Invited paper:

Proximate Aspects of Starvation-Related Morbidity and Mortality Among Young California Sea Lions (*Zalophus californianus*)

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Abstract

Dramatic fluctuations of west coast (USA) California sea lion (CSL) population sizes are well-known historical events. More recently, the episodes have involved extensive losses from pup starvation. However, beyond dams' lactation failure that results from loss of preferred prey, proximate causes of malnutrition-related death among youngest CSL remain somewhat speculative. We hypothesized that age-related differences in presentation status and postmortem pathology would clarify elements of death trajectories. Records of 494 California sea lions included young pups to late adolescents, all present in the same environment and ecology. We identified differential starvation-related impacts among young CSL populations. Rapid and extreme loss of muscle and fat body mass, and resulting emaciation, were the primary overall features of morbidity. More specifically, the death trajectory among young pups was associated more proximately with contributing fluid deficit, hypovolemia, hypoglycemia, and loss of respiratory capacity. The death trajectory among non-pups was associated more proximately with negative effects of forced diet change that resulted in severe-to-overwhelming chronic parasitism. The vast majority of death trajectories appeared to be non-reversible, either in short term or over longer term. The sentinel status of marine mammals such as CSL forewarns of potential threats to local or regional ecology. It is especially concerning if recurring similar events appear to differ causally

from historical patterns. We suggest that starvation events occurring in seemingly new patterns can signal new influences on a marine ecology.

Keywords: Starvation, Morbidity, California Sea Lions

Introduction

Periodic dramatic declines of Channel Islands (USA) California sea lion (*Zalophus californianus;* CSL) populations are long-recognized events (Cass 1985). Historical causes and influences seem to have involved (a) early human subsistence hunting; (b) commercial harvest (for oils); (c) oriental trade; (d) scientific study; and (e) exhibitions such as zoological gardens (Cass 1985). During the early 20th century, part of the CSL harvest may have been directed into pet foods, and until 1972, fishermen (and others) were permitted to kill CSL to reduce predation pressure on market fish (Cass 1985). Thus, past causes for CSL losses appear to have been diverse and thus would be difficult to compare accurately with more recent population mortality events.

California sea lions are opportunistic feeders. When their usual food sources become limited by ecology-disrupting influences, CSL respond by broadening their prey spectrum. Unfortunately, the alternate prey species may be less appropriate nutritionally (Melin et al. 2012). A report from San Miguel rookery (Channel Islands) indicated that nursing CSL frequently succumb to dams' lactation failure, secondary to fluctuating quantity and quality of prey fish that are available (McClatchie et al. 2016). Those authors hypothesized that species-specific changes in circumstantial environmental carrying capacities can favor increased presence of lower energy prey fish (McClatchie et al. 2016). For example, in the case of CSL, populations of anchovy and sardine may decline, while squid and rockfish increase, as observed by investigators (McClatchie et al. 2016).

Other investigators evaluated northeastern Pacific Ocean surface temperatures and the association with CSL body condition, nutritional state, and immune status (Banuet-Martinez et al. 2017). The investigators associated higher sea surface temperatures with poorer CSL body condition, lower average blood glucose, and potential immunopathy that was characterized as lower circulating immunoglobulins (Banuet-Martinez et al. 2017). The authors suggested that elevated sea surface temperatures produce conditions that are detrimental to CSL energy use and functional immunity.

Observations such as the foregoing suggest that terminal events among starving CSL are caused by interacting intrinsic and extrinsic influences, and are associated with external and internal anatomical alterations (Melin et al. 2012; McClatchie et al. 2016; Banuet-

Martinez et al. 2017). Our present report is associated with the Unusual Mortality Event in Southern California (USA), for the years 2013 through 2016. Data from the National Oceanic and Atmospheric Administration (NOAA) suggest that this stranding event may have totaled some 9000 CSL.
(http://www.nmfs.noaa.gov/pr/health/mmume/californiasealions2013.htm)

Specifically, we hypothesized that elements of physical evaluation and postmortem pathology of young CSL pups (age < 1 yr) would differ from older (non-pup age > 1 year) CSL that were present simultaneously in the same marine environment and ecology. Further, we hypothesized that age-related segregation of proximate causes of morbidity and mortality would allow more exact definition of primary intrinsic and extrinsic contributing influences to death trajectories.

