CHAPTER

22

PNEUMONOLOGY

Thomas N. Tully, Jr.
Greg J. Harrison

Companion bird clients frequently seek veterinary care when abnormal respiratory signs are noted in their pets.[1] The rapid progression of many avian respiratory diseases makes early recognition by the client and rapid diagnosis and effective treatment by the practitioner critical. Conversely, chronic rhinitis and air sacculitis can fulminate for many years with subtle clinical changes. The difficulty in distinguishing between clinical signs originating from the upper versus the lower respiratory system makes the diagnosis and treatment of these problems challenging.[52] Successful respiratory therapy depends on rapid diagnosis.[36] The primary cause of a respiratory disease may be complicated by a more severe opportunistic infection that takes advantage of damaged respiratory epithelium. Diagnosis and treatment of avian respiratory disease depend on an understanding of the unique anatomy and physiology of the avian respiratory system and the effect these adaptations have on the clinical signs and the progression of disease. Respiratory health is best maintained by frequent exposure to fresh air and sunlight, a proper plane of nutrition and flighted exercise.

During the initial physical examination, the avian patient should be observed from a distance to evaluate subtle changes in posture, wing position, respiratory rate and respiratory pattern that may indicate an abnormality. A bird uses its intercostal musculature to expand and depress its chest, creating a "bellows" action that moves air into and out of the respiratory system. Any compromise in inspiratory or expiratory effort can affect the bird's posture.[24,35,59,64] Normal respiratory effort in the bird should not be noticeable, and the mouth should remain closed. An increase in abdominal effort or head movement may be recognized in association with increased respiration following exercise, but should return to normal within minutes of ceasing exertional activity. See Chapter 8 for normal avian respiratory rates.

Mild upper respiratory or lung-induced dyspnea is frequently accompanied by open-mouthed breathing with a dilated glottis. Lung and lower respiratory tract problems are usually associated with a rhythmic jerking of the tail (tail-bob). A bird in severe respiratory distress may also move its head forward in an effort to increase air intake. If the respiratory problem is associated with excessive fluid production, bubbling and gurgling sounds are common on both inspiration and expiration (Table 22.1).

Overt signs of respiratory disease are easy to identify and include oculonasal discharge, stained or matted feathers around the nares, sneezing, coughing, dyspnea or audible inspirations or expirations (Figure 22.1). Changes in pitch or vocalization of the patient may indicate problems in the glottis, trachea or syrinx.[24] Shallow, labored breathing in a bird with a weak, altered or absent voice is common with acute zinc toxicosis. Many psittacine birds may mimic the sneeze or cough of household members, which should not be misinterpreted as a sign of respiratory disease.

Dyspnea may be caused by chronic lung or air sac consolidation or by an acute reduction in the amount of air being delivered to the lungs. When dyspnea is induced by protracted respiratory disease, it is usually associated with other clinical signs including weight loss, depression, ocular or nasal discharge, sneezing or wheezing. Acute dyspnea in an apparently healthy bird usually results from exposure to aerosolized toxins, dislocation and movement of plaques in the trachea (from malnutrition or infectious agents) or aspiration of foreign bodies (particularly seed husks or cage litter material). Dyspnea may also be caused by rhinoliths, air sac granulomas, tracheal parasites (particularly in canaries, gallinaceous birds, finches), obesity (loss of air sac volume) or thyroid enlargement secondary to an iodine-deficient diet (Table 22.2).

The Respiratory Tract

Clinically important components of the avian respiratory system include the external nares, operculum, nasal concha, infraorbital sinus, choanal slit, glottis, trachea, syrinx, bronchi, lungs, parabronchi and air sacs (see Anatomy Overlay).

FIG 22.1 A six-month-old Blue-crowned Conure was presented for suspected sinusitis that was not responsive to antibiotics. The bird had moist, matted feathers and dried debris over most of the head region. The area was thoroughly cleaned and severely matted feathers were removed. The discharge was found to be associated with conjunctivitis and not sinusitis. It was determined that the bird had no nasolacrimal ducts. It also had secondary bacterial and fungal conjunctivitis. The bird was successfully managed by frequently flushing the eyes with sterile saline.

Nares

The first areas to examine for respiratory disease are the nares and surrounding tissues. Unilateral or bilateral diseases of the upper respiratory tract are indicated by matted or mildly stained feathers around the nostrils, occluded nares, nasal discharge or a growth or change in size of the nasal opening. Bacterial, fungal, chlamydial and viral infections, neoplasia and trauma are common etiologic agents of upper respiratory disease.[24,35,59] Chronic inflammation may lead to disfiguring lesions of the nares, beak and cere (Figure 22.2).[36] Severe *Knemidokoptes* spp. infection may cause proliferation of the cere that blocks the external nares and causes respiratory difficulties (see Color 24).[50]

Some Arabian falconers dilate the nares in their birds to facilitate air intake and improve hunting performance. With the efficiency of the air exchange system in birds, it is unlikely that this procedure is of any value.

The operculum can be seen just inside the nostril; this cornified flap of tissue (frequently referred to

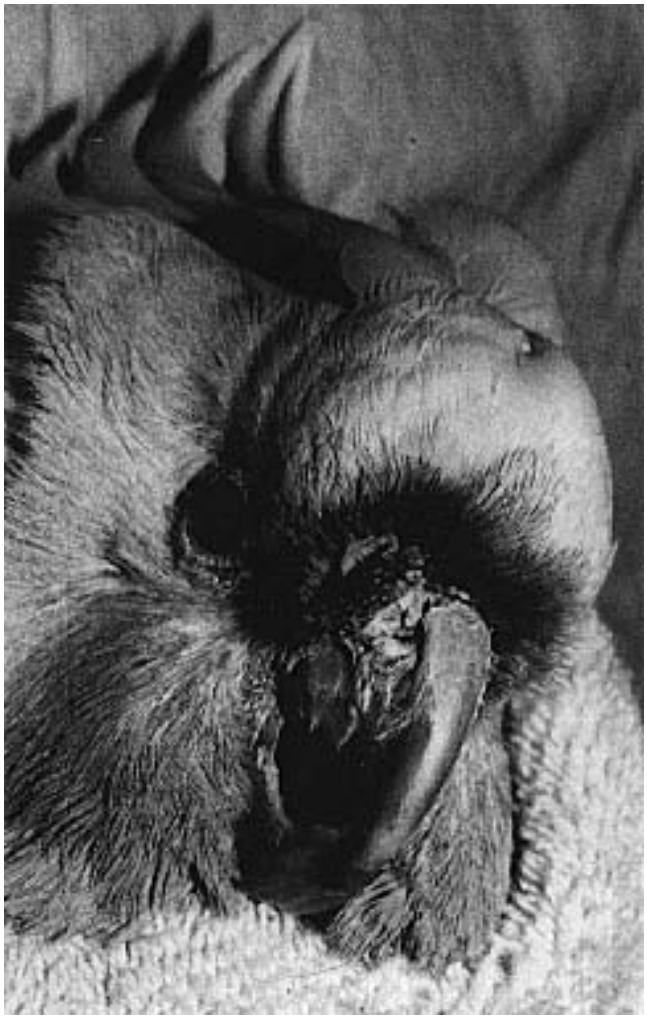


FIG 22.2 A mature, male Major Mitchell's Cockatoo was presented for a proliferative mass involving the right nostril and beak. No other abnormalities were noted by physical examination, radiography or clinicopathologic evaluation. The mass was removed and was determined to be a well encapsulated fungal granuloma. The presence of a beak defect from the cere to the tip indicates the chronicity of the problem.

improperly as bone) should not be mistaken for a foreign body. The operculum should be smooth and dry. Cellular debris can accumulate between the operculum and the wall of the nasal cavity, leading to substantial necrosis of the rostral nasal passages and its associated bone (see Chapter 41).

A septum divides the nasal cavity into two halves, each containing a rostral, middle and caudal nasal concha in most avian species. Air that enters the external nares is warmed and moistened by the highly vascular rostral and middle nasal concha (turbinates), which lie rostral, caudal and ventral to the operculum. The caudal nasal concha does not connect directly to the nasal cavity; instead, it opens dorsally into the infraorbital sinus, which is divided into five diverticuli (rostral, periorbital, infraorbital, mandibular and postorbital) that extend into the upper beak, mandible and portions of the pneumatic skull (see Anatomy Overlay). The infraorbital sinus opens caudally into the cranial and cervical portions of the cervicocephalic air sac and dorsally into the middle nasal concha.[44] The fact that the openings from the caudal nasal concha to the infraorbital sinus and from the infraorbital sinus to the middle conchae are both in a dorsal position may contribute to the common accumulation of pus and cellular debris in the infraorbital sinus in birds with upper respiratory tract disease (Colors 22.2, 22.3, 22.5).[44]

In mammals, the sinuses are contained within the bones of the skull. By contrast, avian sinuses are restricted laterally by the skin and subcutaneous tissues of the face. The sinuses have simple mucous glands and are lined by stratified squamous and ciliated columnar epithelium. Hypovitaminosis A commonly causes squamous metaplasia and hyperkeratosis of the sinuses and nasal passages, leading to granuloma formation.

Sinuses

The infraorbital sinus is the only paranasal sinus in birds and is located lateral to the nasal cavity and surrounding the eyes ventrally.[72] In some birds (insectivorous Passeriformes, Anseriformes and Psittaciformes), the right and left infraorbital sinuses communicate, while in other species (non-insectivorous Passeriformes), the right and left infraorbital sinuses are independent.

The interconnection of the nasal cavity, infraorbital sinuses and the porous calvaria creates a situation in which inflammatory reactions in the sinus or nasal passages can involve most of the anatomic structures of the head. The numerous pockets and extensions of the nasal system make sinus infections difficult to treat. With severe chronic sinusitis, the accumulation of caseous necrotic debris can cause destruction of the nares, nasal cavity, operculum and nasal conchae. This degree of destruction is particularly common in Amazon parrots and African Grey Parrots with aspergillosis sinusitis. Inflammation or accumulation of debris in the infraorbital sinus can lead to periorbital swellings (Color 22.2).

The tube-like nasal cavity turns ventrally at the sinuses, and the air exits the two choanae (internal

TABLE 22.1 Clinical Considerations of Respiratory Disease

UPPER RESPIRATORY DISEASE

Clinical Signs
- Open-mouthed breathing
- Change in voice
- Sneezing
- Sinus swelling
- Rhinorrhea
- Nasal granulomas
- Exercise intolerance
- Dyspnea
- Head-shaking
- Mucopurulent nasal discharge
- Inflamed swollen cere
- Stretching the neck
- Yawning
- Epiphora
- Periophthalmic swellings
- Plugged nares

Diagnostic Protocol
- Thorough review of nutritional status
- Thorough history (exposure to smoke, PTFE)
- Gram's stain of feces; direct fecal smear for parasites; special pathology stains
- Sinus flush
- Cytology of affected area (sinus aspirate, flush, scraping for parasites); Gram's stain
- Radiographs (whole body, sinus views)
- Rhinoscopy (foreign body examination)
- Culture and sensitivity
- Special bacterial and viral diagnostic testing (also for chlamydia, mollicutes)
- Biopsy of lesion
- CBC, biochemistry panels

Normal Flora
- Gram-positive bacteria (eg, *Lactobacillus* spp., *Streptococcus* spp. and *Micrococcus* spp.)
- Small numbers of gram-negative organisms (eg, *E. coli, Bordetella*)
- Occasional non-budding yeast

Abnormal Flora
- Large numbers of gram-negative bacteria (over 5%)
- 10 budding yeast per oil immersion field examined

LOWER RESPIRATORY DISEASE

Clinical Signs
- Tail-bobbing
- Loss of voice
- Change in vocalization
- Labored respiration
- Exercise intolerance
- Coughing
- Sounds on auscultation

Diagnostic Protocol
- Radiographs of lungs, air sacs
- Transtracheal lavage (cytology of sample)
- Laparoscopy; tracheoscopy
- Biopsy of lungs, air sacs
- Suction and cytology
- Culture of trachea, lungs and air sacs
- Surgical intervention (air sac granuloma; tumor; tracheal foreign body)
- Compression reduction of air sacs

Fluid Obtained by Tracheal and Air Sac Lavage
- Normal
  Low cellularity and very few pulmonary macrophages or inflammatory cells
- Abnormal
  Large numbers of heterophils, pulmonary macrophages and other inflammatory cells, bacteria or yeast

nares) at the level of the palate. The middle choanae are just cranial and dorsal to the choanal slit, which courses longitudinally in the dorsal oral cavity or roof of the mouth. The choanae are separated into right and left openings by the nasal septum or vomer bone. The paired entrances of the nasal cavity can be viewed with a rigid or flexible endoscope by directing it through the rostral end of the choanal slit (see Color 13). The choanal slit represents the incomplete fusion of the two bony plates of the hard palate (see Color 8). Birds do not have a soft palate. Instead, air moves from the nasal cavity through the choana via the choanal slit (oropharynx) and then into the rima glottis of the trachea. The configuration of the choanal slit varies with the species, but in all cases the slit should be slightly moist. On the ventral surface of the palate and along the choana are numerous caudally directed choanal papillae, which are most pronounced in gallinaceous species but are also found in most birds (see Color 13).

