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CASE REPORT

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# Novel presentation and pathophysiology of heavy parasitic burdens in Weddell seals (*Leptonychotes weddellii*) during sedation

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## Abstract

**Background** Marine mammals act as sentinel species, with top predators' overall health reflecting their ecosystem, integrated across multiple trophic levels. Yet apparently healthy wild animals may have significant subclinical pathology that goes undetected due to unknown medical histories. Marine mammals, particularly phocid seals, often suffer from heavy parasite burdens. While there are documented cases of severe respiratory infections resulting in complications during sedation, there have been no reports of gastrointestinal parasites contributing to poor outcomes during examinations requiring sedation or anesthesia. This report describes two unique presentations of high intestinal parasite loads that purportedly predisposed Weddell seals (*Leptonychotes weddellii*) to complications under sedation, and characterizes underlying pathology.

**Case presentation** Two adult female Weddell seals exhibited prolonged apnea and vomiting while under intravenous sedation, that led to aspiration and mortality despite resuscitation attempts. Post-mortem examination revealed a severe *Diphyllobothrium* tapeworm impaction in the duodenum, with the parasitic mass causing a partial or complete obstruction. In both cases, the stomach was remarkably distended, suggesting the parasitic mass slowed gastric emptying. Both animals' stomachs contained a high parasite burden with roundworms embedded into the mucosa. Histological analysis identified underlying pathological conditions that were likely parasite related, including chronic pneumonia associated with lungworm infestations, reactive, depleted and fibrosed lymph nodes, granulomatous lymphadenitis and hepatitis. Further examination in one of the animals revealed severe gastritis and necrotizing duodenitis at the site of the cestode infection.

**Conclusions** To our knowledge, this is the first description of a significant gastrointestinal parasitic impaction being linked to acute distress during sedation in a marine mammal. We provide an in-situ depiction of the severe cestode infection. It is noteworthy that both animals in this case study exhibited histopathology consistent with chronic inflammation across multiple organ systems. Whether animals were sufficiently immunocompromised that rapid parasite growth became unchecked, or whether the parasite infestation led to dysfunction in other organs remains unresolved. We discuss the potential for premedication with prokinetic agents that increase esophageal sphincter tone to mitigate complications in future late-summer Weddell seal handlings.

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**Keywords** Cestode, Duodenitis, Gastritis, Inflammation, Nematode, Parasite, Pinniped, Obstruction, Weddell seal

## Introduction

Marine mammals are regarded as environmental sentinels, with the overall health and condition of top predators reflecting that of their ecosystem due to their high trophic level niche, long lifespans, and large fat reserves [1–3]. However, apparently healthy wild animals can have surprisingly high prevalence of disease and/or parasite burdens. Marine mammals have notably large gastrointestinal capacities and long intestinal tracts as compared to terrestrial mammals [4] and species with primarily fish-based diets are known to be heavily parasitized [5–7]. Despite a diversity of parasites found in marine mammals, little is known regarding natural temporal (seasonal or age-class related) fluctuations in parasite burden, changes that accompany long fasts and subsequent refeeding, or linkages between the host immune response and the magnitude of parasite infection. This is particularly relevant given anticipated impacts of climate events and long-term warming on parasite virulence and transmission rates, with downstream consequences to their hosts [8].

Pinnipeds are notably difficult to anesthetize as these species have cardiorespiratory adaptations for breath-holding on long underwater dives that can lead to decompensation while under anesthesia. For example, marine mammals are inherently apneustic breathers that routinely exhibit extreme bradycardia, peripheral vasoconstriction, and drops in core body temperature to achieve low diving metabolic rates [9]. When free-living, pinnipeds are believed to exert conscious control over the initiation and magnitude of the 'dive reflex', with a graded response that corresponds with the anticipated duration of a dive [9]. Each facet of the dive response is tightly regulated, working in concert to prioritize oxygen delivery to the anoxia-intolerant organs, thus extending dive durations. However, a lack of response or discoordination of these systems during anesthesia (e.g., bradycardia without the associated peripheral vasoconstriction) due to inhibited conscious control can lead to hypotension, hypo- or hyperthermia, protracted recoveries, and severe prolonged apneas resulting in death [10–13]. Moreover, pinnipeds have additional adaptations to separate the respiratory from digestive tracts for feeding underwater including a relatively elevated larynx, cartilaginous corniculates angled anteriorly that close off the laryngeal inlet, and exceptionally distensible pharyngeal soft tissue that can easily obstruct the airway and make intubation for resuscitation efforts difficult [14].

Remote field sites, logistics of transportation including rugged terrain or aerial support, and/or cold ambient temperatures all create significant obstacles in maintaining and operating gas anesthesia equipment for reliable use. Because of these constraints, injectable sedatives provide a valuable alternative for immobilization [15, 16] especially in larger species when manual ventilation is not feasible. Still, even closely-related species can respond differently to anesthetic agents, and there may be a narrow margin of safety in some species. For example, among phocid seals, tiletamine-zolazepam is very effective for sedation in grey seals (*Halichoerus grypus*; 0.5-1.0 mg/kg intramuscular IM) and northern elephant seals (*Mirounga angustirostris*, 1.0 mg/kg IM), in maintaining a deep enough plane of anesthesia for examinations while remaining eupneic [11]. However, tiletamine-zolazepam elicits prolonged apnea and/or high mortality rates in Weddell seals (*Leptonychotes weddellii*, 0.75 mg/kg IM [17]) and leopard seals (*Hydrurga leptonyx*, 2.0 mg/kg IM [18]) and ketamine-midazolam and/or butorphanol-midazolam combinations have instead been adopted [19]. These combinations have the advantage of the ability to lower dissociative-to-benzodiazepine ratios, and to use reversable agents in the latter combination [15, 19]. However, underlying pathology can impact the success and efficacy of anesthesia protocols, particularly in wild animals with unknown medical histories [20].

Weddell seals in Erebus Bay (McMurdo Sound), Antarctica (~77°S, 165°E) have historically been a particularly tractable model species for detailed physiological, behavioral, and demographic study owing to their accessibility on the fast-ice within close proximity of research stations [21, 22]. Individuals exhibit a high degree of lifetime site fidelity, allowing for repeated observations days to years apart and through multiple generations [22]. From the pioneering work characterizing the marine mammal aerobic dive limit [23], to investigation of ontogenetic [24, 25], thermoregulation [26], and reproductive biology [27, 28], as well as instrumentation for study of the species' foraging ecology [29–32], numerous research teams have utilized sedation protocols to safely handle Weddell seals of all ages and sexes through the past decades. Thus, there has been substantial protocol development for chemical sedation in Weddell seals specifically, with few complications (e.g., apnea that responded quickly to respiratory stimulant [15]) and these procedures have been adopted through numerous research programs from multiple countries. Here, we report rare cases of acute anesthetic death associated with an underlying cestode duodenal impaction in two apparently healthy Weddell seals. Concomitant parasitic and inflammatory lesions were present. Knowledge and preparedness for this type of complication during immobilization of phocid seals is important for research and

conservation monitoring programs and is likely to be valuable for rehabilitation efforts in additional pinniped species.

