for esophageal stricture [6,7]. Both techniques carry a risk of esophageal rupture or penetration. Most animals require multiple dilations. Surgical options include esophageal resection and anastomosis or esophagoplasty with an inlay patch.

Esophageal Diverticula

Esophageal diverticula are uncommon in dogs and cats. Diverticula are categorized as congenital or acquired. Acquired diverticula are further separated into pulsion or traction types. Pulsion diverticula are characterized by herniation of the mucosa through the muscularis and adventitial layers. They are thought to result from high lumen pressures generated by a functional or mechanical obstruction. In dogs, pulsion diverticula occur almost exclusively in the caudal thoracic (epiphrenic) esophagus. Traction diverticula result from inflammation and adhesion of the esophagus to an adjacent structure or organ causing outward radial traction and formation of a full-thickness pouch in the esophageal wall. Traction diverticula are sometimes associated with a concurrent bronchoesophageal fistula. Clinical signs associated with esophageal diverticula are similar to other esophageal disorders, or they may be an incidental finding without symptoms. Diagnosis is confirmed by contrast esophagogram and/or esophagoscopy. Surgery is directed at understanding and treating the underlying cause(s). In the case of pulsion diverticula, this means ruling out and treating functional or mechanical obstruction. A large pulsion diverticulum may be treated by excision of the herniated mucosa (diverticulectomy) followed by standard two-layer closure of the esophagus. Traction diverticula may not require surgical intervention unless accompanied by a bronchoesophageal fistula.

Esophageal Fistulae

Esophageal fistulae are abnormal communications between the esophagus and trachea, bronchus, or lung parenchyma. Congenital fistulae result from incomplete separation between the digestive tract and tracheobronchial tree. Congenital esophageal fistulae have been reported in dogs and cats. In dogs, acquired esophageal fistulae are most often a sequela to an esophageal foreign body. Bronchoesophageal fistulae are more common than bronchotracheal fistulae. The right caudal lung is most commonly affected in dogs. Bronchoesophageal fistulae may be accompanied by a traction type esophageal diverticula. Clinical signs are referable to both esophageal dysfunction and chronic pulmonary infection. Coughing may be exacerbated

by drinking fluids. Esophageal fistulae require surgical management. The fistula is divided and the esophageal defect is closed primarily. Pathology in the communicating lung lobe is usually extensive necessitating lung lobectomy.

Esophageal Neoplasia

Primary esophageal neoplasia occurs in both dogs and cats. In dogs, the most common location is in the caudal thoracic esophagus. Tumor types include squamous cell carcinoma, leiomyosarcoma, osteosarcoma, fibrosarcoma, undifferentiated sarcoma, and leiomyoma [8, 9]. Sarcomas of the caudal thoracic esophagus in dogs have a strong association with migration of the *Spirocerca lupi* nematode parasite found in tropical and subtropical regions [10]. In cats, the most common primary esophageal neoplasia is squamous cell carcinoma and the most frequent site is the cranial thoracic esophagus. Clinical signs are referable to esophageal dysfunction and the systemic manifestations of neoplasia. Surgical management of esophageal neoplasia is possible in selected cases. Benign tumors such as leiomyomas have been successfully treated by partial thickness longitudinal resection without opening the mucosa [9]. Malignant neoplasms are often advanced at diagnosis and have a high prevalence of metastasis. Resection of malignant esophageal neoplasms in dogs may be possible in selected cases after complete staging to rule out metastasis. Partial-circumference full-thickness esophagectomy with surgical margins has been successful in the management of esophageal sarcomas in dogs [11]. Circumferential resection and anastomosis may be necessary in more advanced esophageal neoplasia. A reliable method for esophageal substitution after extensive resection of the thoracic esophagus has not been demonstrated in small animals, and therefore cannot be recommended.

Surgery of the Esophagus

Surgery of the esophagus presents some unique challenges. Complication rates are historically higher than other portions of the alimentary tract. Several factors are cited as contributing to a higher complication rate, including the absence of a complete serosa or sealing omentum, motion associated with swallowing and respiration, incisional tension caused by inability to mobilize long segments of the esophagus, and the segmental nature of esophageal blood supply and innervation. Despite these challenges, surgery of the esophagus can be successful by acknowledging these limitations and adhering to certain principles. Preservation of the segmental blood supply and innervation of the esophagus is important to successful healing and maintaining function after surgery. Although esophageal blood supply is segmental, the rich network of submucosal vessels is capable of supporting long segments of the esophagus. However, this emphasizes the need for careful tissue handling, avoidance of electrocautery, and accurate suture placement. The esophagus is preferably closed in two layers. The mucosa is closed with knots placed within the lumen to minimize disruption of the submucosal vascular network. Because esophageal musculature in small animals is composed entirely of skeletal muscle, function is completely dependent on maintaining innervation both proximal and distal to the segment undergoing surgery. Injury to any of the major branches of the vagus nerve supplying the esophagus risks esophageal dysfunction and megaesophagus after surgery.

Excessive tension on an esophageal anastomosis increases the likelihood of dehiscence. This devastating complication is primarily avoided by recognizing limitations on how much of the esophagus can be reasonably resected. As a rule of thumb, esophageal resections should not be greater than about 20% of the length of the cranial or caudal thoracic esophagus. After esophageal surgery, the esophagus should be rested by withholding oral food and water for at least 72 hours. During this period nutritional support can be provided via gastrostomy or enterostomy feeding.

The lack of a complete serosal layer is often cited as a reason for higher dehiscence rates associated with surgery on the thoracic esophagus. The importance of this factor may be somewhat overstated, as it has been shown that healing of other portions of alimentary tract is not impaired by removal of the serosa. However, the omentum does likely provide and important safety net in support of healing of the alimentary tract within the abdomen. Omentopexy by mobilization of the omentum through the diaphragm has been shown to support esophageal healing and decrease stricture formation in dogs. This technique should be considered if there is reason to believe that healing might be delayed or compromised [12]. Techniques for mobilizing vascularized omental pedicle grafts in dogs and cats have been described [13].

Approaches to the Thoracic Esophagus

Choosing an appropriate thoracic approach is an important aspect of planning for surgery on the thoracic esophagus. A cranial sternotomy provides

access to the cranial thoracic esophagus from the thoracic inlet to the base of the heart. The esophagus is located to the left of the trachea at the thoracic inlet and crosses to the dorsal aspect of the trachea as it courses toward the heart. A sternotomy can be extended into a ventral cervical incision to expose the cervical esophagus. The cranial thoracic esophagus can also be accessed via a right or left third or fourth thoracotomy.

Exposure from a left thoracotomy requires ventral traction of the brachiocephalic and left subclavian arteries. Care should be taken from a left thoracotomy to avoid injury to the thoracic duct as it courses dorsal to the left subclavian artery. The esophagus normally passes to the right of the ascending aorta, so access to the esophagus over the base of the heart is best gained from a right fifth thoracotomy. Exposure over the base of the heart is facilitated by ventral retraction of the tracheal bifurcation and cranial vena cava. The azygous vein crosses over the right side of the esophagus at the heart base. Passage of stay sutures around the azygous vein aids with retraction. If absolutely necessary, the azygous vein can be ligated and divided. If the aorta develops from a persistent right aortic arch, then the esophagus courses to the left of the aorta as seen with most vascular ring anomalies (see Chapter 13). Preferred access to the caudal thoracic esophagus is via a left seventh to ninth thoracotomy to avoid the caudal vena cava. Care is taken to avoid injury to the dorsal and ventral vagal trunks, which course on the caudal thoracic esophagus.

Esophagotomy

The primary indications for esophagotomy are removal of esophageal foreign bodies, repair of penetrating injuries, correction of diverticula or fistulae, and resection of smaller esophageal tumors. Esophagotomy is usually a longitudinal full-thickness incision in the esophageal wall (Figure 12.1a). Sometimes it may be preferable to make the incision just proximal or distal to the site of a foreign body to avoid tissue that might have been compromised by the foreign body. Circumferential tapes are placed around the esophagus proximal and distal to the planned incision site to help control spillage from the lumen taking care not to injure the vagal trunks (Figure 12.1b). Luminal mucosa is inspected for evidence pressure necrosis or penetrating injury. The incision is closed with interrupted sutures in two layers to allow the mucosa and muscularis to move independently during esophageal contraction and distension. The mucosa and submucosa are closed together with interrupted sutures of 4-0 monofilament suture with the knots placed in the lumen (Figure 12.1c). The muscularis is closed mattress sutures placed loosely to minimize disruption of the blood supply.

Transdiaphragmatic Gastrotomy

An alternative approach for removal of foreign bodies in the caudal thoracic esophagus is by transdiaphragmatic gastrotomy. Access to the stomach is gained by an incision in the diaphragm (Figure 12.2a). The foreign body is grasped and removed through a gastrotomy (Figure 12.2b). This approach avoids the need for an esophagotomy, which tends to heal more slowly and be more susceptible to dehiscence compared to gastrotomy. The transdiaphragmatic approach is preferred over an abdominal approach because it allows direct visualization of the esophagus during foreign body removal and inspection after removal for evidence of penetrating injury or necrosis.

Esophageal Resection and Anastomosis

Esophageal resection and anastomosis is indicted for treatment of localized strictures that have failed serial dilation, severe traumatic injuries or necrosis caused by foreign bodies, and esophageal neoplasia. Circumferential tapes are placed proximal and distal to the planned resection to isolate the portion of the esophagus that is to be resected (Figure 12.3a). Mobilization of the esophagus should be kept to the minimum necessary to accomplish the resection for avoid disruption of blood supply. Vagal nerves are preserved proximal and distal to the resection. Anastomosis is completed in two layers with interrupted sutures. The mucosa and submucosa is closed with 4-0 monofilament sutures with knots placed in the lumen (Figure 12.3b). The muscularis and adventitial layers are closed with interrupted mattress sutures (Figure 12.3c).

Esophageal Patching

On-lay esophageal patching is used to reinforce esophageal incisions that are at risk of dehiscence because of compromised vascularity and/or incisional tension. In-lay esophageal patches are used to increase the luminal circumference of esophageal strictures. Pedicles for patching the thoracic esophagus can be developed locally from the pericardium, intercostal muscles, or diaphragm [14, 15]. Omental pedicle flaps

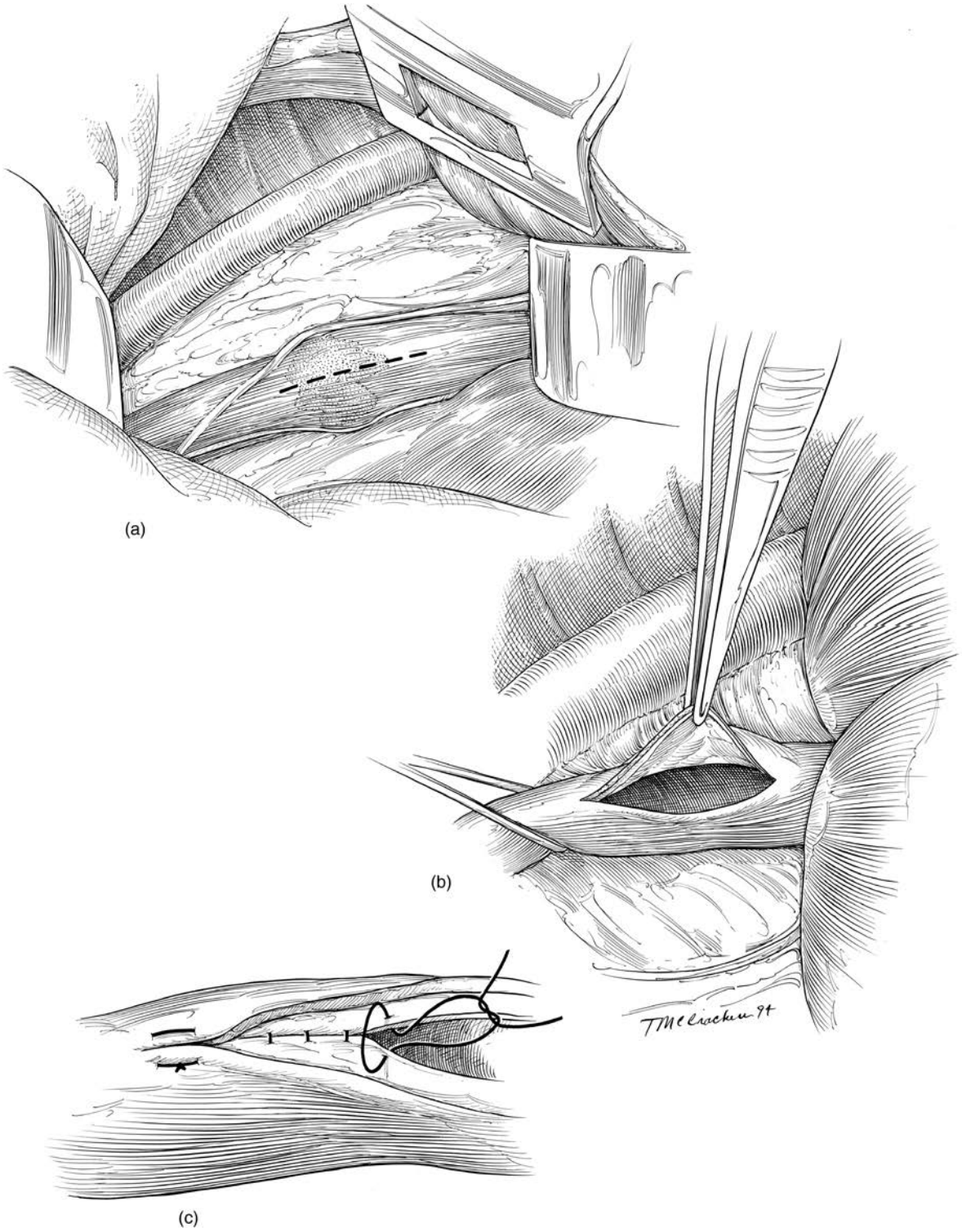

Figure 12.1 Esophagotomy

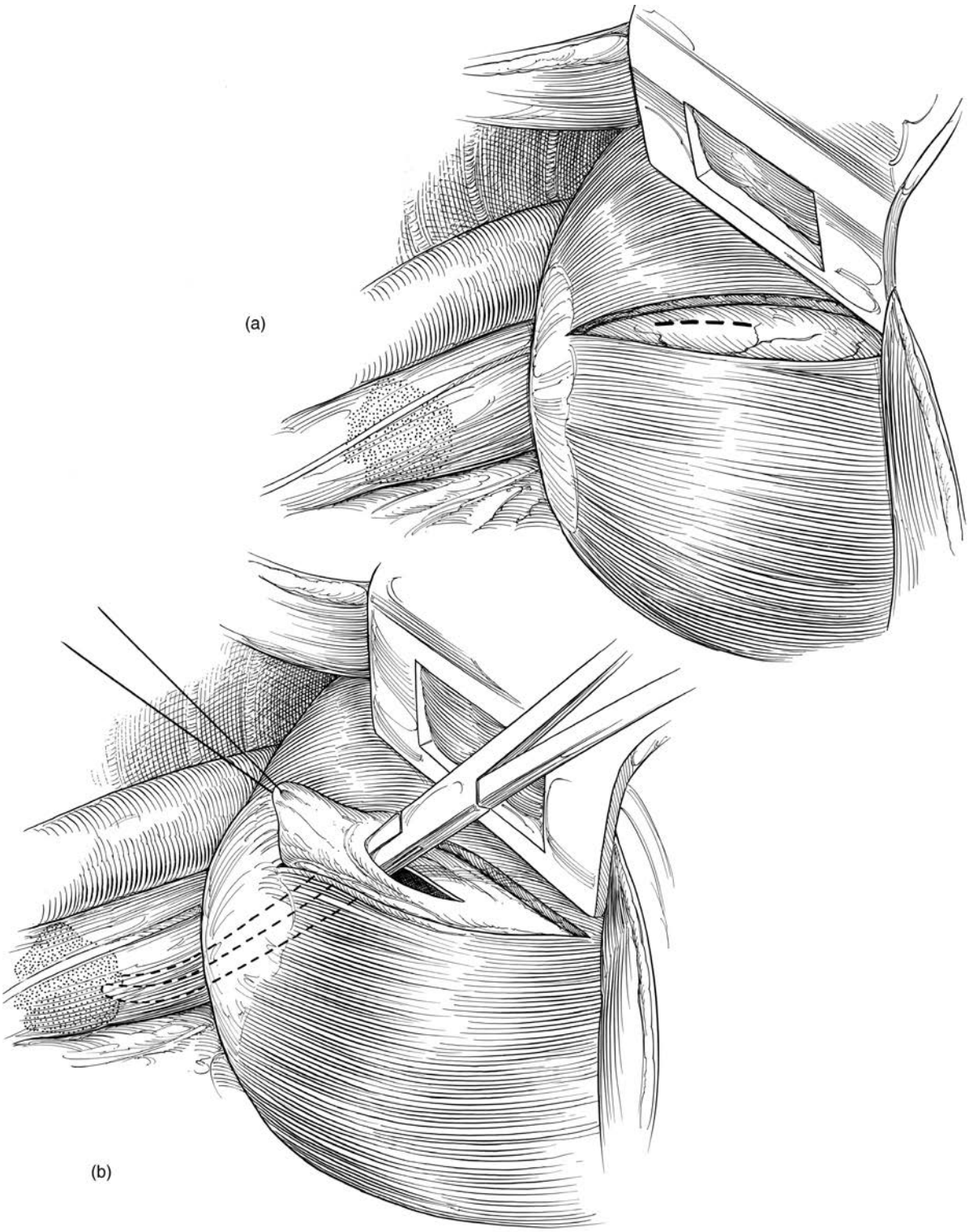

Figure 12.2 Transdiaphragmatic Gastrotomy

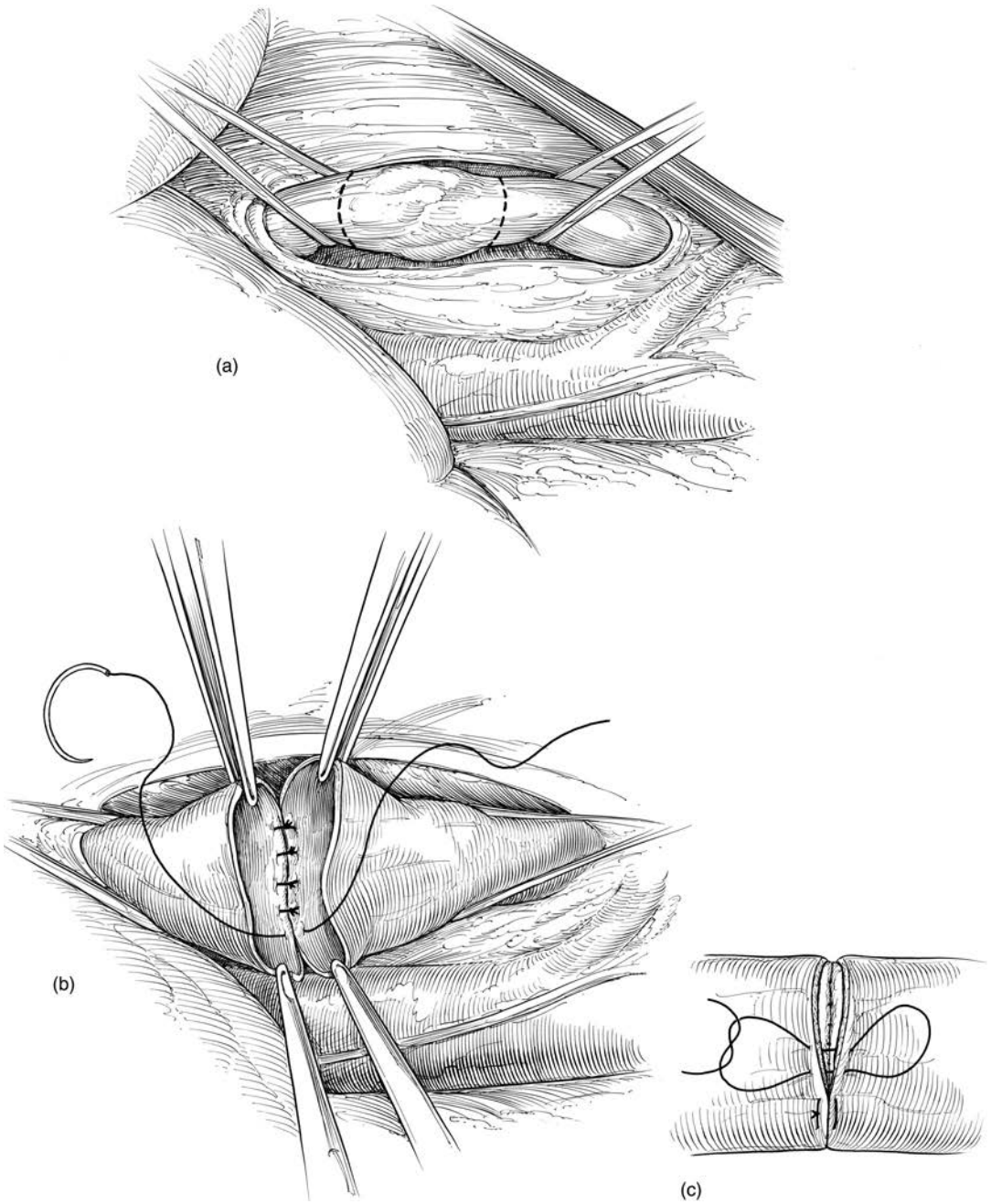

Figure 12.3 Esophageal Resection and Anastomosis

can be brought through the diaphragm to support the caudal thoracic esophagus [12]. Patching of the caudal thoracic esophagus can be accomplished by developing a pedicle of diaphragmatic muscle with its based at the esophageal hiatus (Figure 12.4a). For an in-lay patch, a longitudinal incision is made over the stricture (Figure 12.4b). Sutures are preplaced around the circumference of the incision and edges of the pedicle flap. Preplaced sutures are tied, and the defect in the diaphragm is closed (Figure 12.4c).

Esophageal Substitution

Direct esophageal replacement of the caudal thoracic esophagus with a small intestinal or colonic pedicle graft is prevented by the limited mobility of the vascular pedicle in small animals. A number of techniques for substitution of segments of the thoracic esophagus have been evaluated experimentally in dogs [16–20]. So far, none have been demonstrated to be successful in the clinical setting.

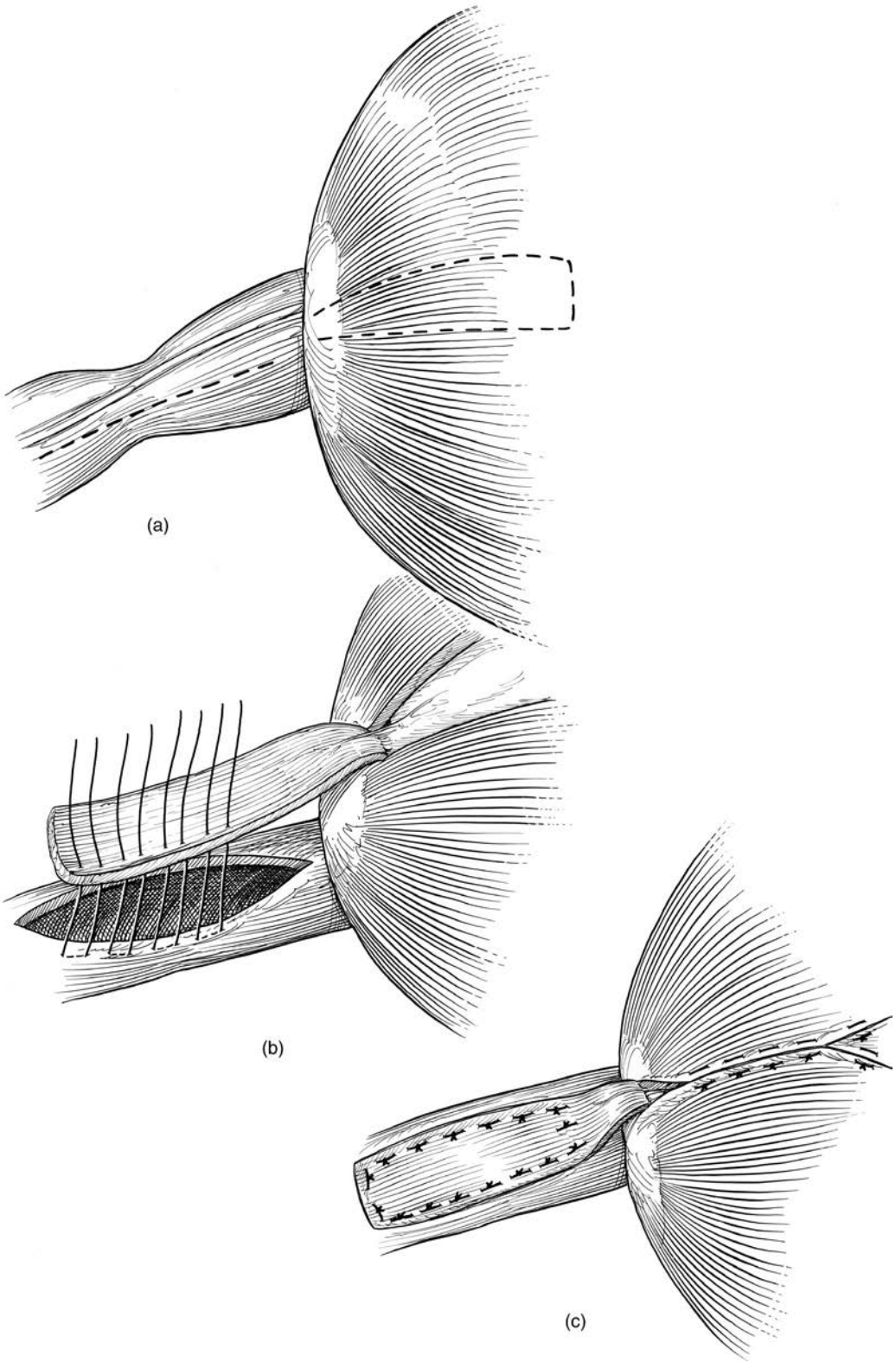

Figure 12.4 Esophageal In-Lay Patch from Diaphragm

References

1 Houlton JEF, Herrtage ME, Taylor PM, and Watkins SB. 1985. Thoracic oesophageal foreign bodies in the dog: A review of ninety cases. *The Journal of Small Animal Practice*. 26:521–36.

2 Gianella P, Pfammatter NS, and Burgener IA. 2009. Oesophageal and gastric endoscopic foreign body removal: Complications and follow-up of 102 dogs. *The Journal of Small Animal Practice*. 50(12): 649–54.

3 Deroy C, Corcuff JB, Billen F, and Hamaide A. 2015. Removal of oesophageal foreign bodies: Comparison between oesophagoscopy and oesophagotomy in 39 dogs. *The Journal of Small Animal Practice*. 56(10): 613–7.

4 Sale CS, and Williams JM. 2006. Results of transthoracic esophagotomy retrieval of esophageal foreign body obstructions in dogs: 14 cases (2000-2004). *Journal of the American Animal Hospital Association*. 42(6):450–6.

5 Adamama-Moraitou KK, Rallis TS, Prassinos NN, and Galatos AD. 2002. Benign esophageal stricture in the dog and cat: A retrospective study of 20 cases. *Canadian Journal Veterinary Research*. 66(1): 55–9.

6 Harai BH, Johnson SE, and Sherding RG. 1995. Endoscopically guided balloon dilatation of benign esophageal strictures in 6 cats and 7 dogs. *Journal of Veterinary Internal Medicine*. 9(5):332–5.

7 Bissett SA, Davis J, Subler K, and Degernes LA. 2009. Risk factors and outcome of bougienage for treatment of benign esophageal strictures in dogs and cats: 28 cases (1995–2004). *Journal of the American Veterinary Medical Association*. 235(7): 844–50.

8 Ranen E, Dank G, Lavy E, Perl S, Lahav D, and Orgad U. 2008. Oesophageal sarcomas in dogs: histological and clinical evaluation. *Veterinary Journal*. 178(1):78–84.

9 Rolfe DS, Twedt DC, and Seim HB. 1994. Chronic regurgitation or vomiting caused by esophageal leiomyoma in three dogs. *Journal of the American Animal Hospital Association*. 30:425–30.

10 Ranen E, Lavy E, Aizenberg I, Perl S, and Harrus S. 2004. Spirocercosis-associated esophageal sarcomas in dogs. A retrospective study of 17 cases (1997-2003). *Veterinary Parasitology*. 119(2–3): 209–21.

11 Ranen E, Shamir MH, Shahar R, and Johnston DE. 2004. Partial esophagectomy with single layer closure for treatment of esophageal sarcomas in 6 dogs. *Veterinary Surgery*. 33(4):428–34.

12 Hayari L, Hershko DD, Shoshani H, Maor R, Mordecovich D, and Shoshani G. 2004. Omentopexy improves vascularization and decreases stricture formation of esophageal anastomoses in a dog model. *Journal of Pediatric Surgergy*. 39(4):540–4.

13 Hosgood G. 1990. The omentum—the forgotten organ: physiology and potential surgical applications in dogs and cats. *Compendium of Continuing Education for the Practicing Veterinarian*. 12:45–51.

14 Hopper CL, Berk PD, and Howes EL. 1963. Strength of esophageal anastomoses repaired with autogenous pericardial grafts. *Surgery Gynecology Obstetrics*. 117:83–6.

15 Bryant LR, and Eiseman B. 1965. Experimental evaluation of intercostal pedicle grafts in esophageal repair. *J Thoracic Cardiovascular Surgery*. 50(5): 626–31.

16 Kuzma AB, Holmberg DL, Miller CW, Barker I, and Roth J. 1989. Esophageal replacement in the dog by microvascular colon transfer. *Veterinary Surgery*. 18(6):439-45.

17 Yamataka A, and Miyano T. 1994. Tubed latissimus dorsi musculocutaneous flaps for thoracic esophageal replacement in dogs: Possible clinical application. *Surgery Today*. 24(9):852–3.

18 Paulo NM, Miranda W, Atayde IB, Siqueira Junior JT, Azevedo EM, Lima FG, Franco LG, and Faria CM. 2007. Reconstruction of thoracic esophagus with pediculated diaphragmatic flap in dogs. *Acta Cirurgica Brasileira*. 22(1):8–11.

19 Moutsouris C, Barouchas G, Karayannacos P, Dontas I, Salakos C, and Skalkeas G. 1987. Tubular musculopleural pedicle grafting of esophageal long gaps in dogs. *Journal Pediatric Surgery*. 22(2):117–9.

20 Henderson RD, Boszko A, Mugashe F, Szczepanski MM, and Marryatt G. 1974. Oesophageal replacement by a gastric tube: an experimental study of the properties of the gastric tube. *British Jounral Surgery*. 61(7):533–7.

13

Vascular Ring Anomalies

Eric Monnet

During normal embryological development, the first and second aortic arches largely regress, except for contributions to the internal and external carotid arteries. The common carotid arteries develop from right and left third aortic arch. The right fourth aortic arch gives rise to the brachiocephalic and right subclavian arteries, whereas the left fourth aortic arch gives rise to the arch of the aorta. The fifth aortic arches regresses bilaterally. The right and left sixth aortic arches give rise to left and right pulmonary arteries respectively. The left sixth aortic arch also gives rise to ductus arteriosus, which regresses after birth to become the ligamentum arteriosum. The left subclavian artery develops from the seventh intersegmental artery. Vascular ring anomalies result when this pattern is broken—more specifically, when one or more of the vascular structures that normally develop from the left fourth or sixth aortic arches develop from the right side. The result is formation of a vascular ring that either completely or partially obstructs of the esophagus from birth.

Types of Vascular Ring Anomaly

Normal embryonic development gives rise to an aorta, ligamentum arteriosum, and left subclavian artery positioned to the left of the esophagus (Figure 13.1a). Several types of vascular ring anomalies have been reported in dogs and cats [1–13]. A persistent right fourth aortic arch resulting in an ascending aorta and aortic arch positioned to the right of the esophagus is the most common vascular ring anomaly in dogs and cats [7]. In this anomaly, the ligamentum arteriosum develops from the left side, resulting in a complete vascular ring (Figure 13.1b). This type of vascular ring anomaly represents 95% of the cases observed [3, 7, 13]. A partial vascular ring is formed when an aberrant left subclavian artery arises from the right side, compressing the esophagus cranial to the base of the heart (Figure 13.1c). Aberrant left subclavian artery occurs either as a sole defect or combination with persistent right fourth aortic arch. Aberrant left subclavian arteries with unusual origin from a patent ductus arteriosus have been described [12,14,15]. Persistence of both fourth aortic arches results in formation of a double aortic arch with entrapment of the esophagus between the arches (Figure 13.1d) [2, 4, 6, 8, 9, 16, 17]. The arch of the aorta can develop normally from the left side, with the ligamentum arteriosum developing from the right side, resulting in a complete vascular ring that is a mirror image of persistent right fourth aortic arch (Figure 13.1e). Lastly, an aberrant right subclavian artery can form from the left side resulting in partial esophageal obstruction (Figure 13.1f) [5,18]. The combination of both a right ligamentum arteriosum and an aberrant right subclavian artery is possible.

Diagnosis

Dogs and cats with a vascular ring anomaly present with a primary complaint of regurgitation, typically starting after transition from liquid to more solid diet. Affected animals are smaller than their littermates despite a good appetite. Intermittent bulging of the neck is often observed. Respiratory signs related to aspiration pneumonia are often present. The presence of a continuous heart murmur suggests that the ductus arteriosus has remained patent, which has implications for surgery.

Thoracic radiographs with and without barium demonstrate a dilation of the esophagus cranial to the heart base. It is not unusual to see foreign materials and food cranial thoracic cavity that narrows at the level of the heart base. Narrowing of the esophagus cranial to the heart base suggests the possibility

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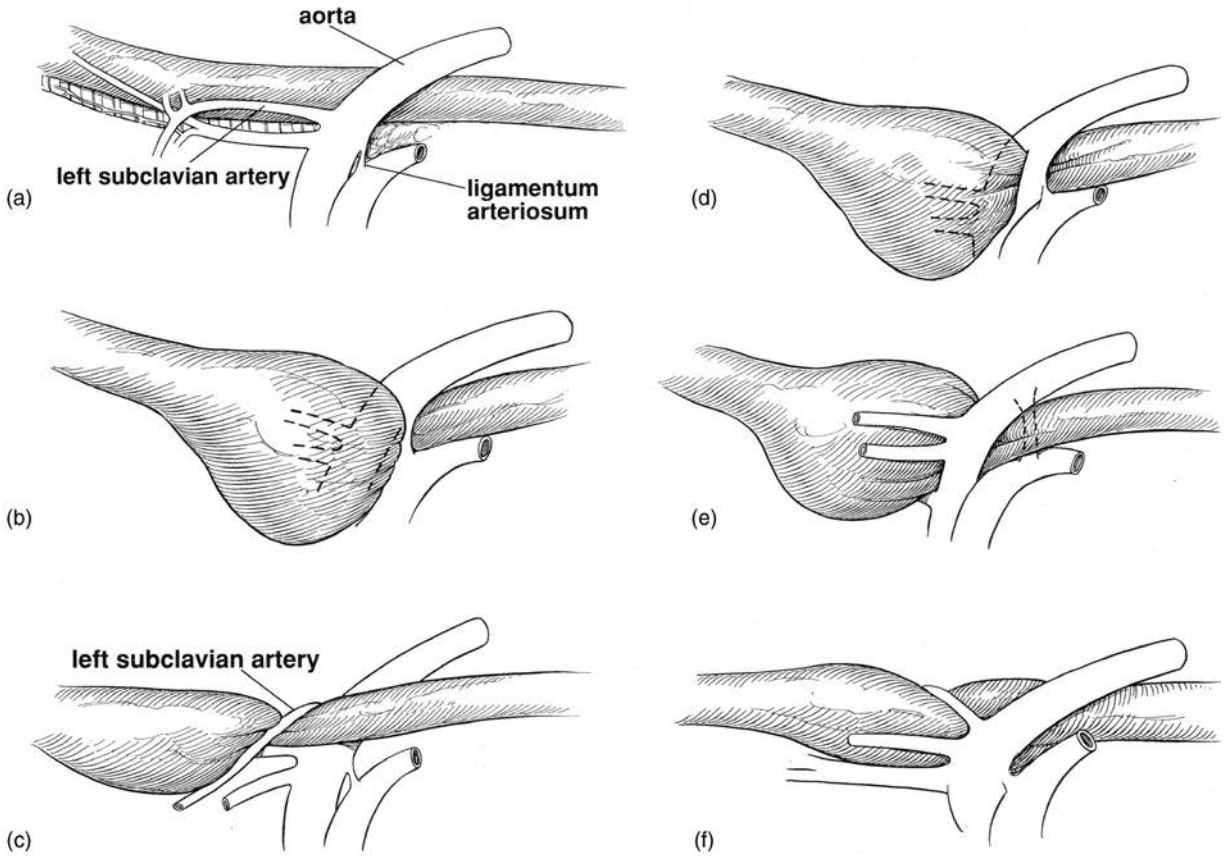

Figure 13.1 Types of Vascular Ring Anomaly

of an aberrant subclavian artery. In a case series of dogs, leftward deviation of the trachea cranial to the heart base on the ventral-dorsal radiograph is present in 100% of the cases with persistent right aortic arch [7]. Narrowing of the trachea on ventrodorsal or dorsoventral views was present in 75% of the cases. The segment of the esophagus distal to the heart base typically is not dilated and exhibits normal peristaltic waves. CT angiography has been used to further define the anatomy of vascular ring anomalies in dogs and cats [19].

Endoscopy of the esophagus can be used to confirm a persistent right aortic arch to the right side of the esophagus (Figure 13.2 white arrows) with a left ligamentum arteriosum (black arrows). This observation helps determine whether the surgical approach should be a left or right thoracotomy. Bilateral pulsations indicate a double aortic arch. In this case, angiography may be necessary to determine which aortic arch is more prominent and thereby the preferred side for the thoracic approach [4, 6, 8, 9, 16, 17].

Surgery

The goal of the surgical treatment is to relieve the esophageal obstruction. Surgical correction of vascular ring anomaly is often palliative because of persistent dysfunction of the cranial esophagus after the obstruction is relieved. Most cases need medical management, including elevated feeding for the rest of their life. Nevertheless, surgery typically significantly decreases the severity of the clinical signs in the majority of cases and owners report satisfaction with the outcome [3, 13]. Surgical correction can be undertaken by intercostal thoracotomy or video-assisted thoracoscopy, depending on type of vascular ring anomaly and patency of the ductus arteriosus.

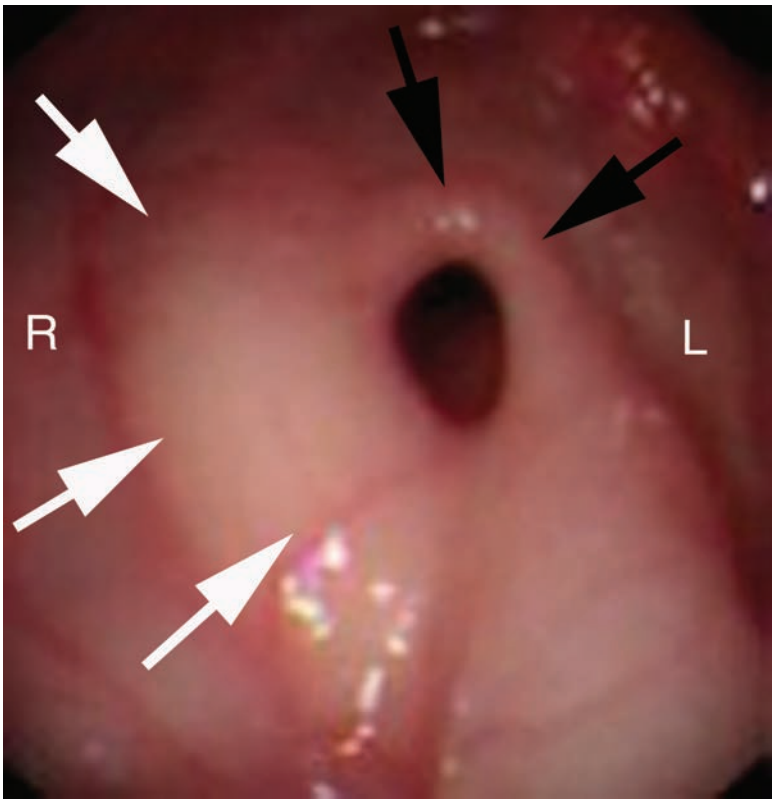

Figure 13.2 Endoscopic View of Persistent Right Aortic Arch

Persistent Right Aortic Arch with Left Ligamentum Arteriosum

Relief of persistent right aortic arch with left ligamentum is accomplished by dividing the ligamentum from a left-sided thoracic approach. Occasionally, a left hemiazygos vein and/or persistent left cranial vena cava will be present. A left hemiazygos vein should be dissected, ligated, and divided because it may contribute to the esophageal obstruction [1]. A persistent left cranial vena cava should be maintained by retracting it dorsally or ventrally to gain access to the ligamentum.

Thoracotomy

The surgical approach to a persistent right aortic arch with left ligamentum is a left fourth or fifth intercostal thoracotomy. The left cranial lung lobe is retracted caudally. The ascending aorta and aortic arch are medial to the esophagus. The esophagus is dilated cranial to the base of the heart. The ligamentum arteriosum is identified between the descending aorta and left pulmonary artery (Figure 13.3a). The vagus nerve coursing over the ligamentum is identified and preserved by retracting it ventrally or dorsally. The pleura and adventitial tissues surrounding the ligamentum arteriosum are dissected with electrocautery. The ligamentum arteriosum is isolated, taking care not to perforate the esophagus. The ligamentum is double ligated and transected between the ligatures (Figure 13.3b). The ligamentum should not be divided without ligation because of the risk of undetected residual patency. After dividing the ligamentum, it is necessary to divide any residual fibrous tissue contributing to obstruction of the esophagus. This is aided by passing a large Foley catheter into the esophagus with the balloon inflated to help identify residual fibers.

Video-Assisted Thoracoscopy

The animal is placed in right lateral recumbency for intercostal thoracoscopy. Four portals will be required. Three portals are placed in the seventh or eighth intercostal spaces and a fourth is placed one intercostal space cranial in the ventral third of the thorax. A retractor or a palpation probe is introduced into the lower portal for retraction of the lungs.

Five millimeter (5 mm) equipment is typically used for the procedure. Because many dogs are very young and weight less than 8 kg, a pediatric set of equipment may be necessary. Pediatric equipment is usually 2.7 mm in diameter. One 5 mm portal will still

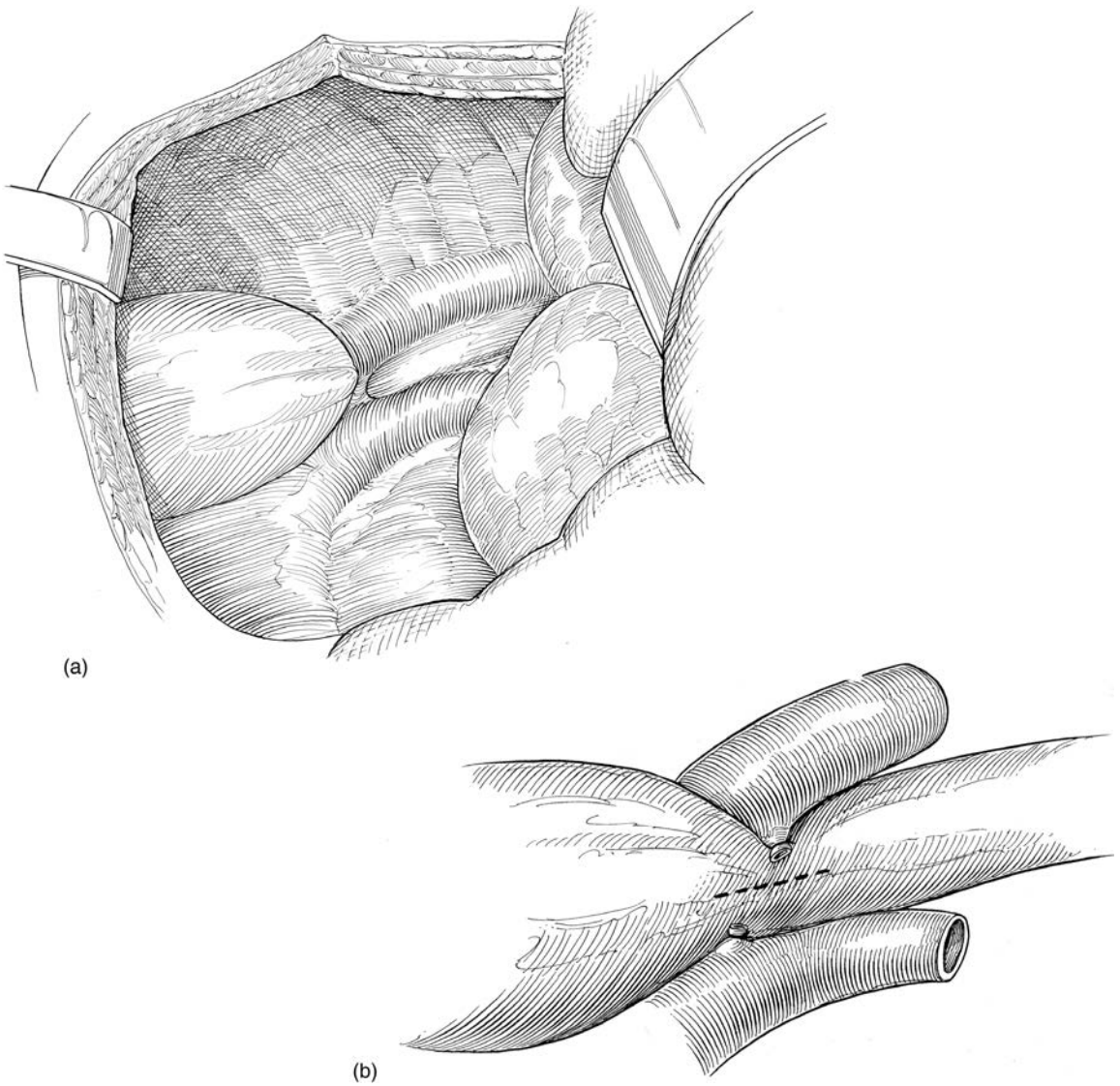

Figure 13.3 Surgery for Persistent Right Aortic Arch

be needed for introduction of a vascular clip or vessel sealant device.

The left cranial lung lobe is retracted ventrally to expose the cranial mediastinum and the heart base (Figure 13.4a). The ligamentum arteriosum is located by identifying the point of narrowing of the dilated esophagus at the base of the heart. A stomach tube can be passed in the esophagus to help identify the ligamentum arteriosum. A palpation probe is used to further localize the ligamentum arteriosum (Figure 13.4b). The vagus nerve coursing over the ligamentum is identified and preserved. The ligamentum arteriosum is isolated by sharp and blunt dissection. Endoscopic 5 mm vascular clips are placed and the ligamentum is transected between the clips (Figure 13.4c). Alternatively, a vessel sealant device can be used to seal the ligamentum before transection. Any remaining fibers contributing to the obstruction of the esophagus are divided by passage of a balloon dilation catheter (Figure 13.4d). A chest tube is placed and the portals are closed.

Aberrant Left or Right Subclavian Artery

An aberrant left or right subclavian artery compresses the esophagus but does not form a complete vascular ring. As a result, symptoms tend to be less severe. Aberrant subclavian arteries are approached with an ipsilateral intercostal thoracotomy. If artery is atrophied it can be ligated and transected. However if the

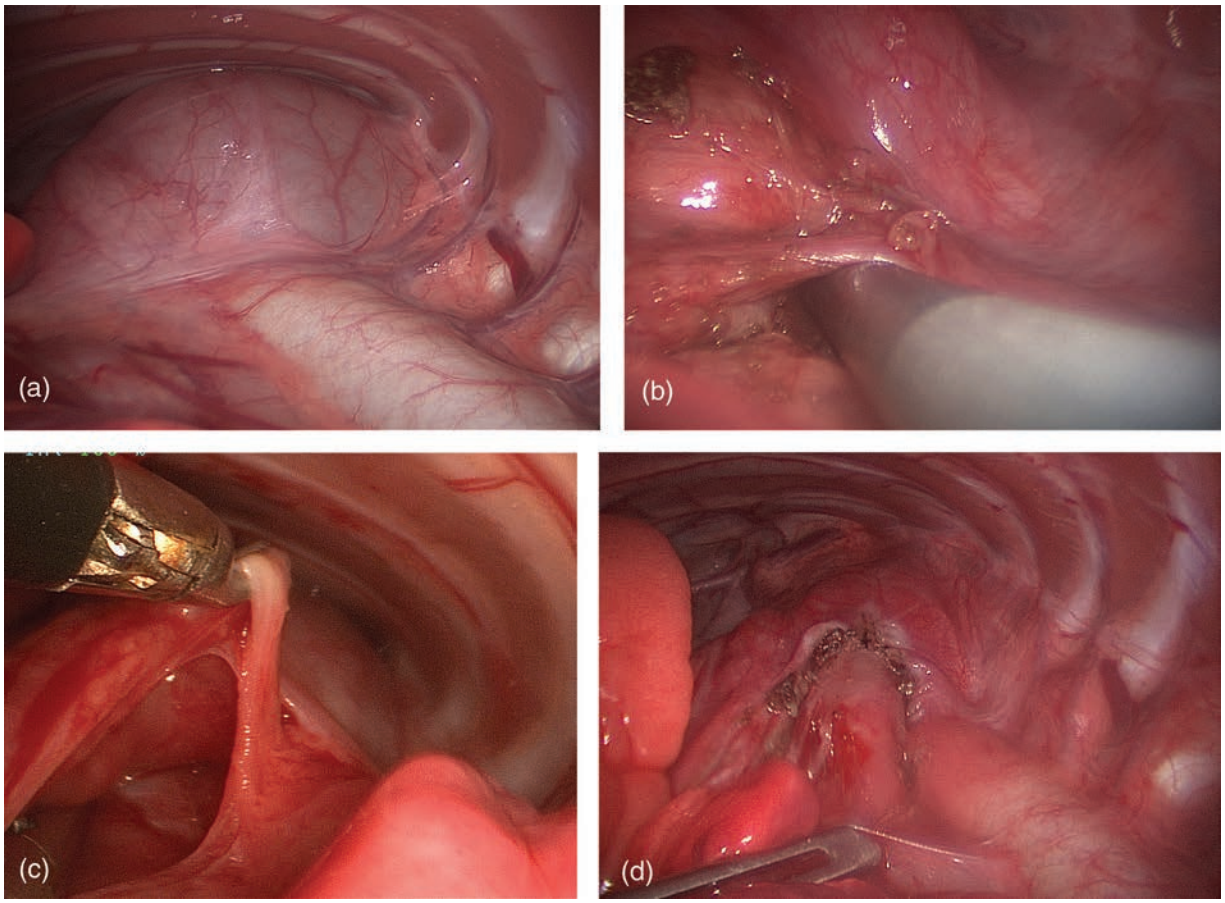

Figure 13.4 Thoracoscopic Surgery for Vascular Ring Anomaly

artery is clearly patent, transection can compromise blood supply to the respective forelimb. Measurement of arterial pressure in the appropriate leg by direct arterial catheterization or Doppler can help determine if the aberrant artery can be safely divided. If the artery is judged to significantly contribute to the blood supply to the forelimb, then the artery should not be ligated and transected. In this case, the artery should be lengthened by implantation of an ePTFE vascular graft.

Persistent Right Ligamentum Arteriosum

Surgical correction of a persistent right ligamentum with a normal left aortic arch is similar to persistent right aortic arch except that the right ligamentum is divided from a right thoracotomy. Holt et al. [5] described successful surgery for this defect from the left side but acknowledged that a right-sided approach is easier.

Double Aortic Arch

Surgical relief of a vascular ring caused by double aortic arch is accomplished by dividing one of the aortic arches. Typically, one of the arches is less dominant, and that arch should be preferentially divided. Preoperative angiography or CT angiography is helpful in identifying which arch should be divided. While it is possible for either arch to be divided from left intercostal thoracotomy, the surgery is more safely performed from a thoracotomy on the same side as the arch that will be divided. Prior to dividing one of the arches, it should be clamped for a period of time to assure that division will be tolerated. Surgery is accomplished by clamping with two vascular clamps and transecting the arch between the clamps similar to the technique described in Chapter 19 for dividing a patent ductus arteriosus. The divided ends are closed with pledget-reinforced mattress sutures of 5-0 polypropylene and oversewn with a simple continuous pattern.

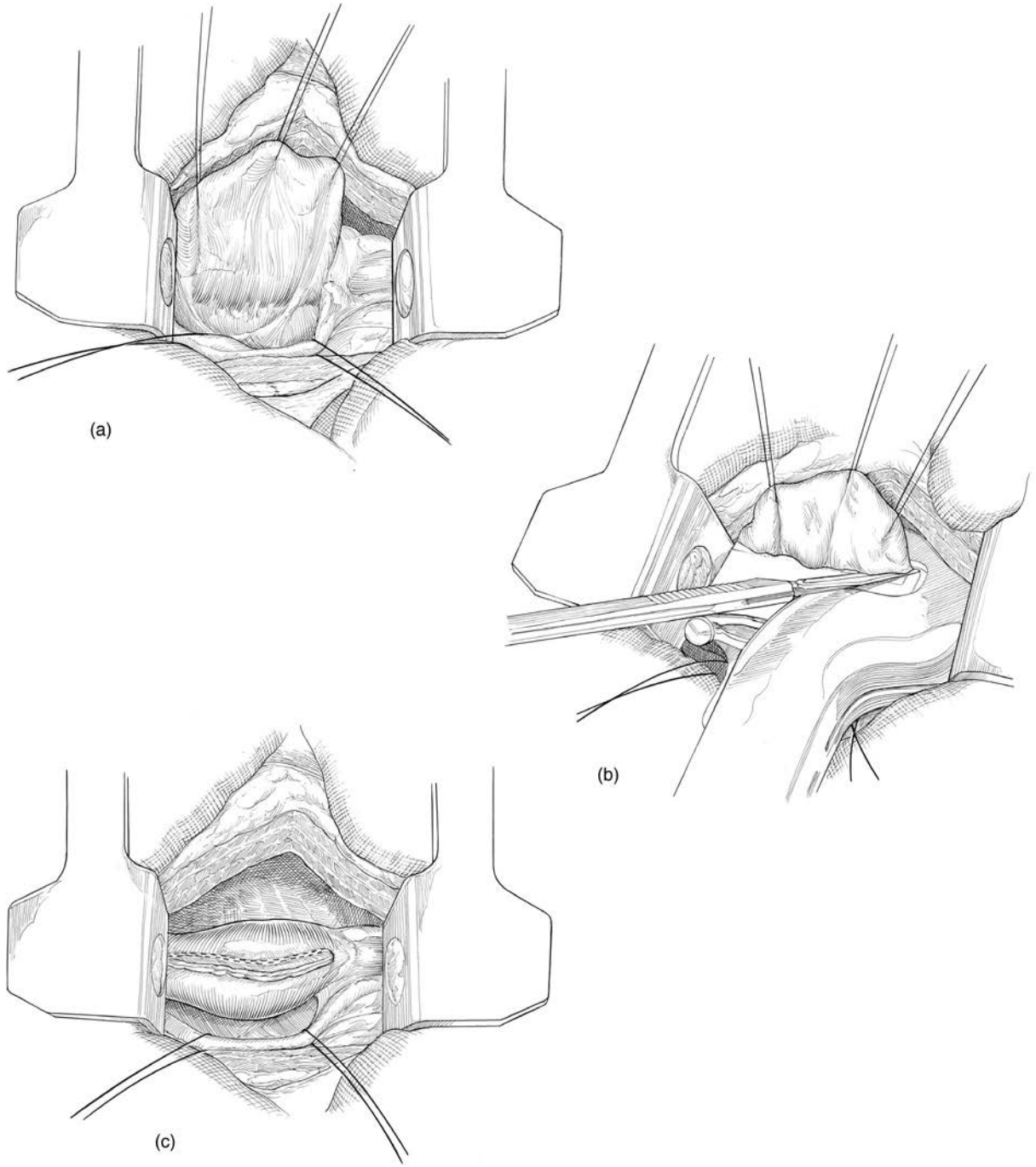

Figure 13.5 Esophageal Resection for Vascular Ring Anomaly

Surgery for Esophageal Dilation

It is generally accepted that peristaltic function will not be fully or even partially restored to the dilated portion of esophagus after surgery. Nevertheless, palliative efforts to decrease the degree of dilation to reduce passive food entrapment are sometimes undertaken. Palliative surgeries that have been performed include longitudinal plication or resection. It is unknown whether either of these techniques changes the prognosis. The author has performed resections of redundant esophagus with success in a limited number of dogs. This is accomplished by placing stay sutures to retract the lateral wall of the

esophagus (Figure 13.5a). A TA stapling device is applied to the long axis of the esophagus to decrease its diameter and the redundant esophagus is removed by incising along the stabling device (Figure 13.5b). The stabling device is released and the esophageal incision is inspected for leakage (Figure 13.5c).

References

1 Ellison GW. 1980. Vascular ring anomalies in the dog. *Compendium of Continuing Education for Veterinarians*. 2:693–705.

2 Martin DG, Ferguson EW, Gunnels RD, Robinowitz M, and Toft JD, 2nd. 1983. Double aortic arch in a dog. *Journal of the American Veterinary Medical Association*. 183(6):697–9.

3 Muldoon MM, Birchard SJ, and Ellison GW. 1997. Long-term results of surgical correction of persistent right aortic arch in dogs: 25 cases (1980–1995). *Journal of the American Veterinary Medical Association*. 210(12):1761–3.

4 Findji L, and Degueurce C. 1999 Symmetrical double aortic arch in a dog. *Veterinary Record*. 145(16):465–6.

5 Holt D, Heldmann E, Michel K, and Buchanan JW. 2000. Esophageal obstruction caused by a left aortic arch and an anomalous right patent ductus arteriosus in two German Shepherd littermates. *Veterinary Surgery*. 29(3):264–70.

6 Ferrigno CR, Ribeiro AA, Rahal SC, Orsi AM, Fioreto ET, Castro MF, et al. 2001. Double aortic arch in a dog (Canis familiaris): a case report. *Anat Histol Embryol*. 30(6):379–81.

7 Buchanan JW. 2004. Tracheal signs and associated vascular anomalies in dogs with persistent right aortic arch. *Journal of Veterinary Internal Medicine*. 18(4):510–4.

8 Du Plessis CJ, Keller N, and Joubert KE. 2006. Symmetrical double aortic arch in a beagle puppy. *Journal of Small Animal Practice*. 47(1): 31–4.

9 Moonan N, Mootoo NF, and Mahler SP. 2007. Double aortic arch with a hypoplastic left arch and patent ductus arteriosus in a dog. *Journal of Veterinary Cardiology*. 9(1):59–61.

10 Shojaei B, Akhtardanesh B, Kheirandish R, and Vosough D. 2001. Megaesophagus caused by an aberrant right subclavian artery and the brachiocephalic trunk in a cat. *Online Journal of Veterinary Research*. 15(1):46–52.

11 Bottorff B, and Sisson DD. 2012. Hypoplastic aberrant left subclavian artery in a dog with a persistent right aortic arch. *Journal of Veterinary Cardiology*. 14(2):381–5.

12 Saunders AB, Winter RL, Griffin JF, Thieman Mankin KM, and Miller MW. 2013. Surgical management of an aberrant left subclavian artery originating from a left patent ductus arteriosus in a dog with a right aortic arch and abnormal branching. *Journal of Veterinary Cardiology*. 15(2):153–9.

13 Krebs IA, Lindsley S, Shaver S, and Macphail C. 2014. Short- and long-term outcome of dogs following surgical correction of a persistent right aortic arch. *Journal of American Animal Hospital Association*. 50(3):181–6.

14 Menzel J, and Distl O. 2011. Unusual vascular ring anomaly associated with a persistent right aortic arch and an aberrant left subclavian artery in German pinschers. *Veterinary Journal*. 187(3): 352–5.

15 Philipp U, Menzel J, and Distl O. 2011. A rare form of persistent right aorta arch in linkage disequilibrium with the DiGeorge critical region on CFA26 in German Pinschers. *The Journal of Heredity*. 102 Suppl 1:S68–73.

16 Yarim M, Gultiken ME, Ozturk S, Sahal M, and Bumin A. 1999 Double aortic arch in a Siamese cat. *Veterinary Pathology*. 36(4):340–1.

17 Vianna ML, and Krahwinkel DJ, Jr. 2004. Double aortic arch in a dog. *Journal of the American Veterinary Medical Association*. 225(8):1222–4.

18 Hurley K, Miller MW, Willard MD, and Boothe HW. 1993. Left aortic arch and right ligamentum arteriosum causing esophageal obstruction in a dog. *Journal of the American Veterinary Medical Association*. 203(3):410–2.

19 Henjes CR, Nolte I, and Wefstaedt P. 2011. Multidetector-row computed tomography of thoracic aortic anomalies in dogs and cats: Patent ductus arteriosus and vascular rings. *BMC Veterinary Research*. 7:57.

14

Trachea

Eric Monnet

Structure and Function

The trachea conducts air from the larynx to the bronchial hilus, traversing the neck, thoracic inlet, and cranial thoracic mediastinum. The tracheal wall is composed of C-shaped cartilage rings with fibroelastic annular ligaments between. A dorsal membrane, composed of smooth muscle and connective tissue, completes the circumferential wall. The epithelium of the trachea is ciliated columnar epithelium with many mucous producing goblet cells. Blood supply to the trachea is segmentally carried by the cranial and caudal thyroid and the bronchoesophageal arteries. The laryngeal recurrent nerves course on each side of the trachea.

The structure of the trachea provides rigidity that resists forces generated by breathing and lateral flexibility to support movement of the neck. Forces associated with breathing include negative intraluminal pressures during inspiration and positive extraluminal pressures on the thoracic trachea during forced expiration and coughing. Both exert a collapsing force on the trachea. Increased upper airway resistance exacerbates negative intraluminal pressures during inspiration, whereas increased lower airway resistance exacerbates positive extraluminal pressure on the thoracic trachea. Because the entire minute volume flows through the relatively small crossectional area of the trachea, it is a point of high airway resistance. High flow velocities in the trachea can contribute to negative intraluminal pressures due to the Bernoulli effect. The mucociliary apparatus of the trachea clears inhaled particles and microbes away from the lower airways and thus represents an important protective mechanism against respiratory infections.

Temporary Tracheostomy

Temporary tracheostomy is indicated to temporarily bypass the upper airway in cases of acute upper airway obstruction. Obstructions could be caused by acute exacerbation of laryngeal paralysis, brachycephalic airway syndrome, neoplasia, and trauma. Temporary tracheostomy is also used to maintain anesthesia for some maxillofacial surgeries and for longer-term ventilatory therapy.

Temporary tracheostomy employs a tube to maintain patency and allow application of positive pressure ventilation when needed. Tracheostomy tubes come with an outer cannula with or without a cuff, an inner cannula, and an obturator (Figure 14.1). A cuff is required for positive pressure ventilation. If positive pressure ventilation is not needed, a cuffless tube is used or the cuff is not inflated to facilitate mucociliary clearance. The obturator facilitates placement of the outer cannula. The obturator is replaced by the inner cannula immediately after tube insertion. The inner cannula allows for regular cleaning of the tube and airway toiletry.

A temporary tracheostomy is typically performed under general anesthesia, but in an emergent situation is performed under sedation and local anesthesia. The patient is placed on dorsal recumbency and a midline cervical incision is made caudal to the larynx. During the dissection, it is helpful for the surgeon to hold the trachea between two fingers to stabilize and push it toward the incision. The sternohyoideus muscles are separated to expose the trachea (Figure 14.2a). A Gelpy retractor can be placed to retract the skin and the sternohyoideus muscles. A transverse incision is made in an annular ligament between tracheal

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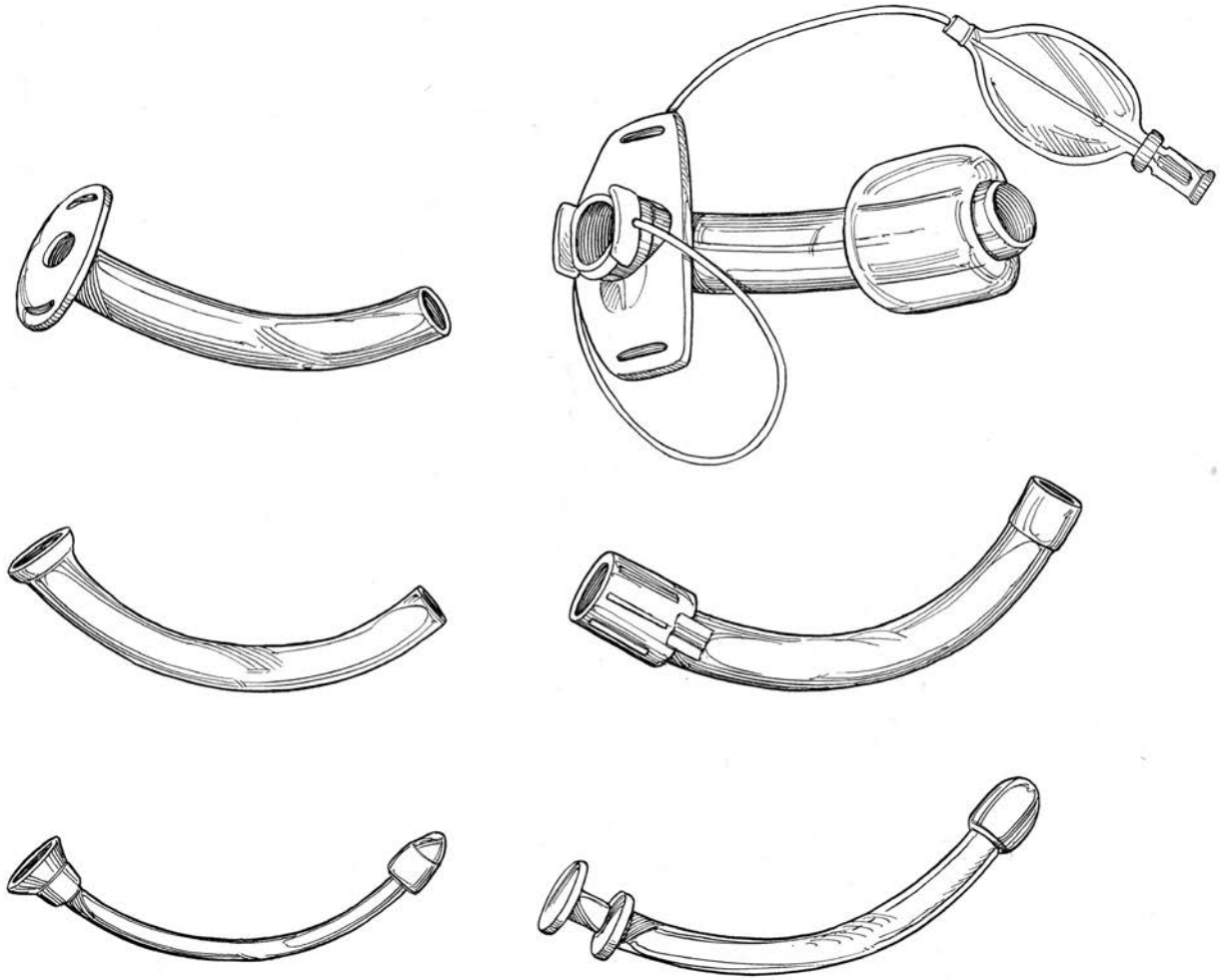

Figure 14.1 Tracheostomy Tubes

rings, avoiding injury to the recurrent laryngeal nerves coursing laterally on the trachea. A heavy monofilament stay suture is placed around the tracheal ring just caudal to the incision. This stay suture is used to open the incision and assist with tube placement (Figure 14.2b). The stay suture is left in place to assist with replacement of the tracheostomy tube should it need changing or become dislodged.

The flanges of the tracheostomy tube are secured with umbilical tape around the neck of the patient. The tube is cleaned every 4 to 6 hours to prevent obstruction with mucous and reduce nosocomial infection. The inner cannula is removed and cleaned in hydrogen peroxide.

After the tracheostomy is no longer needed, the tube is removed. The stay suture is left in place for another 24 hours in case the tracheostomy tube must be reintroduced. The trachea and the soft tissues are left open to heal by second intention. A light bandage is applied over the site. If subcutaneous emphysema develops, the skin and subcutaneous are kept open until the trachea seals.

Permanent Tracheostomy

Permanent tracheostomy is indicated when permanent bypass of the upper airway is required. Brachycephalic syndrome with third-degree laryngeal collapse, laryngeal paralysis with a high risk for aspiration pneumonia, severe laryngeal trauma, and laryngeal neoplasia are the most common indications for a permanent tracheostomy in dogs and cats.

A midline cervical incision is made over the trachea. The sternohyoideus muscles are dissected and separated (Figure 14.3a). The two muscles can be sutured together dorsal to the trachea to elevate and secure the trachea (Figure 14.3b). Additional sutures are placed between the muscles and trachea, taking care to avoid injury to the recurrent laryngeal nerves.