Swollen, inflamed choanal tissues, with a sloughing of the protruding papillae, are common with upper respiratory tract infections (particularly chlamydiosis),[30] and secondarily infected with candidiasis in immunosuppressed states following prolonged illness, malnutrition or improper antibiotic administration. The presence or absence of papillae is not a diagnostic indicator of current respiratory disease, as they seldom regrow after sloughing. Laryngeal le-

TABLE 22.2 Clinical Presentations of Avian Respiratory Disease with Associated Differential Diagnoses

Clinical Presentation	Differential Diagnosis
Sunken eye	Chronic bacterial sinusitis
Enlarged cere	Chronic rhinitis; foreign body; trauma; allergy; airborne irritants (eg, cigarette smoke); malnutrition (chronic); avian poxvirus; knemidokoptes mites; normal female budgerigar
Enlarged cere with or without granuloma formation	Bacterial, mycotic, mycoplasmal, chlamydial; nutritional rhinitis
Rhinorrhea or sneezing	Bacterial infection; mycotic infection; foreign body; toxic insult (smoke); allergy; virus; malnutrition;; chlamydia
Serous sinusitis	Chlamydia or mycoplasma infection; nutritional rhinitis; foreign body; papillomatosis; occluded choana (atresia); uncomplicated viral infections
Mucopurulent sinusitis	Bacterial infection with predominantly gram-negative organisms; mycotic infection (often secondary to serous sinusitis)
Irritated swollen cere with sloughed papillae	Chronic mycotic, bacterial or viral sinusitis; chronic exposure to airborne irritants; chlamydiosis; malnutrition; hypovitaminosis A
Coughing (chronic)	Bacterial, viral, fungal, chlamydial, parasitic, yeast, mycobacterial; ascites; abscess or granuloma; malnutrition; air sac mites; mimicry of humans; airborne toxins (eg, cigarette smoke)
Coughing (acute)	Foreign body inhalation; trauma; upper respiratory infection; abscess or neoplasia in lungs or body cavity; air sac mites; infectious tracheitis; avian viral serositis; mimicry of humans; bleeding into body cavity; sarcocystosis; syringeal granuloma; airborne toxins - PTFE gas
Dyspnea (acute)	Aspergillosis syringeal granuloma; infectious disease; foreign body inhalation; internal bleeding; allergy; toxin inhalation; plugged nares; avian viral serositis; sarcocystosis; anemia
Dyspnea (chronic)	Infectious disease; liver disease; kidney disease; ascites; heart disease; neoplasia; air sacculitis; malnutrition; sarcocystosis (lung edema); proliferative tracheitis; Pacheco's disease virus; pericardial effusion; egg-related peritonitis (binding); hemochromatosis; anemia; obesity; thyroid enlargement, tumors, goiter
Subcutaneous swelling	Overinflation of cervicocephalic air sac; trauma (bite wound); normal (pelicans)
Neonatal sneezing, coughing, dyspnea	Inhalation pneumonia; respiratory foreign body; infections (eg, chlamydia); avian viral serositis; mycotic infection

sions may occur from viral infections (poxvirus, herpesvirus), hyperkeratosis secondary to hypovitaminosis A, candidiasis, trichomoniasis, papillomatosis and neoplasia (Figure 22.3).[24,25,35,36,57,61] Seeds may also become lodged in the choanal slit and cause respiratory signs or constant movement of the tongue in an effort to dislodge the seed.

Trachea

The opening of the larynx, or rima glottis, is not covered by an epiglottis as it is in mammals. The laryngeal cartilages are reduced or absent. The largest laryngeal cartilages are the coracoid cartilages.[3] The larynx does not function for sound production in birds. When a bird breathes, the mouth is closed and the mobile glottis seals with the choanal slit, allowing air to pass into the trachea from the nares (see Anatomy Overlay). There are no vocal cords in the larynx.

The trachea is loosely found on the right side of the neck, ventral to the esophagus. The trachea courses under the crop at the thoracic inlet and terminates into the syrinx. It is the syrinx that serves as the vocal organ (see Anatomy Overlay). The trachea of birds differs from mammals in that it is longer and has a larger diameter — two anatomic considerations for anesthesia. The trachea consists of complete cartilaginous rings in most avian species. These cartilaginous rings may calcify as the bird grows older.[7] The length, configuration and anatomic position of the trachea vary widely among genera. Some birds, like the Whooping Crane, have a trachea that extends to the cloaca, where it doubles back and returns to the thoracic inlet before connecting to the syrinx. Other species (Helmeted Curassow) have a similar configuration, but the trachea courses subcutaneously outside the confines of the sternum.

At the distal end of the trachea is the syrinx, which can be classified as tracheal, tracheobronchial or bronchial depending on the location of fusion of the cartilages.[51] Most psittacine birds have a tracheobronchial-type syrinx in which the last of the tracheal rings fuse into a syringeal box, which joins to the first of the bronchial rings. The shape of the syrinx and the sound it emits are controlled by the bronchial muscles that attach to the syrinx, the first bronchial rings and the bronchotracheal muscles, which extend from the bronchus to the trachea. Sounds are believed to be produced in the syrinx by

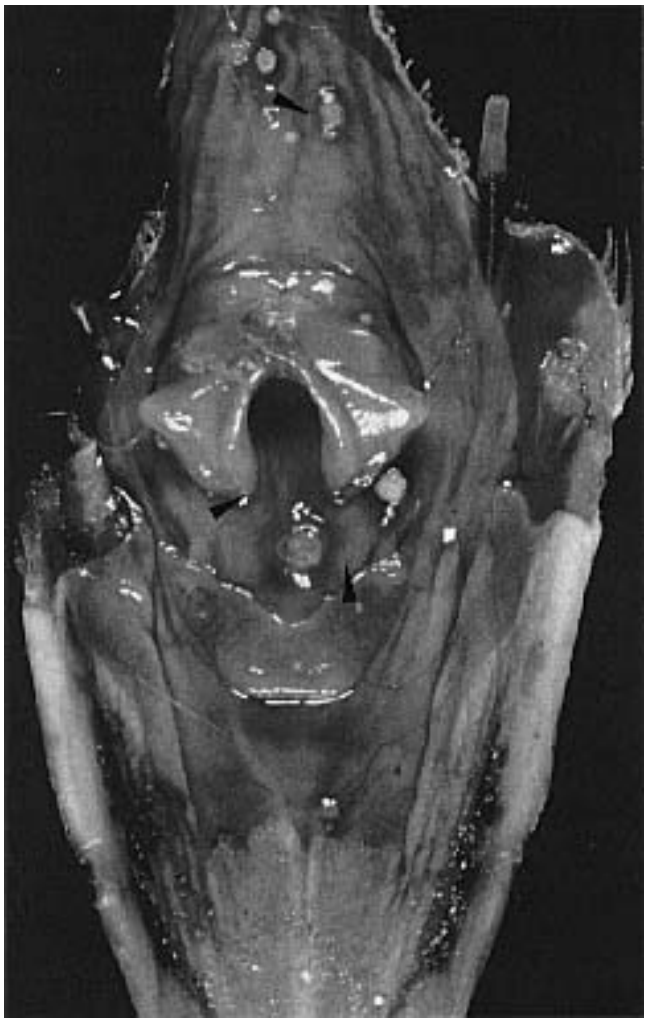


FIG 22.3 Proliferative lesions of the pharyngeal or oral mucosa (arrows) may be caused by some viruses, hypovitaminosis A, *Trichomonas* spp. or *Candida* spp. (this case). Cytology is a useful technique to establish a clinical diagnosis (courtesy of Louise Bauck).

the turbulent flow of expelled air that is forced through syringeal membranes, which form slots.[44] The pitch of the sound is also controlled by the length of the trachea and whether the air sacs are inflated or flattened. A long trachea and inflated air sacs produce a loud, booming, low-frequency sound.

Pathology involving the syrinx is best diagnosed and treated when signs of disease are first recognized. If a bird stops talking or has a voice change it should be evaluated immediately for lesions developing in the perisyringeal area (frequently aspergillosis). Progressive changes recognized clinically as dyspnea, coughing or tracheal discharge are more difficult to successfully resolve (Figure 22.4). The trachea and primary bronchi contain goblet cells and are lined with pseudostratified, ciliated, columnar epithelium. The syringeal mucosa contains bistratified squamous or columnar epithelium that is subject to squamous metaplasia and granuloma formation.[4]

Lungs

The lungs of birds are much different than those of mammals, both morphologically and physiologically (Table 22.3). The paired lungs lie dorsally in the thoracic cavity, extending from the first through the seventh ribs in Psittaciformes; however, the boundaries of the lungs vary, and they may extend to the ilia in some species. The lungs are attached dorsally against the thoracic ribs and vertebrae, where they fill the intercostal space throughout their margins (see Color 14). When removed, the coastal surface of the lung will have an impression of the vertebral ribs (costal sulci). The sixth rib creates the most caudal sulci in Psittaciformes (see Anatomy Overlay).

There are frequent and inaccurate suggestions that the avian lung is fixed and not expandable. While changes in the size or position of the avian lung are limited, it is a dynamic organ that does undergo expansion and contraction during the respiratory cycle.[44]

The lungs are connected to the distal trachea (syrinx) by the primary bronchi, which progressively divide into secondary bronchi and tertiary bronchi (parabronchi) (Figure 22.5). Parabronchi connect to the secondary bronchi and other parabronchi, which have shallow depressions (atria) evenly displayed along their walls. Each depression has three to six funnel-shaped ducts (infundibula), which lead to the air capillaries. The air capillaries form an anastomosing three-dimensional network.[44] The air capillaries are intermittently interwoven with the blood

TABLE 22.3 Differences in Avian and Mammalian Respiratory Systems

Bird	Mammal
No diaphragm	Active diaphragm
Air sacs	No air sacs
Communicating air capillaries	Alveoli (blind sacs)
Syrinx	No syrinx
Complete tracheal rings	Open tracheal rings
No thyroid cartilage	Thyroid cartilage
No laryngeal vocal cords	Laryngeal vocal cords
No epiglottis	Epiglottis
Limited lung expansion	Highly expansible lungs

Pneumonology

Color 22.1
Chronic sinusitis may cause the globe of the eye to retract into its socket (sunken sinus syndrome). The problem is usually unilateral but on occasion may occur bilaterally. The pathogenesis of this lesion is unclear. Some cases will resolve when the sinusitis is resolved (courtesy of L. Karpinski).

Color 22.2
An advanced case of mycoplasmosis in a Galliforme associated with symblepharon, infraorbital sinusitis and rhinitis. The avian sinuses are not restricted laterally by bone (as they are in mammals), and sinusitis is frequently associated with facial swelling (courtesy of R. Korbel).

Color 22.3
Inflammatory reactions in the sinuses can involve most of the structures of the head. In this Blue-fronted Amazon Parrot, chronic sinusitis is associated with an infraorbital sinus fistula, nostril damage, a beak deformity, conjunctivitis and keratitis. Because of the complex extensions of the nasal system, treatment for chronic sinusitis usually requires surgical drainage (courtesy of R. Korbel).

Color 22.4
Cockatiels frequently develop an upper respiratory disease characterized by sinusitis, conjunctivitis or both. *Mycoplasma* spp. and *Chlamydia* sp. are frequently implicated in these cases. Conjunctival scrapings collected for cytology and culture are most useful in identifying an etiologic agent. If an infectious agent cannot be determined, these birds frequently respond to long-term treatment with tylosin eye wash and lincomycin/spectinomycin in the drinking water or an ophthalmic solution containing chlortetracycline (conjunctivitis only) or doxycycline (conjunctivitis and sinusitis).

Color 22.5
An adult cockatiel was presented with a two-week history of sneezing, lethargy and a nasal discharge. The bird had been receiving over-the-counter medications in the water for ten days. A severe conjunctivitis and mucopurulent rhinitis were noted on physical examination. Additionally, several periocular masses were evident. Cytology of samples collected from the masses indicated an accumulation of mixed gram-negative bacteria. The bird did not respond to therapy. At necropsy, granulomatous infraorbital sinusitis was evident.

Color 22.6
A gallinaceous bird was presented with a three-day history of progressive lethargy, anorexia and upper respiratory disease. The bird was part of a backyard flock in which intermittent deaths had been occurring in birds with similar clinical signs. This bird did not respond to therapy. Newcastle disease virus was isolated from the bird. A vaccination program was initiated in the flock.

Color 22.7
A herpesvirus that is serologically distinct from Pacheco's disease virus has been isolated from Amazon parrots with proliferative tracheitis. **a)** The diphtheritic membranes in the trachea cause severe dyspnea and death. In this case, the pharyngeal mucosa is covered with necrotic tissue. **b)** Bronchopneumonia and the presence of necrotic casts in the trachea and bronchi are characteristic (courtesy of Helga Gerlach).

Color 22.8
A cockatoo neonate died seconds after being hand-fed. The chick had placed its glottis on the ending of the feeding tube, and food was deposited in the trachea and lungs.

Color 22.9
A five-month-old ostrich had been losing weight for several days. In an attempt to provide supportive care, the client passed a feeding tube and delivered a liquid-based product. The bird began stretching its neck and became frantic. It died several minutes later. The client had passed the tube into the trachea instead of the esophagus, resulting in asphyxiation (courtesy of Brett Hopkins).

Color 22.10
Mycobacterial pneumonia in a Border Canary (courtesy of Louise Bauck).

Color 22.11
A breeding Umbrella Cockatoo was found dead in its nest box. The bird was in excellent overall condition and had blood-tinged fluid in the mouth. The lungs were edematous and hemorrhagic. Sarcocystosis was diagnosed histologically. Inhalation of noxious gases and fumes from non-stick cooking surfaces can cause similar gross lesions in the lung.


