ABSTRACT: This paper will cover the disease conditions of the reptilian gastrointestinal tract, excluding the pancreas and liver. It is not a comprehensive review. More in-depth disease descriptions can be found in the articles, website, and reference books listed in the literature cited Benson, 1999; de la Navarre, 2003; Jacobson, 2007; Greiner and Mader, 2006; Mehler and Bennett, 2003; Mitchell and Diaz-Figueroa, 2005; Wellehan and Johnson, 2005; Wilson and Carpenter, 1996).

KEY WORDS: lizards, snakes, crocodylia, chelonia, gastrointestinal disease, oral lesions, virus, bacteria, fungus, neoplasm

INTRODUCTION

Common insults to the gastrointestinal system include developmental anomalies, degenerative lesions, obstructions, traumatic injuries, infectious disease agents, and neoplasms. The paper is divided by disease categories and location. A short discussion of these disease conditions in reptiles is found in each section. For the purposes of this paper, chelonia will refer to all of the shelled reptiles. The term “tortoise” will be used to describe the large land animals that have stout elephantine feet. Few references to marine turtles and crocodilian species are included, as these are uncommonly seen in most clinical practices.

DEVELOPMENTAL ANOMALY

A number of congenital disorders of the digestive tract are recognized in reptiles even from the early Cretaceous age (dicephaly) (Buffetaut, 2007). Ophidian dicephaly (craniodichotomy) is presumably the result in incomplete separation of identical twins and is presumed to be the cause in other reptiles (Andreadis and Burghardt, 1993). Rarely this anomaly (dicephaly) has even been used in experimental studies on causes of satiety (Andreadis and Burghardt, 1993). In the oral cavity, developmental defects include mandibular brachygnathism, maxillary brachygnathism, and cleft palate (Mader, 2006). The proposed causes reported are genetic defects, anoxia of the embryo, and rapid thermal changes during internal incubation (Andreadis and Burghardt, 1993; Mader, 2006).
DEGENERATIVE

An atrophic gastritis has been described in a Hermann's tortoise (*Testudo hermanni*) and two red-eared slider turtles (*Trachemys scripta elegans*) (Zwart and vander Gaag, 1981). The prominent feature in all three cases was of atrophy of the gastric glands. In addition all animals had extensive renal lesions with mineralization noted in the gastric muscular tunics and arteries of the submucosa. The mineralization is suspected to have led to tissue edema, inflammation, and the gastric mucosal atrophy.

A physiologic change that may fall under degenerative processes is of the atrophy of the intestinal mucosa and pancreas observed in snakes and chelonians, particularly those that have long periods of time between feeding (Cox and Secor, 2008; Zhalka and Bdolah, 1987). There is reduction in the mass of the intestinal mucosa and depletion of the zymogen granules of the exocrine pancreas (Zhalka and Bdolah, 1987; Cowan, 1968). In studies of boas, pythons, and gila monsters (*Heloderma suspectum*) re-feeding resulted in hypertrophy of the pancreatic acinar and ductal cells as well as lengthening of the intestinal villi (Cox and Secor, 2008; Christel et al., 2007).

OBSTRUCTIVE LESIONS

Lizard

The most common cause of gastrointestinal obstructions is from the ingestion of foreign material (Benson, 1999; Mitchell and Diz-Figueroa, 2005). Generally the foreign material is of inappropriate environmental substrates. A common green iguana (*Iguana iguana*) presented with vomiting after feeding and with subtle signs of abdominal pain. The animal was evaluated radiographically and by ultrasound before an exploratory coeliotomy was performed and a large piece of absorbent cotton was removed from the ileum and cecum. The cotton was part of bandaging material off a companion iguana (Büker et al., 2010).

Inadequate environment in terms of humidity or temperature as well as inappropriate diet can result in fecal obstipation. A savannah monitor (*Varanus exanthematicus*) presented with a 2-month history of lethargy, anorexia, and lack of stool production. Medical therapy was attempted without success. Celiotomy and colonic enterotomy allowed for complete removal of the impacted fecal masses (Franch and Martorell, 2001).

