RESPIRATORY DISTRESS AND THERAPEUTICS

M. Scott Echols, DVM, Diplomate ABVP
Westgate Pet and Bird Hospital
Austin, TX

Respiratory distress is common in avian practice. By classifying the type of respiratory disease, clinicians are better able to determine a cause and resolution. This article attempts to help practitioners classify and treat various forms of respiratory disease in awake and anesthetized birds.

ACUTE RESPIRATORY DISTRESS

Acute respiratory distress is a potentially life-threatening emergency condition seen in birds. Its causes can be divided into large airway disease, parenchymal disease, coelom space disease, and small airway disease. Prior to differentiation, all avian patients experiencing acute respiratory distress should be gently handled to minimize stress and provided with supplemental oxygen in preparation for more aggressive diagnostics.

LARGE AIRWAY DISEASE

Causes of large airway disease include tracheal obstruction (foreign body, fungal or bacterial granuloma, intratracheal neoplasia, stricture, and others) or oropharyngeal masses, granulomas, swelling, or other disorders that obstruct airflow in and out of the trachea. These birds often require rapid anesthetic induction, intubation to get past oropharyngeal masses or an air sac tube if the trachea is obstructed. Diagnostics (eg, tracheal endoscopy, tracheal wash, radiographs) follow once the patient is more stable.

Air Sac Breathing Tube

If the oral cavity or trachea is occluded preventing proper induction or maintenance of anesthesia or the air sacs need to be medicated, an air sac tube can be used. Patients with oral masses, tracheal obstructions, etc should be mask (if possible) or box induced. Anesthesia delivered via air sac breathing tubes has been successfully used in patients as small as zebra finches.

When anesthetized, place the patient in lateral recumbency and quickly, surgically prepare the paralumbar fossa (just behind the last rib). A small skin incision is made over the paralumbar fossa (same site as for surgical sexing) exposing a relatively thin layer of lateral abdominal wall muscles. Use right angle forceps to “punch” through the muscle layer and into the underlying air sac. Place a sterilized endotracheal, red rubber feeding or other tube into the air sac (either caudal thoracic or abdominal air sac, depending on the bird species and placement) and suture (the tube) to the skin. When properly placed, one can hold a down feather over the tube opening to watch for air movement or place a slide and watch for condensation. The tube diameter should be approximately the same size as the patient’s tracheal lumen. Due to progressive micro-organism infection and air sacculitis, air sac breathing tubes should not be left in avian patients more than 5 days. In addition to infection, air sac breathing tubes can result in coelomic organ damage, life-threatening blood loss (from vessel laceration/trauma), air sac damage, and subcutaneous emphysema (which is usually self-limiting).

In studied sulfur-crested cockatoos (Cacatua galerita), delivering isoflurane and oxygen via caudal thoracic air sac intubation provided a reliable method of maintaining anesthesia and resulted in minimal alteration in respiratory function similar to endotracheal tube administration. In the same study, clavicular air sac intubation did not provide adequate ventilation or maintenance of anesthesia.

PARENCHYMAL DISEASE

Causes of parenchymal disease include smoke inhalation; fungal, viral, bacterial and parasitic pneumonia; and cardiogenic pulmonary edema. Bronchodilators (parental or via nebulization) and antibiotics may benefit birds with parenchymal disease. An air sac tube may also improve respiration in these birds, but is often a less dramatic improvement as is seen in birds with large airway disease. If a murmur, arrhythmia or muffled heart sounds are auscultated, cardiogenic edema or pericardial effusion may be present. Furosemide (2–4 mg/kg IV) and nitroglycerine ointment on the tongue may help stabilize the patient for additional diagnostics. Bronchodilators, antibiotics, and antifungals should be considered with pneumonia.

COELOM SPACE DISEASE

Coelom space disease includes those causes of coelomic distension such as liver disease, egg-related and other peritonitis, heart disease, hypoalbuminemia and organomegaly. If fluid is present, coelomocentesis and standard fluid evaluation are recommended. Depending on fluid analysis, antibiotics may be indicated. Ultrasound and radiographs can be beneficial in differentiating coelomic masses, eggs, and fluid from each other. If present, egg binding is treated (eg, ovocentesis, fluid therapy, warmth). Organomegaly often requires a full medical work up to identify the problem and guide therapy.

Coelomocentesis

Coelomocentesis is indicated for birds with free coelomic fluid of unknown type or origin. Due to the coelomic, intestinal-peritoneal and dorsal and ventral hepatic-peritoneal cavities in normal birds, a single free “cavity” does not exist. Attempts at ‘abdominal washes’ are difficult and can result in fluid infusion into the abdominal or caudal thoracic air sacs potentially causing or contributing to respiratory distress. With increasing coelomic fluid, the avian air sacs become compressed, the coelom distends and the patient may develop varying degrees of dyspnea. An affected bird should be restrained with the long axis of its body oriented vertically, or slightly tilted forward, and head up. Use of...
anesthesia may be contraindicated due to potential respiratory compromise.

