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PTYALISM IN DOGS AND CATS – A SHORT REVIEW.

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ANATOMY AND PHYSIOLOGY

Ptyalism (also called hypersalivation or rarely sialorrhea) is defined as pathologic overproduction of saliva. It has also been defined as increased amount of saliva in the oral cavity, which may be caused by decreased clearance of saliva (i.e. inability to retain saliva in the oral cavity or problems with swallowing). There are four paired major salivary glands in dogs and cats. Salivary glands are unique among the digestive glands as no endocrinological regulation is needed. The primary stimulus for salivation is taste and afferent input is carried to the solitary nucleus in the medulla via the facial and glossopharyngeal nerves. Input from other senses, such as smell and sight are also integrated in the solitary nucleus. Parasympathetic efferent pathways for the sublingual and submandibular glands are from the facial nerve via the submandibular ganglion and for the parotid gland from the glossopharyngeal nerve via the otic ganglion. These pathways regulate fluid secretion by releasing acetylcholine at the surface of the salivary gland acinar cells. Macromolecule secretion is regulated by noradrenalin release from sympathetic nerves. This illustrates why thick and ropy saliva can sometimes be seen with increased sympathetic tone in stressed animals, while watery profuse saliva is typically seen with parasympathetic stimulation. Numerous causes may account for hypersalivation and the inciting cause may originate in different locations. The primary cause can originate from the oral cavity, the salivary glands themselves, the esophagus or the gastrointestinal tract. Moreover, hypersalivation can be seen as a consequence of neurologic (peripheral or central), metabolic, infectious or immune-mediated diseases, or a drug reaction.

The obvious clinical sign is drooling. It can vary from mild to severe, intermittent or permanent. In some dogs with longer or curly hair around the face, all that is found on physical examination is a wet mouth.

This is also common in cats. Saliva with a sanguineous or purulent appearance usually points at a local problem in the oral cavity. When taking the history, questions should include possible toxin exposure (plants in the household, concurrent medications of pet and owner), as well as additional gastrointestinal (vomiting, diarrhea, anorexia) and urinary signs (micturition, dripping of urine).

CLINICAL EXAMINATION

A thorough inspection of the oral cavity should be performed and it should be noted that a complete oral inspection in cats and some dogs will require at least sedation or general anesthesia. Inability to open the mouth is most commonly associated with a **masticatory muscle myositis**. In the acute form dogs present with swelling of the jaw muscles, drooling, and pain on opening the mouth (which may not be clinically apparent until the clinician actually examines these painful patients under anesthesia). This condition is limited to the masticatory muscles because they have a molecular structure, called 2M muscle fibers, which are found nowhere else in the dog's body. Masticatory myositis results when the immune system's antibodies specifically target these 2M muscle fibers. Any breed can be affected, but young King Charles Cavalier Spaniels, Samoyeds, Dobermans, German Shepherds and Rottweilers are overpresented.[1] Demonstration of serum 2M autoantibodies confirms the clinical suspicion. An inflammatory myopathy with clinical signs of pharyngeal dysphagia with excessive drooling, marked atrophy of the masticatory muscles, and difficulty swallowing has recently been identified in several young adult Hungarian Vizsla dogs.[2] The underlying etiopathogenesis of this breed-specific polymyositis remains undetermined.

Another differential for the inability to open the mouth in a hypersalivating dog with bilateral painful swellings of the mandible is **craniomandibular osteopathy (CMO)**. It is a non-neoplastic, proliferative bone disease affecting primarily bones of the skull namely, the parietal and occipital bones, the tympanic bulla and the mandibular rami, as well as the temporomandibular joint.[3] It is a self-limiting disease occurring in young dogs (3 to 10 months). CMO can affect either gender and was initially predominantly seen in the West Highland White Terrier, Scottish Terrier, Boston Terrier, Cairn Terrier, and other terrier breeds. Meanwhile CMO has also been diagnosed in large breed dogs, such as the Labrador retriever, Great Dane, Doberman, Boxer, Shetland Sheepdog, and Pyrenean Mountain breeds. Treatment of CMO generally consists of pain management allowing the dog to eat without much discomfort. Providing IV fluids and nutritional support in patients that are unable to eat properly is critical. Although comparatively obvious with additional spasticity in other muscle groups **tetanus** can also cause lockjaw caused by contraction of masticatory muscles, and excessive salivation.

The oral exam includes the inspection of all teeth (abscesses, fractures) and periodontal tissues (although resorptive lesions in cats only very rarely present with hypersalivation), as well as the sublingual check for linear foreign bodies (strings) or sialoceles, as they most frequently affect the sublingual salivary gland or its duct. Sometimes subtle lesions, like small vesicles or bullae can be missed in drooling cats with oral herpes or calici virus infections, only to become evident shortly afterwards. The palatine should be examined closely for focal lesions compatible with eosinophilic granulomas especially in breeds such as Siberian Huskies, Alaskan Malamutes and King Charles Cavalier Spaniels. Ulcerative gingival disease or inflammation of the fauces in cats can be immune-mediated (usually secondary to bacterial plaques), but should also make the clinician consider the possibility of FIV testing in outdoor cats.

