A word from the editor

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The technical problems involved in the re-publication of the treatise were mastered by Konstantin Kambach (Inter-Research). Unavoidably, the print quality of the final product is somewhat inferior to the original.

Otto Kinne
Oldendorf/Luhe
21.01.2009

DISEASES OF MARINE ANIMALS

Volume I GENERAL ASPECTS, PROTOZOA TO GASTROPODA

Volume II INTRODUCTION, BIVALVIA TO SCAPHOPODA

Volume III INTRODUCTION, CEPHALOPODA, CRUSTACEA, etc. TO UROCHORDATA

Volume IV INTRODUCTION, PISCES, REPTILIA, AVES, MAMMALIA

DISEASES OF MARINE ANIMALS

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VOLUME IV, PART 2
Introduction, Reptilia, Aves, Mammalia

1985
BIOLOGISCHE ANSTALT HELGOLAND
Hamburg, Federal Republic of Germany

ISBN 3-9800818-4-2

Printed in Federal Republic of Germany by Westholsteinische Verlagsdruckerei Boyens & Co., D-2240 Heide

FOREWORD

Volume IV* of 'Diseases of Marine Animals' summarizes and evaluates the present status of our knowledge on the diseases of those Pisces, Reptilia, Aves and Mammalia that live permanently or temporarily in marine or brackish habitats. The volume centers on biotic diseases, proliferative disorders, structural abnormalities, and impairments due to environmental stressors. While Volume IV concentrates on marine forms, it includes data on species migrating between marine, brackish and limnic habitats. Wherever considered desirable for completeness and comparison, disease phenomena in fresh-water living relatives are also referred to.

Volume IV consists of 2 parts containing the following chapters:

Part 1

Introduction to Volume IV, Part 1: Pisces
Chapter 1: Diseases of Pisces

Part 2

Introduction to Volume IV, Part 2: Reptilia, Aves and Mammalia
Chapter 2: Diseases of Reptilia
Chapter 3: Diseases of Aves (Marine Birds)
Chapter 4: Diseases of Mammalia: Carnivora
Chapter 5: Diseases of Mammalia: Pinnipedia
Chapter 6: Diseases of Mammalia: Sirenia
Chapter 7: Diseases of Mammalia: Cetacea

I could not avoid having Volume IV appear before Volume III. With all contributions to Volume IV in my hands and knowing that the preparation of Volume III would take another year, I decided not to let the Volume IV contributions age on my desk but to make them available to the scientific community as fresh and early as possible — even at the risk of being critized for breaking the fundamental rule of chronological sequence.

There is more knowledge available on the diseases of fishes than of any other group of aquatic organisms. This fact is mirrowed by the large size of Chapter 1.

Although drawing on different sources, talents and disciplines, Volume IV maintains, as much as possible, the principles of organization and perspectives outlined in the introduction to Volume I.

I acknowledge with pleasure and gratitude the assistance and support received from all contributors to this tome; from Helga Witt, Seetha Murthy and Nancy Norris-Bauer in matters of technical editing; from Martin Söhl and Frau Schritt in the search for literature information.

Oldendorf/Luhe, September 16, 1984 — O. KINNE

* For technical reasons Volume IV will be published before Volume III.

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INTRODUCTION, REPTILIA, AVES, MAMMALIA

INTRODUCTION TO VOLUME IV, PART 2: REPTILIA, AVES, MAMMALIA

O. KINNE

GENERAL ASPECTS

The second and final part of Volume IV* of 'Diseases of Marine Animals', this tome concludes the first attempt in history at documenting and evaluating comprehensively all essential information produced on disease phenomena of the animals inhabiting our oceans and coastal waters.

The information presently available on the diseases of the animals covered in this book — marine reptiles, birds, and mammals (sea otter, pinnipeds, sirenians and whales) — is widely scattered and often restricted to annecdotal reports or superficial descriptions. Much of it was obtained from captive or dead (stranded) individuals. Systematic in-depth studies are rare, and — where performed — of recent date. In fact, we have very little solid knowledge about the diseases and the actual health status of free-living marine reptiles, birds and mammals. This statement includes potential effects of man-made pollutants.

World-wide, much more financial support and a significant increase in pertinent scientific manpower are necessary in order to produce the amount and kind of knowledge needed for sound assessments of (i) the present health status of marine animals; (ii) its bearing on ecological dynamics; (iii) the effects of human impact on and interference with oceans and coastal waters; (iv) the needs and means for protecting sea life (wild-life conservation); (v) the significance of the diseases studied for veterinary and human medicine.

Pain and death rank high among the motivating forces that affect our thinking, culture, religion and art. Pain and premature death result primarily from man's own actions and from diseases (some of man's actions intended to cause pain and death may *per se* be considered a disease). Intensified and deepened disease research conducted with a broad perspective — as has been outlined and emphasized in this treatise — holds the key to a better future for both nature and mankind.

SUMMARIES OF CHAPTER CONTENTS

Comments on Diseases of Reptilia

Very few reptiles inhabit marine waters: some turtles, snakes and the Galapagos iguana. Most of the scanty reports on the diseases of marine reptiles due to micro-

* As already pointed out in the Foreword, for technical reasons Volume III (Cephalopoda, Crustacea etc. to Urochordata) will be published after Volume IV.

organisms stem from the green turtle *Chelonia mydas*, and here detailed information is restricted to individuals maintained in culture. An economically valuable reptile, the green turtle has received concerted attention from ecologists, pathologists and conservationists.

Commercially reared *Chelonia mydas* hatchlings frequently develop grey-patch disease (GPD). This disease manifests itself in 2 different forms: spreading plaques of superficial epidermal macerations which tend to disappear towards the end of the first year, and non-spreading papular lesions which may persist throughout the turtle's life. Apparently caused by a herpes-type virus, GPD can be transmitted experimentally by scratch inoculation and by injection with (bacteria-free) preparations of viral particles. Older turtles appear to develop antibodies against the viral agent.

GPD is considered to be a stress-caused disease. Typical stress-pathogens, herpesviruses are apparently forced to remain latent or dormant under circumstances which favour optimal host development, but tend to turn virulent in hosts exposed to excessive stress (e.g., extreme intensities of environmental factors, maintenance in captive water bodies, crowding) and/or nutritional deficiencies. Consequently, in captive green turtles, the most effective measures for disease prevention and therapy appear to be stringent quarantine procedures, optimal culture conditions, avoidance of overcrowding, proper nutrition, and careful water-quality management. Thus far, GPD has not been reported from free-living green turtles.

No sound information is available on diseases of free-living marine reptiles caused by bacteria. In sharp contrast, diseases due to bacteria abound in reptiles maintained in captivity. Here, more than a dozen bacterial species have been identified as potential pathogens. There are reports on skin lesions, scarring, abscesses and ulcers (in *Chelonia mydas*), superficial lesions (in *Caretta caretta*), and 'mouthrot' in the sea snake *Enhydrina schistosa*. Captive *C. mydas* have also been shown to suffer from tuberculosis and intestinal lesions.

Although fungi (e.g., members of the genera *Sporotrichium, Cladosporium, Paecilomyces*) have been isolated from marine reptiles (notably *Chelonia mydas*), no definitive information is available on fungi-caused diseases of reptiles from the marine environment, except for a case of mycotic pneumonia diagnosed in captive juvenile *C. mydas*.

Diseases of marine reptiles caused by protistans have received very little attention. Our pertinent knowledge is practically nil, except for brief reports on amoebiasis (believed to be due to *Entamoeba invadens*) and on coccidiosis (caused by *Caryospora cheloniae*) in the green turtle *Chelonia mydas*.

Among the metazoan disease agents of marine reptiles, digenean trematodes feature a species-rich spectrum. From *Chelonia mydas* alone members of more than 50 trematode species have been collected. While a fair number of parasitic infestations — especially of intestinal tract, stomach, gall bladder and blood system — have become known from marine turtles and snakes, the information at hand is largely restricted to reports of parasite taxonomy and 'microdistribution' (Vol. I, p. 38). Little is known about parasite life cycles, and next to nothing about details of agent – host interactions or disease phenomena *sensu stricto*. Examples of trematode parasites are *Lophotaspis vallei*, found in the oesophagus and stomach of *Caretta caretta*; members of the digenean genera *Pronocephalus, Cricocephalus, Diaschistorchis, Pleurogonius, Medioporus* and *Pyelosomum*, most of which have been reported from the intestinal tracts

or stomachs of turtles; Paramphistomatidae and Brachycoeliidae, which are widely distributed intestinal turtle parasites; Plagiorchidae, Rhytidodidae and Auridistomatidae, which live in the intestine, gallbladder and/or urinary bladder of turtles; and a variety of blood flukes (of 20 species of spirorchids, 12 are known to parasitize *Chelonia mydas;* 5, *Caretta caretta;* 2, *Eretmochelys 'squamosa';* and 1, *C. japonica).*

Most digenians reported from sea snakes are hemiurids — widely distributed fish parasites. The hemiurid *Lecithochirium dillanei* was found in the alimentary tract of an unidentified sea snake; *Sterrhurus carpentariae,* in the stomach of *Lapemis hardwicki; Hydrophitrema gigantea,* in the tracheal lungs of several sea snakes *(Hydrophis elegans, H. cyanocinctus, Kolpophis annendalei, Aipysurus laevis, Enhydrina valakadyen, Microcephalopsis gracilis.)*

While the Galapagos marine iguana *Amblyrhynchus cristatus* shares trematode parasites of the family Pronocephalidae with chelonian turtles, the sea snakes have no members of trematode families in common with other marine reptiles.

There are no definite reports regarding cestode parasites of marine reptiles, but endoparasitic nematodes of marine turtles have been known for a long time. The ascaridoid *Sulcascaris sulcata* has received appreciable attention. A cosmopolitan, this nematode has been recovered from the stomachs of turtles *(Chelonia mydas, Caretta caretta).* Its eggs pass out with faeces and sink to the sea bottom; the larvae develop in mollusc larvae and, at a length of ca 5 mm, infest the final host (a turtle) *via* ingestion; in the host's stomach they mature in about 5 months, reaching up to some 10 cm in length and 0.1 to 0.2 cm in width. Heavy infestations may lead to lesions, chronic gastritis and ulceration. Other nematodes recovered from the intestinal tracts or stomachs of chelonians *(Caretta caretta)* include *Cucullanus carettae, Kathlania leptura* and *Echinocephalus* sp. Several species of the ascaridoid genera *Goezia* and *Paraheterotyphlum* were found in stomachs of sea snakes such as *Enhydrina schistosa, Lapemis hardwicki, Hydrophis ornatus, H. elegans* and *H. caerulescens.*

Hirudineans may infest marine turtles at high levels of incidence and intensity, and may cause severe damage (e.g., excavations and local destructions of the plastron, removal of blood, transfer of blood parasites, skin and eye destructions). Most of the information available pertains to species of *Ozobranchus* which attach their eggs to the external surfaces of the host, while adults inhabit the corners of the mouth, tissue folds between carapace and plastron, external cloacal region, as well as flipper surface and neck. Among the treatments of leech infestation tested thus far, addition of copper sulphate to the water turned out to be most effective.

Acarina species live on amphibious sea snakes and the Galapagos marine iguana. While the mite *Vatacarus ipoides* may abound in air sacs and respiratory passages of members of the sea snake genus *Laticauda* (up to 100 % infestation; up to 60 mites host^{-1}) no respiratory impairment appeares to prevail, and no gross lesions could be observed in the sea snakes upon autopsy.

Among the crustaceans, species of cirripeds, copepods and decapods associate with chelonians; cirripeds also with sea snakes. The role of these crustaceans appear to range from phoronts over mutualists (most cases) to true (ecto-)parasites (rare). Forms such as *Chelonibia caretta, C. ramosa* and *Tubicinella cheloniae* qualify as potential pathogens.

Tumours and other abnormalities have been reported from marine repti-

lians only in very few cases. Adenoma, papilloma, carcinoma and rhabdomyoma occurred in 5 chelonian species. The papillomas, for example, were located on tail, flippers, axils, neck, eyelids and corneas. In several cases, tumors supported trematodes and leeches. The rare reports of structural abnormalities (e.g., partially or totally lacking flippers of turtles) seem to have been caused by predator (shark) attacks. Other abnormalities in chelonians include albinism, abnormal curvature of the spine and a swayback condition.

Comments on Diseases of Aves (Marine Birds)

While the ecological impact of the few truly marine bird species (i.e., birds directly participating in the turnover of energy and matter in marine habitats) may be considerable, their diseases have received little attention.

Birds inhabiting the free oceans may be expected to suffer less from biotic diseases, especially those due to microbial and protozoan agents, than those inhabiting coastal areas. This may, to some extent, also pertain to diseases caused by metazoan parasites. However, similar to mammals, high-sea birds appear to have their own indigenous helminth fauna (mainly specialized nematodes and cestodes). In addition, they may act as carriers of non-avian parasites.

In contrast, coastal birds exhibit numerous biotic diseases. The situation here is comparable to that in continental (inland) birds. Coastal birds suffer from diseases due to viruses, bacteria, fungi, protistans, and a large variety of metazoan parasites, notably helminths. Thus, single individuals of ducks, oyster catchers, larids or of other respresentatives of the coastal avian fauna may harbor from hundreds to many thousands of helminths. The life cycles of coastal-bird parasites require much more attention from researchers.

Life-cycle stages of coastal birds may significantly affect other living components of coastal ecosystems. In fact, large coastal bird populations, such as develop in man-protected bird sanctuaries, may turn their habitats into real 'épidémiotopes', i.e., into areas of mass development of parasites, resulting in devastating effects on local invertebrates and fishes which share life-cycle stages of avian parasites. Lauckner concludes (p. 637) that the ecological impact of bird trematodes on various levels of coastal ecosystems may by far exceed the potential damage to the fisheries due to bird predation. He calls for a critical re-evaluation of the role of birds as carriers of parasites harmful to marine life and for a reconsideration of the present concept of coastal-bird protection.

Comments on Diseases of Mammalia: Carnivora

The sea otter *Enhydra lutris,* the only representative of carnivores in the marine environment, has received much attention from ecologists and ethologists. However, research on sea-otter diseases has been the focal point of research in only very few instances, and has a relatively short history.

While there are no definitive reports on virus-caused diseases in *Enhydra lutris,* bacteria have been shown, or are suspected, to produce disease conditions such as haemorrhagic gastrointestinal enteritis and infections of bone and paws. Among the fungi, *Coccidioides immitis* may cause coccidioidomycosis. No diseases of sea otters due to protistans have become known.

Metazoan disease agents reported from sea otters include 5 species of digenetic trematodes, 2 (possibly 4) species of cestodes, the nematode *Pseudoterranova (Phocanema) decipiens*, at least 6 species of acanthocephalans, and the nasal mite *Halarachne miroungae*. The very limited information at hand suggests relatively low rates of helminth infestations and a remarkable lack of ectoparasites.

Comments on Diseases of Mammalia: Pinnipedia

Pinnipedia suffer from a number of diseases known from their terrestrial relatives (e.g., swine, dog), thus indirectly documenting closely related evolutionary pathways of the hosts and agents involved. Our present knowledge of the diseases of pinnipeds is young, limited, and superficial. Most of the more definitive information on negative deviations from the normal state is related to conditions induced by man: environmental pollution and maintenance in captivity (i.e., in zoos, oceanaria, circuses).

The intensity of pollution impact tends to increase in toplevel carnivores such as pinnipeds far beyond average levels *via* contaminant accumulation along the food chain. Especially technical organic chemicals, pesticides, and heavy metals can cause a variety of disease phenomena, such as metabolic disturbances and a reduction in the reproductive potential, with the overall result of diminished host defence and increased host morbidity and mortality. The effects of man-made pollution in the marine environment have been reviewed comprehensively in 'Marine Ecology' (Kinne, 1984a, b*).

Maintenance of captive pinnipeds — and here economic even more than scientific aspects — has stimulated disease research and significantly enriched our knowledge on husbandry, medical care and environmental requirements of pinnipeds and, in fact, of marine mammals in general (Kinne, 1977**).

There are only a few reports on virus-caused diseases in pinnipeds, and these are of recent date. The agents involved are pox virus, calciviruses and adenovirus.

Seal-pox virus disease — externally recognizable by raised nodular skin lesions — is usually not fatal. No specific therapy is available; in captivity, optimization of environmental and nutritive conditions (i.e., maximum support of host defence) remains the major method for reducing negative effects of agent virulence.

Among the different serotypes of calciviruses, San Miguel Sea Lion Virus (SMSV) has received most attention. Indistinguishable from the virus causing vesicular exanthema in swine (VESV), the endemicity of SMSV in herds of California sea lions and northern fur seals suggest that VES outbreaks in domestic swine may have a marine source. SMSV has also been detected in prey fish of pinnipeds. In pinnipeds, SMSV causes pustular lesions and, possibly, abortion.

Acute adenoviral hepatitis was reported from *Zalophus californianus*, both agent characteristics and disease etiology being similar to cases of hepatitis in dogs. Attempts to isolate the agent failed and the source of the infection remains unknown.

A large number of pinniped diseases — involving respiratory tract, eyes, ears, teeth, digestive tract, and possibly leading to fatal systemic infections — are caused by bac-

* Kinne, O. (Ed.) (1984a). *Marine Ecology*, Vol. V, Ocean Management, Part 3, Wiley, Chichester
* Kinne, O. (Ed.) (1984b). *Marine Ecology*, Vol. V, Ocean Management, Part 4, Wiley, Chichester
** Kinne, O. (1977). Cultivation of animals. Research cultivation. In O. Kinne (Ed.), *Marine Ecology*, Vol. III, Cultivation, Part 2. Wiley, Chichester, pp. 579–1293

teria. Bacterial agents frequently reported from dermal wounds and abscesses include species of *Staphylococcus, Streptococcus, Pseudomonas, Aeromonas, Proteus* and *Corynebacterium*. Subdermal abscesses have been shown to contain *Escherichia coli, Acinetobacter parapertussis, Alcaligenes faecalis, Micrococcus* sp., *Staphylococcus* sp., and *Streptococcus* sp. *E. coli, Citrobacter* sp., *Corynebacterium pyogenes* and *Micrococcus* sp. were isolated from deep abscesses in free-living California sea lions.

Bacterial infections of respiratory organs can cause severe diseases and significant mortality in wild and captive pinnipeds. Numerous pinnipeds, especially captive ones, die from pneumonia.

Bacterial agents isolated from the lungs of pneumonia-stricken California sea lions included *Escherichia coli, Klebsiella pneumoniae, Serratia* sp., *Salmonella* sp., *Alkalescens dispar, Pseudomonas aeruginosa, Pseudomonas* sp., *Citrobacter* sp., *Acinetobacter woffi, Micrococcus* sp., *Staphylococcus aureus* and *Staphylococcus* sp. Typical symptoms of pneumonia are coughing, nasal discharge, increased respiratory rate, lung sounds and elevated rectal temperature. Application of antibiotics has been successful in early phases of the disease.

Bacterial infections of gastrointestinal areas are common in pinnipeds (e.g., salmonellosis, enteritis, diarrhoea, haemorrhagic enterocolitis, enterotoxaemia). Causative agents include various serotypes of Salmonellae, *Escherichia coli, Streptococcus faecalis,* and *Edwardsiella tarda.*

A common disease of free-living pinnipeds is botulism, a neuroparalytic condition caused by toxins associated with *Clostridium botulinum.* Similar clinical signs, collectively referred to as 'otariid ataxia' (paralysis, staggering movements, extension of head when lying down) result from thiamine deficiency (therapy: thiamine injection), hypoglycaemia (therapy: glucose injection) and enterotoxaemia (therapy: *C. perfringens* antitoxin injection).

Severe-to-fatal myositis of *Zalophus californianus* has been shown to be due to *Clostridium perfringens* which may also infect other pinnipeds and possibly be involved in other diseases. Similarly, fatal effects may result from pasteurellosis (acute haemorrhagic septicaemia, necrotic peritonitis), characterized by anorexia, elevated body temperature, and dyspnoea, and largely restricted to captive individuals.

Further diseases of pinnipeds caused by bacteria include *Erysipelothrix* infections (p. 702), controllable in captivity *via* vaccination; leptospirosis (p. 703), leading to premature parturition (abortion) and considered to be dangerous to man; actinomycoses (p. 705), due to fungus-like bacteria and in some cases of considerable medical concern.

In contrast to bacteria, fungi and protozoans have been shown to be the cause of disease in pinnipeds only in rare cases. Examples of fungal agents are species of *Candida, Coccidioides, Blastomyces, Mucormyces, Histoplasma,* and *Microsporum.* These agents damage skin, mucous membranes and lung, and produce systemic infections. Among the protistans, only Coccidia (species of *Eimeria, Sarcocystis, Toxoplasma*) have thus far been found to cause severe impairment (e.g., diarrhoea, colitis, toxoplasmosis, necrosis of a variety of internal organs) and death.

Metazoans constitute a major source of disease in Pinnipedia. Of the main taxa involved, trematodes are represented only by a moderate number of species; cestodes, by a species-rich and diverse fauna; nematodes, by at least 5 superfamilies with numerous species that may cause heavy damage; acanthocephalans, by some 16 species; insects, by 8

species of sucking lice; Acari, by several species of mites infesting nose, lungs and other host sites; and Cirripedia, by 2 species of *Lepas* attaching themselves to the skin, apparently without causing severe harm.

Trematoda inhabit internal organs, especially intestine, bile duct and liver. Most trematodes associated with pinnipeds belong to the Heterophyidae, Opisthorchiidae and Campulidae. Life-cycles of the Heterophyidae are largely unknown. These trematodes appear to cause limited, if any, detrimental host effects. Life cycles and host effects of opisthorchiids are not known. Campulids may infest pinnipeds at high levels of incidence and intensity; they produce obvious damage (e.g., biliary fibrosis, hepatitis, pericholangitis, bileduct hyperplasia), their life cycles remain to be explored.

Cestoda – largely members of the genus *Diphyllobothrium* – have been reported from the intestines of a considerable number of pinniped species. They seem to be rather unspecific in their choice of final host and may infest one host individual in numbers of hundreds or thousands. Life cycles and pathogenicity require critical attention; damage to hosts appears to be largely restricted to passive obstruction, irritation, inflammation, energy depletion and release of potentially toxic substances.

Nematoda inhabit primarily stomach, lung or heart. They are the most dangerous and deleterious metazoans known to parasitize pinnipeds. Stomachworms produce gastritis, enteritis, diarrhoea, dehydration, anaemia and a variety of toxic effects (for therapeutic measures consult p. 733). Lungworms inhabit bronchioli, alveoli and bronchi; some of them may cause severe-to-lethal damage (e.g., bronchial obstruction, support of microbial infection, anorexia, dyspnoea, vasculitis, bronchopneumonia). Heartworms parasitize heart, blood system, and other organs of numerous pinnipeds; they may produce a variety of structural and functional disorders, not only in the circulatory system but also in other organs such as liver, lungs, kidneys, and spleen. For therapy measures consult Chapter 5. While the life cycles of several nematodes have received appreciable attention, much more research is necessary before we can present a detailed account of the ecological and pathological aspects of nematode-pinniped associations.

Acanthocephala have been collected from most pinniped species — notably members of the acanthocephalan genera *Corynosoma* and *Bolbosoma*. With the exception of *C. strumosum* and *C. semerme*, life cycles are unknown. Assessments of acanthocephalan pathogenicity are equivocal; however, massive infestation and large parasites in small hosts may cause severe damage (e.g., necrosis of the intestinal wall).

Insecta are represented among the pinniped associates by sucking lice. These may inhabit a very high percentage of a given pinniped population at the rate of several-to-hundreds of lice per host individual. The lice feed while their host is at sea and reproduce while it is ashore. The degree of infestation may be a function of host condition. Heavy lice infestations cause serious irritation, anaemia and, in extreme cases, appear to contribute to premature death. Pediculosis is a problem in most free-living pinnipeds. In captivity, it can be controlled easily by application of insecticides (chlorinated hydrocarbons). Lice are important vectors of a number of dangerous disease agents affecting homeothermal animals.

Acarina or mites have caused health problems in feral and, especially, captive pinnipeds. Members of the family Halarachnidae cause nasal infestations and pulmonary acariasis; members of Sarcoptidae and Demodicidae, skin problems (mange). 'Nasal mites' reside in the nasopharyngeal region; they may cause sneezing, coughing, mucosal erosion,

inflammatory oedema, metaplasia of the epithelium, and ulceration. 'Lung mites' inhabit respiratory organs (bronchi, trachea, lung). Diagnostic cues of pulmonary acariasis are sneezing, productive coughing, mouth odor and tenacious sputum; diseased individuals show functional and structural disorders (e.g., excessive exudate, occlusion of bronchi and bronchioles, extensive alveolar collapse, distension of lymphatic vessels in tracheal and bronchial walls, asphyxiation); heavy infestation may be fatal. No specific therapy is available against nasal and pulmonary mite infestation. Effective treatment of mange-like lesions (demodicosis) has been achieved by applying 'ronnel', benzyl benzoate and gamma isomer of benzene hexachloride (p. 765). Cirripedia-pinniped associations, qualify, in essence, as cases of phoresis (Vol. I, p. 19). Cirripeds attach themselves to the external body surface (guard hair, skin of the naked ear or of the dorsal surface). Normal intensities of such epizoite settlement do not seem to cause demonstrable harm to the hosts involved.

Neoplasia have been reported from pinnipeds only in a few cases (Table 5–8, p. 768). In-depth studies are lacking.

Structural abnormalities appear to be extremely rare. The information available reflects incidental findings. No systematic study has, apparently, been performed.

Comments on Diseases of Mammalia: Sirenia

Scattered, anecdotal and of insufficient depth, the knowledge available on the diseases of Sirenia is inadequate even for vague assessments. While assumed to exist, disease-causing viruses and bacteria have not yet been definitely documented, and only 1 fungus *(Epidermophyton floccosum)* has been shown to cause erosions of nose, face, flippers and tail; 3 other fungi appear to have been involved in skin disorders. No reports were available on diseases due to protistans.

Among the metazoans, only a few monostome trematodes have been shown to inhabit sirenians — mainly intestinal tract and stomach. Their life cycles, as well as details of agent/host relations, have remained unknown. Additional host sites inhabited by trematodes include nose, Eustachian tubes and upper lip. Of the nematodes, only *Paradujardinia halicoris* is known to parasitize sirenians (intestine). Crustacean associates of sirenians — copepods and cirripeds — appear to be restricted to external body surfaces.

Comments on Diseases of Mammalia: Cetacea

Cetaceans — Odontocetes and Mysticetes — evolved from land dwelling ancestors (related to cattle, sheep, camels). With these groups they share several diseases. While little information is at hand on the diseases of Mysticetes, the greater availability for observation and research of the smaller Odontocetes (dolphins, porpoises) has yielded some insights into disease phenomena of both captive and wild representatives. Captive Odontocetes are confronted with radically modified environmental conditions; they often suffer from stress syndromes, and tend to encounter much higher densities and a wider spectrum of potential disease agents (including highly resistant pathogens of human origin) than their free-living counterparts (for details consult Kinne, 1977, e.g. p. 1123*). Hence,

* Kinne, O. (1977). Cultivation of animals. Research cultivation. In O. Kinne (Ed.), *Marine Ecology*, Vol. III, Cultivation, Part 2. Wiley, Chichester, pp. 579–1293.

information on disease phenomena obtained from captive whales cannot be extrapolated to the situation in natural habitats without critical examination. Especially in regard to intimate associations between microorganisms and whales, agent/host interactions require much more attention before definitive statements on disease phenomena in feral populations can be made.

Viruses have been shown or suspected to cause lesions (skin lesions, dolphin pox, tattoos) and hepatitis in Odontocetes, and may possibly be involved in a variety of diseases of Mysticetes.

Bacterial disease agents appear to represent a major source of disease in cetaceans, especially in captivity. Among the infections reported are those of integument, respiratory and genito-urinary systems, and blow hole. Bacteria may also be involved in the development of septicemia, mellioidiosis and abscesses.

Wild, stranded and captive cetaceans have been reported to suffer from systemic disease due to fungi. Candidosis, a primary or secondary infection usually involving the fungus *Candida albicans,* is characterized by a large variety of lesions (e.g., esophageal, intestinal, cutaneous) and may lead to anorexia, head shaking and abdominal cramping. In captive cetaceans candidosis appears to be supported by the effects of chlorine; it can be treated by Ketoconazole application (p. 812). Lobomycosis (Lobo's disease), a skin infection caused by the fungus *Loboa loboi,* leads to white cutaneous patches or bumps, and may ultimately cause loss of mobility and probably death; it has been treated successfully by Miconazole application (p. 813).

Possibly due to lack of pertinent research (rather than to absence of malady) no definitive reports on cetacean diseases due to protozoans are available.

A considerable number of metazoan agents, notably helminths, can cause diseases in cetaceans. Nine digenean trematode species of the genus *Nasitrema* infect the head region of small Odontocetes (Eustachian tube, air sinuses, lung, brain). *Nasitrema* infestations in *Tursiops truncatus* have been treated successfully with Bithional® (p. 819). Members of 10 digenean genera inhabit the digestive tract of cetaceans; their pathological potential remains to be investigated. Digeneans invading internal organs are largely restricted to liver, pancreas or hepatopancreatic duct (11 genera); in various cetacean populations they have been shown to cause diseases such as cholangitis, chronic hepatitis, fibrosis, chronic inflammation, and chronic pancreatitis. Strategies of transmission remain to be explored. Among the cestodes, members of the Tetrabothriidae, Diphyllobothriidae and Phyllobothriidae invade cetaceans. Only a few of these tapeworms appear to cause disease. Parasite life cycles, effects on hosts and therapy measures await investigation.

Numerous nematodes, representing 3 families (Pseudaliidae, Heterocheilidae, Crassicaudidae) inhabit representatives of both Odontocetes and Mysticetes in all oceans. They may cause severe diseases of thoracic cavity, head, stomach, intestine, tissue, and internal organs. Dailey considers parasitic nematodes to be a major cause of mortality of cetacean populations. Although more is known about nematodes than about any other cetacean parasite group, agent life cycles and virulence, agent/host interactions, and disease diagnosis require much more attention, as does disease prevention and therapy in captive hosts.

While acanthocephalans have been recovered from 16 cetacean species, there is no unequivocal proof that they may cause severe diseases. The same is true for the large

variety of epizoites and ectoparasites found to live in close association with whales.

In recent years, 14 types of tumours have been reported from a variety of anatomical sites from 9 cetacean species, primarily in older individuals. Congenital abnormalities have become known primarily from captive whales. They include defects of the circulatory system and kidneys, as well as deformed flippers and spine.

Conclusions

The limited in-depth information available on the diseases of marine reptiles, birds and mammals hardly justifies drawing conclusions beyond those formulated in the preceding 'Summaries of Chapter Contents' and in Chapters 2 through 7. However, in this last book of the treatise I wish to call attention to apparently rather substantial changes in the overall significance rating of the circumstances responsible for causing disease.

It seems that we are presently witnessing shifts in the relative importance of the different categories of disease causes. As pointed out in Vol. IV, p. 9 (see also Vol. I, p. 16), the following basic categories of disease causes may be distinguished: (i) critical intensities of abiotic environmental factors; (ii) damage due to coexisting organisms; (iii) nutritional disorders; (iv) physical injuries; (v) circumstances internal to the individual involved. Of these, particularly the first 2 categories are gaining in significance and are likely to continue to do so in the future.

Critical intensities of abiotic environmental factors have attained a new, man-made dimension. Modern man's impact on, and interference with, nature around him has increased dramatically. Especially pollution by, and food production for, some 4.9 billion people tend to modify and destabilize established ecological equilibria and dynamics, and to interfere with the potential of numerous organisms for counteracting or adjusting to disease-causing entities.

Damage due to coexisting organisms (agents) appears to be augmented *via* man-made distortion of evolved, selection-controlled host/agent interactions. In economically important and hence man-protected plants and animals and, of course, in man himself, impairment of evolutionarily acquired defence potentials against biotic disease agents opens up new avenues and opportunities for agent attack, particularly for viruses, bacteria and fungi.

Categories (i) and (ii) — in combination with anthropogenic impact and deformation of natural selection processes — augment the significance of innate (idiopathic, genetic) disorders (Category V). Medical achievements in protecting and supporting individuals against disease may ultimately prepare the scene for disease at the genotype (population) level (Vol. IV, p. 9): reduction of selection pressure tends to impair or to disharmonize genetic components of host defence potentials, including immunity mechanisms.

2. DISEASES OF REPTILIA

G. LAUCKNER

The class Reptilia, which is so diversified with respect to its terrestrial forms, is only sparsely represented in the marine environment — by sea turtles (order Testudines, superfamily Chelonioidea), sea snakes (Serpentes) and the Galapagos marine iguana, the latter 2 in superorder Squamata.

There are only 6 'safe' species of marine turtles in the oceans of the world, although a large number of subspecies, species of doubtful validity and synonyms exist. The 6 species are: *Chelonia mydas,* the green turtle; *Caretta caretta,* the common or Atlantic loggerhead turtle; *Eretmochelys imbricata,* the hawksbill turtle; *Lepidochelys olivacea,* the olive or Pacific ridley; *Lepidochelys kempi,* the bastard turtle or Kemp's (Atlantic) ridley; and *Dermochelys coriacea,* the luth, leatherback, or trunkback turtle (the latter being the largest of the existing marine reptiles).

The first 5 are considered members of the Cheloniidae, the latter members of the Dermochelidae. *Chelonia depressa* and *C. japonica* may be 'forms' of the cosmopolitan *C. mydas* (for lists of synonyms and determinative keys consult Brongersma, 1972, and Ingle and Smith, 1974).

Marine turtles range among the few reptiles which are of economic importance, and of these, the green turtle *Chelonia mydas* is considered "the most valuable reptile in the world" (Carr, 1952, p. 349). The steady decline in the world population of this species during the past century — mainly caused by extensive harvesting of natural stocks (Pax, 1962) — has recently stimulated scientific and commercial interest in the general biology, farming and artificial propagation of these reptiles (Hendrickson, 1958, 1974; Márquez, 1966; Schroeder, 1966; Bustard, 1972; Stickney and co-authors, 1973; Ingle and Smith, 1974; Owens, 1974; Ulrich and Owens, 1974; Wood, 1974; Witham and Futch, 1977; Solomon and Baird, 1979).

Marine turtles may be afflicted by a variety of diseases, both microbial and parasitic. However, at the most, only passing mention is made, in the monographs listed above, of the diseases and parasites of chelonians. Detailed information related to this topic is widely scattered in the literature, and no comprehensive review of the diseases of these important marine vertebrates has ever been published.

The marine Serpentes are represented by the tropical (Indo-Pacific) sea snakes of the family Hydrophiidae. The Laticaudinae (sometimes regarded as an independent family, Laticaudidae) are amphibious and spend considerable time of their life outside the water. The Hydrophiinae, in contrast, are 'true' aquatic reptiles; some are pelagic. For general information on the biology of sea snakes consult Smith (1926) and Dunson (1975); for notes on the (problematic) maintenance of hydrophiids in captivity see Shaw (1962), Klemmer (1967) and Zeiller (1969); and for information on the biology of the Galapagos marine iguana consult Carpenter (1966).

DISEASES CAUSED BY MICROORGANISMS

Agents: Virales

Stress-induced viral affections are well known in vertebrates. Herpes-type viruses have been identified as associated — or causative — agents of numerous skin diseases. Herpesviruses are typical stress pathogens, often remaining latent or dormant in their hosts over extended periods of time, with no obvious harm, becoming virulent only under adverse physiological conditions.

Chelonia mydas hatchlings, maintained under crowded stocking conditions at a commercial turtle farm at Grand Cayman, British West Indies, developed a skin affection termed 'grey-patch disease' because of the greyish colour of the lesions (Haines and co-authors, 1974; Rebell and co-authors, 1975; Haines, 1977, 1978; Fig. 2-1). The eggs from which the hatchlings were obtained were originally collected from the beaches of Surinam, Costa Rica and Ascension Island. Five successive groups of young turtles underwent epizootics of grey-patch disease, from 70 to 95 % of the individuals in a given batch contracting the virosis.

The origin of the eggs did not seem to influence the incidence of the disease, which often led to severe maceration of the skin and death of the hatchlings. Grey-patch disease (GPD) with typical skin lesions usually appeared in the turtles 50 to 80 days after hatching, and peak mortalities involving 2 to 25 % of affected animals occurred 8 to 12 weeks after the onset of external signs (Fig. 2-2). It was considered possible that earlier peaks of mortality (3 to 6 weeks after hatching and well before the onset of skin manifestations) may have been due to a systemic viral infection. Indirect evidence for this comes from laboratory experiments, in which hatchlings were scratch-inoculated (see below). Mortalities in non-inoculated turtles were always chronologically associated with the occurrence of disease signs and, in all likelihood, death of the animals was a direct result of the affection.

The lesions produced consisted of spreading plaques of superficial epidermal maceration with slightly raised borders. In some cases, they eventually covered the entire surface of the skin of the neck and flippers (Fig. 2-1b, c). Occasionally, less extensive lesions resolved spontaneously after approximately 6 weeks duration, but most patches continued to spread for months. The lesions can involve other superficial epidermal structures, such as the shell segments, the eyelids and conjunctival surfaces of the eye itself; secondary bacterial infections are common. Typically, grey-patch lesions disappear before 1 yr of age. There also was another form of the same skin disease, characterized by small non-spreading papular lesions, which may persist in turtles throughout life. Lesions of this kind (Fig. 2-1a) were small and sharply circumscribed, with marked hyperkeratosis and acanthosis of the epidermis (Fig. 2-3a). In the upper half of the epidermis, the nuclei contained homogeneous, faintly basophilic, ground glass-like inclusions (Fig. 2-4), and the remaining nuclear chromatin was condensed into clumps at the periphery of the inclusions. Nuclei containing these inclusions were enlarged and surrounded by an optically empty halo. The upper layers of the epidermis were infiltrated by eosinophilic granulocytes, and the dermis contained an infiltrate of mononucleate cells and eosinophilic granulocytes predominantly around the blood vessels. Few bacteria were seen in the keratin layer (Rebell and co-authors, 1975).

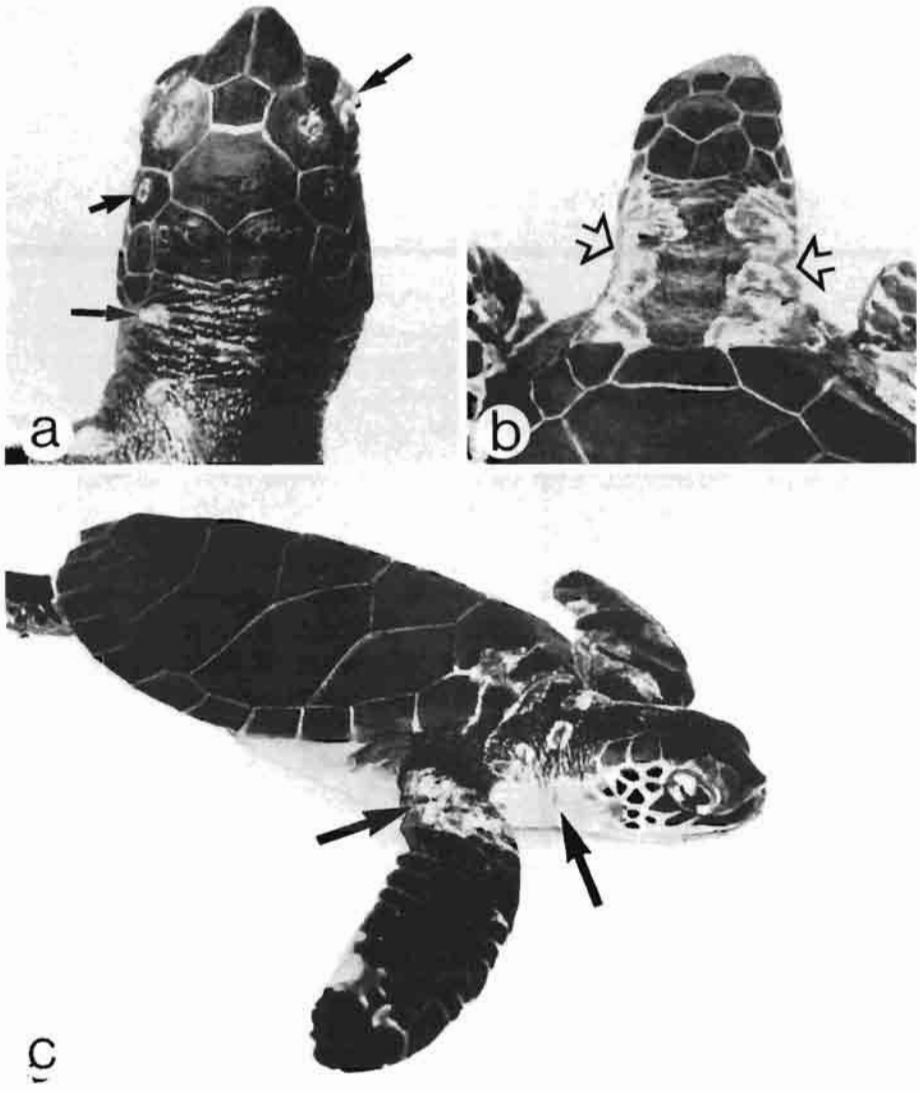


Fig. 2-1: *Chelonia mydas.* Lesions in 2- to 3-month-old hatchlings associated with herpes-type virus infection. (a) Papular lesions; (b) grey-patch lesions with characteristic advancing border; (c) extended lesion. (After Rebell and co-authors, 1975.)

Microscopically, the more diffuse, spreading GPD skin lesions (Fig. 2-1b, c) were similar to the papular lesions, but much more extensive and less sharply circumscribed (Fig. 2-3b). There was marked hyperkeratosis which, in places, had a basket-weave appearance. Deeper portions of the keratin contained residual nuclei with intranuclear inclusions, as well as clumps of Gram-negative rods and occasional conspicuous Gram-positive cocci in the upper portion of the keratin layer. The underlying epidermis was

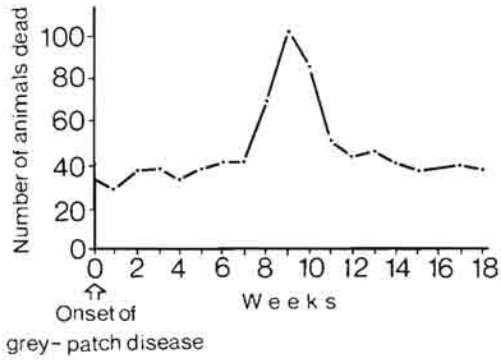


Fig. 2-2: *Chelonia mydas*. Typical course of a grey-patch epizootic. (After Haines and co-authors, 1974.)

acanthotic and had papillary hyperplasia. Throughout the upper half of the epidermis, the nuclei contained inclusions identical with those observed in the papular lesions. The underlying dermis showed increased vascularization with infiltrates of mononuclear cells and eosinophilic granulocytes. In later stages of the disease, the entire epidermis was replaced by a necrotic crust, fibrin and inflammatory cells.

The histopathologic picture of GPD is consistent with a herpes-type virus infection. Electron microscopy revealed the presence of abundant viral particles in thin sections and scrapings of diseased skin portions (Fig. 2-5). The particles (Fig. 2-6a) measured 160 to 180 nm in diameter and contained an electron-dense inner core surrounded by a capsid and an outer envelope. Occasionally, particles containing 2 nucleocapsids surrounded by a single unit membrane were seen (Fig. 2-6a). Negative stained preparations (Fig. 2-6b) revealed enveloped nucleocapsids, 175 to 200 nm in diameter, and naked nucleocapsids (Fig. 2-6d), 105 to 120 nm in diameter. All forms represented in Fig. 2-6 have been described for herpesviruses (Rebell and co-authors, 1975; Koment and Haines, 1977).

GPD could be transmitted experimentally by scratch inoculation and by injection with bacteria-free preparations of viral particles. Resulting lesions were fully developed within 3 to 5 weeks. Hatchlings scratch-inoculated at a very early age (3 to 5 weeks) showed 100 % mortality within 7 days without visible skin lesions. In general, only turtles under 1 yr of age were susceptible to inoculation, which is in agreement with the observation that natural GPD is rarely seen in animals over 1 yr of age. It is likely that older turtles have developed circulating antibodies as a direct result of having survived the disease in their first year of life (Haines and co-authors, 1974; Haines, 1977, 1978).

Various attempts have been made to grow the GPD virus using a large number of mammalian, reptilian and fish cell lines. The only cells in which constant infections showing consistent cytopathic effects were achieved, were *Chelonia mydas* skin cells. Optimal virus replication occurred within 72 h at 25 °C. Material from these cultures, scratch-inoculated into young green turtles, produced new GPD lesions (Koment and Haines, 1977; Haines, 1978).

According to Haines (1977), some response to metabolic inhibitors is seen in severely GPD-affected turtles. More recently, Haines and co-authors (unpubl.; in Haines, 1978) have produced an inactivated vaccine from GPD virus. Intramuscular injections of dead

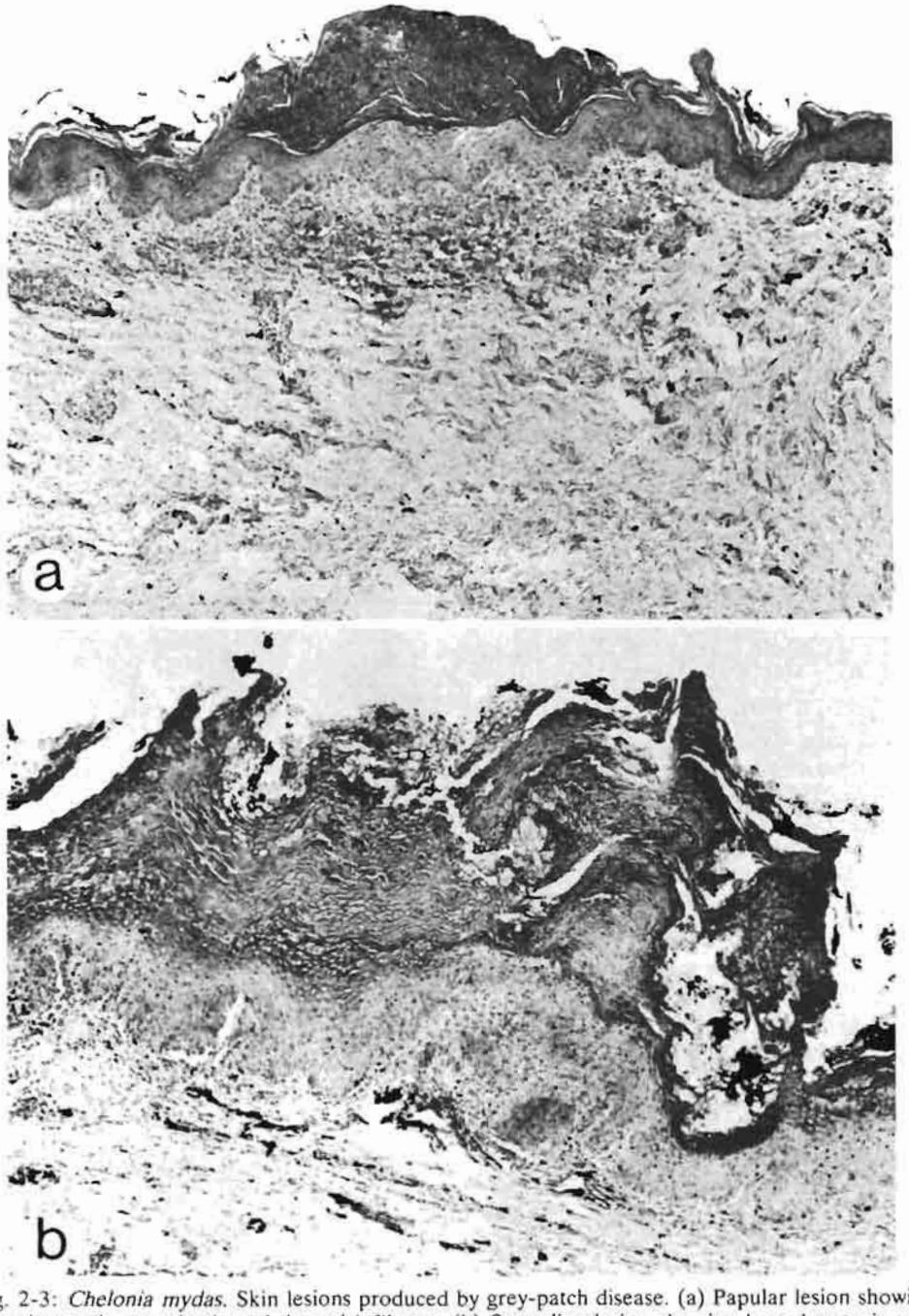


Fig. 2-3: *Chelonia mydas*. Skin lesions produced by grey-patch disease. (a) Papular lesion showing hyperkeratosis, acanthosis and dermal infiltrate. (b) Spreading lesion showing hyperkeratosis with persistent inclusions and local basket-weave appearance, acanthosis, papillomatosis and granulocytes at epidermis-keratin junction. Both H & E stain, original magnification × 38. (After Rebell and co-authors, 1975.)

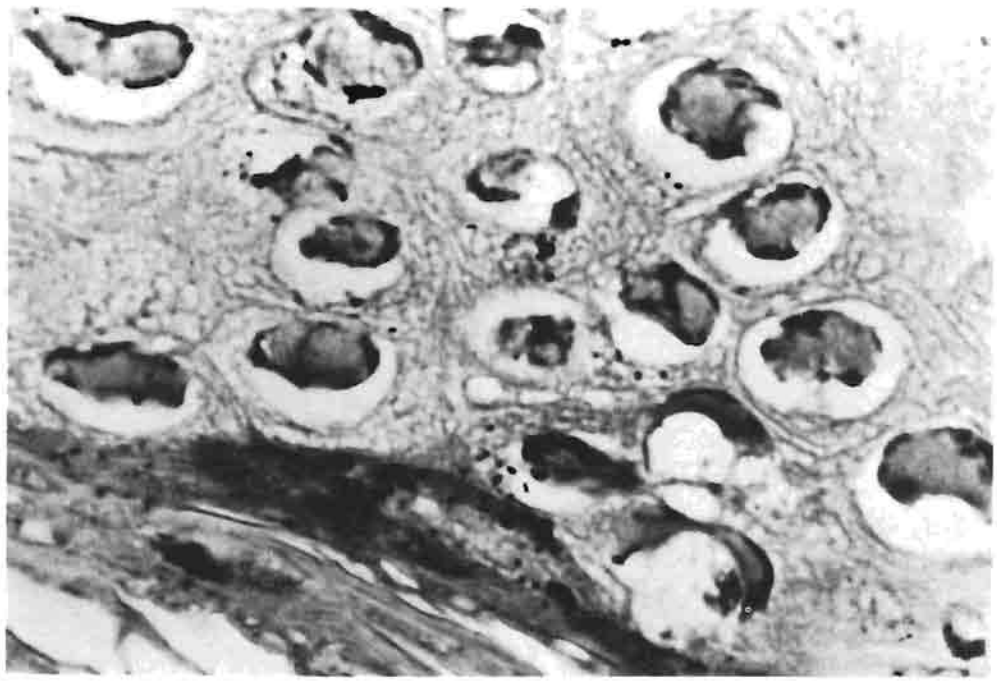


Fig. 2-4: *Chelonia mydas*. Skin section of turtle with papular form of grey-patch disease. Note enlarged nuclei, intranuclear inclusions, clumped chromatin and perinuclear clear space. H & E stain, × 600. (After Rebell and co-authors, 1975.)

GPD virus were found to protect against challenge with active material in inoculated test individuals. Preliminary trials have shown that the inactivated virus vaccine is feasible for farm-wide use.

Preventive measures against outbreaks of GPD include the maintenance of proper sanitary conditions. Under farm conditions, stocking densities of green turtles are usually high. Since GPD is considered a stress-induced disease, reduction of stocking densities is a prerequisite of disease prevention in turtle hatcheries. Reduction of water chlorination also appears to be feasible. Morbidity and mortality levels depend largely upon the degree of stress which the animals undergo (Haines, 1977, 1978).

Among several possible stress factors, elevated water temperature appears to be the most important one. Under hatchery conditions, GPD is more severe in summer when the water temperature of the holding tanks is 4 to 5 °C higher than in the cooler winter (Haines, 1978). In a large-scale heat induction experiment, Haines and Kleese (1977) subjected 4 groups of 8-week-old *Chelonia mydas* to a series of water temperature changes. One of the 4 groups, each of which contained 52 test individuals, was subjected to a temperature increase from 25 to 30 °C at a rate of 1 °C day^{-1}, then left for 3 days at 30 °C, and subsequently subjected to a decrease from 30 to 25 °C at the same rate. A second group was subjected to the same gradual temperature increase, but maintained at 30 °C for the duration of the experiment. A third group was taken from water of 25 °C and placed immediately in water of 30 °C, left for 4 days at 30 °C, and then returned directly to water of 25 °C. These turtles were, in essence, temperature 'shocked'. The fourth group,

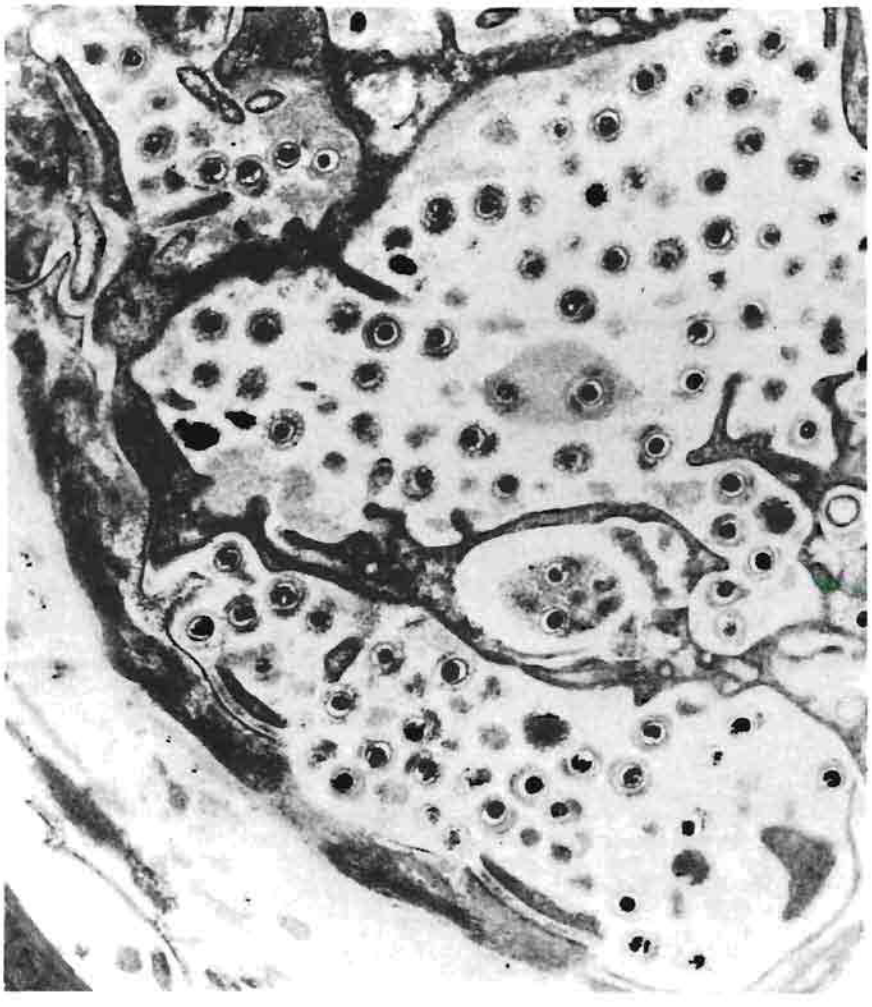


Fig. 2-5: Grey-patch disease (GPD) viral particles in thin section of scraping from GPD lesion in *Chelonia mydas*. Original magnification × 41,000. (After Rebell and co-authors, 1975.)

serving as control, was maintained at a constant temperature of 25 ± 0.5 °C. All turtles were monitored for the appearance of GPD lesions over a period of 48 days.

The turtles subjected to temperature shock treatment and those subjected to a gradual increase to 30 °C, and maintained at 30 °C, had an earlier onset of disease, with more severe lesions, than either the control group or the group of animals subjected to a gradual increase to 30 °C and then brought back down to 25 °C.

These results were considered as good evidence that heat is one of the stress factors which induce GPD, and that higher water temperatures tend to increase the severity of the lesions. No stress factors other than water temperature changes were applied to the turtles. However, under mariculture conditions in a turtle farm, a variety of other stress factors may exert cumulative effects upon disease severity and mortality. Nevertheless, water temperature may be the major stress factor that triggers the development of GPD, and that

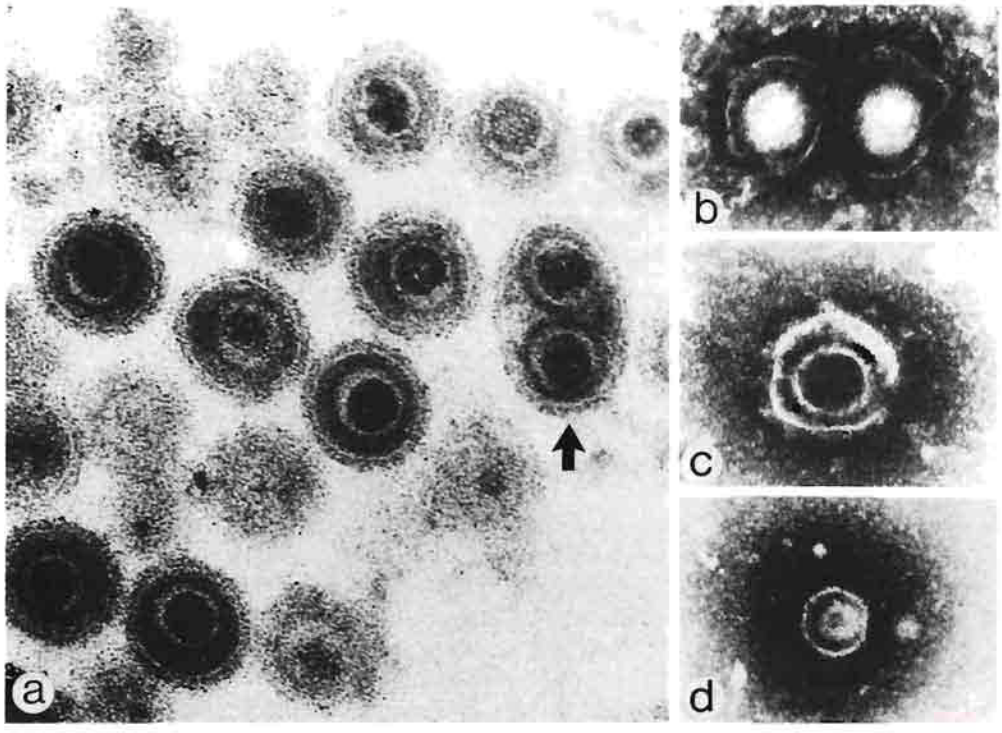


Fig. 2-6: GPD herpes-type viral particles from skin lesion of *Chelonia mydas*. (a) Viral particles from lesion shown in Fig. 2-5 at higher magnification. Note particle containing 2 nucleocapsids surrounded by single unit membrane (arrow). (b) Negatively stained preparation of fully enveloped nucleocapsids. (c) Enveloped empty capsid. (d) Naked full capsid. Original magnifications × 123,000. (After Rebell and co-authors, 1975.)

water temperature manipulation may permit control of the disease under intensive aquaculture conditions (Haines and Kleese, 1977; Haines, 1978).

Since mariculture farming of *Chelonia mydas* seems likely to become a successful industry in the Caribbean, further study of the GPD virus may have considerable economic importance. In this context, it would be important to determine whether the agent is a natural, possibly specific, virus of the green turtle, or whether this reptile is an 'accidental', not fully compatible host for the pathogen. The fact that, under experimental conditions, GPD virus would replicate only in green-turtle skin cell lines (see above), tends to support the first view. The pronounced pathogenicity of GPD for *C. mydas*, on the other hand, suggests poor adaptation or even host-parasite incompatibility.

There has been some speculation about the question whether the herpesviruses are of mono- or polyphyletic origin (Nahmias, 1972; Kaplan, 1973). Some of the peculiarities of the Herpetoviridae suggest that they may have evolved with their hosts. Many members of this group, particularly the cytomegaloviruses, are host-specific. They seldom damage their specific hosts but, when crossing host-species barriers, may produce fulminating and sometimes fatal disease (Dulbecco and Ginsberg, 1973; Buchanan and Richards, 1982). Cytomegaloviruses (and herpesviruses in general) are capable of remaining latent or dormant in a host for extended periods of time, and of becoming reactivated and disease-

producing many years after the initial (in mammals often transplacental) infection, particularly under conditions of stress. The human herpes simplex and herpes (varicella) zoster viruses are well-known examples.

Herpes-type viruses and virus diseases have been reported from several marine invertebrate and vertebrate hosts (for reviews consult Buchanan and Richards, 1982, and Lauckner, 1983), and even from an estuarine fungus, *Thraustochytrium* sp. (Kazama and Schornstein, 1972). Whether these (probably specific) herpesviruses can cross-infect other hosts, is unknown. With respect to the origin of the green-turtle virus, Haines (1978) contributed some interesting observations and experiments. In order to determine whether the agent had been introduced with the original *Chelonia mydas* eggs collected in the field, or whether it was a virus that was maintained on the Gran Cayman turtle farm, eggs were hatched away from the contaminated environment of that mariculture facility. The hatchlings were raised in different locations, i. e., in Bimini (Bahamas), Arizona, and Virginia and Pigeon Keys, Florida. Each group of turtles raised in these diverse locations, under different environmental conditions, developed GPD. In general, however, because these individuals were not in a stressful environment, the lesions were of the less extensive, pustular form.

These findings appear to indicate that the GPD virus is indeed a natural virus of *Chelonia mydas*, which possibly also occurs in the open ocean. Since wild green turtles with GPD skin lesions have, thus far, not been found, the agent must be present in these hosts in its latent, dormant phase.