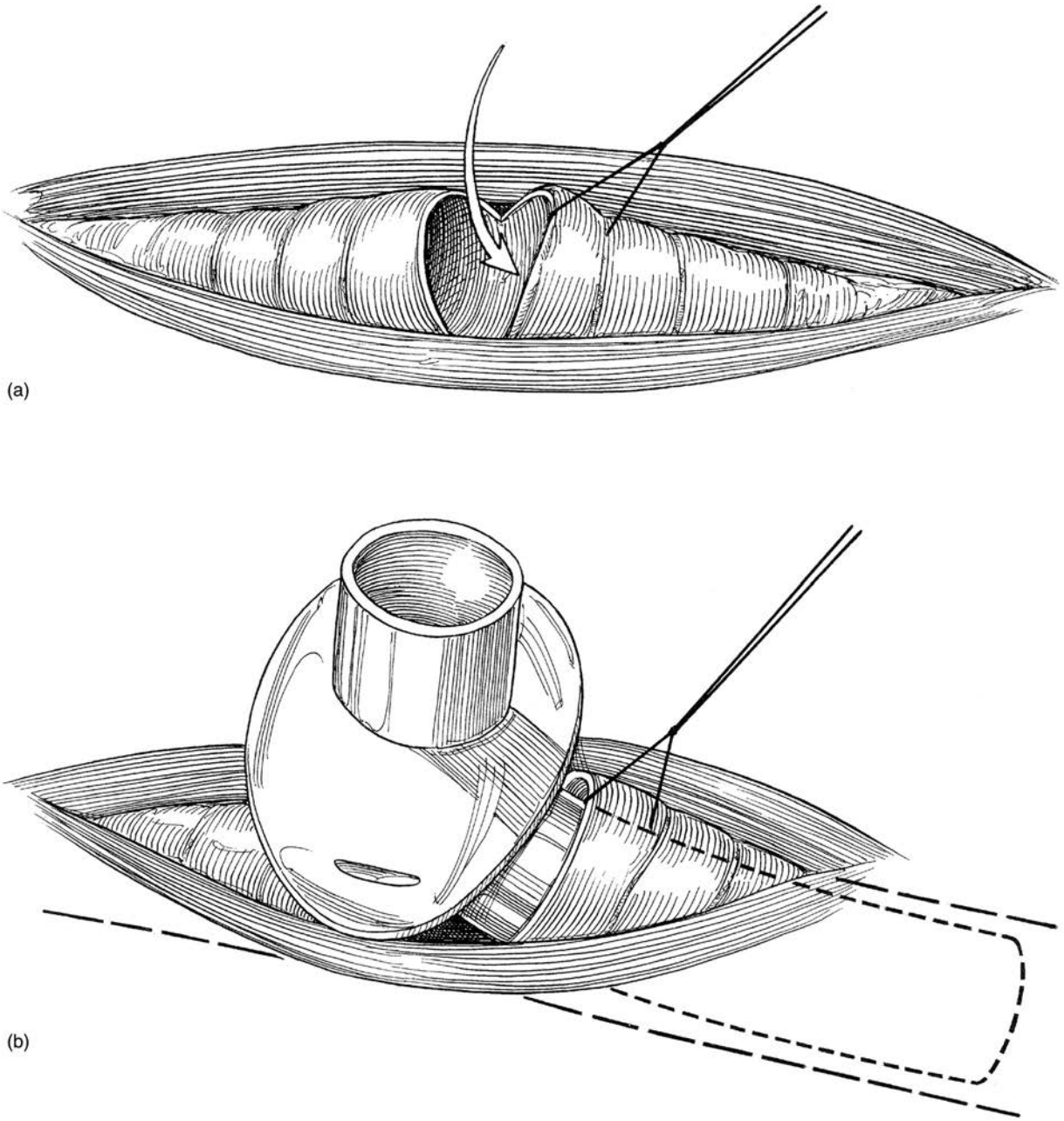

Figure 14.2 Tracheostoomy

The tracheostomy is performed over three tracheal rings, starting at the third or fourth tracheal ring from the larynx. The width of the tracheostomy should be approximately a third of the circumference of the trachea. The tracheal rings are incised with a #15 blade, avoiding penetration of the tracheal epithelium. Using more than one blade to maintain sharpness facilitates the procedure. After the tracheal rings are excised, an H-shaped incision is made in the epithelium. The skin is then sutured directly to the epithelium with simple interrupted sutures of 4-0 monofilament non-absorbable suture (Figure 14.3c). Good apposition between the skin and tracheal epithelium is important to limit the risk of stricture. If redundant skin in the neck might lead to obstruction the tracheostomy site, then it is recommended that excessive skin be excised on each side of the tracheostomy.

Care after surgery is important to the success of permanent tracheostomy. Because air is not filtered or humidified by the upper airways, the tracheal

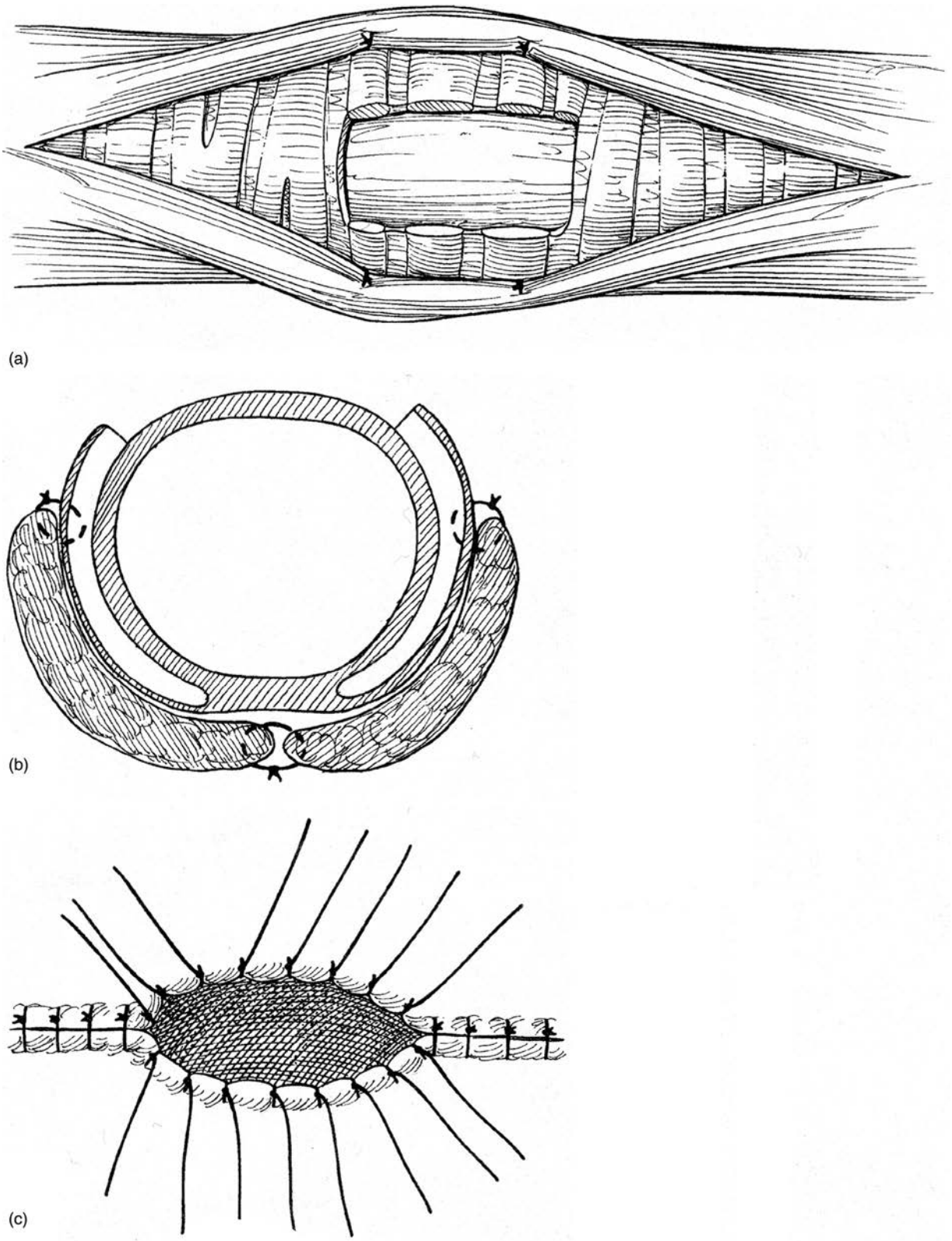

Figure 14.3 Permenent Tracheostomy

epithelium becomes inflamed and tends to increase mucus production. The tracheostomy should to be inspected and cleaned every 6 hours. Application of petroleum jelly on the surrounding skin decreases irritation and facilitates cleaning. Aspiration of the trachea may become necessary to remove mucus from the tracheal lumen. Nebulization with acetylcysteine can help with mucus clearance. Use of an air humidifier multiple times a day is helpful and can be continued at home for at least a month after surgery. After about one month, the epithelium in the trachea remodels and mucous production significantly decreases. Management of a permanent tracheostomy is more difficult in brachycephalic dogs because of a tendency for excessive mucus production and frequent obstruction of the airway. Permanent tracheotomy is possible in cats but has been associated with a high complication rate [1].

Traumatic Tracheal Injury

Bite wounds to the neck can cause severe tracheal trauma resulting in subcutaneous emphysema, pneumomediastinum, and pneumothorax. Conservative management with standard wound care over three days is recommended because small tracheal tears and punctures will often seal without surgery. A thoracostomy tube may be required to manage pneumothorax.

If subcutaneous emphysema or pneumothorax persist, surgical exploration is recommended. Tracheal lacerations are debrided and closed with interrupted sutures of monofilament absorbable suture. Braided sutures should not be used in the trachea because they can cause granuloma formation. Sutures are placed around the rings and not through the rings. If the laceration is wide and affecting more than two tracheal rings, tracheal resection and anastomosis may become necessary. Culture of the surgical site is taken at the time of surgery because of the severe contamination associated with bite wounds. A closed suction drain is placed before closure of the soft tissues. The patient is kept on antibiotic therapy for 10 to 14 days after surgery.

Tracheal Resection and Anastomosis

Tracheal resection and anastomosis is indicated for severe trauma to the trachea, tracheal stenosis, or resection of tracheal tumors. The patient is intubated to the level of the planned resection (Figure 14.4a). The trachea is exposed via a midline ventral cervical incision, cranial median sternotomy, or a right intercostal thoracotomy as needed. Ventral cervical and sternotomy approaches are combined if necessary. The recurrent laryngeal nerves are identified and preserved by dissection away from the lateral trachea. If a right intercostal thoracotomy is used, visualization of the left laryngeal recurrent nerve requires rotation of the trachea. Bilateral iatrogenic trauma to the laryngeal recurrent nerves must be avoided to prevent laryngeal paralysis. Dissection of the trachea is kept to the minimum necessary to minimize disruption to the segmental blood supply and avoid necrosis at the anastomotic site. The amount of trachea that can be safely resected is not clearly defined. It likely varies with the age, with younger animals able to tolerate more resection. Cats tolerate more resection than dogs. Resection of five tracheal rings is generally tolerated. More extensive resections increase the likelihood of anastomotic failure and stenosis. Stay sutures are placed in the distal segment of the trachea (Figure 14.4b). The trachea is transected distal to the segment being resected. Incision in the fibroelastic annular membrane is preferred over the cartilaginous rings. A sterile endotracheal tube at the table is passed into the distal tracheal segment and connected to the anesthesia circuit. The trachea is transected cranially and the segment to be excised is removed (Figure 14.4c). The distal endotracheal tube is removed and the original endotracheal tube is advanced back across the defect and the cuff is inflated. The anastomosis is completed with a simple interrupted suture pattern (Figure 14.4d). If tension is high, a simple continuous suture pattern may be superior to a simple interrupted suture [2]. Monofilament nonabsorbable suture are used to avoid granuloma formation. The sutures are first placed in the dorsal membrane with the knots outside of the lumen of the trachea. Remaining sutures can be placed around or through the rings. Tension-relieving sutures are placed one or two rings away from the anastomosis. The tension-relieving sutures are not exposed to the lumen of the trachea. They are placed around or through the tracheal rings with or without pledgets. If the surgery was performed on the thoracic trachea, a thoracostomy tube should be maintained for 24 hours after surgery to monitor for anastomotic leakage of air.

Tracheal Rupture

Rupture of the trachea can occur between the thoracic inlet and the carina, and usually results from stretching of the trachea with hyperextension of the neck or overinflation of an endotracheal tube cuff. The soft tissues around the trachea typically remain intact creating a pseudo-airway. Thoracic radiographs show

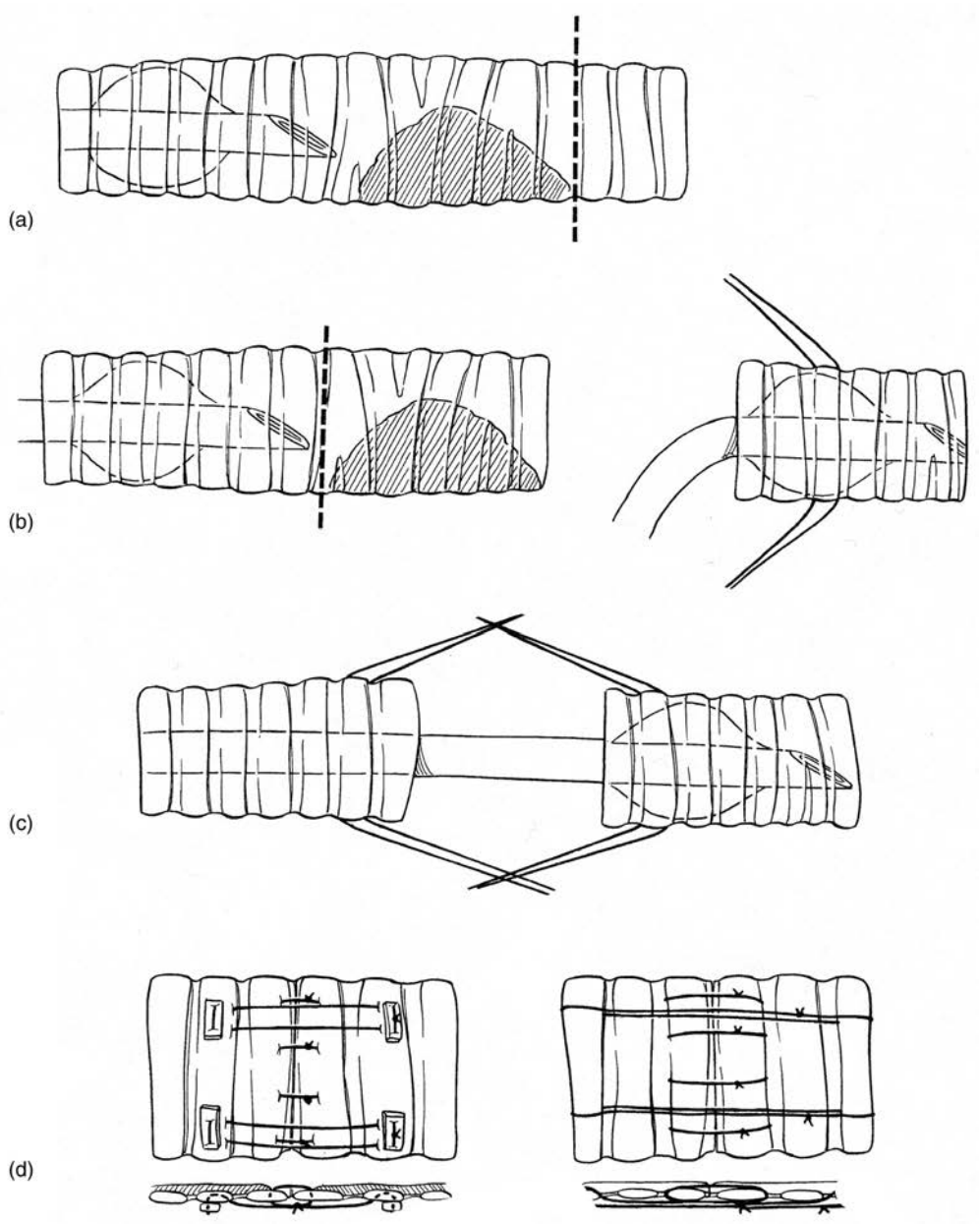

Figure 14.4 Tracheal Resection and Anastamosis

the lack of continuity of the trachea in the cranial mediastinum.

After induction of anesthesia, the patient is intubated with an endotracheal tube that stays cranial to the rupture. A right lateral thoracotomy is used to expose the trachea in the cranial mediastinum. The vagus and phrenic nerves are identified and preserved. The ends of the ruptured trachea are localized by palpation. Stay sutures are placed in the proximal and distal ends. Once the pseudo-airway is opened, an endotracheal tube is advanced in the distal segment to maintain ventilation of the patient. The ruptured ends of the trachea are debrided and sutured with 3-0 monofilament nonabsorbable sutures in a simple interrupted pattern, as described for tracheal anastomosis.

Tracheal Collapse

Dynamic tracheal collapse is a common condition in small dogs. Opinions vary as to whether tracheal

collapse is a primary disorder of the trachea or secondary to other conditions such as upper airway obstruction, chronic respiratory disease, or cardiac disease. Clinical observation and current evidence suggest that perhaps both are correct. The fact that that the condition is typically acquired later in life and almost always occurs with other chronic respiratory and/or cardiac conditions suggests that it is a secondary disease. The predilection in certain breeds of dog supports a hereditary cause, or at least a hereditary predisposition. Changes in the tracheal cartilage such as hypocellularity, low proteoglycan and low glycosaminoglycan content have been documented, but it is unclear whether these are primary or secondary changes [3–5].

Whether or not dynamic collapse of the trachea is primary or secondary, it is clearly exacerbated by several conditions that exert dynamic collapsing forces on the trachea. These include upper airway obstruction, which increases negative transmural pressures within the cervical trachea during inspiration and any respiratory disease associated with chronic coughing. Cough exerts a severe collapsing force on the thoracic trachea, both from very high intrathoracic pressures generated during forced expiration and the Bernoulli effect generated by high-velocity flow within the trachea. Left atrial enlargement associated with chronic cardiac conditions such as degenerative mitral valve disease clearly worsens dynamic tracheal collapse. Rarely static (adynamic) collapse or malformation of the trachea occurs as a hereditary defect in dogs and cats. Diagnosis of tracheal collapse is established based on clinical signs, radiographs, fluoroscopy, and bronchoscopy [6].

Medical treatment of collapsing trachea is directed at the underlying conditions that cause or exacerbate collapsing trachea. Chief among these is chronic coughing associated with chronic respiratory diseases such as chronic bronchitis. Medical therapies include antibiotics, anti-inflammatory drugs, bronchodilators, antitussives drugs, and environmental therapies. When cardiac disease is present, medical therapies to reduce left atrial size are also beneficial [7].

Surgery or interventional therapies for collapsing trachea are indicated when airway obstruction has progressed to the point of interfering with normal breathing. Typically, this is necessary when tracheal collapse has progressed to the point of complete flattening even when severe dynamic forces are not applied (Figure 14.5a). Cough, in and of itself, should not be considered an indication for surgery or intervention. Chronic coughing should be treated medically and typically will not be improved by surgical intervention. Interventions for collapsing trachea include surgical implantation of external prosthetic rings or intraluminal tracheal stenting. Each of these approaches has advantages and disadvantages.

Ring Tracheoplasty

Surgical implantation of external prosthetic rings has the principle advantage of not interfering with mucociliary clearance mechanisms within the tracheal lumen [8–10]. C-shaped polypropylene prosthetic rings can be fashioned from syringe casings (Figure 14.5b). Holes are added to the rings to facilitate suturing. The trachea is approached by ventral midline cervical incision and/or sternotomy. The recurrent laryngeal nerves on the lateral aspects of the trachea are identified and preserved. Dissection of the tracheal is kept to the minimum necessary for ring placement to maintain segmental blood supply. Rings are placed around trachea, excluding the recurrent laryngeal nerves. The open portion of the ring faces ventrally. The trachea is sutured to the ring with 4-0 monofilament nonabsorbable sutures. Sutures are placed in the dorsal membrane by rotation of the trachea. Additional rings are placed 7 to 10 mm apart as needed.

Tracheal Stenting

Tracheal stenting is accomplished with self-expanding intraluminal stents placed under fluoroscopic and/or bronchoscopic guidance [11–15]. The principal advantage of tracheal stenting is its minimal invasiveness compared to surgery. Problems with stent fracture and migration have decreased with experience and improved stents. Determining appropriate stent diameter and length prior to implantation is critical to success. Thoracic radiographs of the entire trachea from the larynx to carina are used to determine stent diameter and length.

Ideally, radiographs are taken with the patient under anesthesia with positive-pressure ventilation to dilate the trachea. The neck is placed in a normal position to prevent extension of the trachea. The diameter of the trachea is measured in the cervical area and the thoracic area. A stent diameter 10% larger than the measured tracheal diameter under positive pressure is chosen. Stent length is measured from 1 cm distal to the larynx to 1 cm proximal to the carina. Because of limitations of stent inventory at most hospitals, measurement of the trachea under anesthesia often requires recovery from anesthesia without intervention. This can be a challenge for some dogs with severe tracheal collapse. An alternate approach used by the author is to take radiographs with the animal

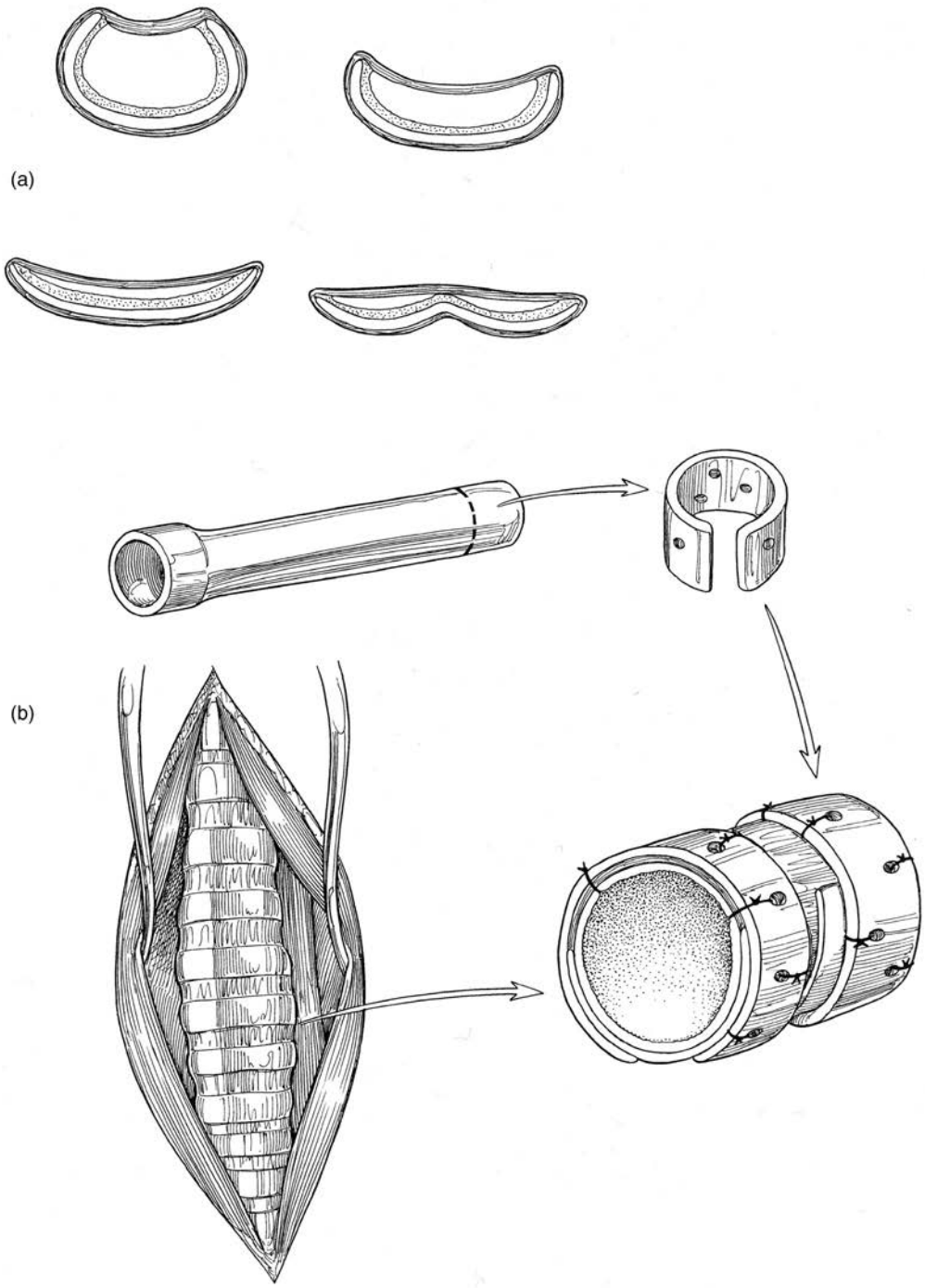

Figure 14.5 Ring Tracheoplasty

awake. Measurements are taken at the level of the larynx and the carina and averaged. This diameter is doubled to obtain the diameter of the stent. The length of the trachea from larynx to carina is measured on the radiographs and 2 cm is subtracted as above.

Tracheal stents are made of self-expanding nickel-titanium metal. They come in two basic types: braided and laser-cut. Braided stents shorten, depending on the degree of expansion. This phenomenon, known as foreshortening, must be taken into consideration

in choosing an appropriate stent length. Manufacturers provide charts of stent length, depending on the final diameter of the stent after deployment. Laser-cut stents have the advantage of exhibiting minimal foreshortening. Stents are supplied in a variety of diameters and lengths. Tapering stents are also now available. Stents are constrained in a delivery catheter for deployment. The deployment of the stent is performed under fluoroscopic guidance. The patient is placed under general anesthesia and intubated. An adapter with a side entry port is placed on the endotracheal tube to allow access to the tracheal lumen. An appropriate-sized guide wire is advanced through the endotracheal tube into the bronchi of a caudal lung lobe (Figure 14.6a). The delivery catheter is advanced over the guidewire into the trachea to the level of the carina. The distal marker of the stent is maintained 1 cm proximal to the carina as the stent is slowly deployed (Figure 14.6b). Stents can be reconstrained if necessary up to a certain point. The manufacturer provides information about the percentage of deployment that cannot be exceeded for reconstraining the stent. Once that point is exceeded, the stent cannot be reconstrained or moved. Deployment of the stent proximately requires that the patient be briefly extubated (Figure 14.6c).

Medical therapy for underlying chronic respiratory disease must be continued after tracheal stenting. If the stent maintains good contact against the wall of the trachea, it will be incorporated into the wall of the trachea over time. Gaps between the wall of the trachea and the stent allow accumulation of mucus, and this will be a source of chronic inflammation and infection. Motion of the stent can cause granuloma formation at the ends of the stent and increases the risk of stent fracture [11,13]. Placement of larger diameter stents that apply significant radial force against the wall of the trachea is the best way to prevent stent movement. Stenting the entire length of the trachea instead of only the collapsed section helps prevent migration. The thoracic inlet is a zone of increased stress on the stent and is a likely site for fracture of the stent. Stent fracture is best treated by placing another stent within the broken stent.

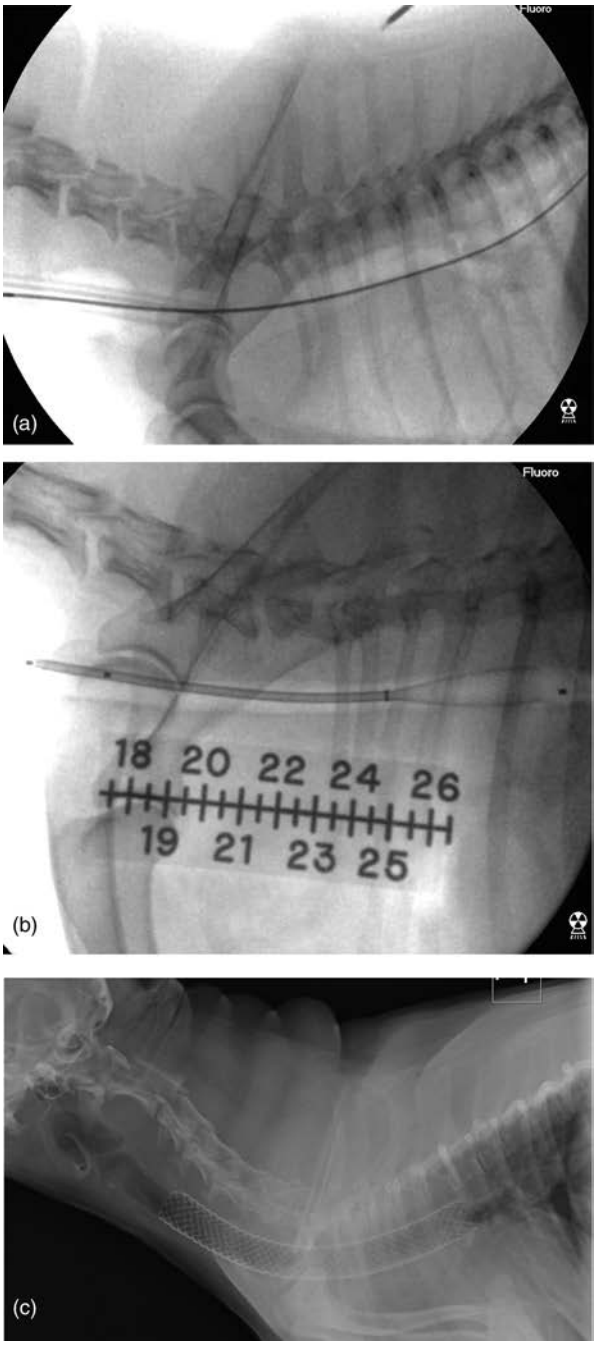

Figure 14.6 Tracheal Stenting for Collapsing Trachea

References

1 Guenther-Yenke CL, and Rozanski EA. 2007. Tracheostomy in cats: 23 cases (1998–2006). *Journal of Feline Medicine and Surgery*. 9(6): 451–7.

2 Demetriou JL, Hughes R, and Sissener TR. 2006. Pullout strength for three suture patterns used for canine tracheal anastomosis. *Veterinary Surgery*. 35(3):278–83.

3 Johnson LR, and Pollard RE. 2010. Tracheal collapse and bronchomalacia in dogs: 58 cases (7/2001-1/2008). *Journal of Veterinary Internal Medicine*. 24(2):298–305.

4 Maggiore AD. Tracheal and airway collapse in dogs. 2014. *Veterinary Clinics of North America - Small Animal Practice*. 44(1):117–27.

5 Tappin SW. Canine tracheal collapse. 2016. *Journal of Small Animal Practice*. 57(1):9–17.

6 Johnson L. 2000 Tracheal collapse. Diagnosis and medical and surgical treatment. *Veterinary Clinics North America—Small Animal Practice*. 30(6): 1253–66.

7 Johnson LR, and Fales WH. 2001. Clinical and microbiologic findings in dogs with bronchoscopically diagnosed tracheal collapse: 37 cases (1990–1995). *Journal American Veterinary Medical Association*. 219(9):1247–50.

8 Buback JL, Boothe HW, and Hobson HP. 1996. Surgical treatment of tracheal collapse in dogs: 90 cases (1983-1993). *Journal American Veterinary Medical Association*. 208(3):380–4.

9 Becker WM, Beal M, Stanley BJ, and Hauptman JG. 2012 Survival after surgery for tracheal collapse and the effect of intrathoracic collapse on survival. *Veterinary Surgery*. 41(4):501–6.

10 Chisnell HK, and Pardo AD. 2014. Long-term outcome, complications and disease progression in 23 dogs after placement of tracheal ring prostheses for treatment of extrathoracic tracheal collapse. *Veterinary Surgery*. 44(1):103–13.

11 Radlinsky MG, Fossum TW, Walker MA, Aufdemorte TB, and Thompson JA. 1997. Evaluation of the Palmaz stent in the trachea and mainstem bronchi of normal dogs. *Veterinary Surgery*. 26(2):99–107.

12 Moritz A, Schneider M, and Bauer N. 2004. Management of advanced tracheal collapse in dogs using intraluminal self-expanding biliary wallstents. *Journal of Veterinary Internal Medicine*. 18(1): 31–42.

13 Sura PA, and Krahwinkel DJ. 2008. Self-expanding nitinol stents for the treatment of tracheal collapse in dogs: 12 cases (2001-2004). *Journal American Veterinary Medical Association*. 232(2):228–36.

14 Durant AM, Sura P, Rohrbach B, and Bohling MW. 2012. Use of nitinol stents for end-stage tracheal collapse in dogs. *Veterinary Surgery*. 41(7): 807–17.

15 Tinga S, Thieman Mankin KM, Peycke LE, and Cohen ND. 2015. Comparison of outcome after use of extra-luminal rings and intra-luminal stents for treatment of tracheal collapse in dogs. *Veterinary Surgery*. 44(7):858–65.

15

Lung

Eric Monnet

Structure and Function

The right and left lung of dogs and cats are separated by a thin mediastinum. The left lung is divided into cranial and caudal lobes (Figure 15.1). The left cranial lung lobe divides incompletely into a cranial and caudal portion that share a lobar bronchus. The right lung is divided into cranial, middle, caudal, and accessory lung lobes. The accessory lung lobe passes dorsal and medial to the caudal vena cava. The ventral portion of the accessory lobe is separated from the rest of the right lung by the plica vena cava, a reflection of pleura containing the caudal vena cava and right phrenic nerve. The trachea divides into two principal bronchi, which, in turn, divide into lobar bronchi supplying each lung lobe. Dichotomous branching continues into segmental bronchi, subsegmental bronchi, terminal bronchioles, respiratory bronchioles, alveolar ducts, alveolar sacs, and pulmonary alveoli.

Pulmonary arteries follow a lobar distribution in close proximity to cranial aspect of the airways. Bronchial artery branches of the bronchoesophageal arteries carry oxygenated blood to the airways down to the level of the respiratory bronchioles where they terminate in the pulmonary capillary bed. Pulmonary veins follow the caudal aspect of the airways and carry blood from the pulmonary capillaries to the left atrium. The lungs receive afferent and efferent innervation from the vagus nerves and sympathetic trunk.

Respiration is accomplished by close coupling between the lung and thoracic wall and diaphragm. Coupling is accomplished by negative pressure within the pleural space. The ventilation and gas exchange functions of the lungs are reviewed in Chapter 1. The lungs receive the entire cardiac output of the right heart. As such, conditions affecting the pulmonary vascular bed can have profound effects on cardiac function overall. The pulmonary vascular bed has built in redundancy that allows recruitment of vessels to accommodate exercise and protect against losses to the pulmonary vascular bed, including lung lobectomy. Pulmonary arteries, unlike systemic arteries, exhibit strong vasoconstriction in response to alveolar hypoxia. This response, known as *hypoxic pulmonary vasoconstriction*, mediates development of hypoxic pulmonary hypertension. Other than this difference, pulmonary arteries respond to the same vasoconstrictive and vasodilatory mechanisms active in systemic arteries. Pulmonary airways contain smooth muscle that mediates bronchoconstriction and bronchodilation in response to parasympathetic and beta-sympathetic stimulation, respectively.

Surgical Conditions of the Lung

Neoplasia is the most common indication for lung lobectomy in dogs and cats. Bronchoalveolar carcinoma represents 85% of the lung tumors in dogs, while adenocarcinoma represents 70% of lung tumors in cats. Other pulmonary tumors are adenocarcinoma and squamous cell carcinoma. Histiocytic sarcoma has an apparent breed predilection for Bernese mountain dogs, although Rottweiler, Golden retriever, and Flat-Coated retriever may be at increased risk also [1]. Thirty-percent of primary lung tumors in dogs are asymptomatic. Coughing is noted in 52% to 93% of canine cases. Dyspnea, lethargy, anorexia, weight loss, hemoptysis, and lameness are the most commonly observed clinical signs in dogs with lung tumors [1–4]. Gastrointestinal signs such as vomiting and diarrhea are mostly observed in cats. Pleural effusion also occurs in cats with lung tumors. When pleural effusion is present cytology of the fluid is helpful in determining the tumor type in cats [5]. Consolidation of lung lobes associated with chronic pneumonia or lung abscesses with or without a migrating foreign body are indications for lung lobectomy. These conditions can be associated with concurrent pyothorax. Finally, spontaneous pneumothorax secondary to pulmonary

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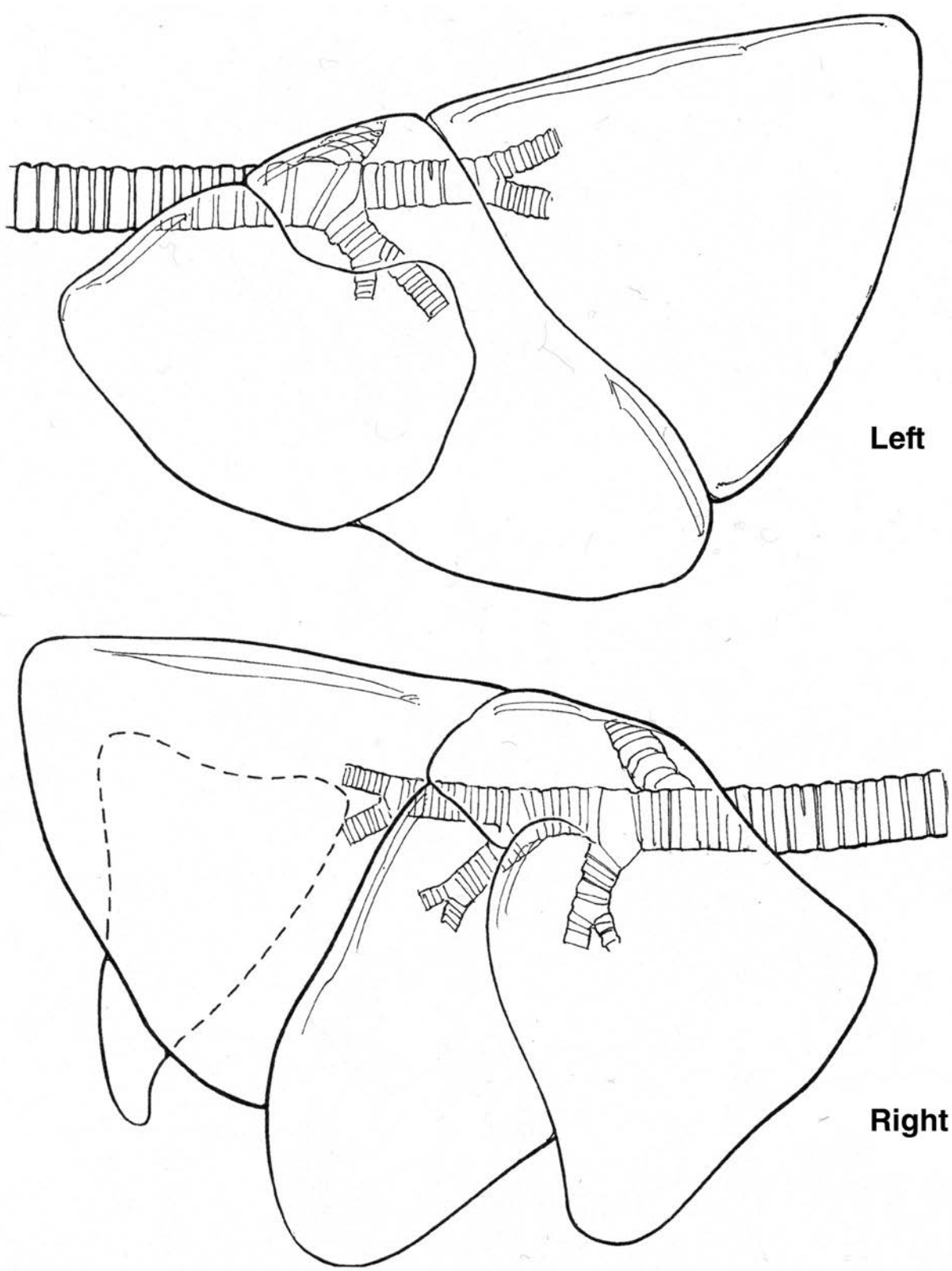


Figure 15.1 Canine Lung Lobes

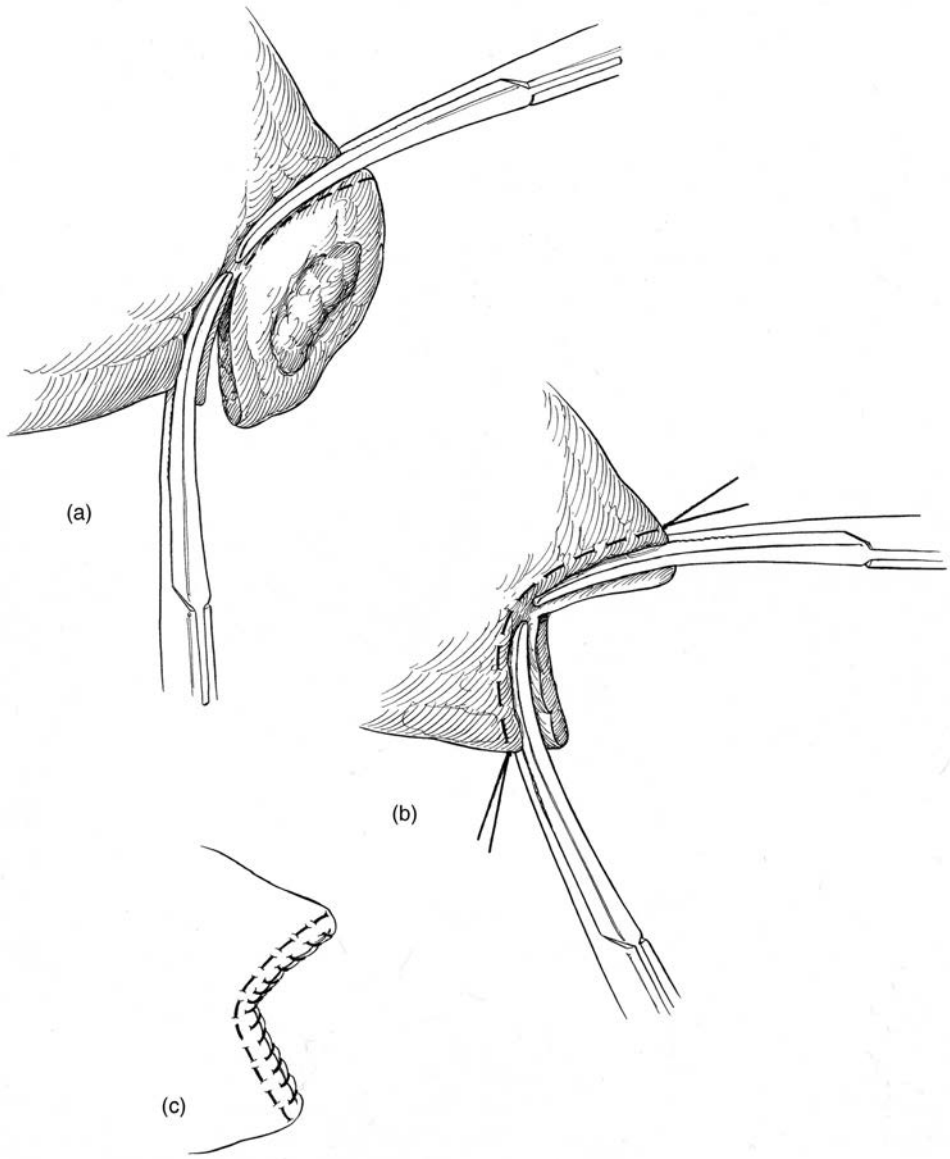


Figure 15.2 Partial Lung Resection

bleb and emphysematous bullae represent indications for partial lung resection or pulmonary lobectomy [6–8].

Diagnosis

Thoracic radiography is the most important diagnostic modality for identifying surgical conditions of the lung. In a canine case series, 83% of the pulmonary tumors were diagnosed on thoracic radiographs [4]; 54% of the cases had a single mass. Thoracic ultrasound is useful for guiding fine-needle aspiration of pulmonary tumors before surgery. CT scanning is gaining importance in the diagnosis of pulmonary conditions, especially for evaluation of lung metastasis. CT scan also provides information about tracheobronchial lymph node enlargement that cannot be appreciated on thoracic radiography. CT guided core biopsy of lung mass can be performed with an accuracy of 92% and a sensitivity of 80% for pulmonary neoplasia [9]. However, the complication rates associated with image-guided biopsy including pneumothorax and pulmonary hemorrhage can be as high as 43% [9].

Primary spontaneous pneumothorax has been shown to have a better long-term outcome if a surgical exploration is performed [6–8]. Radiographs and CT

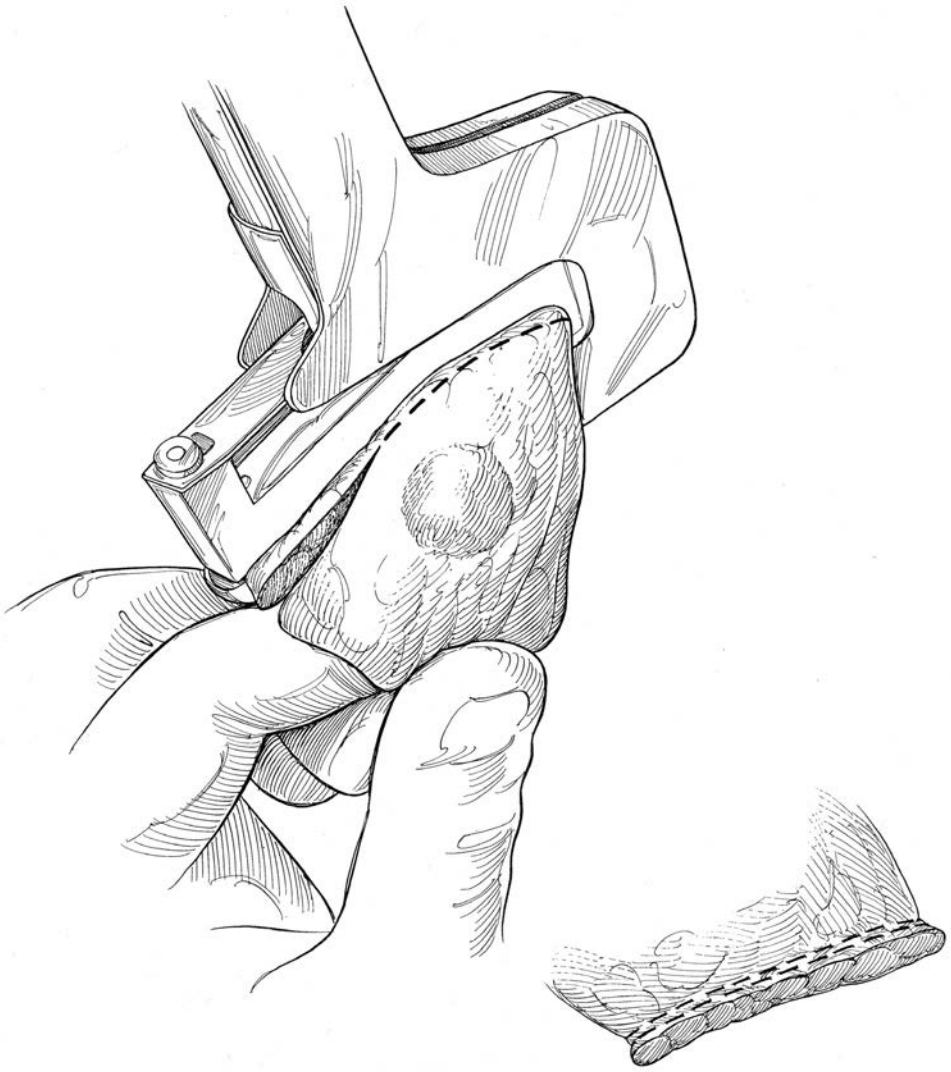


Figure 15.3 Partial Lung Resection with Staples

scans are not sensitive for the diagnosis of bullae and blebs [10, 11]. Cats have an apparent better long-term outcome with conservative management of spontaneous pneumothorax [8].

Lobar pneumonia with lung lobe consolidation is an indication for lung lobectomy after appropriate medical treatment has failed. Bronchoscopy with bronchoalveolar lavage provides specimens for bacterial culture and sensitivity, enabling identification of the infectious agents and guiding appropriate antibiotic therapy before surgery. Antibiotic therapy should be continued for at least three weeks without obvious clinical or radiographic improvement before surgery is considered.

Lung trauma, lung lobe torsion, and bronchoesophageal fistula represent additional indications for lung lobectomy in dogs and cats. Lung lobe torsion most often affects the left cranial and right middle lung lobes [12–17]. Large-breed dogs are more commonly affected; however, pugs have been reported to be at risk for lung lobe torsion [12, 13, 16]. Risk factors for lung lobe torsion include presence or history of pleural effusion, pneumothorax, previous thoracic surgery, trauma, or diaphragmatic hernia.

Surgery

Partial and complete lung lobectomy can be performed via intercostal thoracotomy, median sternotomy, or thoracoscopy. The choice of the approach is based on surgeon experience, the size and location

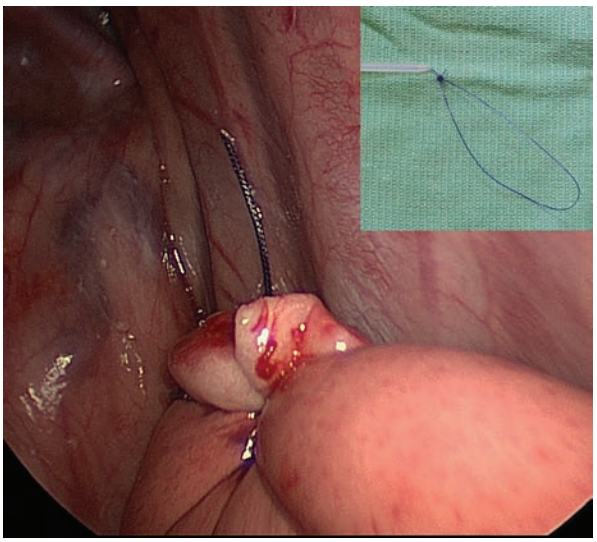


Figure 15.4 Thoracoscopic Lung Biopsy—Loop Technique

of the mass, and the degree of need for complete exploration of the thoracic cavity and its organs. All lung lobes can be resected via video-assisted thoracoscopy. However, only smaller masses well away from the pulmonary hilus can be safely resected by this approach. If a mass is large, then an enlargement of intercostal portal site will be necessary to remove the tumor which defeats the primary purpose of doing a minimally invasive thoracoscopic procedure.

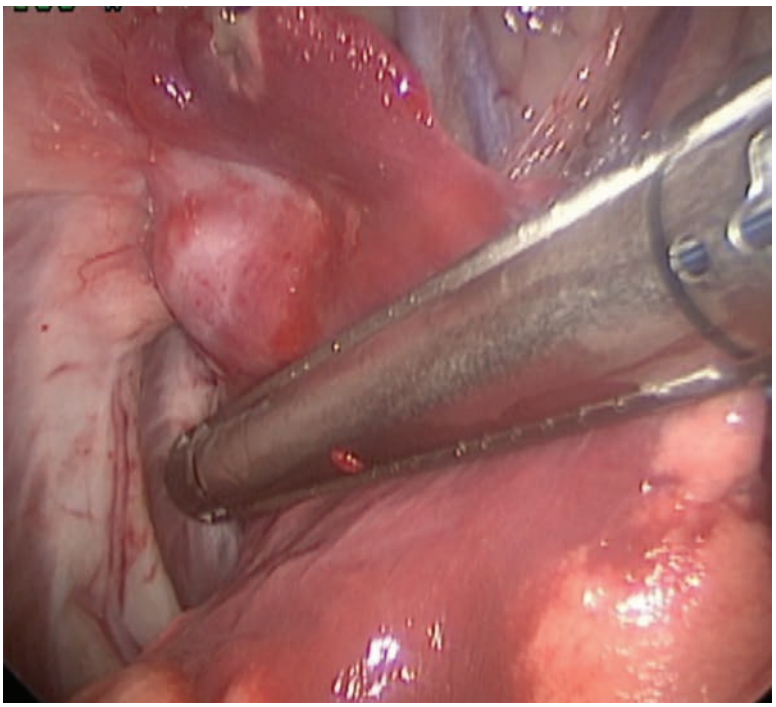
Partial Lung Resection

Partial lung resection is indicated for smaller lesions primarily at the periphery of the lung and for the collection of biopsies. The procedure can be performed either with sutures or stapling devices.

Open partial lung resection with sutures is accomplished with the aid of noncrushing clamps to isolate the area for resection (Figure 15.2a). A simple continuous mattress pattern of 5-0 or 6-0 monofilament suture is placed 3 to 5 mm proximal to the clamp (Figure 15.2b). The clamps are removed and the incision is oversewn with a simple continuous pattern (Figure 15.2c). Good technique is paramount to minimizing air leaks after surgery. This includes small suture with atraumatic needles and carefully following the curvature of the needle to minimize the size of the needle holes. Open partial lung resection can also be accomplished with a thoracoabdominal (TA) stapling device (Figure 15.3), preferably with vascular type staples. Wedge resections can be accomplished with two devices although a hole with be created at the apex of the resection that needs to be closed with sutures.

Thoracoscopic partial lung resection can be accomplished via an intercostal or transdiaphragmatic approach, both of which are described in Chapter 6. One-lung ventilation is not required for partial lung resections. After isolation of the lesion for resection or site for biopsy, a pre-tied loop suture or linear

Figure 15.5 Thoracoscopic Partial Lung Resection with Staples



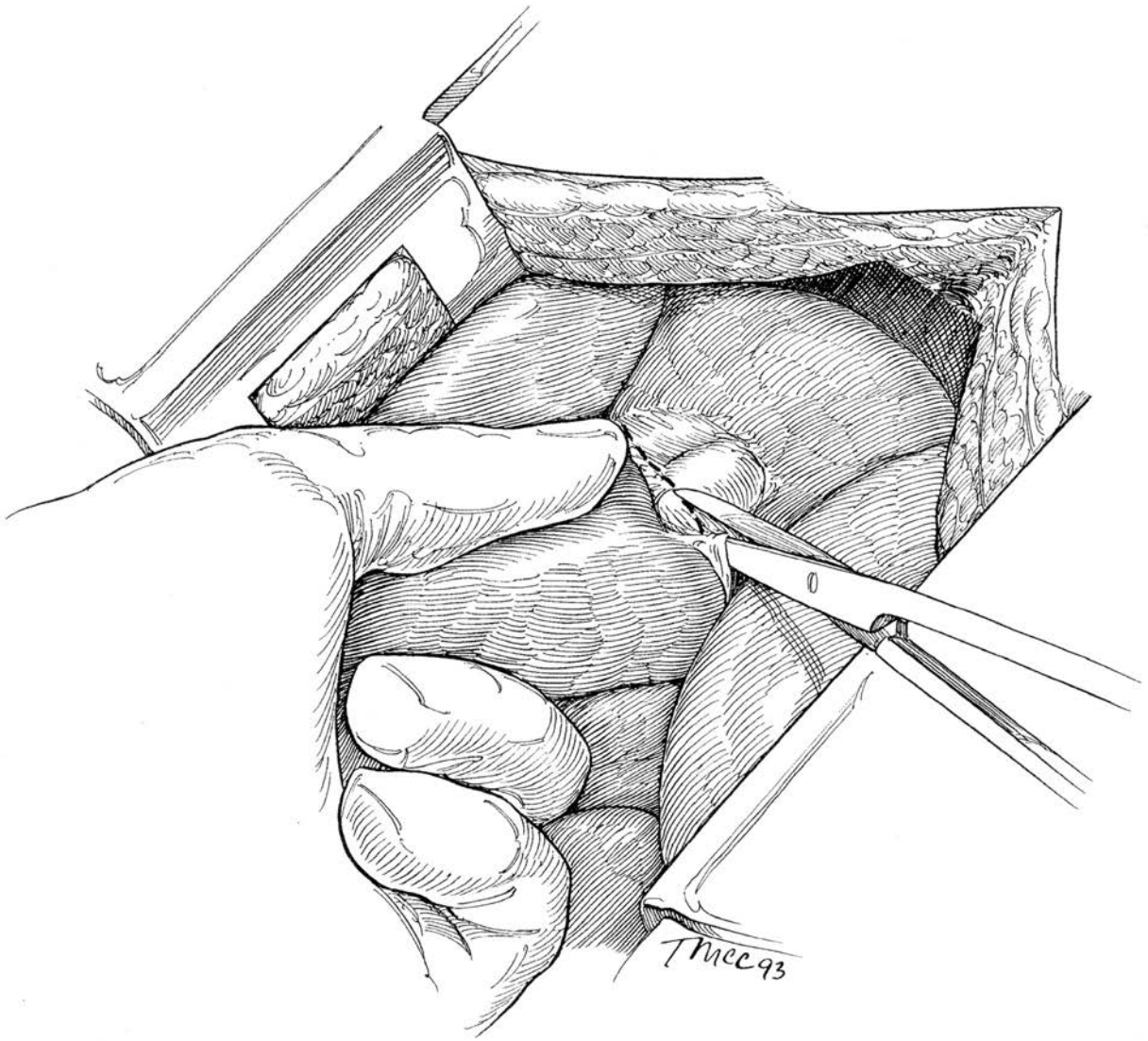


Figure 15.6 Division of Pulmonary Ligament

stapler is used to accomplish the resection. A pre-tied suture is introduced through one portal. A grasping forceps introduced through a separate portal is passed through the loop and then used to grasp the lung undergoing resection (Figure 15.4). Lung tissue is brought through the loop and the loop is then tied around the lung parenchyma. The loop should not be placed further than 2.5 cm from the edge of the lung otherwise bronchi and blood vessels too large to be safety closed with this technique might be included.

Endoscopic linear stapling devices can be used to perform larger partial lung resections (Figure 15.5). The stapling cartridges exist in lengths of 30, 45, and 60 mm. They typically deploy six rows of staples and cut between the middle rows to provide a safe seal.

Vessel sealant devices may be used to safely collect open or thoracoscopic small lung biopsy samples [18]. However, studies of biopsies taken at 3 cm from the edges of the lung leaked at inflation pressures between 10 and 40 cm of water were considered unreliable [19].

Lung Lobectomy

Lung lobectomy can be performed on one or more lung lobes. Lung lobectomy removes both available gas exchange area and pulmonary vascular bed, which can have significant consequences depending on the degree of underlying pulmonary parenchymal or vascular disease present. In general, removal of one or two lung lobes will be well tolerated. Removal of

more than 40% of the total lung risks significant gas exchange impairment and/or development of acute cor pulmonale, depending on the degree of underlying pulmonary pathology.

Lung lobectomy requires reliable closure of the pulmonary vein, pulmonary artery, bronchial artery, and lobar bronchi which can be accomplished with sutures or staples. The pulmonary artery is located cranial and dorsal and the pulmonary vein is located ventral and caudal to the bronchi. For resections of the left caudal or right caudal and the accessory lung lobes, the dorsal ligament is first divided to mobilize the lobe (Figure 15.6). The accessory lung lobe is located medial to caudal vena cava and plica vena cava and is typically removed with the right caudal lung lobe.

Surgical Pulmonary Lobectomy

Classic surgical lung lobectomy is accomplished by ligation of the pulmonary vessels and suturing of the bronchus. The pulmonary artery is isolated, triple ligated, and divided between the middle and distal ligatures (Figure 15.7a). Transfixing the middle suture decreases the risk of ligature slippage. The pulmonary vein is divided in similar fashion. The bronchial artery is also ligated. A noncrushing vascular clamp is placed on the bronchus and it is transected 1 cm distal to

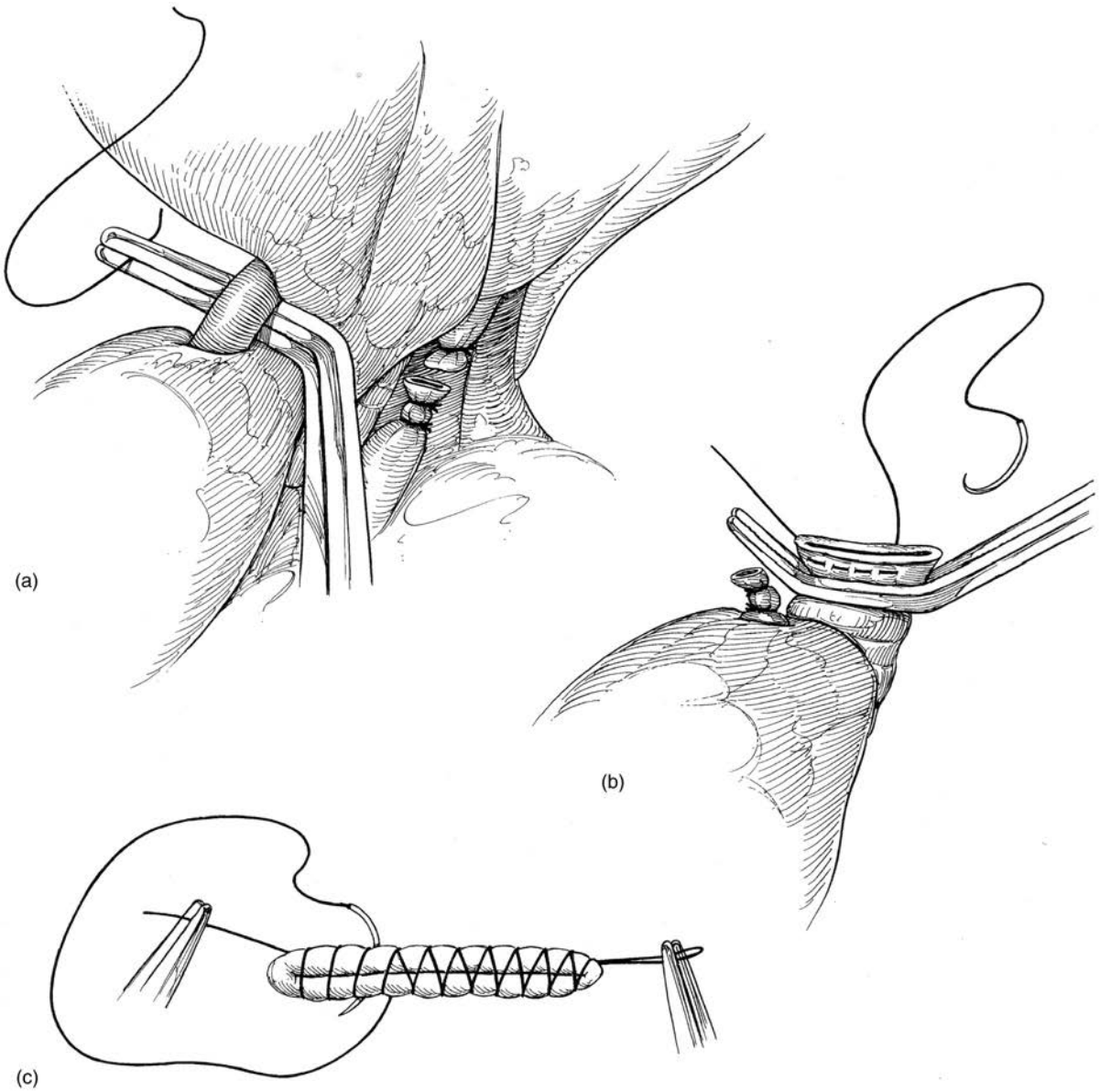


Figure 15.7 Lung Lobectomy

the clamp (Figure 15.7b). The bronchus is closed with continuous mattress suture pattern oversewn with a simple continuous pattern using 4-0 nonabsorbable monofilament suture (Figure 15.7c). The bronchus is checked for leaks by placing it under water and administering a positive pressure inhalation. Lung lobectomy has been performed safely in dogs and cats with TA stapling device [20]. A 30 mm cartridge with or without vascular staples is used. In the author's experience, stapling tends to be associated with more postoperative air leak and/or bleeding, but typically this is self-limiting.

Thoracoscopic Pulmonary Lobectomy

Thoracoscopic lung lobectomy is performed using an intercostal approach. One-lung ventilation facilitates the surgery. The technique for one-lung ventilation is described in detail in Chapter 6. After induction of anesthesia, an endobronchial blocker is placed on the side of the planned lobectomy. Capnography is used to monitor for dislodgement of the balloon into the trachea. One-lung ventilation is initiated in the operating room after the patient has been positioned for surgery.

The patient is positioned in oblique lateral recumbency with the dorsal part of the thoracic cavity elevated. Thoracoscopic lung lobectomy is performed with four intercostal access ports. Three ports are placed in the same intercostal space to minimize the number of intercostal spaces that need to be blocked after surgery and the fourth is placed one space cranial or caudal. For cranial or right middle lung lobectomy, ports are placed in the eighth or ninth intercostal space. For a caudal lung lobectomy, ports are placed in the fourth and fifth intercostal spaces. The dorsal third of the intercostal space should be avoided because the ribs are not compliant enough to allow manipulation of instruments. Five millimeter (5 mm) ports are required for the surgical instruments except the linear stapler, which requires an 11 mm port. The port for the stapler is placed after the hilus of the lung has been identified. For a caudal lung lobectomy the dorsal ligament is divided first to facilitate manipulation of the lung lobe. A fan retractor is used to retract adjacent lung lobes and help identify the hilus of the lung lobe to be resected (Figure 15.8a). The stapler is placed across the hilus keeping the stapler perpendicular to the

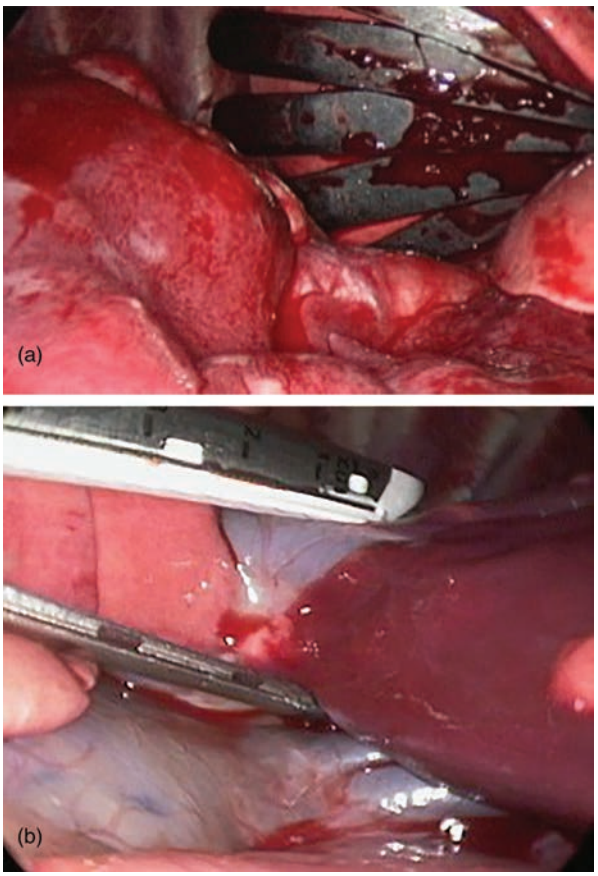


Figure 15.8 Thoracoscopic Lung Lobectomy

hilus to maximize the effective length of the stapling cartridge (Figure 15.8b). Usually, a 60 mm cartridge with 3.5 mm staples is used. If a second cartridge is needed it is overlapped with the first cartridge. Before the staples are fired it is important to make sure that other structures have not been incorporated into the stapler. The resected lung lobe is placed in a retrieving pouch to minimize the risk of contamination or seeding of the thoracic wall. The extracting port site is converted into minimal-incision thoracotomy as required to remove the resected lobe and retrieving pouch. Retraction of the thoracotomy is not used or needed, which decreases postoperative pain. A thoracostomy tube is placed under thoracoscopic visualization. The thoracotomy and cannulation sites are closed in a routine fashion.

References

1 Rebhun RB, and Culp WTN. In: Withrow SJ, Vail DM, and Page RL eds. 2013. *Small Animal Clinical Oncology*. pp 453–62. St Louis: Elseveir, Saunders.

2 Ogilvie GK, Haschek WM, Withrow SJ, Richardson RC, Harvey HJ, Henderson RA, et al. 1989. Classification of primary lung tumors in dogs: 210 cases (1975–1985). *Journal of American Veterinary Medical Association*. 195(1):106–8.

3 Ogilvie GK, Weigel RM, Haschek WM, Withrow SJ, Richardson RC, Harvey HJ, et al. 1989. Prognostic factors for tumor remission and survival in dogs after surgery for primary lung tumor: 76 cases (1975–1985). *Journal of American Veterinary Medical Association*. 195(1):109–12.

4 McNiel EA, Ogilvie GK, Powers BE, Hutchison JM, Salman MD, and Withrow SJ. 1997. Evaluation of prognostic factors for dogs with primary lung tumors: 67 cases (1985–1992). *Journal of the American Veterinary Medical Association*. 211(11): 1422–7.

5 Hahn KA, and McEntee MF. Primary lung tumors in cats: 86 cases (1979–1994). 1997. *Journal of American Veterinary Medical Association*. 211(10): 1257–60.

6 Puerto DA, Brockman DJ, Lindquist C, and Drobatz K. 2002. Surgical and nonsurgical management of and selected risk factors for spontaneous pneumothorax in dogs: 64 cases (1986-1999). *Journal of American Veterinary Medical Association*. 220(11):1670–4.

7 Lipscomb VJ, Hardie RJ, and Dubielzig RR. 2003. Spontaneous pneumothorax caused by pulmonary blebs and bullae in 12 dogs. *Journal of American Animal Hospital Association*. 39(5):435–45.

