

CHRONIC CRANIAL OSTEITIS IN A ROCKY MOUNTAIN BIGHORN SHEEP

Author: TURNER, JACK C.

Source: Journal of Wildlife Diseases, 18(3) : 373-379

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-18.3.373>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

CHRONIC CRANIAL OSTEITIS IN A ROCKY MOUNTAIN BIGHORN SHEEP

JACK C. TURNER, Division of Life Sciences, Geoscience, and Geography, Sam Houston State University, Huntsville, Texas 77341, USA.

Cranial osteopathy in desert bighorn sheep (*Ovis canadensis* spp.) has been described in specimens collected from various southwestern populations (Allred and Bradley, 1965, Trans. Desert Bighorn Council. 9: 75-81; Allred and Bradley, 1966, Trans. Desert Bighorn Council. 10: 86-97; Paul and Bunch, 1978, J. Am. Vet. Med. Assoc. 173: 1178-1180). Cranial osteonecrosis has been diagnosed in 40-70% of the specimens in many regional collections (Bunch et al., 1978, Trans. Desert Bighorn Council. 22: 16-20; Bunch et al., 1978, Proc. N. Am. Wild Sheep and Goat Symp. pp. 261-273; Paul and Bunch, op. cit.) and is estimated to have a 20% overall frequency in most desert bighorn populations (Bunch and Allen, 1981, J. Am. Vet. Med. Assoc. 179: 1150-1152).

Recent observations have resulted in speculation that cranial osteolysis may be initiated by irritation of nasal and sinus epithelium by larvae of the nasal botfly, *Oestrus ovis*, in concert with secondary bacterial infections by *Corynebacterium* sp. (Bunch et al., 1978, Utah Sci. 39: 97-103; Cobbette and Mitchell, 1941, Am. J. Vet. Res. 2: 358-366; Paul and Bunch, op. cit.). Pyogenic osteomyelitis of the paranasal sinuses could potentially result from this invasion, cause perforation of the cranial vault and death from brain abscessation and/or suppurative meningoencephalitis. Consequently, bot-induced osteopathy has been suggested as a major mortality factor for desert bighorn populations (Bunch, 1980, Trans. Desert Bighorn Council. 24: 14-18; Bunch and Allen, op. cit.; Paul and Bunch, op. cit.).

Heretofore, osteolytic lesions similar to those described for desert bighorn sheep have not been observed in the Rocky Mountain bighorn ecotype. This seems unusual since the botfly has a wide distribution and is abundant in the Rocky Mountain area (Capelle, 1966, J. Parasitol. 52: 618-621; Meleney et al., 1962, Am. J. Vet. Res. 23: 1246-1251; Rogers and Knapp, 1973, Environ. Entomol. 2: 11-23). This paper describes an extensive cranial osteolytic condition in a Rocky Mountain bighorn sheep, *O. c. canadensis*, similar cases of which, previously, have been characterized as botfly induced. It is the intention of this article to emphasize caution in the indiscriminate assignment of the botfly as the etiologic agent of mortality by showing that other avenues of infection may result in conditions similar to those purportedly induced by the botfly larvae.

A 3 to 5 yr old, free-ranging Rocky Mountain bighorn ram was repeatedly observed over an interval of several months by Wyoming Game and Fish personnel in Sybille Canyon, Albany County, Wyoming. The ram appeared alert, active and able to negotiate 2.5 m fences to pursue captive bighorn ewes maintained in the canyon. The last time it was observed alive the ram appeared to have an injury or infection involving the right eye and/or corneal process. The ram was not seen for 10-20 days and subsequently was found dead. Advanced autolysis precluded a necropsy; however, a draining abscess caudal and ventral to the right orbit was observed. The right corneal process and horn were separated from the skull. However, no evidence was discernible to suggest a fall was the cause of death. The head was severed from the

carcass by Wyoming Game and Fish personnel, the horn sheathes removed and the skull given to the author for study.

The cleaned skull showed extensive reduction of cranial bone and numerous fistulae. The right cornual process had separated from the skull. Additionally, an anomalous retention of the second deciduous premolar (dPM³) and fistulation of the palatine process of the maxillary bone at the juncture of PM³-PM¹ were found.

Roentgenographic evaluation of the cranial and facial bones and the tooth arcade was made both prior to and after bilateral hemisection of the skull. Additionally, comparisons were made with radiographs of similar aged, unaffected skeletal material.