A parasitic impaction was found in the large intestine of a Fiji island iguana (*Brachylophus vitiensis*) (Kane et al., 1976). At death, a mass of oxyurid parasites identified as *Alaeuris brachylophi* filled the large intestine.

Intestinal intussusception is reported in iguanas (McGuire et al., 1999; Saik et al., 1987), chameleons (*Chamaleo* spp.; Barrie et al., 1993) and in a juvenile blue tongued skink (*Tiliqua scincoides*; Benson, 1999).
Snake

A variety of foreign objects have resulted in obstructive lesions in the digestive tract of snakes (Benson, 1999; Mitchell and Diaz-Figueroa, 2005). A black rat snake (Elaphe obsoleta obsolete) ingested a golf ball which lodged within the stomach (Adams and Sleeman, 2005). It was successfully removed surgically and the snake returned to the wild.

An unusual case of parasitic impaction was reported in an adult male milk snake (Lampropeltis triangulum) (Klaphake et al., 2005). The snake was anorexic for two months and had regurgitated a partially digested mouse. Several fluid-filled coelomic masses were identified by radiographs and ultrasound. A gastric impaction was due to a firm solid white mass containing the eggs of the nematode Kalicepsalus sp and the coelomic masses were granulomatous inflammatory foci secondary to the nematode eggs.

Extra intestinal masses can result in gastrointestinal obstruction. A cystic testicular anomaly in an emerald tree boa (Corallus caninus) resulted in the development of a subsequent stenosis of the small intestine (Kostka et al., 1998).

Chelonia

Intestinal obstruction by foreign bodies is common in sea turtles (Di Bello et al., 2006). Early diagnosis can be complicated by the extended fasting periods and lack of significant clinical signs. In surveys of marine sea turtles, foreign material resulting in gastrointestinal obstructions included nylon fishing line, plastic bags, and fishing hooks (Di Bello et al., 2006; Flint et al., 2009).

A red-eared slider (Trachemys scripta elegans) with a history of ingesting aquarium stones, presented with anorexia, no defecation, and difficulty swimming. Radiographically there was intestinal dilation with numerous radiopaque foreign bodies. Over 100 stones were removed via enterotomy and intestinal perforations were debried and sutured closed (Rahal et al., 1998). In another red-eared slider, sloughed shell material obstructed the small intestine (Gould et al., 1992). The material was surgically removed; however, a month later the turtle re-presented with anorexia and gravel-like foreign material identified radiographically. This material passed after use of an orally administered commercial lubricant.

An obstruction was associated with a 360 degree volvulus of the colon in a hawksbill turtle (Eretmochelys imbricata). Death was most likely the result of focal necrosis and clostridial toxemia occurring secondary to the colonic volvulus (Schumacher et al., 1996).

A review of post-mortem examinations on three free-living green turtles (Chelonia mydas) in the United Arab Emirates identified a duodenal volvulus and stomachs full of fresh seagrass (Hasbun et al., 1998). Based on a lack of other findings, the duodenal volvulus was suspected to be dietary in origin from over-fermentation of ingesta (Hasbun et al., 1998).
An unusual obstructive lesion occurred in a previously stranded green turtle (*Chelonia mydas*). After a 2-mo history of anorexia, intermittent regurgitation, decreased fecal production, and positive buoyancy of the right side, radiographs confirmed gaseous distension of bowel loops suggestive of intestinal obstruction. Surgically a 540 degree volvulus of the small intestine was identified and derotated. An intestinal stricture was present at the site of the volvulus, which was resected. A leiomyoma was identified at the focal stricture (Helmick et al., 2000).

