PROCEEDINGS

ASSOCIATION OF AVIAN VETERINARIANS (AAV)
PRE-CONFERENCE PROGRAM: BEHAVIOR

AND

ASSOCIATION OF EXOTIC MAMMAL
VETERINARIANS (AEMV) SCIENTIFIC PROGRAM

IN CONJUNCTION WITH THE
31ST ANNUAL AAV CONFERENCE & EXPO
WITH AEMV
AUGUST 1, 2010
SAN DIEGO, CALIFORNIA USA

ASSOCIATION OF AVIAN VETERINARIANS
Advancing and Promoting Avian Medicine and Stewardship

AEMV
Association of Exotic Mammal Veterinarians

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Introduction

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Section 1

AAV Pre-conference Program: Behavior

Heather W. Barron, DVM, Dipl ABVP (Avian),
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Best Practices for Behavior Change: P-A-R-R-O-T

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Session #100

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Abstract: Hidden in the word PARROT are 6 core principles of effective, humane behavior management: Power, Approximations, Reinforcement, Repetition, Observable Behavior, and Teaching Opportunities. These principles are relevant to all species of animals. Understanding each principle and arranging the companion environment to reflect them affords animals the best possible chance of achieving behavioral health and sustaining lifelong relationships with their caregivers.

Introduction

Hidden in the word PARROT are 6 core principles of effective, humane behavior management: Power, Approximations, Reinforcement, Repetition, Observable Behavior, and Teaching Opportunities. These principles are relevant to all species of animals. Understanding each principle and arranging the companion environment to reflect them affords animals the best possible chance of achieving behavioral health and sustaining lifelong relationships with their caregivers.

Power

The power to control one's own outcomes is essential to behavioral health.

Behavior is a tool, an evolved mechanism that enables animals to change their environment in some way that has personal value. Research suggests that controlling outcomes is also associated with emotional well-being, even with human babies as young as 3 months old.[1,2] Animals from dozens of species have demonstrated the contrafreeloading phenomenon: they choose working (response-contingent reinforcement) over freeloading (non-contingent reinforcement) to get valued outcomes.[3,4] Therefore, one way to empower animals is by designing more complex environments rich with choices, problems to solve, and purposeful activities that run the gamut from species-typical behaviors (foraging) to novel behaviors (dunking basketballs).

It is the power to say no that may be most overlooked in the lives of companion animals–the freedom to use behavior to escape events perceived as aversive to the individual. Blocking an animal's escape responses works against its nature to behave for an effect. If response blocking persists, animals can learn that their behavior is ineffective. This leads to learned helplessness, depression, learning disabilities, emotional problems, and even suppressed immune activity.[5–7]

We can empower companion animals to say no by ensuring that they have an escape route, a runway, to move away from imposing hands, and unfamiliar people and items, whenever possible. For instance, a 6-in T-perch used for training restricts a parrot's escape but a 36-in perch (with the caregiver at one end of the perch) allows the animal to express its choice to move away. This strategy may seem counterproductive to caregivers who hope to have close relationships with their pets; however, the apparent relationship that results from forced interaction is only an illusion. True relationships are the result of choice and mutually reinforcing interactions, not force. The goal is for animals to choose to approach their caregivers, which can be achieved by following these teaching principles.

Approximations

The key to teaching new behaviors is reinforcing approximations toward the end goal.

People often expect animals to instantaneously perform complete behaviors. For example, many caregivers expect a parrot to perform the complete "step up" behavior just because they (or anyone else for that matter) offer a hand. When animals don't meet this expectation, caregivers too often resort to coercion, such as cornering the bird to make it step up. Coercion can result in big withdrawals from the trust account, which may not be easily recouped.[8]

Animals learn most behaviors better when they are reinforced for smaller, approximate responses that ultimately lead to the end goal. This procedure is called differential reinforcement of successive approximations, also known as shaping. It is the sharpest tool in any teacher's toolkit. To illustrate, the complete step-up behavior for a parrot can be broken into the following graduated sequence of approximations: 1) looking at the hand, 2) leaning toward the hand, 3) moving a foot in the direction of the hand, 4) taking a step toward the hand, 5) taking several steps toward the hand, 6) walking next to the hand, 7) touching the hand with one foot, 8) resting the foot on the hand, 9) transferring weight onto one foot, and 10) bringing the other foot on to the hand.

After describing the complete target behavior in observable, measurable terms, shaping starts by reinforcing the closest approximation the animal already does (eg, looking at the hand). It is well-timed reinforcement that accounts for the animal offering the behavior again. Once the first approximation is performed without hesitation, the next approximation in the sequence is reinforced (eg, leaning toward the hand). The natural variation in responses produces the next, closer approximation (eg, looking with a slight lean). As training progresses from one approximation to the next, the reinforced responses become progressively more like the final behavior.

Shaping requires good mechanical skills to observe and reinforce the small variations in responding that lead to the final behavior. If a learner experiences difficulty at any step along the way, go back to the last successful approximation rather than withhold reinforcement to the point of eliciting frustration. The best ways to create the momentum needed to steadily advance through the approximations is to reinforce smaller approximations, avoid staying on one approximation too long, use stronger reinforcers, and communicate a clearer criterion with more precisely timed reinforcement.

Reinforcement

Reinforcing consequences are essential feedback about how to behave in the future.

Positive reinforcement is not manipulation, and reinforcers are not bribes. Positive reinforcement is the natural process by which behavior is maintained or increased by consequences. Learning is the process of changing behavior based on experience. This flexibility is surely one of the animal kingdom's most astounding survival mechanisms.

The schedule with which reinforcement is delivered matters. To be used effectively, reinforcement should be delivered contingently (only if behavior *x* occurs) and contiguously (closely following the behavior). Continuous reinforcement (a 1:1 ratio of behavior to reinforcer) is best for teaching new behaviors because it clearly communicates the contingency, if behavior *x*, then reinforcer *y*. Intermittent reinforcement (a ratio between 1:1 and 1:0) results in persistence, that is, behavior that, in the absence of reinforcement, continues longer before decreasing (ie, extinguishing). It is quite likely that behind every problem behavior is a lean intermittent reinforcement schedule that accounts for its persistence.

We can reduce problem behaviors without using punishment by applying the matching law that describes the way relative rates of particular behaviors tend to match the relative rate of reinforcement the behaviors produce. This means that we can increase desirable behaviors and decrease problem behaviors just by offering relatively more reinforcement for desirable alternative behaviors. When the environment is arranged so that desirable behaviors are both easier to perform and more reinforcing than undesirable behaviors, animals naturally perform the desirable behaviors more. A related strategy is to withhold the maintaining reinforcer for a problem behavior (extinction) *and* deliver super-sized reinforcers for an alternative behavior. This strategy is called differential reinforcement of alternative behaviors (DRA). A DRA schedule is often more effective and less intrusive than extinction alone or punishment.

When using a secondary (conditioned) reinforcer to contiguously mark the right behavior (eg, a praise word, click, or whistle), closely follow it with a back-up reinforcer every time (eg, a food treat or tactile reinforcer). This "twofer" (click-treat) approach will maintain the secondary reinforcer at full strength. Delivering a secondary reinforcer without consistently pairing it with another reinforcer will eventually drain the strength of the secondary reinforcer, resulting in a weak marker.[9]

Once a behavior is mastered, it is often possible and beneficial to slowly shift from so-called contrived reinforcers (rewards arranged for training sessions) to naturally occurring reinforcers (the spontaneous outcome of behaving). For example, teaching a parrot to step up may initially involve reinforcing approximations with a contrived reinforcer such as sunflower seeds. With each repetition, naturally occurring reinforcers are also available, such as interacting with the caregiver and moving to a new location away from the cage. These naturally occurring consequences can maintain the step-up behavior in the long run. When teaching a new behavior, contrived reinforcers should be considered for the short run, and naturally occurring reinforcers should be considered for the long run.

Repetition

A high rate of repetition across different conditions builds fluent, generalized behavior.

The term *behavioral fluency* describes quick, accurate performance of a behavior. Fluent behaviors are emitted without hesitation in the presence of appropriate cues or conditions. Generalization is the extent to which a behavior occurs in different settings or situations (ie, stimulus generalization). When it comes to good citizenship behaviors–the kind that keep animals in their homes–the goal is fluent, generalized behaviors. Positive practice is the means to achieve this goal. Positive practice refers to high rate of repetition that results in a high rate of reinforcement.

It's easy to underestimate how much positive practice is needed to build a fluent behavior in one setting and to generalize that behavior across different settings and situations. When an animal doesn't respond to a cue or request, people often feel frustrated and insist, "He *knows this behavior*. I've seen him do it a dozen times!" Effective teachers define what an animal knows by observing what it does, and they interpret failure to respond to a request as an indication more positive practice is needed.

It may take hundreds of reinforced repetitions, in several different settings, for an animal to reliably respond to a cue. Research indicates that learning proceeds more successfully when practice is distributed over many short sessions rather than less frequent, long sessions.[10] Therefore, positive practice doesn't need to be very time-consuming. A few quick repetitions a day can build and maintain behavioral fluency. One positive side effect of this approach is the strong bond that develops between teacher and learner due to the highly reinforcing environment associated with positive practice.

Observable behavior

For an objective understanding of behavior, focus on describing observable behavior and observable conditions.

Most caregivers never consider how their descriptions of behavior are really just value labels of what they think an animal *is* rather than what it *does*. They wish for a companion animal that is friendly, is docile, or is sweet. However, we can't really teach animals what to *be*; rather we teach them what *do, given certain conditions.* For example, we can train an animal to approach people, relax when touched, and take food from human hands. If an animal is observed to do these behaviors, then we label it friendly.

Among professionals, there is a tendency to describe behavior in terms of diagnostic labels based on hypothetical, psychological constructs. Ostensibly, these constructs tell us what an animal *has* or *lacks*, such as anxiety, dominance, or motivation. A construct is a concept that is inferred from commonalities among observed phenomena and used to explain those phenomena. However, constructs are abstractions by definition, and abstractions cannot cause behavior. Although constructs can have a place in theory building, and conveniently summarize behaviors with a single word, they lack the specific information we need for an objective understanding of behavior.

Behavioral descriptions are critical to solving behavior problems. Describing what an animal actually does in context (not in the animal) is central to the behavioral level of analysis, the level at which observable behavior and observable conditions act upon one another. The behavioral level of analysis is one piece of the behavior puzzle without which no accounting of behavior is complete.[11]

Teaching opportunities

See teaching opportunities instead of punishment opportunities–misbehavior is a lack of information, motivation or practice.

An important goal (if not *the most* important goal) is for animals to respond to our requests because they possess the skills and the motivation to do so, not because they have to. Animals learn something with each and every interaction they have with humans, and what they learn influences how they behave in the future. There is no off switch where learning is concerned. Therefore, every interaction with an animal is best understood as a teaching opportunity. Instead of punishing problem behaviors, identify what the animal should be doing instead and teach that behavior.

There is a saying among behavior analysts that the rat is never wrong. This expression is meant to remind us that the responsibility for a learner's behavior rests largely with the learner's teachers. As Kay Laurence eloquently wrote, "[It] is not about teaching impressive behaviors; it is about impressive teaching of behaviors."[12]

Conclusion

It is the nature of all animals to use behavior to achieve valued outcomes. Given the *power* to choose, animals can be taught to behave successfully by shaping small *approximations* that lead to the end goal. *Reinforcement* is the crown jewel of teaching. To use it most effectively, positive reinforcers should be certain, swift and strong. A high rate of *repetition* and planned generalization builds fluent behaviors and a strong human-animal bond. To understand behavior, our main focus should be on *observable behavior*, not vague labels or intangible constructs. We need to be aware that information is exchanged in every interaction we have with animals; thus, every interaction is a *teaching opportunity*. These principles represented by the letters P-A-R-R-O-T are fundamental to facilitating behavioral health and building trusting, long-term relationships with the animals in our care.

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Contrafreeloading in Grey Parrots

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Session #105

Summary Style Manuscript

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Foraging opportunities are effective in prevention or reduction of feather damaging behavior (FDB).[1,2] It is, however, not known whether this results from a change in time budget or from fulfillment of a "behavioral need."

The hypothesis that foraging is a behavioral need corresponds with the principle of contrafreeloading (CFL).[3] The purpose of this study was to determine whether CFL is present in parrots.

Twenty clinically healthy grey parrots (10 with FDB, 10 without FDB) were housed individually in standardized cages with 2 feed bowls (high/low), 1 drink bowl, 2 perches, and 2 foraging toys. All birds were fed pellets.

During a 4-week-period, video recordings were made of the parrots, and analyzed for time spent on preening and foraging. Additionally, food consumption from each item (bowls, toys) was determined.

Birds preferred to use the food bowls in the morning and the foraging toy in the afternoon. Parrots differed in their time spent on foraging (223 ± 119 minutes for birds without FDB; 164 ± 68 minutes for birds with FDB). Additionally, parrots without FDB showed a significantly higher consumption of food from the feeder puzzle, both in time and amount (27.2 g [38%] in 83 minutes) compared to birds with FDB (18.2 g [20%] in 36 minutes).

These results demonstrate that CFL occurs in parrots, supporting the hypothesis that provision of foraging opportunities is essential for their well-being. Differences between birds with and without FDB, however, indicate that "needs" and time allocation may be altered in individuals with FDB.

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Training a Harpy Eagle to Present Medical Behaviors

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Session #110

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Abstract: The Dallas Zoo is home to harpy eagles (*Harpia harpyja*). The mature female hatched in 2002 has been on exhibit since 2004. In September 2007 keepers initiated a training program to facilitate optimal care of this individual. Initial training included the use of targets to teach hands-off behaviors such as moving from point A to point B, shifting into a holding area, stepping onto a scale, and flying from perch to perch on cue. Eventually keepers were able to work in the enclosure with the eagle and incorporated these behaviors into educational presentations. As the bird continued to progress, it was decided to train behaviors that would allow the eagle to make appearances outside of the enclosure. This included teaching the bird to wear falconry equipment and sit comfortably on a glove in public areas. Because of the bird's role as an exhibit bird, it was important to the facility that equipment could be removed completely for when the bird was on display. This challenged keepers to train the eagle to allow daily manipulation of feet, extensive touching to the tarsus while anklets and jesses were attached and removed, and learning to step onto a glove voluntarily. By adhering to a strictly positive reinforcement-based training approach, staff members were able to successfully achieve their goals and more. This paper will share the triumphs and challenges of using positive reinforcement to train one of the largest and most powerful species of eagles in the world.

Introduction

The Wings of Wonder section of the Dallas Zoo is home to a variety of raptor and New World vulture species. The recently refurbished exhibit is primarily designed to give zoo guests the opportunity to view these impressive birds of prey in their large, attractive, and naturalistic enclosures. There is an additional goal of obtaining reproductive success with a number of the species on display. To date, a pair of king vultures (*Sarcoramphus papa)* has successfully reproduced while housed at the refurbished Wings of Wonder.

In the past, caring for exhibit and birds in reproduction programs usually involved a "less is more" approach. The idea being that the fewer disturbances a keeper makes in a bird's life, the more likely the bird is to show behaviors indicating comfort and therefore potentially breed. However, with the addition of an animal training consultant and zoo keeping staff members with experience and interest in training, it was decided a different approach could prove successful.

In 2007 it was decided a proactive approach to training would be implemented for the birds in Wings of Wonder. The goal was to train as many birds as possible for behaviors that would facilitate husbandry and daily care. Initially several of the vulture species were identified as excellent training candidates. The criteria used to determine this was the level of comfort in close proximity to keepers and their willingness to accept food from keepers. A female Andean condor (*Vultur gryphus*) was involved in the training program until her transfer to another facility. The king vultures (*S papa)* were successfully trained to target, shift, enter a kennel, present a foot, and allow tactile pressure to the chest. They also bred and raised offspring and participated in training throughout the process. The offspring also learned to target and stand on a scale.[1]

As training proved to produce positive results, it was decided to consider adding other candidates to the training roster. Another bird that demonstrated a high level of comfort in the presence of the keepers was a female harpy eagle (*H harpyja*) named Killa (pronounced "kee-ya").

The Training Candidate

The harpy eagle (*H harpyja*) is one of the largest species of eagle. Harpy eagles can weigh from 10 to 20 pounds and have a 6 1/2-foot wingspan. Their talons are comparable in size to the claws of a grizzly bear. They feed primarily on animals that live in the trees, like sloths, monkeys, opossums, and some reptiles and birds. Harpy eagles are highly maneuverable fliers and strike their prey after a rapid pursuit through the trees.[2]

Killa was hatched in 2002 at the Avian Propagation Center at the San Diego Zoo. Hatched in an incubator, she was then hand-raised using techniques to avoid imprinting on humans. While in the brooder she was covered with a towel, set behind a curtain, and a CD of rainforest sounds was used to drown out the sounds of human voices. During feeding, keepers placed a sheet over their heads and used a harpy eagle puppet to deliver food.[3]

Because of her large size and power, birds such as Killa have traditionally been regarded as potentially dangerous. For safety purposes, the Dallas zoo policy requires that a keeper is only allowed to enter her enclosure if another keeper is present. However, what keepers observed was a bird that appeared quite comfortable in the presence of people. Killa had never been observed to present aggressive behavior. Nor did this bird demonstrate a fear response. This in part may be the result of her experiences as a chick with humans at the Avian Propagation Center. Other individual birds in Wings of Wonder such as the much smaller ornate hawk eagle (*Spizaetus ornatus*) would show overt aggressive behavior, flying directly at keepers. The spectacled owls (*Pulsatrix perspicillata*) showed extreme fear responses and escape behavior when keepers were present. Compared to these individuals, Killa appeared to be a training candidate with excellent potential.

With the permission of the curator, Killa became the next training subject for the Wings of Wonder team.

Initial Training

Traditional raptor training practices often involve capturing and restraining a bird to get a bird on a scale to determine a "starting" weight for training. However, the process of grabbing and restraining a bird against its will is contradictory to the goals of a positive reinforcement training program. Furthermore, knowing the weight of an animal is not necessarily important to reaching training goals. While the information can be of interest for evaluating health, it is not required to train an animal.

Instead keepers tried to ascertain, based on observations, the amount of food Killa typically ate in a day. They also observed her responsiveness to food when she was relaxed and comfortable. More or less food could be added or removed from the diet based on her response during a training session to maintain an adequate level of interest in training. This amount changed in correlation with the time of year. Colder temperatures meant an increase in diet. Warmer weather meant a decrease.

Because Killa showed a high level of comfort around people, she readily took food from keepers in the first training session. Per zoo policy, the food was offered from hemostats as opposed to hands. Training was initiated with keepers outside of the enclosure. The first behavior Killa learned was to target. The target was a blue plastic circle attached to a dog clip. The target could be hung on the outside of the cage near the eagle. At first

food was held near the target. Killa learned to touch the blue target with her beak to earn the food reinforcer. In the early stages of training, the target became a useful tool to call Killa to the front of the cage for keeper talks. This offered zoo guests a great opportunity to see the impressive wingspan of a harpy eagle (*H harpyja*) as she flew to the front of the enclosure.

The target was also used to call her to the back of the cage to work on training other behaviors. To initiate introduction of a kennel, the target was used as a means to pair reinforcers with the kennel. The kennel was placed outside of the enclosure and Killa was reinforced for targeting nearby.

Success Leads to More Training Goals

As hoped, Killa learned quickly and was an eager participant. In addition, her continued calm demeanor helped increase the teams' confidence that the presentation of aggressive behavior would be very unlikely. Because of this, keepers moved from training outside of the enclosure to training inside.

Very quickly, Killa learned to fly to perches when cued by the keepers. This made for an even more dynamic presentation for zoo guests.

Killa also learned to shift into a holding area. This was an important training goal. Colder weather often required birds to be placed in holding. Training Killa to voluntarily enter her holding area made this process stress free and more reliable.

Crate training progressed with the crate now being in the enclosure, along with keepers in the enclosure to help direct Killa into the kennel using the target and food reinforcers.

After months of training, a scale could be used inside the enclosure to get a weight on the eagle. Although it was helpful to know her weight for recordkeeping purposes, it was not integral to her training success.

A Small Setback

One of the challenges for the harpy eagle was her exhibit. On several occasions, Killa was found caught in the netting/wire of her enclosure. This meant keepers had to restrain her and untangle her legs from the netting. Pairing her trainers with this aversive experience had the potential to affect the trusting relationship that had been nurtured via positive reinforcement. Unfortunately, the problem occurred more than once before Killa was moved to another enclosure while the netting was replaced with something more suitable.

The entanglement incidents did cause Killa to show some hesitancy with her trainers, but because of a long positive reinforcement history she was able to return to her consistent performance during training rather quickly.

Training to Wear Falconry Equipment

The move to the new enclosure proved to be a blessing in disguise. Although the enclosure was smaller, it opened up the possibility for some new training goals. Because Killa had been doing so well, the idea of bringing her out of the enclosure for educational opportunities had been suggested. However, her role as an exhibit

(and potentially breeding) bird was also important. To bring Killa out of her enclosure would require the bird to be equipped with anklets and jesses. However it was agreed for her safety, based on her entanglement history, and for her role as an exhibit bird that she not wear equipment while in her enclosure. For these reasons, it was decided to train her to allow keepers to apply anklets, jesses, and any other equipment for excursions outside of the enclosure. These would then need to be removed when she returned to her enclosure.

At the time, to the team's knowledge, training a harpy eagle (*H harpyja*) to accept the level of manipulation required to apply and remove equipment daily had not been attempted before. A shaping plan was devised to achieve the training goal. The steps included the following:

- Touch foot with hand
- Touch foot with anklet
- Drape anklet on foot
- Wrap anklet around leg
- Secure anklet around leg
- Thread jess
- Add swivel and leash
- Step to the glove
- Short hop to the glove
- Walk with the bird on the glove

Training sessions were scheduled to occur once daily. However due to staffing challenges, training could not always take place every day. Despite this, the goal of securing the anklets comfortably around Killa's legs was achieved in 33 training sessions. It took 9 more training sessions for the eagle to be comfortable with the jesses being threaded. Sessions to add the swivel and the leash followed. After 11 more training sessions, Killa was stepping to the glove without hesitation.

Training Killa to accept touching on her feet also lead to some other behavior goals. These included training her to allow a keeper to wrap his hands around her legs and apply some pressure. The goal was to be able to approach and calmly secure Killa's legs if she needed to be captured and restrained. This approach is far less stress-inducing than traditional methods that require birds to be chased, netted, and grabbed for restraint.

An additional bonus to the tactile training was the ability to evolve this into touching Killa's sternum and pectoral muscles. This allowed keepers another means to evaluate her physical condition in a manner that was stress-free for the bird.

Since Killa's initial training, it was discovered that nature cinematographer and falconer Neil Rettig has also had success training a harpy eagle to accept the application of anklets and jesses and allow touching.[4] With more than one harpy eagle responding well to the use of positive reinforcement to train these behaviors, it is possible more trainers will be inspired to consider this as a training goal.

Addressing Aggressive Behavior and Fear Responses

Because Killa continued to be an excellent training subject, it was an important goal to maintain her calm demeanor. Avoiding creating aggressive behavior or fear responses were high priorities. If any activity elicited aggressive behavior, it was immediately discontinued and smaller approximations were employed. The smallest demonstration of body language typically associated with aggressive behavior would cause trainers to reevaluate their strategy. This was especially relevant when introducing touching with hands or the anklets and when manipulating equipment, for example, when threading the jesses.

Another factor that contributed to some aggressive behavior was using large pieces of food. To encourage Killa to hop to a glove and remain for a period of time, a large piece of food was used to lure her initially. However, if Killa was particularly motivated for training she would show some body language indicative of aggressive behavior. For these reasons, trainers quickly switched to using smaller pieces of food and waiting for presentation of the behavior paired with calm body language before delivering reinforcers.

Although some female eagles have been observed to present aggressive behavior at sexual maturity, this has not been observed with Killa. She has also not yet been provided with a nest site, nesting materials, or a mate. Other birds at Wings of Wonder have successfully continued training during breeding and rearing of offspring. It will be interesting to observe if this can be repeated with Killa at some point in the future.

Keepers also did not want to create a fear response. This was especially important when the equipment was on Killa and she was on the glove. Keepers wanted to avoid creating a situation in which the eagle would be likely to fly or bait off the glove. When a bird baits, it is often trying to escape a situation it perceives as uncomfortable. In other cases, a bird may be trying to go towards something, for example, a preferred perch. In Killa's case, her few instances of baiting appeared to be in line with wanting to go away from the situation. They were always associated with the keeper walking around the enclosure with Killa on the glove. Learning to adapt to the movement was a challenging behavior.

In many cases, a bait can be avoided by observing a bird's body language closely and reinforcing the bird for remaining calm or, if possible, removing the bird from the situation. However, if the moment is missed and a bird does bait it can be very challenging for some birds to recover. Restricted movement is not something to which Killa was accustomed. When she did bait, the trainer needed to assist her to get back on the glove and often she was no longer interested in participating in the training session after a bait.

Other Challenges

At this point in Killa's training, another major setback occurred. Killa was found disoriented and wobbly. Because of this she was sent to the veterinary hospital for medication and observation. Training was discontinued for approximately 30 days while she remained at the hospital. She was eventually returned to Wings of Wonder. Her training was resumed. However, she was eased back into the process. Behaviors practiced initially included hopping on the scale, targeting around the enclosure, and crate training.

Along the way, other training challenges occurred, including finding a crate large enough to comfortably hold a large eagle. Another challenge was creating stable portable perches that could withstand the force of a harpy eagle landing and launching into flight. Other setbacks included lack of funds during hard economic times to purchase materials to facilitate training. Because zoo policy required 2 keepers to enter Killa's enclosure, training was sometimes delayed due to lack of staff availability. However, despite these challenges, training did progress.

To date, Killa has successfully learned to target, fly from point A to point B on cue, shift into a holding area, fly to a scale, enter a kennel, allow falconry equipment to be applied/removed, hop to a glove, perch on a glove while a trainer walks around her enclosure, allow her feet to be manipulated, allow a trainer to wrap his hands around her legs and apply pressure, and allow touching of her chest and beak. Killa's training is ongoing and it is the team's hope to continue to add more behaviors to her repertoire.

Conclusion

As the potential of positive reinforcement is explored more, traditionally held beliefs about animal care are challenged. Exhibit birds can function as education program animals, falconry methods can be modified to ensure the most positive methods are utilized and coercion is reduced or eliminated, micromanaging diets and weights is not required to train, and potentially dangerous animals can be trained and trusted to be non-threatening. Most importantly birds, such as Killa can be an example of what is possible when you train with positive reinforcement.

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Post-traumatic Stress Disorder among Parrots in Captivity: Treatment Considerations

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Abstract: Conventionally, the majority of avian diagnoses are rendered in terms of physical and behavioral issues. Only of late, with the emergence of a unitary, vertebrate-common model of brain and mind, is attention drawn to psychological suffering in birds and its pivotal role in avian health. Overlap between mammalian and avian neuropsychologies permits fluid exchange between human and avian research and healthcare. In this expanded view of avian experience, behavior is subsumed within psychology and certain parrot behavioral "problems" reveal as expressions of post-traumatic stress disorder (PTSD). Similar to humans exposed to sustained incarceration and acute trauma, parrots in captivity conform to criteria characterizing Complex PTSD. This paper reviews avian PTSD, its causes, symptoms, and illustrates a therapeutic treatment approach for birds that has been adapted from human models to treat PTSD.