8 Mooney ET, Rozanski EA, King RG, and Sharp CR. 2012. Spontaneous pneumothorax in 35 cats (2001–2010). *Journal of Feline Medicine and Surgery*. 14(6):384–91.

9 Zekas LJ, Crawford JT, and O'Brien RT. 2005 Computed tomography-guided fine-needle aspirate and tissue-core biopsy of intrathoracic lesions in thirty dogs and cats. *Veterinary Radiology & Ultrasound*. 46(3):200–4.

10 Au JJ, Weisman DL, Stefanacci JD, and Palmisano MP. 2006. Use of computed tomography for evaluation of lung lesions associated with spontaneous pneumothorax in dogs: 12 cases (1999–2002). *Journal of American Veterinary Medical Association*. 228(5):733–7.

11 Reetz JA, Caceres AV, Suran JN, Oura TJ, Zwingenberger AL, and Mai W. 2013. Sensitivity, positive predictive value, and interobserver variability of computed tomography in the diagnosis of bullae associated with spontaneous pneumothorax in dogs: 19 cases (2003–2012). *Journal of American Veterinary Medical Association*. 243(2):244–51.

12 Neath PJ, Brockman DJ, and King LG. 2000. Lung lobe torsion in dogs: 22 cases (1981–1999). *Journal of American Veterinary Medical Association*. 217(7): 1041–4.

13 Rooney MB, Lanz O, and Monnet E. 2001. Spontaneous lung lobe torsion in two pugs. *Journal of American Animal Hospital Association*. 37(2): 128–30.

14 Hofeling AD, Jackson AH, Alsup JC, and O'Keefe D. 2004. Spontaneous midlobar lung lobe torsion in a 2-year-old Newfoundland. *Journal of American Animal Hospital Association*. 40(3):220–3.

15 d'Anjou MA, Tidwell AS, and Hecht S. 2005. Radiographic diagnosis of lung lobe torsion. *Veterinary Radiology & Ultrasound*. 46(6): 478–84.

16 Murphy KA, and Brisson BA. 2006. Evaluation of lung lobe torsion in Pugs: 7 cases (1991-2004). *Journal of American Veterinary Medical Association*. 228(1):86–90.

17 McLane MJ, and Buote NJ. 2011. Lung lobe torsion associated with chylothorax in a cat. *Journal of Feline Medicine and Surgery*. 13(2):135–8.

18 Mayhew PD, Culp WT, Pascoe PJ, and Arzi NV. 2012. Use of the Ligasure vessel-sealing device for thoracoscopic peripheral lung biopsy in healthy dogs. *Veterinary Surgery*. 41(4):523–8.

19 Marvel S, and Monnet E. 2013. Ex vivo evaluation of canine lung biopsy techniques. *Veterinary Surgery*. 42(4):473–7.

20 LaRue SM, Withrow SJ, and Wykes PM. 1987. Lung resection using surgical staples in dogs and cats. *Veterinary Surgery*. 16(3):238–40.

16

Diaphragm

E. Christopher Orton

The diaphragm separates the thoracic and abdominal cavities. As such, surgery on the diaphragm represents an intersection between thoracic and abdominal surgery. Regardless of surgical approach, surgery on the diaphragm inevitably invades the thoracic space and directly or indirectly affects thoracic organs and structures. It is therefore appropriate to consider surgical management of the diaphragmatic disorders within the realm of thoracic surgery.

Structure and Function

The diaphragm is composed of a U-shaped central tendon that constitutes about 20% of the diaphragm and two large muscular masses. The ventrolateral muscle mass is composed of radially aligned skeletal muscle that attaches the central tendon to the sternum (pars sternalis) and lateral rib cage (right and left pars costalis). The dorsomedial muscular mass (pars lumbalis) attaches the central tendon to the dorsal rib cage and lumbar spine via the diaphragmatic crura. The right and left diaphragmatic crus are tendinous extensions of the pars lumbalis that project caudally and insert on the third and fourth lumbar vertebrae.

The diaphragm has three passages: the aortic hiatus, the esophageal hiatus, and the caval foramen. Aortic hiatus is bordered dorsally by the lumbar spine and laterally and ventrally by the diaphragmatic crura. The aorta, azygous vein, and cistern of the thoracic duct pass through the aortic hiatus. The esophageal hiatus is located on the midline at the junction of the central tendon and the right and left pars lumbalis muscles. The esophagus and the dorsal and ventral vagal trunks pass through esophageal hiatus. The caval foramen lies within the dorsal portion of the central tendon to the right of midline. The caudal vena cava passes through the caval foramen.

The thoracic surface of the diaphragm is covered by parietal pleura. The pleura reflects off the diaphragm onto the caudal mediastinum, which transmits the esophagus, aorta, and apical portion of the pericardium to the diaphragm; and onto the plica vena cava, which accompanies the caudal vena cava. The diaphragm is innervated by the right and left phrenic nerves, which originate from the cervical nerve roots and enter the thorax through the thoracic inlet. The right phrenic nerve courses along the right lateral aspect of the cranial vena cava, pericardium, and caudal vena cava to innervate the right side of the diaphragm. The left phrenic nerve courses along the left lateral mediastinum and pericardium to innervate the left side of the diaphragm. Contraction of the diaphragm plays a major role in pulmonary ventilation by expanding the volume of the thorax cavity in a caudal direction. This action combines with craniolateral expansion of the thoracic wall by the intrinsic and extrinsic inspiratory thoracic musculature to accomplish normal ventilation during rest and exercise. While it is possible for either the diaphragmatic or thoracic wall phases of ventilation to maintain adequate ventilation in normal animals at rest, in reality, bilateral paralysis of the diaphragm is generally poorly tolerated especially if any degree of pulmonary pathology and accompanying mechanical dysfunction (increased airway resistance or decreased pulmonary compliance, or both) is present. Thus, a very high priority must be placed on preserving the integrity of the phrenic nerves during thoracic surgery.

Surgical Conditions of the Diaphragm

Traumatic Diaphragmatic Hernia

Traumatic diaphragmatic hernia is the most common surgical condition of diaphragm in dogs and cats. Several large case studies in dogs and cats have been reported [1–6]. Blunt automobile trauma is the most common cause, although other types of trauma,

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including penetrating injuries, can result in diaphragmatic hernia. Not surprisingly, diaphragmatic hernia is associated with a high incidence of injury to multiple organs. The most likely site of diaphragmatic rupture is within and/or at the attachment of the ventrolateral muscle mass (pars costa or pars sternalis). Chronic diaphragmatic hernia (> 2 weeks after trauma) occurs with surprising frequency, emphasizing the importance of screening animals that experience significant blunt trauma for this injury [5]. Adhesions necessitating a thoracic approach or combined thoracic-abdominal approach are more likely to occur with chronic diaphragmatic hernias.

A high index of suspicion for diaphragmatic hernia should be carried for dogs and cats experiencing significant blunt trauma. Findings supportive of the diagnosis on physical examination include unilaterally muffled heart and/or lung sounds, dullness on thoracic percussion, tachypnea/dyspnea with paradoxical movement of the abdomen during inspiration, and an empty appearance to the abdomen. Diagnosis is typically confirmed by diagnostic imaging, either radiographs or survey ultrasonography of the pleural space. A lateral thoracic radiograph is generally regarded as the safest and most sensitive view for diagnosis, although two views increase diagnostic sensitivity [7].

Early case series of dogs and cats with traumatic diaphragmatic hernia suggested high mortality rates in animals undergoing surgery [2]. For this reason, some surgeons have historically advocated for delayed surgical repair of traumatic diaphragmatic hernia. However, current thinking is that animals should undergo surgical repair as soon as they can be stabilized, and certainly within 24 hours of presentation. More recent reports suggest mortality rates of about 10% when surgery is undertaken within the first 24 hours [3, 6]. Somewhat higher complication and mortality rates can be expected in animals undergoing repair for chronic traumatic diaphragmatic hernias [5].

Congenital Pleuroperitoneal Diaphragmatic Hernia

Congenital pleuroperitoneal hernia is a rare condition in dogs and cats [8–11]. In dogs, the condition is suspected to have an autosomal recessive mode of inheritance [11]. The defect appears to occur most commonly in the dorsal muscle mass of the diaphragmatic crura (pars lumbalis). The defect can be associated with serious pulmonary anomalies. In older animals, the defect is more likely to be an incidental finding and can mimic a pulmonary mass. Primary repair of congenital pleuroperitoneal diaphragmatic hernia can be undertaken either by direct closure or patching with autogenous grafts or synthetic materials.

Peritoneopericardial Diaphragmatic Hernia

Peritoneopericardial diaphragmatic hernia is a commonly described congenital defect in dogs and cats [12–14]. The defect is found incidentally without apparent clinical signs in up to 40% of cases. Clinical signs when present are related to respiratory or gastrointestinal compromise. Cats are more likely to present with respiratory-related clinical signs, whereas dogs present more frequently with gastrointestinal signs. Cardiovascular signs related to acute or chronic cardiac tamponade are also possible. The diagnosis is readily confirmed by demonstration of abdominal viscera within the confines of the pericardial sac on thoracic radiography or echocardiography. Conservative management has been successfully employed in animals without clinical signs. Animals with clinical signs related to peritoneopericardial hernia should undergo surgical repair. Surgical repair is accomplished by reduction of abdominal viscera and direct closure. Surgery is associated with a high success rate and resolution of clinical signs in both dogs and cats.

Hiatal Hernia

Hiatal hernia is a condition in which an abdominal structure, typically the stomach, passes through the esophageal hiatus into the mediastinum. The condition is more correctly an eventration, because the phrenicoesophageal ligament usually remains intact. Sliding (type I), paraesophageal (type II), and combined (type III) types of hiatal herniation of the stomach have been reported in dogs and cats [15–19]. Concurrent herniation of liver and small intestine (type IV) through the esophageal hiatus has also been reported [20]. English bulldogs and Chinese Shar-pei dogs are predisposed to the condition [21]. Hiatal hernia has been reported as a complication after repair of chronic traumatic diaphragmatic hernia [22]. Paralysis of the diaphragm is also thought to predispose to the condition.

Hiatal hernia is occasionally an incidental finding without apparent clinical signs. Regurgitation secondary to reflux esophagitis and possible secondary megaesophagus is the most common clinical sign. Other clinical signs can include ptyalism, vomiting, anorexia, and weight loss. Respiratory distress and cough caused by secondary aspiration pneumonia are also possible. Clinical signs typically develop at an early age, although they sometimes develop later in life. Diagnosis is suspected based on plain thoracic radiograph, and confirmed by positive-contrast esophagram. Fluoroscopic evaluation may be necessary to document intermittent type I hernias.

Esophagoscopy is useful to evaluate for concurrent reflux esophagitis.

Medical management of hiatal hernia is focused on reducing gastric acidity, mucosal protection, increasing gastric emptying, and augmenting the lower esophageal sphincter. A significant percentage of animals will respond to medical therapy alone [15]. Surgical repair is indicated for animals in which clinical signs are not completely resolved or recur. Surgery consists of reduction of the size of the esophageal hiatus, esophagoplasty, and left-sided gastropexy. Most dogs with a predisposition for hiatal hernia, such as the Shar-pei, will require surgery [21, 23]. Surgery is generally successful at relieving clinical signs associated with hiatal hernia [15, 16, 21, 23, 24].

Surgery of the Diaphragm

Access to the diaphragm can be obtained via midline celiotomy, sternotomy, or lateral thoracotomy; or combinations of these. The most commonly employed approach for diaphragmatic hernia repair is midline celiotomy, which facilitates reduction, inspection, and return of abdominal organs to their normal anatomical position. This approach also allows development of an abdominal muscle flap to close a defect in the diaphragm if necessary. Midline celiotomy can be combined with a sternotomy if more direct access to the thoracic cavity to relieve adhesions of herniated organs or repair thoracic structures is needed. The disadvantage of an abdominal approach is that access is provided to the concave surface of the diaphragm, which makes suturing more difficult. A caudal (eighth to tenth) thoracotomy has been advocated by some surgeons because it provides more direct access the diaphragm facilitating suturing and repair [3]. It also allows direct inspection of thoracic structures. The disadvantages of thoracotomy are that it provides only unilateral access to the diaphragm and it is likely more painful compared to an abdominal approach.

Traumatic Diaphragmatic Hernia Repair

Repair of traumatic diaphragmatic herniation is typically accomplished via a midline celiotomy, which allows inspection of the entire diaphragm and facilitates reduction and inspection of herniated abdominal viscera (Figure 16.1a). Most traumatic diaphragmatic hernias occur within or at the attachment of the ventrolateral muscle mass (pars sternalis or pars costalis) to the body wall, or a combination of these. Herniated abdominal viscera are reduced, inspected, and returned to their normal anatomic position within the abdomen. A subcostal thoracostomy tube is placed lateral to the abdominal incision and positioned in the ventral thoracic cavity prior to closing the hernia. The diaphragm is reconstructed by a combination of repairing ruptures within the muscle and reattaching the diaphragm to the body wall using interrupted mattress sutures (Figure 16.1b). The repair is oversewn with a simple continuous pattern to achieve an airtight seal. The thoracic space is evacuated of air and fluid via the thoracostomy tube. As the abdominal approach is closed, gentle compression is applied to the abdomen to remove as much air as possible from the abdominal cavity. The thoracostomy tube is maintained for at least several hours after surgery to remove any air or fluid that crosses the repair from the abdomen.

Transverse Abdominis Muscle Flap Repair of Diaphragmatic Defects

Defects in the diaphragm can result from severe injury requiring debridement, contracture of chronic traumatic diaphragmatic ruptures, excision of diaphragmatic masses, or congenital diaphragmatic hernias. Such defects can be closed with a muscular flap developed from the transverse abdominis muscle. The repair is performed via a midline celiotomy approach. A rectangular muscle flap is developed with its base at the cranial end of the flap near the normal attachment of the diaphragm to the body wall (Figure 16.2a). A flap is elevated from the underlying abdominal oblique muscle and folded back to cover the defect (Figure 16.2b). The flap is sutured to the diaphragm with interrupted mattress sutures oversewn with a simple continuous pattern. A subcostal thoracostomy tube is placed before closure to the diaphragmatic defect to remove air and fluid from the thoracic space after surgery.

Repair of Peritoneopericardial Hernia

Repair of congenital peritoneopericardial hernia is typically accomplished through the midline celiotomy approach. The defect is found at the midline of the ventral diaphragm. The repair is similar to that described for pleuroperitoneal diaphragmatic hernia repair. Herniated abdominal viscera are reduced from the pericardial sac, inspected and returned to their normal position in the abdomen. Adhesions are gently broken down digitally. If this fails or if bleeding occurs, the midline celiotomy can be extended into a caudal sternotomy and the ventral pericardium can be opened to facilitate exposure and reduction of herniated viscera. After reducing the hernia, the rim of the hernia consisting of the fused pericardial sac and diaphragm are closed in a single layer with mattress sutures. Typically, the pleural space is not

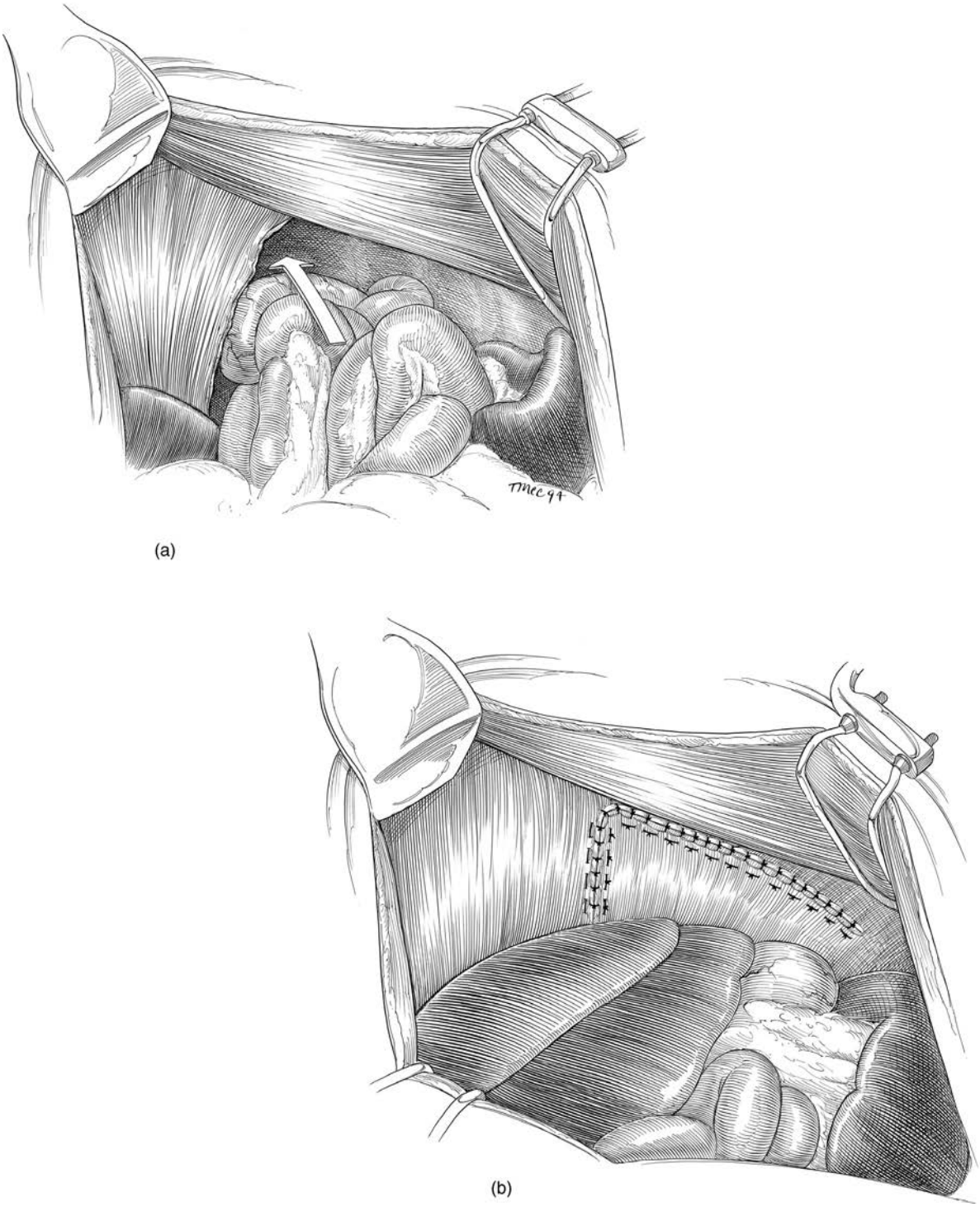

Figure 16.1 Diaphragmatic Hernia Repair

entered during repair of peritoneopericardial hernia so it may not be necessary to place a thoracostomy tube. If the pericardial space is large, evacuation is accomplished by placing a tube across the repair and out the abdominal incision. The tube can then be pulled back into the abdomen to evacuate air from the abdomen after the celiotomy is closed. Enlarging the hernia orifice to facilitate reduction invariably opens the pleural space, and in this case a subcostal thoracostomy tube should be placed prior to surgical repair and closure of the abdomen.

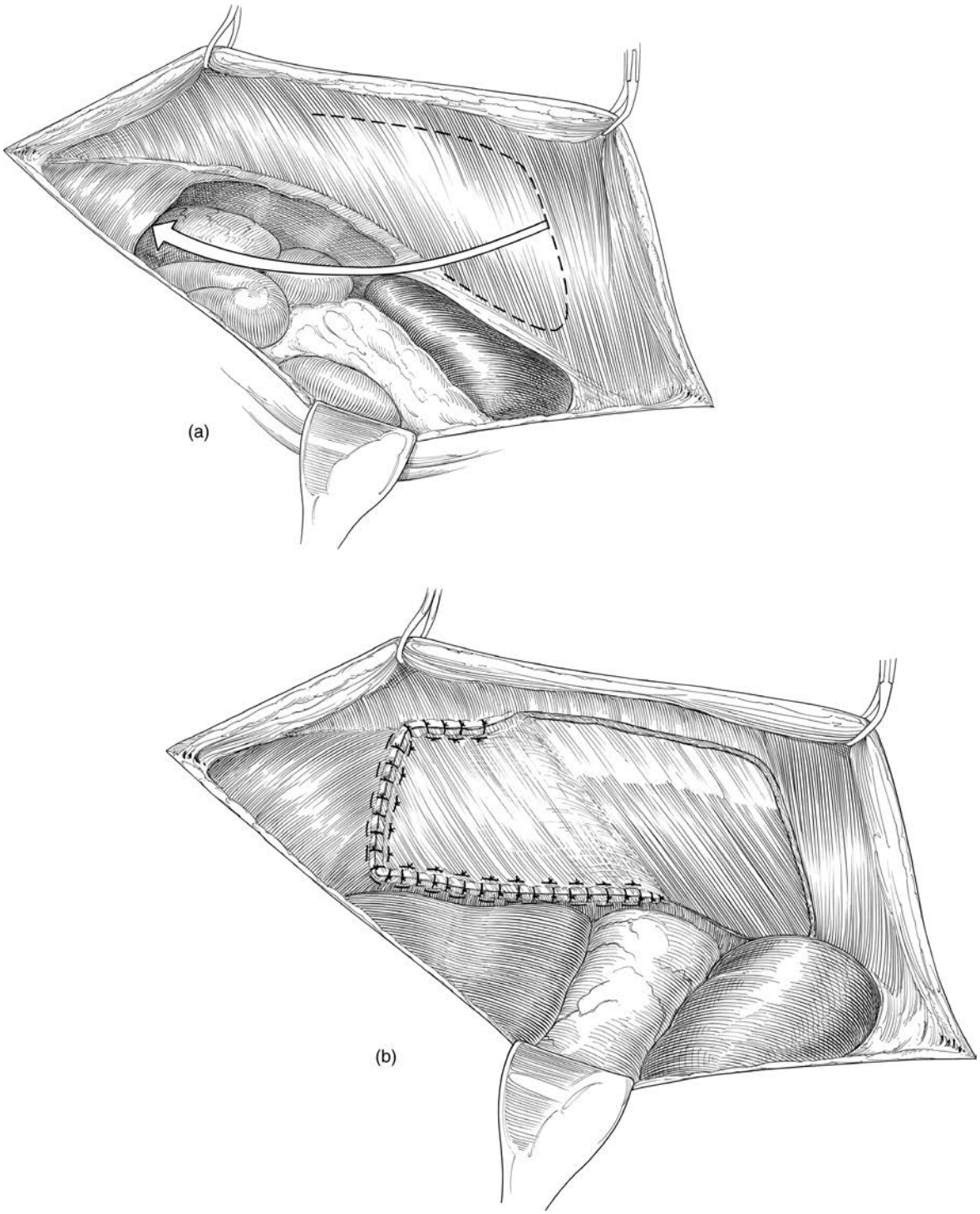

Figure 16.2 Transverse Abdominus Flap Repair of Diaphragm

Repair of Hiatal Hernia

Hiatal hernia is more correctly eventration of the stomach through the esophageal hiatus, rather than a true hernia. A sliding (type I) herniation of the stomach is most common in dogs and cats (Figure 16.3a), although paraesophageal (type II) hernias also occur. The condition results from the combination of a redundant phrenicoesophageal ligament and laxity of the muscular esophageal

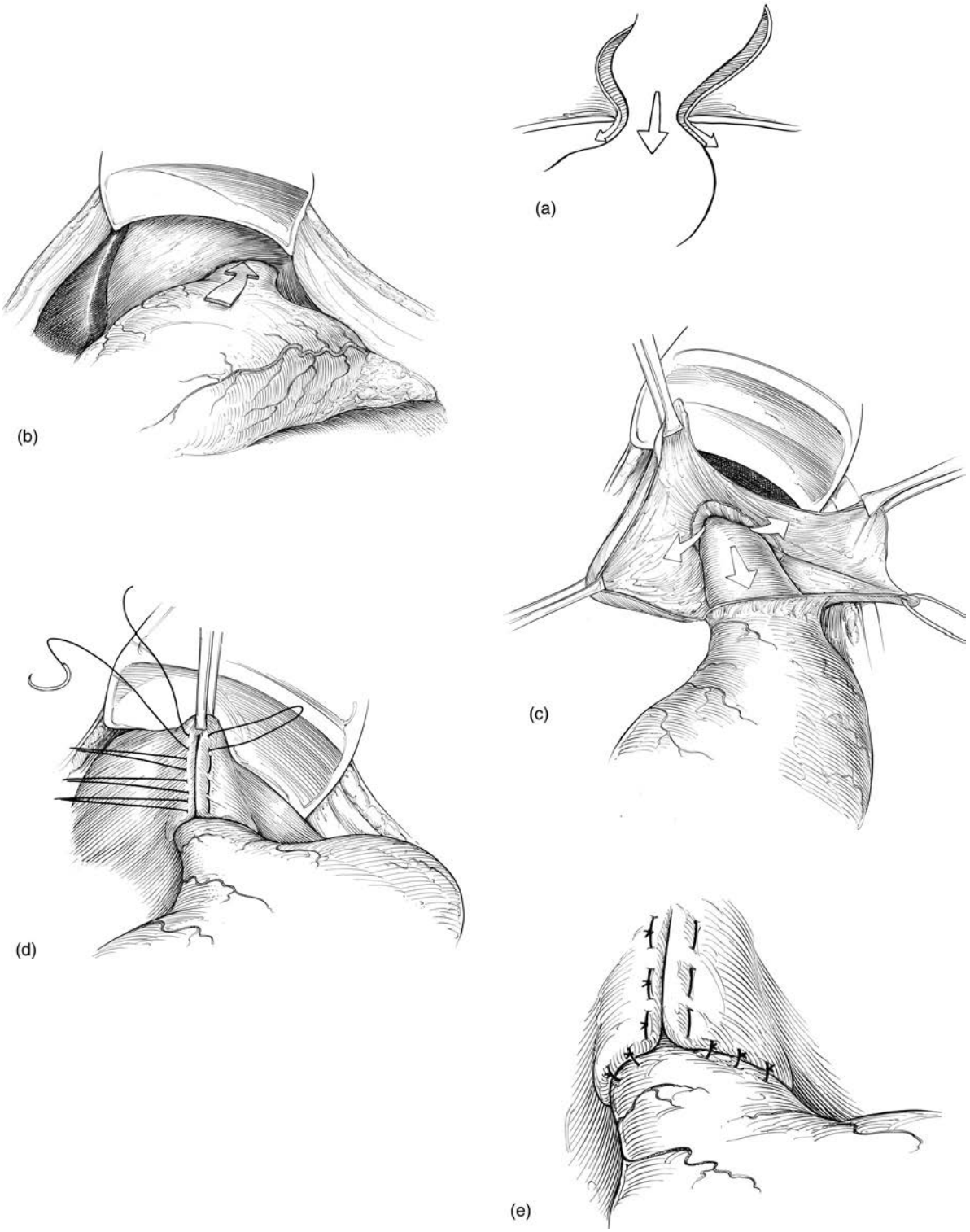

Figure 16.3 Hiatal Hernia Repair

hiatus. Repair of hiatal hernia is accomplished from a midline celiotomy (Figure 16.3b). The stomach is reduced back into the abdomen. Our preference is to excise the redundant phrenicoesophageal ligament, taking care to visualize and preserve the dorsal and ventral vagal trunks (Figure 16.3c). The size of the esophageal hiatus is judiciously reduced by placing mattress sutures in the musculature borders of the esophageal hiatus (Figure 16.3d). Interrupted sutures are placed between the abdominal esophagus and hiatus (esophagoplasty) to further reduce the risk of sliding (Figure 16.3e). A left-sided gastropexy procedure is added to the repair to further reduce the risk of gastric herniation after surgery. Hiatal hernia repair can be combined with a fundoplication procedure to reinforce the integrity of the lower esophageal sphincter if this is judged to be necessary [16, 17]. Evidence supporting effectiveness and need for fundoplication in animals is lacking.

Care after Surgery

Animals undergoing surgical repair for traumatic diaphragmatic hernia will likely have significant ongoing cardiopulmonary compromise related to multiple trauma. Concurrent injuries include pulmonary contusion, direct or indirect cardiac injury, bleeding, systemic inflammatory response, multisystem organ injury, and orthopedic trauma. Continued supportive care after surgery is critical to success. Emphasis is placed on assessment and support of ventilation given the important role the diaphragm plays in ventilation. Adequacy of ventilation is assessed by measurement P_aCO_2. Support of ventilation may be necessary for a period of time after surgery. Supplemental oxygen helps correct impaired pulmonary gas exchange associated with pulmonary injury. Ongoing management of the pleural space is critical after surgery.

Animals undergoing repair for chronic diaphragmatic hernia are at risk for *reexpansion pulmonary edema*. Reexpansion pulmonary edema occurs when lungs that have been atelectatic for an extended period are rapidly reinflated. The mechanism is considered multifactorial but likely involves a combination of decreased surfactant production resulting in high transmural hydrostatic pressures and vascular leak from the release of inflammatory mediators. There is no clear consensus on prevention of reexpansion pulmonary edema. Aggressive correction of chronic pulmonary atelectasis by positive pressure inflations while the thoracic space is open should be avoided. Allowing fluid or air to remain in the pleural space after surgery is controversial and probably should be also avoided. Treatment is best accomplished through supportive therapies by administration of supplemental oxygen, avoiding overadministration of crystalloid fluids, and mechanical ventilatory support if necessary.

References

1 Wilson GP, 3rd, Newton CD, and Burt JK. 1971. A review of 116 diaphragmatic hernias in dogs and cats. *Journal of the American Veterinary Medical Association*. 159(9):1142–5.

2 Garson HL, Dodman NH, and Baker GJ. 1980. Diaphragmatic hernia. Analysis of fifty-six cases in dogs and cats. *The Journal of Small Animal Practice*. 21(9):469–81.

3 Stokhof AA. 1986. Diagnosis and treatment of acquired diaphragmatic hernia by thoracotomy in 49 dogs and 72 cats. *Veterinary Quarterly*. 8(3):177–83.

4 Schmiedt CW, Tobias KM, and Stevenson MA. 2003. Traumatic diaphragmatic hernia in cats: 34 cases (1991-2001). *Journal of the American Veterinary Medical Association*. 222(9):1237–40.

5 Minihan AC, Berg J, and Evans KL. 2004. Chronic diaphragmatic hernia in 34 dogs and 16 cats. *Journal of the American Animal Hospital Association*. 40(1): 51–63.

6 Gibson TW, Brisson BA, and Sears W. 2005. Perioperative survival rates after surgery for diaphragmatic hernia in dogs and cats: 92 cases (1990-2002). *Journal of the American Veterinary Medical Association*. 227(1):105–9.

7 Hyun C. 2004. Radiographic diagnosis of diaphragmatic hernia: review of 60 cases in dogs and cats. *Journal of Veterinary Science*. 5(2):157–62.

8 Feldman DB, Bree MM, and Cohen BJ. 1968. Congenital diaphragmatic hernia in neonatal dogs. *Journal of the American Veterinary Medical Association*. 153(7):942–4.

9 White JD, Tisdall PL, Norris JM, and Malik R. 2003. Diaphragmatic hernia in a cat mimicking a pulmonary mass. *Journal of Feline Medicine and Surgery*. 5(3):197–201.

10 Voges AK, Bertrand S, Hill RC, Neuwirth L, and Schaer M. 1997. True diaphragmatic hernia in a cat. *Veterinary Radiology and Ultrasound*. 38(2):116–9.

11 Valentine BA, Cooper BJ, Dietze AE, and Noden DM. 1988. Canine congenital diaphragmatic hernia. *Journal of Veterinary Internal Medicine*. 2(3): 109–12.

12 Banz AC, and Gottfried SD. 2010. Peritoneopericardial diaphragmatic hernia: a retrospective study of 31 cats and eight dogs. *Journal of the American Animal Hospital Association*. 46(6):398–404.

13 Burns CG, Bergh MS, and McLoughlin MA. 2013 Surgical and nonsurgical treatment of peritoneopericardial diaphragmatic hernia in dogs and cats: 58 cases (1999–2008). *Journal of the American Veterinary Medical Association*. 242(5): 643–50.

14 Reimer SB, Kyles AE, Filipowicz DE, and Gregory CR. 2004. Long-term outcome of cats treated conservatively or surgically for peritoneopericardial diaphragmatic hernia: 66 cases (1987–2002). *Journal of the American Veterinary Medical Association*. 224(5):728–32.

15 Lorinson D, and Bright RM. 1998 Long-term outcome of medical and surgical treatment of hiatal hernias in dogs and cats: 27 cases (1978–1996). *Journal of the American Veterinary Medical Association*. 213(3):381–4.

16 Bright RM, Sackman JE, DeNovo C, and Toal C. 1990. Hiatal hernia in the dog and cat: A retropective study of 16 cases. *The Journal of Small Animal Practice*. 31:244–50.

17 Ellison G, Lewis D, and Philips L. 1987. Esophageal hiatal hernia in small animals: literature review and a modified surgical technique. *Journal of the American Animal Hospital Association*. 23:391–9.

18 Miles KG, Pope ER, and Jergens AE. 1988. Paraesophageal hiatal hernia and pyloric obstruction in a dog. *Journal of the American Veterinary Medical Association*. 193(11): 1437–9.

19 Kirkby KA, Bright RM, and Owen HD. 2005. Paraoesophageal hiatal hernia and megaoesophagus in a three-week-old Alaskan malamute. *The Journal of Small Animal Practice*. 46(8):402–5.

20 Rahal SC, Mamprim MJ, Muniz LM, and Teixeira CR. 2003. Type-4 esophageal hiatal hernia in a Chinese Shar-pei dog. *Veterinary Radiology and Ultrasound*. 44(6):646–7.

21 Guiot LP, Lansdowne JL, Rouppert P, and Stanley BJ. 2008. Hiatal hernia in the dog: a clinical report of four Chinese shar peis. *Journal of the American Animal Hospital Association*. 44(6):335–41.

22 Pratschke KM, Hughes JM, Skelly C, and Bellenger CR. 1998. Hiatal herniation as a complication of chronic diaphragmatic herniation. *The Journal of Small Animal Practice*. 39(1):33–8.

23 Callan MB, Washabau RJ, Saunders HM, Kerr L, Prymak C, and Holt D. 1993. Congenital esophageal hiatal hernia in the Chinese shar-pei dog. *Journal of Veterinary Internal Medicine*. 7(4):210–5.

24 Prymak C, Saunders HM, and Washabau RJ. 1989. Hiatal hernia repair by restoration and stabilization of normal anatomy. An evaluation in four dogs and one cat. *Veterinary Surgery*. 18(5):386–91.

Section V

Cardiac Surgery

17

Pericardium

Eric Monnet and E. Christopher Orton

Structure and Function

The pericardium is a saclike structure that envelopes the heart and origins of the great vessels. It is divided into the parietal and visceral pericardium, with the pericardial cavity in between. The parietal pericardium consists of an outer fibrous portion that blends with the adventitia of the great vessels and an inner serous portion. The fibrous pericardium is around 2 mm thick, has low cellularity, and is composed of collagen and elastin fibers. The collagen fibers are wavy, allowing a degree of stretch until it reaches its elastic limit, after which it stiffens exponentially [1].

The serous pericardium forms a closed mesothelium-lined cavity consisting of the visceral pericardium, which forms the epicardium and the serous portion of the parietal pericardium that lines the inside of the fibrous pericardium. The stromal layer of the visceral pericardium (epicardium) consists predominately of elastic fibers. The apex of the fibrous pericardium is anchored to the ventral diaphragm and sternum by the sternopericardic ligament. The pericardium is supplied by pericardial arteries that course with the sternopericardic ligament and pericardiophrenic arteries that course with the phrenic nerves. Both are branches of the internal thoracic arteries.

The pericardium maintains the heart in its normal anatomic position in the thorax by its attachment to the sternal portion of the diaphragm. The pericardium also constrains cardiac filling and enhances diastolic ventricular coupling. By this mechanism, the pericardium prevents cardiac overdistention and helps balance the output of the right and left ventricles. At low cardiac volumes, intrapericardial pressure is zero or negative, and the pericardium exerts little effect on cardiac filling. An intact pericardium protects against atrial rupture in dogs with mitral insufficiency and myocardial hemorrhage induced by acute right-sided heart failure. It may also prevent the spread of infection or neoplasia from the pleural space to the heart.

The pericardium provides a gliding surface to accommodate heart motion. The pericardial cavity is filled with a variable amount of pericardial fluid. In dogs, fluid volumes range from 1 to 15 mL. Pericardial fluid is an ultrafiltrate of serum that contains phospholipids for lubrication, a protein content of 1.7 to 3.5 g/dL, and colloid osmotic pressure approximately 25% of that seen in serum [2]. Because the pericardium is noncompliant and has a small reserve volume, intrapericardial pressure rises rapidly when the volume increases acutely. Chronic stretching of the pericardium results in remodeling of the extracellular matrix and augmentation of the pericardial volume.

Pathophysiology

Capacitance of the pericardium is influenced by rate of fluid accumulation. Because parietal pericardium is fairly noncompliant, pericardial pressure begins to increase after 5 to 60 mL of fluid accumulates acutely within the pericardial sac. With slow accumulation, the pericardium stretches, permitting augmentation of pericardial volume and rightward shifting of the pressure-volume curve. As a result, the pericardium can accumulate a larger volume of fluid before pressure begins to increase. However, beyond a certain point, pressure increases quickly with small increases in volume. When the pericardium is thickened, as is the case with constrictive pericardial disease, a minor increase in volume causes a significant increase in pericardial pressure [3].

An increase in pericardial pressure increases diastolic pressure within the heart, which in turn reduces cardiac filling and stroke volume. Pericardial pressure first equilibrates with right ventricular filling pressure (right-sided heart tamponade) and then with left ventricular filling pressure (left-sided heart tamponade).

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With tamponade, cardiac output decreases and systemic venous pressure increases, stimulating activation of compensatory neuroendocrine responses to increase vascular volume and maintain blood pressure [3]. With activation of the renin-angiotensin-aldosterone system, sodium and water are retained. Sympathetic stimulation and adrenomedullary catecholamine release produce positive inotropic and chronotropic effects and vasoconstriction. Because atrial wall stretching is limited by tamponade, atrial natriuretic peptide is not released with pericardial effusion and is therefore not available to counteract the effects of the renin-angiotensin-aldosterone system. As a result, cardiac tamponade is associated with increases in systemic venous and portal pressures, causing jugular vein distention, liver congestion, ascites, and peripheral edema secondary to fluid transudation from systemic capillary beds. Although cardiac contractility is not directly affected by tamponade, compression of coronary arteries results in poor myocardial perfusion. When coupled with decreased cardiac output and arterial hypotension, cardiogenic shock and death may result.

Arterial pressures may vary paradoxically with respiration during severe cardiac tamponade. During inspiration, pericardial pressure and right ventricular pressure decrease, facilitating venous return to the right atrium and ventricle and pulmonary blood flow. However, because heart volume is limited by the pericardium, the intraventricular septum shifts to the left. Consequently, left ventricular end-diastolic volume, left heart output, and arterial pressure are decreased during inspiration, resulting in variation of systolic arterial pressures often greater than 10 mm Hg. This phenomenon, known as pulsus paradoxus, can also occur with obstructive lung disease, restrictive cardiomyopathy, constrictive pericarditis, or hypovolemic shock and is therefore not pathognomonic for cardiac tamponade [3–5].

Pericardial Effusion

Pericardial effusions are categorized by characteristics of the accumulated fluid. A transudative pericardial effusion may occur with congestive heart failure, peritoneopericardial diaphragmatic hernia, hypoalbuminemia, or increased vascular permeability [6–11]. An exudate (total protein >2.5 g/dL; total nucleated cell count >5000 cells/μL) results from infectious or noninfectious pericarditis, such as feline infectious peritonitis [12]. Infectious agents can be bacterial, fungal, or viral. Fungal pericarditis is uncommon, with the exception of *Coccidioides immitis* in dogs living in the southwestern United States [13]. Bacterial pericardial effusion has been reported in dogs and is suspected to be secondary to migration of plant materials [12].

Causes of hemorrhagic pericardial effusion include trauma, neoplasia, anticoagulant intoxication, or rupture of the left atrium secondary to mitral valve disease [14, 15]. If an underlying cause cannot be determined, it is classified as idiopathic pericardial effusion. Idiopathic pericardial effusion is considered by some authors to be the most common cause of acute or chronic hemorrhagic pericardial effusion in dogs [16]. Some dogs with idiopathic pericardial effusion may actually have a small undetected intrapericardial tumor or mesothelioma. Although infectious agents are an unlikely cause, influenza type A viral ribonucleic acid was detected in pericardial fluid of 1 in 14 dogs with idiopathic pericardial effusion [7].

The second most common cause of hemorrhagic pericardial effusion is neoplasia of the heart, heart base, or pericardium, with hemangiosarcoma of the right atrium being most common [15,17,18]. Hemangiosarcoma is sometimes multicentric, involving the spleen or liver at the time pericardial effusion is detected. Chemodectoma is the second most common cardiac tumor to cause pericardial effusion and is most often seen in brachycephalic dogs. Hemorrhagic pericardial effusion may also be caused by pericardial mesothelioma. This diffuse neoplasm of the pericardium and other serosal surfaces may be difficult to distinguish from idiopathic pericardial effusion even with pericardial histopathology and immunohistochemistry [19].

Diagnosis

Echocardiography is very sensitive for diagnosis of pericardial effusion and can detect as little as 15 mL of fluid.The classic echocardiographic finding in pericardial effusion is an anechoic space between the epicardium and parietal pericardium. Right and left ventricular dimensions are often diminished, and ventricular walls appear thicker than normal when pericardial effusion is severe and cardiac filling is impaired. Collapse of the right atrium or ventricle during diastole suggests significant increase in intrapericardial pressure and cardiac tamponade. Absence of these findings, however, does not exclude significant impairment of the cardiac function [20, 21].

Echocardiography allows visualization of cardiac masses or myocardial infiltration. The presence of pericardial fluid greatly enhances detection of intrapericardial masses. The location of a mass, although not definitive, is often suggestive of a

diagnosis. Right atrial masses are most likely to be hemangiosarcomas, and masses along the ascending aorta are likely to be chemodectoma. Myocardial infiltration, visible as diffuse hyperechogenicity in a cat, is suggestive of lymphosarcoma [22]. Two-dimensional echocardiography has been reported to be 80% to 90% sensitive for the detection of cardiac masses in dogs; however, false-negative results are possible [24].

Surgery

Surgical options for management of pericardial effusions include subtotal pericardiectomy and pericardial window. Subtotal pericardiectomy offers more definitive resolution of pericardial effusion and has the advantage of more complete removal of pathologic pericardial tissues. A pericardial window is less invasive, but tends to be more palliative compared to pericardiectomy.

Subtotal Pericardiectomy

The goal of subtotal pericardiectomy is excision of the entire parietal pericardium ventral to the phrenic nerves. Subtotal pericardiectomy results in more definitive resolution of pericardial effusion by removing pathologic tissues that are presumably a major source of the effusion. Complications of pericardial windows—such as herniation of the heart, ongoing effusion, or late closure—are avoided. Subtotal pericardiectomy can be accomplished via thoracotomy, sternotomy, or transdiaphragmatic thoracoscopy.

Thoracotomy Approach

Subtotal pericardiectomy can be performed via a right fifth thoracotomy in dogs. The principal advantage of performing pericardiectomy via thoracotomy is the avoidance of intraoperative hemodynamic compromise and postoperative pain associated with sternotomy. It provides better visualization of the vena cavae during excision. It also provides direct access to cardiac structures such as the right auricle in the event that combined cardiac surgical procedures are needed. The principal disadvantages of pericardiectomy via thoracotomy are that it does not allow visualization of the opposite phrenic nerve and it typically requires a brief lifting of the heart to complete the excision.

A T-shape incision is made in the pericardium along the cardiac base 1 cm ventral to the phrenic nerve (Figure 17.1a). The incision is extended toward the apex using an electroscalpel if possible to increase hemostasis (Figure 17.1b). The incision is extended cranially and caudally around base of the heart taking care not to overretract the pericardium to avoid incising the vena cavae (Figure 17.1 c). The incision is then extended to the opposite side while an assistant briefly lifts the apex of the heart (Figure 17.1d). If the pericardium is thickened, then it might not be possible to visualize the left phrenic nerve through the pericardium. In this case, the level of the incision must be estimated. It is best to be conservative and ensure that the incision is well below the opposite phrenic nerve. The heart is released into the thoracic cavity and the sternopericardic ligament and associated blood supply are divided with an electroscalpel or ligated (Figure 17.1e). A thoracostomy tube is placed and the intercostal thoracotomy is closed in a routine fashion.

Sternotomy Approach

Performing a subtotal pericardiectomy via a sternotomy has the principal advantage of allowing direct visualization of both phrenic nerves. A sternotomy approach is required if constrictive pericardial disease is suspected (see section on Constrictive Pericarditis). Pericardiectomy is started from the apex of the heart and extended cranially toward the great vessels. Incision is then extended caudally on each side approximately 1 cm ventral to the phrenic nerves, taking care to identify the vena cavae by not overretracting the pericardial tissues. Electrocautery can be used to control bleeding; however, touching the epicardium with electrocautery should be avoided. The heart is gently retracted to complete the incision caudally. Excision of the pericardium is completed by dividing the sternopericardic ligament and associated vasculature.

Subxyphoid Thoracoscopy

Pericardiectomy can be accomplished by video-assisted thoracoscopy using a transdiaphragmatic subxyphoid approach [24, 25]. The transdiaphragmatic subxyphoid approach gives access to both sides of the thoracic cavity and can be performed with or without one-lung ventilation [23, 24]. One-lung ventilation allows for better visualization of the phrenic nerves, but does not eliminate the risk of phrenic nerve damage [24]. The technique for one-lung ventilation is described in Chapter 6. Induction of one-lung ventilation can induce hypoxemia and the patient should be closely monitored with blood gases during the procedure [24,25]. Intercostal ports are placed in the left and right ninth or tenth intercostal space. The mediastinum is dissected with electrocautery or a vessel sealant device. The pericardium is grasped with 5 mm fine teeth graspers. Usually, two graspers are needed to get hold of the fibrous pericardium and avoid grasping just pericardial or mediastinal fat. If the

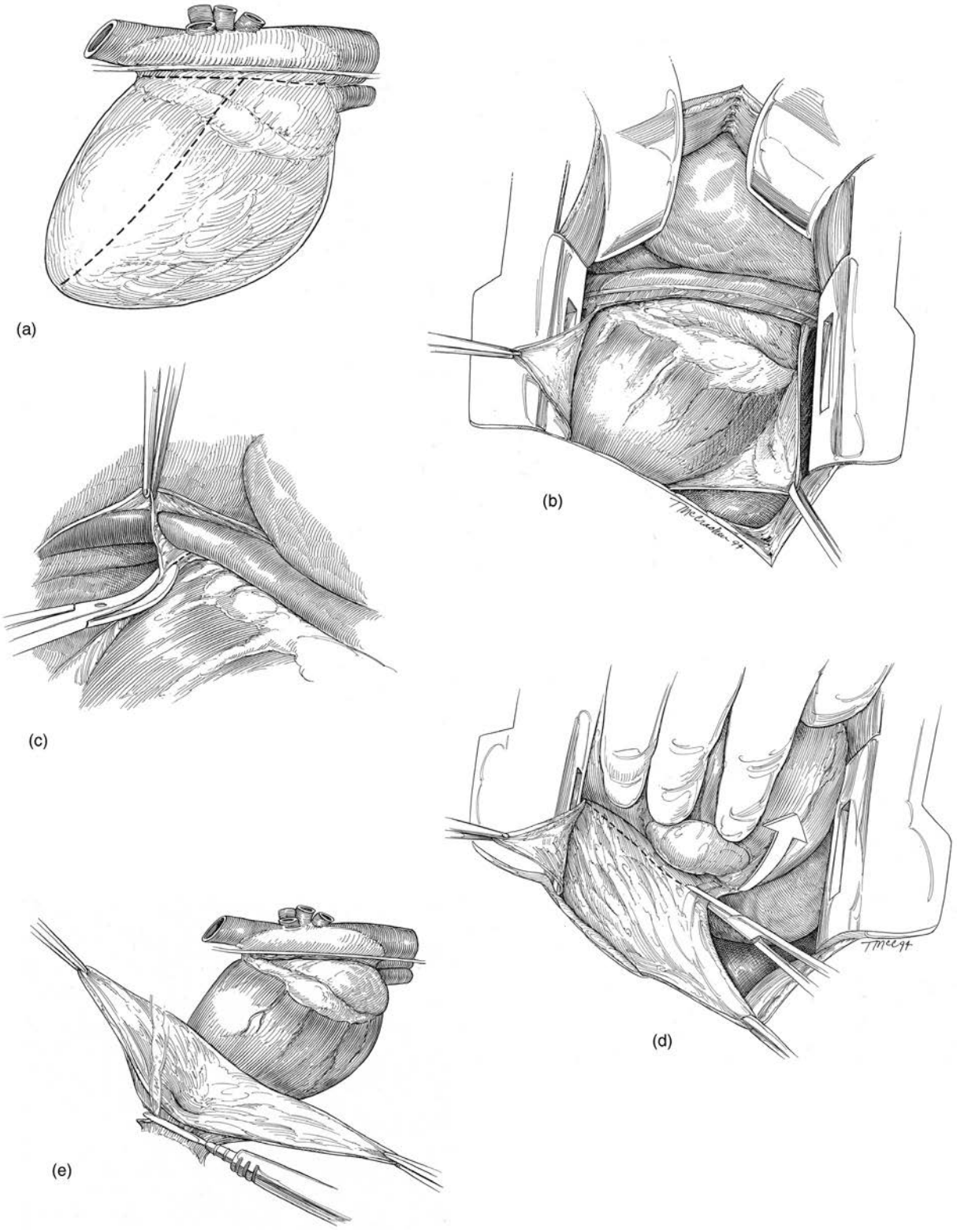

Figure 17.1 Subtotal Pericardiectomy—Right Thoracotomy

pericardium is distended with fluid and under pressure, drainage of the pericardial sac with a long spinal needle decompresses the pericardium and facilitates grasping the fibrous pericardium. It is best to grasp the pericardium toward the cardiac apex to avoid iatrogenic trauma to a possible right atrial mass. The initial incision in the pericardium is performed with scissors. After the pericardium is opened, the resection is continued with electrocautery or a vessel sealant device.

After the initial incision, pericardial fluid can be removed by a sump suction tip introduced through one of the cannulae. Subtotal pericardiectomy is then performed by extending the incision first toward the base of the heart and then on the left and right side of the pericardium, keeping the phrenic nerves in the field of view. This point in the procedure is where one-lung ventilation can be help to visualize phrenic nerves.

The pericardiectomy is completed by connecting the two subphrenic incisions across the cranial border of the pericardium. This is facilitated by changing the position of the electrocautery or vessel sealant device to the transdiaphragmatic ports. The endoscope is placed in one of the intercostal cannulae.

Pericardial Window

Pericardial window is a palliative procedure intended to provide long-term drainage of chronic pericardial effusion into the pleural space. The procedure should be avoided in the setting of acute hemorrhage associated with cardiac neoplasms, as this can result in unchecked bleeding into the pleural space. Pericardial window can be performed by video-assisted thoracoscopy, transdiaphragmatic celiotomy, or minimal-incision thoracotomy.

Subxyphoid Thoracoscopy

Ports for thoracoscopic pericardial window are placed in the transdiaphragmatic and left and right intercostal positions, as described for thoracoscopic pericardiectomy. The mediastinum is dissected. The pericardium is grasped with 5 mm fine teeth graspers (Figure 17.2a). Alternating two graspers is typically required to get hold of the fibrous pericardium. The pericardium is elevated and scissors are used to make the initial incision (Figure 17.2b). The pericardium is lifted away from the heart as the incision is enlarged (Figure 17.2c). The window is expanded cranially and laterally to make a 2 to 4 cm diameter window, depending on patient size (Figure 17.2d). Electrocautery or vessel sealant device is used to enlarge the window to decrease bleeding from the thickened pericardium (Figure 17.2e). The ideal window size is largely based on judgment. It should be large enough to maintain long-term drainage without closing but not large enough to allow herniation of the heart. Herniation of the heart through a window can result in a tethering obstruction of one or both vena cavae.

After completion of the window, the endoscope can be advanced into the pericardial space (pericardioscopy) to inspect the surface of the heart, right atrial appendage, and ascending aorta for evidence of pathology or neoplasia (Figure 17.2f) [26]. Mesothelioma can originate from the visceral pericardium (epicardium). Cardiac masses can be biopsied for histologic typing.

Transdiaphragmatic Celiotomy

Pericardial window can be performed through a midline celiotomy and transdiaphragmatic approach. This approach avoids the need for equipment necessary to perform video-assisted thoracoscopy and potentially diminishes post-operative pain associated with thoracotomy or sternotomy. A cranial midline celiotomy is performed. The liver is retracted caudally with malleable retractors. A midline incision is made in the diaphragm just dorsal to the sternum, and stay sutures are placed to retract the edges of the diaphragm. The pericardial sac is then exposed. An appropriate-sized pericardial window is made over the cardiac apex. This approach alone does not allow inspection of the mediastinum and pericardial space for evidence of underlying pathology and possible biopsy.

Minimal-Incision Thoracotomy

Pericardial window at the cardiac apex can be performed through a left-sided minimal incision thoracotomy described in Chapter 6. This approach provides more direct access to the cardiac apex compared to transdiaphragmatic celiotomy. It can be performed quickly with the patient in lateral recumbency, which is associated with less hemodynamic compromise compared to dorsal recumbency. A ventral minimal-incision thoracotomy is performed in the left eighth or ninth intercostal space at the cardiac apex based on palpation or thoracic imaging (see Figure 6.1). This approach provides immediate access to the apex of the pericardium (Figure 17.3). An appropriate sized window is made in the pericardium with scissors and/or electroscalpel. A thoracostomy tube is placed and the thoracotomy is closed.

Constrictive Pericarditis

Chronic inflammation of the pericardium results in extensive fibrous tissue proliferation and thickening of the pericardium. Severe lesions can constrict the heart, which compromises cardiac filling and

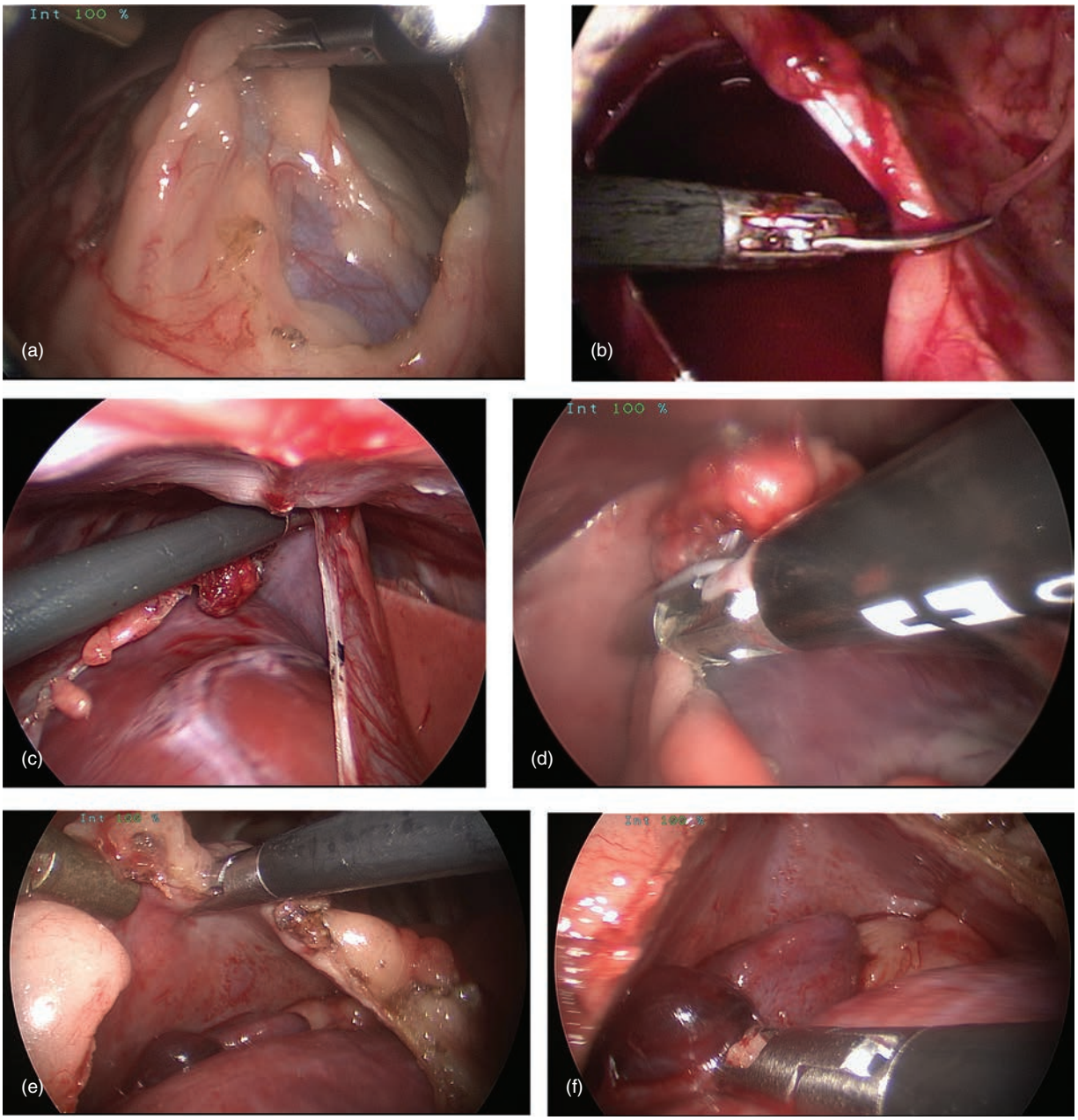

Figure 17.2 Thoracoscopic Pericardial Window

cardiac output. Constrictive pericarditis can be caused by any condition that results in chronic pericarditis. The most common causes include idiopathic pericardial effusion, chronic chylothorax, neoplasia, foreign material (e.g., bullets), and infection (coccidioidomycosis) [27–29]. In most affected animals, the parietal pericardium is more severely affected than visceral pericardium, reaching a thickness of up to 8 mm. In some cases, the visceral and parietal pericardia are both affected, with adhesions developing between them. Effusive-constrictive pericarditis occurs when pericardial fluid is present within the nondistensible pericardial sac. In affected animals, right atrial pressure remains increased after pericardiocentesis because of pericardial constriction by the thickened epicardium.

Pathophysiology

In animals with constrictive pericarditis, early ventricular filling is normal and proceeds rapidly until maximal pericardial distension is reached. In mid- to late-diastole, thickened noncompliant pericardium abruptly limits ventricular filling and produces

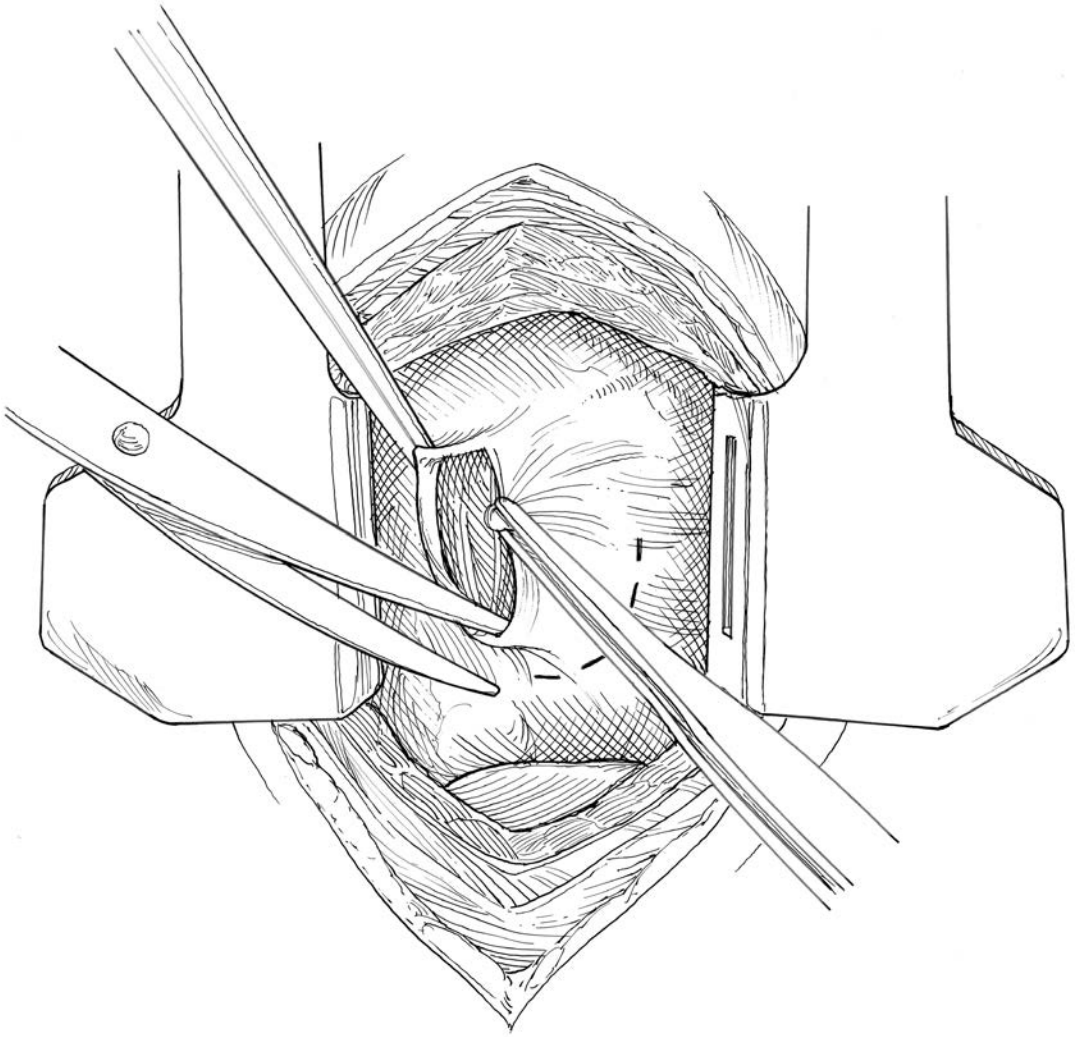

Figure 17.3 Pericardial Window via Minimal-Incision Thoracotomy

equilibration of diastolic pressures in of all four cardiac chambers. As the condition worsens, cardiac output declines. As with pericardial effusion, constrictive pericarditis can result in fluid retention and signs of right-sided congestive heart failure from activation of the renin-angiotensin-aldosterone system.

Central venous pressure does not decrease during inspiration in animals with constrictive pericarditis because negative intrathoracic pressure during inspiration is not transmitted to the cardiac chambers. Persistent increase in jugular venous pressure during inspiration is referred to as Kussmaul sign [1].

Diagnosis

Except in extreme cases, pericardial thickening is difficult to detect with echocardiography. Parietal pericardial thickness may be appreciated if pericardial fluid is present. Changes detected on echocardiography include left ventricular free wall flattening during mid- to late-diastole, early closure of the mitral valve, premature opening of the pulmonary valve, exaggerated interdependence of tricuspid and mitral inflows with respiration, and ventricular septal fluttering [30]. Variation in mitral inflow waves and rapid mitral inflow propagation velocity have been detected with M-mode echocardiography in dogs with effusive constrictive pericarditis.

Definitive diagnosis of constrictive pericarditis is based on measurement of cardiac pressures [1]. Pulmonary wedge pressure, right atrial pressure, and right and left ventricular diastolic pressures are increased and reach near equilibration. Venous pressure tracings may show a rapid, prominent Y descent during right atrial pressure measurement. Right and left ventricular pressure decreases quickly in early diastole because of rapid ventricular filling. The

subsequent increase in ventricular pressure is abruptly halted by pericardial restriction during mid- to late-diastole, resulting in a plateau that persists until the next ventricular systole. This *dip and plateau* or *square root sign* is considered diagnostic for pericardial constriction. Patients that are hypovolemic from diuretics or other causes may require volume loading with crystalloid solution to demonstrate classic hemodynamic changes. If a small amount of pericardial fluid is present, rapid Y descent may be absent.

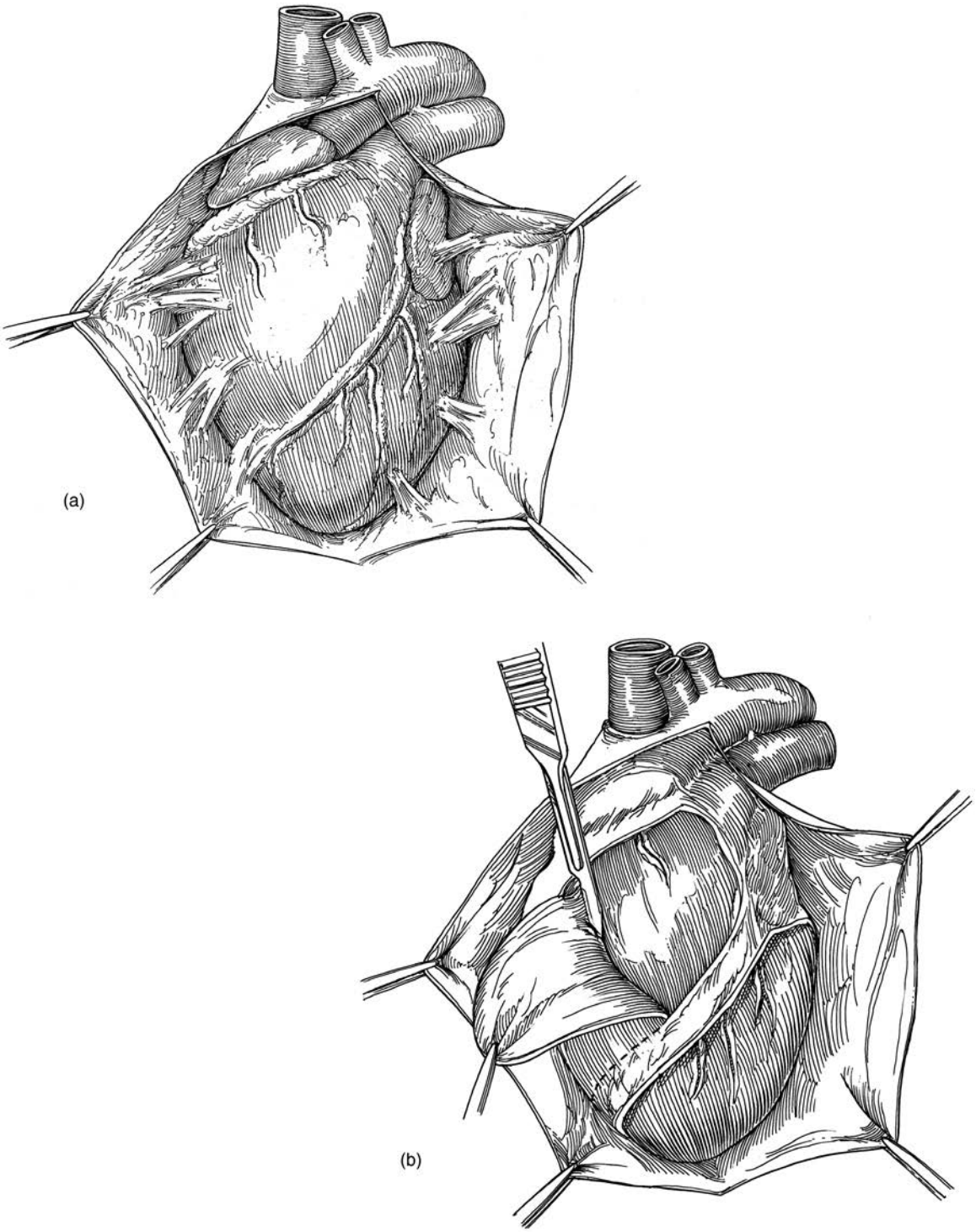

Figure 17.4 Constrictive Pericarditis—Pericardiectomy and Epicardial Decortication

Pericardiectomy and Epicardial Decortication

The only effective treatment for constrictive pericarditis is pericardiectomy with or without epicardial decortication depending on the degree of epicardial fibrous peel formation. Surgery should be performed through a sternotomy to provide best access to all cardiac surfaces. A pericardiectomy is performed, starting with a ventral midline incision of the fibrous pericardium (Figure 17.4a). Adhesions between the parietal pericardium and epicardial surface are broken down with a combination of sharp and gentile blunt dissection. The pericardiectomy is extended laterally to the level of the phrenic nerves. It may be necessary to separate the phrenic nerves from the surface of the pericardium to facilitate a total pericardiectomy in an effort to decompress the atria. If a fibrous peel has formed over the epicardial surface, then epicardial decortication is necessary to relieve constriction of the ventricles. This is accomplished by establishing a dissection plane between the fibrous peel and the epicardial surface of the heart (Figure 17.4b). The fibrous peel is separated through a combination of sharp and gentile blunt dissection. This procedure is only performed over the ventricles, avoiding the immediate area of the ventral interventricular coronary artery. Decortication of the atria should not be attempted.

References

1 LeWinter MM, and Kabbani S. In: Zipes DP, Libby P, Bonow RO, and Braunwald E eds. 2005. *Braunwald's heart disease: A textbook of cardiovascular disease. pp* 1757–80. Philadelphia: Elsevier Saunders.

