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OCULAR, NASO-MAXILLARY, AND NEURAL ANOMALIES IN RACCOONS, *PROCYON LOTOR* (L.)[1]

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ABSTRACT: Congenital ocular and related anomalies were studied in two unrelated young raccoons. One animal was anophthalmic and had severe anomalies of the central nervous system, consisting of meningoencephalocele, pachygyria, hydranencephaly, cerebellar cavitation, syringomyelia, and other defects. A second animal was microphthalmic with congenital defects of the nose, maxilla and teeth. Ocular lesions were severe and included chorioretinal coloboma, retinal folds, disorganized neuroectodermal cell layers, spherophakia, cataract and other defects. The nose had unilateral abnormal epithelium, hair follicles, sweat glands and sebaceous glands, and a lack of parietal cartilage on the affected side.

INTRODUCTION

There are few reports of congenital anomalies in raccoons, despite the fact that these mammals are common in North America. Heidt (1969) described a case of congenital amelia of the forelimbs and lack of a tail in a young female raccoon. Vellard and Penteado (1931) described a raccoon with two tails. Other developmental anomalies reported for this species include congenital diaphragmatic hernia (Sanderson, 1960), renal hypoplasia (Mech and Anderson, 1966), dental anomalies (Knable and Werner, 1964), and skeletal anomalies (Michael, 1968). Albinism, erythrism, and pelage color abnormalities have been the most commonly reported anomalies for the species (Whitney and Underwood, 1952; Neill, 1953; Allen and Neill, 1956; Funderburg, 1961; Michael, 1968; Johnson, 1970).

In this report we describe ocular anomalies in two young raccoons. One animal also had abnormalities of the naso-maxillary area, and another had extensive neural defects.

MATERIALS AND METHODS

Raccoon no. 1 was a 3- to 4-mo-old male presented for evaluation of masses located on the forehead and persistent ankyloblepharon. He had been in captivity for 1 mo and the owners knew nothing about his littermates. The clinical diagnoses were encephalocele and bilateral microphthalmia/anophthalmia. The owners chose euthanasia and the carcass was submitted for necropsy.

The head was removed, skinned and fixed in neutral buffered 10% formalin. The mandible and associated structures were removed and the remaining portion of the head was decalcified. The head with the brain intact was divided in two with a transverse cut just rostral to the medial canthi of the palpebral fissures. The caudal portion was cut transversely into nine approximately equal slabs. Six-μm sections of each of these slabs were prepared in a routine manner, stained with hematoxylin and eosin, and examined microscopically.

Raccoon no. 2 was a 2½- to 3-mo-old female which was live-trapped with her mother in a barn in Thorntown, Indiana. The young raccoon had bilateral anomalies of the eyes and unilateral anomalies of the planum nasale, maxilla and upper teeth, but was in otherwise good health and was of normal size and weight for its age. The animal was killed and the globes were removed and fixed in Zenker's fixative. The planum nasale and adjacent tissue were fixed in neutral buffered 10% formalin. Six-μm sections of the globes were examined microscopically, but the nervous system was not studied.

RESULTS

The gross alterations of raccoon no. 1 were clinically diagnosed as meningoencephalocele and bilateral microphthalmia/anophthalmia. Two elongated dome-shaped, soft, subcutaneous masses, each approximately ½ × 1 × 2 cm, were present on both sides of the dorsomedial frontal region of the head. The right mass was slightly rostral to the left mass. Plain skull radiographs revealed a 2 cm long, soft tissue protrusion overlying an open fontanel between the frontal bones of the skull. The maxilla was slightly asymmetrical with the right dorsal aspect slightly elevated in comparison to the left side. The fontanel between the frontal bones was open and shaped like an asymmetrical heart with a rounded, caudally pointing apex. The