Introduction

Individuals suffer from post-traumatic stress disorder (PTSD) as a result of exposure to psychological trauma. PTSD is defined in the American Psychiatric Association's Diagnostic Criteria from the Diagnostic and Statistical Manual as exposure to a traumatic event when the individual witnessed, experienced, or was confronted with an event that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others; which includes intense fear, helplessness, or horror.[1] PTSD differs from other anxiety disorders because the individual's existence is directly threatened with no perceived avenue of escape. While originally considered to be limited to specific communities, PTSD in humans has been demonstrated to affect 8% of the general population, within which 30% of veterans, 45% of battered women, 50% of sexually abused children, and 35% of adult rape victims are likely to suffer from PTSD at some point in their lifetime. Symptoms that characterize PTSD have been increasingly identified in other animals including elephants,[2,3] chimpanzees,[4,5] wolves, dogs, and parrots.[6]

Symptoms of PTSD are diverse (Table 1) and often co-morbid with other disorders. The phenomenology and assessment of PTSD follows a continuum of complexity and severity (Table 2).[7] Because the precipitating event(s) are life-changing, treatment often evokes and involves philosophical and existential issues. Neuropsychological research among PTSD victims shows that the traumatic event is neurologically "wired" to produce exaggerated responses to mental cues or environmental stimuli, interpreted by the individual as preludes to a recurrence of the original trauma. [8] This neurobiologically shaped survival strategy is more tenacious than other anxiety disorders. Symptom and treatment complexity increase with several factors including adult versus childhood onset, delay between exposure and symptom onset, severity of symptoms, multiple exposures, invasive nature of traumatic events, and potential social stigma associated with traumatic experiences. For example, until the aftermath of the Vietnam War, veteran PTSD was masked by euphemisms such as "combat fatigue," "shell shock," and "soldier's heart" and largely dismissed as non-existent or shameful conditions experienced by those who are "weak" or lacked "normal" coping skills or responses prior to the event.[9]

Table 1. Diagnostic criteria of PTSD (APA 2002).[1]

DSM Criteria		Avian event, symptom or condition in captive setting
A. The victim has been exposed to both:	1. A traumatic event experienced or witnessed, or was confronted that involved actual or threatened death or serious injury; or a threat to the physical integrity of self, family, others	Wild capture Premature weaning Inadequate, non-normative or non-existent nurturing, rearing, learning of critical skills Isolation
	2. The victims' response involved intense fear, helplessness, or horror. *Note:* In children, this may be expressed instead by disorganized or agitated behavior.	Neglect Physical, verbal, psychological abuse Captivity
B. The traumatic event is persistently *re-experienced in one or more of the following ways:*	1. Recurrent and intrusive, distressing recollections of the event, including images, thoughts, or perceptions (*Note:* In young children, repetitive play may occur in which themes or aspects of the trauma are expressed);	May be expressed as inattention, aggression or mood disturbance Inter or intra-specific aggression, biting
	2. Recurrent distressing dreams of the event (*Note:* In children, there may be frightening dreams without recognizable content);	Restless or disturbed sleep patterns; startled, intense fearful awakening
	3. Acting or feeling as if the traumatic event were recurring, including a sense of reliving the experience; illusion, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated (*Note:* In young children, trauma-specific re-enactment may occur);	Inattention, aggression directed at triggers of trauma; Stereotypy and other dissociative disorders including feather destruction, self-injurious behavior, unresponsiveness
	4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event;	Sudden, inexplicable fear or aggression; Hyper-arousal to "neutral" everyday noises or actions, phobias
	5. Physiological reactivity upon exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event	Hyperarousal, hypoarousal. Displaced or projected fear, aggression towards conspecifics, caregivers, inanimate objects in the immediate environment

Table 1 Cont'd. Diagnostic criteria of PTSD (APA 2002)[1]

DSM Criteria		Avian event, symptom or condition in captive setting
C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness as *indicated by two or more of the following:*	A. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by 3 (or more) of the following: 1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma; 2. Efforts to avoid activities, places, or people that arouse recollections of the trauma; 3. Inability to recall an important aspect of the event; 4. Markedly diminished interest or participation in significant activities; 5. Feeling of detachment or estrangement from others; 6. Restricted range of affect (for example, inability to have loving feelings); 7. Sense of foreshortened future (for example, no expectation of having a career, marriage, or a normal life span).	Listless, apathetic, unresponsive to environmental stimuli, socially, nonresponsive, seeks isolation, physical distance from conspecifics and/or caregivers, little affect towards conspecifics or caregivers, anorexic, no interest in external world; anhedonia; withdrawal
D. Persistent symptoms of increased arousal as indicated by two or more of the following:	1. Difficulty falling or staying asleep; 2. Irritability or outbursts of anger; 3. Difficulty concentrating; 4. Hypervigilence; 5. Exaggerated startle response.	Constant agitation, inability to relax/sleep, hyperactivity, hypervigilence, exaggerated startle response, relative calm followed by episodes of aggression or extreme fearfulness
E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than one month.		Behavioral disturbance does not abate or lessen over time, in new environment or new social setting and persists despite months or years of stable, secure, familiar, predictable environment and/or social interactions
F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.		Disturbance impedes healthy social interaction, ability to adjust to changes in environment, coping mechanisms under stress and leads to isolation, depression & neuroses. Severe, negative impact on adaption to captivity, overall quality of life

Severity, that is, the degree to which symptoms affect the life arc of the victim, symptom duration, and the nature of the traumatic event itself, is often linked to psychological compromise during childhood. Many human and avian victims suffering from Complex PTSD have a history of repeated, personal victimization experiences "often beginning with extended childhood abuse and neglect and associated disruption of the parent-childhood attachment system."[7] The National Child Traumatic Stress Network (NCTN) Complex Trauma Working Group reports that "children exposed to complex trauma often experience lifelong problems that place them at risk for additional trauma exposure and cumulative impairment….in childhood, adolescence and adulthood." Further, early trauma extends to diverse domains including the "biological, attachment, affect regulation, dissociation, cognitive, behavioral control, and self-concept".[10,11] Although children exposed to trauma may be diagnosed with PTSD, major depression, and/or some form of anxiety disorder, these diagnoses capture only a limited aspect of the traumatized child's complex self-regulatory and relational impairments. These individuals experience a double loss, the trauma itself and the lack of coping mechanisms developed in the human early developmental (altricial) period and later incidents. Trauma experienced in formative developmental periods is a leverage point in PTSD etiology, but also forms the foundation for model therapeutics.[6] There are multiple methods adopted to treat PTSD (Table 3).

Table 2. Simple/Complex PTSD Comparison (after Lanktree & Briere, 2008).[11]

Simple Trauma	Complex Trauma
Non-interpersonal	Interpersonal
Limited exposure--may be a single incident	Multiple exposures of different types of trauma
Shorter duration	Longer duration
Onset of traumatic exposures more likely to be at later stage of development	Onset of traumatic exposures may have begun at an earlier stage of development
Support of caretaker/family	Less or no support of primary caretaker/family
Secure attachment with primary caretaker(s)	Insecure attachment

Table 3. Human PTSD therapeutic models.

Therapeutic Model	Model Assumptions	Therapeutic Features
Cognitive Behavioral	Problems arise from the way we interpret situations, and resulting thoughts, feelings and behaviors	Treatment includes self monitoring, cognitive restructuring and behavioral experiments
Behavioral Activation	Positive stimuli are lacking in the client's environment and negative stimuli are promoted	Increase activity levels that have positive outcomes and feelings while avoiding negative events and those that remind the person of the traumatic stimuli
Acceptance and Commitment Therapy	The concept that suffering does not arise from experiencing emotional pain; but from the avoidance of the pain itself	Therapy tries to help individuals realize that avoiding emotional pain does not work; stopping the struggle to avoid pain and focus upon alternative positive events
Exposure/Implosion Therapy	That problems arise from an irrational fear and reaction to an identified event	Allowing the individual to be confronted with the stimuli in a controlled environment to eventually desensitize them from the over-reaction to the stimuli
Stress Inoculation Therapy	That problems arise from an irrational fear and reaction to an identified event	A graded development of coping mechanisms, like deep breathing exercises to cope with memories of or re-exposure to the stimuli
Hypnotherapy	That problems arise from a rational response to an irrational, unexpected, unpredictable event	Reprogram emotional and cognitive responses through guided imagery using alternatives of the traumatic event
Cognitive Processing Therapy	Belief that symptoms stem from pre-stimuli beliefs about the self and the world and the creation of "sticking points" from the stimuli	Rational review of these beliefs helps the individual cope with the non-stimuli world and prepare for the potential (if any) of a new similar stimuli.
Eye Movement Desensitization and Reprocessing (EMDR)	Substitutes neuropsychological programming from negative events with alternative memories	While talking about memories, individuals are asked to focus on distractions like eye movements, hand taps, and sounds.
Attachment Theory	When trauma is associated with relational aspects of altrical development	Uses a modification of stress inoculation therapy and a variant of either cognitive behavioral or hypnotherapy

Relational Therapy for Treating Avian PTSD

While there are many suggested approaches to trauma treatment, one of the key factors underlying distress and symptoms is relational trauma. Indeed, one might generalize and consider all trauma in humans and birds alike to derive from one type of relational tear or another. Trauma is the psychoid's (integrated mind-body system) response to changes in the environment that exceed psychological and psychological expectations and capacities. More specifically, parrots in captivity experience relational stress to trauma because of their non-normative existence as captives and their life in socially and emotionally depauperate conditions.

Further, as discussed more fully below, parrots in captivity definitionally conform to diagnosis of PTSD/Complex PTSD. Subsequently, while there may be differential diagnoses, considerations of trauma and how they may inform treatment is central and has compelled the use of trauma-based assessments, namely the Achenbach Child Behavior Checklist (CBCL),[12] Trauma Symptom Checklist for Children (TSCC),[13] and Strange Situation Test.[6] This does not exclude other diagnoses, but is necessary as a fundamental framing in recognition of captivity's defining compromise to avian health. Traumatology also underscores the synoptic framing of psychological diagnoses as opposed to the limited and hence, inaccurate, framing of a purely behavioralist view.

Study site

Psychological assessment, diagnosis, and treatment of birds is conducted at the Avian Care & Recovery Center (ACRC), a sanctuary and training and educational center located on the grounds of The Landing (operated by MAARS), St. Paul, Minnesota. The ACRC was created in collaboration with the Midwest Avian Adoption & Rescue Services, Inc (MAARS; www.maars.org) and the Kerulos Center (www.kerulos.org) to establish a formal program and treatment center that could provide consistent, systematic care for birds suffering form severe psychological trauma. The Landing cares for approximately 100 permanent residents. Staff includes a clinical psychologist and multiple trained, volunteer caregivers. The director and director's assistant (who have day-to day contact with the study subjects and extensive experience working with parrots in recovery) and the clinical psychologist and trained caregivers (referred to in text collectively as *therapists*) conducted on-site assessments and provided structured interviews, and client case and medical histories.

Framing avian PTSD and treatment

Because most rescued birds arriving at The Landing are adults with fully developed faculties, it is important to understand why an assessment developed for human children has been adopted. Often, comparisons are made between parrots and human children. For instance, it is not uncommon to find such descriptions in popular bird magazines and resources: "parrot psychologists often claim that a parrot has the emotions of a 2- or 3-year-old child. A good guideline is that your parrot's intelligence can rival that of a human child between the ages of 1 year and 4 years old" and a " parrot is as smart as a 3-year old child with the emotions of a 2-year-old child." However, these are incorrect analogies and damaging to the bird's well-being.

In the case of human children, caregiver expectations are calibrated to developmental stage without prejudice. Because children's behavior and psychological state are considered normative, they are encouraged, not punished. However, when the psychological state, intelligence, and emotions of adult birds in captivity are judged in terms of another species (human) at another developmental stage (childhood), birds are infantilized and marginalized. They are often meted punishment or subjected to "training" to eliminate the so-called "problem," which is in reality a communication of distress. This attitude strips away the bird's sense of self and agency, experiences that psychologists consider profoundly damaging to any individual.

An accurate interpretation is that these individuals are expressing symptoms of affect dysregulation as a consequence of their experiences in the highly non-normative conditions of captivity. Biting, screaming, asociality, and fear-based responses are somatic communiqués of an adult who is trying to survive under imposed conditions of total control at the hands of his/her captor. From the perspective of traumatology, there are no avian "behavior problems," only birds suffering psychological distress that lack adequate developmental and/or environmental resources to self-heal and/or coping skills that would permit at least some degree of adjustment to the alien, abnormal, dysfunctional environment.

While it is true that early relational and repeated traumas can lead to cognitive and emotional developmental delays, symptoms of birds in captivity are reflective of *the trauma* that has been experienced, not *the victim*. Affect and stress dysregulation that frequently express as hyperarousal, "aggression," and other symptoms result from compromised rearing conditions and abuse. In captive situations, as in the case of human children who depend on their abuser to survive, the victim's sense of self and means of coping are shaped by the traumatic environment and relationship. Subsequently, dysregulated emotions and behavior are indicative of internal responses to distorted external environments–not immaturity or inferior capacities. In fact, when one appreciates the enormous differential between the social and ecological conditions in which birds are forced to live with those historical free-ranging conditions to which they are evolutionarily adapted, one is impressed by the fact that parrots are willing and able to adapt as well as they do.

We choose diagnostic tools developed for human children for adaptation in treatment of parrot PTSD for other reasons. One, similar to human children, parrots in captivity are non-verbal by conventional adult human standards and are silenced by their marginalized positions of power. Verbal narratives that are employed for adult human trauma victims are often not appropriate to obtain information for human children as in the case of parrots. The absence of verbal narrative may have presented a difficult hurdle in the past, but models of communication have changed. Similar to other animals, children were once believed to be incapable of developing PTSD because they did not express typical verbal manifestations of the disorder. Neuropsychologist-analyst Allan Schore and others argue that the exclusion of prosody, facial expression, and unconsciously derived information ignores the majority of affective signals. "The [focus] on the patient's verbal outputs as the primary data of the psychotherapeutic process…delete the essential 'hidden' prosodic cues and visuoaffective transactions that are communicated between patient and therapist." Overall, spoken language is "a relatively poor medium for expressing the quality, intensity and nuancing of emotion and affect in different social situations…the face is thought to have primacy in signaling affective information".[14] A psychological framing of avian symptom contextualizes both behavior and verbal communications as only part of the broader subjective experience and exchange. One of the most important sources of information derives from a bird's symptoms. They speak loud and clear of the bird's inner distress. Inter-species communication is a rich and growing area of research and promises to make strong contributions to avian healthcare practices.[6]

Two, treating both children and wildlife (in captivity) raises many ethical questions concerning implied consent. Captivity in its most non-prejudicial or unqualified form is created and maintained by an intrinsic inequality of power between the captive and the captor–a condition that infers a violation of (implied) consent, therefore, becoming the foundation of event-related trauma and a later diagnosis of PTSD. Symptom resolution and treatment can become complex as a result. The question is, then, does the relationship between captive wildlife and human caretakers/guardians undermine psychological health? Does it form a source of trauma or act to re-traumatize? And does [relative] "freedom" after prolonged captivity and the survival-driven bond between the captive individual and his/her captor only serve to add yet another layer of re-traumatization, again, directed by the inequality of power between the captor and the captive? Is, for instance, the behavior for which a "reward" is provided (such as a pumpkin seed) given to reinforce something that the client (parrot) enjoys, or does the

exchange corrode agency and amount to little more than complex justification for manipulation of one whom is totally powerless and dependent? [15] All of these questions confront the therapist and must be addressed carefully, honestly and without ego or species-ism.

On a practical level, it is important to obtain as much information as possible regarding the trauma victims' history. In the case of "rescued" parrots, it is frequently the case that little is known about what the individual experienced. It is even sometimes difficult to ascertain the parrot's age. For this reason, we adapted Ainsworth's Strange Situation Test.[6,16] The "strange situation" test is a way to relate differences in attachment style to behavior. In the case of humans, a parent and child sit together in a room, and then a stranger enters. After a short while, the parent leaves, then returns to comfort and embrace the child. Based on observed behavior of child and parent during these series of events, 1 of 4 general classes of attachment style may be identified: secure, insecure-avoidant, insecure ambivalent, and disorganized-disoriented attachment. This has been added to the MAARS Avian Quality of Life Assessment (AQLA) to gain insights into developmental history of the parrot and relational patterns and trauma.

Intake and assessment

MAARS has developed and utilizes an Avian Quality of Life Assessment (AQLA) to evaluate incoming birds' physical, mental, and social health organized in 5 categories: mobility, sustenance, physical comfort, psychological adjustment, and social interactions. The AQLA has been used for several years. It was originally inspired by human hospice, nursing, and medical care. More recently we have included an adaptation of queries concerning trauma and psychological state as developed in the Achenbach Child Behavior Checklist (CBCL)[12] and Trauma Symptom Checklist for Children (TSCC).[13] The TSCC is used to assess psychological state of children and teenagers who have experienced trauma. AQLA evaluation is conducted within 2 days of the bird's arrival to gauge general psychological distress and gauge severity/complexity. The test is implemented at intervals during the progress of the bird's recovery to track changes and therapeutic treatment efficacy.

Implementation of relational therapy

After intake, initial evaluation, and preliminary diagnosis, there are 4 sequential steps comprising the treatment path. Each builds upon the other and all are intended to gradually transform the individual psychologically from a collapsed, fearful victim to an agentic state of being.

- *Establishment of a secure environment* (safe, "therapeutic container") through predictable and nurturing sanctuary, free of visual, audio, and tactile threats: an individual cannot recuperate in a state of near constant agitation-arousal. The principle behind the therapeutic container is that it minimizes stress for someone already psychologically and/or physically compromised by an experience or wound. Physically, the therapeutic container usually means a quiet place, soft light, comfortable surroundings, and the ability to sit peacefully with no interruptions or threatening intrusions from the outside world. Yet physical safety alone does not always confer a sense of safety. There is also relational safety to consider. In human treatment, the therapist's task is to cultivate a "therapeutic alliance" with the client that creates a relational holding environment to permit the client to feel safe enough to allow his/her true self to emerge without an excessive fear of vulnerability and exposure.[15] Eventually, the victim should appear calm and exhibit confidence in the routine and conformation of their environment. This sense of security allows temporal healing that offers opportunities for trust building and self-expression.

- *Encouraging the client/victim to determine and hold the locus of control*. Locus of control is a concept first developed by psychologist Julian Rotter (1954)[17] to describe an individual's perception of their own agency, the ability to decide, respond to, and act upon circumstances that affect their well-being

and life. It is related to other concepts such as agency and self-efficacy.[18] By definition, a trauma victim experiences a total lack of control during the event, a perception that lingers and underlies trauma symptoms. The therapist is responsible for providing diverse self-enrichment and empowerment opportunities through self-determined choice (eg, toys, variable forms of perches, different food choices, enclosure door open where parrot may choose to enter or exit at will, etc) and other client preferences to build mental and physical competence that have been lost or severely reduced. Increased competence and confidence developed through the empowering opportunity to choose without reprisal, leads to individual self-repair and revitalization of agency. In theory, this will change over time, finally, culminating in the establishment of psychological equilibrium mirroring original self-capacity. However, in many cases, either because of the severity of the trauma, an experience of early relational trauma, and/or limits of post-trauma context,[5] recovery will be constrained.

- In severe cases of trauma or PTSD symptoms, it may be necessary to employ the use of psychotropic medications, as a temporary or long-term measure, in order to overcome or decrease extreme depression, anxiety or aggression to the extent that the severity of these conditions interfere with the therapist-client relationship or greatly diminishes the client's capacity to integrate the new information necessary for a recovery process and progress. However, as with all aspects of treatment, for all species, the use of such medication should be conservative, clearly indicated, risk vs benefit factors considered, and documented as is appropriate according to the AQLA and modified treatment plan.

- *Increasing enrichment within a relational context* shifts emphasis from building the victim's confidence through physical surrounding to that of the social realm. The concept of locus of control relates beyond the physical realm to include attachment style and sense of self, both of which are culturally contingent.[3]For instance, individuals who have experienced a secure attachment generally have a high internal locus of control: they feel self-empowered and agentic. In contrast, those who experience insecure or ambivalent attachment styles through development tend to feel destabilized by their environment and therefore have a moderate high external locus of control and develop psychological and behavioral coping skills to be able to regain a sense of control, many of which are maladaptive in adult life. However, in collective, interdependent cultures (as opposed to individualistic, independent cultures such as modern western society), the locus of control of a "healthy' self may lie somewhere "in–between," that is, an individual sense of self relates to the "group mind."[3] This is likely the case for flock and herd societies such as those of parrots, elephants, and other social animals. From this point of view, we can understand that rebuilding a flock, whether comprised of birds, humans, or a mixture, is necessary to restore agency for those individuals who are were reared under these circumstances. The subject of who and what comprises the "best" social context for an individual in recovery is complex and requires careful study and observation to avoid re-traumatization.[5,6] The treatment regimen is essentially "parrot-directed"–that is calibrated and initiated by the victim/client, and also advised by what the Strange Situation Test has revealed.[6] This reversal in locus of control at all levels initiates a process of healing by cultivating contexts that can emulate natural settings and procedures as much as possible. Just as in the wild, a bird chooses where s/he goes and what s/he does, the therapeutic regimen mirrors the individual's locus of control in their environment.

- *Hypnotherapeutic and/or desensitization therapy assistance* is implemented once the victim has developed a secure sense of self and competence within their environmental sphere. "Diluted" triggers that may be reminiscent of the trauma are gently reintroduced (eg, the sound of paper bag that was associated with traumatic experience) in a controlled environment to allow to deepen their sense of control and to depotentiate the stressor. This facilitates opportunities for long-term healing – effectively, "re-wiring" neural pathways, learning to distinguish between past and present experiences and/or potential triggers and, finally, self-modulation of the fight-or-flight survival response. Just as in human cases, the client

eventually is allowed to identify the traumatic elements over which they have control; recognize that the traumatic event is unlikely to reoccur; and renew their daily routines with non-avoidant behaviors. Hypnotherapy, used by trained therapists at MAARS, has been found to be a useful adjunct by offering avian clients an environment conducive to the healing process. The stress reduction aspects of hypnotherapy, as well as the focus upon the current moment, allow clients to buffer against hypervigilance and avert focus from common PTSD event triggers. However, given the neurobiologically ingrained nature of trauma, therapists are aware of the potential for inadvertent triggering and symptom reoccurrence. This is an important concept to convey to parrot guardians to prevent possible recriminations against the victim: the parrot is not "being bad" but re-experiencing their painful trauma. At the time of reoccurrence, the victim believes that their life is in danger.

Conclusions

Compelled by multiple lines of research from the neurosciences and the volume of corroborating ethological and clinical data, human psychology/psychiatry and veterinary medicine/ behavior have converged under one conceptual umbrella. A purely behaviorist framing is therefore incomplete as it excludes consideration of the range of parrot subjective experience and symptom of which behavior is only a single expression: a psychological framing is mandated. This is a critical point in the case of birds in captivity as captivity involves not only physical but mental stress. Trauma is psychologically and physically mediated.

Definitionally, all birds in captivity qualify for Complex PTSD. The wild-caught experience meets the criteria for PTSD through involuntary capture and confinement as does captive breeding where parental-altrical and early-nature–based opportunities are missed or compromised. Captivity cannot be cured for those fated to a lifetime without freedom. The result is a plethora of traumatized individuals that enter the veterinarian offices and sanctuaries often labeled with "behavioral problems."

Subsequently, the task of healthcare professionals and caregivers is to create social and ecological habitats that support psychological recovery and well-being. In households and sanctuaries where caregivers are attuned to the psychosocial and ecological needs of parrots, many birds will be able to forge a reasonably fulfilling life. However, others will be plagued by trauma-related symptoms. It is critical that healthcare providers educate parrot guardians as to the underlying causes of behavioral "problems" as symptoms of PTSD and related disorders. Parrots in captivity are responding to experiences of trauma and conditions of chronic stress whose symptoms may reveal overtly (eg, biting, self-mutilation) or covertly (eg, stress-mediated immune disorders). We are all ethically compelled to aid birds negotiate the psychologically demanding life as captives.

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Frustrated by Parrot Behavior? Ten Common Training Mistakes

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Session #125

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Abstract: Many caregivers are facing the same parrot behavior problems, such as screaming for attention fear responses, or aggressive behavior. While there is quality information available to facilitate resolving behavior problems, sometimes this is not enough. Observing the owner and the bird interacting during the problem behavior might reveal a need for additional detail. This paper will explore the 10 most common mistakes caregivers make when trying to address a parrot behavior problem using positive reinforcement training.-

Introduction

Questions pertaining to parrot behavior problems are typically presented via an email, a phone call or an in-person discussion. When reviewing the details of the behavior problem there is usually a fairly clear positive reinforcement solution to the problem. However when the caregiver returns home to work with the bird, it is discovered words of wisdom are just not enough. Observation of the owner and the bird interacting during the problem behavior usually reveal more detailed information is required. While each problem and parrot is an individual and each path to a solution may vary, there are mistakes that caregivers commonly make when trying to address parrot behavior problems.

The following is a list of the 10 most common mistakes caregivers make when trying to fix a parrot behavior problem using positive reinforcement training.

1. Asking For Too Much Too Soon

When training new behaviors, one highly successful approach is to shape the behavior with approximations. This requires identifying a goal behavior, describing what the behavior should look like, then creating a series of small steps or approximations that the parrot can easily accomplish to get the desired result. Typically this approach works extremely well for most people. However when a behavior is particularly challenging these approximations often need to be refined into even easier accomplished steps. For some this can be difficult to visualize. The result is that the caregiver becomes frustrated as the parrot seems unwilling or unable to present the desired behavior. Often the parrot is labeled as stubborn, difficult, dominant, etc. However the likely cause of the problem is that the bird has either not learned what is required, or has not had enough reinforcement history to understand there is value in presenting the approximation toward the desired behavior. As stated by professional dog trainer Peta Clark, "You can be sure that shaping with the smallest approximations is what is behind the most impressive behaviors."[1]

Instead of assuming the bird is unable to learn, or choosing deliberately not to participate, it is helpful to ask "Are the approximations just too big?" In most situations this is the primary problem holding caregivers back from achieving their training goals with their parrots.

2. Expecting the Parrot to Know What You Want

The mistake of expecting the parrot knows what the caregiver wants ties in with asking for too much too soon. It also speaks to motivation and reinforcement history. In some cases it is assumed a parrot knows a behavior because the bird has presented it in the past, therefore it must be trying to "play games" or avoid responding just to annoy the caregiver. Although no one can truly know what a parrot is thinking, it is unlikely the bird has a goal to annoy the caregiver. Furthermore it is not relevant to finding a solution to the problem. Instead noncompliance can be a signal that the consequence for the desired behavior does not hold enough value for the bird compared to the undesired behavior.

When a parrot does not comply, rather than assume it knows what to do, caregivers can have success by going back to basic training strategies. When a parrot is unwilling to present a behavior, the first choice is to ask for a smaller approximation and rebuild the behavior from there. Reinforcing the desired behavior, often heavily, can be critical to getting back on track.

For example a parrot may have a long history of entering a crate for transport. However if the bird chooses not to enter the crate, one appropriate solution would be to use a target to cue the bird to take a step towards the crate. This could be followed by another approximation of placing 1 foot in the crate, followed by 2 feet. The next steps could include cueing the bird to walk towards the back of the crate. Each approximation results in desired positive reinforcers. Usually within one session a behavior that was considered "lost" is recovered.

3. Using Force to Get Behavior

Coercion unfortunately is still quite prevalent in animal training. Parrot literature still recommends training methods that rely on negative reinforcement to create behaviors, or the use of positive punishment to address undesired behavior. These methods usually involve the use of an aversive to gain compliance. The fallout from the use of these methods usually includes fear responses and/or aggressive behavior. We react to coercion by avoiding or escaping from our coercers if we can.[2] Being that most people acquire parrots with the intention of building a relationship with the animal, coercion presents a giant roadblock to attaining that goal.

Because coercion has been generally accepted for so long in parrot training, many caregivers have difficulty recognizing when force is being applied. A small push into the chest of a bird to get it to step up is considered innocuous, when in fact it can be quite trust depleting. The fear of hands so many parrots present is the direct result of hands being used to force birds to participate. Caregivers can have great success with parrots by learning to notice the smallest changes in body language that indicate a parrot is uncomfortable. This may include the slightest attempt to escape or body language that is typically associated with aggressive behavior.

For example many people are challenged by harness training. Caregivers would like to see their parrots wear a harness. Often the harness is applied to the bird despite body language that indicates the bird is uncomfortable. The next time the harness appears the bird flees immediately. Because of the past coercion the difficulty in training this behavior has been increased tremendously. For parrots that are not comfortable with touch, harness training is a behavior that usually requires very small approximations and the absolute avoidance of coercion. The slightest attempt to push past a birds comfort level can create setback after setback.

However what many fail to realize is even the less challenging behaviors are damaged by coercion. Every time a parrot is forced back into its cage, forced onto a hand or shoved into a kennel it can cause the bird to be less

likely to voluntarily present that behavior in the future. In addition it damages the relationship between the parrot and caregiver. Caregivers can make great strides in training by becoming more aware of coercive methods and avoiding them.

4. Forgetting to Reinforce Desired Behavior

This is an extremely easy mistake for caregivers to make. We often take for granted that our companion animals should behave. There is a misconception that parrots should obey a "step up command" or any other command for that matter. In the world of positive reinforcement training commands do not exist. Instead animals are cued to present behavior. If the behavior is presented and if the resulting consequence is a positive reinforcer the behavior will be presented again in the future. The presentation of behavior is contingent on the consequence. If the positive reinforcer is withheld it is understood that eventually the behavior would extinguish itself. Caregivers should ask themselves of what value is it to the parrot to present the requested behavior? A lack of a desired consequence is a very valid reason for a parrot to choose not to participate.

The simple solution is to remember to reinforce every time a parrot is cued or requested to present a behavior. This includes behaviors essential to daily management such as stepping up, stepping down, returning to the cage, etc. The reinforcers can range from food treats, attention, head scratches, access to toys and more. Developing a large repertoire of reinforcers also can help influence the bird's motivation to present behavior. Never knowing what reinforcer might be received can help keep a parrot more interested in presenting behaviors when cued.

5. Blaming the Parrot

While it is true a parrot may have presented a problem behavior (such as biting), identifying the parrot as the culprit often leads to non resolution of the problem. When caregivers take the approach that modifying their own actions can influence the parrot's actions, they discover they have the potential to find solutions to behavior problems.

Parrots are not inherently well-behaved or misbehaved. They are a product of their life experiences. Therefore parrot behavior, good or bad, is often the result of learning. Usually this learning involves interactions with caregivers and resulting consequences. For example if a parrot owner uses coercion to attempt to teach a parrot to step up, it is quite likely the parrot may present aggressive behavior as a means to express its displeasure with the force. If the aggressive behavior causes the hand to be removed the bird has learned biting/lunging when a hand is presented causes the hand to go away. However using a positive reinforcement approach to training, the same bird can learn that taking small steps closer to hand can result in preferred treats. Depending on the experience a parrot has, he can learn hands are to bite or desired consequences happen when you move towards a hand. This is all entirely based on the choices caregivers make when training their parrots.

6. Believing the Problem Is an Internal, Permanent Condition

Many parrot behavior problems are frequently blamed on hormones or genetics. This ties in with mistake #5 Blaming the Parrot. When we give ourselves permission to blame the problem on an internal state or inherent quality of the parrot, we place ourselves on a path of non resolution of a problem. While hormones and species tendencies may influence behavior, they do not have exclusive power over the behavior problem.