2 Goto Y, and LeWinter MM. 1990. Nonuniform regional deformation of the pericardium during the cardiac cycle in dogs. *Circulation Research*. 67(5): 1107–14.

3 Ameli S, and Shah PK. 1991. Cardiac tamponade. Pathophysiology, diagnosis, and management. *Cardiology Clinics*. 9(4):665–74.

4 Hoit BD, Gabel M, and Fowler NO. 1990. Cardiac tamponade in left ventricular dysfunction. *Circulation*. 82(4):1370–6.

5 Hoit BD, and Ramrakhyani K. 1991. Pulmonary venous flow in cardiac tamponade: influence of left ventricular dysfunction and the relation to pulsus paradoxus. *Journal of American Society of Echocardiography*. 4(6):559–70.

6 Lombard CW. 1983. Pericardial disease. *Veterinary Clinics North America Small Animal Practice*. 13(2): 337–53.

7 Berg RJ, Wingfield WE, and Hoopes PJ. 1984. Idiopathic hemorrhagic pericardial effusion in eight dogs. *Journal of American Veterinary Medical Association*. 185(9):988–92.

8 de Madron E, Prymak C, and Hendricks J. 1987. Idiopathic hemorrhagic pericardial effusion with organized thrombi in a dog. *Journal of American Veterinary Medical Association*. 191(3): 324–6.

9 Bouvy BM, and Bjorling DE. 1991. Pericardial effusion in dogs and cats. Part II. Diagnostic approach and treatment. *Compendium on Continuing Education for the Practicing Veterinarian*. 13(4):633–42.

10 de Madron E. 1991. Malignant pericardial effusion in dogs: seven cases clinical, electrocardiographic, radiographic, and echocardiographic aspects. *European Journal of Companion Animal Practice*. 1(1):52–62.

11 Dunning D, Monnet E, Orton EC, and Salman MD. 1998. Analysis of prognostic indicators for dogs with pericardial effusion: 46 cases (1985-1996). *Journal of American Veterinary Medical Association*. 212(8): 1276–80.

12 Aronson LR, and Gregory CR. 1995. Infectious pericardial effusion in five dogs. *Veterinary Surgery*. 24(5):402–7.

13 Heinritz CK, Gilson SD, Soderstrom MJ, Robertson TA, Gorman SC, and Boston RC. 2005. Subtotal pericardectomy and epicardial excision for treatment of coccidioidomycosis-induced effusive-constrictive pericarditis in dogs: 17 cases (1999–2003). *Journal of American Veterinary Medical Association*. 227(3):435–40.

14 Aronsohn M. 1985. Cardiac hemangiosarcoma in the dog: A review of 38 cases. *Journal of the American Veterinary Medical Association*. 187(9): 922–6.

15 Dunning D, Monnet E, Orton EC, and Salman MD. 1998. Analysis of prognostic indicators for dogs with pericardial effusion: 46 cases (1985–1996). *Journal of the American Veterinary Medical Association*. 212(8):1276–80.

16 Zini E, Glaus TM, Bussadori C, Borgarelli M, Santilli RA, Tarducci A, Margiocco ML, Rampazzo A, Meli ML, Maisch B, and Pankuweit S. 2009. Evaluation of the presence of selected viral and bacterial nucleic acids in pericardial samples from dogs with or without idiopathic pericardial effusion. *Veterinary Journal*. 179(2):225–9.

17 Ehrhart N, Ehrhart EJ, Willis J, Sisson D, Constable P, Greenfield C, Manfra-Maretta S, and Hintermeister J. 2002. Analysis of factors affecting survival in dogs with aortic body tumors. *Veterinary Surgery*. 31(1):44–8.

18 Weisse C, Soares N, Beal MW, Steffey MA, Drobatz KJ, and Henry CJ. 2005. Survival times in dogs with right atrial hemangiosarcoma treated by means of surgical resection with or without adjuvant chemotherapy: 23 cases (1986–2000). *Journal of the American Veterinary Medical Association*. 226(4): 575–9.

19 Friedrichs KR, and Young KM. In: Withrow SJ, Vail DM, and Page RL eds. *Small Animal Clinical Oncology*. St Louis: Elsevier; 2013: 111–30.

20 Bonagura JD, and Pipers FS. 1981. Echocardiographic features of pericardial effusion in dogs. *Journal of the American Veterinary Medical Association*. 179(1):49–56.

21 Berry CR, Lombard CW, Hager DA, Ackerman N, and King RR. 1988. Echocardiographic evaluation of cardiac tamponade in dogs before and after pericardiocentesis: four cases (1984–1986). *Journal of the American Veterinary Medical Association*. 192(11):1597–603.

22 Sisson D, and Thomas WP. In: Fox PR, Sisson D, and Moise NS eds. 1988. *Textbook of canine and feline cardiology: Principles and clinical practice*. pp 679–701. Philadelphia: W.B Saunders.

23 Dupre GP, Corlouer JP, and Bouvy B. 2001. Thoracoscopic pericardectomy performed without pulmonary exclusion in 9 dogs. *Veterinary Surgery*. 30(1):21–7.

24 Mayhew KN, Mayhew PD, Sorrell-Raschi L, and Brown DC. 2009. Thoracoscopic Subphrenic Pericardectomy Using Double-Lumen Endobronchial Intubation for Alternating One-Lung Ventilation. *Veterinary Surgery*. 38(8):961–6.

25 Kudnig ST, Monnet E, Riquelme M, Gaynor JS, Corliss D, and Salman MD. 2003. Effect of one-lung ventilation on oxygen delivery in anesthetized dogs with an open thoracic cavity. *American Journal of Veterinary Research*. 64(4):443–8.

26 Skinner OT, Case JB, Ellison GW, and Monnet E. 2014. Pericardioscopic imaging findings in cadaveric dogs: comparison of an apical pericardial window and sub-phrenic pericardectomy. *Veterinary Surgery*. 43(1):45–51.

27 Schwartz A, Wilson GP, Hamlin RL, Swenberg J, Termin P, Thomford NR, and Donovan E. 1971. Constrictive pericarditis in two dogs. *Journal of the American Veterinary Medical Association*. 159(6): 763–76.

28 Shubitz LF, Matz ME, Noon TH, Reggiardo CC, and Bradley GA. 2001. Constrictive pericarditis secondary to Coccidioides immitis infection in a dog. *Journal of the American Veterinary Medical Association*. 218(4):537–40, 26.

29 Monnet E. 2012. In: Monnet E ed. *Small Animal Soft Tissue Surgery*. pp 820–2. Ames: Wiley-Blackwell.

30 Feigenbaum H. 1994. In: Feigenbaum H ed. *Echocardiography*. pp 181–215. Philadelphia: Lea & Febiger.

18

Strategies for Cardiac Surgery

E. Christopher Orton

Because delivery of oxygenated blood to tissues and organs cannot be interrupted more than momentarily, cardiac surgery must employ one or more strategies to allow cardiac corrections to be performed while maintaining, or only briefly interrupting, perfusion of tissues with oxygenated blood. Which strategy is chosen depends on the cardiac correction that needs to be performed. Cardiac surgery strategies include surgeries performed on the beating heart, surgeries performed with brief circulatory arrest during venous inflow occlusion, surgeries performed with the aid of cardiopulmonary bypass, and hybrid cardiac surgeries that combine image-guided interventional techniques with minimal-incision cardiac approaches. Each of these methods has advantages and disadvantages. Each has its own challenges and requirements for equipment and expertise. For some cardiac surgeries, more than one strategy might be feasible. In this case, understanding the pros and cons of each is an important consideration in planning surgery.

Beating Heart Surgery

Many cardiac surgeries can be performed on a beating heart without circulatory or cardiac arrest. These include surgery on structures outside of the heart such as PDA ligation or pulmonary artery banding, surgery performed by passing instruments through small portals controlled by suture tourniquets such as closed valve dilation, and surgery performed with the aid of tangential or side-biting vascular clamps such as systemic-to-pulmonary shunts. A central principle of all cardiac surgery is to plan the surgery so that control of blood loss is always maintained. This is particularly true during beating heart surgery where there are few fallback options once significant bleeding occurs. It is important to have instrumentation (e.g., vascular clamps) available to control hemorrhage in the event that a cardiac structure is inadvertently opened. Vascular clamps should be deeply placed to prevent slippage and to ensure adequate margins for closure of cardiac incisions. Sutures should generally be reinforced with pledgets to prevent them from cutting through cardiac tissues. Advanced planning and a deliberate approach are important in preventing crisis situations during cardiac surgery.

Motion is an intrinsic aspect of beating heart surgery. Cardiac motion adds to both the psychological and technical challenges of cardiac surgery. Gentle compression on the surface of the heart is useful in controlling cardiac motion during suture placement. Placement of fingers within the ring handles of a needle holder, as opposed to *palming* the instrument, generally provides greater instrument control and facilitates suturing on the beating heart. As a general rule, pharmacologic manipulation of the heart rate during beating heart surgery is not necessary, and should be avoided given its inherent risks. An exception is when atrial fibrillation is present. As surgeons become familiar with operating on the beating heart, they soon realize that it is not fundamentally different from surgery on other tissues or organs.

Venous Inflow Occlusion

Venous inflow occlusion is a strategy that provides a brief period of circulatory arrest, allowing for open cardiac repairs that can be performed in a short period of time, generally less than 2 minutes. It is accomplished by placing tourniquets on the vena cavae and azygous vein to stop venous blood return to the heart and provide a brief period of interrupted blood flow within the heart without inducing cardiac arrest. It is indicated for cardiac surgeries that can be reasonably performed in a short period of time. Its principal advantages are its relative simplicity, lack of need for specialized equipment, and minimal cardiopulmonary, metabolic, and hematological derangements

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after surgery [1]. The principal disadvantages of inflow occlusion are the short time available to perform cardiac surgery, motion of the surgical field, and the limited options should delays occur in the completion of surgery. As a result, cardiac surgery performed during venous inflow occlusion must be meticulously planned and executed.

Ideally, the duration of circulatory arrest in a normothermic patient should be 2 minutes or less to minimize the risk of cerebral injury and ventricular fibrillation. If necessary, inflow occlusion can be repeated to allow completion of a cardiac surgery. If repeated, adequate time for recovery of the myocardium must be allowed between inflow occlusions. Circulatory arrest time can be prolonged up to 4 minutes with mild whole body hypothermia (32° to 34°C). However, the risk of cardiac arrest increases as the circulatory arrest time increases, regardless of whether deliberate hypothermia is employed. The temperature should not be allowed to fall below 32°C because the risk of ventricular fibrillation increases significantly below this temperature. After surgery, surface warming with water blankets or beds is used to return the animal to normal temperature.

Venous inflow occlusion can be accomplished from a left or right thoracotomy or median sternotomy depending on the cardiac procedure being performed. Tourniquets are placed on the vena cavae and azygous vein to accomplish inflow occlusion. Direct access to the vena cavae and azygous vein for inflow occlusion is readily achieved from a right thoracotomy (Figure 18.1a). The right phrenic nerve is excluded during placement of tourniquets on the vena cavae to avoid injury to the nerve. Access to the vena cavae and azygous vein for tourniquet placement is also readily achieved from a sternotomy approach by gently retracting the heart to the left before opening the pericardium. Placement of tourniquets for inflow occlusion is more difficult from a left thoracotomy, particularly when the heart is enlarged. The vena cavae and azygous vein are accessed by dissecting through the mediastinum (Figure 18.1b). Dissection to the cranial vena cava is just ventral to the brachiocephalic artery. Access to the azygous vein is gained by dissection dorsal to the aorta and esophagus. A possible alternative approach for venous inflow occlusion from a left thoracotomy is to pass balloon catheters into the cranial and caudal vena cave from a left jugular access and then inflating the balloons to accomplish venous inflow occlusion.

Venous inflow occlusion follows a carefully scripted sequence of steps (Box 18.1). The plan should be communicated to the surgical and anesthesia teams. Drugs and equipment for full cardiac resuscitation should be immediately available after venous inflow occlusion. Ventilation should be discontinued during inflow occlusion to prevent pulmonary blood from being pushed into the surgical field during positive-pressure ventilation. De-airing the heart is a critical step at termination of venous inflow occlusion to avoid a fatal air embolus. This is accomplished by simultaneous release of one tourniquet and a large positive pressure breath (i.e., Valsalva maneuver) just prior to closure of the cardiac incision. Gentle cardiac massage may be necessary after inflow occlusion to help reestablish cardiac function. Digital occlusion of the descending aorta during this period helps direct the available cardiac output to the heart and brain. If ventricular fibrillation occurs, the heart should be defibrillated by direct electrical shock immediately after inflow occlusion is discontinued. Cardiac incisions are generally initially closed with vascular clamps or pre-placed sutures to minimize the length of circulatory arrest times. Placement of stay sutures adjacent to the planned incision site helps control the incision during inflow occlusion and facilitates placement of the vascular clamp. Cardiac incisions are sutured after cardiac function is restored.

Cardiopulmonary Bypass

Cardiopulmonary bypass (CPB) is a strategy where the patient is connected to an extracorporeal system that provides flow of oxygenated blood to the patient allowing the heart to be stopped for surgical repair. CPB provides for a motionless and bloodless operative field, and permits time to perform more complex cardiac repairs. CPB has been employed to treat a variety of cardiac conditions in dogs. The various approaches to CPB in dogs have been reviewed [2]. Successful cardiac repairs have also been reported in cats [3, 4]. Standard CPB provides continuous flow of oxygenated blood to all organs except the heart, which is placed in cardioplegic arrest [5, 6]. Alternate strategies can be employed, including CPB on the beating heart without cardioplegic cardiac arrest [4], low-flow deep hypothermic CPB [7, 8], and deep hypothermic CPB with circulatory arrest. Successful CPB requires a team approach. The primary CPB team consists of the surgical, perfusion, and anesthesia teams. Initiation, maintenance, and discontinuation of CPB require a coordinated and practiced effort between these teams. There must be clear communication between the leaders of each team. The primary surgeon is ultimately responsible for the overall conduct of CPB.

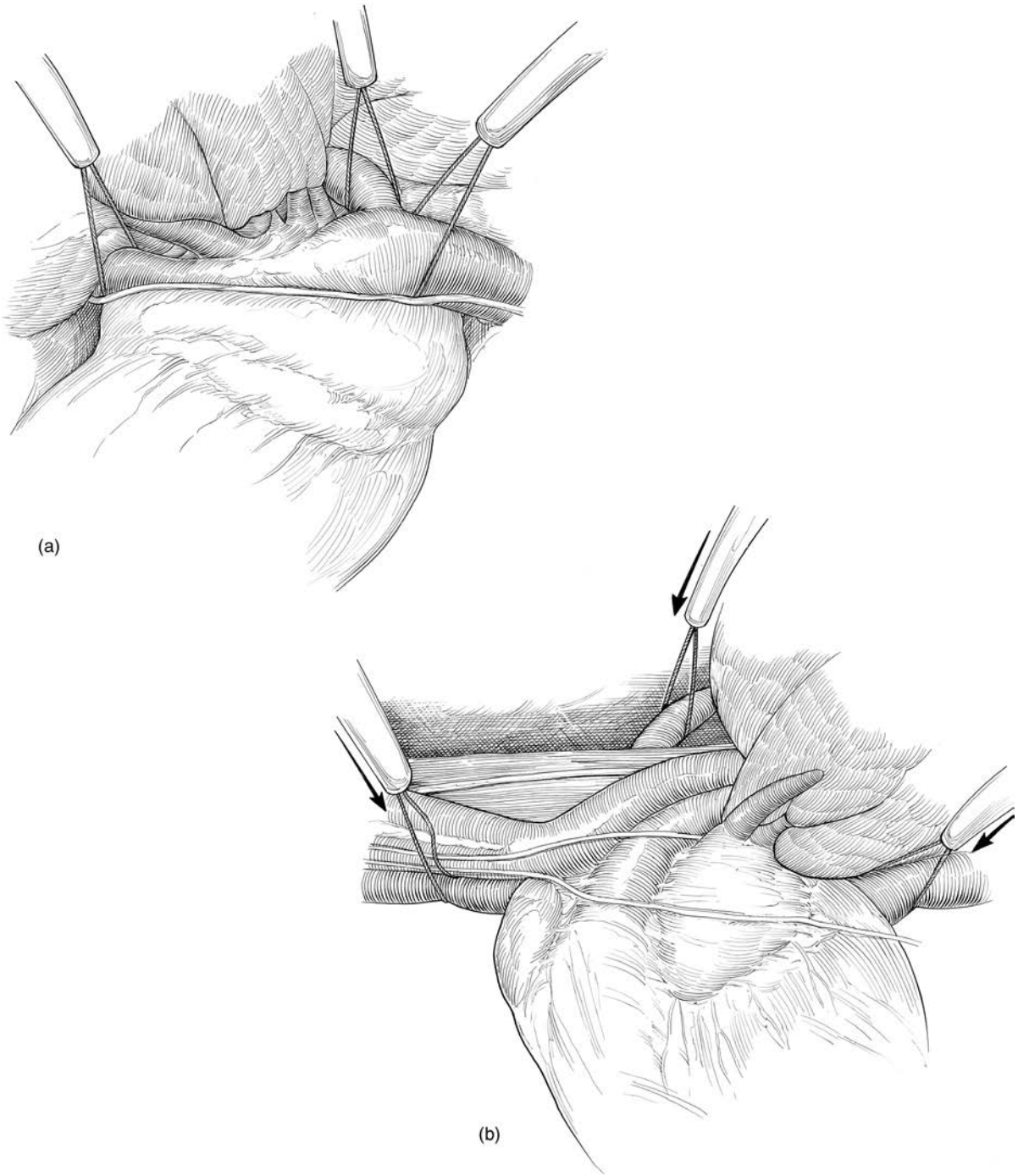


Figure 18.1 Venous Inflow Occlusion

Circuit and Equipment

CPB is accomplished with a heart-lung machine that consists of standard equipment and a disposable bypass circuit. The equipment components of a heart-lung machine include the pumps (four roller pumps and a centrifugal pump), a circulating heater/cooler water bath, an air-oxygen mixer, gas flow meters, and an anesthetic vaporizer (Figure 18.2). The primary bypass circuit consists of a venous line, venous reservoir, centrifugal pump, membrane oxygenator, heat exchanger coils, and arterial line (Figure 18.3). A

Box 18.1 Sequence of Steps for Venous Inflow Occlusion

- Carefully plan and script the surgery.
- Communicate plan to surgery and anesthesia teams.
- Keep core temperature above 32°C.
- Correct acidosis and hypoventilation before circulatory arrest.
- Preplace stay sutures adjacent to planned incision to control incision during inflow occlusion.
- Be prepared for full cardiac resuscitation.
- Initiate inflow occlusion by tightening tourniquets.
- Turn off ventilator during inflow occlusion.
- Allow heart to empty before making cardiotomy.
- Perform cardiotomy.
- Perform cardiac repair within 2 minutes (maximum 4 minutes).
- De-air heart as cardiotomy is closed by releasing a tourniquet and performing a Valsalva.
- Temporarily close cardiotomy with vascular clamp or pre-placed sutures.
- Resuscitate the heart as needed.
- Suture the cardiotomy.
- Check cardiotomy for bleeding.

membrane-type oxygenator should be used to minimize injury to the blood. A pediatric size oxygenator is preferred in dogs < 40 kg to minimize the priming volume of the circuit. During CPB, blood is drawn away from the patient to the reservoir by gravity flow. Blood is then pumped through the oxygenator under pressure and returned to the patient by means of a roller or centrifugal pump. A centrifugal-type pump is currently preferred for the primary perfusion circuit because it is less injurious to the blood. The heater/cooler water bath is used to control body temperature by means of a heat exchanger built into the primary circuit. Blood in the operative field is collected and returned to the reservoir by one or two suction lines driven by secondary roller pumps. An additional vent line is used to keep the heart decompressed and help remove air during weaning from CPB.

Monitoring

Prior to surgery, a multi-lumen venous catheter is placed into a jugular vein to provide central venous access and monitoring of central venous pressure (CVP). An arterial catheter is placed to monitor direct arterial pressure and arterial blood-gas status. The electrocardiogram, arterial blood pressure, CVP, and esophageal and rectal temperatures are monitored continuously during surgery. Arterial and venous blood gases, activated clotting time (ACT), electrolyte concentrations (Na^+, K^+ and ionized Ca^{++}), hematocrit, total protein, and lactate are monitored intermittently during CPB.

Cannulation

The surgical team is responsible for connecting the patient to the CPB circuit. Cannulation for CPB follows a sequential order consisting of an arterial cannula, venous cannula(e), and cardioplegia cannula. The cardioplegia cannula serves to deliver cardioplegia solution to arrest the heart and vent the aortic root during weaning from CPB. An additional vent cannula is sometimes placed in the left ventricle during weaning from bypass if the left heart has been opened. Several options for arterial and venous cannulation are available depending on the thoracic approach and cardiac surgery being performed. Prior to cannulation and initiation of CPB, the animal must undergo complete anticoagulation by administration of IV heparin (Box 18.2). The ACT is monitored every 20 minutes during bypass and maintained above 480 seconds. Oxygenated blood is returned to the patient via the arterial cannula and line. Arterial cannulation is performed prior to other cardiac manipulations. If bleeding occurs before initiation of CPB, blood can be salvaged from the operative field and returned to the patient via the arterial cannula and line. Options for arterial cannulation are the femoral artery, carotid artery, or aorta. Cannulation of a femoral or carotid artery is less technically demanding and is performed before the thoracic cavity is opened. If a thoracotomy approach is chosen, the contralateral femoral artery or the ipsilateral carotid artery is cannulated. Cannulation of a peripheral artery is accomplished by placing proximal and distal controlling tourniquets around the artery (Figure 18.4a). A transverse incision is made in the artery with a #11 blade (Figure 18.4b). A straight arterial cannula is inserted into the artery and secured by tightening the tourniquets and tying the cannula to the tourniquets (Figure 18.4c). Cannula sizes range from 8 to 14 F depending on the size of the animal. Alternatively, an arterial cannula can be placed in the ascending or descending aorta after the thorax is opened. The advantages of aortic cannulation are avoidance of separate incision for arterial cannulation and ability to place a larger and shorter cannula that is less injurious to blood. The disadvantage is that is it technically more difficult and less forgiving of

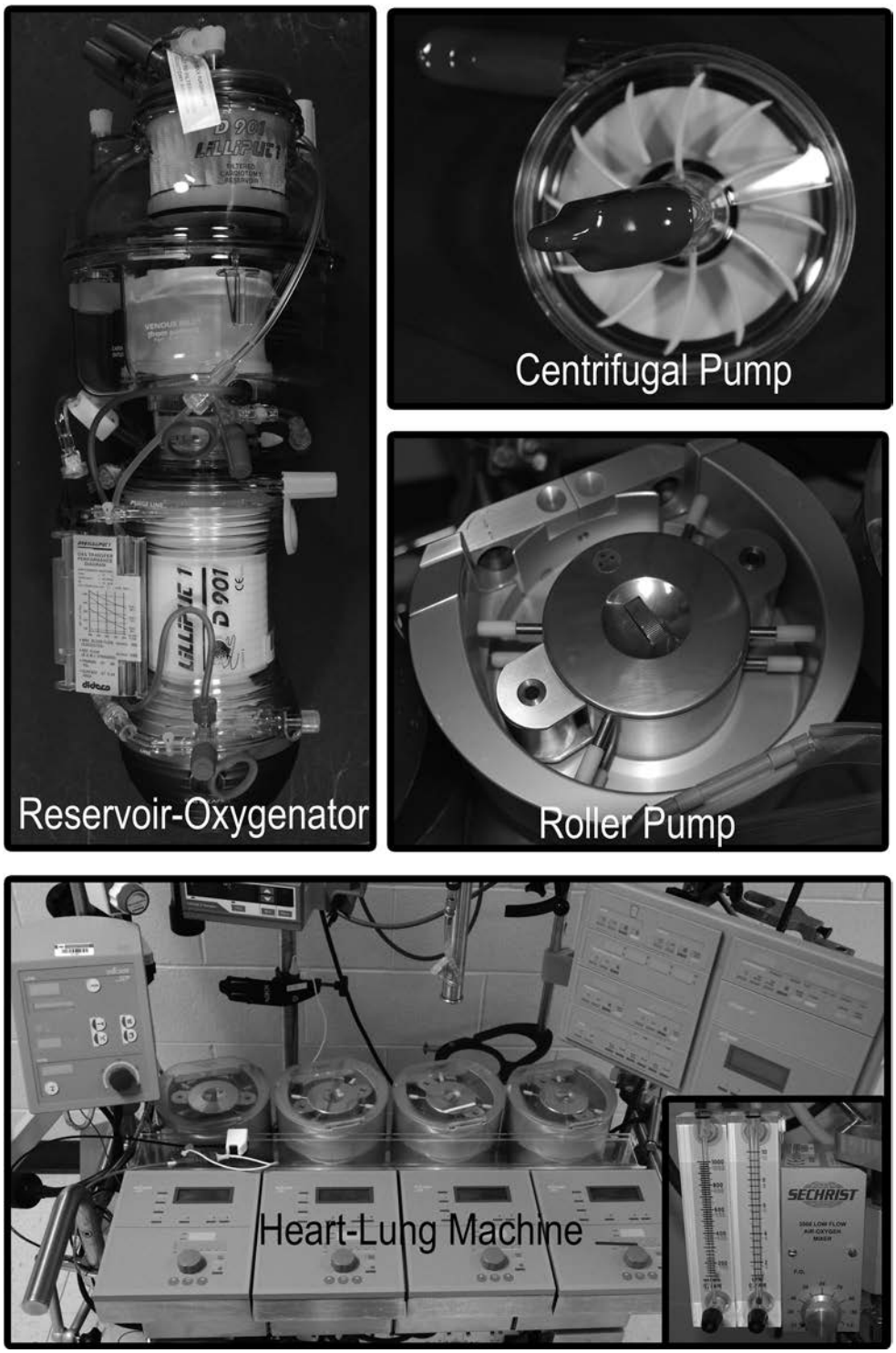


Figure 18.2 Heart-Lung Machine

error. Direct aortic cannulation is accomplished from a left thoracotomy before opening the pericardium (Figure 18.5a). The descending aorta is freed from surrounding loose connective tissues and a tape is passed around it. A pledget reinforced pursestring suture is placed in the wall of the aorta and passed through a tourniquet. A side-biting vascular clamp is placed on the aorta to control bleeding during cannulation and a

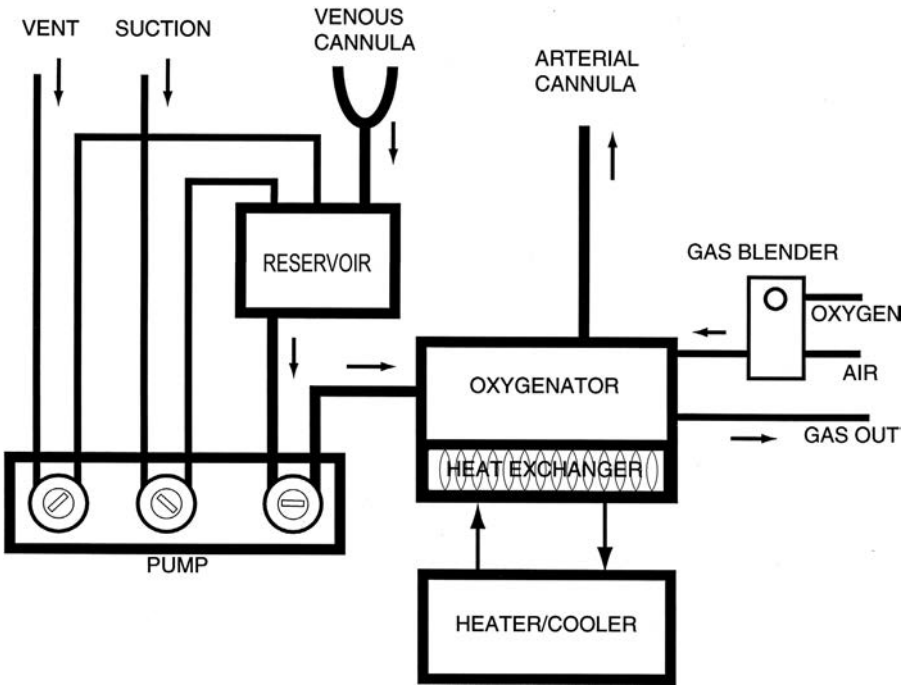


Figure 18.3 Cardiopulmonary Bypass Circuit

longitudinal aortotomy is made. An angled aortic cannula is inserted and secured by tying it to the tourniquet (Figure 18.5b). The cannula is connected to the arterial line. A $^1/_4$-in. arterial line is sufficient for most small animals.

Blood is diverted from the right heart to the CPB circuit by means of one or two venous cannula(e) and a venous line. Venous cannulation is accomplished using different configurations, depending on the cardiac surgery and thoracic approach. Bicaval venous cannulation is required for cardiac surgeries that use a right atrial cardiotomy approach, such as septal defect repairs and tricuspid valve surgery. Bicaval cannulation can be achieved from a right thoracotomy or sternotomy, but not a left thoracotomy. Tape or heavy suture tourniquets are placed around a vena cavae and azygous as for venous inflow occlusion excluding the phrenic nerve (Figure 18.1a). The pericardium is opened to expose the vena cavae and right atrium (Figure 18.6a). Pursestring

Box 18.2 Drugs for Cardiopulmonary Bypass in Dogs

Before Bypass

Heparin sulfate: 300–400 U/kg IV (ACT > 480 seconds)
ε′-aminocaproic acid: 50–100 mg/kg over 30 minutes

During Bypass

ε′-aminocaproic acid: 10–15 mg/kg/hour CRI
Phenylephrine: 0.05–0.1 mg/kg IV (mean BP > 50 mm Hg)
$NaHCO_3$: 0.5–1 mEq/kg IV (correct acidosis, alpha-stat pH)

After Bypass

Dobutamine: 1–10 mcg/kg/min CRI
Epinephrine: 0.05–0.5 mcg/kg/min CRI
Lidocaine: 50–70 mcg/kg/min CRI
Protamine sulfate: 0.5–1.0 mg/100 U heparin slow CRI
Fresh whole blood: 1 unit/25 kg

Cardioplegia

Solution: pH-adjusted (7.4) crystalloid solution
Add: KCl 40 mEq/500 mL (first dose)
KCl 20 mEq/500 mL (subsequent doses)
$NaHCO_3$ 25 mEq/500 mL
Heparinized blood (4:1 blood: crystalloid)
Temperature: 4°C
Volume: 25–50 ml/kg intracoronary @ 20 minutes
Delivery pressure: 100–120 mm Hg

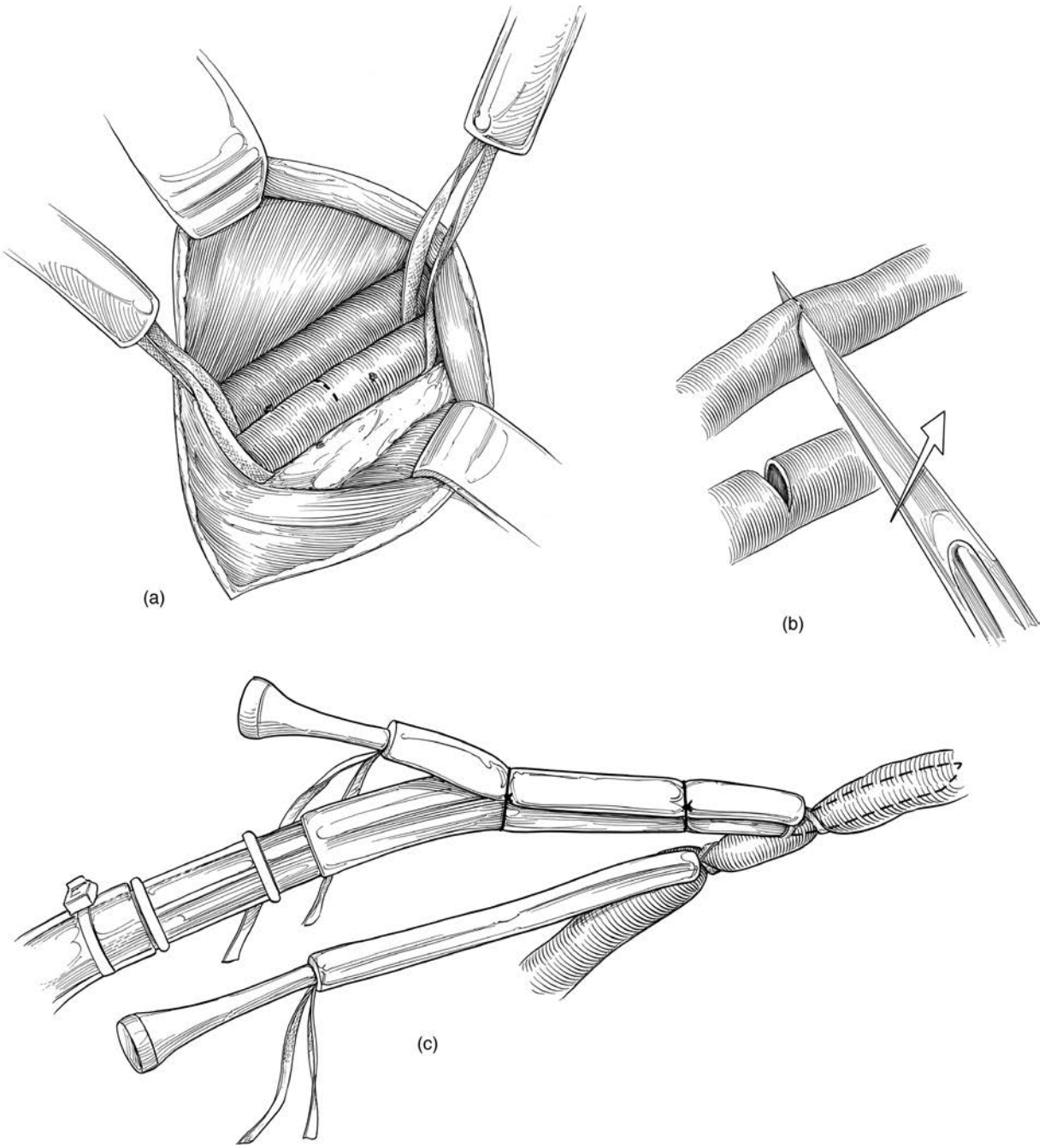
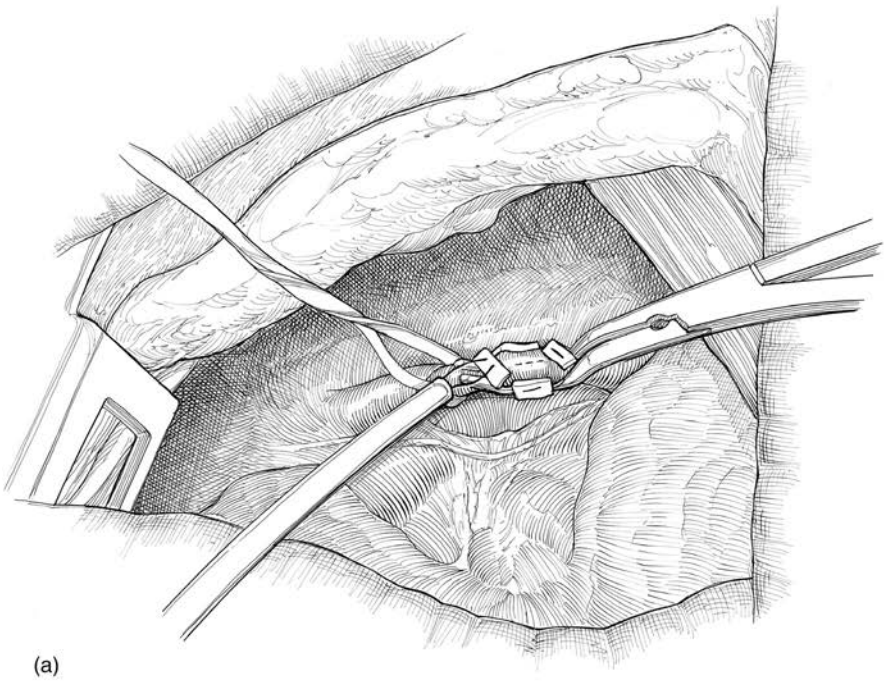
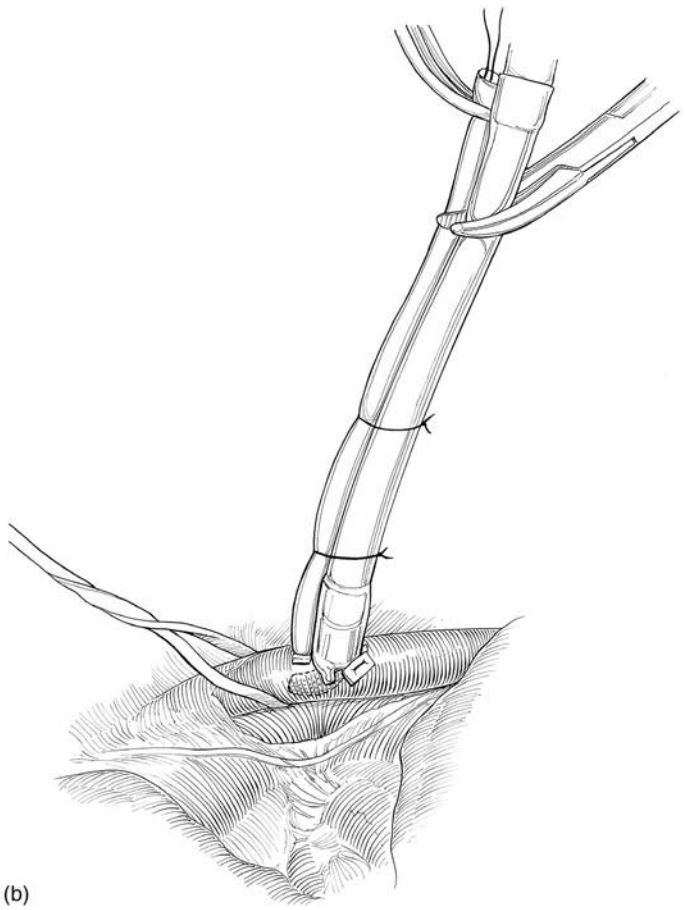


Figure 18.4 Femoral Artery Cannulation

sutures are placed in at junctions of the right atrium with the cranial and caudal vena cavae. The cranial pursestring should avoid the region of the terminal sulcus, which contains the sinus node. Incisions are made within the pursestring sutures with a #11 blade (Figure 18.6b) and then dilated with a hemostat (Figure 18.6c). Bleeding is controlled during the cannulation by grasping the cannulation site with forceps and bringing the edges together between steps. An angled venous cannulae are inserted starting with the caudal vena cava (Figure 18.5d) and secured by tying to the tourniquets (Figure 18.5e). The caval venous cannulae are connected to the venous line by a Y-connector. The caval tourniquets are tightened



(a)



(b)

Figure 18.5 Aortic Cannulation

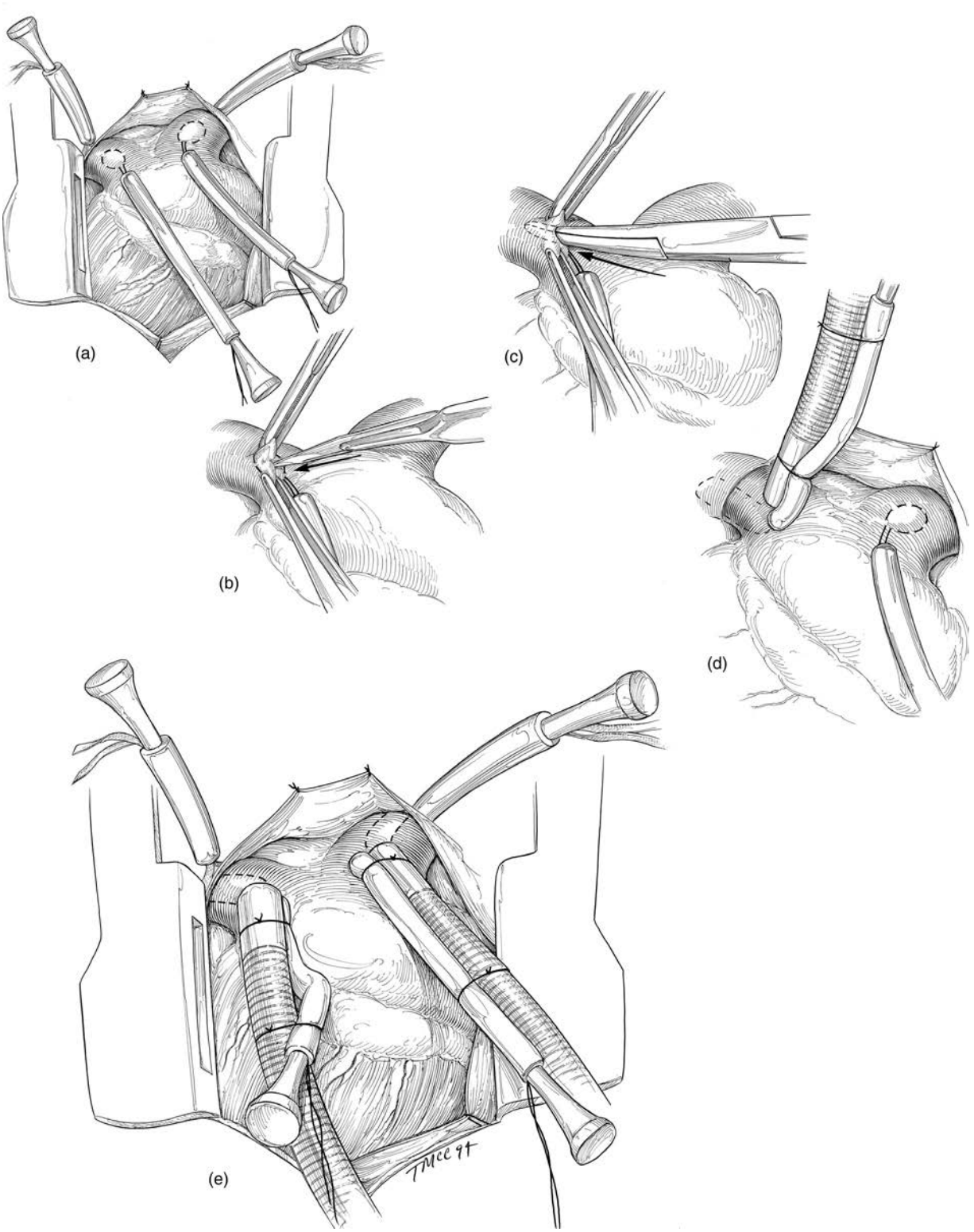


Figure 18.6 Bicaval Venous Cannulation

around the cannulae to exclude blood flow to the right atrium. The venous line can be 3/8 in. or 1/4 in. tubing depending on patient size.

Atrial venous cannulation is used for cardiac repairs of the left heart or when the right atrium does not need to be excluded from blood flow. Atrial cannulation can be accomplished from any thoracic approach and is the only option from a left thoracotomy. A one-stage or two-stage venous cannula is introduced through the right atrial appendage. A two-stage cannula provides more efficient venous drainage by cannulation of both the right atrium and caudal vena cava. From a right thoracotomy, the pericardium is opened, a side-biting vascular clamp is placed on the right auricle, and a pursestring suture is placed in the end of the right auricle (Figure 18.7a). The tip of the auricle is excised and the venous cannula is inserted as the vascular clamp is removed (Figure 18.7b). The tourniquet is tightened and the cannula is secured by tying it to the tourniquet (Figure 18.7c). Atrial venous cannulation from a left thoracotomy follows the same sequential steps as from a right thoracotomy consisting of placement of a pursestring suture (Figure 18.8a), insertion of the venous cannula (Figure 18.8b), and securing the cannula to the tourniquet (Figure 18.8c). The more limited access to the right auricle from the left side makes cannulation more technically difficult from a left thoracotomy.

Final cannulation before CPB consists of placement of a cannula in the ascending aorta for administration of cardioplegia solution and venting during weaning from CPB (Figure 18.9). Cannulation for cardioplegia is accomplished by opening the pericardium and freeing the ascending aorta from loose connective tissues and main pulmonary artery. Dissection of the aorta is accomplished with an electroscalpel to minimize bleeding. A tape is passed round the ascending aorta. A pledget-reinforced mattress suture is placed partial thickness into the wall of the aorta and passed through tourniquets. A second unpledgeted pursestring suture is placed outside the mattress sutures to help control bleeding. A cardioplegia cannula is inserted into the aorta and secured by tying it to the tourniquet. Complete CPB is achieved by placing a crossclamp on the ascending aorta and administration of cardioplegia solution to arrest mechanical systole and cool the myocardium. This cannula also serves to vent the left heart during weaning from CPB.

Sometimes it is beneficial to place an additional vent cannula in the left ventricle to keep it decompressed and aid with de-airing the heart during weaning from CPB. A vent cannula can be placed directly into the left ventricle through a pledgeted mattress suture in the cardiac apex. From a right thoracotomy, a left ventricular vent cannula can be placed directly into the left atrium by placing a mattress suture dorsal to the right atrium (Figure 18.10a). The vent cannula is introduced into the left atrium and across the mitral valve into the left ventricle (Figure 18.10b).

Perfusion

Mild hemodilution is desirable during CPB to decrease systemic vascular resistance and counter the effects of increased blood viscosity during hypothermia. Hemodilution results when crystalloid prime in the bypass circuit mixes with the patient blood. The goal is to decrease the hematocrit to approximately 28%. The hematocrit should not fall below 25% to avoid bleeding complications and systemic inflammatory response after surgery associated with excessive hemodilation. A balanced pH-adjusted (7.4) crystalloid solution is used to prime the circuit. Whole blood can be added to the prime to avoid excessive hemodilution.

Perfusion flow rates during bypass depend on several factors including patient size, temperature, and hematocrit. Perfusion flow should be adjusted to meet the requirements of the patient based on venous oxygen saturation (> 70%) and blood lactate from the blood gas analysis. A general guideline for perfusion flow rates in dogs is 80 to 100 mL/kg/min when dogs are normothermic. Perfusion rate can be decreased during hypothermic CPB. Mean arterial pressure should be 50 to 70 mm of Hg during bypass. Arterial pressure usually falls dramatically for several minutes after initiation of bypass due to the effect of sudden hemodilution on vascular resistance. Phenylephrine can be administered as necessary to increase vascular resistance and maintain adequate perfusion pressure. Standard CPB perfusion strategies with mild to moderate hypothermia can be used for dogs weighing >10 kg. Deep hypothermic (15° to 18°C) low flow (20 mL/kg/min) CPB may be a more appropriate strategy for small dogs (< 10 kg) to decrease adverse effects associated with bypass [7].

Esophageal and rectal temperatures are monitored continuously during CPB. It is generally advantageous to cool to between 25° and 28°C during CPB to decrease the metabolic requirements of the patient. This is accomplished with the heater/cooler water bath and heat exchanger in the pump circuit. The requirement for inhalation and other anesthetic drugs decreases during hypothermia.

During CPB, the oxygenator receives continuous flow of gas mixture of oxygen and nitrogen from an oxygen blender. The ratio of oxygen to nitrogen

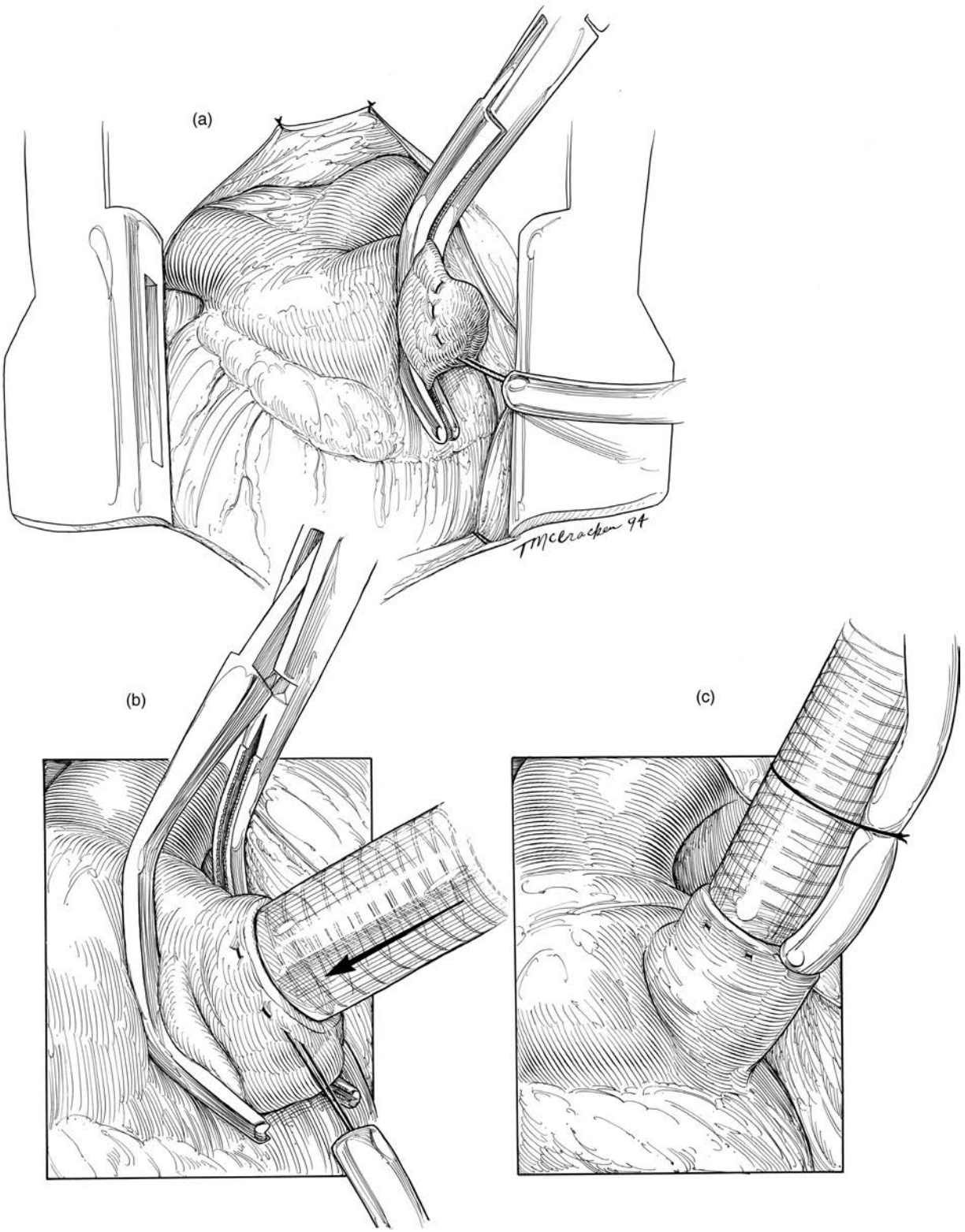


Figure 18.7 Right Atrial Cannulation—Right Thoracotomy

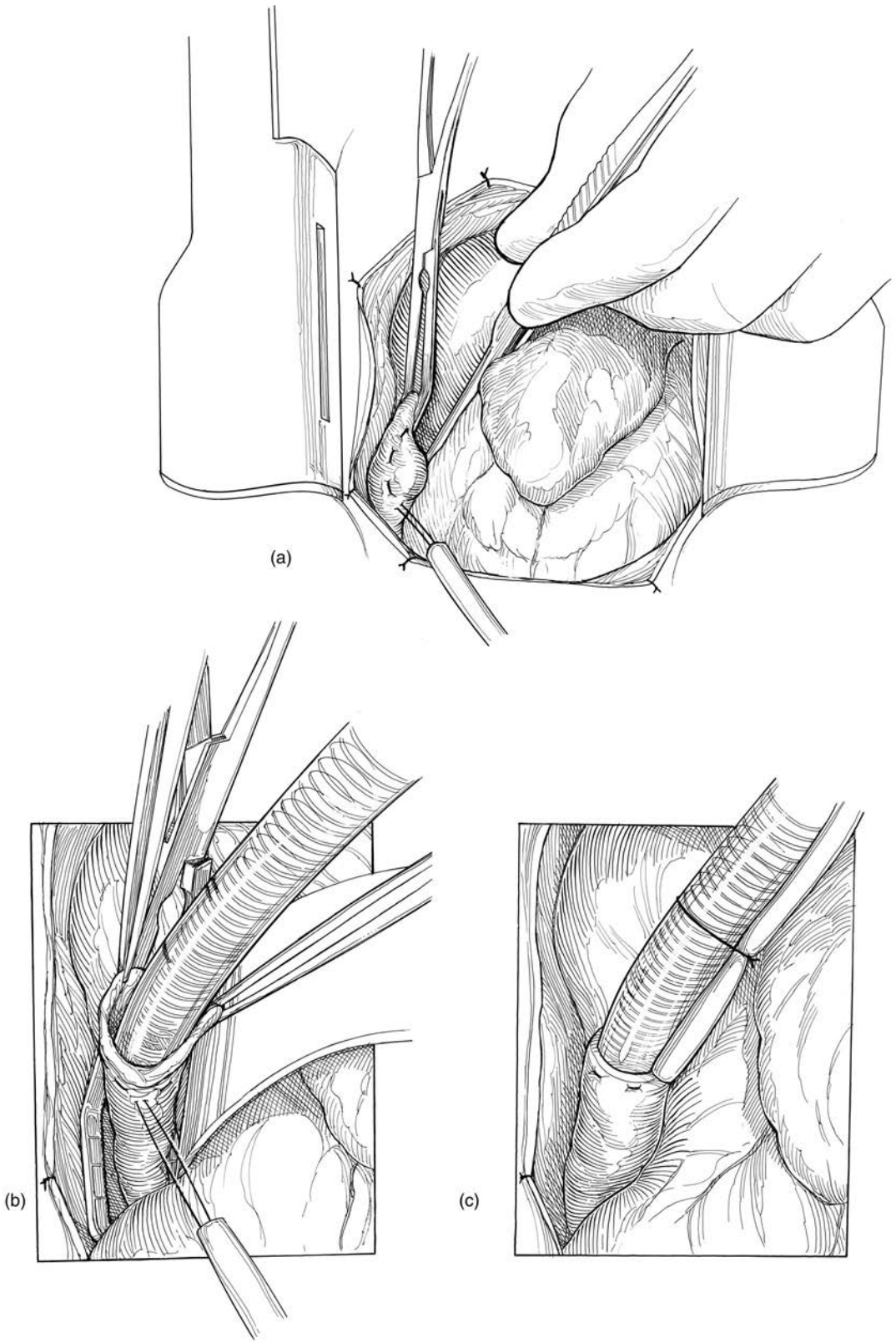


Figure 18.8 Right Atrial Cannulation—Left Thoracotomy

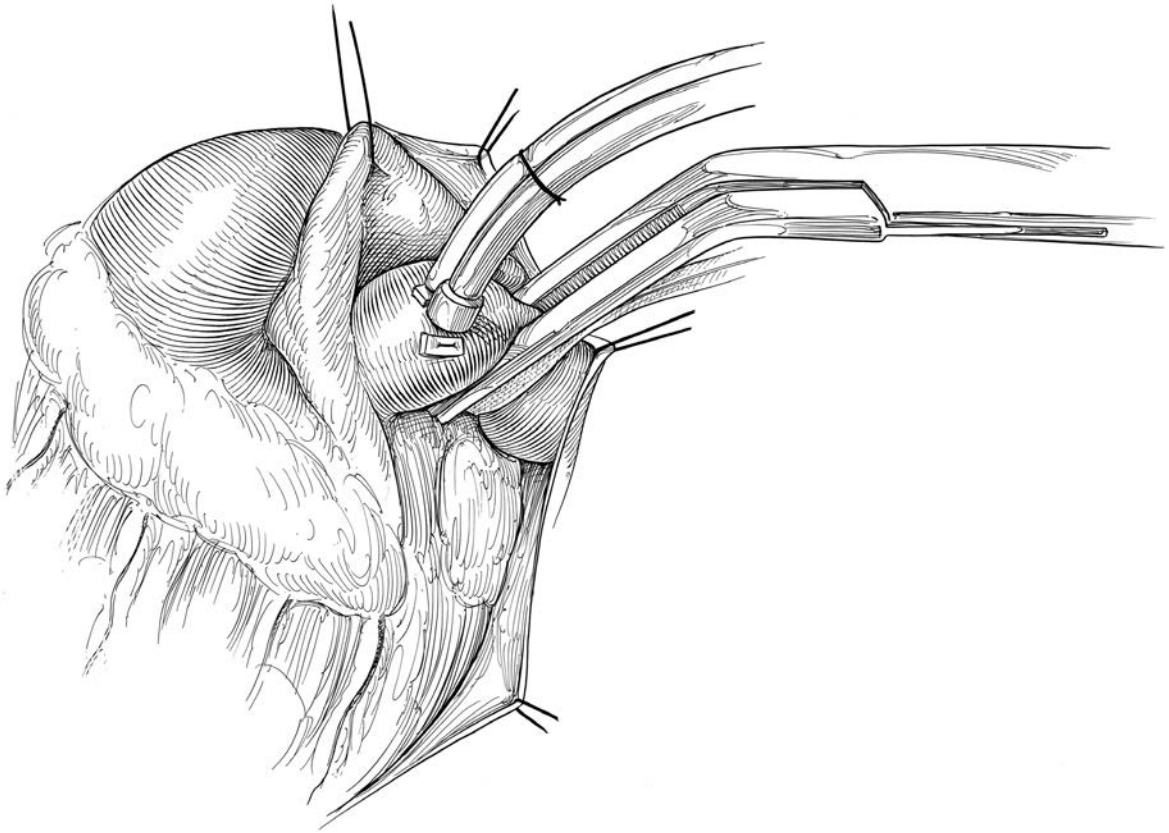


Figure 18.9 Cardioplegia Cannula and Aortic Crossclamp—Right Thoracotomy

should be adjusted to keep the P_aO_2 above 120 mm Hg during CPB. The P_aCO_2 is adjusted during CPB by adjusting total gas flow (L/min) to the oxygenator with the gas flow meter. Blood gases should be measured every 30 minutes during CPB. An alpha-stat strategy for acid-base management is used during CPB to account for the effects of hypothermia [9]. Somewhat counterintuitively this is accomplished by not adjusting the blood gas analysis for body temperature and running the analysis as if the body temperature were 38°C. Severe metabolic acidosis during CPB should be corrected by administration of IV $NaHCO_3$. The balance of evidence in adult humans undergoing CPB favors administration of an antifibrinolytic drug during CPB to counter bypass-induced hyperfibrinlysis and associated bleeding during and after surgery [10, 11]. Although routine antifibrinolytic therapy in human CPB patients has recently come under scrutiny [12] and data in animals is lacking, it has been our practice to administer ε′-aminocaproic acid during CBP in dogs.

Complete CPB is accomplished by cross-clamping the ascending aorta which in turn blocks blood flow to the coronary arteries. Protection of the myocardium from ischemic injury during aortic crossclamping is accomplished by immediate cessation of electromechanical activity and rapid cooling of the myocardium. This is achieved by administration of cold cardioplegia solution into the coronary circulation via the cardioplegia cannula just after the aortic cross-clamp has been placed. The solution contains a high concentration of potassium that arrests the electrical and mechanical activities of the myocardium, thereby greatly reducing its metabolic requirements. Cooling the myocardium to approximately 4° to 8°C further decreases its metabolic requirement. A sanguineous cardioplegia solutions consisting of a mixture of heparinized blood from the bypass circuit and a high-potassium crystalloid solution is used. Ice-cold saline can be placed around the cardiac surface to assist with cooling of the myocardium. All electrical and mechanical activity of the heart should cease after cardioplegia administration. Cardioplegia is repeated every 20 minutes while the aorta is cross-clamped. Ideally, aortic crossclamp time should not exceed 90 minutes.

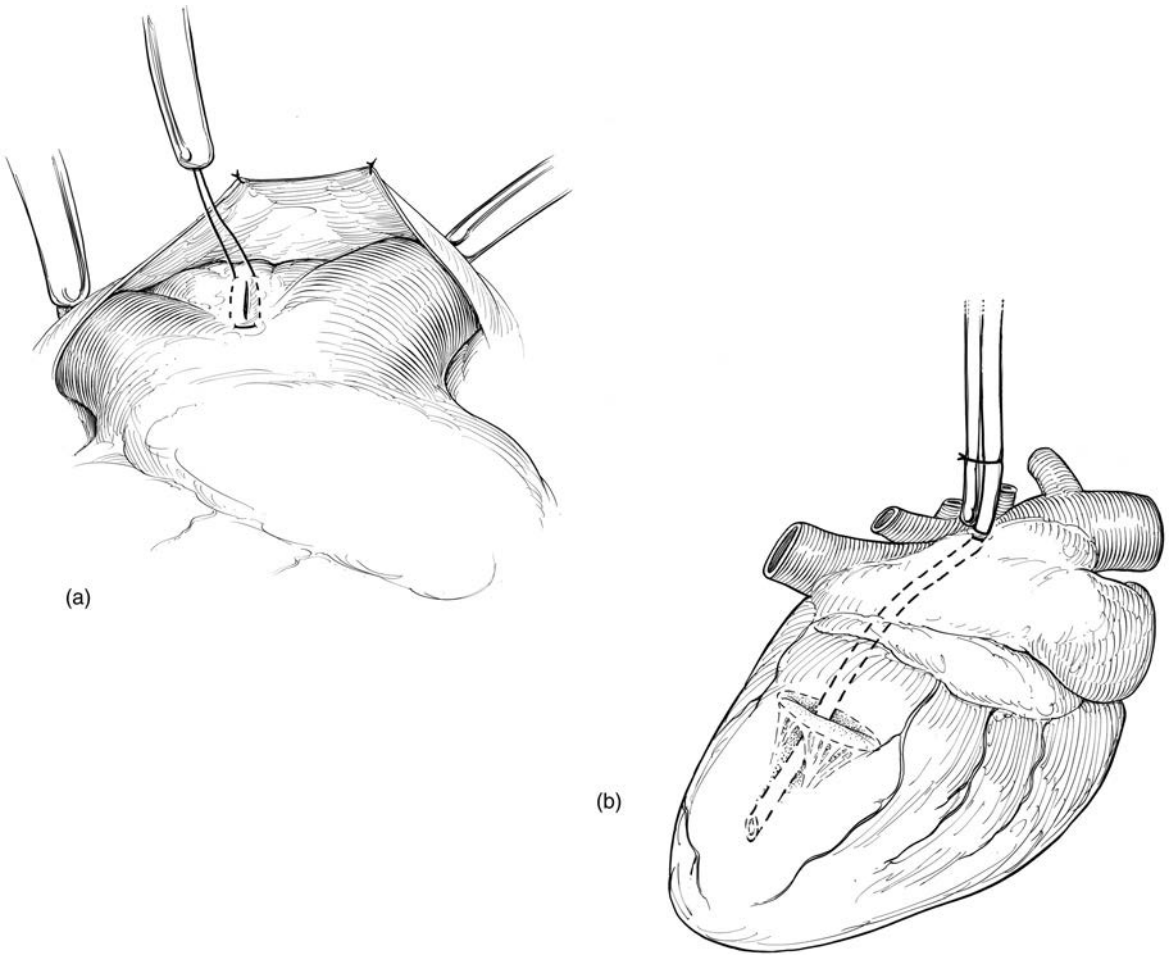


Figure 18.10 Left Ventricular Vent—Right Thoracotomy

Discontinuation of Cardiopulmonary Bypass

Air must be evacuated from the heart before discontinuing CPB. This is accomplished by allowing the cardiac chambers to fill and the incisions to overflow just as the cardiotomy incision is closed. The perfusionist assists with de-airing by briefly diverting blood from the bypass circuit back to the heart and the anesthesiologist assists by applying continuous positive pressure to the lungs to push pulmonary blood back toward the heart. The left ventricle, left atrium, and left auricle are manipulated during de-airing to release any trapped air prior to reestablishing left ventricular ejection. After the aortic cross-clamp is removed the left heart is vented by connecting the vent line from the bypass circuit to the cardioplegia cannula. This helps keep the left ventricle decompressed and scavenges air bubbles ejected from the left heart. A second vent can be placed into left ventricle if necessary to prevent the ventricle from distending during discontinuation of CPB.

If ventricular fibrillation occurs after removal of the crossclamp, the heart is defibrillated with a direct current defibrillator at 20 to 50 joules. If initial attempts at defibrillation are unsuccessful, defibrillation should be repeated every few minutes as the patient is rewarmed until a normal rhythm is restored. Rewarming can begin as the cardiac surgery is being completed, usually while the cardiac incisions are being closed. After the patient is rewarmed to 37°C and an effective cardiac rhythm established, a gradual weaning from CPB is begun. During this period, inotropic and pressor support with drugs such as dobutamine, dopamine, and/or milrinone are usually beneficial to support cardiovascular function. Once the animal has been fully weaned from CPB and is hemodynamically stable, the cannulae are removed in the reverse order, in which they were introduced. Cannulation sites are temporarily closed by tightening the controlling tourniquets. Pursestring sutures are then tied to close cannulation cardiotomy sites after it is clear that the patient will not need to go back on bypass. After cannulae are

removed, protamine sulfate is administered to reverse heparin anticoagulation. Protamine has a very potent hypotensive effect in dogs and must be administered slowly by continuous intravenous infusion. The ACT should return to less than 150 seconds. We routinely administrator fresh whole blood after CPB to restore coagulation factors and functional platelets. The thoracic cavity is closed after confirmation that all cardiotomy incisions are not bleeding.

Care after Cardiopulmonary Bypass

CPB causes a systemic inflammatory response syndrome (SIRS) that has important implications for management after surgery [13]. The SIRS response is initiated by contact with extracorporeal surfaces and is mediated through protein and cellular elements in the blood. Based on the experience in humans, the SIRS response to CPB is more intense in children in part due to their smaller size [14]. A similar effect based on patient size is likely present in animals and is related to the relative surface areas of the perfusion circuit and cardiovascular system of the animal. Understanding that animals must be managed for their underlying cardiac disease and surgery as well as for the SIRS response to CPB is critical to successful post-operative management. Several therapies have been evaluated to prevent or ameliorate the SIRS effect of CPB; however, none have emerged as routine in the management of CPB patients [15].

The first 12 hours after CPB are most critical. Major problems that can occur during this period include bleeding, hypoxemia, hypoventilation, low cardiac output, hypotension, cardiac arrhythmias, low urine output, and electrolyte and acid-base abnormalities. Hemorrhage is a major postoperative concern after CPB. Hemorrhage can be surgical or biological, or both. Surgical hemorrhage results from bleeding from cardiotomy sites that were not adequately closed. This is best prevented by careful inspection of cardiotomy sites prior to closure of the thoracotomy. Even with perfect closure of cardiotomy sites, bleeding can occur after CPB due to the biological effects of CPB. Dilutional and consumptive thrombocytopenia, acquired platelet dysfunction, consumptive and dilutional coagulopathy, and fibrinolysis can all contribute to biological bleeding after surgery. Biological bleeding after CPB is best prevented by avoiding over hemodilution of the patient during CPB and administration of fresh whole blood to restore red blood cells, platelets, and clotting factors in the operating room. Stored whole blood, RBC, or plasma may also be necessary in the postoperative period to restore RBC and coagulation factors. If a cell washer is available, shed blood can be collected, washed, and autotransfused back to the patient. Autotransfusion of unwashed shed blood should be avoided.

Hypoxemia is usually present after CPB. Pulmonary dysfunction, sometimes referred to as *pump lung*, results from pulmonary injury caused by the inflammatory effects of CPB [16]. Managing this pulmonary injury is an important element of post-CPB management. Supplemental oxygen administration (F_IO_2 0.40) is usually indicated for at least 24 hours after CPB. Ventilatory support with positive end-expiratory pressure (5 to 8 cm of water) for 12 hours may be required after surgery, but usually is not necessary. Circulatory support after CPB should include volume support to maintain the CVP between 4 and 10 mm Hg. Bedside echocardiography can also very useful for assessing adequacy of cardiac preload. Because of the generalized increase in vascular permeability after CPB, volume deficits should be corrected primarily with plasma or blood. Administration of synthetic colloid solutions should be avoided after CPB because of their adverse effect on bleeding. Administration of high volumes of crystalloid fluids to restore volume deficits should be avoided after CPB because of risk of worsening pulmonary dysfunction. Hematocrit should be maintained above 30%. In addition to correction of any volume deficits, inotropic and/or pressor support may be necessary to maintain adequate cardiac output and mean systemic blood pressure. Cardiac arrhythmias that impair cardiac function or carry an inappropriate risk of cardiac arrest (e.g., ventricular tachycardia, atrial fibrillation) should be managed medically with appropriate drug therapies. Urine output should be monitored for 12 hours after surgery to ensure adequate renal function. Hypokalemia and hypocalcemia are frequently encountered after CPB and should be corrected.