Agents: Bacteria

Bacterial affections appear to be rare among free-ranging marine reptiles, but are common in captive individuals, mainly in the form of subcutaneous abscessation. A number of microorganisms have been isolated from reptilian abscesses; both pure and mixed cultures have been obtained. *Aeromonas hydrophila, A. aerophila, A. aerogenes, Citrobacter* spp., *Enterobacter* spp., *Escherichia coli, Mycobacterium* spp., *Peptostreptococcus* sp., *Proteus morgani, P. rettgeri, Pseudomonas* spp., *Salmonella marina* and *Serratia* spp. have been identified as etiological agents (Frye, 1974). Stickney and co-authors (1973) and Sindermann (1977a) mentioned an *Aeromonas* sp. infection in tank-held *Caretta caretta*. The organism produced 'superficial lesions' not described in detail. Affections of this kind have not been reported from *Chelonia mydas* maintained in the same indoor running sea water system.

Witham (1974) briefly described a bacterial disease of pen-reared hatchling *C. caretta*. Affected individuals developed necrotic, spreading skin lesions and died within 3 to 7 days. All hatchlings were not infected simultaneously, but within 3 months each had developed the symptoms. None of the older turtles, maintained in the same sea-water system, contracted the disease. *Bacteroides* sp., *Pseudomonas aeruginosa* and *Staphylococcus epidermidis* were isolated from the hatchlings' lesions. As the latter were necrotic, spreading and not walled off, *Bacteroides* sp. was considered the primary pathogen. Of 140 hatchlings, 131 eventually died. The remaining 9 turtles received 0.1 ml injections of potassium penicillin (25,000 USP units ml^{-1}) and streptomycin sulphate (250 mg ml^{-1}). Of these individuals, 3 eventually recovered. Chloramphenicol, the drug of choice against *Bacteroides* spp., might have been more effective and should be tested in future outbreaks (Witham, 1974). *Citrobacter freundi* has been identified as etiological agent of septicaemic

cutaneous ulcerative disease in other turtles; the infection was found to respond to daily injections of chloromycetin succinate (Frye, 1974).

Bacteria may appear as secondary invaders of *Chelonia mydas* skin lesions produced by a herpesvirus (see Section 'Agents: Virales'). Unidentified Gram-negative rods and occasional conspicuous Gram-positive cocci were seen in the upper portion of the keratin layer in skin areas affected by grey-patch disease (Rebell and co-authors, 1975). Occasionally, these organisms may invade the dermal area and deeper tissue. At times, bacterial infection may become so severe as to initiate deep scarring. The bacterial flora of GPD lesions appears to change, depending on the surrounding conditions, and no specific strain of bacterium could be demonstrated as a consistent inhabitant of the damaged skin portions. Because of the contaminated environment of holding tanks, new bacteria come in as old ones are displaced (Haines, 1978).

Viruses or *Clostridium* organisms have been suspected as etiological agents of a condition termed 'floppy-flipper' and involving the skeletal musculature of tank-held *Chelonia mydas*. The affection, listed by Haines (1978) under 'Diseases commonly seen in commercially raised green sea turtles', was not described further.

Ulcerative shell disease (USD), also known as 'shell rot', 'spot disease' or 'rust', is a bacterial disease affecting the scutes of the carapace and the plastron. It tends to be mildly contagious, chronic and self-limiting. The causative agent is the bacterium *Beneckea chitinivora (Bacillus chitinivorus)* (Wallach, 1975, 1976, 1977). Thus far, USD has only been reported from non-marine turtles. However, because *B. chitinivora* is of ubiquitous occurrence in marine environments and produces shell disease in a variety of crustaceans including commercially exploited species (Vol. III; for review consult Rosen, 1970), it should be considered as being potentially capable of producing shell disease in marine turtles.

'Mouth rot', an ulcerative stomatitis, has briefly been reported from captive beaked sea snakes *Enhydrina schistosa*. The disease produces lesions in the jaws, which may progress to the point that the snakes are unable to feed, and die (Murthy and Rama Rao, 1977). Of the 9 species of sea snakes occurring on the Madras (India) coast, *E. schistosa* appears to be the only species afflicted with mouth rot. The etiological agent of the disease has not been identified, but *Aeromonas hydrophila* was the organisms most frequently cultured from mouth-rot lesions in other captive reptiles. Treatment with systemic antibiotics, particularly chloromycetin succinate, proved to be effective for the control of the infection. If left untreated for a sufficiently long time, osteomyelitis will result (Frye, 1974).

Several species of bacteria have been isolated from juvenile mariculture-reared *Chelonia mydas* suffering from mycotic pneumonia, e. g., *Salmonella gatuni* from the lungs and the intestine, *Arizona* organisms from the lungs, liver and kidney, and *Citrobacter* sp. from the intestinal tract. *Salmonella* sp. was recovered in pure culture from an intestinal lesion in one turtle (Jacobson and co-authors, 1979). The role of these bacteria in the overall pathology of the diseased turtles was not established, but the possibility was considered that such infectious agents may initiate pathological processes that ultimately lead to fungal involvement (p. 564).

Respiratory infections are common to all captive reptiles, but they are of particular importance in snakes, turtles and tortoises. Clinical signs are nasal discharge, open mouth breathing, wheezing, inappetance, torpor and, occasionally, loss of normal swimming

equilibrium — a condition also displayed by turtles suffering from mycotic lung infections (p. 564). Since the pulmonary system of chelonians also functions as a hydrostatic organ for maintaining buoyancy, unilateral loss of the functional pulmonary bed will cause the turtle to swim with the nonaerated side lower than the other (Frye, 1974).

Brock and co-authors (1976) reported on a spontaneous outbreak of tuberculosis in captive *Chelonia mydas* hatchlings. The turtles were obtained from the French Frigate Shoals, 800 km west-northwest of Hawaii, and maintained in sea-water tanks at the University of Hawaii. Of 120 hatchlings, 6 developed grossly visible lesions in the lungs, consisting of yellowish-white nodules ranging from 0.5 to 2 mm in diameter. Nodules were also seen in the liver and kidneys of some of the turtles. The lesions consisted of focal granulomas characterized by central necrosis, sometimes with calcification, and infiltrated by macrophages, lymphocytes and Langerhans-type multinucleated giant cells. The foci were surrounded by fibroblastic elements. The animals had succumbed to the infection.

Tissue smears and histological sections of lung, liver, kidney and spleen, stained by the Ziehl-Neelsen method, revealed the presence of acid-fast organisms. The bacilli were identified as *Mycobacterium avium,* Serotype 8. This appears to represent the first instance of an *M. avium* infection in a cold-blooded species. Friedmann (1903) had previously reported on a case of spontaneous tuberculosis affecting an individual of *Caretta caretta ('Chelone corticata')* in the Berlin Aquarium. This turtle had died with tubercle formation and typical giant cells. The causative agent was later described as *Mycobacterium friedmanni* (Reed, 1948).

The occurrence of spontaneous tuberculosis in poikilothermic animals is not unusual. Nigrelli and Vogel (1963) listed 151 species of fish, 11 species of amphibians and 23 species of non-marine reptiles, in which tuberculosis has been observed. However, the only report on mycobacteriosis in a marine reptile, included in this list, is that by Friedmann (1903; see above).

The origin of the *Mycobacterium avium* infection of *Chelonia mydas,* described by Brock and co-authors (1976), remained unknown. The 6 cases of tuberculosis reported by these authors all occurred during a 2-month period, which represents a high attack rate. The severity and consistency of the pneumonic lesions suggest a relatively high susceptibility of marine turtles to this bacillus and, moreover, may indicate that infection occurred by airborne transmission rather than by ingestion of *M. avium.* However, various strains of mycobacteria have on several occasions been isolated from aquarium water (Pattyn and co-authors, 1971). Brock and co-authors (1976) emphasized the risk for natural marine turtle populations arising from release of individuals, which have contracted infectious diseases in captivity. Epizootics initiated by infected turtles released in nature to replenish diminishing stocks of endangered species could counter the well-meaning intentions and efforts of conservationists.

Agents: Fungi

The literature pertaining to vertebrate mycotic diseases contains mainly reports relating to non-reptilian hosts and, as far as the Reptilia are concerned, information stems almost exclusively from terrestrial or freshwater forms (Hunt, 1957; Frank, 1976; Jacobson and co-authors, 1979).

Unidentified fungi have been observed in *Chelonia mydas* skin lesions produced by grey-patch disease (see Section 'Agents: Virales'). These appear as secondary invaders of

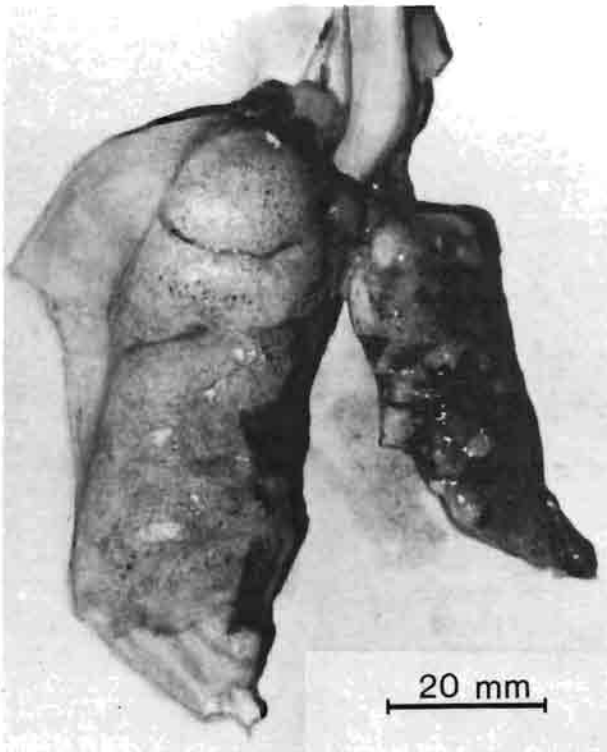


Fig. 2-7: *Chelonia mydas*. Lungs of juvenile turtle with mycotic pneumonia. Right lung contains multiple nodules and is consolidated, left lung is emphysematous. (After Jacobson and co-authors, 1979.)

the keratin layers and may sometimes penetrate down to the dermal area and deeper tissue (Haines, 1978). Superficial skin lesions, believed to be of fungal etiology, occasionally developed on pen-reared *Chelonia mydas*. These were effectively controlled with 5 to 10 % gentian violet solution or potassium permanganate dips. Similar lesions, produced by bacteria (p. 561), did not respond to these drugs (Witham, 1973, 1974; Witham and Futch, 1977).

Ulcerative shell disease or 'shell fungus', a bacterial affection of freshwater turtles (p. 562), has at times been regarded as being of mycotic etiology. However, although a wide variety of bacteria may be isolated from shell-diseased individuals, fungal cultures may be negative (Frye, 1974).

A granulomatous lesion in the lung of 1 out of 120 *Chelonia mydas* caught at French Frigate Shoals, 800 km west-northwest of Hawaii, and subsequently kept in captivity for 6 to 7 months, contained mycelial elements suggestive of a fungal infection (Brock and co-authors, 1976). These organisms may be identical with those reported by Jacobson and co-authors, 1979; see below).

During winter (1977/78 and 1978/79), 4- to 5-month-old green turtles reared commercially on the Cayman Turtle Farm, Grand Cayman (British West Indies), developed a buoyancy abnormality, swimming at an angle to the horizontal. Many of the abnormal turtles chronically lost weight and eventually died. An exudative plug was often

found obstructing their trachea. Highest mortalities, which occurred in January and February, were associated with the lowest mean monthly water temperature (26 °C, in contrast to a summer water temperature of 31 °C).

Severely affected turtles were thin and had multiple small to large, white firm nodules in both lungs, resulting in an elevation of the visceral pleura. The right lung appeared to be more severely affected and was often nodular and collapsed (hence the buoyancy abnormality), whereas the left lung was frequently emphysematous and filled the left coelomic cavity (Fig. 2-7). Several turtles had variably-sized multifocal white nodules throughout the liver that often bulged the capsule.

On histological examination, the nodules appeared as multifocal granulomas (Fig. 2-8), consisting of a central core of caseous material containing branching septate fungal hyphae. The caseous centers of the nodules were surrounded by giant cells, macrophages, leukocytes, fibroblasts and collagen. The granulomas sometimes extended into the airways, and in several instances had ruptured into the air passages along with hyphae and

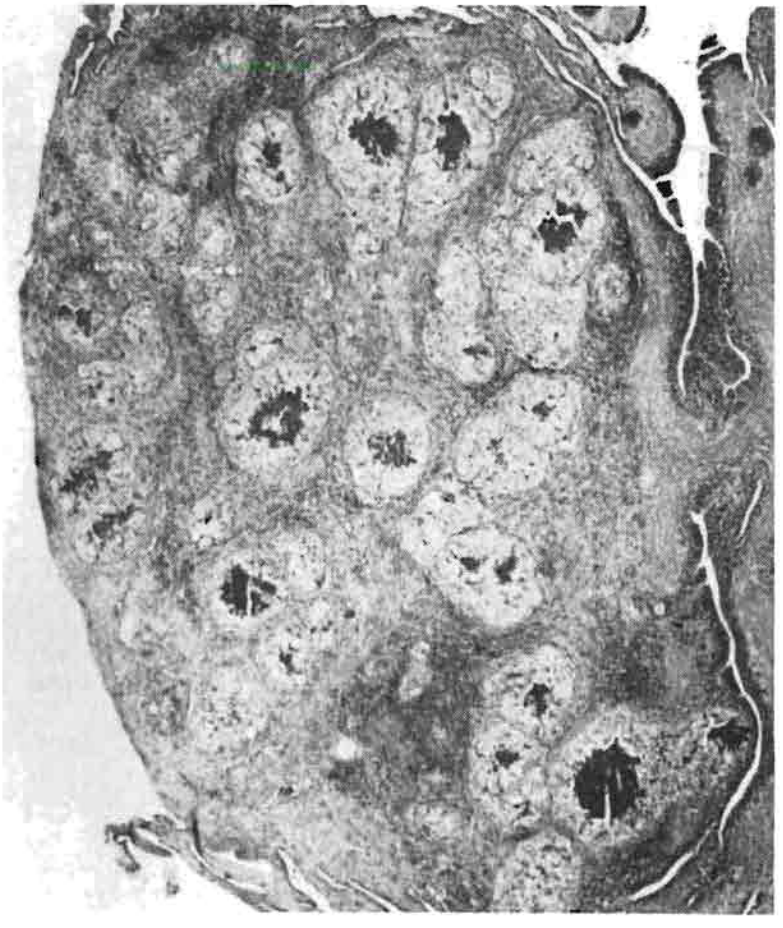


Fig. 2-8: *Chelonia mydas*. Multiple granulomatous inflammatory reactions involving right lung of a turtle with mycotic pneumonia. H & E stain, × 63. (After Jacobson and co-authors, 1979.)

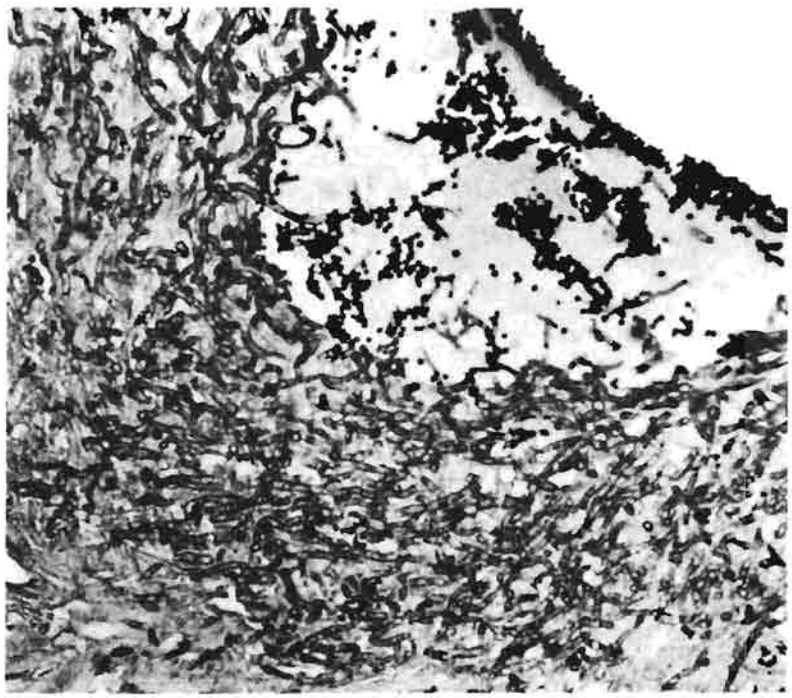


Fig. 2-9: *Chelonia mydas.* Lung pathology caused by mycotic pneumonia, fungal hyphae and spores being released into lumen of an air passage. PAS stain, × 440. (After Jacobson and co-authors, 1979.)

spores (Fig. 2-9). In many sections there was an acute suppurative pneumonic process with necrotic and desquamated lining cells and inflammatory cells filling the terminal airways. In each instance, the emphysematous left lung had dilated airways, with a thinning of the interstitial walls. Some turtles also had multifocal granulomas in the liver.

Fungi of the genera *Sporotrichium, Cladosporium* and *Paecilomyces* were isolated from the lesions. The disease, observed in 29 juvenile *Chelonia mydas,* was diagnosed as mycotic pneumonia, although several species of bacteria were also present in the intestinal tracts and tissues of some of the turtles (p. 562).

The fact that peak mortalities in the hatchery occurred during the month with the lowest water temperatures (February), suggests an effect of the environment on the development of the mycosis. It was considered that at low ambient temperatures (and resultant low body temperatures of these poikilothermic animals), the immune system of the turtles may have been compromised, allowing organisms normally present in the environment or in a latent state within the lungs to initiate an active infection (Jacobson and co-authors, 1979). The authors considered that studies of the seasonal fluctuations in the cell-mediated and humoural immune system may provide significant management-related information.

DISEASES CAUSED BY PROTISTANS

Agents: Sarcomastigophora and Apicomplexa

Next to nothing is known about protozoan diseases of marine turtles and sea snakes, although numerous such diseases have been reported from non-marine reptiles.

Grassé (1924) and Zwart and co-authors (1975) described severe cases of hexamitiasis in terrestrial tortoises, caused by *Hexamita parva.* Flagellates *Monocercomonas colubrorum* were isolated, often in tremendous numbers, from the colon of almost every *Laticauda semifasciata* examined on Amami Island, Japan (Telford, 1976). These flagellates are of common occurrence in nearly every family of squamate reptiles in all parts of the world. The known species of *Monocercomonas* are considered non-pathogenic (Cheng, 1973). Haemoflagellates *Trypanosoma testudinis* have been recorded in *'Testudo esculenta'* (according to Hunt, 1957, a synonym of *Chelonia mydas*).

Recently hatched green turtles *Chelonia mydas* were found to die with signs of amoebiasis, believed to be caused by *Entamoeba invadens.* A 35-cm long *Caretta caretta* individual was similarly affected. Lesions produced in the intestine and the liver, consisting of ulcerations and abscesses, were macroscopically visible and characteristic of *E. invadens* infestation. Amoebae were cultivated from cloacal swabbings (Frank and co-authors, 1976). *E. invadens* is known for its pathogenicity to reptiles. It appears to be ubiquitous in zoological gardens and at times has produced epizootics among captive animals (Geiman and Ratcliffe, 1936; Hill and Neal, 1953). According to Bosch and Deichsel (1972), at least 4 *Entamoeba* strains are pathogenic to reptiles. *E. invadens* cannot survive in sea water. The above marine turtles had, however, been kept under unnatural conditions in freshwater. Frank and co-authors (1976) considered it unlikely that the recently hatched *C. mydas* had been infested naturally. Dunlap (1955, p. 64) very briefly mentioned the occurrence of "numerous amebae resembling *Endamoeba histolytica* in the intestinal contents" of a leatherback turtle *Dermochelys coriacea* that had died in the Audubon Park Zoo in New Orleans. Whether this turtle had been maintained in sea water prior to death has not been reported, although it appears likely. Therefore, the existence of a sea-water strain of *Entamoeba* pathogenic to reptiles cannot be ruled out.

Most of the Galapagos marine iguanas *Amblyrhynchus cristatus* examined by Ayala and Hutchings (1974) harboured unidentified intraerythrocytic cytamoeba-like organisms, which left reddish inclusion cavities in the host-cell cytoplasma. Seven of 12 *A. cristatus* also harboured *Hepatozoon*-like haemogregarines. The protozoans only invaded the erythrocytes, with infestation intensities varying from 1/400 to 1/50,000 cells parasitized. Circumstances suggested ectoparasitic mites or ticks as invertebrate hosts for these blood parasites. Other species of haemogregarines may be transmitted by Hirudinea. Turtle leeches *Ozobranchus margoi* (p. 600), examined by Davies and Chapman (1974), yielded no such organisms. A considerable number of haemogregarines infest freshwater turtles (Popovici-Baznosanu, 1907; Robertson, 1908; Reichenow, 1910). As the sexual stages of these coccidians occur in the leech or the arthropod, the vertebrate represents the intermediate host.

A large number of eimeriine coccidians have been described from freshwater turtles and terrestrial snakes (Pellérdy, 1974). A single species, *Caryospora cheloniae,* is known to infest *Chelonia mydas* (Leibovitz and co-authors, 1978). Initially reported by Rebell and co-authors (1974) but not named, the organism has been identified as a serious

pathogen of recently hatched green turtles raised on the Cayman Turtle Farm, Grand Cayman.

The disease and associated mortality appeared in turtles approximately 30 days after hatching, and ran a 60-day course through the stock hatchlings of the farm. Infested turtles were typically flat, weak and emaciated. Characteristic ellipsoidal coccidian oocysts were abundant in the intestinal tract (Fig. 12a, b). Severely affected hosts were found to pass cylindrical casts of intestinal mucosa and faeces packed with oocysts; and late in the epizootic, emaciated turtles with lusterless shells had the lower gut filled with caseous masses of oocysts (Fig. 2-10). Autopsy revealed plaque-like opaque lesions of the mucosa,

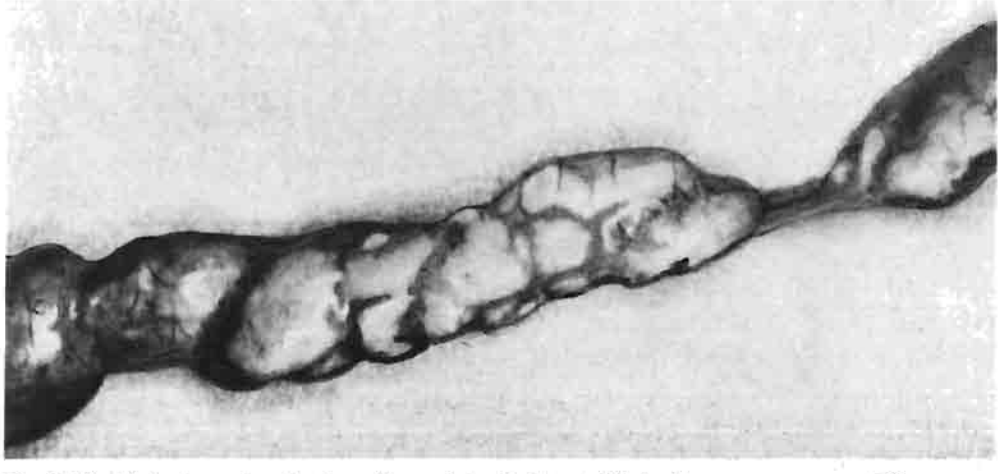


Fig. 2-10: *Chelonia mydas*. Portion of lower intestinal tract filled with caseous masses of *Caryospora cheloniae* oocysts. (After Rebell and co-authors, 1974.)

and gelatinous sloughs of mucosa and oocysts were found essentially throughout the entire length of the intestine distal to the bile-duct opening, with greatest concentrations in the hindgut (Rebell and co-authors, 1974).

Pathological alterations were most obvious in the posterior third of the intestine. The hindgut lumen was greatly dilated and filled with blood, oocysts and tissue debris. The hindgut wall was thinner than normal, thinning being most pronounced where the epithelial folds of the mucosa had sloughed off into the lumen. The tips of the folds were also denuded of their epithelial cover, and free blood was escaping from the blood vessels of the tunica propria into the intestinal lumen. Remnants of the epithelium were distinctly altered, and hyperplasia was pronounced at the margins of the denuded areas. Numerous inflammatory cells, including eosinophils, infiltrated the inflamed mucosal surface. The anterior third (foregut) and the middle third (midgut) of the intestinal tract showed little structural change, although individual developmental stages of *Caryospora cheloniae* were present within some of the epithelial cells at the tips and flanks of the villi of the small intestine (Leibovitz and co-authors, 1978).

Microscopical sections of infested hindgut epithelium revealed the presence of typical coccidian life-cycle stages — trophozoites, macrogamonts, microgametocytes, oocysts, etc. — in intracellular location (Fig. 2-11b). The earliest and smallest stage observed was a

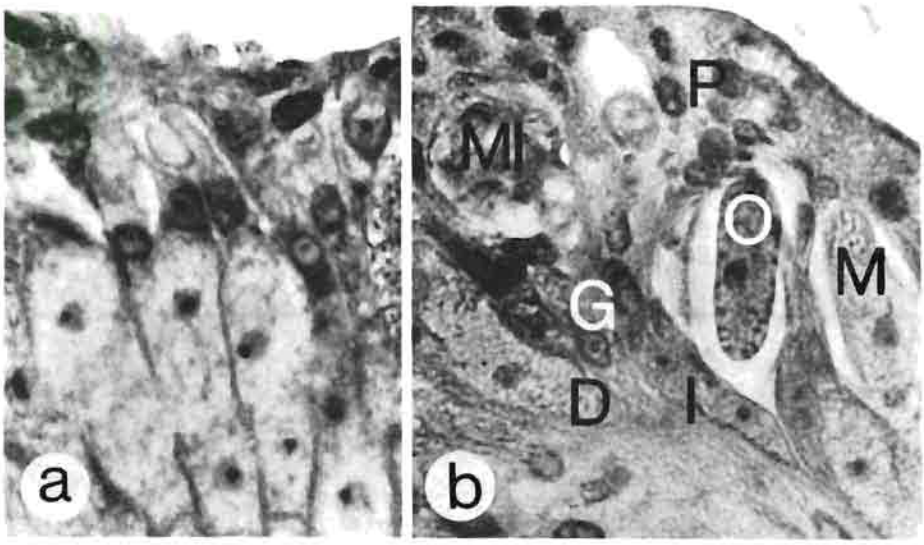


Fig. 2-11: *Chelonia mydas*. Sections of hindgut epithelium infested with *Caryospora cheloniae*. (a) Cords of dividing macrogamonts close to intestinal lumen. × 1,500. (b) Section showing early trophozoite stage (G), immature macrogamonts (I) along basement membrane, dividing macrogamonts (D), mature macrogamonts (M), microgametocyte (MI), oocyst (O) and proliferating epithelial cells (P). × 1,000. (After Leibovitz and co-authors, 1978.)

short, cigar-shaped meront, 10.3 × 8.4 μm (n = 10) in average dimension and with one rounded and one pointed end. The meronts were loosely arranged in groups of 2 to 6 within the middle of the epithelial layer. Binary fission was frequently seen during this stage together with transverse fission of the nucleus, nucleolus and cytoplasm, followed by elongation and separation of the dividing parts (Fig. 2-11a). The dividing segments remained attached, end to end, forming cords of cells. One end of these cords was closer to the intestinal lumen, the other closer to the basement membrane. The cells of the latter were larger, well-differentiated and recognizable as immature or mature macro- or microgamonts. Mature macrogametes, measuring 32.2 × 12.2 μm (n = 10), were closer to the lumen surface.

Developing microgametocytes enlarged into cystic structures, measuring 37.6 × 23.9 μm (n = 10) when fully formed. Mature microgametocytes eventually ruptured at the lumen surface, releasing myriads of biflagellated, curved rod-like microgametes less than 0.25 μm in length.

Following fertilization, unsporulated oocysts became surrounded by a large clear space formed during migration from the ruptured host cell into the intestinal lumen. Experimentally, sporulation could be induced by suspending oocyst-containing faecal material in sea water with an addition of 2.5 % potassium dichromate at 25 °C for 24 h. Unsporulated oocysts were elongate, ellipsoidal in shape, and with smooth walls. As sporulation progressed, granular material of the sporont became more compact and condensed to form a sporoblast (Fig. 2-12a). Sporulated oocysts (Fig. 2-12b), measuring 37.4 × 12.8 μm (n = 5), were monosporocystic and octozoic. However, during the process of sporulation, the double-layered oocyst wall usually disintegrated, releasing the sporulated sporocyst, measuring 34.5 × 12.7 μm (n = 100). Only 6 of 1,000 sporulated

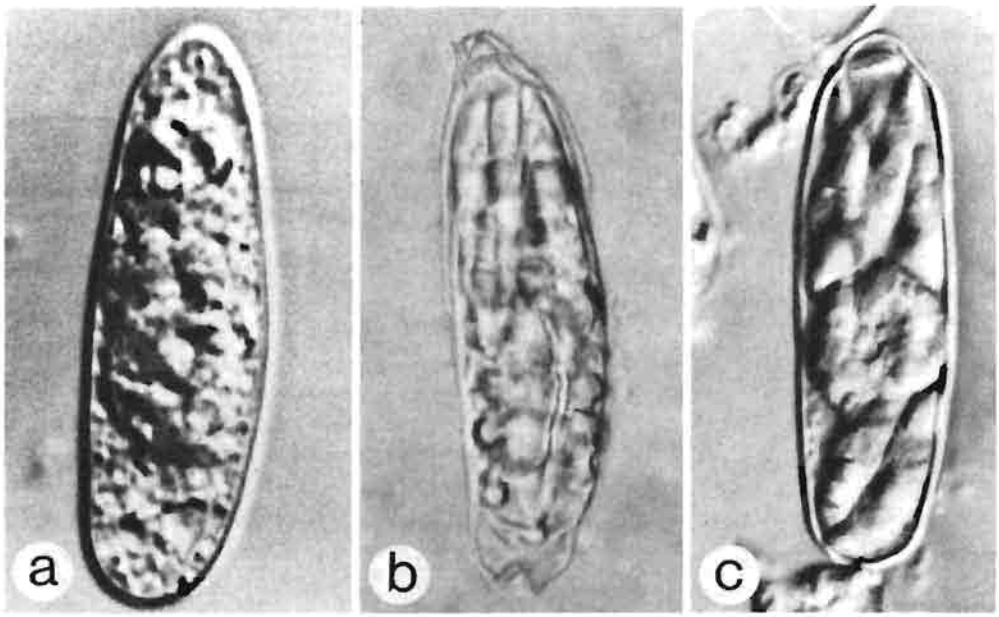


Fig. 2-12: *Caryospora cheloniae* from hindgut of *Chelonia mydas*. (a) Partially sporulated oocyst; (b) monosporocystic, octozoic sporulated oocyst showing thin, transparent, single-layered oocyst wall; (c) sporulated sporocyst. All × 2,000. (After Leibovitz and co-authors, 1978.)

sporocysts examined were contained within intact oocysts. Excysted, completely extended sporozoites were club- or cigar-shaped and 15.8 × 3.8 μm (n = 2) in dimension. Complete sporulation occurred in 19.5 h at 25 °C. Oocysts continued to sporulate after this time, and the greatest percentage increase was noted between 23.5 h and 73.5 h (Leibovitz and co-authors, 1978). Oocysts within the intestinal tract of turtles are invariably in the unsporulated state. Maturation takes place outside the host (Rebell and co-authors, 1974; Sindermann, 1977b).

Thus far, *Caryospora cheloniae* has only been reported from juvenile *Chelonia mydas* cultivated at the Grand Cayman turtle farm. Examination of the intestinal contents of older green turtles at the farm failed to reveal any coccidian infestation. The source of the agent remained unknown. It was suspected that it might have been introduced in the spring of 1973, either in eggs or sand brought from Ascension Island or Surinam or from some other source, or that it may have been enzootic and undetected at the farm until circumstances permitted it to increase to epizootic numbers.

The reported 2 outbreaks of coccidiosis among young *Chelonia mydas* appeared to be unprecendented in the history of the farm, and were attributed to a temporary laxity in maintenance due to an increased workload imposed by the large number of hatchlings obtained. Early in the epizootics, it was found that large amounts of anaerobic food and faecal sludge containing vast numbers of coccidian oocysts had been permitted to accumulate on the bottoms of the affected tanks. Since the severity of coccidial infestations largely depends on the number of sporulated oocysts ingested, unhygienic tank conditions can result in infestations of epizootic proportions. However, once the epizootic had begun, vigorous sanitary measures were not sufficient to control the disease.

The *Caryospora cheloniae* epizootics, which appeared sequentially in 2 groups of hatchlings and were recognizable by an onset of an increase in daily mortality, started on May 29 and June 17, 1973, respectively. After maximal daily mortality had been reached, a gradual decrease to pre-epizootic levels followed. The total course of the mass mortality in the first group of hatchlings was 72 days and in the second group 53 days.

Approximately 1 month after the disease had run its course, apparently healthy survivors still harboured active areas of coccidial invasion in the intestinal tract. However, hatchlings placed in the same stock tanks at this time did not contract the coccidiosis. After approximately three months, most of the surviving turtles appeared to be free of the parasite (Rebell and co-authors, 1974).

At present, no effective treatment of *Caryospora cheloniae* infestations is available. Oral administration of tetracyclines and anticoccidial sulfonamides, as well as intraperitoneal injection, oral administration or overnight soaks with sulfamethazine had no positive effects (Rebell and co-authors, 1974). Sindermann (1977b) suggested trials with avian anticoccidial agents.

DISEASES CAUSED BY METAZOANS

Agents: Trematoda

Marine turtles are hosts for an exceptionally species-rich trematode fauna. With the single exception of an aspidogastrean flatworm, all trematodes of chelonians belong to the Digenea. Quite a few digeneans have been reported from the Galapagos iguana *Amblyrhynchus cristatus* and from hydrophiid sea snakes.

The Aspidogastrea comprise a group of phylogenetically highly interesting helminths. Aspects of their systematic position and host relations have been discussed by Stunkard (1917, 1946, 1962, 1963, 1970), Faust and Tang (1936), Williams (1942), Dollfus (1956, 1958a, b) and Rohde (1972).

Lophotaspis vallei (syn. *L. adhaerens*) is parasitic in the oesophagus and stomach of *Caretta caretta*. First reported under the name *Aspidogaster vallei* from a loggerhead taken off Corfou, Adriatic Sea (Stossich, 1899), it has subsequently been found in the same host species from Alexandria, Mediterranean Sea (Looss, 1901, 1902), from the Gulf of Mexico (Manter, 1932; Wharton, 1939) and from Santos, Brazil (Araujo, 1941). According to Lester and co-authors (1980; see legend accompanying their Fig. 2), *L. vallei* is common in the stomachs of *C. caretta* from Shark Bay, Western Australia. Thus far, it has not been recorded in other chelonians. Worms obtained from *C. caretta* were young and mature adults, 7 to 12 mm in length (Fig. 2-13, 1). Looss (1902), who found 20 of 25 Mediterranean loggerhead turtles to be infested with up to 50 of these worms, has given a very detailed description of the adult. Nothing is known about its pathology. It might be speculated that the large, powerful adhesive disc inflicts at least some injury upon the gastric mucosa to which the worm is firmly attached.

Manter (1932), who rediscovered *Lophotaspis vallei* in the original host at Dry Tortugas, Florida, contributed valuable information to the knowledge of the life cycle of this aspidogastrean. Eggs of *L. vallei* were found to hatch readily in sea water. The emerging larvae, regarded as 'miracidia', measured between 150 and 210 µm in length, depending on the state of contraction or expansion. There was a large sucker at the

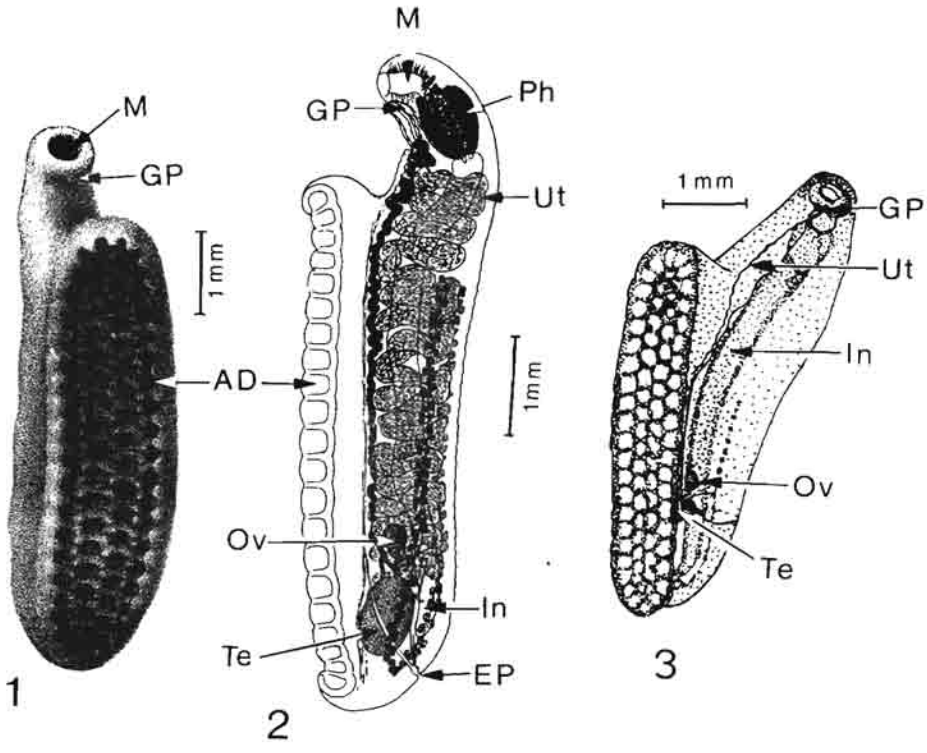


Fig. 2-13: *Lophotaspis vallei* from oesophagus and stomach of *Caretta caretta*. 1: Latero-ventral view of adult; 2: median section of adult; 3: juvenile individual ('nymph'). AD adhesive disc, EP excretory pore, GP genital pore, In intestine, M mouth, Ov ovary, Ph pharynx, Te testis, Ut uterus. (1 and 2 after Looss, 1902; 3 after Wharton, 1939.)

posterior end, and 2 eye spots occurred dorsally, about one-third from the anterior end, which carried an elongate oral sucker. The larva swam rapidly by means of its long cilia, which were limited to 3 supporting plates — 1 at the extreme posterior tip behind the posterior sucker, the other 2 laterally just behind mid-body. It could also creep in a leech-like fashion.

Eventually, Wharton (1939) discovered the postlarvae ('nymphs') of *Lophotaspis vallei* in the mantle cavity of the horse conch *Pleuroploca (Fasciolaria) gigantea* from Florida. The young worms (Fig. 2-13, 3) were essentially alike the adult, though smaller (about 5 mm long) and with reproductive organs not yet fully developed. No adult worms were seen in the conch. There is no redial or sporocyst stage in the Aspidogastrea and, hence, no asexual reproduction as in the Digenea. Development of the worms from egg to adult is direct.

Although the role of the horse conch as an intermediate host of *Lophotaspis vallei* has not been established, such a life-cycle pattern appears likely. However, it is also possible that *Pleuroploca gigantea* is the true definite host of *L. vallei,* and that infestation of the chelonian host occurs merely accidentally — a possibility considered by Stunkard (1963). In fact, numerous aspidogastreans reach sexual maturity in freshwater gastropods and bivalves (for references consult Rohde, 1972). *Aspidogaster conchicola,* unfolding its

entire life cycle in freshwater bivalves mainly of the genera *Anodonta* and *Unio*, is an example well known to every student of zoology. Freshwater turtles and fish have been reported as post-cycle hosts of this aspidogastrean (Faust, 1922; Van Cleave and Williams, 1943).

Other aspidogastreans reach sexual maturity in vertebrates and have only their larval stages in molluscs. Among the marine forms, *Lobatostoma manteri* — the only species for which the life cycle has been worked out more or less completely (Rohde, 1973) — utilizes gastropods *Planaxis sulcatus* and *Cerithium moniliferum* as intermediate hosts. The adult lives in the intestine of the marine teleost *Trachinotus blochi* (Rohde and Sandland, 1973; Rohde, 1975). Adults of *Lophotaspis* spp. have been described from freshwater turtles in North America and China (Ward and Hopkins, 1931; Faust and Tang, 1936), and immature worms have been recovered from marine gastropods *Melo (Cymbium)* sp. and pearl oysters *Pinctada margaritifera* from Australia and Ceylon (Monticelli, 1892; Shipley and Hornell, 1904). Stunkard (1963) pointed out that none of the turtle-infesting species of *Lophotaspis* (excepting *vallei*) have been recorded subsequently — a fact which suggests that they may be secondary or incidental parasites of these reptiles.

The same question arises with respect to the *Caretta caretta* — *Lophotaspis vallei* association. According to Morris (1973), *Pleuroploca gigantea* (previously known as *Fasciolaria gigas*), the gastropod host of *L. vallei* at Dry Tortugas, is a Caribbean species having its northern distributional border in North Carolina waters. Most of the *L. vallei* records are from outside the distributional range of this conch. The carnivorous Fasciolariidae comprise a large family, whose representatives are distributed worldwide in warm seas. Since *L. vallei* has a similar circumtropical and subtropical distribution, its larval stages must be expected to occur in other molluscs, possibly in fasciolariid gastropods, in other parts of the world ocean. The obviously low host specificity of aspidogastreans would favour the acquisition of new hosts and the dispersal of the parasite. However, additional records of larval *L. vallei* are lacking.

The digeneans of marine chelonians are typical amphibian or reptilian parasites. Turtles harbour no representatives of such common trematode families of fish as the Hemiuridae, Opecoelidae, Fellodistomidae, etc. Similarly, they have no digeneans in common with marine mammals. Remarkably numerous digeneans have been reported from marine chelonians. Lists assembled by Hughes and co-authors (1941, 1942) and Ruiz (1943, 1946) show that, up to that time, as many as 51 species of trematodes had been reported from *Chelonia mydas* alone in various parts of the world ocean. Additional species have since been described from this host. Manter (1954a, p. 346) concluded:

> "I believe no other species of host is known to harbor as many as 50 species of trematodes. Surely, no other reptile approaches this number."

Caretta caretta harbours at least 35 species of trematodes, of which 10 are also known from *Chelonia mydas*. At Dry Tortugas, Florida, as many as 15 species (14 digeneans, 1 aspidogastrean) have been described from *C. caretta*, although only a very limited number of hosts have been examined (Linton, 1910; Pratt, 1914; Manter, 1932, 1954a). In the Gulf of Mannar (India) alone, at least 14 species of trematodes have been recorded from *C. mydas* and 6 from *C. caretta* (Simha and Chattopadhyaya, 1970; Chattopadhyaya, 1972a, b; Rao, 1976). Sey (1977) reported 8 species (representing 8 genera) from a total of 37 *C. caretta* and 8 species (representing 7 genera) from 7 (!) *C. mydas* landed at the Alexandria (Egypt) fish market. Although the origin of these hosts

could not be determined, they were believed to have been caught along the Egyptian coasts. Gohar (1934, 1935) listed 25 species of trematodes as parasites of *C. mydas* and 16 as parasites of *C. caretta* from Egyptian waters. Considerably fewer species have been reported from *Eretmochelys imbricata* and *Dermochelys coriacea* (Hughes and co-authors, 1941; Manter, 1954a; Chattopadhyaya, 1972a b; Threlfall, 1979). In Puerto Rican waters, however, *E. imbricata* is host to at least 28 species of digeneans belonging to 20 genera and 10 families (Fischthal and Acholonu, 1976).

Most of the presently valid genera or species of marine-turtle trematodes have already been listed by Looss (1902) in his extensive monograph, although some of these have been shifted between higher taxonomic levels. Representatives of the following families have been reported from marine turtles: Pronocephalidae, Paramphistomatidae, Plagiorchidae, Brachycoeliidae, Angiodictyidae (Microscaphidiidae), Rhytidodidae, Gorgoderidae, Auridistomatidae and Spirorchidae. Most of these are parasitic in the chelonians' intestinal tract; a few have been reported from the gallbladder, the urinary bladder, or the heart and blood vessels. Next to nothing is known about their pathology.

The majority of the digeneans recorded from marine turtles are representatives of the Pronocephalidae, a family largely confined to chelonians. Prominent genera are *Pronocephalus, Cricocephalus, Diaschistorchis, Pleurogonius, Medioporus* and *Pyelosomum.* Most of the known species inhabit the intestine or stomach; some *Pyelosomum* spp. occur in the urinary bladder. Pronocephalids have been reported from *Caretta caretta* in the Gulf of Mexico and at Bermudas (Linton, 1910; Pratt, 1914; Luhman, 1935), from *C. caretta* and *Chelonia mydas* in the Mediterranean, the Indian Ocean, and elsewhere (Braun, 1899a, b, 1901; Looss, 1899, 1901, 1902; Chattopadhyaya, 1972a, b; Sey, 1977), from *C. mydas* in India, Australia, Japan, Nicaragua, Panama, Brazil and the Caribbean (Stephens, 1912; Johnston, 1913; Kobayashi, 1920; Teixeira de Freitas and Lent, 1938; R. K. Mehra, 1939; Caballero y Caballero, 1954; Caballero y Caballero and co-authors, 1955; Gupta, 1961; Mehrotra and Gupta, 1976; Groschaft and co-authors, 1977; Groschaft and Tenora, 1978), from *Chelonia 'japonica'* and *Eretmochelys 'squamosa'* in Japan (Oguro, 1936), from *E. imbricata* in the Gulf of Mexico, in India, Australia and the Bermudas (Johnston, 1913; Barker, 1922; Prudhoe, 1944; Chattopadhyaya, 1972a, b; Fischthal and Acholonu, 1976; Mehrotra and Gupta, 1976) and from *Dermochelys coriacea* in Newfoundland (Threlfall, 1979). H. R. Mehra (1932), R. K. Mehra (1939) and Ruiz (1946) have given general accounts of the Pronocephalidae and have summarized the previous voluminous literature on this family.

The Pronocephalidae are medium-sized to large (up to 10 mm) monostome flukes. As they are equipped with a strong, muscular oral sucker, they must be regarded as being predisposed to cause at least some degree of host pathology. The bright blood-red colour displayed by some species might suggest that they ingest host blood. Sey (1977) observed epithelial destruction of the intestinal mucosa in *Caretta caretta* infested with *Pleurogonius trigonocephalus.* The lesions were covered with caseous debris. Ten of 33 loggerheads harboured these digeneans; numbers ranged from 22 to 173 per host. None of 7 *C. mydas* examined harboured *P. trigonocephalus.*

Although the taxonomic literature referring to the Pronocephalidae is remarkably voluminous, next to nothing is known about their host relations and life cycles. La Rue (1957) included the Pronocephalidae, together with the Notocotylidae, in the newly erected superfamily Notocotyloidea, mainly on the basis of morphological and anatomical

similarities between the cercariae. Notocotylids are parasites of birds. Their cercariae are typically monostomate, apharyngeate and ocellate, have an excretory vesicle with anteriorly fused branches and protrusible cup-shaped attaching structures situated postero-laterally (Fig. 2-14, 1). They develop in rediae in prosobranch gastropods and, upon emergence from the first intermediate host, encyst on solid surfaces in the open water. No second intermediate host is required (Lauckner, 1980). Cable (1956) described *Cercaria caribbea I* from costate horn shells *Cerithidea costata* from Puerto Rico, which is strikingly similar to the larvae of 'normal' notocotylids but distinctly larger (Fig. 2-14, 2). Cercariae of notocotylids described, for example, by Stunkard (1960, 1966, 1967a, b), have body lengths ranging from about 0.16 to 0.35 mm (0.25 mm on the average); the respective adults taken from the intestine of birds are 0.5 to max. 3.6 mm (average about 2.2 mm) in length. If *C. caribbea I* were a larval notocotylid, it would be exceptionally large.

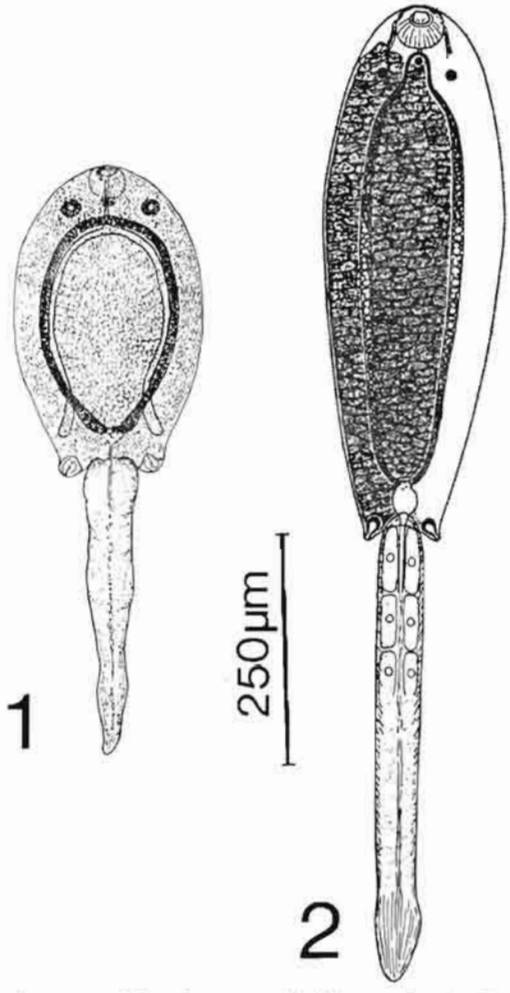


Fig. 2-14: Comparison of notocotylid and pronocephalid cercariae. 1: *Paramonostomum alveatum* from *Hydrobia salsa*; 2: *Cercaria caribbea I* from *Cerithidea costata*. (1 after Stunkard, 1967a; 2 after Cable, 1956.)

Nevertheless, Cable (1956) believed it to be the larva of a bird monostome. *C. caribbea I* was found to encyst on the bottom of the glass dish in which infested horn shells were crushed, as well as on the operculum of the snail host. The author (p. 504) concluded:

> "The ecology of the larva suggests that its adult is not one of the monostomes for which marine turtles are notorious, or either *Pleurogonius candibulus* or *Barisoma erubescens,* two monostomes that were common in angelfishes."

In contrast to Cable (1956), the reviewer feels that there are strong arguments in favour of the assumption that *Cercaria caribbea I* is not a notocotylid but a pronocephalid larva. The cercaria from *Cerithidea costata* is 0.56 to 0.59 mm in length (body *without* tail) and, hence, more than twice as long as an average notocotylid cercaria. Although there is no definite correlation between larval length and adult size in the Digenea, the large size of *C. caribbea I* suggests a relationship with the larger adult pronocephalids (5 to over 10 mm) rather than to the relatively small (0.5 to 3.6 mm) adult notocotylids.

Furthermore, the first intermediate host of *Cercaria caribbea I, Cerithidea costata,* is suspicious. There are many species in that genus, which is widespread and abundant throughout warm seas around the world. Horn shells inhabit shallow waters where they feed on detritus and algae. Pronocephalid cercariae that emerge from the snails could attach to, and encyst on, the vegetation — sea weeds on which marine turtles are known to feed to a large extent. A life-cycle pattern of this kind could explain the ubiquity and great abundance of pronocephalids in chelonians. Another indication suggesting infestation *via* metacercariae encysting on vegetation is the occurrence of pronocephalids in the intestine of the Galapagos marine iguana *Amblyrhynchus cristatus,* which also feeds on sea weeds. Gilbert (1938) described 3 species of pronocephalids from *A. cristatus* captured at Albemarle Island, Galapagos, all of which represented genera which were new to science and not represented in chelonians.

Hunter (1967) described a pronocephalid cercaria from mud snails *Nassarius obsoletus* collected in the Beaufort (North Carolina, USA) area, which she believed to be the larval stage of *Pleurogonius malaclemys,* described by her (Hunter, 1961) from freshwater turtles *Malaclemys terrapin centrata.* Infestations were established in *M. terrapin centrata* and *Chelonia mydas* with metacercariae encysted on the opercula of the host snails. Although Hunter's (1967) cercaria definitely appears to be a larval pronocephalid, her experimental procedures are somewhat dubious and the results are not fully conclusive because at least in the terrapin the experimental infestation was superimposed on a natural one. With respect to Cable's (1956) *Cercaria caribbea I,* the author (p. 40) stated that "it is the only one definitely described as a marine pronocephalid". This statement is incorrect. Cable merely listed it as a "monostome cercaria". As stated above, he believed it to be the larval stage of a bird trematode, which would include it among the notocotylids, not the pronocephalids.

The Paramphistomatidae comprise another important group of amphibian and reptilian parasites. Both the cercariae and the adults are amphistomate, i. e., provided with a conspicuous terminal sucker. All (non-marine) species with a known life history have a two-host cycle similar to that of the Pronocephalidae; the cercariae encyst on the substrate. The Paramphistomatidae are widely distributed intestinal parasites of marine turtles. Numerous species have been reported from *Caretta caretta, Chelonia mydas, C. 'japonica'* and *Eretmochelys imbricata* in various parts of the world ocean (Looss, 1899, 1901, 1902, 1912; Braun, 1901; Travassos, 1934; Teixeira de Freitas and Lent, 1938; Nigrelli, 1941;

Caballero y Caballero and co-authors, 1955; Fischthal and Kuntz, 1975; and others). Their life cycles are unknown, and nothing has been reported on their pathology.

Cymatocarpus undulatus, a representative of the Brachycoeliidae, has been found in chelonians in geographically widely separated waters. First described by Looss (1899) from a loggerhead turtle taken off Abukir, Egyptian Mediterranean coast, *C. undulatus* appears to be specific to *Caretta caretta.* Linton (1910), Pratt (1914) and Luhman (1935) reported it from this host in the Gulf of Mexico. Another member of the genus, *C. solearis,* parasitizes *Chelonia mydas* (Braun, 1901). Individuals of the latter species, described from museum material of unknown geographic origin, are considerably smaller (ca. 2 mm) than *C. undulatus* (over 7 mm).

Linton (1910) recovered "very numerous, approximately 3,000" individuals of *Cymatocarpus undulatus* from the intestine of a single loggerhead turtle captured at Dry Tortugas, Florida. Another turtle harboured 'numerous' worms, a third one 130 specimens. The digestive tract of the heavily infested turtle contained no food, but the intestinal mucosa as far as the rectum was thickly 'peppered' with the worms. The conspicuous dark colour of the trematodes was due to the masses of eggs filling the posterior body portion. The alimentary tract of the second turtle contained fragments of sea weed and *Palinurus* carapaces, that of the third turtle 'vegetable debris'.

The large number of *Cymatocarpus undulatus* recovered by Linton (1910) from a single *Caretta caretta* suggests infestation *via* an intermediate host in which numerous metacercariae had accumulated. The finding of *Palinurus* remains in the stomach of one of the other turtles furthermore suggests the spiny lobster as intermediate host. In fact, Dollfus (1927) reported on the occurrence of metacercariae found encysted in the abdominal musculature of a hermit crab *Pagurus tinctor (P. varipes)* from the Persian Gulf. The cysts were thin-walled, ovoidal in shape, and measured from 0.7 × 1 mm to 1.7 × 2.3 mm. Some 50 metacercariae were recovered from the infested crab. Worms excised from their cysts (Fig. 2-15) were particularly large (up to 7.8 mm long) and showed advanced development. They were readily identifiable as a member of the genus *Cymatocarpus.* Dollfus did not hesitate to assign them to *C. undulatus.* According to Pratt (1914), *C. undulatus* is by far the most numerous and the most constant trematode of *C. caretta* in the Gulf of Mexico. In the Mediterranean, on the other hand, it does not seem to be common (Looss, 1899, 1902).

Orchidasma amphiorchis, another brachycoeliid, has a similarly wide geographic range. It has been reported from the intestinal tract of *Caretta caretta* and *Chelonia mydas* in waters of Brazil, Florida, Japan, Egypt and Italy (Braun 1899a, 1901; Looss, 1899, 1901, 1902; Linton, 1910; Luhman, 1935; Teixeira de Freitas and Lent, 1938; Sey, 1977). Baylis (1928) recorded it from a loggerhead turtle taken as far north as the Lancashire (England) coast. Nothing has been reported on the pathology or the life cycle of *O. amphiorchis.*

Enodiotrema schikhobalovae, a plagiorchid, has been described from *Eretmochelys imbricata* in the Gulf of Mannar, India. More than 200 worms, 0.9 to 1.3 mm in length, were recovered from the oesophagus and stomach of a single hawksbill turtle (Gupta and Mehrotra, 1976). Other plagiorchids are known from *Caretta caretta* and *Chelonia mydas* of different geographic origin (Braun, 1901; Looss, 1899, 1901, 1902; Linton, 1910; Pratt, 1914; Luhman, 1935; Nigrelli, 1941; Euzet and co-authors, 1972; Groschaft and co-authors, 1977; and others). All are parasitic in the alimentary tract of the turtles.

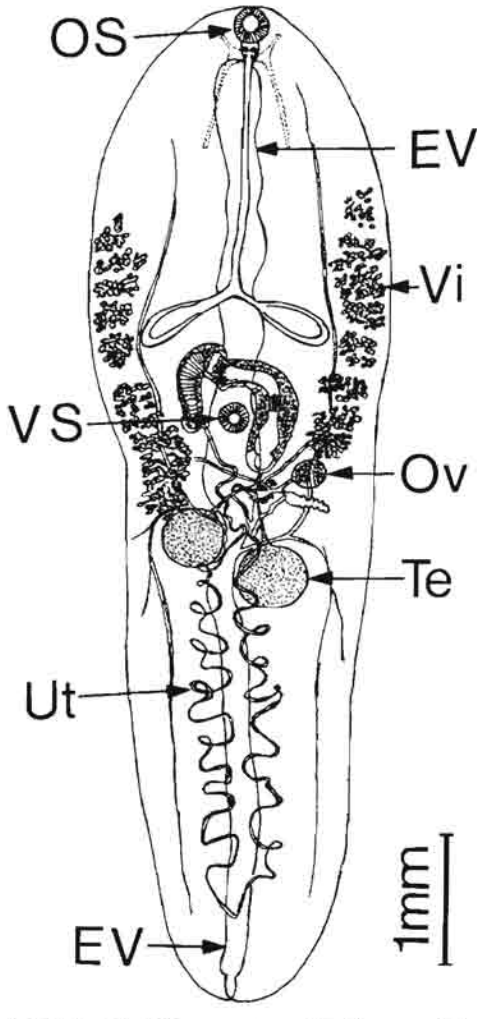


Fig. 2-15: *Cymatocarpus (undulatus?).* Metacercaria excised from cyst in abdominal musculature of *Pagurus tinctor.* EV excretory vesicle, OS oral sucker, Ov ovary, Te testis, Ut uterus, Vi vitellaria, VS ventral sucker. (After Dollfus, 1927.)

Gorgoderids *Phyllodistomum cymbiforme* and pronocephalids *Pyelosomum cochlear* parasitize in the urinary bladder. The life cycles of all of these are unknown. Cercariae of freshwater gorgoderids belong to the 'Cysticercous' or 'Macrocercous' groups. Those of the marine representatives might perhaps be similar.

Some members of the Rhytidodidae inhabit the intestine, others the gallbladder of chelonians (Price, 1939). Heavy *Rhytidodoides similis* infestation was found to be associated with a papillomatous disease of the gallbladder in *Chelonia mydas.* The majority of the flukes, which have an average size of 2.2 × 0.97 mm, lie free in the dark green, thickened and often inspissated bile, but sometimes are attached to the papillomatous parts of the bladder's mucous membrane (Fig. 2-16) or are partly buried in the mucosa.

Worm-inhabited gallbladders may show a wide range of lesions doubtlessly depending

Fig. 2-16: *Rhytidodoides similis* attached to papillomatous lesion in mucous membrane of *Chelonia mydas* gallbladder. (After Smith and co-authors, 1941.)

upon the intensity and duration of the infestation and the susceptibility of the tissues of the host. Any part of the bladder fundus may exhibit thickened papillomatous change in solitary patches or in confluent irregular masses. The most conspicuous type of lesion consisted of papillomatous hyperplasia of the mucous membrane near the entrance of the cystic duct into the gallbladder. In consequence, the cystic end of the bladder may appear greatly thickened and encroaching upon the bladder lumen almost to the point of producing a stenosis or obstruction (Fig. 2-17). Sometimes, the entire gallbladder wall participates in the lesion, the epithelium being thrown up into papillomatous folds (Fig. 2-18). The affected muscularis then shows increased amounts of connective tissue and lymphoid-cell infiltration, and the subserosa may be oedematous and thickened, and contain a large number of dilated capillaries, both vascular and lymphatic. All sorts of irregularly dilated glands are formed in the hyperplastic epithelium. Frequently, glands penetrate down into the submucosa. However, all these growths appeared to be benign.

Yellowish, thick-shelled, embryonated parasite ova, measuring on the average 36 × 72 μm and being surrounded by epithelioid or even giant cells, may be found in the mucous membrane or other parts of the gallbladder wall (Fig. 2-19a). As a result of parasite-produced tissue irritation, mucus production is greatly enhanced (Fig. 2-19b). The characteristic lesions described were believed to arise from mechanical or 'chemical' factors related to the activities of *Rhytidodoides similis* (Smith and co-authors, 1941). Distinct *R. similis*-produced histopathology in the gallbladder of *Chelonia mydas* has also been reported by Caballero y Caballero (1954).