## Case presentations

Two adult female Weddell seals were handled as part of a larger study in Erebus Bay, Antarctica. Particularly given the species' large size, sedation was required for tissue collection for scientific research. Lifetime histories of both animals were known through their inclusion in the 60 + year-long demographic study of the Erebus Bay Weddell seal population [22]. Specimen identification number (SPENO) 14852, hereafter referred to as 'Weddell seal 1' was 16 years old and weighed 334 kg at the time of handling on January 30, 2017; she was a reproductively mature female but had skipped pupping during the study year (referred to as a 'skip-breeder'). 'Weddell seal 2' (SPENO 15253) was 21 years old and weighed 368 kg when handled on January 18, 2023; she had successfully weaned a pup in early December. Both had been successfully handled earlier in the austral summer (Weddell seal 1 during late November; Weddell seal 2 during both late October and November). All prior handlings utilized the same sedation protocols, with no concerns during any of the previous sedation events. Weddell seal 1 was not observed in-between handlings. Weddell seal 2 was observed twice in the two weeks prior to final handling. The animal was resting and appeared to be behaving normally, but could not be approached on foot until found on January 18 due to deteriorating sea ice.

During initial approach and visual observation, animals were apparently healthy and alert, mobile, and active. Both animals had dry pelage at the time of capture indicating that they had been out of the water for some time prior to anesthesia (and Weddell seal 2 had been observed hauled-out at least two hours prior to handling). The seals were captured by hoop net and administered an intramuscular induction dose targeted at ~ 2.0 mg/kg ketamine hydrochloride (KetaVed, Vedco, Inc.) and 0.1 mg/kg midazolam hydrochloride (Almaject, Inc.) based on visually estimated body mass [15]. Weddell seal 1 received 2.04 and 0.10 mg/kg ketamine and midazolam, respectively, and Weddell seal 2 was administered 2.34 and 0.12 mg/kg ketamine and midazolam. While visual mass estimates and doses were slightly overestimated in Weddell seal 2, this was well in range of many successful procedures both in the larger study during late-summer (mean dose: 2.04 ± 0.02 and 0.10 ± 0.001 mg/kg ketamine and midazolam, respectively; range in dose – ketamine: 1.59–2.61 mg/kg; midazolam: 0.08–0.13 mg/kg) and within bounds reported previously during protocol development [15].

The induction dose achieved a light (e.g., still mobile; Weddell seal 1) to moderate (sedated with sluggish movement; Weddell seal 2) level of sedation. Following the induction period, intravenous maintenance doses at a target of 0.5 and 0.025 mg/kg ketamine and midazolam, or midazolam alone [15] (Table S1) were administered to permit initial blood collection from the extradural vein using a 18 gauge x 3.5-6" spinal needle, injection of physiologic tracers, and mass measurement after animals were briefly rolled onto a tarp and weighed using a tripod and hanging scale. Serial blood draws were taken, muscle and blubber biopsies collected, morphometric measurements taken, and a reproductive transrectal ultrasound examination performed, with additional maintenance doses provided as necessary to keep animals sedated (at a moderate-to-deep plane; e.g., small to no body movements) and eupneic [28]. At 10 min following the induction dose, Weddell seal 1 began receiving maintenance IV doses, for 5 total (each 10–17 min apart). Weddell seal 2 did not receive a maintenance dose until 31 min after the induction, and was administered 3 maintenance doses 16–18 min apart. The required maintenance doses for these two cases were approximately average compared with individuals in late-summer in the larger study (mode: 5, mean: 4.5 ± 0.2 SE maintenance doses; range: 0–11), to achieve sedation for 112 ± 0.6 min (range: 101–129 min) followed by an additional 7.6 ± 0.9 min (range: 0–46 min) of recovery time ($n = 104$ successful sedations in January-February).

Animals were maintained in sternal recumbency with respiration (RR) and heart rates (HR) within normal limits for Weddell seals (Weddell seal 1, RR: 12 breaths per minute, HR: 64–65 beats per minute bpm; Weddell seal 2, RR: 10 breaths per minute, HR: 67–75 bpm) [15], with mucous membrane coloration, capillary refill time, and plane of sedation monitored throughout by designated trained personnel and veterinarians. In both cases, at ~ 75–80 min after initial approach and capture, slow and irregular breathing was noted while the animals were still sedated. This was 5 min and 16 min following the final maintenance dose of sedative for Weddell seal 1 and Weddell seal 2, respectively. The animals then became apneic with belching or borborygmi, and within ~ 2–5 min the animals began vomiting large volumes of digesta. Action was taken immediately to clear the airway manually and with bulb syringe suction to improve respiration, and to orient the seal such that the hind end was slightly elevated relative to the head to reduce aspiration of stomach contents.

For Weddell seal 1, intense vomiting prevented successful intubation. Nostril flaring was noted but air was not being passed and multiple doses of doxapram (each 180–200 mg; Hikma Pharmaceuticals) were administered IV; chest compressions were performed. For Weddell seal 2, an intubation attempt was initiated after the animal first became apneic but the seal began vomiting as soon as the

mouth was opened. After the first bout of vomiting, the seal was successfully intubated and provided with supplemental oxygen for active ventilation for ~15 min until nasal movement was observed and the seal was extubated and regular breathing resumed (HR increased to 90–100 bpm) with additional supplemental oxygen provided by nasal cannula. However, 5 min later, there was a sudden drop in heart rate from 100 to 32 bpm immediately followed by more bouts of vomiting; afterwards the animal was re-intubated. In addition to multiple doses of doxapram (each 160–600 mg; Hikma Pharmaceuticals), Weddell seal 2 was administered epinephrine (15 mg; Pivetal), dexamethasone (40 mg; Vedco Inc.), and multiple doses of ephedrine (2.5-5 mg; Endo Inc.), atropine (5–8 mg; Vedco Inc.), and flumazenil (1–2 mg; Hikma Pharmaceuticals) IV (*See* Table S1 for full time records and drug doses administered). Despite all resuscitation efforts the animals never regained spontaneous ventilation and cardiopulmonary arrest was confirmed by a veterinarian after 50 (Weddell seal 1) and 47 min (Weddell seal 2).

