HYPERPLASTIC OTITIS

Medical and Surgical Q & A

Nicole A. Heinrich DVM, DACVD and Lara M. Rasmussen DVM, MS, DACVS
McKeever Dermatology Clinics and Direct Veterinary Surgery
www.mckeevervetderm.com and www.directvetsurg.com

What is hyperplastic otitis (aka cocker spaniel ears, aka bulldog ears)? --NAHeinrich
Hyperplastic otitis refers to the proliferation/thickening of ear canal epithelium. The hyperplasia causes a diffuse, nodular texture to the ear canal lining and also causes the lumen of the ear canal to become partially or even completely occluded.

What causes hyperplasia to develop? --NAHeinrich
Hyperplasia is a result of chronic inflammation, most often due to uncontrolled allergies. The allergy causes inflammation in the ear canal epithelium. The inflammation causes cerumen glands within the epithelium to increase secretion. The inflamed, waxy environment leads to a bacterial or yeast infection. If the infection goes unnoticed or recurs regularly, the epithelium of the ear canal starts to thicken. The ear's response to being constantly under attack by the infection is to create a bigger, better, thicker shield. However, this response backfires on the ear because too much hyperplasia causes the ear canal lumen to become occluded. Some breeds of dog, such as bulldogs and cocker spaniels, are more likely to have a hyperplastic response to inflammation than other breeds. These breeds may form hyperplasia more quickly than others.

What if hyperplastic otitis persists untreated? --NAHeinrich
The longer hyperplastic otitis persists, the more refractory it becomes to treatment. The end result of chronic hyperplasia is calcification of the ear canal. Once the ear canal becomes calcified, medical management is almost completely futile.

How does this condition progress through the ear anatomy? --LMRasmussen
Early stages of hyperplastic otitis are limited to the soft tissues of the external canal. Structural weakness of the canal associated with chronic inflammation and loss of flexibility may result in ruptures of the vertical canal wall and outpouching of debris or overt abscessation.

The chronic inflammation and pathology of the soft tissues of the external canal progresses to dystrophic mineralization (calcification). This loss of canal flexibility may progress to canal separation from the external acoustic meatus (opening to bulla) and deep abscessation.

Progressive canal mineralization and fibrosis will entrap surrounding anatomy, the most pertinent being the facial nerve as it exits the skull and crosses laterally around the vertical canal.

With time, stenosis and occlusion result in debris accumulation and inflammatory changes sufficient to traverse the tympanum into the middle ear. Marked middle ear inflammation or pressure will cause injury to the delicate inner ear mechanisms, often manifesting in cranial nerve signs.

Chronic middle ear inflammation/infection may also erode the bone of the bulla, and in advanced caseous abscessation of the bulla, erosion thru the cranium is possible. *(Ask me sometime about the adrenal-squeeze I enjoyed during one of these advanced cases!!!)*

What does the dog experience as the disease progresses? --LMRasmussen
This is always a difficult question, and perhaps my best answer is an extrapolation from *what changes after surgical recovery*.

- Dogs are brighter, more interactive and more energetic. (Interpretation: they were experiencing chronic pain, headache, TMJ pain, facial neuritis, anemia of chronic disease, malaise from chronic inflammatory state.)
- Chronic keratitis sicca and conjunctivitis, so often seen in Cockers, becomes easier to manage. (Interpretation: their eyes were experiencing very high local bacterial/fungal populations; their immune system was taxed under a chronic inflammatory state.)
- Generalized dermatopathies become more manageable. (Interpretation: same as above.)
- Similar (to preoperative) hearing acuity, though reduced relative to a "normal" ear; still able to respond to owner's voice and environmental cues. (Interpretation: the hearing preoperatively was reduced by the loss of a "sound airway" down the occluded external canal, to the tympanic rupture, and into middle ear debris/granulation. All preoperative and postoperative hearing accomplished via bone conduction.)

How can we treat hyperplastic otitis? --NAHeinrich
The most effective way to treat hyperplastic otitis is to prevent it from occurring. Dogs that present with recurrent otitis must have their allergies addressed. The possibility of a food allergy must be investigated with a diet trial. Environmental allergies can be addressed with allergy testing and hyposensitization or use of medications such as Atopica or Apoquel.

