East Tennessee
Veterinary Medical Association

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REPRODUCTIVE SURGERY

Karen Tobias, DVM, MS, DACVS

Latest Data on Neutering Effects: One of the most recent papers was published in July 2020: *Assisting Decision-Making on Age of Neutering for 35 Breeds of Dogs: Associated Joint Disorders, Cancers, and Urinary Incontinence* https://www.frontiersin.org/articles/10.3389/fvets.2020.00388/full
This study supports findings of the previous studies, noting that occurrence of joint disease in Golden Retrievers, Labrador Retrievers, and German shepherds is much higher if those breeds are neutered at 6 months or younger. In general, age of neutering had no effect on occurrence in joint disease in most small breed dogs or certain giant breed dogs (Great Danes and Irish Wolfhounds). In this paper, overall occurrence of mammary cancer was 6%, but the breed related occurrence varied from 0-15%.

Breed	Joint disease for those Neutered ≤ 6 months	Joint disease for those Intact	Cancer if Neutered ≤ 6 months	Cancer for those Intact
Golden male	18%	3%		
Golden female	25%	8%	26%	14%
Labrador male	22%	8%		
Labrador female	33%	10%		
German shepherd male	33%	2%		
German shepherd female	29%	9%		

The following chart summarizes some of the data from the article so that you can quickly identify the effects of neutering on occurrence of joint disease, cancer, and urinary incontinence and, for Corgis, intervertebral disk disease. The chart also lists the percentages of mammary cancer and pyometra in those breeds. Additionally, I have included a chart at the end of these notes - "Suggested Guidelines by Breed for Age of Neutering" –from this same article; it includes recommendations for neutering age based on their study results. As a summary, neutering after 2 years of age is recommended for Boxer and German shepherd males and females; Cocker spaniel, Doberman, Sheltie, and Shih tzu females; and Irish wolfhound and standard poodle males. Breeds that should be neutered at 12 months or older include male Beagles, Border Collies, Boston terriers, Golden Retrievers, Miniature poodles, and Rottweilers, and female Border Collies, Collies, English springers, and Labrador retrievers. The authors recommended leaving male Dobermans and female Golden Retrievers intact because of the high risk of cancer in those dogs. For 14 breeds, there was no noticeable effect of neutering on cancer, joint disease, or incontinence rates. Interestingly, neutering had no effect on risk of disk disease (IVDD) in male and female dachshunds (risks of 53% and 38%, respectively), but percent affected dogs increased from 3% to 18% when male Corgis were castrated before 6 months of age.

Breed	Joint disease risk with neuter	Cancer risk (neuter = male and female)	Incontinence risk with OHE	Mammary cancer, Pyometra
Aus Cattle dog	7x if OHE <6mo			Pyo 4%, MC 6%
Austr shep		Sl. w/ OHE	1% if OHE <6 mo	Pyo 5%. MC 0-8%
Beagle	7x if castrated<1 year		2% if OHE <6 months	Pyo 2%; MC 0%,
Bern Mntn Dog	3x-6x	2x with OHE <6 mo		Pyo 5%, MC 0%
Border Collie		6x-11x w/neuter<1 yr		Pyo 4%, MC 1%
Boston Terrier		2x with castration	2% if OHE <6 mo	Pyo 7%, MC 2%
Boxer		2x with neuter <2 yrs	1% if spayed	Pyo 2%, MC 0%
Bulldog	Sl. with neuter<6 mo			Pyo 2%, MC 1-2%
Cavalier				Pyo 2%, MC 0-%
Chihuahua				Pyo 2%, MC 1-4%
Cocker	11x w/castr <6 mo	17x w/OHE <2yr (MCT)		Pyo 5%, MC 11%
Collie		with OHE <6 months	13% if OHE at 6-11 mo	Pyo 16%, MC 4%
Corgi	IVDD 6x castr< 6mo			Pyo 0%, MC 8%
Dachshund				Pyo 4%, MC 1%
Doberman	Sl. OHE <12 months	Sl. with castration	25% if OHE <6 mo; 19% if OHE <2 years	Pyo 7%, MC 2-4%
Engl Sprng Span			Sl if OHE <1 year	Pyo 0%, MC 6-15%
German shepherd	3x w/neuter <1 year		9% with OHE <1 year	Pyo 3%, MC 5%
Golden retriever	2x-5x w/neuter <1 yr	1.3x w/castr <6mo; 3x w/OHE at any age		Pyo 4%, MC 1-4%
Great Dane				Pyo 6%, MC 2%
Irish Wolfhnd		3x w/castr at 1-2 yrs		Pyo 5%, MC 0%
Jack Russell				Pyo 1%, MC 1-3%
Labrador	2x w/castration<6 mo or OHE<12 mo		2-3% if OHE ≤1 year	Pyo 2%, MC 1-2%
Maltese				Pyo 0%, MC 0.5%

Min schnauzer				Pyo 4%, MC 0%
Pomeranian				Pyo 7%, MC 1%
Toy Poodle				Pyo 0%, MC 1%
Mini Poodle	9x w/castr 6-11 mo			Pyo 6%, MC 1%
Std Poodle	Sl w/castration <6mo	LSA 6x w/castr at 1 yr		Pyo 2%, MC 4%
Pug				Pyo 5%, MC 0%
Rottweiler	2-3x w/castration <12 mo; 3x with OHE<6 mo	Previous paper: 2x in bone sarc if neuter<1yr	1% if intact; 4%-6% if OHE <6 and 12 mo	Pyo 12%, MC 5-8%
Saint Bernard	17x (100%!) OHE<6mo			Pyo 15%, MC 0%
Sheltie			33% OHE<2yr, 6% <1yr	Pyo 14%, MC 0%
Shih tzu				
West Highland White			14% OHE<6mo, 6% if OHE<12 mo	Pyo 7%, MC 0%
Yorkie				Pyo 7%, MC 1%

Other reproductive notes:

OVE vs. OHE: Ovariectomy (OVE) without hysterectomy is becoming more common, particularly with laparoscopic spays. Ovariectomy alone does not increase the risk of pyometra, since endometritis, pyometra, and stump pyometra require progestagens to develop. Ovariectomy may be less traumatic because incision size is smaller and tissues are handled less; the only increased risk compared to ovariohysterectomy (OHE) is future development of uterine tumors, which are rare in dogs (0.03%). Most of these tumors (90%) are benign leiomyomas; therefore, the true overall incidence of malignant uterine tumors is 0.003%.

Pedicle ties: In high volume spay practices, ovarian pedicles in cats are often "tied on themselves" (pedicle tie), similar to cat castration. Complication rates are low, with hemorrhage noted in 0.28% and usually detected in surgery. Pedicle tie surgeries are about 2 minutes faster than ones with suture ligation.

Ovary-sparing surgery: Hysterectomy alone as a method for preventing pyometra and avoiding the inconvenience of vaginal discharge during heat cycles is offered as an option to ovariohysterectomy. There are no studies on short or long-term outcome of the procedure. When ovary-sparing hysterectomy is performed as an open surgical technique, the incision must be large and caudal enough to see the ovaries and the cervix (Frontiers Vet Sci 2020; https://doi.org/10.3389/fvets.2020.00342). The vessels to the uterine horns (including those between the ovary and uterine horn) are ligated, and the

distal cervix or proximal vagina is clamped, ligated, and transected. The entire uterus must be removed to remove the risk of stump pyometra, so the distal ligation must be at or beyond the caudal cervix, since the cranial cervix contains glandular tissue. Additionally, the tips of the uterine horns must be removed- this can be difficult in small or immature dogs. Dogs undergoing hysterectomy still have ovaries and will therefore experience the hormonal effects of estrus (e.g., vulvar swelling, behavioral changes) and will attract male dogs. There is a risk of vaginal rupture and peritonitis if hysterectomized dogs are allowed to copulate in the first 2 months after surgery.

Scrotal versus prescrotal castration: Scrotal castration can be performed in any size dog. Prescrotal castration is difficult in young dogs because the testicles slide into the inguinal rings, making them hard to find. Dogs undergoing prescrotal castrations and an intradermal closure have greater incidence of self-trauma and longer surgical times than those castrated through a scrotal incision and with a single simple interrupted subcutaneous stitch for closure (Woodruff et al, Vet Med 2015). Caudal scrotal incisions are also no more likely to cause complications than prescrotal incisions (Snell et al, New Z J 2015).

Autoligation techniques in dogs: Similar to cats, hemostasis in small breed dogs can be achieved by tying the cords on themselves. Complication rates of scrotal castrations performed via autoligation are similar to prescrotal castration with suture ligation.

HEAD AND NECK SURGERY

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Tracheal Collapse

Collapsing trachea is a progressive condition in which the supportive cartilage in the trachea and smaller airways start to degenerate and soften. With mild tracheal collapse, the dorsal membrane of the trachea starts to billow inward, partially blocking the airway. As the condition progresses, the C shaped rings of the trachea begin to flatten out, allowing greater airway compromise. During coughing, the weakened walls of the airway slam together, causing inflammation and trauma, which stimulates more coughing. If the cycle of coughing is not broken, the airway can become temporarily obstructed, leading to respiratory distress and even death.

Collapsing trachea is usually diagnosed in toy breed dogs and is particularly common in Yorkshire and Maltese terriers, poodles, and Pomeranians. Clinical signs may be seen as early as 6 months of age, although most dogs are middle age when they present for diagnostics and treatment. Initially the primary sign is coughing, but it can progress to exercise intolerance, wheezy breath sounds, cyanosis, and collapse. In rare cases dog present only with intermittent collapse (similar to a heart problem).

Collapsing trachea is often suspected based on the breed, clinical signs (a classic cough sounds like a "goose honk" if tracheal collapse is severe), and neck palpation (the cough can be stimulated, and sometimes the collapse can be felt). Definitive diagnosis may require thoracic and cervical radiographs, fluoroscopy, or even tracheoscopy. Thoracic radiographs are not always definitively diagnostic for tracheal collapse because other structures (such as the esophagus) can overly the trachea, making it hard to determine the exact shape of the trachea. Tracheoscopy is considered the "gold standard" for diagnosis; however, it requires anesthesia, and some dogs with severe tracheal collapse have trouble recovering from the procedure. Therefore, most veterinarians base their initial diagnosis on clinical signs and radiographs.