Complete necropsies were conducted immediately after death, led by on-site veterinarians (Weddell seal 1: R.M.; Weddell seal 2: S.J.). All activities and findings were reported to the National Marine Fisheries Service Marine Mammal Protection Act permit offices (No. 17411, 23273), Antarctic Conservation Act Offices, and institutional IACUCs.

Tissues including right middle and cranial lung, mediastinal lymph node adhered to the heart, mesenteric, tracheobronchial, and axillary lymph nodes, and parasites from Weddell seal 1; and brain, affected duodenum, ileum, liver, lung, multiple lymph nodes including tracheobronchial, parasites, muscle, spleen, thyroid, tongue, tonsil, ovaries, and uterus from Weddell seal 2 were collected in 10% neutral buffered formalin. Paraffin embedding, sectioning at 4–5 µm and hematoxylin and eosin staining were performed at Histology Consultation Services and analysis was performed at Alaska Veterinary Pathology Services. Gastrointestinal parasite samples from Weddell seal 1 were similarly collected in 10% neutral buffered formalin and subsamples also frozen at -20 °C and sent to the University of California Davis for morphological and genetic identification. Partial 28 S (LSU) rDNA of the D1-3 region, and the complete ITS-1 and ITS-2 regions were amplified and sequenced for nematodes and partial cox-1 (COI) mitochondrial regions were amplified and sequenced for Acanthocephalans. Two specimens of each species were sequenced. The cestodes collected from the duodenal impaction yielded insufficient DNA for PCR amplification.

Blood that had been collected at the time of handling in EDTA vacutainers was analyzed for hematocrit via centrifugation in the field, hemoglobin using the cyanomethemoglobin method, and red and white blood cell counts using Ery-Tic and Leukotic Bluplus kits (Medix™). Blood (heparinized; or collected in serum separate tubes) was also centrifuged to separate and collect plasma or serum, and stored frozen at -80 °C until analysis. Serum cortisol (MP Biomedicals, Inc., ImmunoChem coated tube) and insulin (MilliPore Sigma, Inc., Sensitive Rat) were measured using commercially available radioimmunoassays that were validated for use in Weddell seals using parallelism and standard addition tests; samples were analyzed on a Wizard 2 gamma counter (Perkin Elmer, Inc.). Serum non-esterified fatty acids (ZenBio, Inc.), L-Amino Acids (Sigma-Aldrich, Inc.), and serum iron (Pointe Scientific, Inc.) concentrations were also measured using colorimetric assays. Additional plasma chemistry panels for indices of liver function were analyzed at the Kansas State Veterinary Diagnostic Laboratory (KSVDL); Selenium was also measured from whole blood at KSVDL.

### Incidence of anesthetic complication

These two cases of anesthetic-related fatal complications were rare. Across the larger research programs led by the authors, no anesthesia related mortalities occurred in these programs during the early-summer breeding season (October-December; $n = 104$), and late-summer (January-February) mortality rate was 1.9% (2 of 106 equivalent sedation procedures on adult females with ketamine/midazolam [15] for condition assessments from years 2013–2023). Below, we report that remarkably high parasitic loads concentrated in the gastrointestinal tract may have predisposed two adult Weddell seals to significant complications during sedation.

### Clinical pathology

The seasonal changes in mass observed in these two cases fit patterns expected based on their reproductive histories, with post-partum females recuperating mass across the austral summer while skip-breeding females lose mass during the late-summer annual pelage molt [33]. Both animals appeared to be in good body condition (ultrasound blubber depths were measured prior to anesthetic complications for Weddell seal 2 only, and were typical of healthy individuals [34]; axillary: dorsal – 2.95, lateral – 4.03 cm). While previous work provides a species-specific reference range for most hematological and chemistries of interest [33–37], less information is available during the late summer (January-February) period when both seals exhibited anesthetic complications. Both seals' hematology and chemistries exhibited shifts across the study period but appeared to be within previously reported ranges (Table 1), except for potentially low creatinine and total bilirubin during late summer relative to published studies. Weddell seal 2 exhibited substantially

**Table 1** Mass, hematology, and blood chemistries including liver enzymes from the two Weddell seals. This provides temporal comparison in physiological attributes across the austral summer. Weddell seal 1 was first handled early in the summer as a skip-breeder; Weddell seal 2 was handled across lactation. Both seals were recaptured approximately 2 months later, when anesthesia-related complications arose and the parasitic mass was found. Reference ranges (with notation regarding time of year) are provided for comparison. Data are presented as ranges or mean ± standard deviation