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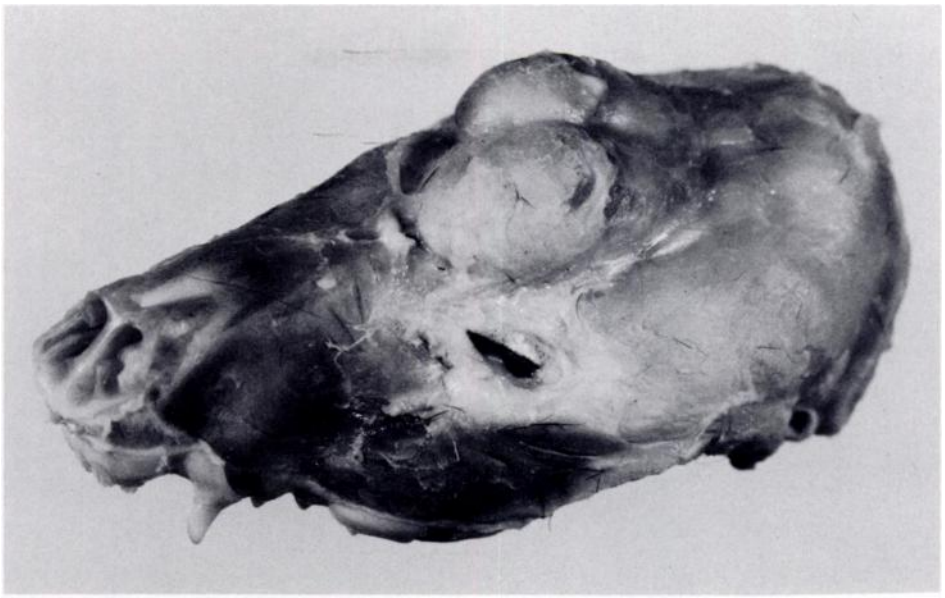

FIGURE 1. Head of raccoon no. 1 with the skin and lower jaw removed. Notice the dorsal bilobed soft tissue protrusion that contains brain tissue and ankyloblepharon.

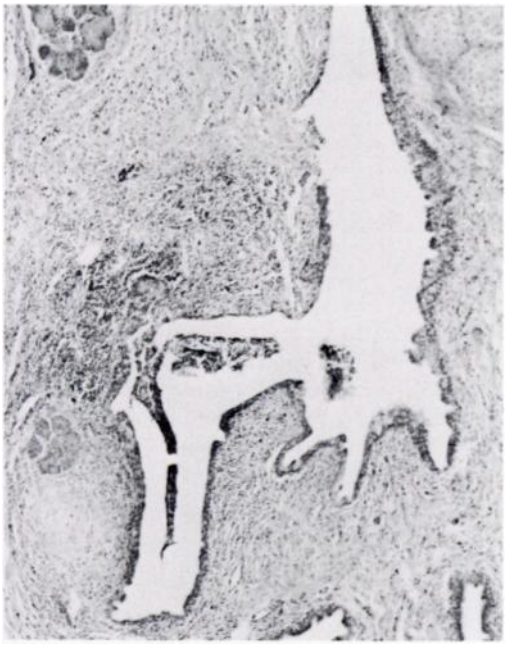

FIGURE 2. Photomicrograph of adnexal ocular structures and palpebral fissure of raccoon no. 1. Notice that no components of the globe are present. H&E stain, ×56.

adjacent frontal and parietal bones were also asymmetrical. The lid margins of both eyes were partially separated, however palpebral fissures were small and no globes were visible (Fig. 1).

Microscopically, the palpebral fissures were irregular and the lid margins were covered by conjunctival epithelium. Subconjunctival connective tissue containing numerous lymphocytes and a multilobulated sebaceous gland. Medial to the palpebral fissure was a multilobular tubuloacinar gland composed of cells having a basilar nucleus and finely granular eosinophilic or basophilic cytoplasm (Fig. 2). The morphological characteristics were consistent with lacrimal gland. No globes were present.

Alterations in the brain (Fig. 3) and spinal cord (Fig. 4) consisted of meningoencephalocele, pachygyria, cavitation of the cerebellum, hydrocephalus (fourth ventricle), syringomyelia, and dorsoventral elongation of the central canal. Frontoparietal lobes of the cerebral cortex covered by meninges were within the soft tissue protrusion and were composed of variable-sized bundles of neural tissue infiltrated and surrounded by fibrous connective tissue. Areas of clearly demarcated white and grey matter were present in the prolapsed tissue just dorsal