Medical management is the mainstay for treatment of tracheal collapse. The first step is to break the coughing cycle. Most often a cough syrup containing a narcotic (hydrocodone) is used. This medication makes the dog sleepy, and some owners do not like the change in personality or loss of appetite. If veterinarians do not have access to hydrocodone and cannot prescribe it through a local pharmacy, butorphanol can be substituted. High or frequent doses or narcotics are usually only used for a few days to a week, and they allow the dog (and its airway) to get some rest. If a narcotic does not stop the cough, we may prescribe oral steroids to help reduce inflammation. Long term use of steroids can cause problems with weight gain, excessive drinking and urinating, liver disease, and increased risk of infection, so dogs are gradually weaned off the steroids. Respiratory infection can worsen the condition, so we may prescribe a short course of antibiotics. Bronchodilators may be used -but only if the bronchioles appear to be affected on radiographs. Since stress and excitement can initiate coughing, we prescribe a sedative (e.g., trazodone) or tranquillizer (e.g., acepromazine) that can be given at home when stress occurs or is expected (e.g., visitors, fireworks, thunderstorms).

Other important aspects of initial management are environmental changes to reduce airway irritation. Dogs with tracheal collapse should not be exposed to wood or cigarette smoke, perfumes, and other airway irritants, either in person or on clothing, rugs, or fabric. They may need humidification if the environment is low in humidity (e.g. during the winter when the heater is on), and they may need air

filtration (e.g., air conditioning) when pollen counts are high. Harnesses should be used instead of leashes and collars. Exercise may need to be limited until coughing has decreased.

The most important part of long-term medical management is weight loss. Many toy breed dogs with tracheal collapse are overweight, and 70% of these dogs will improve dramatically with weight loss. Weight loss can be challenging in a dog on steroids or exercise restriction, so nutritional control is critical for managing these dogs.

When medical management is no longer sufficient, more aggressive intervention with a tracheal stent may be required. In some cases, stents are placed surgically around the trachea ("extraluminal stents") through a long incision in the neck that may extend into the chest. Most commonly, however, an expandable tracheal stent is placed nonsurgically inside the trachea ("intraluminal stent") by a specialist using fluoroscopic or endoscopic guidance. Owners should expect to spend $3800 to $5200 for uncomplicated stent placement. Most dogs undergoing stent placement will spend 1 to 3 nights in the hospital. Because the stents are expensive, the specialist may have only a limited number of sizes. If the appropriate size stent is not available, the dog may need to be hospitalized on sedatives and oxygen until an appropriate size stent can be obtained by express delivery. After stent insertion, the dog is placed on steroids for at least a month, plus sedatives, cough syrup, and potentially other medications. Steroids will be gradually decreased over 4 to 6 weeks to every other day treatment and discontinued if they are no longer needed. Radiographs are often taken 1, 3, 6 and 12 months after stent placement to make sure there are no detectable problems such as stent fractures or granulation tissue.

Because intraluminal tracheal stents are essentially "foreign bodies", they are expected to cause coughing, particularly the first 1 to 3 months after placement. In fact, some dogs cough the rest of their lives after tracheal stent placement and must be kept on steroids to prevent it. Therefore, stents are usually not placed in dogs in which the only sign is coughing. Instead, those dogs are managed medically until their signs are severe enough that they are having bouts of exercise intolerance at a walk, cyanosis, or collapse or are considered to have a poor quality of life. The average lifespan of dogs undergoing stent placement is about 2 years, but many dogs live longer than 4 years.

Besides coughing, complications of stent placement include disruption of the normal mucous flow up and out of the trachea, which could increase the risk of infection or airway obstruction. If the stent is too loose, it can actually be coughed out. If the stent is too short, the trachea can collapse around it. Irritation from the stent may cause "granulation tissue" to grow through and around the stent, blocking airflow. If the dog coughs excessively, the stent may break, requiring placement of a second stent or even euthanasia. Stents do not prevent collapse of the bronchi, and they do not correct laryngeal collapse or elongated soft palate, both of which may be present from longstanding tracheal collapse. In other words, tracheal stenting is not a cure for tracheal collapse, only another means of managing it.

Veterinary consults about stent placement are directed to the Soft Tissue Surgery Service, while consults about diagnosis and treatment of chronic airway disease are directed to the Internal Medicine Service.

Laryngeal Paralysis

Laryngeal paralysis is a well-recognized syndrome in large breed dogs that results in upper airway obstruction from loss of arytenoid cartilage abduction. It is occasionally reported in cats and small dogs. Acquired laryngeal paralysis is seen in older dogs (median age, 9 years), with males affected more often than females. In the United States, Labrador retrievers are commonly affected. Acquired laryngeal paralysis is often part of a syndrome of generalized peripheral neuropathy. In a study at University of

Tennessee, all dogs with acquired laryngeal paralysis had evidence of polyneuropathy on peripheral nerve and muscle biopsies, despite a lack of clinical signs of neuromuscular disease in many of the affected animals. Another study reported 25% of the dogs have neurologic signs by the time of laryngeal paralysis diagnosis, and 100% have signs within a year. Acquired laryngeal paralysis can also occur as a result of trauma, iatrogenic (surgical) injury to the recurrent laryngeal nerve or its cranial laryngeal branch, or compression of the recurrent laryngeal nerve by a cranial mediastinal or cervical mass. A congenital form of the condition has been reported in Bouvier des Flandres, Dalmatians, rottweilers, and Siberian huskies, with onset of clinical signs noted before one year of age.

Dogs with laryngeal paralysis usually present with progressive respiratory signs. Initially, owners may note a voice change, inspiratory stridor, and exercise intolerance. Some animals with peripheral neuropathy may have dysphagia or regurgitation from concurrent esophageal dysfunction. Eventually the dogs can develop severe dyspnea, cyanosis, and syncope. Collapse or respiratory distress may be initiated by hot weather, stress, or heavy exercise.

Animals suspected to have laryngeal paralysis should be examined for evidence of other systemic illnesses that occur as a result or cause of laryngeal paralysis. For instance, all patients should have a thorough neurologic examination, thoracic auscultation, and rectal temperature. Basic blood work is performed to look for evidence of systemic illness (e.g. hypothyroidism, hypoadrenocorticism, sepsis from infection) and to evaluate general health status before anesthesia. Thoracic radiographs are examined for evidence of aspiration pneumonia, pulmonary edema, intrathoracic masses, or esophageal dilation or for etiologies that could cause collapse or respiratory distress (e.g. primary heart or lung disease).

Diagnosis of laryngeal paralysis is usually based on examination of the larynx under a light plane of anesthesia. Normal function can be seen in dogs heavily sedated with dexmedetomidine alone or in conjunction with butorphanol or hydromorphone. Anesthetics used for laryngeal examination may cause significant depression of laryngeal function; false positives have been noted with ketamine/diazepam, acepromazine/propofol, and propofol alone. If patients require acepromazine, propofol, or other drugs that reduce arytenoid motion, respiration can be stimulated by administration of doxapram hydrochloride IV (1.1 mg/kg). The animal must be evaluated for paradoxical motion, an abnormal condition where the arytenoid cartilages are drawn inward on inspiration and forced apart by air during exhalation. This motion can be easily confused with normal laryngeal function.

Treatment depends on the severity of clinical signs. Animals that are in distress are immediately stabilized with sedation and oxygen administered by mask or nasal catheters. An IV catheter is placed for administration of fluids and medications. Hyperthermic animals are topically cooled. Glucocorticoids are administered if laryngeal edema is suspected. Patients are monitored with pulse oximetry, when available. Those that do not respond to treatment may require immediate intubation or emergency tracheostomy or laryngeal tieback. A plastic tube stent has also been used.

Because the recurrent laryngeal nerve controls opening and closing of the glottis and tightening of the vocal cords, dogs with laryngeal paralysis often present with a voice change, coughing, noisy breathing, and sometimes aspiration pneumonia. Most surgical treatments involve permanently holding the airway open, which does nothing to prevent coughing, does not return the voice, and can result in noisy breathing and aspiration pneumonia. Therefore, mildly affected dogs are usually treated conservatively with weight loss, stress reduction, use of antianxiety medications (e.g., trazodone), treatment of any pneumonia, and restriction of exercise or exposure to high ambient temperatures. Because laryngeal

paralysis is a progressive condition, many conservatively-managed animals will require future treatment. Most dogs with acquired laryngeal paralysis will have progression of generalized polyneuropathy signs over the next year.

The most common surgical options include unilateral arytenoid lateralization ("tieback"), partial arytenoidectomy, or vocal fold resection. Laryngeal tieback is considered the technique of choice by most surgeons. Unilateral arytenoid lateralization is usually performed through a lateral cervical incision and is basically a suture replacement of the cricoarytenoideus dorsalis muscle. A suture is passed through the cricoid cartilage and the muscular process of the arytenoid cartilage and is tied gently; this suture will prevent the arytenoid cartilage from being pulled back into the airway. Only one side is "tied back": bilateral arytenoid lateralization is much more likely to result in aspiration and death since it prevents any arytenoid closure during swallowing.

Improvement in respiration is expected in 90% of animals undergoing unilateral arytenoid lateralization, and 70% are still alive 5 years after the surgery. Complications are reported in 10% to 28% of dogs and include aspiration pneumonia (8% to 33%, depending on the paper), coughing and gagging (16%), suture failure or return of clinical signs (4% to 8%), gastric dilatation and volvulus (4%), respiratory distress (2% to 4%), and sudden death (3%). Aspiration pneumonia may occur shortly after surgery or at any time for the remainder of the dog's life, and is more common in dogs with concurrent megaesophagus. Perioperative administration of metoclopramide reduces the risk of postoperative aspiration pneumonia.

Laryngeal Collapse

Laryngeal collapse is usually are result of chronic, highly negative airway pressure, such as that seen with brachycephalic airway syndrome. As a result of airway narrowing, increased resistance, and high negative pressure, the arytenoid cartilages lose their rigidity, allowing them to deviate medially and block the airway. In the early stage of the condition (Grade 1), the laryngeal saccules evert. As the condition worsens, the cuneiform processes are displaced toward midline; eventually, the corniculate processes collapse as well (Grade III). Mild cases respond to correction of associated conditions (stenotic nares, elongated soft palate) and removal of the everted saccules. Moderately to severely affected animals have a more guarded prognosis. These dogs require placement of a temporary tracheostomy tube and some sort of surgical intervention, such as a laryngeal tieback or permanent tracheostomy. About 80% of affected dogs that undergo temporary tracheostomy and unilateral laryngeal tie back do well after surgery, as long as the tracheostomy tube is left in place for at least 24 hours after surgery (so swelling resolves). About 50% of surviving dogs have intermittent episodes of regurgitation.

One thing to note is that repair of brachycephalic syndrome (e.g., staphylectomy and rhinoplasty) may not result in regression of everted laryngeal saccules, so many surgeons remove these saccules (transecting them at their base with scissors) when correcting other brachycephalic defects.

