Reprinted from the JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, Vol. 177, No. 9, Pages 934-937.

Mitral Valve Insufficiency with Congestive Heart Failure in a Pukeko

A 4½-YEAR-OLD MALE pukeko *(Porphyrio melanotus)* weighing 881 g was examined because of respiratory distress. (Pukekos are related to coots and gallinules. The subject of this report was imported from New Zealand, where the species is plentiful in low-lying, swampy regions.) The medical history included 2 episodes of temporary limb stiffening and falling to lateral recumbency. These episodes occurred 4 and 3 weeks earlier, and each episode lasted less than 1 minute.

The pukeko was in a poor nutritional state. Respirations were deep, with moist rales, and at a rate of approximately 70/min. Vomiting and passage of green-tinged feces occurred during examination. The heart rate was irregular and approximately 290/min. Hematologic values did not vary from those of healthy pukekos.

An electrocardiogram (Fig 1) was produced, as described by Sturkie,[1] with the thoracic lead on the central carina and with the bird in left lateral recumbency, after anesthetization with ketamine, halothane, and nitrous oxide.

There was no P wave present. The heart rate was irregular, with R wave to R wave intervals of 0.12 to 0.26 seconds. The QRS width was 0.03 to 0.04 seconds. The QT interval was 0.12 seconds. The RS amplitude varied up to 30%. The electrical axis was −90 degrees. The absence of P waves and the irregular heart rate was interpreted as indicating atrial fibrillation on the basis of criteria used in mammalian electrocardiography.[2]

Ventrodorsal radiography revealed an enlarged left atrium (Fig 2). Barium sulfate cream was administered orally to indicate the position of the esophagus.

The dyspnea was relieved by placing the pukeko in an oxygen tent kept dark and quiet. Digoxin in a pediatric elixir form was administered orally at a dosage of 11.4 μg/kg of body weight, QID. By the following morning (14 hours later), respirations were normal, but dyspnea recurred whenever the pukeko was handled. Despite gradual increases of digoxin dosages (to 50 μg/kg of body weight, TID), changes were not noted on serial electrocardiography. The pukeko gradually lost weight and died after being treated 7 weeks.

At necropsy, the pericardium was adhered to the left ventrolateral body wall and contained approximately 1 ml of clear, straw-colored fluid. The left atrium was dilated to approximately 3 cm diameter, with walls 1–2 mm thick (Fig 3). A nearly spherical thrombus 1.5 cm in diameter was in the left atrial lumen. The left atrioventricular valve was thickened, opaque, nodular, and firm, with a smooth surface. The left ventricular wall was symmetrically hypertrophied, with a decreased lumenal diameter. The pulmonary veins were distended. The right atrium was approximately 1.5 cm in diameter. The liver was firm and contained multiple punctate yellowish lesions randomly distributed in the parenchyma. All other organs appeared normal. Heart blood and liver cultures had no anaerobic or aerobic bacterial growth.

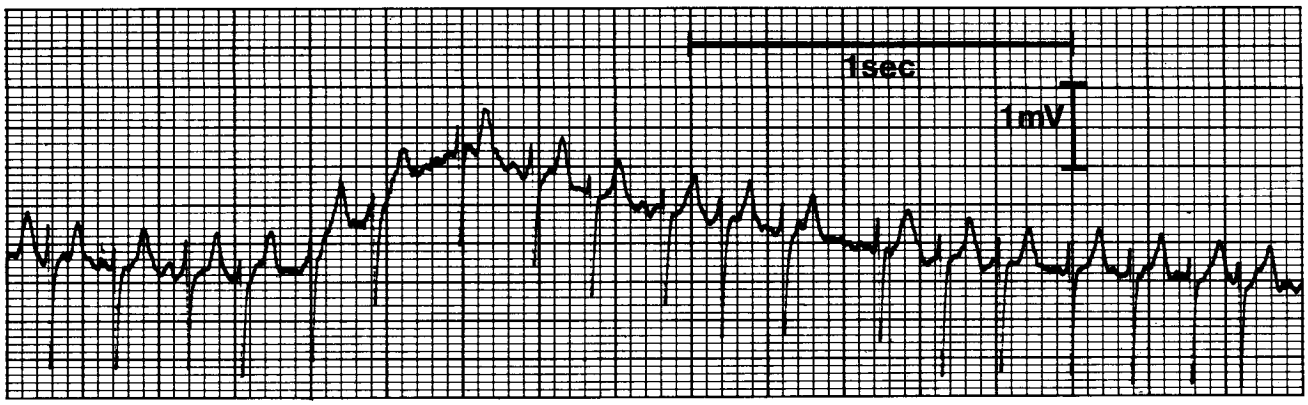

Fig 1—Lead II ECG, showing atrial fibrillation. 50 mm/sec; 10 mm = 1 mV.