Strictures developing from surgical intervention can result in an obstructive enteritis. An adult female Mediterranean spur-thigh tortoise (*Testudo graeca*) presented with a devitalised prolapse of the colon. Resection of the prolapse was performed; however, clinical signs of an obstruction occurred within 16 days. A subsequent coeliotomy identified a stricture at the site of the previous anastomosis (Lloyd, 2003).

*Crocodilia*

Gastric impaction due to food material has been described in captive crocodiles (*Crocodilus niloticus*) (Rogers and Windsor, 1982). Five crocodiles fed only guinea pigs for 4 days died and in the stomach was a compacted mass of matted guinea pig hair.

**ORAL LESIONS**

The oral cavity is probably the most readily examined portion of the digestive tract. Trauma to the oral cavity is a common presentation in captive reptiles. Rostral trauma will occur as the animal rubs the face against cage walls and covers. These lacerations or ulcerations can be portals for secondary bacteria and fungal infections. Thermal burns may occur when overheated prey items are fed. Damage to the beak, teeth, gingiva, or tongue is not uncommon with aggressive encounters with cage-mates, live prey, or examination procedures (Mehler and Bennett, 2003). The oral cavity can be the portal of entry for other diseases involving the digestive tract.

*Lizard*

Confirmed viral stomatitis is uncommon in lizards. A proliferative stomatitis was recognized in green tree monitor lizards (*Varanus prasinus*) from two different collections. DNA in situ hybridization of tissues from three lizards was positive for herpesvirus in the oral mucosa. This virus was designated Varanid herpesvirus 1 (Wellehan et al., 2005).

Plated lizards (*Gerrhosaurus* sp) part of a private collection, developed glossal and labial stomatitis associated with three different herpesviruses, Gerrhosaurid herpesviruses 1, 2, and 3. All three viruses were classified as alphaherpesviruses (Wellehan et al., 2004).

Several cases of a bacterial chelitis in Uromastyx and bearded dragons (*Pogona vitticeps*) have been reported (Mehler and Bennett, 2003; Koplos et al., 2000). The lizards develop a yellowish-brown crust along the margins of the oral cavity with variable degrees of inflammation. A Gram-
positive filamentous or pleomorphic bacilli was identified proliferating in the hyperkeratotic crusts and these were identified as *Devriesea agamarum*, a novel bacterium, in a later study (Hellebuyck et al., 2009). This bacterium was demonstrated to be a facultative pathogen, able to cause a dermatitis when the integrity of the skin is breached. For many lizards the crusts did not appear to result in significant clinical signs; however, a chronic dermatitis leading to a septicemia is possible.

Dental plaque, gingivitis, and periodontitis are significant lesions in captive lizards particularly agamids, chameleons, some species of skinks, and Fijian iguanas (*Brachylophus vitiensis*) (Mehler and Bennett, 2003). As with other species, excessive plaque build-up due to dietary items leads to an anaerobic environment and proliferation of anaerobic and fungal flora. Inflammation of the gingival can progress and involve the periodontal bone and tooth loss (Mehler and Bennett, 2003). Pathologic fractures through areas of osteomyelitis and systemic infections can result in death.

Fungal infections involving the oral cavity are rare. A case of fungal periodontal osteomyelitis in a chameleon (*Furcifer pardalis*) involved the mandible (Heatley et al., 2001). The morphology of the fungal hyphae (branching and septate) suggested an *Aspergillus* spp. Aggressive surgical and medical therapy resulted in resolution of the infection. *Chrysosporium anamorph of Nannizziopsis vriesii* (CANV) has been described in bearded dragons and other reptilian species as a keratinophilic fungus that results in a fatal granulomatous dermatitis. A fungal gingivitis in a bearded dragon associated with this fungal organism (Bowman et al., 2007).

*Cryptosporidium* spp. infection has been associated with aural-pharyngeal polyps in three iguanas (*Iguana iguana*) (Uhl et al., 2001). All iguanas presented for masses protruding from the ear canal. The most likely route of infection was ingestion; however, definitive oocysts were not observed in other locations in these iguanas. Histologically, the masses were composed of fibrous tissue rimmed by oral mucosa containing widely separated ducts, glands, and cysts. The cysts were lined by hyperplastic epithelium and contained numerous round 3–8-μm protozoal organisms, consistent with *Cryptosporidium* sp., at the apexes of the epithelial cells (Uhl et al., 2001).