22.1


22.2

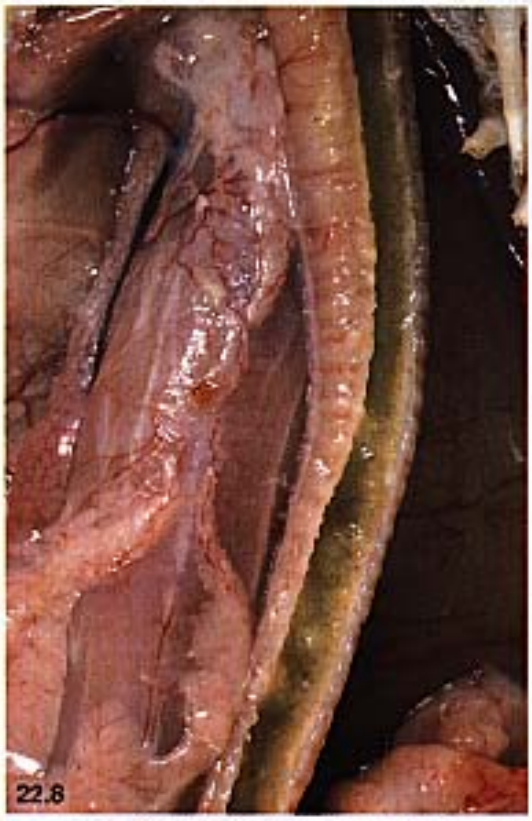

22.3

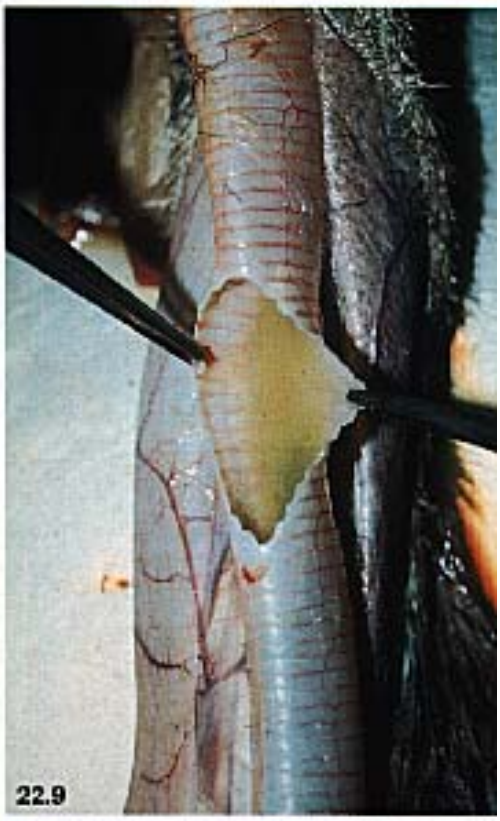

22.4

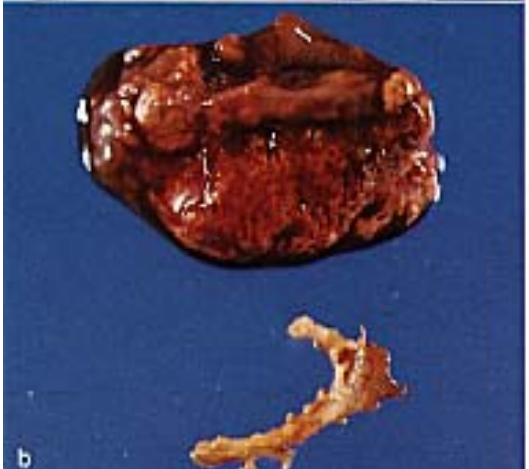

22.5

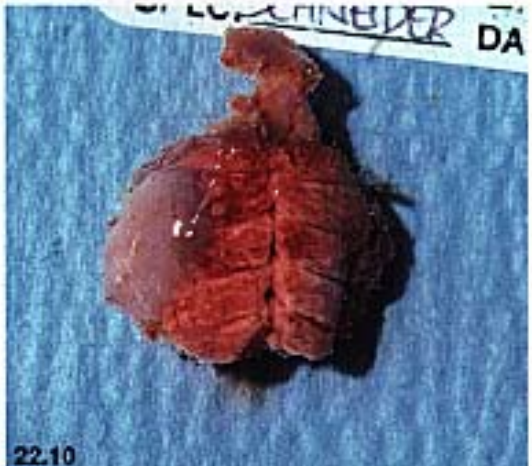

22.6


22.7a

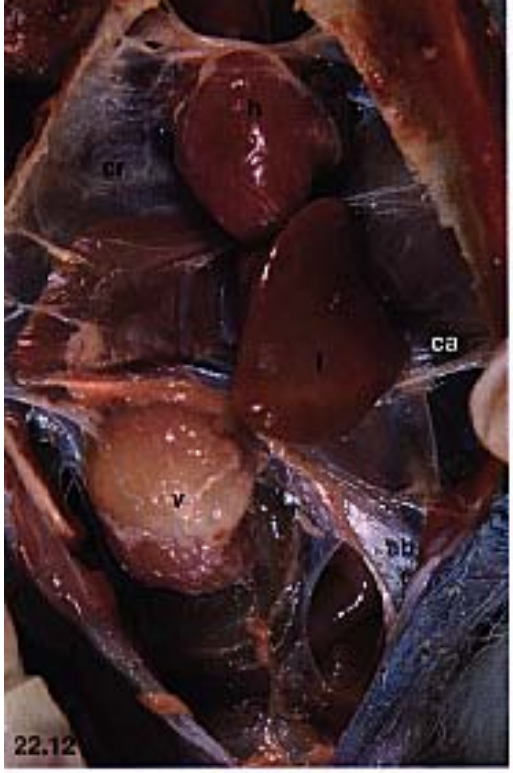

22.8


22.9

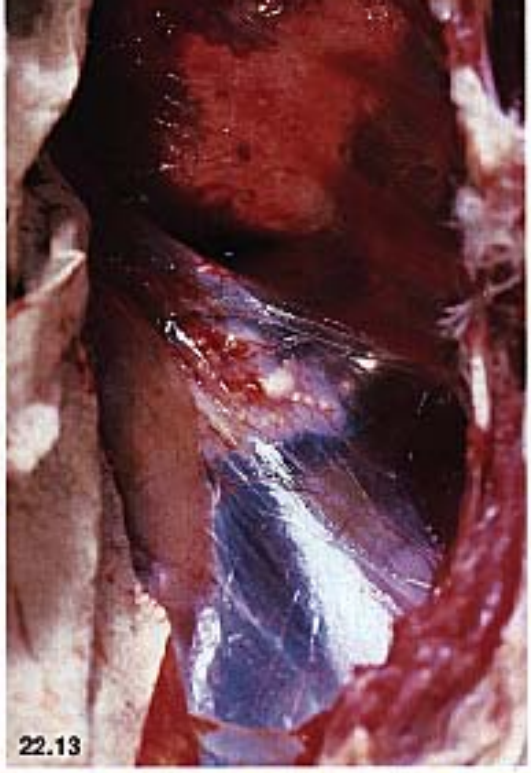

b

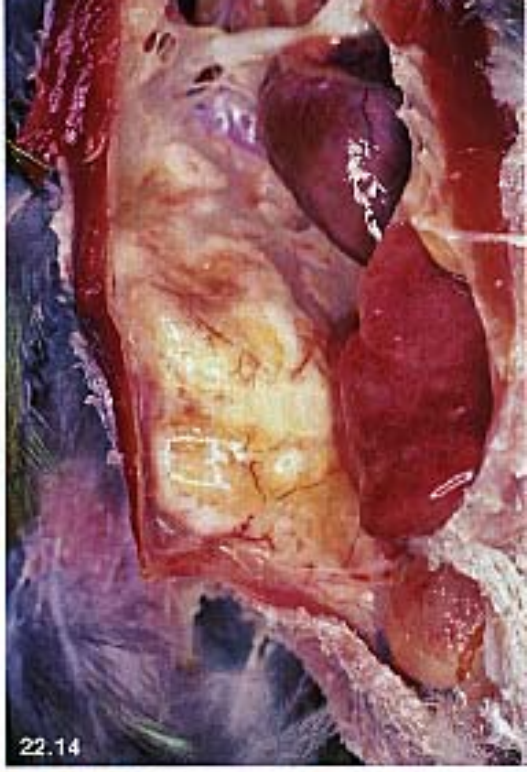

22.10

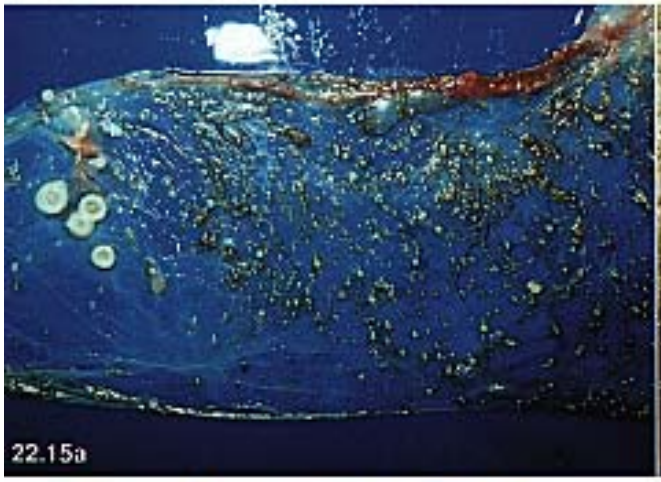

22.11

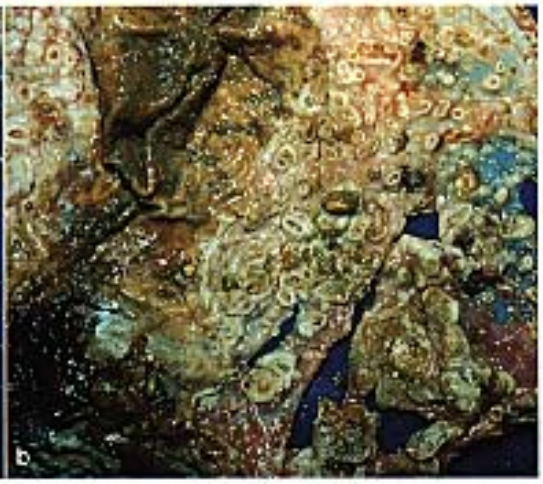


22.12

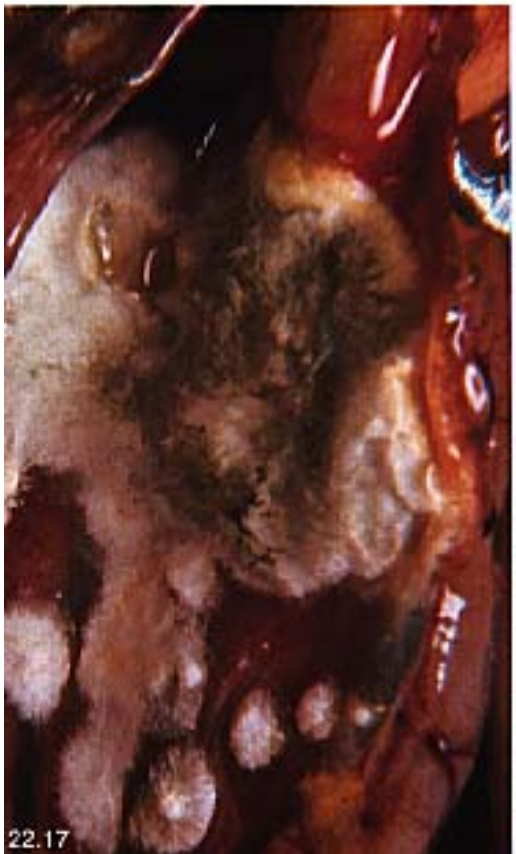

22.13

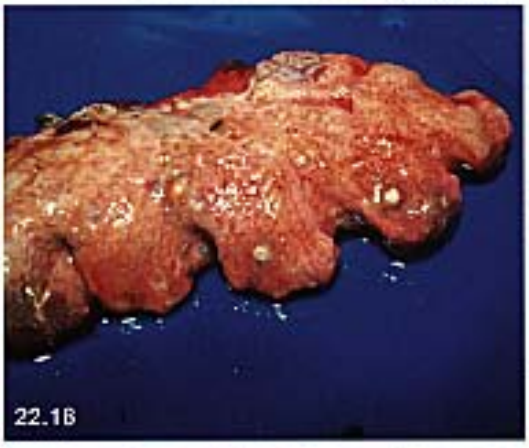

22.14

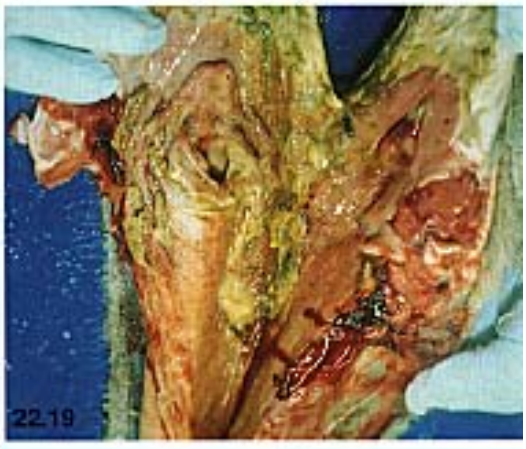

22.15a

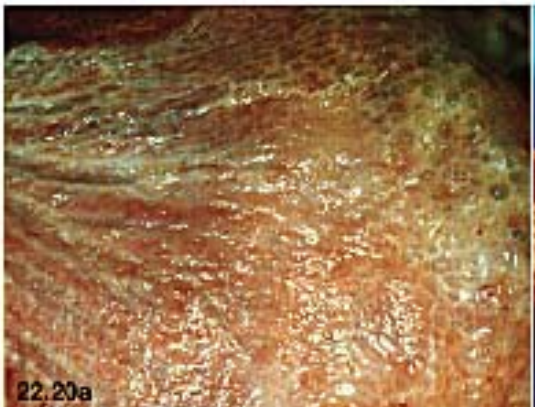

b

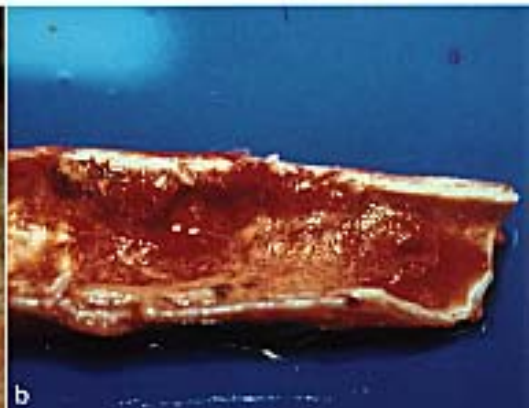

22.16


22.17


22.18


22.19


22.20a


b

Pneumonology

Color 22.12
Normal air sacs should be completely transparent and lack vascularity. Heart (h), liver (l), ventriculus (v), cranial thoracic air sac (cr), caudal thoracic air sac (ca), abdominal air sac (ab) (image is reversed).

Color 22.13
Several white proliferative masses were noted on the cranial thoracic air sac during a routine necropsy of a cockatoo with PBFD. Impression smears stained with new methylene blue revealed branching septate hyphae characteristic of *Aspergillus* spp. These plaques were considered an incidental finding.

Color 22.14
A five-month-old Amazon parrot was presented with a history of progressive dyspnea. The bird had a severe tail-bob, was open-mouthed breathing and was in severe distress. The bird was anesthetized with isoflurane and an air sac tube was placed in the abdominal air sac. Radiographs indicated a soft tissue mass in the right thoracoabdominal cavity. The client chose euthanasia because of the extent of the mass. At necropsy, a large, yellow mass that involved portions of the lung and thoracic air sacs was identified. Histopathology indicated bacterial pneumonia and air sacculitis. Ingesta was also noted, confirming a diagnosis of aspiration pneumonia.

Color 22.15
a) Four white plaques suggestive of aspergillosis in a relatively normal thoracic air sac from an ostrich. **b)** Severe, necrotic fungal air sacculitis in a 22-month-old ostrich. The primary isolate was *Aspergillus* sp., although other unisolated fungi were suspected to be the cause of the black discoloration. This fungal infection was secondary to aspiration of ingesta. Note kernels of corn (courtesy of Brett Hopkins).

Color 22.16
Aspergillosis granulomas in the trachea of a Pintail Duck (courtesy of R.J. Montali).

Color 22.17
Aspergillosis will frequently produce proliferative fluffy lesions in the air sacs. Colonies in the oxygen-rich areas of the lungs and air sacs frequently produce characteristic conidiophores. The aspergillosis lesions in this case were present on the abdominal air sacs of a Buttlehead Duck (courtesy of R.J. Montali).

Color 22.18
Aspergillus spp. pneumonia in a six-month-old emu with progressive dyspnea that was not responsive to antibiotic therapy. Note the multiple white-tan granulomas. The depressions in the lung (costal sulci) represent the areas where the lung folds around the ribs (courtesy of Brett Hopkins).

Color 22.19
Pseudomonas spp. can cause severe respiratory disease in ratites. A two-year-old ostrich hen with a history of respiratory disease had fibrinonecrotic pharyngitis (courtesy of Brett Hopkins).