Materials and Methods

We evaluated records of 494 stranded California sea lions (CSL) that were presented to Pacific Marine Mammal Center (PMMC) at Laguna Beach CA, USA, during 2015 and 2016. Ethical Statement: The PMMC is able to work with, and collect samples from, marine mammals due to its Stranding Agreement (SA), issued by the National Oceanic and Atmospheric Administration (NOAA). Pacific Marine Mammal Center is part of the National Stranding Network West Coast Region that is associated with NOAA's Marine Mammal Health and Stranding Response Program (MMHSRP).

Our study of these data focused on visual CSL status at presentation to PMMC, and morphology at postmortem examination. Observations that were recorded at presentation were extracted from the PMMC database, along with observations and results from gross postmortem evaluation and histopathology (Tables 1, 2). When terminal status was observed in individual CSL, appropriate humane euthanasia was determined and implemented by attending veterinary personnel. Standard practices for veterinary postmortem and histological examinations at PMMC were followed (RHE).

We first segregated data from young CSL pups as a population (n = 393) to be compared to all non-pup CSL (n = 101) in the database (Tables 1, 2). All observational data were expressed as categorical variables. The relationships between variables of interest were described by calculating appropriate percentages from the observed trait counts. Statistical comparisons were done using the two-tailed Fisher's Exact test (GraphPad Software, San Diego CA, 92108, USA) (Gerdin et al. 2016).

Examination for internal and external parasites had accompanied all

evaluations. Based on those results, additional statistical evaluations then were conducted to ascertain whether differential effects on morbidity and mortality could be identified for infections with: (a) *Corynosema spp.* with and without lower bowel hemorrhage; (b) *Contracaecum spp.* with and without gastric ulcers; and (c) *Parafilaroides spp.* with and without histological lung inflammation. These evaluations also were done using Fisher's Exact Test.

Results

Presenting observations (Tables 1, S1 a,b)

Presenting physical and morphological observations that were more prevalent among young pups, compared to non-pups, included hypothermia ($p< 0.0001$); hyperthermia ($p<0.0001$); adipose loss ($p=0.0001$); skeletal muscle loss ($p=0.0001$); and emaciation ($p= 0.0011$). Striking adipose loss (Fig 1) was found in both age groups. Other observations that were more prevalent among young pups included low respiratory rate (≤ 12/minute, $p=0.0073$); elevated heart rate ($p=0.048$); and trauma-associated cutaneous lesions ($p=0.0061$). Bloody feces or diarrhea were more prevalent among non-pups ($p<0.0001$), although few young pups were so-affected.

Initial screening of CSL upon presentation to PMMC also revealed greater prevalence of hypoglycemia among 249 young pups, compared to 42 non-pups ($p=0.0001$, not in Tables). Presenting observations that did not segregate between pups and non-pups, and occurred in at least 15% of each population, included (a) respiratory distress, cough, abnormal respiration (as a group); and (b) dehydration.

Table S1 a,b lists common potential differential diagnoses for each of the observed physical traits to aid future studies, so that important potential environment-specific contributors are not overlooked.

Postmortem evaluation (Tables 2, S2 a,b

The lungworm *Parafilaroides spp.,* with lung tissue inflammation (Fig 2), was more prevalent among non-pups ($p<0.001$), while prevalence of *Parafilaroides spp.* without inflammation did not differ between young pups and non-pups ($p>0.05$). The acanthocephalan parasite *Corynosoma spp.* was associated with colonopathy, with (Fig 3) or without hemorrhage. Both diagnoses were more prevalent among non-pups ($p<0.0001$). The ascarid gastric parasite *Contracaecum spp.* was found with or without mucosal ulceration of parasite attachment sites. Both diagnoses were more prevalent among non-pups ($p<0.0001$). Bowel tapeworm infections or impactions (associated principally with *Taenia* spp.) were more prevalent among non-pups ($p<0.0001$). Non-site-specific lymphadenopathy or lymphadenitis both

were more prevalent among non-pups ($p<0.001$).

Gross and histological nonspecific (not pathognomonic) pneumonopathy were more prevalent among young pups ($p<0.001$), as was the grouping 'hepatopathy or hepatitis' ($p=0.0014$). Observations that did not segregate between pups and non-pups, and occurred in at least 15% of each population, included the grouping 'bronchial and/or lung inflammation, aspiration, and serositis'.