**Snake**

Rare viral infections have involved the oral cavity in snakes. In a palm viper (*Bothriechis marchi*) viral inclusions of adenovirus were present in the mucosal epithelium of the oral cavity and esophagus. These were associated with multifocal subacute esophagitis and stomatitis (Raymond et al., 2002). This snake was concurrently infected with inclusion body disease of boid snakes (IBD).

Numerous cases of an ulcerative stomatitis are described in snakes (Kiel, 1977; Hess and Rudy, 1974; Parker and Migaki, 1981; Stull and Anderson, 1976). Generally there is a history of trauma or inappropriate husbandry/diet and damage to the mucosa that permits secondary bacterial invaders. Malnutrition and deficiency of ascorbic acid are known to lower resistance to
infections (Parker and Migaki, 1981). The most common isolates associated with a stomatitis are predominantly Gram-negative bacteria including *Pseudomonas aeruginosa*, *P. maltophilia*, *Providencia rettgeri*, *Aeromonas hydrophila*, and *Klebsiella* spp. (Mehler and Bennett, 2003; Draper et al., 1981). The initial presentation is of a frothy, fibrinous exudate around the lips with hyperemia and petechia of the oral mucosa. Refusal to eat contributes to generalized debilitation. If left untreated, the lesions progress to a caseous necrosis and even osteomyelitis. An acute septicemia can result in death. Rare cases of mycobacterial granulomas and granulomatous stomatitis are described (Kiel, 1977; Quesenberry et al., 1986).

**Chelonia**

From a review of the lesions in marine turtles the most common lesions in the oral cavity and esophagus were mainly associated with the ingestion of fishing hooks (Oros et al., 2004). The lesions were of ulcerative and necropurulent stomatitis and esophagitis.

Herpesvirus-associated rhinitis, stomatitis, glossitis, tracheitis and bronchopneumonia has been described in a number of tortoises (California desert tortoise, spur-thigh tortoises, green sea turtles, Argentine tortoises [*Geochelone chilensis*], Mediterranean land tortoises, Hermann’s and four-toed tortoises, Greek tortoises [*Testudo graeca*]) (Jacobson et al., 1985; Cooper et al., 1988). This herpes virus infection is frequently complicated by secondary bacterial infections and generally results in severe debilitation of the tortoise. The clinical signs include nasal and ocular discharge, regurgitation, anorexia, and lethargy (Jacobson et al., 1985). Necrotizing lesions of the oral cavity can be extensive including the tongue. The transmission is most likely from infected carriers although the pathogenesis of this viral infection has not been fully described. Treatment of clinical cases with acyclovir 5 per cent ointment appeared encouraging, with a marked improvement in some instances (Cooper et al., 1988).

Necrotizing stomatitis, esophagitis, and/or pharyngitis are characteristic lesions in many naturally occurring chelonian Ranavirus infections (Johnson et al., 2007). The clinical signs from a Ranavirus infection such as ocular and nasal discharge, conjunctivitis, and palpebral edema are similar to those signs caused by infections of *Mycoplasma agassizii* or tortoise herpesviruses. An important complication in diagnosing Ranaviral infections is that inclusion bodies may be an inconsistent finding and should not be relied upon making a differential diagnosis (Johnson et al., 2007). Transmission electron microscopy (TEM) remains a useful diagnostic tool in chelonian Ranavirus infections even in the absence of visible inclusions on histologic section.