Hybrid Cardiac Surgery

Hybrid cardiac surgery is an emerging option for correction of congenital and acquired structural cardiac defects. Hybrid cardiac surgery combines image-guided catheter-based interventional techniques/devices with minimal-incision direct cardiotomy approaches. These minimal-incision cardiotomy approaches can often be combined with minimal-incision thoracic approaches such as combining a minimal-incision left thoracotomy with a transapical cardiotomy. Hybrid cardiac approaches allow for larger delivery systems and more direct access to cardiac defects than would otherwise be possible with traditional percutaneous (transvascular)

interventional cardiology techniques. Hybrid cardiac approaches are particularly well suited for left heart interventions, as they can provide access to the left heart without the need for transseptal puncture. These advantages are particularly useful in smaller animals where traditional percutaneous approaches are often not feasible because of their size. Hybrid cardiac surgeries are performed in an imaging-capable hybrid operating room. Imaging for hybrid cardiac procedures generally includes both

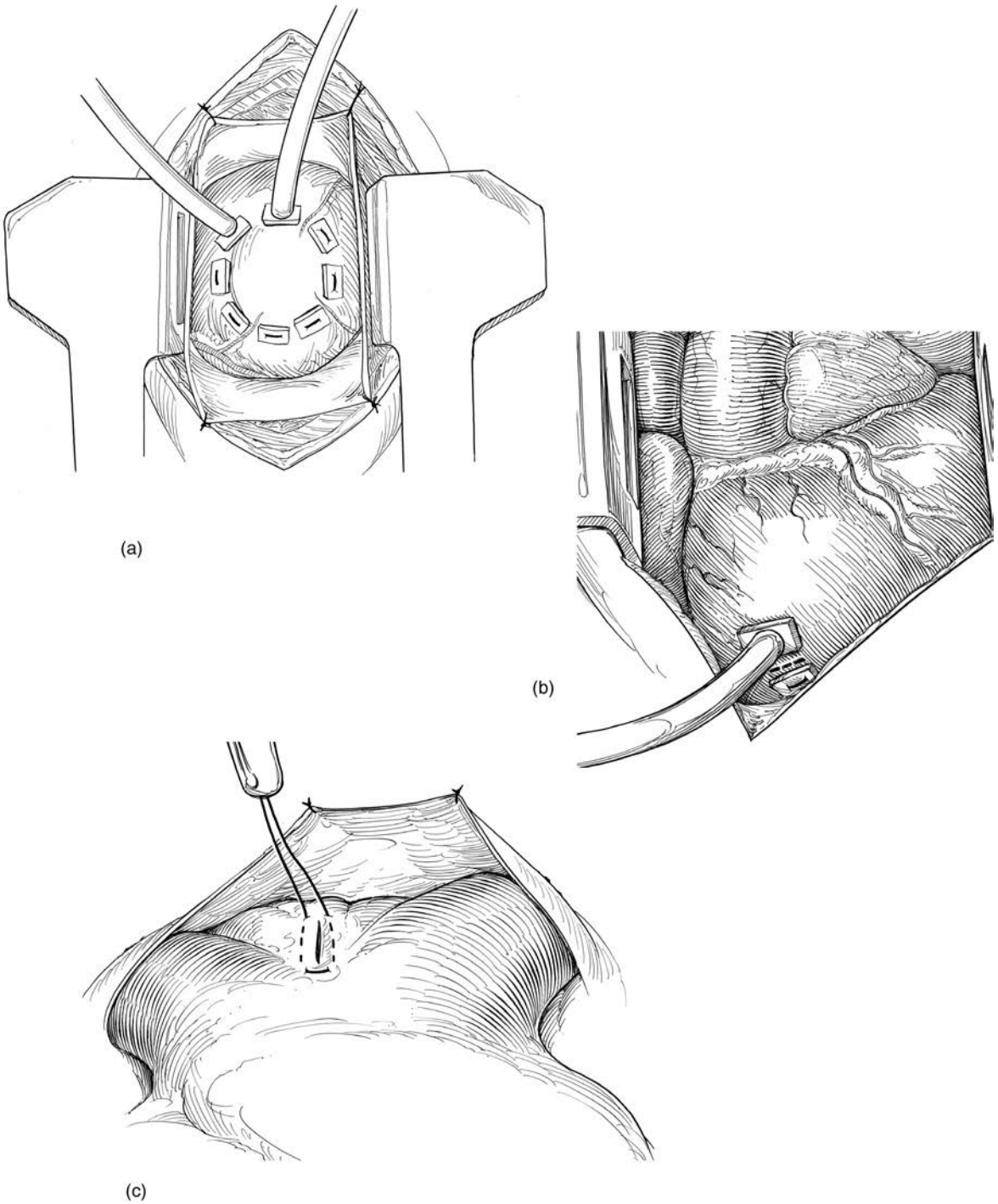


Figure 18.11 Hybrid Cardiac Approaches

fluoroscopy and echocardiography (transesophageal or epicardial). Hybrid operating rooms can also be equipped for video-assisted surgery to enable a fully capable, minimally invasive operating room. Hybrid cardiac surgeries require the skillset of a surgeon and the cardiac imaging and interventional expertise of a cardiologist. Thus, in most instances, hybrid cardiac procedures are best performed by a hybrid cardiac team that includes an interventional cardiologist and a surgeon.

Hybrid cardiac surgery can theoretically be performed through any cardiac chamber or great vessel wall that gives best direct access to a structural defect. A hybrid cardiotomy approach consists of a small incision made within a pledget-reinforced mattress or pursestring suture. Guidewires and catheter-based devices can either be passed through standard introducers or directly into the cardiac chamber without an introducer. Several standard hybrid cardiotomy approaches have been described. Transapical hybrid cardiotomy is performed at the cardiac apex and provides straight direct access to the left ventricle and atrium, mitral valve, and aortic valve (Figure 18.11a). Two pledget-reinforced pursestring sutures are placed in the cardiac apex and passed through tourniquets to control bleeding during introduction of catheters. The sutures are tied to close the cardiotomy when the intervention is completed. The transapical cardiac approach can be combined with a minimal-incision thoracotomy to make for a truly minimally invasive approach. Other hybrid cardiotomy sites include the right ventricular outflow tract (Figure 18.11b), the right or left auricular appendage, and dorsal left atrium from a right thoracotomy (Figure 18.11c). A hybrid approach directly through the left atrial wall from a left thoracotomy should be used cautiously because of the fragility of the atrial wall when the atrium is dilated.

Several hybrid cardiac surgeries have been reported or are in development for small animals including device closure of ventricular septal defect, atrial septal defect, and aorticopulmonary fistula; mitral valve repair or implantation (replacement); balloon dilation and/or stent implantation for congenital and acquired (neoplasia) cardiac obstructions or valve stenosis.

References

1 Hunt GB, Malik R, Bellenger CR, and Pearson MR. 1992. Total venous inflow occlusion in the normothermic dog: a study of haemodynamic, metabolic and neurological consequences. *Research Veterinary Science.* 52(3):371–7.

2 Pelosi A, Anderson LK, Paugh J, Robinson S, and Eyster GE. 2013. Challenges of cardiopulmonary bypass-a review of the veterinary literature. *Veterinary Surgery.* 42(2):119–36.

3 Uechi M, Harada K, Mizukoshi T, Mizuno T, Mizuno M, Ebisawa T, and Ohta Y. 2011. Surgical closure of an atrial septal defect using cardiopulmonary bypass in a cat. *Veterinary Surgery.* 2011;40(4):413–7.

4 Borenstein N, Gouni V, Behr L, Trehiou-Sechi E, Petit A, Misbach C, Raillard M, Retortillo JL, Pouchelon JL, Pierrel A. 2015. Surgical Treatment of Cor Triatriatum Sinister in a Cat Under Cardiopulmonary Bypass. *Veterinary Surgery.* 44(8):964–9.

5 Klement P, del Nido PJ, Mickleborough L, MacKay C, Klement G, and Wilson GJ. 1987. Technique and postoperative management for successful cardiopulmonary bypass and open-heart surgery in dogs. *Journal of the American Veterinary Medical Association.* 190(7):869–74.

6 Orton EC, Mama K, Hellyer P, and Hackett TB. 2001. Open surgical repair of tetralogy of Fallot in dogs. *Journal of the American Veterinary Medical Association.* 219(8):1089–93, 73.

7 Lew LJ, Fowler JD, Egger CM, Thomson DJ, Rosin MW, and Pharr JW. 1997. Deep hypothermic low flow cardiopulmonary bypass in small dogs. *Veterinary Surgery.* 26(4):281–9.

8 Kanemoto I, Taguchi D, Yokoyama S, Mizuno M, Suzuki H, and Kanamoto T. 2010. Open heart surgery with deep hypothermia and cardiopulmonary bypass in small and toy dogs. *Veterinary Surgery.* 39(6):674–9.

9 Bove EL, West HL, and Paskanik AM. 1987. Hypothermic cardiopulmonary bypass: a comparison between alpha and pH stat regulation in the dog. *Journal of Surgical Research* 42(1): 66–73.

10 Pustavoitau A, and Faraday N. 2016. Pro: Antifibrinolytics Should Be Used in Routine Cardiac Cases Using Cardiopulmonary Bypass (Unless Contraindicated). *Journal Cardiothoracic Vascular Anesthesia.* 30(1):245–7.

11 Koster A, Faraoni D, and Levy JH. 2015. Antifibrinolytic Therapy for Cardiac Surgery: An Update. *Anesthesiology.* 123(1):214–21.

12 Harvey R, and Salehi A. 2016. Con: Antifibrinolytics Should Not Be Used Routinely in Low-Risk Cardiac Surgery. *J Cardiothoracic Vascular Anesthesia.* 30(1): 248–51.
13 Warren OJ, Smith AJ, Alexiou C, Rogers PL, Jawad N, Vincent C, Darzi AW, and Athanasiou T. 2009. The inflammatory response to cardiopulmonary bypass: Part 1—mechanisms of pathogenesis. *Journal Cardiothoracic Vascular Anesthesia.* 23(2): 223–31.
14 Kozik DJ, and Tweddell JS. 2006. Characterizing the inflammatory response to cardiopulmonary bypass in children. *The Annals of Thoracic Surgery.* 81(6): S2347–54.
15 Warren OJ, Watret AL, de Wit KL, Alexiou C, Vincent C, Darzi AW, and Athanasiou T. 2009. The inflammatory response to cardiopulmonary bypass: part 2—anti-inflammatory therapeutic strategies. *Journal Cardiothoracic Vascular Anesthesia.* 23(3): 384–93.
16 Clark SC. 2006. Lung injury after cardiopulmonary bypass. *Perfusion.* 21(4):225–8.

19

Patent Ductus Arteriosus

E. Christopher Orton

The ductus arteriosus is a vascular connection between the pulmonary artery and descending aorta that allows venous blood to bypass the collapsed fetal lungs. The ductus arteriosus closes soon after birth during the transition from fetal to extra-uterine life. Continued patency of the ductus arteriosus for more than a few days after birth results in the condition of patent ductus arteriosus (PDA).

PDA is among the most common congenital defects in dogs, accounting for 17% to 21% of congenital malformations in this species [1,2]. The defect also occurs in cats but with a lower prevalence [1]. PDA is seen more commonly in purebred dogs with a predilection for females. Poodles, keeshonds, Maltese, bichon frisé, Yorkshire terrier, cocker spaniels, Pekinese, collies, shelties, Pomeranians, Welsh corgis, and other breeds have an established predisposition for PDA [3]. PDA is proven heritable in poodles and should be considered so in other breeds [4]. The defect is caused by segmental hypoplasia and asymmetry of the ductal smooth muscle mass that results in failure of ductus contraction [4,5].

Pathophysiology

Persistence of the ductus arteriosus after birth allows left-to-right shunting of blood from the aorta to the pulmonary artery. The result is severe volume overload of the left heart leading to left ventricular and atrial dilation, progressive myocardial dysfunction, and left-sided congestive heart failure. As the left ventricle dilates, functional mitral regurgitation develops, adding to the volume overload the left ventricle. Atrial fibrillation is a common late sequela. A majority of dogs and cats with untreated PDA die from progressive heart failure early in life; however, the clinical course without treatment is unpredictable [6,7].

Some animals with PDA develop progressive pulmonary hypertension that can reverse the direction of shunt flow, resulting in hypoxemia and cyanosis that is more intense in the caudal portions of body. Right-to-left PDA may develop as a progressive sequela to chronic pulmonary overcirculation or occur from birth due to persistent pulmonary hypertension and failure of transition to extra-uterine life [8]. Severe pulmonary hypertension diminishes the risk for developing progressive left-sided heart failure, but causes varying degrees of hypoxemia, exercise intolerance, pelvic limb dysfunction, and polycythemia.

Diagnosis

Animals with PDA may be apparently asymptomatic to their owners at initial presentation, exhibiting only mild activity intolerance. Clinical signs are compatible with left-sided heart failure and include severe activity intolerance, exertional tachypnea, or dyspnea. Physical examination reveals a continuous murmur at the left heart base with or without continuous cardiac thrill. The left cardiac impulse is caudally displaced and hyperdynamic. Arterial pulses are bounding due to low arterial diastolic pressure caused by rapid runoff of blood through the ductus during diastole.

Thoracic radiographs show moderate to severe left atrial and ventricular enlargement, enlargement of pulmonary vessels (pulmonary overcirculation), and a characteristic aneurysm of the descending aorta on the DV view. Tall R waves (> 2.5 mV) in a lead II electrocardiogram support the diagnosis. Atrial fibrillation or ventricular ectopy are seen in animals with advanced disease.

Echocardiography confirms a diagnosis of PDA and helps rule out concurrent cardiac defects. Echocardiographic findings include left atrial enlargement, left ventricular chamber dilation, dilation of the main pulmonary artery, increased aortic ejection velocity, and retrograde turbulent flow in the pulmonary artery on

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Doppler analysis. PDA is classified by angiographic appearance into four morphologic types (types I, IIA, IIB and III) based on presence and location of an abrupt narrowing of the ductus [9]. The type III ductal morphology shows an absence of ductal narrowing and currently represents a relative contraindication for interventional ductal closure.

Animals with right-to-left or "reverse" PDA have a different clinical presentation and diagnostic findings. The most common presenting complaints are exercise intolerance and pelvic limb collapse on exercise. Cyanosis is a hallmark physical finding. Classically, the cyanosis is *differential* (i.e., more intense in the caudal mucous membranes), but may be observed cranially as well. Femoral pulses are normal. Usually there is no cardiac murmur, especially if polycythemia is present. Thoracic radiographs show evidence of biventricular enlargement and a markedly enlarged pulmonary artery segment. The pulmonary arteries may appear enlarged and tortuous or normal. The defect is confirmed by echocardiography, demonstrating evidence of severe pulmonary hypertension including severe concentric hypertrophy of the right ventricle. The diagnosis can be confirmed by a venous bubble study demonstrating right-to-left shunting of micro-bubbles into the abdominal aorta.

Indications for Surgery

Based on the poor long-term prognosis without correction, PDA closure is generally strongly indicated in dogs and cats with left-to-right PDA unless shunt flow is clearly documented to be trivial [6,10]. PDA closure can be accomplished by transcatheter intervention or surgery. Both approaches are essentially equally effective, with interventional approaches being less-invasive and therefore currently preferred at most centers for most animals. The preferred interventional approach for PDA closure in dogs is by the transarterial canine ductal occluder. This procedure has a limitation on patient size in that animals must be at least 2.5 kg based on current availability of the device sizes. Closure with the canine ductal occluder is also considered relatively contraindicated in dogs with a type III ductal morphology more common in certain breeds such as the German shepherd dog.

Surgical closure of PDA can be accomplished by ligation or division and suturing of the ductus arteriosus. The advantage of surgical closure is that it is not limited by patient size or PDA morphology type. Surgical closure should be undertaken in dogs and cats as soon as possible after diagnosis, preferably before 16 weeks of age. Older animals should undergo surgery as soon as possible. Even animals with severe secondary myocardial dysfunction and functional mitral regurgitation will benefit from PDA closure. Animals that present in congestive heart failure should be stabilized medically and then undergo closure as soon as pulmonary edema is resolved, usually within 24 to 48 hours. Animals with pulmonary hypertension can undergo PDA ligation so long as pulmonary artery pressures have not reached systemic levels. Pretreatment of "balanced" or bidirectional PDA with oral sildenafil may selectively decrease active pulmonary vasoconstriction and help identify animals that can undergo safe closure of PDA. Closure of a right-to-left PDA is contraindicated.

PDA Ligation

Surgical closure of PDA is most often accomplished by ligation of the ductus arteriosus, which is generally regarded as the safest and most technically feasible approach. PDA ligation is accomplished through a left fourth thoracotomy in the dog and a left fourth or fifth thoracotomy in a cat. The vagus nerve courses over the ductus arteriosus and serves as an anatomic landmark for identification of the ductus arteriosus (Figure 19.1a). The ductus is located predominately outside of the pericardial space. The vagus nerve is isolated at the level of the ductus and gently retracted either dorsally or ventrally with one or two sutures (Figure 19.1b). Rarely a persistent left cranial vena cava may overlie the ductus arteriosus. In this case the vein should be isolated and retracted with the vagus nerve. A persistent left cranial vena cava should not be ligated or divided. Dissection and ligation of the ductus arteriosus is generally accomplished outside of the pericardium. The caudal aspect of the ductus is isolated by passing a right-angle forceps between the descending aorta and left pulmonary artery branch in the transverse plane. Exposure of the cranial aspect of the ductus is facilitated by cutting the loose connective tissues between the ascending aorta and ductus with scissors (Figure 19.1c). A right-angle forceps is passed between the aorta and ductus at a 45° angle to the transverse plane (Figure 19.1d). The right-angle forceps is then passed medial to the ductus in a caudal to cranial direction (Figure 19.1e). The forceps can be guided through the space between the aorta and ductus by palpating the tip of the forceps to assure that the medial wall of the ductus has not been caught by the forceps. The tip of the forceps should be pointed slightly dorsally to avoid injury to

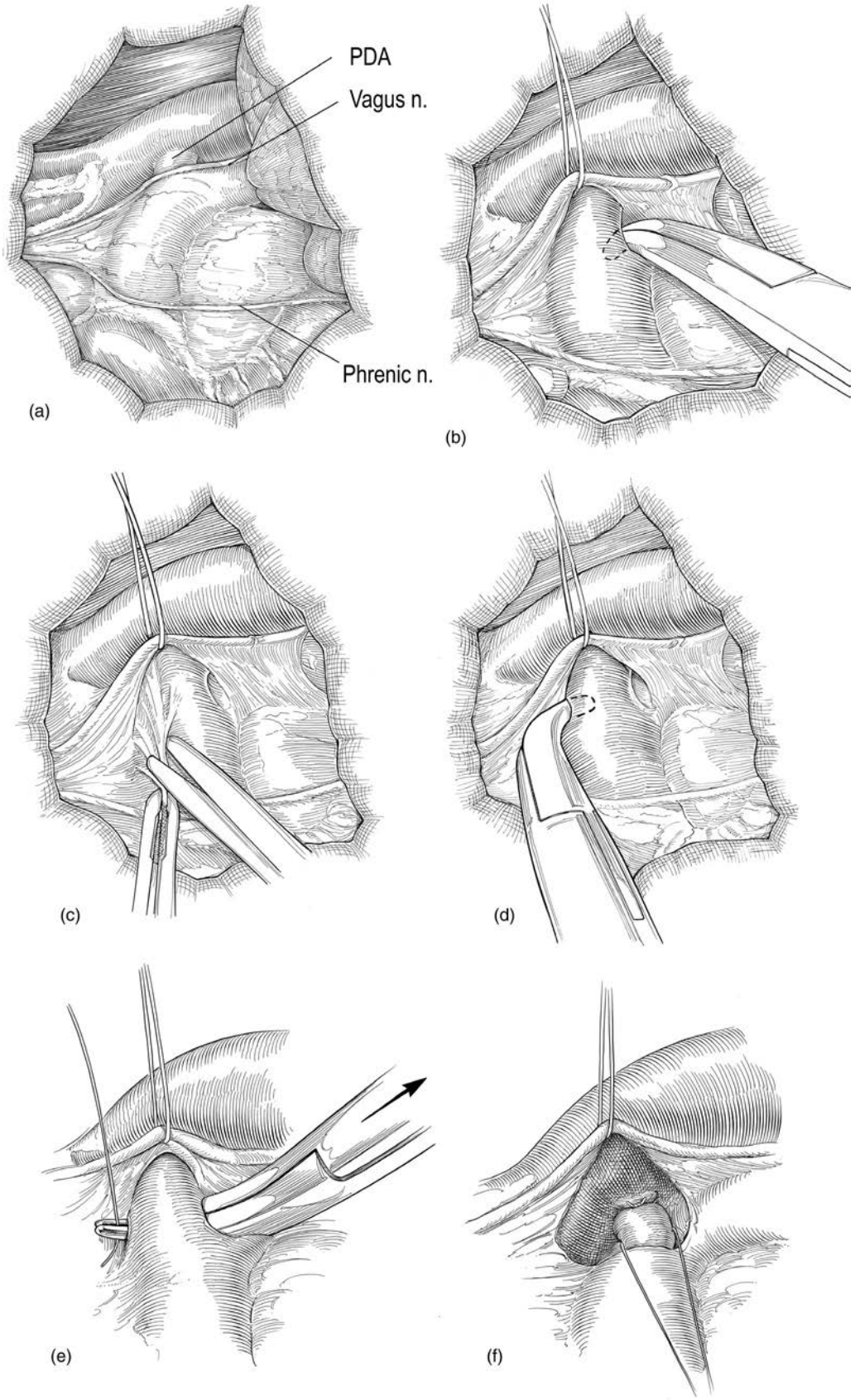

Figure 19.1 Patent Ductus Arteriosus Ligation.

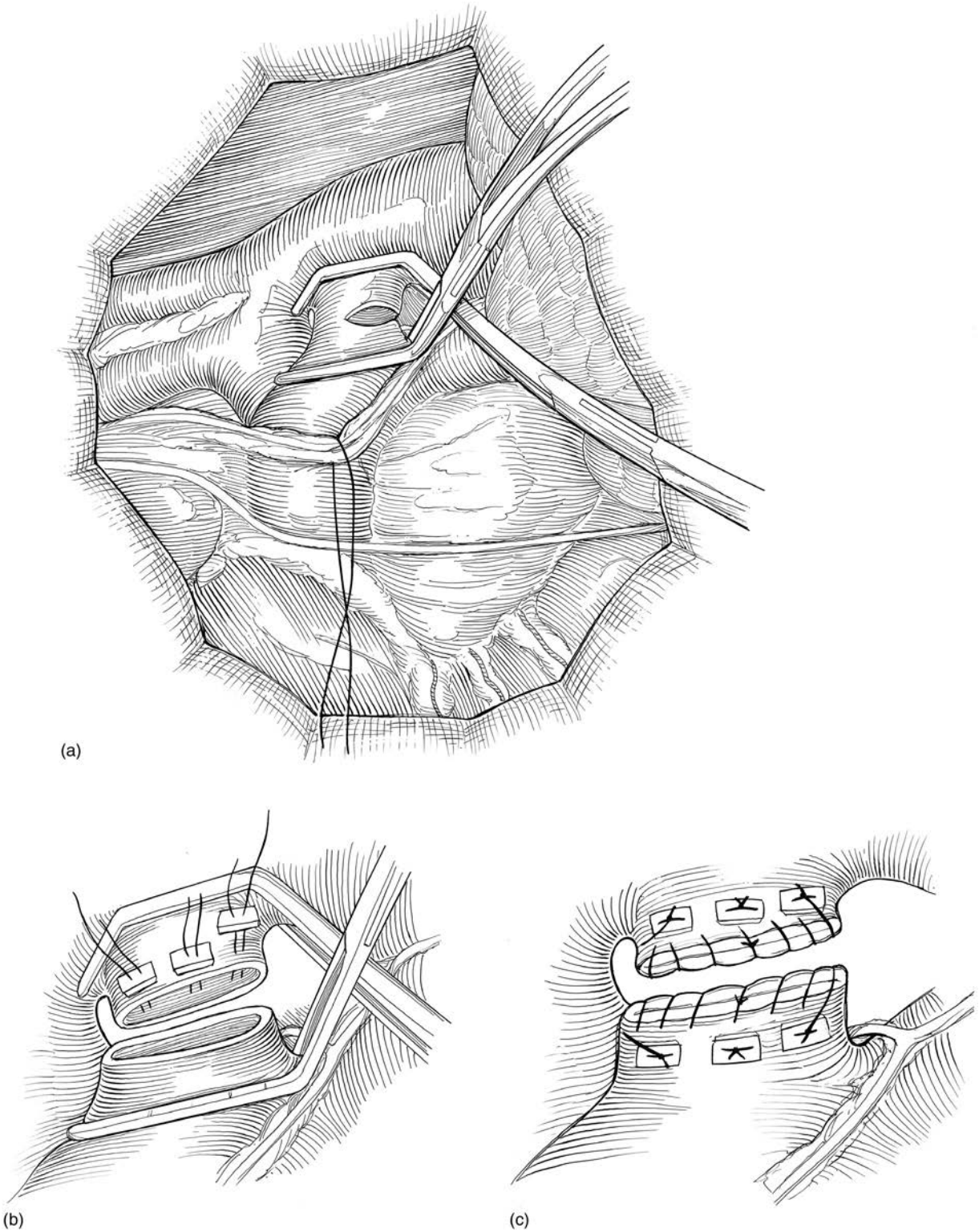

Figure 19.2 Division of Patent Ductus Arteriosus.

the pulmonary artery. Blunt dissection by opening and closing the forceps medial to the ductus should be avoided to prevent injury to the medial wall of the ductus and catastrophic bleeding. The right-angle forceps are used to pass two sutures (# silk) around the ductus. The ductus arteriosus is closed by slowly tightening and tying the silk ligatures (Figure 19.1f).

Division of PDA

Surgical division of a PDA can be considered for closure of large-diameter PDA. Reasons to consider surgical division are a large type III ductus that may be difficult to safely dissect or completely close by ligation. If significant hemorrhage occurs during dissection for PDA ligation, the surgery should be immediately converted to a surgical division.

Surgical division of PDA is accomplished through the same thoracic approach as ligation. The vagus nerve is isolated and retraction ventrally, as with ligation. A tangential vascular clamp is placed deeply on the descending aorta dorsal the ductus (Figure 19.2a). A second angled or tangential vascular clamp is placed on the pulmonary artery just ventral to the ductus. It is generally preferred to place both vascular clamps from a caudal to cranial direction. The ductus is divided between the vascular clamps (Figure 19.2b). The divided ends are closed with pledget-reinforced mattress sutures of 4-0 or 5-0 polypropylene suture and then oversewn with a simple continuous suture pattern (Figure 19.2c).

Expected Outcomes

Surgical mortality rates of 0% to 7% have been reported in retrospective studies involving 50 or more dogs [7, 11–15]. Vascular rupture resulting in severe hemorrhage is the most serious complication associated with PDA ligation and the most common cause of operative mortality [11, 13, 15]. Operative mortality rates approaching 0% are achievable by experienced surgeons even when higher risk cases are included. An unintended consequence of the advent of interventional ductal closure is that experience levels among surgeons is diminishing. PDA closure is associated with favorable short-term and long-term outcomes in the majority of cases, even in older animals or when secondary functional MR or myocardial dysfunction are present [6, 12, 15, 16]. Long-term favorable outcomes can also be expected in cats undergoing surgical ligation of PDA [17]. PDA closure will be curative for most animals and at least strongly palliative for other animals with significant secondary or primary structural heart disease. Some degree of residual ductal flow is not uncommon after surgical ligation of a large ductus arteriosus; however, this usually has minimal consequences so long as the flow is considered trivial.

References

1 Schrope DP. 2015. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. *Journal of Veterinary Cardiology.* 17(3):192–202.

2 Oliveira P, Domenech O, Silva J, Vannini S, Bussadori R, and Bussadori C. 2011. Retrospective review of congenital heart disease in 976 dogs. *Journal of Veterinary Internal medicine.* 2011;25(3): 477–83.

3 Buchanan JW. 1999. Prevalance of cardiovascular disorders. In: Fox PR, Sisson D, and Moise NS eds. *Textbook of Canine and Feline Cardiology*, pp. 457–70 Philadelphia: W.B. Saunders Co.

4 Patterson DF. 1965. Congenital heart disease in the dog. *Annals of the New York Academy Science.* 127(1):541–69.

5 Buchanan JW, and Patterson DF. 2003. Etiology of patent ductus arteriosus in dogs. *Journal of Veterinary Internal Medicine.* 17(2):167–71.

6 Van Israel N, Dukes-McEwan J, and French AT. 2003. Long-term follow-up of dogs with patent ductus arteriosus. *The Journal of Small Animal Practice.* 44(11):480–90.

7 Eyster GE, Eyster JT, Cords GB, and Johnston J. 1976. Patent ductus arteriosus in the dog: Characteristics of occurrence and results of surgery in one hundred consecutive cases. *Journal of the American Veterinary Medical Association.* 168(5): 435–8.

8 Oswald GP, and Orton EC. 1993. Patent ductus arteriosus and pulmonary hypertension in related Pembroke Welsh corgis. *Journal of the American Veterinary Medical Association.* 202(5):761–4.

9 Miller MW, Gordon SG, Saunders AB, Arsenault WG, Meurs KM, Lehmkuhl LB, Bonagura JD, and Fox PR. 2006. Angiographic classification of patent ductus arteriosus morphology in the dog. *Journal of Veterinary Cardiology.* 8(2):109–14.

10 Saunders AB, Gordon SG, Boggess MM, and Miller MW. 2014. Long-term outcome in dogs with patent ductus arteriosus: 520 cases (1994–2009). *Journal of Veterinary Internal Medicine.* 28(2): 401–10.

11 Hunt GB, Simpson DJ, Beck JA, Goldsmid SE, Lawrence D, Pearson MR, and Bellenger CR. 2001. Intraoperative hemorrhage during patent ductus arteriosus ligation in dogs. *Veterinary Surgery.* 30(1):58–63.

12 Bureau S, Monnet E, and Orton EC. 2005. Evaluation of survival rate and prognostic indicators for surgical treatment of left-to-right patent ductus arteriosus in dogs: 52 cases (1995–2003). *Journal of the American Veterinary Medical Association.* 227(11):1794–9.

13 Birchard SJ, Bonagura JD, and Fingland RB. Results of ligation of patent ductus arteriosus in dogs: 201 cases (1969–1988). 1990. *Journal of the American Veterinary Medical Association.* 196(12):2011–3.

14 Buchanan JW. 1994. Patent ductus arteriosus. *Seminars Veterinary Medicine and Surgery (Small Anim).* 9(4):168–76.

15 Goodrich KR, Kyles AE, Kass PH, and Campbell F. 2007. Retrospective comparison of surgical ligation and transarterial catheter occlusion for treatment of patent ductus arteriosus in two hundred and four dogs (1993–2003). *Veterinary Surgery.* 36(1):43–9.

16 Van Israel N, French AT, Dukes-McEwan J, and Welsh EM. 2003. Patent Ductus Arteriosus in the older Dog. *Journal of Veterinary Cardiology.* 5(1): 13–21.

17 Bascunan A, Thieman Mankin KM, Saunders AB, Bright JM, Scharf V, et al. 2017. Patent ductus arteriosus in cats (*Felis catus*): 50 cases (2000–2015). *Journal of Veterinary Cardiology*. 19(1):35–43.

20

Pulmonary and Aortic Valves

E. Christopher Orton

Pulmonary Stenosis and Pulmonary Outflow Obstructions

The pulmonary outflow tract consists of the outflow portion of the right ventricle (infundibulum), pulmonary valve, pulmonary artery (trunk) and the branch pulmonary arteries. Obstructive malformations of the pulmonary outflow track include double-chambered right ventricle, primary infundibular stenosis, dynamic right ventricular obstruction, valvular pulmonary stenosis, pulmonary artery stenosis, stenosis of the branch pulmonary arteries, and combinations of these. Double-chambered right ventricle or mid-right ventricular obstruction consists of an anomalous muscular band at the junction of the inflow and outflow portions of the right ventricle. This defect and its surgical correction are discussed in Chapter 23. Primary infundibular stenosis consists of fibromuscular bands arising within the infundibular portion of the right ventricle and has been described in both dogs and cats [1, 2]. Dynamic right ventricular obstruction (DRVO) is a dynamic mid-systolic obstruction recognized by its "dagger-shaped" Doppler envelope. The condition differs from primary infundibular stenosis in that it lacks a fixed obstruction component. DRVO can result from severe right ventricular wall thickening secondary valvular pulmonary stenosis [3] and has been described as a primary condition in cats [4]. Valvular pulmonary stenosis (PS) represents a constellation of pathologic malformations of the pulmonary valve. "Typical" PS consists of commissural fusion of the valve leaflets with a central office and "doming" of the valve leaflets during systole. The leaflets may be mildly thickened. *Pulmonary valve dysplasia* is characterized by a spectrum of pathologic features, including hypoplasia of the valve annulus, thickening and immobility of the valve leaflets, and asymmetric narrowing at the sinotubular junction. The former has been referred to as a type A defect, whereas the latter is termed a type B defect [5]. In actual fact, these pathologies likely represent the ends of a pathologic spectrum rather than distinct entities, with the degree of annular hypoplasia having the most important implication regarding intervention and prognosis [5–8]. Valvular PS one of the most common congenital heart defects in dogs, accounting for 21% to 32% of congenital malformations in this species [9–11]. Several breeds of dog are overrepresented in various reports of valvular PS including English bulldog, boxer, bull mastiff, cocker spaniel, Labrador retriever, Cavalier King Charles spaniel, several terrier breeds, and others. Valvular PS also occurs in cats but is less common [10]. English bulldogs and boxers have a high concurrent incidence of an anomalous left coronary artery that originates from a single right coronary ostium and courses cranial to the right ventricular outflow tract just ventral to the pulmonic valve [12]. This defect, termed an R2A type anomalous coronary artery, is usually associated with degree of subvalvular PS and has obvious implications regarding interventional options in these breeds. Pulmonary artery stenosis is characterized by a discrete fibrous membranes within the main or branch pulmonary arteries. The condition is reported in dogs [13–15] and cats [16].

Pathophysiology

PS and other pulmonary outflow obstructions cause pressure overload on the right ventricle. The right ventricle responds with concentric wall thickening which may in turn result in dynamic obstruction through the right ventricular outflow tract worsening the obstruction caused by the fixed stenosis [3]. Dogs with mild to moderate obstructions may remain asymptomatic for several years; whereas dogs with severe obstructions show activity intolerance, syncope, right-sided congestive heart failure, and/or sudden cardiac death. Concurrent tricuspid regurgitation increases the

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likelihood of developing congestive heart failure and cardiac related death [17]. It is not uncommon for PS to be accompanied by a right-to-left shunting patent foramen ovale (PFO). Concurrent PFO decreases the likelihood of congestive heart failure, but can be associated with activity-related hypoxemia, cyanosis and polycythemia. The prevalence of concurrent PFO in dogs with PS is reported to be as high as 39% of cases [18].

Diagnosis

Dogs with PS may not show apparent clinical signs for several years depending on severity. Severely affected animals may present with stunted growth, activity intolerance, syncope, or abdominal distension due to ascites. The predominate finding on physical examination is a systolic ejection murmur heard best at the left cardiac base. Signs of right-sided congestion may be present including jugular distension, systemic venous hypertension, hepatomegaly, positive hepatojugular reflex, or ascites. The electrocardiogram usually shows a prominent S wave in leads I, II, and aVF indicative of a right axis shift and right ventricular concentric hypertrophy. Thoracic radiographs show varying degrees of right ventricular and main pulmonary artery segment enlargement.

Diagnosis of PS is confirmed by echocardiography. Echocardiographic findings include right ventricular wall thickening, dilation of the main pulmonary artery, malformation of the pulmonary valve, and an elevated pulmonary ejection velocity. The systolic pressure gradient across the stenosis reflects the severity of the stenosis and can be measured directly by catheterization of the right heart with a flow-directed balloon catheter or indirectly by Doppler echocardiography. In the latter, the systolic pressure gradient (ΔP) across the defect is determined by Doppler measurement of the peak pulmonary ejection velocity (V) and calculation from the modified Bernoulli equation ($\Delta P = 4V^2$).

Indications for Surgery

Prognosis for animals with PS depends on the magnitude of the obstruction and the degree right ventricular hypertrophy. There is general agreement that a Doppler-derived pressure gradient across the defect of 80 mm Hg or greater in an awake dog warrants an intervention [5, 19]. Evidence for intervention in dogs with Doppler-derived pressure gradients of 60 mm Hg or greater is less compelling, but intervention should be considered if clinical signs are present [17, 20–22]. Presence of mild or greater tricuspid regurgitation or right-to-left shunting PFO should also factor into the decision regarding intervention for PS.

Interventional options for valvular PS include transvenous balloon valvuloplasty, transventricular pulmonary valvuloplasty, pulmonary patch-graft, and pulmonary valve bypass conduit. Choice of intervention depends on several considerations including location and morphologic type of pulmonary outflow obstruction, invasiveness of the intervention, availability of equipment, and presence of concurrent cardiac defects. Transvenous balloon valvuloplasty (BV) is accomplished by transvenous (percutaneous) introduction of an appropriate-sized balloon catheter. The balloon catheter is passed through the right heart across the stenosis under fluoroscopic guidance and inflated to tear the fused valve leaflets and effectively increase the central valvular office [7]. BV is generally regarded as an effective intervention for type A (typical) valvular PS, but is considered less effective for type B (dysplastic) PS morphology, especially when annular hypoplasia is present [5, 17, 19, 21, 23, 24].

Closed transventricular "pulmonary valvulotomy" was first reported by Brock in 1948 in three human patients with congenital PS [25] and in a dog with congenital PS in 1953 [26]. With the development of specialized instruments such as the Cooley pediatric valve dilator, the procedure evolved into the current procedure of transventricular pulmonary valvuloplasty (TPV). Prior to the development of percutaneous BV, TPV was commonly performed in dogs with PS [6] and has been employed in dogs with primary infundibular stenosis [1]. TPV has largely been replaced by less invasive BV, but can still be considered as part of a surgical plan if there are other conditions requiring surgical intervention; or if BV is not available, fails, or cannot otherwise be accomplished. Direct comparisons between BV and TPV are not available, but presumably they carry similar indications and results. Pressure gradients are rarely returned to normal by dilation valvuloplasty interventions (i.e., BV and TPV) and initial reductions in the systolic pressure gradient may not be sustained. Dilation valvuloplasty interventions are most likely to be effective for type A valvular PS that is not associated with extensive hypertrophy of the right outflow tract, and less likely to be effective for type B PS with hypoplastic valve annulus. Successful treatment of pulmonary artery stenosis (supravalvular PS) by combined transvenous BV and stent placement has been described [14]. Dilation valvuloplasty interventions are considered relatively contraindicated in dogs with R2A anomalous left coronary artery because of risk of injury to the coronary artery during dilation.

"Conservative" BV has been advocated for dogs with this anomaly.

Pulmonary patch-graft (PPG) is indicated for dogs with severe type B valvular PS, particularly when annular hypoplasia is present. Evidence of dynamic outflow obstruction secondary to severe right ventricular hypertrophy is an additional indication to avoid the possibility of worsening dynamic obstruction caused by only addressing the fixed obstruction, inducing a so called *suicide ventricle* [3]. PPG is most often performed after dogs have failed to be adequately palliated by a percutaneous BV. Several surgical modifications for applying a patch-graft to the right ventricular outflow tract, with or without inflow occlusion, have been described [27–31]. PPG can also be performed with the aid of cardiopulmonary bypass [32]. PPG is absolutely contraindicated in dogs with a R2A anomalous left coronary artery. An anomalous coronary artery is not always visible on the surface of the heart so inspection during surgery is not a reliable way to rule out its presence. The defect must be definitively ruled out by angiography or echocardiography prior to performing PPG—especially in English bulldogs, boxers, and other dogs at risk for this anomaly.

Pulmonary valve bypass (PVB) or right ventricle-to-pulmonary artery conduit consists of placement of a conduit from the right ventricle to the main pulmonary artery providing a bypass for flow around the PS. The conduit would preferentially include a valve and rigid ventricular connector. PVB is the only surgical option for dogs with severe PS with R2A type anomalous coronary artery.

Transventricular Pulmonary Valvuloplasty

Transventricular pulmonary valvuloplasty of the pulmonary valve can be performed through a left fourth thoracotomy or sternotomy. The pericardium over the right outflow tract is opened vertically and sutured to the thoracotomy incision (Figure 20.1a). Locating the pericardial incision on the cranial aspect of the heart will help rotate the right outflow tract into the field when the pericardium is sutured to the incision. A buttressed mattress suture is placed in the right ventricular outflow tract and passed through a tourniquet to control bleeding during the procedure. A stab incision is made in the ventricle and an appropriate-sized valve dilator (e.g., Cooley pediatric valve dilator) is passed into the right ventricle and across the pulmonary valve (Figure 20.1b). The pulmonary valve is dilated several times in at least two planes. The appropriate amount of dilation is subjective and should be at least 1.5 times the estimated width of the valve annulus. A distinct popping sensation should be felt through the instrument as the valve leaflets rupture. The ventriculotomy is closed by tying the mattress suture. This surgical approach can also be used to perform a hybrid transventricular balloon valvuloplasty under fluoroscopic guidance.

Pulmonary Patch-Graft

Pulmonary patch-graft correction of PS by inflow occlusion is performed through a left fifth thoracotomy. Tourniquets are passed around the vena cavae and azygous vein for inflow occlusion as described in Chapter 18 (Figure 18.1b). A partial thickness incision is made in the right ventricular outflow tract (Figure 20.2a). An oval-shaped patch-graft is sutured to the ventriculotomy incision with pledget-reinforced mattress sutures (Figure 20.2b) and simple continuous suture patterns between. Our preferred patch material is 0.4 mm expanded polytetrafluoroethylene (ePTFE) vascular patch, although autogenous pericardium has been used. The patch-graft is then presutured to the pulmonary artery, leaving the sutures loose to allow access to the pulmonary artery for an incision across the outflow track (Figure 20.2c). The ends of the pulmonary artery sutures are passed through a Rommel tourniquet. After initiation of venous inflow occlusion, an incision is made in the pulmonary artery beneath the patch and extended full thickness across the pulmonary valve annulus into the previously made partial-thickness incision in the right ventricle (Figure 20.2 inset). Dysplastic pulmonary valve leaflets are incised or excised as necessary. The heart is de-aired, the patch-graft is closed by pulling the pulmonary artery sutures tight through the tourniquet, and inflow occlusion is discontinued. After circulation is restored, the pulmonary artery sutures are tightened to close the patch (Figure 20.2d). Any areas of leak around the patch-graft can be closed with pledget-reinforced mattress sutures.

Pulmonary patch-graft can also be performed with the aid of cardiopulmonary bypass. In this case, the preferred thoracic approach is median sternotomy. Venous cannulation can be bicaval or by a single two-stage cannula introduced via the right atrial appendage. After initiation of cardiopulmonary bypass, the patch-graft procedure can be performed on the beating heart without cardioplegia.

Pulmonary Valve Bypass

Pulmonary valve bypass consists of placement of a valved conduit from the right ventricle to the pulmonary artery (Figure 20.3). The surgery is performed

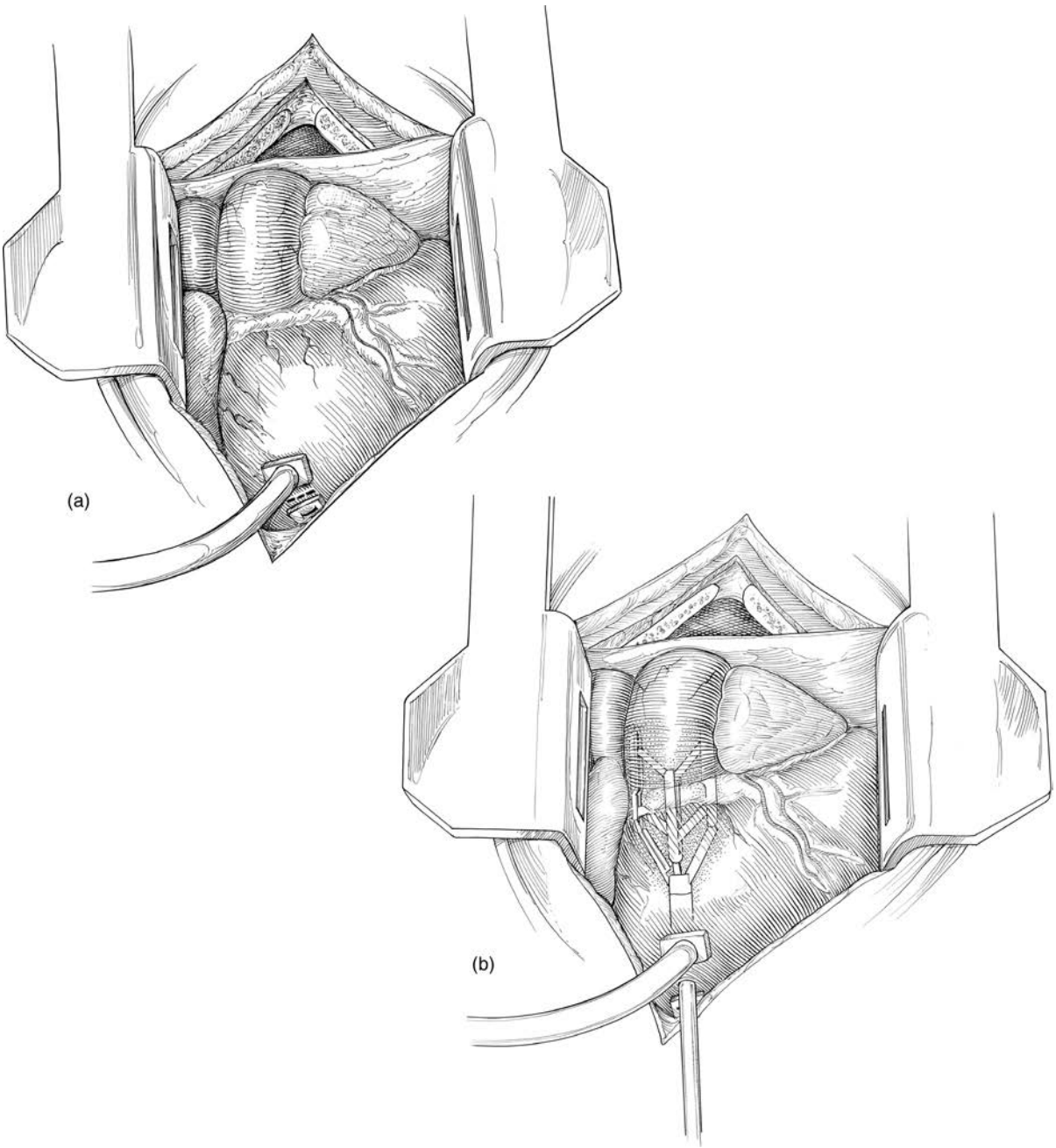

Figure 20.1 Transventricular Pulmonary Valvuloplasty

via left fourth thoracotomy. The surgical technique is as described for aortic valve bypass conduit described later in this chapter. The size of the valved conduit depends on the size of the patient. A 12 mm or 14 mm conduit and connector is appropriate for medium- to large-breed dogs. Dogs are placed on antiplatelet and/or anticoagulation therapy for at least 3 months after surgery.

Outcomes

Surgical correction of PS should be regarded as palliative rather than curative. The degree of palliation depends on the degree of obstruction relief achieved. Ideally, the systolic pressure gradient should be decreased to below 50 mm Hg. In this case, the risk for developing progressive heart failure should be

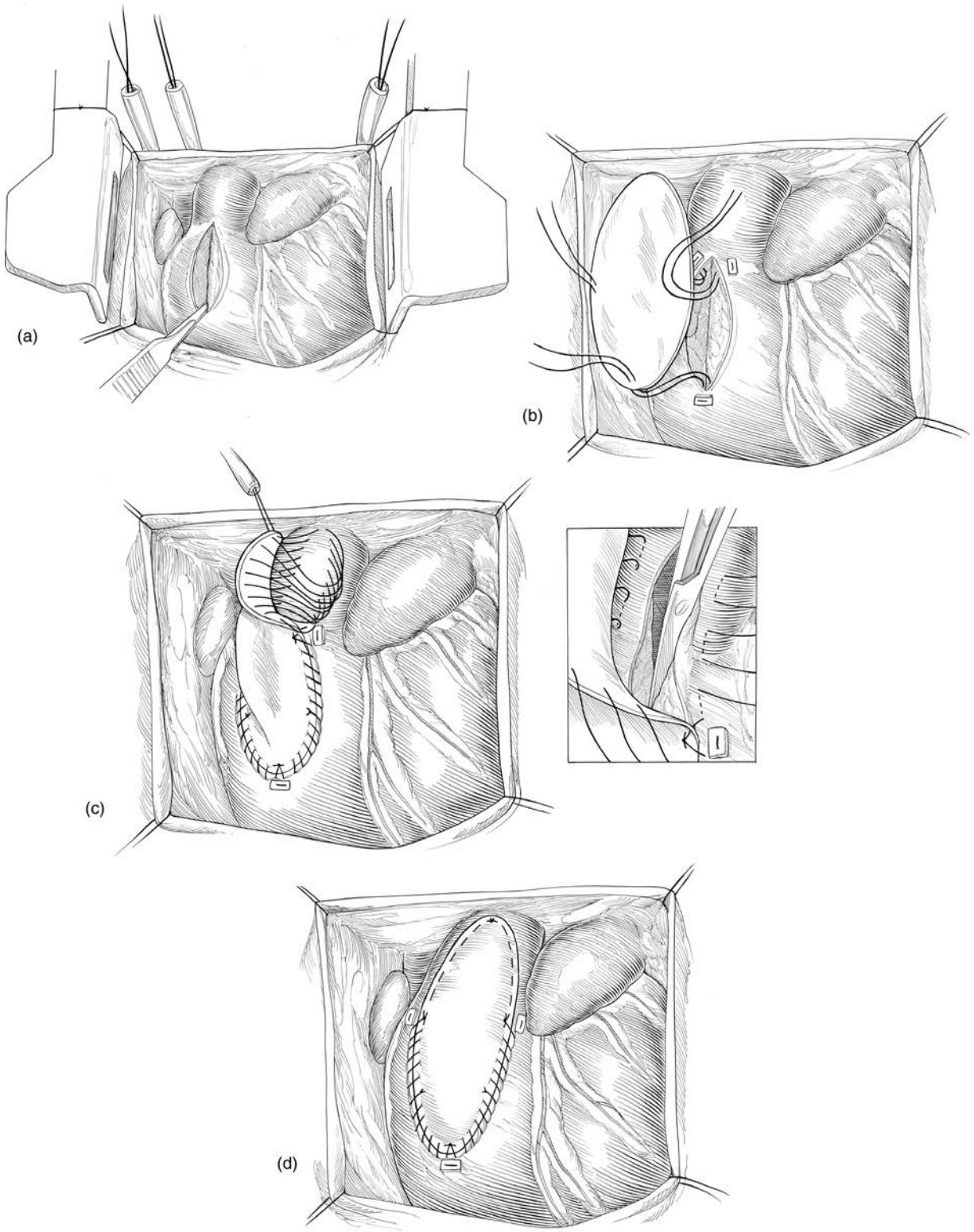

Figure 20.2 Pulmonary Patch-Graft

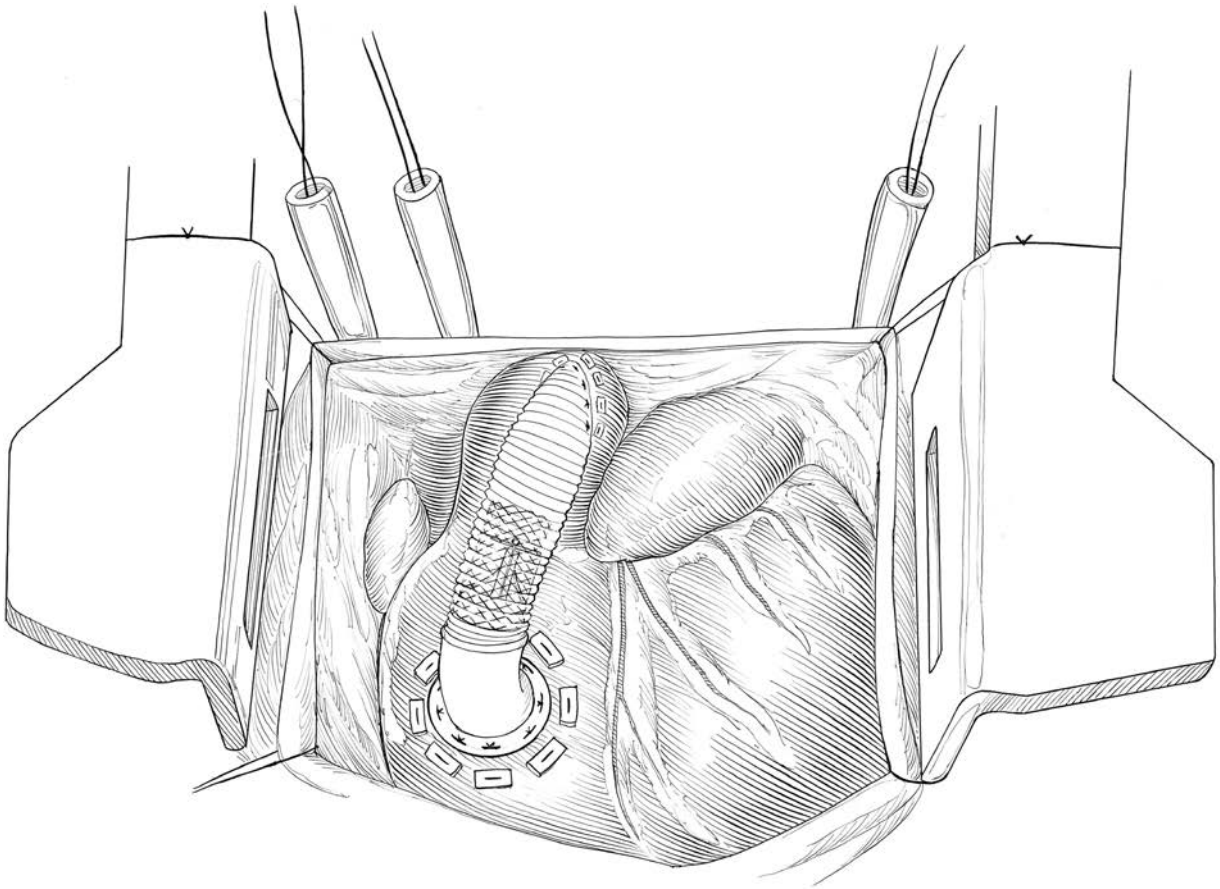

Figure 20.3 Pulmonary Valve Bypass

substantially diminished and exercise tolerance should be improved. The risk for sudden cardiac death likely diminishes, but not is eliminated by a successful surgical correction.

Several studies have documented decreased clinical signs and improved survival in dogs undergoing BV [5, 21, 22]. Results are better for dogs with type A PS morphology than for dogs with type B morphology. Less data are available for surgical dilation of the pulmonary valve, but presumably would carry similar results to BV. Outcomes for pulmonary patch-graft are limited to a few case reports [27, 28, 32, 33]. Pulmonary patch-graft clearly carries a higher operative risk than dilation valvuloplasty interventions regardless of the surgical technique employed. Long-term favorable outcomes in dogs with type B PS morphology have been achieved with PPG surgery. Effectiveness of pulmonary valve bypass conduit surgery has been demonstrated in an experimental canine model [34], but clinical effectiveness has not been documented in dogs.

Subvalvular Aortic Stenosis

Subvalvular aortic stenosis (SAS) is an important congenital defect in dogs accounting for 15% to 25% of congenital defects, depending on the study population [9–11]. Breeds of dogs predisposed to SAS include Newfoundland, German shepherd, boxer, golden retriever, and Rottweiler [9]. It is not uncommon for dogs with SAS to have concurrent valvular PS, especially boxers [35]. Congenital valvular and supravalvular aortic stenosis are reported in dogs and cats, but are less common [10, 36]. The most common cause of acquired aortic stenosis in dogs is valvular endocarditis [37]. There is anecdotal evidence that SAS predisposes dogs to valvular endocarditis [37,38].

The typical morphology for SAS is a discrete fibrous membrane or ring just below the aortic valve. The fibrous membrane originates from the ventricular septum and reflects onto the septal leaflet of the mitral valve sometimes restricting mitral valve closure. Hypertrophy of the ventricular septum can

contribute to the fixed stenosis and/or add a dynamic component to the obstruction. The most advanced morphologic form of the defect manifests as a fixed tunnel-like obstruction of the left ventricular outflow track. It is not uncommon for SAS to be associated with varying degrees of mitral valve dysplasia characterized by restrictive motion of the septal mitral leaflet. The resultant mitral regurgitation increases the likelihood of congestive heart failure and usually accelerates the clinical course of SAS.

Pathophysiology

Aortic stenosis causes pressure overload on the left ventricle. The left ventricle responds through parallel replication of sarcomeres leading to concentric hypertrophy, which serves to normalize systolic afterload through wall thickening and decreased ventricular chamber radius. There is evidence that development of the coronary circulation fails to match the degree of cardiac hypertrophy. Additionally, dogs with SAS have pathologic change within the intramural coronary arteries characterized by intimal and medial hyperplasia [39]. These pathologic changes, combined with elevated pressures within the ventricular wall, cause mismatch between myocardial oxygen demand and delivery, particularly in the subendocardial region of the ventricular wall during systole [40, 41]. The resultant chronic myocardial ischemia leads to fibrosis of the subendocardium and predisposes dogs to ventricular dysrhythmias and sudden cardiac death [38,42,43]. Dogs that avoid sudden cardiac death early in life often develop congestive heart failure later in life. Mitral regurgitation from concurrent mitral valve dysplasia greatly increases the likelihood of developing congestive heart failure as an early manifestation and usually is associated with a rapid clinical course despite medical therapy.

Diagnosis

Presenting history for dogs with SAS is often unremarkable. Historical findings can include activity or exercise intolerance, syncope or near-syncope, and signs related to left-sided congestive heart failure. For some dogs, the first recognized clinical sign is sudden cardiac arrest. Characteristic physical findings are systolic ejection murmur at the left cardiac base and notably weak arterial pulses. The systolic murmur radiates up the carotid arteries. The degree of obstruction associated with SAS is progressive through maturity sometimes causing the systolic murmur to be absent or soft in the first 6 to 12 weeks after birth.

Diagnosis of SAS is confirmed by echocardiography. Left ventricular concentric wall thickening is present depending on severity. A discrete subvalvular membrane or ring can usually be visualized on 2-D echocardiography. Severity is assessed based on the spectral Doppler-derived peak systolic aortic velocity and calculation of the of the instantaneous pressure gradient from the modified Bernoulli equation. Pressure gradients of 80 mm Hg or greater are indicative of severe SAS. Because aortic pressure gradients are flow dependent, they can underestimate the severity of SAS if left ventricular systolic function is compromised. In human patients severity of aortic stenosis is based on determination of the effective orifice area (EOA) of the stenotic valve, either from cardiac catheterization or echocardiography [44]. Doppler-derived EOA indexed to body surface area for assessment of SAS severity has been advocated for dogs [45], but has not yet been widely adopted.

Indications for Surgery

Doppler-derived aortic pressure gradients of 80 mm Hg or greater are generally regarded as indicative of severe SAS. Several studies have documented the risk of sudden cardiac death in dogs with SAS [38, 42, 43, 46]. Interventions for SAS in dogs with SAS include lifetime beta blocker therapy [46], transarterial balloon valvuloplasty [43], transapical aortic valve dilation [47], open resection under cardiopulmonary bypass [48, 49], and aortic valve bypass (left ventricular apico-aortic) conduit [50]. To date, no therapy or intervention has been shown to decrease risk of sudden cardiac death in dogs with SAS [42, 43, 46]. Thus, the most appropriate rationale for intervention is to improve clinical signs related to syncope, significant activity/exercise intolerance or congestive heart failure, rather than to prevent sudden cardiac death. Interventions for SAS should be considered palliative.

Transapical Aortic Valve Dilation

Transapical aortic valve dilation can be performed from a sternotomy approach without image guidance. Position of the valve dilator is confirmed by direct palpation of the ascending aorta. Alternatively, the procedure can be performed via a minimal-incision left thoracotomy (Figure 6.1) under transesophageal echocardiographic (TEE) and/or fluoroscopic image guidance. A pledget-reinforced mattress suture is placed in the left ventricular apex and the suture ends are passed through a tourniquet to control bleeding during the procedure (Figure 20.4). A small stab incision is made and an appropriate sized valve dilator is

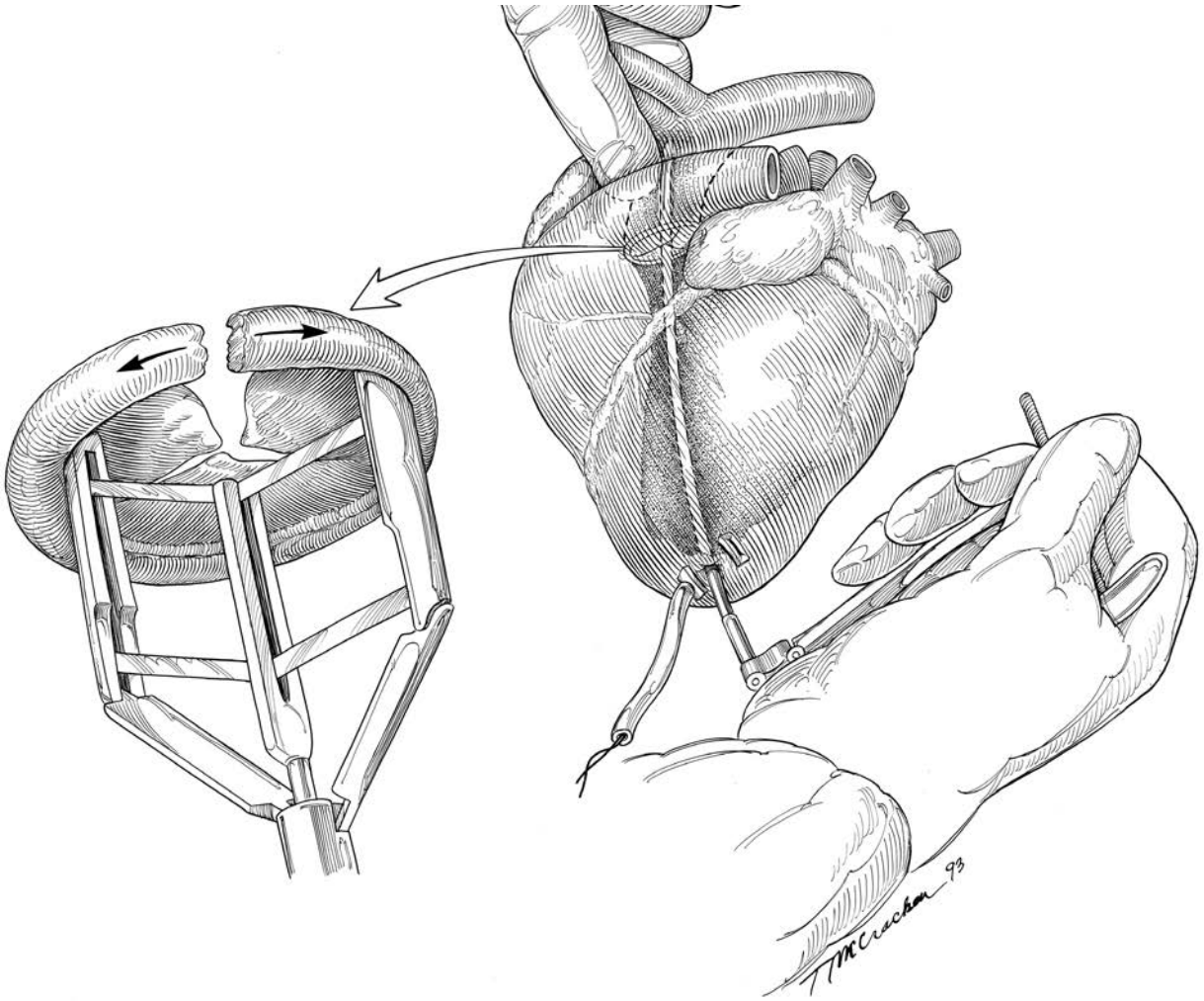

Figure 20.4 Transapical Aortic Valve Dilation

passed into left ventricle and across the aortic valve. Position of the valve dilator is confirmed by palpation of the tip of the valve dilator through the wall of the ascending aorta. Alternatively, the position of the valve dilator can be confirmed by TEE or fluoroscopy. The stenosis is dilated in several planes. The width of the dilator should be at least 1.5 times the measured diameter of the stenosis.

Aortic Valve Bypass (Apico-Aortic) Conduit

Aortic valve bypass (AVB) or left apico-aortic conduit is accomplished by placement of a valved conduit between the left ventricular apex and aorta. The valve can be a bioprosthesis or an aortic allograft. Connection to the left ventricle is accomplished with a rigid connector to prevent collapse of the ventriculotomy site during systole. The conduit can be ePTFE or low-porosity Dacron. Dacron vascular conduits must be preclotted with autologous blood prior to implantation. A 12 mm or 14 mm diameter conduit is appropriate for large- or giant-breed dogs. The main advantage of AVB is that flow through the conduit is entirely additive to native flow through the left outflow track and native valve flow is not diminished, should the conduit fail. AVB can be performed on the beating heart with the aid of brief inflow occlusion.

The bypass conduit can be connected to either the ascending aorta via sternotomy approach or descending aorta via a left thoracotomy. We prefer connection to the ascending aorta because it is more physiologic and the anastomosis is technically easier to perform. After the thoracic approach is complete, the patient is heparinized with 150 U/kg IV bolus. The activated clotting time is kept above 280 seconds during the surgery. Tourniquets are preplaced on the cranial and caudal vena cavae and azygous vein for inflow occlusion (Figure 20.5a). The ascending aorta

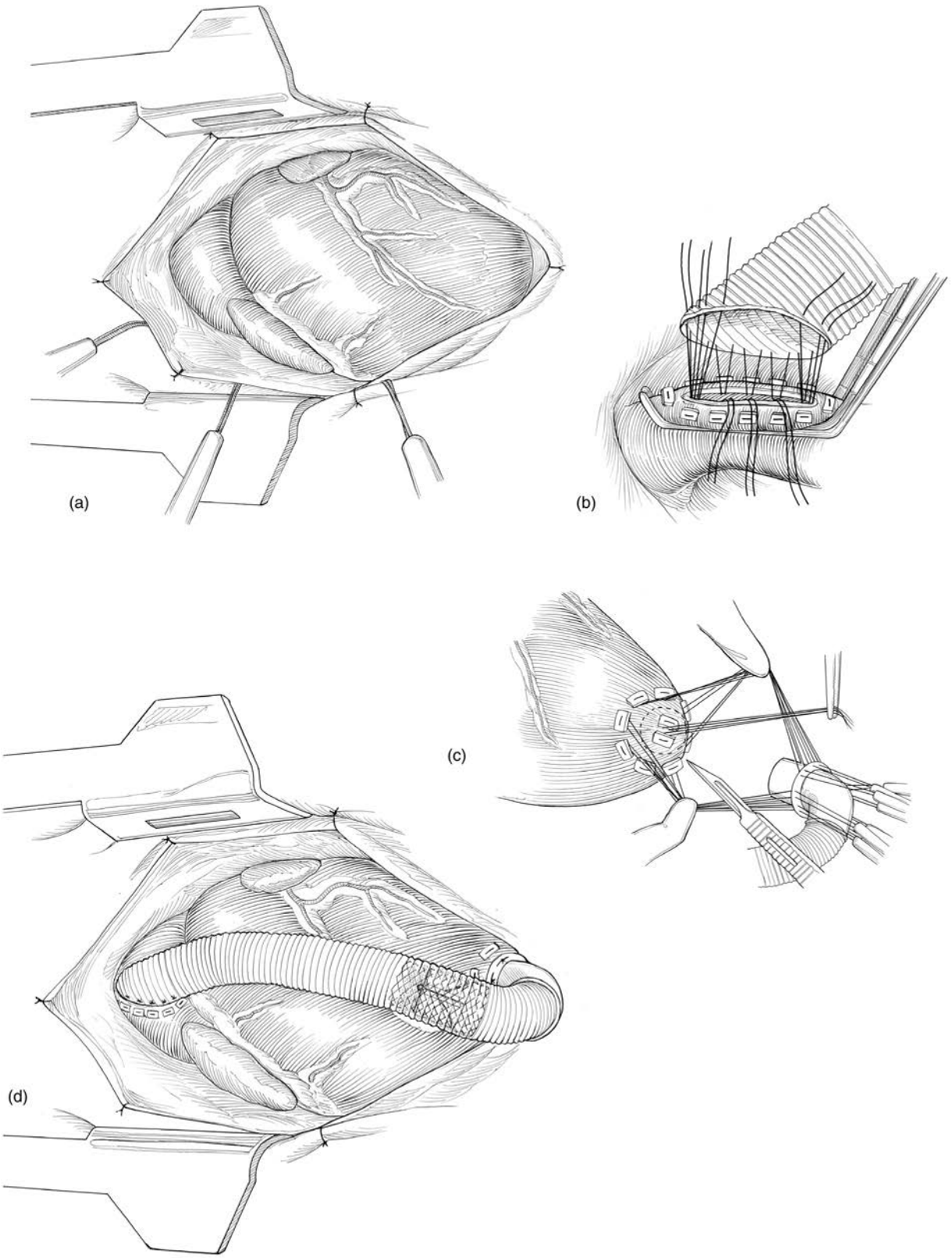

Figure 20.5 Aortic Valve Bypass

is prepared for anastomosis by dissection of loose adventitial tissues and placement of a tangential vascular forceps (Figure 20.5b). The aorta is incised and pledget-reinforced mattress sutures of 4-0 or 5-0 polypropylene are preplaced around the incision. The mattress sutures placed in the conduit and then tied to complete the anastomosis. A straight vascular forceps is placed on the conduit. The aortic clamp is removed and the anastomosis is checked for bleeding. Eight deep-biting mattress sutures of 2-0 polyester are preplaced around the planned ventriculotomy site (Figure 20.5c). The mattress sutures are passed through the sewing ring of the connector and then through tourniquets. Two mattress sutures are placed in the ventricular apex to control the muscular plug during incision. Inflow occlusion is initiated and circular incision is made in the ventricular apex with a #11 scalpel blade or a sharp circular coring knife. The ventricular plug is removed and the connector is placed into the ventricular apex. The heart is de-aired during insertion of the connector by release of one of the venous inflow tourniquets. The connector is temporally secured by tightening tourniquets on the connector mattress sutures. The conduit is de-aired with an 18 g hypodermic needed, and the straight vascular forceps on the conduit are removed to establish flow through the conduit. The procedure is completed by tying mattress sutures to secure the connector (Figure 20.5d). The patient should be placed on anticoagulant therapy for 3 months after surgery and long-term anti-platelet therapy as described for mitral or tricuspid valve replacement (see Chapter 21).

