Introduction
The chronic feline sniffer is a frustrating patient to treat. The longer the course, the more severe the consequences to affected tissues are and the more debilitating it is to the patient. A logical diagnostic plan to differentiate probable etiologies and to rule out nonviral causes results in appropriate therapeutic choices. Even with a viral etiology, therapies to reduce the pathological consequences of the infection may modulate and help control the clinical signs. Novel choices and drug combinations are discussed.

History and Presentation
Chronic, recurrent rhinosinusitis can occur in cats of any age. Cats are presented because of sneezing, nasal discharge, and noisy breathing with or without inappetence. Sneezing occurs because of stimulation of irritant receptors in the nasal and sinus subepithelium. Knowing the timing, onset, duration, and frequency of sneezing can be helpful. With chronic, inflammatory changes this response may be abolished, resulting in accumulation of discharge. Nasal discharge may be serous, mucoid, purulent, or sanguinous. It is helpful to know whether the discharge has changed, whether it changes throughout the day or season, and especially whether it is unilateral or bilateral.

Respiratory patterns and sounds may be abnormal. Clients may comment on the cat sounding hoarse or even silent when meowing or that its purr is different. In general, sounds heard on inspiration are associated with larger airways, whereas expiratory sounds are associated with smaller, lower airways. Snorting occurs with accumulation of discharges in the nasal passages or with secretions coughed into the oropharynx (e.g., from pneumonia). A snoring, stertorous sound is associated with proximal upper respiratory occlusion, such as with a polyp or foreign body obstruction or functional inflammatory obstruction. Stridor is an inspiratory wheeze that reflects changes in the larynx. An expiratory wheeze, crackles, and rales reflect small airway involvement. A complete lack of bronchovesicular sounds occurs when there is pulmonary consolidation or inflammation.

If the breathing is “worse at night,” this could reflect bronchitis or merely the time that the client is at home to observe the cat. Sounds that are worse after exercise or at rest may reflect the severity of the respiratory interference or the movement of secretions. Some cats have seasonal flare-ups, suggesting an allergic or contact irritant component.

Assess facial symmetry both face on and from above the head. Palpate the face to further look for swelling, invagination, or discomfort. Thoroughly evaluate teeth and alveolar bone for evidence of periodontal disease, abscessation, or inflammation. Look at and palpate the hard and soft palate, where feasible, looking for oronasal fistula or mass lesions or ulceration. If a cat retches or yawns, the tonsils may be visualized. By opening the mouth we can evaluate neurologic competency: jaw tone (motor V), position, movements and symmetry of the tongue (XII), and gag reflex (IX, X).

Evaluate nasal passage patency using a small mirror (compact or dental) or a glass slide that has been kept in the freezer. Wisps of cotton are also helpful. Palpate the trachea to see if this elicits a cough. It is helpful to auscult the trachea as well as three locations (dorsal cranial and caudal and cranioventral) bilaterally to define the primary location of the lesion. Occasionally auscultation of the frontal sinuses may be revealing. For this, a small pediatric bell is used. For pulmonary auscultation, use two heads, the standard bell and a plexiglass scope (e.g., UltrascopeTM), as they provide different sensitivities and frequencies.

A thorough physical examination should be performed. Fundic examination should be performed to look for Cryptococcus and other signs of systemic disease. Enlargement of regional lymph nodes or generalized node enlargement should be assessed.

Etiologies and Pathogenesis
Chronic rhinitis may be a sequel to acute rhinitis but it may be a separate condition altogether. It may represent an ineffective immune response to persistent viral infection [1]. Feline herpesvirus 1 (FHV-1) may be the common denominator initiating turbinate resorption, with subsequent secondary bacterial infections and unchecked
inflammation exacerbating the problem. This is especially bad in anatomically predisposed individuals (conformation, anomalies). Irreversible destruction of the turbinates may result in viral or inflammatory mediator-induced cytolysis. Reactivation of herpesvirus from infected trigeminal ganglion may result in recurrent destruction. All of these are possible pathogenic strategies; it is not possible to determine the course/cause in a given individual.

Caliciviruses infection results in a carrier state with continuous shedding for variable periods of time. FHV-1, like other herpesviruses, results in a state of latency, and approximately 80% of infected cats are permanent carriers. Latency accounts for recurrence of clinical signs during periods of physiological or psychological stress.