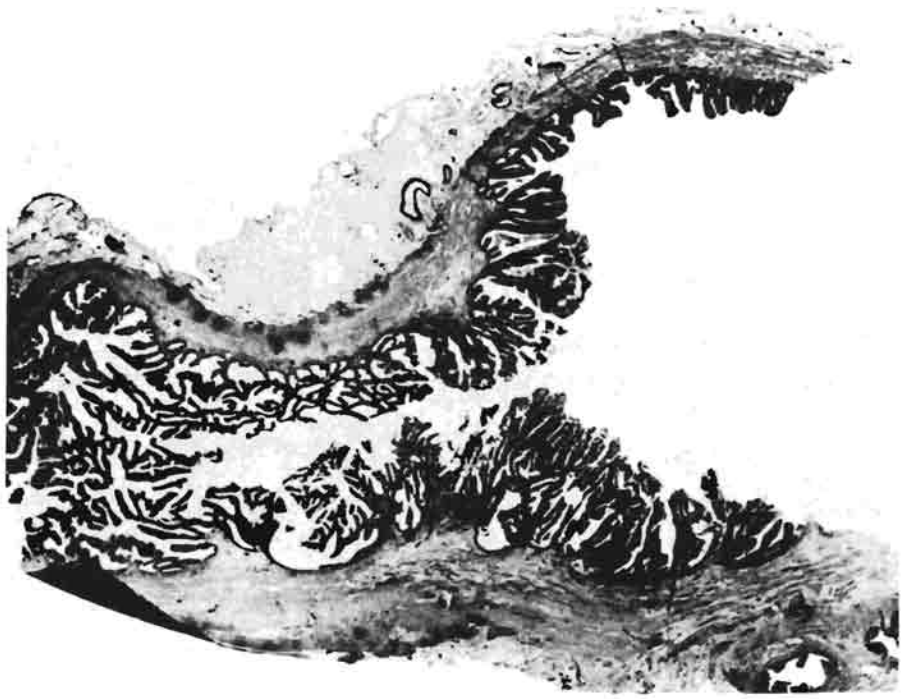


Fig. 2-17: *Chelonia mydas*. Pathology produced by *Rhytidodoides similis* in cystic end of gallbladder. Note stenosis resulting in near-obstruction of bladder lumen. (After Smith and co-authors, 1941.)

Auridistomatids *Calycodes anthos* were recovered from 4 of 33 *Caretta caretta* captured on the Egyptian coasts. From 2 to 8 worms, up to 19 mm in length, occurred in individual hosts. The trematodes, which were attached to the mucous membrane of the gallbladder, produced crater-like lesions and hyperplasia in the bladder epithelium. Loose epithelial cells and whole pieces of detached epithelium were seen surrounding the sites of attachment. Macroscopically, the gallbladder appeared markedly dilated and filled with greenish-brown, jelly-like bile. In the most heavily infested turtles, a deformation of the plastron was apparent (Sey, 1977).

Digeneans of the family Spirorchidae are parasitic in the blood-vascular system and heart of turtles. In contrast to the schistosome blood flukes of homoeothermic vertebrates, spirorchids are hermaphroditic. With a single exception, representatives of this group have not yet been reported from amphibians or non-testudine reptiles (Smith, 1972). Most known species have been described from freshwater turtles of the families Chelydridae, Emydidae and Trionychidae. Members of the genera *Amphiorchis, Carettacola, Haemoxenicon, Hapalotrema, Learedius, Monticellius, Neospirorchis* and *Squaroacetabulum* parasitize Cheloniidae and, with a single dubious exception, appear to be restricted to marine hosts.

As pointed out by Stunkard (1922), probably no other trematode group presents such an unusual specific variety and diversity as the blood flukes. However, with respect to the spirorchid trematodes of marine turtles, as well as sanguinicolid trematodes of marine fish,

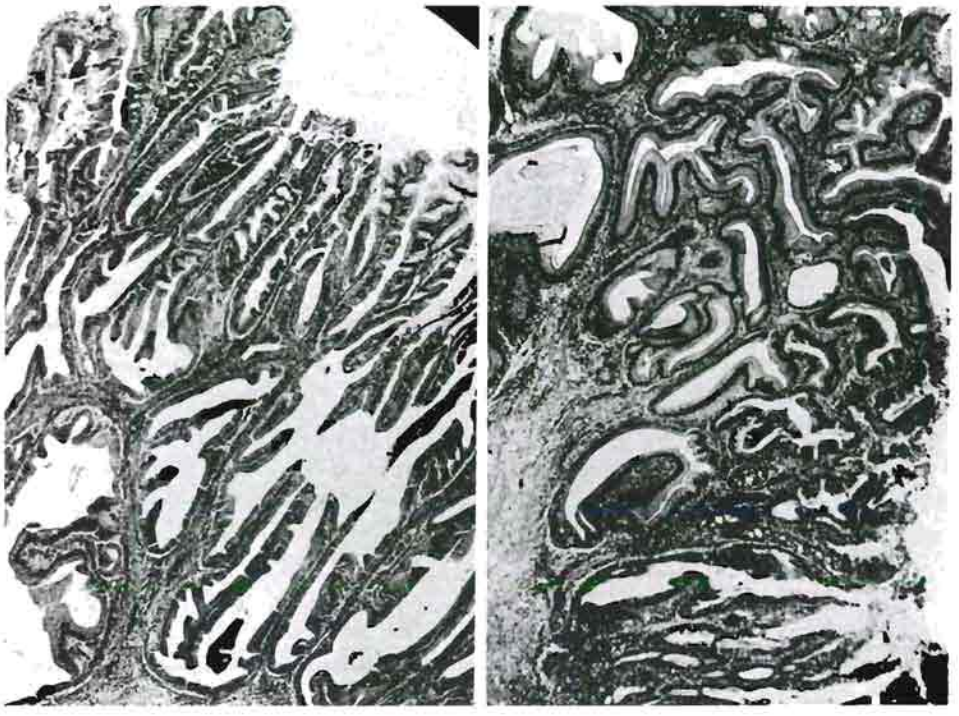


Fig. 2-18: *Chelonia mydas.* Heavy papillomatous proliferation of gallbladder epithelium resulting from irritation by *Rhytidodoides similis.* (After Smith and co-authors, 1941.)

records may be incomplete because — as emphasized by Manter (1963) and Smith (1972) — researchers often do not search for blood flukes.

Of the 20 known species (representing 3 subfamilies and 8 genera) of spirorchids infesting marine turtles, 12 (7 genera) have been reported from *Chelonia mydas,* 5 (3 genera) from *Caretta caretta,* 2 (2 genera) from *Eretmochelys 'squamosa'* and 1 from *Chelonia japonica.* A single species, *Hapalotrema loossi (H. constrictum)* is known to invade more than 1 host species, i. e., *C. caretta* and *C. mydas* (Leared, 1862; Monticelli, 1896; Looss, 1899, 1902; Gohar, 1934, 1935; Price, 1934; Luhman, 1935; Oguro, 1938; H. R. Mehra, 1939; Nigrelli, 1940, 1941; Takeuti, 1942; Manter and Larson, 1950; Martin and Bamberger, 1952; Caballero y Caballero and co-authors, 1955; Simha and Chattopadhyaya, 1970; Rao, 1976; and others). Smith (1972) has given an excellent account of the Spirorchidae. Consult this author for numerous references.

Hapalotrema constrictum (Fig. 2-20), originally named *Distomum constrictum* by Leared (1862), is the first-discovered turtle-blood fluke described, however, from immature specimens recovered in considerable number from the heart of an 'edible' or 'common' turtle. With respect to the unusual site of the worms, Leared (p. 170) erroneously concluded:

> "These immature worms being found in the heart, would imply that they were in the act of migrating, as we can hardly suppose the cavities of the organ to have been their resting-place."

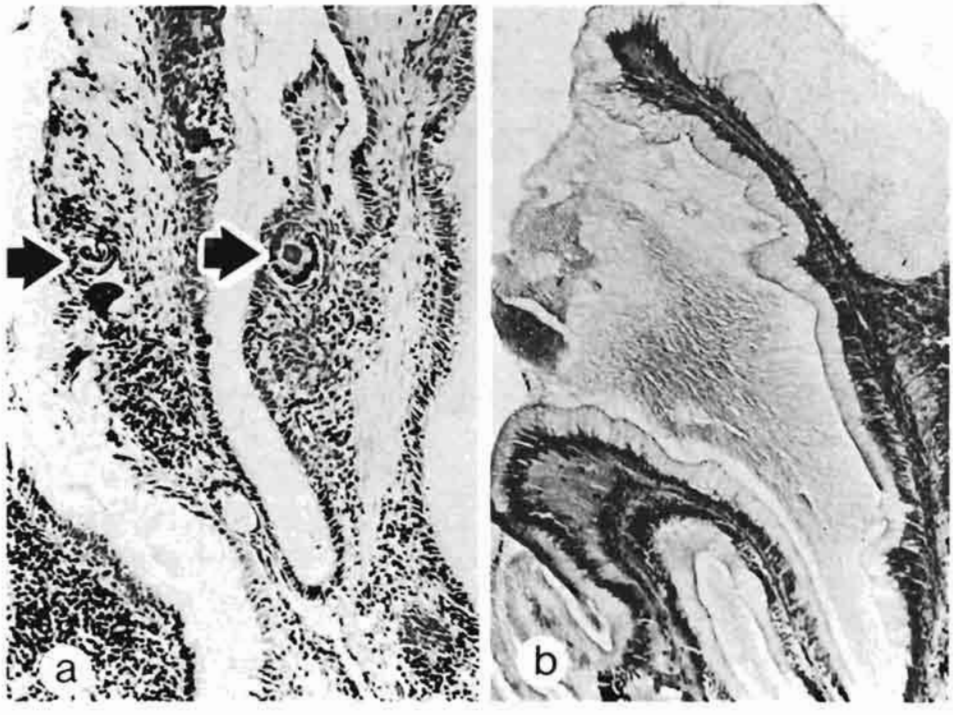


Fig. 2-19: *Chelonia mydas*. (a) Ova (arrows) of *Rhytidodoides similis* embedded in papillomatous tissue of trematode-infested gallbladder; (b) parasite-stimulated hypersecretion resulting in abundant mucus production. (After Smith and co-authors, 1941.)

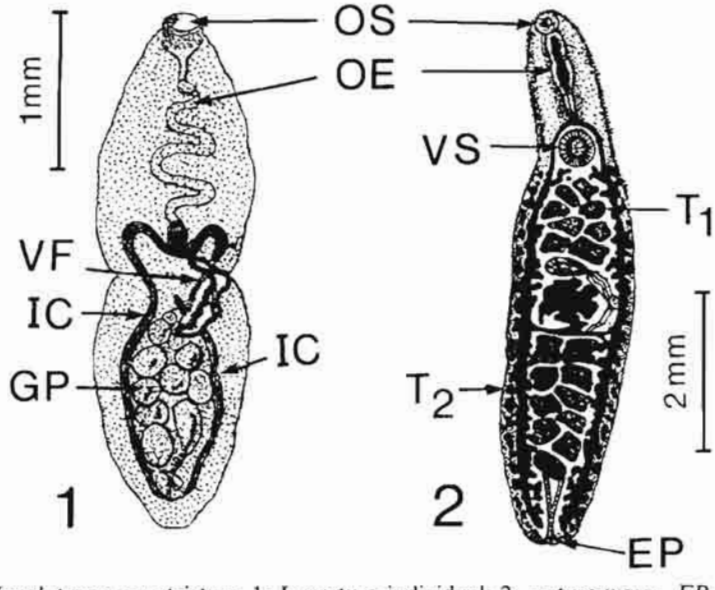


Fig. 2-20: *Hapalotrema constrictum*. 1: Immature individual; 2: mature worm. *EP* excretory pore, *GP* genital primordium, *IC* intestinal caecum, *OE* oesophagus, *OS* oral sucker, $T_{1,2}$ Testes 1 and 2, *VF* ventral sucker, folded, *VS* ventral sucker. (1 after Leared, 1862; 2 after Looss, 1899.)

Neither the origin nor the scientific name of the host turtle were given by the British author. Without indicating the origin of his information, Stunkard (1923) referred to Leared's turtle as *'Thalassochelys corticata' (= Caretta caretta),* but Smith (1972) argued that there are good reasons to believe that it was an individual of *Chelonia mydas.* Similarly, Brongersma (1972), an authority in this field, stated (p. 178) that "in English this species [i. e., *C. mydas*] is also known as the Edible Turtle". Common names of this species in German, Norwegian and Spanish clearly support this contention. Thus, there can be no doubt that *C. mydas* is the type host of *Hapalotrema constrictum.*

Monticelli (1896), who refound Leared's (1862) *Chelonia mydas* blood fluke in Mediterranean *Caretta caretta,* transferred it to the genus *Mesogonimus* and presented a detailed description of its structure. Looss (1899) eventually made it the type species of a new genus, *Hapalotrema. H. constrictum* is by far the most abundant trematode infesting Mediterranean *C. caretta.* Looss (1902) found the characteristic eggs of this species in the tissues of 19 of 20 loggerheads examined. The eggs were also detected in *C. mydas,* even in very small (25 to 30 cm carapace length) individuals.

According to Smith (1972), there is no evidence among the spirorchids of strict 'phylogenetic host specificity' at either generic or specific levels. With respect to the 10 spirorchid species (in 7 genera) reported from *Chelonia mydas,* the author feels that the validity of these taxa merits further investigation.

All freshwater spirorchids with a known life history have a 2-host cycle; their cercariae develop in sporocysts parasitizing gastropods. In spite of the fairly numerous records of adult chelonian blood flukes, not a single marine spirorchid life cycle has as yet been elucidated. As early as 1896, Monticelli, studying *Hapalotrema ('Mesogonimus') constrictum* (the first chelonian blood parasite discovered by Leared, 1862), predicted that *Cercaria dichotoma* might be the larval form. The latter designation is indeed a collective name applied to several unrelated cercariae (see Lauckner, 1983). However, Wall (1940, 1941a, b), who experimentally elucidated the first spirorchid life cycles (i. e., those of *Spirorchis parvus* and *S. elephantis* parasitic in freshwater turtles *Chrysemys picta*), confirmed Monticelli's (1896) suspicion. The larvae of *S. parvus* and *S. elephantis* are furcocercous, brevifurcate, apharyngeate, distome cercariae, which develop in sporocysts in planorbid gastropods *Helisoma* spp. (Cort, 1917). Similar spirorchid larvae have been described from other freshwater snails (O'Roke, 1917; Soparkar, 1921; Sewell, 1922; H. M. Miller, 1924, 1926; E. L. Miller, 1936; Larson, 1961; and others), but have not yet been associated with adult forms (Smith, 1972). Johnston and Beckwith (1947) suspect that several other cercariae, described by Soparkar (1921), McCoy (1929) and Croft (1933) from planorbid and lymnaeid freshwater snails and believed to be cercariae of sanguinicolid blood flukes (see Lauckner, 1983) are, in fact, larval spirorchids. Cercariae of marine spirorchids remain to be discovered. They probably also develop in molluscs, presumably gastropods.

Aside from mere descriptions of adult worms, studies on the life cycles of marine spirorchids are hampered, at least in part, by the fact that the chelonians constitute a group of highly endangered species. Experimental hosts may not be available in sufficient numbers and, if so, should not be sacrificed needlessly. Methods of *in vitro* maintenance and cultivation, as developed with partial success for freshwater spirorchids (Fried, 1965; Fried and Fee, 1964, 1968; Fried and Tornwall, 1965, 1969) open an avenue along which study of marine spirorchids could proceed in the future. Such studies should include the

experimental exposure of selected marine gastropods (suspect of serving as possible first intermediate hosts) to spirorchid miracidia, as successfully accomplished by Goodchild and Fried (1963), Goodchild and Dennis (1967) and Holliman and co-authors (1971) in freshwater snails.

The pathology of adult spirorchids has, thus far, almost exclusively been studied in freshwater turtles. Infestation of *Chrysemys picta* with *Spirorchis* spp. caused severe emaciation, debilitation or even death, depending on worm site and intensity of infestation (Wall, 1941b; Holliman and Fisher, 1968). Heavy brain and spinal cord infestation may lead to necrotic degeneration of nervous tissues with resultant contralateral hemiplegia and loss of reflex ability to withdraw the head (Fisher, 1968; Holliman and Fisher, 1968; Holliman and co-authors, 1971). Similar pathology must be expected to result from spirorchid infestation in marine turtles but remains to be studied. Looss (1902) merely stated that the blood-vessel walls of *Caretta caretta* at the sites of *Hapolotrema constrictum* lodgement were thickened and exhibited irregular protuberances protruding into the vessel lumen. Eggs released by the worms are distributed to various organs *via* the blood stream and may considerably contribute to pathology.

Canton (1861) found yellowish fusiform eggs with long polar filaments (Fig. 2-21) — obviously spirorchid ova and probably those of *Hapalotrema constrictum* (see below) — to adhere firmly to the conjunctivae of the eyes of a 'common turtle'. The eggs measured about 335 μm in total length, including the polar filaments. Like Leared (1862), Canton did not indicate the origin and the scientific name of the turtle. He probably dealt with an individual of *Caretta caretta.* Judging from a list of records assembled by Brongersma (1972), the loggerhead turtle appears to be by far the most common chelonian in North European waters. It has been reported from waters as far north as Shetland, Norway and even Russia (Murmansk, Barents Sea). According to Brongersma (1972, p. 109), "in English this species is also known as the Common Loggerhead". Although having recovered only immature individuals of *H. constrictum* from the heart of *Chelonia mydas,* Leared (1862) observed the presence of trematode ova in the heart blood of the turtle carrying the immature worms. He stated that they were of the same kind as those described and figured by Canton (1861). Obviously, Leared (1862) had overlooked the producers of these eggs, which reach only about 6.5 mm in length when fully mature (Fig. 2-20). Looss (1902) reported on the presence of eggs of *H. constrictum* and 3 other trematodes in blood vessels and tissues of *C. caretta* and *C. mydas,* but was unable to find adult worms other than *H. constrictum.* Fusiform eggs of *Haemoxenicon chelonenecon,* "with a spine at each end", have been observed in large numbers in various tissues of *C. mydas* (Martin and Bamberger, 1952). Smith and Coates (1938) described fibro-epithelial growths of the skin of *C. mydas* (p. 612), which they subsequently (Smith and Coates, 1939) found to be associated with the presence of spirorchid eggs. Similarly, Smith and co-authors (1941) observed papillomatous growths in the gallbladder of green turtles parasitized by spirorchids. Heavy *Learedius learedi* infestations were detected in 65 % of the turtle tissues examined (Nigrelli, 1941). Severe pathology has also been found to be caused by spirorchid eggs in freshwater turtles (Goodchild and Dennis, 1967).

Although it would appear likely that spirorchid eggs like those of other trematodes normally are passed out with the faeces or urine of the host, the above reports indicate that turtle-blood fluke eggs frequently lodge in tissues which have no connection with the exterior. Holliman (1971) failed to detect worm ova in the faeces of turtles parasitized by

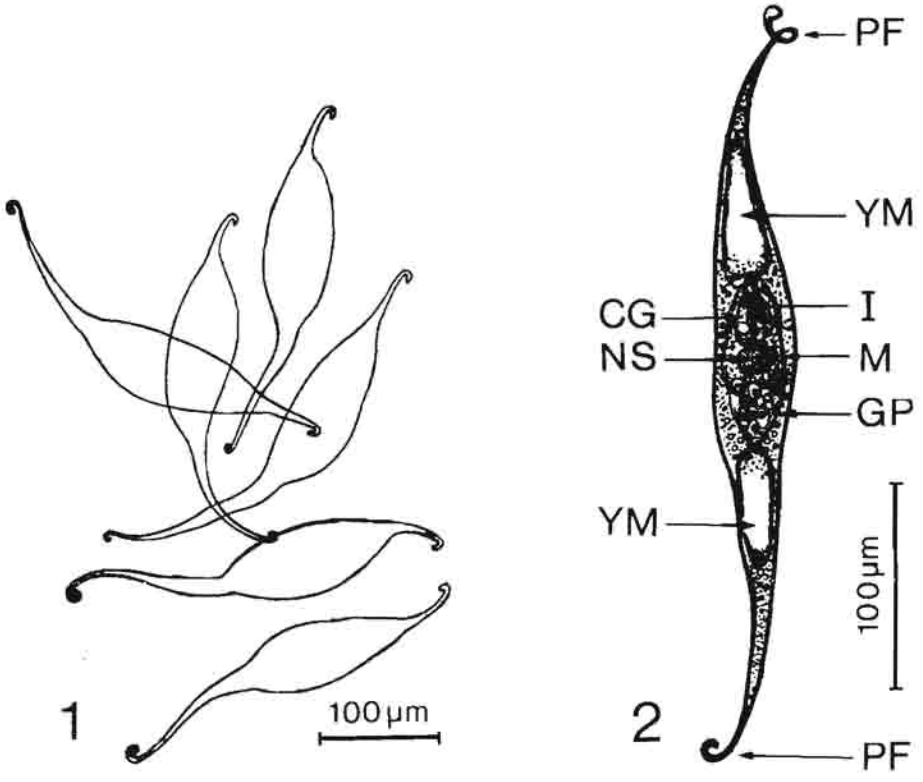


Fig. 2-21: *Hapalotrema constrictum*, eggs. 1: Gross appearance of ova from heart blood of *Chelonia mydas;* 2: structural details of mature egg. *CG* cephalic glands, *GP* genital primordia, *I* intestine, *M* miracidium, *NS* nervous system, *PF* polar filament, *YM* yolk mass. (1 after Canton, 1861; 2 after Looss, 1899.)

spirorchids for extended periods of time. He concluded that either faecal eggs may have been overlooked, or that egg release from infested tissues occurs upon death of the host and subsequent disruption of the turtles' tissues by scavengers.

Next to nothing is known about the turtles' defense mechanisms effective against infestation by Spirorchidae. Considerable irritation by the skin-invading cercariae has been described on several occasions. The avoidance behaviour in response to cercariae included eye blinking, eye wiping with the flippers, jaw snapping and abnormal body movements (Wall, 1941b; Goodchild and Kirk, 1960; Fisher, 1968; Holliman and Fisher, 1968; Holliman and co-authors, 1971). Martin (1967) demonstrated the presence of circulating antibodies in spirorchid-infested turtles. Whether such antibodies are capable of destroying cercariae is not known. Goodchild and Dennis (1967) observed host phagocytosis of *Spirorchis* sp. ova in experimentally infested *Chrysemys picta*. Trematode eggs present in the arteries or tissues became invested with defensive cells, which rapidly softened, penetrated and eventually destroyed the eggs. Macrophages, lymphocytes and fibroblast-like cells were most important in this process.

In contrast to the Galapagos marine iguana *Amblyrhynchus cristatus,* which shares the Pronocephalidae with the chelonians (see above), the Hydrophiidae have no trematode families in common with the other marine reptiles. Curiously, most digeneans

reported from sea snakes are hemiurids, which are known as common and widely distributed parasites of marine teleost fish.

Lecithochirium dillanei, the first hemiurid described from any member of the Hydrophiidae, has been recovered by Nicoll (1918) from the alimentary tract of an "unidentified sea snake *(Distira* sp.)" in North Queensland (Australia) waters. Manter (1947) regarded it as an uncertain species, and Yamaguti (1971) transferred it (probably incorrectly) to the genus *Sterrhurus.*

Bush and Holmes (1979) described *Sterrhurus carpentariae* from the stomach of *Lapemis hardwicki* collected in the Gulf of Carpentaria (Australia). Six of 71 sea snakes were found infested with 1 to 48 (average 15) of these worms.

Nothing is known about the life cycles of these sea-serpent trematodes. Both *Lecithochirium* and *Sterrhurus,* members of the hemiurid subfamily Lecithochiriinae, are well represented in teleosts, mainly in tropical and subtropical waters (Looss, 1907, 1908; Nicoll, 1914; Manter, 1934, 1947, 1954b; Jones, 1943; Crowcroft, 1946; Yamaguti, 1953, 1970, 1971; Manter and Pritchard, 1960; Reid and co-authors, 1965). However, the 2 species described from hydrophiids have not yet been reported from fish. As in other hemiurids, copepods very probably act as first intermediate hosts (for synopsis of literature consult Yamaguti, 1975). Infestation of sea snakes *via* copepods appears unlikely. The findings of Looss (1908) and Nicoll (1914) of encysted, progenetic (ovigerous) *Lecithochirium* spp. in various species of teleosts suggests that sea-snake infestation occurs by ingestion of cyst-carrying fish.

Hydrophitrema gigantica, a hemiurid of the subfamily Pulmoverminae, parasitizes in the tracheal lungs of sea snakes. It is a particularly large worm, adults measuring from 17 to 26 mm in length and from 3 to 4.8 mm in width across the acetabular region. Its body is thickly muscled and provided with a short acetabular peduncle and suckers surrounded by a conspicuous tegumental fold, the latter apparently serving as a supplementary attachment structure, anchoring the oral and ventral suckers into a single adhesive unit (Fig. 2-22, 1, 2). First described by Sandars (1960) from a southeastern Queensland (Australia) *Hydrophis elegans,* it has subsequently been reported from other hydrophiids in the Indo-Pacific region, namely *H. cyanocinctus* in Hong Kong, Taiwan and Malaya, *Kolpophis annandalei* in Indochina, *Aipysurus laevis* in Australia and *Enhydrina valakadyen* and *Microcephalopsis gracilis* in the Bay of Bengal (Vercammen-Grandjean and Heyneman, 1964; Coil, 1965; Madhavi and Hanumantha Rao, 1973, 1974; Ko and co-authors, 1975).

Sandars (1960) recovered 6 specimens of *Hydrophitrema gigantica* from a single *Hydrophis elegans* captured in Moreton Bay, Queensland. Of 17 *H. cyanocinctus* from Penang, Malaya, only 2 were positive and yielded a total of 14 flukes (Vercammen-Grandjean and Heyneman, 1964). In contrast, most *H. cyanocinctus* caught around Hong Kong and examined by Ko and co-authors (1975) were heavily infested with *H. gigantica.* A total of 4,639 worms were recovered from 180 (98.4 %) of 183 snakes. The intensity of infestation varied from 1 to 126 (!), with a median of 26 trematodes per infested snake. The worms varied from 5 to 25 mm in length, with a mean of 14.5 ± 2.2. mm (n = 2,404).

To explain such a high *Hydrophitrema gigantica* prevalence in the Hong Kong sea snakes would obviously require a profound knowledge of the biology of the host and the life cycle of the parasite. Unfortunately, neither of these aspects has been studied. According to Smith (1926) and McCosker (1975), *Hydrophis* spp. feed mainly, if not exclusively, on fish. Specimens of *H. cyanocinctus,* examined by Ko and co-authors

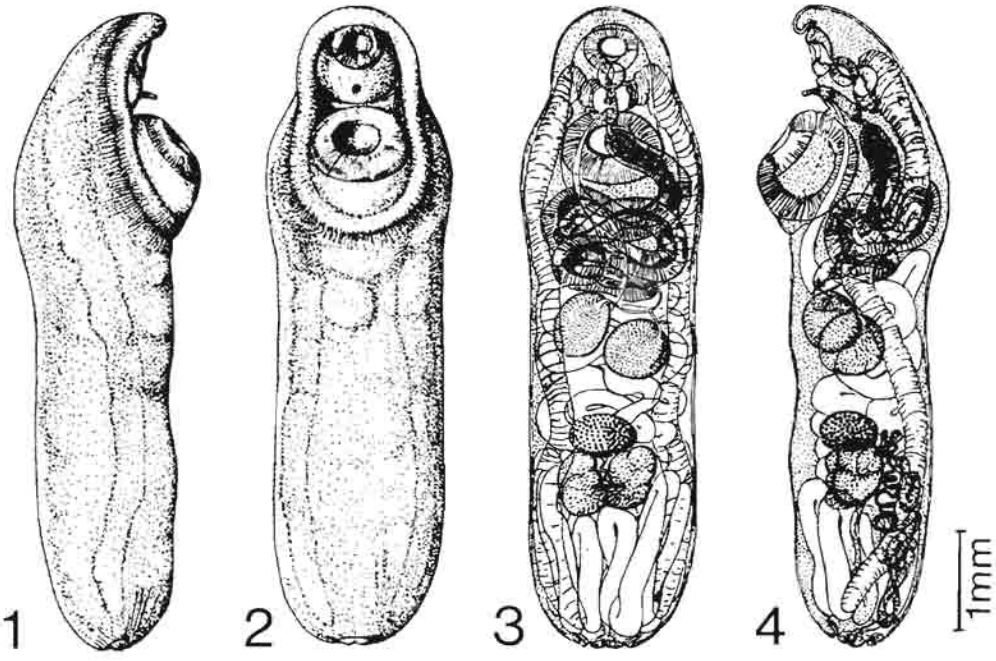


Fig. 2-22: *Hydrophitrema gigantica* from the lung of hydrophiid sea snakes. 1–2: External aspect, lateral and ventral views; 3–4: orientation of principal internal structures. (After Vercammen-Grandjean and Heyneman, 1964.)

(1975), usually had empty stomachs, but occasionally contained remains of snake eels *Ophichthys* sp. The authors considered it to be likely that fish may serve as second intermediate hosts of *H. gigantica*.

In *Hydrophis cyanocinctus,* the worms are usually located in the anterior region of the lung near the heart, i. e., close to the region with the highest vascularization. A dark-brown, mucoid exudate is commonly observed in the lumen of the infested lung. Pinhead haemorrhages occur at the *Hydrophitrema gigantica* attachment sites. Histologically, the intestinal caeca of the digeneans commonly contain a pinkish homogeneous material with haemosiderin. It was concluded that the worms feed mainly on host blood. The mucoid exudate in the lung lumen may contain tissue debris, macrophages and numerous parasite eggs (Fig. 2-23a). Histopathological damages include disruption of the lung epithelium and presence of red blood cells in the lamina propria close to the worm sites, focal necrosis in the mucosa, presence of macrophages with haemosiderin in the lamina propria and submucosa, and occasional hyperplasia of the lung mucosa at the attachment site of the worms' ventral sucker (Fig. 2-23b–e; Ko and co-authors, 1975).

Sandars (1961) erected the hemiurid subfamily Pulmoverminae to accomodate trematodes of the genera *Pulmovermis* and *Hydrophitrema.* The latter genus had previously been assigned by her (Sandars, 1960) to the subgenus Derogenetinae. *Pulmovermis cyanovitellosus,* the type species, has been described by Coil and Kuntz (1960) from *Laticauda semifasciata* caught at Botel Tobago off the southern tip of Taiwan. Eight of 13 hosts examined have been found infested. Like *Hydrophitrema gigantica, P. cyanovitellosus* invades the lungs. In size (max. 24.8 × 1.2 mm) it is only slightly smaller although

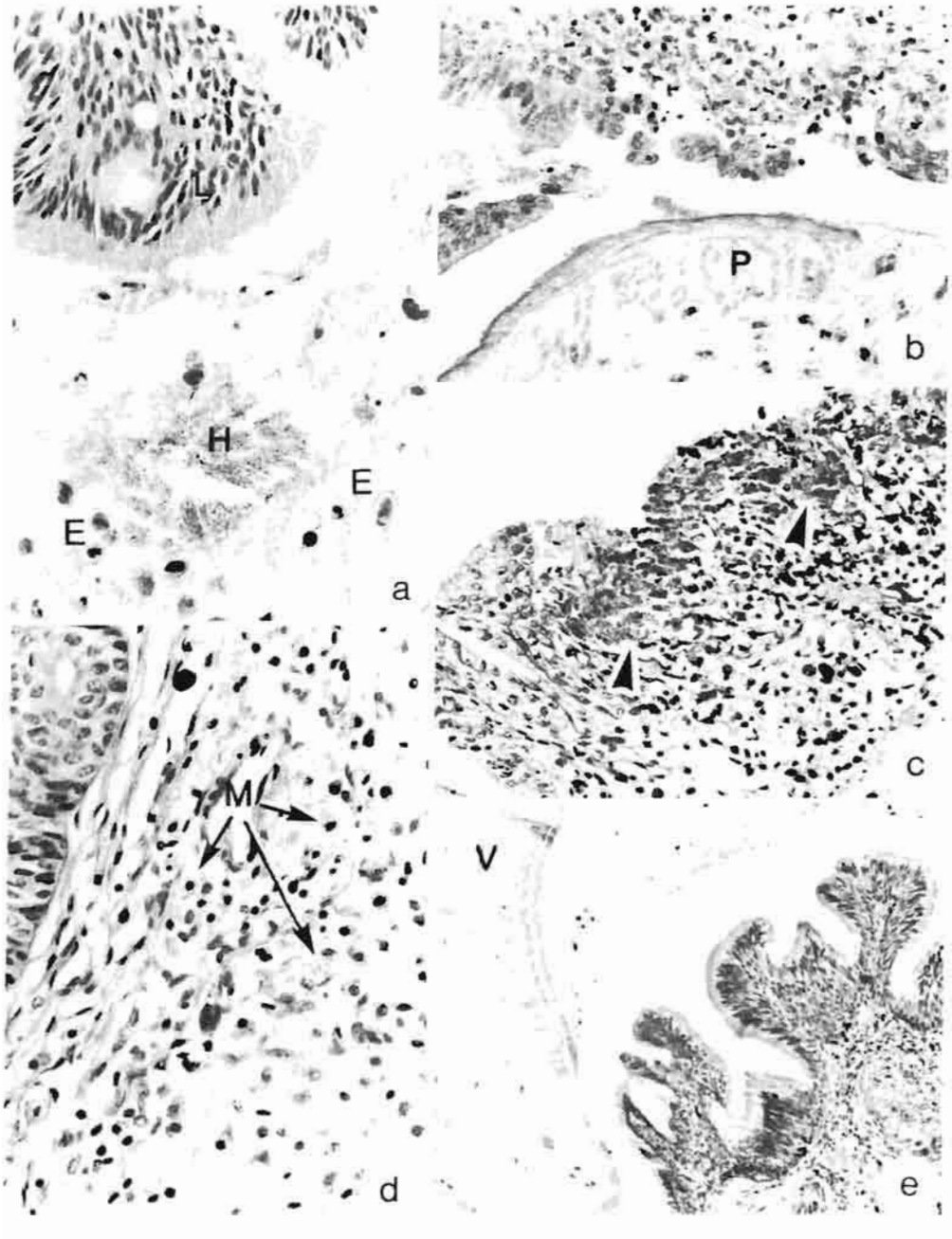


Fig. 2-23: *Hydrophis cyanocinctus*. Lung pathology produced by *Hydrophitrema gigantica*. (a) Presence of worm eggs (E) and haemosiderin (H) in mucoid exudate in lung lumen. L lung epithelium. × 360. (b) Disruption of lung epithelium. P body of parasite. × 225. (c) Eroded area of lung epithelium showing necrosis and haemosiderin (arrows). × 450. (d) Macrophages (M) in lamina propria. × 400. (e) Epithelial hyperplasia at attachment site of ventral sucker (V). × 90. (After Ko and co-authors, 1975.)

distinctly more slender (Coil and Kuntz, 1960; Vercammen-Grandjean and Heyneman, 1964). Nothing has been reported on the pathology of *P. cyanovitellosus,* but presumably it might be similar to that produced by *H. gigantica.*

Obviously unaware of the above publications, Telford (1967) described worms taken from the trachea, lung and air sacs of *Laticauda semifasciata* from Amami Island (Japan) as *Laticaudatrema amamiensis.* Forty-eight of 51 snakes were found to be infested with up to 33 worms per host. *L. amamiensis,* as described by Telford, is likely to represent *Pulmovermis cyanovitellatus* Coil and Kuntz, 1960. Gibson and Bray (1979) listed both *Hydrophitrema* Sandars, 1960, and *Laticaudatrema* Telford, 1967, as synonyms of *Pulmovermis* Coil and Kuntz, 1960 — a view not shared by other workers.

Further digeneans reported from sea snakes include opisthorchiids *Oesophagicola laticaudae,* recovered in "considerable number" from the lower portion of the oesophagus of *Laticauda laticaudata* from Isigaki Zima, Japan. The worms are relatively small, measuring 3.3 to 4.2 × 1.1 to 1.4 mm (Yamaguti, 1933). The same author described clinostomids *Harmotrema laticaudae,* 5.7 to 6.9 mm long × 1.0 to 1.2 mm wide, from the small intestine of the same snake host from the same locality. Telford (1967) reported *O. laticaudae* (in numbers ranging from 14 to 68) from 5 out of 6 *L. laticaudata,* and *H. laticaudae* (in numbers ranging from 0 to 33) from 10 out of 51 *L. semifasciata* from Amami Island (Japan).

Tubangui and Masiluñgan (1935) described opisthorchiids *Opisthorchis ophidiarum* from *Lapemis hardwicki* from the Philippines, and Coil and Kuntz (1960) described acanthostomids *Ateuchocephala marinus* from *Laticauda semifasciata,* and acanthocolpids *Ophiotreminoides orientalis* from *L. colubrina,* both collected at Botel Tobago. The relatively small worms (4.9 × 0.7 mm and 1.4 × 0.7 mm, respectively) inhabit the small intestine of their hosts. Unidentified digeneans have been reported from the trachea of *L. laticaudata* from New Caledonia (Nadchatram and Radovsky, 1971). The intermediate hosts and life cycles of all of these trematodes are unknown, and nothing has been reported on their pathology.

Agents: Cestoda

Marine reptiles appear to be remarkably free of cestode parasites. In his synopsis of the cestodes of vertebrates, Yamaguti (1959) lists various genera and species of tapeworms reported from terrestrial and freshwater reptiles, but does not mention a single record of a cestode known to parasitize marine reptiles. Joyeux and Baer (1936) merely mention pseudophyllideans *Ancistrocephalus imbricatus* (fam. Triaenophoridae) as tapeworms of *Caretta caretta* in the Atlantic and Mediterranean. Other members of this genus are parasitic in marine fish.

Specifically unidentified tetrarhynchidean neoplerocercoids have been found in 2 of 33 *Caretta caretta.* The turtles were obtained from the Alexandria (Egypt) fish market and believed to have been caught along the Egyptian coasts. The cysts enclosing the neoplerocercoids were oval and 2.0 to 2.5 × 1.3 to 1.8 mm in dimension. Large numbers of these cysts were attached, by means of strong connective-tissue fibres, to the serosa of the stomach and to the outer surface of the lungs. On the outside they were covered by stratified connective tissue. Parasite capsules, excised from the cysts, measured 1.0 to 1.4 × 0.8 to 1.0 mm; the contained larvae were 430 to 460 μm in length. Their morphology suggested affinities to *Lacistorhynchus* or *Eutetrarhynchus* (Sey, 1977).

Adult Tetrarhynchidea (Trypanorhyncha) are exclusively parasites of elasmobranchs. Postlarvae (tentaculo-neoplerocercoids) are mostly found in cephalopods and decapods (Vol. III), as well as in teleost fish; occasionally they occur in bivalves or gastropods. No complete trypanorhynch life cycle is presently known, but the currently available information suggests a considerable complexity of possible pathways (Cake, 1975; see also Vol. II, p. 773). Young (1954), Yamaguti (1959) and Mudry and Dailey (1971) presented evidence that the life cycle of *Lacistorhynchus* spp. involves teleosts as second intermediate hosts. Since fish are rare in the diet of *Caretta caretta* (Brongersma, 1972), it appears unlikely that Sey's (1977) larval trypanorhynchs were *Lacistorhynchus*. With *Eutetrarhynchus* the situation is different. Neoplerocercoids of this genus normally occur in molluscivorous decapod crabs (Dollfus, 1936, 1942). Pelseneer (1906, footnote p. 178) found larval *E. ruficolle* in oysters; Cake (1975, 1976) reported *Eutetrarhynchus* sp. neoplerocercoids from 4 species of bivalves and 5 species of molluscivorous gastropods (Vol. II); and Riser (in Dollfus, 1964) observed larvae of the same genus in cephalopods. All these invertebrate groups are part of the normal diet of *C. caretta* (Brongersma, 1972).

Sey (1977) discussed the possibility of *Caretta caretta* being a normal intermediate host for larval Tetrarhynchidea. Although there are numerous records of predation upon loggerheads by sharks, the final hosts of Tetrarhynchidea (Brongersma, 1972; Balazs, 1979), the natural involvement of *C. caretta* in the life cycle of these cestodes appears highly improbable. When consumed, together with the normal intermediate host, by an unsuitable host, trypanorhynch postlarvae usually leave the digestive tract of the latter and encyst or re-encyst in bladder-like structures formed as irritation products of the host's tissues. The occurrence of encysted trypanorhynchs at unusual sites, i. e., on the intestinal serosa and the outer surface of the lungs of the loggerheads examined by Sey (1977), identifies the turtle as a 'wrong' host for the neoplerocercoids. The latter author frequently found remains of crabs in the turtles' alimentary canal. It appears, therefore, reasonable to assume that *C. caretta* is an abnormal paratenic host for *Eutetrarhynchus* spp., accidentally infested by devouring crabs harbouring neoplerocercoids.

Agents: Nematoda

Stomach nematodes parasitizing marine turtles have been known for a long time. The most frequently reported and apparently cosmopolitanly distributed species is the ascaridoid *Sulcascaris sulcata*. In addition, oxyuroids *Kathlania leptura*, cucullanoids *Cucullanus carettae* and spiruroids *Echinocephalus* sp. occur in the alimentary tract of chelonians. Ascaridoids of the genera *Goezia* and *Paraheterotyphlum* have been reported from hydrophiid sea snakes.

Sulcascaris sulcata has originally been described from *Chelonia mydas* as *Ascaris sulcatus* by Rudolphi (1819). Unfortunately, Baylis (1923) transferred it to *Porrocaecum*, a genus erected by Raillet and Henry (1912) to accomodate *P. crassum*, a bird nematode, as type species. According to Hartwich (1957, 1959, 1975), species of *Porrocaecum* are confined to anseriform birds. Baylis' (1923) error probably originates from a misinterpretation of Raillet and Henry's (1912) paper. These authors discussed *Porrocaecum* in connection with nematodes described from iguanas and tortoises. Upon reexamination of Rudolphi's (1819) original material, Hartwich (1957) found it to differ from *Porrocaecum*, and consequently established the new genus *Sulcascaris* to accomodate *S. sulcata* as type

and sole species. His view was rejected by Allison and co-authors (1973), who declared *Sulcascaris* a junior synonym of *Porrocaecum,* but it was confirmed by Sprent (1977), likewise on the basis of a reexamination of Rudolphi's original specimens.

Sulcascaris sulcata is of cosmopolitan distribution in warm and temperate seas. Rudolphi (1819), Stossich (1895) and Sey (1977) reported it from chelonians in the Mediterranean and the northern Red Sea, respectively, Schneider (1866) from turtles in the Caribbean. Teixeira de Freitas and Lent (1946) found it in *Chelonia mydas* from Brazil, Sprent (1977), Lester and co-authors (1980) and Berry and Cannon (1981) in *Caretta caretta* from Queensland and Western Australia. From the occurrence of the larvae, which parasitize molluscs, primarily bivalves (Vol. II), a still wider distribution can be inferred. First noticed in pearl oysters *Margaritifera (= Pinctada)* spp. from Ceylon and described as *Ascaris meleagrinae* by Shipley and Hornell (1904), and as *Paranisakis pectinis* in *Pecten* sp. from North Carolina by Cobb (1930), larval *S. sulcata* have subsequently been recovered from diverse molluscs in various parts of the world's oceans (see Table 13-32 in Vol. II). These data indicate that *S. sulcata* is unspecific with respect to the selection of intermediate hosts — a fact which accounts for its worldwide occurrence.

The specific identity of the larval nematodes in molluscs remained obscure until Sprent (1977), Lichtenfels and co-authors (1977, 1978), Cannon (1978) and Payne and co-authors (1980) assigned the worms, together with similar ones from the stomach of chelonians, to the fourth stage of *Sulcascaris sulcata* by reverse designation from the adult, since earlier stages were not available for study. It remained to Berry and Cannon (1981) to experimentally elucidate the entire life cycle of *S. sulcata.* The authors failed to infest copepods exposed to eggs or larvae, but readily achieved larval infestations in bivalves and gastropods. The larvae (Figs 2-24, 2-25 and 2-26 c–e) were identical to those observed in naturally infested molluscs. Previously, Cannon (1977) had failed to find these worms in 123 species of fish from southeastern Queensland. Cannon (1978) was unable to experimentally infest teleosts and elasmobranchs with fourth-stage larvae, nor did he find worms in 5 species of rays. From this evidence it became clear the *S. sulcata* has a 2-host cycle. Berry and Cannon's (1981) study is the first demonstration of an anisakine life history from a reptile. In brief, it may be summarized as follows (Fig. 2-27; for details on the larvae parasitizing molluscs consult Lauckner, 1983).

Adult *Sulcascaris sulcata* parasitizing in the stomach of *Caretta caretta* and *Chelonia mydas* produce eggs, which are passed out with the faeces. The eggs are negatively buoyant in sea water and consequently sink to the bottom. Two moults occur in the egg to produce third-stage larvae, which hatch spontaneously. Further development of the third-stage larvae occurs in molluscs. After 3 to 4 months, when having attained a length of about 5 mm, the worms moult to fourth-stage larvae, which are infestive to the final host. When ingested by a turtle, fourth-stage larvae liberated from the tissues of the intermediate host attach at the final host's oesophago-gastric junction (Fig. 2-28) and moult to adults within 7 to 21 days. Subsequent growth to mature adults takes at least 5 months (Berry and Cannon, 1981).

Incidence and intensity of *Sulcascaris sulcata* infestation in chelonians may be high. Teixeira de Freitas and Lent (1946) found 'muitos' adult worms in a single individual of *Chelonia mydas* from Brazil. Six of 33 *Caretta caretta* from Egyptian waters harboured between 13 and 128 worms per host (Sey, 1977). Of 6 *C. caretta* from Shark Bay, Western Australia, 5 were found to be infested with *S. sulcata* ranging from 9 to 354 in number. All

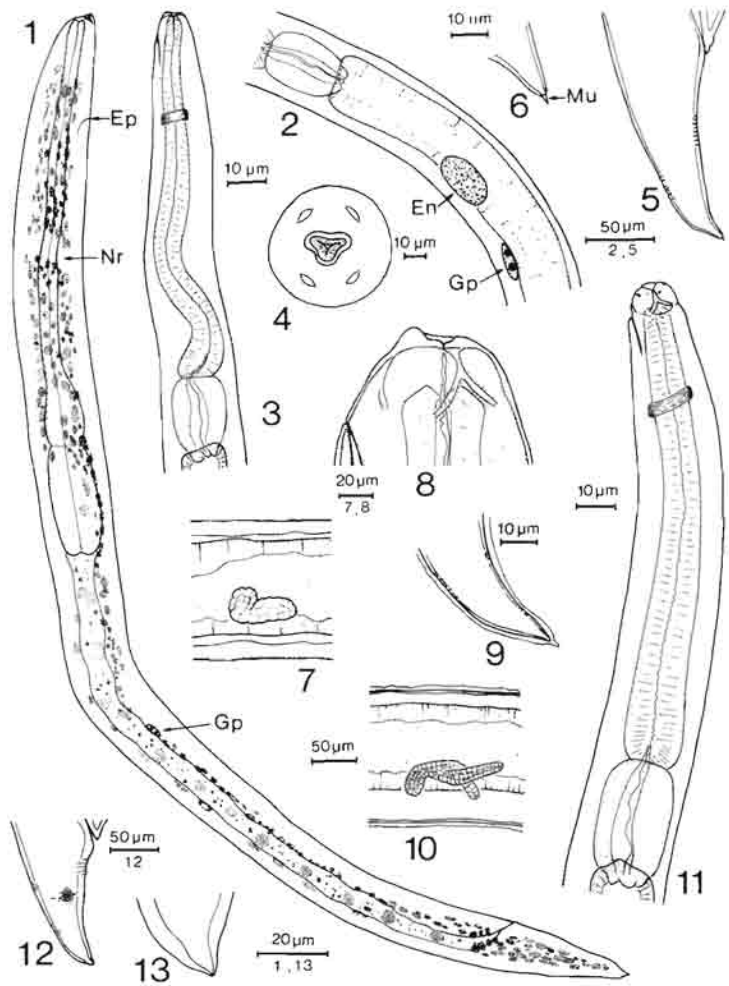


Fig. 2-24: *Sulcascaris sulcata*. 1: Third-stage larva from egg, stained with aceto-orcein to visualize nuclei; 2: third-stage larva from intermediate host *(Melina ephippium)* experimentally infested 55 days previously, lateral view of middle region; 3: third-stage larva from bivalve infested 86 days previously, oesophageal region; 4: same, *en face* view; 5: same, tail showing no apparent phasmid; 6: same, tip of tail showing mucron; 7: same, latero-ventral aspect of middle region exhibiting genital primordium (arrow indicates anterior end); 8: third-stage larva from bivalve host infested 115 days previously, about to moult into fourth-stage larva, head showing third-stage cuticle with fourth-stage lips forming below; 9: same, tip of tail with third- and fourth-stage cuticles; 10: same, latero-ventral aspect of middle region showing genital primordium and third- and fourth-stage cuticles (arrow: anterior end); 11: fourth-stage larva from bivalve host infested 115 days previously, oesophageal region; 12: same, tail showing phasmid; 13: same, tip of tail devoid of mucron. *En* excretory nucleus, *Ep* excretory pore, *Gp* genital primordium, *Mu* mucron, *Nr* nerve ringe. (After Berry and Cannon, 1981.)

hosts were non-gravid females measuring about 1 m in length (Lester and co-authors, 1980). Large numbers of fourth-stage larvae are commonly found with adults (Sprent, 1977).

Although Rudolphi's (1819) type material stems from the green turtle, loggerheads

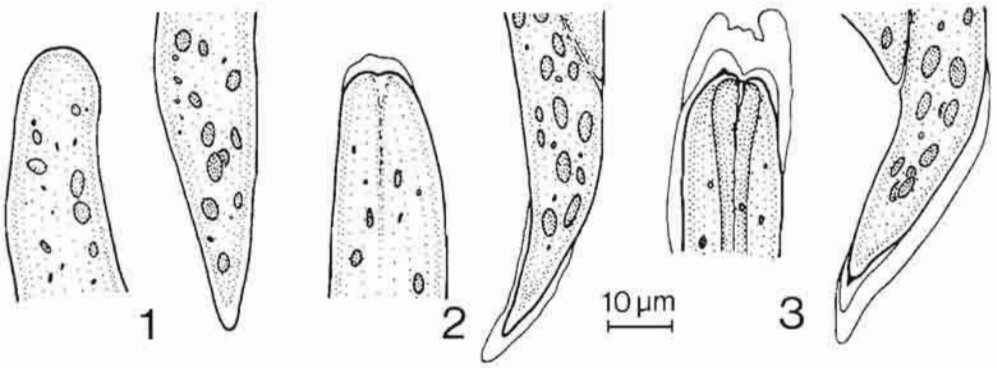
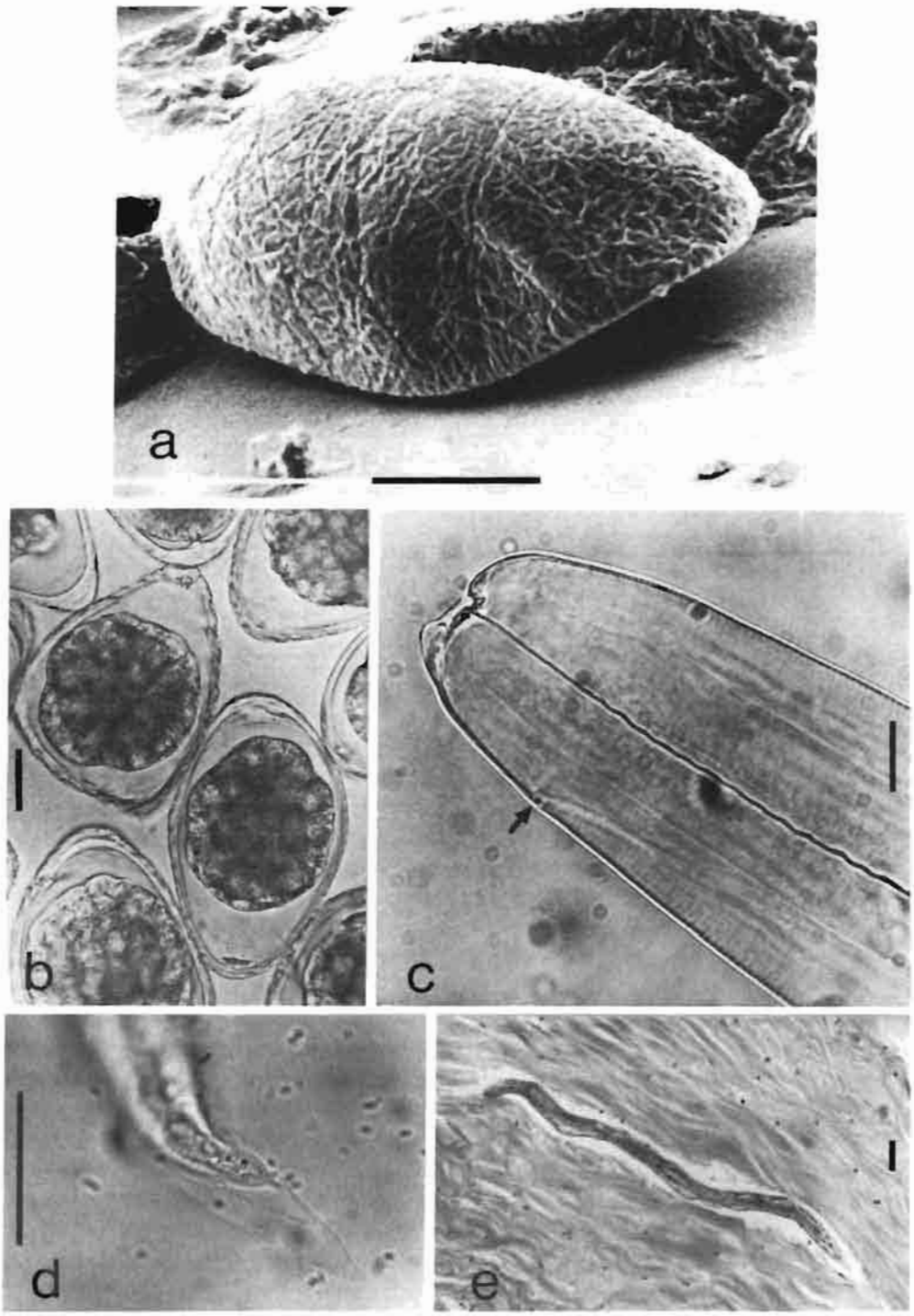


Fig. 2-25: *Sulcascaris sulcata*. Anterior and posterior ends of larvae from eggs. 1: First-stage larva from egg 4 days after being laid, being devoid of internal differentiation; 2: second-stage larva from egg 6 days after being laid, showing little internal differentiation and first moult; 3: third-stage larva from egg 8 days after being laid, exhibiting advanced internal differentiation and first and second moults. (After Berry and Cannon, 1981.)

appear to be more frequently and more heavily parasitized by *Sulcascaris sulcata*. Thus, Sey (1977) recorded the nematode from 6 of 33 *Caretta caretta* but from none of 7 *Chelonia mydas*. Smith and Penn (in Lester and co-authors, 1980) found 5 of 6 loggerheads but none of 3 medium-sized green turtles from Shark Bay to be infested. Limpus (in Lester and co-authors, 1980) has found *S. sulcata* "many times in *C. caretta* from Queensland but never in *C. mydas*". Dietary preferences of the 2 turtles may account for these differences. *C. mydas* is predominantly a herbivore, while *C. caretta* is a voracious carnivore (Brongersma, 1972). This suggests that the loggerhead is the more important final host in the life cycle of *S. sulcata*.

In the turtle host, *Sulcascaris sulcata* may attain a considerable size. Teixeira de Freitas and Lent (1946) found female worms taken from the stomach of *Chelonia mydas* to range from 63 to 105 mm in length and from 0.9 to 2.0 mm in width; males were 40 to 100 mm long and 0.6 to 1.3 mm wide. The measurements of the females, as given by these authors, are well within the range of those of Rudolphi's (1819) original specimens. Sprent (1977) noticed wide length variations in worms of both sexes taken from loggerheads in the western Pacific Ocean and the Mediterranean Sea. However, Berry and Cannon (1981) showed that the size of *S. sulcata* increases conspicuously with age — in females from 49 mm at 7 days p.i. to 111 mm at 190 days p.i. (the latter being mature, egg-laying individuals) and in males from 44 mm at 69 days p.i. to 93 mm at 190 days p.i.

It is evident that such large worms must cause considerable host pathology. In the stomach of *Chelonia mydas*, the presence of large numbers of adult *Sulcascaris sulcata* was found to be associated with macroscopic lesions, the formation of fibrino-purulent exudate and chronic gastritis (Teixeira de Freitas and Lent, 1946). The worms — fourth-stage larvae as well as adults — usually attach in large clusters to the stomach wall, mainly in the region of the oesophago-gastric junction (Fig. 2-28), where they cause severe ulceration (Fig. 2-29). Characteristically, larvae are attached with the head embedded in a caseous plug within a small, raised, haemorrhagic crater from which they are difficult to remove (Fig. 2-29a, b). If forcibly removed, it is usual for the plug to remain attached to the worm encasing the anterior end. At the bottom of each lesion there is a necrotic, hyaline layer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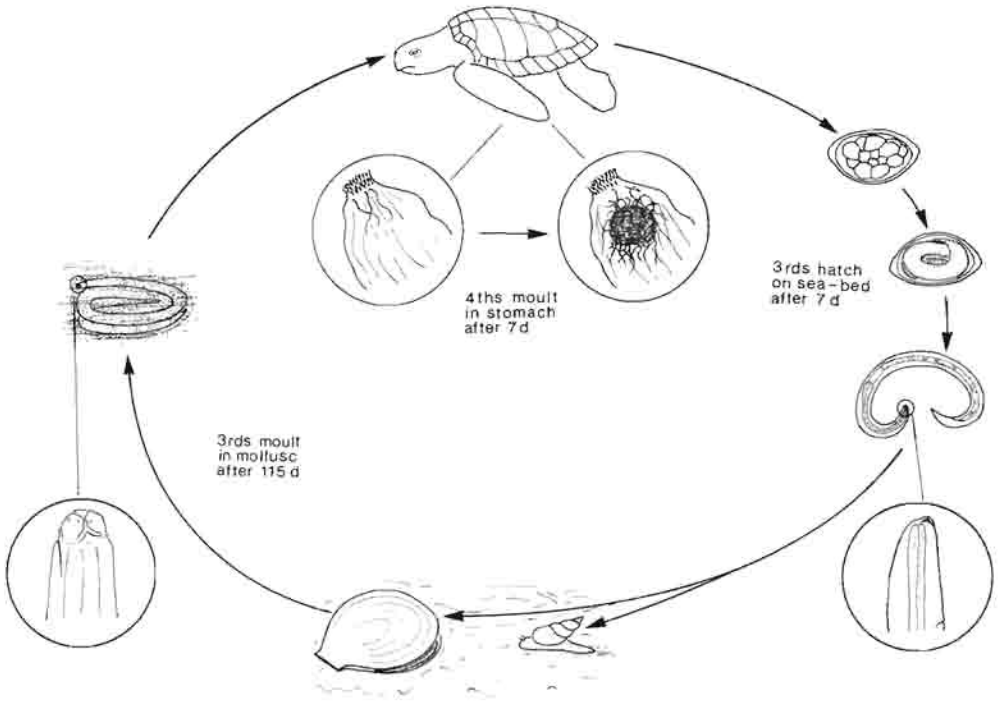

Fig. 2-27: *Sulcascaris sulcata.* Life cycle. Time intervals are minima determined experimentally at 25 °C. (After Berry and Cannon, 1981.)

about 300 to 450 μm in width. In the region of the ulcers, the upper mucosa may be almost completely destroyed (Fig. 2-29c; Sey, 1977; Lester and co-authors, 1980; Berry and Cannon, 1981).

There are several literature inconsistencies concerning anatomical details of *Sulcascaris sulcata* which, among other things, led Allison and co-authors (1973) to invalidate Hartwich's (1957) genus *Sulcascaris.* One of these discrepancies concerns the egg (Fig. 2-26a, b). Dimensions reported by the various authors vary between 40 to 47 × 47 to 79 μm (Sprent, 1977) and 61 to 75 × 86 to 100 μm (Berry and Cannon, 1981). Exact statistical comparison of these data cannot be made because none of the workers indicates the mean and standard deviation of the egg dimensions nor the number measured. As outlined elsewhere (Lauckner, 1980, p. 321), the *range* of measurements is "the poorest of all measures of dispersion" (Simpson and co-authors, 1960). Berry and Cannon (1981) have given exact data ($\bar{x}$, S.D., n) for the larvae of *S. sulcata* but not for the eggs. In the helminths, egg size is a fairly constant character, except in overaged, degenerate worms. Baylis (1923) considers egg size and shape to be of diagnostic value. Therefore, the

Fig. 2-26: *Sulcascaris sulcata.* (a) Scanning electron micrograph of ovum demonstrating rugose surface; (b) eggs 1 day after being laid, exhibiting irregular shapes; (c) head of third-stage larva from bivalve-intermediate host *(Melina ephippium)* experimentally infested 55 days previously, exhibiting reduced anterior boring tooth and excretory pore (arrow); (d) tail of third-stage larva from egg, showing 2 moults; (e) section of third-stage larva in adductor muscle of experimentally infested individual of *M. ephippium,* evoking no apparent host reaction. Bars: 20 μm (a–e). (After Berry and Cannon, 1981.)