**Crocodylia**

Although the lesions of meningoencephalomyelitis and myocardial necrosis are best recognized in West Nile viral infections of American alligators (*Alligator mississippiensis*), other lesions include heterophilic necrotizing stomatitis, glossitis, and pancreatic necrosis (Jacobson et al., 2005). On gross examination of hatchling alligators a necrotizing pharyngitis characterized by fibrinous material and exudates was a frequent finding.
**ESOPHAGUS, STOMACH, INTESTINES**

There are several excellent reviews on the viral infections of reptiles. These reviews provide more details on the various types of viruses and the extent of the lesions (Jacobson, 2007; Wellehan and Johnson, 2005). Endoparasites from protozoa to metazoans have also been extensively reviewed (de la Navarre, 2003; Jacobson, 2007; Greiner and Mader, 2006; Wilson and Carpenter, 1996).

Adenovirus infections are documented in at least twelve different species of reptiles. In contrast to their mammalian and avian counterparts reptilian adenoviruses are not well characterized as to their pathogenic potential and their ability to cause primary disease.

Adenoviral infections of bearded dragons are common and this viral infection has become established in certain U. S. lizard breeding groups of the genus Pogona (bearded dragons) (Kim et al., 2002; Jacobson et al., 1996; Moormann et al., 2009). The common clinical signs in young dragons are a progressive weakness, anorexia, circling, and head tilt (Kim et al., 2002). The lesions are of severe acute coagulative hepatocellular necrosis and the large intranuclear basophilic inclusion bodies in the hepatocytes and enterocytes. It is speculated that there is possible vertical transmission through the egg in utero or at time of oviposition. The adenovirus infections are also reported to be associated with a second virus, Dependovirus (Jacobson et al., 1996). This virus is defective and appears to need the adenovirus to replicate. It is not associated with a disease condition.

Adenovirus was associated with proliferation of tracheal and esophageal mucosa in a 6 month old Jackson’s chameleon (*Chamaeleo jacksoni*). The clinical signs included opisthotonus and anorexia. Histologically, eosinophilic intranuclear inclusion bodies found within ciliated epithelial cells of the esophageal and tracheal mucosa. The inclusions were morphologically consistent with adenovirus by electron microscopy (Jacobson and Gardiner, 1990).

Adenovirus has also been isolated from the intestine of other lizards. An adult male wild caught mountain chameleon (*Chameleo montium*) died after a 28-day history of anorexia. Gross examination revealed marked emaciation and enteric nematodiasis. Histopathologic examination of the small intestine revealed moderate numbers of enterocytes with basophilic, intranuclear inclusions. These were confirmed to be an adenovirus on ultrastructural evaluation. No pathologic changes were associated with the adenoviral infection (Kinsel et al., 1997).

A group of seven juvenile California mountain kingsnakes (*Lampropeltis zonata multicincta*) developed severe acute gastroenteritis. The clinical signs included emesis, dehydration, and sudden death. Histopathology demonstrated segmental acute mucosal necrosis and hyperplasia. The enterocytes had intranuclear inclusions consistent with adenovirus (Wozniak et al., 2000).

Adenovirus infections in a common boa (*Boa constrictor imperator*) and a Mojave rattlesnake (*Crotalus scutulatus scutulatus*) were diagnosed using in situ hybridization (ISH). Adenoviral DNA was observed in the nuclei of hepatocytes, Kupffer cells, endothelial cells, and enterocytes.
Both affected snakes had other concurrent diseases, suggesting that the adenovirus may not have been the primary pathogen (Perkins et al., 2001).

Herpesvirus infections are uncommon in most reptiles with the majority of reports in chelonians (see oral lesions) (Hughes-Hanks et al., 2010). The cases involving the intestinal tract of lizards are limited to monitor lizards (Varanus spp.) (Hughes-Hanks et al., 2010). Two monitor lizards had multifocal necrosis in the lamina propria of the small intestine and in the liver. Many of the degenerate cells contained large, eosinophilic intranuclear inclusions. The enveloped icosahedral virions consistent with herpesvirus were detected by electron microscopy (Hughes-Hanks et al., 2010).