Color 22.20
a) Severe emphysema and collapse of the secondary bronchi and **b)** hemorrhagic pseudomembranous tracheitis caused by *Pseudomonas* spp. in an ostrich (courtesy of Brett Hopkins).

capillary network to form the exchange tissue of the lung.[44] On the ventral surface of the lung, secondary bronchi connect directly to the caudal thoracic and abdominal air sacs through ostium that can be visualized during endoscopy (see Color 13).

Surfactants in the parabronchi function to keep fluids from entering the air capillary area and prevent transudation. These functions combine to maintain the integrity of the delicate blood gas barriers.[44] Dilation and contraction of the bronchi and ostium are controlled by smooth muscles. The innervation to these muscles is non-vagal and can be relaxed with adrenergic drugs.[44]

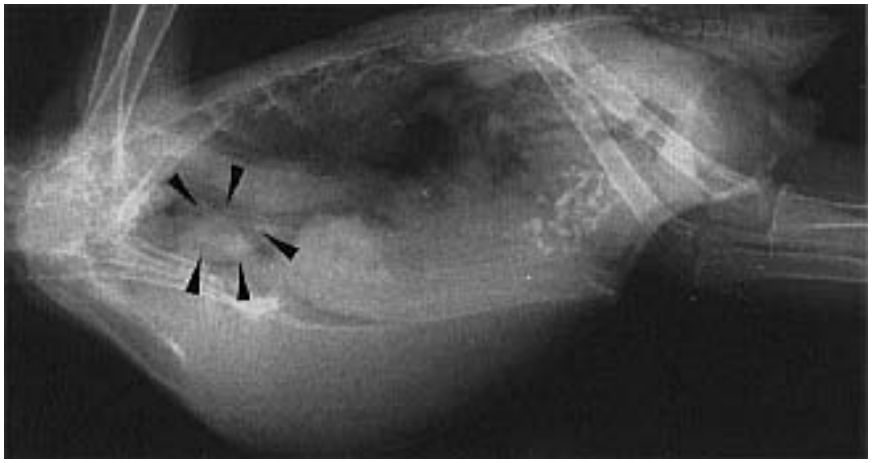


FIG 22.4 An adult African Grey Parrot was presented with acute onset of severe dyspnea accompanied by open-mouthed breathing. Radiographs revealed a soft tissue mass at the level of the syrinx (arrows). The lungs and air sacs were considered radiographically normal. Tracheoscopy was performed with a 2.7 mm endoscope while the animal was maintained on isoflurane anesthesia delivered through an air sac tube. Cultures taken from a syringeal granuloma were positive for *Aspergillus* spp. (courtesy of M. McMillan).

From a functional standpoint, the avian lung is divided into a paleopulmo (which all birds have and which constitutes at least 75% of the lung) and the neopulmo (which some birds have and which makes up no more than 30% of the lung). The neopulmo is absent in penguins, minimally developed in emus, further developed in ducks and psittacine birds and maximally developed in pigeons and gallinaceous and passerine birds. The reasons for this division have not been clearly established, but it has been determined that the neopulmo is less efficient at gas exchange than the paleopulmo.[40] The fact that the neopulmo is less efficient is interesting, considering it is highly developed in one of the fastest flying birds, the pigeon.

Air Sacs

Pulmonary

Most birds have four paired and one unpaired pulmonary air sacs that connect to the lung and create a large respiratory capacity (see Anatomy Overlay). The configuration of the air sacs varies with the species. Most birds, including Psittaciformes, are believed to have four paired air sacs that include the cervical, cranial and caudal thoracic and abdominal air sacs. An unpaired clavicular air sac lies dorsal and caudal to the crop in the thoracic inlet and has both intra- and extrathoracic components. The intrathoracic component surrounds the great vessels, esophagus and syrinx with diverticula into the sternum and sternal ribs. The extrathoracic component represents diverticula into the thoracic girdle (see Anatomy Overlay).

The cranial air sacs are composed of the cervical, clavicular and cranial thoracic air sacs; the caudal air sacs are composed of the caudal thoracic air sac and abdominal air sac. The cranial thoracic air sacs receive air via the medioventral parabronchi and are physiologically components of the paleopulmonic air sac system. The caudal thoracic air sac, on the other hand, gets its air from lateroventral parabronchi and, along with the abdominal air sacs, is part of the neopulmonic air sac system.[44]

Cervicocephalic

The cervicocephalic air sacs are not connected to the lung and are divided into cephalic and cervical portions; they connect to caudal aspects of the infraorbital sinus (see Anatomy Overlay). Extensive cervicocephalic air sac development has been noted in budgerigars, cockatiels, conures, Amazon parrots, macaws and cockatoos. This air sac is absent in diving birds, partially developed in ratites, pigeons and chickens and is well developed in strong-flying avian species. The cervicocephalic air sacs may function as insulating air layers for the retention of heat, to control buoyancy, to reduce the force of impact with the water in fish-eating birds and to support the head during sleep or flight.[44]

In some species, the cephalic portion is large, and in others it is minimally developed. Studies involving budgerigars, conures and cockatiels suggest that the cephalic air sacs arise from the infraorbital sinus and extend dorsally to cap the dorsum of the skull.[72] In the Amazon parrot, the cephalic portion of this air sac

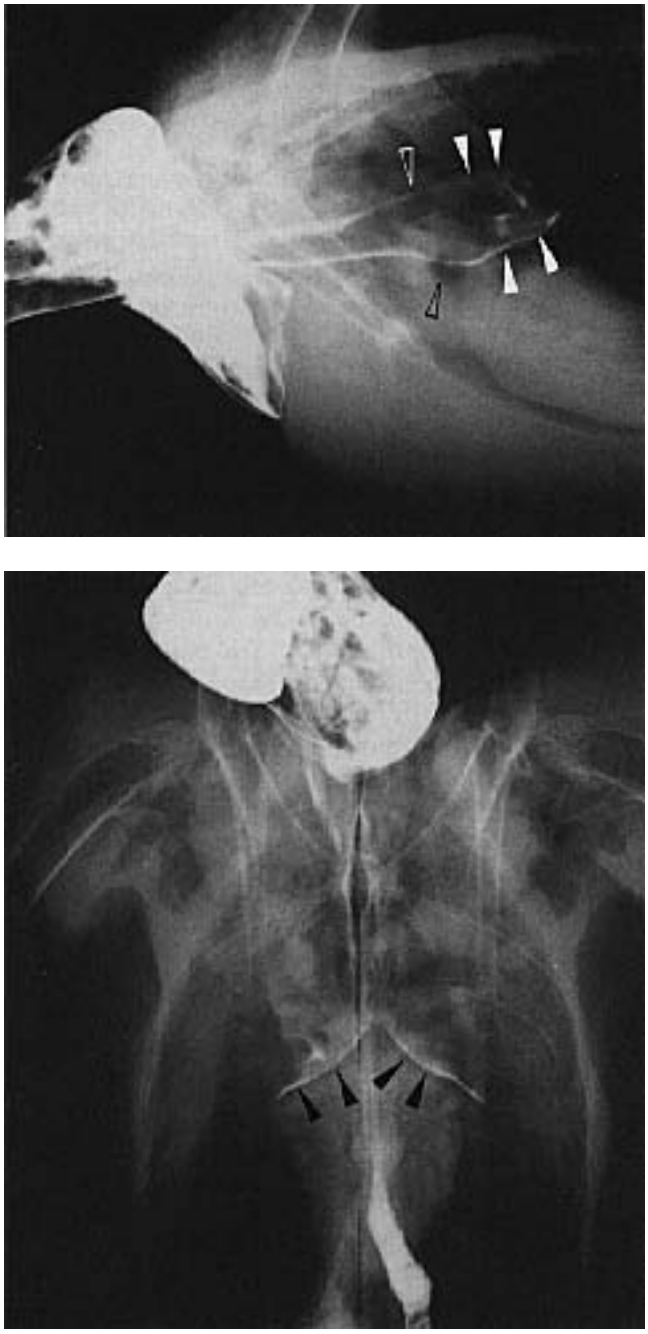


FIG 22.5 The syrinx (open arrow) and primary bronchi (arrows) are clearly visible in this macaw that aspirated barium. The barium could not be detected in the respiratory system on a subsequent radiograph taken 90 minutes later.

extends dorsally adjacent to the occipital bone. The cervical portion extends bilaterally dorsolaterally in the neck from the head to the distal neck (Figure 22.6).[72] The cervical division of the air sac communicates with the cephalic part by a small median pathway. The cervicocephalic air sac covers the lateral aspect of the head and extends caudally over the dorsal cervical region to the shoulder area.

No direct connection has been found between the cervicocephalic air sac system and any of the pulmonary air sacs. All air sacs are thin-walled and lack vascularity. The air sacs of a normal bird are completely transparent (appear similar to clear plastic wrap) (Color 22.12). Any alteration in transparency should be considered abnormal. The presence of blood vessels in the air sacs may be an indication of early inflammation. Blood vessels that transverse inflamed abdominal air sacs must be avoided during surgical procedures.[61] Air sac lesions that are localized and do not alter the flow of air in or out of the air sacs may not cause clinical changes (see Color 13.).

The trachea, primary bronchi and larger secondary bronchi are lined with pseudostratified or simple columnar ciliated epithelium, whereas the air sacs distal to the connection with the lungs are lined with a single layer of simple squamous epithelial cells. The area of the air sacs near the lung may contain simple cuboidal and columnar ciliated epithelium.[44] The poor vascular supply and lack of ciliary transport system within the air sacs hinder parenteral treatment of air sacculitis.[52]

Depending on the species of bird, the humerus, clavicles, coracoids and cervical vertebrae are connected to the respiratory system through extrathoracic diverticula. The sternum and sternal ribs are pneumatized through the intrathoracic diverticula that lie between the coracoid bones. The lungs connect directly to the thoracic vertebrae and their associated ribs. The femur may be pneumatized through a connection with the air sac (see Anatomy Overlay).

Respiratory Physiology

Birds have no functional diaphragm. The thoracic cavity is separated from the abdominal cavity by a thin membrane called the oblique septum. Birds breathe by using the six inspiratory muscles (principally the external intercostales) to pull the ribs cranially, laterally and ventrally and to move the sternum ventrally and cranially, increasing the volume of the thoracoabdominal cavity. These changes in the body wall create a negative pressure with respect to

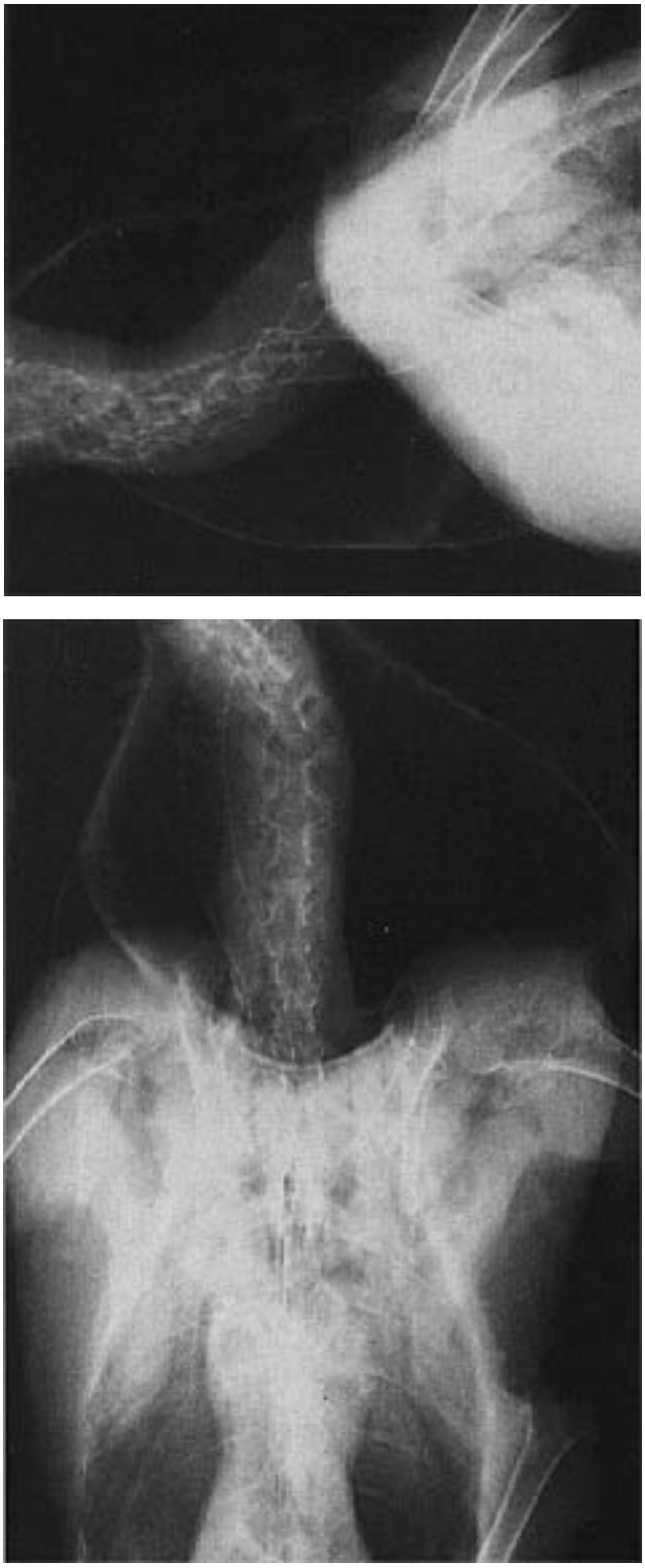


FIG 22.6 A Yellow-naped Amazon Parrot was presented for a persistent swelling in the cervical area. Radiographs indicated gaseous distension of the cervical air sac with no other evidence of respiratory disease. Biopsies and cultures of the air sac were unremarkable. A permanent opening (stent) was created in the air sac to resolve the problem (courtesy of Marjorie McMillan).

the atmospheric air pressure surrounding the bird, causing air to pass through the respiratory system and into the inspiratory portion of the respiratory tract.

The nine expiratory muscles (principally the internal intercostals and abdominals) pull the ribs caudally, raising the sternum and pulling the ribs inward, causing expiration by creating increased internal pressure within the air sacs. This forces air out of the air sacs and back through the parabronchi (caudal air sacs) or trachea (cranial air sacs).[44]

It is frequently discussed that the oxygen (O_2) and carbon dioxide (CO_2) content of air in the caudal air sacs is similar to that in the environment. In reality, the caudal air sacs contain air that is higher in CO_2, possibly because of some dead space gases that remain in the caudal air sac following expiration.[44] The O_2 and CO_2 content of air in the cranial air sacs is similar to that of expired air. The rapid influx of inspired air into the caudal air sacs and the similarity of this air to environmental air have been used to explain the apparent prevalence of air sac infections and pathology in the caudal air sacs versus the cranial air sacs; however, it should be noted that half the inspired air enters the lungs. The prevalence of caudal air sacculitis may be a reflection of the air layering that occurs in this location.