For example a mature parrot may show aggressive behavior towards people who approach his enclosure. It is possible the parrot may be presenting behavior that is comparable to territorial aggression which is usually observed near nest sites. Even though this behavior may be naturally occurring in wild parrots, it can be modified. Caregivers can remove any materials in the cage that simulate nests or encouraging nest building, such as cardboard boxes, enclosed areas, or shreddable toys. The diet can be evaluated to ensure the fat and protein content is not exaggerated and contributing to a tendency to nest. The light cycle can be changed to simulate the cycle typically seen during the non breeding season for that species. Caregiver interactions with the bird can be monitored and altered so that behaviors that are involved in courtship and reproduction are not being reinforced. This may include the caregiver removing themselves from interactions if the bird is regurgitating, panting, or soliciting touch near the tail or cloaca. This response is a negative punisher (also known as a time out from positive reinforcement.) It is meant to decrease the presentation of the undesired behavior.

The caregiver should also plan on reinforcing heavily any behaviors the bird presents that are not related to courtship or sexual behavior. Via these strategies the bird can learn that sexual behaviors cause the caregivers attention to be removed and other behaviors will result in desired attention.

If aggressive behavior persists, caregivers can look at training alternatives to teach the parrot to voluntarily leave the territory when cued. For example instead of asking the parrot to step up onto a hand, a target can be used to teach the parrot to step onto a stick or play stand on wheels. This can then be moved away from the territory to a place where the bird seems less likely to present aggressive behavior. Utilizing all these strategies the formally labeled “hormonal mature male Amazon parrot” can learn to behave in a manner more acceptable for the household.

An interesting observation is that many male Amazon parrots who reside with experienced positive reinforcement trainers do not seem to follow the long held prediction of becoming aggressive upon sexual maturity. Again how one addresses behavior problems when they arise is far more important than the genetics of the animal. As more people learn to skillfully apply positive reinforcement training strategies in their homes, these examples may continue to grow.

7. Expecting Someone Else To Fix It

Because behavior is forever influenced by its consequences, one quick session with a consultant or trainer is usually not enough to change a behavior problem. While valuable information can be gained from a behavior consultation, the key to success is the follow-up in the household. Caregivers should expect to implement information they learn from a consultant every time they interact with their birds. In many cases, the consultant may not be there when the undesired behavior is presented. What happens in those moments the behavior is presented is what will determine what happens in the future. Therefore the caregiver’s role is critical.

Even an extremely well-behaved parrot is under the influence of its life experiences. A new bird may arrive with an excellent repertoire of well-trained behaviors. However training is never done. Parrots will continue to learn for the rest of their lives. Just because a behavior was trained once doesn’t mean it “sticks” forever. Every behavior caregivers want to see repeated needs to be reinforced. Behaviors that are undesired should go unreinforced and/or redirected to acceptable behaviors.

Consultants should be viewed as a source of information to help caregivers apply positive reinforcement training strategies in their homes, as opposed to quick fixers of parrot behavior problems.

8. Working with a Parrot When It Is Not Motivated

In most cases when a parrot is cued to present a behavior the expectation is to have an immediate response. However many parrots learn to pair slow response with the presentation of the behavior. This is truly an example of a problem that is the result of training. It is usually the result of training a parrot when motivation is low. The parrot may understand what behavior to present, but has little motivation to do so. Therefore the presentation may be slow. When the bird does finally present the behavior, it is reinforced. This pairs slow response with presentation of the behavior followed by a reinforcer. In essence the trainer teaches the bird to be slow.[3]

When training a behavior, part of the criteria should be quick response. An animal is more likely to present quick response when motivated for the reinforcer. Early in the training of a new behavior a trainer can make quick response a priority. This is just one reason that timing training sessions for when a bird is motivated is important.

Another reason to work with a parrot when it is motivated for the reinforcers is to avoid creating other undesired behaviors such as aggression or fear responses. If a caregiver feels compelled to push for more from a bird that is not interested, the parrot may respond with aggressive behavior or fear responses. This can cause setbacks in progress towards training goals. For example, many people are eager to get their parrots to interact with a towel. It is often tempting to push for more when training this behavior. If a bird is not responding well to reinforcers and the trainer pushes for another step in his approximations, the result may be enough of a fear response that the trainer will have to go back several approximations and rebuild the behavior. Experienced trainers aim to end the session before interest in reinforcers has diminished greatly.

In general if caregivers are frustrated that a bird is not responding rather than push for more behaviors they may want to evaluate the birds level of interest in participating and consider rescheduling training for another time when the bird is more motivated.

9. Waiting Too Long to Address the Problem

For each parrot behavior problem there is a moment at which the behavior was first presented and reinforced. That first moment of reinforcement can lead to that behavior being presented again. Very quickly the reinforcement history can build to the point that a very annoying problem has developed. At this point, concerted effort may be required to actively reduce the problem behavior and increase other acceptable behaviors.

A great way to avoid such a scenario is to respond the moment undesired behavior presents itself. When a problem behavior is first noticed, that is the moment to respond. A problem behavior should immediately be a signal to take action. The action taken will depend on the problem. For example, if a caregiver steps outside of a room and her parrot screams, she must make a decision in that exact moment on how she will influence that behavior. She may choose to freeze in her tracks, walk away from the bird, etc. If the bird makes a desirable sound, she can choose to immediately give the bird lavish amounts of attention. In this case she would likely influence the bird to present a behavior (other than screaming) to gain attention.

However if she chooses to walk into the room to deliver breakfast anyway because she is running late, she may reinforce the behavior. If this happens on an intermittent schedule (she reinforces the behavior on an unpredictable schedule), the screaming behavior may become very persistent. By paying attention to the exact moment problem behavior occurs and making choices in that moment, caregivers can address behaviors problems as

they arise. Very quickly a parrot can change its behavior onto an acceptable path. This can help caregivers create well-behaved birds gradually and with little effort, instead of backtracking to focus on specific problems that have developed over long periods of time.

10. Getting Stuck In The Past

It is difficult for many caregivers when a parrot's behavior changes dramatically. In many cases the focus is on the way the parrot used to behave as opposed to how the parrot behaves now. There are many reasons why a parrot's behavior may change. Sometimes finding a reason for the change can be helpful, however sometimes that information is not available or obvious. In either situation the starting point for resolution of the behavior problem is up to the parrot.

For example if a parrot suddenly shows a fear response to a family member, it does not help the situation to force the parrot to participate in a manner it may have been receptive to in the past. To rebuild the relationship the family member may need to physically be at a distance from the parrot in order for the parrot to present body language indicative of comfort. Once that starting point is established, a training plan to gradually reintroduce the person paired with positive reinforcers might be implemented. Over time the bird can learn the presence of that family member results in desired consequences. This can evolve into more interaction as trust is rebuilt.

The relationship may not return to exactly how it was prior to the behavior change and that is often difficult for some parrot owners to accept. However with some creative thinking, caregivers may find themselves developing new ways to interact with their parrots that offer a different but rewarding relationship.

Conclusion

There is a tendency to focus on parrots and problem behavior. However there are many people who live successfully with parrots. This is not the result of good genetics, smarter birds or just extremely tolerant caregivers. It is the result of the choices people make when they interact with their parrots and what they do when a behavior problem presents itself. The behavior of our parrots, good or bad, is most often the results of our own actions. By choosing to adhere to a positive reinforcement approach to parrot learning and avoiding the common mistakes discussed in this paper, caregivers can look forward to a problem free household and a rewarding relationship with their parrot based on trust.

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Multidisciplinary Approach to Feather Destructive Behavior

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Session #130

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Abstract: Feather damaging behavior is a difficult problem to diagnose and treat, in part because of the many factors in its etiology. The lack of foraging opportunities, medical factors, and stressors can all play roles. Differentials may fall into inflammatory skin and feather disease categories and those that concern non-inflammatory skin and feather diseases. Hormonal influences, especially those in reaction to stress, may play a role. Feather damaging behavior may also be associated with the reproductive cycle. The model of primate self-injurious behavior persisting despite alternations to environment and treatment may mirror the problems experienced when treating companion birds for feather damaging behavior. A multidisciplinary approach to feather damaging behavior, assessing the bird from the behavioral perspective as well as the clinical, can be of significant value in treatment. Applied behavior analysis is designed to help the clinician understand the context that supports the behavior problem, and a functional assessment of problem behavior provides the basis for building a behavior change plan. Targeting existing alternative or new behaviors and redesigning the environment to promote and maintain them is one component to treatment. A combination of behavioral change and clinical medicine may help to reduce the expression of feather damaging behavior.

Introduction

Feather damaging behavior (FDB) is a difficult problem to diagnose and treat. For this reason, a multidisciplinary approach may be needed to assess the problem as well as provide a variety of modalities to treat the problem. At this point, treatment effectiveness so that the feather damage is resolved completely may range from failure to moderate success. Commonly, success is limited at best. To provide improved diagnosis and treatment, a multidisciplinary approach may enhance clinical outcome.

Feather damaging behavior has been given a variety of names, including feather pecking, feather picking, and feather plucking. Feather pecking should be used for the condition where 1 bird plucks another, a common problem with battery-caged chickens.[1] It is assumed that in this condition, normal foraging behavior of pecking at substrates on the ground is prevented and the pecking behavior is then redirected at the feathers of conspecifics. Feather plucking implies the removal of feathers by "plucking out," which may or may not occur as some just shred the barbs and leave the scapus intact.[2] Feather picking is to pick at the feather, which suggests that only the barbs are picked. Therefore, it may be best to describe this behavior in companion birds as feather damaging behavior (B. Speer, oral communication, February 2003) and to describe those birds mutilating as a separate condition.

In chickens, feather pecking can range from gentle nibbling to vigorous pulling and may lead to the death of the bird. There have been a variety of factors suggested that may contribute to this condition, including a genetic predisposition. Housing conditions including battery cages and stocking density have also been implicated.[3]

Work by Huber-Eisher and Weschsler[4] also found that foraging opportunities reduced the development of feather pecking in chicks, particularly when they were provided straw at an early age. They also observed that the quality and availability of foraging materials influenced feather pecking in chickens.[5]

Foraging and FDB

An inherent behavioral tendency to forage may play an important role in feather damage in companion birds. Snyder et al[6] documented that Puerto Rican Amazon parrots (*Amazona vittata*) spend approximately 4–6 hour per day foraging and they routinely travel several miles between sites. In contrast, companion birds (orange-winged Amazons, [*Amazona amazonica]*) spend approximately 30–72 minutes per day eating a pelleted diet[7] while sitting on a perch. They do this without traveling, manipulating food items, and not attempting to balance their own diet as they would in the wild.[8] It has been suggested that parrots in the wild may be highly motivated to forage and this behavior is present in companion parrots as well. When foraging opportunities are not present, then abnormal behaviors such as FDB may develop.

Orange-winged Amazon parrots that were parent-raised were placed into individual cages at weaning.[8] These parrots were divided into 2 groups. The control group did not have any enrichments while the enriched group received alternating foraging enrichments with physical enrichments. Feather damaging behavior declined over time in those birds provided with enrichments and changes were first noted after 2 weeks but more apparent at 16 weeks. In addition, this study demonstrated that parrots in the enriched condition worked to access supplemental food items an average of 19%–26% of their active time, even though their normal ration was available as a pellet. This would support the findings in chickens[3–5] that in a barren environment and/or one without foraging opportunities, foraging attempts may be redirected towards their plumage as with FDB. The quality and the availability of the foraging enrichments in particular were suggested to contribute to the effectiveness of the response to FDB.

Other factors may also be involved, as well.[8] Foraging may be important for neural development in parrots. A strong relationship was also found between the lack of behavioral opportunity and the development of stereotypic behaviors in parrots, with evidence that there was an underlying neural compromise with stereotypy.[9] An environmentally induced neural dysfunction may underlie these 2 abnormal, repetitive behaviors.[8] The observed rapid change in feather quality with the addition of the enrichments suggests that enrichments can influence neural function. Treatment with psychoactive drugs can also result in rapid improvement in feather quality.[2,10] However, improvements with drug treatment appeared to be transient and long-term administration is fraught with significant problems with side effects. Clinical impression suggests that there is no silver bullet and that this problem of FDB waxes and wanes with treatment so that multimodal therapy may provide enhanced success.

Medical Factors and Stressors in the Development of FDB

Feather damaging behavior may be a consequence of medical factors and stressors that induce possible over-grooming. From a clinical perspective, it is important to determine first if there is true FDB or if the feather changes are a consequence of altered feather condition from a medical problem. Once FDB is established as the .problem, then it must be determined if this results from infectious agent(s) or not. This may be determined, in part, through the history and clinical examination. It is important that the clinical examination includes both a physical examination and a behavioral evaluation. There are 2 important aspects to the physical examination. The first is to document if there are physical changes that can be observed. The second is to determine the ex-

tent of the changes observed. Documentation of these physical exam findings help to determine if there are any infectious or metabolic changes that produce a secondary behavioral condition. A feather scoring system has been developed and can be adapted for clinical use.[11]

After the physical examination, one can narrow the differential list into 1 of 2 possible but unfortunately overlapping general categories for diseases that affect the integumentary system.[12,13] These would be inflammatory skin and feather diseases and non-inflammatory skin and feather diseases that are often assumed to be noninfectious. Feather damaging behavioral conditions could be a subset of either of these 2 categories. Inflammatory skin diseases include viral, fungal, bacterial, and parasitic infections. Viral diseases that affect the integumentary system of psittacines birds include papillomavirus, polyomavirus, avipoxvirus, and psittacine beak and feather disease virus or avian circovirus. Recently, it has been hypothesized that avian bornavirus (ABV) may play a role in FDB and/or self-mutilation. Bornavirus tends to be neurotrophic.[14] It is unclear if replication of the virus in nerves or ganglia and/or the production of G proteins as a consequence of the virus[15] results in neurogenic pain, causing the bird to disrupt their feathers or to mutilate the area that provides innervation to the skin in its dendritic zone.

Dermatitis

Bacterial dermatitis of a primary origin is not common in bird and seems to less commonly induce FDB. This reduced incidence of dermatitis from a bacterial and/or a fungal origin may result from bacteriostatic and mycostatic compounds within the epidermal lipids of the skin, making birds less susceptible to infection. *Staph. aureus* is the most common isolate and is considered to be part of the normal flora. Fungal dermatitis is also considered uncommon in birds as a primary condition. Fungal infections have been described as chronic and are often confined to the neck and head. External parasites often disrupt the feather barbs by chewing of the mite or lice but it is not self directed. This may be difficult to distinguish and is why the history and PE is very important to rule in or out these organisms.

Diagnostic tests are indicated to rule in/out medical problems including infectious disease agents as part of the cause. However, the shotgun approach to diagnostic testing is not warranted either. Each approach should be tailored to the individual patient and its circumstances. Other medical problems associated with FDB include allergies; pain, possibly from trauma, organ failure, or from osteoarthritis; abnormal hormone levels and misdirected reproductive responses secondary to altered hormone issues. Trauma includes several types of conditions including tail trauma, other traumatic conditions to the skin, and damage to blood feathers[12,16] and this trauma may be responsible to induce picking. It is unclear why birds may self pluck with organ malfunction but may be similar to referred pain of mammals. In mammals, the pain experienced on the body wall is the consequence of organ pain that may or may not be from the same spinal cord segment.

Allergies

Allergies have also been suspected as to play a primary or a secondary role in self-inflicted damage. However, the information presented to date is inconclusive and further research is needed to understand allergies and their role in FDB.

Hormonal influences

A number of psittacines with FDB were observed to have lower than normal thyroid levels, suggesting that there may be hormonal influences.[17] While changes in thyroid levels may be linked to FBD, it may be a consequence of changes in corticosteroid levels or altered hypothalamo-hypophyseal responses due to stress. Exogenous cor-

ticosterone can disrupt behaviors associated with breeding, rearing of young, and territoriality in several species and this effect is independent of the HP-gonadal axis.[18] Corticosterone levels influence learning-induced neural plasticity, as they have been found to positively influence long-term avoidance behavior in chicks.[19] These factors are important as we attempt to understand FDB in relation to neural function and control.

HPA Axis and self-injurious behavior

The fact that primates that are handreared from an early age are more prone to self-injurious behavior (SIB) and differ behaviorally and physiologically from normal is disturbing for us as avian clinicians. In the study on self-injurious behavior in primates,[20] maintenance of the injurious behavior persisted or recurred despite attempts to change the environment. This suggests that the SIB confers some benefit to the individual. In humans, self-wounding may abate the escalation of negative emotions[21] and the act of SIB is followed by an immediate sense of relief.[22,23] Tension reduction as recorded as a drop in heart rate post trauma occurred in the monkey model.[20] Researchers found that the heart rate escalated significantly above baseline 30 seconds prior to the biting episode, remained elevated during the bite, and then dropped precipitously to baseline in the 30–60 seconds after the episode was completed. This suggests that biting acts as a physiologic positive reinforcer for an abnormal behavior. Biting's lack of responsiveness to alterations in the environment and to treatment is suggestive of the problems experienced in the response to treatment of companion birds for feather damaging behavior.

Rhesus monkeys with SIB had a decrease in responsiveness to ACTH stimulation and had lower resting cortisol levels. Interestingly, urinary levels were normal. It has been suggested that the low resting cortisol combined with the other physiological attributes in monkeys with SIB may be similar to post-traumatic stress syndrome in humans. These data suggest that exposure to early stresses out of the normal nest box stressors somehow resets the HPA axis and, that once the disorder develops is resistant to treatment.

Reproductive cycle

Birds may also have FDB associated with the reproductive cycle. Use of a small cage, covering the cage at night or for part of the day and long periods of time may suggest to the bird that it acts as a nest box to stimulate reproductive behavior. This may increase stress and other factors so that the bird may pluck an area around the vent which in some species is an incubation patch. Other areas may be affected related to the species. Conure hens often pluck on the lower part of the chest for example.

Lupron, a GNRH agonist, has been shown to reduce FDB in those birds where reproductive behaviors have been expressed as part of the medical history. This agonist may well play a role in the HPA axis that may influence neural processing and behavior in other and currently unknown ways. It is important that avian clinicians gain as much knowledge as possible though the history and clinical examination to help piece the puzzle together.

Functional Assessment

Part of the multidisciplinary approach to FDB is to assess the bird from the behavioral perspective as part of the clinical exam. The behavioral level of analysis is the level at which observable behavior and observable conditions act upon one another. Applied behavior analysis is designed to help the clinician understand the context that supports the behavior problem. The goal is to change the environment so that an alternative and more desirable behavior is supported instead. This technique offers a framework for the clinician to use to assess and develop a treatment plan for the particular patient.

The first step is to observe and define the problem or target behavior, in this case is self-directed feather damage. The next step is to determine the distant and immediate events or conditions that precede the problem behavior. These preceding events, called antecedents, are predictors of the behavior. For example, when, where, and with whom is the problem behavior more likely to occur. And then the flip side is equally important, when is the patient most successful or when does the problem *not* occur? Then the clinician needs to identity the consequences that maintain the behavior or what does the bird gain by behaving this way? Reinforcers can be tangible, social, sensory, and escape. This may be a response from the clients or an inherited behavior of the bird. In this scenario, it is important to determine if the functional assessment and the behavioral outcome have a medical component and how much that affects the problem behavior.

The outcome of a good functional assessment is four-fold: First is a description of the problem behavior in unambiguous, observable terms. Second is the identification of the general and immediate physical and environmental conditions in which the problem behavior is likely to occur, and also the conditions under which it does not occur. Third is information about the purpose the behavior serves the animal, or the outcomes or consequences that reinforce (ie, maintain or increase) the behavior. Fourth is the development of a summary statement in which the information is combined. The functional assessment of problem behavior provides the basis for building a behavior change plan, which entails targeting existing alternative or new behaviors and redesigning the environment to promote and maintain them. From the identification of these 4 conditions, a summary statement that describes the relationships among the antecedent predictors, the behavior, and the consequence for each major situation in which the behavior occurs should be formulated. It should be added to the medical record and provided to the owner so that everyone is clear and has a basis of operation.[24]

Developing Treatment Plans

In formulating the treatment plan for attempting to reduce FDB, this behavior change plan should also include how the physical and social context of the environment will be altered to enhance an effective change. Redesigning the environment also will require the patient and the owner and those that interact with the bird to learn new skills to make the FD behavior less likely to occur. This behavior support plan should spell out how the environment will be redesigned to promote and maintain an alternative or replacement behavior. This plan may include medications that you prescribe, dietary changes, foraging and enrichment, exercise, training sessions, changes in the physical environment, schedule changes, and what and how the owner and others will use rewards and punishers. Some birds, for example, that are sensitive to the cage as a nest box may need to have more time out with more exercise. Showering frequently may also reduce the desire to breed as most psittacines do not nest during the rainy season.

When discussing the bird and its plucking, most owners are not aware of the immediately preceding event nor the consequence that ensues, as it is important to determine if there are immediate events that contribute to feather damage. In dissecting these important components that make up the functional assessment, giving examples are helpful along with having the owner provide video clips of the "situation" that they think is occurring. It may take a second or third visit, armed with these new pieces of information and short video clips to literally write out the distant and immediate events that precede the feather damaging behavior and the consequences that maintain it. Once the owner is clear that these 3 components of the functional assessment are defined, then the behavioral treatment plan can be formulated.

The behavioral treatment plan should have a clear view of what the desired behavior is and methods to achieve this goal. It should not focus on what the bird should not do (ie, not chew its feathers). To get to the desired behavior (such as manipulating food linked to other "toys" and foraging), the old problem behavior should

become irrelevant, ineffective and inefficient. This is particularly important with feather damage from stressors in the home environment and difficult to accomplish. As seen in the studies by Meehan and Mench and those in primates, self-damage may be reinforcing. Studies in orange winged Amazons indicate that with the introduction of foraging, feather scores improved but did not return to normal feathering. While it did improve feather scores, it took weeks for changes to occur. Therefore, the owner must realize that the feathers may not return to normal and that it takes time to observe changes. However, not all birds suffer from self-induced feather damage that is rearing induced. Because there are many reasons that FDB occurs, it is important to work through the history, exam, and a functional assessment.

The functional assessment plan should fit into the abilities of the caregiver's skill level and their ability to provide the environment to meet the needs of the behavior change. An effective plan should help the avian patient while positively enforcing the caregivers for a better outcome from both a short- and a long-term perspective. Strategies should be based on the most positive and humane while being the least intrusive. This often involves successive steps to get to the desired goal using differential reinforcement or shaping.

There are preliminary strategies that can serve as the framework for developing the treatment plan. The question is: Can I do something different or change something in the environment so that the behavior doesn't occur in the first place? The options could be -are there adjustments that can be made related to *when* the problem behavior is likely to occur? It may be that these adjustments could be made that are related to *where* the problem behavior is likely to occur. Adjustments could be made that are related to the *activity* that is occurring when the problem behavior is occurring. It may be that you should consider changing the *people* present when the problem behavior is likely to occur.

One important and often successful technique is to teach/re-teach a behavior to replace the problem behavior. A common example is to replace biting with a "lean away" from the bird. This helps the owner understand the subtle behaviors of our common companion birds. Foraging is one area that occupies the time budget of a companion bird while providing part of its normal behavioral repertoire. It is one addition to the treatment protocol for most companion birds. An area that often needs adjusting is the bird's environment. By adding, removing or altering a(n) item(s) or conditions of the past. For example, conures often go into breeding behavior when kept in a small cage with a cover at night for it replicates the social context of a large nest box to them. The owner may not know that the bird is going into breeding behavior but interprets the action as " the bird is more bitey, particularly when trying to get the bird out of the cage, and the bird is screaming more." Getting them out, exercising them, and not providing a night cover help reduce the conditions for breeding.

Birds that are caged in home environments need to express some of their innate behaviors to improve their welfare. Foraging for food is a natural behavior that occupies a large portion of their daily time budget of our commonly kept companion species. Additionally, birds in the wild are athletes that expend a considerable amount of energy to fly. Our companion psittacines have evolved in a highly social environment and therefore have a relatively large forebrain for complex cognition. For these reasons, the avian clinician needs to address all of these issues when designing a treatment plan for changing a problem behavior to one that is more socially acceptable replacement behavior.

Following is a treatment design plan that can be modified for the individual patient.[24]

Setting Behavior Targets and Designing a Behavior-Change Plan

After the functional assessment summary statements have been developed, the primary caregiver can answer following questions to plan the behavior-change program:

1. Replacement behavior: What existing alternative behavior would meet the same purpose for the animal?
    a. Rather than _____
    (Identify the problem behavior)
    b. This animal can _____
    (Identify the replacement behavior)

Example: Rather than biting my hand, this parrot can lean away.

2. Desired behavior: What behavior do you ultimately want the parrot to exhibit?
    a. When _____
    (Summarize antecedents)
    b. This animal_____
    (Identify desired behavior)
    c. In order to _____
    (Summarize "payoffs")
    Example: When I offer my hand, this parrot can step up, in order to get a ride to the play tree.
3. What has been tried so far to change the problem behavior?
4. Preliminary strategies: Can I do something different or change something in the environment so that the behavior doesn't occur in the first place?
    a. I could make adjustments related to WHEN the problem behavior is likely to occur by:
    b. I could make adjustments related to WHERE the problem behavior is likely to occur by:
    c. I could make adjustments related to the ACTIVITY during which the problem behavior is likely to occur by:
    d. I could make adjustments related to the PEOPLE present when the problem behavior is likely to occur by:
    e. I could teach/re-teach a behavior such as:
    f. I could adjust some aspect of the environment by adding, removing or changing an item or condition such as:
    g. Other adjustments that can be made are:
5. Training strategies: What skill(s) will the animal need to be taught in order to successfully demonstrate the replacement/desired behavior? _____
    a. Who will provide the training?
    b. When will the training take place?
    c. Where will the training take place?
    d. How often will training take place?
    e. How and how often will opportunities for practice be provided?
6. Reinforcement procedures: What will I do to increase the occurrence of the replacement/desired behavior?
    a. Identify potential reinforcers: What preferred items, activities, or people might be used as incentives in an intervention for this animal?
    b. Establish specific behavior criteria: What exactly must the animal do to earn the above reinforcers?
    c. Determine the schedule of reinforcement: How frequently can the animal earn the above reinforcers? Typically, continuous reinforcement is best (that is, a reinforcer for every correct behavior).
7. Reduction procedures: What will I do to decrease the occurrence of the problem behavior?
    a. I will ignore all occurrences, immediately attending to something else, by:
    b. I will stop and redirect each occurrence of the behavior by:

c. I will implement time out from positive reinforcement by:
d. Other strategies:

8. Implementation details: What other details or explanations would help another person implement this plan accurately and consistently?
9. Tracking change: How can I monitor the animal's behavior so I have a reliable record of progress and can continue or modify the plan as needed?
    a. Describe exactly how data will be collected and recorded.
        i. Frequency count of the target behaviors across the day.
        ii. Frequency count from ___:___ am/pm to ___:___am/pm
        iii. Timing duration of target behaviors.
        iv. Other
10. Evaluating outcomes: This program will be considered successful if what outcome is achieved by both the animal and the caregivers, under what conditions?

In performing a case work-up with a functional plan the outcome may range from determining that the plucking is a pain response to an area and its treatment is purely medical to the other extreme where the patient responds to changes in its environment and through redirected behaviors, the self damage is reduced or extinguished. For example, a female maroon-bellied conure (*Pyrrhura frontalis*) that was presented for FDB of the caudal abdomen was assessed both from a medical and functionally.

Based on the history the antecedents were: the owner had caged the bird more recently due to a change in job, so covered the cage more to try to get more sleep as the bird vocalized more. The owner had given the bird boxes to chew to keep her occupied. The behaviors were those that are observed commonly during breeding: vocalization, chewing and plucking around the vent for a brood patch. The consequence is the behaviors are expressed with normal breeding activity and occurred in the natural time of year for breeding of conures. The summary statement would be: When I set the stage for breeding by providing that type of environment, the pyrrhura conure goes into its normal breeding mode that includes FDB as a consequence of its environmental conditions.

The treatment design plan for this patient was based on changing the environment away from that that tends to reinforce nesting or breeding behaviors to those that are self maintenance thereby changing its time budget. The owner had a friend get the bird out of the cage more during the day. In addition, the conure was exercised several times a day by flying in the house. The cage was not covered. There was no chewing without the purpose of feeding so foraging became her mode for obtaining her food on a daily basis. This further redirected her manipulating her feathers with her beak and tongue, part of the natural behavior of foraging. This precision beak and tongue manipulation is part of the normal and lengthy time budget of foraging as discussed in the first part of this paper. When there is reduced foraging and only an empty box to chew with other environmental cues associated with breeding, the hen may overpreen the caudal part of the abdomen. Changing its environment along with changes in the time budget were designed to redirect the need to manipulate feathers to its natural behavior of foraging. In addition, the bird was showered daily to mimic the rainy season as many psittacines go out of lay with its onset. From the medical portion of the plan, depo Lupron was administered initially to enhance success of the behavioral change plan.

Other cases will be discussed in the masterclass to provide a framework for clinicians to use in not only their workup and treatment plan for birds that feather damage but as a basic model for other behavior problems.