Table S2 a,b lists common potential differential diagnoses for each of the observations to aid future diagnostic evaluations, so that important potential environment-specific contributors are not overlooked.

Discussion

Observational findings

Hypoglycemia in young mammals often is associated with hypothermia, hypoxia, and dehydration (Farstad 1983; Lawler and Bebiak 1986), evidence for all of which was observed frequently upon presentation of young CSL pups. Rapid, extreme loss of adipose and skeletal muscle results in severe emaciation (Fig 1). Critical physiological implications include failure of intrinsic energy metabolism and body insulation, and consequent inability to maintain core body temperature (Wilmer et al. 2006). Secondary systemic complications, such as electrolyte and acid-base disturbances, would contribute strongly to morbidity in the foregoing circumstances (Lawler 1994). With further respect to hypothermia, cardiovascular and respiratory depression are expected as usual consequences (being thus further evidence for hypoxia) (Armstrong et al. 2005), along with diminishing neurological function (Armstrong et al. 2005; Oncken et al. 2001; Danzl 2005; Todd and Powell 2009). During our initial observations, we made additionally a tentative association of greater mortality with visible respiratory rate below 12/min. Data evaluation confirmed respiratory depression as another signal for impending death among young pups.

The environmental impact of ambient temperature on emaciated CSL pups is clear from presenting observations and morbidity-mortality outcomes. Failure of homeothermic processes was an important component of complete metabolic collapse and the irreversible death trajectory. Insulating body fat is lost or does not form because of severe chronic starvation-induced malnutrition (Wilmer et al. 2006). Core metabolic heat that is generated by a healthy metabolism is lost also, particularly the more basal heat that appears to derive from soft tissue organs of the thorax and abdomen (Wilmer et al. 2006). Worsening loss of muscle mass complicates the process of morbidity further, as extensive loss of heat that is produced by active skeletal muscle removes much of the remaining starving CSL heat-generating capacity (Wilmer et al. 2006). Thus, the unshielded CSL pup body in a cold marine environment largely is devoid

of thermic response capacity, thus becoming increasingly helpless.

Degrees of hyperthermia could result from transport stress or elevated ambient temperature (in context of moving from colder water to warmer air terrestrially, as the seal strands) that could be exacerbated by pre-existing severe physiological dysregulation (Wilmer et al., 2006). That is, hyperthermia can cause CSL pup death, but probably is not a direct consequence of starvation.

Parasitism

Non-pups revealed increased prevalence of *Parafilaroides spp.* (with lung inflammation, Fig 2) and *Corynosoma spp. (*with bowel hemorrhage, Fig. 3). Multifocal-to-disseminated, severe inflammatory responses that are secondary to ruptured *Parafilaroides spp.* cystic structures suggest that swimming, diving, and feeding behavior would be impaired sufficiently to exacerbate starvation-related morbidity.

On a population basis, environment-related, forced divergence from usual food sources likely leads to levels of *Parafilaroides spp.* lung parasitism that far exceed average expected burdens, due primarily to more frequent prey-related exposures to the parasite. The latter also could lead to overwhelming stress-related suppression of immune capacity (Banuet-Martinez et al. 2017). By July of a given year, yearling sea lions are reported to have 94% prevalence of lung *Parafilaroides spp* (Grieg et al., 2005).

Non-pups and older CSL likely develop comparatively greater tolerance of exposure to a more gradually increasing parasite burden. However, superimposed diminished environmental availability of preferred prey, with unavoidable diet shifting, predisposes to morbidity and mortality associated with a more rapidly-developing parasite overburden and more extreme respiratory complications.

Histological nonspecific pneumonopathy was more prevalent among young pups. A reasonable argument could suggest that nonspecific pneumonopathy may have causes other than parasitism and should be expected at low population prevalence. This observation should not be overlooked as a potential contributor to morbidity in the presence of extreme ecology-related challenges.

When present at high density, *Corynosoma spp.* are very likely to cause morbidity and mortality from severe blood loss. It has been reported that *Corynosoma spp.* prevalence can reach 100% in yearling and adult California sea lions (Lisitsyna et al. 2018). The latter observations also support the idea that gradual parasite accumulation over time may be more tolerable than rapidly-increasing burdens in an imposed nutrient-poor environment.

