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Acute Myocarditis in a Captive African Elephant (Loxodonta africana)

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This report describes the death of a captive African elephant at the Western Plains Zoo, Dubbo, in central New South Wales. The pathological features were consistent with an acute myocarditis, probably of viral etiology.

A 19-vr-old female African elephant died suddenly at the Western Plains Zoo, Dubbo in October 1984. The elephant was born in Africa and taken to a zoo in South Carolina, U.S.A. at an early age. It arrived in Dubbo via southern California in February 1982. The elephant was one of a group of four being fed oaten hay and horse pellets and housed at night. It was considered to be in good health until the time of death. On the morning of death the elephant was dull and unresponsive and showed intermittent tremor of the distal end of the trunk. Clinical examination revealed normal heart rate and temperature. The elephant collapsed and died within 1 hr of examination.

At necropsy the lungs were severely congested and contained frothy, fibrinous fluid. Mediastinal lymph nodes were hemorrhagic. There was 200 ml of bloodstained fluid in the pericardial sac and ecchymoses were present on the epicardial surface of the left ventricle. In the myocardium of the left ventricle was an area of pallor approximately 1 cm in diameter. The myocardium of the right ventricle was soft and flabby. The liver appeared swollen and mottled and there was a slight

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increase in peritoneal fluid. The post-mortem findings were consistent with cardiac failure and pulmonary insufficiency.

Microscopic sections of the left ventricle revealed diffuse acute myocarditis characterized by edema of the interstitium and a mixed cellular infiltrate of polymorphonuclear leucocytes, lymphocytes and histiocytes (Fig. 1). Myofibers were swollen and their fibrils fragmented. Mineralization of necrotic areas was not observed.

The lesions were interpreted to be acute myocarditis and myocardial degeneration, probably of viral etiology. Although viral culture was not undertaken the most likely diagnosis is encephalomyocarditis virus (EMCV) infection. This virus was responsible for an extensive outbreak of mortalities in pigs throughout the central west of New South Wales during 1984 (Seaman et al., 1986, Aust. Vet. J. 63: 292). The disease occurred in association with a plague of mice (Mus musculus) throughout the central and southern areas of the State. Outbreaks of EMCV disease in pigs from the Dubbo area were confirmed in association with the mouse plague (R. F. Webb, pers. comm.) and the oaten hay fed to the elephants at the Western Plains Zoo had been contaminated by mice. Histological lesions similar to those in this case have been described in suckling pigs that died acutely of EMCV disease (Seaman et al., 1986, op. cit.).

There is one published report of EMCV infection in captive elephants (Simpson et



FIGURE 1. Photomicrograph of the myocardium of an elephant with diffuse acute myocarditis. There is edema of the interstitium and a mixed infiltration of polymorphonuclear leucocytes, lymphocytes and histiocytes. H&E, ×200.

al., 1977, J. Am. Vet. Med. Assoc. 171: 902–904). That incident also involved African elephants, but the lesions described were more of a nonsuppurative myocar-

ditis typical of the subacute to chronic disease associated with EMCV infection in grower pigs (Acland and Littlejohns, 1975, Aust. Vet. J. 51: 409–415).

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Kyphosis in the Marsh Rice Rat (Oryzomys palustris)

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Genetic kyphosis is an autosomal recessive abnormality of the spine first described in domestic laboratory mice (*Mus musculus*) by Dickinson and Meikle (1973, Lancet 1: 1186). Mice homozygous for the condition exhibit progressive erosion of the

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thoracic vertebrae, resulting in an S-shaped dorsal kyphosis commonly known as "hunchback" or "humpback." The defect worsens with age; respiration is impaired, feeding is hindered, adult body weight is reduced by as much as 30%, and fecundity decreases. Recessive homozygosity can be detected in mice not yet weaned by their defective "placing re-