The ventral coelom is aseptically prepared and a small gauge needle (21- to 27-gauge) is inserted on the right side of the body and any fluid present is aspirated. As with other avian fluids, the aspirated sample should be processed quickly and may be used for microbiological, cytologic, and biochemical analysis.

SMALL AIRWAY DISEASE

Small airway disease is often the most difficult to manage and may be due to inhalation of respiratory irritants such as Teflon fumes, aerosols, smoke, and candle suet. These respiratory irritants cause acute bronchoconstriction leading to cough and acute respiratory disease. This series of events creates a need for immediate therapy and bronchodilators are often the first line drugs. Because of a faster onset of action, nebulized bronchodilators are the first choice. Bronchodilators that stimulate beta-adrenergic receptors in bronchial smooth muscle (causing smooth muscle relaxation) are considered the most effective for treating acute bronchoconstriction and respiratory distress. Lichtenberger recommends the beta-agonist terbutaline first given as an aerosol or second, given IM (0.01 mg/kg).

Several nebulization options exist. Jet nebulizers are inexpensive; the most common and create a gas stream that draws drug solution up a tube and then pulverizes the mixture to form an aerosol spray. Ultrasonic nebulizers are more expensive and generate an aerosol using an ultra-high frequency vibrating piezoelectric crystal at the bottom of the drug solution. While ultrasonic nebulizers have faster nebulization time, smaller particle size, quieter operation and longer product life than jet nebulizers, studies in humans have shown little difference between the two types. Metered dose inhalers (MDI) can be used for rapid local administration of bronchodilators (such as albuterol). However, the actual dose of drug deposited in the lower airway is unknown using a MDI. Some MDI come with a soft facemask that can be used with larger psittacine birds.

Oxygen Administration

Facial or environmentally increased (cage) oxygen is essential for many critical avian patients. The indications for supplemental oxygen may include any type of respiratory or cardiac disease, shock, postsurgical recovery, and patient stress. Oxygen delivered by facial mask may not be well tolerated in some fractious, disoriented, and wild patients. If a specifically designed “oxygen cage” is not available, most incubators, cages and aquariums can be modified so that supplemental oxygen can be provided in a closed environment. For most critical avian patients kept in a closed chamber, a 40% oxygen saturation level is recommended. Pure oxygen is acceptable for short-term use when given via a facemask or in an enclosed environment.

It should be noted that oxygen “toxicity” in the form of pulmonary oxidative stress and toxicity with histologic changes in the lungs has been noted in budgerigars. The studied birds were kept in a high oxygen environment (95% O2) for as little as 3 hours and developed signs of oxidative stress, perivascular edema, bronchial pneumonia and depletion of body antioxidants (carotenoids, alpha tocopherol) that worsened with prolonged oxygen exposure. This recent information further supports limiting the exposure to pure supplemental oxygen in avian patients.

RESPIRATORY DISTRESS WHILE UNDER ANESTHESIA

Birds in respiratory distress are frequently encountered in avian practice. Not only are birds brought in for emergency evaluation, but respiratory disease may develop during anesthesia. It is important for the attending clinician to recognize normal and abnormal respiratory patterns for birds undergoing anesthetic procedures and how to deal with problems when they arise.

Abnormal Breathing Patterns Should Be Identified and Resolved as Soon as Possible

Pathologic conditions such as pain (surgical manipulation), lung hemorrhage, and overheating can induce rapid breathing (tachypnea). With painful conditions, tachypnea usually resolves by inducing a deeper plane of anesthesia. Tachypnea associated with blood-filled lungs (hemorrhage) responds poorly to increasing the anesthetic dose. If lung hemorrhage is suspected or identified (visualized via endoscopy or laparoscopy), anesthesia should be stopped or continued only with great caution. In birds, lung hemorrhage can be fatal, especially if progressive; its cause is poorly understood and no treatments are currently described. Increased respiration due to overheating is most commonly noted when the avian patient is “light” or upon recovery, but sometimes, hyperthermia contributes to mild tachypnea during deeper anesthetic planes.

Dyspnea, or difficult and labored breathing, often indicates poor ventilation. Tachypnea and dyspnea can be very similar, are often difficult to distinguish and share some common causes. Underlying diseases (heart, lung, and air sac disorders), as opposed to human induced overheating and pain, are more frequently associated with dyspnea in birds. Dyspnea may occur due to an obstruction (such as a mucus plug) in the trachea or endotracheal tube, poor cardiovascular perfusion (heart disease and anemia), fluid or blood-filled lungs (pulmonary edema, inflammation and hemorrhage) and decreased or diseased air sac space resulting from improper restraint/positioning, ascites, bleeding, organomegaly, other abdominal masses and air sacculitis. Correction of dyspnea is aimed at identifying and addressing the underlying cause.