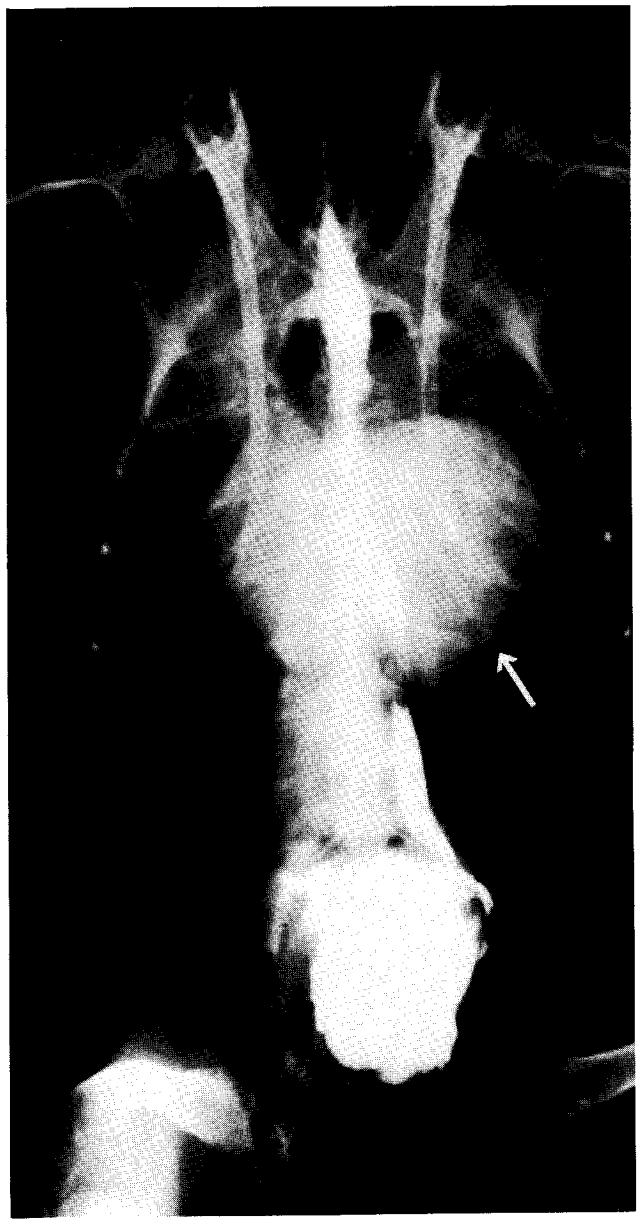

Fig 2—Ventrodorsal radiograph, showing left atrial enlargement (arrow). Barium sulfate cream was administered orally.

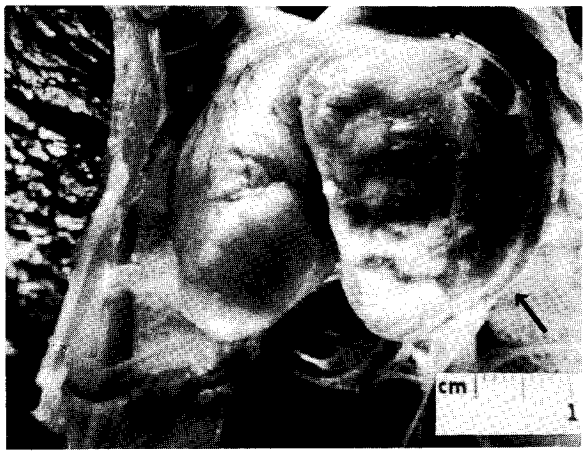

Fig 3—Postmortem view of heart, showing markedly enlarged left atrium (arrow).

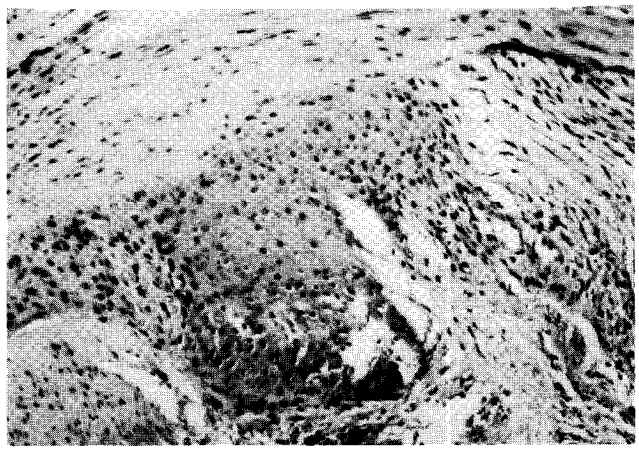

Fig 4—Photomicrograph of mitral valve, showing focus of cartilaginous metaplasia. H&E stain; ×32.