Neither gastric ulceration secondary to *Contracaecum spp.* presence, nor gut tapeworm infestations, appeared to contribute strongly to population

morbidity. Also relating to population-level starvation, observed cutaneous wounds likely were caused by conspecific competition for a limited food supply.

Non-segregating observations

Pathology traits that did not segregate between young pups and non-pups (Tables 1, 2) may represent more widely-occurring pathology that can occur in stranding (or normal) CSL (Tables S1 a,b; S2 a,b). We evaluated observations that occurred in at least 15% of both populations, including elevated heart rate; 'grouped respiratory distress; cough; abnormal respiration'; and dehydration. It must be recognized that each of these observations could be related to parasite burden and/or other primary and secondary respiratory or other pathology. However, the lack of significant differences between young pups and non-pups suggests that they may be incidental observations, whether to be expected or not.

An equally viable alternative argument might be that non-segregating observations in a relatively localized population do remain consistent with complications of advanced starvation (Morley et al., 2006) that exerted its most profound effects on young pups, but had clear implications for population stability and survival at all ages. Concerning differential diagnosis, the latter ideas underscore the importance of incorporating effects of changing ecological circumstances into the evaluation process, at individual (Tables S1 a,b; S2 a,b) and population levels.

CONCLUDING THOUGHTS

The models constructed by Mc Clatchie et al. (2016) were informed by incorporating various multi-year data that included available prey species, commercial fishing (catch rates by species), and foraging ranges (with contemporaneous prey species presence) of tagged reproducing females. Pup weight was expressed as the mean gender-specific weight at age 14 weeks at San Miguel Island from 2001 to 2011. The model predictions of pup weight from dams' forage quality revealed high correlation between higher-quality forage (sardines and anchovy; Table 3) and higher pup weight estimates ($r_{\sim}$ 0.86), as compared to pup weight results associated with greater intake of squid and rockfish (McClatchie et al. 2016). Remarkably, the model predictions were supportable over 5^{o} of latitude (i.e., the effect was not localized geographically).

Time-based variability has indicated that environmental factors other than forage quality also contribute to pup weight outcomes (Melin et al. 2012; McClatchie et al. 2016; Banuet-Martinez et al. 2017). One concern is that scientifically valid controlled body weight studies in wild species in stressful environments are very difficult to design and execute. Nonetheless, suggested examples of these additional (non-forage) influences have included effects of carrying capacity

and population growth trends (McClatchie et al. 2016). Consequently, variable effects on pup morbidity and mortality are expected to occur as the outcomes of interacting stressors. The latter point seems clear in the light of our observations.

Extrinsic (environmental) pressures and population morbidity and mortality

One of the three strongest El Nino-Southern Oscillation climate events since 1950 occurred during 2014-2016 (Pacific ENSO Update 2016). Such extreme events have been more frequent generally, because of climate change (Cai et al. 2014). Among the many consequences in the California Current ecosystem was a rise in sea surface temperature (SST) that resulted in decreased phyto- and zoo-plankton (Bakun et al. 2015). This in turn led to decreased survival and recruitment among anchovy and sardine populations (Lluch-Belda et al. 1986; Deyle et al. 2013; Lindegren et al. 2013). Nursing adult female CSL depend primarily and heavily on anchovy and sardine prey to support adequate lactation. Similarly, weanling CSL require those same prey populations to learn successful foraging (Keledjian and Mesnick 2013; McClatchie et al. 2016) (Table 3).

The nutritional stress hypothesis in marine apex predators holds that a shift to prey species having suboptimal nutritional value leads to adverse physiological states (Trites and Donnelly 2003; Whitfield 2008). These effects are most dramatic in young, growing animals, as evidenced by studies in seabirds (Romano et al. 2006) and Steller sea lions (Trites and Donnelly 2003). In the CSL, the forced shift from anchovies and sardines to squid and rockfish (McClatchie et al. 2016; Robinson et al. 2018) resulted in much lower intake of lipids, protein, and energy (Table 3). Table 3 includes multiple-sourced data that demonstrate much higher content of lipid, protein, and energy from sardines and anchovy, compared to squid and rockfish. Thus, a forced diet shift to squid and rockfish is very likely to lead to nutritional deficits.