Outcomes

Transapical aortic valve dilation decreases intraoperative peak-to-peak transaortic pressure gradients in dogs with SAS by about 80%; however, these decreases are not sustained by 3 to 6 months post-procedure [47]. Thus, transapical aortic valve dilation should not be regarded as a definitive treatment, but rather as a bridge to more definitive surgery especially in immature dogs. Similar results have been reported for dogs with severe SAS undergoing transarterial balloon valvuloplasty [43].

Open resection of SAS in dogs results in sustained decreases in transaortic pressure gradients for periods of 12 months or longer after surgery and improved exercise tolerance [48]; however, this surgery does not significantly decrease risk of sudden cardiac death [42]. For this reason, we no longer perform this surgery.

Hemodynamic benefit and positive clinical outcomes have been reported in high-risk human patients undergoing off-pump aortic valve bypass surgery for aortic stenosis [50, 51]. Other than an early case series [52], limited clinical outcome data are available for aortic valve bypass conduits in dogs with SAS. Effectiveness of AVB has been demonstrated for up to 6 months in a canine experimental model of aortic stenosis [53]. Off-pump aortic valve bypass deserves further exploration as a treatment for dogs with symptomatic severe SAS.

Aortic Insufficiency

Aortic insufficiency (AI) is a relatively uncommon structural heart condition in animals; however, its consequences tend to be devastating compared to other valvular heart conditions. Primary AI is an unusual congenital valve defect. More often, AI is associated with other congenital defects such as ventricular septal defect or subvalvular aortic stenosis. The most common acquired cause of severe AI in dogs is endocarditis [37, 54]. Significant AI causes a severe and often overwhelming volume overload of the left ventricle compared to an equal regurgitant volume of mitral regurgitation. This is because the regurgitant volume must be pumped against a higher aortic pressures, similar to patent ductus arteriosus. The result is congestive heart failure that tends to have a rapid clinical course. The most important physical findings are a soft blowing-type diastolic murmur at the left heart base and notability hyperkinetic pulses caused by rapid fall in diastolic pressures. Concurrent aortic stenosis causes left base "to-and-fro" type systolic and diastolic murmur that can be confused with a continuous murmur. Diagnosis is confirmed by 2-D and Doppler echocardiography. Severity of AI is based on spectral and color-flow Doppler interrogation of the aortic valve, the magnitude of left ventricular dilation, and the pressure half-time of regurgitant aortic flow.

Surgical or interventional options for severe AI have historically been unavailable for animals; thus, there is limited published experience for treatment of AI. Open aortic valve replacement is theoretically possible in a larger dog, but there is no reported experience in dogs. A potential palliative option for severe AI in dogs is heterotopic valve implantation in the descending aorta. The advantage of this approach is that it can be performed on the beating heart without the need for cardiopulmonary bypass and there is no theoretical limitation on patient size. The procedure was first performed in a human patient in 1953 prior to the advent of cardiopulmonary bypass [55], and its utility has since been reaffirmed as an option for human patients that cannot undergo orthotopic aortic valve

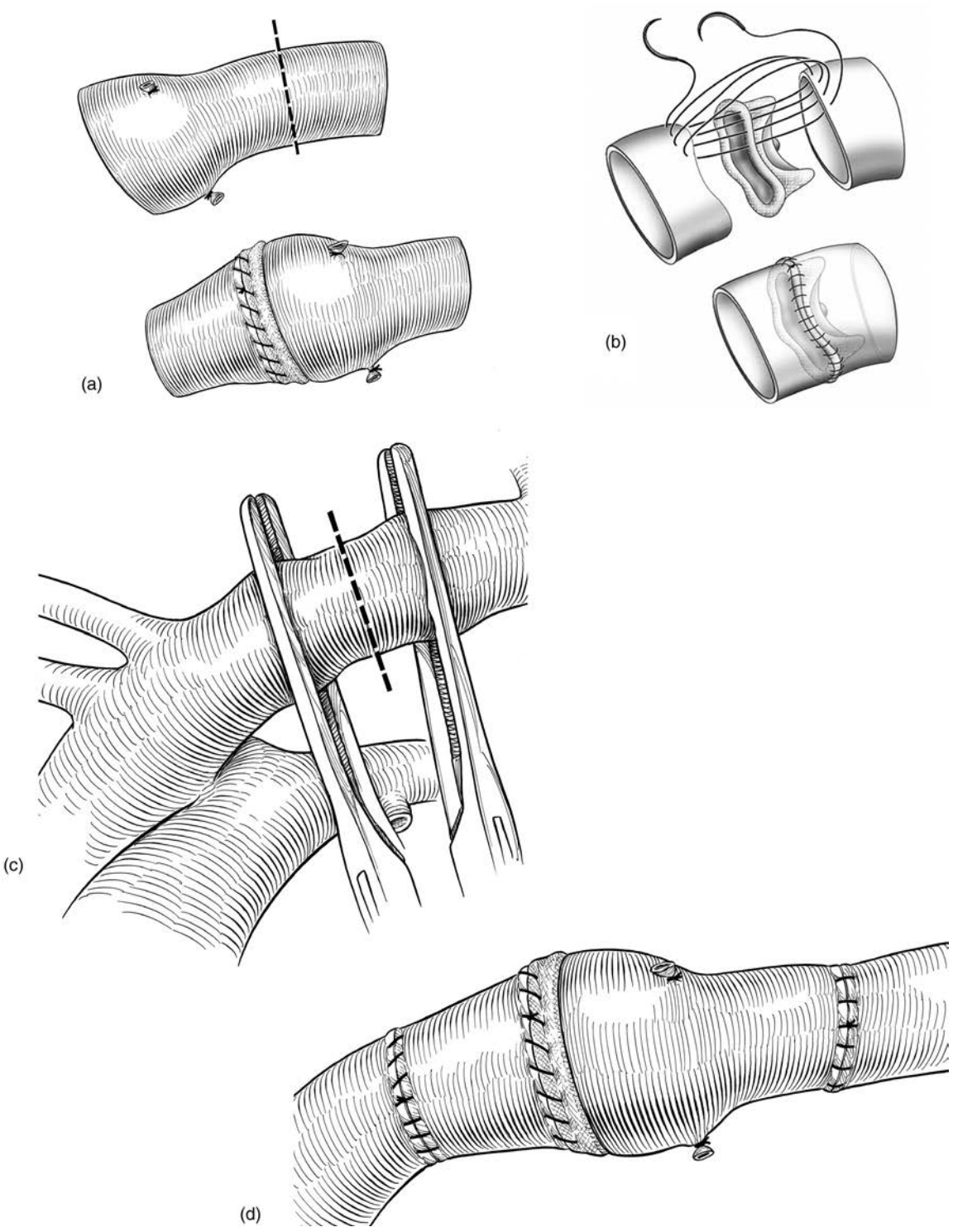

Figure 20.6 Heterotopic Aortic Valve Implantation

replacement [56]. We have reported significant palliation in a dog with to severe AI secondary to endocarditis undergoing this surgery [57].

Heterotopic Aortic Valve Implantation

The diameter of the descending aorta just distal to the aortic arch is measured by echocardiography or CT angiography. Options for heterotopic aortic valve implantation (AVI) include an aortic allograft conduit or an ePTFE conduit with a porcine bioprosthesis. Clinical experience with canine aortic allographs is limited, but their effectiveness has been documented experimentally in dogs [51]. An appropriate-sized cadaveric aortic allograft is harvested, including the septal mitral valve leaflet and muscular shelf that forms the base of the allograft. The mitral leaflet and muscular shelf are trimmed, and a portion of the aorta is transferred and sewn to the base of the allograft to form a conduit (Figure 20.6a). The anastomosis at the base of the allograft should be reinforced with a stripe of surgical felt. The coronary ostia are closed by ligation or pledget-reinforced mattress sutures. The allograft is either fixed with glutaraldehyde [51] or put through a sodium dodecyl sulfate-based treatment protocol prior to implantation to reduce immunogenicity of the graft. Alternatively, a valved conduit can be fashioned from an appropriate-sized ePTFE conduit and a porcine bioprosthesis (Figure 20.6b). Because of limitations on available sizes for commercial aortic bioprostheses, this option is limited to larger dogs with aortic diameters of 18 mm or greater.

AVI is performed via a left fifth intercostal. The proximal descending aorta is isolated outside of the pericardium. Controlling tapes are passed around the aorta to facilitate crossclamp placement. The anastomotic site is cleaned of loose adventitial tissue. Intravenous heparin (150 U/kg) is administered. Vascular clamps are placed proximal and distal to the anastomotic site and the aorta is divided (Figure 20.6c). The conduit is inserted between the divided ends of the aorta with simple continuous suture patterns of 4-0 polypropylene or PTFE suture (Figure 20.6d). Crossclamp time must be kept under 16 minutes to avoid injury to the spinal cord and abdominal organs. Alternatively, an arterial site proximal and distal to the surgery site can be cannulated and a temporary bypass circuit set up to provide distal flow during the aortic crossclamp and anastomoses.

References

1 Minors SL, O'Grady MR, Williams RM, and O'Sullivan ML. 2006. Clinical and echocardiographic features of primary infundibular stenosis with intact ventricular septum in dogs. *Journal of Veterinary Internal Medicine.* 20(6): 1344–50.

2 Schrope DP. 2008. Primary pulmonic infundibular stenosis in 12 cats: Natural history and the effects of balloon valvuloplasty. *Journal of Veterinary Cardiology.* 10(1):33–43.

3 Aprea F, Clark L, Whitley NT, and Oliveira P. 2014. Presumed "suicidal right ventricle" in a Cocker Spaniel dog undergoing pulmonary valve balloon dilation and its prevention in an analogous case. *Veterinary Anaesthesia and Analgesia.* 41(4): 438–9.

4 Rishniw M, and Thomas WP. 2002. Dynamic right ventricular outflow obstruction: a new cause of systolic murmurs in cats. *Journal of Veterinary Internal Medicine.* 16(5):547–52.

5 Bussadori C, DeMadron E, Santilli RA, and Borgarelli M. 2001. Balloon valvuloplasty in 30 dogs with pulmonic stenosis: effect of valve morphology and annular size on initial and 1-year outcome. *Journal of Veterinary Internal Medicine.* 15(6): 553–8.

6 Fingland RB, Bonagura JD, and Myer CW. 1986. Pulmonic stenosis in the dog: 29 cases (1975–1984). *Journal of the American Veterinary Medical Association.* 189(2):218–26.

7 Schrope DP. 2005. Balloon valvuloplasty of valvular pulmonic stenosis in the dog. *Clinical Techniques in Small Animal Practice.* 20(3):182–95.

8 Patterson DF, Haskins ME, and Schnarr WR. 1981. Hereditary dysplasia of the pulmonary valve in beagle dogs. Pathologic and genetic studies. *The American Journal of Cardiology.* 47(3):631–41.

9 Buchanan JW. 1999. Pravalence of cardiovascular disorders, In: Fox PR, Sisson D, and Moise NS eds. *Textbook of Canine and Feline Cardiology*. Pp. 457–70. Philadelphia: W.B. Saunders Co.

10 Schrope DP. 2015. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. *Journal of Veterinary Cardiology.* 17(3):192–202.

11 Oliveira P, Domenech O, Silva J, Vannini S, Bussadori R, and Bussadori C. 2011. Retrospective review of congenital heart disease in 976 dogs.

Journal of Veterinary Internal Medicine. 25(3): 477–83.
12 Buchanan JW. 1990. Pulmonic stenosis caused by single coronary artery in dogs: four cases (1965-1984). *Journal of the American Veterinary Medical Association.* 196(1):115–20.
13 Soda A, Tanaka R, Saida Y, and Yamane Y. 2009. Successful surgical correction of supravalvular pulmonary stenosis under beating heart using a cardiopulmonary bypass system in a dog. *Journal of Veterinary Medical Science.* 71(2):203–6.
14 Griffiths LG, Bright JM, and Chan KC. 2006. Transcatheter intravascular stent placement to relieve supravalvular pulmonic stenosis. *Journal of Veterinary Cardiology.* 8(2):145–55.
15 Treseder JR, and Jung S. 2016. Balloon dilation of congenital supravalvular pulmonic stenosis in a dog. *Journal of Veterinary Science*.
16 Schrope DP, and Kelch WJ. 2007. Clinical and echocardiographic findings of pulmonary artery stenosis in seven cats. *Journal of Veterinary Cardiology.* 9(2):83–9.
17 Francis AJ, Johnson MJ, Culshaw GC, Corcoran BM, Martin MW, and French AT. 2011. Outcome in 55 dogs with pulmonic stenosis that did not undergo balloon valvuloplasty or surgery. *The Journal of Small Animal Practice.* 52(6):282–8.
18 Fujii Y, Nishimoto Y, Sunahara H, Takano H, and Aoki T. 2012. Prevalence of patent foramen ovale with right-to-left shunting in dogs with pulmonic stenosis. *Journal of Veterinary Internal Medicine.* 26(1):183–5.
19 Johnson MS, and Martin M. 2004. Results of balloon valvuloplasty in 40 dogs with pulmonic stenosis. *The Journal of Small Animal Practice.* 45(3):148–53.
20 Dennis S. 2012. Evidence for increased probability of cardiac death in dogs with pulmonic stenosis questionable. *The Journal of Small Animal Practice.* 53(5):304–6; author reply 6–9.
21 Locatelli C, Spalla I, Domenech O, Sala E, Brambilla PG, and Bussadori C. 2013. Pulmonic stenosis in dogs: survival and risk factors in a retrospective cohort of patients. *The Journal of Small Animal Practice.* 54(9):445–52.
22 Johnson MS, Martin M, Edwards D, French A, and Henley W. 2004. Pulmonic stenosis in dogs: balloon dilation improves clinical outcome. *Journal of Veterinary Internal Medicine.* 18(5):656–62.
23 Bussadori C, Domenech O, Longo A, Pradelli D, and Bussadori R. 2002. Percutaneous catheter-based treatment of pulmonic stenosis and patent ductus arteriosus in a dog. *Journal of Veterinary Cardiology.* 4(2):29–34.
24 Ristic JM, Marin CJ, Baines EA, and Herrtage ME. 2001. Congenital Pulmonic Stenosis a Retrospective study of 24 cases seen between 1990-1999. *Journal of Veterinary Cardiology.* 3(2):13–9.
25 Brock RC. 1948. Pulmonary valvulotomy for the relief of congenital pulmonary stenosis; report of three cases. *British Medical Journal.* 1(4562):1121–6.
26 Ripps JH, and Henderson AR. 1953. Congenital pulmonic valvular stenosis in a dog; report of a case successfully treated surgically. *Journal of the American Veterinary Medical Association.* 123(919): 292–6.
27 Orton EC, Bruecker KA, and McCracken TO. 1990. An open patch-graft technique for correction of pulmonic stenosis in the dog. *Veterinary Surgery.* 19(2):148–54.
28 Breznock EM, and Wood GL. 1976. A patch-graft technique for correction of pulmonic stenosis in dogs. *Journal of the American Veterinary Medical Association.* 169(10):1090–4.
29 Hunt GB, Pearson MR, Bellenger CR, and Malik R. 1993. Use of a modified open patch-graft technique and valvulectomy for correction of severe pulmonic stenosis in dogs: eight consecutive cases. *Australian Veterinary Journal.* 70(7):244–8.
30 Krahwinkel DJ, and Sackman JE. 1998 In: Bojrab MJ ed. *Current Techniques in Small Animal Surgery*, pp. 662–71. Baltimore: Williams $ Wilkins.
31 Shores A, and Weirich WE. 1985. A modified pericardial patch graft technique for correction of pulmonic stenosis in the dog. *Journal of the American Animal Hospital Association.* 21: 809–12.
32 Tanaka R, Shimizu M, Hoshi K, Soda A, Saida Y, Takashima K, and Yamane Y. 2009. Efficacy of open patch-grafting under cardiopulmonary bypass for pulmonic stenosis in small dogs. *Australian Veterinary Journal.* 87(3):88–93.
33 Staudte KL, Gibson NR, Read RA, and Edwards GA. 2004. Evaluation of closed pericardial patch grafting for management of severe pulmonic stenosis. *Australian Veterinary Journal.* 82(1–2):33–7.
34 Saida Y, Tanaka R, Fukushima R, Hira S, Hoshi K, Soda A, Iizuka T, Ishikawa T, Nishimura T, and Yamane Y. 2009 Histological study of right ventricle-pulmonary artery valved conduit implantation (RPVC) in dogs with pulmonic stenosis. *The Journal of Veterinary Medical Science.* 71(4):409–15.
35 Kander M, Paslawska U, Staszczyk M, Cepiel A, Paslawski R, Mazur G, and Noszczyk-Nowak A. 2015. Retrospective analysis of co-occurrence of congenital aortic stenosis and pulmonary artery stenosis in dogs. *Pol J Vet Sci.* 18(4):841–5.

36 Hollmer M, Willesen JL, Jensen AT, and Koch J. 2008. Aortic stenosis in the Dogue de Bordeaux. *The Journal of Small Animal Practice.* 49(9):432–7.

37 Sisson D, and Thomas WP. 1984. Endocarditis of the aortic valve in the dog. *Journal of the American Veterinary Medical Association.* 184(5):570–7.

38 Kienle RD, Thomas WP, and Pion PD. 1994. The natural clinical history of canine congenital subaortic stenosis. *Journal of Veterinary Internal Medicine.* 8(6):423–31.

39 Flickinger GL, and Patterson DF. 1967. Coronary lesions associated with congenital subaortic stenosis in the dog. *Journal of Pathology Bacteriology.* 93(1): 133–40.

40 Pyle RL, Lowensohn HS, Khouri EM, Gregg DE, and Patterson DF. 1973. Left circumflex coronary artery hemodynamics in conscious dogs with congenital subaortic stenosis. *Circulation Research.* 33(1):34–8.

41 Borkon AM, Jones M, Bell JH, and Pierce JE. 1982. Regional myocardial blood flow in left ventricular hypertrophy. An experimental investigation in Newfoundland dogs with congenital subaortic stenosis. *Journal of Thoracic Cardiovascular Surgery.* 84(6):876–85.

42 Orton EC, Herndon GD, Boon JA, Gaynor JS, Hackett TB, and Monnet E. 2000. Influence of open surgical correction on intermediate-term outcome in dogs with subvalvular aortic stenosis: 44 cases (1991–1998). *Journal of the American Veterinary Medical Association.* 216(3):364–7.

43 Meurs KM, Lehmkuhl LB, and Bonagura JD. 2005. Survival times in dogs with severe subvalvular aortic stenosis treated with balloon valvuloplasty or atenolol. *Journal of the American Veterinary Medical Association.* 227(3):420–4.

44 Carabello BA, and Grossman W. 2014 In: Moscucci M ed. *Grossman & Baim's Cardiac Catheterization, Angiography, and Intervention*, pp. 272–83. Wolters Kluwer / Lippincott Willaims & Wilkins.

45 Belanger MC, Di Fruscia R, Dumesnil JG, and Pibarot P. 2001. Usefulness of the indexed effective orifice area in the assessment of subaortic stenosis in the dog. *Journal of Veterinary Internal Medicine.* 15(5):430–7.

46 Eason BD, Fine DM, Leeder D, Stauthammer C, Lamb K, and Tobias AH. 2014. Influence of beta blockers on survival in dogs with severe subaortic stenosis. *Journal of Veterinary Internal Medicine.* 28(3):857–62.

47 Linn K, and Orton EC. 1992. Closed transventricular dilation of discrete subvalvular aortic stenosis in dogs. *Veterinary Surgery.* 21(6):441–5.

48 Monnet E, Orton EC, Gaynor JS, Boon J, Wagner A, Linn K, Eddleman LA, and Brevard S. 1996. Open resection for subvalvular aortic stenosis in dogs. *Journal of the American Veterinary Medical Association.* 209(7):1255–61.

49 Komtebedde J, Ilkiw JE, Follette DM, Breznock EM, and Tobias AH 1993. Resection of subvalvular aortic stenosis. Surgical and perioperative management in seven dogs. *Veterinary Surgery*. 22(6):419–30.

50 Thourani VH, Keeling WB, Guyton RA, Dara A, Hurst SD, and Lattouf OM. 2001. Outcomes of off-pump aortic valve bypass surgery for the relief of aortic stenosis in adults. *The Annals of Thoracic Surgery.* 91(1):131–6.

51 Vliek CJ, Balaras E, Li S, Lin JY, Young CA, DeFilippi CR, Griffith BP, and Gammie JS. 2010. Early and midterm hemodynamics after aortic valve bypass (apicoaortic conduit) surgery. *The Annals of Thoracic Surgery.* 90(1):136–43.

52 Breznock EM, Whiting P, Pendray D, Thomas B, Strack D, Bauer T, Koblik P, Hornof W, and Ludders JD. 1983. Valved apico-aortic conduit for relief of left ventricular hypertension caused by discrete subaortic stenosis in dogs. *Journal of the American Veterinary Medical Association.* 182(1):51–6.

53 Hirao H, Inoue T, Hoshi K, Kobayashi M, Shimamura S, Shimizu M, Tanaka R, Takashima K, Mori Y, Noishiki Y, et al. 2005. An experimental study of apico-aortic valved conduit (AAVC) for surgical treatment of aortic stenosis in dogs. *The Journal of Veterinary Medical Science.* 67(4):357–62.

54 Calvert CA. 1982. Valvular bacterial endocarditis in the dog. *Journal of the American Veterinary Medical Association.* 180(9):1080–4.

55 Hufnagel CA, and Harvey WP. 1953. The surgical correction of aortic regurgitation preliminary report. *Bull Georgetown Univ Med Cent.* 6(3):60–1.

56 Cale AR, Sang CT, Campanella C, and Cameron EW. 1993. Hufnagel revisited: a descending thoracic aortic valve to treat prosthetic valve insufficiency. *The Annals of Thoracic Surgery.* 55(5):1218–21.

57 Arai S, Wright BD, Miyake Y, Boon JA, and Orton EC. 2007. Heterotopic implantation of a porcine bioprosthetic heart valve in a dog with aortic valve endocarditis. *Journal of the American Veterinary Medical Association.* 231(5):727–30.

21

Tricuspid and Mitral Valves

E. Christopher Orton

The tricuspid and mitral valves comprise the atrioventricular valves that allow forward filling of the ventricles during diastole and prevent backward flow into the atria during systole in the right and left heart, respectively. Anatomically both valves are bileaflet valves in small animals supported by chordae tendineae and papillary muscles. Each valve has a septal leaflet and a mural (parietal) leaflet. Disorders of these valves manifest functionally as valvular regurgitation or valvular stenosis, or both. Valve regurgitation is classified functionally as resulting from annular dilation (type I defect), leaflet prolapse (type II defect), or restrictive leaflet motion (type III defect) [1].

Disorders of the tricuspid and mitral valve in dogs and cats include congenital valve dysplasia, acquired valve degeneration, and functional regurgitation. The most common functional abnormality of congenital valve dysplasia is valvular regurgitation secondary to restrictive leaflet motion. The primary mechanism of regurgitation in degenerative valve disease is leaflet prolapse. In both congenital dysplasia and degenerative valve disease, secondary dilation of the valve annulus contributes to regurgitation as the ventricle dilates. Congenital stenosis of the tricuspid and mitral valve is rare and usually caused by commissural fusion of the leaflets. Functional (secondary) tricuspid and mitral regurgitation results from primary cardiomyopathies or severe acute pressure overload of the ventricle such as acute cor pulmonale. Each of these conditions present unique challenges for medical and surgical intervention in small animals.

Congenital Tricuspid Valve Dysplasia

Tricuspid valve dysplasia (TVD) is a congenital malformation of the tricuspid valve that occurs in dogs and cats. The defect accounts for approximately 5% of congenital cardiac malformations in both species [2, 3]. Labrador retrievers, golden retrievers, German shepherds, and other large breeds of dog have a predilection for TVD [3]. A spectrum of pathologic malformations of the leaflets, chordae, and papillary muscles have been described [4]. The typical morphology consists of direct insertion of the mural leaflet to elongated or malformed papillary muscles and complete fusion or tethering of the septal leaflet to ventricular septum by shortened or absent chordae. These morphologic changes combine to severely restrict the closing motion of the valve leaflets and allow wide-open tricuspid regurgitation (TR). The resultant volume overload causes progressive severe right heart dilation, which, in turn, dilates the valve annulus, further worsening the TR. Atrial fibrillation is a common late sequela. Most dogs with severe TR develop progressive right-sided congestive heart failure within the first 3 years of life. Tricuspid stenosis, with or without TR, is a less common manifestation of TVD and is usually caused by commissural fusion of the valve leaflets.

Diagnosis

Findings at presentation range from asymptomatic to severe activity intolerance with abdominal distension. Physical findings include a right apical systolic murmur, jugular pulsations, and signs referable to right heart failure including ascites, systemic venous distension, and peripheral edema. Thoracic radiographs reveal moderate to extremely severe cardiomegaly. Tall P waves and "splintered" QRS complexes are typical findings on the ECG. Sinus tachycardia or atrial fibrillation are common rhythm disturbances. Echocardiography confirms the diagnosis by demonstrating typical malformations and restrictive motion of the valve apparatus. Moderate to severe dilation of the right atrium and ventricle are typical. Spectral and color-flow Doppler echocardiography confirm severe

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tricuspid regurgitation. Calculation of the transvalvular pressure gradient from the peak regurgitant velocity tends to overestimate the gradient because the assumptions of the modified Bernoulli equation are not held. As a result Doppler-based estimates of right ventricular systolic pressure are not reliable in this condition. Presence of concurrent tricuspid stenosis is identified by calculation of pressure half-times from the tricuspid inflow velocity profile [5].

Indications

Surgical options for TVD in dogs are tricuspid valve replacement or valve repair. Surgical intervention is based on the degree of right heart dilation and the likelihood of developing congestive heart failure. Presence of moderate to severe right atrial and ventricular dilation and hepatic venous congestion are indications for surgery. Ideally, surgical intervention should be

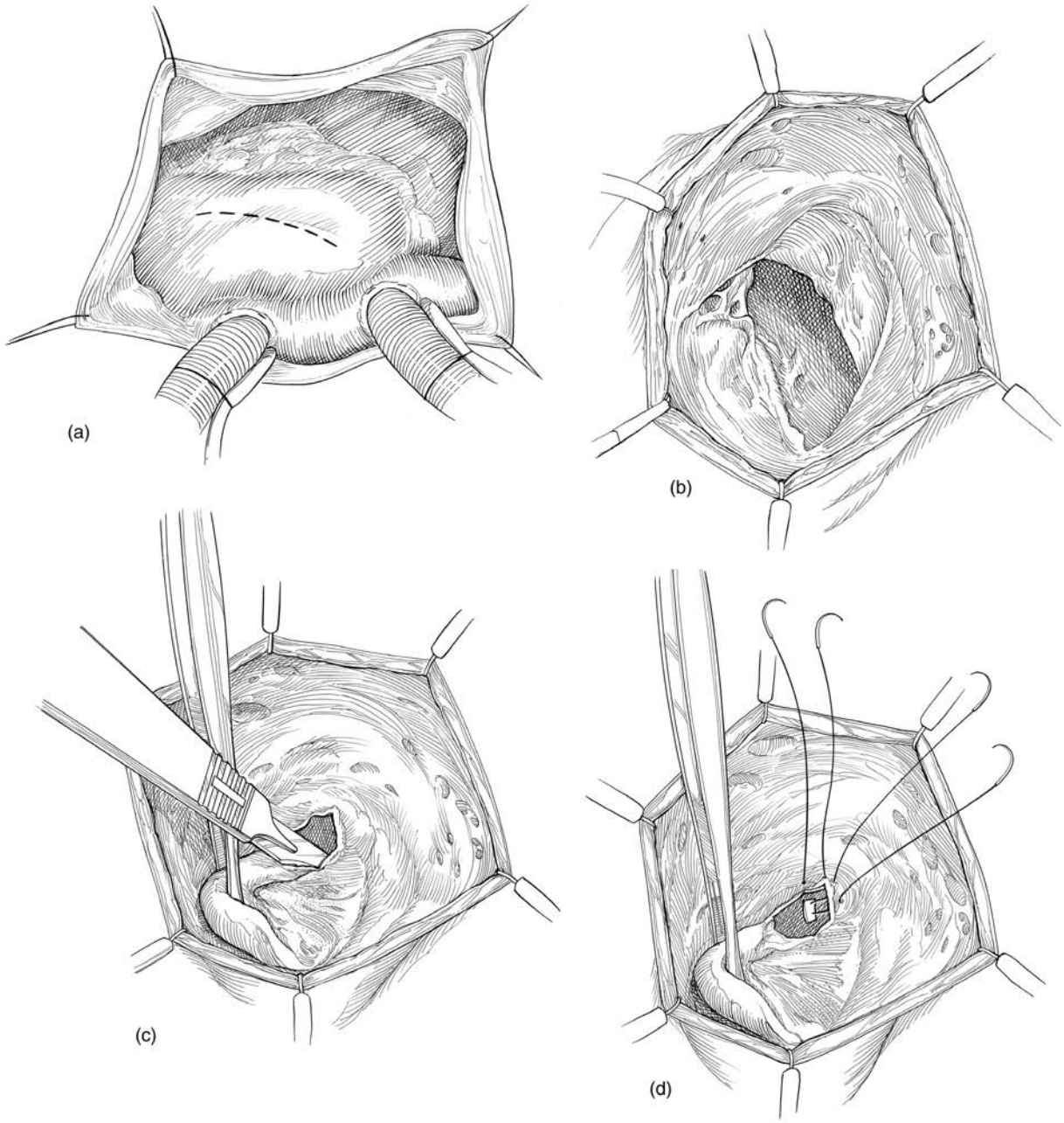


Figure 21.1 Tricuspid Valve Replacement

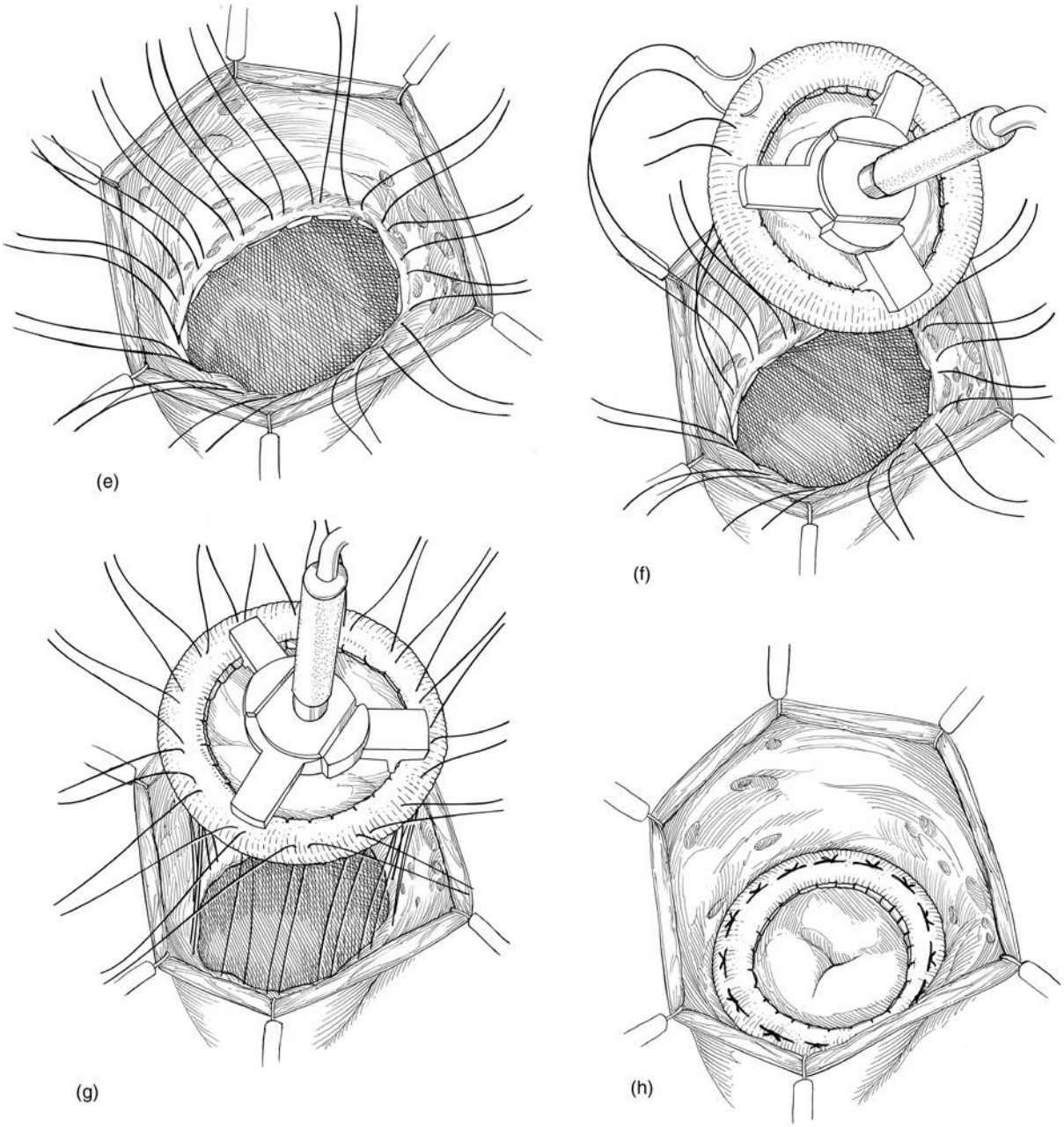


Figure 21.1 (*Continued*)

undertaken before onset of congestive heart failure. Once right-sided congestive heart failure becomes medically refractory or atrial fibrillation becomes longstanding, dogs become poor candidates for surgery.

Tricuspid Valve Replacement

Tricuspid valve replacement with a bioprosthetic valve is accomplished with the aid of cardiopulmonary bypass. The thoracic approach is a right fifth thoracotomy. Bicaval venous cannulation for cardiopulmonary bypass is necessary to isolate the right atrium (Figure 21.1a). The surgery is performed with the aorta crossclamped and antegrade cardioplegia. The cardiac approach is via a right atriotomy to expose the atrial side of the tricuspid valve (Figure 21.1b). The valve leaflets are excised 2 to 3 mm from the annulus (Figure 21.1c). Alternatively the mural leaflet of the tricuspid valve can be imbricated

rather than excised to support right ventricular function. Double-armed 3-0 or 2-0 pledget-reinforced polyester mattress sutures are placed into the annulus with the pledget on the ventricular side of the annulus (Figure 21.1d). The space within each mattress suture (12 to 16 total) can be wide to reduce the annular diameter, but the space between mattress sutures is kept small to prevent paravalvular leak (Figure 21.1e). Mattress sutures are passed through the sewing ring of the valve prosthesis (Figure 21.1f) assuring that the sutures are evenly spaced around circumference of the valve prosthesis and that the space between sutures is again kept small to prevent paravalvular leak (Figure 21.1g). The valve is seated into annulus and the sutures are tied (Figure 21.1h). The atriotomy is closed with a continuous horizontal mattress pattern oversewn with a simple continuous pattern. Our current practice is to administer combined oral anticoagulant and antiplatelet therapy for 3 months after surgery (Table 21.1). Antiplatelet therapy is continued for at least 12 months after surgery.

Tricuspid Valve Repair

Thoracic and cardiac approaches and CPB cannulation are the same as for tricuspid valve replacement. The valve apparatus is inspected for typical malformations and reparability. If the valve is deemed unrepairable, the surgery should be converted to a valve replacement. Papillary muscles that insert directly onto the mural leaflet are isolated and divided close to the leaflet margin (Figure 21.2a). A pledget-reinforced ePTFE mattress suture is placed in the proximal end of the divided papillary muscle (Figure 21.2b). The suture is then passed through the distal end of the divided papillary muscle but is not tied. This technique is repeated for each papillary insertion onto the mural leaflet. The tethered septal leaflet is freed from the septum by dividing shortened abnormal chordae that prevent leaflet motion (Figure 21.2c). Pledget-reinforced mattress sutures of ePTFE are preplaced in the ventricular septum to re-suspend the freed septal leaflet (Figure 21.2d). Before the leaflet sutures are tied, a ribbon annuloplasty is placed to reduce the diameter of the tricuspid orifice (Figure 21.2e). This is accomplished by placing sutures into the mural portion of the annulus from commissure to commissure. The sutures are passed through a ribbon of ePTFE vascular material of predetermined length and then tied. Typically, the tricuspid orifice is reduced by 30% to 40%, equivalent to a 28 mm to 32 mm orifice diameter for large breed dogs. Chordae replacement sutures are then passed through the margin of the septal leaflet and tied (Figure 21.2f). Length is judged by holding the edge of the leaflet to the opposing annulus while the suture is tied. The repair is completed by tying the sutures in the mural leaflet using the same guideline as for the septal leaflet (Figure 21.2g). The patient is placed on oral antiplatelet therapy for 3 months after surgery. Anticoagulation therapy is typically unnecessary unless thrombus begins to form on the repair.

Expected Outcomes

Favorable outcomes have been reported for dogs with TVD undergoing bioprosthetic tricuspid valve replacement [6]. In our experience, survival periods of up to 7 years after surgery are a reasonable expectation. Biological tolerance and long-term performance in dogs appears to be better for porcine aortic bioprostheses compared to bovine pericardial bioprostheses. Outcomes for tricuspid valve repair in dogs have not yet been reported; however, in our experience, short-term outcomes after tricuspid valve repair have been very promising and we now prefer valve repair over replacement for TVD in dogs when possible.

Mitral Regurgitation

Mitral regurgitation (MR) is classified etiologically as *primary (organic)* caused by a primary leaflet abnormality or *functional (secondary)* associated with cardiomyopathy. Examples of primary MR include degenerative (myxomatous) disease, endocarditis, and congenital valve dysplasia. Functional MR results from remodeling and/or dysfunction of the left ventricle. In dilated forms of primary cardiomyopathy, functional MR results from the combined effects of displacement of the papillary apparatus and dilation of the mitral annulus. MR is also classified functionally according to its underlying functional cause as *Type I (annular dilation), Type II (leaflet prolapse),* and *Type III (restrictive leaflet motion)* [1]. Interventional and surgical therapies for correction of MR take into consideration both its etiologic and functional classification.

Degenerative mitral valve disease is the most important cardiac disease in dogs and is a leading cause of death and disability in older dogs. The estimated overall prevalence of degenerative MR is 7% of the canine population [7]. It is characterized by varying degrees of nodular thickening and distortion of the valve leaflets, elongation and rupture of the chordae tendineae, and dilation of the valve annulus. Mitral regurgitation results from the combined functional abnormalities of leaflet prolapse (type II defect) and annular dilation (type I defect).

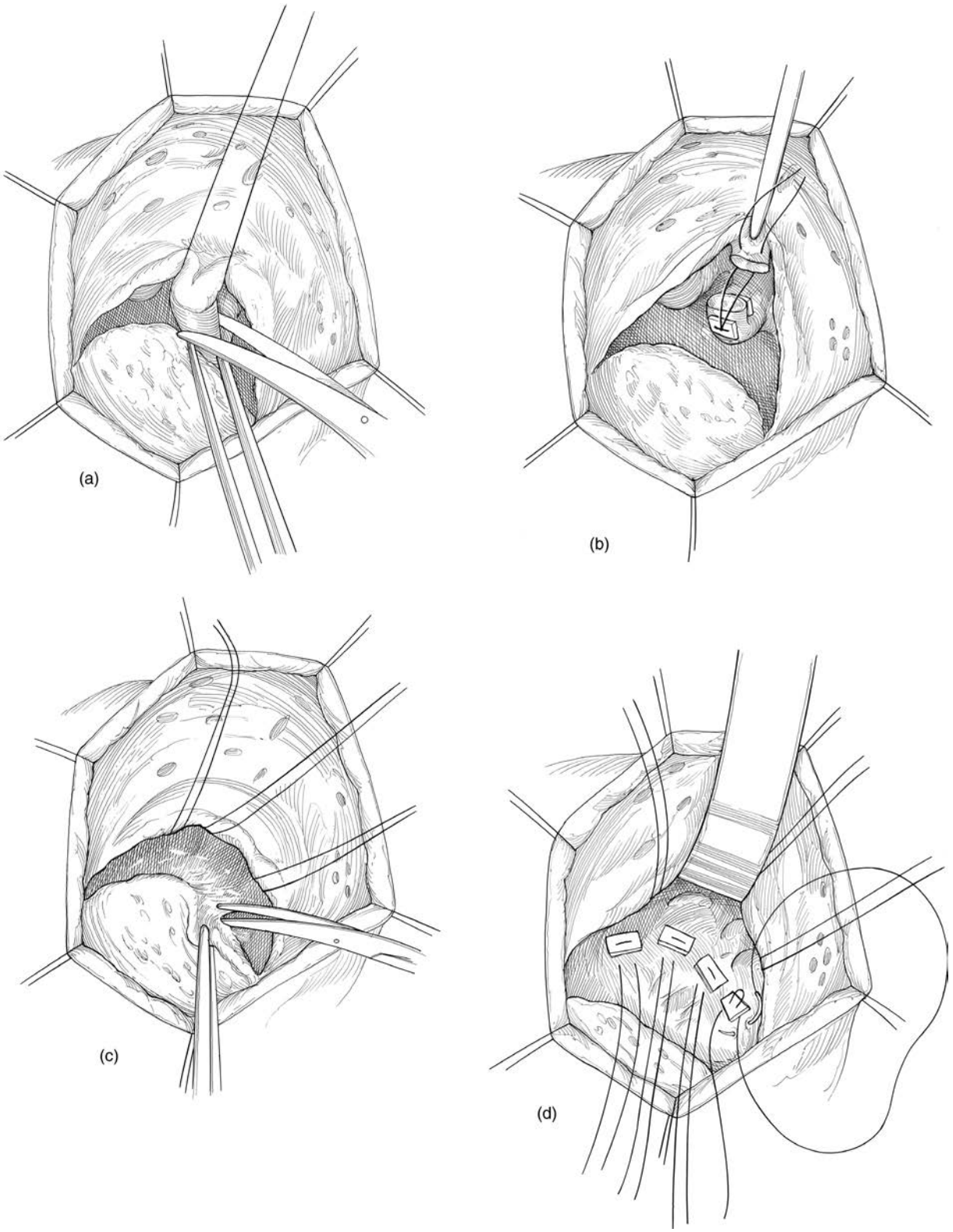
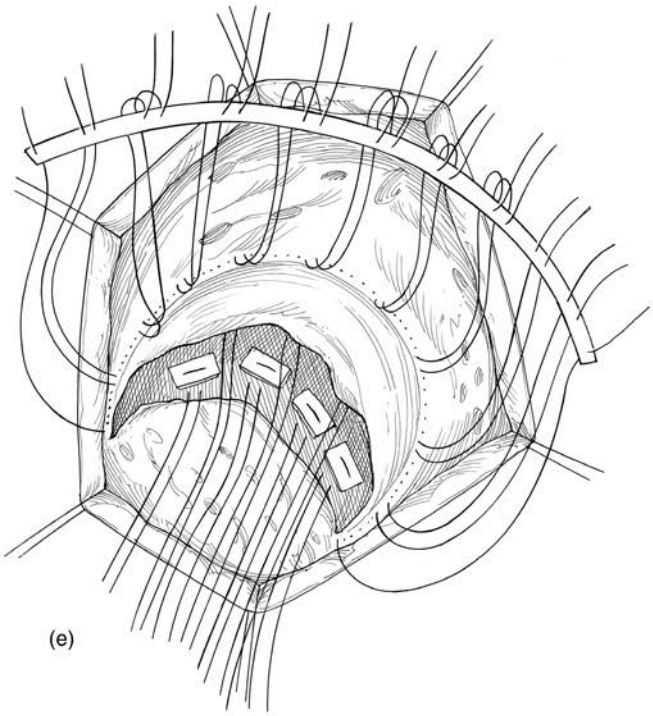
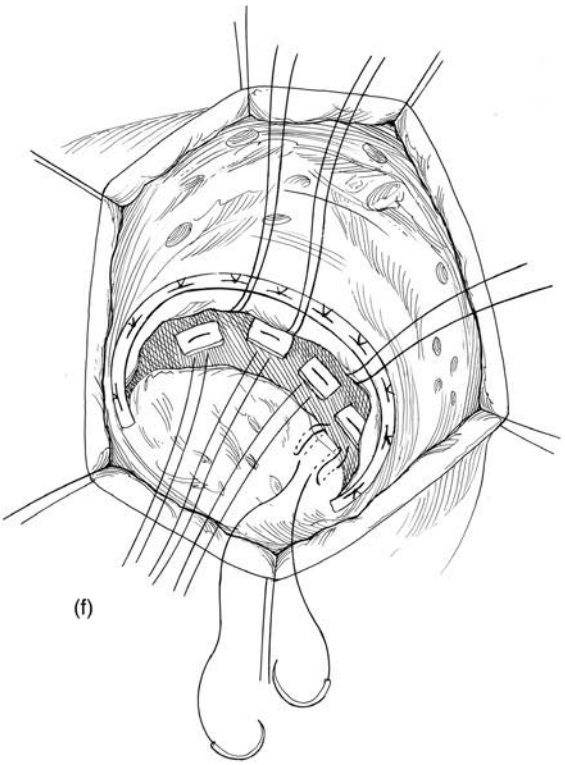


Figure 21.2 Tricuspid Valve Repair





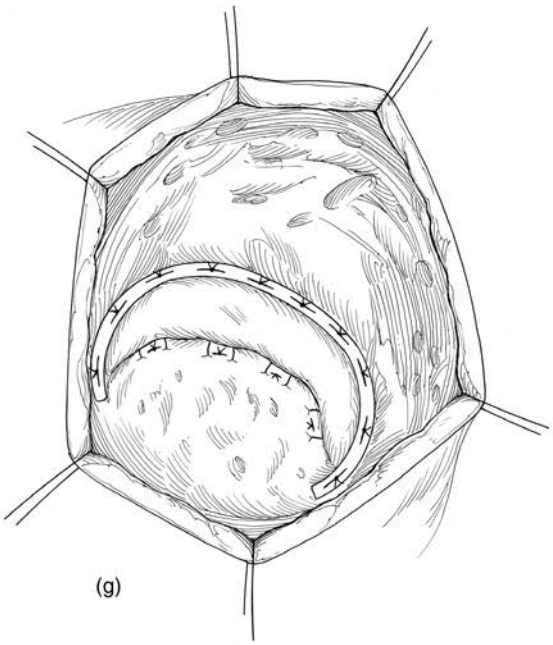


Figure 21.2 (*Continued*)

Table 21.1 Antithrombotic Drugs

Drug	Action	Dose	Monitoring
Apixaban[a,b]	Anticoagulant—Factor Xa inhibition	Dog: 0.5 mg/kg PO q 12 h Cat: 0.1 mg/kg PO q 12 h	Prothrombin time Factor Xa activity
Aspirin[c]	Antiplatelet—Cyclooxygenase inhibition	Dog: 1–5 mg/kg PO q 12–24 h	Platelet aggregometry (Collagen)
Clopidogrel[d,e]	Antiplatelet—ADP receptor inhibition	Dog: 1–2 mg/kg PO q 24 h Cat: 18.75 mg PO q 24 h	Platelet aggregometry (ADP)
Heparin	Anticoagulant—Factor III & X inhibition	Dog: 150–300 U/kg IV bolus 20–50 U/kg/h CRI Cat: same	Activated clotting time aPTT Thromboelastography (TF)
Warfarin[f]	Anticoagulant—Vitamin K inhibition	Dog: 0.05–0.1 mg/kg PO q 24 h	INR based on PT

TF = Tissue factor; ADP = Adenosine diphosphate; aPTT = Activated partial thromboplastin time; INR = International normalized ratio; PT = Prothrombin time

[a]He K, Luettgen JM, Zhang D, He B, Grace JE, et al. 2011. Preclinical pharmacokinetics and pharmacodynamics of apixaban, a potent and selective factor Xa inhibitor. *European Journal Drug Metabolism Pharmacokinetics.* 36(3):129–39.

[b]Myers JA, Wittenburg LA, Olver CS, Martinez CM, and Bright JM. 2015. Pharmacokinetics and pharmacodynamics of the factor Xa inhibitor apixaban after oral and intravenous administration to cats. *American Journal of Veterinary Research.* 76(8):732–8.

[c]Brainard BM, Meredith CP, Callan MB, Budsberg SC, Shofer FS, Driessen B, and Otto CM. 2007. Changes in platelet function, hemostasis, and prostaglandin expression after treatment with nonsteroidal anti-inflammatory drugs with various cyclooxygenase selectivities in dogs. *American Journal of Veterinary Research.* 68(3):251–7.

[d]Brainard BM, Kleine SA, Papich MG, and Budsberg SC. 2010 Pharmacodynamic and pharmacokinetic evaluation of clopidogrel and the carboxylic acid metabolite SR 26334 in healthy dogs. *American Journal of Veterinary Research.* 71(7):822–30.

[e]Teuber M, and Mischke R. Influence of a low dosage of clopidogrel on platelet function in cats as measured by the platelet function analyser PFA-100 and the multiplate analyser. 2016/ *Research Veterinary Science.* 109(149–56.

[f]Winter RL, Sedacca CD, Adams A, and Orton EC. 2012. Aortic thrombosis in dogs: presentation, therapy, and outcome in 26 cases. *Journal of Veterinary Cardiology.* 14(2):333–42.

Congenital mitral dysplasia occurs in both cats and dogs [2]. In dogs, the defect has a predilection for large and giant breeds of dog. A spectrum of abnormalities are possible, including shortening and thickening of the leaflets, cleft lesions of the leaflets, malformations (fusion, shortening, elongation, abnormal insertion, or absence) of the chordae tendineae, and malformation or malpositioning of papillary muscles. Mitral regurgitation results most often from restrictive leaflet motion (type III defect) and secondary annular dilation (type I defect). Mitral stenosis is a less common manifestation that accompanies mitral regurgitation in some cases.

Pathophysiology

Mitral regurgitation causes a low-pressure volume overload of the left ventricle. Cardiomyocytes respond by series replication of sarcomeres, leading to cellular elongation, eccentric hypertrophy, and chamber dilation. The response is initially adaptive, but becomes maladaptive as thickening of the ventricular free wall and septum fail to match chamber dilation, leading to increase ventricular afterload. Dilation of the mitral valve annulus contributes to the progression of MR. Left-sided congestive heart failure eventually develops in response to the volume overload and cardiac remodeling. Systolic dysfunction due to decreased contractility and the effects of remodeling on afterload contribute to the progression of heart failure. Atrial fibrillation may occur, further compromising cardiac function. Median survival with medical therapy alone is about 8 months after the onset of congestive heart failure [8].

Diagnosis

The characteristic physical finding of MR is a systolic regurgitant-type murmur at the left cardiac apex. Intensity of the regurgitant murmur typically reflects MR severity. High-pitched murmurs or musical "whoops" signal the presence of early MR. The femoral pulses may be normal or weak depending on severity. Auscultation of the lungs may reveal pulmonary crackles if pulmonary edema is present. Sinus tachycardia, premature atrial complexes, focal or multifocal atrial tachycardia, or atrial fibrillation are the most common secondary rhythm disturbances.

Thoracic radiographs show cardiomegaly with left atrial enlargement. Distension of the pulmonary veins with or without a perihilar interstitial or alveolar pattern is present, depending on the degree of heart failure. Diagnosis is confirmed based on characteristic echocardiographic findings of the mitral valve apparatus. For degenerative mitral valve disease these changes include nodular thickening and prolapse of one or both leaflets. Ruptured chordae with leaflet flail may also be present. In mitral valve dysplasia a variety of malformations of the mitral valve apparatus may be apparent, including thickened and shortened chordae tendineae and aberrant chordal attachments, all of which generally combine to restrict leaflet motion during systole. The degree of cardiac remodeling generally reflects the severity of MR and include left atrial dilation reflected as an increase in LA:Ao ratio and left ventricular dilation with varying degrees of ventricular wall thickening. Indices of left ventricular function such as fractional shortening or ejection fraction are above normal early, but become normal or subnormal as secondary systolic dysfunction develops. Doppler echocardiography confirms MR. Turbulent flow patterns on color-flow Doppler are often eccentric early in the disease due to asymmetrical prolapse of the leaflets. Severity of MR is graded based on the area of turbulence flow within the left atrium. Turbulent flow patterns greater than 50% of the area of the left atrium are indicative of severe MR.

Indications of Surgery

Indications for mitral valve surgery are diuretic-dependent congestive heart failure (Stage C) or significant left ventricular remodeling with activity intolerance (Stage B2). Severe chronic inflammatory airway disease with or without collapsing trachea is a relative contraindication for surgery. Dogs with secondary systolic dysfunction or atrial fibrillation can tolerate mitral valve surgery, but will require more intense supportive care after surgery. Dogs with medically refractory congestive heart failure (Stage D) are poor candidates for surgical intervention.

Surgical options for MR secondary to degenerative disease or valve dysplasia are valve replacement or valve repair. Mitral valve repair has the advantages of not requiring a prosthesis, not requiring anticoagulation therapy after surgery, and better preservation of systolic function. Disadvantages of valve repair are its technical difficulty and less predictable outcome, depending on surgeon experience. Valve replacement has the advantages of being technically easier to perform than valve repair and certainty of complete correction of MR. The disadvantage of valve replacement is its requirement for a valve prosthesis and their associated shortcomings including early thrombosis or late failure. Mitral valve replacement is also restricted to larger dogs (> 10 kg) based on size availability of commercial valve prostheses (18 mm or larger). Options for mitral valve replacement are a glutaraldehyde-fixed bioprostheses or mechanical valves. Bioprosthetic valves have the principle advantage of only requiring 3 months of anticoagulation therapy after surgery. Bioprosthetic valves carry a risk for non-immune inflammatory rejection (known as inflammatory pannus) in dogs that can render the valve nonfunctional within a few months to several years after surgery. The actual incidence of this complication in dogs is not known, but seems to be less frequent with porcine aortic bioprostheses compared to bovine pericardial prostheses. Mechanical valves have excellent durability, but are thrombogenic and require lifetime anticoagulation therapy. The bileaflet tilting disc design is currently favored because of superior flow characteristics and lower thrombogenicity. However mechanical valves are generally avoided in dogs because of the need for life-time anticoagulation therapy and the high incidence of thromboembolic complications [9].

Surgical Approaches to the Mitral Valve

The approach for mitral valve surgery can be either a right or left fifth thoracotomy. A left thoracotomy generally provides better access to the mitral valve through the left atrial free wall (Figure 21.3a). Venous cannulation is accomplished with a single two-stage cannula passed through the right atrial appendage from the left side. The cardiac approach is via a left lateral atriotomy. With a right thoracotomy approach, bicaval venous cannulation can be employed and access to the mitral valve is through the left atrium dorsal to the interatrial groove (Figure 21.3b). A right thoracotomy provides more direct access for venous cannulation however access to the mitral valve through the dorsal approach is more limited. A left thoracotomy provides better surgical access to the mitral valve is therefore the preferred approach. A right thoracotomy is employed if it is necessary to perform an additional surgical repair requiring access through the right atrium.

Mitral Valve Replacement

The technique for mitral valve replacement is similar to that described for replacing the tricuspid valve. It is strongly recommended that at least some of the mitral apparatus be preserved to help support left ventricular

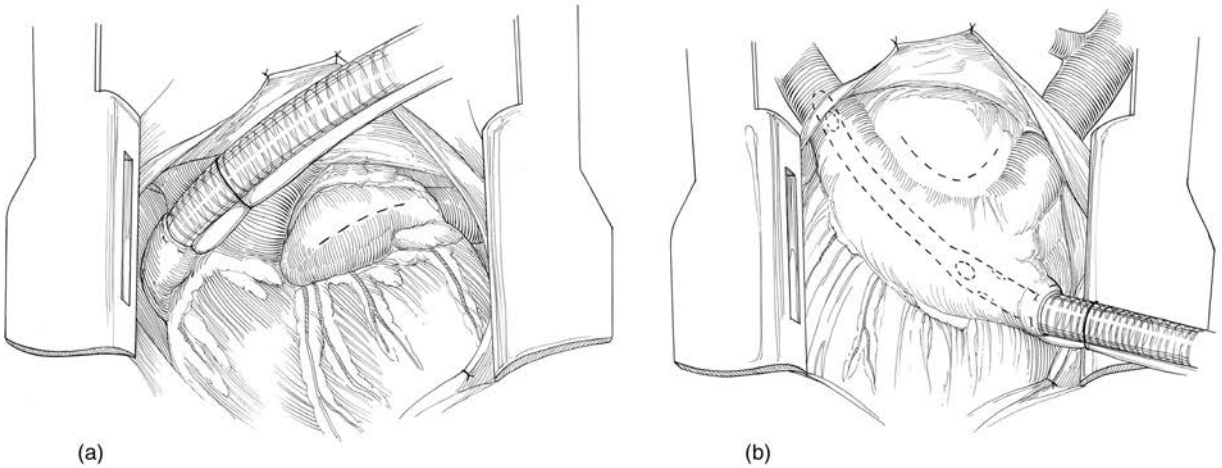


Figure 21.3 Mitral Valve Approaches

function after surgery. The left atrium is opened to expose the mitral valve (Figure 21.4a). The size of the mitral annulus is measured with sizing obturators and an appropriate-sized prosthesis is chosen for implantation. The septal leaflet and associated chordae tendineae are excised leaving a few millimeter margin of leaflet at the annulus (Figure 21.4b). The mural leaflet and its associated chordal attachments are preserved by plicating the leaflet into the annular mattress sutures. Twelve to 15 pledget-reinforced mattress sutures are placed into the valve annulus (Figure 21.4c). The mattress sutures are then passed through the sewing ring of the prosthesis assuring that they are evenly distributed around circumference of the prosthesis. Distance within each mattress suture can be as wide as necessary; however, distance between mattress sutures is kept small to prevent paravalvular leak. The prosthesis is seated into the valve annulus and the sutures are tied (Figure 21.4d). The left atrium is de-aired and closed with a continuous horizontal mattress pattern oversewn with a simple continuous pattern.

Mitral Valve Repair

Mitral valve repair employs a variety of techniques to correct the underlying functional causes of MR. For degenerative MR this requires addressing dilation of the valve annulus and correction of leaflet prolapse. Repair of mitral valve dysplasia is generally directed at annular dilation and correction of restrictive leaflet motion. The success of mitral valve repair depends on accurate preoperative and intraoperative assessment of mitral valve function. Annular dilation is assumed to be a component of mitral regurgitation whenever substantial left heart dilation is present. Mitral valve leaflets are assessed for coaptation, evidence of prolapse, or restrictive motion before surgery by transthoracic and/or transesophageal echocardiography. Color-flow patterns of regurgitant flow are also informative. Leaflet prolapse causes an eccentric regurgitant jet directed away from a prolapsing leaflet. Assessment of valve function during surgery is accomplished by infusing cold saline into the ventricle to assess valve competence.

Annuloplasty corrects annular dilation that is an inevitable component of severe MR and therefore is necessary in virtually all mitral valve repairs regardless of underlying etiology. For functional MR, annuloplasty may be the only repair necessary. Dilation of the annulus occurs over the mural and commissural regions of the annulus. The septal region of the annulus is fixed by the fibrous base of the heart and does not dilate. Thus, it is the mural and commissural regions of the annulus that must be reduced by annuloplasty. A variety of annuloplasty techniques have been developed and advocated. The simplest involve plication of the annulus with running sutures in the commissural and mural portions of the annulus. In humans, ring annuloplasty has been shown to be more durable and is therefore preferred. A variety of rings have been developed including rigid, flexible, saddle-shaped, complete, and partial rings. Commercial annuloplasty rings can be used in larger dogs, but for most dogs a partial flexible "ring" or "ribbon" fashioned from cardiovascular graft materials such as ePTFE is sufficient. The size (length) of the ring is based on the area of the septal leaflet with the goal of reducing the area of the annulus to the approximate area of the septal leaflet. Partial

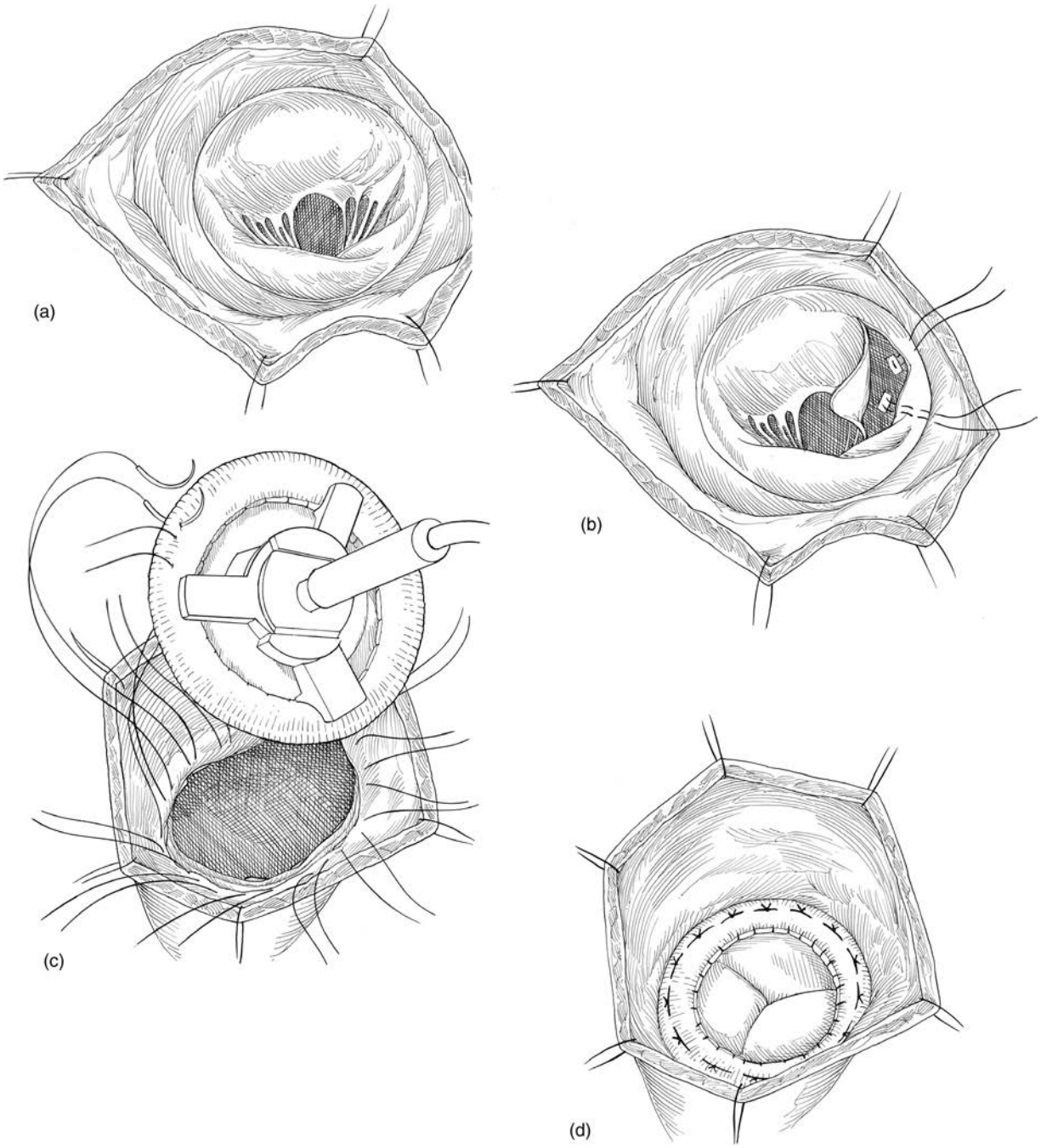


Figure 21.4 Mitral Valve Replacement

ring/ribbon annuloplasty is achieved by placing 3-0 or 4-0 sutures into the valve annulus 1 to 2 mm back from the leaflet insertion (Figure 21.5a). Sutures in the mural portion of the annulus are placed with wide bites to reduce the circumference of this region when the ribbon is seated. If a complete ring is used, sutures are placed across the base of the septal leaflet, taking care to avoid the aortic valve that lies in close proximity. Sutures are placed so as not to reduce the annulus over the septal region. If a partial ring/ribbon technique is used, then anchoring sutures are placed just beyond the commissural regions of the annulus. Sutures are passed through the ring/ribbon. The ring/ribbon is seated into position and the sutures are tied (Figure 21.5b). Annuloplasty reestablishes appropriate coaptation of the valve leaflets.

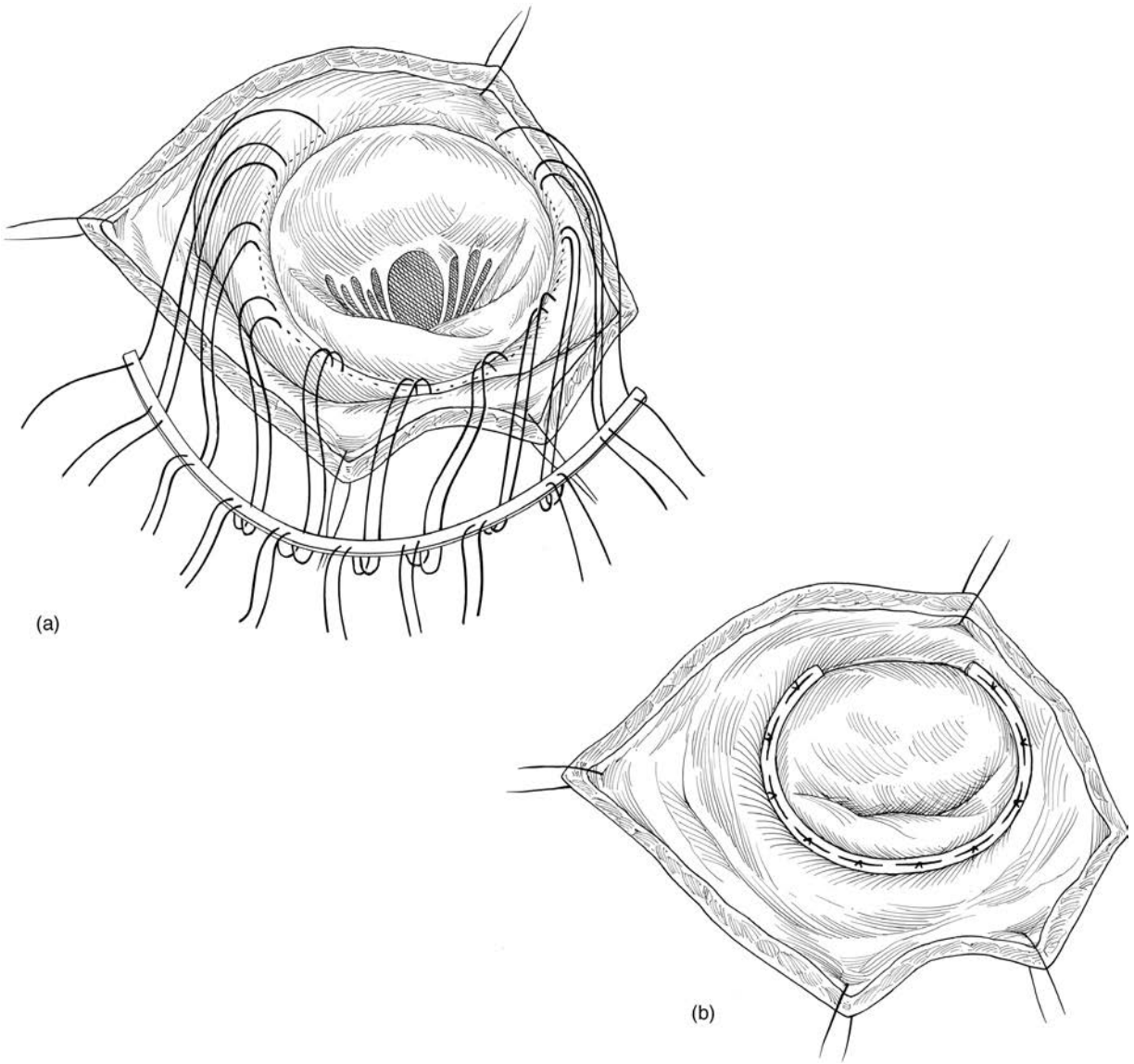


Figure 21.5 Mitral Ribbon Annuloplasty

Several repair techniques have been described to correct leaflet prolapse. Of these techniques, placement of artificial chordae is currently the preferred technique in dogs [10, 11]. Artificial chordae consist of 5-0 or 4-0 ePTFE monofilament suture. Pledget-reinforced ePTFE sutures are placed in the tip papillary muscle and then brought through the leaflet margin (Figure 21.6a). Correction of prolapse is focused primarily on the septal leaflet. Generally, at least two artificial chordae are placed in the septal leaflet, with at least one originating in each papillary muscle. The most important aspect of the technique is judging the length of the artificial chordae. A general guide is that the edge of the valve leaflet should just reach the plane of the opposing annulus after tying down the annuloplasty ring (Figure 21.6b).