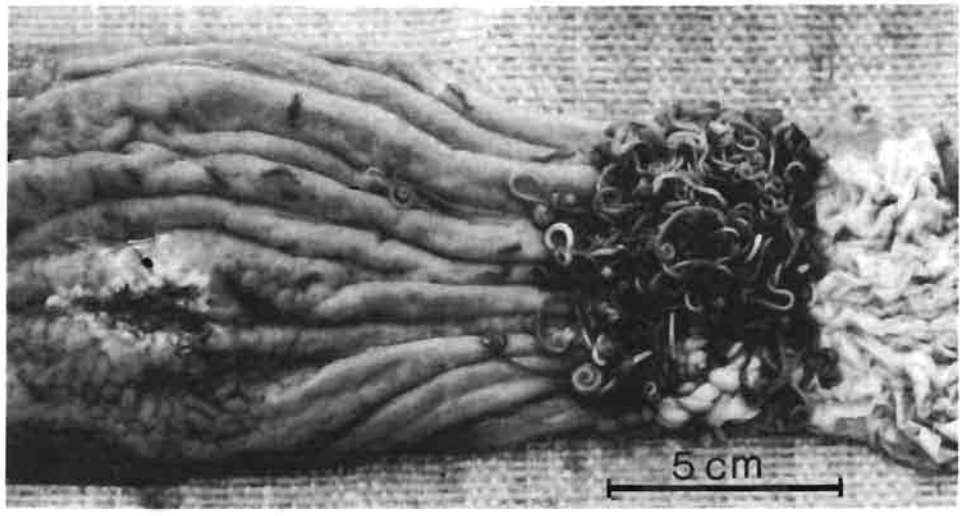

Fig. 2-28: *Caretta caretta.* Opened crop (to right) and stomach showing large clump of adults and fourth-stage larvae of *Sulcascaris sulcata,* attached in region of oesophago-gastric junction. On the left stomach ulcer (arrow) with its caseous white exudate. (After Lester and co-authors, 1980.)

extreme variability of the egg size of *S. sulcata,* as indicated above, awaits explanation. Allison and co-authors (1973) believed it to represent inherent variations in this species. Sprent (1977, p. 383) cautiously concluded:

> "As to whether there is only one species in the genus *Sulcascaris,* a conclusion must await the examination of more specimens. Two male specimens . . . from Australian waters had spicules whose length was twice the length of the ejaculatory duct and more than 8 % of the body length, but otherwise corresponded with the description of *sulcata.*"

A few other nematodes have been reported from chelonians. Baylis (1923) described cucullanoids *Cucullanus carettae* from *Caretta caretta.* The worms occur, attached by their anterior end with the body and tail projecting into the lumen, in the upper 1 to 1.5 m of the intestine. Sometimes the worms may be completely ensheathed by host tissue. Minor haemorrhages mark the attachment sites. Five out of 6 loggerheads from Shark Bay, Western Australia, were found to harbour this species. Numbers per host ranged from 1 to 68 (Lester and co-authors, 1980).

Kathlania leptura, an oxyuroid, has been recovered from 6 of 33 *Caretta caretta* but from none of 7 *Chelonia mydas* captured in Egyptian waters. The infestation intensity ranged from 80 to 240 worms per host (Sey, 1977). One of 6 loggerheads from Shark Bay harboured 200 of these worms, which were swimming freely in the contents of the lower

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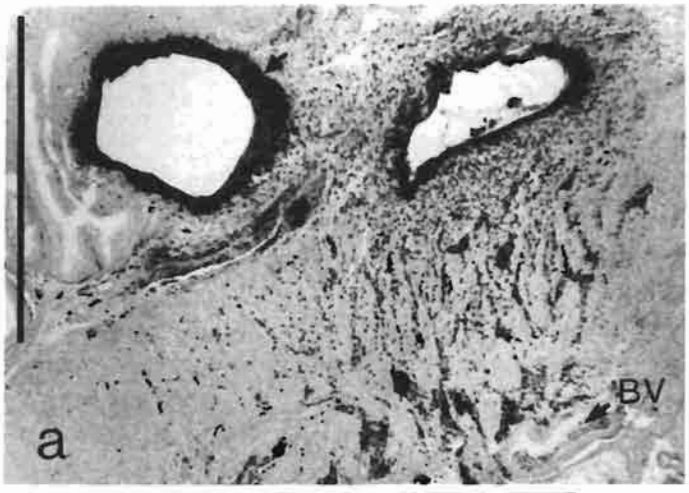
Fig. 2-29: *Caretta caretta.* Pathology produced by *Sulcascaris sulcata.* (a) Section through stomach of turtle experimentally infested with fourth-stage larvae 14 days previously, showing characteristic fourth-stage larva attachment site lesions (arrows). Note distinct infiltration of blood cells from blood vessel (b.v.); (b) fourth-stage larva attachment sites (arrows) at oesophago-gastric junction of turtle experimentally infested with fourth-stage larvae 21 days previously; (c) section through stomach of adult host exhibiting lesion (arrows) caused by natural *S. sulcata* infestation. Note almost complete destruction of upper mucosa. Bars: 1 mm. (After Berry and Cannon, 1981.)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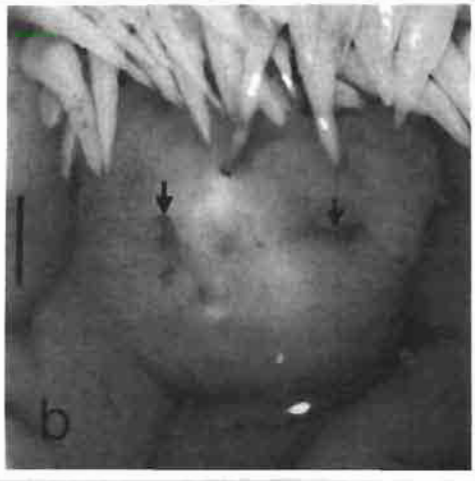
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part of the intestine. Another turtle contained some 5,000 immature oxyuroids (Lester and co-authors, 1980).

A few immature individuals of *Echinocephalus* sp., 16 to 19 mm in length, were found in the stomach of 1 *Caretta caretta* from Shark Bay and in the intestine of two. The anterior one-third of the gnathostomes was embedded in a whitish sleeve of host tissue in the mucosa. Around the attachment site the gut wall felt firmer than normal. Two larval *Echinocephalus* sp., 11 and 18 mm in length and apparently identical to those found in the loggerhead, were dissected from 10 scallops *Amusium balloti* from the same area (Lester and co-authors, 1980). The worms were not identified to species, but they may represent *E. sinensis,* a gnathostome maturing in the spiral valve of elasmobranchs (Ko, 1975). The larvae occur abundantly in oysters *Crassostrea gigas* from Hong Kong (Ko and co-authors, 1974, 1975; Ko, 1976). Cheng (1975) described them as *E. crassostreai* (Vol. II). The occurrence of immature *Echinocephalus* sp. in turtles is certainly accidental.

Maricultured *Chelonia mydas* may sometimes become infested with 'worms' transmitted *via* food fish and, as a consequence, may display stunted growth (Clayton, 1975). Smith and Wootten (1978) assume that the worms referred to by Clayton were larval anisakids, possibly including *Anisakis.*

Ascaridoids of the genera *Goezia* and *Paraheterotyphlum* parasitize members of the Hydrophiidae. *G. holmesi* has been identified as a stomach nematode of sea snakes *Enhydrina schistosa, Lapemis hardwicki, Hydrophis ornatus, H. elegans* and *H. caerulescens* from the Gulf of Carpentaria, Northern Australia. The worms are fairly small; males measure 2.7 to 5.5 mm, females 2.6 to 6.7 mm in length. Their body surface is covered with spines arranged in transverse rows, a feature characteristic of the genus (Sprent, 1978a). With a single exception, all other known species of *Goezia* are parasites of teleost fish, mainly in freshwater (Dollfus, 1935; Rasheed, 1965; Sprent, 1978a).

Nothing is known about the life cycle of *Goezia holmesi,* and only limited knowledge has been gained on that of freshwater species. From the available information (summarized by Hartwich, 1975, and Sprent, 1978a) it appears that copepods act as intermediate hosts and teleosts become infested by ingesting copepods harbouring third-stage larvae. In addition, teleosts may facultatively function as paratenic hosts. As sea snakes are largely piscivorous (McCosker, 1975), they probably acquire *G. holmesi* by devouring fish harbouring encysted third-stage larvae. In addition to adult worms, immature specimens of *G. holmesi,* 1.7 to 2.0 mm long, have been recovered from *Lapemis hardwicki.* Their undeveloped reproductive organs indicated that they were fourth-stage larvae (Sprent, 1978a).

In freshwater fish, mainly in predatory species, the presence of *Goezia* spp. is sometimes associated with the occurrence of tumour-like nodules in the stomach wall, which are inhabited by the worms (MacCallum, 1921; Hartwich, 1975). In sea snakes, *G. holmesi* was found loose within the stomach and appeared to be doing no harm (Sprent, 1978a).

Ascaridoids *Paraheterotyphlum australe* are parasitic in the stomach of various sea snakes. Originally described by Johnston and Mawson (1948) from Australian *Pelamis (Hydrus) platurus,* the species has subsequently been found in *Aipysurus laevis, Hydrophis elegans, Astrotia stokesi* and *Disteira kingi* from the east coast of Australia (Sprent, 1978b). These nematodes are large, slender worms, males measuring 29 to 87 mm and females 82 to 168 mm in length. *P. ophiophagos,* in size similar to *P. australe,* has been

reported from *Laticauda colubrina* and *L. semifasciata* from Taiwan (Schmidt and Kuntz, 1973; Sprent, 1978b). The present knowledge of distribution suggests that these 2 ascaridoids occur in 2 separate centres of sea-snake dispersal.

In addition to adult *Paraheterotyphlum* in the stomach, larvae probably referable to this genus were found in other body regions of hydrophiids. The smallest (probably third-stage) larva, 5.9 mm in length, was collected from the lung tissue of a young *Pelamis platurus* from Queensland, Australia. Larger specimens, 14 to 19 mm long and likely to represent third-stage *P. australe,* were recovered from the trachea of *Aipysurus laevis.* Fourth-stage larvae, 15 to 28 mm in length, occurred in the stomach of several sea snakes (Sprent, 1978b).

No information appears to be available on the life cycle of *Paraheterotyphlum* spp. As the worms from sea snakes are clearly members of the Raphidascarinae, their development might follow the general pattern of other members of the subfamily (for summary of the mainly Russian literature on the life cycle of *Raphidascaris acus* consult Hartwich, 1975). Raphidascarines are typically parasitic in the stomach or intestine of teleost fish, mainly from freshwater. Second-stage larvae, developing outside hosts, invade various invertebrates including gastropods, oligochaetes and copepods, in which they grow but do not moult into third-stage larvae. The latter stage is attained only in teleosts (the true intermediate hosts), which may either be invaded directly or *via* infested invertebrate food organisms. Maturity is reached in predatory fish, which devour benthivorous or planktivorous fish harbouring third-stage larvae.

This general raphidascarine life-cycle pattern led Schmidt and Kuntz (1973) to discuss the possibility as to whether *Paraheterotyphlum australe* and *P. ophiophagos* are genuine fish parasites, their occurrence in hydrophiids representing cases of accidental parasitism resulting from the ingestion of fish. Sprent (1978b), in contrast, concluded that the uniformity of the specimens studied by him makes it seem likely that a well-established host-parasite relationship has evolved between *Paraheterotyphlum* spp. and sea snakes. The author furthermore states (p. 166):

> "... this conclusion is supported by the absence of species in this genus of ascaridoids from other fish-eating reptiles, for example, the Homalopsinae. *Paraheterotyphlum* spp. may have been derived from an ancestral form using fish as definitive hosts. It seems likely that an evolutionary extension of the host succession has occurred, whereby the former definitive host has now become the intermediate host."

Diaphanocephalids *Kalicephalus laticaudae* have been reported from *Laticauda laticaudata* at Isigaki Island (Japan) and Taitung Hsien (Taiwan), from *L. colubrina* collected at Lan Yu Island (Taiwan), and from *L. semifasciata* collected at Amami Island (Japan) (Yamaguti, 1935; Schad, 1962; Telford, 1967). Infestations with these cosmopolitan reptilian nematodes may have been aquired by the amphibious *Laticauda* spp. while basking on rocks or on the beach. Telford reported *K. laticaudae* from 13 of 51 Amami Island *L. semifasciata.*

Agents: Hirudinea

The leeches range among the most deleterious metazoan parasites of vertebrates, not only because of their blood-sucking mode of feeding, but because they may act as vectors

for blood parasites (p. 567). Members of the Ozobranchidae (previously contained in the Piscicolidae) parasitize marine turtles.

Ozobranchus branchiatus, originally described by Menzies (1791) as *Hirudo branchiata,* is a rare warmwater species of apparent circumtropical distribution. Menzies found it "in great abundance adhering to a turtle, in the Pacific Ocean, between the tropics". It has subsequently been reported from the Gulf of Mexico, Australia, Japan, Borneo, Sumatra, the Malay Peninsula and India (Baird, 1869; Oka, 1910; MacCallum and MacCallum, 1918; Nigrelli, 1941, 1942; Nigrelli and Smith, 1943; Hendrickson, 1958; Sanjeeva Raj and Penner, 1962; Ghosh and co-authors, 1963; de Silva and Fernando, 1965; Soós, 1965; Richardson, 1969; Sawyer and co-authors, 1975). Thus far, *Chelonia mydas* is the sole host reported for this leech.

Ozobranchus margoi, first reported by Apáthy (1890) from *Caretta caretta* from the Gulf of Naples (Italy) as *Pseudobranchellion margoi,* has a broad host spectrum. Records are from *C. caretta, Chelonia mydas, Lepidochelys olivacea, L. kempi* and *Eretmochelys imbricata* taken in the Mediterranean, in Japan, Australia and in tropical or subtropical waters of the Atlantic and Pacific Oceans or maintained in captivity (Blanchard, 1894; Oka, 1927; Cordero, 1929; Ringuelet, 1944; Richardson, 1969; Davies and Chapman, 1974; Sawyer and co-authors, 1975; Davies, 1978). Schwartz (1974) found *O. margoi* on *C. caretta* taken as far north as Cape Lookout, North Carolina. Curiously, Oka (1927) reported this species from both *Lepidochelys olivacea* and dolphins *Delphinus longirostris* taken off the coast of Kyushu (Japan). Previously, he (Oka, 1910) had listed *Delphinus* sp. as host for *O. branchiatus.* These records may encompass either cases of accidental parasitism or misidentifications. With respect to turtles, *O. margoi* appears to exhibit no host preference, all 85 marine turtles (representing 4 species) crowded together in a pool being infested. The leeches were neither found on various species of local terrestrial tortoises nor on elasmobranch or teleost fish (Davies and Chapman, 1974).

Like other leeches, *Ozobranchus* spp. are provided with a powerful posterior sucker ascertaining firm attachment to the body surface of the host, and a smaller anterior sucker acting as a 'muscular pump' in the process of obtaining a blood meal from the host. Furthermore, there are 2 eye spots at the anterior end and 2 conspicuous rows of external gills (branchiae) on each body side (Fig. 2-30). For details of the morphology and anatomy of *O. branchiatus* consult MacCallum and MacCallum (1918), Sanjeeva Raj and Penner (1962), Nigrelli and Smith (1943) and Richardson (1969), for that of *O. margoi* Cordero (1929), Richardson (1969) and Davies (1978).

Although *Ozobranchus branchiatus* and *O. margoi* are easily distinguished by the presence of 7 pairs of branchiae on the former and 5 pairs on the latter (Menzies, 1791; Apáthy, 1890), there has been considerable confusion about these 2 species in the literature. Some records may be misidentifications. The confusion started right away with Menzies' (1791) original description of *O. branchiatus.* The author described the worms as having 7 pairs of "pellucid branchy bristles" (i. e., the branchiae), but showed only 5 pairs in his illustrations. The paucity of distributional data, combined with the possible confusion mentioned above, renders speculations about the distributional patterns of both species tenuous (Davies and Chapman, 1974).

Incidence and intensity of infestation with *Ozobranchus* spp. in turtles may be high, the associated pathology severe. Nigrelli and Smith (1943), who observed the presence of *O. branchiatus* on *Chelonia mydas* to be associated with the occurrence of fibro-epithelial

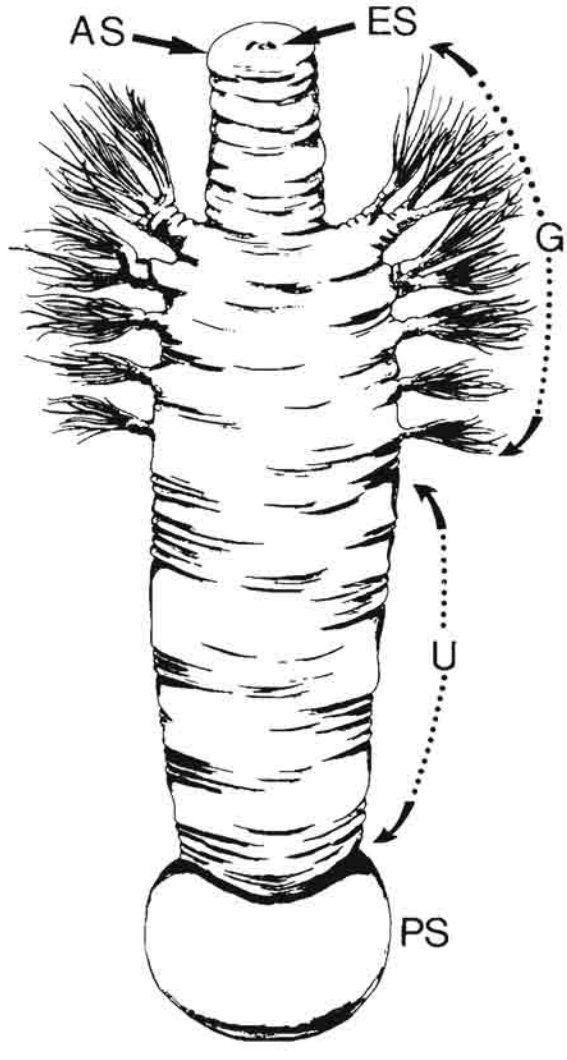

Fig. 2-30: *Ozobranchus margoi*. External morphology. As anterior sucker, ES eye spots, G gills, PS posterior sucker, U urosome. (After Davies, 1978.)

tumours, reported densities of up to 50 leeches per 1.3 cm^2 of tumour surface. Williamson (in Sawyer and co-authors, 1975) observed *O. margoi* on nearly all of 25 adult *Caretta caretta* tagged on Wassaw Island, Georgia. Some turtles carried over 100 leeches. Still larger numbers may occur on turtles held in captivity. Thus, Davies and Chapman (1974) estimated the number of *O. margoi* present on individual turtles crowded in a pool at the Miami Seaquarium to be in the range of 800 to 900 in many cases. As *Ozobranchus* spp., unlike most leeches, complete their entire life cycle on chelonians, epizootic outbreaks of hirudiniasis may occur in captive hosts.

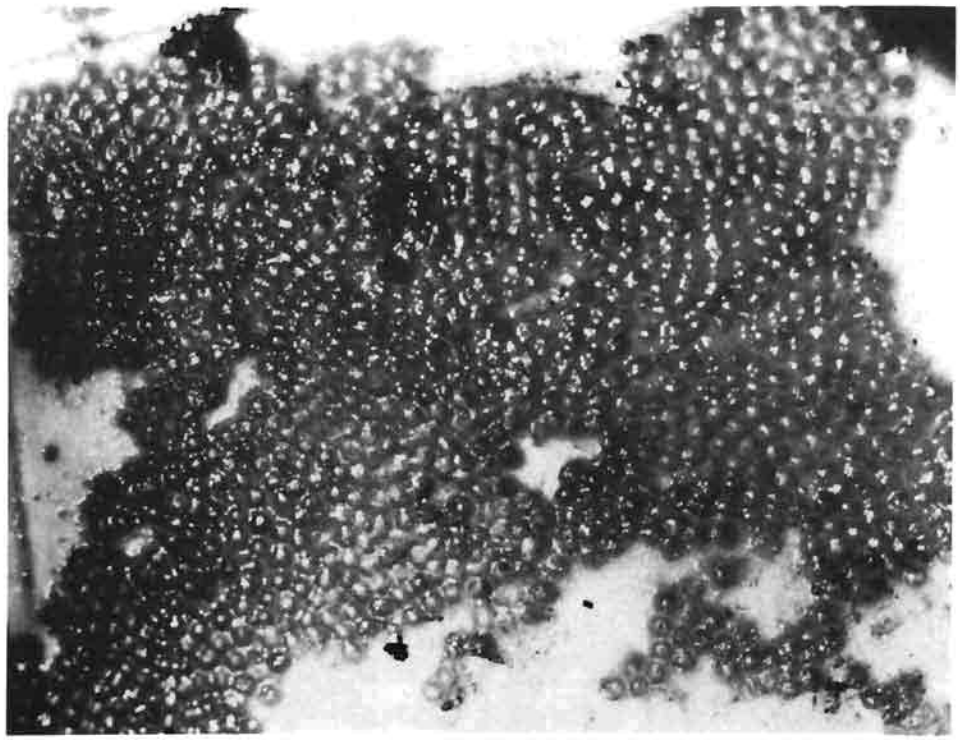

Fig. 2-31: *Ozobranchus margoi.* Egg clusters on plastron of *Caretta caretta.* (After Davies and Chapman, 1974.)

Ozobranchus spp. attach their eggs to the host, sometimes in large clusters covered with sclerous cementing material (Fig. 2-31). Adult worms may be found in the corners of the mouth or in the external cloacal region, on the ventral surface of the flippers, on the neck and in the folds of the soft tissue between the carapace and the plastron (Fig. 2-32). They usually do not occur on the carapace.

Ozobranchus branchiatus, ranging in length from 3.5 to 11 mm (Ghosh and co-authors, 1963; Sawyer and co-authors, 1975), is much smaller than *O. margoi,* for which species a length range of 4 to 30 mm has been recorded (Apáthy, 1890; Cordero, 1929; Davies and Chapman, 1974; Sawyer and co-authors, 1975). The pathology produced appears (although not invariably) to be correlated with the number of leeches per host and the size of the parasites. Nigrelli (1941, 1942) and Nigrelli and Smith (1943) suggested an association of *O. branchiatus* with fibropapillomata observed on the skin of *Chelonia mydas* (p. 612). Hendrickson (1958) and Sanjeeva Raj and Penner (1962) reported a similar condition. Tumorous growths were not seen on any of the turtles infested with *O. margoi* (Davies and Chapman, 1974; Schwartz, 1974). In spite of extremely high parasite densities — 800 to 900 leeches per host in many cases —, the only host pathology observed by the latter authors was the excavation and destruction of the plastron at the sites of attachment (Fig. 2-33). All specimens of *O. margoi* examined had blood in their guts. The massive leech infestation in these captive hosts was first noticed when the turtles continually attempted to haul out.

Fig. 2-32: *Ozobranchus margoi.* Distribution of adult worms on posterior flippers, on external cloaca and in folds of soft tissue between carapace and plastron of *Caretta caretta.* (After Davies and Chapman, 1974.)

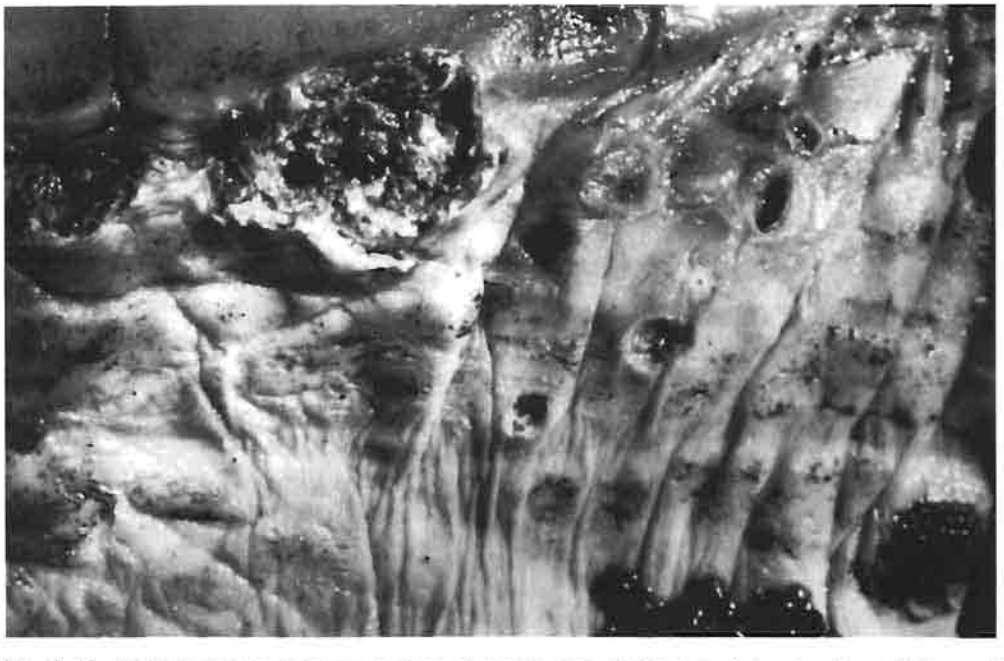

Fig. 2-33: *Caretta caretta.* Lesions at sites of attachment of *Ozobranchus margoi* on plastron of *Caretta caretta.* (After Davies and Chapman, 1974.)

Schwartz (1974) witnessed an epizootic outbreak of hirudiniasis in tank-held *Chelonia mydas* and *Caretta caretta* at the Institute of Marine Sciences, Morehead City, North Carolina. Leeches had apparently been introduced with loggerhead turtles trawled from inside the bight of Cape Lookout on May 26, 1972. One of 7 turtles had numerous *Ozobranchus margoi* in the axil of each rear flipper, as well as in the cloacal opening. No leeches were known to be on the other loggerheads or on 2 *C. mydas* sharing the tank. The first weeks after the introduction of the infested *C. caretta* all went smoothly, the turtles feeding readily on scraps of fish, shrimp or blue crabs, until on July 18 all 9 turtles were suddenly found to be heavily infested with leeches. The green turtles were most heavily afflicted in the throat area as well as in the axils of the flippers. Their plastons were sheeted with egg clusters, while masses of adult leeches had often worked their way under the scales of the plastron. The younger green turtle succumbed 3 days after the discovery of the epizootic.

By July 24, the largest female *Caretta caretta* in the tank were so devastated that their eyes had been eaten away to reveal the internal bone and skull. Leeches filled every possible crevice — mouth, nostrils, flippers, cloaca, etc. Large masses of *Ozobranchus margoi* clung over all the body surface including the carapace. Three of the 4 female loggerheads succumbed between July 24 and August 2. All turtles stopped feeding during the height of the epizootic (Schwartz, 1974).

Treatment of leech infestation was attempted in several ways. Davies and Chapman (1974) applied 10 % formalin directly to the worms and their egg masses on beached turtles, which were subsequently kept out of the water for at least 3 h. This treatment was not highly effective. The second method consisted of addition of copper sulphate to the pool water. A 0.3 ppm concentration was maintained for 6 h. The procedure, repeated twice, 7 days apart, eliminated both the leeches and their eggs from the hosts. However, a few worms or eggs appear to have survived the treatment, because 3 months later, *Ozobranchus margoi* was again found in low numbers on most turtles. Of 86 infested turtles 3 died — whether as a result of the infestation or of the treatment remained unknown.

Attempts to control the *Ozobranchus margoi* epizootic described by Schwartz (1974) by direct application of concentrated iodine simply relieved the afflicted areas for 2 or 3 days before reinfestation occurred in the same areas. In contrast, copper sulphate, added (in unspecified concentration) to the tank water, was a definite cure. Within 5 h most leeches had dropped off the turtles and after 10 h most were dead. None were found alive by the end of the 60-h treatment period. Apparently, also the eggs had been killed since no reinfestation of the surviving loggerheads occurred during the following 4.5 months.

Aside from their direct pathogenic action, Hirudinea may act as vectors of blood protozoans. Leeches are known as invertebrate hosts for haemoflagellates of the family Trypanosomatidae and coccidians of the family Haemogregarinidae. Leech-transmitted haemogregarines and trypanosomes are known from freshwater turtles (Robertson, 1908; Reichenow, 1910). Davies and Chapman (1974) examined blood samples taken from *Ozobranchus margoi,* as well as directly from their turtle hosts. No blood parasites were found.

Agents: Acarina

Trombiculid mites — arachnids of the order Acarina — are well-known parasites of vertebrates. The life cycle of these arthropods normally includes 7 stages — egg, deutovum, larva, nymphophane (prenymphal quiescent stage), nymph, teliophane (preadult quiescent stage) and adult. The 6-legged larvae, commonly known as 'chiggers', are parasitic; 8-legged nymphs and adults are free-living. The chiggers attach themselves to various vertebrates to suck blood causing intense itching and local irritation. In some instances, larval mites serve as vectors of typhus or other infectious diseases. Larval 6-legged trombiculid mites have been reported from amphibious sea snakes and from the Galapagos marine iguana.

Vatacarus ipoides, an unusual larval mite, is parasitic in the air sacs and respiratory passages (trachea and lungs) of *Laticauda* spp. Southcott (1957), who first described it from New Caledonian *Laticauda laticaudata,* was struck by the bizarre appearance of the larva (Fig. 2-34). Instead of being typically acarine in shape, the chiggers were vermiform, which led Southcott to propose a new family, the Vatacaridae, for the genus. Vercammen-Grandjean (1960), however, identified *V. ipoides* as a member of the Trombiculidae and named it *Eutrombicola ipoides. In vitro* studies, conducted by Nadchatram and Audy (1965), yielded surprising results: In *V. ipoides,* the active nymphal stage is by-passed, and larvae give rise to adults with only a single intermediate moulting stage. Hence, there are only 5 stages (instead of 7) in the life cycle of this mite, of which 2 are active stages.

The larval *Vatacarus ipoides* living in the respiratory passages of *Laticauda* spp. are elongate, maggot-like in shape (Fig. 2-34) and orange-coloured. Larger specimens may reach a length of 4.5 mm and a width of 1.5 mm. When extended, their body is capable of increasing considerably in length. Engorged individuals become grossly distended followed

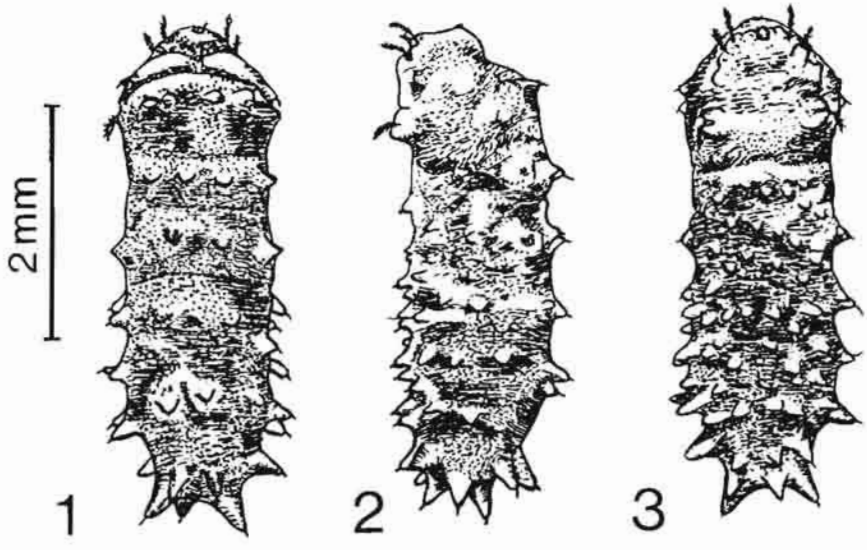

Fig. 2-34: *Vatacarus ipoides.* Six-legged larva from air-sac of *Laticauda laticaudata.* 1: Dorsal view; 2: lateral aspect; 3: ventral view. (After Southcott, 1957.)

by development of large papillae on the body. The papillae have an obvious function enabling the larva to move in mucus in the manner of maggots. Within the superfamily Trombidioidea, *V. ipoides* shows the most complete adaptation to endoparasitism thus far observed (Southcott, 1957; Nadchatram and Audy, 1965).

Although *Vatacarus ipoides* has successfully been reared to adult in the laboratory, no other life-cycle stages except the 6-legged larvae are known from nature. As the amphibious Laticaudinae spend considerable time outside the water, infestations presumably occur on land. As indicated by laboratory experiments conducted by Nadchatram and Audy (1965), adult *V. ipoides* appear to have a very short life span, perishing even before the eggs are hatched. The entire life cycle appears to be completed in 45 to 50 days.

Incidence and intensity of infestation with *Vatacarus ipoides* may be high. Thus, all of 118 *Laticauda colubrina,* collected by Nadchatram and Audy (1965) from tidal reefs off Singapore, were found to be infested. A total of some 2,000 larvae were recovered from these hosts, the maximum number collected from a single sea snake being 60 (mean approx. 17). The 100 % infestation, as observed in *L. colubrina,* might reflect the gregarious habits displayed by these reptiles when being out of the water. All above snakes were collected at low tide, while they were resting in rockholes and treeholes. Similarly, Telford (1967) found all of *L. laticaudata* from Amami Island (Japan) to be infested with *V. ipoides,* in numbers ranging from 9 to 36. Forty-three out of 51 *L. semifasciata* from the same locality were also infested, the maximum number of mites per host being 121.

Vatacarus kuntzi, apparently the only other species of the genus known thus far, is distinguished from *V. ipoides* by its considerably larger size. Engorged larvae, taken from the trachea of *Laticauda colubrina* at Botel Tobago (a small island near Taiwan), measured 5.0 to 6.2 × 2.0 to 2.3 mm (Nadchatram and Radovsky, 1971).

As *Laticauda* is the only genus of sea snakes which is landgoing, *Vatacarus* spp. are unlikely to be found in aquatic hydrophiids. Nadchatram and Audy (1965) examined over 30 individuals representing 5 species of true sea snakes and 37 specimens of 3 species of freshwater or tidal-stream snakes. No *Vatacarus* were found. The genus appears to be restricted to laticaudine sea snakes. *V. intermedius, V. amblyrhynchus* and *V. intranasalis,* 3 species reported from the nasal fossae of the Galapagos marine iguana *Amblyrhynchus cristatus* (Vercammen-Grandjean, 1965; Vercammen-Grandjean and Watkins, 1965), have subsequently been accomodated in the genus *Iguanacarus,* which had previously been regarded as a subgenus of *Vatacarus* (Nadchatram, 1980). All of these are considerably larger than *Vatacarus* spp. Postlarval stages of *Iguanacarus* are unknown. All 5 individuals of *A. cristatus,* autopsied by Vercammen-Grandjean (1965), were heavily infested with *I. amblyrhynchus* and *I. intranasalis* and, in addition, harboured more than a dozen of other (specifically undetermined) species of acarines.

Another member of the genus *Iguanacarus, I. alexfaini,* has recently been described from New Guinean *Laticauda laticaudata* and *L. colubrina* (Nadchatram, 1980).

Next to nothing has been reported on the pathology caused by larval *Vatacarus* and *Iguanacarus.* No respiratory impairment appeared to result from *V. ipoides* infestation, and no gross lesions were observed upon autopsy. When captured, infested *Laticauda laticaudata* were quite vigorous (Rageau, in Southcott, 1957). As pointed out by Southcott (1957), there need not necessarily be a macroscopically discernible tissue reaction to larval mite feeding in a vertebrate host. From the structure of its mouthparts and its affinities, *V. ipoides* was (certainly correctly) believed to feed upon the host's body fluids. Of over

2,000 larval mites collected from 118 *L. colubrina*, not a single one was found in an unfed state (Nadchatram and Audy, 1965).

In addition to mites, ticks (Ixodidae) are commonly found on *Laticauda* spp.; they do not occur on hydrophiids. One species, *Amblyomma nitidum*, is specific to laticaudines (Hirst and Hirst, 1910; Rageau, 1967; Wilson, 1970; Zann and co-authors, 1975), while other members of the genus are parasites of birds. Another tick, *Aponomma* sp., has been reported from New Guinean *Laticauda colubrina*. Normally, it is characteristic of terrestrial reptiles and widespread in New Guinea (Wilson, in Zann and co-authors, 1975). The amphibious mode of life of *Laticauda* probably makes it prone to parasitization by acarines. Like mites, ticks are known as vectors of a variety of serious diseases caused by viruses, bacteria, rickettsiae and protozoans (Arthur, 1962, 1966).

Agents: Crustacea

Cirripeds, copepods and even decapods are known as epizoic associates of chelonians; cirripeds have been reported from sea snakes.

Barnacles of the genera *Chelonibia, Conchoderma, Platylepas, Cylindrolepas, Stephanolepas* and *Stomatolepas* are the most frequently encountered organisms on the carapace and flippers of sea turtles. Of these, *Chelonibia* and *Platylepas* have the broadest 'host' spectrum; they have also been reported from sea snakes and sirenians (Darwin, 1851, 1854; Daniel, 1953; Ross and Newman, 1967; Zann, 1975). Monroe and Limpus (1979), who reported 17 species of barnacles (representing 3 families) from 5 species of chelonians, have provided a key to the cirripeds found on turtles in Queensland waters, as well as an excellent general account of the barnacles living on marine turtles.

Members of the encrusting Balanidae and the stalked Lepadidae may be regarded as facultative epizoans, while the Coronulidae, commonly known as 'turtle' or 'whale' barnacles, are obligate associates of a wide range of hosts (Ross and Newman, 1967). *Balanus trigonus*, up to 11.5 mm in basal diameter, and *B. variegatus*, up to 9 mm in basal diameter, occur on *Caretta caretta*. *B. trigonus* is cosmopolitan on hard substrates of various kinds in tropical and subtropical waters (Pope, 1945; Stubbings, 1967; Relini, 1968; Monroe and Limpus, 1979). Brongersma (1972) recorded the small (max. 4.3 mm diameter) 'Australian barnacle' *Elminius modestus* from an individual of *Chelonia mydas* washed ashore on the Dutch coast.

'Goose barnacles' of the family Lepadidae are typically found on driftwood or attached to boats. Monroe and Limpus (1979) reported *Lepas anatifera, L. anserifera* and *L. hilli* from Queensland *Caretta caretta*. From the same host, these authors recorded the 'whale barnacles' *Conchoderma virgatum* and *C. auritum*. According to Darwin (1851), *C. auritum* is extremely common in all parts of the world. Hubbs (1977) found *C. virgatum* on Pacific ridleys *Lepidochelys olivacea*, caught off La Jolla, California. The stalked barnacles were attached in the loose area of the pelvic axil, oddly with the normally free end embedded. *C. virgatum chelonophilum* has been recovered from the mouth of Queensland *C. caretta* (Monroe and Limpus, 1979).

As stated, the obligately epizoic Coronulidae (with subfamilies Chelonibiinae, Coronulinae and Platylepadinae) have a wide range of hosts including turtles, sea snakes, sirenians, crustaceans, molluscs and fish (Ross and Newman, 1967). The large encrusting *Chelonibia testudinaria*, which measures up to 60 mm in rostro-carinal diameter, has been

recorded on *Caretta caretta, Chelonia mydas, 'Chelonia depressa', Eretmochelys imbricata* and *Lepidochelys kempi*. It occurred on about one-third of the *C. mydas* from Malaya and Sarawak, examined by Hendrickson (1958). The barnacle, which shows no signs of embedding or erosion, is cosmopolitan in tropical and warm temperate seas (Pilsbry, 1916; Walker, 1978; Monroe and Limpus, 1979). In contrast, *Chelonibia caretta*, up to 35 mm in basal diameter, may occur embedded in tissues of *C. caretta* and *Eretmochelys imbricata*. It is an apparently tropical species (Pilsbry, 1916; Korschelt, 1933; Stubbings, 1967; Monroe and Limpus, 1979). Distinct pathology has also been reported for *Chelonibia ramosa*, found embedded in the carapace of a *Chelonia mydas* individual (Korschelt, 1933).

Killingley and Lutcavage (1983) observed variations in the ^{18}O and ^{13}C composition of the calcitic shells of *Chelonibia testudinaria*, epizoic on the carapaces of *Caretta caretta*, which were shown to reflect the environmental conditions under which the turtles had lived. Isotopic profiles from barnacle shell material can thus be utilized to reconstruct movements of *C. caretta* between the open ocean and brackish water regimes.

The subfamily Platylepadinae (in family Coronulidae) includes encrusting and burrowing forms. *Platylepas hexastylos* has been recorded from a variety of chelonian and sirenian hosts (Darwin, 1854; Pilsbry, 1916; Hiro, 1937). Hundreds of *P. hexastylos* were found to encrust the carapace, plastron, dorsal portions of the head and neck, and the dorsal surface of all flippers of an individual of *Chelonia mydas* captured in Chincoteague Bay, Maryland. Specimens ranged from 3 to 15 mm in diameter (Schwartz, 1960). Monroe and Limpus (1979) reported the species from Queensland *Caretta caretta, Eretmochelys imbricata, C. mydas* and *'C. depressa'*. Other species of turtle-inhabiting *Platylepas* include *P. coriacea, P. decorata* and *P. multidecorata*, reported from chelonians in various tropical and subtropical waters (Darwin, 1854; Nilsson-Cantell, 1921; Daniel, 1962). According to Monroe and Limpus (1979), *P. multidecorata* is a doubtful species. *P. coriacea* appears to be confined to *Dermochelys coriacea*, while *P. hexastylos* and *P. decorata* seem to be excluded from this host.

Large numbers of *Cylindrolepas darwini* were found widely attached, largely to exposed parts of the head, body and flippers, of Pacific ridleys *Lepidochelys olivacea* caught off La Jolla, California (Hubbs, 1977).

Encrusting barnacles obviously have little if any detrimental effect on their carriers except in cases where encrustations on the head interfere with the action of the eyelids (Hendrickson, 1958). In contrast, burrowing forms are said to erode 'tumorous' cavities through the carapace (Clark, 1965). Burrowing *Tubicinella cheloniae ('Stephanolepas muricata')* have been considered the most damaging 'ectoparasites' of *Chelonia mydas* from Malaya and Sarawak. The barnacles occur deeply embedded in a sort of leathery tissue, which replaced the normal hard scutes of the turtle's carapace. In a heavily affected green turtle, these lesions formed irregular patches totalling perhaps 900 cm^2 on the dorsal portion of the carapace. The cirripeds, which measured up to 4 cm in length, lay embedded with their exposed ends approximately flush with the surface of the leathery tumour-like tissue which, in turn, was raised up to about 1.5 cm above the general carapace level. The longest specimens had apparently penetrated completely through the carapace to the host's body cavity. All holes, which were left after the forcible extraction of the barnacles by prising them out with a screwdriver, filled with blood, and the blood in the deeper holes moved in and out with the movements of the turtle. *S. muricata* has a hard cylindrical shell

with numerous irregular processes projecting out at right angles from the shell. These processes were embedded in what bone remained in the area (Hendrickson, 1958).

On another occasion, an individual of *Chelonia mydas* was found with a large, mushroom-shaped, leathery growth on roughly the highest point of its carapace dome. This growth, measuring about 10 × 15 cm, was attached to the carapace by a relatively small stalk. It was impossible to remove the growth in its entirety. It was rooted very strongly through the carapace scutes into the underlying bone, and the 'root' was highly calcified. The mass contained a number of cirripeds considered to represent *Stephanolepas muricata*. After its forcible removal, a hole about 2.5 cm wide and 2 cm deep remained. However, very little host blood was lost during the extraction. After the removal of the tumour-like growth, the turtle remained in good health. At later dates, it was seen to return to the same beach where it had been caught, and to lay eggs. Nevertheless, it was assumed that severe *S. muricata* infestation might be lethal to turtles (Hendrickson, 1958).

According to Monroe and Limpus (1979), the above record of *Stephanolepas muricata* is a misidentification. The authors redescribed Hendrickson's (1958) barnacles as *Tubicinella cheloniae*. From *S. muricata*, as first described by Fischer (1886), it is distinguished (among other characters) by its solid porose shell, which is non-porose and fragile in *S. muricata*. The latter species (or forms considered to be identical to it) has been reported from *Chelonia mydas*, *Eretmochelys imbricata* and *Caretta caretta* (Fischer, 1886; Pilsbry, 1916; Monroe and Limpus, 1979). Records by Nilsson-Cantell (1932) and Newman and co-authors (1969) are probably referable to *T. cheloniae*, as described by Monroe and Limpus (1979). The latter authors obtained all their *S. muricata* material from the leading edge of the carpal area of the front flippers of Queensland *C. mydas*, *C. caretta* and *E. imbricata*, where the barnacles burrowed through or between the epidermal scales.

Stomatolepas spp. occur embedded in the soft skin portions of chelonians. Most of these barnacles have been recorded under the name *S. elegans*, but beyond doubt include other species *(S. praegustator, S. dermochelys)*. Barnacles of this genus mostly invade *Lepidochelys olivacea* and *Dermochelys coriacea*, occasionally also *Caretta caretta* and *Chelonia mydas*. Records of *Stomatolepas* spp. are from geographically widely separated areas including temperate waters, i. e., Australia, Japan, Malaya, Senegal, Tortugas (Gulf of Mexico), Nova Scotia (Canada), the Netherlands, Eire, England, Scotland, Norway, Italy and Malta (Pilsbry, 1910; Hiro, 1936; Stubbings, 1965, 1967; Zullo and Bleakney, 1966; Relini, 1968; Holthuis, 1969; McCann, 1969; Brongersma, 1972; Smaldon and Lyster, 1976; Lanfranco, 1979; Monroe and Limpus, 1979).

Stomatolepas praegustator may be found in the gullet as well as in the soft skin of the neck and the base of the front flippers of *Lepidochelys olivacea* and *Caretta caretta* (Pilsbry, 1910; Monroe and Limpus, 1979). *S. dermochelys*, previously frequently confounded with *S. elegans*, has thus far only been reported from the soft skin at the base of the flippers and tail, as well as from the palate of *Dermochelys coriacea; S. transversa* invades the median groove of the plastron of *Chelonia mydas* (Zullo and Bleakney, 1966; Holthuis, 1969; McCann, 1969; Brongersma, 1972; Smaldon and Lyster, 1976; Monroe and Limpus, 1979).

Cirripeds may also attach to marine reptiles other than turtles. 'Goose barnacles' of the genus *Octolasmis* (syn. *Dichelaspis*) associate mainly with decapod crustaceans, but *O. grayi* appears to be restricted to sea snakes. Darwin (1851) originally described it from

Pelamis bicolor (= *P. platurus*); Annandale (1909) reported it from 7 species of hydrophiids from the Indian Ocean and the Arabian Sea. Jeffries and Voris (1976) misidentified sea-snake barnacles studied by them as *Dichelaspis pellucida* but, without making reference to their previous error, later (Jeffries and Voris, 1979) tacitly assigned them to *O. grayi*. The authors screened 1,364 sea snakes from the Straits of Malacca and the South China Sea, representing 17 species (9 genera), for epizoites. Two hundred and thirty-four individuals (17 %), representing 13 species (8 genera) of snakes, bore *O. grayi*. No barnacles were found on *Cerberus rhynchops* (n = 28), *Hydrophis inornatus* (n = 2), *H. melanosoma* (n = 76) and *H. ornatus* (n = 20). Three of the host species examined were represented by relatively large numbers of individuals. Twenty-five percent of 345 *Lapemis hardwicki,* 13 % of 326 *Enhydrina schistosa* and 27 % of 128 *Hydrophis fasciatus* were found to carry *Octolasmis grayi*. The number of barnacles per snake varied from 1 to 62, but most hosts had only a few (1 to 3). *O. grayi* may attach to every region of the snake's skin, but occurred in greatest abundance on the posterior quarter of the body and the tail. The cirripeds grow to about 14 mm total length and become reproductively active at about 3.3 mm length. The cement pad secreted by *O. grayi* has an intimate relationship to the skin's scale surface and its micro-ornamentation but apparently does not permeate its surface (Jeffries and Voris 1979).

Unlike *Octolasmis grayi, Platylepas* invades the skin more deeply. In contrast to the above-mentioned members of the genus, *P. ophiophilus* is found only on sea snakes. Zann (1975) recorded it from 46 of 90 Indo-Pacific hydrophiids representing 5 genera and 7 species, with numbers ranging from 1 to 585. Most of the barnacles were attached to the posterior two-thirds of the snakes' body.

In view of the fact that sea snakes shed their skin at regular intervals (in the range of some 2 to 6 weeks), hydrophiid-specific barnacles must adjust their life cycle very precisely to the peculiarities of their hosts. It is thought that the great majority of barnacles are shed with the old skin. Hence, growth to adulthood and reproduction must be completed within 2 subsequent sheddings. However, some well-attached individuals, particularly the firmly anchored *Platylepas ophiophilus,* may be retained after shedding. The attachment ribs of these barnacles may then grow deeply into the body of the host and survive subsequent sheddings. Such deeply embedded *P. ophiophilus* may inflict some damage on the deeper tissues of the snake, and small quantities of fresh blood may be seen surrounding the penetrating ribs (Zann, 1975). Although the host's scales may be damaged, and in spite of a tissue response to the barnacle's penetrating ribs, the relationship is not parasitic but clearly phoretic.

Other pedunculate barnacles found attached to the skin of sea snakes include *Lepas anserifera* (on *Aipysurus laevis*), *L. anatifera* (on *Emydocephalus annulatus*) and *Conchoderma* spp. (on *Lapemis hardwicki* and *Pelamis platurus*) (Darwin, 1851, 1854; Gotto, 1969; Bennett, 1971; Zann and co-authors, 1975). Cantor (1840), Wall (1921) and Dean (1938) mentioned further cirriped-hydrophiid associations.

In addition to barnacles, a wide variety of other — accidental — fouling organisms have been reported from sea snakes. These include hydrozoans, serpulid polychaetes, bryozoans and even filamentous algae (Cantor, 1840; Smith, 1926; Cuffey, 1971; Kropach and Soule, 1973; Zann and co-authors, 1975; Jeffries and Voris, 1979)..

Clark (1965) reported on an unusual association between a decapod crustacean and a marine turtle. A small hawksbill *Eretmochelys imbricata,* hand-caught near Sarasota,

Florida, in a weak and diseased condition, exhibited a deformation of some of the horny shields of the carapace. The last neutral shield formed a little peaked 'house'. The floor of this protuberance had been eaten away through the carapace to the flesh, and a small stone crab *Menippe mercenaria* was nestled in the depression. The author concluded that, since the adult stone crab has powerful claws, undoubtedly even a small individual can pick its way through the carapace of a turtle that has been weakened by other causes. Hubbs (1977) reported on a similarly unusual association between a crab, *Planes cyaneus*, and a Pacific ridley. Although capable of swimming, the normally pelagic crab was found concealed in the pectoral axil of the turtle.

On a few occasions, small isopods *Eurydice* sp. were found clinging to the margins of the eyelids of *Chelonia mydas* from Malaya and Sarawak. They were not obviously associated with lesions or other signs of pathology (Hendrickson, 1958).

TUMOURS AND ABNORMALITIES

Benign and malignant growths appear to be rare among the Reptilia. Murray (1908) stated that no malignant neoplasms had been reported from reptiles up to that time, and Scott and Beattie (1927) pointed out that of 6,000 reptiles of various species autopsied at the London Zoological Gardens from 1900 to 1925, only 2 showed tumours as the cause of death. In their review of neoplasia in cold-blooded vertebrates, Lucké and Schlumberger (1949) listed tumours diagnosed as adenoma, papilloma, carcinoma and rhabdomyoma from 5 species of "Chelonia (turtles)".

Smith and Coates (1938) observed multiple warty growths of the skin in a single large individual of *Chelonia mydas* maintained at the New York Aquarium. The turtle originated from Key West, Florida. None of 4 other chelonians — 2 green turtles and 2 loggerheads — sharing the tank with the diseased green turtle, had lesions of this kind, although an exposure to possible infection had existed for more than a year. Subsequently, similar cutaneous fibro-epithelial growths were observed in 3 of some 200 wild *C. mydas* caught in the waters south of Key West.

The tumours were distributed in the region over the dorsal part of the neck in both pigmented and non-pigmented areas, as well as in the light grey or whitish areas of both axils and both groins. Several small warty excrescences occurred in the outer regions of the upper eyelids involving the conjunctivae. According to their microscopic architecture and tissue involvement, the growths were classified as papillomata and fibromata. At times, both forms existed in a combined form of tumour. In size, the lesions varied from 3 to 30 mm in diameter; one particularly large growth was 8 × 6 × 5 cm in dimension. When arising from pigmented skin portions, the tumours were dark grey or black in colour, due to large numbers of perivascular melanophores. There was no evidence of malignant changes in these fibro-epithelial turtle tumours, mitotic figures being few in number. No parasites were observed in the lesions, nor were there any cell inclusions suggesting viral etiology (Smith and Coates, 1938).

Lucké (1938) studied multiple large papillomas, similar to those described by Smith and Coates (1938), and developing on 3 *Chelonia mydas* individuals caught off Cape Sable (Florida). The tumours were located on the tail, flippers, axils, neck, eyelids and corneas. In one instance, bilateral corneal growths had produced blindness. The connective-tissue component became more prominent as the tumours increased in size, and their cellularity

gave evidence of active proliferation. Although this never yielded the picture commonly associated with malignant connective-tissue tumours, numerous spherical masses, 3 to 5 cm in diameter, occurred in the lungs of one of the turtles. These masses were composed of dense fibrous tissue covered by ciliated, columnar, respiratory epithelium. Whether they were primary tumours of the lung or whether they represented metastatic dissemination of the neoplastic connective tissue of the skin papillomas could not be determined. An origin *in situ* appeared most probable (Lucké, 1938; Schlumberger and Lucké, 1948).

In a subsequent study, based on the examination of larger amounts of material (about 250 tumours), Smith and Coates (1939) diagnosed the fibro-epithelial *Chelonia mydas* tumours as a 'neoplastic disease'. In one of the growths studied, taken from the lower eyelid of a turtle, an irregular adenomatous change occurred, which was suggestive of early malignancy. In addition, trematode ova, 260 to 310 μm in length and 30 to 40 μm in width, were observed in more than half of the tumours. The ova were identified as belonging to the blood-vascular trematode *Hapalotrema constrictum* (see Section 'Agents: Trematoda'). They were present in both the papillomatous and fibromatous type of growth but not in healthy tissues bordering the tumours. Frequently, they occupied tissue spaces resembling small venules or lymphatics. The authors concluded that the parasite eggs were deposited in pre-existing vascular tumour tissue by the migrating blood fluke (which, however, was not found in the tumours examined), and that they were probably not of primary importance as a factor in the etiology of the disease.

In another subsequent study, Nigrelli (1942) and Nigrelli and Smith (1943) found leeches *Ozobranchus branchiatus* (see Section 'Agents: Hirudinea') to be associated with the presence of fibro-epithelial tumours in *Chelonia mydas*. The growths were of the same type as described by Smith and Coates (1938; see above). As many as 50 *O. branchiatus* were found attached to one-half inch square of tumour surface. The leeches, which were attached to the growths by means of their posterior suckers, usually buried so deep in the crypts of the papillomata that in many instances it was difficult to distinguish between the growth proper and the worms. The authors pointed out that the leeches may act as vectors of viral or other parasitic organisms, which may be the causative agents of the tumours. However, microscopic examination of sectioned and stained leeches revealed no evidence of such agents.

Nigrelli (1942) and Nigrelli and Smith (1943) further argued that, since leeches feed on blood and prevent their host's fluids from clotting by the buccal secretion of hirudin, such a continuous flow of blood makes an excellent medium supplying slow-growing tumour cells with the necessary nutriment. Moreover, there are reports in the literature which indicate that hirudin, *per se*, may have an effect on tumorous growths.

In the reviewer's opinion, a more simple relation appears more likely: Since the fibro-epithelial turtle tumours are highly vascularized (Smith and Coates, 1938), they represent ideal attachment sites supplying the worms with plenty of host blood. The higher abundance of leeches on the tumours is, therefore, not at all surprising. With respect to the accumulation of trematode ova in tumour tissue, as observed by Smith and Coates (1939), it appears likely that the ova circulating in the blood-vascular system of *Hapalotrema constrictum*-infested turtles are simply being trapped in the highly vascularized portions of these growths.

Smith and co-authors (1941) described a papillomatous disease of the gallbladder of *Chelonia mydas*, caused by *Rhytidodoides similis* infestation (see Section 'Agents: Tre-

matoda'). Although the authors argue that malignancy might readily be expected as a result of these chronic hyperplastic lesions, no such malignant invasive changes have as yet been noted in approximately 100 trematode-infested turtle gallbladders examined.

There are very few reports on abnormalities in marine turtles. Most of these refer to partially or totally lacking flippers. Obviously, such 'abnormalities' result from attack by predators, mainly sharks (Brongersma, 1972). Fletemeyer (1977) and Limpus and co-authors (1979) briefly reported on 2 cases of albinism, one in a hatchling *Chelonia mydas*, the other in a breeding *Caretta caretta*.

Kyphosis — abnormal curvature of the spine resulting in a humpback appearance — has repeatedly been observed in chelonians (Carr, 1952). Lordosis, a swayback condition, was found to develop in significant numbers of pen-reared *Chelonia mydas*, although none of the affected individuals exhibited this abnormality at the time of hatching. However, swaybacked turtles appeared otherwise normal, and none died in captivity (Witham and Futch, 1977).

Acknowledgements. I am grateful to M. Söhl and G. Hoops for valuable assistance in literature procurement, to H. Lauckner for typing the manuscript and to M. Söhl for technical assistance in the preparation and improvement of the illustrations.

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3. DISEASES OF AVES (MARINE BIRDS)

G. LAUCKNER

Among the animals allied to the sea with respect to their activities and life cycles, birds have the weakest affinities to the marine environment. Of the 156 currently recognized avian families, only 9 are specialized as *sea*birds in the strict sense — the Speniscidae (penguins), Diomedeidae (albatrosses), Procellariidae (petrels), Hydrobatidae (storm-petrels), Pelecanoididae (diving petrels), Phaethontidae (tropicbirds), Sulidae (boobies), Fregatidae (frigatebirds), Alcidae (auks), and a few species of Sternidae (terns). Members of these families share the following characteristics: They derive all their food from the sea, they void virtually all their faeces into the sea, and when individuals die they do so at sea. They participate in marine energy cycles during all aspects of their life except for the time spent in breeding and nesting activities. Because of these characteristics, members of the above families should be recognized as true marine organisms (Ainley, 1980).

It must be borne in mind, however, that there is a gliding transition from the typical high-sea birds at one end of the scale over a large group of birds occupying mainly coastal habitats to the (mainly charadriiform) shore- and wading birds, which have the weakest affinities to the open water. Thus, the Gaviidae (loons) Phalacrocoracidae (cormorants), Pelecanidae (pelicans), Stercorariidae (skuas), Rhynchopidae (skimmers) and the majority of the Sternidae (terns), Laridae (gulls) and Anatidae (ducks) occupy a distinctly intermediate position between these extremes. Since representatives of these families are involved only part of the time in marine energy cycles, and since they remove energy from marine ecosystems that is not returned, they cannot by regarded as marine organisms in the strict sense. As pointed out by Ainley (1980), the designation 'seabirds' for this group is derived from a general but rather misleading conception shared by most people. The 'sea gull' is a typical example. Birds fitting into the above category will be termed 'coastal birds' here, which simultaneously marks them off from the genuinely marine '*sea* birds', as well as from the '*inland* birds'.

As secondary or tertiary carnivores (i.e., predators at the second or third trophic levels; Ainley and Sanger, 1979) with high metabolic rates and food consumption, piscivorous seabirds occupy an important position in marine food webs, equivalent to that of larger fish and mammals. Observations and estimations of the order of magnitude of seabird predation on marine fish (Sanger, 1972; Idyll, 1973; Wiens and Scott, 1975; Prévost, 1976; Laws, 1977; Furness, 1978a, b, 1982; Furness and Cooper, 1982) suggest an immense impact of seabirds on marine ecosystems and resources. Thus, Furness (1978b) estimated that great skuas *Catharacta skua* and Arctic terns *Sterna paradisea* remove approximately 29 % of the total annual fish production within a 45 km radius of their colonies. Similarly, Wiens and Scott (1975) estimated that common murres *Uria aalge* consume some 11 % of the pelagic fish produced annually in Oregon waters. Although these, as well as similar estimations have received some criticism (Bourne, 1983), the role of piscivorous seabirds in marine ecosystems must be regarded as

important. The impact of shorebirds (which are mainly primary or secondary carnivores) on littoral faunas is no less severe. As an example, oystercatchers *Haematopus ostralegus* and Eider ducks *Somateria mollissima* remove some 20 to 30 % of the cockles *Cardium (Cerastoderma) edule* from North Sea tidal flats (Drinnan, 1957; Davidson, 1967, 1968; Swennen, 1976; Sutherland, 1982).

Nevertheless, in their discussions and models of enery flow in oceans and coastal waters, marine biologists rarely make specific mention of seabirds. Ainley (1980, p. 52), criticizing this situation, concluded:

> "In the light of these real and potential impacts, the fact that marine ecologists generally overlook seabirds, is surprising".

As a consequence of this notorious lack of interest in seabird biology, little is known about the parasites of the genuinely marine species, and next to nothing about their microbial and protozoal diseases. Like the mammals of the high seas, they may be expected to be relatively free of microbial and protozoan pathogens, and the same holds true for metazoan parasites. High-sea birds have an indigenous helminth fauna, which mainly comprises nematodes and cestodes and is poor in species. On the other hand, marine birds can become carriers of non-avian parasites. As they share their food with the mammals of the sea, they may accidentally acquire immature stages of mammalian parasites transmitted by fish.

Thus, nematodes *Pseudoterranova (Phocanema, 'Porrocaecum') decipiens* have been reported on several occasions from penguins, cormorants, and other fish-eating birds, as well as from scavangers, such as sheathbills, from high-latitude waters (Johnston and Mawson, 1945, 1953; Mawson, 1953; Kreis, 1958; Howie and co-authors, 1968; Jones and Williams, 1969b). *P. decipiens* is a common and cosmopolitan parasite of seals (see Chapter 5). Both the adult and the larvae have been recorded under a variety of generic and specific names from numerous species of fish including Antarctic forms (Baylis, 1929; Johnston, 1938; Johnston and Mawson, 1945; Myers, 1959). In seabirds, the 'wrong' hosts for *P. decipiens,* this nematode does not attain sexual maturity.

Similarly, acanthocephalans of the genus *Corynosoma* have been reported from cormorants from the southern hemisphere (Johnston and Edmonds, 1953). An example is *C. hamanni,* which parasitizes South Georgian and South Orkney Island sheathbills *Chionis alba* (Howie and co-authors, 1968; Jones and Williams, 1969b; Williams and co-authors, 1974). The 'true' final hosts of these worms are marine mammals (Edmonds, 1957; Golvan, 1959), the specimens isolated from birds being all immature. Larval *Corynosoma* spp., including *C. hamanni,* occur abundantly encapsulated in teleost fish from the same waters (Baylis, 1929; Zdzitowiecki, 1978). As sheathbills are scavengers, they may acquire their *C. hamanni* infestations by ingesting either fish or littoral amphipods, the probable intermediate hosts of these acanthocephalans.

While the helminth parasites of marine mammals have received considerable attention, those of marine birds — and particularly those from the polar regions — have largely been neglected. There is a notorious lack of life-cycle studies. Only a few Antarctic invertebrates have thus far been screened for intermediate stages of vertebrate parasites. Some species of polar seabirds, such as penguins and petrels, consume large amounts of euphausiid crustaceans (Emison, 1968; Mougin, 1975). A number of larval helminths have been reported from these invertebrates, but all appear to have their adult stages in non-avian final hosts (Lauckner, in prep.).