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Section 2

Pre-conference Program: AEMV Scientific Program

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Efficacy and Pharmacokinetics of Selamectin in the Pet Rabbit

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Session #150

Summary Style Manuscript

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Selamectin is a broad-spectrum topical parasiticide that provides greater than 90% efficacy against fleas infesting dogs and cats for at least 30 days. However, while data is scant it is also being widely used as an external parasiticide in a variety of exotic mammal species, including domestic rabbits (*Oryctolagus cuniculus*). This study, therefore, was conducted to determine the efficacy and pharmacokinetics of selamectin against flea (*Ctenocephalides felis*) infestations on rabbits. While selamectin at both 10 and 20 mg/kg was > 91.24% effective against existing flea infestations on rabbits at 7 days, it did not provide for prolonged residual activity. In addition to the efficacy study, plasma concentrations of selamectin were determined with liquid chromatography and mass spectrometry. Based on the pharmacokinetics, a greater than proportional drug exposure, approximately threefold increase, occurred when the dose was doubled, suggesting dose independent absorption. Further increases in the dose may result in greater than expected plasma concentrations of selamectin in rabbits. The short duration of efficacy of selamectin was most likely due to the short terminal half-life after topical administration in rabbits (~1 day) compared to dogs (11.1 days) and cats (8.25 days).

Standardization of Renal Physiology Parameters in Guinea Pigs via Urinalysis

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Session #152

Summary Style Manuscript

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Introduction

Guinea pigs (*Cavia porcellus*) are considered by most veterinarians to be hystricomorph rodents originating from South America.[1] Some biologists, however, do not believe that guinea pigs should be included in the mammalian order Rodentia.[2] Whether taxonomically considered a rodent or not, they are closely related to the chinchilla, nutria, capybara, and porcupine.[3,4] Cavies originated in the grasslands of the Andes Mountains as a culinary delicacy.[4] Both domesticated and wild cavies are still kept in many regions of South America for food, companions, and good luck. They were brought to Europe in the 16th century and quickly became popular as pets.[3,4] They are used in research (Hartley and Duncan-Hartley strains),[4] kept as pets, bred, and shown by fanciers.

Guinea pigs have 2 retroperitoneal kidneys (right is cranial to left) surrounded by dense adipose tissue.[5,6] The renal pelvis is large and has a single longitudinal papilla.[1,5] Ureters drain urine from each kidney into the bladder and a urethra carries urine from the bladder. The urethra is longer, narrower, and less distensible in the male cavy, thus predisposing them to urolithiasis problems as well as obstructions due to coagulated seminal secretions.[5,6] The urine of guinea pigs is usually alkaline and cloudy due to the presence of crystals (calcium carbonate and ammonium phosphate).[1,5–9] Urogenital diseases are not uncommon in both male and female cavies[10,11]; therefore, diagnostic testing of the urine is an important aspect of health assessment. Although there are references to the general appearance of guinea pig urine,[1,5–9] there have been no published data related to normal (standardized) urine values in the literature. Much frustration has resulted from this lack of documented normal values when attempting to evaluate cavy urinalysis results. The goal of this research project was to determine normal values for urinalysis in non-laboratory, domestic, guinea pigs.

Materials and Methods

Urine samples were collected from 50 guinea pigs representing 8 different breeds (Table 1). Males and females were equally represented and ranged in age from 3.5 months to 4 years. All animals were fed the same standard alfalfa-based diet (Table 2) and housed under the same husbandry conditions. Each animal was placed into a clean plastic container with a plastic grate on the bottom to prevent contamination of urine samples. Samples were collected via sterile pipette immediately after voiding. Samples were immediately processed using reagent strips (N-Multistix SG/ Bayer), a refractometer for specific gravity assessment and sediment analysis. Each urine sample was examined grossly and microscopically, with and without stain (Sedi-stain). Urine samples were centrifuged at ~3000 rpm for 6 minutes prior to sediment analysis. Slides were evaluated for the presence of red blood cells, white blood cells, epithelial cells, renal casts, bacteria, and crystals. The average and standard deviation for each urinary test parameter was determined.

Table 1. Eight breeds of guinea pigs used in urinalysis study (total = 50).

American
American satin
Coronet
Peruvian
Peruvian satin
Silkie
Silkie satin
Texel
Mixed breed

Table 2. Diet used in all test subjects during test period.

Brand of feed	Type of feed	Percent of diet
Purina: Mazuri	Guinea pig feed	50%
Nutrena: Ringmaster	Rabbit feed (+probiotics)	50%
Washington State local	Grass hay	Free choice
Trader Joe (Darwin's)	Vitamin C crystals	15 mg/oz in drinking water (twice a week)

Results

Gross examination of the urine samples revealed cloudy urine in all test subjects. The urine color ranged from pale yellow to orange/tan. There was no visible blood in any of the test subjects included in the study (see discussion section for information regarding expressing the bladder to obtain samples). The pH of all urine samples was alkaline (Table 3). The average urine specific gravity, tested by refractometer, was slightly higher in males than females, 1.032 and 1.030, respectively. There was no correlation found between the specific gravity results from the reagent strips and the refractometer for each sample ($P > 0.05$). The following reagent strip chemistry results were all negative: leukocytes, nitrite, urobilinogen, ketones, bilirubin, and glucose. The blood reagent strip results ranged from negative, in 47/50 samples, to non-hemolyzed trace. The protein reagent strip results ranged from trace to 2000, with an average of 525 (mg/dl).

Results of sediment analysis revealed a range from 0 to 4 WBC/HPF. There were 0–3 RBC/HPF and 0–5 epithelial cells/HPF. Renal casts were rarely seen in test subject sediment analysis (0–2/HPF). All samples but 4/50 had visible bacteria in the sediment. Cocci were predominantly seen (47/50) and ranged from occasional numbers to too numerous to count. Three of the samples had bacterial rods in low numbers. Crystals were found in all samples: 45/50 had calcium carbonate crystals in moderate to large numbers. Twenty five of the samples had amorphous crystals. Phosphate crystals were found in 16/50 samples, ranging from only a few to too numerous to count. Calcium oxalate crystals were found in 3/50 samples in low numbers (Table 3).

Table 3. Urinalysis results for guinea pigs.

Test	Technique	Average	Range	Standard deviation
Appearance	Gross exam	Cloudy	Yellow–orange/tan	
Specific gravity	Refractometer	1.030	1.005–1.050	0.012848
Leukocytes	Reagent strip	Neg		
Nitrite	Reagent strip	Neg		
Urobilinogen	Reagent strip	Neg		
Protein	Reagent strip	525 mg/dl	Trace–2000	764.4442
pH	Reagent strip	8.44	6.5–8.5	0.333197
Blood	Reagent strip	Neg–NH trace		
Specific gravity	Reagent strip	1.005	1.000–1.025	0.006343
Ketones	Reagent strip	Neg		
Bilirubin	Reagent strip	Neg		
Glucose	Reagent strip	Neg		
WBC	Per HPF	0.9	0–4	0.995596
RBC	Per HPF	0.06	0–3	0.272431
Epi cells	Per HPF	1.16	0–5	0.84572
Casts	Per HPF	0.125	0–2	0.25
Bacteria	Per HPF	Mod number	None–TNTC	Cocci
Crystals		Many	Mod–TNTC	CaCarb
		Moderate	None–TNTC	Phosphate
		Rare	None–Few	Ca Oxalate

WBC = white blood cells; RBC = red blood cells; Epi cells = epithelial cells; HPF = high power field; TNTC = too numerous to count.

Discussion

Early in the course of this project, we attempted to obtain urine samples by gently expressing the bladder of guinea pigs that were reluctant to urinate. Every expressed urine sample had blood in it. These animals were tested 1–2 weeks later by voided sample and no blood was seen in their urine. For this reason, we recommend voided samples or cystocentesis rather than expressed samples. Cleaning the vent area with antimicrobial soap, rinsing well, and towel drying is recommended to help prevent contamination of samples. We also recommend analyzing the sample as soon as it is voided, if possible, to prevent contamination. Guinea pigs have a tendency to defecate in or near their urine, so timely retrieval of samples is important to prevent fecal contamination. All cavies produce cloudy urine with many calcium carbonate crystals. Our findings indicated that phosphate crystals were also common in guinea pig urine; however, calcium oxalate is seen only rarely in their urine. Although the reagent strips are very convenient and accurate, the specific gravity results are inaccurate and should not be trusted. A refractometer provides an accurate measure of urine specific gravity. Protein in the urine was found in all samples. This could be due to fecal contamination or other non-bladder related sources. Future studies will be performed to compare voided samples to those taken by cystocentesis (this will help prevent contamination related errors in analysis).

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Use of the Diagnostic Peritoneal Lavage in the Domestic Ferret

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Session #155

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Abstract: Effusive diseases of the abdominal cavity are not uncommon in the domestic ferret (*Mustela putorius furo*). Certain diseases, such as gastrointestinal perforation, often require laparotomy as soon as the patient is hemodynamically stable. A rapid diagnosis is essential for a positive outcome in these cases. The diagnostic peritoneal lavage (DPL) is a sensitive and specific diagnostic tool in human and veterinary medicine when insufficient ascitic fluid exists to obtain a diagnosis from abdominocentesis or ultrasound-guided aspiration alone. The DPL should be considered as a diagnostic tool for the diagnosis of hollow organ perforation, hemoabdomen, and metastatic abdominal neoplasia in the domestic ferret.

Introduction

The domestic ferret (*Mustela putorius furo*) is a popular companion pet and experimental research animal. Diseases involving the abdominal viscera are relatively common in this species. Diagnostic modalities to evaluate the abdomen that have widespread use in clinical practice include survey radiography, ultrasonography, and abdominocentesis. Computed tomography (CT) and magnetic resonance imaging (MRI) can also provide helpful diagnostic information but are generally less accessible to most practitioners and incur greater expense.

Abdominal fluid can be categorized as a transudate, modified transudate, or exudate. Modified transudates and exudates can be further divided into septic, inflammatory, hemorrhagic, and neoplastic fluids.[1] Septic peritonitis is caused by free bacteria in the abdominal cavity from perforating foreign bodies, erosive neoplasia, or gastrointestinal (GI) tract necrosis following obstruction, torsion, or introduction through the body wall. Liver, kidney, prostate, and uterine infections can also serve as a source of bacteria.[2] Chemical peritonitis can be caused by sterile irritating substances such as bile, urine, and pancreatic enzymes. Chemical peritonitis can lead to septic peritonitis.[2] The diagnostic peritoneal lavage (DPL) is a widely utilized diagnostic technique in human and veterinary medicine for the evaluation of abdominal fluid that accumulates with hollow organ rupture, abdominal hemorrhage, and metastatic neoplasia.[3] In humans, the DPL is a more sensitive diagnostic test than abdominocentesis, as less fluid is required for detection (1.0–4.4 ml/kg vs 5.2–6.6 ml/kg). In dogs and cats, the DPL has a far greater accuracy (94.6%) when compared with abdominocentesis (47.3%).[1,4] In dogs, at least 5 to 6 ml/kg of free abdominal fluid must be present in order to get a positive abdominocentesis.

The Diagnostic Peritoneal Lavage in Human Medicine

Until the 1960s, blunt abdominal trauma (BAT) in humans was primarily diagnosed by physical examination and radiographic findings. The DPL was first described in 1965, and to this day is the diagnostic test of choice for the diagnosis of BAT at some medical institutions. Many centers, however, have replaced the DPL with ultrasound (US) and abdominal CT. The relative sensitivity and specificity of DPL, US, and CT are still debated, but there are significant benefits, limitations, and risks for all 3 diagnostic modalities (Table 1).[3,5]

Table 1. Comparative benefits, limitations, and risks of diagnostic peritoneal lavage (DPL), abdominal ultrasound (US), and computed tomography (CT).[3,5]

Diagnostic modality	Benefits	Limitations	Risks
DPL	Most sensitive indicator of hollow organ rupture	Lacks organ specificity Cannot evaluate retroperitoneum	Most invasive of the 3 techniques
US	Noninvasive Rapid Easily repeated	User dependent	
CT	Evaluates the retroperitoneum better than DPL, US	Costly Not widely available Sensitivity and specificity less than DPL for hollow organ perforation	Requires hemodynamically stable patient

In human medicine, the DPL is predominantly used to evaluate penetrating abdominal injuries that may lead to hollow organ perforation or abdominal hemorrhage.[3] Intraoperative peritoneal lavage and DPL have also been described as diagnostic tools to screen for metastatic abdominal neoplasia, such as gastric carcinoma, prostatic carcinoma, biliary tract cancer, and colorectal cancer.

Three techniques in humans are described–open (surgical incisions though skin and body wall), closed (blind percutaenous catheter insertion), and semi-open (initial skin incision with blind insertion of the catheter through the body wall).[3] A number of catheters specifically made for performing the DPL in humans are commercially available. The immediate appearance of blood or the aspiration of 10 ml of blood or greater is a positive aspirate and laparotomy is indicated. In the adult, a standard volume of infusion is 1 L of warm, sterile isotonic saline. In the pediatric patient, a volume of 10–15 ml/kg is recommended. Adequate return is considered 30% of instilled fluid.[3] A positive result in the adult requires one or more of the following criteria[3]: 10 ml of whole blood or greater; WBC >500/µl; RBC >100,000/µl; and/or presence of enteric matter or the presence of bacteria in stained samples.

The DPL in Veterinary Medicine

In veterinary medicine, the definitive diagnosis of acute abdomen (acute abdominal pain, abdominal distension, collapse) can be challenging.[1] Often, patients are hemodynamically unstable and emergency laparotomy carries significant anesthetic risks. It is frequently difficult to determine if the benefits of emergency surgery outweigh the risks of anesthesia and a negative exploratory. The DPL is indicated when 4-quadrant abdominocentesis fails to yield a diagnostic fluid sample or when ultrasound cannot detect free fluid but persistent abdominal pain or fever suggest the presence of peritonitis.[1,2]

Prior to conducting abdominocentesis or DPL, the urinary bladder should be emptied. Survey radiographs should be taken first.[1] If possible, ultrasound should be done prior to the DPL, to evaluate for smaller fluid pockets that

can be aspirated with ultrasound guidance.[1] Although technically a bit more challenging than abdominocentesis, disadvantages of the DPL are few.[6] Caution is advised for patients in respiratory distress, as the fluid volume may put pressure on the diaphragm, and for patients with previous abdominal surgery and preexisting coagulopathy.[3,6] Potential risks include introducing or spreading infection, tearing the GI tract, and hemoabdomen.[1]

Technique

The technique for performing the DPL in the dog and cat has been described previously.[1,3,6] Animals typically require sedation for DPL. A combination of intravenous opioids and inhalant anesthesia can be used, depending on the medical condition of the patient. Patients are generally placed in lateral recumbency.[1] The skin 1–3 cm caudal to the umbilicus on the midline is shaved and prepped. The skin and linea are instilled with 2% lidocaine and a small stab skin incision is made. A catheter (18- to 20-gauge over-the-needle intravenous catheter, peritoneal dialysis catheter, or peritoneal lavage catheter) is inserted 6–10 cm into the abdominal cavity and directed caudodorsally. Catheters that lack a trocar or stiff needle may require a second stab or surgical incision through the body wall. If a needle or stylette is present, it is removed at this time and the catheter is aspirated. If no fluid is retrieved, 20–22 ml/kg of warmed sterile isotonic saline is instilled. The patient is rocked and/or the belly is massaged for up to 1 to 2 minutes. The fluid is then aspirated. Fluid recovery is variable, depending on the amount of peritoneal inflammation. Failure to obtain a significant volume is common, but it is also possible to recover up to 80% of the volume instilled. Re-instillation of up to half the original fluid volume can be performed. Systemically administered analgesia is warranted after the DPL.

Sample handling and interpretation

Samples should be collected in sterile transport tubes or culturettes for microbiologic culture and sensitivity.[2] Fluid samples should also be placed into EDTA tubes for cytologic analysis. A clot tube can be used for biochemical analysis. Fluid analysis for the DPL is performed the same as for abdominocentesis, although caution is advised in interpretation of cell counts and biochemical analyses since the sample will be diluted.[1,2] Normal peritoneal fluid is a clear fluid with a total protein of less than 3.0 g/dl and the total volume is typically less than 1 ml/kg. The total cell count is < 2500 cells/µl, with the neutrophil count at < 500/µl. Approximately 50% of cells are monocytes and 40% are lymphocytes.[2]

The presence of toxic or degenerative neutrophils with intracellular bacteria indicates septic peritonitis, and a laparotomy should be performed as soon as the patient is hemodynamically stable.[2] A false positive result for bacteria may occur if the GI tract is inadvertently entered during sample collection.[1] Abdominal effusions in animals with peritonitis are associated with higher lactate (dogs) and lower glucose than serum. A packed cell volume (PCV) of greater than 2% or RBC > 200,000 cells/µl indicates significant hemorrhage. A PCV of >1% but < 2% is equivocal, and a PCV < 1% is considered insignificant. If hemorrhagic fluid is aspirated, it should be evaluated for clotting. A false positive could occur if the spleen or a blood vessel is entered with the needle or catheter, and this blood will clot upon sitting.[1] Free, non-clotting blood is most consistent with trauma, ruptured splenic tumor, or coagulopathy.[6]

With urinary leakage (eg, bladder rupture), creatinine and potassium concentrations will be higher in peritoneal fluid than in the serum.[6] The presence of bilirubin is suggestive of bile peritonitis.[2] With pancreatitis, amylase and lipase will be higher in abdominal fluid. With chyloabdomen, concentrations of triglycerides and cholesterol will be higher than in serum.

Indications for Use of the DPL in the Domestic Ferret

The indications for which the DPL is used in other species also exist in the domestic ferret.[7]

Hollow organ perforation

Perforation of gastric ulcers: Gastric and duodenal ulceration have been reported in laboratory and companion ferrets. Reported causes include foreign body or toxin ingestion, and treatment with ulcerogenic drugs such as nonsteroidal and steroidal anti-inflammaties.[8] Gastric ulcers have the potential of rupture, which can lead to septic peritonitis.

Perforation of gastrointestinal foreign bodies: Sharp objects can perforate the GI tract and result in septic peritonitis.[9] Intestinal obstruction can lead to intestinal necrosis and rupture.

Perforation of gastrointestinal tumors: The most common neoplasm of the ferret gastrointestinal tract is lymphoma.[10] Gastrointestinal perforation and unexpected death have been reported with intestinal lymphoma.[11] Primary neoplasms of the GI tract tend to be malignant and aggressive, such as gastric and intestinal adenocarcinoma.[10]

Ruptured prostatic cysts and abscesses: Prostatic cysts and abscesses are occasionally seen in male ferrets that have adrenocortical disease. These cysts and abscesses can become quite large, often exceeding the urinary bladder in size. These structures have the potential for rupture. Septic peritonitis can also occur from surgical dehiscence after marsupialization or omentalization of these structures.

Gastrointestinal surgical dehiscence: One author (Powers) has seen one case of septic peritonitis secondary to dehiscence of an intestinal resection/anastamosis for removal of a foreign body.

Metastatic neoplasia

The DPL can also be used to screen for metastatic neoplasia within the abdominal cavity. Neoplasia of the abdominal viscera has been reported in ferrets. Two ferrets diagnosed with mesothelioma had abdominal effusion, and the tumors had metastasized widely.[12] Because mesothelial cells may be seen in any abdominal tap, and because phagocytic, activated mesothelial cells can be difficult to differentiate from macrophages, caution in interpretation of their finding is warranted.[7,10] A ferret with exocrine pancreatic carcinoma and carcinomatosis had ascites with mast cells, small lymphocytes, macrophages, and neutrophils observed within the abdominocentesis fluid.[13] Another ferret with ascites had a poorly differentiated pancreatic adenocarcinoma with regional lymphatic, hepatic, enteric, and pulmonary metastasis.[14]

Hemoabdomen

The DPL has potential to be a useful screening tool for hemoabdomen in the ferret. Several cases of hemoabdomen in the ferret have been described, including iatrogenic splenic rupture after abdominal palpation.[15] In another case,[16] a ferret with suspected trauma had non-clotting blood collected by abdominocentesis using a 4-quadrant technique.

Other

Other potential indications for the DPL in the domestic ferret include chemical peritonitis such as that seen with bladder perforation. No reports could be found for other causes of peritonitis in the ferret, such as bile peritoni-

tis, pancreatitis, hepatic abscess, and ruptured pyometra. Recently, a coronavirus-associated disease resembling feline infectious peritonitis has been recognized in ferrets.[17] However, ferret systemic coronavirus has not yet consistently been associated with serous abdominal effusion typically seen with the "wet form" of FIP.

Suggested Technique for Diagnostic Peritoneal Lavage (DPL) in the Ferret
Heavily sedate the ferret with injectable anesthetic agents (eg, propofol, butorphanol) or gas anesthetics (eg, isoflurane, sevoflurane)
Place the ferret in dorsal recumbency (easier for identification and retraction of the spleen)
Empty the urinary bladder, if possible
Identify the margins of the spleen
Shave and prep the skin just caudal and, if necessary, to the right of the umbilicus and caudal to the spleen. If the spleen extends over the umbilicus and to the right of midline, gently palpate and manually retract the spleen cranially. Shave and prep the skin caudal to the caudal margin of the retracted spleen.
Infuse the catheter site (skin, body wall) with 2% lidocaine (not exceeding 1-2 mg/kg)
Make a small stab incision through the skin
Grasp the underlying body wall with a sterile pair of forceps, elevating the tissues from the abdominal cavity. Continued cranial retraction of the spleen may be necessary at this stage
Insert a sterile 18- to 20-gauge over-the needle catheter through the body wall, making certain to avoid the spleen. A sterile 5 to 8 French red rubber catheter can also be used, but a second stab incision must then be made through the body wall. Peritoneal dialysis and DPL catheters can be used but are usually not necessary.
If desired, use a sterile scalpel blade to fenestrate the catheter prior to insertion in order to improve recovery of fluid. Be sure to remove all cut pieces of catheter prior to insertion.
Advance the catheter caudally and dorsally. If the catheter has been fenestrated, advance the catheter beyond the last fenestration
Remove the stylette or needle (if one is present)
Aspirate the catheter
If no fluid is retrieved, instill 20–22 ml/kg of warm sterile isotonic saline
Gently rock the ferret back and forth and/or massage the belly for up to 1 to 2 minutes
Aspirate instilled fluid and place into sterile container(s) for analysis
Repeat instillation of up to one-half the initial fluid can be performed if necessary
Remove the catheter, suture or glue incision(s) closed, if necessary

Ongoing Study

The authors have found the DPL to be an invaluable diagnostic tool in cases of peritonitis in ferrets. The authors are soliciting further case material for which a DPL was used as a diagnostic modality (contact first author [Powers] at miloplume@gmail.com)

The following criteria must be met:

- The diagnosis ultimately was one of the following:
  - Gastrointestinal perforation from a foreign body, neoplasm, trauma, or dehisced surgical site
  - Prostate cyst or abscess rupture
  - Urinary bladder rupture
  - Gall bladder rupture
  - Hemoabdomen
  - Abdominal metastatic neoplasia
- Ferrets had evidence of disease based on history, exam findings, and preliminary diagnostics
- Ferrets lacked obvious abdominal distension from ascites
- Survey abdominal radiographs were taken prior to the DPL
- If ultrasound was performed, the ferret lacked a moderate to large volume of free peritoneal fluid or the fluid was inaccessible by ultrasound-guided aspiration
- A DPL was performed prior to surgery or necropsy

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Additional references are available upon request.

A Novel Mycoplasma Isolated from Pet Ferrets (*Mustela putorius furo*)

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In 2006, a pet vendor in the Seattle area began importing ferrets from a single breeder in eastern Canada, for distribution to pet stores. The ferrets would arrive at 6 weeks of age, with most surgical sites incompletely healed. All these ferrets can be identified by a single green tattoo in the right ear in the shape of the letter "I". The ferrets had received a single vaccination (DISTEM R-TC, Schering Plough) at 5 weeks of age prior to shipping, but otherwise no medical treatments. Shipments of 150–200 kits would arrive every 2–3 weeks, except during periods of inclement weather when shipping was not permitted. It was noted that a few of the kits would exhibit non-productive coughing, and a few would arrive with mild conjunctivitis that usually cleared within a few days while being held at the vendor. As these ferrets were going to local pet stores, the author (Johnson-Delaney) began to see a number presented at the clinic as well as being surrendered to the Washington Ferret Rescue and Shelter that had chronic coughing. The coughing could be described as paroxysmal, honking, and violent, often with the ferret actually moving backwards during the process. Clinical diagnostic testing included bronchiolar lavage and cytology, cultures for bacterial, canine distemper PCR/serology, influenza serology, radiographs, heartworm antigen screening, ultrasonography of heart and thorax, with many tests repeated. Results were negative on all heartworm screening and distemper/influenza tests. Bronchiolar lavage and cytology yielded some inflammatory cells but cultures yielded only various non-pathogenic bacteria, and ultrasonography showed no abnormalities in the thorax. Radiographs showed mild bronchiolar patterns with possible thickening of bronchi, but these changes were not diagnostic.[1,2] As some of the ferrets aged, the coughing generally progressed with more frequent bouts. Tonsillitis often accompanied ferrets with severe coughing episodes. Complete blood counts and chemistries were within normal ranges.[1,2]

Treatments with broad spectrum antibiotics, bronchodilators, expectorants, nonsteroidal antiinflammatories, nebulization, and, in some, low-dose prednisolone were tried that temporarily decreased the clinical symptoms, but they failed to alleviate recurrences. Some shipments of the kits had severe upper respiratory disease as well, including exudative conjunctivitis, nasal discharge, and sneezing in addition to the coughing. Beginning in 2008, a number of these kits also had heavy infections of *Coccidia* species with accompanying diarrhea. Treatment for the coccidia did not affect the respiratory signs.[3] The first death in one of the more severely affected ferrets in April of 2009 that had been unresponsive to all treatment modalities was submitted for necropsy. The 2-year-old spayed female was diagnosed with a severe nonsuppurative bronchitis. The histologic changes of the lung were consistent with a primary bronchiolar insult resembling lesions seen in rats with chronic murine pneumonia caused by *Mycoplasma pulmonis*.[4,5] The lung lesions did not resemble the allergic bronchitis seen in ferrets as there was no smooth muscle hypertrophy, contraction of the bronchioles, or eosinophil infiltrate. The tissue was sent for immunohistochemistry and was found negative for influenza virus but positive for *Mycoplasma* species.

Additional swabs from conjunctiva of affected ferrets as well as bronchiolar lavage specimens from affected ferrets from the above breeder of various ages ranging from 8 weeks of age to 4 years of age were collected, as well as specimens from non-symptomatic ferrets from the same breeder. All were positive on PCR and culture for the *Mycoplasma* organism. The gene sequencing indicates that this is a novel *Mycoplasma* species and there may be more than one strain. At the time of this writing, the assigned name has not been finalized.

Further histopathology is needed to fully characterize the disease. Transmission studies are also planned. At this time, if practitioners are presented with a coughing ferret that is under 4 years of age they should contact Dr Johnson-Delaney (clinical treatment, antemortem diagnostics) and Drs Garner and Kuipel for pathology.

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Investigations into the LH-receptor Sequence in the Ferret Adrenal Gland

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Session #160

Summary Style Manuscript

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Hyperadrenocorticism is a common disease in pet ferrets, in which increased concentrations of gonadotropins play a pivotal role in the formation of adrenocortical neoplasia.[1]

The current study was set out to determine the full-length luteinizing hormone receptor (LHR) sequence in ferrets using conventional PCR techniques in ferret testes. After elucidation of the full-length sequence, the amount of LHR mRNA was measured by real-time RT-PCR. Western blotting was subsequently used to determine the amount of LHR protein in healthy and neoplastic ferret adrenal glands.

In addition to determining the full-length ferret LHR sequence (fLHR), a major splice variant, skipping exon 9, was detected [fLHR(exon 9)]. Equal amounts of fLHR and fLHR(exon 9) mRNA were present in healthy adrenal glands. However, in neoplastic adrenal glands the expression of fLHR mRNA was significantly higher in comparison to fLHR(exon 9). With western blotting, an equal amount of LHR protein was found in neoplastic and healthy adrenal glands.

Based on these findings we conclude that, although the amount of LHR protein is unaltered during neoplastic transformation, a shift is present in mRNA expression from fLHR(exon 9) in normal adrenal glands to fLHR mRNA in neoplastic ferret adrenal glands. Analagous to findings in human ovaries,[2] decreased expression of fLHR(exon 9) may enable gonadotrophins to bind to ferret LHR and exert a biological effect in ferret adrenocortical tumors.

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Brain Tumors in African Hedgehogs (*Atelerix albiventris*)

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Summary Style Manuscript

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Hedgehogs are popular in the pet trade and in zoologic exhibits, and are commonly presented to veterinarians for health care. Common problems in hedgehogs that present with central nervous signs include wobbly hedgehog syndrome and intervertebral disc disease.[1,2] Neoplasia is common in hedgehogs but brain tumors have not been described to the authors' knowledge, although 1 gemistocytic astrocytoma of the spinal cord has been reported.[3,4] A review of the files at Northwest ZooPath identified 6 brain tumors in hedgehogs. Three were male and 3 were female. Age range was 3 to 5 years, and average age was 3.5 years, although precise age was not known for 2 adults. Clinical signs included paresis (1), bilateral front limb rigidity (1), ataxia (1), bilateral central blindness (1), weight loss (1), unilateral hind limb paresis (1), lateral recumbency (1), and thrashing (1). One died, 3 were euthanized, and manner of death was not reported for 2. Histologically, tumors were classified as astrocytomas (3), gemistocytic astrocytoma (2), and microglioma (1). All tumors were invasive malignancies associated with destruction of the regional neuropil. The astrocytomas had considerable cellular anaplasia and were located in the cerebellum (2) and brainstem (3). The microglioma was in the cerebrum. The gemistocytic astrocytomas were in the anterior brain stem (1) and hippocampus (1). Metastatic lesions were not seen for any of the tumors. Concurrent disease processes included chronic renal disease (2), pyoderma (2), adrenal cortical hyperplasia (2), spongiform change in brain and spinal cord (1), degenerative disc disease (1), hepatic lipidosis (1), degenerative cardiac disease (1), sepsis (1), lymphoplasmacytic enteritis (1), chronic urocystitis(1), and cataract (1). Although the prevalence of brain tumors appears to be low in hedgehogs, these neoplasms should be considered in the differential diagnosis for those patients with central nervous system signs.