Primary bacterial agents include Bordetella bronchiseptica, commonly found as a commensal without causing morbidity. Mycoplasma spp. may be cultured from some individuals, but their true incidence remains unknown due to the difficulty of isolating these fastidious organisms. Chlamydophyliosis is not common, and infection is limited to varying degrees of conjunctivitis. L-forms may also be involved but require specific targeted culture techniques for verification.

In one study [2] aerobic bacteria were cultured from biopsy samples from twice as many clinically affected cats (4/10) as controls (2/7), while anaerobic infection occurred in only the affected cats (2/10). Flush samples were collected from the same cats, with aerobes in 5/7 controls and 9/10 affected cats; anaerobes in 3/10 and Mycoplasma spp. in 2/10 affected cats. Interestingly, FHV-1 was not cultured from any of the cats, but viral DNA was detected in 4/7 control and 3/10 affected cats by PCR, implying that the virus was not viable.

Less frequently isolated bacteria worthy of journal publication have included Actinomyces sp., Haemophilus sp. [3], and Capnocytophaga sp. [4]. Bartonella henselae is commonly detected by serology (antibody titres), yet its true role in the chronically infected cat is not as relevant as its serologic exposure. One study [5] showed that “Serological screening for Bartonella antibodies may not be useful for the identification of bacteremic cats (positive predictive value = 46.4%), but the lack of antibodies to B. henselae was highly predictive of the absence of bacteremia (negative predictive value = 89.7%).”

The fact that cats on antibiotics often improve clinically would support the role of bacteria; the fact that signs recur, despite therapy, implies that bacteria are only part of the cause of the illness. When antimicrobial therapy of 7–10 days duration fails to result in resolution of disease, then a thorough diagnostic work-up should be recommended.

The main fungal organisms causing chronic upper respiratory disease are Cryptococcus neoformans var. neoformans and gattii. These classically cause severe inflammation resulting in facial deformity and skin ulceration along with unilateral (> bilateral) nasal discharge. Aspergillosis sp. and Penicillium spp. have also been isolated [6].

Trauma, congenital and conformational aspects, polyps, periodontal disease, and foreign bodies all predispose to chronic infection [7]. Any factors contributing to alterations in the structure or function of the upper airways, be they primary inflammation (lymphoplasmacytic rhinitis) or that secondary to the noxious effects of infection, will compromise normal function and predispose to chronic damage if the cat is unable to resolve the underlying factors. Chondritis and osteomyelitis are often sequelae to infection/inflammation. There is some suggestion that chronic rhinitis/sinusitis may predispose to nasal lymphoma in cats.

Neoplasia further alters function and form, allowing secondary changes, which may be more worrisome to the client than the underlying cancer. If there are concurrent stressors (suboptimal nutrition, social distress, environmental factors) or outright immunocompromise/suppression (e.g., retroviruses), the likelihood of infectious agent involvement and the inability to clear these is increased. Most feline nasal tumors are malignant. They tend to be locally invasive (frontal and paranasal sinuses) without metastasizing distantly. Similar to other types of cancer in cats, the older cat is overrepresented. Clinical signs will vary depending on the location of the tumor. Nasal tumors result in sneezing and unilateral nasal discharge; nasopharyngeal masses present with stertorous respiration. Further signs include variable facial deformity, epistaxis, and epiphora.

Diagnostics
When rhinitis or rhinosinusitis is a recurrent or chronic problem, a logical and thorough diagnostic plan should be followed. Start with a minimum database of a CBC, serum biochemistry, retroviral serology, urinalysis, and blood pressure determination, if not already done in the earlier examination. If rhinoscopy is considered or if epistaxis has
been part of the process, a coagulation panel should be performed. Any medications affecting hemostasis (e.g., aspirin, alpha antagonists) should be temporarily discontinued. If regionally appropriate, perform Aspergillus and Cryptococcus serologic titres. If lymph nodes are enlarged, cytologic specimens should be collected to use for staging in case neoplasia is diagnosed by histopathology.

Skull radiography or CT/MRI to image dentition, nasal passages, and sinuses as well as bone health will require general anaesthesia. Conventional radiography underestimates the extent of disease. Probe all periodontal pockets, retract the soft palate to look for polyps, and palpate the soft palate. Three standard radiographic views should be exposed using high detail films and screens. 1) An open mouth ventrodorsal view assesses the nasal cavity and bullae. Symmetry is essential for evaluation of changes. 2) A lateral view allows evaluation of the frontal sinuses; if a change is suspected, it may be followed by an oblique lateral view to focus on the sinus in question. 3) A skyline view of the frontal sinuses is valuable and is performed with the cat in dorsal recumbency, pulling the mandible out of the way.