Topical therapy is an extremely important component of addressing allergic otitis. The normal ear is able to clean itself via epithelial cell turnover. The inflamed, hyperplastic ear is unable to clean itself, which results in the accumulation of waxy debris within the canal. The ears should be cleaned weekly with products such as TrizUltra+keto (if prone to yeast otitis) EpiOtic Advanced (if prone to bacterial otitis) or Douxo Micellar (if ear is sensitive to other cleaners). Ears may need to be cleaned as often as daily when active infection is present

Can hyperplasia be reversed? --NAHeinrich
Oftentimes, the veterinarian is presented with a patient with otitis that has progressed beyond the simple inflammatory phase and is fully in the chronically inflamed, hyperplastic phase.

There are 3 phases of treatment for the hyperplastic ear. They are as follows:

First step: Reduce hyperplasia to open the canal lumen.
Multimodal therapy is often required to reduce hyperplasia. It is most successful if the underlying allergy can be addressed. Oral steroids (1mg/kg PO q 24 hr x 1-2 weeks, then 1mg/kg PO q 48 hr x 2 weeks) combined with topical Synotic® applied to the hyperplastic tissue 2-3 times per day x 1 month is effective for most cases to reduce the hyperplasia. Atopica® or Apoquel® can be used to prevent the hyperplasia from recurring. Sometimes Atopica® or Apoquel® can be used in place of oral steroids to reduce hyperplasia; however, neither is as reliable as steroids. Apoquel® seems to be less effective than Atopica® in ability to address hyperplasia, but is case dependent.

It often takes 4 weeks to reduce hyperplasia by 50%. Hyperplasia can be reduced further over the course of months if the underlying allergy can be controlled very well and if topical therapy is diligently administered.

If the hyperplasia is unable to be reduced to open the canal lumen by 50% or if the hyperplasia returns quickly upon reducing steroids, total ear canal ablation (TECA) should be strongly considered.

If there are patient factors (unable to receive steroids, impossible to medicate, sensitive stomach, etc.) that make it difficult to use medication to reduce the hyperplasia, then TECA should be strongly considered.

Second step: Resolve infection.
Infection can be resolved once the canal lumen is open. Topical steroids and antibiotics should be used, and oral antibiotics should be used in cases where otitis media is suspected. It may take 6 to 8 weeks to clear the infection after the hyperplasia has been reduced. An ear culture is often very helpful in the selection of antibiotic therapy. Ear cytology repeated throughout the course of treating the infection is essential for determining when the infection has been resolved.

If infection cannot be resolved, then TECA should be strongly considered.

Third step: Prevent recurrence.
Control of the underlying allergy with diet, medication and/or allergen specific immunotherapy combined with topical therapy will successfully prevent recurrence of hyperplastic otitis in many cases. Topical therapy includes cleaning the ear canals at least weekly. Additionally, administration of topical steroid in the ear (typically 3 times per week) is very helpful for preventing inflammation and subsequent hyperplasia formation.

If recurrence cannot be prevented, then TECA should be strongly considered.

Many cases of hyperplastic otitis respond well to medical management. Some cases are refractory to therapy despite best efforts.

If a therapy fails at **any** of these steps, then TECA should be strongly considered.

Who is the best candidate for medical management of hyperplastic otitis? --NAHeinrich
The best candidates for medical management of hyperplastic otitis are those who are *early in the disease process*. Dogs who are showing a propensity towards recurrent otitis (3 ear infections per year), chronic otitis (an ear infection that requires more than 4 weeks to resolve) and early tendency to form hyperplasia (narrowing of canal lumen, cobblestone architecture to canal epithelium) are superb candidates for medical management. These dogs often respond extremely well and are able to have healthy, pain-free, functional ears. Once dogs show signs that they are predisposed to recurrent, chronic and/or hyperplastic otitis, they need to have their underlying allergy addressed. Finding the best way to address the underlying allergy may take a few tries as not every dog responds in the same way to hydrolyzed protein foods, allergen specific immunotherapy or allergy medications. Dogs with recurrent, chronic and/or hyperplastic otitis often need topical therapy in addition to their allergy therapy.

What patient/owner parameters suggest TECA as a good therapeutic option? --LMRasmussen
Mineralized canal
The first thing I do when presented with a candidate for a TECA is place my fingers gently around the base of their ears. If I encounter stone, my mind is made up. Dystrophic mineralization of the external canals indicates long standing, intractable changes to the tissues.

Occluded hyperplastic canal that has failed medical management
If a canal will not open back up with appropriate medical management, the spiral to end-stage disease is inevitable. Surgical treatment early in this spiral is logical.