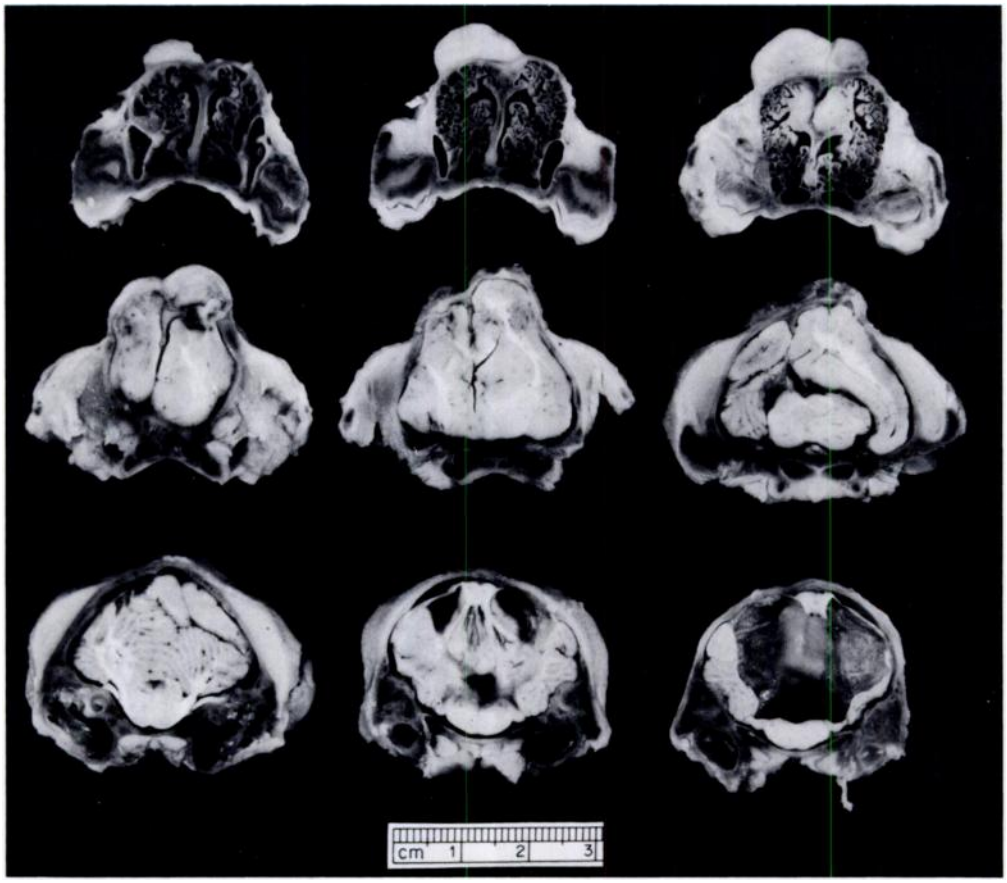

FIGURE 3. Transverse sections of the head of raccoon no. 1 arranged sequentially from top to bottom, starting at the top left and moving to the right. The first section is just rostral to the orbits and the last section is at the caudal aspect of the tympanic bullae. Notice the partial protrusion of the brain through the cranium, the distortion of the brain and the cavitation of the cerebellum (bottom right section).

to the cranial osseous defect but the majority of the parenchyma was disorganized. Intracranial neural parenchyma was also very disorganized. Cerebral sulci and gyri were very few in number, although the layered organization of white and grey matter was present. Identification of structures was difficult because of rostrocaudal malpositioning of the brain. This was probably due to the partial prolapse of the brain, distention of the fourth ventricle and cavitation of the caudal, middle and dorsal lobes of the cerebellum. The caudal cranial fossa, especially the left side, was filled by a thin-walled cavity lined by atrophic cerebellum with the layers gradually diminishing in thickness to four cells. The third ventricle was elongated dorsoventrally and distended. The cystic space was lined by multilayered ependymal cells and the adjacent parenchyma was very thin.

Spinal cord alterations consisted of dorsal extension of grey matter into the dorsal funiculus of the cervical cord, slight asymmetry of the thoracic dorsal funiculus, and myelodysplasia of the lumbar cord. The lumbar central canal was extended dorsally and the dorsal funiculus was asymmetrical. A unilateral crescent-shaped cavitation was present between the lumbar dorsal funiculus on the dorsal horn. The cavitation was Y-shaped in the cranial aspect of the third lumbar segment and continuous with the central canal. Due to the cavitation, the dorsal funiculus was very distorted and caused the dorsal

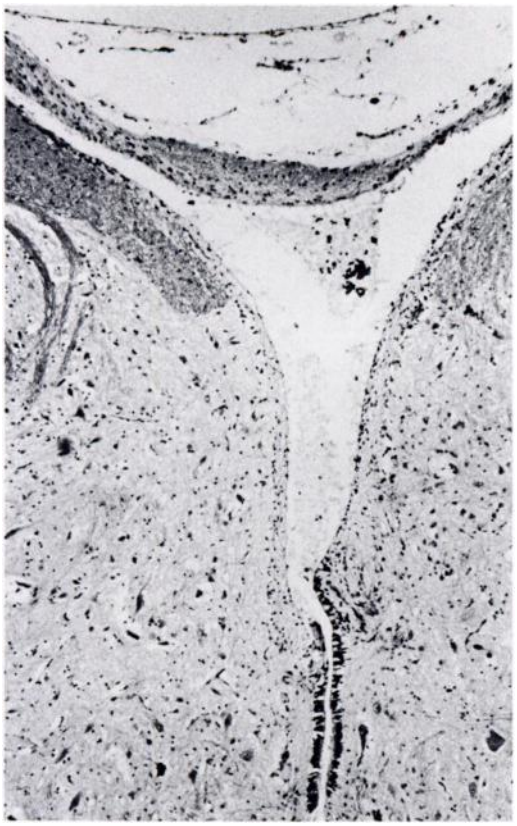

FIGURE 4. Photomicrograph of centrodorsal portion of 6th lumbar spinal cord segment of raccoon no. 1. Notice the dorsomedial syringomyelia and collapse of the dorsal funiculus. H&E stain, ×56.