Stenotic Nares

Stenotic nares are most commonly seen as a component of brachycephalic syndrome in short nosed dogs and cats. Predisposed breeds include English and French bulldogs, Pugs, Boston terriers, Pekingese, Cavalier King Charles, and Norwich terrier dogs and Persian and Himalayan cats. Shih tzus may present with severe clinical signs as early as 6 to 8 weeks of age; at that age, they tend not to have other components of brachycephalic syndrome (e.g. elongated soft palate), but those components are likely to develop if the condition is not treated early. The cause of stenotic nares in dogs is axial deviation of the

dorsolateral nasal cartilage and its associate skin and mucosa (the “wing” or alar fold of the nostril). The negative pressure produced by this airway blockage instigates severe stress (from suction) on the soft palate, larynx, and trachea and can result in development of tissue swelling and airway collapse. Many animals will also have abnormal conchal development; in fact, the alar folds are actually extensions of the ventral nasal concha, which must also be addressed when surgery is performed. In cats, thickening or ”bunching” of the ventrolateral nostril skin and SQ adds to stenosis. About 20% of brachycephalic dogs and cats will have “nasopharyngeal” turbinates that protrude down into the nasopharynx, blocking nasal flow of air. In Europe, nasopharyngeal turbinates are most common in pugs. Repair of stenotic nares in animals with nasopharyngeal turbinates may decrease but not resolve the clinical signs.

Clinical signs of stenotic nares include inspiratory dyspnea, recurrent nasal infections, and sometimes exercise intolerance or poor appetite. Neonates with stenotic nares may have difficulty nursing. With increased negative pressure, affected animals may also develop digestive signs such as hiatal hernia.

Stenotic nares are easily diagnosed on physical examination. Evaluation of concurrent conditions will require anesthesia (e.g., elongated soft palate, laryngeal collapse), radiographs (e.g., hypoplastic trachea), and CT or scoping (e.g., nasopharyngeal turbinates).

In puppies or cats with small alar folds, stenotic nares can be widened by amputation of the alar folds. When performed with a scalpel blade, this is known as the “Trader technique”. In puppies, the ventral half of the fold, with associated ventral nasal concha, is excised with a #11 blade inserted at 40° ventrolateral angle. Bleeding is controlled with digital pressure. The subsequent white scar will gradually regain pigment over 6 months.

In older dogs and some cats, a wedge or punch of tissue is taken out of the central or lateral portion of the alar fold and rostral extent of the ventral nasal concha with a number 11 blade. The remaining gap is apposed with 4-0 or 5-0 rapidly absorbable suture in an interrupted pattern. The sutures are cut short and left in place; they usually fall out in 1-2 weeks. Another technique in cats is to remove skin and subcutis on the ventrolateral margins of the nose and advance the nasal mucosa outward to close the defect.

Salivary Mucoceles

Sialoceles, or salivary mucoceles, are abnormal collections of saliva within tissues. Sialoceles are usually caused by subcutaneous leakage from the mandibular and sublingual salivary glands or ducts. Most affected dogs present with a fluctuant, nonpainful, subcutaneous swelling located in the intermandibular region or ventrally along the proximal cervical region. Fluid can leak submucosally from the rostral portion of the salivary glands and ducts, resulting in a sublingual swelling, or ranula. Treatment for sialoceles is sialadenectomy (removal of the affected glands and ducts). Diagnosis of a salivary mucocele is made by aspirating viscus, yellow tinged fluid (that looks like saliva and contains mucin) from the swelling, or by confirming salivary gland disease with ultrasound or a CT.

The mandibular and sublingual salivary glands are located at the bifurcation of jugular vein. The mandibular lymph nodes lie rostral and ventral to the mandibular salivary gland. The mandibular and sublingual salivary glands are enclosed within the same capsule, which is deep to maxillary and linguofacial veins. Their ducts run adjacent to one another, passing between the masseter and digastricus muscles and the over dorsomedial surface of the mylohyoid muscle. The lingual branch of the trigeminal nerve crosses their lateral surfaces before the ducts reach the oral cavity.

The sublingual salivary gland consists of several lobulated masses. The larger portion sits on the rostral surface of the mandibular gland and drains into the main sublingual duct. Rostrally, a 1 x 3 cm cluster of lobules lies under the oral mucosa. These lobules also drain into the main sublingual duct via four to six smaller ducts. During sialadenectomy, both of these monostomatic portions of the sublingual salivary gland are removed. The polystomatic portion of the sublingual salivary gland consists of 6 to 12 small lobules that drain directly into the oral cavity. These are usually left in place during surgery.

Unilateral sialadenectomy is usually performed with the dog in lateral or dorsolateral recumbency. Dogs undergoing unilateral sialadenectomy are clipped from the angle of the mandible to the midcervical region and from the base of the pinna to ventral midline. A towel can be placed under the neck to elevate the affected side. Since the sublingual salivary gland or duct is usually the culprit, and since the small glandular lobules along the rostral duct can be the source of the saliva, extending the dissection beyond the digastricus muscle may improve removal of all the gland. This is accomplished after dissecting out the main portions of the sublingual and mandibular salivary glands by inserting closed hemostats from medial to lateral under the digastricus muscle, grasping the duct after transecting it to separate it from the monostomatic glands, and pulling it under the digastricus medially. Dissection can then continue along the duct farther rostrally. Once glands and ducts are removed, a drain is placed before closure to manage the dead space posteropatively.

URETHRAL SURGERY

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General Principles:

Healing of the urethra is dependent on urethral continuity and local urine extravasation. If a strip of urethra remains intact and urine is diverted from the site, the urethral mucosa can seal the wound in one week. If the urethra is completely transected, the mucosal ends will retract and fibrous tissue fills the intervening gap, obstructing the urethra. Primary repair is therefore required with complete urethral transection. Urethral injuries can seal within 3 days but heal best when urine is diverted from tears or surgical sites. Urine diversion can be performed with transurethral catheters or cystostomy tubes; either method will decrease the risk of stricture compared to primary anastomosis alone. Risk of urethral stricture formation is increased with tension on the anastomosis or use of oversized urethral catheters.

Surgical Approach:

The distal urethra can be approached through a perineal or scrotal approach (males) or through an episiotomy (females). Perineal and episiotomy approaches are often performed with the animal in sternal recumbency with its rear legs hanging over the end of a tilted, padded table. If identification or catheterization of the urethra is expected to be difficult, concurrent cystotomy is needed, or a mass is present that extends into the abdominal cavity, surgery is performed with the animal in dorsal recumbency. If necessary, the rear legs of the animal are pulled cranially to provide a perineal view; this is particularly useful in cats during perineal urethrostomy. In female dogs, the perivulvar area is clipped and prepped and included in the draped field to allow retrograde or antegrade catheterization. In male dogs, the prepuce is included in the prep and sterile field.

The proximal urethra can often be reached through a caudal abdominal incision. The distal urethra in a male can be approached by prescrotal or scrotal incisions or an incision into the penile body itself, with hemostasis facilitated by tourniquet placement. In a female, the distal urethra can be approached through the vestibule, with exposure improved using an episiotomy.

Approach to the midportion of the urethra may require pelvic osteotomy or ostectomy. Pubic osteotomy alone (a raised flap incorporating the pubic bone) will expose the cranial half of the intrapelvic urethra. Adductor muscular attachments are left in place along one edge of the pubis to improve blood supply and stability. If possible, a portion of the prepubic tendon can also be left attached to the pubic flap. The bone flap is reflected ventrally and caudally during surgery. If a wider exposure is required, a bilateral pubic and ischial osteotomy (saggital pubic osteotomy) or symphyseal ostectomy can be performed. For saggital pubic osteotomy, the pubis is divided along its length with an oscillating saw and its edges gently separated with a rib spreader. The edges are wired back together once the intrapelvic procedure is

complete. With bilateral pubic and ischial osteotomy, muscular attachments are left in place along one lateral margin of the bone flap, and the flap is reflected laterally along these attachments. The prepubic tendon can be secured to the bone fragment through additional drill holes or reapposed as needed along its midline and then sutured caudally to the adductors. Pubic ostectomy (removal) can be performed with rongeurs or burr. The cranial half of the pubis can be removed to expose the urethra immediately caudal to the prostate and trigone, and the caudal half of the pubis can be removed to expose more distal urethra (this is reported in cats with high perineal urethrostomies). If more extensive exposure is required, the entire pubic and ischial symphysis can be removed to produce a gap that is 2 to 3 cm wide, depending on the size of the animal; rib retractors can be used to widen the gap even more. A portion of the cranial (acetabular) branch of the pubis medial to the iliopubic eminence should be left intact with the attached prepubic tendons. During closure, the prepubic tendon and adductors are apposed along the midline and the tendon is sutured caudally to the cranial edge of the adductors.

Urethral Repair

Urethral tears and lacerations are usually secondary to trauma, urethral catheterization, or calculi but can also occur inadvertently during surgery. If a strip of the urethral wall remains intact, most patients can be treated with urinary diversion. If the urethra cannot be catheterized retrograde, a cystotomy is performed and a catheter passed antegrade through the bladder and out the urethra; a Foley catheter is tied to the catheter and pulled retrograde into the bladder. If the urethra is completely transected, primary repair is required. Urethral resection and anastomosis is occasionally performed to resolve strictures or remove tumors. Urinary diversion is important for healing and should be maintained for a minimum of 3 to 5 days, depending on the health of the urethral tissues and the amount of tension on the anastomosis. Urine should be cultured after catheters are removed. In male dogs undergoing intrapelvic urethral resection and anastomosis, there was no significant difference in urethral healing in those that had an indwelling urethral catheter, cystostomy tube, or a combination of both after surgery.

Prescrotal Urethotomy

Under general anesthesia, most urethral calculi can be retropulsed into the urinary bladder and removed with cystoscopy, cystoscopically assisted cystotomy, or open cystotomy. Those that cannot be shifted are usually lodged within the urethra at the caudal end of the os penis. Many of these calculi become embedded within the mucosa and are not easily removed, even through a urethrotomy; in these dogs, scrotal urethrostomy is usually performed. In a few dogs, the calculus can be dislodged through a prescrotal urethral incision. Prescrotal urethrotomies are usually closed primarily to reduce postoperative hemorrhage. Urethral incisions that are left open to heal by second intention will bleed for 3 to 14 days, particularly when animals are excited. Rarely, dogs may undergo permanent prescrotal urethrostomies. Owners should be warned of the potential for urine scald along the scrotum and inner thighs.