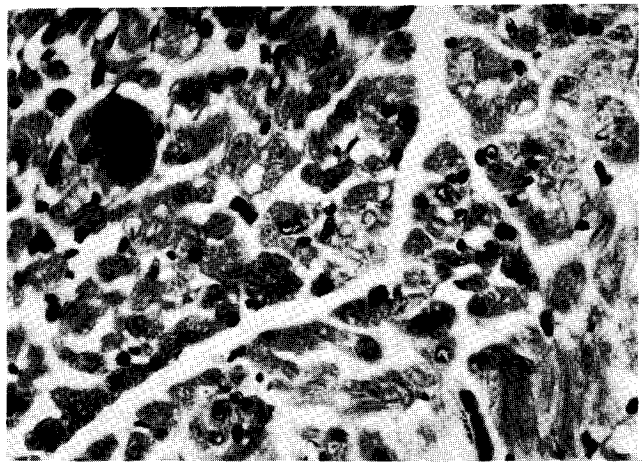

Fig 5—Photomicrograph of myocardium, showing focus of fiber hypertrophy, vacuolization, and degeneration. H&E stain; ×102.

Sections of all organs were fixed in buffered formalin, stained with hematoxylin and eosin, and examined microscopically. The mitral valve leaflets had multifocal hemorrhages and several regions of cartilaginous metaplasia (Fig 4). Lesions of multifocal noninflammatory cardiomyopathy included fiber hypertrophy, vacuolization, and degeneration (Fig 5). There were a few areas of noninflammatory endocardial thickening. The thrombus in the left atrium was confirmed microscopically. There was focal arteriosclerosis with cartilaginous metaplasia in the ascending aorta. Except for mild anthracosis and rare foci of mineralization, the lungs were normal. The tracheal cartilage was partially mineralized. Liver changes were centrilobular vacuolization and mild hepatocellular atrophy. All other tissues were normal.

The valvular cartilaginous metaplasia with multifocal hemorrhages, the left ventricular hypertrophy, and the left atrial dilatation supported the diagnosis of mitral endocardiosis followed by valvular insufficiency.[3] Mitral systolic regurgitation would increase left atrial pressure and would contribute to the left atrial and pulmonic vein distension.

Atrial fibrillation is a common sequela to atrial enlargement in mammals.[4] Because of the similarities of mammalian and avian cardiac electrical conducting systems,[1] atrial fibrillation in this case was assumed to be the result of the atrial dilatation.

The formation of the thrombus may have been the result of altered left atrial blood flow due to the mitral valve insufficiency, atrial dilatation, and atrial fibrillation.[3] Presumably, this thrombus acted as a ball valve and exacerbated the abnormalities by causing mitral stenosis during diastole.

The right atrial dilatation and the liver lesions[5] are consistent with right-sided heart failure due to chronic congestive heart failure. The pulmonary edema characteristic of congestive heart failure in mammals could not be demonstrated grossly or microscopically, possibly because of anatomic differences in the avian lung. The clinical signs (dyspnea and moist rales) in this case corresponded to those expected in mammals with congestive heart failure.—*Bruce A. Beehler, DVM, Richard J. Montali, DVM, and Mitchell Bush, DVM, National Zoological Park, Smithsonian Institution, Washington, DC 20008.*

---

1. Sturkie PD: Heart: contraction, conduction, and electrocardiography, in Sturkie PD (ed): *Avian Physiology*, ed 3. New York, Springer-Verlag, 1976, pp 111–114.
2. Bolton GR: *Handbook of Canine Electrocardiography.* Philadelphia, WB Saunders Co, 1975, pp 120–122.
3. Jubb KVP, Kennedy PC: *Pathology of Domestic Animals*, ed 2. New York, Academic Press Inc, 1970, p 114.
4. Guyton AC: *Textbook of Medical Physiology,* ed 4. Philadelphia, WB Saunders Co, 1971, p 353.
5. Jubb KVP, Kennedy PC: *Pathology of Domestic Animals,* ed 2. New York, Academic Press Inc, 1970, p 199.