In pigeons, barely detectable tail movements have been shown to be associated with inspiration (minimally) and expiration. The M. caudofemoralis, M. pubocaudalis internus and M. pubocaudalis were found to be involved with expiration by depressing the pelvis and uropygium and compressing the thoracoabdominal cavity. The M. longissimus dorsi was found to be involved in inspiration.[6] These findings would suggest that tail-bobbing is an exaggerated movement of a normal component of inspiration and expiration and is a reflection of an increased amount of work necessary to ventilate the lungs. The tail muscles seem to be most involved in respiration when a bird is resting on its keel, or the sternum is fixed in position.

If a bird is unable to move its ribs, it will rapidly suffocate. This can occur with an overly aggressive restraint or by the surgeon resting his hands on the body cavity during surgery. Bandages that encompass the body cavity can also interfere with breathing, particularly if they are wrapped tightly around the caudal portion of the sternum or ribs.

It is frequently discussed in veterinary literature that inspired air flows through the parabronchi or primary bronchus and directly into the caudal air sacs, thus bypassing the gas exchange portion of the lungs. This statement is not completely accurate. On inspiration, one-half of the inspired air volume goes to the lung and the other half goes to the caudal air sacs. The air that is already in the lungs enters the cranial air sacs. On expiration, the ambient air that is in the caudal air sacs enters the lungs. Although not clearly stated in any physiology reference, the air that is in the lungs must exit through the trachea along with the air that is in the cranial air sac. For this system to function, the volume of the caudal air sacs, the lungs and the cranial air sacs must be equal (each contains one-half of a total volume of inspired air at any one time) (Figure 22.7).

Two respiratory cycles are necessary for the one-half volume of air that enters the air sacs to move totally through the avian respiratory tract. Superficially, this would appear to be relatively inefficient, but in reality, it is much more efficient than the mammalian system. In birds, fresh air (fresh air delivered directly to the lungs on inspiration or fresh air delivered directly to the lungs on expiration from the caudal air sacs) enters the lungs on both inspiration and expiration.[44]

Some studies suggest that birds have a fluid valving system that controls the unidirectional air flow through the lungs and air sacs.[45] Other authors suggest that pressure differentials between the cranial and caudal air sac systems control the movement of air through the respiratory tract.[44]

Gas Exchange

The air capillaries are present in all birds. In some species, the parabronchi are divided into two systems. In these birds, the paleopulmonic parabronchi are the major sites of gas exchange, and air flows unidirectionally through these passages on inspiration and expiration. In the neoplumonic parabronchi, air passes bidirectionally through both phases of the respiratory cycle. Gas exchange occurs in the air capillaries. These air tubes branch and anastomose with each other, creating an extensive network. They are richly entwined with blood vessels, which form a blood gas barrier.[60] The pulmonary artery transports the less oxygenated blood into the interperibronchial arteries of the lungs. The interperibronchial arteries are located in the area of gas exchange and are arranged so that blood flows perpendicular to the air capillaries.

TABLE 22.4 Influences on Respiratory Rate

Increase Respiration	Decrease Respiration
Restraint	Anesthesia
Hyperthermia	Hypothermia
Low CO_2 levels in inspired air	Inhalation of toxic fumes
Increased respiratory dead space	High CO_2 levels in inspired air
Obesity	Severing of vagus nerve(s)
Pain	Air sac oxygen administration
Exercise	Sleep

This barrier has the same three components as in mammals; however, it is anatomically not as thick as in mammals due to the reduced width of the epithelial cells and decreased tube size, which is much smaller that the mammalian alveolus. The efficiency of gas exchange is thus greater. However, with infection and its associated inflammation, a greater ventilation perfusion mismatch can occur.

In the paleopulmonic system, a current of parallel tubes of air moves in one direction counter to pulmonic vessels, allowing gas exchange to occur with greater efficiency than in the neopulmonic system or in the alveolus of mammals. In the latter case, air moves in both directions in the parabronchi, mixing oxygenated air with air having a higher partial pressure of carbon dioxide. These physiologic and anatomic components account for a 20% increase in the diffusion capacity for oxygen in birds when compared to mammals.[44]

The respiratory cycle is controlled principally by sensitive CO_2 pulmonary receptors. Interestingly, these receptors have been shown to be inhibited by halothane.[44] Other receptors that are integrally involved in controlling respiration include dermal pain receptors, thermoreceptors (control panting), chemoreceptors, baroreceptors in the aorta and mechanicoreceptors in the respiratory tract (see Chapter 39).[44]

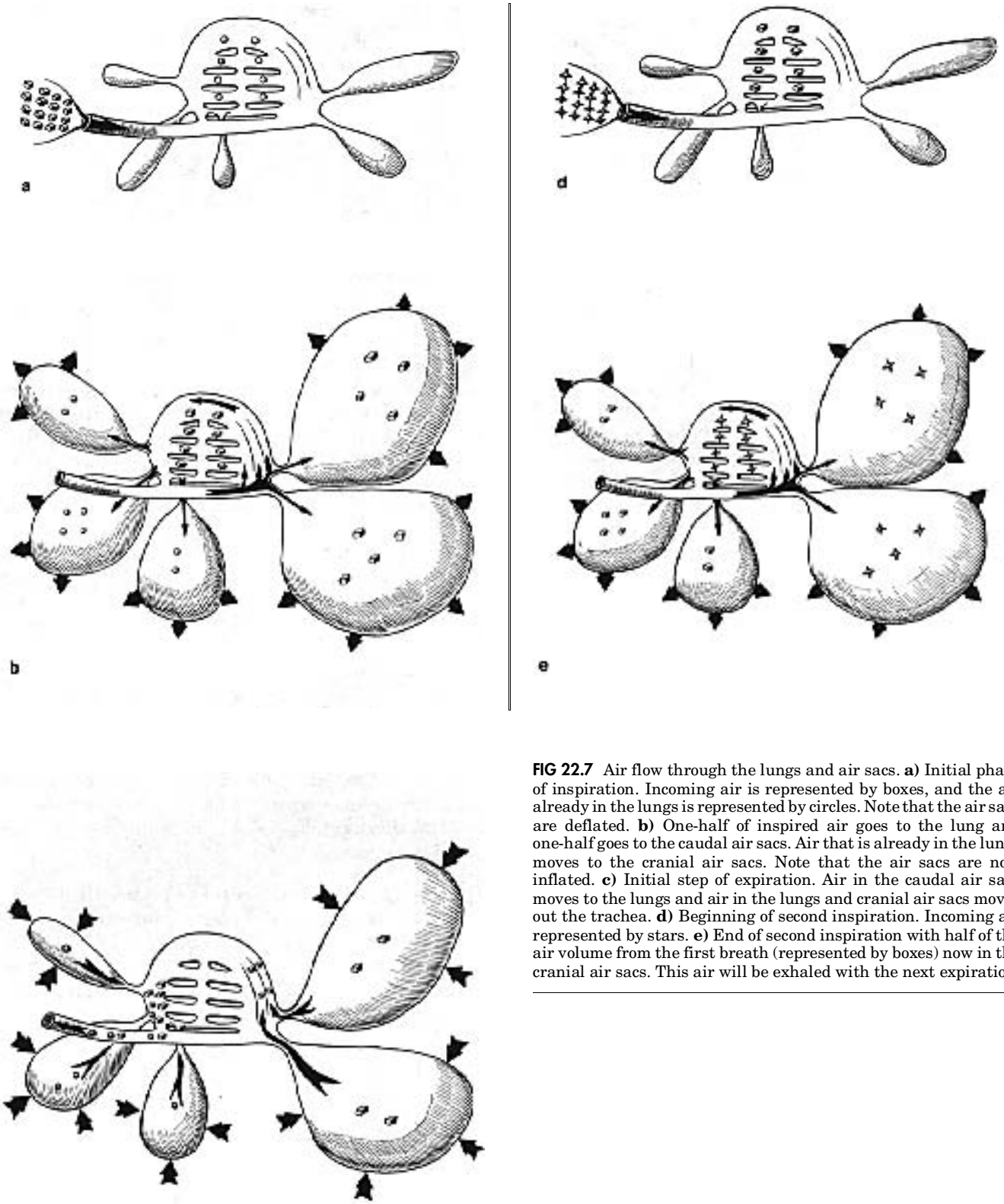


FIG 22.7 Air flow through the lungs and air sacs. **a)** Initial phase of inspiration. Incoming air is represented by boxes, and the air already in the lungs is represented by circles. Note that the air sacs are deflated. **b)** One-half of inspired air goes to the lung and one-half goes to the caudal air sacs. Air that is already in the lungs moves to the cranial air sacs. Note that the air sacs are now inflated. **c)** Initial step of expiration. Air in the caudal air sacs moves to the lungs and air in the lungs and cranial air sacs moves out the trachea. **d)** Beginning of second inspiration. Incoming air represented by stars. **e)** End of second inspiration with half of the air volume from the first breath (represented by boxes) now in the cranial air sacs. This air will be exhaled with the next expiration.

Diagnostic Techniques

Auscultation

The sinuses, trachea, lung, thoracic air sacs and abdominal air sacs can be auscultated using a pediatric stethoscope. Audible sounds on inspiration generally correlate with upper respiratory tract disease, while sounds on expiration are more commonly associated with lower respiratory tract diseases.[35,53,59] Because air moves through the lungs continuously and the air capillaries do not collapse and expand to the same degree as alveoli, a "smacking" sound characteristic of pneumonia in mammals does not occur in birds (Figure 22.8). However, mild respiratory lesions may be associated with audible respiratory sounds, while auscultation may be normal in patients with severe air sac pathology. Placing a thin towel around the bird and ascultating through the towel will actually enhance the clinician's ability to detect respiratory sounds.

With bacterial, fungal and parasitic diseases, harsh sounds may be heard on auscultation when air moves through narrowed parabronchi. Air sac pathology is best detected by placing the stethoscope along the lateral and dorsal body wall. An increased respiratory rate, particularly with dyspnea, is indicative of respiratory tract pathology, and harsh sounds may indicate chronic air sac or parabronchi pathology.[65] Use of a small amount of wing flapping (exercise) serves to increase respiratory rate and accentuate pathologic sounds. The amount of time for the bird to return to normal respiration (respiratory recovery time) is usually under two minutes even in obese birds. Prolonged respiratory recovery time is an indication that further diagnostic tests are necessary.

Imaging

Radiography and endoscopy (with biopsy and culture) are the most effective diagnostic techniques for avian respiratory disease. Radiographically, generalized air sacculitis may be recognized by the appearance of air sac lines on lateral radiographs. Radiographic interpretation of the avian respiratory tract is different from mammals. Interstitial patterns, air bronchograms and atelectasis do not occur in avian radiography.[54] Radiographs are usually of little value in diagnosing acute sinus infections but may be of value, particularly with respect to documenting involvement of bones in the head, with chronic inflammatory processes. Rhinography and sinography are helpful in the diagnosis of upper respiratory tract problems (see Chapter 12).[54]

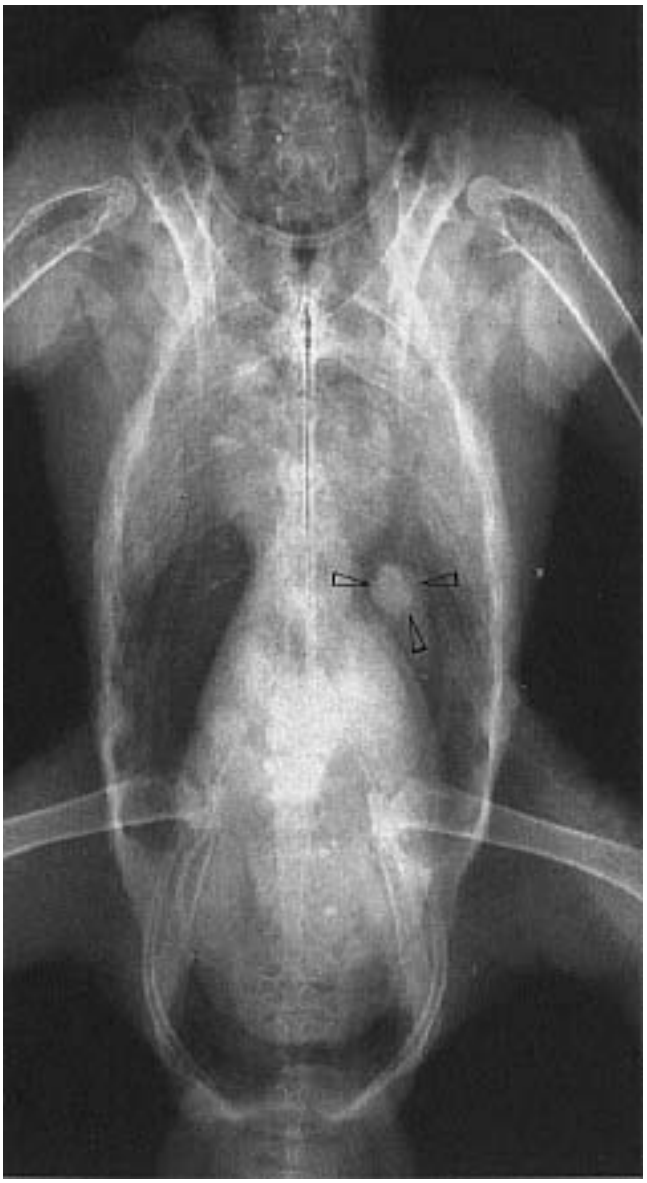


FIG 22.8 A Blue and Gold Macaw was presented with progressive dyspnea of one month's duration. The bird had stopped eating and was depressed for two days before evaluation. Radiographs indicated a soft tissue mass in the left thoracic air sac region (arrows). The only change that could be detected by auscultation was decreased lung sounds. Endoscopy revealed a granulomatous mass and diffuse air sacculitis. The mass was surgically removed and the bird responded to therapy with broad-spectrum antibiotics.

The ventrodorsal view should be used to assess the subtle disorders of the lung and air sacs. Radiographically, soft tissue masses are commonly associ-

ated with upper respiratory signs, pneumonia and air sacculitis (Figure 22.8).[54] On a lateral radiograph, the trachea of normal toucans and mynah birds deviates ventrally, which should not be misinterpreted as a displacement caused by a soft tissue mass. In many ducks, the male has an enlargement on the left side of the syrinx (syringeal bulla) that is not found in the female.

Sample Collection

The minimum database for respiratory problems includes cytology of samples collected from the affected area, a CBC, biochemistries, radiographs and, when indicated, endoscopy.

A sinus aspirate is important in determining the cause of sinusitis. There are several techniques that allow for minimal sample contamination and maximum microbial and cytologic examination. A patient must not move during this procedure or severe damage to the globe of the eye can occur (see Chapter 10). Aspiration of the right and left infraorbital sinuses is needed for diagnostic procedures in some passerines.[44] Samples collected from the rostral portion of the choanal slit may provide some useful information on the organisms present in the respiratory passages. Samples collected from the caudal choanal slit are of little diagnostic value with respect to the sinuses or nasal passages.