Scrotal Urethrostomy

Persistent or severe hemorrhage and dehiscence are uncommon after scrotal urethrostomy when the urethrocutaneous apposition is performed with a continuous pattern, suture bites include mucosa, and

postoperative self-mutilation is prevented. A rapidly absorbable monofilament synthetic suture can be used for closure; sutures are usually not visible 3 weeks after surgery and therefore do not need removal. Addition of a mattress suture at the cranial and caudal extents of the urethrostomy may prevent hemorrhage from the sites, since the “crotches” of the incisions are often missed during urethrocutaneous apposition.

Perineal Urethrostomy (PU)

For cats with concurrent cystic calculi and urethral obstruction or those with strictured PU sites, surgery is most easily performed with the cat on its back and the rear legs pulled forward so that the bladder and urethra can be approached simultaneously. The veterinarian must take care to incise the correct side of the penile body- the one closest to the anus- when opening the urethra. As with dogs, the PU site can be closed with a simple continuous pattern with rapidly absorbable monofilament. However, preplacement of the first 3 to 5 sutures in an interrupted pattern will help secure the urethral mucosa to the skin appropriately. When preplacing the top 3 sutures, bites should be taken as close to the top (“crotch”) of the urethrostomy incision as possible. The first 2 sutures pass through this “crotch” area and angle out to grab skin at the 10 and 2 o’clock positions. The top suture also passes through this area and angles out slightly higher (a less acute angle at about the 1 o’clock position) instead of grabbing the skin directly dorsal to the urethrotomy. This will prevent kinking of the skin and shortening of the distance between the urethrostomy and the anus. Perineal urethrostomy is not always required for cats with obstructions; in fact, 73% of cats in one study responded to treatment with sedatives, analgesics, intermittent cystocentesis, subcutaneous administration of fluids, and a stress free environment.

Perineal urethrostomy through an episiotomy can be performed in female dogs that require vaginal and distal urethral resection (e.g. for leiomyomas). In female dogs the distal urethra can be resected and the proximal end anastomosed onto the remaining vagina (vaginourethroplasty) or the vestibule. Because of the aggressive nature of TCCs, resection is not recommended for dogs with distal urethral transitional cell carcinomas; instead, those dogs may benefit from urethral stenting and appropriate chemotherapy.

Subpubic and Antepubic Urethrostomies:

When the urethral end is too short to reach the skin without tension, it can be brought cranial to the pubis (antepubic urethrostomy), or a portion of the pubis can be removed with rongeurs (subpubic urethrostomy). Because of the curve produced in the urethra,subpubic urethrostomy does not always reduce the length of urethra needed to reach skin. Antepubic urethrostomy has a high complication rate (incontinence, urine scald) and is considered a salvage procedure. In some species, transposition of the urethra to the prepuce may reduce the risk of urine scald.

Urethral Prolapse:

Repair techniques for urethral prolapse include resection and anastomosis or urethropexy. For anastomosis, fine rapidly absorbable suture is recommended. Recurrence (57%) has been reported with both procedures, and postoperative hemorrhage (39%) is also common.

Urinary Incontinence

Hydraulic occluders are becoming more popular for treatment of incontinence caused by urethral sphincter mechanism incompetence. A silicone cuff is placed around the proximal urethra about 2 cm distal to the bladder neck and is attached by tubing to a subcutaneous vascular access port inserted along the caudal abdomen. Many dogs do not need any cuff inflation because of local pressure in the area from the cuff or resultant scar tissue. Those that need extra pressure to maintain continence must be managed cautiously; sometimes addition of as little as 0.1 ml of saline to the cuff can result in obstruction of the dog.

SURGERY OF THE DIGESTIVE TRACT IN SMALL ANIMALS

Karen Tobias, DVM, MS, DACVS

Prophylactic antibiotics

To be effective, prophylactic antibiotics should be administered so they have reached peak concentrations before the incision is made- that means IV injection between 30 and 60 minutes before the incision. They should be repeated IV at 1.5 times the half-life (e.g., every 90 minutes for cefazolin in dogs) if the surgery is still ongoing, or they can be given earlier via an alternate route (e.g., one dose of cefazolin IV and one dose subcutaneously 30 minutes before surgery) so that the second antibiotic peak occurs when the IV dose concentration is dropping. The antibiotic choice should be based on the expected contaminant. For instance, the colon and rectum are more likely to have anaerobic bacteria, so enrofloxacin would not be sufficiently broad spectrum for prophylaxis. Prophylactic antibiotics are discontinued after surgery. If the animal already has an infection somewhere or has serious intraoperative contamination, switch to a therapeutic antibiotic regimen.

Preoperative preparation

For rectal and colonic surgeries, enemas will help empty the area, but they should NOT be performed the day of surgery. Firm poop is easier to control than leaky, runny feces. For dogs with diarrhea, a purse string suture may be used to keep intraoperative leakage to a minimum. Plan in advance for things like feeding tubes. For instance, if you want to place a gastrostomy tube intraoperatively, the animal will need to be clipped and prepped higher on its side. If you want to place a nasogastric tube, have your technician pass it during surgery so you can palpate the tip in the stomach.

Reducing contamination and tissue trauma intraoperatively

Before opening contaminated viscera, set aside surgical instruments and sponges to be used for closure (or have 2 instrument packs). Designate a portion of your table to be “clean” and a portion to be “dirty”. Always perform the “clean” procedures (e.g. liver or lymph node biopsies, spays, gastropexies) first. Once the contaminated viscera is opened, use on the instruments and area of the table dedicated to “dirty” work. When the viscera is closed, you will need to count the dirty sponges and laparotomy pads and remove them from around the surgical site, clear off the dirty instruments, change your gloves, cover any contaminated areas with clean drapes/towels, and use the clean instruments and sponges during closure.

Isolate the surgical site with laparotomy pads that are moistened in the areas they will touch the inside of the animal. Do not soak the whole laparotomy pad if one portion will be laying on the animal’s skin or your cloth drapes- this will encourage hypothermia and bacterial contamination through the drape. Keep the viscera moist- the heat and exposure to lights and room air will dry them out and increase surface inflammation, which can lead to ileus and peritonitis.

Specialized atraumatic clamps (e.g., Doyen) can be used to occlude the healthy intestine to prevent luminal contents from entering the surgery area. Other nontraumatic options include umbilical tape, Penrose drains, or an assistant's fingers. The healthy intestine should be occlude at least 4 cm away from where the cut will be made: when intestine is transected it contracts, and shorter ends may slip through the clamps. Stay sutures can be placed in the stomach to hold it up out of the abdomen and to tense the gastric wall during incision.

If the viscera is opened outside of the abdomen (e.g., you do an enterotomy with the intestine pulled out of the abdomen and isolated with laparotomy pads) and you have prevented any contamination from entering the abdomen, you may not need to lavage the abdomen (focal contamination may be better than spreading it around with lavage!). If the abdomen is contaminated, lavage it until the fluid comes out looking clean. In a Great Dane with a ruptured stomach, this may mean you will need 10 to 20 liters of warm, sterile saline!

Suture materials and patterns in small animals

Most often the digestive tract of small animals is closed with 3-0 or 4-0 monofilament, intermediate or slowly absorbable suture (PDS, Maxon, Monocryl, Biosyn, etc.). Suture material should be on a taper or tapercut needle to prevent tissue tearing. Nonabsorbable material should not be used: the viscera are usually sufficiently strong by 10-17 days after surgery to be considered "healed". Additionally, if placed in a continuous pattern, nonasbsorbable suture can actually result in subsequent grass/fiber/hair obstructions when the suture works its way through the intestine and hangs into the lumen.

Most commonly, an appositional closure is used in the esophagus, intestine, gastric cardia, and pylorus/pyloric antrum. Inverting and 2 layer closures are only used in small animals when obstruction will not occur from luminal narrowing by the suture/suture pattern. Two layer closures are often used in the body of the stomach, with the first layer being a mucosal closure, because there is a lot more luminal space and the mucosa is very thick, vascular, and redundant.

Continuous suture patterns are acceptable in all parts of the gastrointestinal tract, as long as knots are square and secure and the tissues are healthy. There is no significant difference in complication rates when comparing intestinal anastomoses performed with continuous patterns versus interrupted patterns. However, if the health of the tissue along the incisional edge of the intestine is questionable, interrupted sutures are safer. Tissue bites of at least one layer of the closure should include the submucosa, which is the holding layer of the digestive tract. Tissue bite width and distance between bites depends on the thickness of the viscera: bites may be 2 mm wide and 2 mm apart in a ferret intestinal anastomosis, whereas they may be 3 to 4 mm wide and apart in a dog intestinal anastomosis.

Special tips for GI surgery

When possible, gastrointestinal incisions are made in the least vascular part of the GI tract. For example, a gastrotomy for foreign body removal or for gastrostomy tube placement is made in the body of the

stomach half way between the greater and lesser curvatures. An enterotomy or intestinal biopsy is performed on the antimesenteric surface. However, the incision should also be made in the healthiest area possible. For example, in a dog with a racquetball obstructing its intestine, the healthiest area of intestine around the area is downstream of the racquetball. However, the racquetball may be too large to milk downstream out of an enterotomy made in the skinnier intestinal segment. The surgeon will have to determine what location is the safest for the incision but will allow foreign body removal.

For intestinal resection and anastomosis, one end of the intestine may be wider than the other. Luminal disparity can be corrected by cutting the antimesenteric side of the narrow end so that the opening of the intestine on that side is widened.

Intestinal closures can be tested for leakage by occluding the intestinal lumen to either side of the closure with atraumatic clamps/fingers/etc. and distending the intestine with sterile saline injected into the lumen with a syringe. If leakage of saline occurs between sutures, additional sutures can be added. If leakage occurs through suture holes, additional sutures may not help but omentalization might. Omentum provides blood supply, healing factors, and a nice seal to gastrointestinal incisions. Once an enterotomy or anastomosis is completed, a portion of the omentum can be tacked over the site with simple interrupted sutures that go into the seromuscular layer of the intestine.

Permanent, planned adhesions in dogs and cats require muscle-to-muscle contact. To be sure you are making a permanent adhesion between the viscera and the abdominal wall (e.g., gastropexy), the peritoneum must be cut, and the serosa must be cut. The edges of each incision are sewn together so there is muscle-to-muscle contact. In some cases, cutting the serosa is scary (e.g., colopexy), so the peritoneum is cut and the serosa is “scarified” (traumatized by scraping or rubbing) to try and increase the likelihood of adhesion.