aspect of the lumbar cord to be flat. The cavitation extended caudally and the adjacent parenchyma was atrophic. In the region of the fourth lumbar segment, the dorsal aspect of the dorsal funiculus was thin and collapsed ventrally, compressing the cavity. A second unilateral cavity was present in the dorsal funiculus of the fifth lumbar segment and the dorsal median septum was deviated toward the nonaffected side. The central canal was slightly elongated dorsoventrally and a dorsomedially oriented section of grey matter was present in the fasciculus gracilus. The dorsal aspect of the spinal cord collapsed ventrally in the region of this ectopic section of grey matter and the central canal became less elongated in caudal segments. A third cavity was present in the sixth lumbar spinal cord segment which was open into the central canal. The cavity occupied the central half of the dorsal funiculus and was bordered dorsally by a thin layer of neural parenchyma. The dorsomedial portion of spinal cord was deviated ventrally thus giving the cavity a "Y" appearance (Fig. 4). Sacral spinal cord segments were not remarkable and no vertebral malformations were present.

Grossly, the eyes of raccoon no. 2 appeared microphthalmic and the corneas were opaque (Fig. 5). The planum nasale was deviated and the right half was reduced in size, abnormally pigmented (lighter in color, mottled) and covered with hair (Fig. 5). Adjacent nasal pelage was unilaterally abnormal in color and marking pattern. The right maxilla was slightly deformed and there was malpositioning of the right upper canine tooth. The palate was intact.

Microscopically, the globes were found to be microphthalmic with marked morphological disorganization. Multiple sections of the globes were examined but the plane of sectioning was oblique and not all ocular components were available for examination. Histopathological alterations in one globe consisted of a chorioretinal coloboma, retinal folds and nonspecific disorganization of ocular structures. The chorioretinal coloboma was adjacent to the optic nerve and was lined by a scant membranous outer layer with occasional neuroectodermal cells and a thin inner membrane continuous with the inner limiting membrane of the retina. A small amount of liquified vitreous was present in the cavity consisting of a collapsed, fine, collagenous framework (Fig. 6). The wall of the microphthalmic globe was irregular in thickness and composed of collagenous connective tissue bundles with scattered pigmented areas, multiple foci of skeletal muscle bundles and evaginations of the retina which formed isolated pockets of disorganized pleomorphic neuroectodermal cells and variable-sized cysts. The cysts were lined by a single layer of low cuboidal to low columnar epithelial cells which resembled neuroectodermal cells. Intimately associated with the cysts were multiple foci of cells with indistinct cytoplasm and round, deeply basophilic nuclei. The cells were arranged in stratified layers and were of probable neuroectodermal origin. The retina was redundant and folded (Fig. 7). The cornea and lens were not available for examination.

Histopathologic abnormalities in the other microphthalmic globe consisted of spherophak-

FIGURE 5. Raccoon no. 2 with microphthalmia, lateral deviation of the nose, unilateral nasal anomaly and pelage abnormality.

ia, cataract, and severe morphological disorganization. The lens was spherical and had cells along the posterior subcapsular border. Additional cataractous changes were diffuse throughout the cortex and consisted of disorganization of lenticular fibers, variable-sized bladder cells, Morgagnian globules, generalized swelling of lenticular fibers and occasional areas of liquifactive necrosis (Fig. 8).

Approximately one-half of the lens' capsule was bordered externally by disorganized proliferated fibrovascular tissue which was continuous with irregular intertwining bundles of collagenous connective tissue (Fig. 8). The connective tissue surrounded variable-sized cysts lined by disorganized neuroectodermal cells. Some cystic spaces were lined by well differentiated retina. Irregular bundles of skeletal muscle were intermixed with the collagenous connective tissue and one focus of disorganized, heavily pigmented epithelium and fibrous connective tissue was intermixed with the collagen strands (Fig. 9). This may have been a scleral staphyloma since the pigmented tissue was adjacent to a vascularized, pigmented structure resembling iris. Definitive identification of all of the congenital anomalies was not possible due to the severe disorganization of the ocular tissues.