The cadaverous residues from the sinuses and nasal cavity were inspected with a dissection microscope. Recovered sclerotized material was prepared (Bils, 1974, *Electron Microscopy, a Laboratory Manual and Handbook*. Western Publishing Co., Los Angeles, California. 278 pp.) for scanning electron microscopic (SEM) comparisons with residues recovered from mascerated specimens of third instar bot larvae.

The second premolar (PM³) of the right dental arcade was impacted by the horizontal eruption of the second deciduous premolar (dPM³) (Fig. 1). The PM² was forced rostrally in the maxilla. The crown of the dPM³ erupted into the PM¹ resulting in an apparent pressure resorption of the PM¹ root and periapical infection. A tract of osteolytic bone extended into the palatal wing of the maxilla at the base of PM¹ and into the alveolar capsule of M¹, through the dorso-lateral plate of the maxilla and into the maxillary sinus (Fig. 1).

Radiologically, there was an obvious decreased density in the right cranial

bones when compared to radiographs of the left side, or to cranial bones of similar aged, unaffected animals (Fig. 2). The right cornual process □ was separated from the cranium at the posterior margins of the frontal-parietal suture by a cleft 0.6-3.0 cm wide that extended into the dorso-posterior orbit, at the supra-orbital process of the frontal and malar bone and at the mid-line frontal suture by a 1-2 cm crevice extending through the supraorbital foramen into the dorsal aspect of the orbit. The trabeculae of the right cornual process were completely absent.

Integrity of the right caudal and rostral frontal sinus was lost, as was the differentiation of the three diverticula of the caudal frontal sinus (Fig. 2). Communication of the frontal sinus with the nasal cavity, dorsal nasal concha and maxillary sinus was by way of several fistulae, 0.3-1.2 cm in diameter, through the ethmoid, at the medial side of the orbit and the ethmoidal meatus.

The median septum separating right and left frontal sinuses was greatly reduced and perforated, 0.8-1.6 cm in diameter, at four sites allowing communication between them. The left frontal sinus was less degenerate; trabeculae had not lost their integrity.

The left frontal bone was perforated by a 1×2 cm fistula (Fig. 2). Bone of the fistula's ventral margin was proliferative and had formed an extensive spiked osteophyte.

The right side of the calvarium was penetrated nine times by fistulae, 0.3-1.5 cm in diameter, mainly within the parietal and occipital bones. Penetration into the cranium occurred from the cornual diverticula of the caudal frontal sinus near the dorso-medial junction of the frontal and parietal bone (Fig. 3). The cranial surface of the parietal bone's ventro-caudal margin was eroded

□ Anatomy and terminology follows the conventions of: MAY, N.S. 1970, *The Anatomy of the Sheep*. 3rd Ed. University of Queensland Press, Brisbane, Australia, 369pp.