There are a few records of the occurrence of larval trematodes in Antarctic invertebrates (Martin, 1952; Graefe, 1968, 1969). Compared with the conditions prevailing in lower latitudes, prevalences of larval stages in polar invertebrates appear to be negligible. Low temperatures seem to be the main factor controlling digenean development in Antarctic waters (Szidat, 1965). Of 353 individuals of *Laevilittorina caliginosa*, collected in the intertidal zone of Bahía Esperanza (Antarctica), only 2 (0.57 %) harboured rediae of a notocotylid bird trematode, presumably *Paramonostomum antarcticum*. Six hundred fifty-seven further intertidal gastropods, representing 6 species, yielded not a single digenean infestation (Graefe, 1968). This appears to be the only record of larval stages of an avian trematode in the Antarctic, the trematodes studied by Martin (1952) and Graefe (1969) being parasites of fish. Two species of adult notocotylids, one of which possibly represents the mature stage of the cercaria found in *L. caliginosa*, occur in Antarctic and Subantarctic *Chionis alba* (Graefe, 1968; Jones and Williams, 1968, 1969a). Gymnophallids — believed to represent *Gymnophallus deliciosus*, a common gall-bladder trematode of seabirds on the northern hemisphere — were the only other digeneans found in the sheathbills. Diving petrels *Pelecanoides georgicus* and *P. urinatrix* did not harbour any digenean (Jones and Williams, 1969b; Williams and co-authors, 1974).

Renicola sloani, a potentially pathogenic kidney trematode (see below), has been recovered from several species of penguins (Campbell and Sloan, 1943; Wright, 1954). The birds, however, had been held captive in British zoos for extended periods of time prior to autopsy, and no doubt had acquired their kidney parasites in captivity. Renicolids have not been reported from the free-ranging Antarctic and Subantarctic seabirds examined by Graefe (1968), Jones and Williams (1969a, b) and Williams and co-authors (1974). Larval renicolids have not been found in invertebrates from the same areas either.

Quite different conditions are encountered in coastal birds: They spend considerable time, if not most of their time, on land and they reproduce in terrestrial habitats. Coastal birds may establish breeding colonies or roosting places far inland, and continental species may frequent the sea's littoral fringe or the wide tidal flats for feeding. Many species perform seasonal migrations between coastal and inland habitats. Others undertake more ephemeral excursions between marine and non-marine habitats and feeding grounds, as vividly mirrored by their 'mixed' parasitofauna consisting of terrestrial, limnic and marine forms.

Coastal birds have many — particularly microbial — diseases in common with continental birds, acquired during their sojourn on land or in freshwater. Some of these are typical 'poultry diseases' (Davis and co-authors, 1971). Thus, infection with *Pasteurella multocida*, the causative agent of fowl cholera, has been found responsible for repeated epizootics among Eider ducks *Somateria mollissima* and other coastal birds (Klukas and Locke, 1970; Locke and co-authors, 1970; Korschgen and co-authors, 1978). Two hypotheses have been advanced regarding the source of this outbreak: Contagion by domestic poultry or by refuse from international shipping (Gershman and co-authors, 1964).

Salmonellosis — also frequent among coastal birds, particularly gulls — is acquired in a similar way. Aspergillosis is another common source of avian mortality. The causative agent, *Aspergillus fumigatus*, is widely distributed as a saprophyte on decaying vegetation. Birds usually become afflicted with aspergillosis at their roosting places, but epizootic outbreaks have also been observed in zoo-held birds (Herman and Bolander, 1943; Herman and Rosen, 1947; Poulding, 1952; Appleby, 1962; Rosen, 1964).

Birds in general are known as hosts for arboviruses, a group of agents potentially harmful to man (Work, 1963; Stamm, 1966; Main and co-authors, 1976). Arthropod-transmitted arboviruses do not appear to have been positively detected in seabirds, but laboratory tests have shown that no avian species is really refractory to any of these pathogens. The isolation of Hughes virus from marine bird ticks (Philip, 1965) indicates that arboviral agents may exist in sea birds. As hosts of a species-rich ectoparasite fauna (Fitzpatrick and Threlfall, 1977), coastal birds are predisposed to serve as carriers of arthropod-transmitted diseases (Arthur, 1962).

Among the protozoan parasites of birds, Coccidia may have some significance. In its acute form, coccidiosis can be a fatal avian disease (Scholtyseck, 1956; Scholtyseck and Przygodda, 1956; Stephens, 1965). Of the few coccidians reported from coastal birds (for checklist see Pellérdy, 1974), most appear to be non-pathogenic. *Eimeria lari* causes some pathology in the small intestinal epithelium of *Larus argentatus*. *E. somateriae* produces moderate lesions in the kidney epithelium of *Somateria mollissima*. *E. bucephalae*, however, has been identified as a highly pathogenic species causing heavy summer mortalities among *Bucephala clangula* in Denmark (Christiansen and Madsen, 1948; Christiansen, 1952; Schwalbach, 1959).

With respect to the special status of coastal birds, as outlined above, their diseases will not be discussed here in detail. In the following, emphasis will be placed on their role as carriers of metazoan (helminth) parasites, which have their intermediate stages in genuinely marine animals.

In contrast to high-sea birds, coastal birds are known to harbour an exceptionally rich helminth fauna, as witnessed by the large number of host-parasite lists published. Among these metazoan parasites, the Trematoda clearly predominate, followed (in descending order) by the Cestoda, the Nematoda, the Acanthocephala and, eventually, such exotic forms as the Pentastomida. The number of helminths recorded from individual bird species distinctly increases with the increasing diversity of their food items. Gulls, with their extremely wide food spectrum and feeding habits (Spärck, 1951; Meijering, 1954; Bergman, 1960; Ehlert, 1961; Harris, 1965; Löhmer and Vauk, 1969; Bakke, 1970, 1972c; Hatch, 1970; Hartwig, 1971; Spaans, 1971; Kock, 1974; Davis, 1975; Hartwig and Söhl, 1975, 1979) provide an excellent example of such a relationship.

Pemberton (1963) identified *Larus argentatus* from Britain as host for 21 species of helminths — 7 trematodes, 6 cestodes, 7 nematodes and 1 pentastomid. In the same investigation, *L. fuscus* was found to harbour even 12 species of trematodes. Loos-Frank (1971) reported 18 species of Digenea from *L. argentatus* on the East Frisian coast alone. A checklist of the parasitic helminths of *L. canus* (Bakke, 1972a) records 65 species of trematodes, 34 species of cestodes, 26 species of nematodes and 3 species of acanthocephalans. Similar parasite lists exist for other coastal birds.

The majority of these helminths are believed to cause little if any pathology in their avian hosts, at least if not present in excessive numbers. Mass mortalities of birds due to trematode infestation have been documented for non-marine forms, such as *Sphaeridiotrema* spp. and *Plagiorchis laricola* (Price, 1934; Heinemann, 1936; Foggie, 1937; Szidat, 1937; Burns, 1961). These, like many other species, have their closely related counterparts in coastal birds, and one might wonder why these should not be capable of producing similar pathology and mortality. However, most papers on bird helminths are concerned with species records and descriptions, putting aside pathological

considerations. It is certainly not incidental that, as a rule, significant numbers of intestinal parasites are more frequently recovered from moribund than from healthy-looking avian hosts.

Relatively mild pathology has been reported for renicolid trematodes invading the kidneys of marine and coastal birds (Timon-David, 1933; Wright, 1955; Riley and Wynne Owen, 1972). Hill (1952, 1954), however, believed *Renicola* sp. to cause debility and possibly death of its avian host; and Campbell and Sloan (1943) considered that very heavy infestations with *Renicola* sp. might be fatal to penguins.

Local host-tissue reactions are common in response to intestinal trematodes present in low-to-moderate numbers. However, excessively high parasite burdens may cause destruction of large areas of intestinal mucosa, inflammation of considerable portions of the mucosal epithelium, hyperaemia, dilation of tissues and intestinal blood vessels and, eventually, host death. Thus, an adult individual of *Larus fuscus*, found dying in an extremely emaciated state on the beach of Sylt (German North Sea coast), exhibited, upon autopsy, massive enteritis. The intestinal lumen contained considerable quantities of free blood. The empty small intestine of this single animal yielded over 4,400 adult *Cryptocotyle lingua* in almost 'pure culture', a figure hitherto not reported in the literature. One gains an impression of the amount of observable intestinal damage if one realizes that this bulk of worms had been crowded together along a 50-cm portion of the intestinal tract. In addition to the large number of *C. lingua*, a few microphallid trematodes (26), acanthocephalans (2), cestodes (11) and stomach nematodes (13) were recovered from the alimentary tract of the above lesser black-backed gull. No other macroscopically or microscopically detectable disease agents were found. Although the *Tetrabothrius* cestodes present must be regarded as being potentially capable of depriving their host of its vitamin B_{12} reserves (von Brand, 1967), debility and death of the gull was considered to be primarily due to the unusually heavy *C. lingua* infestation (Lauckner, pers. obs.).

Vast numbers of helminth parasites have been recovered from Eider ducks *Somateria mollissima* primarily affected by an oil spill. Total numbers of worms — including echinostomatid, gymnophallid, microphallid, psilostomatid and notocotylid trematodes, various cestodes, nematodes and acanthocephalans — ranged from 8,300 to 92,600 (!), while counts in ducks shot on the beach and considered to be in good health state, were 1 or 2 orders of magnitude lower. The worm burdens present in the emaciated ducks were believed to have contributed to, if not caused, the death of the animals (Lauckner and co-authors, in prep.).

The occurrence of *Profilicollis botulus* (Fig. 3-1) in the above Eiders (max. 1,915 per host) may be of significance. This species and possibly other members of the Acanthocephala have been found responsible for recurrent mass mortalities of *Somateria mollissima* in Scotland, Sweden, Denmark, Finland, the Netherlands and the USA (Christiansen, 1948; Grenquist, 1951; Thom and Garden, 1955; Clark and co-authors, 1958; Swennen and van den Broek, 1960; Rayski and Garden, 1961; Garden and co-authors, 1964; Persson, 1974).

Unlike their — generally minor — *direct* effects on coast bird populations, helminth parasites with an alternation of hosts may have profound *indirect* effects on the whole littoral ecosystem. They may even exert control over entire intermediate host populations. Evidence of the destructive potential which bird trematodes develop in their intermediate hosts — at the individual as well as the population level — has been presented by

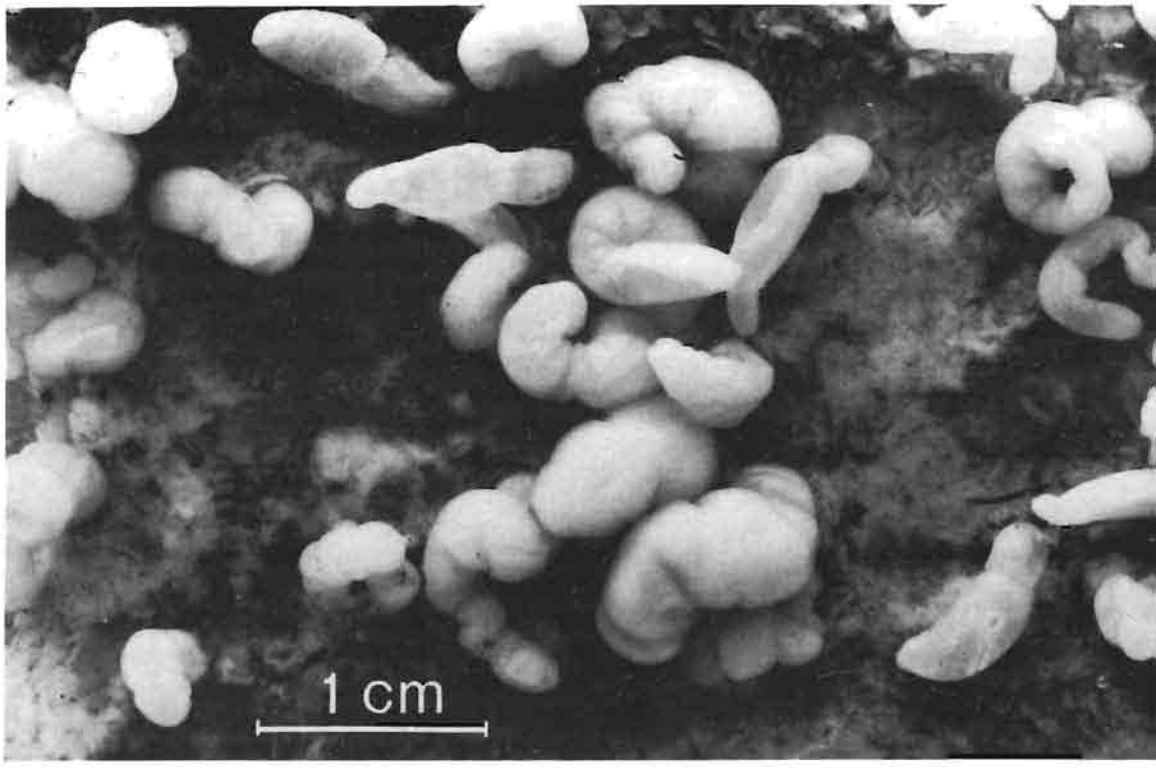

Fig. 3-1: *Somateria mollissima.* Heavy intestinal infestation with acanthocephalans *Profilicollis botulus.* (Original.)

Lauckner (1983, 1984a). The cases discussed in these papers represent characteristic self-accelerating processes. In brief, establishment of a given parasite in an ecosystem may commence with the introduction of a few infestive stages (miracidia or eggs deposited by a definite-host individual) and resultant low infestation rates in the first intermediate host. This is followed by an initially slow build-up of metacercarial infestations in the second intermediate host(s). As more and more second intermediate hosts become infested, the chance for the parasite to reach a definite host increases. The ensuing heavier worm burden of the definite hosts results in a higher density of worm eggs in the littoral environment which, in turn, entails increasing infestation prevalences in the first intermediate host, and so on.

The self-accelerating dynamics of the process may become considerably enhanced by the facts that (i) heavily infested second intermediate hosts display behavioural alterations, which render them more vulnerable to consumption by the definite host, and (ii) the definite host(s) may switch readily to this now easily available food item. In plaintext: Cockles *Cardium (Cerastoderma)* spp., heavily parasitized by metacercariae of *Himasthla elongata,* are no longer able to burrow into the sediment (Lauckner, 1983, 1984a). Moribund Eider ducks, collected from the tidal flats of Sylt (see above), were found to harbour juvenile and adult, ovigerous *H. elongata* in numbers varying from 6,100 to 89,400 (!) worms per host. Analyses of faecal droppings and stomach contents of these

ducks confirmed that they had been feeding almost exclusively on *C. edule* during their last days of life. From the viewpoint of energy expenditure it is evident that the ducks (which had been pre-damaged, at least to some extent, by crude oil) had chosen that food item (i. e., the heavily trematode-infested, non-burrowing cockles), which became available to them with the minimum waste of energy required for food collection. It is also evident that such massive definite-host infestations must result in high levels of first and second-intermediate host infestations. The cockle beds around Sylt have been grossly depleted during the past 10 years, for reasons thus far poorly understood (Reise, 1981). The number of *Somateria mollissima* breeding pairs in that area, on the other hand, has more than doubled during the period 1979–1982 (Anonymous, 1980, 1983), not to mention the increased abundance of *Larus argentatus*, the other main definite host of *Himasthla elongata* (Loos-Frank, 1967). It may be speculated that the latter parasite exerts, to a large extent, control over the cockle populations around Sylt and possibly in other areas of the Wadden Sea.

There is every indication that bird trematodes are responsible for the disappearance or the control of other faunal elements on the tidal flats of Sylt.

Case 1: *Nassarius reticulatus*. — The mud-flat snail has a continuous distribution along the North Sea coasts (Ankel, 1936). Wohlenberg (1937) and Schröder (1971) reported it in low population densities from Sylt. In 1971, Schröder found 8 of 22 (36 %) *N. reticulatus* to be infested with rediae and cercariae of a yet undescribed species of *Himasthla*. In 1972, Lauckner (unpubl.) observed the same parasite in 43 of 58 (74 %) mud-flat snails from Sylt. In 1973/74, snails were found only on rare occasions, and most of these were infested. Reise (1976) no longer recorded *N. reticulatus* from stations where it had occurred previously, according to Wohlenberg (1937). At present, it is close to extinction at Sylt. Molluscs infested with trematode rediae or sporocysts generally undergo 'parasitic castration' (Lauckner, 1980, 1983) and are thus removed from the breeding population. It appears that the *N. reticulatus* population of Sylt has not survived the heavy trematode invasion of 1971–1974.

Case 2: *Scrobicularia plana*. – James and co-authors (1977) identified this bivalve as first intermediate host for *Meiogymnophallus minutus*. Infested individuals become castrated. At Sylt, the *S. plana* population has been reduced, during the past years, to extremely low numbers, presumably due to control by *M. minutus*. Concomitantly, metacercarial infestations in *Cardium edule*, the second intermediate host, have decreased far beyond previous levels. Lauckner (1971) found all of 3,600 *C. edule* from Sylt to be infested with an average of 200 metacercariae per host. In 1984, the few remaining cockles had less than 20 larval *M. minutus* each, and some were even devoid of this digenean. Bowers and James (1967) and Lauckner (1972) consider *M. minutus* a significant parasite of *C. edule*. Its main definite host is the oystercatcher *Haematopus ostralegus* (Bowers and James, 1967).

Case 3: *Corophium* spp. – Populations of these amphipods are known to undergo marked fluctuations. In several areas of the tidal flats at Sylt, *Corophium* population densities appear to have declined during the past few years. The reasons for this are unknown, and quantitative data are not available. Individuals of *C. volutator*, examined in the reviewer's laboratory during the summer of 1975, exhibited extremely high incidences and intensities of infestation with larval trematodes (microphallids) and cestodes. Some of the crustaceans were so riddled with larval worms, crowded in the body cavity and even in

the appendages, that it was difficult to understand how these hosts could have remained alive. However, many of the more heavily infested amphipods died shortly after their transfer to the laboratory. Unfortunately, these pilot studies were discontinued. It remains, therefore, unknown whether the recent *Corophium* decline at Sylt is caused by helminth infestations, but findings reported by Muus (1967) lend support to this conjecture.

The Danish worker observed a drastic breakdown of a *Corophium volutator* population in Nivå Bay, Denmark, obviously caused by "a malignant cercaria infection" (Muus, 1967, p. 127). Larger amphipods harboured up to 40 cysts, which could fill the abdomen completely. The course of the Nivå Bay epizootic was swift and dramatic: By July 3, only a few *C. volutator* had cysts, but on July 24, metacercariae were found in over 50 % of the amphipods, and in August 100 % of the individuals were infested. Only a small proportion of the population survived. Since 0-group flatfish feed to a large extent, sometimes almost exclusively, on corophiids, the ecological consequences of such a serious depletion of a main food source become obvious.

While the pathology of larval trematodes in their invertebrate second intermediate hosts is fairly well documented (for detailed information consult Lauckner, 1983; see also fourthcoming Vol. III), the effects of larval cestodes remain to be studied. Investigations of this kind are strongly hampered by the fact that next to nothing is known about the life cycles of marine cestodes, whereby experimental infestation studies are largely precluded. With respect to the larval forms found in *Corophium volutator* (see above), there are good reasons to believe that all of them have their adult stages in coastal birds. Vauk-Hentzelt (1979) reported 10 species of cestodes, representing 8 genera, from North Sea (Helgoland) *Larus argentatus* alone. Reimer (1971) described 4 cysticercoids from Baltic Sea crustaceans and speculated on their possible identity, but the true relationships of these forms remain obscure.

All of the above bird trematodes and cestodes utilize bottom invertebrates as intermediate hosts, and their impact on the ecosystem in terms of destruction or 'parasitic castration' of intermediate hosts can be evaluated fairly well (compare Lauckner, 1983, 1984a). Difficulties arise where such highly motile animals as fish are involved as intermediate hosts of avian trematodes.

Cryptocotyle lingua represents one such example. Its life cycle involves the common periwinkle *Littorina littorea* as first host, and various species of teleost fish as second intermediate hosts. Piscivorous sea- and coastal birds are final hosts (Stunkard, 1930). *C. lingua* is an ecologically highly successful parasite, as indicated by the pronounced spatial and temporal stability of its intermediate- and final-stage populations. Its adult has been recorded in high infestation incidences and intensities from sea- and coastal birds all over the northern hemisphere. It is the most common helminth parasite of piscivorous gulls (e.g., Linton, 1915; Williams, 1961; Reimer, 1962; Pemberton, 1963; van den Broek and Jansen, 1964, 1970; Guildal, 1964, 1968; Harris, 1964; Threlfall, 1967, 1968a, b; Loos-Frank, 1971; Bakke, 1972a, b, c; Reimer and Pav, 1973).

The rediae and cercariae of *Cryptocotyle lingua* are constant associates of *Littorina littorea* throughout its distributional range. An apparent low degree of host specificity of the first larval stages is indicated by the fact that, in addition to *L. littorea* (the main first intermediate host), other members of the genus (*L. saxatilis* and *L. obtusata* on both sides of the Atlantic Ocean, *L. sitkana* and *L. scutulata* on the North American Pacific coast) are utilized as primary hosts. For local *L. littorea* populations, infestation incidences in excess

of 80 % have been reported (e.g., Ching, 1960, 1978; Sindermann and Farrin, 1962; Chubrik, 1966; Selikman, 1966; James, 1968a, b; Werding, 1969; Robson and Williams, 1970; Stunkard, 1970; Pohley, 1976; Lauckner, 1977, 1984b).

Cryptocotyle lingua infestation in the second (fish-)intermediate host merits special attention from the economic point of view. Numerous populations of food fish, particularly those from inshore waters, exhibit high prevalences of metacercarial attack. Infestation incidences close to, or in excess of, 90 % have been recorded for cod, whiting, plaice and flounder from the North and Baltic Seas (MacKenzie, 1968; Möller, 1972, 1974, 1975; van den Broek, 1979). Möller (1975) counted up to 500 *C. lingua* metacercariae in individual infested Baltic cod, while Lauckner (1984b) estimated the metacercarial burden of an exceptionally heavily infested 38-cm long *Gadus morhua* from the same area to be in the range of 38,000 to 40,000. Similar levels of *C. lingua* infestation have previously been reported by Linton (1940), who counted a total of 76,800 metacercariae in a 32-cm long window-pane flounder *Lophopsetta maculata* from the Woods Hole (Massachusetts, USA) region.

Migratory fish may rapidly acquire significant *Cryptocotyle lingua* burdens as they move inshore (Sindermann, 1965, 1966; Shotter, 1973). Herring *Clupea harengus*, taken off the coast of Maine, may at times appear liberally speckled or 'peppered' due to the prominent pigment spots forming around each metacercaria encysted beneath the skin. Invasion of the eyes may produce 'pop-eye' individuals. Experimental challenge with massive numbers of *C. lingua* cercariae killed 1-year-old herring in 15 days and 2-year-old individuals in 30 days (Sindermann and Rosenfield, 1954; Sindermann and Farrin, 1962). MacKenzie (1968) attributed an observed 100 % mortality among caged 0-group plaice *Pleuronectes platessa* to massive natural *C. lingua* infestation. Lesions produced by cercariae penetrating the skin of the fish host may become foci of secondary bacterial infections (McQueen and co-authors, 1973).

The impact of *Cryptocotyle lingua* on natural fish populations is not known. Sindermann (1965) considers it an important parasite of western North Atlantic herring. Reimer and Jessen (1972) found samples of *Clupea harengus* from the open North Sea to be free of the parasite. But, as stated above, inshore fish are almost invariably heavily infested. *C. lingua*-caused mortality may be particularly significant in larvae and 0-group fish. A single *C. lingua* cercaria is sufficient to kill a larval herring (Lauckner, 1974; Fig. 3-2).

Fishery statistics make one believe that the proportion of fish removed from North Sea stocks by seabird predation is in the range of some 0.25 to 4 % (Bourne, 1983). Much higher figures have been calculated by other authorities (see above). Losses caused *indirectly* by sea- and coastal birds as carriers of the adult stages of fish-pathogenic parasites are possibly very much higher. Attempts to estimate parasite-(and in particular *C. lingua*-)caused fish mortality would be a challenging field for the model-makers in ecology and fisheries. It could eventually lead to a dissection of the obscure 'natural mortality' of the fisheries statistics into two well defined components — old-age mortality and disease-caused mortality.

As a starting point for model considerations of this kind, the following facts and figures may be useful:

Larids, the maine definite hosts of *Cryptocotyle lingua*, may harbour several hundred or sometimes several thousand worms per host. Adult worms have a life span of at least 2 weeks and contain, at a given time, some 180 fully developed eggs. Many thousands of

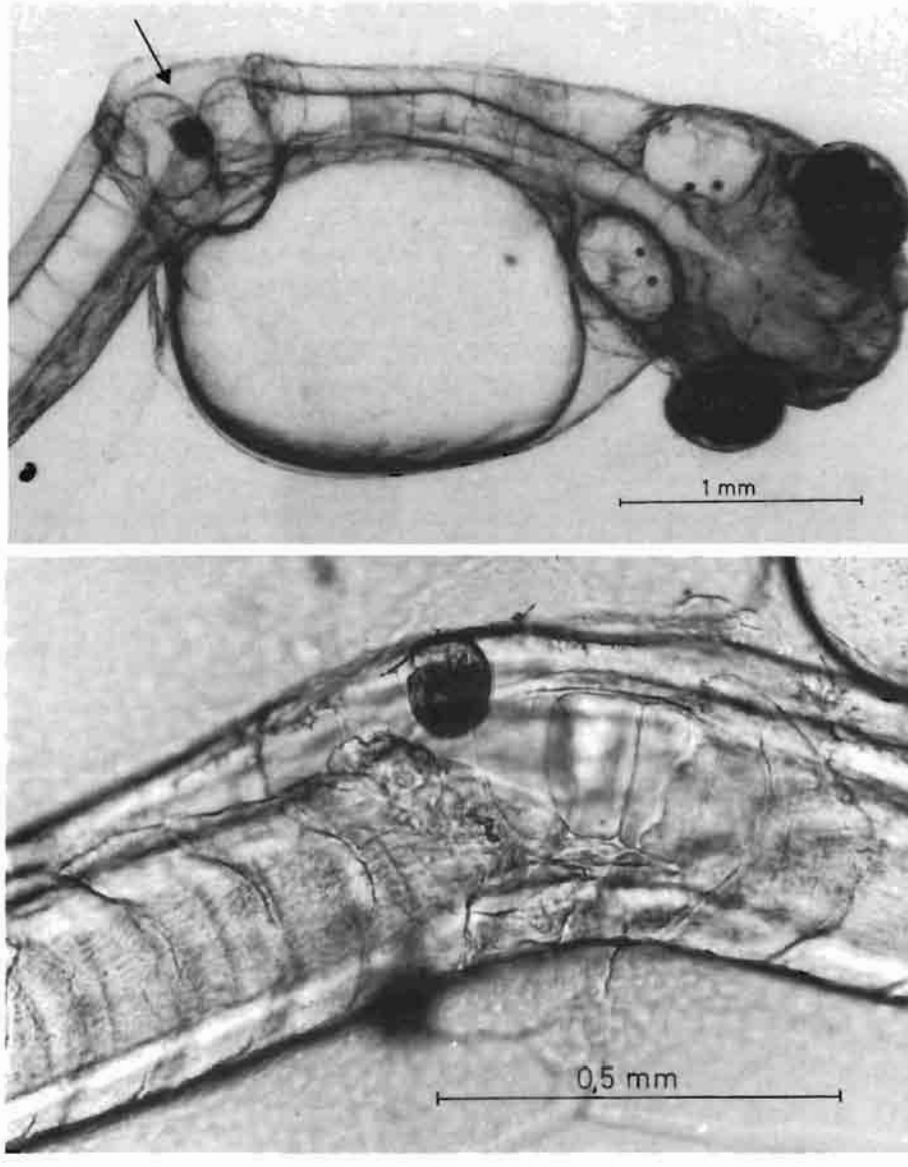

Fig. 3-2: *Clupea harengus*. (a) Yolk-sac larva showing destruction of notochord caused by a single *Cryptocotyle lingua* metacercaria (arrow). (b) Massive host-tissue destruction around encysted metacercaria, produced by proteolytic enzymes released by cercaria during penetration process. (Original.)

eggs are produced by a single worm during its life. A single egg suffices to initiate an infestation in the first intermediate host, a *Littorina* individual. Littorinids are among the most abundant littoral gastropods. Data on population densities and spatial distribution are available in the literature (for bibliography see Pettitt, 1974, 1979). Incidences of infestation with *C. lingua* in individual *Littorina* populations are usually in the range of

some 10 to 20 % (but locally well above 50 %) of the adult snails. Each infested periwinkle may shed up to 3,000 infestive cercariae per day or some 5,500,000 cercariae during its 5 years of life (Meyerhof and Rothschild, 1940; Rothschild, 1942; Sindermann and Rosenfield, 1954). A single cercaria may kill a larval fish and a moderate number a juvenile individual.

Actual incidences of trematode infestation in the hosts involved in the life cycle of a particular parasite may be influenced by the behaviour of the final hosts (Hoff, 1941). Synantropism displayed by gulls may lead to a massing of snail infestations near urban developments and sewage plants (Lauckner, 1977, 1982). Natural preserves and bird sanctuaries with their undisturbed animal life may turn into real 'épidémiotopes', i.e., foci of massive parasitic development (Bartoli, 1974; Bartoli and Prévot, 1976). Lauckner (1984a) concluded that, in certain coastal environments, particularly on tidal flats, general, indiscriminate coastal-bird protection is incompatible with the maintenance of stable ecological conditions. Data presented by James and co-authors (1977) point into the same direction.

In conclusion, whatever might be the order of magnitude of *direct* damage done to the fisheries in terms of energy removal by bird predation, this damage can probably be considered negligible as compared to the resource depletion caused by the impact of bird trematodes on the various levels of the marine ecosystem. It appears that we have to reevaluate the role of birds as carriers of parasites whose intermediate stages are harmful to marine fauna. We may also have to reconsider our present concept of coastal-bird protection.

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4. DISEASES OF MAMMALIA: CARNIVORA

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In contrast to the vast number of terrestrial and freshwater species of Mammalia known worldwide, only some 110 species are purely marine. Scheffer and Rice (1963) and Rice and Scheffer (1968) have assembled lists containing some 117 species of living and recently extinct marine mammals of the world, including a few freshwater representatives of the predominantly marine groups. In oceans and coastal waters, the phylum Mammalia is represented by 5 orders (in parentheses: number of species) — the Carnivora (1), Pinnipedia (33), Sirenia (5), Mysticeti (10) and Odontoceti (68). Consult Scheffer and Rice (1963) and Rice and Scheffer (1968) for extended general bibliography.

The sea otter, *Enhydra lutris,* a member of the order Carnivora, suborder Fissipedia, family Mustelidae, is the sole representative of the genus and the sole marine carnivore. It is the largest member of the family, which includes some 70 terrestrial or limnic species, but is the smallest of all marine mammals. *E. lutris* is the most specialized mustelid, being adapted to a narrow ecological niche in the marine environment. It never enters estuarine or freshwater.

The geographic distribution of *Enhydra lutris* formerly ranged from Morro Hermoso, Baja California (Mexico) northward along the North American Pacific coast to Prince William Sound and the south shore of the Alaskan Peninsula, throughout the Aleutian, Pribilof, and Commander Islands, along the southeast coast of Kamchatka (USSR) and through the Kuril Islands to northern Hokkaido (Japan). According to Merriam (1904), Bolin (1938), Miller and Kellogg (1955), Hall and Kelson (1959), Davis and Lidicker (1975) and others, 2 distinct forms of *E. lutris* exist along the North American Pacific coast — a northern subspecies, *E. lutris lutris* L., formerly ranging from Vancouver Island to the end of the Aleutian chain, and a southern subspecies, *E. lutris nereis* Merriam, ranging from the Strait of Juan de Fuca southward, formerly extending into Baja California, Mexico. Barabash-Nikiforov and co-authors (1947) and Roest (1973, 1976) recognized an additional subspecies or race, *E. lutris gracilis* Bechstein, the Kuril-Kamchatka sea otter. Scheffer and Wilke (1950), Rice and Scheffer (1968) and Kenyon (1969), on the other hand, assume that no subspecies of *E. lutris* are recognizable. Consideration of the genetic status of the various geographic populations of the sea otter may play a role in the transplantation and 'mixing' of individuals of different origin (Miller, 1980).

Vigorous persecution, mainly by fur hunters, has led to an almost complete extermination of *Enhydra lutris* in North American Pacific waters around 1900. In 1911, total protection was given, at least on paper, and during the last decades the sea otter populations have undergone marked recovery in certain areas, such as Amchitka Island, Alaska (Kirkpatrick and co-authors, 1955). A similar return of the sea otter has been noticed in California. From a single remnant of 50 to 100 individuals near Point Sur in the early 1900's, the California population now totals around 2,000 individuals, with an

average yearly increase of 5 to 6%. In Alaska, sea-otter population densities increase at a slightly faster rate — from a remnant of 1,000 to 2,000 individuals in 6 isolated areas to presently 100,000 or 140,000. Under full protection under state law, the return of *E. lutris* has been a success, and a viable and safe population now occupies 290 km of coastline from Santa Cruz to Pismo Beach, California (Miller, 1980).

Numerous studies have been devoted to the biology, ecology and ethology of *Enhydra lutris*, both in nature and in captivity. The reader is referred to Bolin (1938), Fisher (1939, 1940), Kirkpatrick and co-authors (1955), Limbaugh (1961), Jones (1965), Ebert (1968), Lowry and Pearse (1973), Sandegren and co-authors (1973), Houk and Geibel (1974), Estes and Palmisano (1974), Calkins and Lent (1975), Dayton (1975), Miller and co-authors (1975), Estes (1977), Loughlin (1977), Shimek and Monk (1977), Palmisano and Estes (1977), Calkins (1978), Estes and co-authors (1978, 1982), Simenstad and co-authors (1978), Duggins (1980), Breen and co-authors (1982) and Packard and Ribic (1982). Consult these authors for numerous further references.

Sea otters prey mainly upon molluscs (preferably abalones *Haliotis* spp.) and sea urchins. Wherever *Enhydra lutris* reappears, there is a dramatic decrease in the abundance of these invertebrates, accompanied by a profound change in the structure and organization of the nearshore communities. Predation upon abalones may reduce the population below a minimum level required for commercial exploitation. Sea urchins structure shallow subtidal communities by consuming most macroscopic algae (Kitching and Ebling, 1961; Himmelman and Steele, 1971; Estes and Palmisano, 1974; Breen and Mann, 1976a,b; Lang and Mann, 1976; Mann, 1977; Vance, 1979; Bernstein and co-authors, 1981; Chapman, 1981; Wharton and Mann, 1981; Dean and co-authors, 1984; and others). The result of a removal of these herbivorous invertebrates is usually a well-developed macroalgal flora and associated invertebrate and fish fauna where otters are abundant. Upon examination of 198 *E. lutris* scats from the Aleutian Islands, Murie (1940) found *Strongylocentrotus droebachiensis* to be by far the most important food item; sea urchin remains were contained in 188 of the samples, making up 74.9% of the total volume of the droppings. At locations where *E. lutris* is scarce or absent, sea urchins are large and abundant, and kelp is absent or heavily grazed (Mann and Breen, 1972; Breen and Mann, 1976a,b; Mann, 1977; Simenstad and co-authors, 1978; Estes and co-authors, 1978, 1982; Palmisano, 1983). The ecological role of *E. lutris* is particularly evident in areas into which sea otters have recently been transplanted. Rapid and extensive modification of algal species composition may be followed by a dramatic increase in kelp biomass (Duggins, 1980; Breen and co-authors, 1982). A similar recovery of the subtidal vegetation exposed to sea-urchin predation has been demonstrated to result from experimental removal of echinoid grazers by divers (Kitching and Ebling, 1961; Jones and Kain, 1967; Paine and Vadas, 1969; Sammarco and co-authors, 1974; Vance, 1979; Himmelman and co-authors, 1983) or from decimation by predators and/or disease (Pearse and Hines, 1979; Boudouresque and co-authors, 1981; Tegner and Dayton, 1981; Miller and Colodey, 1983; Scheibling and Stephenson, 1984).

In its natural environment, *Enhydra lutris* appears to have few enemies. Apart from apparently infrequent shark attacks and depredation of pups by bald eagles (Orr, 1959; Sherrod and co-authors, 1975; Fay and co-authors, 1978; Ames and Morejohn, 1980), losses due to predation appear to be minimal. Barabash-Nikiforov and co-authors (1947), Lensink (1962), Kenyon (1969), Morejohn and co-authors (1975), Schneider and Faro

(1975) and Fay and co-authors (1978) identified food shortage and resultant energy loss as the main cause of sea-otter mortalities. About 70% of the deaths were in immature animals. Primary causes of mortality seemed to be a combination of dental problems and severe winter storms preventing adequate access to food sources. In many cases, starvation is more acute than chronic. In general, there are no known enzootic diseases associated with *E. lutris* as is characteristic of pinnipeds, although food deprivation and starvation may be serious factors predisposing these sensible animals to health disorders.

DISEASES CAUSED BY MICROORGANISMS

Agents: Virales

Marine mammal medicine is a fairly recent addition to the field of veterinary medicine, and this holds particularly true for the discipline of virology. Not a single virus had been isolated from any marine mammal prior to 1968 (Watkins and co-authors, 1969). Through the medical care of captive marine mammals, particularly of Odontoceti and Pinnipedia, much has been learned about the diseases of these animals (Miller and Ridgway, 1963; Ridgway, 1965, 1972; Hubbard, 1968a, 1969; Hubbard and Poulter, 1968; Harrison, 1974; Dailey, 1978; Sweeney, 1978a,b,c,d). In contrast, the study of pathology in *Enhydra lutris,* particularly that of microbial diseases, is still in a virgin state.

Knowledge of viral diseases of pinnipeds and cetaceans has increased considerably during the past decade (Smith and co-authors, 1973, 1979; Smith and Skilling, 1979). However, as far as the reviewer has been able to determine, no disease of viral etiology has as yet been reported from *Enhydra lutris.* It appears that no special search has been made for such agents in the sea otter. On the other hand, a variety of pathological conditions — including degeneration of the liver and kidneys — have been described, which are highly suggestive of viral involvement. Viruses of this kind have been detected in pinnipeds and cetaceans, and some have been shown to be transmissible to domestic animals (Smith and co-authors, 1973; Smith and Skilling, 1979). Therefore, the occurrence of viral diseases in *E. lutris* appears highly probable, but remains to be discovered.

Agents: Bacteria

One of the most commonly reported causes of death in *Enhydra lutris* is haemorrhagic gastroenteritis (Rausch, 1953; Hubbard, 1969; Kenyon, 1969, 1972). Most sea otters found dead on Amchitka Island, Alaska, displayed terminal symptoms of enteritis similar to enterotoxaemia diagnosed in northern fur seals *Callorhinus ursinus,* harbour seals *Phoca vitulina* and Steller sea lions *Eumetopias jubatus* (Keyes, 1963, 1965; Hubbard, 1968b, 1969). From the intestine of enteritic fur seals, *Clostridium perfringens* has been isolated in pure culture (Keyes, 1963). Similar or identical micro-organisms are believed to be involved in sea-otter enteritis. Clostridia have mainly been implicated as causative agents of myositis in marine mammals. Thus far, *C. perfringens* is the sole species positively identified in marine-mammal diseases (Greenwood and Taylor, 1978). Haemorrhagic enteritis in pinnipeds and cetaceans has mainly been attributed to *Pasteurella* and *Leptospira* infections (Smith and co-authors, 1977; Sweeney, 1978a). The agent(s)

responsible for similar conditions in *E. lutris* have, apparently, never been identified with certainty.

Whatever the causative agent of haemorrhagic enteritis in *Enhydra lutris* may be, symptoms displayed by the sea otter in the final stage of the disease are (i) inability to stand or walk, (ii) semicomatose or very lethargic state, (iii) hiccup-like convulsions, and (iv) vibrissae extended rigidly forward. Necropsy almost always reveals intestinal lesions of variable extent. Juveniles may die while lesions are moderate, affecting only some 10 cm of the small intestine. Infected intestinal areas become blackish or greenish-black. In adults, long sections of the small intestine show this condition. Voiding of black faecal matter is typical in the final stage of the disease. Weight loss, probably caused by dehydration and starvation, characteristically accompanies enteritis. A captive young male otter, for instance, who had died showing symptoms of enteritis, lost 26% of its body weight within 3 days (Kenyon, 1969).

Gross pathological changes found in sea otters dying with symptoms of enteritis may include severe hyperaemia of the small intestine and a tightly adherent layer of fibrinous exudate. Gastritis is usually associated with enteritis. Petechiae are noted occasionally on the liver and, in some cases, the kidneys are hyperaemic.

Histopathological inspections often disclose severe hyperaemia of the villi, as well as massive desquamation of the mucosal epithelium. Leukocytic infiltration, however, is mostly lacking, and bacteria are not observed in the zone of inflammation (Rausch, 1953).

It was concluded that a species of *Clostridium* might be present in all sea otters, which becomes virulent when the animal is subjected to stress, such as starvation or nervous tension (Kenyon, 1969). However, bacteriological studies, conducted by Rausch (1953), gave equivocal results. Inoculations were taken from all appropriate organs, as well as from the gastrointestinal tract of enteritic sea otters. Coliform organisms predominated in all cultures. In a few cases, Gram-negative anaerogenic bacilli, sharing some antigenic properties with the *Shigella* group, were isolated. Serologically, negative results were obtained in Polysalmonella-typing attempts, and the possibility of involvement of other, yet undetected micro-organisms was taken into consideration.

Gastrointestinal enteritis is also the major mortality source in captive sea otters. Male otters were generally found to be less hardy and more susceptible to enteritis than females. When weakened animals exhibiting symptoms of the disease (black faeces) were placed in a pool of clean water and furnished adequate food, some of them recovered (Kenyon, 1969). Rausch (1953) concluded that ordinarily sea-otter gastroenteritis is associated with the presence of trematodes *Microphallus pirum* (see below), but in a few cases fatal enteritis without concomitant trematode infestation was noted. A correlation between *M. pirum* and gastroenteritis appears dubious.

In the absence of any detectable etiological agent, Rausch (1953) termed the disease 'idiopathic' (i.e., spontaneously arising) enteritis. However, comparison of the entirety of the symptoms of sea-otter gastroenteritis with the so-called 'salmon-poisoning disease' of dogs shows striking similarities (although this does not necessarily mean that both conditions are etiologically identical).

'Salmon poisoning disease' is a rickettsial malady transmitted by trematode parasites (see section 'Agents: Trematoda'). Thus far, there are no reports on the occurrence of a clearly diagnosed rickettsiosis in *Enhydra lutris*. *Nanophyetus salmincola*, the platyhelminth vector of the causative agent, *Neorickettsia helminthoeca*, was found to develop in

another mustelid, *Mustela vison* (mink), but did not reach maturity in this host (Donham and co-authors, 1926). The latter fact, however, does not necessarily preclude transmission of *N. helminthoeca* (if present) to the mustelid host.

Rickettsiae are small (0.3 to 2 μm), pleomorphic coccobacilli. As most of them are obligate intracellular prokaryotes that can survive only for short periods of time outside animal cells, rickettsiae were long considered to occupy a special taxonomic niche between the Eubacteria and the Virales. Several properties, however, clearly identify them as members of the Bacteria (Davis and co-authors, 1973).

In dogs suffering from 'salmon poisoning disease', 'coccobacillary elementary bodies', i.e., rickettsial organisms, have been found in numerous reticular cells of the reticuloendothelial tissues. These appeared either as morula-like masses or were diffusely scattered throughout the cytoplasm (Philip and co-authors, 1954a). As far as the reviewer has been able to determine, *Enhydra lutris* has never been examined for the presence of similar intracellular pathogens, and in fact, such small organisms are easily overlooked.

Among the fish-intermediate hosts of *Nanophyetus salmincola* (the *Neorickettsia helminthoeca* vector) there are salmon, trout, sculpins and other marine and estuarine fish (Gebhardt and co-authors, 1966; Millemann and Knapp, 1970b). All of these are known as dietary items of *Enhydra lutris* and should, therefore, be considered as being potentially capable of transmitting a rickettsial disease to the sea otter. Cordy and Gorham (1950) presented evidence that *N. helminthoeca* can survive in aberrant hosts without producing overt disease. After 3 blind serial passages of the pathogen through mice, lethal infections were produced in a dog and a fox using inocula prepared from these experimental hosts.

In spite of its distinct pathogenicity for dogs and foxes, *Neorickettsia helminthoeca* is not known to produce disease in homoeothermic animals other than canines, and probably does not affect *Enhydra lutris*. However, a similar organism might be involved in the etiology of haemorrhagic gastroenteritis of the sea otter. *E. lutris* in the field feeds to a large extent on molluscs, and these invertebrates are potential (carrier-) hosts for rickettsiae. Numerous, yet unidentified, rickettsiae have been detected in marine bivalves (Vol. II, p. 505). Molluscs are favourite hosts for helminth parasites, and these, in their turn, are known to transmit bacteria and viruses (Stefanski, 1959). In this context, the observation by Rausch (1953) of the apparent association of haemorrhagic gastroenteritis with the presence of *Microphallus pirum* (see section 'Agents: Trematoda') in the sea-otter intestine merits special attention. All known species of rickettsiae are serious disease agents affecting warm-blooded animals and man. Our present knowledge of the significance of rickettsioses in marine mammals is in a barren state. Therefore, future studies on *E. lutris* diseases should include screening for the presence of rickettsial pathogens. As these micro-organisms respond readily to treatment with Terramycin, Aureomycin or Tetracycline(s), enteritic sea otters should be submitted to therapy with these antibiotics.

Bacterial bone infection is another source of mortality in *Enhydra lutris*. Particularly old adults sometimes exhibit intensive dental attrition resulting in necrosis and jaw infection. In extreme cases, the teeth may be worn to the gumline. Resulting bacterial infiltration may cause considerable osteolysis of infected jaws, as well as fatty degeneration and sclerosis of the liver. Evidence of bone infection was found in about 72% of a sample of dead adult otters (Kenyon, 1969).

Bacterial paw infection has been observed in both wild and captive sea otters. In captivity, infections developed in all extremities while, in the field, they were mostly

restricted to the forepaws. Apparently, paw infections mainly result from bacterial infiltration into puncture wounds caused by sea-urchin spines and acquired during food gathering (Kenyon, 1969).

Agents: Fungi

Marine mammals may be affected, and succumb to, systemic mycoses. Infective agents generally include *Mucormyces, Histoplasma, Blastomyces* and *Coccidioides* (Sweeney, 1978a). *C. immitis*, the most infectious of these agents, has also been reported from *Enhydra lutris*.

An adult male sea otter, found in an emaciated and diseased state at Atascadero

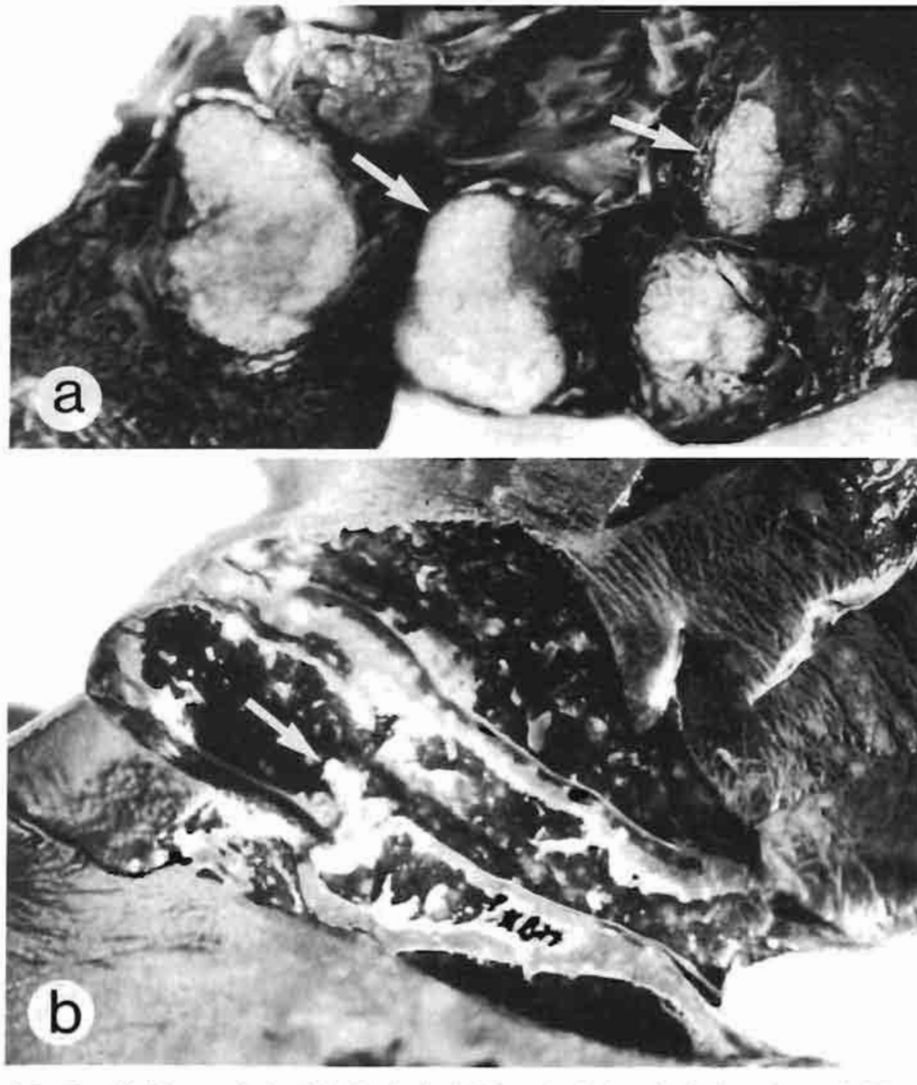

Fig. 4-1: Coccidioidomycosis in *Enhydra lutris*. (a) Granular lesions in the lung (arrows); (b) spleen with numerous white foci on both surface and cut section (arrows). (After Cornell and co-authors, 1979.)

Beach, California, was brought to Sea World, San Diego (USA), for examination and medical treatment. The animal was quite thin, weighing only 19 kg, ataxic and apathetic toward food. A deep laceration at the distal end of the tail exposed the caudal vertebrae. Administration of several drugs initially improved the health state, but eventually the otter died.

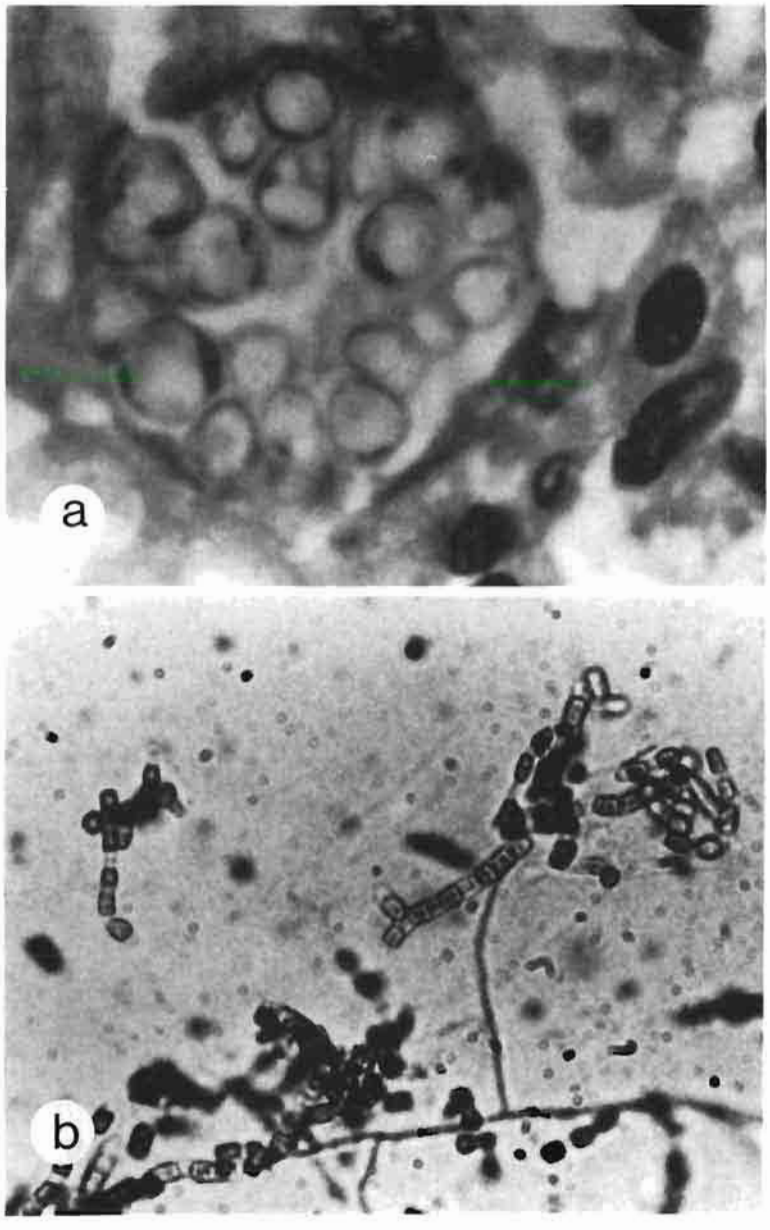

Fig. 4-2: *Coccidioides immitis.* (a) Fungal spherules containing endospores in lung tissue of *Enhydra lutris;* (b) arthrospores in culture from sea-otter lymph nodes. (After Cornell and co-authors, 1979.)

Necropsy revealed inflammation of the mucosa of the trachea, bronchi and bronchioles. Both lungs had marked interstitial emphysema. One lung lobe contained a large (4.5 × 2.2 cm) irregularly shaped, granular lesion fused to the normal lung. The lesion consisted of a whitish capsule, about 3 mm thick, and surrounding a reddish hardened granular center (Fig. 4-1 a). The apical and mesenteric lymph nodes were inflamed and oedematous. Numerous white foci, about 1 mm in diameter, were found on the surface and within the parenchyma of the spleen (Fig. 4-1 b). The liver was friable and the central lobe was greyish-tinged.

The intestinal tract showed severe enteritis and heavy acanthocephalan (*Corynosoma* sp.) infestation. Histological examination revealed a disseminated infection by a spherical-shaped fungus ranging in size from 25 to 50 μm and characterized by a double contoured capsule and endosporulation. Host response consisted of granulomatous reaction comprised primarily of epithelioid cells, as well as marked necrosis. Lesions and fungi were found in lymph nodes, liver, lung and spleen.

The condition was diagnosed as coccidioidomycosis by the morphology of fungal spherules seen in tissue sections (Fig. 4-2 a) and subsequently confirmed by the isolation of *Coccidioides immitis* (Fig. 4-2 b) from frozen lung and pulmonary lymphatic tissue (Cornell and co-authors, 1979). Fungal isolates from the diseased sea otter were found to be capable of establishing infections in mice (Walch, in Cornell and co-authors, 1979).

Coccidioides immitis has a predilection for growth in desert soils and is enzootic in the arid regions of the southwestern United States. Its windborne arthrospores (Fig. 4-2 b) readily infect homoeothermic animals including man (Davis and co-authors, 1973). Under laboratory conditions, the spores will survive in sea water and saturated sodium chloride solutions (Friedman and co-authors, 1962; Dzawachiszwili and co-authors, 1964; Egeberg and co-authors, 1964). Reed and co-authors (1976) described a case of coccidioidomycosis in a captive California sea lion *Zalophus californianus*. The above report by Cornell and co-authors (1979) is the first record of *C. immitis* from a free-ranging marine mammal.

DISEASES CAUSED BY METAZOANS

Agents: Trematoda

Thus far, only 5 species of digenetic trematodes have been reported from *Enhydra lutris* — a remarkably low figure (which, however, appears to reflect lack of scientific scrutiny rather than a sparse digenean fauna) when compared with the large number of flatworms described from species of pinnipeds and cetaceans. Four of these occur in the intestine; the fifth inhabits the gall bladder. Four of the trematodes have previously been described from Pinnipedia, which indicates a common source of infestation, i.e., identical food items.

Heterophyids *Pricetrema zalophi* — common parasites of seals and sea lions — have been recorded but once from the small intestine of an Amchitka sea otter. The minute but heavily spined worms, which measure about 435 × 260 μm, appear to have no detrimen-

tal effect on *Enhydra lutris* (Rausch and Locker, 1951; Rausch, 1953). Neiland (1961), however, suspected that heavy *P. zalophi* infestation may be an aggravating factor in the parasite-induced pathology of fur seals. More than 1,000 of these worms were recovered from an extremely emaciated *Callorhinus ursinus* pup that had been found dead on the shore at Valdez, Alaska. Willey and Stunkard (1942) reported on severe pathology in dogs produced by another marine heterophyid trematode, *Cryptocotyle lingua*. Since the Heterophyidae show remarkable uniformity in morphology and host relationships, similar pathology must be expected to result from heavy *P. zalophi* infestation in sea otters.

The life cycle and intermediate hosts of *Pricetrema zalophi* are unknown but, as in other heterophyids, fishes beyond doubt act as secondary hosts. Initially reported from *Zalophus californianus* as *Apophallus zalophi* by Price (1932), it was renamed by Ciurea (1933). Subsequent workers (Neiland, 1961; Keyes, 1965; Dailey and Hill, 1970) reported the parasite from northern fur seals *Callorhinus ursinus* and Steller sea lions *Eumetopias jubatus*. Its rare occurrence in *Enhydra lutris* may indicate that the sea otter is not a normal host for *P. zalophi*.

Opisthorchiids *Phocitrema fusiforme*, originally described by Goto and Ozaki (1930) from a captive ringed seal *Pusa (Phoca) hispida* in Japan, are similarly rare parasites of *Enhydra lutris* (Rausch and Locker, 1951; Rausch, 1953). The adult worm measures about 1.16 mm in length and 0.55 mm in width, the tegument is covered with minute spines. Its life cycle and intermediate hosts are unknown. *P. fusiforme* has been reported to occur in low abundance in seals and sea lions (Afanasiev, 1941; Neiland, 1961; Keyes, 1965; Fay and co-authors, 1978). According to Rausch (1953), it is commonly found in Aleutian Island foxes. There is nothing on pathology.

Microphallus pirum, first described by Afanasiev (1941) from Bering and Mednii Island sea otters and Arctic foxes *Alopex lagopus beringensis*, but misidentified by that author as a heterophyid and named *Paraheterophyes pirum*, is the only digenean of *Enhydra lutris* known to cause overt pathology. Its taxonomic status is confused. Belopolskaja (1952) clearly identified Afanasiev's (1941) *Paraheterophyes pirum* as a microphallid and assigned it to the genus *Spelotrema*. Unaware of the Russian authors' publications, Rausch and Locker (1951) redescribed microphallids from Amchitka sea otters as *Microphallus enhydrae*. Rausch (1953) changed its name into *M. pirum*. However, adult worms described by the Russian authors measured 700 to 800 μm in length and were said to have unusually large eggs (25 to 30 μm), whereas the American workers' worms measured only 350 to 450 μm in length and had much smaller ova (21 to 26 μm long by 11 to 16 μm wide). Further morphological differences led Deblock (1971) to consider both as separate species. In contrast, Ching (1965) — although noting differences in size, as well as in morphology, between *M. pirum* and *M. enhydrae* — regarded the latter as a junior synonym of *M. pirum*, as did Margolis and Dailey (1972).

There are several studies on the life cycle of *Microphallus* spp. from *Enhydra lutris*. Afanasiev (1941), assuming that he was dealing with a heterophyid, erroneously believed fishes to act as second intermediate hosts of *M. pirum* (as would be typical of a heterophyid). Schiller (1954) isolated encysted microphallid metacercariae, 392 to 490 μm in diameter, from 46 of 51 hermit crabs *Pagurus hirsutiusculus* collected at Amchitka Island, as well as from crabs of the genus *Telmessus* from Kodiak Island, Alaska. Larval worms excised from their metacercarial cysts were about 580 μm in length (i.e., distinctly larger than the adults of *M. enhydrae*) and believed to be *M. pirum*. Subsequently, Schiller

(1959) purported to describe the life cycle of *M. pirum* as involving rock snails *Thais emarginata* as first and hermit crabs as second intermediate hosts. But, as noted by Cable (1963), Deblock and Rosé (1965) and Stunkard (1968), Schiller's experimental procedures and interpretations were faulty; he confused larval stages of a microphallid and a renicolid trematode, which he regarded as an 'early stage' and a 'completely developed' form of one and the same species. Biguet and co-authors (1958) erroneously synonymized *M. pirum* with *M. excellens* and suggested *M. enhydrae* to be close to, or identical with, *M. pygmaeus* — an idea strongly rejected by Ching (1965) who, however, made comparisons with *M. pirum,* not with *M. enhydrae*(!). In the reviewer's opinion there appears to be strong evidence that *E. lutris* is host for 2 species of the genus *Microphallus.* In view of the reported pathogenicity of *M. pirum* in *E. lutris,* details of the life cycles of sea-otter infesting microphallids should be worked out.