Intractable bacterial otitis
In today's world of concerns for antibiotic resistance, the superbugs that grow in chronic hyperplastic otitis cases are standouts! If appropriate medical management has failed to create a canal free from the need for "stronger" antibiotics with each passing season, then surgical removal of that source of infection is warranted (before those superbugs increase the postoperative risk!)

Recurrent hyperplastic otitis
TECA is indeed a salvage procedure, but one with an incredibly generous risk:benefit ratio. I'll take it over an FHO any day! If recurrence of canal occlusion develops into a habit, surgical removal of the offender earlier (than end-stage) may offer a better long-term outcome.

Owner treatment fatigue
Ongoing maintenance costs perhaps come to mind first in this regard. But I hear more often that owners do not like "hurting" their dog with ear treatments every day. I can empathize

with that feeling. Dogs will become head-shy, avoid their owners at treatment time, and at times react dramatically by trying to bite their owners.

Painful, subdued, irritable patient
The subtle changes that occur over time with advanced hyperplastic otitis are difficult to appreciate in real-time. Retrospectively, they are obvious. But as they develop, most owners rationalize the changes as "getting older". The nice dog turned grumpy, and the perky dog turned distant are not easily attributed to an ear problem. These changes, if indeed related, can be reversed.

The TECA procedure, in brief. --LMRasmussen
A technically challenging procedure with such positive outcomes to put it in the top three of my favorite procedures. An incision is made around the opening of the external canal to include all active hyperplastic epithelium (occasionally, this includes much of the pinna, and a planned pinnectomy is a logical addition to the procedure.) Dissection of the external canal continues proximally toward the skull, using caution to isolate and preserve the facial nerve as it courses around the lateral aspect of the vertical canal. Abscesses are preserved intact when possible and resected with the canal *en bloc*. The horizontal canal is transected at the external acoustic meatus, and the entire canal removed. The external acoustic meatus is debrided, and the lateral bulla walls removed. Approximately 50% of the bulla surface area is removed to allow for thorough debridement of debris, granulation and the epithelial lining of the bulla, as well as promote access for cells, vasculature and antibiotics necessary for clearing deep infection. The bulla and surgical wound are lavaged and evacuated. A sample of the "clean" wound/bulla is taken for culture and sensitivity. The surgical wound is closed primarily (with or without—more commonly—a drain). A head bandage/stockinette hood is maintained for 2wks to protect the incision and restrain the pinna from flapping which perpetuates head shaking and discomfort.

What complications are associated with TECA? --LMRasmussen
There are three main complications:

1) *Persist surgical wound infection*. This is a risk typically in end-stage cases on chronic steroids and with markedly resistant bacteria at the time of surgery. An incision that is not dry within 3-5days is the first indication.
2) *Transient or permanent facial palsy*, manifesting primarily as a weak/absent ability to blink. Topical lubricant is the treatment of choice. If the blink has not returned in 3wks, it is unlikely to return and chronic use of eye lubrication is often necessary. Transient palsy is seen in most patient (likely secondary to bupivacaine local blockade and surgical traction). Permanent palsy is seen in approximately 10% of cases, in my experience.
3) *Chronic, deep sterile/infected abscess formation*. The source is presumed to be remnant epithelium lining the bulla (+/- bacteria), that sloughs and generates debris overtime. Much of this likely occurs and evacuates via the Eustachian tubes; if in sufficient volume, an inflammatory reaction may develop and "head up" near the skin surface. Treatment as a simple abscess (stab/lavage/drain/culture/antibiotics) is usually curative; rare cases will maintain a tiny draining tract with minimal exudate longterm. This eventuality is quite rare in my experience, developing in less than 5% of cases.

4) *Devitalized pinna* (secondary to loss of blood supply during dissection/resection) has been reported, but not in my experience.

What does the owner experience postoperatively? --LMRasmussen
After 1-2wks of recovery, the vast majority of owners report high levels of satisfaction with their choice.

- Universally, owners report a happier, more interactive pet; often quoted as saying "she is like a puppy again!"
- Life with their pet is more pleasurable because the foul odors are gone, and the daily ear treatment "battles" are ended.
- Concerns regarding hearing "loss" do not seem to be recognized or commented upon postoperatively.

Hyperplastic otitis is a challenge—for the dog, the owner, the veterinarian, the dermatologist and the surgeon. Please feel free to call us to discuss options and plans as you navigate this challenging disease with your clients. –NAHeinrich and LMRasmussen