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Source: Journal of Wildlife Diseases, 41(3) : 654-658

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-41.3.654

Journal of Wildlife Diseases, 41(3), 2005, pp. 654–658

Congenital Hemicerebral Anomaly in a Stranded Pacific Harbor Seal (*Phoca vitulina richardsi*)

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ABSTRACT: A stranded 5-month-old female Pacific harbor seal (*Phoca vitulina richardsi*) was presented displaying tachypnea and diminished lung sounds. No neurological abnormalities were noted. The animal was treated for verminous pneumonia, but died 2 wk later. Gross necropsy examination revealed a severe obstructive verminous pneumonia associated with large numbers of *Otostrongylus circumlitus.* In addition, the majority of the right cerebral hemisphere was absent, with hypoplasia of the left cerebellar hemisphere, absence of the right pyramid, and malformation of the right occipital bone. Histopathologic findings included multifocal thrombosis and inflammation of pulmonary arteries, verminous pneumonia, and mild vacuolation of the subependymal white matter in the third ventricle representing swelling of myelin sheaths and edema. This is the first report of a hemicerebral anomaly in a marine mammal.

Key words: agenesis, brain, congenital defect, hydranencephaly, Pacific harbor seal, *Phoca vitulina*.

Congenital defects are physiological or structural abnormalities present at birth as a result of faulty development, infection, genetics, or injury (Slauson and Cooper, 2002). Although congenital defects are commonly observed in terrestrial mammals (Leipold and Troyer, 1995), there are relatively few reports of congenital defects in marine mammals. The majority of the latter lesions have been found on postmortem examination, and therefore are restricted to gross anatomical defects (Gulland et al., 2001). Congenital neurological abnormalities that have been noted in marine mammals include cerebellar hypoplasia and hydrocephalus in a northern fur seal (*Callorhinus ursinus*) (Spraker in Gulland et al., 2001), spina bifida in a fossil mysticete (Fordyce and Watson, 1998), and hydrocephalus in northern elephant seals (*Mirounga angustirostris*) (Griner, 1983; Trupkiewicz et al., 1997). Congenital abnormalities identified in harbor seals (*Phoca vitulina*) include cleft palate (Suzuki et al., 1992), ectrodactyly (Tarasoff and Pierard, 1970), abnormal tooth number (Coyler, 1936; Suzuki et al., 1990), alopecia and dental aplasia (King, 1964), and penile deviation (Spraker et al., 1994). To the best of our knowledge, no brain malformation of harbor seals has been reported. This paper describes the postmortem finding of absence of the majority of the right cerebral hemisphere in a Pacific harbor seal.

A female 5-mo-old weaned Pacific harbor seal was stranded along the central California coast at Moss Landing Marine Laboratory in Monterey County, California (36°48'N, 121°47'W) on 29 July 2002. The animal was brought to The Marine Mammal Center in Sausalito, California showing signs of lethargy, weight loss, tachypnea, and diminished lung sounds on auscultation. Supportive care and treatment with antibiotics and anthelmintics were started for suspected verminous pneumonia associated with *Otostrongylus circumlitus.* Two wk later, on 12 August 2002, she was found dead and postmortem examination was performed immediately.

Gross necropsy findings included approximately 5 ml of red foam and live nematodes with a morphology consistent with *O. circumlitus* in the trachea, hilus, bronchi, bronchioles, pulmonary artery, and pulmonary arterioles. Multifocal, well-demarcated areas of hemorrhage were noted in both lung lobes, involving full thickness of lung parenchyma. Histopathologic findings in the lungs included mul-