Any **toxic** / **caustic agent** can cause oral mucosal changes, and therapy is usually supportive. A surgical biopsy should be taken from lesions that do not heal with symptomatic treatment. Erosive oral mucosal lesions occur after ingestion of parts of calcium oxalate raphide containing household plants (elephant's ear (Caladium sp.), dumb cane (**Dieffenbachia sp**.), philodendron, peace lily) in dogs and cats. Even a small dose of calcium oxalate is enough to cause intense sensations of burning in the mouth and throat.[4] The stalks of plants in the

Dieffenbachia genus produce the most severe oxalate reactions. The needle-like oxalate crystals produce pain and swelling when they contact the lips, tongue, and oral mucosa.[4]
The tongue should always be palpated, as increased rigidity can be a hint for a primary muscular problem (with hindered clearance of saliva) and a creatine kinase level should be measured. Following the oral cavity, a maxillofacial exam should be performed and checked for asymmetry, swellings, and cranial nerve functions. If the animal cannot close its mouth, the temporomandibular joints should be radiographed in order to rule out a luxation or fracture. Neurologic differentials for a hanging jaw or decreased jaw tone include trigeminal nerve dysfunction (usually trigeminal neuritis, rarely nerve sheath tumor) and less likely botulism.[5]
The salivary glands should be examined for size, firmness and possibly painfulness. Enlarged salivary glands can be a physiological hypertrophy in response to chronic stimulation or it can be secondary to autonomic neuropathies. In dogs a rare disorder called **sialoadenosis or hypersialosis** exists that may be completely Phenobarbital-responsive. It is hypothesized that this clinical entity may in fact be an unusual form of limbic epilepsy.[6] However before starting a hypersalivating dog with enlarged salivary glands that may present with additional retching or vomiting on Phenobarbital, care should be taken to closely examine the esophagus of these patients, as a **primary esophageal problem** (esophagitis, esophageal foreign body or diverticuli, spirocerca lupi infestation, megaesophagus) is often the cause.[7] When the associated disease is successfully treated, the salivary glands return to normal and all clinical signs resolve.[7] It is hypothesized that an afferent vagal reflex may be involved, and that the mechanism of disease is similar to the neural pathogenesis suggested for hypertrophic osteopathy; in this instance, the efferent targets are the salivary glands rather than the limbs.
Even though neoplastic salivary gland disease (most often adenocarcinoma) is another differential for enlarged salivary glands, generally this clinical entity does not cause ptyalism.
Once oral and maxillofacial causes have been ruled out, further diagnostics are indicated. Chest radiographs are useful to rule out esophageal foreign bodies or demonstrate a dilated gas or fluid-filled esophagus. Esophagitis, esophageal foreign bodies and strictures, as well as motility disorders can all lead to hypersalivation through activation of the esophagosalivary reflex. This is a vagally mediated neural reflex demonstrated by perfusing acid into the esophagus, which stimulates salivation. Another important disease in cats with ptyalism that require chest radiographs is **pyothorax**. Hypersalivation was a common physical examination finding in nonsurviving cats in one study describing 80 cats with pyothorax.[8] The mechanism of this remains unclear. The authors of that study discussed hypersalivation as a marker of disease severity, possibly reflecting increased pain, nausea, or secondary hepatic dysfunction. Another thought was, that the large volume of pleural effusion may make swallowing difficult for cats with pyothorax.
If the cause of the ptyalism has not been identified at his point, diagnostic testing with a minimum data base including a CBC, serum biochemistry, and urinalysis is needed. Special attention should be paid to liver enzymes and indirect liver function tests (albumin, glucose, cholesterol), as **hepatobiliary disease** can present with ptyalism in dogs and cats. This is especially the case when the patients suffer from **hepatic encephalopathy** (HE). Hepatic lipidosis (hyperbilirubinemia, high alkaline phosphatase and transaminases, mild γGT elevation) and portosystemic shunts (normal bilirubin, minimally elevated transaminases) are the two liver disorders most commonly associated with hypersalivation in cats. A liver function test (postprandial serum bile acids or plasma ammonium concentrations) and hepatic imaging should be performed. It is important to note that running serum bile acids is of no use in any icteric patient, as they will be high in cholestatic disease. Dogs with portosystemic shunts may also preset with ptyalism, although this more commonly seen in cats at the author's institution. Acute hepatic necrosis ("acute liver failure") and extrahepatic cholestatic disease is more often associated with ptyalism in dogs.
The finding of ptyalism in a very sick dog or cat, or a patient with hypotension should also always evoke suspicion of **sepsis**. During sepsis the sensitivity of salivary glands to autonomic stimulation is increased.
Abdominal pain due to visceral stretch is another reason for ptyalism in dogs and cats seen by the author. Intestinal obstructions due to a foreign body, intussusception, acutely obstructing ureteroliths or choleliths need to be ruled through abdominal ultrasonography.
If the animal shows additional changes in behaviour, **rabies** may also be considered.

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