The functional relationship between environmental variability (both physical and biological) and marine mammal mortality highlights the pressing need for detailed necropsy examinations and surveillance of population health. It is our view that modeling studies of the synergistic relationship between sea surface temperature and commercial fishing (Lindegren et al., 2013) also could lead to (a) better understanding of the various drivers that are proximate to marine mammal morbidity and mortality; (b) improved CSL population management; (c) and more efficient predicting of CSL responses to climate change (Meager and Limpus 2014).

Summarily, in the foregoing context, we suggest that the death trajectory

among the *young starving* CSL pups that we evaluated was the outcome of severe metabolic collapse from rapidly-developing, severe, readily-visible disseminated loss of body adipose and muscle mass. Associated and immediately proximate metabolic effects of hypothermia, hypoglycemia, hypovolemia, dehydration, and respiratory-associated hypoxia, defined the terminal stage. On the other hand, while the death trajectory among *non-pups* also appeared to relate to similar short-term, severe loss of body mass components, the disastrous proximate parasitic consequences of forced diet change (in this population, *Parafilaroides spp.* and *Corynosema spp.*) defined the death trajectory.

It is important that we recognize the limitations imposed by the inability to identify contemporaneous healthy control subjects in the same aquatic environments. A further complication of considering such a "control" population is that voluntary relocation of CSL that had remained sufficiently healthy to relocate could bias data acquired from the remaining population. Lastly, another limitation is that little or no prior history was available from presenting CSL, just as with most wild animals.

Marine mammals, and particularly CSL, have been considered to be sentinel species that reflect marine environmental health (Bossart 2012; Lindegren et al. 2013). In this context, the morphology and pathobiology that we have described here extends our understanding of interacting effects of age, environmental characteristics and impacts, and host/parasite relationships of this top predator of the coastal marine ecosystem.

Considering these findings more broadly, we first must ask whether re-occurring events will have the same cause(s)? Secondly, will environmental associations with morbidity and mortality be more or less variable? Thus, it is very important to recognize that the details of young CSL morbidity and mortality during future events may or may not align with the present or historical outcomes. Where they do not, such events could signal a further-altered marine ecology that must be evaluated anew.

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Table (1): Physical Evaluations of 494 California Sea Lions Presented to PMMC[1] 2015-2016

	Pups	Pups	Non-Pups	Non-Pups	
	393 (N)	(%)	101 (N)	(%)	p for N
Observation					
hypothermia	221	56.2	24	23.8	<0.0001
hyperthermia	44	11.2	2	2.0	<0.0001
bloody feces or diarrhea	3	0.8	13	12.9	<0.0001
severe adipose loss	391	99.5	93	92.1	0.0001
severe skeletal muscle loss	291	74.0	45	44.6	0.0001
emaciation (severe adipose/muscle loss)	217	55.2	37	36.6	0.0011
wound/abscess/fishhook/bites/pox	169	43.0	28	27.7	0.0061
respirations ≤ 12/minute	208	52.9	38	37.6	0.0073
tachycardia	99	25.2	16	15.8	0.048
comatose/unconscious	36	9.2	16	15.8	0.067
respiratory distress/abnormal pattern/cough	106	27.0	35	34.6	0.139
ocular trauma/infection	23	5.9	10	9.9	0.177
lethargy	31	7.9	12	11.9	0.234
dehydration	96	24.4	20	19.8	0.360
epiphora	28	7.1	4	4.0	0.364
oronasal discharge/inflammation	29	7.4	10	9.9	0.410
bradycardia	34	8.7	10	9.9	0.696
seizures/trembling	58	14.8	13	12.9	0.751
unresponsive	33	8.4	8	7.9	1.000
[1]Pacific Marine Mammal Center					

Table (2): Post-mortem Evaluations of 494 California Sea Lions Presented to PMMC[1] 2015-2016