Postoperative care

Besides the normal considerations (analgesics, fluids as needed, correction of electrolyte abnormalities, maintenance of blood glucose, incisional care, monitoring for complications), animals undergoing gastrointestinal surgery may have special issues. For instance, some of these animals are nauseated and may need anti-emetics (e.g. maropitant). Use of pure mu opioids (morphine, hydromorphone, fentanyl) can slow GI motility, resulting in functional ileus. Intestinal dilation is painful and can stress surgical closures. Therefore, steps should be taken to try and improve GI tract function: animals should be walked to get things moving, they can be switched to buprenorphine if injectable opioids are needed, and they can be fed and offered water as soon as they are upright, ready to eat, and not nauseous. If a gastric or esophageal feeding tube has been placed, it is used only after the animal can hold itself upright. We do not know if dogs and cats undergoing GI procedures require a bland diet after surgery, but we do know that mucosal health relies on feeding the intestine directly (vs. IV).

If gastric or esophageal mucosal healing is a concern, animals may be given proton pump inhibitors (e.g. omeprazole, pantoprazole) to reduce gastric acid production. Use of nonsteroidal anti-inflammatories is considered on a case by case basis: for instance, these drugs are avoided in dogs with gastrointestinal ulcers or gastropexy for GDV (gastric dilatation volvulus correction), but they are sometimes used in young healthy dogs and cats that have undergone an uncomplicated foreign body removal.

Complications

The greatest concern after surgery of the esophagus, stomach, or intestine is incisional failure and leakage, which will lead to infection (peritonitis in the abdomen, pleuritis in the chest), septic shock, and death. Gastrotomy incisions rarely fail unless there is something wrong with the animal's tissues (e.g., neoplasia, vascular damage). Major complications have been reported in 5% to 12% of animals undergoing intestinal biopsy and include dehiscence, peritonitis, and hemorrhage. Clinical signs of intestinal leakage usually occur 1.5-3 days after enterotomy but may become apparent as late as 9 days after surgery. Complications of intestinal resection and anastomosis include dehiscence, leakage, infection, ileus, and short bowel syndrome. Anastomotic leakage rates were 3% and 11% in animals undergoing continuous and interrupted sutured anastomoses, respectively. Risk of dehiscence is increased in animals that have severe hypoalbuminemia or pre-existing peritonitis. Dehiscence of anastomoses is usually detected 1.5 to 5 days after surgery and may result from poor surgical technique or presence of diseased tissue at the anastomotic ends.

Feline Cardiomyopathy – an update ETVMA: 09/26/2021

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Introduction

Myocardial disease is an important cause of morbidity and mortality in domestic cats. Feline myocardial disease sometimes occurs in association with non-cardiac disease but when that is the case, it often is subclinical; idiopathic myocardial disease is more commonly responsible for clinical signs. Cardiomyopathy (CM) has been defined as a myocardial disorder in which the heart muscle is structurally and functionally abnormal in the absence of other cardiovascular diseases sufficient to cause the observed myocardial abnormality.

Recently proposed schema for classification of human cardiomyopathies have emphasized the cause or molecular basis of myocardial disease. Although genetic etiologic factors are likely important, feline CM remains largely idiopathic. Accordingly, the use of morphopathologic/ functional designations remains valid. Specifically, hypertrophic cardiomyopathy (HCM) is defined by diffuse or regional hypertrophy of a nondilated ventricle in the absence of hemodynamic stimuli for hypertrophy. Dilated cardiomyopathy (DCM) is characterized by left or biventricular dilation associated with diminished systolic myocardial function. Restrictive cardiomyopathy (RCM) is functionally defined by diminished ventricular compliance; the ventricle may have a normal or nearly normal appearance but left or biatrial dilation are consistent features. Arrhythmogenic right ventricular cardiomyopathy (ARVC) is characterized by fatty or fibrofatty replacement of right and sometimes left ventricular myocardium and often, arrhythmias. During this session, the pathophysiology, diagnosis and therapy of hypertrophic cardiomyopathy (HCM), the most common feline myocardial disease, will be emphasized.

Etiopathogensis

It is accepted that that HCM in humans is primarily a genetic disease that is associated with numerous mutations of genes that encode sarcomeric proteins. HCM is inherited in Maine coon cats and Ragdoll cats, and in cats of these breeds is associated with distinct mutations of the myosin binding protein C (MYBPC) gene. Familial occurrence of HCM has been observed in other purebred lines and in mixbreed cats; it is therefore possible that feline HCM generally is a genetic disorder. The causes of the other forms of primary myocardial disease that are observed in the cat have not been established. It is possible that some examples of RCM represent the sequela of endomyocardial inflammation or perhaps, an alternate phenotypic expression of mutations that are associated with HCM. The prevalence of feline DCM decreased radically after the recognition of the association between this disorder and nutritional taurine deficiency, but idiopathic DCM is still sporadically observed.

Epidemiology
Several investigators have retrospectively evaluated the population characteristics of feline HCM. HCM is not exclusively a geriatric disease; patients of all ages can be affected and the median age at the time of detection in one report was 4.6 years. Males are more often affected than are females. A substantive proportion - between 33 and 55% - of cats with HCM are subclinical (asymptomatic) when the disease is identified. The prevalence of HCM in apparently healthy cats is close to 15%. This prevalence is seemingly high but consistent with the current understanding of the HCM in humans; it is now accepted that HCM has a broad spectrum of phenotypic expression, often occurs in a subclinical form and is not inevitably associated with progression and poor outcome.

Pathophysiology
Diastolic dysfunction is thought to be the primary pathophysiologic mechanism responsible for clinical signs in HCM. Diastolic function refers to the ability of the ventricle to fill at low pressures. The primary determinants of diastolic function are the active process of myocardial relaxation and a mechanical property of the ventricle known as compliance. Diastolic dysfunction results in increased ventricular filling pressures when ventricular volumes are normal or small. High filling pressures are reflected “upstream” potentially resulting in atrial dilation and the development of pulmonary edema or pleural effusion. In feline CM, atrial dilation almost invariably precedes the development of congestive signs. Functional abnormalities in HCM are not limited to diastole. Although the clinical implications have been debated, most patients with HCM exhibit a valve motion abnormality – systolic anterior motion of the mitral valve or, SAM – that causes obstruction of left ventricular outflow. In affected cats, hydrodynamic forces, of which drag is most important, cause systolic movement of the mitral leaflets toward the interventricular septum. This abnormal valvular orientation causes dynamic, as opposed to fixed, obstruction of the left ventricular outflow and typically, concurrent mitral valve regurgitation. In addition, some patients, presumably those with long-standing HCM, develop systolic myocardial dysfunction resulting in a cardiac phenotype that has been referred to as “end-stage HCM” or “burnt-out HCM”.

Clinical Presentation / Diagnosis
Feline CM is identified when abnormalities are detected during physical examination of apparently healthy cats, when congestive heart failure (CHF) develops, or when CM is complicated by systemic thromboembolism. Many, but not all, cats with HCM have cardiac murmurs, but it is relevant that murmurs can develop in cats in which cardiac disease is absent. Furthermore, murmurs in cats, whether related to cardiac disease or not, are often labile, meaning that the intensity can change from moment to moment. Murmurs in cats can be provoked by increases in sympathetic activation, and an increase in murmur intensity documented during serial examinations does not indicate worsening of disease. Retrospectively evaluated case series have identified an association between the administration of corticosteroids and the development of CHF in cats. Some affected cats may have had pre-existing, but clinically silent, HCM but this

has not been established. The association is relevant because the long-term prognosis of corticosteroid associated CHF might be superior than for more typical presentations. Patients with congestive heart failure typically are presented for evaluation of respiratory distress caused by pulmonary edema or pleural effusion. Cats with CHF rarely cough. Hypothermia is frequently recorded. Tachypnea is generally evident and pulmonary auscultation may disclose adventitious lungs sounds in patients with edema or attenuated sounds in those with pleural effusions. While tachycardia caused by sympathetic activation is commonly observed in canine patients with heart failure, heart rates of cats with CHF do not differ from those of healthy cats and fairly often, feline patients with heart failure are bradycardic. Cardiac auscultation may reveal murmurs, gallop sounds and sometimes tachycarrhythmia, but these findings are not consistently present.
In the cat, radiographic patterns of specific chamber enlargement are not distinct. However, the chest film may reveal consequences of cardiac dysfunction; pleural effusion can be identified and the finding of pulmonary opacities together with cardiomegaly provides a non-invasive diagnosis of heart failure. Echocardiography is the only non-invasive method that can definitively characterize feline CM. Congestive heart failure is a clinical and/or radiographic diagnosis; the presence of heart failure cannot be determined based on echocardiographic data alone.
Blood concentrations of cardiac biomarkers including endothelin, atrial natriuretic peptide (ANP), B-type natriuretic peptide (BNP) and troponin have been evaluated in veterinary patients. Circulating BNP concentration has a particular role in the diagnostic evaluation of patients suspected to have heart failure. This hormone is released by atrial and ventricular cardiomyocytes in response to increases in ventricular filling pressures; potentially, it is a blood-borne diagnostic marker of the heart failure state. The diagnostic accuracy of the quantitative NT-BNP assay, for identification of moderate or severe subclinical CM or identification of cardiac causes of respiratory distress is relatively high. However, the need to submit samples to a central laboratory is a disadvantage in the urgent/emergent setting. The point of care assay provides a binary – normal/ abnormal – result and has a role when feline patients are presented for evaluation of respiratory distress, it is not possible to safely obtain diagnostic chest films and point of care echocardiographic examination is not available. Optimally, abnormal NT-BNP results are investigated echocardiographically.

Therapy
In order to facilitate the development of therapeutic guidelines, a modification of the AHA/ ACVIM scheme for staging of heart disease can be applied to feline CM.

The role of drug therapy in patients with subclinical (asymptomatic) Stage B CM is uncertain. It is likely that most patients with HCM have slowly progressive or even non-progressive, disease. Furthermore, there is currently no published evidence that any agent can slow the progression of HCM. Based on this, periodic echocardiographic re-evaluation rather than therapy is appropriate when faced with patients that have Stage B1 disease; meaning, subclinical HCM with left atrial dimensions that are normal, or

reflect only mild enlargement. In patients with stage B2 disease – those that have distinct left atrial enlargement – administration of clopidogrel is reasonable, in hopes of decreasing the probability of arterial thromboembolism [ATE]. It should be noted however, that evidence to support this approach is indirect and the incidence of ATE in patients with subclinical echocardiographically documented CM is not very high. Beta-blockade might also have a role in the management of subclinical HCM, particularly when SAM is evident. However, a recent open-label, nonrandomized trial failed to show a benefit of atenolol in this scenario.