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Cryptococcal Meningitis and Optic Nerve Neuritis in a Chinchilla (*Chinchilla lanigera*)

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Session #165

Summary Style Manuscript

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A 13-year-old chinchilla (*Chinchilla lanigera*) with no previous significant medical history was subdued for 1 day before presenting with lethargy, anorexia, and mild intermittent rear leg tremors. The chinchilla was visual and able to move normally. Radiographic and ultrasound images were within normal limits, but attempts at phlebotomy were unsuccessful. Subcutaneous fluids and presumptive anti-inflammatory and antimicrobial treatments were begun. On day 4, the animal started to fall to the right side (often paddling when lateral), appeared blind, and had intention tremors. A limited biochemical panel revealed normal calcium (8.5 mg/dl), elevated creatine phosphokinase (19,824 IU/L), and hyperglycemia (252–411 mg/dl over the day). Because of consistent weight declines, assisted feeding was instituted. On day 6, there was a slow worsening with increased lateral recumbency, a right ear droop, and more pronounced intention tremors. There was no menace reflex bilaterally, and no dazzle reflex in the right eye. Serial blood glucose readings ranged from 349–359 mg/dl and were not affected by a single insulin injection of 0.15 IU. On day 7, the animal was very weak, in lateral recumbency, and could no longer swallow, so euthanasia was performed.

Histiocytic meningitis and optic neuritis with myriad intra-lesional yeasts morphologically consistent with *Cryptococcus* species were documented at necropsy. The site of origin was not evident. There was focal necrosis of peripancreatic fat, but significant pancreatitis or changes in pancreatic islet cells that could explain hyperglycemia were not observed.

No previously published reports of cryptococcosis in chinchillas were found.

Hymenolepis nana in Black-tailed Prairie Dogs (*Cynomys ludivicianus*)

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Session #168

Summary Style Manuscript

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Hymenolepis nana is a cestode that infects mainly mice and rats and can cause clinical disease in humans, especially in children.[1–3]

Emaciated prairie dog pups (n = 6) were presented with anorexia, facial edema, facial alopecia, wet chins, and pale mucous membranes. All pups were purchased at the same pet store. At the time of presentation, one owner had already lost 8 animals over several weeks. Fecal examination revealed *Hymenolepis nana* infestation in 5 animals. Necropsy of one prairie dog revealed edematous skin, ascites, pulmonary edema, and severe cachexia.

Hematology of one animal showed reduced PCV (23.5%; reference range, 36%–54%), decreased RBC (4.15 x 10^6/µl; reference range, 5.9–9.4 x 10^6/µl), and decreased Hb (7.29 g/dl; reference range, 12.7–19.6 g/dl). Abnormalities on the biochemical profile of the same animal included hypoproteinemia (4.6g/dl; reference range, 5.8–8.1 g/dl), low albumine (2.12 g/dl; reference range 2.4-3.9 g/dl), elevated AST (175 IU/L; reference range, 16–53 IU/L), and elevated bilirubin, total (0.6 mg/dl; reference range, 0.1–0.3 mg/dl).[4]

Treatment included praziquantel (10 mg/kg SC every 10 days), protein supplementation, and supportive care.[5]

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Multiple Facial Surgeries in a Pet Rabbit

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Session #170

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Abstract: A challenging case involved in a 2-year-old female lop rabbit. The disease started as an extensive odontogenic facial abscess. Complications led to chronic purulent rhinitis; empyema of the alveolar bulla; retrobulbar abscess; empyema of the tympanic bulla; and empyema of the maxillary recess. Treatment required multiple facial surgeries including: excision of the facial abscess; extraction of the left maxillary cheek teeth and lateral maxillotomy for treatment of the retrobulbar abscess; lateral otostomy; and monolateral rhinostomy. This case represents the first report of a successful surgical treatment of a retrobulbar abscess without enucleation of the eye globe.

Introduction

Rabbits are a herbivorous species with elodont (open-rooted) teeth.[1,2] Since these continuously growing and erupting teeth do not develop a true anatomical root, this portion is referred to as the *apex*. The portion of the tooth below the gingiva is named *reserve crown*, and the continuously renewed crown is called a *clinical crown*.[1,2] The normal dental formula of rabbits is I2/1 C0/0 P3/2 M3/3 for a total of 28 teeth.[1] Premolars and molars are anatomically indistinguishable, and are therefore simply termed *cheek teeth*.[1,2] In rabbits, a peculiar bone structure called the *alveolar bulla* includes the reserve crown and apexes of the 4 caudal cheek teeth (the 3rd premolar, and the 3 molar teeth).[1,3,4] Normal grinding motions during chewing produce sufficient wear to keep incisors at the proper length in the normal animal. Similarly, normal crushing of food produces adequate wearing of cheek teeth in the normal animal.[2]

The *tympanic bulla* is well developed in rabbits. Unlike the alveolar bulla, it is normally an empty anatomic structure.[1,3] The tympanic bulla is connected laterally with the ear canal, entering the alveolar bulla through the external acoustic meatus; medially it connects with the pharynx through the Eustachian tubes.[5]

Rabbits have 3 lacrimal glands. The main lacrimal gland lies in the caudal and dorsal portion of the orbital fossa. The large accessory lacrimal gland is located in the lower part of the orbit. The superficial and the deep gland of the third eyelid are associated with the nictating membrane, and lie cranially close to the eye globe.[6]

Conchae (otherwise named *turbinates*) are cartilagineous and mucosal structures filling the nasal cavities.[3] They outline empty spaces (*meati*) and blind cavities (*sinuses*). A ventral meatus, a dorsal meatus, and 2 paranasal sinuses (the conchal and maxillary sinuses) have been described in rabbits.[6] At the cranial end of each sinus is a single opening into the dorsal and the ventral meatus, respectively. Rabbits lack a frontal sinus.[6]

Dental disease is a result of anatomical or physiologic abnormalities of incisor teeth, cheek teeth, or both. Any process interfering with normal eruption of, or wearing of continuously growing teeth, will result in dental disease. Diagnosis of dental disease is greatly enhanced by 3 modalities: thorough examination of the oral cavity, radiology (including computed tomography), and endoscopic evaluation.[2]

Magnetic resonance imaging (MRI) has recently become available for small mammals. It is the elective diagnostic imaging modality of soft tissues because images are of superior quality compared with computed tomographic images of soft tissues.[7–9] Computed tomography (CT) is also very useful for detailed diagnosis of acquired dental disease and its complications, mostly when involved bones and related structures are investigated. CT is also very useful in investigating nasal cavities.

Abnormalities in dental disease can include deviations of the cheek teeth occlusal plane; sharp spurs or spikes on lingual or buccal aspects of cheek teeth; loose or fractured teeth; widened interproximal spaces; and damage to the tongue and adjacent soft tissues.[2]

Periapical infections are common in pet rabbits, and they represent an important portion of the acquired dental disease syndrome.[3,10] Due to the peculiar anatomy of the teeth and the relationship between the reserve crown and the maxillary and mandibular alveolar bones, periapical infections spread to surrounding bone and soft tissues, producing osteomyelitis, abscesses, or empyema of natural cavities.[3] These complications must be considered during diagnosis and prognosis, as in early stages, initial focus of infection can be very subtle and completely asymptomatic. A number of surgical techniques and options have been proposed beyond simple and usually ineffective incision of the abscess and flushing of the purulent material.[3,10] The surgical treatment of choice is that which addresses the 3 typical pathologic conditions related to periapical infections: the presence of a capsule; the presence of necrotic tissue–soft, dental, or bony; and the presence of the osteomyelitis.[3,10] Ideally, the surgical technique should allow frequent postoperative debridement of the surgical site, flushing and application of antiseptics or other products to promote healing, and allow constant direct monitoring of healing until it occurs by second intention. Marsupialization of the surgical site allows these surgical principles to be applied in cases of osteomyelitis of the skull.

The surgical technique and the postoperative management described above represent treatment options for the more common periapical infections. However, this technique is not appropriate in all cases. Retrobulbar abscesses in pet rabbits are a common consequence of dental disease due to the anatomical relationship between the apexes of the maxillary cheek teeth and the orbital fossa. When a periapical infection involves 1 or more of the 4 caudal cheek teeth, the alveolar bulla acts as a preformed bone cavity and can fill with food debris, tooth fragments, and pus. If the thin dorsal cortical bone of alveolar bulla is perforated, a retrobulbar abscess may occur.[3] The extraoral (lateral) approach is extremely difficult to use for abscesses of the mandibular teeth, or of the most cranial maxillary cheek teeth, because the zygomatic arch limits the approach; as a result, the marsupialization technique cannot be applied. However, an intraoral approach to the alveolar bulla through the tooth socket after extraction of the diseased maxillary cheek tooth/teeth is feasible and has been reported.[11] A lateral extraoral surgical approach to the alveolar bulla by partial excision of the zygomatic arch has been reported by the author in a 5-year-old male Flemish giant rabbit.[4,12] Alternative surgical techniques for treatment of retrobulbr abscess with a parabulbar approach combined[13] or not[14] with intraoral extraction have been reported.[13,14] Rhinitis and sinusitis have been reported as the most common forms of pasteurellosis.[5] In addition, infection may spread from the nares to the middle ears through the Eustachian tubes. Most rabbits with rhinitis also have otitis media.[5]

Surgical techniques for lateral ear canal resection (permanent otostomy) and for total ear canal ablation (TECA) associated with ostectomy of the tympanic bulla for treatment of otitis externa and media in rabbits have been described by the author.[15] A ventral approach for osteotomy of the tympanic bulla is also reported.[16] Ventral bulla osteotomy is indicated for the management of otitis media without otitis externa.

Rhinotomy for treatment of non-odontogenic rhinitis has been reported.[17] The author has performed permanent and temporary rhinostomy for treatment of chronic, septic rhinitis following complicated end-stage dental disease, and for surgical access to the nasal meati.

Case Report

First presentation and surgical treatment of the facial abscess

A 2-year-old intact female lop rabbit was presented with a extensive facial swelling (Fig 1). The history did not report any significant information, with the exception of inappropriate nutrition including limited vegetables, a seed and cereal mix, bread, and no hay. The rabbit was still eating, but the quantity consumed was less than was normal. The rabbit was alert and responsive, body temperature was within normal range (37.5°C), and mild dehydration was present. The lump extended over the entire left masseteric region, from the lower eyelid to the mandible. Palpation of the ventral margin of the left mandible did not reveal bone deformities. The intraoral inspection performed during physical exam showed malocclusion of cheek teeth. In particular, there was excessive elongation of the left maxillary first premolar with a sharp buccal spur, and subsequent ulceration of the buccal mucosa. Tentative diagnosis based on physical exam was non-periapical, dental-related facial abscess.

The rabbit underwent anesthesia for radiography of the skull, intraoral inspection and treatment, and surgical debridement of the facial abscess. General anesthesia was induced with butorphanol (0.4 mg/kg SC), dexmedetomidine (40 g/kg IM), and ketamine (20 mg/kg IM). Oxygen was administered by face mask.

Radiographs of the skull and teeth showed signs of acquired dental disease of cheek teeth, not consistent with periapical infection, and confirmed coronal elongation and a spike at the left first premolar.

Figure 1. Facial swelling at presentation, after partial shaving. The lump extended over the whole left masseteric region, from the lower eyelid to the mandible. The rabbit is shown after sedation. Used with permission from Vittorio Capello, DVM.

Oral endoscopy confirmed elongation of clinical crowns of the cheek teeth. In particular, the left maxillary premolar was bent on the buccal side, forming a sharp spike that created a small ulceration of the mucosa near the origin of the abscess. Intraoral treatment was performed with coronal reduction of cheek teeth arcades, combined with extraction of the left maxillary premolar. Pus coming out through the oral fistula confirmed the communication with the abscess. After the intraoral procedure, orotracheal intubation was performed using a 2.5-mm uncuffed endotracheal tube, The rabbit was placed in lateral recumbency on the right side and prepared aseptically for surgery.

After incision of the skin over the swelling, careful blunt dissection of the abscess capsule was performed. After thorough excision of most of the abscess, it was necessary to open it in order to handle it properly and complete surgical removal. The abscess was filled with a malodorous, fluid pus. The owner declined culture and sensitivity. Complete removal of the abscess with its capsule led to wide exposure of the masseter muscle, the zygomatic area, and the caudal portion of the buccal area.

The oral/extraoral fistula from the buccal lesion was clearly visible just cranial to the aponeurotic insertion of the masseteric muscle over the zygomatic arch.

Due to the impossibility of marsupialization of such a wide defect, a cosmetic surgical reconstruction of the skin was attempted after thorough flushing of the previously infected area. Marsupialization was limited to the small area around the oral/extraoral fistula, with the intent to allow flushing and healing by second intention. The owner declined patient boarding; therefore, the rabbit was sent home on antibiotic and analgesic treatment (procaine penicilline, 40,000 IU/kg SC q24h, and meloxicam 0.2 mg/kg OS q12h). The patient was able to eat without assistance 6 hours after surgery.

Follow-up and second surgical debridement

The first recheck was performed 3 days after surgery. Failure of the cosmetic reconstruction of the skin was evident; a large portion of it was necrotic. Black, dry skin and a foul smell were present. Overall patient condition was otherwise very good.

A second surgical debridement was recommended, but the owner declined any further treatment and elected euthanasia. The alternative of surrender was offered, and the owner agreed. The rabbit underwent a second, short, surgical procedure in order to remove the necrotic flap of skin. After debridement of small parts of necrotic tissues, the defect was left open to heal by secondary intention. Postoperative systemic antibiotic and analgesic treament was continued. Locally, the rabbit was treated with frequent flushing of saline solution and application of cream containing quaternary ammonium suspended in aloe vera distillate and monoglyceride of fatty acid (HEALx Soother Plus, Harrison's Pet Products, Lake Worth, FL, USA) twice a day.

Constant granulating process occurred postoperatively, allowing complete healing in 18 days, with minimal scar retraction of the skin and of the lower eyelid.

Diagnosis and medical management of rhinitis, and ovariohysterectomy

When complete healing was achieved, the rabbit was scheduled for elective neutering. However, a couple of days earlier, the rabbit began sneezing and a mucopurulent, unilateral left nasal discharge became evident. Symptomatic treatment with a second trial of procaine penicilline (40,000 IU/kg SC q24h) combined with nebulization with acetylcysteine was administered, and ovariohysterectomy was delayed.

In about a week, both signs and symptoms disappeared, and neutering was rescheduled. The rabbit underwent MRI of the skull to allow visualization of the nasal cavities. Magnetic resonance imaging was performed with a scanner equipped with a 0.2-Tesla magnet. Complete filling of the left nasal cavity with dense material was

evident in all 3 scanning planes (transverse, dorsal, and sagittal). The right nasal cavity appeared completely normal, showing evident unilaterality. Following MRI, the rabbit underwent elective ovariohysterectomy.

Diagnosis of retrobulbar abscess and empyema of the maxillary recess

Over the following weeks, the rabbit appeared normal with no relapse of symptoms of rhinitis.

Three months after the first surgical procedure and more than 2 months after healing, the rabbit developed severe, left unilateral exhophtalmus that developed over a 3-day period. Exophtalmus, conjunctivitis, mild keratitis, and episcleritis were also present (Fig 2). The palpebral reflex was absent due to the prominent exophtalmus; the corneal reflex was reduced but still present. Overall patient condition was good, and the rabbit was eating and acting normally. Because of the rabbit's quiet attitude, the exophtalmic eye was not protected with a Elizabethan collar. Systemic antibiotic treatment with procaine penicilline was started, and topical conservative treatment with "artificial tears" and piroxicam eye drops was administered.

Accurate differential diagnosis between retrobulbar and parabulbar abscess was pursued, with the goal of preventing enucleation of the eye globe. The rabbit was scheduled for ultrasonography of the eye globe, and for magnetic resonant imaging and computed tomography of the skull.

The sonogram of the eye globe demonstrated the presence of a thick, echogenic structure behind the anechoic, normal eye globe. Echogenicity was not compatible with fluid; therefore, those features were consistent with a capsulated abscess, filled by dense pus.

Figure 2. Marked exophtalmus, conjunctivitis, prolapse of the third eyelid, and keratitis. Used with permission from Vittorio Capello, DVM.

The magnetic resonsance exam confirmed the presence of a 1.3 x 1.7-cm, dense, retrobulbar mass. The transverse scanning plane demonstrated the marked exophtalmus; the dorsal scanning plane showed the mass was medio-caudal compared with the position of eye globe, positioned just cranial to the cerebral techa. The series on this plane demonstrated marked improvement of the empyema of the left nasal cavity.

Edema and/or involvement of the peribulbar tissues and the accessory lacrimal gland were also evident.

The axial series of computed tomography demonstrated perforation of the left alveolar bulla from the apices of maxillary CT2, CT3, and CT5. Empyema and osteolysis of the maxillary recess was present cranial to the alveolar bulla. The analysis of caudal slices showed radiodense material in the deep portion of the left ear canal and inside the alveolar bulla (Fig 3).

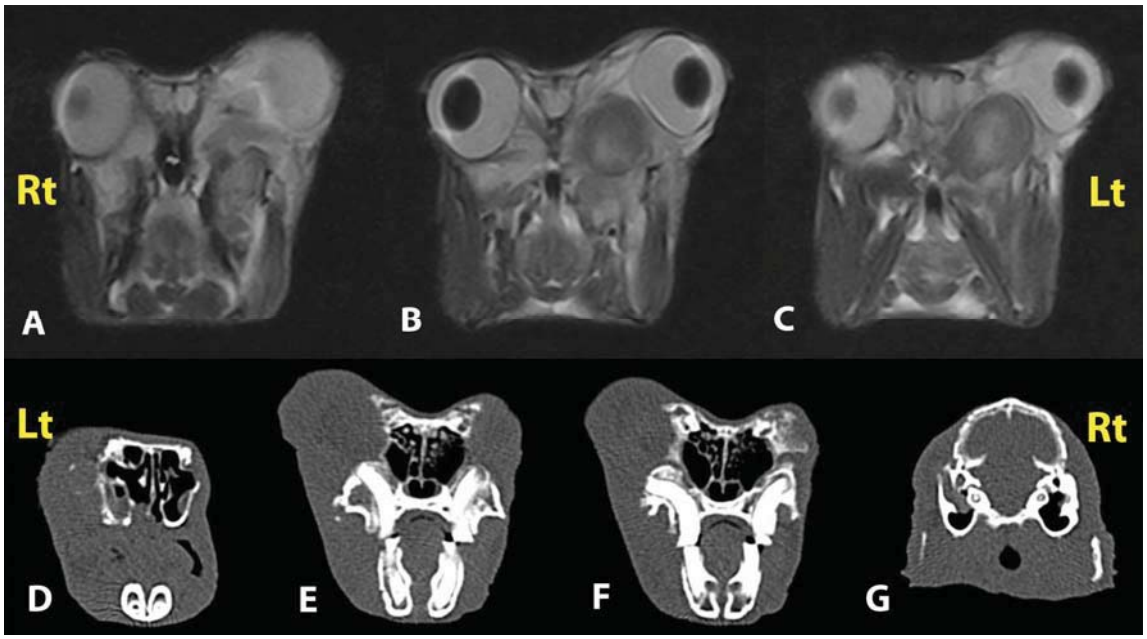

Figure 3. Magnetic resonance imaging (MRI) and computed tomography (CT) of the skull.
(A, B, C) Axial projection, from rostral to caudal.
(A) The retrobulbar abscess is visible as a darker mass under the left eye globe. Note the mass begins caudal to the most caudal maxillary cheek teeth.
(D, E, F, G) Axial view from rostral to caudal.
(D) Section of the nasal cavity shows empyema of the maxillary recess on the right side periapical growth and deformity of CT2 (E) and CT3 (F). In (F), note the comparison between the perforated alveolar bulla (left) and the normal bony contour (right).
(G) Radiodense material in the deep portion of the ear canal and inside the alveolar bulla.
Used with permission from Vittorio Capello, DVM and Alberto Cauduro, DVM.

Surgical management of the retrobulbar abscess

Surgical treatment was planned for treatment of the retrobulbar abscess. Because enucleation of the eye globe was not considered as the first surgical option, intraoral approach to the abscess through the alveolar bulla was attempted.

The rabbit underwent general anesthesia with the same anesthetic protocol listed above. Extraction of left maxillary CT2 was performed, but no pus was released from the abscess after extraction. The remaining cheek teeth of the maxillary arcade were extracted as well, both because they were loose, and because of the report from CT scan. After extraction of the entire cheek tooth arcade, the attempt to incise the capsule of the abscess and release the pus through the oral cavity was unsuccessful, using both a Crossley's luxator for cheek teeth and an 18-gauge contoured needle. Apposition of the gingiva was achieved with 2 interrupted 4-0 absorbable sutures.

Lateral approach to the retrobulbar space was then performed with maxillotomy of the alveolar bulla. A horizontal skin incision was performed parallel and just dorsal to the dorsal margin of the zygomatich arch. Blunt dissection of the subcutaneous tissue led to exposure of the ventral border of the eye globe. Careful para- and ventrobulbar dissection of the ventral straight and the ventral oblique muscles of the bulb of the eye led to access the alveolar bulla. Entering the alveolar bulla allowed incison of the abscess capsule. Removal of the pus, gentle debridement, and flushing allowed the retrobulbar abscess to empty. Even though retrobulbar pressure was now relieved, replacement of the eye globe in the normal anatomic position was not yet possible becuase of swelling in retrobulbar and parabulbar soft tissues. Suture of the muscle layer and the skin, respectively, with adsborbable and non-adsorbable 3-0 suture material completed the surgical procedure. Evident exophtalmus was still present after surgery, and topical treatment for both keratitis and conjunctivitis was continued. Procaine penicilline at the dosage reported above and prednisolone (1.5 mg/kg q24h IM) were administered as systemic treatment.

Recovery from this surgical procedure was uneventful, and the rabbit was able to eat on its own the following day. The main goal of reduction of the exophtalmus was achieved in about 3 days, while 2 minor complications did occur. Dehiscence of the suture was managed with local flushing, debridement, and application of healing cream (HEALx Soother Plus, Harrison's Pet Products) twice a day. Focal keratitis involved the whole surface of the cornea, so local treatment with piroxicam eye drops was increased to every 6 hours. About a week later, the affected left eye was completely recovered from the severe exophtalmus, conjunctivitis, and keratitis. A mild degree of retraction of the lower eyelid occurred.

Surgical management of the otitis externa

Several days after recovery from the surgical procedure for the retrobulbar abscess, thick, white exudate from the ear canal became evident. Mild intermittent head tilt on the left side unassociated with other neurologic signs was present. Considering the previous information provided by computed tomography, otitis externa and media was diagnosed.

The rabbit was scheduled for lateral ear canal resection. Before the sugical procedure, intraoral endoscopic recheck showed dehiscence of the suture of the gingiva placed after extraction of the maxillary cheek teeth. The mucosal wound was cleaned of food debris, then flushed and debrided with a Williger bone curette. A second suture was not attempted. The diseased homolateral mandibular cheek teeth arcade did not show regrowth, so coronal reduction was not needed in this case. Otoscopy was performed before ear canal resection using a 2.7-mm rigid endoscope. It was unremarkable because of the presence of waxy debris, pus, and stricture of the vertical portion of the ear canal. Lateral ear canal resection was performed utilizing the technique reported by the author.

Recovery from anesthesia and surgery was uneventful, and healing of suture margins occurred in about 3 weeks.

Rhinostomy and second maxillotomy

About 2 months later, a white and dense discharge from the left nasal opening was evident, together with mild exophthalmus and a white discharge from the proximal opening of the nasolacrimal duct. There was also mild swelling at the site of the previous maxillotomy. In consideration of the latent diagnosis of chronic, monolater rhinitis and homolateral empyema of the maxillary recess, permanent rhinostomy was scheduled, with the goal of cleaning and flushing the nasal cavity and the maxillary recess and establishing a patent access to the left nasal cavity for further medication. Before the surgical procedure, intraoral endoscopic recheck showed that the open alveolar bulla was filled with thick, granulating tissue.

The rabbit was placed in sternal recumbency for rhinostomy. A skin incision was performed on the midline of the dorsal nasal region, and blunt dissection of the subcutaneus tissue led to exposure of the periosteum of the nasal bones. Osteotomy of the left nasal bone was performed with the tip of a #11 scalpel blade, following the anatomic junctions with the incisive bone on the lateral side, and with the right nasal bone on the medial side. Ostectomy was completed burring the caudal end and dissecting the cartilagineous rostral end of the left nasal bone. Part of the necrotic turbinates were removed using a small needle holder as a rongeur. Mucus, dense pus, and clots were thorougly flushed and removed. Flushing of the left maxillary recess was performed in the caudolateral part of the nasal cavity. Apposition of the skin over the rhinostomy site was performed; two 4-0 nonabsorbable sutures were used to transfix the periosteal membrane. The maxillotomy site was surgically addressed as well. A communication was present with the maxillary recess through the perforated surface of the maxillary bone, but there was no longer a communication with the alveolar bulla. Antibiotic and anti-inflammatory treatment was administered.

The postoperative period was uneventful. The stoma allowed postoperative flushing of the nasal cavity, and the rabbit was able to breath more easily. Clinical signs and symptoms of rhinitis disappeared in about 2 weeks, and the stoma was progressively reduced by concentric healing of the skin.

The final follow-up about 5 months after the first surgical procedure showed complete resolution of the many facial abscesses, empyemas, and of the exophtalmus (Fig 4).

The rabbit died suddenly about 4 months later, following acute gastrointestinal bloat of unknown origin.

Discussion

Reported anatomic terminology of rabbit sinuses is not completely correct. A *sinus* is defined as an empty space inside cavitary bones, but in actuality such thick and developed skull bones are not present in rabbits. *Meatus* is a space between the turbinates. A dead-end, blind space inside the nasal cavity is better defined as a *recess*. *Maxillary recess* is therefore the terminology proposed by the author.

Rabbits can present with huge facial swellings. Owner neglect, long hair, and lack of symptoms may lead owners to present these patients very late. With the exception of abscesses following bites or scratches from cats when those species are housemates, facial abscesses in rabbits are always directly or indirectly related to dental disease.

The typical pattern of acquired dental disease makes the maxillary clinical crowns curve laterally and the mandibular crowns curve medially. While lesions can occur anywhere, mandibular teeth usually produce tongue lesions, while maxillary teeth produce buccal lesions. Extraction of the left maxillary premolar was performed in order to prevent regrowth in the same abnormal, bent direction.

Figure 4. Final follow-up. The frontal view shows long-term reduction of the exophtalmus, in comparison with the normal right eye. Used with permission from Vittorio Capello, DVM.

In the author's experience, fluid and malodorous pus is related to gangrenous anerobic bacteria. Healing by second intention is more challenging when these bacteria are involved, and this was likely that case, even though culture and sensitivity were not performed. On retrospect, the cosmetic reconstruction of the skin might not have been the best surgical choice, but healing by second intention of such a wide defect was not compatible with postoperative care provided at home by the owner. This patient represents a typical example where postoperative management and boarding must be discussed with the owners in detail, and must be made mandatory after an invasive surgical procedure.

Septic rhinitis is common in rabbits, and may follow local infections such as dental abscesses. In this case, the evident homolaterality with previous abscesses supported the pathophysiology of contiguity, even if no fistulas were present from the abscess to the nasal cavity or to the maxillary recess.

Because purulent exudate in rabbits is very thick, and density may be compared with those of soft tissues, the magnetic diagnostic imaging modality was elected. A disadvantage of MRI compared with CT is prolonged scanning time; however, in this stable patient it was not a concern. Resolution of diagnostic imaging is a concern for small exotic mammals, and an MR scanner capable of a 1.3-Tesla magnetic field has been recommended over scanners with "weaker" fields capable of only 0.2 or 0.4 Tesla.[7] Despite the 0.2-Tesla magnet available, resolution was optimized at 512 x 512 pixels, resulting in an image not inferior to standard CT images. This case represents a good example where combined diagnostic imaging was critical for detailed diagnosis. Among radiographic modalities, the diagnostic potential of CT is superior to traditional radiographs for challenging

dental cases and their complications; this is true even as high-quality films are still critical in investigating dental disease, especially of the mandible. Both CT and MRI are advanced diagnostic imaging options, but each has specific advantages in regard to bony and dental structures, and of soft tissues, respectively. This is why they should ideally be performed in combination.