Reovirus, an unenveloped RNA virus, produces relatively nonpathogenic infections in animals. A group of six juvenile, captive-bred, Arizona mountain king snakes (Lampropeltis pyromelana) presented underweight and with regurgitation for several weeks. There was significant atrophy of coelomic cavity fat pads. Histologically, the significant lesions were acute necrotizing colitis and hepatitis with syncytial cell formation. By electron microscopy numerous viral particles were found in the cytoplasm of the intestinal epithelium and in hepatocytes. The morphology suggested a reovirus. The remaining two sick animals in the group responded to supportive care including tube-feeding, intra-coelomic fluids, and ceftazidime injections (Reavill et al., 2003).

Inclusion body disease (IBD) of boid snakes can be histologically recognized in lining mucosal epithelial and lymphoid cells aggregates of the esophagus, as well as within the mucosal cells of the stomach and less frequently in the intestines (Garner and Raymond, 2004). The eosinophilic intracytoplasmic inclusions have been described in boas and pythons (family Boidae), palm vipers (Bothriechis marchi) and corn snakes (Elaphe guttata) (Vanraeynest et al., 2006; Fleming et al., 2003; Raymond et al., 2001). It is important to note that these inclusions do not always confirm IBD, as the inclusions in the corn snakes did not have consistent morphology when examined by electron microscopic (Fleming, et al., 2003). This disease can be responsible for clinical signs including chronic regurgitation, incoordination, loss of righting reflexes, paresis, and an increased incidence of secondary infections such as stomatitis and pneumonia. Clinical signs referable to the central nervous system are more prominent in the Pythoninae subfamily (Schumacher et al., 1994; Carlisle-Nowak et al., 1998). Antemortem diagnostics include CBC’s and biopsies of the esophagus (especially of lymphocytic stromal aggregates or “tonsils”), gastric mucosa, and liver (Garner and Raymond, 2004). The etiologic agent of IBD is suspected to be a retrovirus (Jacobson et al., 2001; Huder et al., 2002; Wozniak et al., 2000). At this time the mode of transmission is unknown; however, consider fecal/oral contamination, airborne via respiratory discharges, or by the snake mite as a vector (Schumacher et al., 1994).

From a sea turtle study of diseases, the lesions involving the intestines were of catarrhal, fibrinous, necropurulent and necrotising enteritis (Oros et al., 2004). A wide range of Gram-negative and Gram-positive bacteria, including Bacillus species, Escherichia coli, Pasteurella species, Proteus species, Staphylococcus species, Streptococcus species and Vibrio alginolyticus were isolated from the lesions. All the cases of necrotising enteritis were associated with intestinal intussusception caused by the ingestion of monofilament fishing lines.
Salmonella enteritis has importance from its zoonotic potential. There are many excellent written reviews (Johnson-Delaney, 2006; Mitchell, 2006) and one available from the website of the Association of Reptile and Amphibian Veterinarians: (http://www.arav.org/ECOMARAV//timssnet/journals/Salmonella.cfm).

A fungal colitis was associated with an intussusception in a chameleon (Shalev et al., 1977). The lizard presented with a rectal prolapse that was surgically replaced. The fungal organisms had phycomycotic hyphae.

Cryptosporidiosis is a well-known gastrointestinal disease of snakes and lizards. Hypertrophic or proliferative gastritis is a common manifestation of Cryptosporidium infections in snakes, and atropic gastritis has been described in lacertas (Uhl et al., 2001). Polymerase chain reaction (PCR) has detected Cryptosporidium varanii (saurophilum) in corn snakes (Pantherophis guttatus) and in leopard geckos (Eublepharis macularius). Cryptosporidium serpentis was found in other leopard geckos. A Cryptosporidium sp. “lizard genotype” was reported in one leopard gecko and one corn snake (Richter et al., 2011). Pseudoparasitic cryptosporidian species from those ingested with the prey (pseudoparasites) have been described in snakes (Richter et al., 2011).