Tracheal Lavage

Tracheal lavage is indicated when pathology of the trachea or lower respiratory system is suspected. The procedure is relatively simple but requires general anesthesia in most avian patients.[40] A normal wash should be low in cellularity with a minimum of pulmonary macrophages or inflammatory cells.[13]

Increased numbers of heterophils, pulmonary macrophages and other inflammatory cells in the lavage fluid are clinically important.[13] In a severely dyspneic bird, a large-gauge hypodermic needle or a respiratory catheter placed in the abdominal air sacs will help the patient breathe while the procedure is performed.

An intratracheal wash is performed by placing the bird in dorsal recumbency and passing a sterile, soft plastic or rubber tube (eg, Rob-nel catheter) through the glottis into the trachea, ending near the syrinx (just caudal to the thoracic inlet). A sterile saline solution (0.5 to 1.0 ml/kg body weight) is infused into the trachea and reaspirated in the sterile syringe attached to the tube.[40]

A sterile endotracheal tube may be placed within the trachea prior to inserting the lavage tube to prevent sample contamination as the lavage tube is passed through the oral cavity. Tracheal swab samples for microbiology evaluation may be taken by passing a small sterile cotton swab directly into the trachea.

A transtracheal lavage can be performed by sterilely placing an 18 to 22 ga Teflon indwelling catheter through the skin and into the trachea. The bird is held parallel to the floor and fluid is instilled and immediately removed. This procedure can be performed in some patients without anesthesia.

Endoscopy

An endoscope may be used to diagnose respiratory problems associated with the trachea, air sacs or lungs. Small-diameter, rigid or flexible endoscopes can be inserted to the syrinx in some birds. A 1.9 mm endoscope easily passes to the syrinx in a 200 g parrot while a 1.7 mm scope will not reach the syrinx in a cockatiel.

Endoscopic evaluation of the air sacs can be performed on both the right and left side of the patient. The caudal surface of the lung, which normally appears pale pink and spongy, may also be observed during this procedure (see Chapter 13).

Diffuse air sacculitis, recognized endoscopically as vascularized, transluscent, thickened air sacs, commonly occurs with chlamydiosis, some viral diseases, poor air quality, bacterial infections and localized fungal infections. Granulomatous air sacculitis is difficult to resolve without surgery.

Air Sac Diagnostics

Cultures or biopsies of the air sacs can best be obtained using endoscopically guided procedures. Specially designed brushes are commercially available that will transverse the length of a sterile channel in the endoscope, eliminating the problem of coordinating the position of a separate endoscope and sample collecting device (see Chapter 13). Feather picking over the air sacs may be an indication of irritation that requires further investigation. The lung can also be biopsied using an endoscope (see Chapter 13).

A cytologic sample can be collected from the air sacs by passing a tube through an endoscopic cannula, lavaging with sterile LRS and immediately reaspi-

rating. Sterile cotton swabs may be used to obtain samples for bacterial or fungal cultures using the same technique.[40]

Lung Biopsy

Lung biopsies may be diagnostic in some cases of toxin inhalation and microbial or parasitic infections. This procedure does create the potential for localized pulmonary hemorrhage and should be performed with minimal trauma to the lungs (see Chapter 13).[41]

The approach to the lung can be achieved through either the caudal thoracic air sac or via an intercostal approach through the third intercostal space. Approaching through the caudal thoracic air sac provides the best view of the caudal aspect of the lungs while the intercostal approach is used to access the dorsolateral portion of the lung. In experimental pigeons, mild to moderate pulmonary hemorrhage occurred at the biopsy site using a 2.7 Fr (best biopsy sample but more severe hemorrhage) or 5 Fr biopsy forceps. The procedure is not without risk and should be considered only when other diagnostic techniques are ineffective or when a biopsy is necessary to determine and initiate life-preserving therapy.[41]

Aerosol Therapy

The use of a therapeutic solution that has been atomized into a fine mist is effective in treating upper respiratory tract infections.[66] Humidification, vaporization and nebulization are three types of aerosol therapy that have been used successfully to treat avian respiratory problems.

If the relative humidity of the environmental air is low, then humidification of inspired air may improve the efficiency of the mucociliary blanket.[66] In the clinical setting, humidification is used in conjunction with a therapeutic agent but can be prescribed without additives for home treatment. Any source of cool, moist air could be used.

Vaporization is a form of aerosol therapy that utilizes cool or warm mist to deliver topical medications to the mucous membranes.[66] Vaporized particles are large and do not reach the lower respiratory system. Eucalyptus-based products, available as over-the-counter medications for human vaporizers, may cause mucosal irritation in birds and should not be used.

Nebulization can be used to augment systemic therapy of some respiratory tract diseases. Nebulization can help maintain proper hydration of the respiratory epithelium, break up necrotic debris and deliver antimicrobial agents to the upper respiratory tract and portions of the lower respiratory tract. Nebulization therapy is indicated in birds exhibiting sinusitis, rhinitis, pharyngitis and bronchitis. Depending on the agents delivered, nebulization can be used three to four times per day for 10 to 15 minutes for each session. Therapy should be continued for three days after all clinical signs have been resolved.

The equipment needed for nebulization therapy includes an air compressor or some source of O_2, an enclosed chamber and an infant (human) nebulizer. The most important piece of equipment is the air compressor. An inexpensive reliable unit is commercially available,[a] which should satisfy most nebulization requirements. At least two sizes of nebulization chambers should be maintained, one for larger patients and one for small birds. It has been shown that nebulization can be used to deliver antimicrobial agents to the lungs and some portions of the air sacs if the particle size is less than 0.5 microns in diameter.

All medications delivered to birds by nebulization are used empirically and should be based at least on results obtained from culture and sensitivity (Table 22.5). Mucolytic agents should be used only with infections localized to the sinuses and trachea. Amphotericin B, gentamicin, polymyxin B and tylosin have been found to be poorly absorbed from the respiratory epithelium, and these agents are used principally for their local effects. However, penetration of nebulized antibiotic particles into avian lung parenchyma and onto air sac surfaces may be effective.

The addition of DMSO to the nebulization solution was found to increase the local and systemic concentration of nebulized tylosin.[40] However, the systemic effects of inhaling DMSO have not been evaluated. Nebulized tylosin required one hour to reach therapeutic concentrations in the air sacs and lungs of pigeons and quail.[67]

Other therapeutic agents that have been reportedly used for nebulization therapy in birds include acetylcysteine, sodium tris-EDTA, levamisole phosphate and corticosteroids.[12] The use of immunosuppressive drugs such as corticosteroids in nebulization therapy should be avoided. Acetylcysteine may be added to

nebulization therapy for upper respiratory diseases in which the exudates can be physiologically removed, but it should not be used for lower respiratory treatment because of a bird's inability to rapidly remove exudates from the air sacs.

TABLE 22.5 Medications Commonly Used in Nebulization Therapy[5]

Drug	Dosage
*Amphotericin B	100 mg in 15 ml saline
Chloramphenicol succinate	200 mg in 15 ml saline
Erythromycin	200 mg in 10 ml saline
*Gentamicin	50 mg in 10 ml saline
*Polymyxin B	333,000 IU in 5 ml saline
Spectinomycin	200 mg in 15 ml saline
Sulfa dimethoxine	200 mg in 15 ml saline
*Tylosin	100 mg in 10 ml saline, 1 g in 50 ml DMSO
*Amikacin	50 mg in 10 ml saline
Enrofloxacin	100 mg in 10 ml saline

These drugs may also be delivered by transtracheal injection.
*Poorly absorbed from the respiratory epithelium; provides primarily topical therapy.

Specific Respiratory Diseases

Table 22.6 lists the most common etiologic agents associated with respiratory diseases in birds (see Figure 22.11).

Nutritional Disorders

Hypovitaminosis A has been associated with hyperkeratosis, abscessation of the palatine salivary glands and other oral salivary glands and respiratory lesions in psittacine birds (see Color 8.).[56,68] Improving a bird's diet and providing oral vitamin supplementation and parenteral administration of vitamin A will prevent and eliminate nutritional deficiencies, support ill patients and speed recovery time in patients with respiratory infections. It should be noted that with the widespread use of formulated diets, hypovitaminosis A is less commonly encountered than it was a decade ago.

TABLE 22.6 Selected Etiologic Agents of Avian Respiratory Disease

BACTERIAL
- *Chlamydia psittaci*
- *E. coli*
- *Mycoplasma*
- *Pseudomonas*
- *Klebsiella*
- *Salmonella*
- *Mycobacterium avium*
- *Proteus*
- *Haemophilus*
- *Bordetella avium*
- *Pasteurella*
- *Streptococcus*
- *Staphylococcus*

NUTRITIONAL
- Vitamin A deficiency
- Iodine deficiency
- Obesity
- General malnutrition

TOXIC
- Polytetrafluroethylene gas
- Formaldehyde
- Quaternary ammonium
- Creosote
- Chlorinated biphenyl
- Carbon monoxide
- Cigarette smoke
- Naphthalene
- High ammonia
- Airborne particulate matter
- Zinc

VIRAL
- Adenovirus
- Paramyxovirus
- Laryngotracheitis virus
- Influenza virus
- Infectious bronchitis virus
- Avian poxvirus

PARASITIC
- *Sternostoma* (tracheal mites)
- *Cytodites* (tracheal mites)
- *Cyathostoma*
- *Syngamus*
- *Sematospiculum* (nematode)
- Cryptosporidia
- *Trichomonas*
- Coccidia (systemic)
- Hematozoa
- *Knemidokoptes* (scaly face mites)
- *Sarcocystis*

FUNGAL
- *Aspergillus*
- *Candida*
- *Mucor*
- *Cryptococcus*

Infectious Organisms

Chlamydia psittaci and *Mycoplasma* spp. are obligate intracellular organisms with a predilection for respiratory epithelium and have been implicated in cases of rhinorrhea, infraorbital sinusitis and inflamed choanae.[34,73] Both organisms may persist as low-grade upper respiratory tract infections. This is particularly common in birds that are treated with immunosuppressive, over-the-counter antibiotics.

Mycoplasma spp. have been proposed as causes of upper respiratory and ocular infections in cockatiels and budgerigars, although documented cases are rare (see Color 22.2 and Chapter 38).[27,30,36] *C. psittaci* and *Mycoplasma* spp. organisms are difficult to isolate and can cause similar clinical signs, which complicate a definitive diagnosis. These organisms have also been isolated from tissues of clinically asymptomatic birds.[65] Birds with suggestive clinical signs frequently respond to treatment with tetracyclines, tylosin or spectinomycin. These drugs are rarely effective against microbial organisms other than *Chlamydia* or *Mycoplasma* spp.

Bacteria

Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, *Pasteurella multocida*, *Yersinia pseudotuberculosis* and *Salmonella* spp. are gram-negative bacteria frequently isolated from birds with respiratory tract infections (Color 22.1).[55,63] Gram-negative bacterial infections cause a mucopurulent or thick serous drainage in comparison to the rhinorrhea (clear nasal discharge) associated with uncomplicated *C. psittaci* infections (see Chapter 33).

Serous nasal discharges may result from foreign bodies, allergies, uncomplicated viral, bacterial, fungal or chlamydial infections and with developmental defects or injuries that block the normal drainage of the sinuses into the oral cavity. With most infectious agents, the discharge will turn rapidly from serous to mucopurulent.

Pathogenic gram-positive bacteria commonly associated with respiratory infections include strains of *Streptococcus* spp. and *Staphylococcus* spp.[30] *Mycobacterium tuberculosis* was recovered from the nasal cavity and infraorbital sinuses of a Red-lored Amazon Parrot (Color 22.10).[5]

Under most circumstances, *Streptococcus* spp. and *Staphylococcus* spp. would be considered normal bacterial flora. However, pure isolates of *Staphylococcus* spp. and *Streptococcus* spp. have been associated with respiratory and intestinal tract infections.[30] Common nonpathogenic bacteria isolated from the respiratory tract of psittacines include *Bacillus* spp., *Corynebacterium* spp. and *Lactobacillus* spp. (see Chapter 33).[3,65] Upper respiratory infections caused by spirochetes have been seen in cockatiels. This organism can be demonstrated on wet mount smears (see Chapter 10).

Mycotic Organisms

Systemic avian mycotic infections can be difficult to treat. *Aspergillus* spp. are ubiquitous fungal organisms and are common pathogens in the respiratory system of immune-incompetent birds.[42,55] African Grey Parrots, cockatoos, Amazon parrots, raptors, penguins, turkeys, swans and other waterfowl (see Chapter 37) seem to be more susceptible than most psittacine species to this mycotic infection (see Chapter 35). Birds with a history of stress, unsanitary conditions or malnutrition and birds affected by oil spills or other toxins are most susceptible.[61,65] Infections may be acute, chronic or associated with mycotic tracheitis.[29,33] Mycotic granulomas may be found in the nasal cavity, oropharynx, glottal opening, tracheal bifurcation (syrinx), lungs or air sacs.[42,55,62,65]

Clinical signs with acute mycotic tracheitis include dyspnea (mild to severe) and a white discharge originating from the glottis (Color 22.16).[41,55] Fungal hyphae may be seen cytologically in specimens taken from the choana or trachea. Tail-bobbing and peracute severe respiratory distress are common with chronic lower respiratory tract involvement if the passages from the air sacs to the lungs is occluded.[65] Secondary infections may occur on organs in contact with infected air sacs, which might include the liver, kidneys, intestinal serosa and gonads (see Chapter 35). *Candida* spp. infections originating in the oral pharyngeal cavity may extend into the proximal trachea and infraorbital sinuses resulting in varying degrees of dyspnea.[14,39,62] Infected birds may temporarily respond to antibiotics (alleviation of secondary bacteria) but fail to recover. In these cases, samples from the affected area should be evaluated by cytology and culture for the presence of fungal pathogens.

Although not common in psittacine birds, a few cases of respiratory cryptococcosis have been described.[15,23,32,69] Affected birds were depressed with severe dyspnea and were unresponsive to treatment. Necropsy findings indicated gelatinous myxomatous material in the nasal cavity, infraorbital sinus and air sacs.[61]

Rhinosporidiosis is most frequently associated with waterfowl from estuary habitats.[11,17] These and other less common saprophytic fungi have been associated with rhinitis and sinusitis.[11]

Trichosporon beigelli (trichosporonosis), *Absidia* sp. (mucormycosis) and *Nocardia asteroides* (nocardiosis) have been isolated from the lungs and air sacs of birds with respiratory signs.[9,19,47,70]

Parasites

Disseminated cases of trichomoniasis may involve the upper respiratory system, trachea and air sacs causing dyspnea and respiratory distress.[58] In the oropharynx and ventral choanal surface, lesions may appear as white or yellow caseous nodules or ulcers[61] (see Chapter 36).