| Physiology, Hematology, or Chemistries | Weddell seal 1 SPENO 14852 | | Weddell seal 2 SPENO 15253 | | | |
|---|---|---|---|---|---|---|
| | Skip-Breed (11/29/16) | Late Summer (01/30/17) | Early Lactation (10/27/22) | Late Lactation (11/25/22) | Late Summer (01/18/23) | Reference Range |
| Mass (kg) | 390 | 334 | 488 | 321 | 368 | 190–554 (Oct-Feb; [33]) |
| Hematocrit (%) | 65 | 57.5 | 58.5 | 58.3 | 56.8 | 43-72.3 (Oct-Feb; [33]) |
| Hemoglobin (g $dL^{-1}$ whole blood) | 23.9 | 21.6 | 20.6 | 19.8 | 21.9 | 18.1–31.6 (Oct-Feb; [33]) |
| Red Blood Cell (x$10^6$ per mL) | 3.85 | 4.04 | 3.47 | 2.86 | 3.03 | 2.68–5.23 (Oct-Feb; [33]) |
| White Blood Cell (x$10^3$ per mL) | --- | --- | 8.00 | 13.85 | 7.88 | 5.88–17.6 (Oct-Feb; Rzucidlo *unpubl*) |
| Cortisol (μg $dL^{-1}$) | 124.5 | 162.7 | 172.3 | 154.1 | 173.4 | 71.9-236.6 (Oct-Feb; Rzucidlo *unpubl*) |
| L-Amino Acids (nmol $\mu L^{-1}$) | 4.07 | 4.55 | 2.79 | 2.50 | 5.03 | 1.71–8.61 (Oct-Feb; Rzucidlo *unpubl*) |
| Non-esterified Fatty Acids (μM) | 488.9 | 559.9 | 288.4 | 972.1 | 2167.4 | 178.1-2227.1 (Oct-Feb; Rzucidlo *unpubl*) |
| Insulin (ng $mL^{-1}$) | 0.073 | 0.090 | 0.086 | 0.045 | 0.070 | 0.039–0.551 (Oct-Feb; Rzucidlo *unpubl*) |
| Glucose (mg $dL^{-1}$) | 63 | 76 | 98 | 78 | 90 | 56–180 (Oct-Dec; [34]); 99 ± 18 (Dec-Jan; [35]) |
| Total Protein (g $dL^{-1}$) | 8.6 | 8.0 | 8.7 | 7.4 | 8.1 | 8.0-11.8 (Oct-Dec; [34]); 7.4 ± 0.8 (Dec-Jan; [35]) |
| Albumin (g $dL^{-1}$) | 2.9 | 3.0 | 3.7 | 3.3 | 3.1 | 3.2–6.1 (Oct-Dec; [34]); 2.5 ± 0.3 (Dec-Jan; [35]) |
| Globulin (calculated) (g $dL^{-1}$) | 5.7 | 5.0 | 5.0 | 4.1 | 5 | 3.2–6.5 (Oct-Dec; [34]); 4.9 ± 0.6 (Dec-Jan; [35]) |
| Urea nitrogen (mg $dL^{-1}$) | 19 | 34 | 12 | 9 | 39 | 11–43 (Oct-Dec; [34]); 22 ± 6.7 (Dec-Jan; [35]) |
| Creatinine (mg $dL^{-1}$) | 1.6 | 0.9 | 1.2 | 1.2 | 0.7 | 1.3-2.0 (Oct-Dec; [34]); 1.4 ± 0.3 (Dec-Jan; [35]) |
| Total Bilirubin (mg $dL^{-1}$) | 0.4 | 0.2 | 0.9 | 1.2 | < 0.2 | 0.4–0.8 (Oct-Dec; [34]); 0.6 ± 0.3 (Dec-Jan; [35]) |
| Serum iron (μg $dL^{-1}$) | 53.4 | 129.8 | 208.8 | 134.0 | 35.4 | 23.2-441.1 (Oct-Feb [33]) |
| Alanine transaminase P5P (U $L^{-1}$) | 61 | 49 | 29 | 13 | 38 | 46.4 ± 26.4 (Dec-Jan; [35]) |
| Alkaline phosphatase (U $L^{-1}$) | 256 | 149 | 296 | 752 | 375 | 480.8 ± 302.6 (Dec-Jan; [35]) |
| Gamma glutamyltransferase (U $L^{-1}$) | < 3 | < 3 | 5 | 4 | 8 | 7.8 ± 15.1 (Dec-Jan; [35]) |
| Aspartate transaminase P5P (U $L^{-1}$) | 61 | 44 | 58 | 30 | 47 | 46.4 ± 26.4 (Dec-Jan; [35]) |
| Cholesterol (mg $dL^{-1}$) | 197 | 180 | 389 | 374 | 296 | 368 ± 97 (Dec-Jan; [35]) |
| Potassium (mmol $L^{-1}$) | 4.3 | 3.8 | 3.9 | 4 | 3.7 | 3.6–5.8 (Oct-Dec; [34]); 5.1 ± 0.5 (Dec-Jan; [35]) |
| Magnesium (mg $dL^{-1}$) | 2.2 | 2.0 | 1.5 | 1.8 | 2.3 | 3.4–6.3 (Jan-Feb; [36]) |
| Selenium (ppm) | 1.61 | 1.64 | 1.12 | 1.14 | 1.37 | 0.52–2.26; *Harbor seal [37] |

[33] Shero et al. 2022

[34] Mellish et al. 2011

[35] Yochem et al. 2009

[36] Tryland et al. 2012

[37] Griesel et al. 2008 *Weddell seal data were unavailable; thus values are presented from another phocid (harbor seal)

higher white blood cell counts during late lactation, and both seals had higher cortisol concentrations during late summer which also aligned with general seasonal patterns in the species [38]. Similar to visual observations suggesting the animals had been apparently health upon approach, blood chemistries also appeared overall within normal limits.

### Stomach distention and parasitic impaction

Necropsy revealed similar gross presentation and lesions in both cases. The most striking findings were that the stomachs of both animals were vastly distended to ~ 3x normal size, even though Weddell seal 1 and Weddell seal 2 had vomited ~ 12 and 5 L of fluid, respectively (Fig. 1a-b). Both seals had large parasite burdens in the stomach, with roundworms deeply embedded into the mucosa

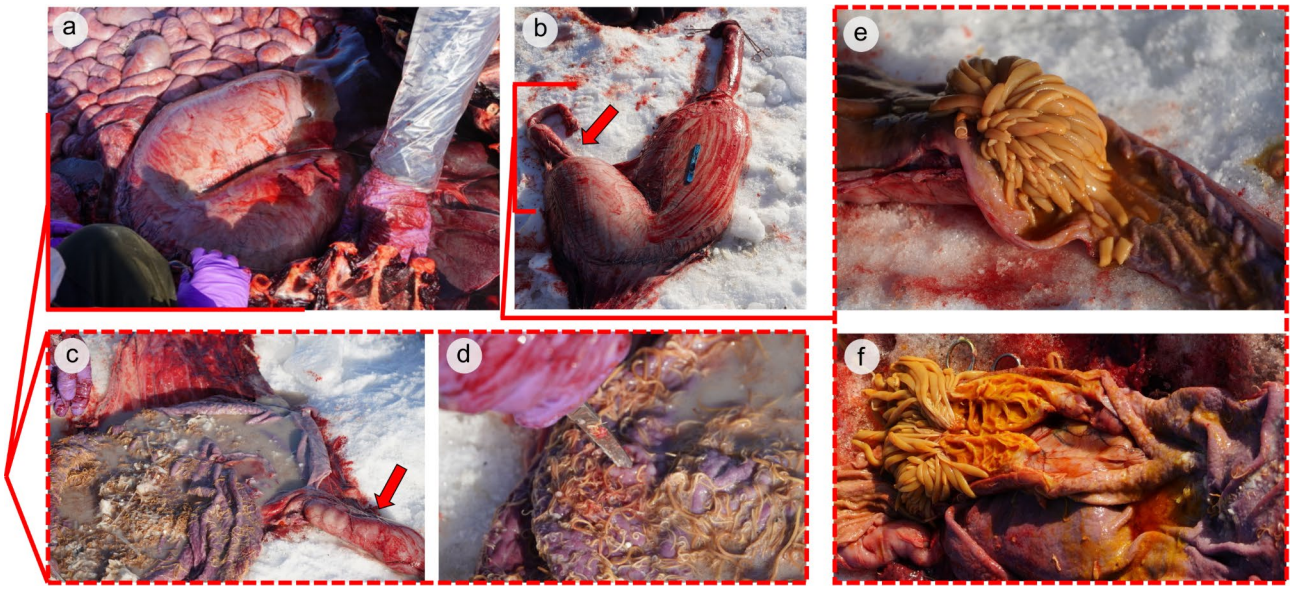


**Fig. 1** Depiction of gastric distention and large quantities of nematodes, and the cestode duodenal impaction. The distended stomach is shown (**a**) in-situ and (**b**) removed with the hard impaction (*denoted with arrow*) just past the pyloric sphincter. Within the stomach (**c**) were ingested spiny processes (*parasitic mass is again denoted with arrow)* and (**d**) a heavy parasite load embedded into the mucosa and volcanic ulcers (*dissection scissors are pointing to one ulcer*). Dissection of the hard mass revealed (**e**) an impaction of cestodes with a (**f**) thickening of the intestines and possible obstruction of the bile duct. Images taken from Weddell seal 2 (SPENO 15253) necropsy