Palpebral conjunctiva was present but cornea and a definitive ciliary body were not identified. Periorbital adipose tissue and a multilobulated, tubuloacinar lacrimal gland were present peripheral to the microphthalmic globe.

The principal histopathologic abnormality in the planum nasale was the presence of abnormal epithelium, numerous hair follicles, sebaceous glands and sweat glands in the integument of the right nostril (Fig. 10). The epithelium was reduced to a thin layer of stratified squamous cells and there was a sharp line of demarcation between this abnormal integument and the normal nasal integument on the left side. Most adnexal structures were well differentiated, however, there were several hair follicles that were surrounded by numerous blood filled vascular spaces and a rim of dense connective tissue. There were multifocal aggregates of inflammatory cells consisting of macrophages and lymphocytes with a few neutro-

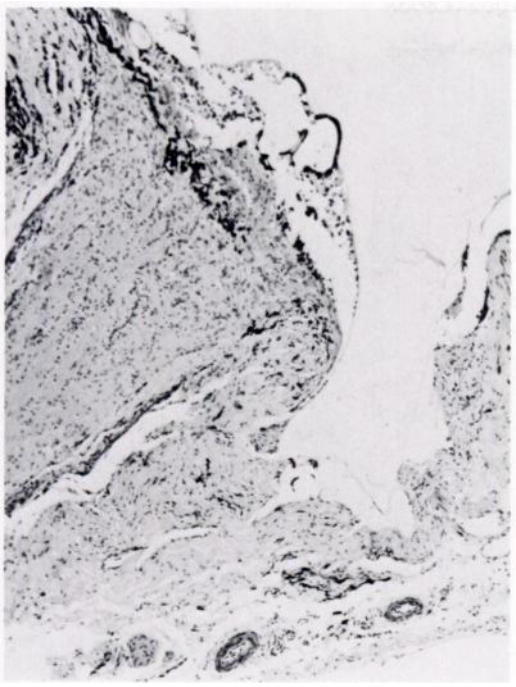

FIGURE 6. Photomicrograph of a chorioretinal coloboma adjacent to the optic nerve and containing prolapsed vitreous, in raccoon no. 2. The membrane lining and defect is continuous with the inner limiting membrane of the retina. H&E stain, ×56.

phils. Multiple transverse sections through the nose revealed discontinuous development of the nasal cartilage. In one section, there was complete absence of the parietal cartilage, and in other sections there were isolated islands of cartilage. One section also had a focus of osseous metaplasia.

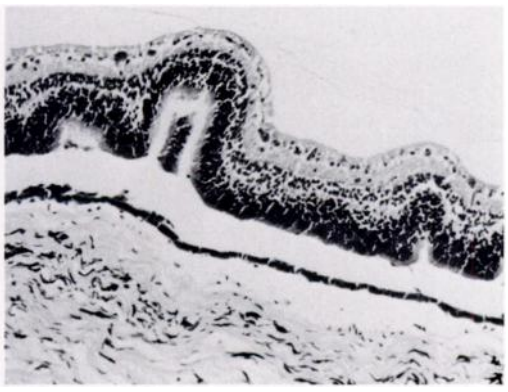

FIGURE 7. Photomicrograph of retinal folds in a microphthalmic globe of raccoon no. 2. H&E stain, ×88.2.

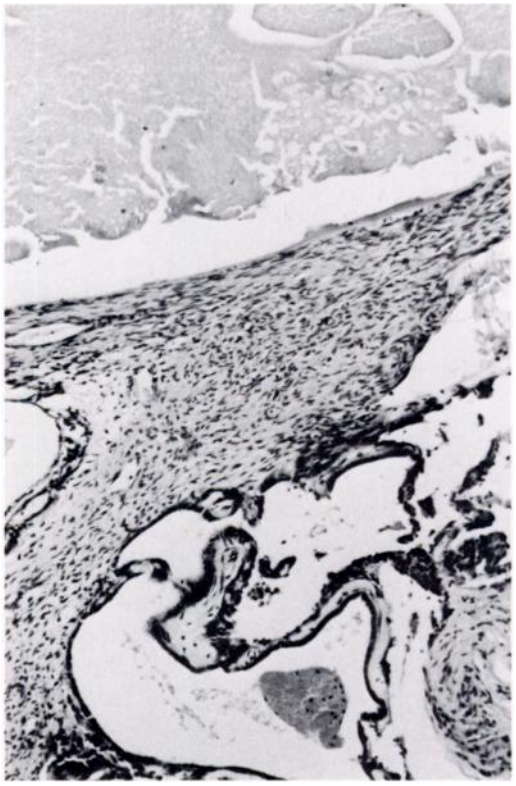

FIGURE 8. Photomicrograph of a cataractous lens, proliferated fibrovascular tissue and cysts lined by disorganized neuroectodermal cells of a globe from raccoon no. 2. H&E stain, ×56.