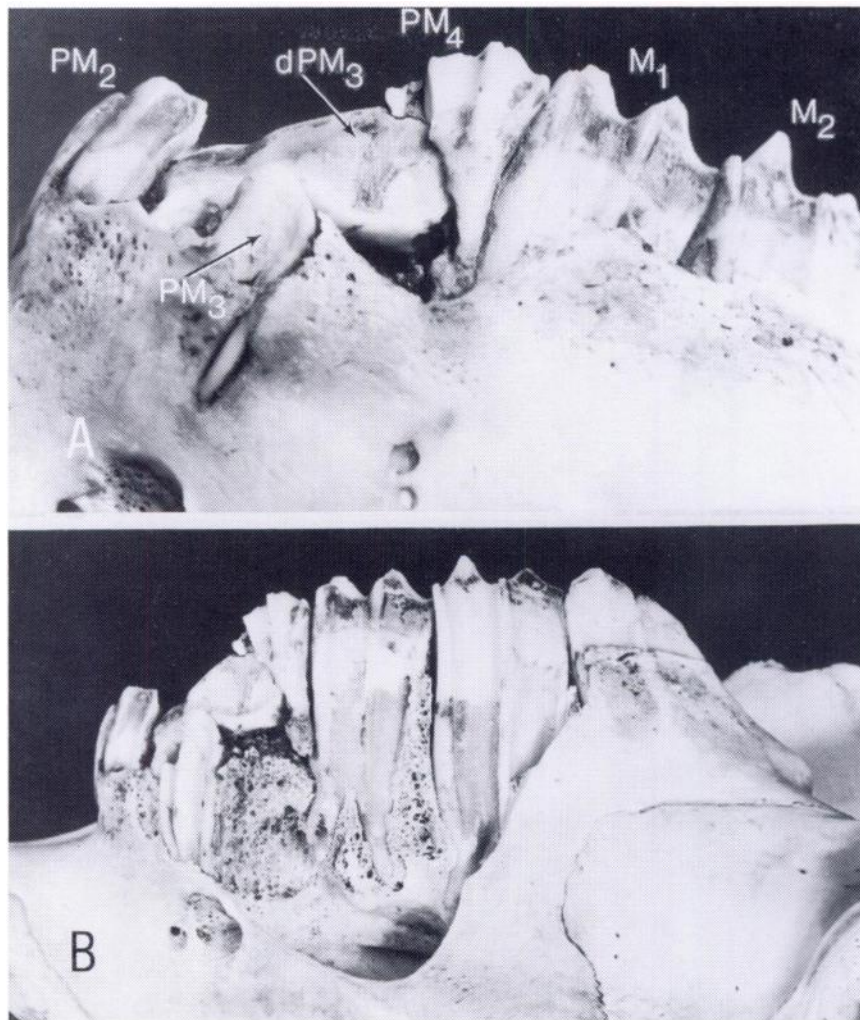


FIGURE 1. Partial buccal view showing the horizontal eruption of the dPM³, impactation of the PM⁴, an apparent pressure resorption of PM¹ and periapical infection of PM³ (A). Removal of the maxillary shield revealed a tract of osteolytic bone penetrating the maxillary sinus (B).

through the petrosal crest laterally and into the petrous part of the temporal bone and occipital bone ventrally and dorso-laterally, respectively. The extensive loss of bone either destroyed or impaired the right temporal meatus, accessory foramina, the postglenoid, mastoid,

stylomastoid and condyloid foramina and condyloid canal.

All soft tissues removed from the sinial and nasal cavities prior to and during the skull's preparation had lost cellular integrity. Sclerotized material was only recovered from the nasal passage. Scan-