(Fig. 1c). There were two ascaridoid gastric nematodes, morphologically identified as species of *Contraceacum*. These were molecularly identified as *Contraceacum osculatum* and *C. radiatum* based on high identity for the D1-D3 (NCBI GenBank Accession numbers: PQ556322-PQ556325) and ITS sequence regions (Accession numbers: PQ549595-PQ549598) [39]. Weddell seal 2 also had eight volcanic ulcers (Fig. 1d, ~1 cm).

Approximately 4 cm caudal to the pyloric sphincter, both animals had a hard mass in the duodenum that was ~10 cm in length. Upon dissection, it was revealed that the mass was a compaction of cestodes (i.e., tapeworms) with scolices attached to the mucosa, causing a thickening of the intestine and may have obstructed the bile duct (Fig. 1e-f). The cestodes found specifically at the duodenal impaction site had solid parenchymal bodies with calcareous corpuscles. The collected tapeworms were immature, but morphologically consistent with species in *Diphyllobothrium*. In Weddell seal 2, the digestive tract surrounding the parasitic impaction was characterized histologically. The pyloric section of the stomach displayed evidence of chronic, focally severe ulcerative gastritis, the submucosa was greatly thickened by fibrosis, with lymphocytes, and plasma cells and macrophages. Portions of the stomach and duodenum contained deeply eosinophilic necrotic debris on the surface of the ulceration (Fig. 2). In combination with blunted villi, there were large numbers of lymphocytes, plasma cells, macrophages, and karyorrhectic debris in the lamina propria. Taken together, the animal had chronic, focally severe, lymphoplasmacytic and necrotizing gastritis and duodenitis associated with parasitic infection.

The cestode mass appeared to cause a complete obstruction in Weddell seal 1 and no digesta was present in the small or large intestines; only bile-like mucus was noted. No consolidated feces were present. The parasitic mass only created a partial obstruction for Weddell seal 2 with digesta further down the digestive tract. Morphological examination also suggested that one species of Acanthocephalan from the genus *Corynosoma* was present in the intestines, and COI sequences were obtained (Accession numbers: PQ549669-PQ549670). Based on characteristics of the proboscis, it appeared most likely to be *C. hamanni* (previously known as *C. antarcticum*). However, there is no published sequence for molecular comparison of this particular species. Morphologically, this could also be *C. semerme* (but sequencing did not provide a match to this species) or *C. peposacae* (but this has not been found previously in Weddell seals). Taken together, all sections of the digestive tract contained heavy parasite loads embedded in the mucosa in both seals.

#### Oral cavity, and regurgitate in the esophagus

There were no abnormal findings in the oral cavity and pharynx in Weddell seal 1. However, Weddell seal 2's tongue appeared enlarged upon examination and was sampled for histology. There was lymphoplasmacytic perivascular diffuse reaction subjacent to the epithelium, with lymphocytic exocytosis. This was associated with

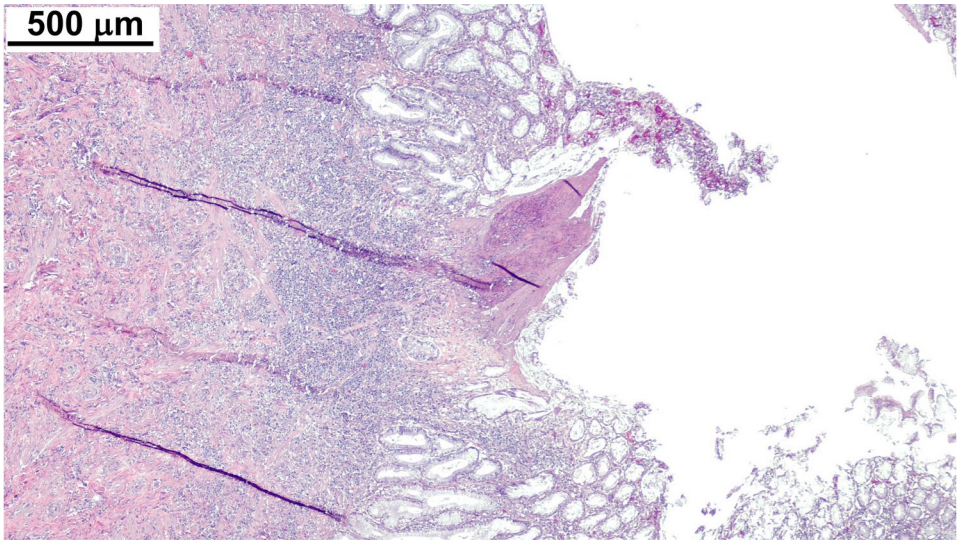


**Fig. 2** Severe duodenal ulceration at the site of cestode impaction. Histological examination revealed necrotic debris and chronic inflammation. Specimen is from Weddell seal 2 (SPENO 15253)

patches of vacuolated superficial epithelial cells and parakeratotic hyperkeratosis (Fig. S1). The cause of this lesion is unknown, however it is suggestive of a viral lesion, and the tonsil was also reactive to depleted. Weddell seal 2 had also regurgitated large portions of fish tissue, which was presumed to be from Antarctic toothfish (*Dissostichus mawsoni*) based on size. These fragments included a cranium (Fig. S2), and necropsy revealed a large pectoral fin, bones, and a number of spiny processes that had formed a large obstruction in the esophagus (Fig. S2). This is notable as Weddell seals are generally thought to behead these fish and remove spiny processes prior to consumption [40].

#### Respiratory system confirms aspiration and an additional lungworm infection

Both animals' tracheas had significant hemorrhage and ingesta that extended into the bronchi indicative of aspiration and consistent with the clinical history. This was further reflected in the histopathology from both seals showing gastrointestinal contents and mixed bacterial colonies within the bronchi.