Microphallus sp. (probably *pirum*; Fig. 4-3) was collected from 21 of 31 sea otters on Amchitka Island. The worms, which measured between 260 and 936 μm in length, were sometimes present in the small intestine in extremely large numbers. Heavily infested otters exhibited enteritis in varying degrees of severity. Semi-fluid stools containing a large proportion of blood were characteristic. *Post mortem* examination of such animals disclosed pronounced hyperaemia of the villi, and the dark brown to blackish colour of the

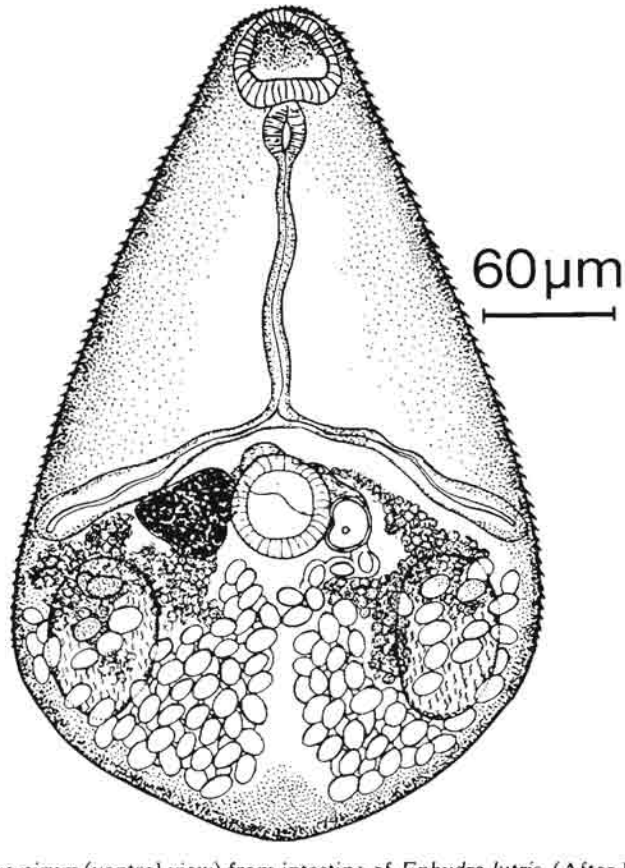

Fig. 4-3: *Microphallus pirum* (ventral view) from intestine of *Enhydra lutris.* (After Rausch, 1953.)

intestinal contents indicated heavy extravasation of blood. Gastritis, although relatively mild, was noted in most cases. A maximum of 30 *M. pirum* per mm^2 of mucosal surface were counted.

Histological examination of gut material disclosed severe mucosal hyperaemia, as well as extensive desquamation of mucosal epithelium. The worms were found at all levels in the intestinal wall (Fig. 4-4). Maximum numbers occurred in the mucosa, but some had

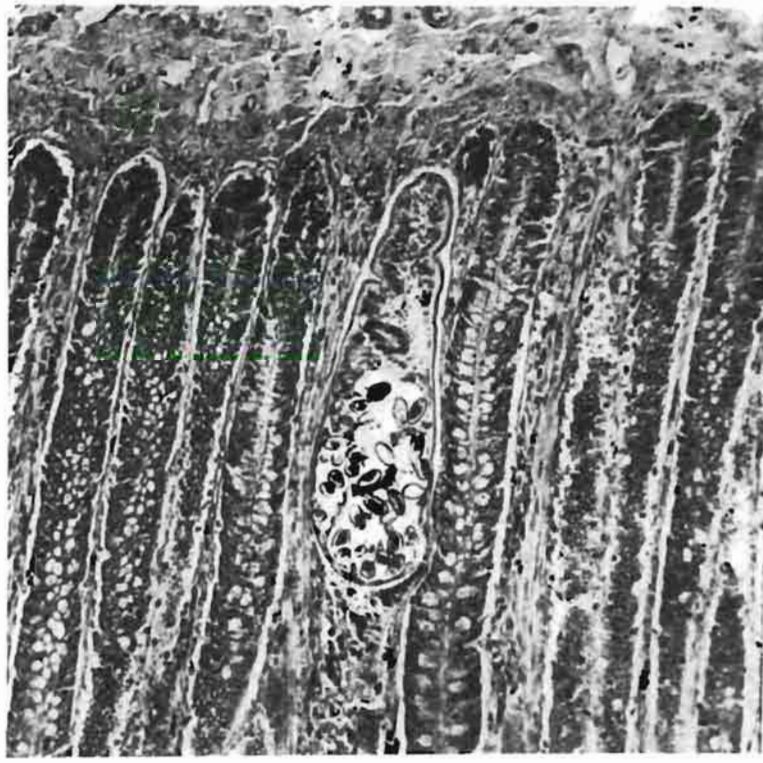

Fig. 4-4: *Microphallus pirum in situ* in mucosa of *Enhydra lutris* intestine. (After Rausch, 1953.)

penetrated the muscularis externa. Complete intestinal perforation was also observed, with worms located on the serosal surface. In general, there was little if any leukocytic infiltration, indicating that the trematodes were actively migrating through the tissue. On the basis of the observed mechanical action alone it was concluded that *Microphallus pirum* is distinctly pathogenic. However, although bacteria were not much in evidence anywhere and were not seen to invade living host tissue along with the trematodes, it was conceded that at least some of the effects attributed to *M. pirum* may have been caused by some other (unspecified) pathogen (Rausch, 1953; p. 648).

Another potentially harmful intestinal parasite of *Enhydra lutris* is *Nanophyetus* sp. (Troglotrematidae), reported from Commander Island sea otters (Afanasiev, 1941). The worm has not yet been recorded from Aleutian animals (Rausch, 1953), although *Nanophyetus salmincola* is a common parasite of North American Pacific fish, which act as second intermediate hosts. Infestations with metacercariae of that species are acquired in

freshwater and are carried to the sea by catadromous salmon and estuarine fish (Donham and co-authors, 1926; Simms, 1933; Bennington and Pratt, 1960; Farrell and co-authors, 1964; Baldwin and co-authors, 1967). Gebhardt and co-authors (1966) and Millemann and Knapp (1970b) have assembled lists of natural and experimental fish-intermediate hosts of *N. salmincola*.

Nanophyetus salmincola is relatively small. Adult worms taken from the intestine of dogs measured about 0.8 to 1.1 mm in length and 0.3 to 0.5 mm in width (Witenberg, 1932). In the alimentary tract of the vertebrate final host, *Nanophyetus salmincola* causes but moderate direct pathology (Chapin, 1926; Hoeppli, 1926; Cordy and Gorham, 1950). But the helminth is the carrier of a rickettsial hyperparasite, *Neorickettsia helminthoeca* (Vol. II, p. 510), which causes a fatal disease in canines termed 'salmon poisoning disease' (Donham, 1925; Donham and co-authors, 1926; Simms and co-authors, 1931a,b; Witenberg, 1932; Philip and co-authors, 1953, 1954a,b; Philip, 1955; Millemann and co-authors, 1964; Millemann and Knapp, 1970a,b; and others).

In addition to dogs, foxes, coyotes, cats, bears and pigs, a wide variety of other warm-blooded vertebrates have been found to harbour natural *Nanophyetus salmincola* infestations or could be infested experimentally (Cram, 1926; Donham and co-authors, 1926; Millemann and Knapp, 1970a). In contrast to dogs and foxes, for which the helminth-transmitted rickettsiosis is fatal in about 90% of the cases, *Neorickettsia helminthoeca* causes less severe if any pathology in other hosts. Skrjabin and Podjapolskaja (1931) described natural *Nanophyetus schikhobalowi* infestations from humans in Siberia (USSR). Subsequent workers (Witenberg, 1932; and others) regard the latter name as a synonym of *N. salmincola*. Although humans can be infested experimentally with *N. salmincola*, they do not appear to contract the rickettsiosis (Philip, 1955, 1958). At least the remote possibility exists that haemorrhagic gastroenteritis, a common and frequently fatal disease of *Enhydra lutris*, might be caused by *N. helminthoeca* or a similar micro-organism (p. 648).

Orthosplanchnus fraterculus, a campulid trematode first described by Odhner (1906) from *Odobenus rosmarus* at Spitsbergen, is a common gall-bladder parasite of walruses and bearded seals in the northern Pacific (Rausch and Locker, 1951; Neiland, 1961; Keyes, 1965; Fay and co-authors, 1978). Rausch (1953) identified it as the most common trematode of *Enhydra lutris* from Amchitka Island, Alaska, 28 of 31 otters examined being infested, some heavily. The author concluded that *E. lutris* regularly becomes infested during its first year of life.

Orthosplanchnus fraterculus is by far the largest digenean parasitizing *Enhydra lutris*. Adult worms recovered from *Odobenus rosmarus* by Odhner (1906) were about 4 to 4.5 mm in length and 0.5 to 0.63 mm in width, but Rausch and Locker (1951) obtained specimens 6 to 7 mm in length from the gall bladder of Amchitka sea otters.

In heavy infestations, the lumen of the gall bladder of *Enhydra lutris* was found to be filled almost completely by hundreds of *Orthosplanchnus fraterculus*. The worms attach themselves firmly to the mucosa, with the posterior end projecting into the bladder lumen. Considerable quantities of host tissue are taken into the muscular openings of the worms' oral and ventral suckers. The long (16 μm) integumental spines embedded in the gall-bladder wall cause chronic irritation resulting in extensive necrosis of the mucosa (Fig. 4-5). Fibrotic nodules, up to 2 mm in size, were noted. Fibrocytic proliferation may bring about occlusion of the entire bladder lumen which, however, did not appear to have

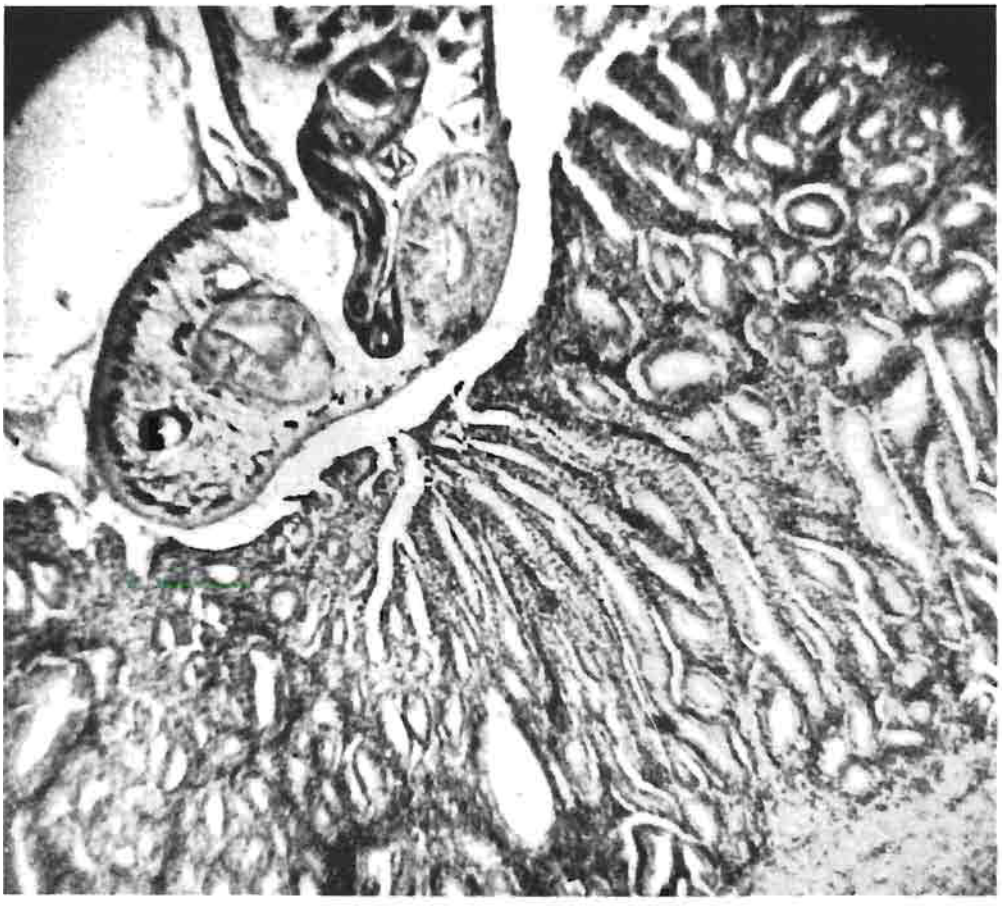

Fig. 4-5: *Enhydra lutris.* Transverse section of gall bladder with specimen of *Orthosplanchnus fraterculus in situ.* (After Rausch, 1953.)

serious consequences. Accumulation of bile distal to the occluded portion was not observed, and there was no tendency toward occlusion of the cystic duct. Hyperaemia was never marked. Mucosal hypertrophy may also be characteristic of *O. fraterculus* infestation, but adequate material from non-infested otters was not available for comparison (due to the infestation incidence, in the inspected otters, of 90%). The large worm eggs, about 0.1 × 0.55 mm in size, were often seen entrapped by the host tissue reaction (Fig. 4-6; Rausch, 1953). In spite of these pathological changes, *O. fraterculus* was regarded as but mildly pathogenic. Fay (in Kenyon, 1969), however, observed extreme *O. fraterculus*-caused fibrosis and near-occlusion of bile ducts in several portions of the liver of a subadult female sea otter found dead at Patten Bay, Montague Island. Distinct pathology of *O. fraterculus,* aggravated by secondary bacterial infection with *Staphylococcus epidermidis* and *Edwardsiella tarda,* has also been reported for bearded seals *Erignathus barbatus* by Fay and co-authors (1978). The intermediate hosts of *O. fraterculus* are unknown.

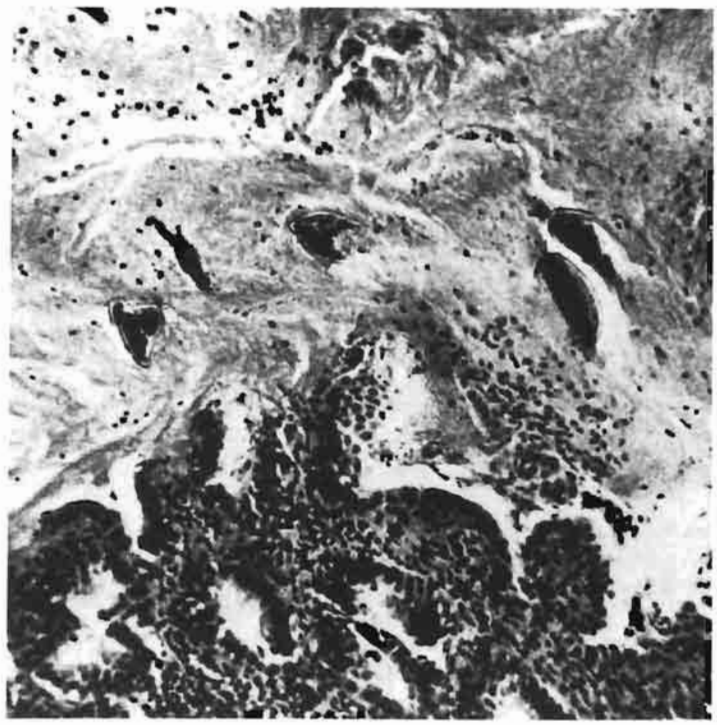

Fig. 4-6: *Enhydra lutris.* Gall bladder showing fibrosis and entrapped eggs of *Orthosplanchnus fraterculus.* (After Rausch, 1953.)

Agents: Cestoda

Diplogonoporus tetrapterus, a small diphyllobothriid cestode, has been reported from a few sea otters captured at Amchitka Island and a dead otter from Montague Island (Rausch, 1964). *D. tetrapterus* is a common parasite of Steller sea lions *Eumetopias jubatus* and other pinnipeds (Dailey and Brownell, 1972; Margolis and Dailey, 1972; Fay and co-authors, 1978). Its life cycle is unknown.

A single immature individual of another diphyllobothriid, *Pyramicocephalus phocarum,* has been recovered from the intestine of an Amchitka sea otter. The specimen was only 58 mm long. No gastric or intestinal lesion characteristic of the attachment of *P. phocarum* in other hosts was apparent (Rausch, 1953). This cestode is a very common parasite of *Erignathus barbatus,* the only host from which adult specimens have been obtained. In the bearded seal, dense *P. phocarum* populations may cause severe inflammation of the upper duodenum (Fay and co-authors, 1978). The life cycle of *P. phocarum* is unknown.

Two unidentified cestodes, found by Barabash-Nikiforov (1935) in the small intestine of a single Commander Island sea otter, are possibly attributable to *Pyramicocephalus phocarum.* Remarkably, Afanasiev (1941) reported no cestodes from Commander Island otters examined by him.

It may reasonably be concluded that *Enhydra lutris* does not appear to have a

characteristic cestode fauna. Tapeworm infestations in sea otters seem to be rare, apparently accidental, and of no pathological significance.

Agents: Nematoda

Pseudoterranova (Phocanema) decipiens, an anisakine stomach nematode normally maturing in pinnipeds, is the most deleterious helminth parasite of *Enhydra lutris.* The species has an extremely broad intermediate-, paratenic- and final-host spectrum and is cosmopolitan in distribution. Its taxonomic status has caused maximum confusion, both the larvae and the adult worms having been described under a variety of generic and specific names (for lists of synonyms consult Johnston, 1938; Johnston and Mawson, 1945; and Myers, 1960).

Krabbe (1878a,b) named worms taken from the stomach of various North Atlantic seals *Ascaris decipiens.* Baylis (1920) transferred the species to the genus *Porrocaecum* — an ill-fated error because *Porrocaecum* spp. are restricted to birds (Hartwich, 1959, 1975). Johnston and Mawson (1945; see footnote), in their turn, erroneously assigned it to *Terranova,* a badly defined genus established by Leiper and Atkinson (1914) to accomodate a single female nematode parasitic in an elasmobranch fish (!). Johnston and Mawson (1945) furthermore believed their worms, taken from Antarctic seals, to be identical with *Filaria piscium,* a larval nematode described from teleosts by Rudolphi (1809), and created the new combination *Terranova piscium.* Eventually, Myers (1959) erected the genus *Phocanema* to include *P. decipiens* as type (and sole) species. In one of her subsequent papers, Myers (1975) presented a synopsis of the generic diagnoses of *Terranova, Porrocaecum, Phocanema* and other genera in the Anisakidae, some of which are monotypic and, as emphasized by the author, need a critical review to determine their validity. It should be pointed out, in this context, that Hartwich (1975) included *Phocanema* in the Anisakidae, Anisakinae, but removed *Porrocaecum* from the latter taxon and included it in the Toxocaridae.

In neglect of the clarifying publications of Myers (1959) and Hartwich (1959, 1975), many subsequent workers erroneously continue to include *Phocanema decipiens* in the (bird-nematode) genus *Porrocaecum* (e.g., Scott and Black, 1960; Berland, 1961; Rae, 1963, 1972; Dailey and Brownell, 1972) or, more frequently, in the obscure (elasmobranch-nematode) genus *Terranova* (e.g., Delyamure, 1961; Kenyon, 1969, 1972; Young and Lowe, 1969; McClelland and Ronald, 1970, 1974; Margolis and Dailey, 1972; Suzuki and co-authors, 1972; and others). Recently, Gibson and Colin (1982), criticizing the present disagreement concerning the conception and validity of *Terranova* and the related anisakid genera *Pseudoterranova, Phocanema* and *Pulchrascaris,* suggested the acceptance of *Pseudoterranova* for *Terranova*-like nematodes from marine mammals, with *Phocanema* as a synonym of *Pseudoterranova.*

Pseudoterranova decipiens deserves special interest because its larvae, commonly known as 'codworms' (Rae, 1963, 1972; Platt, 1975, 1976) are capable of infesting man. Human cases have been documented in the literature (e.g., Buckley, 1951; Kagei and co-authors, 1972; Little and MacPhail, 1972; Suzuki and co-authors, 1972; Kates, 1973;

It was not Mozgovoy (1951) who made the erroneous nomenclatorial combination for the first time, as suggested by Margolis and Dailey (1972).

Little and Most, 1973; Jackson, 1975). Chitwood (1970), Davey (1972), Cheng (1973b, 1976), and Myers (1976) have reviewed the potential public health importance of *P. decipiens* and other marine nematodes known to cause human 'anisakiasis'.

In spite of the numerous studies, the life cycle of *Pseudoterranova decipiens* is not completely understood. Sexually mature adults occur in a variety of pinnipeds (Stiles and Hassall, 1899; Dailey and Brownell, 1972; Margolis and Dailey, 1972), and the infestive fourth-stage larvae in various species of fish (Schiller, 1954; Scott, 1954; Margolis, 1956; Scott and Black, 1960; consult the above-cited authors for numerous further references). Scott (1953) achieved the first experimental *P. decipiens* infestation in *Phoca vitulina*. Considerable insight into the physiology and the moulting cycle of the codworm has been gained by *in vitro* cultivation of eggs, larvae and adults (Scott, 1955; Townsley and co-authors, 1963; Davey, 1965, 1969, 1971; Davey and Kan, 1968; Kan and Davey, 1968a,b; Davey and Sommerville, 1974; McClelland and Ronald, 1970, 1974; Bier, 1976).

Myers (1960) obtained experimental infestations with second-stage larve of *Pseudoterranova decipiens* in a large number of invertebrates including amphipods, mysids, shrimps and molluscs (Vols II and III). No further development of the nematodes occurred and the larvae survived in these hosts for not more than 24 h. Second-stage larvae in amphipods and mysids, transferred to fish, disappeared from the intestines of these hosts within 24 h. No natural *P. decipiens* infestations were detected in invertebrate field samples, although hundreds of specimens were examined. Scott and Black (1960) screened over 8,000 mysids from Bras d'Or Lakes, Nova Scotia, Canada, for the presence of larval nematodes. Although 110 worms were found, only 1 certainly and 4 dubiously appeared to be *'Porrocaecum'*. The others were 'clearly' larval *Contracaecum* (Scott, 1957). As pointed out by Smith (1983), the *'Porrocaecum'* found by Scott and Black (1960) were, in fact, larval *Hysterothylacium* (previously known as *Thynnascaris*; see also Deardorff and Overstreet, 1981). Their 'larval *Contracaecum*' may also have been *Hysterothylacium*, as suspected by Smith (1983). To make the confusion complete, Uspenskaja (1960) claimed to have found larval *P. decipiens* in the haemocoel of armoured shrimp *Sclerocrangon borealis* from the Barents Sea. She constructed a hypothetical life cycle involving benthic crustaceans as 'first intermediate hosts' and fish as 'second intermediate hosts'. McClelland and Ronald (1973), however, believe that the Russian author might have been mistaken, because her larvae differ morphologically from typical *P. decipiens*. Based on this meagre evidence, Platt (1976) inferred a *P. decipiens* life cycle similar to that hypothesized by Uspenskaja (1960).

The discovery by Martin (1921), Scott (1954) and Scott and Black (1960) of larval *Pseudoterranova decipiens* in plankton-feeding fish such as *Osmerus eperlanus* and *O. mordax* might point toward another possible sequence of life-cycle stages of this nematode: Smelt feed on copepods and other plankton organisms such as ctenophores. Copepods are known to serve as hosts for second-stage larvae of various species of nematodes, most of which have been identified inadequately or incorrectly, due to lack of distinguishing morphological characters (Apstein, 1911; Wülker, 1929, 1930a,b; see also Vol. III). Smelt and other plankton feeders may, therefore, acquire *P. decipiens* infestations by ingesting copepods harbouring second-stage larve. Unfortunately, copepods have apparently not yet been inspected for the presence of larval *P. decipiens*. Myers (1960) did not include these crustaceans in her list of potential hosts for this species. The next

important step in this presumed life-cycle sequence — transfer of *P. decipiens* second-stage larvae from smelt to cod and other predatory fish — has already been verified experimentally by Scott (1954).

Aside from taking up *Pseudoterranova decipiens* infestations by devouring infested planktivorous fish, cod may also acquire nematodes by feeding directly on infested plankton organisms other than copepods (e.g., cnidarians, ctenophores or chaetognaths; Vol. III). Ctenophores, for example, regularly function as paratenic hosts for second- (and possibly third-) stage larvae of nematodes (Lauckner, 1980). Cod are known to feed voraciously on ctenophores, sometimes to gorging. Recent evidence suggests that ctenophores constitute a more important item in the diet of fish than previously expected. Thus, the stomachs of 27 of 50 chum salmon *Oncorhynchus keta*, caught in British Columbia (Canada) waters, contained almost entirely ctenophore remains, and in 1 chinook salmon *O. tshawytscha*, ctenophore remains constituted over 95% of the volume of the stomach contents (Black and Low, 1983).

Barabash-Nikiforov (1935) was the first who reported *'Porrocaecum decipiens'* from *Enhydra lutris* on the Commander Islands. One sea otter had 17 small and 3 large nematodes in the stomach, 19 small worms in the small intestine, 7 small specimens in the large intestine and 6 small worms in the rectum. Another otter had large nematodes in the oesophagus (1), in the stomach (13) and in the small intestine (2). The author did not state as to whether the 'large' worms were adults, nor did he indicate the developmental stage of the 'small' worms, which probably were third- or early fourth-stage larvae.

Among sea otters found dead on beaches of Amchitka Island, 2 of 27 juveniles and 5 of 14 adults harboured *Pseudoterranova decipiens* (Kenyon, 1969). Heavy infestation appears to debilitate *Enhydra lutris,* but the symptoms associated with nematode infestation could not be defined clearly (Rausch, 1953). Thus, 50 otters captured at Amchitka and believed to be in good health when killed, had *P. decipiens* and *Microphallus pirum* infestations, but there were no gross lesions in the infested individuals. On the other hand, among 7 adult Amchitka otters only 1 appeared to be in poor condition when captured. Only the ill individual was infested with an appreciable number of *P. decipiens* (65 worms) and *M. pirum* (many worms), but there was no sure indication that this otter's poor condition was a result of parasitic infestation (Kenyon, 1969).

Pseudoterranova decipiens, recovered from *Enhydra lutris,* appear all to be larvae or immature worms. Development of reproductive organs seems to occur only in the 'true' (pinniped) final host. Similarly, Scott and Fisher (1958) reported on the occurrence of *P. decipiens* in the common porpoise, *Phocoena phocoena,* from the Bay of Fundy (Canada). No trace of reproductive organs was seen in these nematodes. Rausch (1953) stated that adult *P. decipiens* do occur in *E. lutris* but are not commonly found. It appears likely that these worms are actually advanced fourth-stage larvae or immature adults. Ovigerous female *P. decipiens* have not positively been reported from the sea otter.

The pathogenicity of *Pseudoterranova decipiens* in *Enhydra lutris* largely depends on the developmental stage of the worms. The earliest stage found in the otter — second-stage larvae having a 'cephalic spike' (i.e., a cuticular boring tooth; Fig. 4-7) — appears to be the most pathogenic. The presence of these worms was always associated with intestinal perforation and seemed to be directly responsible for all *E. lutris* deaths known to have resulted from nematode infestation (Rausch, 1953; Fig. 4-8). Second-stage larvae of *P. decipiens* recovered from the musculature of greenlings *Lebius superciliosus* ranged from

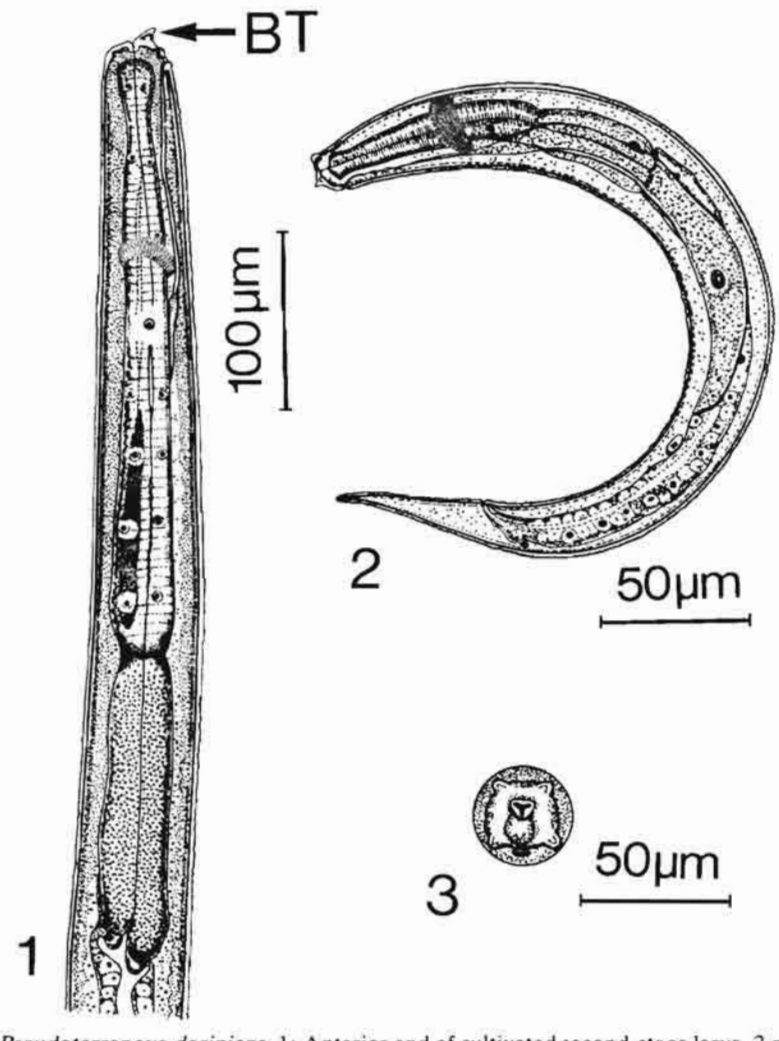

Fig. 4-7: *Pseudoterranova decipiens*. 1: Anterior end of cultivated second-stage larva, 2 mm in length (lateral view); 2: cultivated second-stage larva, 350 µm in length (lateral view); 3: *en face* view of 2 mm long larva. Note cuticular boring tooth (BT). (After McClelland and Ronald, 1974.)

30 to 50 mm in length (Schiller, 1954). Later larval stages, possessing fully developed lips (but still sexually immature) were usually found attached in closely associated aggregations in the sea-otter stomach. These were believed to be only mildly pathogenic (Fig. 4-9). Worms considered to be adults were not observed attached to the stomach mucosa nor associated in any way with intestinal perforation. These appeared to be non-pathogenic (Rausch, 1953). Young and Lowe (1969) reported similar minimal pathology from grey seals *Halichoerus grypus* infested with adult *P. decipiens*.

Sea otters captured in the terminal stages of *Pseudoterranova decipiens* infestation showed depression and weakness. However, they often fed readily up to a few hours preceding death. Both adult *Enhydra lutris* and young individuals were similarly affected by nematodes (Rausch, 1953). Fay and co-authors (1978) identified parasitization by

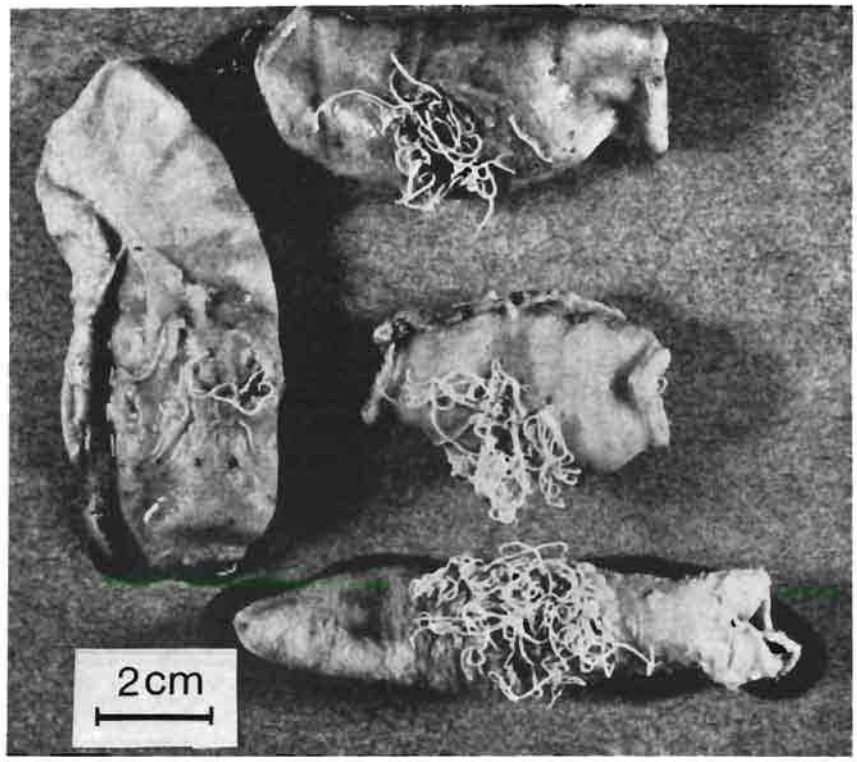

Fig. 4-8: *Enhydra lutris.* Gross specimens of duodenum showing appearance of perforations caused by second-stage larvae of *Pseudoterranova decipiens.* Note nematodes protruding through perforations from intestinal lumen. (After Rausch, 1953.)

"stomach nematodes causing ulcers" as one of the major causes of mortality in *E. lutris.* Similar ulcers have been observed in *P. decipiens*-infested northern fur seals *Callorhinus ursinus* from Alaska (Keyes, 1965) and South American sea lions *Otaria byronia* (= *O. flavescens*) from Chile, but the relation between the worms and the ulcers was not clear (Cattan and co-authors, 1976).

Generalized peritonitis appears to be the eventual cause of death in the case of intestinal perforation by *Pseudoterranova decipiens.* Upon opening the ventral abdominal wall, numerous larval nematodes may be seen lying free on the surface of the greater omentum, between it and the abdominal wall. The omentum may adhere to the inflamed area. In a few cases, the omentum itself was directly invaded by nematode larvae. The body cavity of such otters usually contains a quantity (up to 1,000 ml) of a thin, often blood-stained, fluid, and a fibro-purulent exudate may coat the serous membranes. The fluid in the body cavity may have an admixture of food remnants that evidently had oozed out of the intestine by way of the perforations. Frequently, firm visceral adhesion, accompanied by extensive fibrosis, is observed. Multiple perforations of the small intestine (Fig. 4-10) are the rule, and direct invasion of spleen, liver or other organs may occur (Rausch, 1953; Kenyon, 1969).

The intestinal perforations are surrounded by large inflamed areas, which are greatly congested and contrast markedly with the healthy tissue. Incision of the intestine at the site

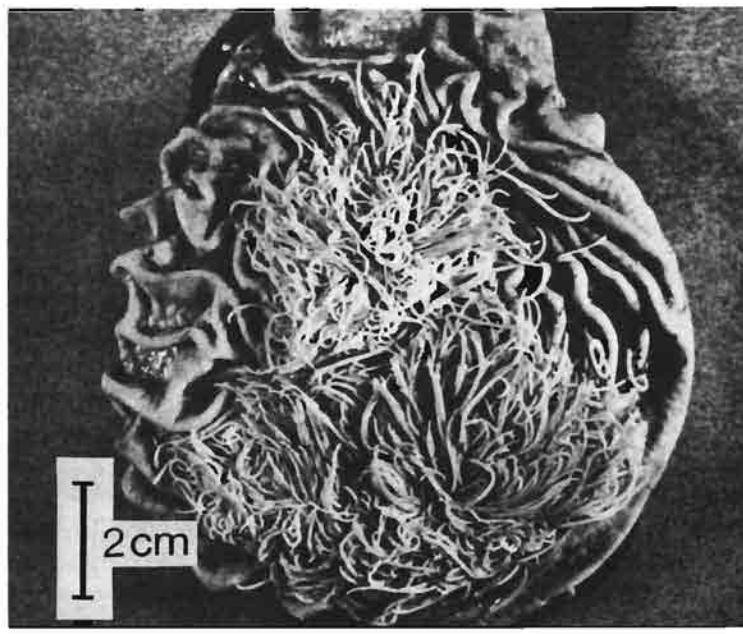

Fig. 4-9: *Enhydra lutris.* Aggregations of *Pseudoterranova decipiens* in stomach. Two larval stages are represented. (After Rausch, 1953.)

of perforations reveals dense aggregations of larvae, which appear as firmly anchored masses occluding the lumen of the intestine to a significant degree (Fig. 4-8). In the stomach, later larval stages predominate. The anterior ends of the worms are firmly embedded in the gastric wall, which may be eroded to the depth of the muscularis mucosae or, in rare cases, of the muscularis externa, in the immediate area of attachment (Fig. 4-9), but total perforation of the stomach appears to be the exception. Tissue liquefaction, apparently caused by the contents of the nematodes' oesophageal glands, occurs at the sites where the worm heads are embedded, resulting in the formation of cavities surrounded by hyperaemic tissue (Rausch, 1953; Kenyon, 1969).

The way by which *Enhydra lutris* contracts *Pseudoterranova decipiens* infestations is not clear. The apparent preponderance of early (= second-stage) larvae might suggest uptake *via* an invertebrate 'carrier' host in which the development of the worms is arrested. As stated, various invertebrates, including molluscs, can be infested experimentally with *P. decipiens.* From the absence of larvae less than 10 mm long from cod in the Bras d'Or Lakes, Nova Scotia (Canada), Scott and Black (1960) concluded that a host preceding the teleost host must be involved in the life cycle of *P. decipiens.* What appear to have been second-stage larvae have also been recovered from planktivorous fish, such as smelt (see above), as well as from greenlings *Lebius superciliosus.* The latter fish host also harboured third-stage larvae (Schiller, 1954). Rausch (1953) observed sea otters feeding on still-living *L. superciliosus.* Schiller (1954) stated that greenlings occur frequently in the diet of *E. lutris* and probably constitute the most important source of nematode infestation. However, the smallest *P. decipiens* (second-stage) larvae recovered from *L. superciliosus* musculature were 30 mm in length, whereas the smallest worms recovered from *E. lutris* measured 18 to 30 mm. Schiller believed that these small individuals may be located in the

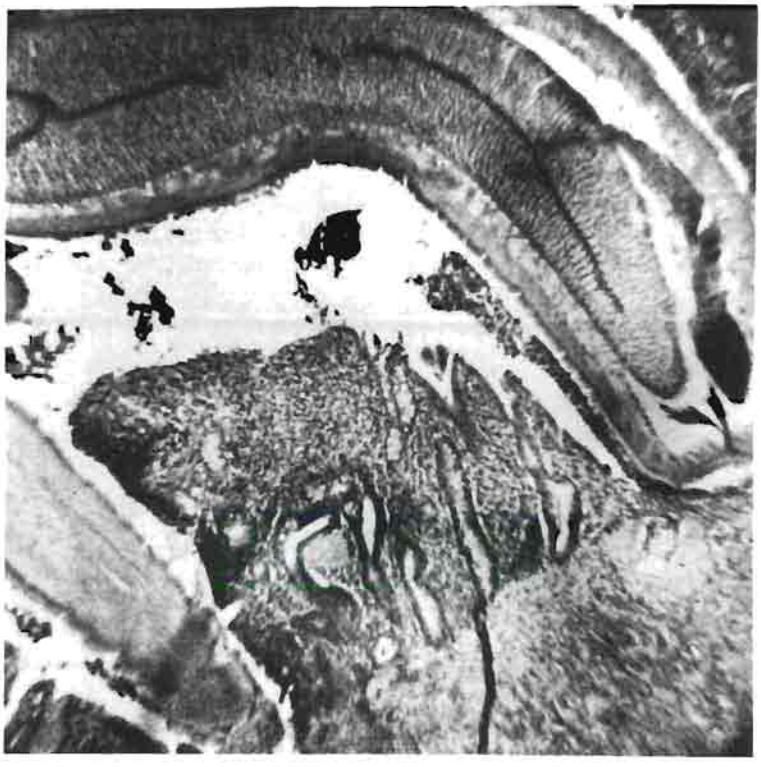

Fig. 4-10: *Enhydra lutris.* Small intestine near perforation, showing invasion by second-stage larvae of *Pseudoterranova decipiens.* (After Rausch, 1953.)

gastrointestinal tract and/or abdomen of the fish host prior to their establishment in the musculature. Fourth-stage larvae, over 50 mm in length, with lips completely developed and devoid of a boring tooth, as well as (apparently sexually immature) young adult worms, were only recovered from the stomach of *E. lutris,* mature nematodes only from pinnipeds. The largest male *P. decipiens* from *E. lutris* were about 78 mm, females about 110 mm in length (Schiller, 1954).

Although second-stage larvae of *Pseudoterranova decipiens* seem to constitute a considerable proportion of the total nematode burden of *Enhydra lutris,* there is evidence that passage through a fish-intermediate host (with concomitant development to the third larval stage) is an essential physiological prerequisite for the completion of larval growth and development. Thus, cultivated *P. decipiens* larvae in the advanced second stage were not infestive to seals, whereas third-stage larvae from cod musculature were (McClelland and Ronald, 1974).

Schiller (1954) observed that second-stage larvae when excised from fish musculature became very active and, when placed free upon a piece of fish muscle and covered with another sizeable piece of the same tissue, re-entered and completely embedded themselves in the muscle in less than 10 min. This behaviour might explain, in part, the pathogenicity of such early-stage larvae in the sea otter, as reported by Rausch (1953). Similar intestinal pathology, caused by larval *Pseudoterranova decipiens,* is not observed in pinnipeds, the 'true' natural definite hosts of this nematode.

Sea otters in captivity sometimes became heavily (and often lethally) infested with *Pseudoterranova decipiens via* fish food contaminated with larval worms. Infestations could be circumvented by routine freezing of the fish prior to feeding. Also, piperazine citrate was administered as a precautionary measure to captive sea otters by injecting an aqueous solution into fish before feeding. However, the effectiveness of this vermifuge was difficult to evaluate, but it had no ill effect on the sea otters (Kenyon, 1969).

Agents: Acanthocephala

Enhydra lutris is host for at least 6 species of thorny-headed worms of the genera *Corynosoma* and *Polymorphus. Corynosoma* spp. (in subfamily Polymorphinae) are abundantly represented in marine mammals (Lyster, 1940; Lincicome, 1943; Van Cleave, 1953a,b; Margolis, 1956; Golvan, 1959; Neiland, 1962; Dailey and Brownell, 1972; Margolis and Dailey, 1972; and others), while *Polymorphus* spp. normally parasitize birds.

The general acanthocephalan life cycle involves an invertebrate intermediate host (usually an arthropod), a vertebrate final host, and sometimes an invertebrate or vertebrate transport host. An egg (actually a shelled embryo), eaten by the arthropod, hatches into an acanthor larva, which actively penetrates the gut wall and becomes located in the haemocoel where it develops into the so-called acanthella stage and further into a juvenile known as a 'cystacanth'. When the invertebrate is ingested by a compatible final host, the cystacanth loses its protecting sheath and develops into an adult. If the infested arthropod is eaten by a host that is not conducive for development of the worm to sexual maturity, the cystacanth may reencyst in this animal, which thereby becomes a paratenic or transport host. Although a paratenic host is not normally essential for completion of the acanthocephalan's life cycle, it may serve as a link in the food chain and carry the immature parasite to a suitable definite host (Nybelin, 1924a,b; Cheng, 1973a; Noble and Noble, 1976). For general information on the biology, ecology and pathology of Acanthocephala consult Petrochenko (1956), Nicholas (1967, 1973) and Crompton (1970).

The life cycle of *Corynosoma* spp. parasitic as adults in marine mammals involves a crustacean (usually an amphipod) as regular intermediate host and various species of fish as paratenic hosts (Golvan, 1959). While the crustacean intermediate hosts are largely unknown, late-stage juveniles have been recovered from various species of fish (Schiller, 1954; Neiland, 1962). Accidental infestations with *Corynosoma* spp. have been reported from dogs, mink and even man. Immature worms showing arrested development may occur in fish-eating birds (Lyster, 1940; Dollfus, 1953; Van Cleave, 1953a,b; Schiller, 1954; Schmidt, 1971).

Corynosoma strumosum and *C. villosum,* first recorded from *Enhydra lutris* by Rausch (1953) and Van Cleave (1953a,b), respectively, are also common intestinal parasites of otariid, odobenid and phocid pinnipeds, indicating a common food source (Lyster, 1940; Rausch, 1953; Van Cleave, 1953a,b; Margolis, 1954, 1956; Neiland, 1962; Dailey and Brownell, 1972; Margolis and Dailey, 1972, Fay and co-authors, 1978). In *C. strumosum,* both sexes are not conspicuously different in size, and comparatively small, reaching from 5 to 7, occasionally 9 mm, in length. In *C. villosum,* the females (6.4 to 8.4 mm) are larger than the males (3.5 to 6.4 mm) (Van Cleave, 1953b). According to Golvan (1959), females of *C. villosum* may reach a length of 13 mm.

Immature individuals of *Corynosoma strumosum* have been recovered from 16 of 116

starry flounders *Platichthys stellatus*, as well as from 2 of 30 two-lined flounders *Lepidopsetta bilineata* from Departure Bay, British Columbia (Canada). As many as 120 cystacanths occurred in a single starry flounder (Ekbaum, 1938). Ward and Winter (1952) reported juvenile *Corynosoma* spp. from Californian yellowfin croaker *Umbrina roncador*. Meyer (1931) reported larval *C. strumosum* from sculpins *Cottus* (= *Myoxocephalus*) *quadriramis* and herring *Clupea harengus*, and Dollfus (1953) listed Atlantic cod *Gadus morhua* as paratenic host for this parasite.

In the Baltic Sea, amphipods *Pontoporeia affinis* have been identified as intermediate host of *Corynosoma strumosum* (Nuorteva, 1965, 1966, see below). *P. affinis*, a 'glacial relict' (Ekman, 1953), is restricted to the brackish waters of the Baltic and Caspian Seas. The abundant occurrence of adult *C. strumosum* in marine mammals from the Atlantic and Pacific Oceans indicates that other crustaceans, probably other species of amphipods, have taken up the position of the first intermediate host of this acanthocephalan in these waters. The intermediate and paratenic hosts of *C. villosum* are unknown. Schiller (1954) found most of the greenlings *Lebius superciliosus* and sculpins *Hemilepidotus hemilepidotus* from Constantine Harbor, Alaska, to harbour late-stage *Corynosoma* larvae which, however, were not identified to species. As early as 1933, Nybelin concluded that fish are not essential hosts in the life cycles of *Corynosoma* spp.

Morosov (1940) and Afanasiev (1941) described *Corynosoma enhydri* from sea otters of the Commander Islands. In the opinion of Van Cleave (in Rausch, 1953), the description of this species is inadequate. Van Cleave (1953b) recognized what appears to have been the same species in a sea otter from Simeonof Island off the southern coast of the Alaska Peninsula. Neiland (1962) restudied and named it *C. macrosomum*. In a mimeographed report, Jellison and Neiland (1965) recognized the latter as a synonym of *C. enhydri* Morosov, 1940. Hennessy and Morejohn (1977) reported this species from California sea otters. With a body length of 16.5 to 28.0 mm for females and 9.0 to 18.5 mm for males, *C. enhydri* is the largest of all mammalian species of *Corynosoma*. Its intermediate and paratenic hosts are unknown.

While *Corynosoma strumosum* and *C. villosum* have most frequently been found in pinnipeds, there is no published evidence of *C. enhydri* occurring in its adult stage in any mammalian host other than *Enhydra lutris*. Further — specifically unidentified — *Corynosoma* have been reported by Rausch (1953) from individuals of *E. lutris* collected on Amchitka, Aleutian Islands. They were believed to differ from both *C. strumosum* and *C. enhydri*. Rausch and Locker (1951) briefly mentioned another, yet unnamed *Corynosoma* sp. from Amchitka sea otters.

In addition to *Corynosoma enhydri*, Hennessy and Morejohn (1977) reported 3 acanthocephalans of the genus *Polymorphus* from California sea otters. The most common species was *P. kenti*, originally described by Van Cleave (1947) from the herring gull *Larus argentatus*, collected in the North Atlantic off New Brunswick, Canada. The (unexpected) occurrence of adult *P. kenti* in *Enhydra lutris* represents a new host record. Previously, Reish (1950) had reported adult *P. kenti* from gulls *L. occidentalis* and *L. glaucescens*, and acanthella larvae from sand crabs *Emerita analoga* collected in Oregon. Eighty-two of 86 mature crabs (= 95%) were infested with up to 17 (median: 3) larvae per host. Acanthellas fed to laboratory rats survived and developed into juvenile worms in these animals, which indicates lack of host specificity. Obviously, sea otters acquire *P. kenti* infestations by feeding on crab intermediate hosts. Hennessy and Morejohn (1977) found remains of

anomuran sand crabs, *Emerita* and *Blepharipoda*, among the contents of the gastrointestinal tract of *E. lutris*.

The 2 other *Polymorphus* species present in California sea otters were *P. major* and *P. altmani*, common parasites of diving ducks from the North American Pacific and European Atlantic and adjacent coasts (Lundström, 1942; Perry, 1942). The worms recovered by Hennessy and Morejohn (1977) from *Enhydra lutris* were all immature, which may indicate arrested development in these unnatural hosts.

Intensities of *Corynosoma* spp. infestation in *Enhydra lutris* may be high. Thus, Neiland (1962) recovered 60 specimens of the particularly large species *C. enhydri* from a single adult female; and the same author (in Kenyon, 1969) found a total of 271 *Corynosoma* sp. in the intestinal tracts of 8 adult individuals, chosen at random from 150 sea otters shot at Amchitka. In these, the numbers of acanthocephalans ranged from 1 to 98. Golvan (1959) recovered more than 100 juvenile and adult *C. villosum* from a single male *E. lutris*.

Extremely high acanthocephalan infestation incidences have been observed by Hennessy and Morejohn (1977) in sea otters recovered from the central California coast between Santa Cruz and Morro Bay. This may have been due to the fact that all otters examined were dead or moribund at the time of collection. Of 30 immature and adult female *E. lutris* examined, 29 (97%) were infested with *Corynosoma enhydri*, while 30 of 37 (81%) immature and adult male otters carried these parasites. In addition, 4 of 7 (57%) female pups and 1 of 6 (17%) male pups were infested.

Corynosoma enhydri infestation intensities, as observed by Hennessy and Morejohn (1977) during a 5-year period, exceed anything previously reported in the literature. As indicated above, these figures may be due to the fact that only dead or moribund sea otters were examined. The number of worms per host ranged from 0 to 4,864 in immature and adult females and from 0 to 4,972 in immature and adult males. The average *C. enhydri* load of the female otter (exclusive pups) was 638 worms per host, while that of the same age grouping of male otters was only 5. Heaviest infestation intensities were encountered from March to May (Fig. 4-11).

Polymorphus infestations were considerably lower in the sea otters examined by Hennessy and Morejohn (1977), which certainly indicates that *Enhydra lutris* is an accidental host for these bird acanthocephalans. *P. kenti* was the most common worm of this genus in both incidence and intensity of infestation, 4 sea otters — 2 males and 2 females — being infested with 3, 6, 8 and 206 worms per host. *P. major*, the next most common worm of this genus, occurred in 3 males with infestation intensities of 2, 2 and 6 worms per host. *P. altmani* was found in only 1 otter (6 specimens).

The heavy cystacanth burden encountered in some fish — for example, 120 larvae of *Corynosoma strumosum* recovered by Ekbaum (1938) from a single *Platichthys stellatus* in British Columbia waters — shows that *Enhydra lutris* may acquire sudden extremely heavy acanthocephalan infestations by devouring a single infested fish.

There is remarkably little information on the pathology of Acanthocephala in *Enhydra lutris*. On the basis of autopsies and histological studies, Rausch (1953, p. 601) concluded:

> "The two species of Corynosoma [*C. strumosum* and *Corynosoma* sp.] reported here are unimportant parasites insofar as the sea otter is concerned. It is conceivable that the presence of great numbers of *Corynosoma* spp. might

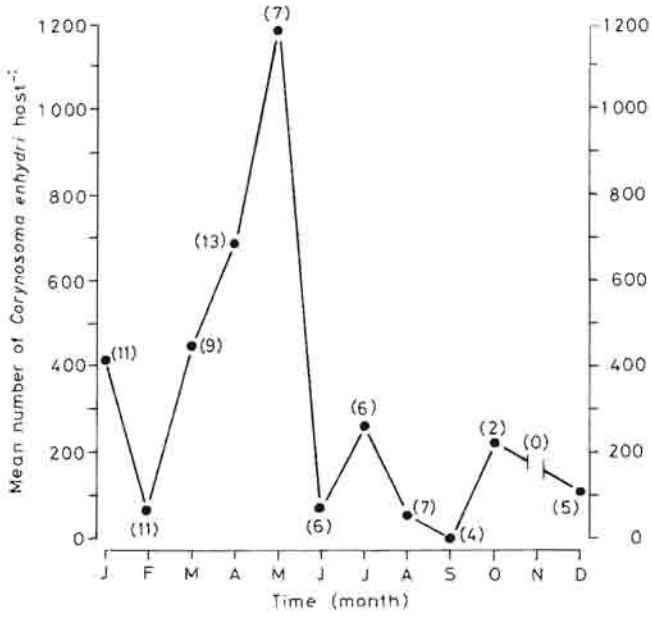

Fig. 4-11: *Enhydra lutris*. Monthly *Corynosoma enhydri* numbers from 42 male and 39 female sea otters summed over a 5-yr period. In brackets: number of hosts examined. (Compiled from data presented by Hennessy and Morejohn, 1977.)

result in enough inflammation and fibrosis of the host intestine to bring about a serious effect. Generally speaking, however, the closely localized tissue reaction caused by these Acanthocephala is inadequate in amount to affect the host, at least in any recognizable manner."

In view of the known — sometimes lethal — damage to the vertebrate host by acanthocephalans (Petrochenko, 1956; Nicholas, 1967, 1973; and others), this statement is more than surprising. In the definite host, the hooked proboscis of the worms (Fig. 4-12) usually penetrates deeply into the gut wall, producing traumatic injury, ulcers and necrosis. In some cases, the proboscis pierces the intestine to such an extent that it comes to lie beneath the serosa. Disruption of intestinal innervation as well as secretory and motor function may result.

Female acanthocephalans usually penetrate the intestinal wall more deeply than males. The histopathology produced can directly be related to the depth of proboscidal insertion (Chaicharn and Bullock, 1967). The mechanical damage done to the host intestine may be aggravated by secondary infections due to enteric bacteria. Moreover, the involvement of toxic secretions, as first suggested by Van Cleave (1919), cannot be ruled out entirely. Miller and Dunagan (1971) studied the morphology of the rostellar hooks of *Macracanthorhynchus hirudinaceus* (an acanthocephalan of swine) by means of scanning electron microscopy. The hooks were found to have a well-defined groove along the greater curvature, proximal to which lies a pore. The latter is continuous with gland-like tissue structures. The authors argued that the combination of these 3 elements — groove, tube-like opening, acid-staining pockets of tissue — is provocative and suggestive of a secretion delivered by means of the hooks. *M. hirudinaceus* produces a strong inflamma-

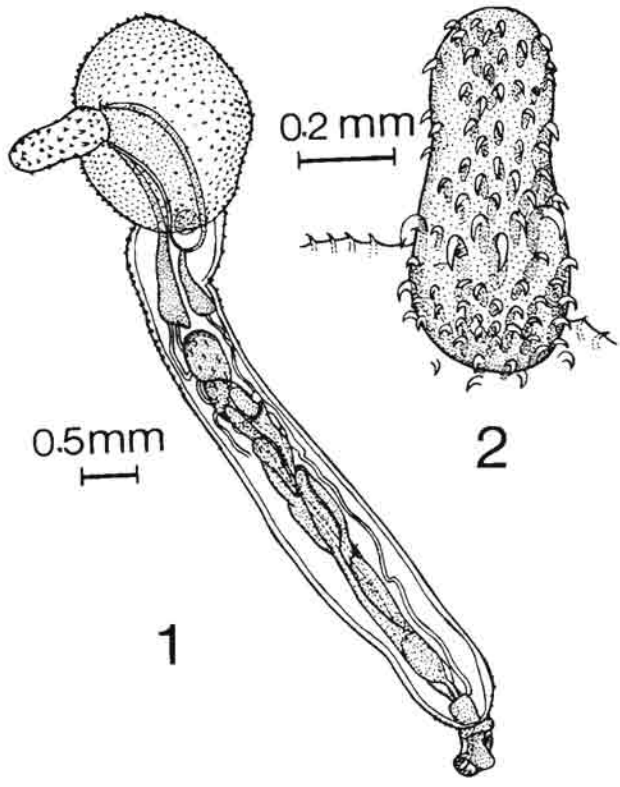

Fig. 4-12: *Corynosoma strumosum*. 1: male; 2: proboscis of same. (After Lyster, 1940.)

tory host response, which was found to be lacking in other acanthocephalans devoid of such a pore and groove.

Pathological damage caused by *Corynosoma strumosum* has been reported for pinnipeds (Mohr, 1952; and others). Nuorteva (1966) identified *C. strumosum* as a lethal parasite of farmed mink *Mustela vison* in Finland. During an epizootic, obviously initiated by the use of cystacanth-carrying food fish, hundreds of young mink died of bloody diarrhoea and consequent anaemia. Autopsy revealed the presence of *C. strumosum* in the hindgut of dead mink. Survivors also harboured *C. semerme* which, however, appeared to be less pathogenic to *M. vison* than *C. strumosum*.

Both acanthocephalans are common intestinal parasites of pinnipeds occurring in North Atlantic waters (Mohr, 1952; Sprehn, 1966a,b), and the principal host for both species in Finnish waters is the ringed seal *Pusa (Phoca) hispida*. Amphipods *Pontoporeia affinis* have been identified as intermediate hosts, and various species of marine as well as fresh- or brackish-water fish as potential paratenic hosts (Nuorteva, 1965). Sprehn (1956) also reported *Corynosoma strumosum* from farmed mink in Germany, discovered upon autopsy of succumbed hosts. There can be no doubt that *C. strumosum* is highly pathogenic to mink. Since *Mustela vison* and *Enhydra lutris* are closely related members of the family Mustelidae, the pathology of these acanthocephalans in sea otters must be expected to be similar to that produced in mink.

In the study of Hennessy and Morejohn (1977), *Corynosoma enhydri* was easily

removed from the intestinal mucosa, where it penetrated only slightly. Ulcerative lesions were observed in the intestinal walls of only a few otters. This condition appears to reflect a balanced parasite-host relation. It should be remembered (see above) that *C. enhydri* has not yet been reported from any host other than *Enhydra lutris,* the apparent 'true' definite host.

In contrast, the pathology produced by *Polymorphus* spp. may be more severe. Worms of this genus attach securely to the host's intestine with their large, bulbous proboscis deeply embedded in the intestinal wall. The one otter carrying 206 *P. kenti* (see above) had a necrotic ulceration, which had perforated the intestinal wall with many *P. kenti* attached. It was considered that this heavy infestation may have contributed to the eventual death of this sea otter (Hennessy and Morejohn, 1977).

Severe pathology and even lethal damage to the host have also been reported for Acanthocephala parasitizing fish (e.g., Wurmbach, 1937; Prakash and Adams, 1960; Bullock, 1961; Esch and Huffines, 1973) and birds (e.g., Clark and co-authors, 1958; Swennen and van den Broek, 1960; Rayski and Garden, 1961; Garden and co-authors, 1964). There can be no doubt that the Acanthocephala represent a group of highly pathogenic parasites.

Agents: Acarina

Enhydra lutris is remarkably free of arthropods and other ectoparasites, with the exception of probably fortuitous infestations by a nasal mite, *Halarachne miroungae* (Kenyon, 1969, 1972). Originally described by Ferris (1925) from the nasal passages of the northern elephant seal *Mirounga angustirostris,* this arthropod has subsequently also been reported as a common parasite of harbour seals *Phoca vitulina* (Doetschman, 1941; Newell, 1947).

Mites of the genus *Halarachne* are generally believed to be host-specific at the generic level and to be confined to the earless seals (Pinnipedia, Phocidae). Kenyon and co-authors (1965), however, found that at least 1 species of the genus is capable of infesting non-pinniped hosts in the wild. Of approximately 200 sea otters from Amchitka Island (Alaska), autopsied over a period of several years, 6 were found to harbour nasal mites. Infestations were light, with 1 to 7 parasites (females and larvae) per host, and the observed effects of the parasite were said to be always mild. Comparison of these specimens with *H. miroungae* from *Phoca vitulina* revealed no significant differences. The female of *H. miroungae* (Fig. 4-13), taken from the original host, measures 2.25 mm in length (Ferris, 1925).

Kenyon and co-authors (1965) assumed that cross-infestation on hauling-out areas used by both sea otters and seals probably accounts for the otters' infestation with these nasal mites. Their findings indicate that host specificity of halarachnids is not as narrow as generally thought.

In addition to the rare and light *Halarachne miroungae* infestations of *Enhydra lutris* in the field, Kenyon and co-authors (1965) observed an extremely heavy infestation in a single captive sea otter. Over 3,000 of these mites were recovered from a female that had died in a Seattle (Washington) zoo after having been kept in captivity for over 6 years. In this individual, the presence of the parasites was associated with severe disability and believed to be contributory to death. At autopsy, the nasal passages were found to be crowded with mites, and the mucosa showed severe reddening. Moreover, the turbinates

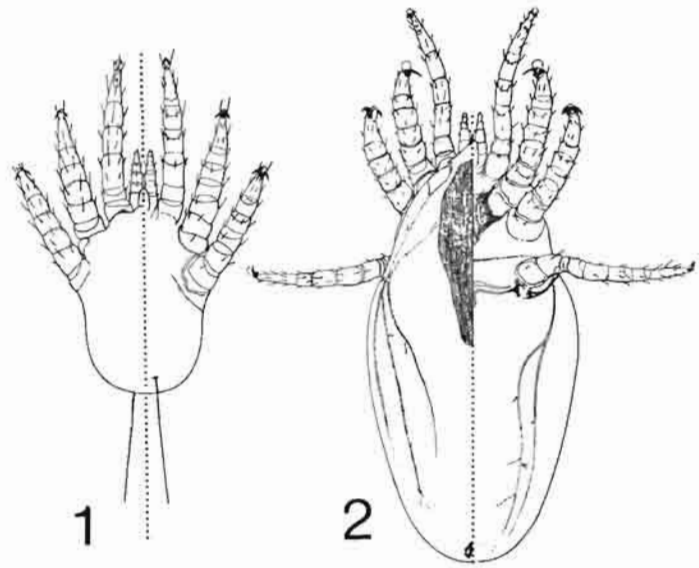

Fig. 4-13: *Halarachne miroungae*. 1: Female, dorsal (left half) and ventral (right half) aspect; 2: first-stage nymph, dorsal (left half) and ventral (right half) view. (After Ferris, 1925.)

had been destroyed, leaving a nearly unobstructed void from external nares to posterior nasal passages. Presumably, osteolysis had occurred because of severe mucosal irritation by the parasites. The immediate death of the female appeared to have been caused by lung congestion.