In this specific case, a tentative diagnosis of retrobulbar abscess based on history, clinical signs, and ultrasonography was apparent; nevertheless, detailed visual information about size and positioning may be critical for prognosis and surgical planning, especially when the intent is to salvage the eye globe through surgical procedures. Computed tomography did not allow additional information about the size and position of the retrobulbar mass, but it did provide more visual information about the nasal cavity, the left maxillary cheek teeth, the alveolar bulla, as well as the deep ear canal and the tympanic bulla. Periapical infections and facial abscesses may involve more anatomical structures of the skull. This case demonstrated how scanning of the whole skull is recommended even if specific clinical signs are not present, rather than CT scanning of a selected section of the skull. Scanning options like slice thinkness and the reconstruction index must be optimized in order to get the most detailed visual information.[18]

Failure to incise the capsule of the retrobulbar abscess from the oral cavity through the tympanic bulla was in some ways expected. Unlike other case reports,[4,11,12] this rabbit was not affected by deformity and empyema of the alveolar bulla, but it had a true retrobulbar abscess, positioned dorsal and caudal the alveolar bulla, as properly outlined by CT and MRI. Also, proper long and curved instruments effective in accessing the deepest portion of the alveoral bulla and of the retrobulbar space from the oral cavity were not available. The use of a flexible endoscope with operating side channel might have been useful in this case. Extraction of the maxillary cheek teeth represented a proper indication because 3 of the remaining 5 were loose, and they had elongated and deformed reserve crowns.

Unlike a case previously reported by the author,[4,12] partial ostectomy of the zygomatic arch was not needed. Severe exophtalmus pushing the eye globe in a dorsal position allowed entrance to the alveolar bulla. Marsupialization and postoperative flushing was not an option both because of the anatomic position, and because the eye globe was expected to regain anatomic position.

Despite the fact that the use of steroids is not recommended in rabbits, and contraindications do exist in case of degenerative diseases of the eye, prednisolone was administered in order to maximize anti-inflammatory activity. No adverse symptoms or contraindications have been recorded in this patient.

Extraction of the entire cheek teeth arcade has been anecdotally reported, and has been performed when indicated by the author. This degree of extraction is generally not desired, but it might be necessary when empyema of the alveolar bulla and/or a retrobulbar abscess are present. In addition, when teeth are loose, a kind of "domino effect" is experienced during the extraction procedure, where extraction of a mesial cheek tooth makes the adjacent, distal cheek tooth loose as well, forcing extraction of the whole arcade. This is particularly evident when maxillary CT3 is extracted, because the reserve crown of the first molar tooth (CT3) and of the other 3 distal molar teeth are located inside the alveolar bulla. Pet rabbits appear to tolerate multiple extractions well, and they are usually able to eat normally if the contralateral cheek teeth arcades are in normal or good condition. Ideally, repeated coronal reduction of the opposing cheek teeth arcade must be performed at regular intervals of about 4–6 months. Many rabbits needing extensive extraction are affected by dental disease of the opposing cheek teeth arcade; therefore, coronal reduction may not be needed, or may be needed infrequently.

Many different bacterial involvements of periocular structures can lead to the clinical sign of exophtalmus. All those etiologies have been generally reported as "retrobulbar abscess." Even if this pathologic condition is relatively common in pet rabbits as a complication of dental disease, a proper classification must be outlined

and discussed not only because details of the clinical sign can be different (the exophtalmus shows different features), but mostly because many different surgical approaches can be pursued depending on the type of abscess. Periapical infections of maxillary premolar teeth (CT1 and CT2) usually lead to maxillary (zygomatic) abscesses. In these cases, mild exophtalmus can occasionally be present. Periapical infection of the maxillary molar teeth (CT3 through CT6), in which the reserve crowns are included inside the alveolar bulla, can lead to at least 5 different complications (single or combined): initial empyema of the alveolar bulla; true retrobulbar abscess; parabulbar abscess located in the infraorbital or the caudal area of the eye globe; and empyema of the maxillary recess. All of them may lead to a certain degree of exophtlmus. In advanced cases of end-stage dental disease, empyema of the alveolar bulla and of the maxillary recess can lead to chronic septic rhinitis and empyema of the nasal cavity.

Classification, clinical signs and symptoms, potential involvement of adjacent structures, and surgical approaches are reported in Table 1.

A mild degree of retraction of the lower eyelid occurred. It was the eventual outcome of the second surgical debridement of the large abscess, and to a minor extent of the maxillotomy access performed during surgical treatment of the retrobulbar abscess. Despite partial exposure of the ventral conjunctiva, the cornea was not eventually affected by this functional defect of the lower eyelid.

Otitis externa, with possible involvement of the middle ear and the tympanic bulla, is common in lop-eared rabbits. It is impossible to state if this was an underlying, asymptomatic condition; but since clinical signs were not present at the first clinical exam, in this case pathophysiology might have been from contiguity from adjacent dental disease, through the Eustachian tube.[5]

In the author's experience, suture of the gingiva after extraction of cheek teeth is feasible but difficult because of the narrow opening of the rabbit oral cavity. Dehiscence of the suture is a common outcome, probably due to incessant chewing movements of the rabbit. Rabbits are anisognathics; therefore alternate mastication between the 2 sides of cheek teeth arcades is physiologic, regardless of the absence or poor condition of affected teeth. On the other hand, there is no evidence that force feeding through a nasogastric tube would have granted a proper healing, and the option to withold food to a rabbit patient able to eat was not considered. Despite dehiscence and the impossibility of performing frequent flushing, the wound can heal by secondary intention. Impaction of food inside the alveolar bulla may or may not be a complication. In this case, proper healing did occur.

Otoscopy is rarely helpful in cases of advanced disease of the ear canal, and CT is superior for diagnosing abnormal conditions of the horizontal portion of the ear canal and the tympanic bulla. Otoscopy can be useful in early cases, when preventative lateral ear canal resection is still a possibility.

Evaulation of clinical signs and information provided by CT did not indicate the need for ostectomy of the tympanic bulla. In particular, ventral bulla osteotomy is indicated to manage otitis media without otitis externa, and this was not this patient's case.

Retrobulbar abscesses are commonly diagnosed when they produce increased pressure behind the eye globe and/or panophthalmitis. Advanced cases presenting late in the course of the disease result in loss of the orbit and necessitate enucleation. This procedure allows dorsal surgical access to the alveolar bulla, but also risks exposure of the optical nerve and the optic foramen, and is also not ideal for aesthetic reasons.

The surgical treatment of the case reported here represents the first, long-term successful treatment of a true retrobulbar abscess obtained without enucleation of the eye globe.

Table 1. Classification of maxillary odontogenic abscesses, clinical signs, and symptoms, potential involvement of adjacent structures, and surgical approaches.

Etiology	Clinical signs	Clinical symptoms	Denomination	Surgical approach
Periapical infection of maxillary premolar teeth (CT1 and/or CT2)	Swelling of the rostral maxillary area	Epiphora Mild exophtalmus (occasionally)	1) Maxillary abscess 2) Zygomatic abscess	Intraoral: extraction of CT1 and/or CT2 Extraoral: surgical, extraction of apical fragments when present
Bacterial dacryocystitis	Swelling of the rostral maxillary area	Epiphora Mild exophtalmus (rarely)	1) Maxillary abscess 2) Zygomatic abscess	Extraoral: surgical debridement
Periapical infection of maxillary molar teeth (CT3 through CT6)		Mild exophtalmus	Empyema of the alveolar bulla	Intraoral: extraction of the affected maxillary tooth/teeth Extraoral: maxillotomy of the alveolar bulla via partial ostectomy of the zygomatic arch
Periapical infection of maxillary molar teeth (CT3 through CT6)	Severe exophtalmus Keratitis Conjunctivitis Panophtalmitis		Retrobulbar abscess	Enucleation of the eye globe Intraoral: extraction of the affected maxillary tooth/teeth and possible access to the retrobulbar abscess Extraoral: maxillotomy of the alveolar bulla
Periapical infection of maxillary molar teeth (CT3 through CT6) and possible involvement of the accessory lacrimal gland	Swelling of the infraorbital area	Exophtalmus from mild to severe	1) Parabulbar abscess (cranial) 2) Infraorbital abscess	Intraoral: extraction of the affected maxillary tooth/teeth and possible access to the parabulbar abscess Extraoral: Parabulbar approach
Periapical infection of maxillary molar teeth (CT3 through CT6) and possible involvement of	Swelling of the caudal orbital and parietal area	Exophtalmus from mild to severe	Parabulbar abscess (caudal)	Intraoral: extraction of the affected maxillary tooth/teeth and possible access to the parabulbar abscess

Table 1 Cont'd. Classification of maxillary odontogenic abscesses, clinical signs, and symptoms, potential involvement of adjacent structures, and surgical approaches.

Etiology	Clinical signs	Clinical symptoms	Denomination	Surgical approach
the lacrimal gland				Extraoral: Parabulbar approach
Periapical infection of maxillary molar teeth (usually CT1 through CT3) and involvement of the maxillary recess	Swelling of the ventral maxillary area	Epiphora Mild exophtalmus	Empyema of the maxillary recess	Intraoral: extraction of the affected maxillary tooth/teeth Extraoral: maxillotomy of the maxillary recess via osteotomy of the perforated surface of the maxilla
Periapical infection of maxillary molar teeth (usually CT1 through CT3), involvement of the maxillary recess and the nasal cavity	Possible swelling of the ventral maxillary area (not always)	Epiphora Mild exophtalmus Nasal purulent discharge Sneezing	Empyema of the maxillary recess Empyema of the nasal cavity	Intraoral: extraction of the affected maxillary tooth/teeth Extraoral: permanent rhinostomy (unilateral or bilateral) maybe combined with maxillotomy of the maxillary recess via osteotomy of the perforated surface of the maxilla

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Rabbit Thoracic Surgery

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Abstract: Thoractomy in rabbits is mainly indicated to treat thymoma/thymic lymphoma or pulmonary abscesses. Endotracheal intubation is essential for thoracic surgery and can be challenging in rabbits. The 2 most commonly used approaches to the chest in rabbits are the ventral midline, which is used for cranial mediastinal masses, and the lateral approach which is used for lung lobectomy. Postoperative management is vital for a successful outcome.

For thoracic surgery in rabbits, venous access is essential. Crystalloid fluid therapy is recommended during surgery at a standard anesthesia rate of 10 ml/kg/hr. An intravenous or intraosseous catheter is also needed if there is a cardiac emergency or for blood transfusion. The average blood volume of a rabbit is 57 ml/kg BW and loss of 15%–20% of the blood volume results in a dangerous catecholamine release. Hemodynamic collapse and shock occur after loss of 20%–30% of the blood volume. While rabbits do have different blood types, typing is not routinely done. A cross-match is recommended before administering a transfusion. It is best to be prepared to administer a transfusion before beginning thoracic surgery in rabbits. Once there is a need, there is no time to identify a donor, collect blood from the donor, and administer the blood to the patient. If blood is not available, a colloid or a blood substitute (hemoglobin-based) might be beneficial.

Anesthesia

As the saying goes, the best anesthesia is the one the anesthetist is most comfortable with. It is best to use drugs and protocols you are familiar with and comfortable with when performing thoracic surgery in rabbits rather than trying new things. I will review my protocol but must emphasize it is only what I am comfortable with and is not necessarily the "right way."

I give 0.02 mg/kg buprenorphine IM along with 0.05 mg/kg midazolam IM. Most rabbits become calm and relaxed after receiving both buprenorphine and midazolam. It is usually feasible to place an IV catheter before inducing anesthesia. Because nonsteroidal anti-inflammatory drugs can have serious renal consequences if the patient becomes hypotensive, I prefer to give meloxicam after the patient has recovered from the anesthesia. Midazolam can be reversed with flumazenil at 0.1 mg/kg IV and the reversal should be drawn and ready to give in the case of an emergency. Even after 1–2 hrs of surgery, many rabbits are overly sedate in recovery due to the effects of the midazolam, so I reverse it. Once reversed, they are more alert, interactive, and willing to begin eating.

Propofol (10 mg/kg or less) IV rapidly induces anesthesia but animals induced with propofol can become apneic. If the patient cannot be intubated quickly, this could be a problem. Alternatively, anesthesia can be induced with isoflurane or sevoflurane gas using either a mask or chamber. The preanesthetic protocol generally allows for manual restraint without excess patient stress for mask induction.

For thoracic surgery, endotracheal intubation is absolutely necessary. Two commonly used techniques for endotracheal intubation of rabbits are using a semi-rigid endoscope and the blind method. A small semi-rigid

endoscope (1.0–1.5 mm) can serve as a stylet within the endotracheal tube and allow visualization of the glottis. Advance the endotracheal tube (available in sizes as small as 1 mm), while listening for respiratory sounds. When the tube is at the larynx (clear and loud breath sounds), have an assistant place 0.1 ml of 2% lidocaine into the tube and blow on the tube to spray the lidocaine onto the glottis to prevent laryngospasm. Wait 5 minutes for it to take effect. Place the endoscope inside the endotracheal tube and apply sterile lubricant onto the tube. Hold the head tipped back as far as possible and have an assistant pull the tongue out to open the pharynx. Advance the scope into the mouth to visualize the glottis. Direct the scope between the arytenoids and advance the scope into the trachea. You will see the tracheal rings, which will provide a visual confirmation that you are within the trachea. Slide the tube off the scope and remove the endoscope. Confirm tube placement (moving the rebreathing bag, end-tidal CO_2, tube fogging) and secure the tube in place. Because the tube is easily dislodged during patient positioning, etc, consider placing butterfly tape around the tube at the mouth and suturing it to the upper and lower lip to secure it in place.

An alternative technique is blind intubation, which works well in rabbits that weigh more than 1.0 kg. In this size rabbit, you should be able to get a 2.0-mm endotracheal tube placed. These are the smallest firm tubes available. The 1.5- and 1.0-mm endotracheal tubes are not firm and bend when they hit the larynx, making it difficult to place them using the blind technique. There are 3 key hints to success with blind intubation. First, lidocaine is used as described above to minimize laryngospasms. Be sure to wait a full 5 minutes to allow it to work. Tip the head way back so that the path from the front of the mouth through the mouth and into the trachea is a straight shot. Advance the tube to the level of the glottis. At this location, you should hear loud, clear breathing sounds. Erratic, muffled sounds usually indicate the tube is heading down the esophagus. Listen to the rhythm and get in tune with the in, out, in, out… On inspiration (when the arytenoids are open), slip the tube into the trachea. Because of the lidocaine, the patient may not cough or react as the tube is advanced into the trachea. Make sure to confirm the tube is in the trachea as described above.

Once the patient is intubated, it should be ventilated. It is best not to ventilate to pressures greater than 20 cm H_2O. A nonrebreathing system is most commonly used in rabbits and most do not have a manometer, so the anesthetist does not know to what pressure they are ventilating the patient. Anesthetists are more likely to ventilate to pressures that are too high rather than too low. The lungs should only be inflated to a level that prevents atelectasis. The lungs should not be inflated so that they billow out of the chest and become pale to white in color. Mechanical ventilators are available that can accommodate the smaller tidal volumes of such small patients. Be sure to confirm that a particular unit is able to deliver the correct volumes and pressures for patients weighing less than 5 kg. During surgery, having the patient on a mechanical ventilator can be problematic. When dissecting near vital structures, the surgeon does not want lungs billowing up unexpectedly and will ask that the patient be manually ventilated so they can control when the lungs inflate. If the patient is on a mechanical ventilator, make sure the anesthetist knows how to efficiently switch from mechanical to manual ventilation.

In-depth monitoring of the patient's status during surgery is vital. If possible, body temperature, electrocardiogram, ventilation, end expiratory carbon dioxide, blood pressure, hemoglobin saturation with oxygen, and blood gas levels should be monitored during the thoracic surgery and appropriate measures taken if clinically important alterations occur. If direct blood pressure monitoring is available, it is relatively easy to place an arterial catheter in rabbits. They have a medial saphenous artery that can be catheterized. This will allow blood pressure to be monitored during the procedure. Indirect blood pressure monitors are less reliable but can document trends in the patient's blood pressure.

Thymoma and Thymic Lymphoma

In rabbits, the thymus persists in adults and is easily identified cranial and ventral to the heart on both the right and left sides. Thymomas arise from the thymic epithelial cells and can be subdivided based on the epithelial component of the mass. They are considered histologically benign; however, clinically they are considered benign or malignant based on their invasiveness and prognosis for surgical excision. Metastasis can occur but is quite rare. Because the thymus is a site of lymphocyte production, lymphocytes are a large component of the mass making it difficult to differentiate between thymoma and thymic lymphoma (small mature lymphocytes in thymomas, large lymphoblasts in thymic lymphoma). While one report indicates they occur in young rabbits,[1] clinical reports indicate they occur most commonly in older pet rabbits (6–10 years old) and they have been reported to comprise 8% of neoplasms in 55 rabbits.[2] In 3 rabbits, a genetic basis for thymoma has been identified and was associated with immunodeficiency and hemolytic anemia.[3]

Clinical signs of thymoma are related to the mass effect. In humans, dogs, and cats, dysphagia, regurgitation, and coughing are common; however, these are not reported in rabbits. Other clinical signs are related to precaval syndrome (the result of compression of the cranial vena cava) and these signs are most common in rabbits along with respiratory compromise secondary to the mass compressing the lungs. Signs of the precaval syndrome include edema of the head, neck, and front limbs, and jugular distention. Because rabbits have an orbital sinus, many present with exophthalmos with a thymoma. The heart and lung sounds are diminished in the cranial and ventral fields and there is decreased compressibility of the cranioventral chest due to the mass. Hematologic and biochemistry values are often within normal limits. In other species, hypercalcemia has been reported as a paraneoplastic syndrome. In one report in a rabbit, hypercalcemia was described as a paraneoplastic syndrome, but the authors did not address the differences in calcium metabolism between rabbits and other mammals.[4] Hyperglobulinemia and lymphocytosis are also common in dogs but have not been evaluated in rabbits with thymoma.

Thoracic radiographs are strongly indicative of thymoma demonstrating a large cranial mediastinal mass. It is difficult to define the border between the mass and the heart and a misdiagnosis of an enlarged cardiac silhouette can be made. The mass is often large enough to cause caudodorsal displacement of the heart and lungs as well as elevation of the trachea. Myasthenia gravis, which is associated with thymoma development in humans, dogs, and cats, has not been reported in rabbits to date. Computed tomography and magnetic resonance imaging have not yet been adequately evaluated in diagnosing and evaluating thymomas. Ultrasound is a useful diagnostic tool. Thymomas tend to have mixed echogenicity and cavitations. Thymic lymphoma is typically a more homogenous, hypoechoic mass without cavitations. Ultrasound can also direct a core needle biopsy instrument to obtain a core sample (more diagnostic) while avoiding cavitations and major blood vessels. Aspirate cytologic evaluation can be suggestive of thymoma; however, Withrow comments this method is "unreliable" for diagnosing thymomas because aspirate cytologies typically exfoliate the lymphoid, not the epithelial components.[5] Mast cells are also commonly present on cytologic evaluation. Cytokeratin stain is very helpful in diagnosing thymoma because it stains the epithelial and not the lymphoid components.

The size of the mass is not of prognostic value; rather its invasiveness is a significant factor. Ultrasound can be helpful to define major structures involved but is not helpful in evaluating invasiveness. Most thymomas surround but do not invade thoracic structures and shell out easily. In dogs, it is estimated that approximately 70% are resectable (not invasive) and in cats it seems fewer are invasive than in dogs.[5] In humans, 65% are reported to be noninvasive. In the rabbits (approximately 15) from which I have surgically removed thymomas, only one was considered invasive, suggesting they behave more like thymomas in cats.

The treatment of choice for thymomas is surgical excision. The recommended surgical approach is a ventral midline thoracotomy to allow access to both sides of the cranial thoracic cavity.

Ventral Thoracotomy

The first report of surgical excision of a thymoma in a rabbit used a lateral thoracotomy.[6] That rabbit survived the surgery but died in the immediate postoperative period. The disadvantage of trying to remove a large cranial mediastinal mass through a lateral thoracotomy is the limited exposure. A ventral midline thoracotomy allows exposure to both sides of the chest, the important vessels and nerves in the cranial mediastinum (jugulars, carotids, cranial vena cava, brachiocephalics, aorta, vagus nerves, trachea, esophagus, etc), and exposure of the cranial most aspects of the chest. The thoracotomy can be extended caudally as far as needed to achieve the necessary exposure. The sternebrae of rabbits are too narrow to split longitudinally (median sternotomy), so the term median sternotomy is technically not applicable.

The rabbit is placed in dorsal recumbency and the skin incision is made beginning mid-cervical (it must be 2–3 cm cranial to the manubrium) extending caudally along the manubrium to the xiphoid. A common error is not initiating the incision far enough cranially. There is a paired subcutaneous muscle (cutaneous muscle of the neck) that is on each side of midline.[7] The sternohyoideus and sternocephalicus muscles in the cervical region are separated on midline using blunt dissection to the level of the manubrium. Because the mass generally extends to the cranial extent of the thoracic inlet, a hemostat is used to bluntly dissect dorsal to (under) the manubrium to create a space into which scissors can be inserted. Small, blunt scissors (Metzenbaum or Mayo) are inserted and used to cut the costal cartilages at the sternebrae along one side. Rabbits generally have 6 sternebrae and only the first 6 pair of ribs are attached directly to the sternebrae. This dissection is continued to caudal to the heart undermining dorsal to (under) the sternebrae as the dissection progresses caudally. Ideally, the last 1–2 costal cartilages attaching to the sternebrae are not transected to provide more stability to the rib cage/chest postoperatively. This stability provided is also considered to decrease postoperative pain. This may not be possible because the sternebrae do not extend very far caudally and it may not be possible to remove a large mass without cutting all costal cartilages attached to the sternum.

A retractor is used to spread the 2 sides of the chest, providing exposure to the mass. Be careful not to damage the intercostal muscles on the side where the costal cartilages have been cut by putting too much force to open the chest. My preference is to use a Lone Star Stay Retractor (Lone Star Medical Products, Stafford, TX, USA). To remove the mass, dissection must be meticulous and can be time-consuming. Cotton-tipped applicators are useful for lifting the mass away from important structures without damaging blood vessels. Hemostatic clips are also very useful for this dissection. Place 2 clips and cut between them. At the thoracic inlet, these masses generally wrap around the subclavian artery and vein, as well as the jugular veins and carotid arteries. It is best to avoid needing to ligate these vessels.

Once the mass is removed the area is check for hemorrhage and the lungs are checked for air leaks prior to closing. An intercostal chest tube (5–10 Fr) is placed for control of the pleural space postoperatively. The cut ribs are not specifically reattached. Suture is passed through the intercostal muscles, then around the sternebrae and opposite rib in a figure-of-8 pattern. A second layer of sutures is placed in the insertions of the pectoral muscles along the ventral midline in a simple continuous pattern. This layer should create an air-tight seal. The subcutaneous tissues and skin are closed routinely. The chest is evacuated and the removed tissue is submitted for cultures and histologic evaluation to direct long term therapy.

Lung Lobe Abscesses

Lung abscesses occur occasionally in rabbits. In dogs and cats, medical management (long-term antibiotic therapy) and surgical management (lung lobectomy) have reasonably good success rates. It is more difficult to control

abscesses in rabbits with antibiotic therapy only. As such, it is logical to recommend lung lobectomy as part of the management of lung abscesses in rabbits. Clinical signs include lethargy, depression, decreased appetite, weight loss, and other nonspecific signs of illness. Rabbits generally do not cough, but sneezing may be part of the history. Affected rabbits may have a fever and increased white blood cell count on clinical examination. Thoracic auscultation reveals diminished sounds in the affected lung field.

Radiographs are generally consistent with a consolidated lobe. Ultrasound shows a cavitated lesion in the affected lung and will allow collection of fine needle aspirate samples for culture and cytology, allowing diagnostic confirmation. If there is well-aerated lung between the lesion and the ultrasound probe, the air will interfere with accurate imaging. Computed tomography is also very useful especially with contrast. Many of the computed tomography units available acquire images very rapidly, allowing imaging while the rabbit is under sedation. After contrast administration, the center of the abscessed lobe (pus-filled area) will not enhance because it does not have a blood supply, while the area surrounding the pus is associated with inflammation and enhances with contrast. Computed tomography can also be used to guide a needle into the lesion to obtain samples for culture and cytology. It is best to get culture results and initiate antibiotic therapy prior to surgery so that an appropriate antibiotic can be used before manipulating the abscess. If the patient's condition indicates a more urgent need for surgical intervention, a Gram stain may help direct antibiotic therapy. A variety of organisms are isolated from abscesses in rabbits including anaerobic bacteria.

Lateral Thoracotomy

Approximately 44% of rabbits have 12 ribs and 56% have 13 ribs.[8] Only the first 6 are directly attached by the costal cartilage to the sternum and the last 2 usually have no attachment to the sternum (floating). The muscular anatomy of the lateral thorax of rabbits is similar to that of dogs and cats. The latissimus dorsi lies under the skin and over the scalenus and serratus ventralis muscles. The scalenus muscle extends caudally across the fourth intercostal space as a fan-shaped muscle and the caudal end of the last belly inserts on the fifth rib. The serratus ventralis extends farther caudal to about the tenth rib, but its bellies are parallel to the ribs so it is not necessary to transect them for an intercostal thoracotomy. The internal and external intercostal muscles are similar to those of small animals and the intercostal vessels and nerves are along the cranial aspect of the intercostal space.

Rabbits have only 2 lung lobes on the left: the left cranial and the left caudal. The left cranial lobe has a small fissure that defines the cranial portion of the cranial lobe and the caudal portion of the cranial lobe. The division is incomplete and both portions are supplied by a single bronchus. On the right side there are 4 lobes: cranial, middle, caudal, and accessory. The right middle lobe is mainly ventral to the right cranial lobe. The accessory lobe is caudal to the middle lobe and ventral to the caudal lobe, lying along the esophagus and caudal vena cava.

The location of the lateral thoracotomy will depend on the location of the lesion; however, for a lung lobectomy a fourth or fifth intercostal thoracotomy is generally used because the hilus is in this area. For caudal lobes a fifth intercostal thoracotomy is used. As an external landmark, the caudodorsal border of the scapula is at approximately the level of the fourth intercostal space. The incision is made through the skin and subcutaneous tissues. The location of the desired intercostal space is identified and the latissimus dorsi muscle incised from ventral to dorsal, transecting the ventral two-thirds of the muscle. Bleeding from the vessels within the muscle can be controlled with electrosurgery or hemostatic clips. Once the latissimus is incised, insert your finger under it cranially until you can curl it around the first rib into the thoracic inlet. Then count intercostal spaces caudally until you confirm that you know the location of the correct intercostal space. The serratus ventralis is separated at the appropriate intercostal space and the scalenus is transected along the fifth rib (if a fourth intercostal thoracotomy). The intercostal muscles are incised, being careful not to cut the internal thoracic vessels ventrally and the azygous vein dorsally. Once the chest is entered, manual or mechanical ventilation is required.

The affected lung lobe is identified and elevated out of the chest cavity. It is very important to do this gently if there is an abscess to minimize the risk of pus going out the bronchus into other lung lobes! Do not squeeze or manipulate the affected lobe excessively. The bronchi and pulmonary artery and vein are small and can be ligated, clipped, or sealed (Harmonic Scalpel or Ligasure). If a portion of the lobe is not affected, a partial lobectomy can be performed using a thoracoabdominal stapling device. The stapler will seal across the pulmonary parenchyma, allowing a portion of the lung to be transected and removed distal to the rows of staples. Carefully inspect the stump for hemorrhage and air leaks prior to closure. Consider placing an intercostal nerve block for postoperative analgesia prior to closing the chest cavity. Place an intercostal chest tube as described for control of the pleural space postoperatively.

I have found intracavitary bupivicaine to be helpful in the postoperative period. The first dose can be given prior to closing the thoracotomy. The maximum dose of bupivicaine is 2 mg/kg. This can be divided, with some being used for an intercostal nerve block and the rest being placed into the chest cavity as an intracavitary block. For intracavitary anesthesia, the bupivicaine is injected via the chest tube. The volume is small and will need to be flushed into the chest with saline. Before putting the bupivicaine into the tube, determine the volume of saline needed to completely fill the chest tube. After injecting the bupivicaine into the tube, flush it all into the chest with that volume of sterile saline.

Place sutures around the ribs cranial and caudal to the thoracotomy and pre-place them before tying any. Do not over-tighten them. Remember there is normally a space (intercostal) between ribs so they are not meant to be touching or overlapping. Close the scalenus (if transected) and serratus ventralis, then the latissimus dorsi with a simple continuous pattern of an absorbable material. The removed tissue is submitted for cultures (aerobic, anaerobic, and fungal) and histologic evaluation to direct long-term therapy.

Postoperative Care

During recovery from anesthesia, administer another dose of buprenorphine (0.02 mg/kg). If bupivicaine was used as an intercostal block or intracavitary prior to closing, do not administer additional bupivicaine for 8 hours. Intracavitary bupivicaine can be repeated every 8 hours. Instill no more than 2 mg/kg of bupivicaine into the chest tube followed by the predetermined volume of sterile saline. Bupivicaine provides topical anesthesia for approximately 8 hours, so it should not be used more frequently. Record the volume of both bupivicaine and saline put into the chest so when fluid and air are evacuated via the tube, you know the volume of fluid you put into the chest. The chest tube should be aspirated for fluid and air (record amounts recovered) every hour, or less initially. The interval is extended if the amount of fluid and air is minimal. Remember the presence of a chest tube will induce an inflammatory reaction that will result in pleural effusion at a rate up to 2 ml/kg/day.

Administer meloxicam (0.1–0.2 mg/kg PO q24h) once the rabbit has recovered for anesthesia and there is little risk of a drop in systemic blood pressure. Continue IV fluids at a maintenance rate until the rabbit is eating and drinking well (at least overnight). Offer food as soon as possible in recovery. If the patient does not eat within 4 hours, institute syringe feeding to minimize the risk of gastrointestinal ileus. Consider using prokinetic agents such as metoclopramide if there is concern about gastrointestinal stasis. Continue antibiotic therapy for 10–14 days.