Edge-to-edge (E-to-E) repair was first described by Alfieri and has become a useful addition to the armamentarium for valve repair [12]. E-to-E repair for leaflet prolapse is accomplished by suturing the free edge of the prolapsing leaflet to the free edge of the facing leaflet at the point of greatest prolapse (Figure 21.7). This correction results in a double orifice valve and can reduce the effective office area for mitral inflow. The E-to-E technique can be combined with a more conservative annuloplasty to avoid creating mitral stenosis. The E-to-E technique can also be used to correct regurgitation in the commissural regions,

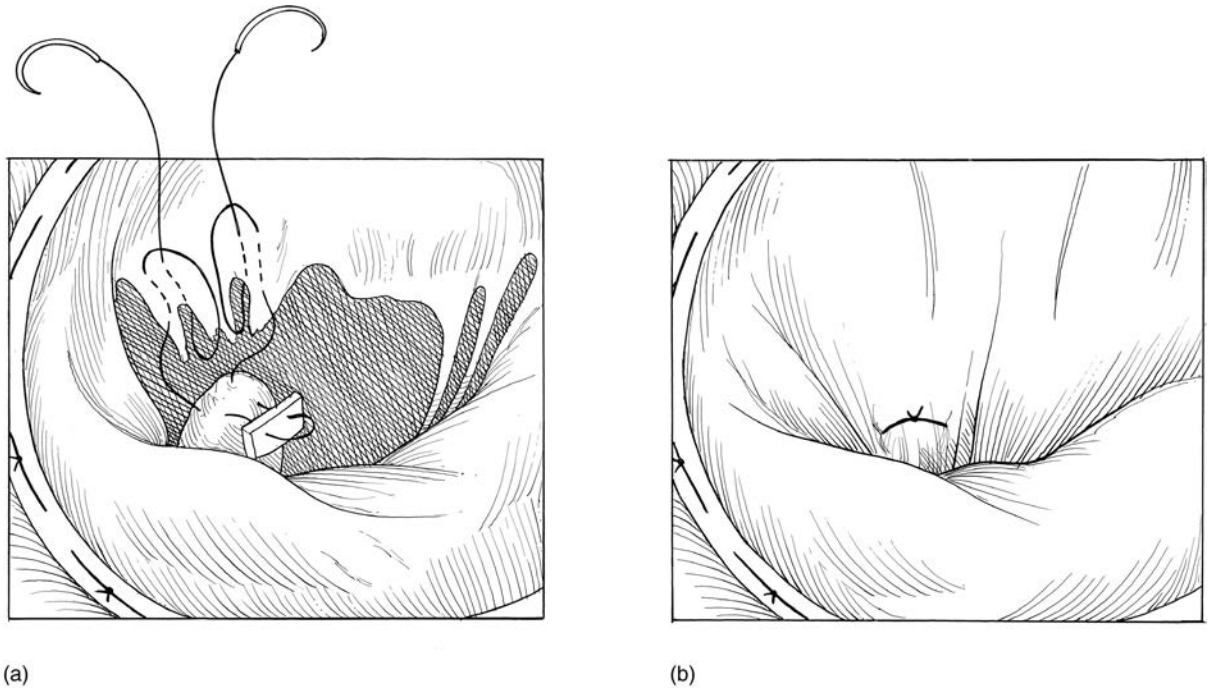


Figure 21.6 Mitral Valve Repair—Artificial Chordae

which can be difficult to repair with other techniques. E-to-E repair is technically simpler than artificial chordae repair but is considered less optimal from a functional and durability standpoint. Nevertheless, E-to-E repair can be employed as a secondary technique when needed.

Surgical repair of mitral valve dysplasia can be challenging, depending on the spectrum of morphologies present. In general, these malformations combine to restrict leaflet closure during systole. A variety or repairs have been described to correct restrictive leaflet motion associated with mitral valve dysplasia [13]. Aberrant chordae tendineae that are restricting leaflet closure are divided. If dividing an aberrant chordae results in leaflet prolapse, this can be addressed by placement of an artificial chordae.

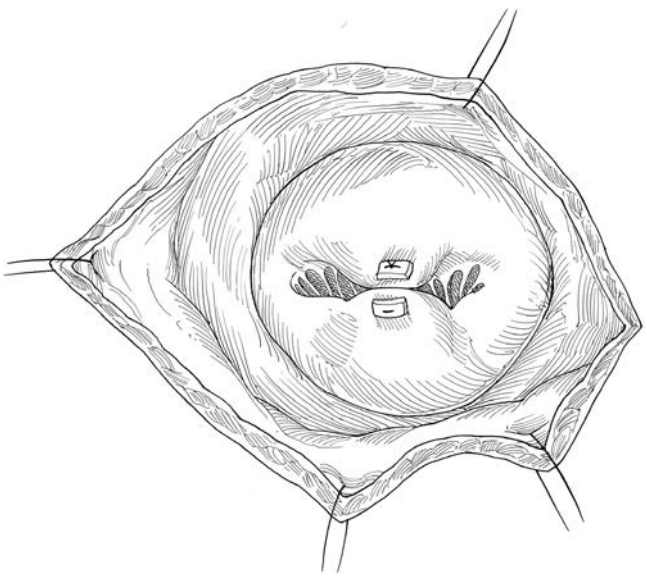


Figure 21.7 Mitral Valve Repair—Edge-to-Edge

Fusion and shortening of the chordae tendineae can be addressed by fenestration of the fused chordae and splitting of the papillary muscle to effectively lengthen the chordal apparatus. If this fails to adequately mobilize the valve apparatus, then the papillary muscle can be divided and lengthened with a large ePTFE mattress suture, as described for correction of tricuspid valve dysplasia. Commissurotomy may be necessary if valvular mitral stenosis is present. Lastly, a ring annuloplasty will usually be necessary to correct secondary annular dilation.

Outcomes

Despite good short-term results, replacement of the mitral valve with a mechanical prosthesis should be avoided due the need for lifetime anticoagulation therapy and high incidence of thrombosis [9]. Clinical results for dogs undergoing bioprosthetic mitral valve replacement are limited to a case report [14], although there is experimental evidence that bioprosthetic valves perform well in dogs in the mitral position [15]. Our own clinical experience with bioprosthetic mitral valve replacement in larger dogs is favorable, with survival periods of 5 years or greater.

Successful mitral valve repair has been reported in dogs with degenerative mitral valve disease and congenital mitral dysplasia [10,11,16,17]. Success rates in cases series range from 75% to > 90% and will likely improve with case experience. In our own experience, dogs have survived for periods of more than 7 years after mitral valve repair.

References

1 Carpentier A. 1983. Cardiac valve surgery—The "French correction." *Journal of Thoracic Cardiovascular Surgery.* 86(3):323–37.

2 Schrope DP. 2015. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. *Journal of Veterinary Cardiology.* 17(3):192–202.

3 Buchanan JW. 1999. Pravalence of cardiovascular disorders. In: Fox PR, Sisson D, and Moise NS eds. *Textbook of Canine and Feline Cardiology*. pp. 457–70 Philadelphia: W.B. Saunders Co.

4 Liu SK, and Tilley LP. 1976. Dysplasia of the tricuspid valve in the dog and cat. *Journal of the American Veterinary Medical Association.* 169(6): 623–30.

5 Oyama MA, Weidman JA, and Cole SG. 2008. Calculation of pressure half-time. *Journal of Veterinary Cardiology.* 10(1):57–60.

6 Arai S, Griffiths LG, Mama K, Hackett TB, Monnet E, Boon JA, Carter L, and Orton EC. 2011. Bioprosthesis valve replacement in dogs with congenital tricuspid valve dysplasia: technique and outcome. *Journal of Veterinary Cardiology.* 13(2): 91–9.

7 Atkins C, Bonagura J, Ettinger S, Fox P, Gordon S, Haggstrom J, Hamlin R, Keene B, Luis-Fuentes V, and Stepien R. 2009. Guidelines for the diagnosis and treatment of canine chronic valvular heart disease. *Journal Veterinary Internal Medicine.* 23(6):1142–50.

8 Haggstrom J, Boswood A, O'Grady M, Jons O, Smith S, Swift S, Borgarelli M, Gavaghan B, Kresken JG, Patteson M, et al. 2008. Effect of pimobendan or benazepril hydrochloride on survival times in dogs with congestive heart failure caused by naturally occurring myxomatous mitral valve disease: the QUEST study. *Journal of Veterinary Internal Medicine.* 22(5):1124–35.

9 Orton EC, Hackett TB, Mama K, and Boon JA. 2005. Technique and outcome of mitral valve replacement in dogs. *Journal of the American Veterinary Medical Association.* 226(9):1508–11.

10 Griffiths LG, Orton EC, and Boon JA. 2004. Evaluation of techniques and outcomes of mitral valve repair in dogs. *Journal of the American Veterinary Medical Association.* 224(12): 1941–5.

11 Uechi M, Mizukoshi T, Mizuno T, Mizuno M, Harada K, Ebisawa T, Takeuchi J, Sawada T, Uchida S, Shinoda A, et al. 2012. Mitral valve repair under cardiopulmonary bypass in small-breed dogs: 48 cases (2006–2009). *Journal of the American Veterinary Medical Association.* 240(10):1194–201.

12 Alfieri O, and De Bonis M. 2010. The role of the edge-to-edge repair in the surgical treatment of mitral regurgitation. *Journal of Cardiac Surgery.* 25(5):536–41.

13 Carpentier A, and Brizard C. 2006. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 573–98. Chichester: John Wiley & Sons.

14 Behr L, Chetboul V, Sampedrano CC, Vassiliki G, Pouchelon JL, Laborde F, and Borenstein N. 2007. Beating heart mitral valve replacement with a bovine pericardial bioprosthesis for treatment of mitral valve dysplasia in a Bull Terrier. *Veterinary Surgery.* 36(3):190–8.

15 Takashima K, Soda A, Tanaka R, and Yamane Y. 2008. Long-term clinical evaluation of mitral valve replacement with porcine bioprosthetic valves in dogs. *The Journal of veterinary Medical Science.* 70(3):279–83.

16 Mizuno T, Mizukoshi T, and Uechi M. 2013. Long-term outcome in dogs undergoing mitral valve repair with suture annuloplasty and chordae tendinae replacement. *The Journal of Small Animal Practice.* 54(2):104–7.

17 Boggs LS, Dewan SJ, and Ballard SE. 1996. Mitral valve reconstruction in a toy-breed dog. *Journal of the American Veterinary Medical Association.* 209(11):1872–6.

22

Congenital Septal Defects

E. Christopher Orton

Congenital septal defects include ventricular septal defect (VSD), atrial septal defect (ASD), atrioventricular septal defect (AVSD), and tetralogy of Fallot. The natural history of these defects depends on their size, location, and blood flow direction and magnitude. Normal intracardiac pressures dictate that shunt flow is left-to-right, unless right heart pressures are elevated by an intracardiac obstruction, pulmonary hypertension, or right-sided heart failure. Left-to-right shunt flow causes a volume overload of the right or left ventricle, or both, depending on the size and location of defect. Volume overload can lead to congestive heart failure, depending on magnitude. Large left-to-right shunts also induce pulmonary vasoconstriction and vascular remodeling leading to progressive pulmonary hypertension. In some cases, pulmonary hypertension can be severe enough to reverse shunt flow through the defect. Right-to-left shunt flow causes hypoxemia, cyanosis, and progressive polycythemia, depending on shunt fraction. Palliative and corrective surgeries are available for septal defects that are determined to be hemodynamically significant.

Ventricular Septal Defect

Isolated ventricular septal defect is a congenital defect that results from incomplete closure of the membranous and/or muscular ventricular septum. The anatomy of VSD in humans has been extensively described [1]. *Perimembranous VSD* are most prevalent in companion animals. Perimembranous defects are bordered by the central fibrous body and either the inlet, trabecular, or infundibular (outlet) portion of the muscular septum. These defects are subcategorized as *perimembranous inlet VSD*, *perimembranous trabecular VSD*, and *perimembranous infundibular VSD*, respectively. They are medial to the septal tricuspid leaflet and closely associated with the conduction tissues of the heart. *Muscular VSD* are less prevalent in small animals, are bordered entirely by muscle, and occur within the inlet, trabecular, or infundibular muscular septum. *Doubly committed subarterial VSD* occurs within the infundibular (outlet) muscular septum that is shared by the right and left ventricles. The upper border of the defect is in fibrous continuity with the pulmonary and aortic valves, separated only by a thin rim of fibrous tissue. These defects are associated with a high incidence of aortic sufficiency secondary to prolapse of aortic valve.

VSD accounts for about 7.5% to 15% of congenital heart defects in dogs and is among the most common congenital malformations in cats [2–4]. The etiology of VSD is incompletely understood, but is suspected to be heritable particularly when it occurs in a breed with a high prevalence. English springer spaniel, Lakeland terrier, West Highland white terrier, Basset hound, English bulldog, Akita, and Shih Tzu have a predisposition for the defect.

Pathophysiology

The pathophysiology and natural history of VSD depend on its size and location. Large VSD allow significant left-to-right shunting of blood that overloads the left and possibly right heart eventually causing progressive heart failure. High flow VSD can also trigger pulmonary vasoconstriction and vascular remodeling leading to progressive pulmonary hypertension. In some cases pulmonary arterial pressures can become suprasystemic resulting in right-to-left shunt flow. This can occur shortly after birth or develop as a late sequela. Residence at altitude increases the likelihood and accelerates the development of pulmonary hypertension; and this should be a consideration for surgical intervention. Aortic insufficiency can develop as a secondary abnormality, particularly with

Small Animal Thoracic Surgery, First Edition. E. Christopher Orton and Eric Monnet.

Companion website: www.wiley.com/go/orton/thoracic

doubly committed subarterial VSD. Aortic insufficiency results from prolapse of the right coronary aortic cusp into the defect and adds to the volume overload of the left ventricle accelerating the course toward congestive heart failure.

Diagnosis

Young animals with VSD may not be symptomatic at first presentation. Larger VSD will eventually cause signs related to left-sided heart failure. VSD produces a systolic murmur loudest at the right cranial sternum. A systolic murmur is also heard well at the left heart base due to relative pulmonary stenosis caused by high flow across the pulmonary valve. A diastolic murmur at the left heart base suggests the presence of significant aortic insufficiency and gives the murmur a continuous quality that can be confused with patent ductus arteriosus. Thoracic radiographs reveal varying degrees of left or biventricular heart enlargement, depending on the size and location of the defect. The degree of pulmonary vascular enlargement generally reflects the magnitude of the left-to-right shunt.

Larger VSD can be visualized directly on two-dimensional echocardiography. Perimembranous VSD are located high in the ventricular septum medial to the septal leaflet of the tricuspid valve on the right side. Color-flow Doppler is helpful in demonstrating turbulent flow through smaller defects. The direction and velocity of shunt flow is determined by interrogation with spectral Doppler. Absence of significant left-sided heart dilation, a high left-to-right peak shunt velocity through the defect (> 5.0 M/s), and a normal peak pulmonary ejection velocity (< 1.5 M/s) suggest that the VSD may be "hemodynamically restrictive," warranting a good prognosis without the need for intervention. Large "hemodynamically significant" or "nonrestrictive" defects are associated with prominent left-sided heart enlargement, lower peak shunt velocities across the defect (< 4.0 M/s), and an elevated peak pulmonary ejection velocity (> 2.0 M/s). Peak pulmonary ejection velocities can be elevated by large volume shunt flows (i.e., relative pulmonary stenosis) or by actual concurrent congenital pulmonary stenosis, so this finding must be interpreted in light of other findings. Concurrent mild to moderate pulmonary stenosis can be a favorable finding, as it elevates right ventricular systolic pressure and diminishes shunt flow through the septal defect. Thus, it is important to distinguish between elevated pulmonary velocities due to high shunt flow and actual mild to moderate pulmonary stenosis that may be moderating shunt flow. The pulmonary valve should be carefully evaluated for structural abnormalities if the peak pulmonary ejection velocity is elevated. A low peak shunt velocity across the defect can be caused by a nonrestrictive VSD that allows a degree of equilibration of right and left ventricular pressures, or it can signal the presence of elevated right ventricular pressure due to pulmonary hypertension or pulmonary stenosis. It is important to look for supporting evidence of pulmonary hypertension, including thickening right ventricular wall, higher than predicted tricuspid regurgitation, or pulmonic insufficiency velocities, and/or characteristic changes in the pulmonic ejection velocity profile.

Classic quantification of intracardiac shunts including VSD is determined by oximetry and calculation of the pulmonary-to-systemic shunt flow ratio (Q_P/Q_S). This is accomplished by selective right heart catheterization and demonstration of a "step up" in blood oxygen saturation from upstream to downstream of the shunt. The simplified formula for calculation of the shunt flow ratio is:

$$Q_P/Q_S = (S_aO_2 - S_vO_2)/(S_{pv}O_2 - S_{pa}O_2) \quad (22.1)$$

where S_aO_2, S_vO_2, $S_{pv}O_2$ and $S_{pa}O_2$ are systemic arterial, mixed venous, pulmonary venous, and pulmonary arterial blood oxygen saturation, respectively. In the case of VSD, S_vO_2 samples are collected from the right atrium. $S_{pv}O_2$ saturation is assumed to be the same as systemic arterial so long as $S_aO_2 \geq 95\%$.

Indications for Surgery

Surgical intervention is indicated for high flow VSD to prevent or slow onset of congestive heart failure and/or progressive pulmonary hypertension. Several parameters suggest that a VSD is hemodynamically significant, including radiographic evidence of pulmonary vascular enlargement, radiographic or echocardiographic evidence of left atrial and ventricular chamber dilation, lower than predicted spectral Doppler-measured shunt flow velocity across the defect, and/or elevated peak pulmonary ejection velocity. If there is doubt about the hemodynamic significance of a VSD, then right heart catheterization should be performed to determine the Q_P/Q_S. Based on guidelines in human infants, $Q_P/Q_S < 1.5$ suggests that a VSD is restrictive and argues against the need for intervention [5]. Similarly, favorable prognosis has been reported for dogs and cats with $Q_P/Q_S < 1.5$ [6]. A $Q_P/Q_S > 2.0$ indicates a large left-to-right shunt and is generally an indication for intervention. The ratio of VSD to aorta diameter (VSD:Ao) correlates with Q_P/Q_S in dogs and cats [6]. VSD:Ao < 0.4 is associated with a favorable prognosis [6]. The presence of

moderate or greater aortic insufficiency is an indication for surgical intervention even if the shunt flow is considered mild to moderate because of the likelihood that it will be progressive.

Definitive closure of VSD can be considered for animals with moderate pulmonary hypertension so long as shunt flow remains left-to-right. Surgical intervention is contraindicated for right-to-left VSD secondary to pulmonary hypertension. Palliative pulmonary artery banding must be undertaken before the onset of significant pulmonary hypertension. Presence of moderate or greater pulmonary hypertension diminishes the benefit of pulmonary artery banding, although it can still be considered in an effort to stop or slow progression of pulmonary vascular remodeling.

Pulmonary Artery Banding

Pulmonary artery banding is a palliative surgical intervention for large left-to-right VSD and other complex cardiac defects associated with high pulmonary blood flow, including complete atrioventricular septal defect and double-outlet ventricle without pulmonary stenosis [7]. Pulmonary artery banding for isolated VSD in humans has been largely abandoned in favor of early definitive VSD closure. In animals, pulmonary artery banding is a viable option for long-term palliation of VSD. The goal of pulmonary artery banding is to increase right ventricular systolic pressure and thereby decrease the pressure gradient driving shunt flow. The principal hazard is overtightening the band and reversal of shunt flow.

Pulmonary artery banding is accomplished through a left fourth thoracotomy in a dog or a left fourth or fifth thoracotomy in a cat. The pericardium is opened transversely ventral to the phrenic nerve and sutured to the thoracotomy incision (Figure 22.1a). The pulmonary artery is separated from the aorta cranially and caudally by a combination of sharp and blunt dissection of the loose adventitial connective tissue that is shared by the pulmonary artery and aorta. Caudally dissection is facilitated by gentle retraction of the left auricle with a wide retractor or vascular clamp (Figure 22.1b). A right-angle forceps is passed behind the pulmonary artery between the pulmonary artery and aorta. Cotton tape or #1 silk suture is passed around the pulmonary artery dorsal to the pulmonary valve. Judging the degree of tightening of the band is facilitated by placing a catheter into the pulmonary artery distal to the band for measurement of pulmonary arterial pressure and $S_{pa}O_2$. Catheter placement is accomplished through a 5-0 pledget-reinforced mattress suture or simple pursestring suture passed through a Rommel tourniquet (Figure 22.1c).

Several criteria are considered to determine the degree of pulmonary artery constriction (Box 22.1). As the pulmonary artery band is tightened, systemic arterial pressure increases while the pulmonary arterial pressure distal to the band decreases. The optimal degree of constriction is achieved when the increase in systemic arterial pressure just reaches a plateau (usually an increase of 10–20 mm Hg); and pulmonary arterial systolic pressure returns toward normal (ideally 30 mm Hg assuming that significant pulmonary vascular remodeling is not present). Inotropic support should be avoided if possible during band tightening, as this may alter the balance between systemic and pulmonary pressures and resistances. Additional guidelines include a decrease in $S_{pa}O_2$ of 5% to 10%, a decrease in the peak shunt velocity measured by intraoperative epicardial or transesophageal echocardiography. Peak shunt velocity should not be decreased to > 2.5 M/s. S_aO_2 should be monitored before and after banding to assure that it has not changed as a result of shunt reversal. As a general rule, optimal banding requires an approximate 50% to 66% reduction in the diameter of the pulmonary artery, although this will vary, depending on the degree of pulmonary artery dilation. The pulmonary band is secured by tying the suture, and the catheter site is closed by tying mattress suture (Figure 21.1d).

Open Repair of VSD

Open repair of VSD is accomplished with the aid of cardiopulmonary bypass [5]. A right atriotomy approach is chosen to close most isolated VSD of the perimembranous and inlet muscular types (Figure 22.2a). A right fifth thoracotomy provides the most direct access to the right atrium. Arterial cannulation for cardiopulmonary bypass is in the left femoral artery, right carotid artery, or aorta. Bicaval venous cannulation with caval tourniquets is required to accomplish complete isolation of the right atrium. Incision in the right atrium is made parallel to the atrioventricular groove away from the sulcus terminalis, which contains the sinoatrial node. A perimembranous VSD is located medial to the septal leaflet of the tricuspid valve, although the precise location depends on the specific subtype of perimembranous defect (Figure 22.2b).

For perimembranous defects, the conduction tissues are located in close proximity with the dorsal and caudal borders of the defect between the defect and coronary sinus (Figure 22.2c). These conduction

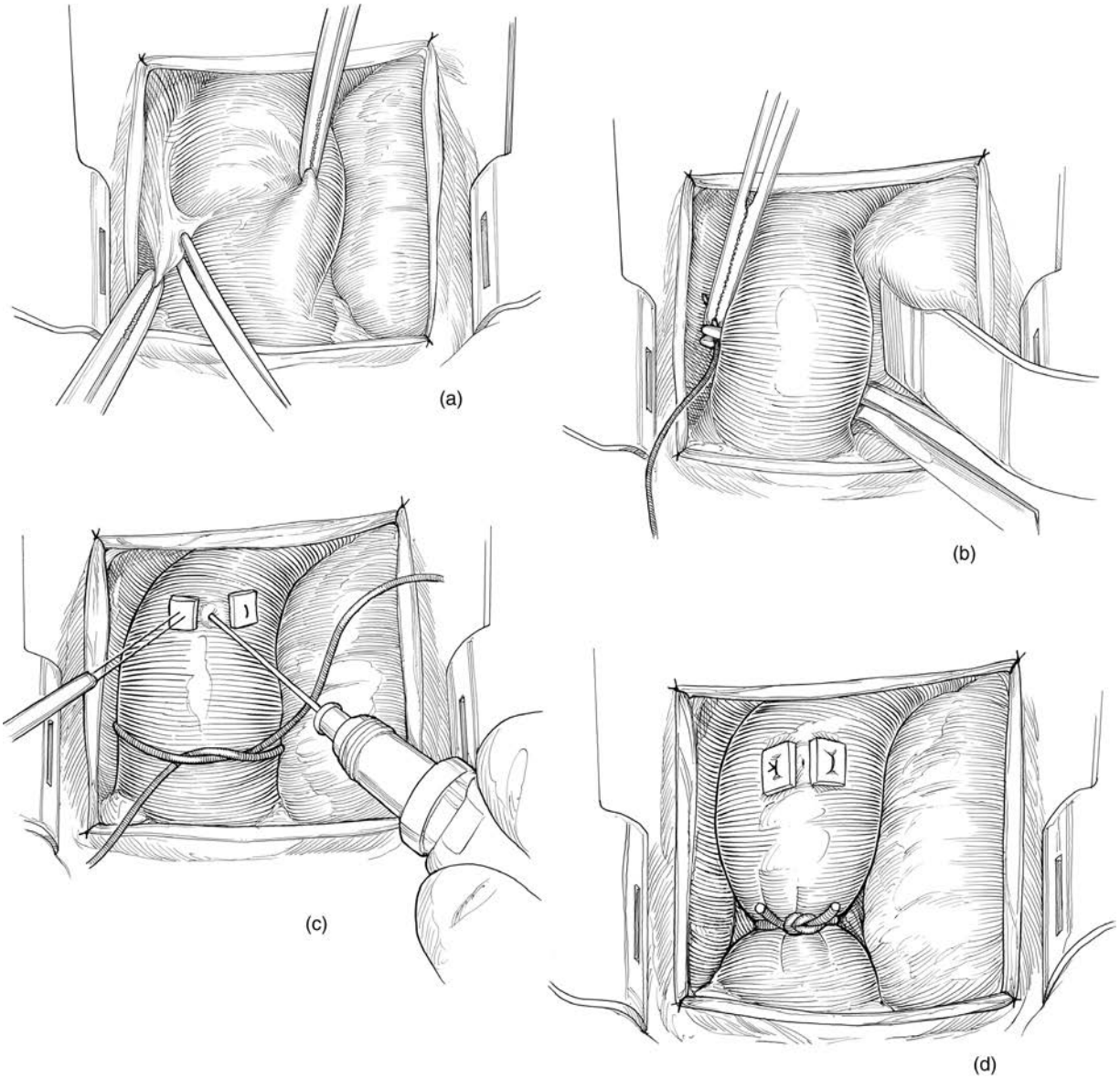


Figure 22.1 Pulmonary Artery Banding

tissues must be avoided to prevent atrioventricular block after surgery. The anatomy of conduction tissues in relation to different types of VSD should be reviewed prior to undertaking closure of a VSD [8]. The defect is exposed by gentle retraction of the septal tricuspid leaflet with a stay suture. The defect is closed with Daron velour or ePTFE patch, tailored to the size and shape of the defect. The patch should

Box 22.1 Guidelines for Pulmonary Artery Banding

- Increase systemic arterial pressure to just reach a plateau.
- Decrease pulmonary arterial systolic pressure toward 30 mm Hg measured by a distal pulmonary artery catheter.
- Decrease in pulmonary artery oxygen saturation collected from distal pulmonary artery catheter by 5% to 10%.
- Decrease peak shunt velocity determined by transesophageal or epicardial Doppler echocardiography toward, but not less than, 2.5 M/s.

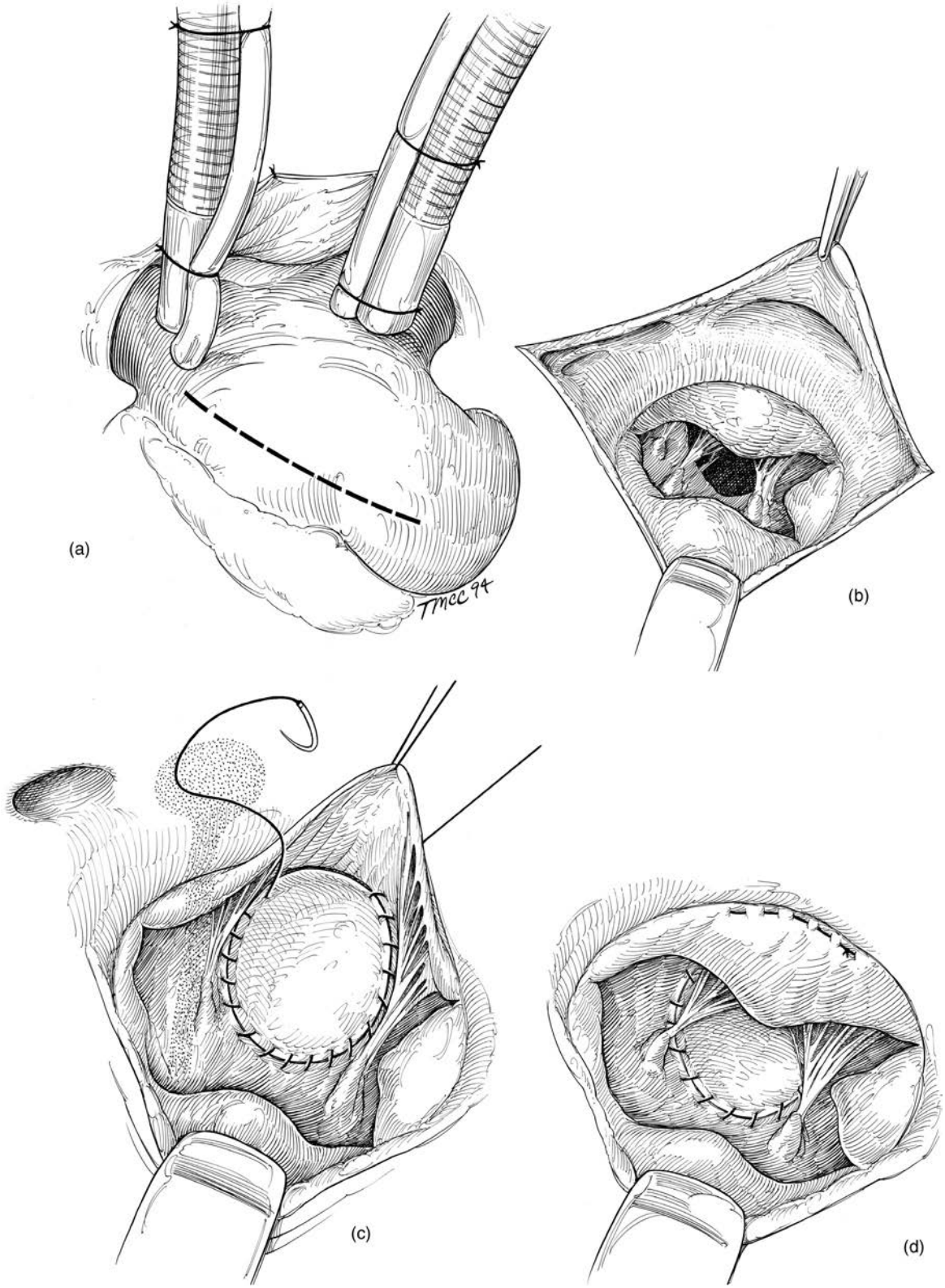


Figure 22.2 Ventricular Septal Defect Repair

be wider than the defect caudally and dorsally to facilitate avoidance of the conduction tissues by extending the patch beyond the caudodorsal border of the patch. The patch can be sutured with a continuous or interrupted mattress pattern of 5-0 synthetic suture, double-armed with small semi-circle needles. Interrupted mattress suture pattern has the advantage of allowing preplacement of the sutures in the defect and then "parachuting" the patch into the defect. Mattress sutures should be buttressed with small pledgets. Sutures should not be placed in the caudodorsal border of the defect to avoid injury to the conduction tissues. In this area, sutures should be places 3–5 mm away from the edge of the defect on the right ventricular side. The defect usually extends dorsally to the tricuspid valve annulus, and in this case, sutures are placed through the base of the tricuspid leaflet and tied on the atrial side of the leaflet (Figure 22.2d). The left heart should be de-aired prior to final closure of the patch. After the defect is closed, the right atrial incision is closed with a continuous mattress pattern oversewn with a continuous suture pattern.

Perimembranous infundibular (defect seen with tetralogy of Fallot), infundibular muscular, and doubly committed subarterial defects can be corrected through an incision in the right ventricular outflow tract. In this case, a sternotomy approach provides best access to the right ventriculotomy. Bicaval venous cannulation should be employed with placement of the caudal vena caval cannula through the right atrial appendage. Doubly committed subarterial defects may require repair of the aortic valve to correct aortic insufficiency [5].

Outcomes

A majority of dogs and cats with VSD have restrictive shunt flow and a favorable prognosis without intervention [6]. Long-term or lifetime palliation of hemodynamically significant VSD can be achieved with pulmonary artery banding in dogs and cats. Palliation of VSD has been reported in dogs and cats for up to 7 years after surgery [9], which is consistent with our experience. The most serious complication is overtightening of the band, leading to reversal of shunt flow and hypoxemia. This complication can be avoided initially by careful assessment of hemodynamic parameters during banding. Progressive narrowing of the band and late reversal of shunt flow can result from growth of the animal, progressive fibrosis of the band, progressive right ventricular hypertrophy, or any combination of these.

Definitive closure of VSD under cardiopulmonary bypass is considered curative in humans so long as it is undertaken prior to development of severe pulmonary hypertension or secondary myocardial failure. Although outcome results in animals have been limited to case reports, experience at our institution after definitive closure of VSD in dogs has been associated with long-term favorable outcome. Transcatheter and hybrid surgical-transcatheter closure of VSD has been reported in dogs and will likely emerge as option in the future [10–12].

Atrial and Atrioventricular Septal Defect

Atrial septal defects (ASD) occur in the septum secundum. Types of secundum ASD include *patent foramen ovale*, *fossa ovalis type ASD* (or *ostium secundum ASD*), *sinus venosus ASD,* and *coronary sinus ASD* [13]. Fossa ovalis type ASD may have a complete septal rim or may extend dorsally, leaving little or no dorsal rim. Large fossa ovalis ASD may also extend caudally to the orifice of the caudal vena cava. In this case, the right pulmonary veins will be close to the rim of the defect. Sinus venosus ASD are located cranial and dorsal in the atrial septum at the junction of the cranial vena cava and are usually accompanied by *anomalous pulmonary venous return* of one or two of the right cranial right pulmonary veins into the cranial vena cava. Coronary sinus ASD or unroofed coronary sinus results from an incomplete separation between the coronary sinus and the left atrium due to the persistence of a left cranial vena cava.

Atrioventricular septal defects (AVSD) represent a spectrum of malformations that involve the septum primum, the inlet portion of ventricular septum, and the atrioventricular (AV) valves [14]. This group of defects is subdivided into partial, intermediate, and complete forms. *Partial AVSD* consists of an ostium primum type ASD in the ventral atrial septum just dorsal to separately formed atrioventricular valves. The defect is generally large and hemodynamically unrestrictive. Partial AVSD is typically associated with a malformation of the mitral valve, described as either a *cleft* in the septal mitral leaflet or as *trileaflet* mitral valve. This malformation can cause mitral regurgitation of varying severity. The tricuspid valve may also have a trileaflet structure that is not typical of the normal tricuspid valve in animals. *Complete AVSD* (formally known as an endocardial cushion defect) consists of an ostium primum ASD above, a VSD below, and a single AV valve that is common to the right and left ventricle. The single AV valve has six leaflets suspended above the unattached crest of the ventricular septum. *Intermediate AVSD* lies between the partial and complete forms consisting of an ostium primum

ASD, VSD, and incomplete separation of the mitral and tricuspid valves.

Pathophysiology

Uncomplicated ASD is associated with left-to-right shunt that places a volume overload the right side of the heart. Often, ASD is well tolerated until animals reach maturity and beyond. Progressive right heart dilation and right-sided congestive heart failure result, depending on defect size and shunt ratio ($Q_P/Q_S > 3$). Bidirectional or right-to-left shunt cause varying degrees of oxygen-unresponsive hypoxemia and occur when right atrial pressures are increased by congestive heart failure, pulmonary hypertension, or concurrent right-sided structural heart defects (e.g., pulmonary stenosis). The atrial septal defect of partial AVSD is often large and hemodynamically significant. Partial AVSD may be accompanied by severe concurrent mitral regurgitation that adds a significant volume overload on the left heart and increases the likelihood of congestive heart failure early in life. A majority of animals with complete AVSD will die before reaching maturity.

Diagnosis

Animals with ASD can remain asymptomatic for several years. Large defects cause signs related to exercise intolerance, right heart failure, or chronic hypoxemia. The principal physical finding is a left cardiac base systolic murmur caused by high-velocity flow through the pulmonary valve (relative pulmonary stenosis). Partial AVSD may also have a murmur consistent with mitral regurgitation. Cyanosis may be present, particularly with exertion, depending on the degree of bidirectional shunting present. Thoracic radiographs reveal enlargement of the right heart and pulmonary vessels. Definitive diagnosis of ASD and AVSD is obtained by echocardiography. Large defects are readily visualized directly on two-dimensional echocardiography. Doppler echocardiography identifies the magnitude and direction of flow through defects.

Indications for Surgery

Intervention for ASD and AVSD is based on clinical signs and the hemodynamic significance of the defect. Indications for surgery include right ventricular dilation, activity intolerance, congestive heart failure, and evidence of progressive pulmonary arterial hypertension. In humans, a $Q_PQ_S > 3$ is considered indication for surgery [13]. Septal shunt flow velocity > 0.45 M/s suggests that shunt flow is hemodynamically significant [15]. Transcatheter occlusion of fossa ovalis ASD, either by transvascular or a hybrid transatrial approach, is an emerging option for small animals so long as the defect has an adequate rim dorsally [16]. Transcatheter closure of AVSD is generally not an option because of the lack of a ventral rim and proximity to the atrioventricular valves. Open surgical closure of secundum type ASD and partial AVSD can be undertaken with the aid of cardiopulmonary bypass. Pulmonary artery banding is not generally an option because of the risk for reversal of shunt flow through the defect.

Open Repair for ASD and AVSD

Open repair of ASD or partial AVSD is accomplished with the aid of cardiopulmonary bypass through a right fifth thoracotomy. Cannulation for cardiopulmonary bypass is as described for VSD. The defect is approached through a right atriotomy (Figure 22.3a). The septal defect is closed with ePTFE patch or autogenous pericardium. If pericardium is used it is harvested at the time that the pericardium is opened, cleaned, and placed on a saline-moistened towel. Handling of the pericardium is improved by trimming the pericardium and towel to their final shape together. The pericardial patch should be oriented with the serous surface leftward. The patch is placed with a simple continuous suture pattern of 5-0 polypropylene suture. For fossa ovalis type defects, the suture line is started in the dorsal-caudal corner of defect visualizing the office of the caudal vena cava and Eustachian valve. Deep stitches in this area should be avoided to prevent injury to the aorta. During closure, it is best not to aspirate blood below the rim of the defect to minimize entry of air into the left heart. Air should be removed from the left heart prior to closure of the patch. For sinus venosus defects, the cranial vena cava should be inspected for anomalous pulmonary veins prior to cannulation. If anomalous pulmonary veins are present, caval cannulation should be extended cranially to allow repair of the anomalous veins. This is accomplished by extending the patch closure to the right of the ostia of the anomalous veins, functionally leaving their flow on the leftward side of the patch. Partial AV septal defects require repair of the mitral valve prior to closing the septal defect. The cleft in the mitral valve is closed directly with interrupted 5-0 or 6-0 polypropylene sutures (Figure 22.3b). Mitral annuloplasty may be necessary if the mitral annulus is dilated. The atrioventricular node and conduction tissues are located in the atrial wall between the defect

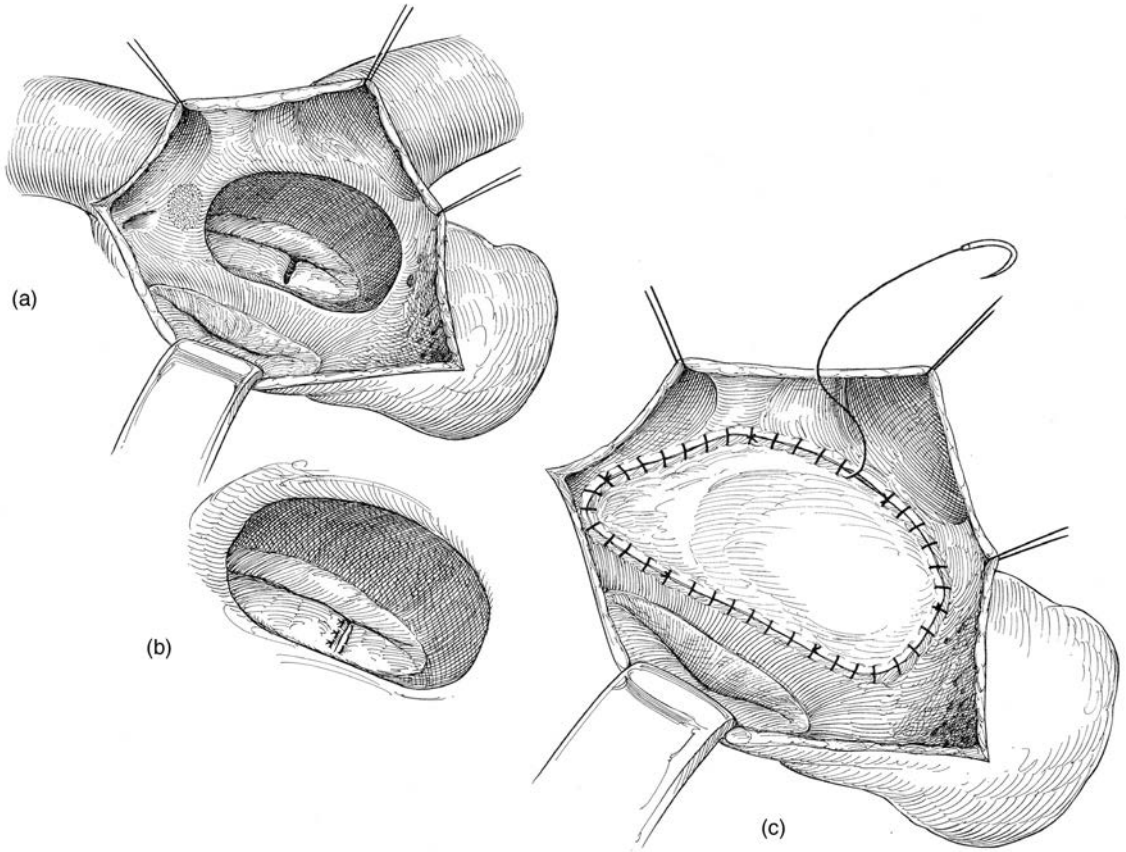


Figure 22.3 Atrioventricular Septal Defect Repair

and the coronary sinus. To avoid injury to these structures, the patch is extended along the atrioventricular valve annulus and then caudally around the coronary sinus, leaving it and the conduction tissues on the leftward side of the patch (Figure 22.3c). The right atrium is closed as described for VSD.

Outcomes

Successful surgical correction has been described for ASD and partial AVSD in dogs [17–19]. Open repair of ASD or AVSD can be considered curative assuming that pulmonary hypertension or secondary myocardial failure is not severe at the time of surgery. For partial AVSD, long-term outcome also depends on the severity of mitral regurgitation and the success of mitral valve repair. As with VSD, complete atrioventricular block is a possible operative complication of open repair.

Tetralogy of Fallot

The classic components of the "tetrad" of *tetralogy of Fallot* include a perimembranous infundibular VSD, an overriding aorta, right ventricular outflow tract obstruction, and right ventricular hypertrophy. All of these components are thought to result from a single morphologic anomaly; namely the cranial and leftward displacement of the infundibular septum [20]. The VSD of tetralogy of Fallot is large, unrestrictive, and in fibrous continuity with the aortic valve. The conduction tissues penetrate along the caudoventral margin of the defect. Displacement of the infundibular septum results in obstruction of the right ventricular outflow tract through the formation of a prominent subpulmonary muscular band(s) or sleeve. Formation of this subpulmonary muscular sleeve is the result of complex fusion of normal right ventricular structures (septoparietal and septomarginal trabeculations) and is a required element of tetralogy of Fallot. Valvular pulmonary stenosis sometimes, but not invariably, contributes to the outflow obstruction through varying degrees of leaflet dysplasia and annular hypoplasia. The main pulmonary artery may also be hypoplastic. Other cardiac defects can accompany tetralogy of Fallot. These include patent foramen ovale, secundum ASD, AVSD, patent ductus arteriosus, and anomalous origin of the coronary arteries.

Tetralogy of Fallot is the most common cyanotic heart defect in companion animals, but accounts for

only about 1% to 4% of cardiac malformations in dogs and cats overall [2–4]. Tetralogy occurs in cats and a variety of canine breeds including keeshonds, poodles, schnauzers, terriers, collies, and shelties. In keeshonds, tetralogy of Fallot is genetically transmitted as part of the spectrum of conotruncal defects [21].

Pathophysiology

From a functional standpoint, tetralogy of Fallot can be simplified into the combined effects of right ventricular outflow obstruction and a septal defect. The pathophysiologic consequences of tetralogy depend on the relative severity of these two defects. At one end of the spectrum are animals with a large septal defect and mild right outflow obstruction (often these are actually a combined VSD and valvular pulmonary stenosis rather than true tetralogy of Fallot). In these cases, the functional result is similar to a hemodynamically unrestricted VSD. At the other end of the spectrum are animals with severe right ventricular outflow tract obstruction and severe right-to-left shunt. The result is moderate to severe oxygen-unresponsive hypoxemia, cyanosis, exercise intolerance, and progressive polycythemia. A shortened life span can be expected due to complications associated with chronic hypoxemia, polycythemia, and sudden cardiac death. In the middle of the spectrum are animals where the right outflow obstruction and VSD are hemodynamically balanced, analogous to a VSD after pulmonary artery banding. Such defects may be well-tolerated for a period of time and do not warrant intervention. A majority of dogs and cats with tetralogy of Fallot present with clinical signs and have an unfavorable prognosis without intervention [22].

Diagnosis

Clinical findings at presentation for classic tetralogy of Fallot include moderate to severe exercise intolerance, exertional tachypnea, and syncope. The most prominent physical finding is moderate to severe cyanosis that is unresponsive to supplemental oxygen. Cyanosis typically worsens with activity or exercise. Systolic murmurs may be present at the left cardiac base and right cranial sternum. If polycythemia is severe, murmurs may be soft or not heard. Electrocardiogram usually shows a right axis shift in the frontal plan (deep S wave in leads I, II, and aVF) supportive of severe right ventricular hypertrophy. Thoracic radiographs show evidence of right ventricular enlargement and diminutive pulmonary vessels.

Two-dimensional echocardiography demonstrates all the elements of a tetralogy of Fallot, including right ventricular hypertrophy, subpulmonary right ventricular outflow obstruction, perimembranous VSD, and an overriding aorta. Doppler interrogation of the septal defect reveals the direction and magnitude of shunt flow.

Indications for Surgery

The long-term prognosis for tetralogy of Fallot depends on the severity of right-to-left shunt. Some animals with mild to moderate shunt may live for a period of time without intervention, although they will be moderately to severely exercise intolerant. Animals with resting cyanosis and progressive polycythemia will likely succumb to the effects of the defect early in life [22]. Surgery should be considered for animals with cyanosis to lessen clinical signs and prolong life. Debilitating exercise intolerance, polycythemia (hematocrit > 60%) and hypoxemia at rest (S_aO_2 < 75%) are objective indications for intervention. Animals with predominately left-to-right shunt may function reasonably well so long as the shunt flow remains low and does not cause heart failure, and so long as progressive right ventricular outflow obstruction does not reverse the shunt.

Palliative interventions for tetralogy of Fallot include isolated interventions for right outflow obstruction and/or creation of a systemic-to-pulmonary shunt distal to the outflow obstruction. In humans, these procedures have been employed for short-term palliation before corrective repair. In animals, palliative interventions can be regarded as definitive procedures for long-term palliation. The goal of palliative approaches is to increase pulmonary blood flow without creating an overwhelming left-to-right shunt. Interventions aimed at relieving right outflow obstruction alone risk overcorrection and creation of an overwhelming left-to-right shunt. From this standpoint, a transvascular balloon valvuloplasty or transventricular dilation valvuloplasty is indicated over a more definitive surgery such as a transannular patch valvuloplasty. Pulmonic dilation valvuloplasty is more likely to be effective if valvular pulmonary stenosis is a component of the defect.

Systemic-to-pulmonary shunts increase pulmonary blood flow to a predictable degree and thereby improve arterial oxygen saturation while lessening the risk of excessive pulmonary over circulation. Several systemic-to-pulmonic shunts have been devised for palliation of tetralogy of Fallot including the Blalock Taussig (left subclavian artery-to-pulmonary artery anastomosis), Potts (aorticopulmonary anastomosis), Waterston (aorta-to-right pulmonary artery anastomosis), and Glenn (vena caval-to-pulmonary arterial anastomosis) shunts [23]. Definitive open

correction for tetralogy of Fallot can be undertaken in dogs with curative intent.

Modified Blalock-Taussig Shunt

The Blalock-Taussig shunt is the most physiologically appropriate and technically feasible palliative procedure for tetralogy of Fallot in animals. The magnitude of increase in pulmonary blood flow is governed by the diameter of the left subclavian artery, which is known to be appropriate in humans and seems to work well in animals. The original Blalock-Taussig shunt consisted of dividing the left subclavian artery and performing an end-to-side anastomosis to the pulmonary artery. In animals, the left subclavian artery does not have sufficient length to reach the pulmonary without kinking at its origin on the aorta—therefore, a modification of the original procedure is necessary.

Two modifications of the classic Blalock-Taussig procedure can be used in animals. In one modification, the left subclavian artery is harvested a free autogenous vascular graft (Figure 22.4a). The

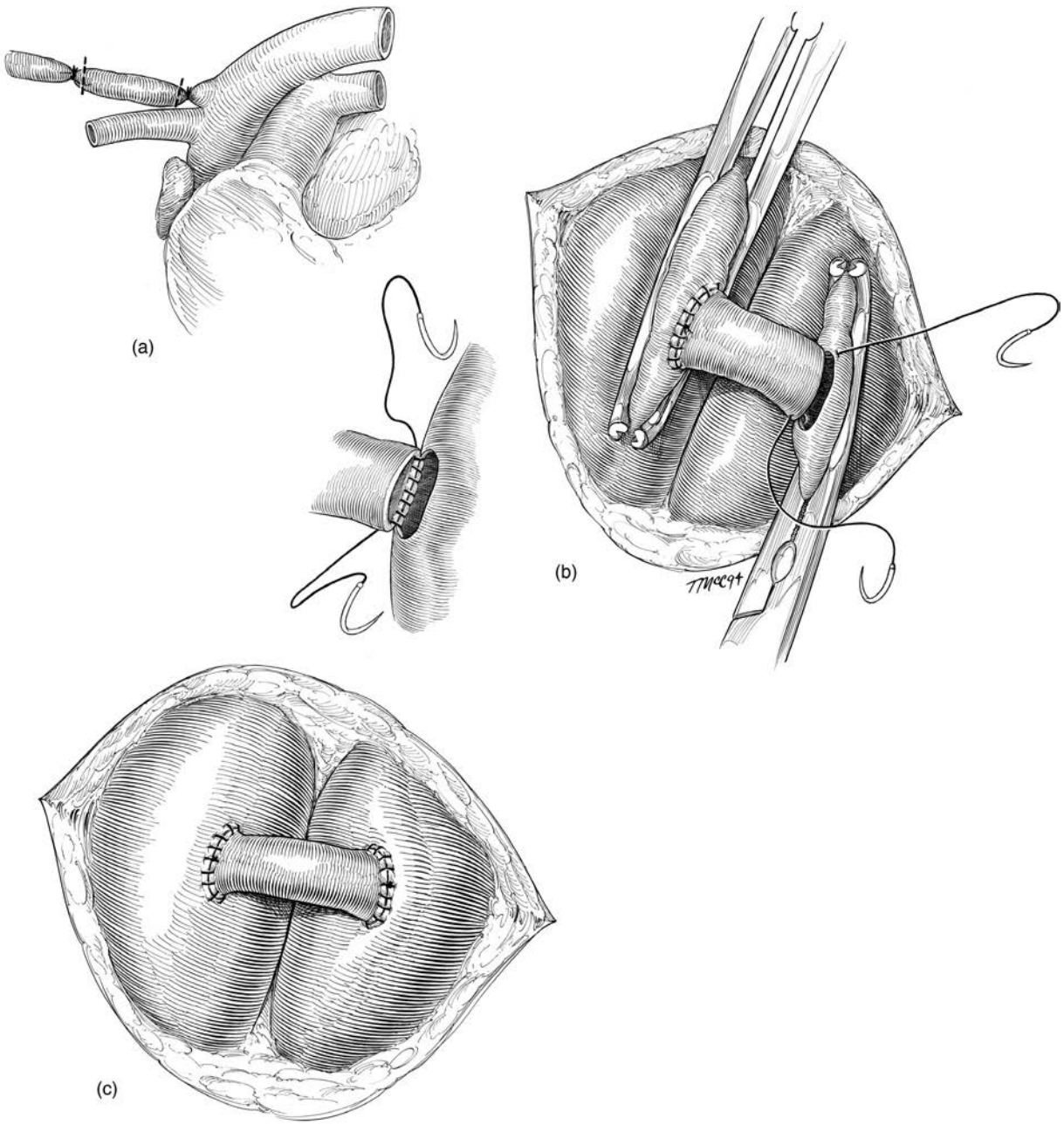


Figure 22.4 Modified Blalock-Taussig Shunt (Aorta to Pulmonary Artery)

pericardium is opened and tangential vascular clamps are placed on the ascending aorta and pulmonary artery (Figure 22.4b). Incisions are made within the vascular clamps and end-to-side anastomosis are performed using a simple continuous patterns of 5-0 or 6-0 polypropylene suture (Figure 22.4c). This modification has the advantage of not requiring a synthetic graft which is susceptible to thrombosis when small diameter grafts are required, and therefore may be preferred for small dogs (< 5 kg) and cats. A second modification inserts a 5 or 6 mm ePTFE vascular graft between the left subclavian artery and pulmonary artery. Vascular clamps are placed to isolate a segment of the left subclavian artery (Figure 22.5a). An end-to-side anastomosis is performed between the vascular graft and left subclavian artery. The shunt is completed by performing an end-to-side anastomosis between the vascular graft and pulmonary artery (Figure 22.5b). In this modification, pulmonary blood flow is still governed by the size of the subclavian artery and it has the advantage of preserving the subclavian artery. Both procedures are accomplished through a left fourth thoracotomy. The pericardium is opened and sutured to the thoracotomy incision. Heparin (150 U/kg) is administered intravenously just prior to starting the anastomoses.

Open Repair of Tetralogy of Fallot

Definitive repair of tetralogy of Fallot can be undertaken with the aid of cardiopulmonary bypass [24]. The preferred surgical approach in dogs is by median sternotomy [25]. Venous cannulation should be bicaval with direct cannulation of the cranial vena cava. The caudal vena cava cannula is introduced via the right atrial appendage. The repair is accomplished with the aorta crossclamped during cardioplegic arrest. In smaller animals where total circulatory arrest CPB will be employed, a single atriocaval cannula can be used for cooling/warming, and then it can be removed during repair of the VSD. The most technically feasible cardiac approach in animals is a longitudinal ventriculotomy in the right ventricular outflow tract (Figure 22.6a). The infundibular muscular band is excised with a scalpel (Figure 22.6b). Patch closure of the VSD is accomplished, taking care to avoid injury to the tricuspid valve, conduction tissues located in the caudoventral rim, and the aortic

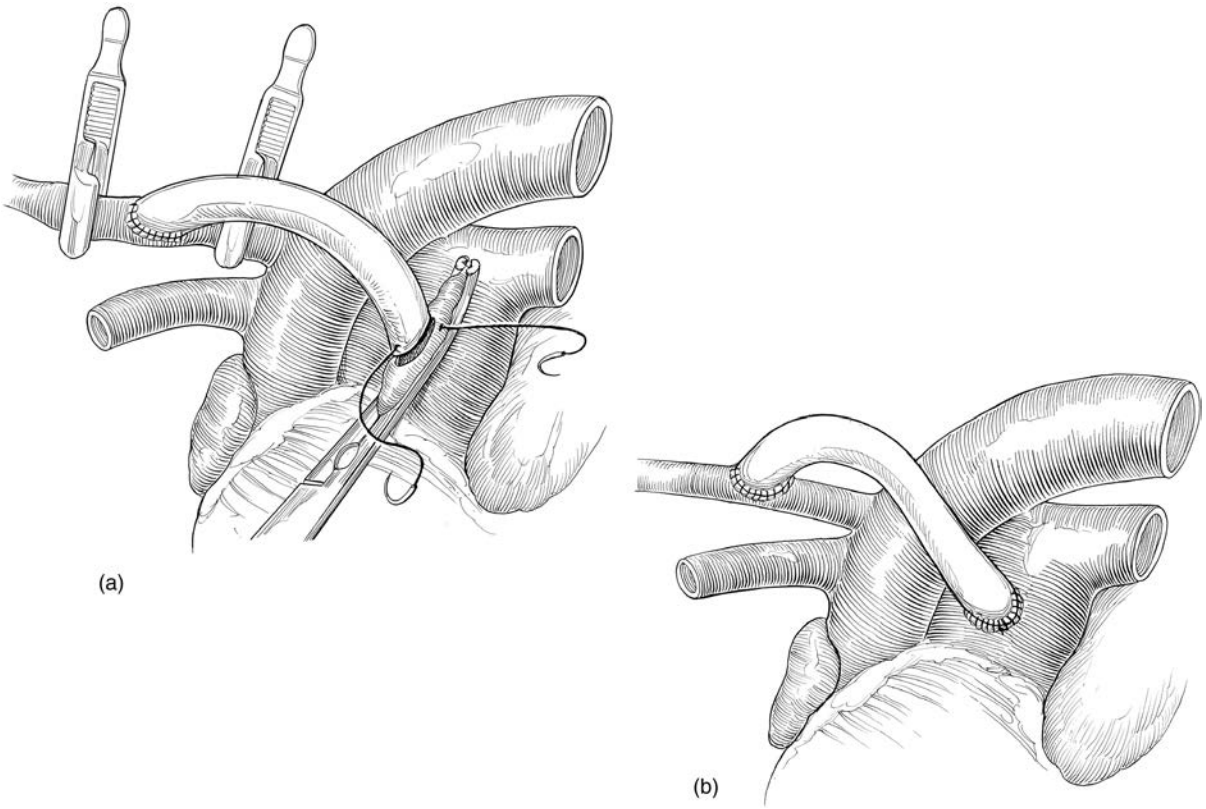


Figure 22.5 Modified Blalock-Taussig Shunt (Subclavian Artery to Pulmonary Artery)

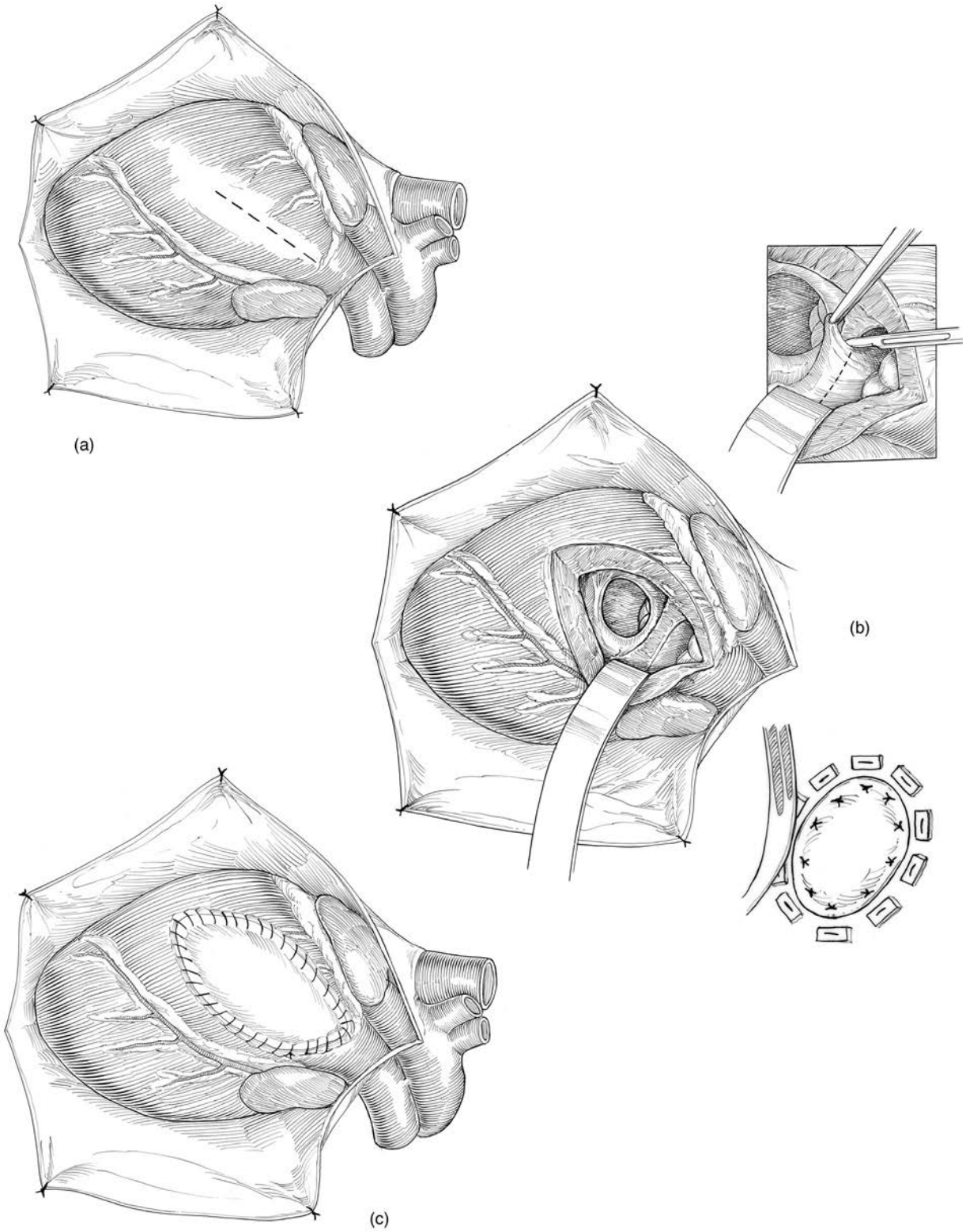


Figure 22.6 Tetralogy of Fallot Repair

valve dorsally. An oval patch-graft is inserted into the ventriculotomy incision to relieve the right ventricular outflow tract obstruction (Figure 22.6c). In cases where the outflow obstruction consists entirely of a subpulmonary fibromuscular band, the patch does not need to extend across the annulus of the pulmonic valve. If valvular pulmonic stenosis is also present, the pulmonic patch-graft is extended across the pulmonary valve annulus into the pulmonary artery.

Outcomes

Long-term palliation of tetralogy of Fallot by modified Blalock-Taussig shunt has been reported in dogs [26]. Successful definitive correction for tetralogy of Fallot has also been reported in dogs [25]. In our experience successful correction of tetralogy of Fallot in dogs is associated with resolution of clinical signs and a normal life expectancy after surgery.

References

1 Anderson RH, Sarwark A, Spicer DE, and Backer CL. 2014. Exercises in anatomy: The normal heart. *Multimedia Manual of Cardiothoracic Surgery*.

2 Buchanan JW. 1999. Prevalance of cardiovascular disorders. In: Fox PR, Sisson D, and Moise NS eds. *Textbook of Canine and Feline Cardiology*. pp. 457–70. Philadelphia: W.B. Saunders Co.

3 Oliveira P, Domenech O, Silva J, Vannini S, Bussadori R, and Bussadori C. 2011. Retrospective review of congenital heart disease in 976 dogs. *Journal of Veterinary Internal Medicine.* 25(3): 477–83.

4 Schrope DP. 2015. Prevalence of congenital heart disease in 76,301 mixed-breed dogs and 57,025 mixed-breed cats. *Journal of Veterinary Cardiology.* 17(3):192–202.

5 van Doorn C, and de Leval M. 2006. Ventricular septal defects. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 355–71. Chichester: John Wiley & Sons.

6 Bomassi E, Misbach C, Tissier R, Gouni V, Trehiou-Sechi E, Petit AM, Desmyter A, Damoiseaux C, Pouchelon JL, and Chetboul V. 2015. Signalment, clinical features, echocardiographic findings, and outcome of dogs and cats with ventricular septal defects: 109 cases (1992–2013). *Journal of the American Veterinary Medical Association.* 247(2):166–75.

7 Tsang VT, and Stark J. 2006. Pulmonary artery banding. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 262–70. Chichester: John Wiley & Sons.

8 Anderson RH, and Becker AE. 2006. Anatomy of ventricular septal defects and their conduction tissues. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 121–43. Chichester: John Wiley & Sons.

9 Eyster GE, Whipple RD, Anderson LK, Evans AT, and O'Handley P. 1977. Pulmonary artery banding for ventricular septal defect in dogs and cats. *Journal of the American Veterinary Medical Association.* 170(4):434–8.

10 Durham JA, Scansen BA, Bonagura JD, Schober KE, Cheatham SL, and Cheatham JP. 2015. Iatrogenic embolization and transcatheter retrieval of a ventricular septal defect occluder in a dog. *Journal of Veterinary Cardiology.* 17(4):304–13.

11 Saunders AB, Carlson JA, Nelson DA, Gordon SG, and Miller MW. 2013. Hybrid technique for ventricular septal defect closure in a dog using an Amplatzer(R) Duct Occluder II. *Journal of Veterinary Cardiology.* 15(3):217–24.

12 Bussadori C, Carminati M, and Domenech O. 2007. Transcatheter closure of a perimembranous ventricular septal defect in a dog. *Journal of Veterinary Internal Medicine.* 21(6):1396–400.

13 Stark J, and Tsang VT. 2006. Secumdum atrial septal defect and partial anomalous pulmonary venous return, In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 343–54. Chichester: John Wiley & Sons.

14 Pacifico AD. 2006. Atrio-ventricular septal defects. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 373–86. Chichester: John Wiley & Sons.

15 Marx GR, Allen HD, Goldberg SJ, and Flinn CJ. 1985. Transatrial septal velocity measurement by Doppler echocardiography in atrial septal defect: correlation with Qp:Qs ratio. *The American Journal of Cardiology.* 55(9):1162–7.

16 Sanders RA, Hogan DE, Green HW, 3rd, Hoyer MH, and Puppel DA. 2005. Transcatheter closure of an atrial septal defect in a dog. *Journal of the American Veterinary Medical Association.* 227(3): 430–4.

17 Akiyama M, Tanaka R, Maruo K, and Yamane Y. 2005. Surgical correction of a partial atrioventricular septal defect with a ventricular septal defect in a dog. *Journal of the American Animal Hospital Association.* 41(2):137–43.

18 Eyster GE, Anderson LK, Krehbeil JD, Whipple RD, Blanchard GL, Dickason J, Braden TD, Bohonowych R, and Harris F. 1976. Surgical repair of atrial septal defect in a dog. *Journal of the American Veterinary Medical Association.* 169(10):1081–4.

19 Monnet E, Orton EC, Gaynor J, Boon J, Peterson D, and Guadagnoli M. 1997. Diagnosis and surgical repair of partial atrioventricular septal defects in two dogs. *Journal of the American Veterinary Medical Association.* 211(5):569–72.

20 Anderson RH, Sarwark A, Spicer DE, and Backer CL. 2014. Exercises in anatomy: Tetralogy of Fallot. *Multimedia Manual of Cardiothoracic Surgery.*

21 Patterson DF, Pyle RL, Van Mierop L, Melbin J, and Olson M. 1974. Hereditary defects of the conotruncal septum in Keeshond dogs: pathologic and genetic studies. *The American Journal of Cardiology.* 34(2):187–205.

22 Chetboul V, Pitsch I, Tissier R, Gouni V, Misbach C, Trehiou-Sechi E, Petit AM, Damoiseaux C, Pouchelon JL, Desquilbet L, et al. 2016. Epidemiological, clinical, and echocardiographic features and survival times of dogs and cats with tetralogy of Fallot: 31 cases (2003–2014). *Journal of the American Veterinary Medical Association.* 249(8):909–17.

23 van Doorn C, and de Leval M. 2006. Systemic-to-pulmonary artery shunts. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 251–60. Chichester: John Wiley & Sons.

24 Bove E, L., and Hirsch JC. 2006. Tetralogy of Fallot. In: Stark J, de Leval M, and Tsang VT eds. *Surgery for Congenital Defects*. pp. 399–410. Chichester: John Wiley & Sons.