Cryptosporidiosis was found in other leopard geckos. A Cryptosporidium sp. “lizard genotype” was reported in one leopard gecko and one corn snake (Richter et al., 2011). Pseudoparasitic cryptosporidian species from those ingested with the prey (pseudoparasites) have been described in snakes (Richter et al., 2011).

Two Russian tortoises (Agrionemys [Testudo] horsfieldii) and a pancake tortoise (Malacochersus tornieri), all from separate collections had histologic evidence of intestinal cryptosporidiosis and one with gastric cryptosporidiosis. A mild inflammation was associated with the infections; however, only one case of the intestinal case had mucosal hyperplasia. Consensus Cryptosporidium sp. PCR and sequencing was used to characterize the Cryptosporidium sp. present in these three tortoises. This may be an under-recognized problem in tortoises (Griffin et al., 2010).

Reptilian amebiasis is caused by Entamoeba species. E invadens protozoan is described as resulting in high morbidity and mortality among some snakes, lizards, and tortoises (Chia et al., 2009; Donaldson et al., 1975; Jacobson et al., 1983). It is common to see secondary bacterial infections associated with these organisms. The infection begins in the intestinal tract producing colonic ulcers. As more of the gastrointestinal tract is involved finally there will be hematogenous spread to the liver via the portal vein. The lumen of the intestinal tract will usually be filled with fibrinonecrotic plaques. An unusual case of amoebiasis resulting from a combination of direct invasion via skin wounds and hematogenous spread was reported in common water monitor lizard (Varanus salvator) (Chia et al., 2009). Entamoeba can be transmitted by other reptiles who are inapparent carriers and unaffected by the organism. Transmission is by ingestion of the protozoal cysts. Mixing reptile species should be discouraged to prevent the transmission of such organisms that may be deadly in other species (Cowan, 1968). Examination for cysts or trophozoites (positive Lugol’s iodine) in fecal material is suggested for all contact animals.

Other Entamoeba species have also been associated with enteric disease (Richter et al., 2008). In a possible immunocompromised boa constrictor with inclusions of inclusion body disease (IBD),
a species closely matching *E. ranarum* by gene sequencing was associated with a diphtheroid colitis (Richter et al., 2008).

The coccidia *Isopora amphiboluri* is most common in Bearded Dragons and can be associated with clinical signs (Kim et al., 2002). It has a direct life cycle and can predispose to other more serious diseases. An adverse synergistic effect of the coccidiosis with the adenoviral infection may be responsible for increased morbidity and mortality (Kim et al., 2002).

Adult spirorchiid flukes (blood flukes) inhabit the cardiovascular system (heart and blood vessels) of many freshwater (Goodchild and Dennis, 1967; Johnson et al., 1998) and marine turtles (Gordon et al., 1998). These are analogous to the schistosomes of birds and mammals. Eggs deposited by the adults are carried through the circulatory system and can block small blood vessels in many organs producing microgranulomas. As the eggs migrate to the lumen of the intestines, this can lead to necrosis and bacteremia. In freshwater turtles, the intermediate host is a snail. The intermediate host for marine turtle spirorchiid flukes is as yet unknown. Treatment with praziquantel in freshwater turtles has been reported (Johnson et al., 1998).

In two monitor lizards (*Varanus* spp.) multiple, nonsegmented, acoelomate trematodes with a thin cuticle and anterior suckers were present in the intestinal lumen (Hughes-Hanks et al., 2010). The clinical significance of the infection is not well understood.

Oxyurids are common parasites in lizards, chelonians and some snakes. In snakes, care must be taken not to mistake rodent pinworm eggs for those parasitic for reptiles. These parasites are usually not associated with any pathology and treatment is usually not indicated. Oxyurids have a direct life cycle and in captivity, large numbers can accumulate within the colon putting some reptiles at possible risk for impaction (de la Navarre, 2003). In one case of an iguana, large numbers of oxyurids (*Alaeuris brachylophis*) in the colon were associated with small intestinal intussusception (McGuire et al., 1999). Severe infestations associated with poor diet and deficiencies of husbandry can result in debilitation and death in iguanas (Loukopoulos et al., 2007).