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Maxillary Cheek Tooth Elodontoma in a Guinea Pig

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Session #175

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Abstract: Elodontoma of a maxillary cheek tooth was diagnosed in a guinea pig presented as a referral for treatment of dental disease. Diagnosis and treatment included diagnostic imaging, intraoral dental procedures, and extraoral extraction. Odontogenic tumors (odontomas) and other dysplastic changes (pseudo-odontomas) of incisor teeth have been reported in squirrels and other rodent species. This is the first reported case of odontoma in a guinea pig, and the first reported case affecting an elodont cheek tooth.

Introduction

All rodent species have one pair of well-developed, strongly curved maxillary and mandibular incisor teeth, representing the most known anatomical peculiarity of this order.[1–3] Incisor teeth vary in shape, length, color, and thickness among species and are elodont (open rooted, continuously growing) teeth. All rodents lack canine teeth and there is a diastema between the incisor and the first premolar (or molar) tooth. Premolar and molar teeth are anatomically indistinguishable, and are therefore simply called "cheek teeth."[1] The hundreds of species belonging to the Rodentia order are grouped into 3 suborders ("guinea pig-like," "rat-like," and "squirrel-like"). These suborders have different dental formulas, but from the standpoint of the dental anatomy and physiology, they can be simply reduced to 2 groups. The porcupine-like rodent species have both elodont incisor and cheek teeth. The other 2 groups have elodont incisor teeth, but anelodont (rooted, not growing throughout life) cheek teeth.[1] A very important anatomical peculiarity of cheek teeth of guinea pigs is that they are curved; the mandibular have a buccal (lateral) convexity, while the maxillary have a palatal (medial) convexity.[1] This results in a 30^{o} oblique occlusal plane that normally slopes from dorsal to ventral, lateral to medial. Incisor teeth of squirrels are much bigger, longer, and thicker than incisor teeth of the other 2 groups.

Guinea pigs are commonly presented with symptoms suggesting dental disease or directly related to it, such as reduced food intake, anorexia, or dysphagia.[4] The primary cause of dental disease in the guinea pig is insufficient or improper wearing of cheek teeth due to inappropriate diet, in particular, due to lack of fiber. The history of guinea pigs with dental disease often includes improper feeding of seed mixes and diets low in fiber. [4]

A slight alteration of the sloped occlusal plane of cheek teeth is enough to hamper chewing, and a slight overgrowth of clinical crowns is enough to interfere with movements of the tongue and swallowing. This is why guinea pigs are more prone than rabbits to apparent onset of anorexia due to dental disease.[4]

Malocclusion of incisor teeth often presents as excessive elongation or lateral deviation of the clinical crown of mandibular incisors. As primary incisor malocclusion is rare and occasionally follows traumatic injuries, dental disease of cheek teeth should be investigated and addressed as well. In most cases, malocclusion of incisor teeth is actually secondary to acquired dental disease of cheek teeth. [4]

Complete inspection and proper diagnosis of guinea pig dental disease should be performed under general anesthesia. Effective anesthetic protocols for induction and maintenance of general anesthesia for this species, which cannot be easily intubated, are reported elsewhere.

Diagnosis of dental disease in guinea pigs requires 2 modalities: radiology and oral endoscopy.[4] A very good to optimal complete radiologic study must include 5 basic projections (lateral, oblique in 2 directions, ventrodorsal, and rostrocaudal) using very high definition cassettes and films such as those for mammography, as standard radiographic cassettes and films will not produce adequate detail.

Thorough inspection of the oral cavity is greatly enhanced by oral endoscopy. Other magnification devices are helpful but not always sufficient, and many lesions can be missed without the help of stomatoscopy. Modern spiral computed tomography (CT) scan are capable of excellent details also in small species like guinea pigs.[5–8] In particular, 3-dimensional volume and surface renderings of the skull can provide tremendous information for diagnosis of dental disease or related problems in these species, like abnormalities of the temporomandibular joint.

As with rabbits, excessive crown elongation and malocclusion of cheek teeth arcades is common in guinea pigs, but with different characteristics. Due to the peculiar orientation of the cheek teeth, mandibular cheek teeth crowns always elongate lingually, and maxillary crowns always laterally. [4]

Overgrowth of clinical crowns is not as evident as it is in rabbits, and spike or spur formation is much less frequent. Mandibular cheek teeth tend to bend lingually over the tongue, hampering movements. A typical "bridge-like" malocclusion occurs when the first mandibular premolars occlude or even cross each other. The sharp border of the entire maxillary arcade can create severe discomfort to the buccal mucosa. It should be kept in mind that even very early malocclusion and subsequent alteration of the sloped occlusal planes is enough to elicit reduced food intake or anorexia in this species.[4]

While most disease of the cheek teeth of guinea pigs involves the clinical crowns, periapical changes are occasionally seen as well. Bony tumors of the skull and teeth have not been reported in the guinea pigs.

Unless the guinea pig is presented in very poor general condition, the prognosis for treatment of dental malocclusion of cheek teeth is fair to good. Malocclusion of incisor teeth is usually addressed as a consequence, and extraction is rarely needed. Unlike rabbits, guinea pigs may not improve immediately after a dental treatment, due to stretching of the masticatory muscles, dysfunction of the temporomandibular joint, and associated pain and inflammation. Patients that do not immediately return to eating may not wear teeth down to the degree required to prevent repeated overgrowth. These patients may require additional dental treatment until the soft tissues heal and the patient is able to eat enough high-fiber food to allow normal wearing.

Occlusal adjustment of cheek teeth in guinea pigs should be performed with the tip of a delicate, slightly abrasive bur. [4] The goals are to shorten the elongated clinical crowns, and to restore the proper oblique occlusal plane, which is a critical aspect of treatment in this species. Unfortunately, this procedure does not positively affect the function of masticatory muscles and temporomandibular joint. Due to the normal curvature of cheek teeth in guinea pigs, extraction is virtually impossible, unless the tooth is loose secondary to periodontal infection. Unfortunately, diseased teeth fracture easily, making complete extraction extremely difficult. Extraoral extraction of cheek teeth has been reported in rabbits and chinchillas.[9] This technique has not been reported in guinea pigs, but surgical anatomy and basic principles of surgery are similar to the abovementioned species.

Odontomas have been reported in dogs, horses, primates,[10] and many rodent species, both companion and laboratory animals: rats,[11] mice,[12] prairie dogs,[2,4,13–17] degus,[18] and tree squirrels.[19,20] The author also encountered radiographic abnormalities consistent with this lesion in a pet rabbit, a chinchilla, a chipmunk, and a citellus *(Spermophilus citellus)*. In all rodent cases, the incisor teeth were affected. Odontoma has not been previously reported in guinea pigs.

Terminology about this dental disease has not been clear in the past. The term "odontoma" was meant to include both odontogenic tumors (invasive and locally destructive) and the odontogenic dysplasia of incisor teeth, which frequently occurs in prairie dogs and other squirrels (mostly locally compressive on the cranial portion of nasal cavities). According with the *Histological classification of tumors of the alimentary system of domestic animals,* 2 different kinds of odontomas are classified.[10] The non-neoplastic, dysplastic malformation that occurs when normal tooth eruption is impaired or arrested has been properly named *pseudo-odontoma*.[2] Apical growth continues, causing primary deformation of the apex and of the reserve crown, and secondary abnormalities of the surrounding structures like the incisive bone. The outcome is severe apical deformity and folds of newformed tooth substance, especially on the labial surface of the reserve crown, acting as a space-occupying mass and leading to progressive obstruction of the nasal openings.[1,14]

The term *elodontoma* has been suggested because those dental changes occur with elodont incisor teeth.[20]

The pathogenesis of odontomas in rodents is not clear. Different hypotheses have been proposed, including osteopetrosis (an autosomal recessive disease) in rats and mice.[11,12] Unlike the case in rabbits, metabolic bone disease has not been investigated and reported as an underlying cause of dental disease in rodent species.[4,14] Nevertheless, the author has diagnosed and treated dental dysplasia of a mandibular incisor tooth in a citellus with concerrent metabolic bone disease.

In prairie dogs and other squirrels, repeated trauma, fractures, and subsequent acquired dental disease are the most important cause of dysplastic changes (pseudo-odontomas). Trauma can be the result of constant chewing of cage bars, fractures, or improper trimming of maloccluded incisor teeth.[1,2]

The most common presenting symptoms in prairie dogs and other rodents are reduced activity, reduced food intake and stool production, and respiratory distress.[1,2,4,13,15–18] True dyspnea is common, but milder symptoms include sneezing or a unique snoring sound sometimes referred to as "reverse sneezing." Respiratory symptoms are related to reduced nasal air passage in obligate nasal breathing species.[1,2] Weight loss or emaciation can often be present at this stage as well.

Different surgical techniques have been reported for treatment of pseudo-odontomas in prairie dogs.[2,4,13,17] Extraction of incisor teeth through an extraoral approach is similar to that described for rabbits.[13] Nevertheless, this technique is rarely feasible because of severe deformity of both the reserve crown and apex of the affected tooth. Alternative approaches are intraoral transpalatal; extraoral lateral, creating a fenestration of the incisive bone; and extraoral dorsal by rhinotomy.[2,9] Palliative treatment by dorsal rhinostomy and positioning of a tubular stent can be considered when radiographs or CT reveal that the maxillary incisor teeth are fractured, or when the preoperative plan is to minimize risks related to anesthesia and surgical treatment.[4,17] It should be considered this surgical option does not stop the dysplastic or the neoplastic process.

Case Report

History

A 3.5-year-old, neutered male guinea pig *(Cavia porcellus)* was presented for a second opinion. Basing on clinical symptoms related to dental disease, the guinea pig underwent oral inspection and coronal reduction of cheek teeth under general anesthesia. Little to no clinical improvement was achieved after dental procedures, therefore the guinea pig was referred for additional diagnostics performed by CT of the skull. Basing on the tomographic report, intraoral extraction of the right maxillary premolar tooth was performed. Again, very little improvement

occurred, and the guinea pig was sent for the second opinion. During this period, the guinea pig was fed with greens and supported with Oxbow Critical Care.

Computed tomography performed before extraction revealed severe bone lysis and periosteal reaction of the right maxilla, cranial to the zygomatic arch. Involvement of the cranial portion of the right nasal cavity was present as well. Both the clinical and reserve crown of the right maxillary premolar were abnormally elongated. The zygomatic arch and the orbital fossa were not involved.

Those radiographic signs were consistent with periapical infection and possible osteomyelitis of the maxillary bone following dental disease of right maxillary CT1. Differential diagnosis included bony neoplasia.

Physical exam and diagnostic tests

On physical exam, the guinea pig was bright, alert, and responsive. Body weight was 850 g. A hard and moderately painful swelling was present at the right zygomatic area, associated with mild epiphora (Fig 1). Sneezing, breathing noise, nasal discharge, or other symptoms related to involvement of nasal cavities were not present. The rest of the physical exam was unremarkable.

Figure 1. Facial swelling in the zygomatic region and mild epiphora. Used by permission from Vittorio Capello, DVM.

The guinea pig underwent general anesthesia to obtain skull radiographs, collect a blood sample, and perform oral endoscopy. The blood work was unremarkable. Skull radiographs showed evident bone lysis and periosteal reaction cranial to the right zygomatic arch in all of the 5 standard projections (in particular, the vendrodorsal). The oblique projections did not show abnormalities of the apices of cheek teeth. The clinical crown of the right maxillary premolar was missing because of previous extraction, but part of the reserve crown was apparent radiographically (Fig 2a). The occlusal plane of cheek teeth arcades was normal.

Oral endoscopy demonstrated a slight overgrowth of the left mandibular CT1 and absence of the clinical crown of right maxillary CT1 because of previous extraction by the referring veterinarian. The gingiva had healed over it. No interproximal space, fistula, or pus were present.

The diagnosis based on physical exam and diagnostic imaging was osteomyelitis of the maxillary bone following dental disease of right maxillary CT1. Differential diagnoses included bony neoplasia. Surgical debridement of the bone through an extraoral approach was recommended at this point.

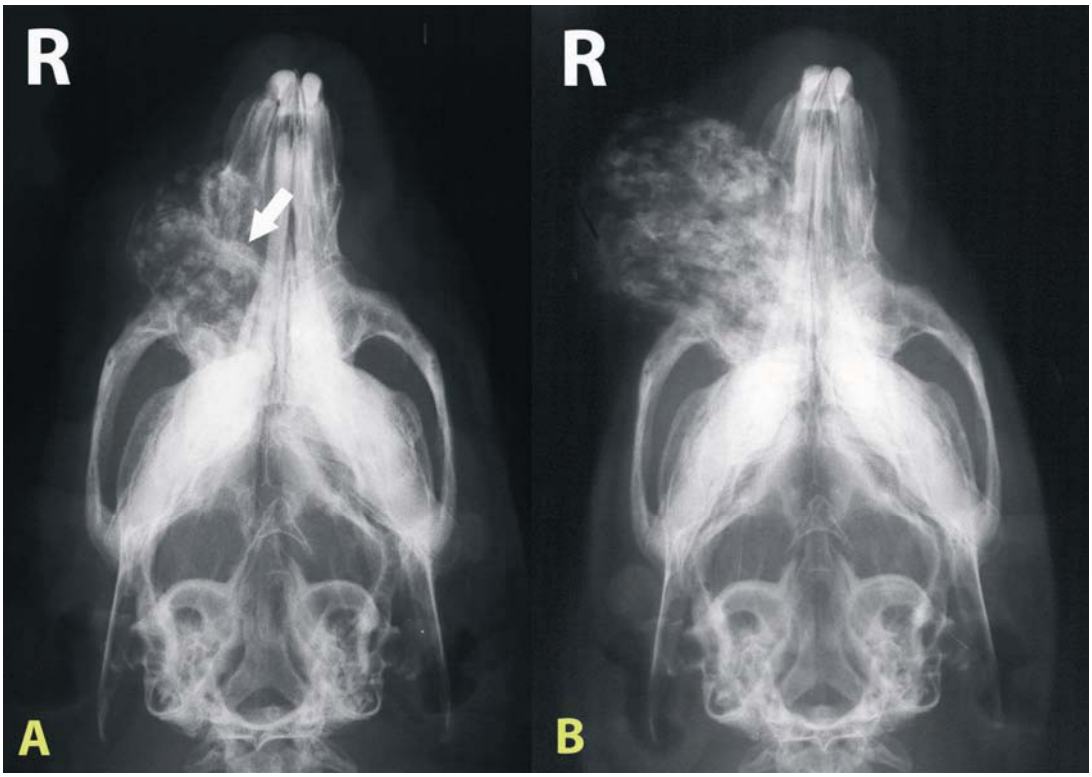

Figure 2. Radiographs of the skull, ventrodorsal projection. A: Collected during second opinion. Bone lysis, periosteal reaction, and reserve crown of the right maxillary premolar (arrow) are visible. B: Collected during anesthesia before euthanasia. Bone abnormalities have increased in size. Used by permission from Vittorio Capello, DVM.

Follow-up and surgical treatment

No significant clinical improvement occurred during the following weeks, and the guinea pig was generally fine, even if unable to eat completely on his own. Because the facial swelling was slowly increasing, exploratory surgery was elected and performed a month after the second opinion.

The capsule of the swelling was exposed by blunt dissection in the zygomatic area, and dissected close to the bony attachment. No pus was present, but the capsule included fibrous tissue. After thorough debridement, extraction of the apex and the reserve crown of maxillary CT1 was performed (Fig 3).

No fistula was present through the oral cavity or to the nasal cavity. The surgical site underwent a thorough surgical debridement and flushing, and despite no evident purulent infection, the surgical procedure was finalized with marsupialization of the surgical site in order to elicit healing by secondary intention. Culture/sensitivity testing was not performed at this point because there was no pus. A tissue biopsy sample was not performed at this time because of the clear presence of a "foreign body" (the tooth) and the desire to prevent formation of a larger cavity.

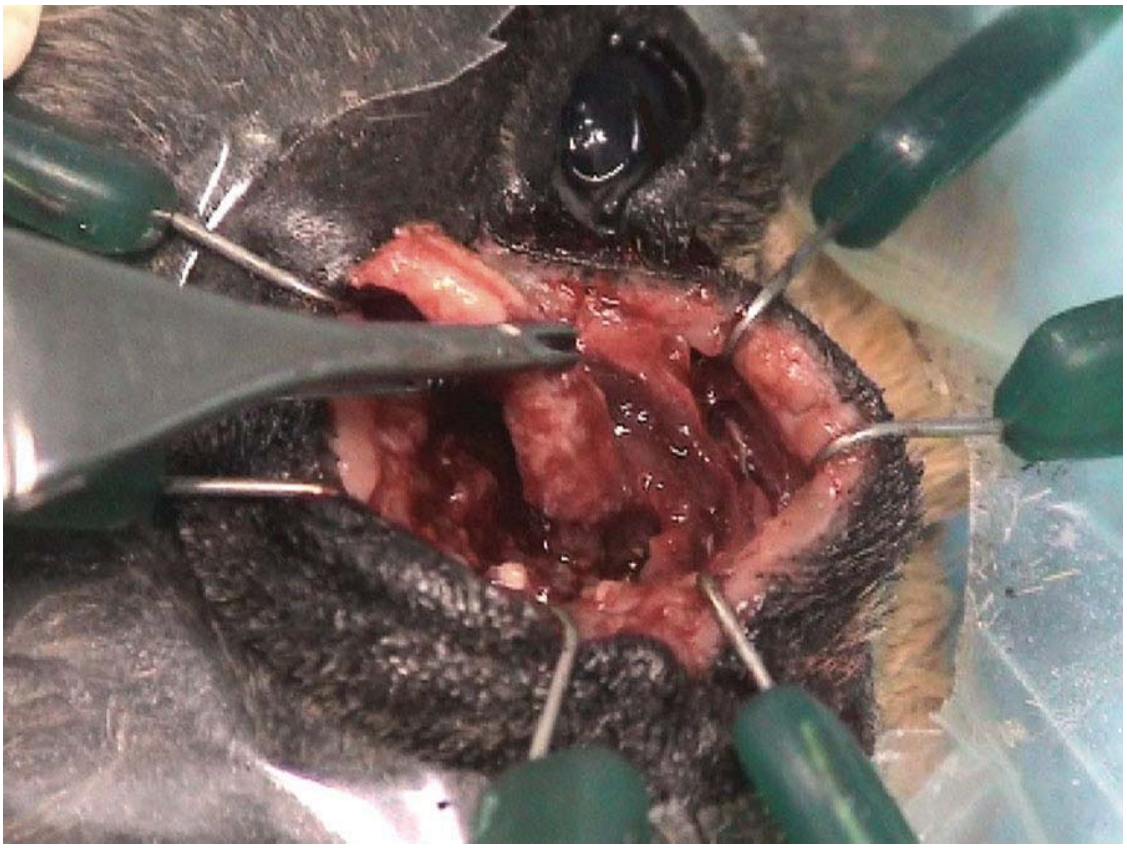

Figure 3. Extraoral extraction of the right maxillary premolar. The head of the guinea pig is positioned in lateral recumbency, with the nose at the bottom of the image. The apex of the tooth is on the top left. Used by permission from Vittorio Capello, DVM.

The diagnosis at this stage was periapical granuloma following dental disease, fracture, and incomplete extraction of tight maxillary premolar tooth. A postoperative oral endoscopy recheck showed a mild decrease of the palatal swelling, after removal of the tooth, and postoperative radiographs clearly demonstrated the complete removal of the diseased maxillary tooth.

The guinea pig underwent daily flushing and debridement of the surgical site. On day 21 after surgery, the surgical site was almost completely healed. An intraoral recheck did not show any significant difference with the 2 stomatoscopies performed earlier, and the palatal swelling was stable. Skull radiographs showed increased radiodensity, with increased deposit of calcium. Differential diagnosis of neoplasia was reconsidered, and a tissue biopsy was recommended, but the owners declined this option.

During the following days the patient was stable, with no clinical improvement. Labored breathing due to compression on the right nasal cavity varied from day to day; breathing eased with nebulization.

Overall patient conditions worsened during the following 4 weeks, mostly because of increased labored breathing, and the owners elected euthanasia. Necropsy was not performed, but a bisopsy sample was submitted for final diagnosis. Histopathology reported neoplastic conglomerates of haphazardly arranged odontogenic hard and soft tissues. The neoplastic mass was infiltrative and consisted of odontogenic epithelium closely associated with dentine and cementum-like mineralized tissue. The overall histopathologic pattern was consistent with complex odontoma (Fig 4).

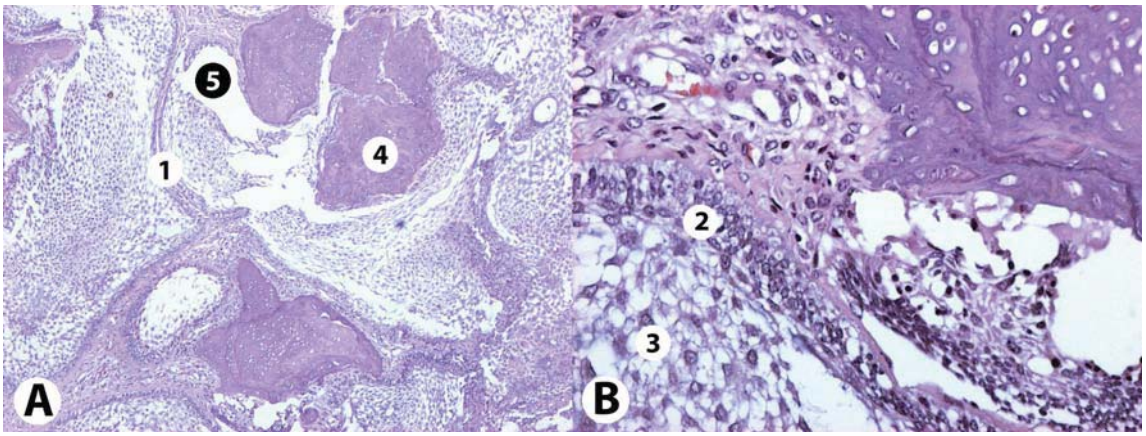

Figure 4. Histopathology of the complex odontoma (A: x100; B: x200) The neoplastic mass (2 x 2.5 cm) has moderate cellularity, is not encapsulated, is infiltrative, and extends to the cutting borders, with moderate fibrous stroma.

The tumor consists of columnar odontogenic epithelium (A, 1) with prominent peripheral palisading (B, 2) and stellate reticulum-like areas (B, 3) closely linked with dentine and cementum-like mineralized tissue (Figs 2 and 4). Empty spaces (Figs 2 and 5) previously occupied by fully mineralized enamel are also visible after decalcification. Used by permission from Gabriele Ghisleni, DVM, Dipl ECVCP.

Discussion

Clinical signs and symptoms of dental disease in guinea pigs are often nonspecific, and their onset is usually shown through subtle changes, such as reduced food intake or diffferent selections of food.

A thorough dental exam including a full set of radiographic projections and stomotoscopy is recommended. Computed tomography has received great attention in exotic veterinary medicine during the past few years. Thanks to the advent of new spiral CT scanners and the availability of post-capture image manipulation software, this diagnostic imaging modality has become feasible, practical, and extremely useful as an adjunct to standard radiology even in small mammals. Optimal traditional radiographs (both digital and analog) still provide higher resolution, but CT allows visualization of important details impossible to be seen on radiographs. The main advantage of CT over traditional radiography is the visualization of internal anatomy, without superimposition of adjacent and overlying structures; this is particularly useful in evaluating complications of dental disease such as facial abscesses, osteomyelitis, and odontomas. In selected cases, CT provides detailed diagnostic information that is critical for proper prognosis and surgical planning of difficult dental cases.

From the standpoint of imaging, this patient underwent complete diagnostic assessment (including CT), both during the first and the second opinion.

Extraction of cheek teeth is very challenging in guinea pigs because of their peculiar anatomy. This intrinsic difficulty probably led to failure of the first dental extraction procedure, where just the clinical crown was extracted, but the reserve crown remained in situ. Because of the short time between the first dental procedure and the second evaluation, a possible regrowth of the reserve crown appears unlikely.

Extraction of cheek teeth through the extraoral approach has not been reported in guinea pigs, but in this case it was relatively easy and effective, because the apex of the affected tooth was in the core of the zygomatic swelling.

Even if an odontogenic tumor has not been included in the differential diagnosis, a core tissue biopsy performed during surgery might have led to earlier intra vitam diagnosis. Nevertheless, the complexity of an odontogenic tumor requires a good biopsy sample, and postsurgical healing of a marsupialization site is always a clinical concern, especially in a small patient like the guinea pig.

On retrospective, a fine needle aspirate would have not provided definitive diagnostic information.

Postmortem histopathologic diagnosis of elodontoma was interesting, both because odontomas have not been previously reported in the guinea pig and because odontomas have not been previously reported in teeth other than incisors, even considering other rodent species. Nevertheless, it is interesting to note that premolar and molar teeth of guinea pigs are elodont teeth, like the incisor teeth of all the other rodent species. However, the chewing movements and function of cheek teeth are substantially different than those of incisors. Therefore in this case, a pathophysiologic hypothesis of repeated trauma and fractures, a frequent etiology in squirrel-like rodents, is questionable.

It is not clear if the trauma related to incomplete extraction of the right maxillary premolar led to development of the odontoma; alternatively, the odontogenic tumor already in progress could have caused malocclusion of that tooth. The first option is the most likely, considering that CT performed before extraction indicated bone lysis. However, it is impossible to guess if this dental disease started as periapical infection and osteomyelitis, and eventually turned to elodontoma after incomplete extraction.

In addition of being the first reported case of an odontogenic tumor in the guinea pig species, the definition of "elodontoma" fits this case, extending this term also to open-rooted cheek teeth.

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Ferret Preputial Tumors

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Summary Style Manuscript

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Apocrine sweat glands ("scent glands") in domestic species are associated with each hair follicle complex over the body.[1] These also include the specialized apocrine glands such as the ceruminous glands in the ears, glands of Moll in the eyelids, anal sac apocrine glands, and mammary glands. They produce a proteinaceous, white odorless milky fluid that mixes with sebum when expelled to form a protective emulsion acting as a physical and chemical barrier. On the ferret, large numbers of scent glands occur around the head, neck, prepuce, and vulva.[2]

The neoplastic lesions arising from the epithelial component include benign adenomas and cystadenomas, and malignant carcinomas and adenocarcinomas. From the literature the malignant tumors are described as locally aggressive neoplasms with a moderate potential for metastasis.[2] The prepuce is one of the most common sites reported.[3,4]

Preputial apocrine tumors including carcinomas, adenocarcinomas, and cystadenocarcinoma were identified in 42 cases. Although only 18 (42.8%) were listed as neutered males, it is suspected that this number is higher. From a follow-up survey returned on 16 cases, 5 were lost to follow up, 4 had recurrence after surgical removal at 2–4 months, and 4 had no recurrence at follow-up from 6 days to 2 years. Three were euthanized and the sample submitted postmortem. A perineal urethrostomy was performed in 3 cases, 2 of which had tumor recurrence.

A tumor mass involving the prepuce of ferrets should warrant prompt surgical removal with wide and deep surgical margins.

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Deslorelin Implants as an Alternative for Surgical Castration in Ferrets

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Session #180

Summary Style Manuscript

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Deslorelin implants are shown to be a good alternative for surgical castration in male laboratory ferrets.[1] However, the effect in female ferrets, as well as the longevity of the implants in both sexes, has not been demonstrated. In this study, a telephone survey was used to monitor the longevity and potential side effects of an implant containing 4.7 mg deslorelin in 132 privately owned ferrets (72 ♂, 60 ♀).

Owners were very satisfied with the effect of chemical castration in their ferrets. In male ferrets, the duration of action (from 301 to over 1249 days) was reported to be lower compared to that in females (637 to 872 days) at the time of the phone call. This might be the result of owners determining the effectiveness of the implant based on sexual behavior. Hormone concentrations, however, were measured in 2 ferrets in which the owner claimed the implants were no longer functioning. All androgen concentrations were below the detection limit of the assay. It is therefore likely that the implants were still functional in these ferrets. The duration of action may thus be at least 2 years in both sexes.

Because deslorelin induces estrus in bitches,[2] a double-blind, placebo-controlled study was performed to see whether 2 mg medroxyprogesteron acetate or a placebo, given 2 days prior to placement of the implant, would prevent estrus in jills (n = 27). No significant difference was found between the 2 groups.

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Gabapentin for Tail Trauma in a Syrian Hamster (*Mesocricetus auratus*)

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Session #183

Summary Style Manuscript

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Gabapentin, designed as an anticonvulsant, has gained widespread use in human medicine[1] and recently in veterinary medicine for treating neuropathic pain[2] and allodynia. While research with this drug has been conducted in rats and mice for human medicine, little information is available on its clinical use in treating rodents for pain. This case illustrates the effect of this drug as part of a multi-modal protocol for treating tail trauma in a 20-month-old female Syrian hamster (*Mesocricetus auratus)*. The hamster presented with a presumptive self-traumatized lesion that appeared as an ulceration on the distal end of the tail. It was placed on trimethoprim/sulfamethoxazole (TMS) and was discharged. Over the next 5 days, the mutilation continued despite the addition of buprenorphine and meloxicam. The hamster's tail was now chewed to less than 0.5 cm in length. It was hospitalized for monitoring and treatment. Gabapentin, which was specially formulated by the pharmacy at Western College of Veterinary Medicine (The University of Saskatchewan, Saskatoon, Canada), was added to try and stop the wound from progressing. There was no published dose for hamsters or myomorph rodents, so dosing was extrapolated by combining the information available from research dosages on rats and mice[3–5] with published dosages for treating pain in dogs and cats.[6] The dosage given to the hamster was 50 mg/kg PO q24h. After adding gabapentin, the wound improved. Three laser treatments, 1 application of silver sulfadiazine, and 1 acupuncture procedure were performed. However, these treatments were not continued after discharge. The hamster went home on gabapentin, meloxicam, and TMS and improved until full resolution.