The source of the mites for this sea otter remained undiscovered. It was suspected that cross infestation from phocid seals kept in nearby, but separate, enclosures could have occurred. In any way does the massive affection show that *Halarachne miroungae* infestations in *Enhydra lutris* need not be inconsequential or transient (Kenyon and co-authors, 1965). With respect to the severity of the infestation it should be noted that the diseased sea otter had been held under abnormal conditions in a freshwater pool, and that the maintenance of *E. lutris* in captivity is always problematic (Kirkpatrick and co-authors, 1955; Kenyon, 1969). According to Furman and Dailey (1980), the sea-otter mites belong to *H. halichoeri*, not to *H. miroungae*.

NEOPLASIA

A young female sea otter, found dead on Amchitka Island, Alaska, revealed abnormal tissue growth in the viscera and a quantity of dark, apparently bloody, fluid in the body cavity. Throughout the small intestine, the bowel wall and the adjacent mesentery were irregularly thickened with firm, grey tissue which appeared to be neoplastic. The tumour-like tissue infiltrated and thickened the bowel wall in some areas and extended out into the mesentery, which was also thickened and grey. Toward the distal end, the mesentery contained a large coarsely nodular mass, 8 × 6 × 4 cm in dimension which, on section, was solid and grey. Small, solid nodular masses scattered through the mesentery were apparently other involved lymph nodes. Microscopic sections of enlarged lymph nodes

showed local areas of apparent neoplastic invasion, the tumour partially replacing the nodal substance. The condition was diagnosed as a leiomyoma or leiomyosarcoma (Kenyon, 1969). However, the presence of mitotic figures in the presumed tumorous tissue has not positively been reported, and the involvement of the lymph nodes in the manner described may be taken as reminiscent of a rickettsial infection similar to 'salmon poisoning disease' in canines (p. 648).

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5. DISEASES OF MAMMALIA: PINNIPEDIA

G. LAUCKNER

Among the 110 presently recognized species of marine mammals, the Pinnipedia represent the second largest group, following the toothed whales (Odontoceti). According to Rice and Scheffer (1968), the order Pinnipedia now contains 33 valid species — 12 in the family Otariidae ('eared seals'), 20 in the family Phocidae ('earless seals') and 1 (the walrus) in the monotypic family Odobenidae. Geographic races exist in several species. A number of synonyms (listed by Scheffer, 1958) and misidentifications sometimes impede the exact identification of the species dealt with, particularly in older reports. The generic and specific names adopted in this review are those considered valid by Rice and Scheffer (1968).

As an animal group of great commercial importance, the Pinnipedia have attracted the interest of numerous scientists. For general information on their biology, the reader is referred to Scheffer and Slipp (1944), Mohr (1952), Brooks (1954), Scheffer (1958), Pike (1961), King (1964), Harrison and King (1965), Nishiwaki (1972), Ridgway (1972a), Hewer (1974), Eley and Burns (1976) and Laws (1977).

Heavy persecution by fur hunters until the middle of the 20th century has brought a number of pinniped species close to extinction. The common designation 'fur seals' literally stands for the fate of these animals. Today, seal hunting is largely prohibited or restricted by federal and international regulations, but considerable numbers of Arctic seals continue to be harvested for their pelts. Seals may interfere with local fisheries, and in some areas population control has been proposed to protect commercial fish stocks (Anonymous, 1981; Harwood, 1984). In Norway, seal attacks on marine fish farms constitute a growing problem (Anonymous, 1984). The situation is aggravated by the fact that increased seal-predation pressure upon caged salmon leads to a rapid build-up of roundworm infestation in the fish, which act as intermediate hosts for these seal parasites (see section 'Agents: Nematoda'). Heavy parasitization reduces the market value of the salmon.

Since the diminishing of human persecution of pinnipeds, seal populations have become progressively more affected by pollution. Morbidity, reproductive failure and mortality due to man-made environmental contaminants represent a continuing and increasing problem for the Pinnipedia worldwide. As top carnivores, these mammals accumulate high levels of organochlorine residues and heavy metals in their tissues. The ecotoxicity of these substances and their effects on marine life are well documented. Both groups of pollutants have a strong immunosuppressive capacity. Alarming declines of the harbour seal population in the North Sea, particularly in the Dutch part of the Wadden Sea, have been traced to environmental pollution. The literature on this subject is voluminous. For detailed information and further references, the reader is referred to Holden and Marsden (1967), Wassermann and co-authors (1969), Le Boeuf and Bonnell (1971), De Long and co-authors (1973), Kim and co-authors (1974), Koeman and co-

authors (1975), Gilmartin and co-authors (1976), Helle and co-authors (1976a, b), Drescher and co-authors (1977), Drescher (1978b), Helle (1978), Olsson (1978), Møhlenberg and Jensen (1980) and Reijnders (1980, 1981).

During the past decades, and particularly with the advent of the oceanaria, there has been an increase in public interest and familiarity with seals, sea lions and walruses. There is also a developing interest in pinnipeds as research animals. This interest centers on the ability of these diving mammals to descend to depths exceeding 350 m and to perform a number of underwater tasks (Saunders and Hubbard, 1966; Hubbard and Poulter, 1968; Ridgway, 1972a, 1976; Kooyman and co-authors, 1980). Maintenance of pinnipeds in captivity entails a wide variety of medical problems. Husbandry and medical care of marine mammals have made considerable progress during the past decades though, and knowledge of their parasites and diseases is increasing steadily.

While the cetaceans most closely resemble cattle internally, seals and sea lions are more like the dog in anatomy and behaviour (Ridgway, 1965). In contrast to the Cetacea, the Pinnipedia spend considerable time out of the water and have adapted to both aquatic and terrestrial habitats. Beyond doubt, the pinniped ancestors have been terrestrial animals. The phylogenetic history of the group is also reflected by the fact that pinnipeds have a number of genera or even species of helminth parasites in common with terrestrial carnivores (Delyamure, 1955, 1959, 1966).

Among the earliest general accounts of disease in captive pinnipeds are those of Fox (1923) and Dobberstein (1936). Little was known, at that time, about diseases of feral populations. For general information on health problems of captive and feral pinnipeds, the reader is referred to Rigdon and Drager (1955), Brown (1962), Hubbard (1965, 1966, 1968b, 1969), Poulter and co-authors (1965), Ridgway (1965, 1972a, b), Keyes (1966), Wallach (1970, 1972), Palumbo and co-authors, 1971; Gray (1972), Sweeney (1973, 1974a, b, 1977, 1978a, b, c, d), Harrison (1974), Ridgway and co-authors (1975), Hammond and Elsner (1977), Fay and co-authors (1978), Geraci (1978a), Conklin (1979) and Howard (1983), to mention only a few. These publications deal with various aspects, i.e., non-biotic, nutritional, endocrine, metabolic and cardiovascular diseases, disease diagnosis and management, intoxications, drugs, anaesthesia, handling, husbandry, general medical care, blood values, anatomy, surgery, etc. — topics, the treatment of which is beyond the scope of this review, which centers on the biotic diseases and parasites of Pinnipedia.

DISEASES CAUSED BY MICROORGANISMS

Agents: Virales

Information on virus diseases of marine mammals in general is scanty; the field of marine-mammal virology is still in its relative infancy (Britt and Howard, 1983).

Established cell lines facilitating mammalian-virus research are not available from any marine mammal species, with the exception of a sea-lion skin-cell line established from a biopsy of focal dermatitis (Britt and Howard, 1983). Pioneering experiments have been conducted by Cecil and Nigrelli (1970). The authors grew cell cultures from mesentery, lung, heart and kidney tissue of a grey seal *Halichoerus grypus*. Human viruses from the picorna-, herpes-, adeno- and reovirus groups were shown to replicate in these cells. The

kidney culture appeared to have undergone cellular transformation in showing (i) loss of contact inhibition, (ii) altered morphology, (iii) increased growth rate and (iv) persistence in serial subcultures.

Not a single virus had been isolated from any marine mammal prior to 1968 (Watkins and co-authors, 1969; Smith and Skilling, 1979). More recently, a pox virus, different serotypes of caliciviruses and an adenovirus have been reported from pinnipeds.

Seal pox has for the first time been diagnosed by Wilson and co-authors (1969) in a captive 1-year-old female California sea lion. Subsequent surveys revealed the occurrence of the disease in free-living *Zalophus californianus,* in feral and captive *Phoca vitulina,* and in captive *Otaria byronia* (= *O. flavescens*) (Wilson 1970; Wilson and Poglayen-Neuwall, 1971; Wilson and co-authors, 1972a, b, c; Dunn and Spotte, 1974).

Seal-pox skin lesions appear as raised nodules, about 1.5 to 2 cm high and 5 to 20 mm in diameter, which are distributed over the entire body surface, mainly the head and neck (Fig. 5–1). Grossly, the lesions resemble those produced by *Dermatophilus congolensis,* the etiological agent of cutaneous streptothricosis (see section 'Agents: Bacteria'). Histologically, the pox lesions, which often suppurate, are characterized by parakeratosis, hyperkeratosis, hypertrophy and hyperplasia of the stratum spinosum (Fig. 5–2, a). The dermis is infiltrated by cords of epithelial cells, many of which contain one or more

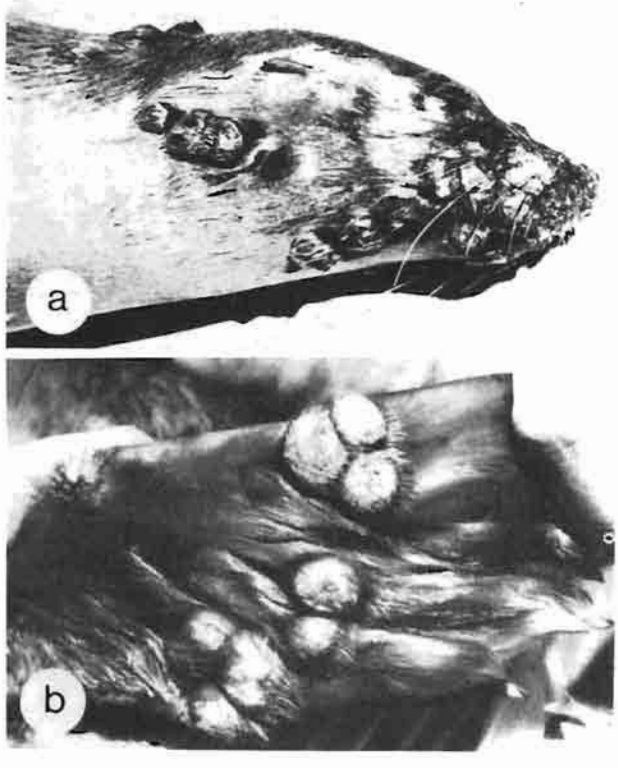


Fig. 5–1: Seal pox. (a) Distribution of pox lesions on head and neck of a 1-year-old female *Zalophus californianus;* (b) large pox nodules on flipper of an approximately 4-month-old *Phoca vitulina.* (a after Wilson and co-authors, 1969; b after Wilson and co-authors, 1972c.)

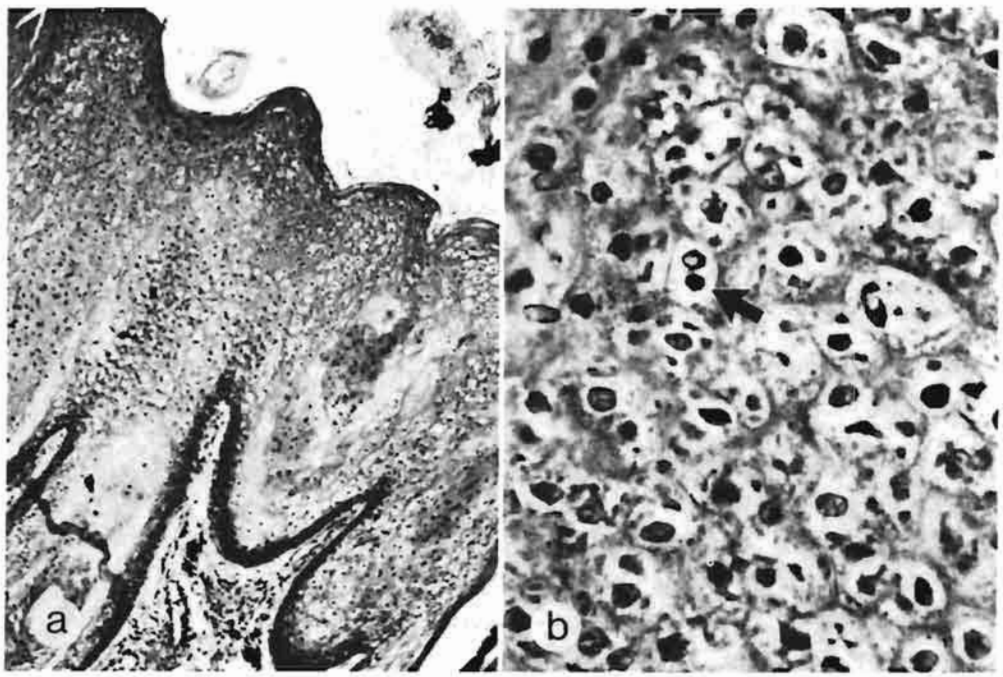


Fig. 5–2: Seal pox. (a) Section of a pox lesion in a 1-year-old female *Zalophus californianus* showing hypertrophy, hyperplasia and vacuolation of cells of the stratum spinosum; H & E stain, × 58. (b) Vacuolated, degenerating cells of the stratum spinosum containing numerous eosinophilic intracytoplasmic inclusions (arrow); H & E stain, × 600. (After Wilson and co-authors, 1969.)

eosinophilic intracytoplasmic inclusion bodies, 2 to 15 μm in diameter (Figs 5–2, b, and 5–3).

Ultrastructural examination of seal-pox material revealed the presence, in the lesions, of dumbbell-shaped pox virions. Employing negative stained preparations, mulberry (M) and capsular (C) forms could be distinguished. The M form, averaging 312 nm in length and 194 nm in width, had a beaded surface and a serrated outline. The C form, averaging 353 nm in length and 196 nm in width, also had a serrated periphery but was not beaded (Fig. 5–4).

The presence of such M and C forms in proliferative epithelial lesions of pinnipeds corresponds to reports of similar particles in other pox virus morphological studies. However, a precise classification of the seal poxvirus into one of the pox virus subgroups has not been possible. The orderly pattern of the surface filaments, as well as the size and shape, revealed a similarity of the M form to Orf virus (the etiological agent of contagious pustular dermatitis of sheep and Bovine Papular Stomatitis poxviruses, which are accomodated in the paravaccinia subgroup (Wilson and Sweeney, 1970).

Seal pox in *Otaria flavescens* differed markedly from all other cases examined. While the usual cutaneous lesions in *Zalophus californianus* and *Phoca vitulina* proliferate outward, the lesions in *O. flavescens* are distinctive downward proliferative masses. Also, the intracytoplasmic inclusion bodies are larger and occur 1 to a cell. The virions measure about 250 nm in length and 200 nm in width and are rectangular in shape. In their

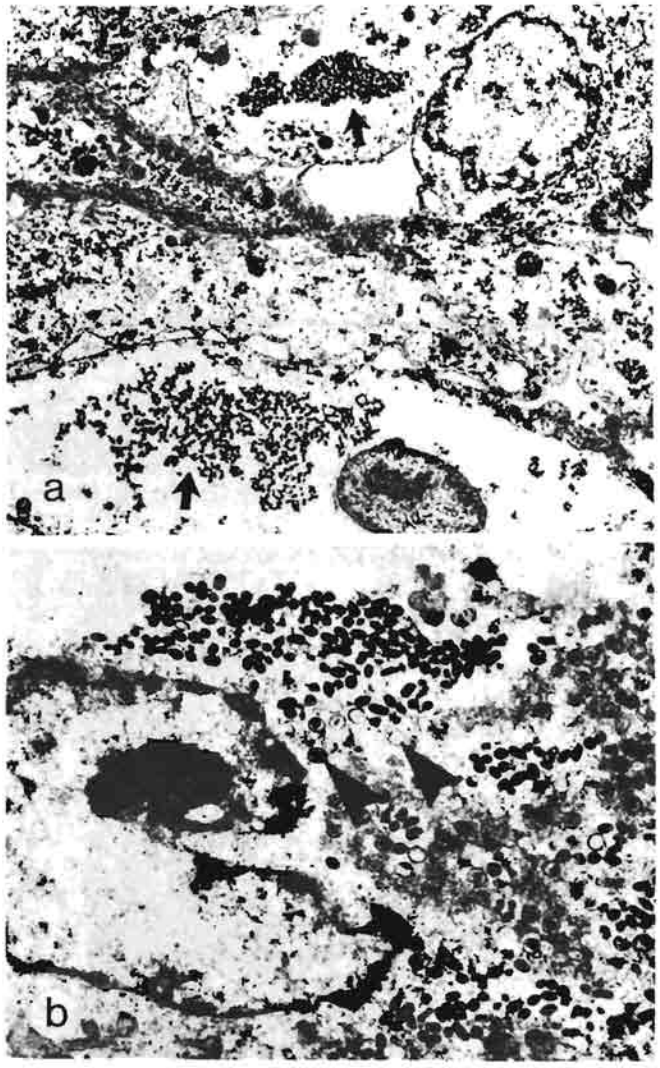


Fig. 5–3: Seal pox. (a) Electron micrograph of an infected cell of the stratum spinosum of a 1-year-old female *Zalophus californianus* showing nucleus and intracytoplasmic inclusion composed of dense, granular material and foci of mature virions (arrows); × 8,450. (b) Poxvirus-infected epithelial cell of a 4-month-old *Phoca vitulina* showing chromatin clumping, margination and irregular nuclear membrane. Large intractoplasmic inclusion body contains various viral developmental forms (arrows); × 12,000. (a after Wilson and co-authors, 1969; b after Wilson and co-authors, 1972c.)

appearance, the *O. flavescens* pox were similar to molluscum contagiosum, a poxvirus diseases of man (Wilson and Poglayen-Neuwall, 1971).

Poxvirus infections in man and lower animals can cause significant mortality. Uncomplicated cases in seals are not known to be fatal but may cause a high morbidity rate. Individuals with diffuse pox lesions have died, but the infections were not considered the cause of death. At the height of the clinical disease, which occurs about 4 weeks post

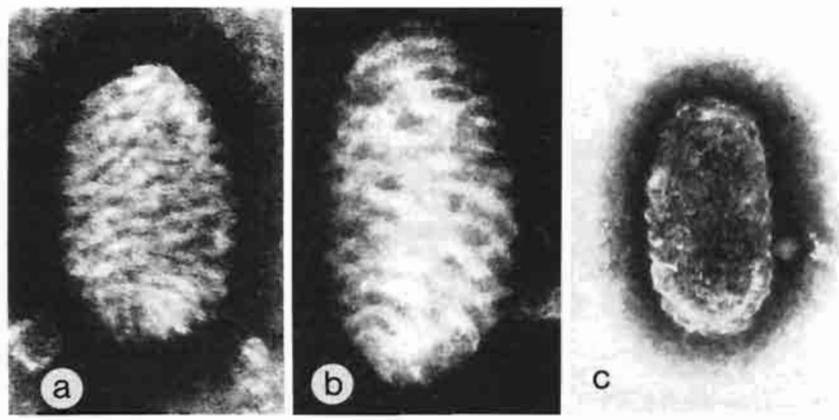


Fig. 5–4: Seal poxvirus. (a) 'M' form showing surface filaments and marginal scalloping; phosphotungstic acid stain, pH 4.3, × 130,000. (b) 'M' form illustrating orderly pattern of surface filaments; phosphotungstic acid, pH 7.0, × 173,000. (c) 'C' form showing capsule with serrated margins enclosing inner body; phosphotungstic acid, pH 4.3, × 91,000. (After Wilson and Sweeney, 1970.)

infection, the skin may be covered by several hundred nodules, involving a large proportion of the body surface. Dehiscence of most nodules may follow the clinical peak, resulting in generalized pustulosis. However, within about 15 weeks, seals usually recover from the disease. Healed lesions may remain partially alopecic. There is no known treatment except intensive nursing and elimination of any concurrent disease that may cause debilitation of the host (Wilson and Poglayen-Neuwall, 1971; Wilson and co-authors, 1972c; Dunn and Spotte, 1974; Ridgway and co-authors, 1975).

Seal-pox prevalence in feral pinnipeds may be higher than could generally be inferred from the examination of captive individuals. According to Wilson and co-authors (1972b), seals with solitary and multiple skin nodules are captured frequently. However, individuals with multiple lesions are often released rather than retained and shipped to exhibits. Results from a sealpox questionnaire survey, conducted by the above authors, indicated the frequent occurrence of similar skin lesions in captive Steller sea lions and elephant seals. The etiology of these skin conditions was not determined. However, the presence of inclusion bodies in hypertrophied epidermal cells of a spotted seal *Phoca largha* from the Bering Sea suggested the presence of a viral agent (Fay and co-authors, 1978). As seal pox can be highly contagious, infected new arrivals represent a potential source of infection for zoos and exhibits. Seal pox has not been reported outside North America but has been suspected in Britain (Greenwood and co-authors, 1974).

Several serotypes of caliciviruses have been isolated from vesicular lesions and aborted foetuses of California sea lions and northern fur seals, as well as from clinically normal and orphaned elephant seal pups. The first isolate was obtained in 1972 from an aborting California sea lion on San Miguel Island, California, and was consequently designated San Miguel Sea Lion Virus (SMSV) (Smith and co-authors, 1973). SMSV, a single-stranded RNA virus about 34 nm in diameter (Fig. 5–5), was found to be indistinguishable from VESV, a virus producing vesicular exanthema in swine. Both agents are members of the Picornaviridae. When injected intradermally into pigs, SMSV causes lesions identical to those produced by VESV. The structure as well as the biochemical and

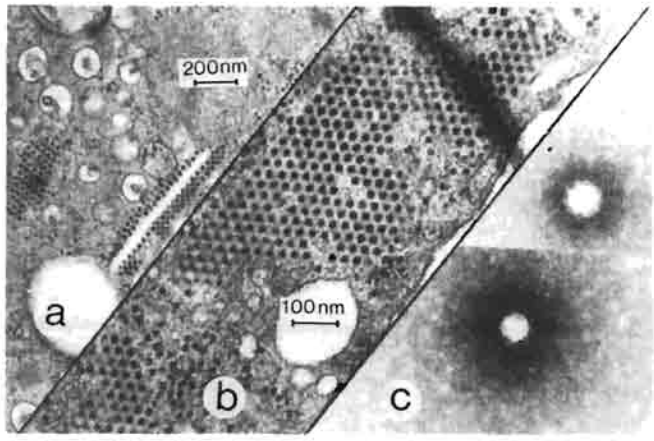


Fig. 5–5: San Miguel sea lion virus (SMSV). Electron micrographs of virus-infected Vero cells 12 h after inoculation. (a) Viral particles in tubular configuration similar to that seen with VESV Type H_{54}, × 28,400; (b) a crystalline lattice in cytoplasm, × 57,600; (c) negative stained preparations of SMSV virus showing typical calicivirus morphology. (After Smith and co-authors, 1973.)

biophysical properties of SMSV have been studied in detail by Schaffer and Soergel (1973), Madin and co-authors (1976), Smith and co-authors (1976, 1977a, 1979), Breese and Dardiri (1977) and Burroughs and co-authors (1978). Six serotypes — SMSV-1, -2, -4, -5, -6 and -7 — have been identified, 3 of which can be shared by both marine and terrestrial hosts (Table 5-1). At least the Types 4, 5 and 6 are believed to be antigenic variants of the vesicular exanthema of swine virus. It is not quite clear, however, whether

Table 5-1
Summary of test results for SMSV neutralizing antibodies in 11 mammalian species using 100 $TCID_{50}$ of virus (Compiled from Akers and co-authors, 1974; Prato and co-authors, 1974; Smith and co-authors, 1976, 1979; Madin and co-authors, 1976)

Species	Antigenic types of SMSV				
	SMSV-1	SMSV-2	SMSV-4	SMSV-5	SMSV-6
Grey whales	–	+	–	+	–
Whales — Sperm, Sei, Fin and White	–	–	–	–	NT
California sea lion	+	+	+	+	+
Northern fur seal	+	+	+	+	+
Northern elephant seal	–	+	NT*	NT	–
Bearded seal	–	–	–	–	NT
Harbour seal	–	–	–	–	NT
Steller sea lion	–	+	–	+	NT
Ringed seal	–	–	–	–	NT
Walrus	–	–	–	–	NT
Feral sheep	–	+	–	–	NT
Feral goat	–	–	–	–	NT
Feral swine	+	+	–	+	–

* NT not tested

this is true, or whether SMSV is in fact a virus of marine origin simply possessing properties very similar to VESV. Bankowski (1981) considers the pinniped and swine viroses to have the same etiology.

The close relationship between the marine- and terrestrial-mammal caliciviruses warrants special attention. From 1932 to 1934, repeated outbreaks of vesicular exanthema were observed in domestic swine in California. Losses attributed to the disease were related to abortion, reduced conception rate and baby pig death due to agalactia and weight loss. The infected animals had been fed raw garbage containing various sorts of food scraps. In 1952, the disease spread to all major swine-producing areas of the United States. Since 1956, when federal regulations prohibited the feeding of raw garbage to swine, there have been no reported outbreaks of VES in the USA. Natural VESV infections have not been reported from outside the USA, except once in Iceland where it appeared in swine fed raw garbage containing pork scraps obtained from a US military base (Sawyer, 1976). The garbage component carrying the virus has not been identified, but a marine origin of the agent was suspected.

In fact, Sawyer and co-authors (1978) isolated SMSV from an animal food product obtained from ground carcasses of northern fur seals harvested on St. Paul Island, Alaska. Prato and co-authors (1974) presented serological evidence for the transmission of SMSV Types 1 and 2 between California sea lions, northern fur seals and feral pigs from the Californian Channel Islands. The authors concluded that the finding of SMSV antibodies in these hosts may throw light on the successive outbreaks of VES in domestic swine in the early 1930's; and that the endemicity of SMSV in pinniped herds suggests that VES outbreaks in domestic swine originated from a marine source. Smith and co-authors (1976, 1979) and Smith and Latham (1978) furthermore speculated that there exists, for the above serotypes of caliciviruses, an ocean reservoir unique in some ways to California. The authors postulated that some of the fish eaten by marine mammals form large populations, but have a geographic range limited to southern California and may represent the primary reservoir of the virus. However, 137 sera from 19 species of fish common to the California coast proved negative for neutralizing antibodies to SMSV Serotypes 1, 2, 4 and 5 (Madin and co-authors, 1976; Smith and co-authors, 1976).

Eventually, however, a new serotype of SMSV — Type 7 — was detected in the opaleye *Girella nigricans,* a common prey of pinnipeds in southern California waters. When inoculated into pigs, the virus produced a condition identical to VES. Serotype SMSV-7 was also isolated from 4 elephant seals and from 1 sea-lion liver fluke *Zalophotrema hepaticum.* A second calicivirus isolated from naturally infected *G. nigricans* proved to be SMSV-6, and infections of this teleost species with serotype SMSV-5 were obtained experimentally (Smith and co-authors, 1978, 1980a, b, 1981; Smith, 1981). Smith and co-authors (1980a) also presented some evidence for the possible involvement of the sea-lion lungworm *Parafilaroides decorus* as a linkage between its piscine intermediate and mammalian final hosts in the cycle of SMSV-5. Although SMSV is capable of replication in *G. nigricans,* it apparently does not harm its fish host and can, therefore, be termed a 'minimal fish virus'. Perhaps the fish-virus relationship is one of long standing and the virulence of the agent for mammals one of recent encounter and hence of virulence (Wolf, 1984). Thus far, *G. nigricans* is the only fish host from which SMSV has been isolated. The range of the species extends from Point Conception (near Santa Barbara) southward along the California coast into Mexican waters. This places the opaleye within

the geographic location where marine calicivirus activity appears to be most intensive (Smith and co-authors, 1980b). The occurrence of a virus in both a piscine and mammalian host, as reported for SMSV, is not unique. Thus, Officer (1964) has demonstrated the ability of a fish (rainbow trout) cell line to support the growth of mammalian (Venezuelan and Eastern equine encephalitis, VEE and EEE) viruses.

Aside from causing pustular lesions in pinnipeds, SMSV has been suspected to provoke abortions in *Zalophus californianus*. Recurrent high incidences of premature parturition have been observed among California sea lions on San Miguel Island, and the first isolate of SMSV was made from a female aborting about 60 days before full term (Smith and co-authors, 1973). VESV, the terrestrial counterpart of SMSV, is known to cause abortion in swine. The premature parturient San Miguel sea lions, however, also had high incidences of *Leptospira pomona* infections (which may also cause abortion; see section 'Agents: Bacteria'), as well as tissue-p,p'-DDE levels up to 7.6 times higher, and PCB residue levels up to 4.4 times higher than full-term females (Gilmartin and co-authors, 1976). These data suggest an interrelationship of disease agents and environmental contaminants as the cause of premature parturition in *Z. californianus*.

Acute viral hepatitis was diagnosed in 5 *Zalophus californianus* stranded along the Los Angeles, California, coast. The sea lions were emaciated and dehydrated and showed clinical signs of pneumonia. Their livers were hyperaemic, and acute coagulation necrosis affected 20 to 80 % of each lobule, with minimal leukocytic infiltration. The hepatocyte cytoplasm in the necrotic areas appeared eosinophilic and homogeneous, with loss of

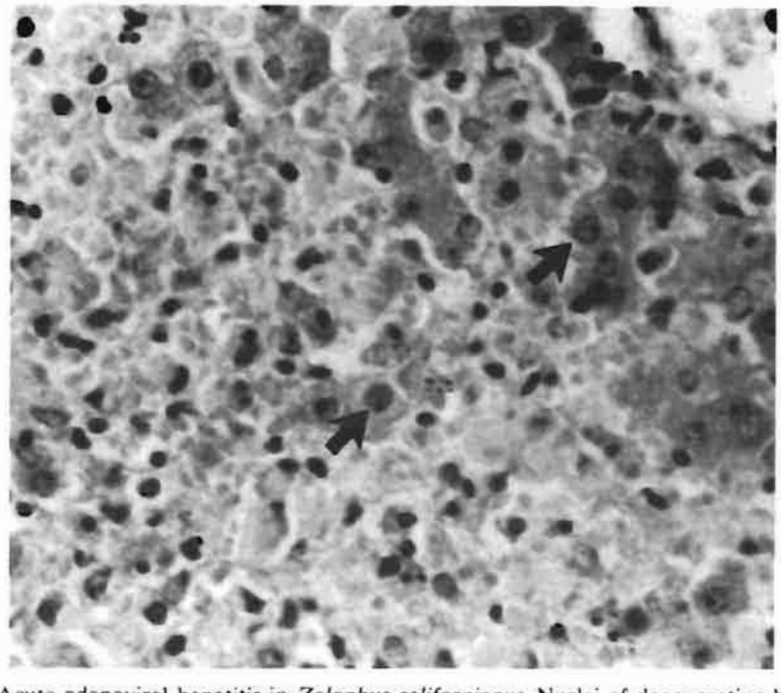


Fig. 5–6: Acute adenoviral hepatitis in *Zalophus californianus*. Nuclei of degenerating hepatocytes with homogeneous inclusions (arrows). Viable hepatocytes stain dark. H & E stain, × 360. (After Britt and co-authors, 1979.)

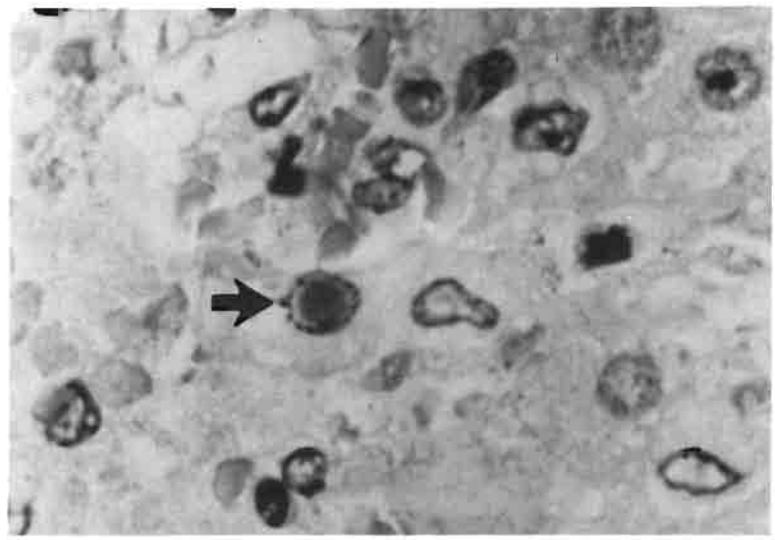


Fig. 5–7: Condensed inclusion body in nucleus of *Zalophus californianus* hepatocyte (arrow), with margination and clumping of chromatin. H & E stain, × 1,000. (After Britt and Howard, 1983.)

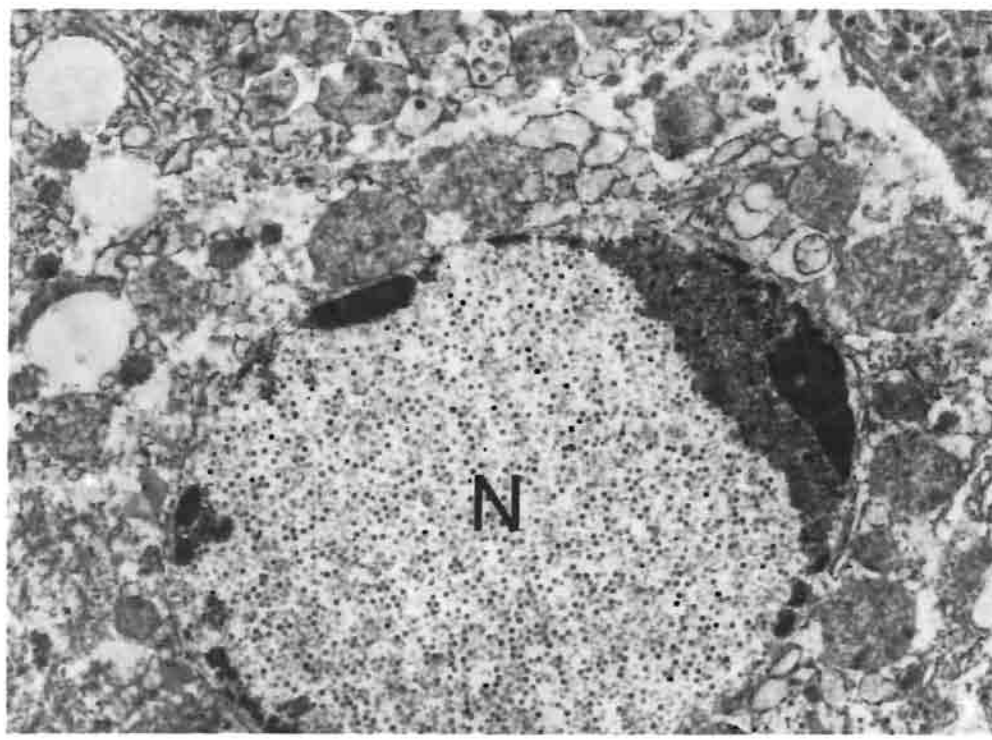


Fig. 5–8: *Zalophus californianus*. Ultrastructure of adenovirus-infected hepatocyte. Nucleus (N) contains numerous viral particles, chromatin has clumped around nuclear membrane, cytoplasm contains lipid droplets and swollen organellae. Uranyl acetate and alkaline bismuth subnitrate, × 9,200. (After Britt and co-authors, 1979.)

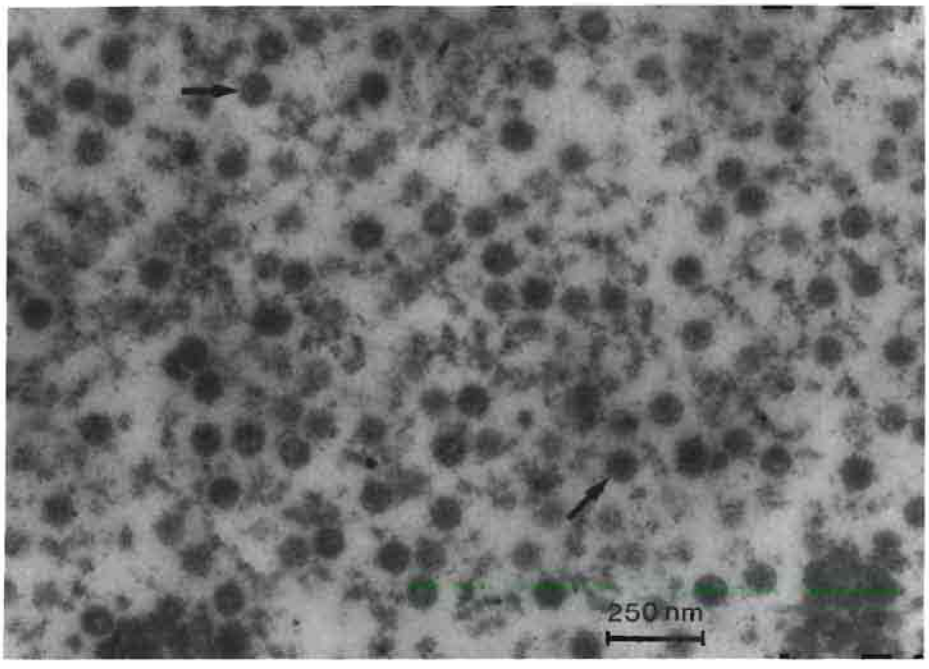


Fig. 5–9: *Zalophus californianus* adenoviruses at higher magification. (Courtesy Dr. J. O. Britt.)

normal architecture. There was early cytolysis in the center of the most severely affected necrotic areas, and karyorrhexis was occasionally seen. Light microscopy revealed large intranuclear inclusion bodies in hepatocytes, the nuclear membrane of which had undergone partial degeneration (Figs 5–6 and 5–7).

Ultrastructural examination revealed the nuclear inclusion bodies to be composed of masses of viral particles, 70 to 75 nm in diameter, with a distinct nucleocapsid core and a capsid coat but no envelope (Figs 5–8 and 5–9). Their size and morphological features were characteristic of the adenovirus group. The hepatitis in these sea lions resembled infectious canine hepatitis (ICH), the morphological features of both diseases being similar. This is the first viral disease of marine mammals shown to be lethal (Britt and co-authors, 1979; Britt and Howard, 1983). Attempts to isolate and adapt the virus to cell-culture systems were unsuccessful.

The source of the infection remained unknown. However, climatic conditions preceding the stranding of the sea lions included an excessive amount of rainfall in the area, which resulted in the dumping of untreated sewage into the offshore waters of the Los Angeles coast. Whether this factor played any role in the disease incident remains speculative (Britt and co-authors, 1979). Subsequent to this outbreak there have been only 2 additional cases of adenoviral hepatitis in captive southern Californian sea lions, and none in the last few years (J. O. Britt, pers. comm.).

Dierauf and co-authors (1981) reported a single case (seen in 1980) in a northern Californian sea lion, which had been in captivity for 28 days.

None of the hepatitic sea lions had exposure to canines, but some of the first 5 individuals examined by Britt and co-authors (1979) had overlapping periods fo confine-

ment to a care center, and Cases 6 und 7 were in capticity together 2 years later (Britt and Howard, 1983). Presumably, the virus is contagious to other sea lions but apparently not highly so, or perhaps the number of susceptible individuals in the population at any given time is low.

An influenza A virus, similar to fowl plague influenza virus, has recently been implicated as the causative agent of a mass mortality in a dense *Phoca vitulina* population on Cape Cod, Massachusetts. The harbour-seal disease had an acute to peracute course, with death often occurring within hours or days. Affected individuals appeared emaciated, and some discharged white or bloody froth from the airways. Subcutaneous emphysema in these seals resulted from air escaping from ruptured lung parenchyma. Microscopically, there was severe haemorrhagic alveolitis with necrotizing bronchitis and bronchiolitis (Geraci and co-authors, 1982).

The role played in the condition by the influenza virus was not quite clear. In an experimentally infected seal, it produced only mild disease. The feral individuals, however, had concurrent bacterial infections of different types in the lungs, a mycoplasma organism being the only consistent bacterial isolate. It may be necessary to have a synergism between another agent and the influenza virus to induce severe fatal disease. The natural course of this virus in the harbour seal population is not yet known. The disease was first observed during the seal-mass mortality in December 1979, tapered off as the animals dispersed during northward migration, and was not seen after October 1980. Retrospectively, the influenza virus was incriminated in pneumonia outbreaks in other seal populations. Antibodies to the agent have been found in grey seals *Halichoerus grypus* more than 500 miles north of Cape Cod. It has been postulated that the influenza A virus is transmitted by migratory sea birds (Geraci and co-authors, 1982; Britt and Howard, 1983).

In 1955, a mass dying of crabeater seals *Lobodon carcinophagus* occurred in a population wintering on the sea ice of the Crown Prince Gustav Channel, Antarctica. The disease, which was apparently quite contagious, spread rapidly through the population. Except for post-mortem changes, the condition of the corpses appeared reasonable, indicating previous good health. The stomachs were, however, empty, and intestines nearly so. The kidneys were somewhat enlarged, and two of the corpses examined had white flecks on the external surface of their kidneys and livers. One constant finding was acute congestion of the lungs. There were also areas of collapse, consolidation and emphysema. Spleens were engorged with blood and exhibited large amounts of haemosiderin, probably indicating extensive haemolysis. The kidneys showed evidence of acute nephritis.

Although observations were made shortly after the normal pupping season, no live pups were seen. Instead, numerous abortions were noted. The average mortality of *Lobodon carcinophagus* amounted to 85 %, but in some places, the figure was as high as 97 %. The disease, the effect of which may have been enhanced by abnormal crowding and partial starvation, was apparently specific to crabeaters and did not spread to intermingled Weddell seals *Leptonychotes weddelli*. Diseased seal meat was eaten by dogs and man with no ill effects. Although detailed and critical histological examination was not possible, owing to the decomposition of the material, it was concluded that the deaths were attributable to disease, probably a virus infection (Laws and Taylor, 1957).

However, as criticized by Ridgway and co-authors (1975), the term 'viral disease' is often misused to identify conditions of unknown origin. Some of these conditions may in

fact be due to viruses, but as such they are difficult to diagnose and probably become masked by secondary microbial or parasitic invasion. Mass mortalities among marine mammals may also have completely different causes. Thus, in the early 19th century, a mass dying of South African fur seals *Arctocephalus pusillus* occurred on Possession Island, Southwest Africa. The event had been witnessed by Captain Benjamin Morrell, an American southseaman, who visited the island in September 1828. An analysis of his report led Wyatt (1980) to hypothesize that a toxic dinoflagellate bloom was responsible for the catastrophe.

Agents: Bacteria

Bacteria have been implicated in a large number of pinniped diseases. They may cause primary infections of the respiratory tract, eyes, ears, teeth and digestive tract, produce fatal systemic infections, or appear as secondary invaders in superficial dermal lesions or deep wounds. As many of these agents have the potential of invading, and causing disease in, mammals other than pinnipeds, their possible hazard to zoo personnel and visitors should be taken into consideration.

As pinnipeds are notoriously 'dirty' animals, bacterial infections of the skin are common to them. Severe dermal and subdermal lesions may result from minor scratches or puncture wounds contracted during intrasexual fighting. Pups frequently receive severe bites from both adult males and females (Coulson and Hickling, 1964; Bonner, 1972). Bacterial infection of such deep wounds often leads to systemic infection and, eventually, death of the suscept. Organisms most frequently isolated from dermal wounds and abscesses include strains of *Staphylococcus, Streptococcus, Pseudomonas, Aeromonas, Proteus* and *Corynebacterium* (Brown, 1962; Eriksen, 1962; Larsen, 1962; Appleby, 1964; Ronald and co-authors, 1970; Wallach, 1972; Jedlička and Hojovcová, 1973; Sweeney, 1973; Anderson and co-authors, 1974; Brandes and co-authors, 1974; Greenwood and co-authors, 1974; Clausen, 1978; Drescher, 1978a; Reichel and Mayer, 1978; Stroud and Roffe, 1979; and others).

Of 65 common seals *Phoca vitulina* from Danish waters, 22 (34 %) had skin lesions ranging from circular wounds a few cm in diameter to ulcerations 30 × 50 cm in extension. In 2 cases the lesions were so severe that they were believed to have caused the death of the victims. The wounds yielded a streptococcus of unidentified serotype (Clausen, 1978; Heide-Jørgensen, 1979). Umbilical infections, sometimes resulting in purulent peritonitis and death are not uncommon to seals. Beta-haemolytic and other streptococci have been isolated from such lesions (Larsen, 1962; Appleby, 1964; Blessing and Eickhoff, 1967). Specifically unidentified β-haemolytic streptococci constitute a large proportion of the bacteria isolated from suppurative skin wounds in other pinnipeds (Simpson and Gardner, 1972; Brandes and co-authors, 1974).

Many of the skin lesions observed in seals appear to result, and spread, from infection foci in the severed umbilical cord or the umbilicus itself. Of 78 juvenile *Phoca vitulina* from the German North Sea coast, 56 had skin infections confined to the umbilical region (Fig. 5-10), 15 had additional wounds in other body regions, but in only 7 individuals with skin infections was the umbilical region unaffected. Developing ulcerations may attain a diameter of 20 cm within half a year (Drescher, 1978a).

Escherichia coli, Acinetobacter parapertussis, Alcaligenes faecalis, Micrococcus sp., *Staphylococcus* sp. and *Streptococcus* sp. have been isolated from subdermal abscesses,

Fig. 5–10: *Phoca vitulina.* Approximately 11-month-old individual with severe bacterial skin lesion (arrow) in umbilical region. (After Drescher, 1978a.)

E. coli, Citrobacter sp., *Corynebacterium pyogenes* and *Micrococcus* sp. from deep abscesses in sick, free-living California sea lions (Simpson and Gardner, 1972; Sweeney and Gilmartin, 1974). Van Pelt and Dieterich (1973) isolated a coagulase-positive *Staphylococcus aureus* from a young male harbour seal *Phoca vitulina richardi,* which had died from generalized subcutaneous abscessation followed by acute systemic infection. Similar staphylococcal granulomas have been reported from *Pagophilus groenlandicus* (Figs 5–11; Wilson and Long, 1970). The disease is commonly known as 'botryomycosis'.

Corynebacterium phocae has been found to produce haemorrhagic subcutaneous lesions comparable to swine erysipelas (see below) in *Pagophilus groenlandicus, Pusa hispida, Erignathus barbatus* and *Cystophora cristata* (Svenkerud and co-authors, 1951). In grey and common seals from the Dee estuary, England, the same organism caused skin erosions but no subcutaneous abscesses as described in the above species of seals. No evidence was found to connect the fairly high incidence (about 15 %) of dermal lesions with any environmental factor. It was thought that they develop as a result of the infection of minor wounds (Anderson and co-authors, 1974).

In 1970–71, an epizootic destroyed hundreds, or perhaps thousands, of Galapagos sea lions *Zalophus californianus wollebaeki.* Predominant features of the disease, which was speculated to be of possible bacterial etiology, were the formation of non-umbilicated multiple suppurative cutaneous nodules, debilitation and loss of motor power. Necropsy of

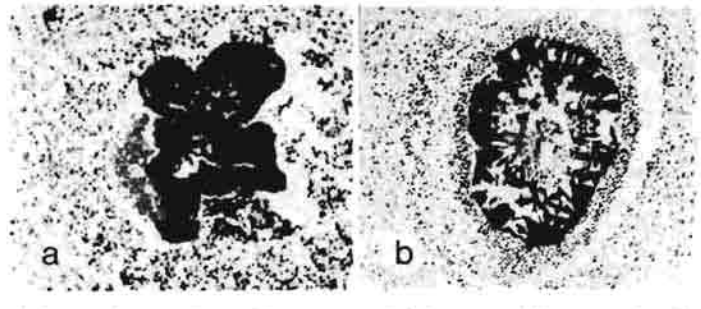


Fig. 5–11: Staphylococcal granuloma (botryomycosis) in *Pagophilus groenlandicus.* (a) Sulphur granule in center of a pulmonary granulomatous lesion; H & E stain, × 82. (b) Masses of Gram-positive cocci in center of an intestinal granulomatous lesion; Brown and Brenn, × 82. (After Wilson and Long, 1970.)

an individual in an advanced (pre-terminal) stage of disease disclosed suppurative cellulitis, with leukocytic invasion extending to all layers of the epidermis. Although several species of bacteria were isolated from the blood, *Pseudomonas aeruginosa* was the predominant organism. It was the sole agent present in nodular pus (Rand, 1975).

Bacterial eye infections are common to both captive and feral pinnipeds (Gallacher and Waters, 1964; Cansdale, 1970; Bonner, 1972). Ocular sepsis was the most frequent of all infections in young grey seals *Halichoerus grypus* at Farne Islands, England. Of 277 pups examined, 47 (17 %) had septic eyes (Ogilvie, 1957, 1959). Organisms isolated from affected eyes include *Staphylococcus albus, Pseudomonas aeruginosa,* haemolytic streptococci and *Neisseria* sp. (Appleby, 1964; Bonner, 1970, 1972; Beumer and co-authors, 1972). Simmons (in Bonner, 1972) isolated haemolytic and non-haemolytic staphylococci, α- and β-haemolytic streptococci, *Corynebacterium* sp. and *Neisseria* sp. from both infected and apparently non-infected eyes of Farne Island grey seal pups. Eye trouble is very common in captive pinnipeds and may, at least in part, result from the unnatural maintenance of these marine animals in freshwater (Ulmer, 1962; Ruempler, 1976).

Dental problems — mostly in the form of abscessed teeth — may occur in pinnipeds as well as in any other carnivore, with similar means of diagnosis and therapy. Of special concern, however, is the problem of tusk abrasion and infection in *Odobenus rosmarus.* Both wild and captive walruses frequently abrade their tusks to the gumline, thus exposing the pulpa. The resultant pulpitis is characterized by an acute swelling over one or the other tusk alveolus within the maxillary bone, as well as cellulitis, microabscessation and inflammatory cell infiltration. Necrosis of periosteum and superficial bone (osteomyelitis) has also been observed. Streptococci have often been identified as causative agents, but *Staphylococcus aureus, Clostridium* sp. and coliforms have also been isolated from tusk abscesses (Brown, 1962; Brown and Asper, 1965, 1966; Bartsch and Frueh, 1971; Sweeney, 1973; Brandes and co-authors, 1974; Ruempler, 1976). The disease usually runs a chronic course but, in its acute form, may exceptionally result in death within a few days of the onset of clinical signs. Stirling (1969) identified tooth wear with subsequent infection as a significant mortality factor in adult Weddell seals *Leptonychotes weddelli.*

Bacterial infections of the respiratory tract and lungs are a major health problem in both captive and feral pinnipeds, and constitute a source of significant mortality, particularly in wild and freshly caught individuals (Sweeney, 1973). According to Ridgway and co-authors (1975, p. 335), pneumonia is "a common killer of most marine mammals in captivity". Despite its frequent occurrence, there have been no studies on the exact course of events leading to fulminating clinical pneumonia in pinnipeds. Pulmonary diseases are commonly secondary to some other problem. They may appear as a secondary manifestation of some totally unrelated debilitating disease, or as a consequence of extensive lung or respiratory-tract damage resulting from mechanical manipulation (e.g., endotracheal intubation); or they may accompany destructive parasitic (lung worm) invasion. Bacteria of all descriptions have been implicated in pinniped pneumonia, and it would be unreasonable to list them by name (Ridgway and co-authors, 1975). Most of the known causative agents have been shown to be Gram-negative and can be controlled with broad-spectrum antibiotics. A sputum or tracheal culture with antibiotic sensitivity should be obtained and treatment directed accordingly (see below).

In diseased California sea lions stranded on southern California beaches, the predominant lesions were in the respiratory tract. Bacterial infections appeared to affect mainly

younger individuals. Thus, of 51 sea lions examined by Sweeney and Gilmartin (1974), 3 were over 2 years of age, 40 were between 15 and 20 months, and 8 were between 3 and 6 months old. In these groups, pneumonia-pneumonitis was diagnosed in 1, 33 and 5 cases, respectively. Organisms isolated from infected lungs included *Escherichia coli, Klebsiella pneumoniae, Serratia* sp., *Salmonella* sp., *Alkalescens dispar, Pseudomonas aeruginosa, Pseudomonas* sp., *Citrobacter* sp., *Acinetobacter woffi, Micrococcus* sp., *Staphylococcus aureus,* and *Staphylococcus* sp. In sea lions under 2 years of age, bacterial pneumonia is frequently, although not necessarily, associated with lungworm infestation (MacDonald and Gilchrist, 1969; Sweeney, 1973, 1978b; Kaufmann, 1974; Sweeney and Gilmartin, 1974). *Pasteurella multocida, Corynebacterium* spp., *Staphylococcus* spp., *Streptococcus* spp., *Salmonella typhimurium* and *Mycobacterium tuberculosus* have been isolated from other pinnipeds suffering from bronchopneumonia (Eriksen, 1962; Larsen, 1962; Wallach, 1972; Ridgway and co-authors, 1975).

Beta streptococci were associated with upper respiratory infections in captive California sea lions (Cordes and O'Hara, 1979) and *Escherichia coli* with bronchitis in captive common seals (Blessing and Rummeld, 1969).

Vylegzhanin (1963) reported on a mass mortality of Caspian seals *Pusa caspica* due to infection by *Diplococcus phocididae caspii.* In the winter of 1955–1956, about 30,000 seals — 80 % of the adult population — succumbed to the infection.

The clinical course of pneumonia may be acute or chronic. Symptoms include coughing, nasal discharge, rapid respiratory rate, lung sounds on auscultation, elevated rectal temperature and elevated WBC with an increase of immature granulocytes. Pleuritis or empyema may occur secondary to pneumonia. Treatment includes the administration of broad-spectrum antibiotics (tetracyclines, chloramphenicol, penicillin/streptomycin or sulfadimethoxide). However, by the time the disease becomes clinically obvious, it may have progressed to an irreversible stage (Eriksen, 1962; Larsen, 1962; Wallach, 1972; Ridgway and co-authors, 1975).

At St. Kilda Island, Scotland, bacterial pneumonia due to pneumococci has been identified as the major cause of morbidity and mortality of feral grey seals *Halichoerus grypus.* Dead seal pups, examined from a rookery at Mol Ghiasgar, all had patchy pneumonic consolidation of the lungs. Slides prepared from the sputum of diseased pups showed large numbers of pneumococci and small numbers of other bacteria. Bacterial pneumonia appeared to be a primary epizootic at that rookery. Grey seal mortality at St. Kilda was estimated to be higher than 50 %, and pneumonia was considered to be the major contributing cause. Diseased pups put in an outhouse and injected with 300,000 units of penicillin twice daily quickly recovered and could be returned to the sea after a few days (Gallacher and Waters, 1964).

Gastrointestinal infections are also common to pinnipeds. A considerable number of bacteria have been implicated as causative or accompanying agents of gastroenteritis in these animals. Salmonellae of different serotypes have repeatedly been isolated from the stools of feral pinnipeds. Blood and viscera of 5 of 12 sick *Callorhinus ursinus* pups, collected on the Pribilof Islands, yielded *Salmonella enteritidis.* Bacillary dysentery (salmonellosis) was considered to contribute significantly to the excessive fur-seal pup mortality observed on the islands (Jellison and Milner, 1958; King, 1964). Similarly, paratyphoid infection (= salmonellosis) was believed to be the major cause of death of fur-seal pups on Beringa Island, USSR (Melikhov and co-authors, 1961). *S. newport* was

isolated from the liver but not from the intestine of a New Zealand fur seal *Arctocephalus forsteri,* which had died from severe enteritis (Cordes and O'Hara, 1979). Schroeder and co-authors (1973) recorded *S. typhimurium, S. enteritidis* and *S. newport* infections in California sea lions stranded on the beaches of Los Angeles County, California.

An increasing incidence of isolation of *Salmonella* spp. from sick sea lions and fur seals stranded on the beaches of southern California led Gilmartin and co-authors (1979) to screen feral individuals from San Miguel Island for the prevalence of these organisms. Rectal swabs were collected from 90 *Callorhinus ursinus* and 50 *Zalophus californianus* pups. Three serotypes — *S. newport, S. heidelberg* and *S. oranienburg* — were recovered from 33 % of the fur seals and 40 % of the sea lions (Table 5-2). The authors concluded

Table 5-2
Salmonella spp. Frequency of isolation of 3 serotypes from 90 northern fur-seal and 50 California sea-lion pups (After Gilmartin and co-authors, 1979)

Salmonella serotypes	California sea lions	Northern fur seals
newport	16	22
heidelberg	–	4
oranienburg	3	–
newport and *oranienburg*	1	2
oranienburg and *heidelberg*	–	1
newport and *heidelberg*	–	1

that the high prevalence of these potential pathogens in a pinniped population is not surprising, as the rookeries are heavily contaminated with faecal material. Western gulls *Larus occidentalis,* also present in the rookeries, may be responsible to some extent for spreading the salmonellae about the beaches, since gulls have repeatedly been implicated as carriers (Berg and Anderson, 1972; Wuthe, 1972; Williams and co-authors, 1976). Gilmartin and co-authors (1979) furthermore concluded that mortality due directly to *Salmonella* in the young pups examined by them does not appear to be a problem. It may, though, have significance as a disease problem in pups or older individuals, which have been debilitated for other reasons. However, it could not be excluded that salmonellosis might be involved in the high rate of abortion observed in the San Miguel Island sea - lion population.

Escherichia coli was isolated in pure culture from the faeces of a New Zealand fur seal *Arctocephalus forsteri* exhibiting light diarrhoea (Cordes and O'Hara, 1979). Bacteria cultured from small intestines of northern fur seals *Callorhinus ursinus* from Pribilof Island, Alaska, were *E. coli, Proteus mirabilis* and *Streptococcus faecalis.* In these animals, the morbidity of gastrointestinal infection (44.0 %) was much higher than the mortality from that cause (4.6 %). Necrotic enteritis was the most common form, making up half of the cases, followed by catarrhal enteritis and haemorrhagic enterocolitis (Keyes, 1965). A haemolytic strain of *E. coli* was isolated from the small intestine of a captive northern fur seal, which died from acute enterotoxaemia (Van Pelt and Ohata, 1974).

Edwardsiella tarda is another member of the Enterobacteriaceae pathogenic to pinnipeds. It appears to be an opportunistic invader of sick or injured individuals. Thus, it

has been isolated from the mesenteric lymph nodes of a captive *Zalophus californianus*, which died from streptococcal pneumonia (Wallace and co-authors, 1966). *E. tarda* has been isolated from peritoneal exudate of an individual of *Eumetopias jubatus* found in a weakened condition on rocks near Florence, Oregon, as well as from the liver of a *Zalophus californianus*, which had become stranded and died at Agate Beach near Newport, Oregon. The Steller sea lion was suffering from fibrinopurulent peritonitis resulting from a perforated ulcer in the posterior colon. The California sea lion died from fibrinopurulent peritonitis and abscessation of the lumbar lymph nodes (Coles and co-authors, 1978). *E. tarda* infections may be contracted by contact with gull faeces, from which the agent has been cultivated (Berg and Anderson, 1972).

Botulism, a neuroparalytic condition caused by toxins associated with the growth of *Clostridium botulinum*, is a common disease of free-ranging animals, particularly water fowl. It may also be the cause of a number of 'unexplained' deaths in wild and captive pinnipeds. *C. botulinum*, an anaerobic, motile, Gram-positive spore-forming rod, exists in the form of 8 serotypes, each of which elaborates an immunologically distinct neurotoxin. Botulinum toxin is the most potent toxin known. Botulism is ordinarily not an infectious disease but rather an intoxication.

Clinical signs of botulism are generally those of a symmetrical bulbar paralysis. An initial period of listlessness is followed by staggering movements, extension of the head when lying down ('limberneck') and gradually increasing paralysis. Gastrointestinal signs are variable. Dysphagia and pharyngeal paralysis are common. Death usually results from paralysis of the respiratory muscles and may occur within 24 h, or as late as 7 or 8 days following intoxication. Characteristically, post-mortem lesions are absent or minimal (Donadio and co-authors, 1971).

A series of — initially unexplained — deaths occurred in 1968 and 1969 among the California sea lions of the Kansas City Zoo. The diseased animals stopped feeding several days prior to death. Their attitude towards food suggested that they were hungry but unable to swallow. In all of the 7 cases, death was very sudden. *Clostridium botulinum* was confirmed as the causative agent in the final 2 cases. The bacterium appeared to be endemic at the zoo, as cases of 'limberneck' in the duck population there were not uncommon. It was concluded that anaerobic conditions possibly existing at the bottom of the sea - lion pool during summer might have favoured the reported outbreaks of botulism (Wagner and Mann, 1978). The authors point out that sea lions with dysphagia, followed by 'sudden death', should be considered botulism suspects, particularly if necropsy examination reveals no immediate cause of death.

Another species of *Clostridium*, *C. perfringens*, has been identified as the causative agent of acute, severe and sometimes fatal myositis in *Zalophus californianus*. The disease is characterized by sudden unilateral swelling of the neck region. The lesions may extend from the ramus of the mandible to the shoulder, and the entire affected area may become pruritic. An intense leukocytic response is typical (Sweeney, 1978a). Pinnipeds have notoriously dirty fur and skins, and despite thorough disinfection prior to injections, abscess formation at the sites of needle penetration is quite common. Greenwood and Taylor (1978) reported on a California sea lion, which developed slight skin swellings after having been injected twice in the gluteal muscles with a multivitamin preparation. During an attempt to obtain a blood sample from the deep gluteal vein, offensive-smelling bloody pus was aspirated. The sea lion died the next day and the autopsy revealed an area of

myonecrosis, extending deep into the muscle mass and accompanied by the development of gas and pus. *C. perfringens* Type A was isolated from the lesion. There were also toxic changes in the visceral organs.

The conditions required for the growth of *Clostridium perfringens* are typically devitalized tissue, anaerobic conditions with poor blood supply and high glucose concentrations. It appears that diving mammals provide ideal tissue conditions in their muscles for this type of infection by virtue of their high circulating glucose levels and anaerobic metabolism during breathholding. Clostridial infections therefore present a considerable risk to captive marine mammals. Excision, drainage and antibiotic therapy of clostridial muscle lesions are sometimes effective. Immunization, however, should be routine, and this should include protection against *C. perfringens* toxins (Greenwood and Taylor, 1978; Sweeney, 1978b).