Helminths are common findings in surveys of chelonians (Keymer, 1978). In most cases the nematodes are non-pathogenic. A viviparous pinworm-like nematode of the genus *Protrachadis* (Family Atractidae) was associated with a lymphoplasmacytic enterocolitis in red-footed tortoises (*Geochelone carbonaria*) and leopard tortoises (*Geochelone pardalis*) (Rideout et al., 1987). Clinical signs were either nonspecific, consisting of anorexia, lethargy, and depression, or were absent.

Penned green turtles (*Chelonia mydas*) became infected with a larval nematode (*Anisakis* sp. Type I) most likely from the food source, sardine (*Harengula ovalis*) (Burke and Rodgers, 1982). The larvae were associated with hemorrhagic ulcers in the pyloroduodenal junction of the alimentary tract.
**TUMOR**

The majority of the adenocarcinoma/carcinoma gastrointestinal tract tumors are reported in snakes and rarely in lizards and chelonians (Jessup, 1980; Hernandez-Divers and Garner, 2003; Jacobson and Ackerman, 1981; Latimer and Rich, 1998; Schlumberger and Lucke, 1948; Martein et al., 1994; Oros et al., 2004). These tumors will result in variable clinical signs of the digestive tract including constipation, regurgitation, and progressive enlargement of the coelom. In snakes the differential should include gastric cryptosporidia infections.

Metastases occur late in reptilian species. There is one report of a metastatic intestinal adenocarcinoma to the liver in an emerald tree boa (*Corallus canius*) (Oros et al., 2004) and the authors have identified metastases from gastric carcinomas into the intestine and liver of a carpet python (*Morelia spilota*) and in the liver of two bearded dragons (*Pogona vitticeps*).

Therapy has rarely been attempted as many cases present in a terminal condition. A transmural mucinous colonic adenocarcinoma with scirrhous reaction in a corn snake (*Elaphe guttata guttata*) was surgically resected. The snake presented with a history of constipation and a palpable mass in the distal coelomic cavity. The mass was nodular firm and white encircling the distal colon proximal to the cloaca (Latimer and Rich, 1998). The surgery alleviated the intestinal obstruction for at least four months, after which the snake was lost to follow-up (Latimer and Rich, 1998). A Burmese python (*Python molurus bivittatus*) was euthanatized because of complications following resection of a segmental colonic adenocarcinoma (Chandra et al., 2001). A European viper with a cloaca and oviduct adenocarcinoma died after photodynamic therapy with chloro-aluminum sulfonated phthalocyanine (Roberts et al., 1991).

Squamous cell carcinomas of the oral cavity (frequently involving the lips) are more commonly reported in snakes (Hill, 1977; Roberts et al., 1991). The tumors have poorly defined borders and are associated with hemorrhage and necrosis of the surrounding tissues. These lesions lead to a chronic stomatitis and may contain caseous material. Common clinical signs for tumors in these locations include stomatitis, anorexia, and oral discharge.

There are several reports of a highly malignant gastric neuroendocrine carcinomas in young bearded dragons (*Pogona vitticeps*) (Ritter et al., 2009; Lyons et al., 2010). These tumors readily metastasize to the liver. The common clinical signs are anorexia, vomiting, hyperglycemia, and anemia.

Malignant lymphoma (lymphosarcoma) represents the majority of the documented cases in reptiles. These neoplasms can involve many organs. In many reptiles it may initially present as cutaneous or oral tissue masses that are found to be metastatic disease. Grossly, they are gray-white or cream masses that are soft to relatively firm. Necrosis and hemorrhage may be present (Hernandez-Divers and Garner, 2003; Effron et al., 1977; Oros et al., 2001).
REFERENCES