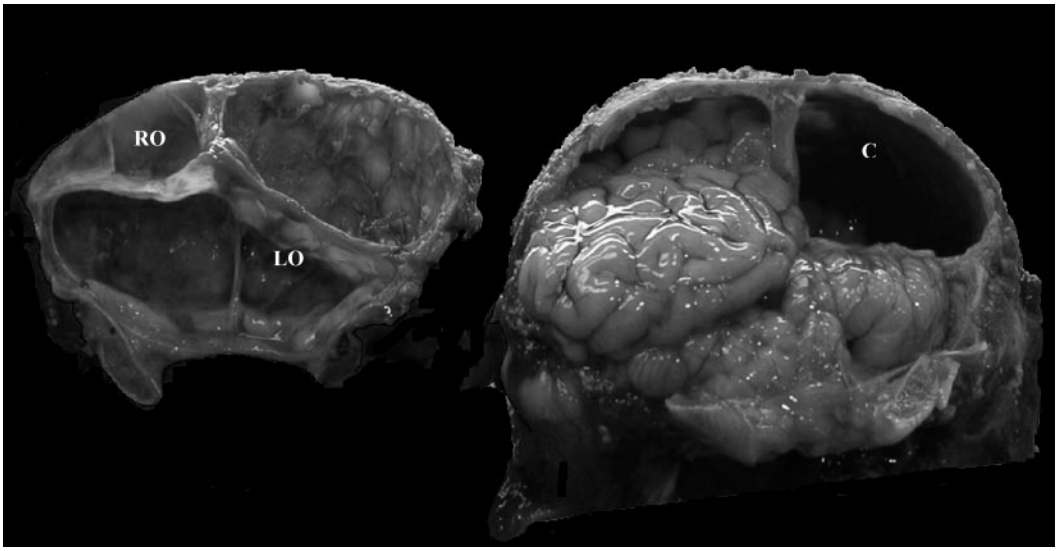

FIGURE 1. Transverse cut of the caudal aspect of the skull. On the left photo, the right occipital bone (RO) and the portion of the cranial cavity it normally covers are reduced in size. The space for the left cerebellar hemisphere ventral to the tentorium is also reduced in size (LO). On the right photo, there is absence of the right cerebrum as evidenced by the empty calvaria (C).

tifocal subacute to chronic thrombosis of small, medium, and large pulmonary arteries, intra-alveolar and intrabronchial nematodes with mild to moderate accompanying pyogranulomatous pneumonia, and moderate bronchial epithelial goblet cell hyperplasia.

The occipital bone on the right side of the skull was distorted and approximately half the size of the contralateral bone (Fig. 1). Upon intracranial examination, the right side of the cranial cavity contained approximately 10 ml of free, clear yellow fluid and there was absence of the majority of the right cerebrum (Fig. 2). Portions of the right olfactory peduncle, caudate nucleus, and hippocampus were still present. The left cerebellar hemisphere also appeared smaller than the right hemisphere. The left cerebrum and right cerebellar hemisphere appeared normal in size. There was mild enlargement of the left lateral ventricle. All cranial nerves appeared to be paired with no grossly detectable atrophy. Examination of the midbrain revealed the absence of the right crus cerebri. Examination of the medulla showed absence of the right pyramidal tract. The crus cerebri and pyramid normally contain projection fibers from the cerebral neocortex. The absence of the right neocortex in this seal explains the absence of the right crus cerebri and pyramid. Histopathologic examination of the brain revealed multifocal scalloped areas of mild vacuolation in the subependymal white matter of the third ventricle with mild astrogliosis, consistent with edema and swelling of myelin sheaths.

Infection with the metastrongyle nematode, *O. circumlitus*, is well recognized as a common pathogen in harbor seals (Geraci, 1978). Heavy infestations cause obstructive bronchitis and bronchiolitis (Lauckner, 1985), with the possibility of secondary infections, resulting in pneumonia or pulmonary abscesses (van der Kamp, 1987). The clinical signs of respiratory disease and death of this seal from verminous pneumonia is typical for juvenile seals in this area (Gulland et al., 1997). The apparent lack of clinical neurological abnormalities in this case, however, was surprising. The reorganizational