Both seals also had chronic pneumonia due to lungworm infestation. Bronchioles were cuffed by lymphocytes, plasma cells, and eosinophils and fibrosis, and interlobular septa were expanded by fibrosis and mixed inflammatory cells including histiocytes, reactive endothelial cells, and eosinophils (Fig. 3a). Alveoli contained variable degrees of histiocytic, neutrophilic, and eosinophilic inflammation. Some lobules were largely replaced by fibrosis, exuberant inflammation with areas of plump spindle cells forming vascular clefts and lacunae (reactive endothelial cells) along with some accumulations of reactive spindeloid to rounded cells with abundant cytoplasm, consisting of histiocytes, sometimes centered on necrosis and profiles of presumed nematode larvae (i.e., parasitic granulomas) (Fig. 3b). In Weddell seal 2, nematode adults were also present in some alveoli with minimal inflammatory cell reaction (Fig. 3c). These nematodes had prominent lateral cords, polymyarian coelomyarian musculature consistent with *Parafilaroides* sp. or other metastrongylids.

#### Lymphatic system and organ tissues with possible parasite-related reactivity and lesions

Both seals had multiple reactive lymph nodes that were likely to be parasite related. For example, observed chronic pneumonia and lungworm infestation was associated with marked lymphadenitis and fibrosis of the draining lymph nodes. Submucosal glands in the lungs were often markedly distended and were hyperplastic in both cases.

Weddell seal 1 had multiple enlarged lymph nodes that were firm upon palpation. There was one enlarged structure at the dorsal aspect of the heart (Fig. S3) that was likely a mediastinal lymph node scarred to the pericardium. The architecture of the tracheobronchial and presumably hilar or mediastinal lymph nodes was largely replaced by a scirrhous reaction (Fig. 4a) and axillary lymph nodes were enlarged and discharged a milky fluid on cross section. Follicles, when present, were hyalinized and many severely depleted. In some areas, there were masses of spindeloid to rounded cells with a moderate amount of amphophilic cytoplasm and indistinct cytoplasmic borders, some with areas of central necrosis. Eosinophils and lymphocytes were also present, consistent with parasitic granulomas. In particular, there were numerous firm, enlarged and reactive lymph nodes in the

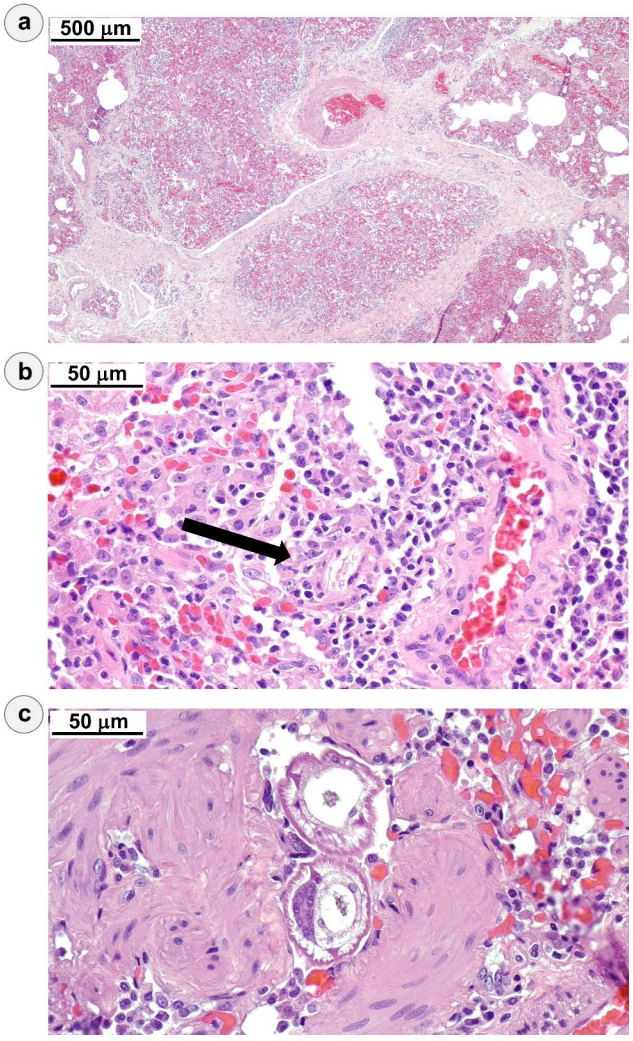


**Fig. 3** Histological specimens showing parasite related chronic septal pneumonia. (**a**) An area of exuberate reactive endothelial cells and (**b**) area of granulomatous inflammation with a central parasite larva (*arrow*) in Weddell seal 1 (SPENO 14852). (**c**) An example of an adult nematode in an affected bronchiole in Weddell seal 2 (SPENO 15253)

mesentery (Fig. 4b). The sampled mesenteric lymph node contained small lymphocytes expanding the paracortex, formation of primary and secondary follicles, and expansion of the cords with plasma cells and eosinophils, likely a reaction to the gastrointestinal parasites.

There were additional remarkable findings throughout other tissues for Weddell seal 2. In sampled skeletal muscle, fibers were swollen, hypereosinophilic and contained contraction band necrosis indicating mild acute degenerative myopathy. There was also marked congestion, extramedullary hematopoiesis and hemosiderosis of the spleen. And while the liver appeared visibly normal, palpation revealed hard masses and friable tissue. Histological analysis showed that hepatocytes contained refractile golden brown pigment granules (likely hemosiderin or ceroid/lipofuscin) indicative of mild chronic pigmentary hepatopathy and there was one eosinophilic granuloma which was likely parasite related.

## Discussion and conclusions

In this case report, the large impaction of tapeworms in the duodenum of the two Weddell seals appeared to lead to partial (Weddell seal 2) or complete (Weddell seal 1) mechanical obstruction of the pylorus. Retention of gastric contents coincided with observations that the stomach had become vastly distended and may have predisposed the animals to vomiting during anesthesia procedures. To our knowledge, this case represents the first report of heavy gastrointestinal parasitic loads being associated with anesthetic complications in marine mammals, either in the wild or in managed care.