	Pups	Pups	Non-Pups	Non-Pups	
	393 (N)	(%)	101 (N)	(%)	p for N
Observation					
colonopathy (Corynosoma)	22	5.6	26	25.7	<0.0001
colonopathy, hemorrhagic (Corynosoma)	7	1.8	25	24.8	<0.0001
gastropathy, non-ulcerative (Contracaecum)	23	5.9	24	23.8	<0.0001
gastropathy, ulcerative (Contracaecum)	24	6.1	21	20.8	<0.0001
bowel tapeworms/tapeworm impaction	1	0.3	8	7.9	<0.0001
pneumonopathy, inflammatory (Parafilaroides)	80	20.4	53	52.5	<0.001
pneumonopathy, non-specific	67	17.0	15	14.9	<0.001
lymphadenopathy/lymphadenitis	8	2.0	14	13.9	<0.001
hepatopathy/hepatitis	54	13.7	3	3.0	0.0014
splenomegaly/trauma/reactive histology	2	0.5	3	3.0	0.06
tracheal hemorrhage	1	0.3	2	2.0	0.108
pleuritis	6	1.5	4	4.0	0.127
bronchial & lung inflammation/aspiration/serositis	85	21.6	16	15.8	0.216
pneumonopathy, non-inflammatory (Parafilaroides)	64	16.3	21	20.8	0.302
ascites	10	2.5	1	1.0	0.475
hyposplenism/atrophy/splenopathy	48	12.2	10	9.9	0.605
gastric ulceration	5	1.3	2	2.0	0.636
gastric foreign body, inanimate	8	2.0	3	3.0	0.704
respiratory mites	12	3.1	4	4.0	0.752
thoracic effusion/exudate/hemorrhage	20	5.1	6	5.9	0.802
gastric crustaceans	9	2.3	2	2.0	1.000
[1]Pacific Marine Mammal Center					

Table 3. Macronutrient Profiles for Four Marine Prey Fish

Macronutrient (g)/100g	Sardine[1]	Anchovy[2]	Squid[3]	Rockfish[4]
Lipids	13.8	9.5	1.3	1.6
Protein	19.2	29.0	15.9	18.7
Carbohydrate	0.0	0.0	2.6	0.0
Kilocalories	213.0	210.0	88.5	94.2

[1]https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/sardines
[2]www.precisionnutrition.com/encyclopedia/food/anchovies
[3]https://www.eatthismuch.com/food/nutrition/calamari-california-market-squid,568042/
[4]https://nutritiondata.self.com/facts/finfish-and-shellfish-products/4096/2

Table (S1a):

Table S1. Observations of 494 California Sea Lions 2015-2016	Pacific Marine Mammal Center
Physical Evaluation Observations	**Differential Diagnosis**
hypothermia[1-3]	loss of fat for insulation and energy, exposure, dehydration, organ disease (renal, heart, hypothyroidism, hypoadrenocorticism),
	sepsis, malnutrition, hypoglycemia, trauma, central nervous system disorders
hyperthermia[4,5]	environment (transport stress, heat), seizures, many infections, endocrinopathy
bloody feces/diarrhea[6,7,19]	hypoxic or acid-base damage to bowel, various infectious agents, colitis, parasitism, environmental toxicity, coagulopathy,
	immunopathy, foreign body, neoplasia, dysbiosis
adipose loss (mostly severe)	adipose loss for energy metabolism early, followed by increasing muscle loss
skeletal muscle loss (mostly severe)	adipose loss for energy metabolism early; expect less muscle loss in older seals (more body fat?)
emaciation (severe adipose and muscle loss)	severe loss of body fat and muscle
cutaneous wounds/abscesses/fishhooks/bites/pox	trauma, conspecific aggression, foreign bodies, many primary and secondary infections
respiratory depression[1,8]	≤12/minute predicts high short-term mortality in our database; many causes include severe malnutrition, sepsis, shock, hypothermia
tachycardia[9]	many causes include fright, dehydration, hyperthermia, exertion, respiratory disease, cardiac disease, acid-base disturbances, pain,
	increased blood volume
comatose/unconscious[11]	trauma, hyperthermia, sepsis, hepatic encephalopathy, hypoglycemia, severe dehydration
respiratory distress/cough/abnormal pattern[10]	cardiac/lung disease, respiratory obstruction/inflammation/infection, allergy, hemorrhage, neoplasia, foreign bodies, pain, fear
ocular trauma/infection	trauma, many infectious agents, some parasites, foreign bodies
lethargy[12]	many causes include anemia, cardiovascular/respiratory disease, hypometabolic state, metabolic collapse, hypothermia, fever, pain,
	hypoglycemia, sepsis, fluid-electrolyte imbalance, severe dehydration, immunopathy, starvation, endocrinopathy, malnutrition
dehydration	anorexia, exposure, pain, fever, environmental hyperthermia, hypothermia, transport, stranding, malnutrition,
	many metabolic diseases
epiphora[13]	trauma, congenital defects, sinusitis, rhinitis, blepharitis, conjunctivitis, anteriour uveitis, foreign body
	pneumonitis/pneumonia, conjunctivits, congenital defects, nasolacrimal duct disorders, facial nerve paralysis
ornasal discharge/inflammation[14,15]	sinusitis, rhinitis, trauma, parasitism/migrating parasites, dental damage/disease, oral ulceration/infection, focal osteomyelitis,
	pneumonitis/pneumonia, pain, uremia, foreign body, facial nerve damage
bradycardia[1,9]	metabolic/circulatory collapse, intracranial disease, shock, agonal state, advanced dehydration/hypothermia, heart disease
	central nervous system trauma or pressure increase
seizures/trembling[16-18]	epilepsy, hyperthermia, hypoglycemia, hypoxia, hypocalcemia, hyperkalemia, hyper- and hypothyroidism, polycythermia, uremia
	hepatic encephalopathy, environmental toxicity, various CNS disorders, various neuropathies, hyper- and hypoadreoncorticism
unresponsive[11]	comatose, unconscious, prolonged seizures, cardiopulmonary disease, trauma, severe sepsis, hypoglycemia,
	severe hypothermia, encephalopathy, metabolic collapse, severe dehydration