Following imaging, samples should be harvested. Michiels et al. [8] evaluated the records of 40 cats who had undergone rhinoscopy for chronic nasal disease to compare relative diagnostic yield. Specimens in 17 cases were collected by brush cytology (higher yield than flush cytology). Concurrent biopsies were collected for histopathologic evaluation. Only 25% of the cases showed agreement. The conclusion was that cytology (even brush cytology) does not appear to be a reliable means for the detection of chronic inflammation and evaluation of chronic rhinitis in cats.

The small size of cats makes scoping challenging: a flexible endoscope may be retroflexed around the soft palate if retraction of the soft palate using a dental mirror was unrevealing. To evaluate the more rostral portions of the nasal passage, a rigid 1.9 mm arthroscope with a 30 degree viewing angle may be used if a small flexible scope is unavailable. Irrigation with sterile saline is essential for optimal visualization. Mucus exudation, a polyp or mass, foreign body, or “webbing” (nasopharyngeal stenosis) may be seen.

If unilateral disease is present, evaluation of the unaffected side first is recommended. Normal turbinate mucosa should be pale pink and smooth. Hyperemia, irregular turbinate surfaces, and moderate amounts of discharge suggest pathology. Fungal plaques may be seen and biopsied. While adenocarcinoma or sarcoma appear as a discrete mass, lymphoma may present as a mass or as a diffuse infiltrate. Even if the mucosa looks normal, biopsies should be taken in a cat with chronic disease, as gross appearance may be misleading. The entire cavity (rostral and caudal) should be examined before biopsying to avoid bleeding, which interferes with visualization. Sedation may be desirable upon recovery, and overnight hospitalization prevents excessive movement, allowing hemostasis to occur.

Concurrently, samples should be collected for culture. Aerobic and anaerobic cultures may be set up, but results must be interpreted with caution because there are large numbers of normal flora in the nasal cavity. One can improve diagnostic yield by obtaining cultures from deep within the nasal cavity, avoiding superficial contamination. Calicivirus identification requires virus isolation (VI). VI may also be attempted for FHV-1; however, exposure to FHV-1, Chlamydia, and Mycoplasma may be determined using PCR. A recent study [9] assessed the relative sensitivity of PCR assays for the detection of FHV-1 DNA in clinical samples and commercial vaccines. It concluded that none of the assays was able to distinguish between wild-type virus and vaccine virus. Additionally, test sensitivity (detection limits and rates) varies greatly between the tests used.

Before recovering the patient from anaesthesia, flush gently and thoroughly to remove and aspirate the discharge to help the patient during and after recovery. The endotracheal tube must be well cuffed, and the oropharynx should be packed with a known number of swabs to prevent fluid aspiration.

**Therapeutics: Specific**
Practitioners frequently choose antibiotics to treat the cat with upper respiratory disease. But do we know what organism is involved? If multiple organisms are grown on culture, the significance of the growth is questionable. Should a single bacterial species grow on culture that is NOT a normal commensal, sensitivity results may be used. Therapy should be continued for 6–8 weeks without changing the antibiotic if there is an initial positive response to the antibiotic, so the antibiotic should be safe for long-term use. Antibiotics should be chosen that reach the site of infection at effective therapeutic concentrations. Antibiotics that penetrate cartilage and bone are of value, making
amoxicillin-clavulanic acid, clindamycin, and chloramphenicol reasonable choices. Clindamycin, doxycycline, and chloramphenicol are effective against Mycoplasma spp.; metronidazole and doxycycline modulate the immune response, thereby reducing inflammation somewhat. Doxycycline is effective against Chlamydia phila and L-forms. Azithromycin (5-10 mg/kg PO q24h for 5 days, then q72h long term) is popular because of its long duration of action. Pulse or intermittent therapy (e.g., 1 week/month) predisposes to the development of antibiotic resistance and cannot be recommended. Administration of antibiotic ophthalmic drops may be included because they can be used as direct topical therapy to the nasal passage.