Heart failure is a clinical syndrome characterized by high venous pressures and/or low cardiac output that results from cardiac disease. CHF results in clinical signs related to tissue edema or body cavity effusions. Pulmonary edema is a consequence of left ventricular failure. In cats, pleural effusions may result from left ventricular or biventricular heart disease. For patients that have developed congestive signs, those with Stage C CM, general, supportive measures are indicated. Supplemental oxygen can be administered through use of an oxygen administration cage or, if the patient is sufficiently tolerant, by mask, or nasal insufflation. Pleurocentesis should be performed when physical, radiographic or sonographic findings confirm that pleural effusion is responsible for respiratory distress.
In general, intravenous fluids should not be administered to patients with frank congestion. In the setting of CHF, infusion of fluid further increases venous pressures but does not improve cardiac performance. When cardiogenic pulmonary edema is present, diuretic administration is indicated. For acute decompensation, the intravenous route is preferred but intramuscular administration is appropriate when resistance to manual restraint or other factors make intravenous administration difficult or impossible. Preload reduction is used in the setting of heart failure because it may effectively eliminate clinical signs related to congestion. In general however, preload reduction does not improve cardiac performance. Indeed, aggressive reduction in filling pressures can decrease stroke volume potentially resulting in hypotension. Other than furosemide, for which efficacy is assumed, there are no medical interventions that have demonstrated efficacy in the management of feline heart failure. Because of this, the use of additional cardioactive agents in the management of acute decompensated heart failure is difficult to justify. Exceptions to this might be the use of antiarrhythmic agents for management of pathologic arrhythmias that contribute to the development of congestive signs, or perhaps the administration of pimbendan to patients with low output heart failure.

Evidence that supports administration of ancillary, medical therapy to patients with stage C is lacking. The results of a multicenter, randomized, placebo-controlled trial that had been designed to evaluate the relative efficacy of atenolol, diltiazem and enalapril in feline patients with diastolic heart failure have been reported but not published. The primary end-point of the trial was recurrence of congestive signs and none of the agents was superior to placebo in this regard, although atenolol was inferior. Administration of pimobendan to cats with CM has been retrospectively evaluated, and the drug is

seemingly tolerated. The results of a prospective, double-blind placebo controlled trial have recently been reported. For patients enrolled in this exploratory trial, pimobendan administration did not lead to improved 180-day outcome.

Arterial thromboembolism is a serious and often unexpected complication of feline CM. It is unexpected because ATE is the first clinical manifestation of CM in the majority of patients in which it occurs. Emergent care of patients with ATE most importantly is supportive and includes analgesia. ATE is almost always associated with advanced CM that has resulted in left atrial enlargement and diagnostic evaluation to identify evidence of edema/effusion is important. Parenteral administration of anticoagulant therapy – unfractionated or low molecular weight heparin is reasonable in the short-term. The results of a clinical trial that compared aspirin to clopidogrel for secondary prevention of ATE provided strong evidence of the superiority of clopidogrel.

Prognosis

The results of a recent retrospective analysis of risk and mortality in HCM provide useful information regarding prognosis associated with subclinical [Stage B] disease. In general, the condition is benign but has important long-term clinical consequences. Over the course of follow-up, which for some patients extended 10 years, total cardiovascular mortality was 28%. Survival after the development of congestive heart failure or the occurrence of ATE was relatively brief with a median survival of less than one year.

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Arrhythmogenic Right Ventricular (Boxer Dog) Cardiomyopathy

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Although congestive heart failure is occasionally observed, heart muscle disease in North American boxer dogs is principally characterized by ventricular tachyarrhythmia (VTA). Often, arrhythmias are incidentally detected during routine examination but the disorder may become clinically apparent when syncope or sudden unexpected death is a consequence of ventricular tachycardia. The cause, clinical presentation as well as the diagnostic and therapeutic approaches to this common disorder will be reviewed during this session.

ETIOPATHOGENESIS

Cardiomyopathy has been defined as a myocardial disorder in which the heart muscle is structurally and functionally abnormal in the absence of other cardiovascular diseases sufficient to cause the observed myocardial abnormality. Boxer dog cardiomyopathy is a breed-associated myocardial disease that is typically characterized by electrical dysfunction. It is generally accepted that the syndrome of inherited VTA observed in Boxer dogs is a form of arrhythmogenic right ventricular cardiomyopathy (ARVC).

ARVC is histologically characterized by fatty or fibrofatty replacement of right and sometimes left ventricular myocardium. The genetic basis of this disease in affected human beings has been extensively investigated. Numerous causative mutations have been identified although most affect genes that encode desmosomal proteins. VTA in boxer dogs is familial; the results of pedigree analysis are compatible with an autosomal dominant mode of inheritance. Meurs et al. recently identified a mutation of the striatin gene that is strongly associated with the ARVC phenotype in boxer dogs. The pathogenesis of ARVC is believed to involve disruption of cellcell connections that result in cell death and impaired electrical coupling. The right ventricle is predominantly affected but left ventricular involvement is also observed. The phenotypic expression of ARVC mutations is diverse, which may be explained by genotypic heterogeneity, variable expressivity and perhaps environmental influences. Although the disease has a genetic basis, clinical signs associated with ARVC generally have an adult onset.

CLINICAL PRESENTATION

In the 1980's Harpster classified the clinical presentation of Boxer dog cardiomyopathy:

- **Category I** - ventricular arrhythmias occur in the absence of clinical signs; these patients are identified when presented for routine veterinary care or for evaluation of non-cardiac illness
- **Category II** - syncope/collapse is observed ○ **Category III** – congestive heart failure resulting from systolic myocardial dysfunction

In some Boxer dogs, there is progression from Class I to Classes II or III but this is not inevitable. Many Boxer dogs with ARVC are presented with VTA in the absence of myocardial dysfunction; that is, the echocardiogram is normal or is subtly abnormal. Dilated cardiomyopathy is observed in boxer dogs but in many geographical regions appears to be less common than ARVC. However, recently published data demonstrate a strong association between the striatin mutation and echocardiographically evident myocardial dysfunction, suggesting that dilated cardiomyopathy and VTA in boxer dogs may be different phenotypic expressions of the same desmosomal defect.

History/Physical Findings

ARVC is often incidentally identified when cardiac arrhythmias are detected during routine veterinary examination. If the disease becomes clinically evident, episodes of weakness or syncope are the signs most often reported by pet-owners although tachypnea or abdominal distention due to ascites may also be observed. Premature beats and paroxysmal tachycardia are evident on physical examination. Murmurs are occasionally heard but most affected patients have minimally altered cardiac structure. Accordingly, murmurs – directly resulting from ARVC – are uncommon. Many outwardly healthy boxer dogs have soft basilar systolic murmurs that reflect either a narrow aortic root – a breed-associated trait – or mild forms of aortic stenosis. However, these murmurs do not have a known relationship to ARVC.

Electrocardiographic Findings

The electrocardiographic hallmark of boxer dog ARVC is the occurrence of VTA characterized by premature ventricular complexes (VPC) and in many cases, ventricular couplets and ventricular tachycardia. Most, but not all, ventricular ectopic complexes in affected boxer dogs have a left-bundle block configuration; that is, the QRS is wide and upright with a negative Twave in lead II. Some affected boxers have relatively few VPC that occur singly and infrequently while others have numerous VPC and sustained paroxysms of ventricular tachycardia (VT).

Ambulatory Electrocardiography

In the management of canine ARVC, 24 hour ambulatory electrocardiographic (Holter) monitoring can provide data that: support, or refine a provisional diagnosis, clarify the cause of syncope and guide antiarrhythmic therapy. The use of ambulatory electrocardiographic event recorders also has a role in the assessment of patients with known or suspected ARVC. These devices are digital loop recorders that can be affixed, by adhesive electrode patches, to patients for days or even weeks. A button on the device interrupts the digital loop and can be pressed if a pet-owner observes an episode of weakness or syncope. The preserved electrocardiographic data sheds light on the cause of intermittent clinical signs by providing a clear association between cardiac rhythm and patient behavior. Implantable loop recorders intended for placement within a surgically created subcutaneous pocket are also available and can usefully document electrocardiographic events that are associated with infrequent clinical episodes.

Echocardiographic Findings

In most affected individuals, the echocardiogram is normal or nearly so. Right atrial and right ventricular dilation can be observed but these findings are neither consistent nor essential for the diagnosis. Systolic myocardial dysfunction, with or without left ventricular dilation, is sometimes observed but it is not known if this is the result of histologic left ventricular involvement, the consequence of persistent tachycardia – tachycardia-induced cardiomyopathy – or reflects a distinct disease process.

DIAGNOSTIC APPROACH

The diagnostic and therapeutic approach to the patient suspected to have ARVC is determined by the clinical presentation. When a patient is urgently presented after a recent syncopal episode and a persistent, rapid ventricular tachycardia is evident, a limited diagnostic approach – perhaps electrocardiography alone – might determine the initial therapeutic strategy. More extensive diagnostic evaluation is generally appropriate for patients that are clinically stable when presented. This diagnostic evaluation may include: thoracic radiography, echocardiography, ambulatory electrocardiography, abdominal sonography and assessment of laboratory data.

When encountered in a mature boxer dog, the occurrence of ventricular ectopy of typical QRS configuration suggests the diagnosis of ARVC. However, other cardiac disorders and indeed, non-cardiac disease, can also predispose to VTA. It is important to recognize that there is a subpopulation of boxer dogs with and without ARVC that experience syncopal episodes that are associated with transient, presumably reflex-mediated, bradycardia. Often these events are precipitated by exertion. When syncopal episodes are relatively infrequent, it is not always possible or practical to define the electrocardiographic rhythm during events. It is probably reasonable to assume that ventricular tachycardia is the cause of syncope when frequent, complex VTA characterized by paroxysms of VT are electrocardiographically documented. However, syncope associated with bradycardia may become more frequent if agents such as sotalol are administered. Therefore, caution is appropriate when making suppositions regarding the cause of fainting when resting electrocardiography fails to reveal dramatic arrhythmias.

THERAPY

Arrhythmias are clinically important because they can cause clinical signs - such as syncope - and because they can cause sudden unexpected cardiac death (SCD). SCD is commonly caused by rapid VT that degenerates to ventricular fibrillation (VF) - VF is a pulseless rhythm that is lethal unless promptly terminated.

There are essentially three reasons to treat arrhythmias:

1) clinical signs are associated with the arrhythmia
2) there is reason to believe that the patient is at risk of sudden death and that treatment will prevent this
3) the burden of arrhythmia places the patient at risk for the development of tachycardia induced cardiomyopathy

The subject of antiarrhythmic therapy is complex because risk factors for poor outcome are largely unknown and while accepting that “absence of evidence is not evidence of absence”, there are no data indicate that the commonly chosen antiarrhythmic agents can prevent sudden death. It is therefore important to consider the relative risk: benefit of antiarrhythmic therapy prior to the administration of antiarrhythmic agents. It is reasonable to treat arrhythmias when they are associated with clinical signs or when the arrhythmia is sufficiently severe that the development of clinical signs can be anticipated. The treatment of subclinical (“asymptomatic”) arrhythmias must be considered more carefully.