It is noteworthy that although Weddell seals are most often studied during the pupping and breeding season (October to December, when females lose > 30% of their body mass while nursing) [21], both the cases in this report with anesthetic complications predisposed by heavy parasite infections were animals handled during the late-summer period (January to February; when females were handled to assess how well they had recuperated after rearing offspring, energetics associated with the annual pelage molt, and factors influencing the ability to initiate a new pregnancy for the next year). From the few opportunistic adult necropsies the authors have conducted at the study site, all were during the pupping and nursing period (October to December; $n = 5$; M. Shero & A. Hindle *pers. observation*) and the gastrointestinal parasite mass was not observed. Similarly, throughout the larger research program for which the incidents in this report occurred, the authors did not encounter any adult Weddell seals that went into cardiorespiratory arrest during anesthesia in October-December (using identical anesthesia protocols as described in the *Case Presentation* section; M. Shero, A. Hindle, J. Burns *pers. observation*). There were two other potential cases from previous Weddell seal research projects in Erebus Bay that may be relevant to findings in this report and similarly occurred in late-summer. In January 2011 (chemical immobilization with 1.0 mg/kg tiletamine/zolazepam IM followed by 0.5 mg/kg ketamine and 0.025 mg/kg diazepam IV) and in March 2019 (using same sedation protocols as this report) two adult female Weddell seals died during sedation after prolonged apnea despite resuscitation efforts including intubation and supplemental oxygen; however, no vomiting had occurred in either instance. In both cases, the stomach contained substantial fluid and large quantities of roundworms were embedded in the mucosa. The intestines were not examined in the former instance, but in the latter, there was a mass of tapeworms in the duodenum that was smaller than cases observed in this report (M. Shero, *pers. observation*). Further,

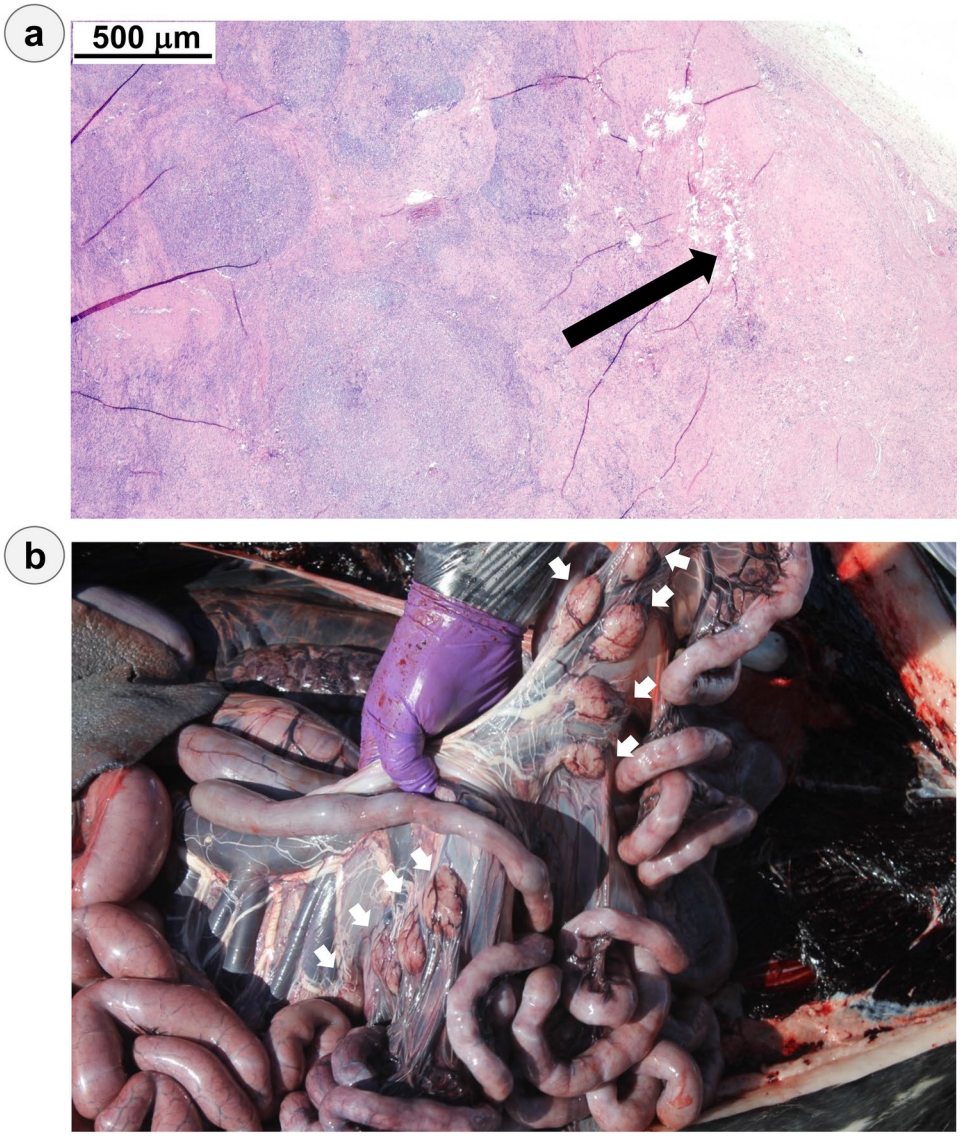


**Fig. 4** Both seals had numerous reactive lymph nodes. (**a**) The cortex was replaced with fibrosis (*arrow*) in the lymph node scarred to the pericardium in Weddell seal 1 (SPENO 14852). (**b**) This same seal also had remarkably enlarged mesenteric lymph nodes (denoted with *arrows*)

regurgitated stomach contents and roundworms can frequently be seen on the sea ice at Weddell seal haul-outs during late summer but not earlier in the year (M. Shero, *pers. observation*).

Future efforts should better characterize annual shifts in parasitic infrapopulation growth in the Weddell seal, that may reflect seasonal foraging patterns as all the intestinal parasites identified in this study have marine life cycles, with a fish, squid, or crustacean intermediate host [41–43]. Post-partum Weddell seals limit their foraging activities during the October-November lactation period, but then begin intensively foraging immediately post-weaning and regain almost 1 kg of body mass per day [33, 38, 44]. Conversely, females that fail to produce a pup (i.e., 'skip-breeders') lose mass during the mid-to-late summer period due to reductions in foraging activities. Thus, Weddell seal 1 was a skip-breeder and lost mass across the summer period, while Weddell seal 2 had reared a pup and proceeded to gain mass very rapidly. Still, both seals had similar parasite infections and almost all the observed cestodes were immature. It is possible that gastrointestinal parasite intensity increases during late summer due to re-feeding and increased intake of a predominantly marine fish diet (i.e., *Pleuragramma antarcticum*, *Dissostichus mawsoni*; [45, 46]), or seasonal shifts in prey species/abundance associated with the Ross Sea's summer phytoplankton bloom [47]. Small, immature cestodes could be the result of recent (re-)infection, or may also be due to resource limitations associated with localized 'crowding' at the site of infection [48].