Should Cryptococcus sp. or Aspergillus sp. be cultured, specific antifungal protocols should be followed (discussed elsewhere). If an allergic component is suspected because of seasonal recurrence, antihistamines may be considered. Chlorpheniramine maleate 1–2 mg/cat PO q12h may be used. Less sedative antihistamines (e.g., Allegra™, Claritin™) selectively inhibit peripheral H1 receptors.

For FHV-1 infection, administration of the intranasal herpes and calicivirus vaccine 2 to 3 times a year may be beneficial in stimulating local immunity. L-lysine helps to reduce the frequency of herpesviral recrudescence by competing with arginine needed for viral replication. The dose is 250 (kittens)–500 (adults) mg PO q12h long term. Interferon alpha at 30 units PO q24h may also help modulate FHV-1 infection. Similarly, ophthalmic administration of alpha interferon in saline has been recommended for cats with herpes virus keratitis or conjunctivitis. Acyclovir is an anti-herpes drug used in humans. Because of potential toxicity in cats, it should only be used in cats with confirmed herpes infection and should be started at a low dose (10–25 mg/kg PO q12h), monitoring the CBC every 2–3 weeks.

Polyps and foreign bodies should be removed. A novel approach to the removal of a polyp originating in a frontal sinus was recently reported [10]. Because of the small size of the patient, an endoscope was passed orad through the cardia of the stomach, into the esophagus and oropharynx, allowing retrieval of several polypoid masses. Nasopharyngeal stenosis/“webbing” requires surgical resection via a transpalatine approach. Like polyps, webs may reoccur. Dental disease should be treated, repairing fistulae if present.

Surgical drainage and flushing may be warranted for some patients with chronic sinusitis. After openings are drilled into the frontal sinus, histopathologic samples and bacterial samples may be collected. Trypsin-containing solutions may help break up heavy mucus. Sinus ablation has also been described in which the frontal sinus is opened by bone flap, the mucoperiosteal lining is removed, necrotic nasal turbinates are removed, the opening between the sinus and nasal passages is obliterated with a piece of temporal muscle fascia, and the frontal sinus is packed with a piece of ventral abdominal fat. This technique has shown success, and fat is preferable to the use of polymethylmethacrylate.

**Therapeutics: Nonspecific**

Maintaining hydration is essential for tissue perfusion, but also to make secretions less viscous and to improve cell function (e.g., their ability to clear mucus via the muco-ciliary apparatus). Thus, humidifying the air around patients with chronic airway narrowing is beneficial, be it by steaming the bathroom or instilling saline into the nostrils to stimulate sneezing and clearance of the nasal passages. Oral decongestants include diphenhydramine HCl 2–4mg/kg PO q8h, dimenhydrinate 4mg/cat PO q8h, or pseudoephedrine 1 mg/kg PO q8h. Nasal decongestant drops are challenging to administer but can be very helpful (pediatric Otrivin™ = 0.05% xylometazoline 1 drop into each nostril SID for 3 days only to avoid rebound congestion).

Anti-inflammatories play a role. By reducing airway swelling, breathing improves and less secretion is produced, making the patient more comfortable. Glucocorticoids may help by retarding leukocyte function and migration, blocking phospholipase A, decreasing release of lytic enzymes, and suppressing delayed hypersensitivity reactions. This makes them candidates for use in lymphoplasmacytic rhinitis, the most common form of chronic rhinitis. Because the condition itself is not life-threatening, glucocorticoids should be used intermittently rather than continuously long-term. The author uses prednisolone daily for a week, and reduces to q48h over the next week. The concern with the use of glucocorticoids is the possibility that they might result in recrudescence of the virus or virus shedding. Non-steroidal anti-inflammatories are alternate options. Piroxicam (0.3 mg/kg PO q48h) may help. Leukotriene blockers may also be considered to reduce inflammatory cell infiltration. Singulair™: 0.25mg-0.5mg/kg q24h (= 1/8th of a 10 mg tab); Accolate™: 0.5mg-1mg/kg q12-24h.
It is critical to pay attention to nutrition in quality, balance, and quantity. In addition to the frequently used antihistamine, anti-serotonin drug cyproheptadine (1 mg PO q12h), mirtazapine at 3–4 mg/cat PO q72h is a newly recognized appetite stimulant for cats.

Acupuncture may be a useful adjunctive therapy.

**Prognosis**
It is important that clients understand that a cat with chronic rhinitis/rhinosinusitis will never be cured. With ongoing management, the patient’s quality of life can be improved, with a reduction in sneezing and nasal discharge.

*References available upon request.*