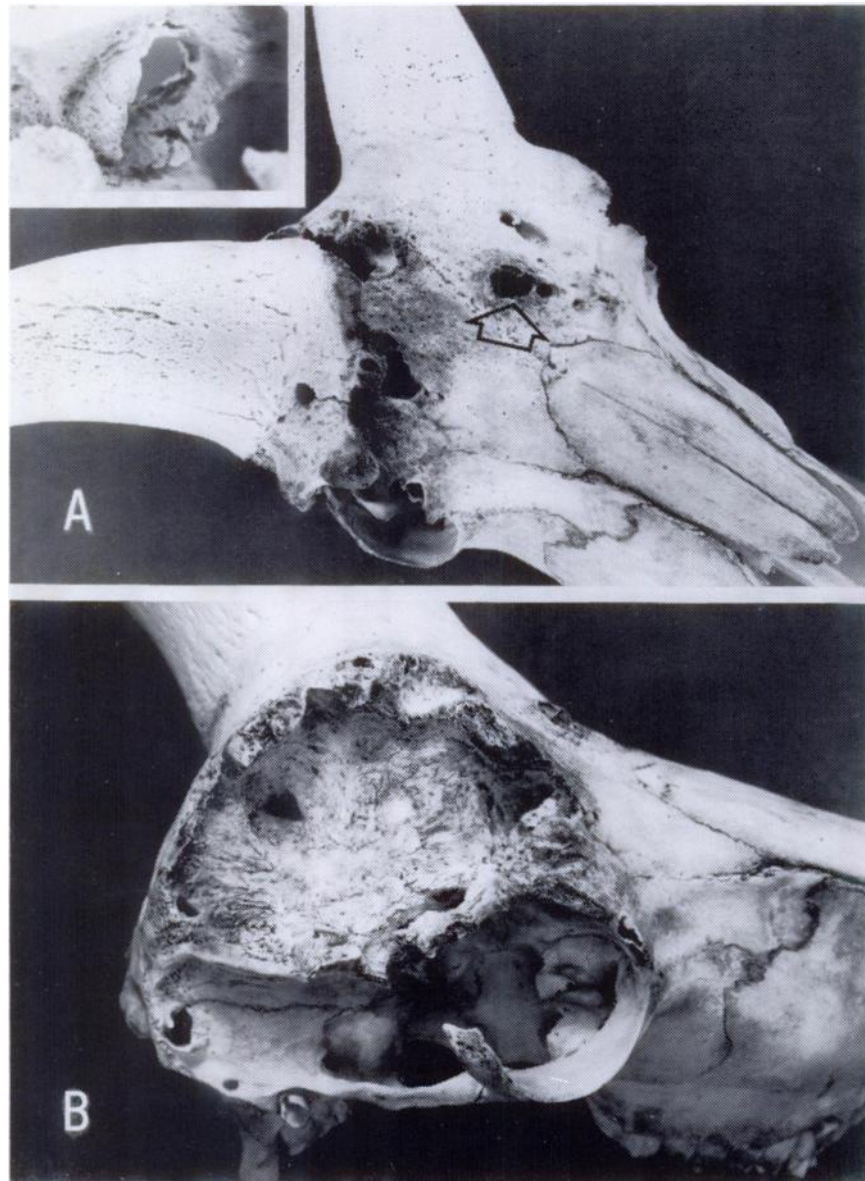


FIGURE 2. Extent of osteolysis on the dorsal skull surface showing lytic cleft separating the skull and right cornual process and a 1×2 cm fistula (arrow) in the left temporal bone (A). Ventral bone surface surrounding the 1×2 cm fistula in the left temporal bone was proliferative and formed a spiked osteophyte (A, insert). Sagittal view of the right frontal sinus showing loss of trabeculate partitions and integrity of frontal diverticula (B).

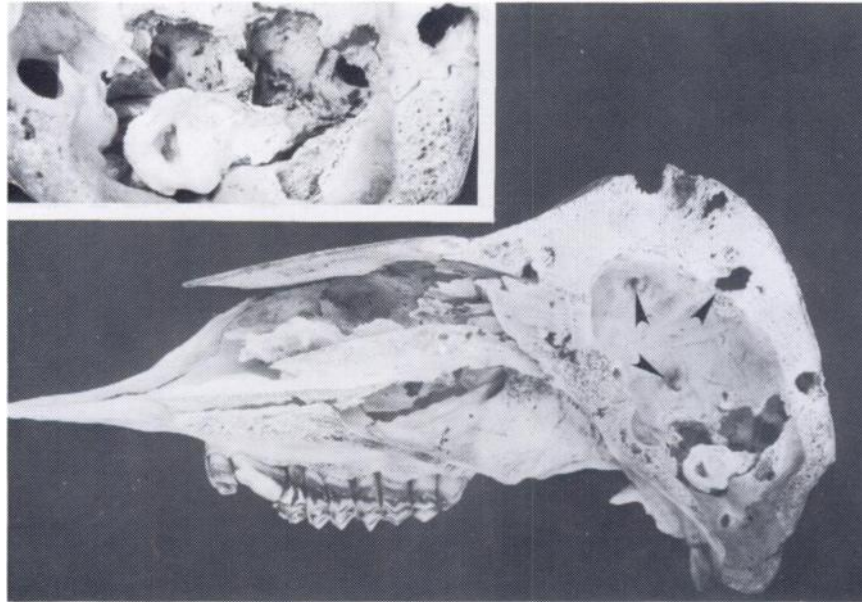


FIGURE 3. Right hemisection of skull revealing reduced bone in the median septum and three fistulae (arrows) penetrating the cranial vault. The loss of bone from the temporal and cranial surface (insert) probably resulted from bacterial action and the accumulation of suppurative toxins.

ning electron microscopic comparisons of the recovered sclerites resembled sclerites from third instar bot larvae. Peritremes or cephalopharyngeal skeletons were not recovered.

The extensive degeneration of cranial bone probably resulted from virulent progressive osteomyelitis, the focus of which resided within the right rostral and caudal frontal sinuses. Osteomyelitis and osteolysis, resulting from chronic osteitis, were a likely consequence of toxins, produced by bacterial action, and ischemia caused by the disruption of local circulation. Loss of cranial trabecular bone probably occurred in response to the suppurative inflammatory reaction (Jubb and Kenedy, 1970, *Pathology of Domestic Animals*, Vol. I, 2nd ed., Academic Press, New York, 593 pp.). The extensive loss was facilitated by the abundance of

cranial trabecular bone, its large surface to mass ratio and vascularization.

The continued thinning of the frontal bone's ventro-medial surface probably resulted in penetration of the brain encasement. The suppurative material probably collected in the ventro-caudal portion of the brain case and produced an osteolysis of the temporal and occipital cranial surface. This undoubtedly caused the observed fistualization and contributed to ischemic necrosis of both bony and subcutaneous tissues. Death presumably came from central nervous system involvement in the infectious process.

