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Source: Journal of Wildlife Diseases, 41(4): 825-828

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-41.4.825

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Fallot's Tetralogy in a European Brown Bear (Ursus arctos)

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ABSTRACT: A congenital heart malformation was diagnosed in a free-ranging adult female European brown bear (*Ursus arctos*) found dead due to intraspecific violence. At necropsy, the heart had all four features of Fallot's tetralogy. No further malformations were identified in the animal, which was of normal size and weight for its age, in normal body condition, and had probably borne young. The heart condition was considered to have contributed to death during an attack by another bear.

Key words: Congenital, European brown bear, Fallot's tetralogy, heart malformation, in-traspecific, pathology, *Ursus arctos*.

Fallot's tetralogy is an important congenital heart malformation seen in humans and in animals, with reports in horses, cattle, dogs, swine, sheep, llama, inbred rat strains, and cats (Michaëlsson et al., 2000). Very few cases of malformations in bears have been reported. Congenital hydrocephalus in two black bears (Ursus americanus), a 2-yr-old (Halstead and Kiel, 1962) and a 2-mo-old (Kearney, 1968), and a possibly congenital hydrocephalus with ependymitis and encephalitis in a 7-wk-old Asian black bear (Ursus thibetanus) (Sorjonen et al., 1982) have been published, as well as a report of supernumerary mammae in 2 of 61 examined female black bears (Erickson, 1960). This is, to the best of our knowledge, the first published report of a cardiac malformation in a bear.

In September 2004, a European brown bear (*Ursus arctos*) was found dead outside the town of Jokkmokk in northern Sweden (66.6°N, 19.8°E). A local wildlife officer inspected the site and concluded that intraspecific violence was the probable cause of death. The body was sent to the Department of Wildlife at the National Veterinary Institute for necropsy and tissue sampling before final storage at the Museum of Natural History, according to Swedish Environmental Agency's regulations for the management of dead freeranging bears.

The bear was a 7-yr-old adult female according to dental cementum annulations, with a body weight of 120 kg and ample body fat reserves. Multiple bite puncture wounds with superficial involvement of subcutaneous tissues were evident on the head, neck, and shoulders, as were parallel claw wounds over the back. Part of the greater omentum was protruding through a 10 cm, roughly round perforation of skin on the ventral abdomen. Wound patterns corresponded in shape and size to bear claw and teeth damage, confirming attack by another bear. The lungs were severely congested. Liver and other internal organs, excluding the heart, were normal. One corpus luteum was present in each ovary, as well as several corpora albicantia. Gross and microscopic examination of the six mammary glands showed no signs of active milk production, but the size of the glands indicated that the female previously had been suckled. The stomach and intestines contained approximately 10 kg of berries, mainly blueberry (Vaccinium myr*tillus*) and crowberry (*Empetrum nigrum*), but no remains of animal origin.

Externally, the heart had no obvious changes in size or shape. Internally, a subvalvular and valvular pulmonary stenosis was found, caused by hypertrophy of the infundibulum and the supraventricular crest (Fig. 1). The outflow diameter was restricted to approximately 10 mm at the level of the valves, about half the size of a normal brown bear heart. The pulmonary trunk widened abruptly into a poststenotic dilatation. An overriding aorta was centrally positioned above the septum, which had a large, oval 30×20 mm semimembranous ventricular septum defect. Severe, somewhat variable concentric right ven-

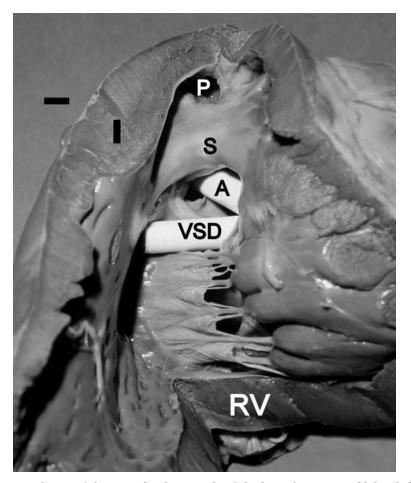


FIGURE 1. Apical view of the opened right ventricle of the heart from a 7-yr-old female brown bear, showing all four features of the congenital heart malformation Fallot's tetralogy: subvalvular pulmonary stenosis due to myocardial hypertrophy of the infundibulum (I), including the supraventricular crest (S) and stenosis of the pulmonary valves (P); large ventricular septal defect (white tubing with VSD); an overriding aorta (white tubing with A); and hypertrophy of the right ventricular free wall (RV). Horizontal bar=1 cm. Photo: Bengt Ekberg, National Veterinary Institute.

tricular wall hypertrophy, up to four times the thickness of a comparable normal brown bear heart, was also noted. In conclusion, all four features of a Fallot's tetralogy were identified. Malformations in other organs were not found. Histology was not performed on internal organs because of moderately advanced autolysis.

Ventricular septal defects (VSDs) are the most common congenital heart malformation in both humans and in most domestic species (Michaëlsson et al., 2000; Schoen, 2005). Fallot's tetralogy is a malformation that includes a large nonrestrictive VSD in combination with three other defined changes in the heart: biventricular connection of the aorta, obstruction of the right ventricular outflow tract caused by a severe subvalvular pulmonary stenosis, and, as a sequel to the restricted outflow, right ventricular hypertrophy. The cyanotic form of Fallot's tetralogy is due to rightto-left ventricular shunting of blood and reduced blood flow to the lungs. In domestic animals, this congenital heart disease invariably leads to clinical disease, where affected animals fatigue easily, are usually cyanotic, and usually have retarded growth rate. In dogs, a genetic breed predisposition for a defect resembling tetralogy of Fallot has been described in Keeshond (Robinson and Maxie, 1993).

The cause of death in the bear in this case was probably not the bear-inflicted wounds, because they could not be considered directly lethal. It was therefore concluded that the bear probably died primarily of circulatory collapse, in which the existing heart malformation played a significant part. It is not clear why another bear attacked this female. The heart condition may have reached the point where the female became clinically affected and cyanotic due to stress or physical exertion. If so, she may have shown aberrant behavior during contact with another bear, which in turn may have triggered an overly aggressive attack. An adult brown bear normally lives solitarily and avoids close contact with other bears, except for mating in May through June. The bear in this case was killed in September. Another possibility is that the female was protecting weaned yearlings still following her. Bears are presumed to be induced ovulators. With corpora albicantia in the ovaries, and with mammary glands indicating suckling young in the past, it is probable that the bear had borne young (Tsubota et al., 1989). However, as the bear had never been immobilized or tagged, no other information on her previous history was available. Brown bear females in Sweden usually give birth every second or third year (Sandegren and Swenson, 1997), depending on when she weans her cubs, because a lactating female does not come into heat. The situation when females are killed seems to arise when a male bear takes over a new home range. By killing the cubs, the male can mate earlier and increase his reproductive success (Swenson et al., 1997). Females have then been killed when trying to defend the cubs from the attacking male. Several cases of intraspecific deaths in which males have killed adult female bears have previously been recorded in Sweden (Zedrosser et al., 2004; Mörner et al., 2005). It has also been recorded that the males have fed from the carcasses of the killed female bears.

In conclusion, the first cardiac malformation described in a bear is a Fallot's tetralogy found in an adult female brown bear that obviously had managed to survive in the wild for 7 yr, had fed well, and had presumably bred, before being killed by another bear.

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- 328 JOURNAL OF WILDLIFE DISEASES, VOL. 41, NO. 4, OCTOBER 2005
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Received for publication 20 December 2004.