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Table (S2a):

Table S2. Observations of 494 California Sea Lions 2015-2016	Pacific Marine Mammal Center
Post-mortem Evaluation Observations	**Differential Diagnosis**
colonopathy (*Corynosema*)	
colonopathy hemorrhagic (*Corynosema*)	
gastropathy non-ulcerative (*Contracaecum*)	
gastropathy ulcerative (*Contracaecum*)	
bowel tapeworms/tapeworm impaction	
pneumonopathy, inflammation (*Parafilaroides*)	
pneumonopathy	usually a nonspecific diagnosis, often idiopathic: infectious diseases, environment-based toxicities, parasitism,
	many metabolic diseases, intrinsic cardiopulmonary dysfunction, various hypersensitivities
lymphadenopathy/lymphadenitis[1]	infection bacterial/viral/fungal, neoplasia, granulomatous or inflammatory change, leukocyte proliferation, foreign body
hepatopathy/hepatitis[2,3]	numerous infections and inflammatory disorders, some heart diseases, lipidosis, environmental toxicity, vascular disorders,
	neoplasia, hemolysis, immunopathy, endocrine diseases, pancreatitis, hypo- and hyperthyroidism, hyperadrenocorticism,
	diabetes mellitus, trauma, thermal injury
splenomegaly/trauma/reactive histology[4,5]	congestive heart failure, neoplasia, torsion, inflammation, immunopathy, post-trauma, foreign body, endocarditis, amyloidosis,
	numerous infections and parasitisms, discospondylitis, extramedullary hematopoesis
tracheal hemorrhage[6]	parasitism, infection (viral-bacterial-fungal), trauma, tracheal mass, severe non-infectious respiratory disease, hypersensitivity
	inhalant environmental toxicity
pleuritis[7-11]	numerous infectious agents and parasites especially with lung disease, vasculopathy, neoplasia, trauma, bile, chylothorax
	migrating foreign body, fibrosing (multiple causes)
bronchial/lung inflammation/aspiration/serositis[12-14]	trauma, severe pulmonary disease, coagulopathy, infection (viral-bacterial-fungal), parasitism, aspiration, hypersensitivity,
	hemorrhage
pneumonopathy, no inflamation (*Parafilaroides*)	
ascites[15,16]	liver cirrhosis, congestive heart failure, renal disease, hypoproteinemia, serum hypo-osmolality, some cases of peritonitis,
	abdominal neoplasia, pancreatitis, portal circulatory disorders, excess total body sodium and water, some cases of peritonitis,
hyposplenism/atrophy/splenopathy	hypovolemia, severe emaciation, severe malnutrition, severe hemorrhage, physiological contraction, fibrosis, shock
gastric ulceration[17]	gastric or upper GI parasites, severe stress, upper GI infection (viral, bacterial, fungal), environmental toxicity, chronic gastritis,
	foreign body, some neurologic disorders, liver or renal disease, gastric hyperacidity, shock
gastric foreign body, inanimate	
respiratory mites	
thoracic effusion/exudate/blood[18,19]	cardiopulmonary-liver-renal disease, increased vascular-lymphatic permeability, pancreatitis, trauma, serositis, various
	infections or parasites, fibrosing pleuritis, upper airway obstruction, coagulopathy, empyema, neoplasia, low oncotic pressure,
	hypoproteinemia-hypoalbuminemia, hypervolemia, hypertension, chylothorax
gastric crustaceans	