The tracheal mite, *Sternostoma tracheacolum* may cause severe respiratory signs in finches and canaries. Symptoms include vocalization changes, a characteristic clicking during respiration, tail-bobbing and dyspnea.[50] Severe cases lead to weakness and death. The mite may be present in any location of the

respiratory system. Transtracheal illumination may be helpful in diagnosing infections. The identification of eggs in mucus from the trachea is diagnostic (see Chapter 36).

Gapeworms (*Syngamus trachea)* inhabit the trachea and glottis area of an infected bird. Clinical signs include dyspnea and changes in vocalization. Visualization of large, bright-red helminths that are in a Y-configuration in the glottal opening are indicative of infection.[2] The earthworm is the primary vector for *Syngamus trachea*, and infections occur following ingestion of the worm. This parasite most often infects ground-dwelling species, usually in zoo and aviary situations.

Sarcocystis falcatula is a coccidian parasite with an obligatory two-host life cycle.[16,21] This parasite causes an acute, fulminating, hemorrhagic, interstitial pneumonia.[16] The clinical presentation may range from respiratory distress and severe dyspnea to peracute death with no premonitory signs (Color 22.11; see Chapter 36).

Systemic microfilaria, trematodes, nematodes and cryptosporidia are other parasites that have been documented in the respiratory system of companion birds.[2,38] These parasites may be incidental findings on necropsy or may cause varying degrees of upper or lower respiratory distress.

Inhalation Toxicosis

Birds are sensitive to inhaled toxins and have historically been used as sentinel animals to detect toxic levels of poisonous gases. Clients should be educated with respect to the adverse effects that fumes from common household compounds can have on their companion birds. The clinical changes following inhalation of household fumes may include irritation of mucous membranes, conjunctivitis, rhinitis, dyspnea or peracute death (see Chapter 37).

Cigarette Smoke

Passive exposure to cigarette smoke is a common cause of primary respiratory problems in birds as well as a common complicating factor in other respiratory illnesses. Exposure to cigarette smoke can cause a mixture of clinical problems including conjunctivitis, sinusitis, air sacculitis, rhinitis and dermatitis. Diagnosis and treatment of respiratory disease in birds that are exposed to cigarette smoke are difficult, if not impossible. In many cases, complete cessation of all respiratory signs occurs from several weeks to several months after the bird is removed from an environment contaminated with cigarette smoke.

Rhinitis and Sinusitis

Rhinitis may be caused by chemical, bacterial, fungal, chlamydial or viral pathogens. Precipitating environmental factors may include cigarette smoke, excessive powder down, dust from organic debris (bedding, flooring substrate), nutritional deficiencies and inappropriate use of antibiotics, all of which may damage the mucosa of the upper respiratory tract allowing pathogens to colonize. South American Psittaciformes that are exposed to the dander of cockatoos and cockatiels may develop a severe allergic pneumonitis. Antibiotics should be used with caution in mild undiagnosed rhinitis. Prolonged or inappropriate use can predispose the patient to secondary bacterial or fungal infections.

A seasonal occurrence (primarily winter months) of serous nasal discharge, mild sneezing and erythromatous nostrils has been described in some Psittaciformes (particularly South American species) maintained in cold, dry northern environments.[5] Similar lesions are seen when the heat or air conditioning systems are first turned on, which might suggest the accumulation of debris or respiratory irritants (stale gases) in the duct system. These birds can frequently be maintained with conservative therapy by increasing humidity, as long as the discharge remains serous and no pathogens are demonstrated. Birds are susceptible to influenza-A virus and could, theoretically, be infected through exposure to diseased members of the household (see Chapter 32).

Miscellaneous Conditions

Choanal Atresia

An African Grey Parrot chick with bilateral serous nasal discharge starting at four days of age was found to have choanal atresia (Figure 22.9). Fluids introduced into the nasal cavity did not enter the mouth and a rhinogram (nasal sinus contrast study) indicated that there was no communication between the nasal passage and the choanal slit (see Figure 12.42). Endoscopy of the choanal slit and surrounding structures revealed an intact membrane covering the choana at the level of the palate. All other oral structures were normal. Similarly, an Umbrella Cockatoo with a three-year history of intermittent serous to mucopurulent oculonasal discharge was found to have a deformed hard palate with no choanal slit. The roof of the mouth was flat and bony,

FIG 22.9 An African Grey Parrot was presented with a life-long history of serous to mucopurulent nasal discharge that was unresponsive to antibiotics. Fluid introduced to the nasal cavity would not pass into the oral cavity, and endoscopy and rhinography were used to document choanal atresia (courtesy of Cheryl Greenacre).

with papillae scattered randomly. Sterile saline introduced into the nostrils would not pass from the nares to the oral cavity. Contrast media placed in the nares stopped abruptly at the level of a thickened palate, confirming the diagnosis of choanal atresia (see Figure 12.43).[31]

Proliferative Nasal Granulomas (Rhinoliths)

Proliferative nasal granulomas have been documented in numerous psittacine species, but are particularly common in African Grey Parrots.[18] Pathogenic organisms isolated from these granulomatous growths included *E. coli*, *Enterobacter* spp., *Pseudomonas* spp. *Klebsiella* spp., *Aspergillus* spp. and *Candida* spp. Malnutrition and poor air quality play principal roles in initiating this lesion.

Upper respiratory disease, wheezing, sneezing and insufflation of the infraorbital air sacs on expiration can be early clinical changes associated with the accumulation of debris in the nares. Subtle lesions can best be detected by examining the area around the operculum using magnification. It is best to remove accumulating necrotic debris by probing and flushing before it accumulates and alters the architecture of the nares or sinus passages (see Chapter 41).[14] Recurrence is common unless dietary and management changes are made in conjunction with aggressive parenteral, topical and nebulization therapy.

Advanced lesions require removal of granulomatous tissue, frequently resulting in a large tissue defect (atrophic rhinitis; Figure 22.10). Given the vascularity of the affected area, manipulation of the affected tissue must be augmented with magnification.

Sunken Eye Sinusitis

A syndrome characterized by periorbital depression (sunken sinus syndrome) has been described as a sequela to sinusitis in macaws, conures and emus. Progressive collapse of the epithelium into the infraorbital sinus around the eye is typical (Color 22.1). Gram-negative organisms have been isolated from the infraorbital sinuses and choana of affected birds. The pathogenesis of this lesion is unclear. Ocular pathology or radiologic changes consistent with bone involvement are uncommon.

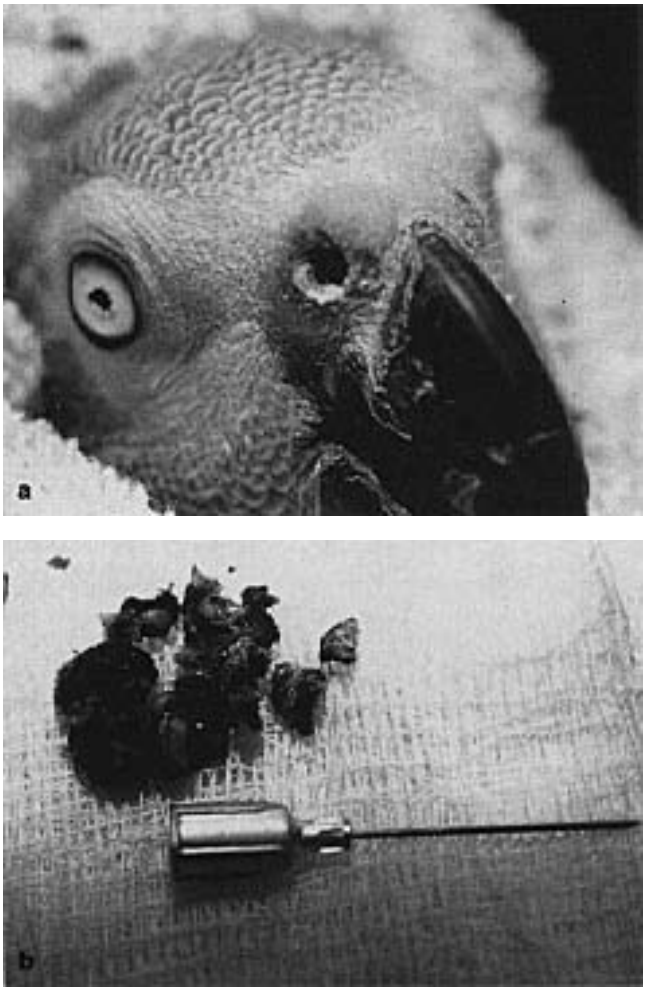


FIG 22.10 **a)** Proliferative nasal granuloma and advanced atrophic rhinitis in an African Grey Parrot. Note the enlargement of the nostril, absence of the operculum and swelling of the perinasal tissues. **b)** Necrotic material that was removed from the nostril of this bird.

Upper Respiratory Disease

Clinical Presentation

Dyspnea, rhinorrhea, purulent nasal discharge, periocular swelling, voice change, open-mouthed breathing, coughing, sneezing

Diagnostic Techniques

History
External examination of nares, choana, pharynx, larynx and trachea for hyperkeratosis, mites, granulomas
Palpation of neck and thoracic inlet, transillumination of trachea
Choanal Gram's stain and culture
Transtracheal wash, suction, cytology
Sinus flush and culture
Radiographs
Endoscopy

Rhinitis

1. Bacteria
2. Fungi
3. Reovirus
4. Parasites
5. Malnutrition
6. Chlamydia
7. Toxins

Sinusitis

1. Bacteria
2. Fungi
3. Hypovitaminosis A
4. Papilloma
5. Chlamydia
6. Mycoplasma
7. Toxins

Tracheitis

1. Amazon Tracheitis Virus
2. Avian Pox
   a. *Agapornis* Pox
   b. Psittacine Pox
   c. Amazon Pox
   d. Budgerigar Pox
3. Parasites
4. Malnutrition
5. Chlamydia
6. Toxins

Laryngitis

1. Herpesvirus
2. Poxviruses
3. Haemophilus-like organisms
4. Hypovitaminosis A

Lower Respiratory Disease

Clinical Presentation

Coughing, dyspnea, open-mouthed breathing, tail-bobbing, low exercise tolerance, depression

Diagnostic Techniques

History
Auscultation of lungs and air sacs
Radiography
Laparoscopy
Culture, flush or biopsy
Visualize lungs and air sacs

Nonrespiratory Diseases with Respiratory Signs

1. Ascites: liver disease, renal disease, neoplasia
2. Hemocoelom: trauma, vitamin K deficiency
3. Malnutrition
4. Obesity
5. Goiter
6. Cardiomyopathy: heart failure
7. Paramyxovirus, herpesvirus, reovirus
8. Hemochromatosis
9. Egg-related peritonitis

Foreign Body Inhalation

Parasites

Air Sacculitis

1. Bacteria
2. Fungi
3. Canary pox
4. Paramyxovirus
5. Chlamydia
6. Mycoplasma

Pneumonia

1. Bacteria
2. Fungi
3. Virus
4. Mycobacterium
5. Parasite (Sarcocystis)
6. Toxin

Respiratory Abscess

1. Bacteria
2. Fungi

Allergy

FIG 22.11 Differential diagnoses of upper and lower respiratory disease.

It has been suggested that this lesion may occur because a vacuum develops within the infraorbital sinus due to blockage of small diverticuli secondary to the host's inflammatory response to infectious agents. Once an infection is resolved and the sinus pathways are patent, the collapsed sinus should return to normal. Within a flock of one hundred six-month-old emus, two birds developed this syndrome, suggesting a low prevalence of the problem in a given population (see Chapter 48).

Foreign Body Inhalation

The inhalation of foreign bodies (seeds, granulomatous plaques, splinters and toys) occasionally occurs in companion birds.[54] The acute onset of mild to severe dyspnea in an otherwise healthy bird is a suggestive finding (Color 22.8). A thorough endoscopically assisted examination of the nares, choana, glottis, trachea and syrinx is helpful in the diagnosis of foreign body inhalation. Tumors, granulomas, abscesses and papillomas (glottis and choana) may cause varying degrees of dyspnea.[18]

The methods chosen to remove a foreign body will depend on the size of the patient. In birds weighing over 300 g, an endoscope can be used to suction or guide grasping forceps in the removal of some foreign bodies. Once the foreign body is localized, a 30 ga needle can be passed through the trachea distal to the mass to prevent it from moving further down the trachea. Some foreign bodies that cannot be removed by grasping may be flushed out of the trachea by holding the bird upside down and infusing fluids through a small tube placed in the trachea or through a transtracheal needle passed caudal to the mass. In some smaller birds, the distance to the syrinx can be estimated and marked on an appropriate-sized tube. The tube is then passed blindly to this predetermined level and suction is applied to remove accumulated debris.[48] If all other methods of removal fail, a tracheotomy is necessary (see Chapter 41).

Proliferative Tracheitis

Dyspnea, rales, pseudomembranous tracheitis, conjunctivitis and sinusitis have been described as clinical signs associated with proliferative tracheitis in psittacine birds. A herpesvirus with group-serologic relations to the infectious laryngotracheitis virus (ILT) has been shown to cause this lesion in *Amazona* spp (Color 22.7).[28] Swabs of the glottis and proximal trachea for cytology culture and viral isolation are necessary for diagnosis. Antiviral therapy utilizing acyclovir may be helpful along with supportive therapy and antibiotics.[65] This disease is rarely reported in the USA, and has been described only in smuggled or recently imported Amazon parrots (see Chapter 32).

Air Sacculitis

Bacterial and fungal organisms are commonly associated with acute and chronic air sac infections.[62] The air sacs are poorly vascularized and have no clearance mechanism (mucociliary blanket), which complicates the treatment of air sacculitis. Air sac infections are best treated aggressively with therapeutic agents that are chosen based on culture and sensitivity. Surgical debridement may be necessary to resolve air sac infections that result in the formation of masses.

Subcutaneous Emphysema

Subcutaneous emphysema can occur following damage to any air sac system but is most common with damage to the cervicocephalic, abdominal or caudal thoracic air sacs (Figure 22.12). Trauma, malnutrition and infectious agents have been implicated as causes of subcutaneous emphysema (see Chapter 41). In addition, the cervicocephalic air sac may distend as a result of rhinitis, which causes occlusion of the nasal passage or damage to the outflow tracts. The resulting lesion looks clinically like subcutaneous emphysema as the air sacs progressively inflate with each successive expiration.

When the air is removed with a needle, the sac will deflate but will typically reinflate with subsequent respiratory cycles. Initially, these problems can be managed by wrapping the area with a loose, self-adherent bandage. If the problem persists, long-term management can be achieved by inserting a Teflon stent in the dorsal wall of the air sac that allows air to escape. In some cases, the damage to the sac will repair itself and the stent can be removed. In other cases, the stent must remain in place permanently (see Chapter 41).