Emergent therapy of VTA associated with ARVC generally consists of parenteral therapy with agents such as lidocaine or procainamide. The pathology underlying ARVC is irreversible. Therefore, palliative therapy for canine ARVC consists of the chronic administration of oral antiarrhythmic agents. In canine ARVC, sotalol – a potassium channel antagonist with betablocking properties - is the most efficacious single agent in terms of arrhythmia suppression. Whether or not this efficacy translates to a reduction in sudden death, is not known. Data from ambulatory electrocardiographic monitoring is often use to evaluate the effectiveness of therapy, though the value of this approach has not been demonstrated and it is relevant that the frequency of ventricular arrhythmia in affected boxers might vary by as much as 75% independent of therapy. Monitoring of clinical signs is a practical method by which effectiveness of therapy can be judged. In clinically stable patients, careful consideration of the relationship between arrhythmia and clinical signs should guide therapy as recently published data suggest that ventricular tachycardia is not the most important cause of collapse in boxer dogs.
It is not possible to make definitive statements with regard to prognosis. Occurrence of syncope and more frequent or complex VTA are associated with poor outcome. However, studies of the natural history of boxer ARVC provide evidence that survival of affected dogs does not differ from that of unaffected boxer dogs.

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Diagnosis and Management of Pulmonary Hypertension
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Introduction

Pulmonary hypertension (PH), defined by abnormally great pressures in the pulmonary vasculature, is a hemodynamic state that can develop in association with a variety of cardiovascular, respiratory and systemic diseases. Clinical signs of PH include cough, tachypnea and exertional syncope. These signs are not diagnostically specific and because of this, the identification of patients for which PH is an appropriate therapeutic target can be difficult. During this presentation, the pathophysiology and therapy of PH will be addressed; clinical findings which help to distinguish patients with primary left heart disease from those with pulmonary arterial hypertension will be outlined.

Pathogenesis

In healthy individuals, pulmonary vascular resistance (PVR) – the hydraulic forces that must be overcome for a pressure difference to result in flow – is lower than is systemic vascular resistance. As a result, pulmonary arterial pressures are lower than systemic arterial pressures. Pulmonary hypertension (PH) refers to the abnormally great pressures in the pulmonary vascular system. In people, PH is defined by a mean pulmonary artery pressure that exceeds 25 mmHg. Mean pulmonary artery pressure can be only be obtained invasively, by right heart catheterization (RHC). Diagnostic RHC is rarely performed in veterinary patients, and the diagnosis is generally based on echocardiographic findings.

Pulmonary artery pressure (PAP) is related to pulmonary blood flow (Q) and PVR according to Ohm's Law: PAP_{mean} - LAP_{mean} = Q x PVR, where: LAP = left atrial pressure, Q = cardiac output [flow] and PVR = pulmonary vascular resistance. None of these quantities is routinely measured, but the concept of PVR is important because it relates to therapeutic approach. PH can result from a rise in PVR, an increase in pulmonary blood flow, an increase in LAP or combinations of these factors.

Pathophysiology

When PH results from high PVR, it is described as "pre-capillary PH"; vasoconstriction and/or vascular remodeling are the principal causes of high PVR, although pulmonary thrombosis can contribute. Left atrial hypertension resulting from left heart disease is the primary cause of "post-capillary PH'. The right ventricle must generate a pressure that is adequate to propel the stroke volume not just to the lungs, but to the left atrium; as a result, a pathologic increase in left atrial pressure necessitates an increase in right ventricular and pulmonary arterial systolic pressure. In some patients with left heart

disease, the increase in right ventricular – and therefore pulmonary arterial – pressure, initiates a cascade of vasoconstriction and vascular remodeling, increasing PVR and resulting in PAP that is disproportionately high relative to left atrial pressure. PH imposes a pressure load on the right ventricle and potentially results in right ventricular hypertrophy, functional pulmonary and tricuspid valve regurgitation, myocardial dysfunction and right-sided congestive heart failure.

Clinical presentation

Clinical signs associated with PH include cough, tachypnea, exercise intolerance, syncope, and abdominal distention due to ascites. It is relevant that these clinical signs are associated with PH, but the association is not necessarily causal. For example, cough is unlikely to result directly from PH, but is more apt to reflect an underlying disease that has caused PH.

Physical findings may include adventitious lung sounds and a right apical systolic murmur resulting from tricuspid valve regurgitation. Pulmonary valve regurgitation is commonly detected echocardiographically but is rarely audible. When the clinical presentation includes respiratory distress and a right apical murmur, PH [and pulmonary thromboembolism] should be diagnostic considerations because the therapeutic approach to these disorders is different from that which is appropriate for left-sided congestive failure.

Diagnostic Evaluation

The definitive diagnosis of PH is through direct measurement of pulmonary artery pressures, but in veterinary patients, the diagnosis is more based on echocardiographic findings.

<u>Echocardiography</u>

Echocardiographic evidence of tricuspid valve regurgitation (TR) is commonly observed in patients with PH. The velocity of the TR jet, obtained by continuouswave Doppler, is related to the systolic pressure difference between the right atrium and the right ventricle by the simplified Bernoulli equation ($\Delta P = 4v^2$ where ΔP is the pressure difference and v is the velocity of the regurgitant jet measured by Doppler echocardiography).

In the absence of pulmonary stenosis (PS), right ventricular and pulmonary artery pressures are *equal* during systole. Thus, measurement of the velocity of the TR jet provide a noninvasive *estimate* of systolic pulmonary artery pressure. The diagnostic accuracy of other echocardiographic variables including various systolic time intervals, tricuspid annuluar plane systolic excursion (TAPSE) as well the fractional change in dimensions of the right pulmonary artery have been evaluated. In general, these surrogate measures can provide supportive evidence and are considered in addition to the velocity of TR or when TR is absent. It is axiomatic that they are less accurate than the criterion [“gold”] standard to which they have been compared. Partly because of the inaccuracy of

Doppler echocardiographic estimation of PAP, the ACVIM consensus panel recommended a ***probabilistic approach*** to the diagnosis of PH. That is, the velocity of TR is considered in the context of other echocardiographic variables, and a low, intermediate or high diagnostic probability of PH is assigned based on these findings. Echocardiographic evaluation of left atrial size is generally used to determine if pulmonary hypertension is the result of high pulmonary vascular resistance ["pre-capillary"] or is the consequence of increases in left atrial pressure resulting from left heart disease ["post-capillary"]. In patients for which there is an intermediate or high probability of PH, normal or diminished left atrial dimensions provide indirect evidence that PH is pre-capillary; left atrial enlargement provides evidence that the PH is at least partly post-capillary.

An etiologic classification of types of PH, based on pathophysiologic mechanisms, recently was proposed:

Classification

- Group 1 - PAH
- idiopathic pulmonary arterial hypertension
- Group 2 - LHD
- canine mitral valve disease
- Group 3 - hypoxia
- canine respiratory tract disease
- high mountain disease
- Group 4 - thrombotic
- Group 5 - parasitic
- heart worm disease
- Group 6 - multifactorial/unclear mechanisms

Reinero C, et al. ACVIM consensus statement guidelines for the diagnosis , classification , treatment , and monitoring of pulmonary hypertension in dogs. 2020; (January):1–25.

Therapy of Pulmonary Hypertension

Treatment of causative or underlying disorders such as heartworm disease or specific pulmonary diseases is essential. Various vasodilators have been used in attempts to decrease pulmonary vascular resistance, but sildenafil is the agent that use most often in canine patients. Sildenafil is an inhibitor of phosphodiesterase type 5 and is a relatively selective dilator of pulmonary arterioles; it generally is indicated when clinical signs result from pre-capillary pulmonary hypertension

There are numerous causes of PH, but after Dirofilariasis is excluded, the distinction between pre- and post-capillary PH is the most important therapeutically relevant

goal. In general, the initial therapeutic approach to patients with postcapillary PH is directed toward optimization of treatment for left-heart disease. Agents such as sildenafil that result in relatively selective dilation of pulmonary arterioles are intended to reduce PVR. The resultant increase in pulmonary blood flow can potentially raise left ventricular filling pressures and precipitate the development of cardiogenic pulmonary edema. As a result, agents such as sildenafil should not be used, or used only with caution, in patients with left heart disease, and generally only after resolution of pulmonary edema.
In contrast, when clinical signs are the result of high pulmonary vascular resistance and decreased systemic output, as is often the case in pre-capillary PH, the use of specific therapies such as sildenafil often is appropriate.

Idiopathic Pulmonary Arterial Hypertension

For mostly valid reasons, aggressive diagnostic evaluation including advanced diagnostic imaging and lung biopsy are seldom performed in patients with respiratory distress and PH. Arguably then, pre-capillary PH, or pulmonary arterial hypertension (PAH), often is idiopathic only because diagnostic evaluation is incomplete. Regardless, there is an important syndrome of PAH in patients that do not have Dirofilariasis or a history of chronic respiratory disease, which is typically observed in older, small- breed dogs. Brachycephalic dogs might be over-represented. Clinical signs include cough, tachypnea/respiratory distress, and syncope, the latter typically observed on exertion. The precise cause is unknown but the pathogenesis relates to vasoconstriction / vascular remodeling that causes an increase in PVR, and the development of PAH. As implied, the diagnosis is presumptive and based on exclusion of known causes of PH, such as heartworm disease. The syndrome is echocardiographically characterized by a normal or small left atrium / left ventricle - caused by diminished pulmonary venous return - and evidence of PH.

The prognosis is generally poor, although some patients respond favorably to administration of sildenafil and supplementary oxygen. The distinction between PAH and acutely decompensated left-sided heart failure can be challenging, but important because diuretic treatment is potentially harmful in the setting of idiopathic PAH. In these patients, the clinical signs are not the result of high pulmonary venous pressures. Conversely, as stated, agents such as sildenafil can harm in the setting of severe left sided heart disease. If echocardiography is not available, it is important to consider the possibility that idiopathic PAH is responsible for clinical signs such as tachypnea/ respiratory distress/exertional syncope, particularly when: ○ distinct radiographic left atrial enlargement is absent ○ there is a lack of response to diuretic therapy ○ a cardiac murmur that is loudest over the *right* apex is identified.

Additional Readings / Complete References Available On Request

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Current management of canine congenital heart disease
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Introduction

This session is intended to provide a synopsis of the current diagnostic and therapeutic approach to congenital heart disease (CHD). The management of *canine* CHD, including the use of minimally invasive, transcatheter techniques in the most commonly occurring malformations will be emphasized.