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Fig (1): Postmortem ventral subcutaneous view of deceased California sea lion pup showing adipose depletion (blue arrows) and skeletal muscle loss (green arrows)

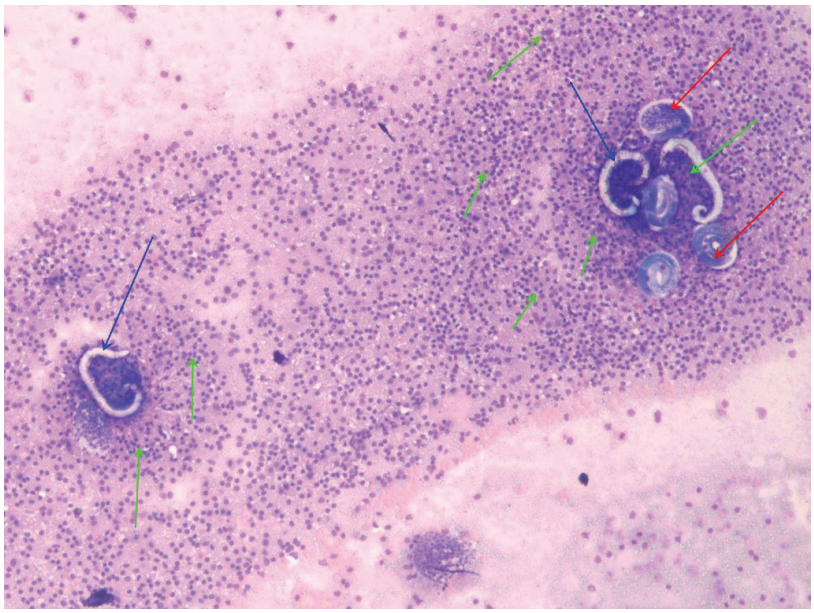

Fig (2): Photomicrograph (400x) of California sea lion pup lung showing Parafilaroides ova (red arrows) with free larvae (blue arrows) and surrounding inflammatory response (green arrows)

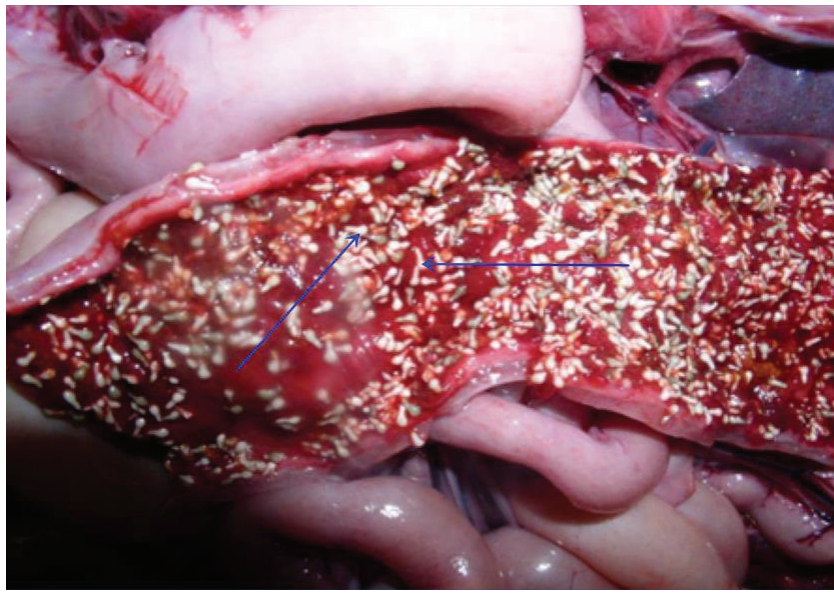

Fig (3): Photograph of California sea lion pup showing *Corynosema* attached to hemorrhagic colon wall in large numbers (blue arrows show individual parasites)