The cause(s) of the described cranial osteopathy is far from unequivocal. The observed periapical infection of the PM¹, its erosion of the maxillary medial plate and penetration of infection into the maxillary sinus may have contributed to

a generalized chronic paranasal sinusitis. Indeed, dental anomalies, coexistent with cranial osteolysis, are not uncommon within southwestern desert bighorn populations (Allred and Bradley, 1965, *op. cit.*; Allred and Bradley, 1966, *op. cit.*). Unresolved sinial empyema, resulting from occluded sinial orifices, can cause osteitis and osteomyelitis (Jubb and Kennedy, *op. cit.*).

Oestrid myiasis accompanied by secondary bacterial infection of the affected nasopharyngeal and sinial cavities has been the implied cause of massive cranial deformities observed in skeletal collections from southwestern desert bighorn populations (Bunch and Allen, *op. cit.*; Bunch, *op. cit.*; Bunch et al., 1978, *Utah Sci.* 39: 97; Paul and Bunch, *op. cit.*). Unfortunately, frequency of oestrid related cranial osteopathy predicted from these collections is difficult to assess due to loss of soft tissues, spatial and temporal biases, inadequacy of field data and relative inabundance of study material. Oestrid larvae have been recovered from only two bighorns suffering cranial bone degeneration (Bunch et al., 1978, *Proc. N. Am. Wild Sheep and Goat Symp.* p. 261; Paul and Bunch, *op. cit.*).

North American wild sheep probably are infrequent hosts to oestrid flies. Oestrid larvae have previously been recovered from Rocky Mountain bighorn; however, no significant disease-related problems have been attributed to their occurrence (Capelle, *op. cit.*; Cobbette and Mitchell, *op. cit.*; Couey, 1958, *Montana Fish and Game Comm. Bull.* No. 2, 90 pp; Honess and Frost, 1942, *Wyoming Game and Fish Dept. Bull.* No. 1, 126 pp; Scott, 1942, *J. Mammal.* 23: 345-346). Numerous bighorn sheep studies from Wyoming and Colorado have not disclosed a bot parasitism problem similar to those suggested to occur in the desert southwest (Hibler and Thorne, *pers. comm.*). Similarly, skeletal collections from both states reveal no unusual

cranial deformities (Walker, *pers. comm.*).

Ecological and physiological tolerance by the parasite and microhabitat conditions within related hosts may establish limitations of parasitism within the same ecological area. For example, the suggested predisposition of desert bighorn to aberrant bot parasitism (Bunch and Allen, *op. cit.*; Paul and Bunch, *op. cit.*) may relate to the thermal conditions within the nasal passage. Oestrid larvae are deposited within the nasal passage during summer months. Thermocouple recordings of nasal passage surface temperatures indicate the anterior passages of desert bighorn to be substantially warmer than those of Rocky Mountain sheep (Turner, *unpubl. data*). Larval selection of an optimal thermal environment within the desert sheep's nasal passage may restrict larval development to the upper nasal sinus and extremities of the paranasal sinuses. Increased larval size would prohibit escape from sinial passages through the ethmoidal meatus. Death of the larvae would provide a necrotic focus for infection.

Cranial deformities in desert bighorn have in only a few documented instances implicated bot parasitism as the causal agent. The absence of any demonstration of Koch's postulates of bodily response to infection casts doubt on the severity of bot parasitism to desert sheep survival. Similarly, bot parasitism occurs in Rocky Mountain bighorn, but it does not appear to pose a serious threat to their survival.

I wish to thank Dr. E.T. Thorne, Wyoming Game and Fish Research Laboratory, Dr. E.S. Williams, Wild Animal Disease Center, Colorado State University, and Dr. H. Harlow, Department of Zoology and Physiology, University of Wyoming, for their review of the manuscript. The technical advice and assistance of Mr. H.A. Dawson, Sybille Wildlife Research Unit, Dr. W.P. Meleney, United States Livestock Insect

Laboratory, United States Department of Agriculture, Dr. C. Hibler, Wild Animal Disease Center, Colorado State University, Dr. M. Robinson, Assistant Pathologist, Texas State Veterinary Medical Diagnostic Laboratory, and Dr. R. Ressler, Huntsville, Texas, was greatly appreciated.

Received for publication 28 September 1981