25 Orton EC, Mama K, Hellyer P, and Hackett TB. 2001. Open surgical repair of tetralogy of Fallot in dogs. *Journal of the American Veterinary Medical Association.* 219(8):1089–93.

26 Brockman DJ, Holt DE, Gaynor JW, and Theman TE. 2007. Long-term palliation of tetralogy of Fallot in dogs by use of a modified Blalock-Taussig shunt. *Journal of the American Veterinary Medical Association.* 231(5):721–6.

23

Cor Triatriatum and Double-Chambered Right Ventricle

E. Christopher Orton

Obstructive congenital cardiac defects include cardiac valve stenosis and nonvalvular congenital obstructions. Stenosis of the cardiac valves can be subvalvular, valvular, or supravalvular. Surgical management of these conditions are reviewed in Chapters 20 and 21. Nonvalvular congenital obstructions in small companion animals are uncommon and include cor triatriatum and double-chamber right ventricle.

Cor Triatriatum

Cor triatriatum is an uncommon congenital anomaly in which the right atrium (cor triatriatum dexter) or left atrium (cor triatriatum sinister) is divided into two chambers by a persistent fold of tissue or membrane creating a heart with three atria (cor triatriatum). Typically, the proximal chamber (sinus venarum) receives all or part of the venous blood flow, while the distal chamber contains the fossa ovalis, atrioventricular valve, and auricular appendage. The membrane may have a single restrictive opening, multiple small openings (fenestrations), or may be intact (imperforate). To date, cor triatriatum dexter (CTD) has only been reported in dogs, whereas cor triatriatum sinister (CTS) has only been reported in cats. In dogs, CTD has an apparent breed predilection the chow breed [1].

Cor triatriatum causes obstruction of venous blood flow to the right or left heart resulting in moderate to severe venous congestion and edema proximal to the obstruction. In the typical anomaly of CTD in dogs, the obstruction occurs between the caudal vena cava and the tricuspid valve leaving cranial vena caval and azygous venous flow unobstructed. It is possible for a portion of the fossa ovalis to be proximal to the membrane resulting in right-to-left shunt flow through the foramen ovale [2]. Physical examination reveals evidence of venous distension in the veins of the hind limb and abdomen, but not the neck and forelimb. Usually, severe medically refractory ascites is present at a young age. A cardiac murmur is typically not heard. Abdominal ultrasound confirms presence of ascites and shows evidence of post-hepatic venous congestion (distended and noncollapsing hepatic veins). CTD is confirmed by transthoracic echocardiography, which demonstrates the persistent membrane within the right atrium caudal to the tricuspid valve and turbulent venous flow within the true right atrial chamber on color-flow Doppler. In cats with CTS, the membrane is dorsal to the fossa ovalis and ostium of the left auricle, distinguishing it from supravalvular mitral stenosis. The anomaly typically obstructs pulmonary venous flow from the entire lung and causes severe pulmonary edema. The typical presentation is a kitten with pulmonary edema. Anomaly is confirmed by transthoracic echocardiography. The prognosis is generally guarded to poor without intervention.

CTD in dogs can be corrected surgically by membranectomy during venous inflow occlusion [1, 3, 4]. Surgical correction during cardiopulmonary bypass has also been reported [5]. More recently interventional approaches using balloon dilation, dilation with cutting balloon and/or balloon-expandable stent have been reported for successful relief of CTD in dogs [2,6,7]. In cats, relief of CTS in cats has been reported by echocardiography-guided dilation of the membrane with a valve dilator [8] or cutting balloon [9] using a hybrid transatrial approach. Successful treatment of CTS by membranectomy during cardiopulmonary bypass has also been reported in a cat [10].

Membranectomy for Cor Triatriatum Dexter

Surgical correction of CTD can be accomplished by membranectomy performed through a right atriotomy during brief circulatory arrest with inflow occlusion. The surgery is performed through a right fifth thoracotomy. Tourniquets are placed around the cranial and caudal vena cavae and azygous vein for

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inflow occlusion (Figure 18.1). The pericardium is opened ventral to the phrenic nerve. The location of the membrane may be evident on the outside of the heart by an indentation and/or by palpation through the atrial free wall. Stay sutures are placed in the lateral atrial wall to control the atriotomy incision during inflow occlusion (Figure 23.1a). The atrium is opened transversely across the defect during inflow occlusion. The membrane is incised to partially excised (Figure 23.1b). A tangential vascular clamp is used to close the atriotomy as inflow occlusion is discontinued (Figure 23.1c). Venous blood should flow from the atriotomy as the clamp is placed to remove air from the heart. The atriotomy is closed with a continuous horizontal mattress pattern oversewn with a simple continuous pattern (Figure 23.1d).

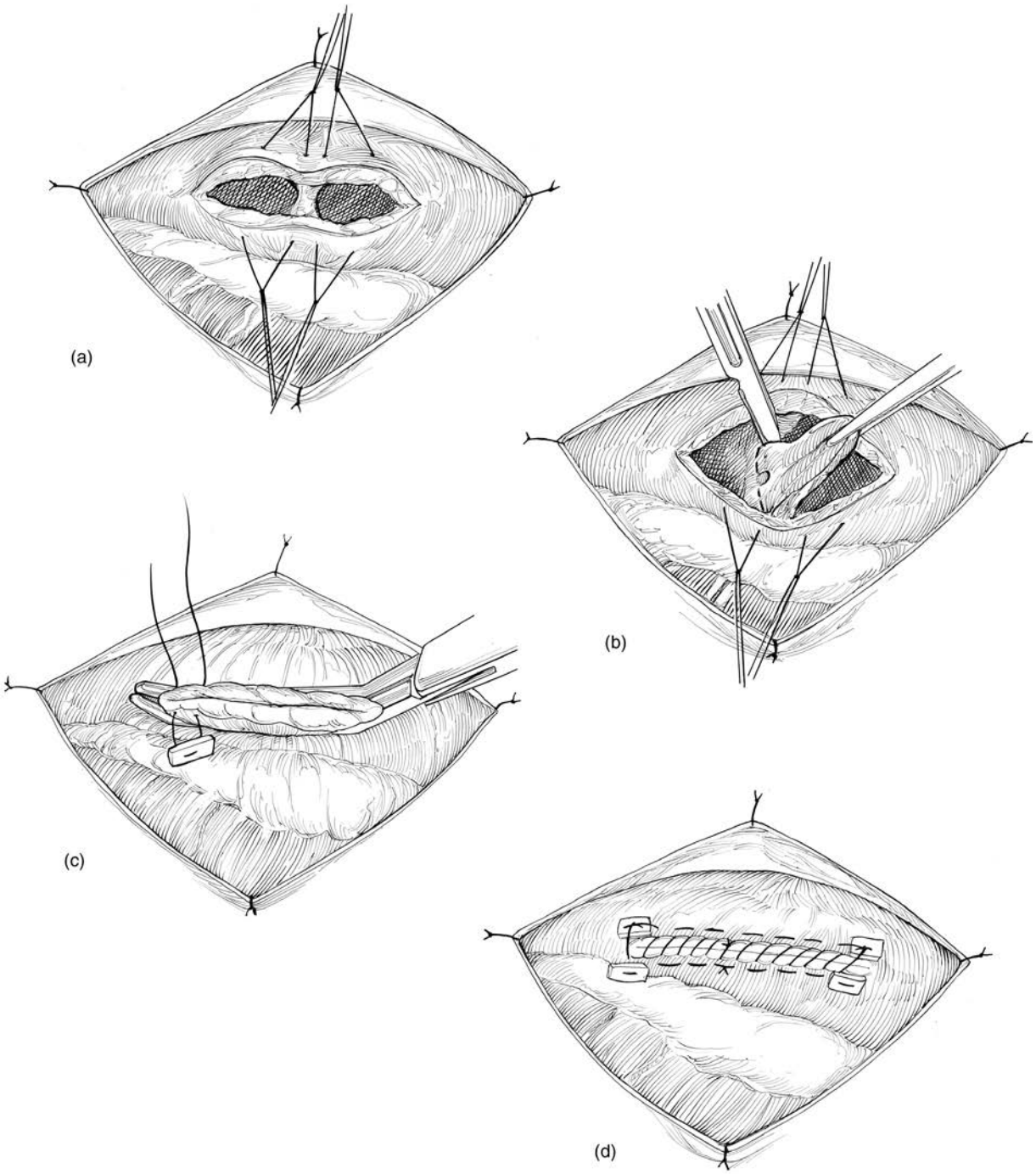

Figure 23.1 Cor Triatriatum Dexter Repair

Expected Outcomes

Clinical outcomes for cor triatriatum surgery in dogs and cats are limited to case reports and small case series. Reports in dogs with CTD undergoing membranectomy with venous inflow occlusion report resolution of ascites without late-term complications [1, 3, 4, 11]. Surgery is considered curative. Successful palliation with resolution of pulmonary edema in two cats with CTS by echo-guided hybrid transatrial dilation with a valve dilator or cutting balloon has been reported [8, 9].

Double-Chambered Right Ventricle

Double-chambered right ventricle (DCRV), also known as right mid-ventricular obstruction, is a congenital cardiac anomaly reported in dogs and cats [12–14]. The anomaly consists of a fibromuscular band at the junction of the inflow and outflow portions of the right ventricle and is distinguished from primary infundibular stenosis by its close proximity to the tricuspid valve annulus and apparatus. The obstruction causes thickening of the right ventricular wall proximal to band giving the right ventricle a "double-chambered" appearance. Two morphologic forms of the fibromuscular band have been described and both forms are reported in dogs [14]. In the oblique form, the fibromuscular band extends to the cardiac apex, whereas in the transverse form the band extends to the free wall at the base of the right ventricular outflow tract. In humans DCRV is associated with a high prevalence of concurrent ventricular septal defect. Concurrent ventricular septal defect is reported in dogs and cats, but presence of this defect is not a required element of the diagnosis. Other congenital cardiac anomalies are often reported in dogs and cats with DCRV including tricuspid valve dysplasia, atrial septal defect, patent foremen ovale, cor triatriatum, and aortic valve anomalies.

The pathophysiology of DCRV is similar to that of pulmonary stenosis. The defect causes pressure overload and concentric hypertrophy of the inflow, but not outflow, portion of the right ventricle. Animals with DCRV are at risk for sudden cardiac death or progressive right-sided congestive heart failure. The malformation is sometimes accompanied by primary or secondary tricuspid regurgitation, which increases the likelihood of heart failure and worsens the prognosis.

Animals with DCRV may be asymptomatic at presentation or may exhibit activity intolerance, syncope, or symptoms referable to right heart failure (abdominal distension or dyspnea secondary to pleural effusion). A systolic murmur is heard that localizes to either the left or right hemithorax, or both. Distention of the jugular veins may be present in animals exhibiting early right-sided congestion. Cyanosis may be present secondary to a right-to-left shunting patent foramen ovale. Echocardiography demonstrates a fibromuscular at the base of the right ventricular outflow tract in close proximity to the tricuspid valve. Marked concentric hypertrophy of the interventricular septum (IVS) and right ventricular free wall with flattening of the IVS is present. Right ventricular wall thickening is absent in the infundibular region distal to the obstruction. Color-flow Doppler demonstrates turbulent flow in the right ventricular outflow tract distal to the obstruction. Spectral Doppler reveals high velocity flow across the obstruction with calculated pressure gradients across the obstruction of 50 to 200 mm Hg.

Indications for surgery are essentially the same as for pulmonary stenosis, although dogs with DCRV may be less tolerant of moderate pressure gradients compared to dogs with pulmonary stenosis [13]. A pressure gradient > 70 mm Hg, concurrent tricuspid regurgitation, presence of exercise/activity intolerance, syncope, or early congestive heart failure are reasons to consider surgery. Dogs with medically refractory heart failure or atrial fibrillation are poor candidates for surgery. Surgical correction of DCRV is performed with the aid of cardiopulmonary bypass. The surgery consists of excision of the muscular band through a longitudinal ventriculotomy, with or without patch-grafting of the ventriculotomy [13, 14]. Because of the proximity of the tricuspid valve apparatus and difficulty in achieving complete excision of the obstructing fibromuscular band, our preference is to close the ventriculotomy with a patch-graft.

Surgery for Double-Chambered Right Ventricle

Surgical correction of DCRV in dogs is accomplished during cardiopulmonary bypass. A sternotomy approach provides the best access to the defect. Standard arterial and bicaval venous cannulation for cardiopulmonary bypass are performed. The procedure can be performed with or without aortic crossclamp and standard cardioplegia. The pericardium is opened and sutured to the incision to expose the right ventricular outflow tract (Figure 23.2a). The location of the obstruction can be determined by palpation of the right ventricle. A longitudinal incision spanning the obstruction is made in the right ventricular outflow tract (Figure 23.2b). The fibromuscular band is excised, taking care not to injure the tricuspid valve apparatus, which lies in close proximity to the band (Figure 23.2 inset). The

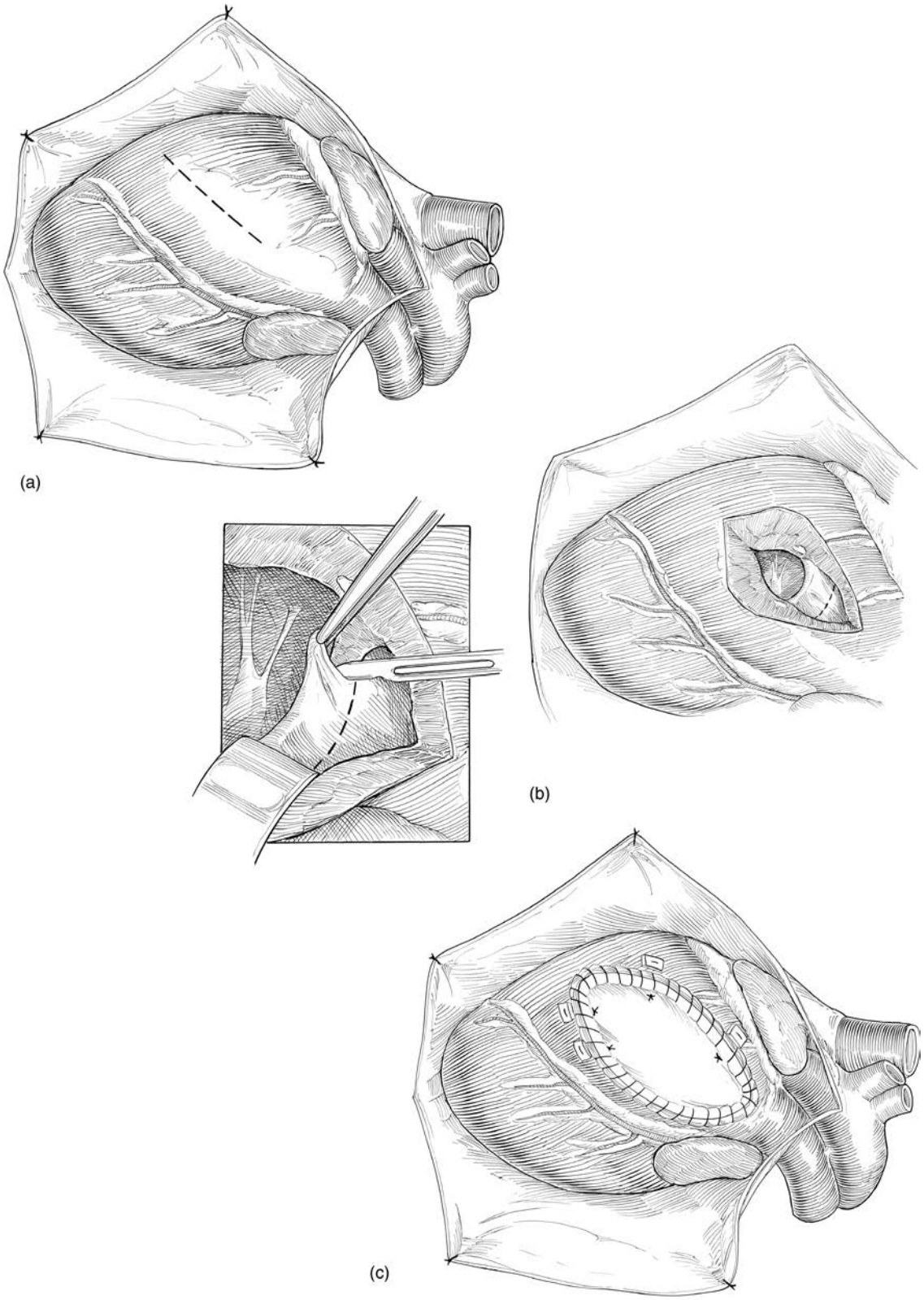

Figure 23.2 Double-Chambered Right Ventricle Repair

ventriculotomy is closed by insertion of an oval-shaped ePTFE patch into the incision to enlarge the effective cross-sectional area across the defect (Figure 23.2c). Suture pattern for the closure can be a simple continuous pattern of 4-0 polypropylene or ePTFE suture. Incisional bleeding is controlled by placement of pledget-reinforced mattress sutures.

Expected Outcome

Surgical correction of DCRV in dogs with follow-up periods of up to 5 years has been reported [13–15]. Long-term resolution of signs related to exercise intolerance, syncope, or early congestive heart failure is a reasonable expectation for surgery. Continued medical management of congestive heart failure may be necessary in dogs with established heart failure at the time of surgery. As with other obstructive cardiac defects, the risk for sudden cardiac death is likely diminished but not eliminated by surgery.

Successful correction of DCRV in a cat by partial excision of the band and placement of a pericardial patch-graft during venous inflow occlusion has been reported [16]. This cat had resolution of chylothorax for a period of 3 years after surgery at the time of the report.

References

1 Fossum TW, and Miller MW. 1994. Cor triatriatum and caval anomalies. *Seminars Veterinary Medicine and Surgery (Small Animal).* 9(4):177–84.

2 Johnson MS, Martin M, De Giovanni JV, Boswood A, and Swift S. 2004. Management of cor triatriatum dexter by balloon dilatation in three dogs. *The Journal of Small Animal Practice.* 45(1):16–20.

3 Tobias AH, Thomas WP, Kittleson MD, and Komtebedde J. 1993. Cor triatriatum dexter in two dogs. *Journal of the American Veterinary Medical Association.* 202(2):285–90.

4 Mitten RW, Edwards GA, and Rishniw M. 2001. Diagnosis and management of cor triatriatum dexter in a Pyrenean mountain dog and an Akita Inu. *Australian Veterinary Journal.* 79(3):177–80.

5 Tanaka R, Hoshi K, Shimizu M, Hirao H, Akiyama M, Kobayashi M, Machida N, Maruo K, and Yamane Y. 2003. Surgical correction of cor triatriatum dexter in a dog under extracorporeal circulation. *The Journal of Small Animal Practice.* 44(8):370–3.

6 Barncord K, Stauthammer C, Moen SL, Hanson M, and Gruenstein DH. 2016. Stent placement for palliation of cor triatriatum dexter in a dog with suspected patent foramen ovale. *Journal of Veterinary Cardiology.* 18(1):79–87.

7 Leblanc N, Defrancesco TC, Adams AK, Atkins CE, Tou SP, Fudge JC, and Keene BW. 2012. Cutting balloon catheterization for interventional treatment of cor triatriatum dexter: 2 cases. *Journal of Veterinary Cardiology.* 14(4):525–30.

8 Wander KW, Monnet E, and Orton EC. 1998. Surgical correction of cor triatriatum sinister in a kitten. *Journal of the American Animal Hospital Association.* 34(5):383–6.

9 Stern JA, Tou SP, Barker PC, Hill KD, Lodge AJ, Mathews KG, and Keene BW. 2013. Hybrid cutting balloon dilatation for treatment of cor triatriatum sinister in a cat. *Journal of Veterinary Cardiology.* 15(3):205–10.

10 Borenstein N, Gouni V, Behr L, Trehiou-Sechi E, Petit A, Misbach C, Raillard M, Retortillo JL, Pouchelon JL, Pierrel A, et al. 2015. Surgical Treatment of Cor Triatriatum Sinister in a Cat Under Cardiopulmonary Bypass. *Veterinary Surgery.* 44(8):964–9.

11 Chanoit G, Bublot I, and Viguier E. Transient tricuspid valve regurgitation following surgical treatment of cor triatriatum dexter in a dog. 2009. *The Journal of Small Animal Practice.* 50(5):241–5.

12 Koffas H, Fuentes VL, Boswood A, Connolly DJ, Brockman DJ, Bonagura JD, Meurs KM, Koplitz S, and Baumwart R. Double chambered right ventricle in 9 cats. 2007. *Journal of Veterinary Internal Medicine.* 21(1):76–80.

13 Martin JM, Orton EC, Boon JA, Mama KR, Gaynor JS, and Bright JM. 2002. Surgical correction of double-chambered right ventricle in dogs. *Journal of the American Veterinary Medical Association.* 220(6):770–4, 68.

14 Fukushima R, Tanaka R, Suzuki S, Hamabe R, Machida N, Nakao S, Saida Y, Takashima K, Matsumoto H, Koyama H, et al. 2011. Epidemiological and morphological studies of double-chambered right ventricle in dogs. *The Journal of Veterinary Medical Science.* 73(10): 1287–93.

15 Willard MD, and Eyster GE. 1981. Double-chambered right ventricle in two dogs. *Journal of the American Veterinary Medical Association.* 178(5): 486–8.

16 Brockman DJ, Borer KE, Baines SJ, Hughes D, and Luis Fuentes V. 2009. Partial right ventriculectomy using the incised patch technique to treat double chambered right ventricle and chylothorax in a cat. *Veterinary Surgery.* 38(5):631–5.

24

Cardiac Neoplasia

E. Christopher Orton

Cardiac neoplasms account for a relatively small portion of neoplasms in dogs and cats [1, 2]. From a surgical perspective, they can be classified as primarily intracavitary, mural, or extracardiac. Clinical manifestations of cardiac tumors, depending on their type and location, include pericardial effusion with acute or chronic cardiac tamponade, obstruction of blood flow within the heart, and/or interference with the electromechanical function of the heart. Hemangiosarcoma is the most common cardiac neoplasia in dogs, followed by aortic body tumors [1]. A variety of other primary neoplasms have been reported in dogs, including fibrosarcoma, chondrosarcoma, rhabdomyosarcoma, ectopic thyroid carcinoma, fibroma, myxoma, and myxosarcoma. Lymphosarcoma and metastatic neoplasia are the most frequent cardiac neoplasias in cats [2].

Surgery plays a limited role in the management of cardiac neoplasia. Most mural cardiac tumors are not amendable to surgical resection, particularly if they involve or cross the atrioventricular junction. An exception is right auricular hemangiosarcoma, which can be palliated in selected cases by surgical resection of the right auricle (auriculectomy). Intracavitary masses typically manifest by obstructing blood flow within the heart. Some of these masses are amendable to surgical resection with venous inflow occlusion or cardiopulmonary bypass if they have a discrete attachment on echocardiography. Tumors at the heart base (chemodectoma, paraganglioma, ectopic thyroid carcinoma) are the most common extracardiac neoplasms in dogs. Manifestations of heart base masses include pericardial effusion and obstruction of blood flow by extracardiac compression and/or invasion of cardiac structures at the base of the heart (vena cavae, right or left atrium, pulmonary arteries). Surgery plays a very limited role in the management of heart base tumors.

Hemangiosarcoma

Hemangiosarcoma (HSA) is the most common cardiac neoplasia in dogs accounting for about two-thirds of cardiac neoplasia in this species [1]. The right atrial appendage is the most frequent cardiac site for cardiac HSA, although it can occur in any part of the heart, including the heart base. The most common clinical presentation for right auricular HSA is acute or chronic cardiac tamponade, resulting from intrapericardial hemorrhage or effusion. Echocardiography confirms pericardial bleeding or effusion and usually demonstrates a mass on the right auricle. Micrometastasis is assumed to be present in virtually all cases at the time of diagnosis, and this neoplasia is regarded as universally fatal. Multiple suspected primary sites, including the heart and spleen, are reported to occur in as many as 25% of cases of canine HSA. Pericardiocentesis provides temporary relief from acute or chronic tamponade. Complete staging is indicated before considering further surgical intervention. Staging should include imaging of the thorax and abdomen, complete blood count, and coagulation profile. Anemia and/or coagulopathy is present in up to 50% of cases, with obvious implications for surgery. Excision of the right auricular mass and subtotal pericardiectomy can be considered in selected cases. Median survival time after excision of right atrial hemangiosarcoma without adjuvant chemotherapy is reported to be approximately 4 months [3]. Survival times are significantly extended by adjuvant chemotherapy if clean surgical margins at the primary surgery site can be achieved [3–5]. Pericardiectomy or pericardial window without surgical excision of the primary tumor is controversial, as it risks major unchecked bleeding into the pleural space. Pericardiectomy without excision of the primary mass does not prolong survival [6].

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Right Auriculectomy

Resection of the right auricle (auriculectomy) can be considered for palliation of hemangiosarcoma. The primary goals are to reduce the tumor burden to a microscopic level and to decrease the risk of recurrent hemorrhage from the tumor site. The surgery is usually combined with a subtotal pericardiectomy and can be performed from either a sternotomy or right thoracotomy. Masses that are primarily restricted to the auricular appendage are the most amendable to resection. Masses that cross the atrioventricular groove are not resectable. A tangential vascular clamp is placed across the base of the right auricle (Figure 24.1a). The auricular appendage and mass are excised distal to the vascular clamp, and a continuous mattress suture line is placed on the proximal side to the clamp (Figure 24.1b). The vascular clamp is removed and the incision is oversewn with a continuous suture pattern (Figure 24.1c). Autogenous pericardial patch-grafting, with or without venous inflow occlusion, has been used to extend the area of resection onto the right atrial wall or control bleeding at the surgical site [7–9].

Intracavitary Cardiac Masses

Intracavitary masses that grow large enough to obstruct blood flow within the heart are occasionally seen in dogs. Dogs with these tumors typically present with clinical signs related to congestive heart failure and/or syncope. These tumors typically exhibit varying degrees of mobility within the heart and have a stalk-like attachment to internal cardiac structures on echocardiography. The most common locations are the right atrium, in close proximity to the tricuspid valve, and the right ventricular outflow tract, in close proximity to the pulmonary valve. The reported tumor types for these intracavitary masses include myxoma, thyroid carcinomas, and chondrosarcoma [10–13]. Removal of well-defined intracavitary masses with the aid of venous inflow occlusion or cardiopulmonary bypass is possible in selected cases.

Removal of Intracavitary Cardiac Masses

Surgical excision of intracavitary cardiac masses can be accomplished during brief circulatory arrest with venous inflow occlusion. Cardiac masses that appear as well-defined ball-like masses with a degree of motility within the cardiac lumen and a definable stalk-like attachment are most amendable to surgical excision during brief circulatory arrest. Tumors that are closely associated with the tricuspid valve are removed via a right thoracotomy and right atriotomy. Tourniquets are placed on the vena cavae and azygous vein for inflow occlusion (Figure 18.1a). The pericardium is opened ventral to the phrenic nerve and sutured to the incision. Loose stay sutures are preplaced on both sides of the planned atriotomy site to control the incision during inflow occlusion (Figure 24.2a). Inflow occlusion is initiated, the right atrium is opened, and the mass is grasped and excised (Figure 24.2b). The heart is de-aired as the atriotomy is closed with a tangential vascular clamp (Figure 24.2c). After cardiac function is restored, the atriotomy is closed with a pledget-reinforced continuous mattress suture pattern of 4-0 suture. The vascular clamp is removed and the incision is oversewn with a simple continuous pattern (Figure 24.2d).

Intracavitary tumors in the right ventricular outflow tract are approached via a sternotomy and removed through a direct right outflow ventriculotomy and/or pulmonary arteriotomy. The surgical technique is as described above for removing tumors through a right atriotomy. The ventriculotomy/pulmonary arteriotomy is temporarily closed with a tangential vascular clamp. The ventriculotomy is then closed with interrupted pledget-reinforced mattress sutures.

Heart Base Tumors

The majority of heart base tumors in dogs are aortic body tumors [14–16]. Ectopic thyroid neoplasia accounts for 5% to 10% of canine heart base tumors [17]. Other reported types of heart base tumor include mesenchymoma and hemangiosarcoma [5, 18]. Aortic body tumors occur most often in older brachycephalic dogs, including boxers, English bulldogs, Boston terriers, and also German shepherds [1, 16, 19]. Chronic hypoxia associated with residence at altitude has been postulated to stimulate development of chemodectoma in dogs and humans [19, 20]. This mechanism is also thought to explain the higher incidence in brachycephalic breeds.

Aortic body tumors are located at the base of the heart between the outer wall of the ascending aorta and surrounding cardiac structures including the pulmonary arteries, right atrium, left atrium, or any combination of these. A strong presumptive diagnosis of aortic body tumor can be made based on this typical location; however, retrospective analysis suggests that presumptive diagnosis of cardiac tumors is only moderately accurate [21].

Aortic body tumors are highly vascular, slow growing, and moderately locally invasive. Initial growth

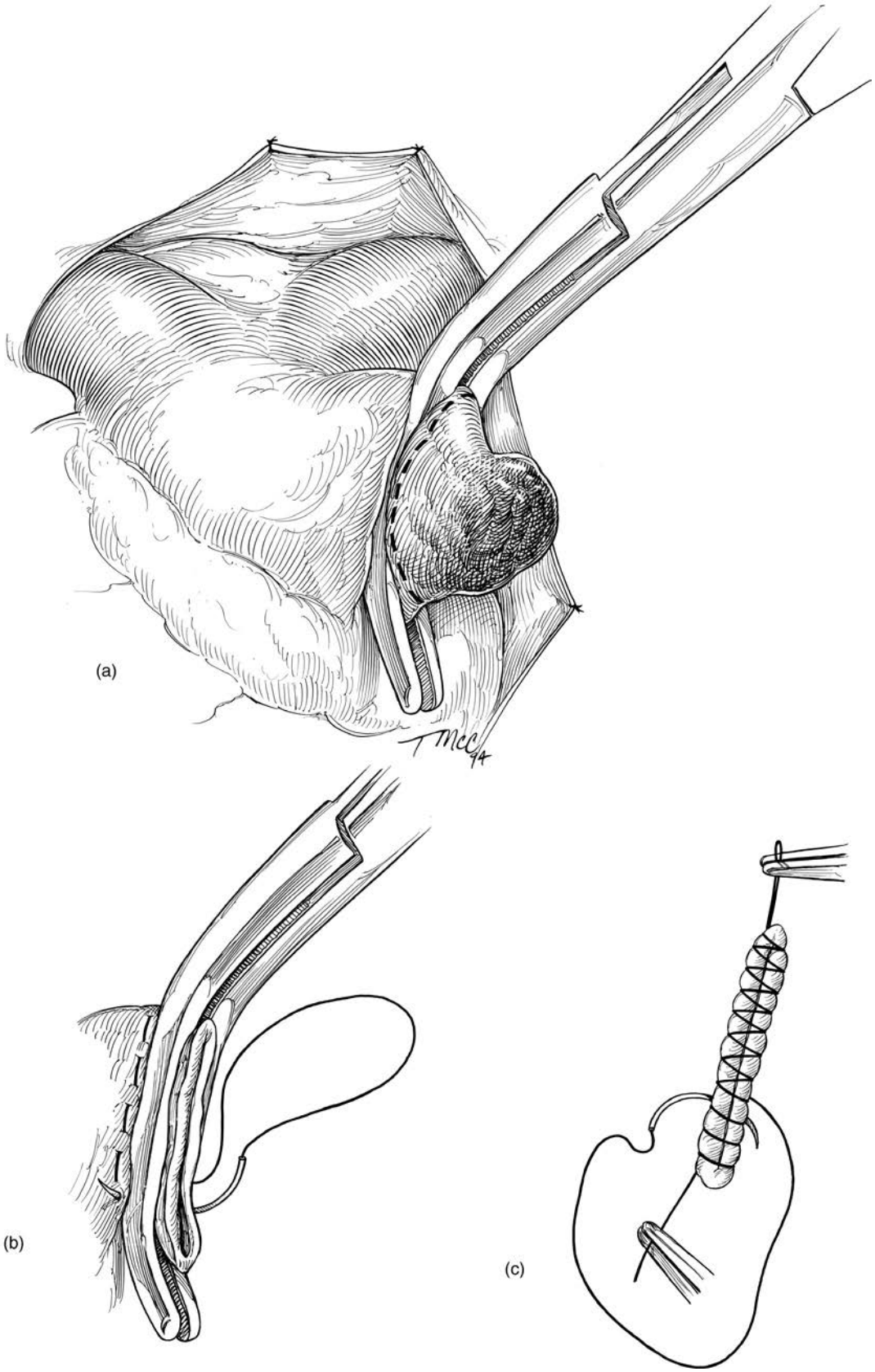

Figure 24.1 Right Auriculectomy

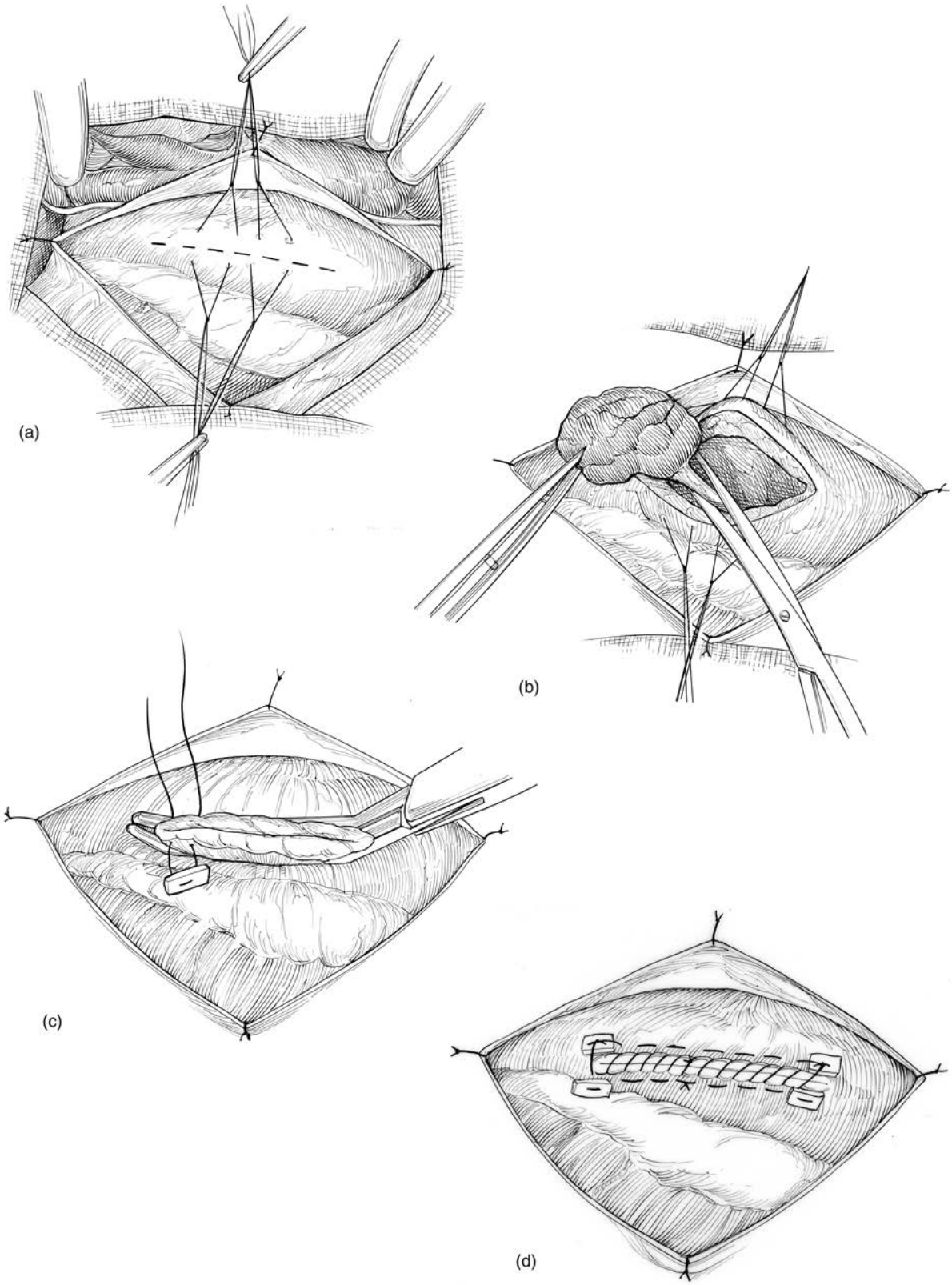

Figure 24.2 Intracavitary Cardiac Mass Removal

of aortic body tumors displaces surrounding cardiac structures without causing adverse effects on cardiac function. Late in their course, aortic body tumors can grow into cardiac chambers, eventually resulting in obstruction to blood flow and clinical signs related to heart failure. Aortic body tumors also cause pericardial effusion, and this is the most likely reason for clinical signs early in their course. Aortic body tumors are sometimes discovered early as an incidental finding on thoracic radiography or echocardiography performed for other reasons. Surgical resection plays a limited role in management of heart base tumors. Resection of small heart base tumors is generally not indicated due to their very slow growth and the high morbidity associated with attempts at surgical resection. Morbidity includes bleeding and injury to the vagal nerves, causing laryngeal paralysis and/or megaesophagus. Large heart base tumors that have reached the point of obstructing blood flow are not amendable to surgical resection. Pericardiectomy or pericardial window prolongs survival in dogs with aortic body tumors and should be considered as a palliative procedure, regardless of whether pericardial effusion is present [14, 15]. Stereotactic radiation therapy is an emerging option for dogs with heart base tumors [22]. In selected cases, obstructed blood flow can be relieved by intracavitary stenting of the vena cavae or pulmonary arteries.

References

1 Ware WA, and Hopper DL. 1999. Cardiac tumors in dogs: 1982–1995. *Journal of Veterinary Internal Medicine.* 13(2):95–103.

2 Tilley LP, Bond B, and Patnaik AK. 1981. Cardiovascular tumors in the cat. *Journal of the American Animal Hospital Association.* 17: 1009–21.

3 Weisse C, Soares N, Beal MW, Steffey MA, Drobatz KJ, and Henry CJ. 2005. Survival times in dogs with right atrial hemangiosarcoma treated by means of surgical resection with or without adjuvant chemotherapy: 23 cases (1986-2000). *Journal of the American Veterinary Medical Association.* 226(4): 575–9.

4 Mullin CM, Arkans MA, Sammarco CD, Vail DM, Britton BM, Vickery KR, Risbon RE, Lachowicz J, Burgess KE, Manley CA, et al. 2014. Doxorubicin chemotherapy for presumptive cardiac hemangiosarcoma in dogsdagger. *Veterinary Comparative Oncology.* 14(4):e171–83.

5 Yamamoto S, Hoshi K, Hirakawa A, Chimura S, Kobayashi M, and Machida N. 2013. Epidemiological, clinical and pathological features of primary cardiac hemangiosarcoma in dogs: a review of 51 cases. *The Journal of Veterinary Medical Science.* 75(11):1433–41.

6 Dunning D, Monnet E, Orton EC, and Salman MD. 1998. Analysis of prognostic indicators for dogs with pericardial effusion: 46 cases (1985–1996). *Journal of the American Veterinary Medical Association.* 212(8):1276–80.

7 Verbeke F, Binst D, Stegen L, Waelbers T, de Rooster H, and Van Goethem B. 2012. Total venous inflow occlusion and pericardial auto-graft reconstruction for right atrial hemangiosarcoma resection in a dog. *Canadian Veterinary Journal.* 53(10):1114–8.

8 Morges M, Worley DR, Withrow SJ, and Monnet E. 2011. Pericardial free patch grafting as a rescue technique in surgical management of right atrial HSA. *Journal of the American Animal Hospital Association.* 47(3):224–8.

9 Brisson BA, and Holmberg DL. 2001. Use of pericardial patch graft reconstruction of the right atrium for treatment of hemangiosarcoma in a dog. *Journal of the American Veterinary Medical Association.* 218(5):723–5.

10 Bright JM, Toal RL, and Blackford LA. 1990. Right ventricular outflow obstruction caused by primary cardiac neoplasia. Clinical features in two dogs. *Journal of Veterinary Internal Medicine.* 4(1):12–6.

11 Bracha S, Caron I, Holmberg DL, O'Grady MR, O'Sullivan LM, Brisson BA, and Stalker MJ. 2009. Ectopic thyroid carcinoma causing right ventricular outflow tract obstruction in a dog. *Journal of the American Animal Hospital Association.* 45(3): 138–41.

12 Worley DR, Orton EC, and Kroner KT. 2016. Inflow Venous Occlusion for Intracardiac Resection of an Occluding Right Ventricular Tumor. *Journal of the American Animal Hospital Association.* 52(4): 259–64.

13 Kohnken R, Durham JA, Premanandan C, and Scansen BA. 2015. Aortic chondroid neoplasia in two Labrador Retriever dogs. *Journal of Veterinary Cardiology.* 17(4):314–20.

14 Ehrhart N, Ehrhart EJ, Willis J, Sisson D, Constable P, Greenfield C, Manfra-Maretta S, and Hintermeister J. 2002. Analysis of factors affecting survival in dogs with aortic body tumors. *Veterinary Surgery.* 31(1):44–8.

15 Vicari ED, Brown DC, Holt DE, and Brockman DJ. 2001. Survival times of and prognostic indicators for

dogs with heart base masses: 25 cases (1986–1999). *Journal of the American Veterinary Medical Association.* 219(4):485–7.

16 Hayes HM, and Sass B. 1988. Chemoreceptor neoplasia: a study of the epidemiological features of 357 canine cases. *Zentralbl Veterinarmed A.* 35(6): 401–8.

17 Constantino-Casas P, Rodriguez-Martinez HA, and Gutierrez Diaz-Ceballos ME. 1996. A case report and review: the gross, histological and immunohistochemical characteristics of a carcinoma of ectopic thyroid in a dog. *British Veterinary Journal.* 152(6):669–72.

18 Gomez-Laguna J, Barranco I, Rodriguez-Gomez IM, Blanco B, Guil-Luna S, Carrasco L, and de las Mulas JM. 2012. Malignant mesenchymoma of the heart base in a dog with infiltration of the pericardium and metastasis to the lung. *Journal Comparative Pathology.* 147(2-3):195–8.

19 Patnaik AK, Liu SK, Hurvitz AI, and McClelland AJ. 1975. Canine chemodectoma (extra-adrenal paragangliomas)—A comparative study. *The Journal of Small Animal Practice.* 16(12): 785–801.

20 Hayes HM. 1975. An hypothesis for the aetiology of canine chemoreceptor system neoplasms, based upon an epidemiological study of 73 cases among hospital patients. *The Journal of Small Animal Practice.* 16(5):337–43.

21 Rajagopalan V, Jesty SA, Craig LE, Gompf R. 2013. Comparison of presumptive ecocardiographic diagnosis of cardiac tumors in dogs. *Journal of Veterinary Internal Medicine.* 27(5):1092–6.

22 Rancilio NJ, Higuchi T, Gagnon J, and McNiel EA. 2012. Use of three-dimensional conformal radiation therapy for treatment of a heart base chemodectoma in a dog. *Journal of the American Veterinary Medical Association.* 241(4):472–6.

25

Epicardial Pacemaker Implantation

Eric Monnet

Pacemaker therapy is indicated mostly for the treatment of symptomatic bradycardia in small animal medicine [1–4]. Other applications in humans include treatment of tachycardia, atrial fibrillation, and ventricular fibrillation. Pacemaker implantation has also been used for resynchronization therapy in patients with severe left ventricular dilation.

Atrioventricular (AV) block, sick sinus syndrome and permanent atrial standstill are the most common conditions treated in veterinary medicine [3–5]. By maintaining the heart rate above a certain limit, the cardiac output will be improved. Persistent atrial standstill is the result of a muscular dystrophy syndrome involving the myocardium of the atria and ventricles. It can also affect the scapulo-humeral skeletal muscles. Springer spaniels are predisposed [6]. Atrial standstill is characterized by the absence of P-waves on the electrocardiogram. Sick sinus syndrome results from degeneration and malfunction of the sinus node. Combinations of bradycardia, tachycardia, and long sinus pauses are seen on the ECG. Third-degree AV block occurs when conduction in the AV node and/or His bundle is blocked. Intrinsic disease of the AV node or His bundle induces a complete failure of the conduction from the atria to the ventricles [5].

Dogs with bradyarrhythmias typically present with exercise intolerance and syncope. Occasionally, they will present with signs related to congestive heart failure. On physical examination, heart rate is very low (30 to 40 bpm). Blood work should be performed to rule out electrolyte imbalance that could interfere with cardiac electrical activity. Thoracic radiographs and echocardiography are indicated to evaluate cardiac dilation, cardiac function, and other thoracic pathology.

Epicardial Pacing

Cardiac pacing can be accomplished by placement of an endocardial or epicardial lead. Endocardial pacing is accomplished by a transvenous placement of an endocardial and is preferred in most cases because it does not require thoracic surgery. However, endocardial pacing is sometimes not feasible due to patient size or relative contraindication due to risk of lead thrombosis from hypercoagulation syndromes. In this case, epicardial pacing can be accomplished by surgical placement of epicardial lead(s) on the surface of the heart. Electrical impulses are produced by a generator and delivered to the epicardium to induce depolarization and contraction. A pulse generator and a lead with an epicardial electrode are required to complete the circuit.

Pulse Generator

The pulse generator must produce an electrical impulse at the appropriate time to capture the myocardium and avoid interfere with intrinsic rhythms. To accomplish this requires both sensing and impulse-generating functions. The sensing function recognizes intrinsic cardiac electrical activity. The sensing unit in the generator couples with an amplifying unit to provide sensing information to the generator. The generator delivers electrical impulses of varying amplitude and duration to capture the myocardium. These functions are programmable after implantation. The generator is powered by lithium batteries.

Ideally, the generator should recognize changing levels of physical activity to be able to adapt its impulse rate to the needs of the patient [7]. Rate

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responsive pacemakers detect physiological or biophysical parameters affected by the metabolic demand. Most generators detect movement with a piezo-electric crystal. Vibrations of the surrounding tissue activate the crystal that will induce incremental augmentation of the heart rate up to a predetermined limit. Temperature, mixed venous oxygen saturation, and accelerometers have also been used to adjust heart rate with exercise.

A five-letter code is used to describe the function of a pacemaker. The first letter describes the chamber that is being paced (V: ventricle, A: atrium, D: both). The second letter describes the chamber that is being sensed (V: ventricle, A: atrium, D: both). The third letter describes the response of the pacemaker to the sensing of intrinsic electrical activity in the chamber sensed. It can be an inhibition of the pacemaker or triggering an electrical impulse to capture the paced chamber (I: inhibition, T: triggering, D: both). The fourth letter indicates that the generator is rate responsive (R: rate responsive) [2, 8]. The fifth letter is used for treatment of tachycardia. Most pacemakers used in veterinary medicine are VVIR if implanted with an epicardial lead on the left ventricle. When electrodes are placed in the atria and ventricle, the function is usually DDDR.

Lead Wire

The lead connects the pulse generator to the myocardium [9]. The lead includes a connector to connect the lead to the pulse generator, conductors, insulation, and one or two electrodes. Epicardial leads can be unipolar or bipolar. With a unipolar lead the electrical circuit is completed from the distal electrode in the epicardium, through the body of the patient, and back to the metallic casing of the pulse generator. The electrode in contact with the epicardium is the cathode. The casing of the generator is the anode. With bipolar leads, both the cathode and anode are in contact with the epicardium. Therefore the circuit is shorter which in turn reduces the total impedance of the circuit and the amount of energy used. Also bipolar electrodes are subject to less interference from other electrical activity such as muscle contraction.

The epicardial electrodes are fixed to the epicardium either with sutures or with a screw-in mechanism. The screw-in mechanism is more traumatic to the epicardium and has a tendency to induce arrhythmias in the postoperative period. Usually, screw-in electrodes require 2.5 turns to be well anchored in the myocardium (Figure 25.1). Sutured electrodes are anchored with mattress sutures of 4-0 monofilament nonabsorbable suture placed in the myocardium (Figure 25.2). One epicardial unipolar electrode provides for unipolar pacing, or a second epicardial lead can be added to accomplish bipolar pacing.

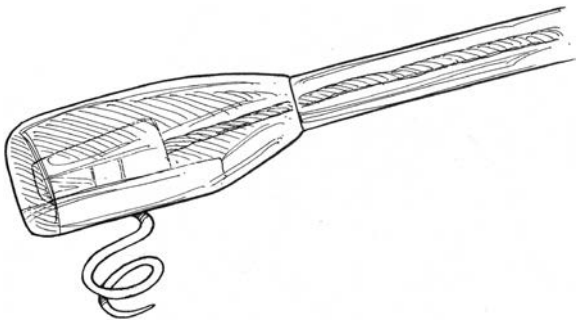

Figure 25.1 Epicardial Electrode—Screw-In Type

Epicardial leads are usually fixed to the left ventricle for ventricular capture and sensing. An electrode can also be attached to the right atrium for sensing to accomplish dual chamber pacing [6]. Atrial electrodes must be the suture-type electrode, since the wall of the atrium is too thin to accept a screw-in electrode.

Epicardial Pacemaker Implantation

Epicardial leads can be implanted with a mini-thoracotomy or a transdiaphragmatic approach after a cranial laparotomy. A thoracotomy is required if a right atrial electrode is placed for dual-chamber pacing. Thoracoscopy can be used for implantation of screw-in electrodes over the left ventricle.

Induction of general anesthesia may further slow a bradycardia or even suppress the escape rhythm, in the case of third-degree AV block. Thus, some form of temporary external pacing is usually indicated during implantation. This can be accomplished with external electrodes applied on each side of the thoracic cavity. This technique allows pacing of the heart; however, the electrical stimulation will capture the muscles of the thoracic wall and induce contraction of the thorax, which can interfere with the surgery.

Temporary pacing with a transvenous endocardial lead is also possible. These endocardial leads typically have a small balloon at the end of the lead to help flow-direct the electrode to the apex of the right ventricle. Fluoroscopy can also be used to direct the lead. Chemical stimulation with isoproterenol can be used to increase heart rate. Dopamine or dobutamine (5 mcg/kg/min) can be used to increase arterial blood pressure. However, chemical stimulation can be unpredictable, since the myocardium may escape

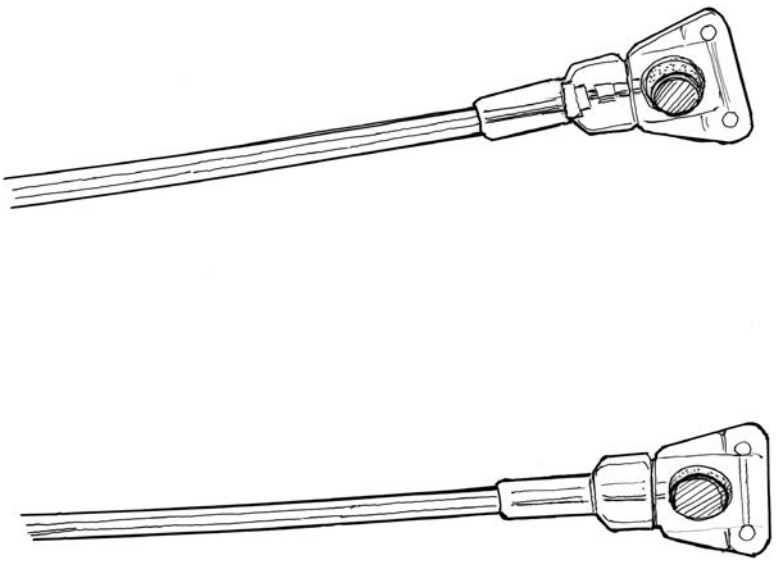

Figure 25.2 Epicardial Electrode—Suture Type

the chronotropic stimulation from inotropic drugs. In patients with third-degree AV block, it is paramount not to use lidocaine before the pacemaker and the lead are implanted because it can suppress the escape rhythm.

Transdiaphragmatic Approach

After a cranial midline laparotomy, Balfour retractors are placed to widely open the abdominal cavity (Figure 25.3a). A pocket to house the pacemaker is created in the transverse abdominal muscle caudal to the last rib on the left abdominal wall. Electrocautery can be used to create the pocket. After the electrode and the pacemaker are connected, electrocautery should be avoided. The diaphragm is opened on midline to expose the pericardium (Figure 25.3b). Stay sutures are placed on the diaphragm for retraction. The pericardium is opened approximately 2 cm to expose the apex of the left ventricle. Additional stay sutures are placed to stabilize the pericardium and the heart. The cranial interventricular (paraconal) coronary artery is identified and avoided as it wraps around the apex of the left ventricle. If unipolar stimulation is used a single epicardial electrode is placed either with the screw-in mechanism or with a 4-0 monofilament nonabsorbable suture. The electrode is sutured to the epicardium with a mattress suture using the tip of the electrode to reinforce the suture by tying it over the tip of the electrode. If a bipolar stimulation will be used, a second unipolar electrode is placed in similar fashion.

The lead is then connected to the pacemaker. The lead is secured in the pacemaker with a screw and a calibrated wrench. The pacemaker is then placed in the pocket created between transverse and internal abdominal oblique muscles (Figure 25.3c). The electrical circuit is completed when the generator makes contact with the body wall. If bipolar pacing is used, a Y connector will be used to connect the two electrodes to the pacemaker. If a sensing lead is placed on the atrium then it is connected to the sensing terminal on the pulse generator. The pocket is closed with 4-0 monofilament absorbable suture. A thoracostomy tube is placed and the diaphragm closed with a 4-0 monofilament nonabsorbable suture. It is important to keep a small loop of lead in the thorax to reduce tension on the lead/myocardial interface during the cardiac and respiratory cycle. Also, it is also important to avoid damage the insulation on the lead while suturing the diaphragm and the pocket. A puncture in the lead will induce leakage of electrical current and increase the risk of failure to capture. The abdominal cavity is close in a routine fashion (Figure 25.3d).

Mini-Thoracotomy Approach

A minimal-incision thoracotomy described in Chapter 6 can be performed at the apex of the left ventricle to secure the lead at the apex of the left ventricle (Figure 6.1). A 4 cm skin incision is made in the ventral part of the seventh or eighth intercostal space in the left side. The external abdominal oblique is retracted dorsally and the rectus abdominis muscle is incised. The intercostal muscles are incised and small retractors are used to spread the rib apart. The pericardium is then incised and two stay sutures are used to stabilize the pericardium and the heart. Electrodes are secured with mattress sutures reinforced by tying the suture over the end of the electrode (Figure 25.4).

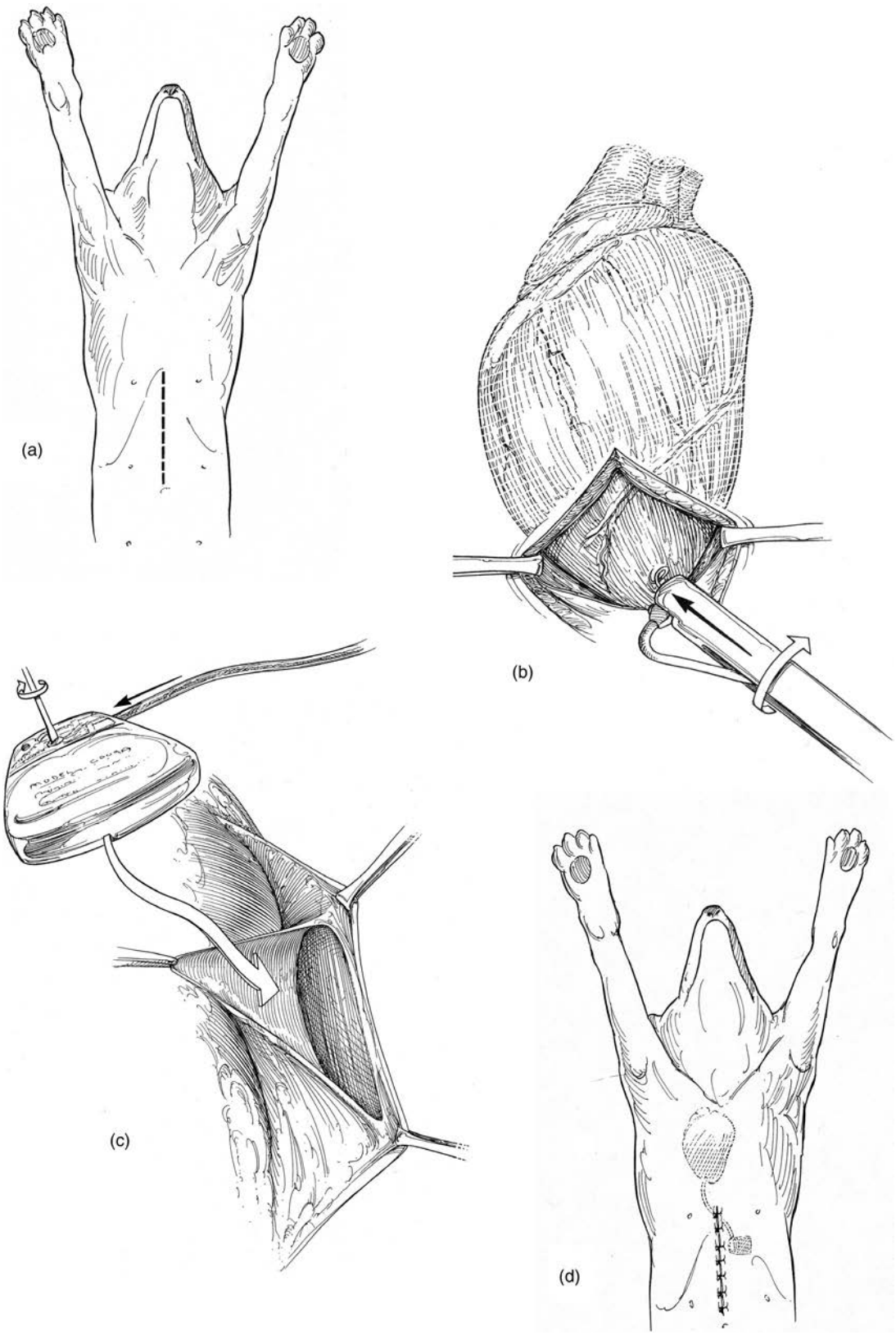

Figure 25.3 Epicardial Pacemaker Implantation—Transdiaphragmatic

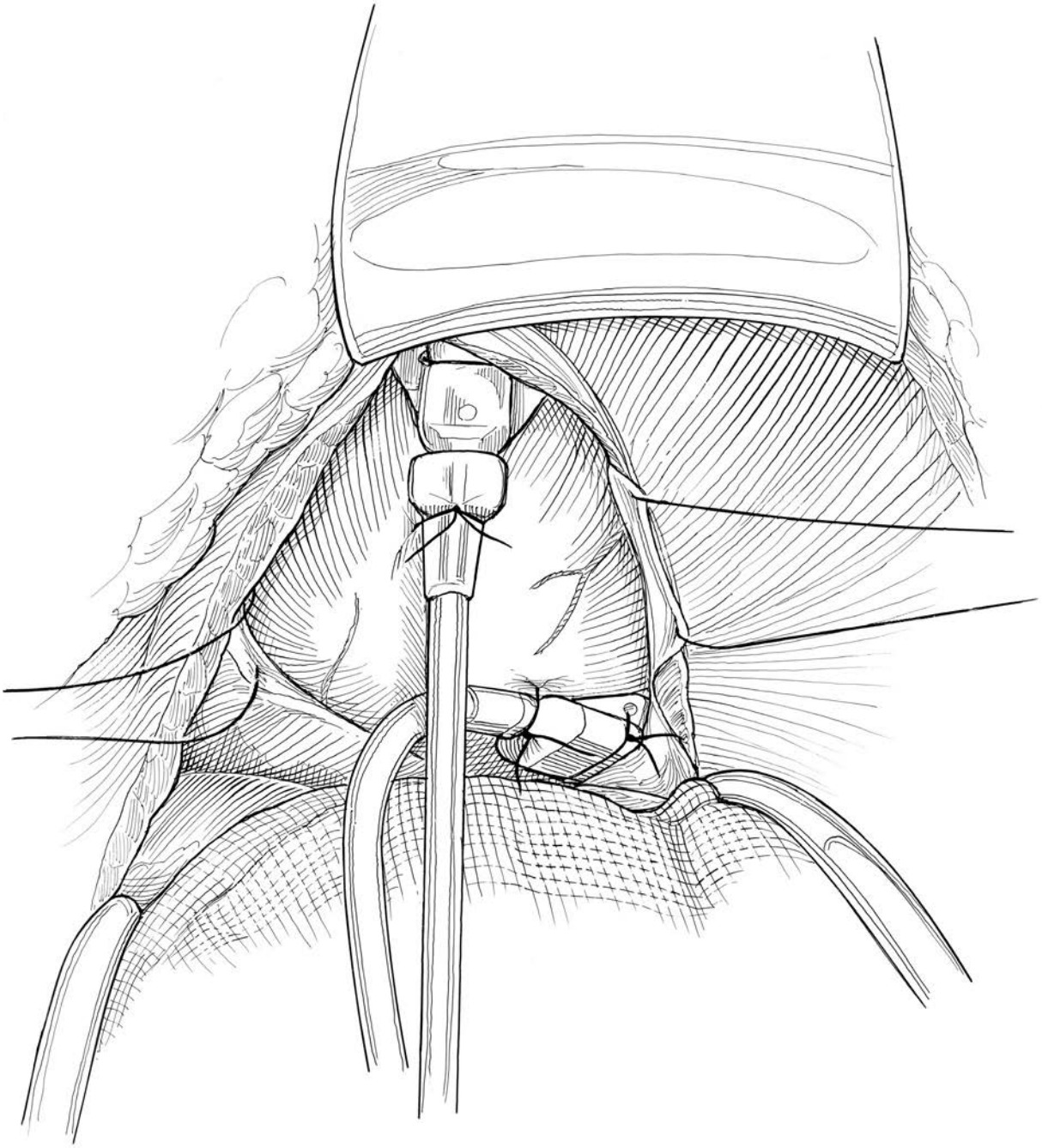

Figure 25.4 Bipolar Epicardial Pacemaker Implantation—Suture-Type Electrodes

An incision is made in the lateral abdominal wall and a pocket for the generator is created between the external and internal abdominal oblique muscles. The electrode is tunneled from the thoracic cavity subcostally to the incisional pocket in the lateral abdominal wall using a tunneling instrument. A thoracostomy tube is placed and the intercostal thoracotomy closed in a routine fashion.

Post-Operative Care

The patient is monitored for 24 hours with an ECG to assure the pacemaker is working properly. The thoracostomy tube is maintained usually for 6 hours after surgery. A pacemaker should be able to sense intrinsic myocardial electrical activity and capture the myocardium when an impulse is delivered. Depending

on pacemaker programming, when electrical activity is sensed, the pacemaker will either trigger an impulse to coincide with the ventricular refractory period or inhibit itself. The pacing generator can be interrogated and reprogrammed by telemetry any time after surgery. Interrogation provides information on the battery life of the pacemaker and lead impedance. Appropriate impedance of an unipolar electrode is between 250 and 1,000 ohms, whereas impedance for bipolar electrodes is between 250 and 500 ohms.

Failure of pacing results in recurrence of the clinical signs. Failure of pacing can be due to failure to capture, inappropriate sensing, or battery failure. Failure to sense (under sensing) and/or capture can result from increased impedance at the lead/myocardium interface, lead dislodgment, or lead fracture.

Failure to capture resulting from high lead impedance is identified on the ECG as electrical spike on the electrocardiogram not followed by a QRS complex. Increasing the power output of the pacemaker should resolve this problem. Failure to capture because of a broken lead or dislodgment of the electrode usually will not produce an electrical spike on the ECG. A thoracic radiograph is useful to evaluate the position of the electrodes (Figure 25.5). Measurement of lead impedance will reveal an impedance > 1,000 ohms if the lead is broken or dislodged. If the lead is leaking current from a defect in insulation, the impedance will decrease to < 250 ohms. In this case the lead will have to be replaced.

Failure to sense or undersensing can result in triggering an electrical impulses at inappropriate times inducing fusion or pseudo-fusion complexes. A fusion complex occurs when the ventricle is stimulated by an electrical impulse at the same time as spontaneous QRS complex, while a pseudo-fusion beat occurs when the electrical impulse is delivered during the QRS complex during the refractory period. The sensing level can be adjusted to increase detection intrinsic ventricular electrical activity.

Oversensing is the result of detection of electrical activity that is unrelated to cardiac activity (e.g., skeletal muscle activity) or inappropriate sensing of the cardiac P- or T-wave. Over-sensing results in failure of the generator to deliver an appropriate impulse. The problem is addressed by reprograming the generator to have a higher sensing threshold. Oversensing is more likely to occur with a monopolar electrode than with bipolar electrodes because more muscular mass is included in the circuit.

The output of the pacemaker should be set to capture the heart without excessive drain on the battery. Batteries are usually set to last 5 years. Pacemakers are typically set to deliver an impulse at 1.0 volt, with a pulse width of 0.5 second. When the battery is exhausted, pacing will gradually shut down and clinical signs will recur.

Lack of synchronization between atrial and ventricular contraction during ventricular pacing can significantly affect cardiac output and arterial pressure. Contraction of the atrium against a closed mitral valve can result in pacemaker syndrome and congestive heart failure. It can be difficult to determine if the progression of a preexisting congestive heart

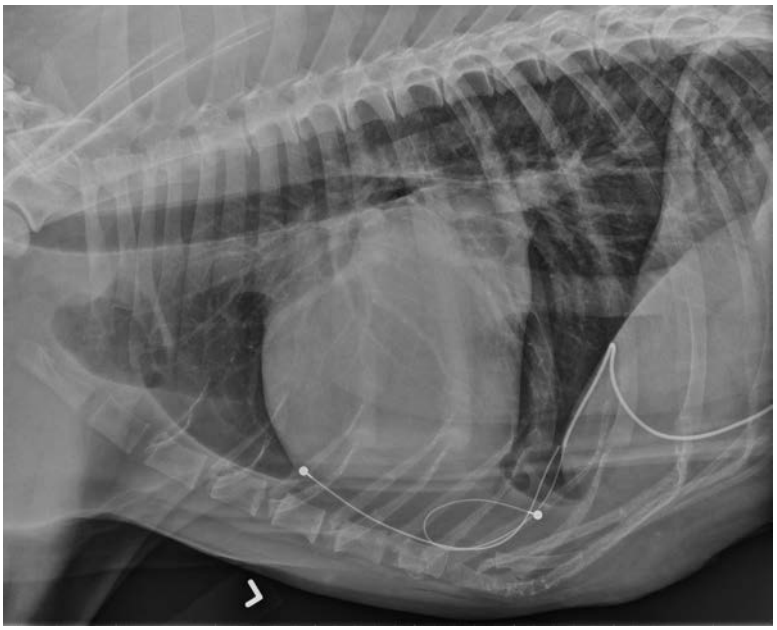

Figure 25.5 Radiograph—Pacemaker Lead Detachment

failure is occurring or if pacemaker syndrome has been induced. If the patient did not have clinical signs of congestive heart failure before implantation of the pacemaker, the index of suspicion for a pacemaker syndrome should increase if congestive heart failure develops after implantation of a pacemaker. Echocardiography to evaluate the timing of atrial contraction against a closed AV valve may be helpful in evaluating for pacemaker syndrome. The only effective treatment of pacemaker syndrome is the implantation of a dual chamber pacemaker that will allow synchronization between atrium and ventricle. Patients with sick sinus syndrome are at greater risk to develop pacemaker syndrome. In those cases, slowing down the pacing rate to the lowest level that prevents syncope might help palliate development of congestive heart failure.

References

1 Fox PR, Moise NS, Woodfield JA, and Darke PG. 1991. Techniques and complications of pacemaker implantation in four cats. *Journal Amercian Veterinary Medical Assocation.* 199(12): 1742–53.

2 Moise NS. 1999. In: Fox PR, Sisson D, and Moise NS, eds. *Textbook of Canine and Feline Cardiology*. pp. 400–25 Philadelphia: W.B. Saunders.

3 Wess G, Thomas WP, Berger DM, and Kittleson MD. 2006. Applications, complications, and outcomes of transvenous pacemaker implantation in 105 dogs (1997–2002). *Journal of Veterinary Internal Medicine.* 20(4):877–84.

4 Johnson MS, Martin MW, and Henley W. 2007. Results of pacemaker implantation in 104 dogs. *Journal Small Animal Practice.* 48(1): 4–11.

5 Moise NS. 1999 In: Fox PR, Sisson D, and Moise NS eds. *Textbook of Canine and Feline Cardiology*. pp. 331–85 Philadelphia: W.B. Saunders.

6 Weder, C., E. Monnet, M. Ames and J. Bright. 2015. Permanent dual chamber epicardial pacemaker implantation in two dogs with complete atrioventricular block. *Journal Veterinary Cardiology.* 17(2):154–160.

7 Benditt DG, and Duncan JL. 1995. In: Ellenbogen BG, Kay GN, and Wilkoff BL eds. *Clinical Cardiac Pacing*. pp. 167–86 Philadelphia: W.B. Saunders.

8 Bernstein AD, and Parsonnet V. 1995. In: Ellenbogen BG, Kay GN, and Wilkoff BL eds. *Clinical Cardiac Pacing*. pp. 279–83 Philadelphia: W.B. Saunders.

9 Mond HG, and Helland JR. 1995. In: Ellenbogen KA, Kay GN, and Wilkoff BL eds. *Clinical Cardiac Pacing*. pp. 69–90. Philadelphia: W.B. Saunders.

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