Apnea, or cessation of breathing, is common in anesthetized birds and can be associated with multiple conditions. If apneic birds are not restored (artificially or
naturally) to breathing, death may soon follow and faster than is noted in cats and dogs. Apnea is commonly due to poor ventilation, taping the beak closed (diving birds), excessive anesthetic, and hypothermia, but may also be associated with hypoglycemia, hypovolemia (blood or other fluid loss), and other underlying metabolic and systemic disturbances. Apnea is frequently preceded by decreased respiration. Identifying and correcting the causes of decreased respiration will help the monitoring nurse prevent apnea or respond more quickly, should it (apnea) occur.

When a bird’s respiration begins to drop, first evaluate the patient’s anesthetic dose, surgical conditions (eg, disrupting air sacs/lungs, excessive blood loss), and monitoring tools (eg, body temperature, heart rate). Correct obvious deficits as best as possible by lowering anesthetic dose, providing fluid support, maintaining patient body heat, and so on as needed. If decreased respiration is progressive, auscultate the lungs and trachea and determine if the passages are clear. Tracheal mucus plugs will often produce a “gurgling” sound that may only be heard with the aid of a stethoscope. Lung hemorrhage and edema may also result in abnormal respiratory sounds and should be considered as well. Remove the endotracheal tube (if present) and check for blood or mucus plugs that may be contributing to poor ventilation. In general, endotracheal tubes may need to be replaced every 30 minutes to decrease plug formation. Air sac tubes are an exception, as they rarely occlude with mucus and can be left in for days without problems. However, air sac tubes can become plugged with blood, other fluids and organ tissue and should be checked for patency as needed. The surgeon should be notified when the patient is becoming apneic so he or she can identify and resolve any surgical conditions that may be interfering with respiration.

If respiration decreases to the point of apnea, several steps should be taken to restore breathing. Quickly evaluate the patient as discussed in the above paragraph. If breathing cannot be restored, turn off all anesthetic gas, maintain oxygen flow, and manually ventilate the patient. When manually ventilating birds, provide enough pressure (8–12 cm H₂O) to create normal inspiratory depth and 10–12 respirations per minute. Some feel that artificial ventilation should be routinely provided to all anesthetized birds. To prevent trauma to the air sacs when providing positive pressure ventilation, limit the pressures to 15–20 cm H₂O.

In birds, decreased ventilation results in increased PaCO₂ which directly affects pH and acid–base balance. The expected response in an awake, healthy bird is to increase ventilation, which decreases PaCO₂ and increases pH. Birds must adequately ventilate to maintain proper acid-base balance. Anesthetized birds may not properly ventilate themselves and often require artificial ventilation. In ducks, the common parameters such as respiratory frequency and tidal volume alone cannot be used to monitor acid-base balance. Studies in Amazon parrots have clearly shown that intermittent positive pressure ventilation will decrease PaCO₂, which subsequently affects pH and acid–base balance. Even birds that are self-ventilating while under anesthesia should be assisted with 4 to 6 respirations per minute.

The sternum may be moved ventrally and dorsally to provide ventilation if no endotracheal tube or sealed face mask is being used. If the air sacs have been exposed as part of surgery, the surgeon may need to temporarily “close” the surgical site with a finger to help permit more normal ventilation. If the patient is apneic, the surgeon must attempt to limit overall surgical time while the attending nurse increases the oxygen flow rate and continues assisted ventilation. Sometimes continued tissue manipulation will help the patient respond and begin breathing. Patients that become apneic and are restored should be maintained on a very light plane of anesthesia for the remainder of the procedure.

Cardiac Arrest Often Follows Prolonged Apnea or Dyspnea

Maintaining normal fluid volume, proper oxygenation, ventilation and monitoring, minimal use of cardiodepressant drugs, and appropriate anesthetic depth are all needed to help prevent cardiac arrest. Certainly, some underlying conditions may adversely affect the heart and may be precipitated by anesthesia. If cardiac arrest occurs, begin cardiopulmonary resuscitation. If possible, place an endotracheal tube (if not already present) and restore ventilation. Doxapram, a positive inotrope, can be given (to help stimulate respiration. Also consider Hetastarch at 5 mL/kg intravenously (IV) or intrathecally (IO) to increase blood pressure and improve organ perfusion. Manipulate the sternum, as described above, to assist circulation and ventilation. Due to anatomy, direct cardiac massage is very difficult. Intravenous, intratracheal, or intracardiac epinephrine (0.5–1.0 mL/ kg of 1:1,000) can be tried to stimulate the heart. Unfortunately, birds are very difficult to resuscitate once cardiac arrest occurs.

REFERENCES