DISCUSSION

Microphthalmia/anophthalmia is a frequent congenital ocular defect among domestic animals, especially collies and swine, but reports in wild animals are scarce except for the white-tailed deer (*Odocoileus virginianus*) (Priester et al., 1970; Selby et al., 1971; Fulton et al., 1977). Canine microphthalmic globes often have cataracts, staphylomas, and single or multiple excavations within the walls. The neurosensory retina frequently exhibits dysplasia with rosettes and extrascleral retinal tissue may be found between scleral and orbital tissues (Carter, 1981). As exhibited by ocular lesions in raccoon no. 2, microphthalmia in raccoons may also be accompanied by these severe and extensive ocular defects.

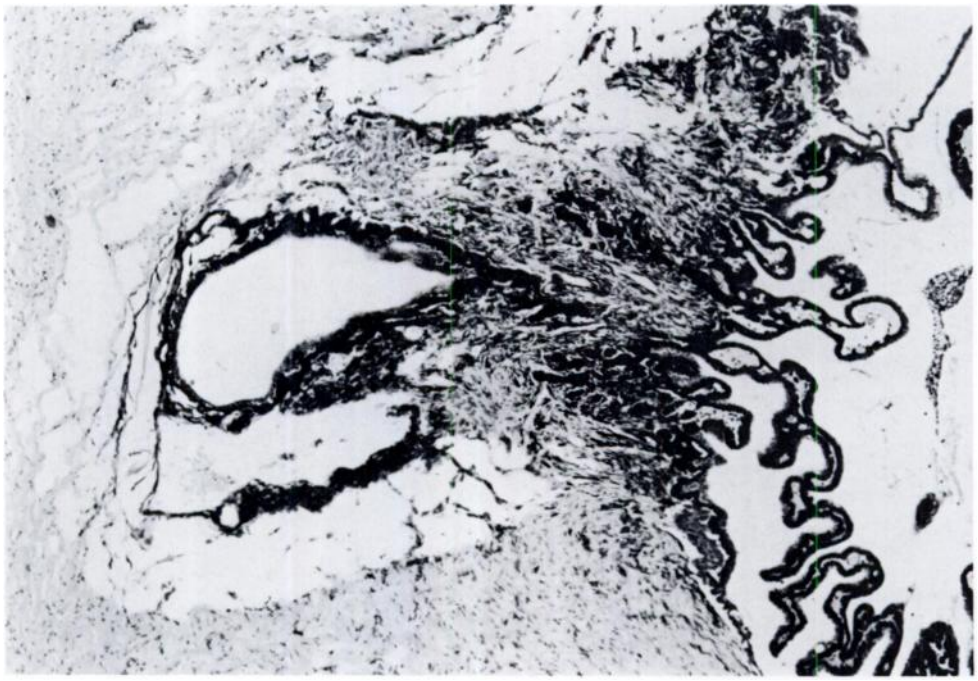

FIGURE 9. Photomicrograph of heavily pigmented epithelium and fibrous connective tissue interwoven in collagenous connective tissue in the wall of a microphthalmic globe of raccoon no. 2, suggestive of a scleral staphyloma. H&E stain, ×56.

Anophthalmia may arise embryologically from lack of formation of the forebrain and optic nerve outgrowth, lack of optic vesicle formation, or formation of the optic vesicle with subsequent degeneration (Weleber, 1975). Lack of forebrain development is not compatible with life. Lack of optic vesicle formation is not necessarily lethal and occurs when the optic pit fails to invaginate. The optic vesicle is the source of the neuroectodermal structures of the globe and is necessary for stimulation of the surface ectoderm to differentiate into other structures of the globe. Either lack of optic vesicle formation or degeneration of the optic vesicle is the probable cause of the anophthalmia in raccoon no. 1.

Microphthalmia may result from either a lack of proper development of the secondary vitreous, which influences the growth of the globe, or from a failure of closure of the embryonic fissure and secondary arrest of the growth of the globe (Weleber, 1975). Since the brain from raccoon no. 2 was not examined, both mechanisms must be considered in the pathogenesis of the raccoon's microphthalmia.