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Canine Distemper: a Review and Case of Canine Distemper-like Disease in a Vaccinated Ferret

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Session #185

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Abstract: Canine distemper virus, a ubiquitous *Morbillivirus*, causes significant morbidity and nearly 100% mortality in domestic ferrets (*Mustela putorius furo*). Vaccination is routinely performed providing protection against clinical disease; however, vaccine efficacy is dependent on host, vaccine, and viral factors. Positive antibody titers result from natural exposure to canine distemper virus and vaccination. A suspected case of canine distemper in a vaccinated ferret will be presented to illustrate the usefulness of titers when evaluating ferrets in the clinical setting.

Introduction and Review

Canine distemper virus (CDV), a *Morbillivirus* in the family Paramyxoviridae, is a cosmopolitan large enveloped single-stranded RNA virus and is closely related to the measles virus. Canine distemper is a disease affecting multiple families in the Order Carnivora including Canidae, Mustelidae, Procyonidae, Hyaenidae, Mephitidae, Ailuridae, Viverridae, Ursidae, Obobenidae, Otariidae, and Phocidae.[1] Several large cats in the family Felidae are also known to be susceptible to CDV. Transmission through aerosolization of respiratory exudates is most often implicated in transmission of CDV, but the virus can also be isolated from tissues and secretions, including urine. Affected animals can shed CDV for 60–90 days postinfection and contact with infected individuals, whether or not they are showing clinical signs of disease, is responsible for the perpetuation of canine distemper in animal populations.[1] The virus is not resistant to environmental factors; exposure to ultraviolet light, temperatures greater than 50°F, drying, and routine disinfection processes are usually effective in killing the virus.

Canine distemper in dogs is highly variable with reports of 25%–75% of infected dogs clearing the infection without developing clinical signs of disease.[1] Factors determining susceptibility and severity of disease include the age of the animal, immune status including presence of maternal antibodies, and viral virulence. Infection results when body secretions from an infected animal come in contact with a susceptible animal's respiratory epithelium. Transmission can also occur through direct contact with an infected individual or through contact with contaminated fomites.[2] Within 24 hours, viral replication occurs in host macrophages and spreads to local lymphoid tissue. Clinical signs develop 4–6 days postinfection as viral multiplication occurs systemically. Clinical signs include high fever and leukopenia secondary to viral damage to lymphoid cells. By day 8 postinfection, dogs with poor immune status undergo hematogenous spread of CDV to epithelial and central nervous system tissue, allowing the virus to be shed in body secretions. Clinical signs associated with viremia are often severe and may lead to death. Dogs that mount an appropriate immune response and develop antibodies to CDV may have a resolution of clinical signs around 2 weeks postinfection. Viral spread to the central nervous system most likely occurs in all viremic dogs but is dependent on the degree of the animal's immune response. Central nervous system signs may develop days to months post recovery and are typically progressive leading to the demise or euthanasia of the dog. Recovery from canine distemper in dogs is associated with subsequent immunity however this protection may be compromised if the animal is exposed to a highly virulent strain of CDV, a large quantity of virus, or if the dog is immunocompromised or stressed.[1]

In 1926, experiments demonstrated that CDV was the same virus responsible for distemper in ferrets (*Mustela putorius furo*) and proved that airborne transmission between ferrets and dogs was readily achieved.[3] Distemper in ferrets is generally considered nearly 100% fatal to infected animals with an incubation period of 7–10 days.[2] Clinical signs of distemper in ferrets don't vary significantly between affected individuals.[1] Hyperthermia is often the first clinical sign with temperatures >105°F (40.5°C) in infected ferrets. Listlessness, serous oculonasal discharge, and anorexia appear early in clinical course of disease. Within 1–2 days, erythema and rash on the chin and in the inguinal region appear. Erythema and hyperkeratosis of the foot pads may or may not develop.[2] An orange tinge to the skin may occur in animals that develop secondary bacterial dermatitis. The character of the ocular and nasal discharge changes to mucopurulent as the disease progresses and by the third day of clinical signs, the eyes may be crusted shut. Pronounced weakness with persistent fever and anorexia is often evident by day 5 of clinical signs. Secondary bacterial pneumonia, maldigestion and diarrhea, and melena often compound canine distemper in ferrets. Death occurs around 2 weeks postexposure, 5–7 days after the onset of clinical signs, although this varies depending on the strain of CDV.[2] In the early studies documenting canine distemper in ferrets, neurological signs were reportedly seen in 7% of ferrets and developed as early as the fourth day of clinical signs (14 days postexposure).[3] The affected ferrets generally had more mild clinical signs and may have been showing signs of recovery. Neurological manifestation of canine distemper in ferrets is characterized by ataxia, muscle tremors, seizures, and coma. As in dogs, these signs are progressive and fatal in the ferrets. Treatment of canine distemper in ferrets is supportive, with antibiotic therapy implemented to combat bacterial infections that occur secondary to immunosuppression.

Diagnosis of distemper is often based on signalment of the animal, history, including vaccination history, and clinical signs. Antemortem diagnostic testing includes hematology, immunocytology, and immunologic testing.[1] Because of the predilection of CDV for lymphatic tissue and the subsequent tissue destruction caused by the cell-mediated response, an absolute lymphopenia plus or minus a leukopenia is often apparent. Inclusions consistent with CDV may be seen in circulating lymphocytes when stained with Wright's stain.[1] Serum chemistry analysis results are non-specific but may reveal a low albumin/globulin ratio. Immunofluorescent antibody (IFA) testing of infected tissue may be positive for CDV. Acutely, IFA may detect antigen in the buffy coat as early as 2–5 days postinfection but is not effective after 14 days due to neutralization by the rising antibody titer. Conjunctival scrapings may also be positive during this time period; however, false-negative results are common. Viral detection by IFA may be possible using epithelial tissue such as skin and foot pad for at least 60 days.[1] Viral isolation is challenging but reverse-transcriptase-polymerase chain reaction to detect viral antigen has been used successfully to detect CDV in infected ferrets.[4]

Vaccination against canine distemper is routinely recommended for ferrets. Historically, modified-live virus (MLV) vaccines were used to induce protection in naïve ferrets to CDV. Fervac-D (United Vaccines, Inc, Madison, WI, USA) was a MLV vaccine attenuated through serial passes through chick embryo cell culture. However, potential for reversion to virulence is always possible with MLV vaccines. Use of multivalent vaccines developed for use in domestic dogs have been used but may represent risks related to vaccine-induced disease or failure to infer protection.[2] Purevax Ferret Distemper vaccine (Merial, Inc, Athens, GA, USA) is currently the only USDA-approved canine distemper vaccine for ferrets. This vaccine uses a recombinant canarypox vector to express viral glycoproteins of CDV and induce immunity in the vaccinates. Vaccine efficacy is dependent on multiple factors including vaccine handling, competence of the host immune system, and virulence of the pathogen. Complete protection against infection is not attained with any vaccine. Maternal antibody to CDV in the kit will interfere with vaccine-induced immunity. The half-life of maternal antibody to CDV in ferrets is 9.43 days, resulting in a recommended vaccination schedule for kits at 6, 10, and 14 weeks of age.[5] Research demonstrates that despite decreasing titer to CDV in dogs, immunity to canine distemper post booster vaccination lasts as long as 7 years.[1] Current American Animal Hospital Association guidelines recommend one canine distemper vaccination at a year of age followed by revaccination every 3 or more years (www.aahanet.org/PublicDocuments/VaccineGuidelines06Revised.pdf).

Typically, annual boosters are recommended for ferrets 1 year of age and older. However, ferrets challenged with CDV following vaccination with MLV vaccine demonstrated immunity for greater than 5 years.[6]

Serum neutralizing antibodies to CDV are evident in ferrets with natural exposure and postvaccination. A correlation between antibody titer and protection against canine distemper has not been established in the ferret but is thought to be ≥1:100 in dogs.[7] A study evaluating MLV and killed vaccines in black-footed ferret hybrids resulted in signs of canine distemper following virus challenge in 3 ferrets that had been vaccinated with the killed vaccine and had serum neutralizing antibody titers of 1:32, 1:64, and 1:256, respectively.[8] In 2 independent studies, serum neutralizing antibody titers to CDV in naïve ferrets were <1:4.[4,9] When comparing inactivated, MLV, and canarypox vectored recombinant CDV vaccines, protective immunity was provided by all 3 against canine distemper in ferrets when challenged with CDV. Serum neutralizing antibody titers to CDV were similar for each vaccine type after 2 vaccinations (inactivated: inverse geometric mean (GM) 229, reference range 128–256; MLV: GM 276, reference range 172–512; canarypox: GM 266, reference range 96–512).[9] Additionally, antibody titer to CDV did not change postchallenge in the ferrets vaccinated with the MLV vaccine but did rise slightly in ferrets vaccinated with inactivated and canarypox vaccines.

Case Report

On May 4, 2009, a 4.5-year-old female spayed ferret presented to the University of Illinois Veterinary Teaching Hospital Emergency Service for acute onset of profound weakness and disorientation. The ferret was privately owned and housed in a small room with another female spayed ferret of the same age. Both ferrets were obtained from a pet store at the same time (March 2005) and had markings indicating that they originated from Marshall Farms; however, their relatedness was unknown. The ferrets were fed a commercial ferret food. Also residing in the same household but not in the ferrets' room were a variety of reptiles and a newly acquired mixed-breed dog. The ferrets did not have direct contact with the reptiles but did have exercise time in the communal living space and some limited nose-nose contact with the dog. According to the owner, the dog was obtained from a shelter and was current on vaccinations. Vaccine history for the ferrets included 2 Purevax Ferret Distemper vaccines administered subcutaneously on October 4, 2007 and October 2, 2008 and a rabies vaccine (Imrab 3, Merial) administered subcutaneously on October 4, 2007. No problems associated with vaccination were noted in either ferret.

When the affected ferret was presented for emergent care, the owner reported that both ferrets had been normal when last seen that morning. Physical exam findings included a body temperature of 102.1°F, heart rate of 240 beats per minute (bpm), respiratory rate of 48 breaths per minute, and a weight of 0.92 kg. Body condition was noted to be optimal. Generalized weakness was observed and the ferret was dragging the hind limbs, although motor function was apparent and no pain was elicited on palpation of the legs or spine. The top differential diagnosis was hypoglycemic crisis secondary to insulinoma. Initial hematology results were within normal parameters with a packed cell volume (PCV) of 50% (reference range, 47%–51%[10]), total protein (measured by refractometer) of 7.0 g/dl (reference range, 5.3–7.2 g/dl[10]), and blood glucose of 109 mg/dl (reference range, 62.5–134 mg/dl[10]). Initial therapy included assisted feeding of a palatable gruel (Critical Care for Carnivores, Oxbow Enterprises, Inc, Murdock, NE, USA) in frequently administered small volumes, which the ferret readily accepted. The ferret continued to demonstrate generalized weakness overnight, was progressively becoming tachycardic (250 bpm), and continued to be tachypneic (~42 breaths per minute). The differential diagnosis list at that time included insulinoma (although this not supported by the blood glucose value on admission), cardiomyopathy (although no arrhythmia or murmurs were noted), trauma (although no history of trauma was reported), and other systemic diseases.

Twelve hours postpresentation, the rectal temperature was 102.3°F, heart rate was 250 bpm, and respiratory rate was 32 breaths per minute. Blood was collected through jugular venipuncture for hematology. A complete blood count demonstrated mild anemia (PCV 35.1%), and lymphopenia ($0.3x10^3$ cells/µl; reference range, 1.475–5.590 $x10^3$ cells/µl[10]) despite a normal total white blood cell count ($7.59x10^3$ cells/µl; reference range 2.5–$8.6x10^3$ cells/µl[10]). These findings were not significantly changed when measured 48 hours later. Serum chemistry evaluation revealed that the ferret was normoglycemic (132 mg/dl) and had a serum total protein level within normal range (5.6 g/dl); however, it also had an albumin:globulin ratio of 0.7 with hypoalbuminemia (albumin 2.3 g/dl; reference range, 2.6–3.8 g/dl[10]) and hyperglobulinemia (3.3 g/dl). Radiographic evaluation of the thorax revealed mild cardiomegaly and mild mixed interstitial-bronchial pattern in the caudal lung lobes indicative of early pulmonary disease or edema. Echocardiography was within normal limits. Tachycardia of non-cardiogenic origin was again noted (290 bpm). Abdominal ultrasound revealed hypoechoic, possibly cystic, enlarged mesenteric lymph nodes, which were aspirated for cytology. Cytology of the lymph node aspirates revealed low cellularity but indicated possible suppurative lymphadenitis. The ferret was continued on assisted feeding and was administered amoxicillin (20 mg/kg PO q12h; Amoxi-drops, Pfizer Animal Health, New York, NY, USA). Twenty-four hours postadmission, the ferret became hyperthermic with a rectal temperature of 103.9°F. The hyperthermia, ranging from 103.1–105.1°F, persisted for 84 hours until the ferret became normothermic.

Hospitalization and supportive care included assisted feeding and fluid therapy (60 ml/kg/d SC; lactated Ringer's solution, Baxter Healthcare Corp., Deerfield, IL, USA). Amoxicillin was discontinued after 4 doses due to the worsening of clinical signs and the ferret was started on chloramphenicol (25 mg/kg PO q12h; compounded by licensed pharmacy, St. Joseph Apothecary, St. Joseph, IL, USA). Three days after admission, the ferret was placed on meloxicam (0.2 mg/kg PO q24h; Metacam, Boehringer Ingelheim, St. Joseph, MO, USA) for anti-inflammatory effects in an effort to control the hyperthermia and ranitidine (24 mg/kg PO q8h; VistaPharm, Birmingham, AL, USA) as a H2 receptor antagonist to control gastric irritation. A multivitamin solution (0.22 ml/kg PO q24h; Pet Tinnic, Pfizer Animal Health) was also administered.

Forty-four hours postadmission, the ferret developed serous bilateral ocular and nasal discharge and blepharospasm. Eighty-four hours postadmission, it was noted that there was some irregularity with mild flaking of the skin of the palmar and plantar foot pads. The pads had a yellow discoloration that was also noted in the inguinal region. The ferret remained lethargic for the duration of the hospitalization but it was noted to be ataxic 5 days postadmission. This coincided with the return to normal body temperature. By day 6 postadmission, the ferret was more alert and active, was self-feeding small amounts of food, weighed 0.955 kg, no longer had ocular or nasal discharge, and was normothermic. Heart and respiratory rates continued to be mildly elevated. The ferret was discharged from the hospital 7 days after admission for home care. Ranitidine, meloxicam, chloramphenicol, vitamin supplementation, and assisted feedings were continued at home. Of note is that the companion ferret was hospitalized with the affected ferret for the entire duration and remained clinically normal.

The ferret was presented for an evaluation 11 days after being discharged (18 days postpresentation) and was found to be alert and active. Its body weight was 0.715 kg. Rectal temperature was 102.3°F, heart rate was 160 bpm, and the respiratory rate was 48–100 breaths per minute, depending on activity. The ferret had developed dark watery stool since being discharged. A complete blood count revealed a leukocytosis ($14.8x10^3$ cells/µl). The lymphocyte count ($1.62x10^3$ cells/µl) had returned to normal limits but an eosinophilia had developed ($1.76x10^3$ cells/µl; reference range 0.2–0.5 $x10^3$/µl[10]). Thoracic radiographs were unchanged since the previous study. All medications were discontinued at this time due to concern that the diarrhea might be related to antibiotic disruption of gastrointestinal biota.

Twenty-nine days after being discharged (36 days postpresentation), the ferret was alert and active but was becoming cachexic, with a body weight of 0.644 kg, despite a healthy appetite. All clinical parameters were within normal limits other than the presence of inflammation around the anus. The previous yellow discoloration of the inguinal area and footpads was resolved. Complete blood cell evaluation revealed a persistent leukocytosis ($15.6x10^3$ cells/µl) with eosinophilia ($2.0x10^3$ cells/µl) and monocytosis ($1.15x10^3$ cells/µl; reference range $0.1–0.2x10^3$ cells/µl[10]). Lymphopenia and anemia were not noted. The persistent leukocytosis caused concern that the ferret was suffering from a secondary gastrointestinal tract infection, so antibiotics were prescribed (amoxicillin-clavulanic acid, Clavamox Drops, 12.5 mg/kg PO q12h for 14 days, Pfizer Animal Health).

A recheck evaluation 2 months after discharge revealed that the ferret was gaining weight (0.804 kg) and had a good appetite. The white blood cell count was still elevated ($14.9x10^3$ cells/µl); however, the eosinophilia and monocytosis were resolving. By 3 months post discharge from hospital, the white blood cell count was within normal reference ranges and the ferret was normal on physical exam. Blood was collected by jugular venipuncture from the ferret as well as from the companion ferret and submitted to Cornell University Animal Health Diagnostic Center, Ithaca, NY, USA for serum neutralizing antibody titer testing for CDV. The affected ferret's CDV antibody titer was 1:3072 and the companion ferret's titer was reported as 1:1024. These values were remeasured 9 months post discharge. The affected ferret's titer had not changed; however, the companion ferret's titer was decreased to 1:768.

Discussion

Canine distemper in ferrets is generally considered to have 100% mortality and vaccination against canine distemper protects ferrets from contracting the disease. Hyperthermia, ocular and nasal discharge, malaise, and anorexia are clinical signs associated with several diseases that affect ferrets and often practitioners must consider history when making diagnoses. Canine distemper was considered unlikely in the clinical case presented in this paper because of the known vaccination history and apparent lack of potential exposure. Table 1 compares the clinical signs observed in the affected ferret with clinical signs associated with the differential diagnoses.

Table 1. Clinical signs observed in affected ferret compared with clinical signs associated with differential diagnoses.

Clinical Sign	Affected Ferret	Canine Distemper	Influenza	Myofasciitis
Fever	+	+	+	+
Oculonasal discharge	+	+	+	+
Anorexia	+	+	+	+
Tachypnea	+	-	-	+
Tachycardia	+	-	-	+
Lower airway disease	+	+	+	+/-
Weight loss	+	-	-	+
Yellowing, hyperkeratosis	+	+	-	-
Reluctance to move-ataxia	+	+	-	+
Leukopenia	+	+	+	-
Recovery	+	-	+	-

The tachycardia and tachypnea observed in the affected ferret were reminiscent of myofasciitis in ferrets treated by the author. However, ferrets diagnosed with myofasciitis are typically < 24 months of age, although the oldest confirmed case was in a 4-year-old ferret (K. Ramsell, written communication, May 2009). Ferrets with myofasciitis eventually succumb to the disease. Influenza was also considered as a possible cause of the clinical signs observed in the affected ferret. In April 2009, the first human cases of H1N1 influenza were being reported in Mexico and by the time the affected ferret was presented for emergency care, the disease was becoming widespread in the United States. Ferrets are susceptible to influenza and in October 2009 a confirmed diagnosis of H1N1 influenza in a ferret was reported where the ferret had contracted the infection from its human owner (Center for Disease Control, www.flu.gov/, accessed April 28, 2010). Influenza was ruled out in the affected ferret due to a lack of exposure with persons or animals demonstrating clinical signs of influenza. Canine distemper in the affected ferret was suspected when it developed changes to the footpads. Classic hyperkeratosis of the footpads never developed but a distinct yellow discoloration was apparent on the footpads and the skin of the inguinal area. Unfortunately, the owner's financial resources were committed to providing treatment and hospitalized care, so definitive testing was not performed.

Serum neutralizing antibody titers to CDV were measured in the affected and companion ferrets post convalescence. Variability in immune response to vaccination between individuals is expected; however, both ferrets were the same age, living in the same environment, and had received the same batch of vaccine, in the same manner, at the same times. The affected ferret's antibody titer was 3 dilutions higher than the companion ferret's titer. Results of 618 serum neutralizing antibody titers to CDV from the Cornell University Animal Health Diagnostic Center were evaluated. The serum antibody titers were transformed (log) before statistical analysis. The inverse geometric mean (313.99, 90% confidence interval 16–3072, range <4–6144) was similar to those reported in the previously described studies, although no data was available regarding vaccination history or natural exposure to CDV for these animals. A definitive diagnosis of canine distemper in the affected ferret cannot be made since testing was not performed to directly detect the presence of CDV. However, based on clinical signs, disease progression, and antibody titer data, canine distemper was strongly suspected in this ferret.

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Sedation of Exotic Companion Mammals

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Session #188

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Abstract: Sedation is an attractive option for exotic companion mammals when general anesthesia poses unacceptable risk. Newer drug alternatives, in combination with local or regional analgesia, allow completion of minor diagnostic or treatment procedures without general anesthesia.

Introduction

General anesthesia is commonly utilized in exotic pet medicine for procedures beyond surgery, such as for sample collection and radiography. General anesthesia is not without risk. A recent retrospective study of anesthetic death rates in the UK suggested anesthetic- or sedation-related death rates in healthy patients of 0.05% for dogs, 0.11% for cats, and 0.73% in rabbits within 48 hours of the procedure. Death rate increased more than 10 times in patients considered sick prior to anesthesia. However, comparison of anesthesia vs sedation demonstrated anesthetic death rate to be 2–2.5 times higher than sedation in all 3 species.

Why Sedate?

Sedation in general is a safer procedure than general anesthesia and is often adequate for minor procedures such as phlebotomy, placement of a catheter, and diagnostic imaging. The addition of local analgesia reduces discomfort associated with painful procedures such as intraosseous catheterization. Sedation becomes even more important in those patients for which anesthesia presents moderate to significant risk, in particular the ill or critical patient.

General anesthesia involves risk, even under the best circumstances. The advent of safer inhalant agents was a boon to exotic animal medicine. However, inhalant agents are naturally hypotensive, and untoward effects are dose-dependant. The use of inhalant agents as sole anesthetics necessitates higher doses, thus incurs higher risk. No other branch of veterinary medicine uses inhalants as sole agents for anesthesia for what are considered obvious risks. Therefore, exotic mammal practitioners should consider methods to reduce risk, which include the use of pre-anesthetic agents and analgesics to reduce the effective amounts of inhalant gases, and the use of sedation as an alternative to complete anesthesia when possible.

The following is a description of drugs currently used by the author and others for sedation of exotic companion mammals.

Midazolam

Midazolam is a benzodiazepine tranquilizer with sedation and anti-anxiety properties. Effects on blood pressure and other parameters are dose-dependant, but relatively mild. Midazolam can be administered intravenously and intramuscularly. Midazolam has anamnestic effects in human patients, which have also been demonstrated in rats. These effects could be expected to be of benefit in other mammal patients as well.

Opioids

Opioid agents are analgesic, but also provide sedation and work synergistically with midazolam. The effects of these drugs can be reversed with administration of naloxone. Choice of drug depends upon duration of effect desired and anticipated level of discomfort. In cases where an opioid is used solely to provide sedation along with midazolam, and discomfort is minimal, butorphanol is preferable due to short duration of action. Other longer-acting agents such as buprenorphine and hydromorphone can be considered when additional or longer acting analgesia is required.

Ketamine

At low doses, ketamine is safe and can be an effective addition to midazolam and an opioid for the purpose of sedation. Ketamine also has analgesic effects at lower doses.

Etomidate

Etomidate is a sedative and hypnotic agent used for induction of general anesthesia. It is primarily used for human patients with compromised cardiopulmonary function due to minimal hemodynamic effects, when compared to other IV induction agents. The main drawbacks are the need for IV administration, and frequent muscle movements and possible seizure-like activity, which can be abated with pre-administration of benzodiazepenes such as midazolam. This drug has no analgesic effects.

Lidocaine/bupivacaine

Local infusion of lidocaine/bupivacaine drugs facilitate procedures such as phlebotomy and catheterization. Local topical anesthetic gels, sprays, and topical refrigerants are also useful.

Specific uses for sedation in an exotic practice are outlined below.

Collection of Diagnostic Samples

While sedation alone does not allow collection of diagnostic samples from every patient, it is often sufficient. Even in those that ultimately require general anesthesia, sedation allows reduction of anesthetic agents according to the "multi-modal" effect. In the author's experience, the greatest benefit of sedation for collection of diagnostic samples is the use of low-dose agents (butorphanol and midazolam) in debilitated patients where sample collection is of prime importance for diagnosis and formulation of a treatment plan, but general anesthesia entails higher risk. Other specific examples include higher doses of the same agents, plus the addition of low-dose ketamine in clinically stable but fractious rabbits and small rodents. Dosages and drug combinations depend on anticipated level of discomfort and stress, and overall patient condition (Table 1).

Table 1. Drug dosages used by the author for sedation of exotic companion mammals.

Drug Class	Drug	Dosage (mg/kg)	Route	Comments
Benzodiazepine	Midazolam	0.25–0.50	IV, IM	
Opioids	Butorphanol		IV, IM	Short acting.
	Rabbit/Chin/ Guinea pig	0.1–0.3		This drug is very sedating in ferrets; consider lower doses.
	Ferret	0.1–0.2		
	Rat	0.5–1.0		
	Mouse	1.0–1.0		
NMDA antagonist	Ketamine	1–10	IM	Used in addition to midazolam and an opioid for additional sedation.
Sedative/Hypnotic agent	Etomidate	1–2	IV	Must use with benzodiazepine to prevent seizures. Short acting induction agent.
Local anesthetic	Lidocaine Bupivacaine	1–2 each	Local block or infusion	Enhances patient comfort for procedures such as phlebotomy and catheterization.

Placement of an IV Or IO Catheter

Midazolam combined with butorphanol, when combined with local anesthesia over the catheterization site, is often adequate, especially in debilitated patients. Local anesthesia can be administered in the form of injectable lidocaine, or topical anesthetic products applied 10 minutes prior to catheterization. To use injectable lidocaine for IV catheterization, roll the skin over the vein laterally or medially, and infuse lidocaine into the subcutaneous and dermal tissue. Massage the area of infusion to diffuse the lidocaine bleb, which should now be directly over the vein. Wait 10 minutes and place the catheter.

In-hospital use to facilitate acceptance of an IV line or urinary catheter

Midazolam in combination with butorphanol or buprenorphine often prevents disruption of IV or urinary catheters during hospitalization. Butorphanol can produce profound sedation in ferrets, and should be used at lower dosages (Table 1).

Diagnostic Imaging

Ultrasound and radiography are often facilitated with a combination of midazolam and butorphanol, plus the addition of low-dose ketamine if needed, along with proper manual restraint. Proper radioprotection much be used when considering manual restraint for radiography in small exotic mammal patients. (It should also be noted

that some states completely prohibit personnel in the room during radiography). This is accomplished with the use of radioprotective gloves and careful collimation of the primary beam over the area of interest excluding the restrainer's hands.

Recommended Reading

Reprinted in part from the Proceedings of the North American Veterinary Conference, 2009 and 2010.

1. Lennox AM. Venipuncture in small exotic companion mammals. Paper presented at: North American Veterinary Conference–Clinician's Brief; October 2001.

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3. Broadbelt D, Blissett K, Hammond R. The risk of death: the confidential enquiry into perioperative small animal fatalities. *Vet Anesth Analgesia*. 2008;35:365-373.

4. Etomidate: Medscape. Available at: www.druglib.com/druginfo/amidate. Accessed June 2008.

Secondary Nutritional Hyperparathyroidism with Fibrous Osteodystrophy in 3 Guinea Pigs

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Summary Style Manuscript

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This report describes fibrous osteodystrophy associated with secondary nutritional hyperparathyroism in 3 pet guinea pigs (*Cavia porcellus*) from different households purposefully placed on low-calcium diets to prevent urolithiasis. Fibrous osteodystrophy is caused by primary or secondary hyperparathyroidism and results in increased osteoclastic resorption of bone and replacement by fibrous tissue due to an imbalance in the Ca:P ratio. Diets with a low Ca:P ratio cause reduced growth rates, stiffness of joints, pathological fractures, and calcification of soft tissues.

Clinical signs in the guinea pigs included anorexia or difficulty eating, lethargy, difficulty walking, and an unwillingness to move. This disease must be differentiated from hypovitaminosis C as clinical signs can appear quite similar. Diagnosis was based upon history, physical examination findings, plasma total and ionized Ca concentrations, serum 25-hydroxyvitamin D and parathyroid concentrations, and radiographs. Treatment was aimed at stabilizing the patients and normalizing Ca:P ratios. Supportive care with assisted feedings, fluids, analgesics (opioids and non-steroidal anti-inflammatories), and supplemental calcium were provided. Despite aggressive supportive care, all 3 guinea pigs were euthanized within 12 months of diagnosis. Necropsy findings included severely thinned trabecular bone, marked osteoclastic activity, resorption of cortical bone and extensive replacement with fibrous connective tissue, and hyperplastic parathyroid glands, consistent with secondary nutritional hyperparathyroidism.

While vitamin D deficiency, Ca malabsorption, or other Ca metabolism disturbances may also play a role in the onset of fibrous osteodystrophy, caution should be used when recommending dietary calcium reductions for prevention of urolithiasis in guinea pigs.

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