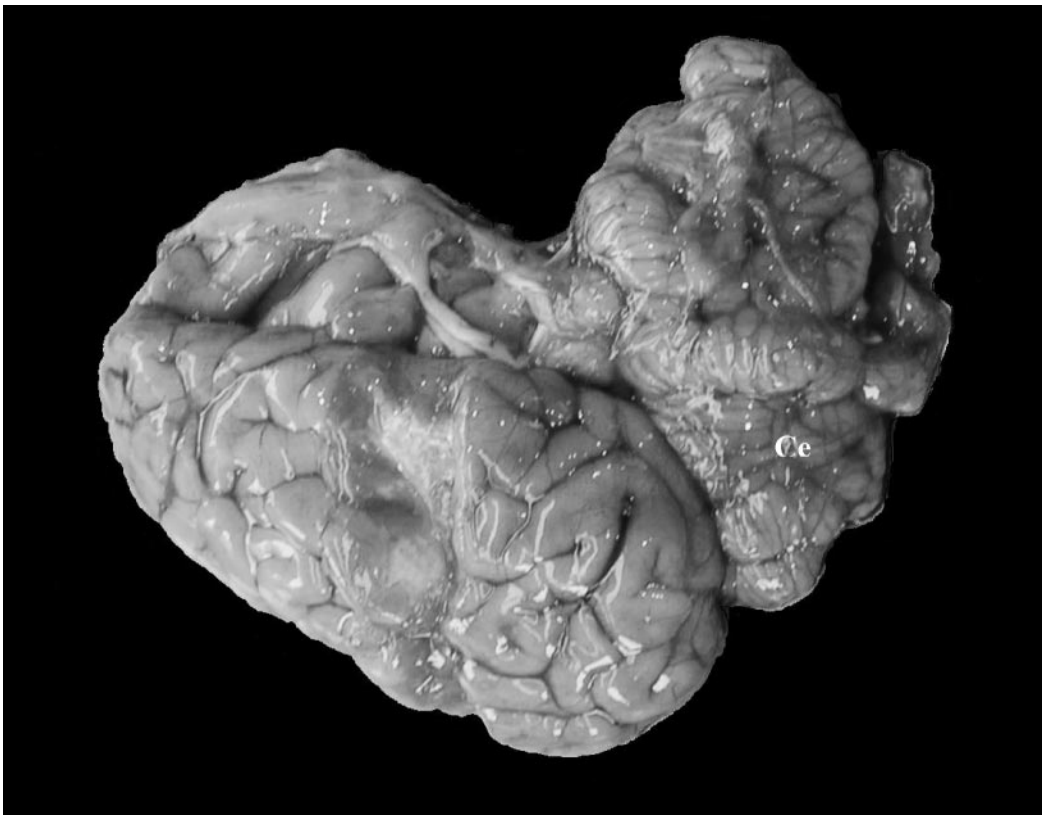

FIGURE 2. Brain removed from the skull. The neocortical portion of the right cerebral lobe is absent, and the left cerebellar hemisphere (Ce) is reduced in size.

potential of the developing brain allows immature animals of many species to sustain damage to large areas of the brain and show remarkably little functional deficits (Chugani et al., 1996). A case of a 6-yr-old boy with unilateral cortical malformation had magnetic resonance imaging (MRI) findings that revealed that the unaffected hemisphere retained motor control of both hands (Staudt et al., 2001). It is likely that neuronal development during fetal growth in the seal allowed for similar cortical representation, resulting in apparently normal neurological signs. Domestic animals are much more dependent on their brain stem for survival than the cerebrum. The same may be true for the harbor seal, and this seal still had a normal left cerebrum.

Syndromes that can result in severe loss of cerebral tissue include hemicerebral anencephaly and hemihydranencephaly. Anencephaly is the term used for failure of cerebral development with concurrent skull malformation, and is a relatively common finding in human feti (Birnbacher et al., 2002). However, to the best of our knowledge there is no report of a nearly complete absence of a cerebrum (i.e., hemicerebral agenesis) in any mammalian species. There is a report of hemicerebellar agenesis in a 38-yr-old woman found incidentally on MRI, with no concurrent clinical signs of cerebellar abnormalities on neurological examination (Erdongan et al., 2002). This case was believed to be the result of intrauterine destruction of the cerebellum.

Another congenital abnormality, hemihydranencephaly—complete or partial destruction of one cerebral hemisphere with transformation into a membranous sac containing cerebrospinal fluid—has also

been described in humans (Greco et al., 2001). Its development has been ascribed to a number of causes, including infections, irradiations, fetal anoxia, medications, and twin–twin transfusion, leading to a vascular disruption (Greco et al., 2001). Reports of cerebral hemiatrophy caused by multiple developmental venous anomalies also exist (Uchino et al., 2001). This marine mammal case represents hemihydranencephaly rather than anencephaly because of the presence of the right olfactory peduncle, caudate nucleus, and hippocampus, and the fluid-filled right side of the cranial cavity. The majority of the absent cerebral tissue receives its blood supply from the right side of the cerebral arterial circle. In utero compromise of this vasculature could explain the extensive right-side hydranencephaly in this animal. With either congenital vascular malformation or vascular thrombosis, blood supply to brain tissues would cease, leading to tissue necrosis and eventual hydranencephaly. As a result of the prenatal loss of this cerebral tissue, the absence of any neocortical projection fibers accounts for the absence of the right crus cerebri and pyramid. Normally the right cerebrum has abundant neurons that project to the left cerebellum via the pons; the absence of these accounts for the left cerebellar hypoplasia. Although the leptomeninges and dorsal cerebral cortical tissue were not seen grossly in the right skull, and are usually developed in cases of hydranencephaly (Nau et al., 1979), it is possible that they could have been so thin that they were easily destroyed during the necropsy.

In domestic animals, a similar hydranencephaly occurs bilaterally from in utero infection of the fetus with the Akabane, bluetongue, and Cache Valley viruses (Summers et al., 1995). In the case of this harbor seal, no associated infectious etiology was identified.

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Received for publication 16 June 2004.