The mechanisms of anophthalmia/microphthalmia in animals involve genetic, infectious, and environmental factors. Anophthalmia has been reported to be inherited in

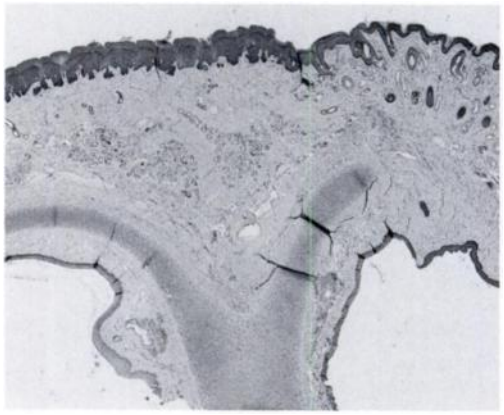

FIGURE 10. Photomicrograph of a transverse section of the nose from raccoon no. 2. Notice the lack of symmetry, the lack of parietal cartilage, abnormal epithelium, and presence of hair follicles, sweat glands and sebaceous glands on the affected side. H&E stain, ×10.

laboratory mice (Chase, 1944; Konyukhov and Vakhruskeva, 1969) and hamsters (Robinson, 1962). Microphthalmia has been reported to be inherited in Australian shepherd dogs (Gelatt and Veith, 1970), guinea pigs (Komich, 1971), laboratory mice (Roberts, 1967), and chickens (Wright and Carr, 1965). Anophthalmia was produced in laboratory rat pups by exposing their dams to nickel carbonyl (Sunderman et al., 1979) and in kittens of a queen treated with griseofluvin during pregnancy (Scott et al., 1975). Microphthalmia in animals has been associated with a variety of environmental factors including vitamin A deficiency in pigs (Watt and Barlow, 1956), lambs of ewes which grazed seleniferous pastures (Rosenfeld and Beath, 1947) and calves of cows infected with bovine virus diarrhea virus while pregnant (Bistner et al., 1970).

The formation of the brain and spinal cord anomalies, like the ocular anomalies, may have been associated with genetic, infections, or environmental factors. An example of inherited syringomyelia and hydromyelia is the disease, spinal dysraphism in weimaraners (McGrath, 1965). The pathogenesis of syringomyelia may be due to a disturbance in the outflow of cerebral spinal fluid from the fourth ventricle, resulting in an increased cerebrospinal fluid pressure, hydromyelia, and subsequent rupture of the dilated ependymal canal. Syringomyelia may also result from an anomalous vascular pattern which leads to ischemia, and subsequent myelomalacia and cavity formation.

Hydranencephaly, like hydromyelia, may also result from an interference with the blood supply to the brain; this has been seen in lambs, associated with bluetongue virus infection (de Lahunta, 1977). The germinal neuroectodermal cells of the mantle layer migrate outward to form the glia and neurons of the cerebral cortex. These cells are most abundant at the end of the first trimester in lamb fetuses and especially susceptible to infection by bluetongue virus. When pregnant ewes are exposed to the virus during the first trimester these cells undergo necrosis and fail to contribute to the substance of the cerebral cortex. Differentiated cells may also undergo necrosis and result in cavitation. Hydrancephaly has also been produced in puppies due to prenatal occlusion or agenesis of the carotid artery. The syringomyelia and hydrocephaly present in raccoon no. 1 may have resulted from similar pathogeneses, although the exact factor or agent is unknown.

Development of the ocular anomalies in raccoon no. 1 may have occurred at a different time in gestation than some of the brain anomalies. Anophthalmia occurs early in the development of an embryo, during the organogenic period of growth (Weleber, 1975). A brain anomaly like pachygyria occurs later in gestation and has been associated with a disturbance in the migration of neuroblasts to the periphery of the cerebral cortex. This migration occurs up to the last third of gestation and until then, the brain is free of gyri or sulci (Escourolle and Pirier, 1978).

Anophthalmia, exencephaly and other congenital anomalies occurred in kittens of queens and pups of rats treated with griseofulvin during pregnancy (Klein and Beall, 1972; Scott et al., 1975). Anophthalmia and exencephaly did not occur together in any one offspring, in siblings, or in offspring of other dams given the same treatment. This suggests that these two lesions occur independently. If the lesions in raccoon no. 1 were the result of exposure to an in utero toxin, then it is possible that the exposure lasted for a certain length of the gestation or was present at multiple intervals.

The planum nasale of raccoon no. 2 was partially dysplastic due to the presence of hairy skin instead of nasal skin and the absence of nasal cartilage. These findings are suggestive of a lack of normal differentiation without hypoplasia or aplasia of the planum nasale. The pathogenesis of this ectodermal dysplasia is unknown.

