LETTER TO THE EDITOR

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LETTER TO THE EDITOR . . .

Letter to the Editor Concerning Arnold-Chiari Malformation in a Captive African Lion Cub

In the July 1998 issue of the Journal of Wildlife Diseases, an Arnold-Chiari malformation was reported in a captive African lion cub (*Panthera leo*) (Shamir et al., 1998). The authors described a Chiari type 2 malformation in a 9-mo-old lioness, with head tilt, progressive ataxia and tremors. The neuropathologic findings were summarized as herniation of the cerebellar vermis, caudal diaplacement of the medulla, and histologically loss of granule and Purkinje cells in the herniated folia.

We have observed nearly identical gross and histologic lesions in eight captive African lions of both sexes and varying ages (but <2-yr-old) (Chandra et al., 1999). Preliminary investigation in three of the eight lions (littermates) revealed skull malformation and cerebellar herniation. Based on the preliminary investigation, the disease in these cubs was reported as Arnold-Chiari-'like'-malformation (Papendick et al., 1995). Similar lesions have been reported in captive African lions in Europe (Baker and Lyon, 1977; Perrin-Raybaud et al., 1973; Tuch and Pohlenz, 1973), Australia (O'Sullivan et al., 1977) and South Africa (Bartsch et al., 1975) implicating vitamin A deficiency as a possible cause (based on the low vitamin A concentrations in the livers of affected animals). The clinical signs, gross and histologic lesions also were generally similar to the lioness described by Shamir and co-authors.

In light of our findings and the previous reports, it appears that the lesions reported in the African lioness may be partly or solely due to deficiency of vitamin A (Shamir et al., 1998). Additionally, as mentioned by Shamir et al. (1998), Arnold-Chairi malformation is almost always associated with spina bifida and/or a protruding meningomyelocele in humans and calves. This feature was neither evident in the lioness of their report nor any of the lions in our study and other reports. The changes in these lions have similarities with lesions of hypovitaminosis A in puppies (Mellanby, 1941, 1950), calves (Blakemore et al., 1957; Summers et al., 1995), pigs (Carrigan et al., 1988; Summers et al., 1995) and birds (Howell and Thompson, 1967, 1970), further substantiating the role of vitamin A in the genesis of these lesions. However, the lack of clinical signs or lesions in the male littermate of their report remains unexplained. The recognition of this entity in captive lions should help in the identification of future cases of this unusual condition.

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