Etiology of CHD

The cause of canine CHD is largely unknown although a genetic basis has been proven for a few specific malformations. Barring identification of a genetic mutation that is consistently associated with an abnormal phenotype, planned breeding studies or careful evaluation of accurate pedigrees are necessary to demonstrate genetic transmission of a congenital malformation. Indeed this has been accomplished for a few defects. For example, subvalvular aortic stenosis is an inherited trait in Newfoundland dogs, genetic transmission of pulmonic stenosis has been demonstrated in the beagle hound and a spectrum of conotruncal malformations that includes Tetralogy of Fallot is inherited in keeshonden. Pronounced breed predispositions are recognized for some forms of canine CHD and in these cases, it is probable that the defect has a genetic basis.

Recognition and Diagnosis of Congenital Heart Disease

The patient history of those with CHD rarely provides specific findings and the majority of canine patients with CHD are free of clinical signs when the disorder is first detected. Importantly, normal growth and lack of clinical signs do not imply that CHD is of no clinical importance nor necessarily imply a favorable long-term prognosis. This serves to emphasize the importance of accurate diagnosis even for cases in which clinical signs are absent. The vast majority of cardiac malformations result in cardiac murmurs. Therefore, congenital heart disease usually is first identified when outwardly healthy puppies or kittens are presented for routine veterinary evaluation. It is noteworthy that some normal puppies and kittens have murmurs that do not result from cardiac disease. These murmurs, known as innocent murmurs, are soft, always systolic, and usually heard best over the left heart base. The intensity of an innocent murmur may vary from day to day or even from moment to moment in association with changes in heart rate. Murmurs that are innocent generally become inaudible before the patient is 8 months of age. While congenital disease can result in a soft murmur, a loud cardiac murmur, or one that is diastolic or continuous, invariably suggests the presence of cardiac disease and further diagnostic investigation is indicated. Further evaluation may include

electrocardiography and thoracic radiography but these tests rarely provide diagnostically specific information. Doppler echocardiography performed by an experienced examiner provides a definitive, non-invasive diagnosis in practically all cases of CHD.

THERAPY OF CONGENITAL HEART DISEASE

Optimally, treatment of CHD is through surgical methods or interventional cardiac catheterization techniques. Medical therapy is apt to be palliative only.

Surgical Techniques / Cardiopulmonary Bypass

The availability of cardiopulmonary bypass - or rather, its lack - is one factor that limits the effective management of CHD in veterinary medicine. A few defects can be surgically managed without the need for bypass; a PDA for example, can be ligated without entering the circulation. However, defects that require access to the left ventricle and/or prolonged manipulation can only be performed with cardiopulmonary bypass. Surgical procedures that require cardiopulmonary bypass currently are performed only at a few veterinary institutions in North America and elsewhere.

Interventional Catheterization Techniques

Originally, cardiac catheterization - the art and science of manipulating catheters within the cardiovascular system - was a diagnostic technique. Usually under fluoroscopic guidance, catheters can be used to selectively deliver dye, to measure blood oxygen contents and to directly measure intracardiac pressures. Beginning in the 1960's, a number of resourceful pediatric cardiologists introduced catheterization techniques that were intended to treat or "intervene". The techniques that are used most often in veterinary cardiology are pulmonic balloon valvuloplasty and transcatheter occlusion of patent ductus arteriosus

PATENT DUCTUS ARTERIOSUS (PDA)

The ductus arteriosus (DA) connects the ventral aspect of the descending aorta to the bifurcation of the main pulmonary artery. During fetal life, the DA diverts the majority of the right ventricular stroke volume to the aorta. In normal individuals, closure of the ductus occurs within days of birth; the process is complex but involves a prostaglandin cascade. Failure of the duct to close, which in most cases is explained by a lack, or relative lack, of ductus specific smooth muscle, results in a persistently patent DA or, PDA. A genetic basis for failed closure of the DA has been documented in miniature / toy poodles. When the duct is the only defect, and pulmonary vascular resistance decreases following birth, blood shunts from the high pressure / high resistance systemic circulation to the low pressure / low resistance pulmonary circulation. Therefore, the shunt direction is from left-to-right; this increases pulmonary blood flow *and* pulmonary venous return imposing a volume load on the left atrium and ventricle. The development of myocardial dysfunction, mitral valve regurgitation and pulmonary edema are potential consequences of the shunt. PDA results in a continuous murmur; that is, a murmur that begins in systole and continues**,** without interruption**,** into diastole.

When the shunt is substantial, the arterial pulse is hyperkinetic or, “bounding”. Electrocardiography often discloses evidence of left ventricular hypertrophy. Radiographically, there is left-sided cardiomegaly that is roughly commensurate with the size of the shunt**.** Additional findings may include prominence of the main pulmonary artery, proximal aorta and left atrium. The diagnosis is confirmed echocardiographically. Specific findings of course depend on the size of the shunt but typically include left atrial and left ventricular enlargement. Doppler examination provides evidence of a continuous flow disturbance within the main pulmonary artery. The ductus, and certainly the ductal orifice of the pulmonary artery**,** can be identified in almost all cases.

Therapy

A minority of patients have a small, well-tolerated duct but usually, intervention is indicated when a PDA is identified in a dog that is younger than 24 months old. Surgical ligation can be performed without cardiopulmonary bypass and though minimally invasive transcatheter occlusion has become routine, surgical ligation remains an appropriate therapeutic approach that is associated with low mortality.

Transcatheter PDA occlusion using different devices and subtly different techniques has been reported. Thrombotic Gianturco coils were widely used until two veterinary cardiologists, Ngyuenba and Tobias, in collaboration with a manufacturer of cardiovascular devices, developed a metallic plug, the ACDO, or Amplatz Canine Ductal Occluder, that was specifically designed to occlude the canine ductus. Use of this device has almost completely supplanted the use of the Gianturco coil in veterinary practice. The ACDO can be used to successfully occlude PDA over the broad range of ductal size and morphologies.

Numerous variations on the basic technique of transcatheter ductal occlusion have been reported. Most often the devices are deployed within the ductus after retrograde catheterization of the aorta. Briefly, after induction of general anesthesia, access to the femoral artery is most often obtained after a small inguinal incision but vascular access can be percutaneously obtained using the modified Seldinger technique. Using fluoroscopic guidance, an angiographic catheter and/or vascular sheath is advanced to the ascending aorta and an angiogram recorded after injection of contrast material in the proximal descending aorta. Then, the device is advanced through a catheter or vascular delivery sheath and deployed within the ductus. Major complications of transcatheter intervention for PDA include intra-operative death, incomplete occlusion, post-procedural hemolysis, and coil migration. Mortality associated with transcatheter intervention for PDA generally is quite low, near 2%, although higher mortality has been reported in small studies that specifically recruited high risk patients. Body size is an important determinant of the suitability of the technique. Because of the size of the delivery devices, transcatheter occlusion of PDA in patients that weight less than 3 kg is problematic.

SUBVAVULAR AORTIC STENOSIS (SAS)

In dogs, left ventricular outflow tract obstruction most commonly results from the presence of a subvavular fibrous or fibrocartilaginous ring that develops in the first

weeks of life. The pressure gradient across the obstruction, which can be measured - by cardiac catheterization - or estimated - by Doppler echocardiography - is used as a clinical measure of stenosis severity. Pressure gradients that are less than 40 mmHG are mild and those greater than 100 mmHg are severe; intermediate gradients are described as moderate. Aortic stenosis is most common in large breed dogs including Golden retrievers, Rottweilers, as well as Boxer, Newfoundland and German Shepherd dogs. Clinical signs in puppies are uncommon; syncope and sudden death are observed in young adults with severe obstructions.
Cardiopulmonary bypass is required for surgical repair. Although a new technique in which a “cutting balloon” is used might have promise, published data suggest that neither surgical correction nor balloon dilation improve survival relative to medical therapy consisting atenolol.
Evidence that administration of atenolol is superior to placebo or no treatment is lacking.

PULMONIC STENOSIS (PS)
Right ventricular outflow tract obstruction usually results from valvular dysplasia. PS occurs commonly in terriers, English Bulldogs, miniature schnauzers and Samoyeds.
Therapy
PS can be treated surgically but is more often addressed by transcatheter balloon dilation. The pressure gradient that represents an indication for intervention is not known with precision although it is known that balloon dilation confers a survival benefit for those with gradients that exceed 80 mmHg. Balloon dilation of PS is performed under general anesthesia. After aseptic preparation of the groin or cervical region, access to the jugular or femoral vein is typically obtained percutaneously. After hemodynamic and angiographic studies, an end-hole diagnostic catheter is guided fluoroscopically into the pulmonary artery. The end-hole catheter is then exchanged for a balloon dilation catheter over a long wire-guide. Balloon catheters are available from manufacturers in numerous sizes; appropriate dimensions are determined by the size of the patient and echocardiographic (or angiographic) measurement of the pulmonic valve annulus. Several inflations with saline-diluted contrast material are performed after the balloon has been positioned across the stenosis. The inflation is observed fluoroscopically; the obstructive valve results in the appearance of a “waist”; ideally, the waist disappears suddenly during the first inflation and is not observed during subsequent attempts. After balloon inflation, catheters are withdrawn and patient is recovered. Most patients are discharged the day after the procedure which permits echocardiographic re-evaluation after complete recovery from anesthesia. Success seems to depend to a great extent on the nature of the stenosis; patients with isolated valvular stenosis in which there is fusion of otherwise normal valve cusps tend to benefit the most from the procedure.

VENTRICULAR SEPTAL DEFECT
A defect of the interventricular septum results in a communication between the left and right ventricles. Presumably because of its complex embryonic derivation, most

ventricular septal defects (VSD) involve the membranous part of the septum; generally these defects are subaortic with a right ventricular orifice that is immediately subjacent to the septal tricuspid leaflet. The clinical importance of the defect depends on: the size of the defect and, the presence or absence of other concurrent defects. When the VSD is the only cardiovascular lesion, blood shunts from left-to-right; this increases pulmonary blood flow and pulmonary venous return resulting in a volume load on the left atrium and left ventricle. A VSD results in a systolic murmur; this is because the pressure difference between the ventricles drops to nearly zero during diastole.

Therapy

Definitive repair requires cardiopulmonary bypass but pulmonary artery banding is occasionally used as a palliative surgical intervention. Transcatheter occlusion of VSD is routinely practiced in pediatric cardiology and transcatheter closure in dogs has been reported. Most VSD in dogs are small, well tolerated and do not require treatment.

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