The species of gastrointestinal parasites identified in these cases have been previously documented in Weddell seals [42]. Indeed, numerous studies have observed heavy infestations of cestodes in the pylorus, bile duct, and anterior portion of the intestinal lumen [5, 49, 50]. *Diphyllobothrium* (previously *Glandicephalus*) *perfoliatus* appeared to be exclusively isolated from the bile duct and duodenum in Weddell seals as was observed in these cases, while other cestodes such as *D. lashleyi* and *D. mobile* exhibited less of a pattern in their distribution throughout the intestines [49]. *D. perfoliatus* also appears to exhibit specificity for the Weddell seal host [7]. Beverley-Burton (1970) [49] found large variation in parasite loads, and 4 of 7 adult Weddell seals had *D. perfoliatus*, with up to 976 specimens (356 mature) in the anterior portion of the intestines. Similarly, Yochem et al. (2009) [35] found anisakis nematode and *Diphyllobothrid* cestode ova in 65% and 67% of Weddell seal adult and weaned pup fecal flotations, respectively. While Weddell seals have been the focus of numerous physiological and ecological studies that have required sedation since the 1960's, to our knowledge, the described anesthetic complications have not previously been observed. It remains unknown whether contemporary Weddell seal parasite prevalence and/or intensity has increased within the Erebus Bay population relative to historic observations.

Generally, intestinal cestode parasites have not been considered to be detrimental to health and body condition in Antarctic seals [5] but there has been relatively little study of their impacts. Intestinal parasites are known to elicit enterospasms causing acute obstructions [51], yet there have been no studies of in-vivo cestode activity (i.e., dynamics of contractile or locomotory movement) within pinniped hosts. In other marine mammal species, cestodes have been reported to elicit local inflammatory responses at attachment sites [43], and have been associated with decreased animal growth in juvenile Hawaiian monk seals (*Monachus schauinslandi*) [52]. There are prior reports of acanthocephalans also causing localized inflammation in marine mammals, as well as fatal perforated ulcerations stemming from Anisakid nematodes infections [53].

Individuals with gastrointestinal disease are at higher risk of gastroesophageal reflux during anesthetic procedures and postoperative vomiting [54]. Risks also increase with longer duration of anesthesia, changes in body position during anesthetic procedures, and intraabdominal procedures [55]. Anesthetic agents also often decrease lower esophageal tone, but studies have shown that premedication with acepromazine or metoclopramide reduced incidences of gastroesophageal reflux in domestic animals [55, 56]. Acepromazine has been used for its tranquilizing properties in sea otters and some cetaceans, with mixed responses as it sometimes resulted in vasodilation and subsequent hypothermia [57]. Premedication with metoclopramide as a prokinetic to increase lower esophageal sphincter tone and increase gastric motility has been successful in preventing stress-related gastroenteritis and anesthesia associated ileus in sea otters and pinnipeds [57, 58] (dose 0.15–0.2 mg/kg in phocids). Incorporation of prokinetics into anesthesia protocols for Weddell seals in late-summer may prove useful in mitigating complications from high gastrointestinal parasite loads. This would likely introduce much less risk to the animal handlings when compared to pre-emptive intubation, which the authors do not recommend in this species with the chemical immobilization protocols used in this program. Laryngeal vagal reflexes can become activated during manipulation of the airway under light to moderate sedation [16], and thus introducing and maintaining the endotracheal (ET) tube would result in lengthening sedation times and increasing the amounts of anesthetic reagent administered than would otherwise be necessary (particularly for less-invasive procedures such as blood draws, morphometrics). Moreover, it has been documented in other phocid seals that the ET tube itself can induce apnea in anesthetized animals [59] and this was similarly observed in Weddell seal 2. In early resuscitation efforts, spontaneous ventilation only resumed once the seal was extubated, at which point breathing began immediately. In either instance, manual ventilation in the field (with Ambu bags; no/little supplemental oxygen provided using demand valves) is not an effective means to support cardiopulmonary function during hypoventilation in large phocid seals [57], and thus measures that are least likely to increase incidence of apnea should be pursued.

The two individual Weddell seals in this report had underlying pathology across multiple organ tissues (liver, spleen, stomach, intestines, and lungs) including edema, congestion, necrosis and numerous reactive lymph nodes that appeared parasite-related. For example, the observed larval and adult lungworms and parasite-related pneumonia in both seals could have exacerbated anesthetic complications in these cases. *Parafilaroides* lungworms have been found across numerous pinniped species [5], and lungworms have been observed in Weddell seals but were not identified to species [60] nor was there previous discussion of the severity of the reaction. In the two cases reported here, the reaction in the Weddell seal's pulmonary lymph nodes were surprisingly severe with loss of much of the architecture to fibrosis and granulomatous inflammation in the one case examined histologically. Whether the cases reported here are more severe than previously seen remains unknown.

In summary, observations in this case report suggest that gastrointestinal parasite impaction in a marine mammal may be associated with increased morbidity

and mortality during sedation, through slowed digestion rates, severe vomiting, and risk of aspiration. These cases also draw attention to less conspicuous impacts of heavy parasite burdens to pinniped health. Although both Weddell seals in this case report appeared visibly healthy upon initial approach, observed pathology from necropsy and histological analysis revealed moderate (mesenteric) to severe (pulmonary) lymphatic inflammation, chronic pneumonia, distended stomachs, and a firm mass of cestodes within the duodenum associated with necrosis. As yet, we do not know the extent that intensity of infestations would decrease nutrient absorption efficiencies from the gut (and corresponding increases in foraging efforts that would be needed to meet equivalent energetic demands), trajectories of the infections (seasonal or inter-annual), or long-term consequences of the observed parasite infections to individual longevity and/or fitness within this marine mammal population.

**Abbreviations**

| | |
|---|---|
| SPENO | Specimen number |
| HR | Heart rate |
| IM | Intramuscular |
| IV | Intravenous |
| RR | Respiration rate |

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12917-025-04740-w.

Supplementary Material 1

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**Author contributions**

MRS, JMB, SJ conceptualized the study; all authors conducted the study; MRS, RM, CLR, ACK, JMB, SJ conducted field work; KBH performed histopathology, SN performed parasite identifications, CLR, ACK, AGH, MRS designed and performed laboratory analyses; MRS, KBH, SJ, JMB provided interpretation of results; MRS, KBH drafted the manuscript; all authors reviewed and approved the final manuscript.

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**Data availability**

All data used in the current study are available from the corresponding author on reasonable request. Genomic data have been deposited in the NCBI GenBank public repository prior to publication.

## Declarations

**Ethics approval and consent to participate**

All protocols were approved by the U.S. National Marine Fisheries Service under permits 17411 and 23273; Antarctic Conservation Act permits, and Institutional Animal Care and Use Committees.

**Consent for publication**

Not applicable.

**Competing interests**

The authors declare no competing interests.

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