Based on the severity of the neural anomalies, it is doubtful that raccoon no. 1 would have survived either in the wild or in captivity. It was probably separated from its mother for a short period of time before human acquisition and then was given good nursing care. Raccoon no. 2, however, if raised by its mother or by humans to weaning, quite possibly could have survived and functioned in the wild (up to some point) or captivity, despite the ocular defects, by relying on other senses. Sunquist et al. (1969) using radiotelemetry, monitored the movements and behavior patterns of a blind adult male raccoon and compared them to a normal counterpart. Of interest were their findings of minimal differences in the activity patterns, travel rates, length of movement periods, and

area of home ranges between the blind and normal raccoons. None of these differences appeared to be functionally significant and probably no greater than would be found among normal animals. The blind animal moved about with no apparent difficulty and rarely bumped into trees or other objects, although off roads his behavior and movement patterns were slightly erratic (Sunquist et al., 1969). Two of us (KRK, WAV) also have experience with a young female pet raccoon which functioned well in captivity despite bilateral cataracts and nearly total blindness, and which was eventually released into the wild (Levandoski, pers. comm.).

Raccoons, like domestic animals, may have severe congenital ocular and neural anomalies which may be accompanied by other defects. The extent of these anomalies in raccoon populations is not known and the relationship of these defects to genetic, infectious or environmental factors has not been established.

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BOOK REVIEW . . .

Diseases of the Reptilia, John E. Cooper and Oliphant F. Jackson, eds. Academic Press, New York, USA. 1981. Vol. I, 408 pp. Vol. II, 232 pp. Vol. I, $62.50 US; Vol. II, $41.00 US.

This multi-authored two-volume work is intended to be an "up-to-date guide to diseases of reptiles" with emphasis on captive reptiles. The editors state their hope that the book will provide accessible data on diseases of reptiles. To these ends, the books are successful. The books contain contributions from clinicians, pathologists, microbiologists, parasitologists, and zoologists.

The volumes are divided into four primary sections: Background, Infectious Diseases, Non-infectious Diseases, and Clinical Aspects. The Background Section contains chapters on anatomy and physiology, pathology and histopathological techniques, and microbiology and laboratory techniques.

The remainder of Volume I is devoted to infectious diseases and includes discussions of viruses, bacteria, fungi, actinomycetes, protozoa, endoparasites, and ectoparasites. This section provides an excellent review of the existing body of knowledge concerning the infectious agents affecting reptiles. The three chapters on parasites are comprehensive and probably represent the most complete assemblage of reptilian parasite/host records published to date. Some new material from the various author's files is scattered throughout the text. Among the most notable in this regard, is the publication of twenty-five new cases of fungal disease from the records of the London Zoo. Unfortunately, and presumably due to the delay between preparation of the manuscripts and final publication, several recent important discoveries in reptilian virology are not included.

Traumatic and physical diseases, nutritional diseases, neoplastic diseases, congenital and developmental diseases, and other miscellaneous conditions are covered in the Non-infectious Diseases Section. The chapter on neoplastic diseases is particularly well-done with an extensive listing, in chart form, of all previously published reports of reptilian neoplasms.

The section on Clinical Aspects contains information on diagnosis, treatment, anesthesia, and surgery. The final chapter of the book is a noble attempt to bring order to the diverse and often conflicting information concerning drugs and dosages. Much of the Clinical Aspects Section, however, lacks the detailed discussion found elsewhere in the book. Coverage of surgical techniques is superficial, and persons contemplating a surgical procedure on a reptile for the first time, will not find the information particularly helpful. There is virtually no discussion on hematology or clinical chemistry other than a brief resumé of sampling techniques.

In the book as a whole, little emphasis is placed on important environmental factors such as temperature, humidity, and photoperiod and their relationship to reptilian disease processes. Future editions could be improved by the inclusion of a chapter devoted to these environmental influences which so profoundly affect the pathogenesis and course of reptilian disease.

The book is conservatively illustrated with black and white photographs and line drawings. More illustrations would enhance the text, although certain individual chapters are supplemented with an abundance of illustrative material. Charts summarizing and/or augmenting information presented in the text are used to good advantage.

Extensive bibliographies are provided at the end of each chapter, and of themselves constitute a valuable contribution to those pursuing the study of the diseases of reptiles. Titles of articles are not included in the references. This omission, for which the editors apologize, creates more of an inconvenience than serious deficiency, however.

Clinicians and vivarium personnel, though they may value this book as a reference, are apt to find its overall impact disappointing. The real strength of the book lies in its use as a broad reference reflecting the state of the art, and as a guide to the existing literature on reptile diseases. The book should serve the scientific community well in this capacity.

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