Product Mentioned in the Text

a. Devilbiss Health Care Inc, Somerset, PA

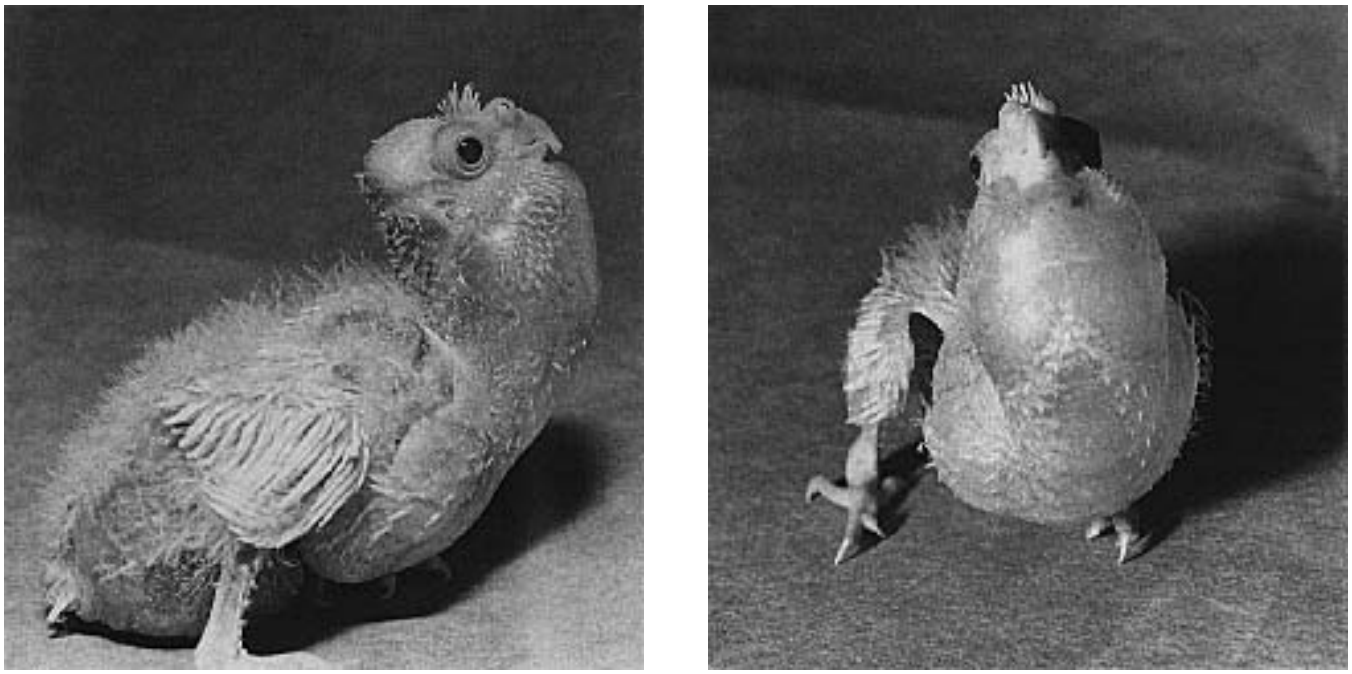


FIG 22.12 Hyperinflation of the cervical air sacs in a cockatiel. The etiology was not determined. The bird was successfully managed by making an incision into the air sacs allowing them to deflate. The rents were kept open for ten days and the problem resolved.

References and Suggested Reading

1. AAV Practice Survey. AAV Today 1(3):98-99, 1987.
2. **Barnes HJ:** Parasitology. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 478-479.
3. **Bangert RL, Cho BR, Widders PR, et al:** A survey of aerobic bacteria and fungi in the feces of healthy Psittacine birds. Avian Dis 32:46, 1988.
4. **Banks WJ:** Histology and Comparative Organology: A Text Atlas. Baltimore, Williams and Wilkins Company, 1974.
5. **Bauck L, Hillyer E, Hoefer H:** Rhinitis: Case reports. Proc Assoc Avian Vet, 1992, pp 134-139.
6. **Baumel JJ:** Functional morphology of the tail apparatus of the pigeon. Adv Anat Embryol Cell Biol 110:1-115, 1988.
7. **Bignon F:** Contribution to the study of pneumacity of birds: The cervicocephalic air cells of the birds and their relation with the bones of the head. Mem Soc Zool Franc 2:260-318, 1889.
8. **Brown R:** Sinus articular and subcutaneous *Mycobacterium faberculosis* infection in a juvenile red-lored Amazon parrot. Proc Assoc Avian Vet, 1990, pp 305-308.
9. **Burn EW, Huchzermeyer FW, Made VD:** Mucormycosis in a parrot. Mod Vet Pract 63(12):961-962, 1982.
10. **Butler JP, Banzell RB, Fredberg JJ:** Inspiratory valving in avian bronchi: Aerodynamic considerations. Resp Physiol 72:241-256, 1988.
11. **Campbell T:** Mycotic diseases. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 464-470.
12. **Campbell T:** EDTA-Tris buffer lavage for treating psittacine birds with *Pseudomonas aeruginosa* infections: A Case Report. Comp Cont Ed 7:598-604, 1985.
13. **Campbell T:** Cytology. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery, WB Saunders Co, Philadelphia, 1986, pp 250-251.
14. **Chute HL:** Fungal infections. *In* Hofstad MS, et al (eds): Diseases of Poultry. Ames, Iowa State University Press, 1972, pp 458-463.
15. **Clipsham RC, Brill TO:** Disseminated cytoccocosis in a macaw. J Am Vet Med Assoc 171(9):989-990, 1977.
16. **Clubb SL, Frenkel JK, Gardiner CH, Graham DL:** An acute fatal illness in old world psittacine birds associated with *Sarcocystis falcatula* of opossums. Proc Assoc Avian Vet, 1986, pp 139-150.
17. **Courtney CH, Forrester DJ, White FH:** Rhinosporidiosis in a wood duck. J Am Vet Med Assoc 171(9):989-990, 1977.
18. **Crane SW, Jacobson E, Shields RP:** Cryosurgical removal of an intranasal granuloma from an African grey parrot. Vet Med Sm Anim Clin 75(3):499-501, 1980.
19. **Dawson CO, Wheeldon EB, McNeil PE:** Air sac and renal mucormycosis in an African grey parrot (*Psittacus erithacus*). Avian Dis 20(3):593-600, 1976.
20. **Drewes LA, Flammer K:** Clinical microbiology. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders, 1986, p 158.
21. **Dubey JP, Speer CA, Fayer P:** Sarcocystosis of animals and man. Boca Raton, CRC Press, 1989.
22. **Emanouelson S:** Avian trichomoniasis. *In* Kirk RW (ed): Current Veterinary Therapy VIII, Philadelphia, WB Saunders Co, 1983, pp 619-621.
23. **Ensley PK, Anderson MP, Fletcher KC:** Cryptococcosis in a male Becarri's crowned pigeon. J Am Vet Med Assoc 175(9):992-994, 1979.
24. **Fillipich L:** Examining your birds and giving first aid. *In* MacWhirter P (ed): Every Bird: A Guide to Bird Health. Melbourne, Australia, In Kata Press, 1987, pp 1-17.
25. **Flammer K:** Oropharyngeal diseases in caged birds. *In* Kirk RW (ed): Current Veterinary Therapy IX. Philadelphia, WB Saunders Co, 1983, pp 617-619.
26. **Frenkel JK:** Toxoplasmosis. *In* Kirk, RW (ed): Current Veterinary Therapy VI. Philadelphia, WB Saunders Co, pp 1318-1324.
27. **Gaskin JM:** Mycoplasmosis of caged birds. Proc 1st Intl Conf Zoo & Avian Med, Hawaii, 1987, p 57.
28. **Gaskin JM:** Psittacine viral diseases: A perspective. J Zoo Wildl Med 20(3):249, 1989.
29. **Gaskin JM, Homer BL, Eskelund KH:** Some unofficial thoughts on avian viral serositis. Proc Assoc Avian Vet, Chicago, 1991.
30. **Gerlach H:** Bacterial diseases. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 408-453.
31. **Greenacre CB, Watson E, Ritchie BW:** Choanal atresia in an African grey parrot (*Psittacus erithacus erithacus*) and an Umbrella Cockatoo (*Cacatua alba*). J Assoc Avian Vet 7(1):19-22, 1993.
32. **Grinder LA, Walch HA:** Cryptococcosis in columbiformes at the San Diego Zoo. J Wildl Dis 14:389-394, 1978.
33. **Harris DJ:** Laboratory testing in pet avian medicine. Vet Clin No Am Sm Anim Prac 21(6):1154, 1991.
34. **Harris JM:** Zoonotic diseases of birds. Vet Clin No Am Sm Anim Prac 21(6):1297, 1991.
35. **Harrison GJ, Harrison LR, Fudge AM:** Preliminary evaluation of a case. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 107-108.
36. **Harrison GJ, et al:** Symptomatic therapy and emergency medicine. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 370-371.
37. **Hillyer EV, et al:** An outbreak of sarcocystis in a collection of psittacines. J Zoo Wildl Med 22(4):434-445, 1991.
38. **Hillyer EV, Quesenberry KE, Baier KE:** Systemic microfilariasis in an umbrella cockatoo. Proc Assoc Avian Vet, 1988, pp 201-202.
39. **Humphreys PN:** Debilitating syndrome in budgerigars. Vet Rec 101:248-249, 1977.
40. **Hunter DB:** An introduction to the avian respiratory system: A diagnostic approach and common diseases. Introduction to Avian Medicine and Surgery. Assoc Avian Vet, New Orleans, 1992.
41. **Hunter DB, Taylor M:** Lung biopsy as a diagnostic technique in avian medicine. Proc Assoc Avian Vet, 1992, pp 207-210.
42. **Jenkins J:** Aspergillosis. Proc Assoc Avian Vet Chicago, 1991, pp 328-330.
43. **Klein PN, Galey FD:** The challenge of toxicologic investigation in birds. Proc Assoc Avian Vet, 1989, 139-142.
44. **King AS, McLelland J:** Form and Function in Birds Vol 4. San Diego, Academic Press, 1989.
45. **Kueth DO:** Fluid mechanical valving of air flow in bird lungs. J Exp Biol 136:1-2, 1988.

46. **Locke D, Bush M:** Tylosin aerosol therapy in quail and pigeons. J Zoo Anim Med 15:67-72, 1984.
47. **Long P, Choi G, Silberman M:** Nocardiosis in two Pesquet's parrots (*Psittrichas fulgidus*). Avian Dis 27(3):855-859, 1983.
48. **Lumeij JT:** A Contribution to Clinical Investigative Methods for Birds with Special Reference to the Racing Pigeon. PhD Thesis. Faculty Vet Med, Univ of Utrect, The Netherlands, 1987.
49. **Lutz M:** Use of human interferon in a case of systemic papillomatosis. Proc Assoc Avian Vet, New Orleans, 1992.
50. **McCluggage DM:** Parasitology in caged birds. Proc Assoc Avian Vet, Seattle, 1989, pp 97-100.
51. **McKibben JS, Harrison GJ:** Clinical anatomy with emphasis on the Amazon parrot. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 31-66.
52. **McKiern BC:** Principles of respiratory therapy. *In* Kirk RW (ed): Current Veterinary Therapy VIII. Philadelphia, WB Saunders Co, 1983, pp 216-221.
53. **McKiern BC:** Lower respiratory tract diseases. *In* Ettinger SJ (ed): Textbook of Veterinary Internal Medicine. Philadelphia, WB Saunders Co, 1983, pp 760-828.
54. **McMillan MC:** Radiology of avian respiratory diseases. *In* Johnston DE (ed): Exotic Animal Medicine in Practice. Trenton, 1991, pp 88-98.
55. **Olsen GH:** Avian respiratory system disorders. Proc Assoc Avian Vet, Seattle, 1989, p 434.
56. **Perry R, Gill J, Cross G:** Disorders of the avian integument. Vet Clin No Am Sm Anim Prac 21(6):1307, 1991.
57. **Rae MA, Duimstra JR, Snyder SP:** Pulmonary silicosis in a blue and gold macaw (*Ara ararauna*). Proc Assoc Avian Vet, Chicago, 1991.
58. **Ramsay EC, Drew ML, Johnson B:** Trichomoniasis in a flock of budgerigars. Proc Assoc Avian Vet, 1990, pp 309-311.
59. **Rich G:** Basic history taking and the avian physical examination. Vet Clin No Am Sm Anim Pract Small 21(6):1135-1145, 1991.
60. **Ringer RK:** Selected physiology for the avian practitioner. *In* Harrison GJ, Harrison, LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 72-73.
61. **Ritchie BW:** Avian therapeutics. Proc Assoc Avian Vet, Phoenix, 1990.
62. **Rosskopf WJ, Woerpel R:** Pet avian conditions and syndromes of the most frequently presented species seen in practice. Vet Clin No Am Sm Anim Prac 21(6):1189-1209, 1991.
63. **Rosskopf WJ, Woerpel RW, Lane R, et al:** A survey of antibiotic efficacy for gram negative bacterial isolates from pet psittacine birds from May 1984 to February 1985. Proc Assoc Avian Vet, Boulder, 1985.
64. **Schaer M, Akerman N:** Diagnostic approach to the patient with respiratory disease. *In* Ettinger SJ (ed): Textbook of Veterinary Internal Medicine 2nd ed. Philadelphia, WB Saunders, 1983, pp 655-672.
65. **Spenser EL:** Common infectious diseases of psittacine birds seen in practice. Vet Clin No Am Sm Anim Pract 21(6):1223-1224, 1991.
66. **Spink RR:** Aerosol therapy. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 376-379.
67. **Spink RR:** Nebulization therapy in cage bird medicine. Vet Med Small Anim Clin 75:791-795, 1980.
68. **Steiner CV, Davis RB:** Caged Bird Medicine, Selected Topics. Ames, Iowa State University Press, 1981, p 42.
69. **Takeshita K, Fenwick B, Wong A:** Cryptococcosis in captive cockatoos. Proc Assoc Avian Vet, Miami, 1986, pp 133-138.
70. **Taylor MT:** Systemic trichosporonosis in a green-winged macaw. Proc Assoc Avian Vet, Houston, 1988.
71. **Tungerman PF, Schwartzman RM:** Veterinary Medical Mycology. Philadelphia, Lea and Febiger, 1972, pp 61-74.
72. **Walsh MT:** Clinical manifestations of cervicocephalic air sacs of psittacines. Comp Cont Ed 9:783-789, 1984.
73. **Wyrick PB, Richmond SJ:** Biology of chlamydia: Reports from the symposium on avian chlamydiosis. J Am Vet Med Assoc 195(11):1507-1511, 1989.