There are other reports on *Clostridium perfringens* infections in pinnipeds. McBee (1960) isolated the organism from the large intestine of a Weddell seal *Leptonychotes weddelli* from the Ross Sea area of Antarctica, and Keyes (1965) found it in heart blood of northern fur seals *Callorhinus ursinus* on the Pribilof Islands suffering from gastrointestinal infection. *C. perfringens* was present in moderate counts in the intestine of a captive walrus *Odobenus rosmarus* that had died from chronic pulpitis (Brandes and co-authors, 1974). A case of *C. perfringens*-associated peritonitis and septicaemia in a harbour seal, following rupture of the pancreatic duct, has been described by Grafton (1967). Necrotic haemorrhagic enteritis was the cause of death in a young, recently captured northern fur seal from the Pribilof Islands. *C. perfringens* was isolated in pure culture from a loop of affected intestine (Keyes, 1963). Abscesses observed by Poulter and co-authors (1965) in young Steller sea lions are highly suggestive of *C. perfringens* involvement. Hubbard (1968a, b) detected the agent in abscesses in *Eumetopias jubatus* pups on Año Nuevo Island. The chronic, relatively asymptomatic nature of the affections implies that free-living sea lions have good natural immunity to clostridial infection. However, it is generally assumed that this high degree of natural immunity is broken down when pups, already stressed by being orphaned, are further stressed by imperfect artificial diets (Poulter and co-authors, 1965; Hubbard and Poulter, 1968).

A resulting condition, clostridial enterotoxaemia, has been observed only in Steller sea lion *(Eumetopias jubatus)* and northern fur seal *(Callorhinus ursinus)* pups fed improper milk substitutes. The disease (which actually represents an intoxication) may manifest itself in a peracute and a subacute form. In its peracute form it rapidly leads to severe 'otariid ataxia' (see below) associated with clonic seizures. The chronic form is characterized by a more gradual onset of weakness, depression and physical collapse within 48 h. Diagnosis must rely upon recognition of the clinical symptoms and the detection of *Clostridium perfringens* in Gram-stained faecal smears. The clinical signs may be misleading because 'otariid ataxia' (Hubbard, 1968a, b) describes a set of neurologically similar symptoms having different etiologies. Thiamine deficiency, hypoglycaemia and enterotoxaemia may all produce the signs. The first 2 conditions can be prevented by adequate nutrition and can be cured by thiamine and glucose injections. The latter condition readily responds to *C. perfringens* antitoxin injection. Once otariid ataxia is noted, all 3 therapies should be initiated immediately. Differential diagnosis is obtained from faecal examination.

The earliest manifestation of otariid ataxia is an abnormally high carriage of the rear

toes, the animals leaving the impression that the tips of the flippers are sensitive to touching the ground. This is followed in 1 to 3 days by loss of equilibrium, 'running staggers' and violent falling. Later there is extreme weakness and inability to rise. If the cause is clostridial enterotoxaemia, response to therapy with multivalent *Clostridium perfringens* antitoxin will be dramatic, with improvement coming in a matter of hours. The disease is nearly 100 % fatal if not treated, and therapy is nearly 100 % effective if applied early (Hubbard, 1966, 1968a, b; Wallach, 1972; Sweeney, 1973).

Pasteurellosis, manifesting itself in the form of acute haemorrhagic septicaemia or necrotic peritonitis, is another cause of significant mortality in pinnipeds. Most of the reported cases appear to be attributable to *Pasteurella multocida*. This small, non-motile, Gram-negative coccobacillus affects a large variety of birds and mammals. It has been described by Pasteur as the cause of fowl cholera. The disease is transmissible to man.

Almost all described cases of pasteurellosis in pinnipeds are from captive seals and sea lions, mostly from individuals held in European and North American zoos (Eriksen, 1962; Larsen, 1962; Wolinski and Landowski, 1962; Keyes and co-authors, 1968; Bonner, 1970). Outbreaks of the disease have been associated with stress, starvation or improper husbandry.

Pasteurellosis is characterized by a sudden onset, anorexia, elevated rectal temperature, dyspnoea and elevated WBC. In acute cases, progress of the disease is rapid and death of the suscept may occur within 3 to 4 days. Although the agent is sensitive to several antibiotics (chloramphenicol, oxytetracyclines), there is usually little time to initiate an effective therapeutic regimen. In the more chronic forms of the disease, seizures and respiratory distress, associated with pyothorax or peritonitis, have been reported. In the latter case, drainage of accumulated exudate is necessary. Prophylactic administration of *Pasteurella* bacterin may protect freshly caught pinnipeds (Wolinski and Landowski, 1962; Wallach, 1972; Sweeney, 1978a).

Staphylococcal septicaemia was the cause of death in a captive leopard seal *Hydrurga leptonyx* (Cordes and O'Hara, 1979). Bonner (1970) recorded septicaemia associated with *Pasteurella multocida, P. haemolytica (= Vibrio parahaemolyticus)* and *Escherichia coli* in starved feral Cornish seal pups. The author concluded that these organisms were probably normally present as non-pathogens, and became virulent when the animals' resistance decreased as a result of starvation.

A pure culture of *Staphylococcus aureus* was isolated from peritoneal exudate, and both *S. aureus* and *Edwardsiella tarda* were obtained from the liver of a California sea lion which had succumbed to fibrinopurulent peritonitis and abscessation of the lumbar lymph nodes (Coles and co-authors, 1978).

On rare occasions, *Erysipelothrix* infections have been reported from pinnipeds. The Gram-positive, nonsporulating, microaerophilic bacillus is the causative agent of a serious economic disease of domestic animals (pig, sheep, turkey, duck) known as swine erysipelas, erysipeloid or erysipeloid of Rosenbach (Wellmann, 1957). The bacterium has also been isolated from marine crabs, fish and cetaceans. In its localized cutaneous form, erysipeloid is characterized by spreading erythematous skin eruptions. Less commonly, the disease may assume a severe generalized cutaneous form or a systemic (septaemic) form often followed by endocarditis. *Erysipelothrix* is of terrestrial origin; it has been described under a variety of specific names — most frequently *rhusiopathiae, insidiosa* and *muriseptica*. On the basis of a study of the cultural characteristics of 50 strains of *Erysipelothrix,*

Langford and Hansen (1954) concluded that they are biochemically unseparable, and that the valid name of the agent is *E. insidiosa*.

Erysipelothrix insidiosa can cause fatal septicaemia in cetaceans (Seibold and Neal, 1956; Geraci and co-authors, 1966). Benkovsky and Golovina (1971) isolated the agent from various organs of 2 dead 5- to 6-month-old *Callorhinus ursinus* pups stranded at Sakhalin Island, Sea of Okhotsk (USSR). Marine mammals probably contract systemic infections by ingestion of contaminated fish (Niewiarowski, 1952; Wellmann, 1957; Geraci and co-authors, 1966). The experimental transmission of erysipelas to houseflies and bloodsucking arthropods (Wellmann, 1955) suggests another possible route of infection for captive marine mammals. *E. insidiosa* can also affect humans; it constitutes an occupational hazard of fish handlers, veterinarians, etc. (Sheard and Dicks, 1949; Sneath and co-authors, 1951). Infections contracted by contact with pinniped carcasses are known as 'seal fingers'. Similar 'whale fingers' occur among whalers (King, 1964). In its cutaneous form, erysipeloid (in humans) is a self-limiting condition. Penicillin treatment is relatively effective (Price and Bennett, 1951).

In captive marine mammals, control of the disease has been achieved by vaccination with either killed or modified live vaccines. Both types are available commercially for swine vaccination. The modified live vaccine offers better protection for a longer time than does the bacterin (Gilmartin and co-authors, 1971). However, because of the 'inexperience' of the immune system of newly captured individuals, 1st-time vaccination should be with the bacterin, followed in 6 months by application of the modified live vaccine. The procedure is not without attendant problems. Thus, dolphins have died from acute anaphylaxis on revaccination. Epinephrine and corticosteroids should be readily available if such a reaction should occur (Medway, 1980).

Leptospirosis is a zoonosis of worldwide occurrence, rodents and domestic animals providing the principal reservoirs (Schröder, 1975). The causative agents, *Leptospira* spp., belong to the Spirochaetes, Treponemataceae — motile organisms which have a morphology quite different from that of other bacteria in being extremely long, flexible, filamentous cells of characteristic spiral, or coiled-spring, shape (Hardy, 1973). Several cases of — sometimes fatal — leptospirosis have been reported from pinnipeds. All strains isolated from these marine animals appear to be serologically most similar to, or identical with, *L. pomona*, a leptospire of swine (Northway, 1972; Medway, 1980). In terrestrial animals, *Leptospira pomona* is transmitted by ticks (Arthur, 1962).

Vedros and co-authors (1971) observed an outbreak of leptospirosis among young male *Zalophus californianus* along the northern California coast. Clinically, the diseased sea lions were depressed and exhibited varying degrees of posterior limb paresis. Other symptoms were anorexia, icterus, erosion and haemorrhages of the oral cavity, elevated rectal temperature and convulsions in stressed individuals. In acute infections, WBC counts were elevated and accompanied by an increase in immature granulocytes. In the terminal stage, however, counts dropped below 3,000. About 10 % of 700 sea lions examined were affected. Schroeder and co-authors (1973) diagnosed leptospirosis in 12 of 64 *Z. californianus* stranded on the beaches of Los Angeles County between June 1970 and May 1973.

Leptospira pomona has been isolated from kidneys and urine of clinically sick individuals. Detection of the agent requires dark-field observation or staining with special silver-salt stains. Grossly, the affected kidney is slightly swollen and hard with light brown

cortices. Histologically there is interstitial nephritis with an infiltration of the cortico-medullar junction by lymphocytic and plasmacytic mononuclears. Serologically, titers to *L. pomona* of 1 : 10,000 have been demonstrated (McIlhattan and co-authors, 1971; Vedros and co-authors, 1971; Sweeney, 1978a). According to Sweeney (1978b), leptospirosis is endemic on California sea-lion rookeries; it is the most prevalent urinary tract disease of *Zalophus californianus.*

Smith and co-authors (1977b) detected *Leptospira pomona* in kidney sections of a 13-year-old northern fur seal *(Callorhinus ursinus)* bull with chronic interstitial nephritis. The animal exhibited an antibody titer of 1 : 640 and was shedding *L. pomona* in the urine. Serums, collected from 1,059 adult and juvenile northern fur seals on the St. Paul Island (Bering Sea) rookeries, were screened for leptospiral antibody. These tests revealed a herd prevalence of leptospirosis ranging from 7 to 15.4 % for adult females and 3- to 4-year-old bachelor bulls, whereas nursing pups averaging 4 months of age had a prevalence of 2 %.

Three newborn pups displayed multiple haemorrhagic lesions identical to those observed in aborted sea lion pups (see below). Detection of *Leptospira pomona* in the liver and kidney of the fur-seal pups led the authors to associate the so-called 'multiple haemorrhagic perinatal complex' with acute leptospirosis of the newborn. Two of the above *Callorhinus ursinus* pups died within a few hours of birth; the third was stillborn with the placenta attached. Microscopic examination of the placenta revealed focal necrosis with scarring and polymorphonuclear infiltration, as well as the presence of silver-positive forms resembling *Leptospira.* These findings suggest, in the above 3 cases, transplacental infection. However, this route appears to be the exception. Low *L. pomona* titers normally found in pre-pelagic pups appeared to indicate that leptospirosis is not normally acquired on the breeding rookeries but rather subsequent to the pups leaving the rookeries, presumably *via* the food chain during their first pelagic cycle (Smith and co-authors, 1977).

Leptospira pomona infections may cause reproductive failure in California sea lions and northern fur seals. Smith and co-authors (1974a, b) isolated leptospires from the placenta of a sea lion, which aborted about 30 days before full term. Gross lesions of the aborted foetus included subcapsular haemorrhage of the kidney, free blood in the peritoneal cavity and a friable liver with extensive capsular haemorrhage. Examination of the liver of a dead newborn fur seal revealed lesions and bacterial organisms identical to those of the sea lion pup (see above). Of 20 adult female *Zalophus californianus* examined on San Miguel Island, California, 10 had aborted approximately 30 to 60 days before full term (Smith and co-authors, 1974b). Fay and co-authors (1978) suggest that leptospirosis may be linked with high foetal mortality in Alaskan *Eumetopias jubatus. L. pomona* serum antibodies were detected in 2 of 6 Steller sea lions but in none of 103 seals belonging to 5 species, nor in 56 walruses. According to Sweeney (1978a), there is no specific therapy for leptospirosis in pinnipeds. A standard *L. pomona* bacterin may be used preventively, but has not become a standard management procedure. There appear to be no reports on the occurrence of leptospirosis in captive pinnipeds.

Leptospirosis may not be the sole disease responsible for premature parturition in pinnipeds. Abortions were also observed in California sea lions having statistically significantly higher tissue chlorinated hydrocarbon residues and polychlorinated biphenyls than full-term parturient females, as well as in individuals infected with San Miguel Sea Lion Virus (see section 'Agents: Virales') (De Long and co-authors, 1973; Gilmartin and

co-authors, 1976). The latter virus is indistinguishable from Vesicular Exanthema of Swine Virus (VESV). Although evidence presented by the above authors suggests an interrelationship of disease agents and environmental contaminants as the cause of premature parturition in sea lions, it should be borne in mind that both VESV and *L. pomona* alone are known as cause of abortion in several non-pinniped mammals.

Leptospirosis of marine-mammal origin is considered to be potentially hazardous to man. A *Leptospira pomona* isolate from *Zalophus californianus* had a marked cytotoxic effect on monolayers of human embryonic lung (HEL) cells when compared to another *Leptospira* of the serogroup Pomona isolated from a terrestrial mammal. As emphasized by Smith and co-authors (1974b), the zoonotic potential of pinniped leptospirosis has not yet been explored. It could be of considerable public health significance, especially on St. Paul Island, where the fur seal harvest places a large percentage of the human population in intimate contact with fur seals which may be carrying *L. pomona*. It appears noteworthy, in this context, that individuals participating in fur-seal harvests have been shown serologically to have had contact with the disease agent (Medway, 1980).

Several cases of actinomycete infections — both superficial and systemic — have been reported from pinnipeds. As the Gram-positive, fungus-like Actinomycetales were long regarded as fungi, the resulting diseases are misleadingly called 'actinomycoses'. Dermatophilosis, a skin affection caused by the actinomycete *Dermatophilus congolensis*, is a common actinomycosis of domestic animals. Frese and Weber (1971) observed the disease in two 1-year-old captive South American sea lions *Otaria flavescens* (syn. *O. byronia*). Skin efflorescences, 3 to 20 mm in diameter (Fig. 5-12), were restricted to the superficial layers of the epidermis (Fig. 5-13); only rarely were the upper portions of the corium affected. *D. congolensis* hyphae appeared in skin preparations (Fig. 5-14), and identification of the organism was ascertained by cultural methods. Macroscopic resemblance of dermatophilosis to seal-pox lesions (see section 'Agents: Virales') requires proper differential diagnosis (Frese and co-authors, 1972). Another case of actinomycosis in *O. flavescens* has been reported by Schmitt (1962).

Actinomycetes of the genus *Nocardia* are of considerable medical concern. The organisms are regularly isolable from soil and there are, accordingly, 2 common modes of establishing infections. Pulmonary nocardiosis arises from inhalation of bacteria, while contamination of skin wounds results in chronic subcutaneous abscesses (mycetomas) (Kobayashi, 1973). One species pathogenic to man, *N. asteroides*, has been implicated in pinniped pneumonia, while another (presumed) species of *Nocardia* has been identified as the causative agent of a systemic infection in a leopard seal.

Nodular purulent lesions in the subcutaneous tissue and muscle of the head and back of a leopard seal *Hydrurga leptonyx* from New Zealand were found to contain clumps of thin filamentous organisms tentatively identified as *Nocardia* sp. In places, these organisms extended beyound the zone of leukocytes, and colonies were also present in veins in the adjacent tissue. The lesions were congested, and haemorrhagic areas were present. Nodular lesions similar to those in the subcutis were also found in the kidney where they extended through the entire thickness of the cortex to involve the medulla. Veins and perivascular lymphatics contained both leukocytes and organisms. Sections of lung also showed suppurative lesions, and the surrounding parenchyma was congested and oedematous; there was an associated catarrhal broncho-pneumonia (Davis and co-authors, 1977).

Nocardia asteroides has been isolated from pinnipeds suffering from 'mycotic

Fig. 5–12: *Otaria flavescens.* (a) Extensive dermal lesions caused by actinomycetes *Dermatophilus congolensis;* (b) multiple, partially confluent lesions in head region. (After Frese and Weber, 1971.)

Fig. 5–13: *Otaria flavescens*. Section of *Dermatophilus congolensis*-infected skin showing distinct crust formation and restriction of pathological changes to superficial skin layers; × 1.5. (After Frese and Weber, 1971.)

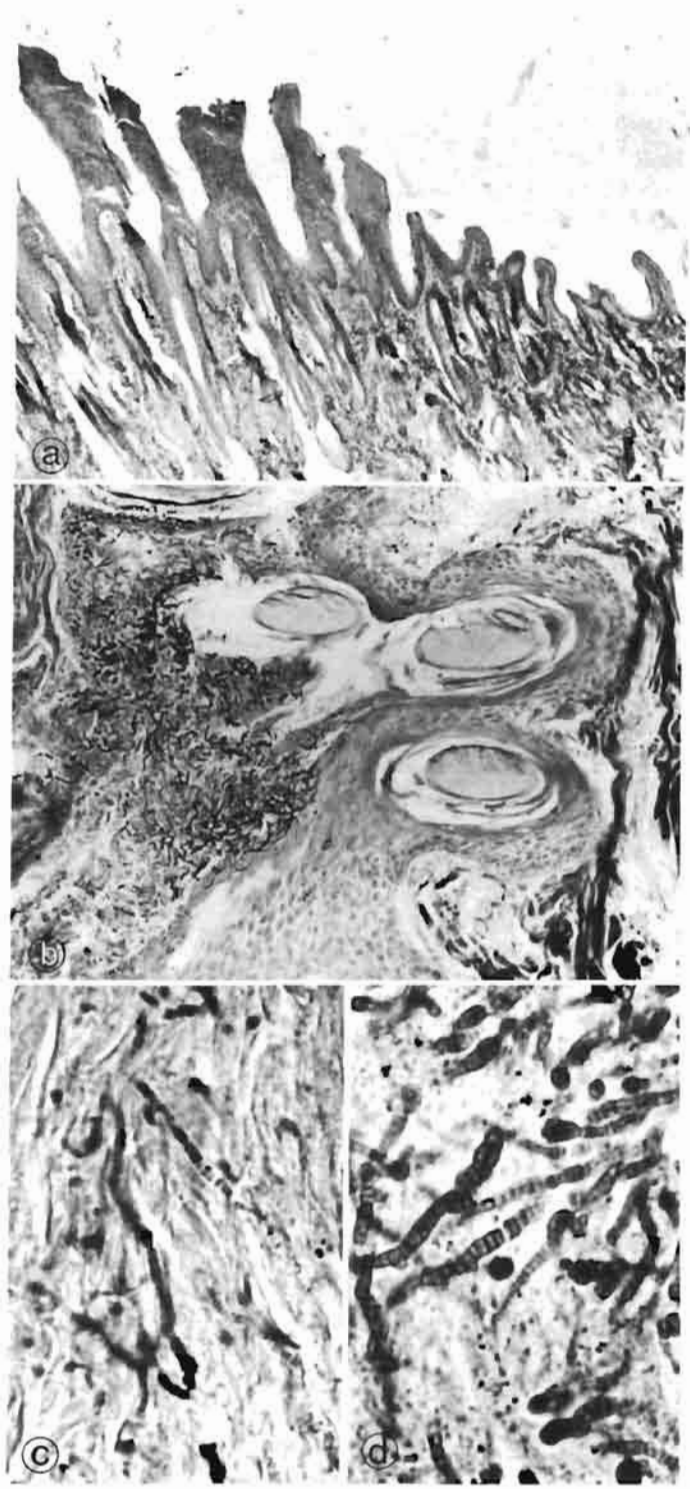
pneumonia'. Symptomology is the same as that reported for other bacterial pneumonias. Pulmonary nocardiosis may be diagnosed by finding the organisms in stained smears of nasal exudate or sputum. Specific treatment for *Nocardia* in pinnipeds includes oral administration of sulfadiazine at a rate of 2 g day^{-1} for 2 to 4 months (Wallach, 1972). In spite of the ubiquity of the causative agent, nocardiosis is a rare disease. However, once becoming evident, infections tend to be progressive and fatal. Therefore, prognosis is guarded.

Unidentified actinomycetes were isolated from necrotic foci in the liver of a captive fur seal. The animal, which was said to have died from a generalized infection, also exhibited splenomegalia; organisms were also found in the spleen (Larsen, 1962). Keyes (1965) isolated an organism tentatively identified as *Actinomyces* sp. from bite wounds in northern fur seals *Callorhinus ursinus*.

Agents: Rickettsiae, Chlamydiae and Mycoplasmas

According to Ridgway and co-authors (1975), there have been no confirmed reports of pinniped diseases attributable to these organisms. Without presenting details, Ridgway (1972b) lists one reference (pers. comm. by R. K. Farrell) to 'salmon disease' in pinnipeds. However, the presence of the causative agent, *Neorickettsia helminthoeca*, in pinnipeds does not appear to have been demonstrated positively (see also Chapter 4).

Two moderately virulent chlamydiae belonging to the PLV (psittacosis/lympho-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granuloma venereum) or bedsonial group of agents have been isolated from northern fur seals *Callorhinus ursinus* on the Pribilof Islands, Alaska. One of the isolates was obtained from the spleen of a 6-year-old female seal (with a serum titer of 1 : 8), the other one from the spleen of a male pup (without antibodies). The fur-seal isolates were immunogenic but non-pathogenic for pigeons and guinea pigs; they were fatal to ricebirds in 4 to 5 days. Of 161 serum specimens collected from subadult and adult *C. ursinus*, 129 yielded antibody titers as the reciprocal of serum dilations ranging from 1 : 2 to 1 : 128. Forty-five percent gave titers of 1 : 16 or greater. In contrast, 65 (90 %) of 72 pup sera reacted negatively; the remainder gave titers of 1 : 2 or 1 : 4, indicating that pups may acquire infections before they depart to sea. It was suspected, although not proven, that the fur-seal agents may, through close contact, cause asymptomatic infections in the human population of the Pribilof Islands (Eddie and co-authors, 1966).

Six *Mycoplasma* isolations have been made from northern fur seals *Callorhinus ursinus* on St. Paul Island, Alaska, but have not been fully characterized (Smith and co-authors, 1974b).

Agents: Fungi

Fungi may affect the skin, mucous membranes and lung, or may produce systemic infections. In contrast to bacterial infections, mycoses are rather rare in pinnipeds. The causative agents generally include *Candida, Coccidioides, Blastomyces, Mucormyces, Histoplasma* and *Microsporum* (Williamson and co-authors, 1963; Appleby, 1964; Nakagawa and co-authors, 1967; Sweeney, 1974a, 1978a; Farnsworth and co-authors, 1975; Jarofke and Klös, 1975; Ridgway and co-authors, 1975; Reed and co-authors, 1976; Reichel and Mayer, 1978). Dermatomycoses are common in zoo-held pinnipeds, but have also been observed in free-ranging seals. Entrance ports for the ubiquitous, opportunistic fungal agents are injuries inflicted by predators or during intrasexual fighting. Large, frequently suppurative wounds observed in wild pinnipeds form centers of secondary bacterial and/or fungal infection (Huey, 1924; Lindsay, 1937; Lugg, 1966; Bonner, 1970; Stirling, 1971a; Greenwood and co-authors, 1974; Fay and co-authors, 1978). Pinnipeds are particularly at risk when hauling in or out over sharp-edged stones or rocks overgrown with barnacles, mussels, etc.

As shown by Waldorf and Vedros (1978), the skin fat of *Callorhinus ursinus* (and probably other pinnipeds) may aid in the protection against dermatophytes. The fatty-acid composition of skin scrapings taken from northern fur seals showed a high proportion of shorter-chain saturated fatty acids (caprylic, nonanoic, undecanoic, lauric, tridecanoic, myristic acid) and a high concentration of palmitic acid. Several of these exhibited distinct antifungal activity. Inhibition increased with increasing chain length to C_{13}–C_{15}. Dermatomycoses can be kept under control in captive pinnipeds, but may be a serious debilitating factor in field populations.

Fig. 5–14: *Otaria flavescens*. (a) Margin of *Dermatophilus congolensis*-produced efflorescence showing formation of purulent exudate and parakeratotic masses resulting in apparent proliferation of interfollicular epidermal portions, × 5,3; (b) numerous *D. congolensis* hyphae in skin adjacent to follicle orifice. Grocott stain, × 134; (c) Brown & Brenn-stained section showing *D. congolensis* in characteristic rouleau-like form, × 1,340. (After Frese and Weber, 1971.)

Candida albicans, the organism most frequently cultivated from pinniped dermal lesions, is well-recognized as a primary or opportunistic pathogen in humans and homoeothermic animals (McCarty, 1973; Odds, 1979; Rippon, 1982). Acute candidiasis manifests itself by the appearance of numerous circular or polygonal, sometimes confluent, erosions of the skin, which may spread to the eyes, mucous membranes of the oral cavity and rectum, and affect the nail beds. Involved skin regions frequently become alopecic. Dermal candidiasis has been reported from a variety of pinnipeds including *Phoca vitulina, Pusa sibirica* and *Mirounga leonina* (Nakagawa and co-authors, 1967; Jarofke and Klös, 1975; Reichel and Mayer, 1978; Buck, 1983). In heavily debilitated suscepts, *C. albicans* infections may become systemic (McCarty, 1973; Odds, 1979).

Candidiasis appears to affect mainly captive pinnipeds, probably due to (i) unnatural maintenance of these marine creatures in freshwater pools, (ii) accumulation of large numbers of infective spores in the water under conditions of confinement, and (iii) increased survival of *Candida albicans* in water of reduced salinity. It is generally accepted that the yeast does not survive and multiply outside hosts (Ahearn, 1973; Rippon, 1982). Lupascu and Zarma (1974), however, maintain that *C. albicans* can survive in sea water for at least 6 months and retain its ability to produce mycelia in Sabouraud agar. Similarly, Claus and co-authors (1967, p. 712) concluded that "lower fungi like *C. albicans* do not only survive if exposed to sea water but retain their viability and infectivity to mice almost indefinitely in this environment".

Candida albicans has been isolated from sea water, as well as from marine invertebrates collected from inshore areas (Madri and co-authors, 1971; Buck and co-authors, 1977). As colony counts decrease rapidly in sea-water samples taken at increasing distances from polluted urban sites (Panades and co-authors, 1976), pinnipeds living on the open coast should presumably run little risk to acquire water-borne *C. albicans* infections. However, there is increasing evidence that the yeast is a common inhabitant of the digestive tracts of birds, particularly gulls (Kawakita and Van Uden, 1965; Cragg and Clayton, 1971; Rippon, 1982). Buck (1983) isolated *C. albicans* from 78 % of fresh gull droppings collected on the Connecticut coast and from 38 % of samples taken in Florida. As gulls frequently share the same rocks with beached pinnipeds and appear as scavengers in seal and sea lion rookeries, they can present a serious health hazard for free-ranging pinnipeds if they are carriers of *C. albicans*.

Cutaneous as well as systemic candidiasis is also common in captive cetaceans (p. 810). Systemic infections appear to be acquired *via* the oral route and may be provoked by entrance of the pathogen through oral or oesophageal wounds inflicted by the spines of ingested feed fish (Dunn and co-authors, 1982). Since *C. albicans* has repeatedly been isolated from aquaria maintaining captive cetaceans (Buck, 1980; Medway, 1980; Dunn and co-authors, 1982), its potential hazard to the health of marine-mammal caretakers and — last not least — to visitors of dolphinaria should not be overlooked.

Candidiasis can be treated with polyene antibiotics. Nystatin is generally applied locally to accessible cutaneous lesions, and amphotericin B has been used effectively in the treatment of systemic infections (McCarty, 1973). However, a *Candida albicans* dermatomycosis in *Pusa hispida* did not respond to nystatin. The drug was similarly ineffective in the treatment of chronic cutaneous candidiasis in bottlenose dolphins *Tursiops truncatus*. In the ringed seal, clodantoin, a modern antimycotic, proved to be effective after only 5 consecutive applications (Jarofke and Klös, 1975; Nakeeb and co-authors, 1977).

Other drugs applied successfully in the treatment of cutaneous lesions were Ectimar® and Ampho-Moronal® suspensions (Bayer AG); eye infections were cured with clotrimazol ointment (Reichel and Mayer, 1978). Ketoconazole (Janssen Pharmaceutics) has proven to be effective in the combat of systemic infections (p. 812).

Dermatomycoses of marine mammals need not necessarily be caused by *Candida albicans*. In addition to that yeast, Brandes and co-authors (1974) isolated *C. reukaufi* and *C. pityrosporum* from the skin of captive walruses. Wright and co-authors (1979) identified another yeast, *C. tropicalis*, as causative agent of mycotic sinusitis in a captive Atlantic bottlenose dolphin. Unidentified yeasts were isolated from discharging eyes of grey seals *Halichoerus grypus*. Although ocular affections are common to seals, resolution normally occurs and permanent damage is rare (Appleby, 1964).

Farnsworth and co-authors (1975) isolated dermatophytes *Microsporum canis* from cutaneous lesions in a zoo-held harbour seal. The diseased animal exhibited generalized thinning of the hair, with alopecia about the face and nose. Progressing pustular lesions with serous exudate developed subsequently. The disease process observed could be confused with bacterial dermatitis, demodicosis (see below), pediculosis, and areas of rubbing with secondary infection. As initially pyoderma associated with bacterial infection was suspected, tetracycline (12.5 mg kg^{-1} body weight) was administered orally once a day for 5 days. The treatment brought about temporary relief, but eventually the pustular lesions recurred. Hairs epilated from the margins of lesions and examined in potassium hydroxide solution revealed the presence of ectothrix-type arthrospores, and culture for dermatophytes revealed *M. canis*. The condition readily responded to a 45-day therapy with griseofulvin (15 mg kg^{-1} body weight). Although *M. canis* is primarily a parasite of domesticated and wild animals, it can also infect humans (McCarty, 1973).

A fungal skin disease, observed in several northern elephant seals *Mirounga angustirostris*, was characterized by local depigmentation. The lesions were 0.5 to 2 cm in diameter and neither raised nor depressed from the skin surface. They always disappeared with the moult and apparently responded to griseofulvin therapy as well; they were not pyritic and did not spread locally or to other body parts. Histologically, an unidentified fungus was found (Sweeney, 1973).

In contrast to dermatomycoses, systemic mycotic infections appear to be very rare in pinnipeds. Reed and co-authors (1976) reported on a case of fatal coccidioidomycosis in a California sea lion held in a Tucson zoo (Arizona, USA). The animal had been anorexic for several days prior to death. Initially, a clostridial infection was suspected because of haemorrhagic myositis and oedema. At necropsy, patchy areas of consolidation were seen in the lungs; the liver was pale and contained multiple white foci. Similar foci occurred in the spleen and kidney, while upon gross inspection all other organs appeared essentially normal. Histological examination revealed the presence of multiple discrete suppurative granulomas in the liver and spleen. These were composed of many small aggregates of degenerated polymorphonuclear neutrophils surrounded by epithelioid cells and fibrous tissue. Some of the epithelioid cells had coalesced to form multinucleate giant cells resembling the Langerhans type. Within the granulomas were large (20 to 30 μm), thick-walled yeast spherules having the morphological features of *Coccidioides immitis* and containing numerous endospores (Fig. 4-2). Although neither fungal spherules nor hyphae were found in the lungs, there was a mild chronic pneumonitis characterized by small aggregates of lymphocytes, plasma cells, histiocytes and fibrous tissue. The infected kidney

also revealed mild chronic inflammation. Chronicity and malignancy of the disease were indicated by the fact that initial lung lesions were in the process of healing, while metastatic lesions had developed in the liver, spleen and kidney.

Coccidioidomycosis, also known as coccidioidal granuloma, Valley fever, San Joaquin Valley fever, Posada's disease, California disease and desert rheumatism, is a dust-borne disease prevalent in the southwestern United States. Its causative agent, *Coccidioides immitis,* is a dimorphic fungus assuming both a saprophytic (mycelial) and parasitic (spherule) phase. Its conspicuous spherules have initially been mistaken for a protozoan — hence the name. Transmission occurs *via* cask-shaped arthrospores, which grow out from the mycelial phase of the fungus. When confined to the lungs, coccidioidomycosis is usually self-limiting and heals with scarring. The disseminated disease, however, is invariably fatal if not treated. Prolonged administration of amphotericin B is the therapy of choice, but failures may occur due to the nephrotoxic effects of that compound (McCarty, 1973). The disease has been reported from a considerable number of terrestrial homoeothermic animals, as well as from man. The only other record from the marine environment is from a sea otter (p. 650).

Blastomyces dermatidis, a heterothallic ascomycete, is the causative agent of mycotic pneumonia known as North American blastomycosis. The disease affects terrestrial mammals and man. Williamson and co-authors (1959) reported it from a captive northern fur seal *Eumetopias jubatus.* During an 8-year period (1954–1962), this was the only case observed at the Chicago Zoological Park (Williamson and co-authors, 1963). The symptomology of blastomycosis is the same as that typical of bacterial pneumonia (see above). The disease may be diagnosed by finding the fungus in stained smears of nasal exudate or sputum, or by radiographic examination of the thorax. Like coccidioidomycosis, blastomycosis can be treated with amphotericin B (Wallach, 1972; Sweeney, 1978a). In general, pinnipeds succumb to systemic mycoses with somewhat lower prevalence than cetaceans (Sweeney, 1978a).

A fungus, *Scopulariopsis* sp., isolated from 3 California sea lions on San Miguel Island, California, did not produce observable disease in these pinnipeds (Smith and co-authors, 1974b). Fungi of the same genus have repeatedly been isolated from the normal skin of US Navy divers during a 60-day saturation dive (Levine and co-authors, 1970). *Scopulariopsis* spp. have been found to be associated with superficial and deep mycotic lesions in man (Conant and co-authors, 1954).

Histoplasmosis is a mycotic infection primarily involving the reticuloendothelial system of man and animals. The causative agent, *Histoplasma capsulatum,* appears in infected cells as small, oval yeast cells, about 1 to 3 μm in diameter, and usually located within macrophages and reticuloendothelial cells. The organism is dimorphic; when cultivated on Sabouraud's or other media, it forms slowly growing mycelial colonies. Virulence is associated with the yeast phase. Wilson and co-authors (1974) diagnosed histoplasmosis in an adult female harp seal *Pagophilus groenlandicus,* which had died in captivity after 7 years. During the last 2 to 3 months of its life, the seal had a profuse yellow mucous nasal discharge and was frequently in respiratory distress. Pertinent microscopic lesions, consisting of large and small areas of coagulative and caseous necrosis, were present in the submandibular lymph node masses, the lungs and the liver. *H. capsulatum* was cultivated from these lesions.

In nature, *Histoplasma capsulatum* exists as a soil saprophyte. Inhalation of conidia

leads to pulmonary infection, which may spread to other organs. Diagnosis of histoplasmosis is based on serological evidence or demonstration of the organism in tissues.

DISEASES CAUSED BY PROTOZOANS

Marine mammals on the whole are remarkably free of protozoan parasites and protozoal diseases. The only agents of this group of organisms reported from Pinnipedia are eimeriine Coccidia of the genera *Eimeria, Sarcocystis* and *Toxoplasma.*

Eimeria phocae was found responsible for the death of a young male harbour seal *Phoca vitulina concolor* and for severe coccidiosis in a young female. Both seals, estimated to have been about 2 months of age, had been captured in Portland, Maine (USA), on June 20, 1972. They were housed together in the same tank and maintained on frozen mackerel. On August 10, the male pup was found to be lethargic, anorectic, and to have profuse, mucinous-bloody diarrhoea. Faecal examination revealed many unsporulated coccidial oocysts. Bacteriologic culture of rectal swab specimens yielded negative results for *Salmonella* and *Shigella,* as well as for other bacterial pathogens. The pup died of severe coccidiosis after a 2-day illness.

At necropsy, the pup was in good physical condition. Gross lesions were only observed in the colon and in the liver. The mucosal surface of the colon was dark red and covered with blood-tinged mucus. Smears of the contents revealed numerous desquamated, necrotic epithelial cells, erythrocytes and large numbers of unsporulated coccidial oocysts. The capsular surface of the liver had dark red to white mottlings. Pale to white areas bulged slightly from the cut surface, with the darker red areas being more depressed. *Streptococcus* sp., *Pseudomonas* sp. and *Escherichia coli* were isolated from the liver and heart blood.

Histologically, sections of the colon showed severe, diffuse, haemorrhagic necrosis of the mucosal surface and the lamina propria (Fig. 5-15, a). Large numbers of developing or mature microgamonts, macrogamonts and oocysts were found in the lamina propria, causing massive destruction of mucosa (Fig. 5-15, b). Eosinophils abounded in the colonic mucosa, and numerous Gram-positive bacterial colonies were in the lamina propria, crypts, and on the necrotic mucosal surface. The liver contained many foci of coagulative necrosis and haemorrhage — lesions probably caused by secondary bacterial infection and toxins from the haemorrhagic colitis. A few microgamonts and oocysts were also found in the mucosa of the distal ileum.

On August 31 (21 days after the death of the male pup), the female seal was anorectic and lethargic, and had an increased frequency of regurgitation. Examination of faecal smears was negative for coccidian oocysts but revealed numerous Gram-positive organisms, later identified as *Clostridium perfringens.* On September 4, the pup developed bloody diarrhoea, and oocysts, free or trapped in masses of desquamated intestinal epithelial linings, appeared in the faeces. A complex medication, involving neomycin sulphate, sulfonamides, kaolin, pectin, furazolidine and aminopropazien fumarate, was initiated to circumvent complications from secondary bacterial infection of the damaged intestine. As a consequence, *C. perfringens* decreased markedly.

In spite of probable reinfestation due to the confined quarters, diarrhoea diminished 2 days after initiation of the treatment, and the pup gradually recovered. It continuously passed oocysts in large quantities for 6 days. Counts then decreased drastically for another

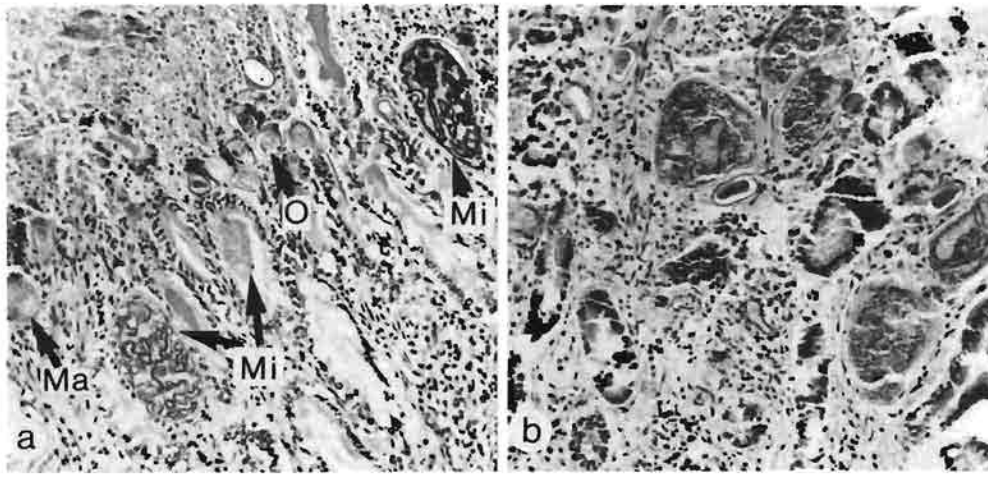

Fig. 5–15: *Phoca vitulina*. (a) Section of colon affected by *Eimeria phocae*, showing developing macrogamonts (Ma), microgamonts (Mi) and oocysts (O) in lamina propria of mucosa; × 160, H & E stain. Note necrotic mucosal surface. (b) Section of colonic mucosa showing presence of numerous mature microgamonts and oocysts in lamina propria; × 140, H & E stain. (After Hsu and co-authors, 1974b.)

4 days, and oocysts disappeared completely on the 11th day after their first appearence in the faeces. Assuming that the female pup had contracted the infestation from its tank mate, the prepatent period of the coccidian would be in the range of 20 to 25 days, while the patent period lasts about 11 days (Hsu and co-authors, 1974b).

The causative agent was described as a new species, *Eimeria phocae*. Its sporulated oocysts (Figs 5-16, b, c, d and 5-18) measure 43.9 × 26.6 μm (n = 100), the sporocysts 16.3 × 8.9 μm (n = 100). The sporozoites are elongate, banana-shaped, and lie lengthwise head-to-tail in the sporocysts. No schizont stages were observed in the intestinal tract of the male pup that had died. However, large numbers of developing or mature microgamonts (Fig. 5-16, e, f; Fig. 5-17, a, b), macrogamonts and fully developed oocysts were found in the lamina propria of villi in the colon (Fig. 5-15, a). Developing microgamonts vary in shape and size, measuring 90 × 65 μm on the average. Macrogametes, also present in the lamina propria, were 36 × 25 μm in dimension, i.e., much smaller than the microgamonts. Most oocysts were found to sporulate in 48 to 52 h at 26°C (Hsu and co-authors, 1974a).

Although *Eimeria phocae* is apparently pathogenic in causing acute, diffuse, haemorrhagic, necrotizing colitis, hosts in good general physical condition can survive an infestation. The rapid development and uncertain outcome of the disease, however, should alert investigators to the problems which can be anticipated when pinnipeds are maintained in circulating water systems. Faeces should be examined for parasites when confined pinnipeds are noted to have gastrointestinal problems. Initial therapy should be directed against the bacterial component of the disease.

Coccidia, particularly the oocysts, are able to withstand exposure to various chemical and physical agents, which are deleterious to most other organisms. More recently, effective anticoccidials have been developed (Hammond and Long, 1973; Pellérdy, 1974;

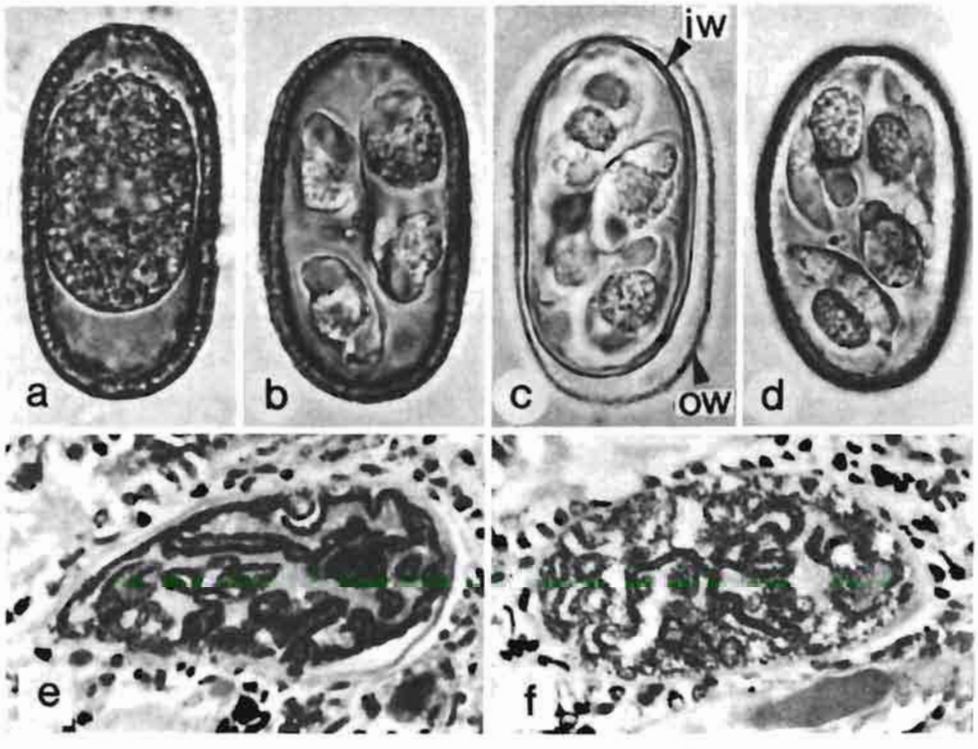

Fig. 5–16: *Eimera phocae.* (a) Fresh unsporulated oocyst, × 1,100; (b) sporulated oocyst containing 4 sporocysts with 2 sporozoites in each, × 930; (c) sporulated oocyst treated with 4 % sodium hypochlorite solution for 5 min (ow: outer oocyst wall, iw: inner oocyst wall), × 1,010; (d) sporulated oocyst showing sporozoites and large residua within sporocysts, × 1,010; (e, f) developing microgamonts containg invaginated cords of dividing nuclei, × 465. (After Hsu and co-authors, 1974a.)

Ryley and Wilson, 1975). A specific therapy for coccidiosis in marine mammals has not yet been reported.

There are 2 very brief reports on the occurrence of *Sarcocystis* sp. in pinnipeds. Using standard histological techniques, Brown and co-authors (1974a) found numerous sarcosporidial cysts in the masseter muscle of 1 adolescent male *Callorhinus ursinus* among a group of 30 pups and 2 adult seals surveyed on St. Paul Island, Alaska. The cysts closely resembled the sarcosporidia seen in other animals. There was no tissue reaction around the intact cysts, and no ruptured ones were detected. Bishop (1979, p. 288) states that "small numbers of sarcocysts were present in the skeletal muscle of the tongue" of a bearded seal *Erignathus barbatus* salvaged from ice in the Chukchi Sea.

Although sarcosporidiosis is common in domestic and wild terrestrial animals, it is infrequent in marine mammals (Cowan, 1966; Owen and Kakulas, 1968; Akao, 1970; see also Chapter 7). Its significance in pinnipeds is uncertain. Sarcocystis has been suspected as etiological agent of equine protozoal myeloencephalitis (Simpson and Mayhew, 1980).

Toxoplasma gondii has been incriminated as the cause of death in a 10-day-old California sea lion at the Philadelphia Zoological Garden. The diagnosis was based on finding organisms in sections of heart, lung, liver and abdominal lymph nodes. Tissues

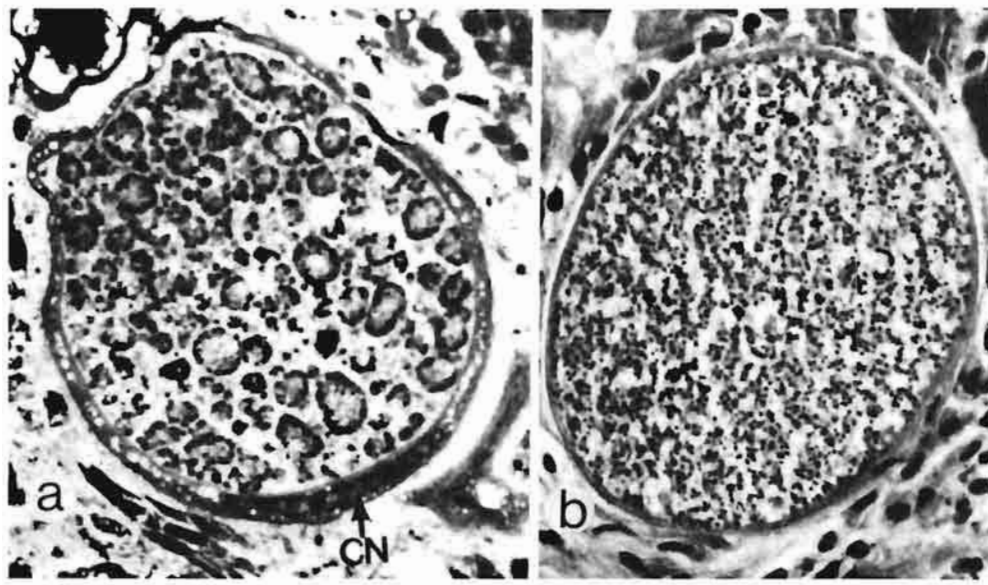

Fig. 5–17: *Eimeria phocae*. (a) Section of almost mature microgamont showing circular arrangement of developing microgametes and host-cell nucleus (CN), × 810; (b) mature microgamont containing numerous tiny microgrametes, × 810. (After Hsu and co-authors, 1974a.)

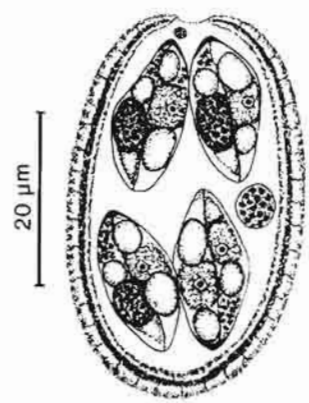

Fig. 5–18: *Eimeria phocae*. Sporulated oocyst containing 4 sporocysts and oocyst residuum; sporocysts each with 2 sporozoites and large sporocyst residuum. (After Hsu and co-authors, 1974a.)

from 43 other sea lions that had died spontaneously were found to be free of *Toxoplasma* cysts (Ratcliffe and Worth, 1951).

A second case of toxoplasmosis was diagnosed in a *Zalophus californianus* female that had been held in captivity for about 4 years at the University of Hawaii, Honolulu. After having recovered from a nutritional disease (thiamine deficiency), the animal initially returned to normal, but then became nervous and anorectic again, and died. At necropsy, the most significant observations were haemorrhagic pancreas and kidneys, dark-red to black bile in the gallbladder, stomach lining stained yellowish orange, and emphysematous edges of the lungs.

Histologically, the heart had necrotic myocarditis characterized by multiple areas of coagulative necrosis surrounded by mononuclear leukocytic infiltration (Fig. 5-19, a). *Toxoplasma gondii* cysts were present in cardiac muscle fibers (Fig. 5-19, b). Necrosis of the smooth-muscle cells and mononuclear leukocytic infiltration were also observed in the tunica muscularis of the stomach (Fig. 5-19, c). Tachyzoites of *T. gondii* were numerous in cytoplasm of smooth-muscle cells (Fig. 5-19, d). Large multifocal areas of coagulative necrosis were present in the pancreas, and interstitial oedema was found in the lungs, but *T. gondii* was not demonstrated in either of the organs. Liver, kidney, and adrenal and thyroid glands were normal (Migaki and co-authors, 1977).

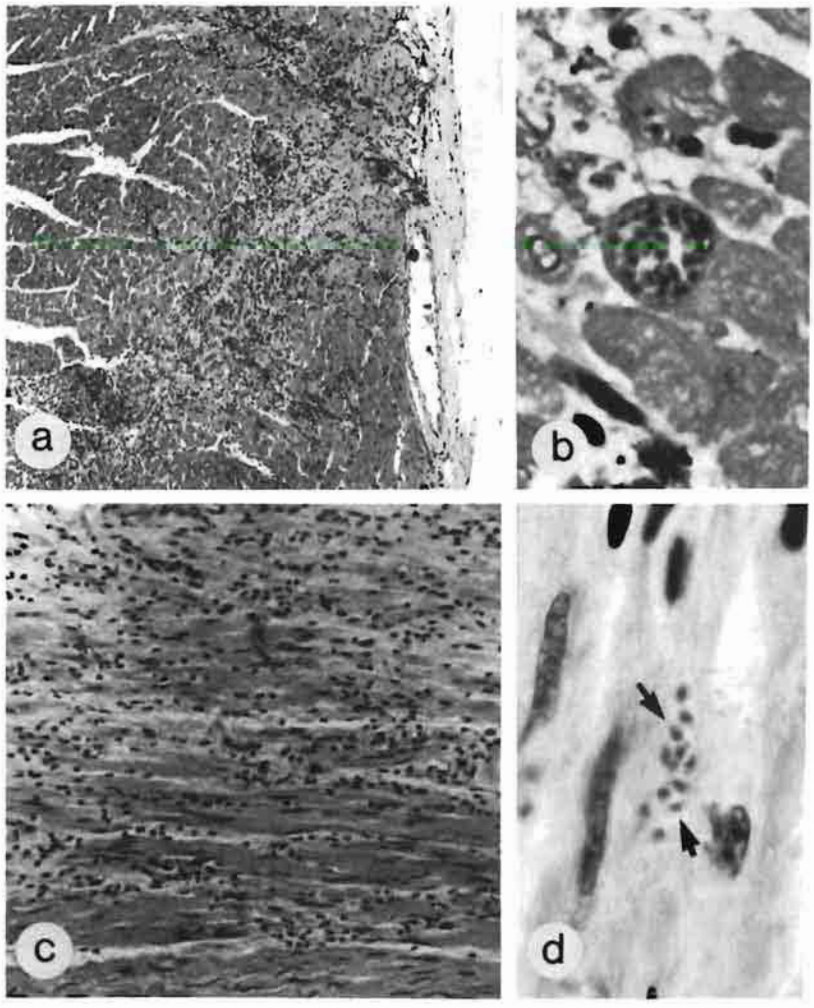

Fig. 5–19: *Zalophus californianus*. (A) Focal necrosis in heart; × 56, H & E stain. (b) *Toxoplasma gondii* cyst in sarcoplasm of a cardiac muscle fiber; × 1,040, H & E stain. (c) Necrosis and leukocytic infiltration of the tunica muscularis of stomach; × 140, H & E stain. (d) Tachyzoites (arrows) of *T. gondii* in smooth-muscle cells of stomach; × 1,080, H & E stain. (After Migaki and co-authors, 1977.)

The source of the *Toxoplasma gondii* infestation in the 2 above-described cases was not established. However, the sea lion maintained at the University of Hawaii was housed close to a site where research studies on toxoplasmosis in cats were being conducted, and common caretakers attended both groups of animals. It was, therefore, considered possible that the infestation resulted from contamination by feline faeces *via* the animal caretakers (for details on the life cycle, transmission, etc., of *T. gondii* consult Frenkel, 1973).

Van Pelt and Dieterich (1973), however, reported on toxoplasmosis in a newborn harbour seal *Phoca vitulina richardi*. The pup had been captured at Cold Bay, Alaska, when it was approximately 1 h old. It lived in captivity until it died from a generalized staphylococcal infection at an age of 23 days. Foci of necrosis were scattered throughout the liver parenchyma. Present in, and adjacent to, necrotic foci were numerous cysts identified as those of *Toxoplasma gondii*. The cysts were intracytoplasmic; however, a few loose aggregates of trophozoites were occasionally observed in foci of liver necrosis. The seal in this study was never knowingly exposed to domestic of feral felines nor their faecal material. Transplacental passage of *Toxoplasma gondii* was considered as the probable avenue of infestation. The prevalence of toxoplasmosis among adult female, feral harbour seals has not been determined. Proliferation of *T. gondii* in the young pup may have been exacerbated by the debilitating effects of the systemic bacterial infection and by the administration of a corticosteroid preparation (dexamethasone) during treatment (Van Pelt and Dieterich, 1973).

DISEASES CAUSED BY METAZOANS

Agents: Trematoda

A moderate number of trematodes — mostly members of the Heterophyidae, Opisthorchiidae and Campulidae — have been reported from pinnipeds. The former inhabit the intestine, the latter two the bile ducts and liver.

Heterophyids *Phocitrema fusiforme* are common intestinal parasites of *Pusa hispida, Phoca largha, Callorhinus ursinus, Erignathus barbatus* and *Eumetopias jubatus* in northern Pacific and Arctic waters (Goto and Ozaki, 1930; Price, 1932; Neiland, 1961; Delyamure and Popov, 1974; Kovalenko, 1975; Popov, 1975a, b, c; Yurakhno and Treshchev, 1975; Delyamure and co-authors, 1976; Fay and co-authors, 1978; Goltsev and co-authors, 1978).

The heterophyid genus *Pricetrema* is represented by several species. *P. zalophi*, originally named *Apophallus zalophi*, parasitizes in the intestine of *Zalophus californianus, Eumetopias jubatus, Callorhinus ursinus* and *Mirounga angustirostris; P. erignathi, P. phocae* and *P. eumetopii* have been described from *Erignathus barbatus, Phoca vitulina richardi* and *Eumetopias jubatus*, respectively (Price, 1932; Neiland, 1961; Keyes, 1965; Yurakhno, 1969; Dailey and Hill, 1970; Delyamure and Popov, 1975b; Yurakhno and Treshchev, 1975; Delyamure and co-authors, 1976; Fay and co-authors, 1978; Shults, 1978; Stroud and Dailey, 1978).

Apophallus donicus and *Phagicola septentrionalis* have been described from *Phoca vitulina* in Europe (Ransom, 1920; Price, 1932; van den Broek, 1967), *Stictodora ubelakeri* from *Zalophus californianus* in the Pacific (Dailey, 1969; Dailey and Hill, 1970). In addition to these, several other digeneans — mostly heterophyids — have occasionally

been reported from pinnipeds. Some of these worms are typically encountered in birds. Heterophyids *Nanophyetus salmincola* have been recovered from the intestine of 1 of 7 *Zalophus californianus* stranded along the Oregon (USA) coast. This trematode has not been reported previously in wild pinnipeds, although its presence was suspected because sea lions prey on metacercaria-infested salmonids from streams located in the enzootic area of this parasite (Stroud and Dailey, 1978). *N. salmincola* is of potential economic concern because it is the vector of *Neorickettsia helminthoeca,* the causative agent of 'salmon-poisoning disease' of canines (pp. 648 and 655).

The life cycles of the pinniped-invading heterophyids are unknown but, as in other members of the family, beyound doubt involve a gastropod as first and various species of fish as second intermediate hosts. The adult worms are notoriously unspecific with respect to the choice of their final hosts. Martin (1950) concluded that *Phocitremoides ovale* (a species similar to *Phocitrema fusiforme*) can develop in seals as well as in piscivorous birds, and this certainly holds true for other members of the group.

Of particular importance is the occurrence of *Cryptocotyle lingua* in several species of pinnipeds. This heterophyid is of considerable economic concern because of its interference with commercial fisheries (Lauckner, 1984a, b). Its life cycle normally involved periwinkles *Littorina littorea* as first intermediate, various species of fish as second intermediate and piscivorous birds (mainly gulls) as final hosts (Vol. II: Chapter 13 and Vol. IV, Chapter 1). However, adult *C. lingua* have also been reported from mink in Britain and Germany, from foxes in Denmark, Germany, Canada and Japan, from dogs in Denmark, Britain, Japan and USA, from cats in Denmark and even from man in Greenland (Ransom, 1920; Nicoll, 1923; Prell, 1928; Price, 1932; Willey and Stunkard, 1942; Christensen and co-authors, 1946; Christensen and Roth, 1949; Babero and Rausch, 1952; Sprehn, 1956; McTaggart, 1958; Babbott and co-authors, 1961; Guildal and Clausen, 1973; Kitamura and Machida, 1973; Kamiya and co-authors, 1975a, b; Smith, 1978). Nicoll (1923), Mohr (1952), van den Broek and Wensvoort (1959), van den Broek (1963) and Sprehn (1966a, b) reported *C. lingua* from North Sea *Phoca vitulina;* Stschupakov (1936) and Kurochkin (1975) found it in Caspian *Pusa caspica.* Heterophyids recorded as *Cryptocotyle jejuna* from Alaskan *Callorhinus ursinus* by Neiland (1961), Keyes (1965) and Dailey (1975b) are very probably also referable to the above species. *C. lingua* is the most frequent trematode of *Phoca vitulina* in the Dutch Wadden Sea (van den Broek and Wensvoort, 1959). It appears that pinnipeds constitute significant reservoir hosts for this economically important parasite.

In general, heterophyids appear to produce little if any pathology in pinniped hosts. Although over 100,000 *Pricetrema zalophi* have been recovered from a northern elephant seal *Mirounga angustirostris,* lesions associated with these parasites were not observed in histologic sections of the intestine. Similarly, no inflammatory response could be detected in histological preparations of *Phoca vitulina* intestine infested with *Rossicotrema (= Apophallus) venustum* (Stroud and Dailey, 1978). In grey seals from the Isle of Man, it occurred in dense patches in the duodenum and ileum (Duncan, 1956). Neiland (1961), however, suspected that heavy *P. zalophi* infestation may be an aggravating factor in parasite-related pathology. He recovered over 1,000 *P. zalophi* from the small intestine of an extremely emaciated *Callorhinus ursinus* pup, which appeared to have succumbed to its heavy parasite burden consisting of trematodes, cestodes, nematodes and acanthocephalans. Neiland also pointed out that several other heterophyids are known to cause

considerable mechanical damage to the epithelial lining of the small intestine. *Cryptocotyle lingua,* for instance, if present in sufficient numbers, may denude the intestinal epithelium, produce a copious exudation of mucus, cause pressure atrophy, necrosis, sloughing of tissue, hyperaemia, infiltration of eosinophils and plasma cells and hyperplasia (Willey and Stunkard, 1942).

Opisthorchiids *Opisthorchis tenuicollis* and *Metorchis albidus* inhabit the bile ducts of *Erignathus barbatus* and *Halichoerus grypus; Pseudamphistomum truncatum* occurs in the same location in *Phoca vitulina, Pusa hispida, Halichoerus grypus* and *Pagophilus groenlandicus* (Nicoll, 1923; Price, 1932; Mohr, 1952; Sprehn, 1966a, b; Kurochkin, 1975). Nothing has been reported on their pathology. Their life cycles are unknown but, as in other opisthorchiids, fish are likely to act as second intermediate hosts.

The Campulidae represent a family closely related to the Fasciolidae of terrestrial mammals but restricted to marine mammals. Like the latter they inhabit the liver, bile ducts and gallbladder. *Zalophotrema hepaticum* parasitizes in the liver of *Zalophus californianus* and *Eumetopias jubatus, Hadwenius* (syn. *Odhneriella*) *rossica* in the bile ducts of *Odobenus rosmarus* (Stunkard and Alvey, 1929, 1930; Price, 1932; Sprehn, 1966a, b; Stroud and Dailey, 1978). *Orthosplanchnus arcticus* inhabits the gallbladder of *Pusa hispida, Phoca largha, Erignathus barbatus* and *O. rosmarus. O. fraterculus* has been reported from *O. rosmarus, E. barbatus* and *Eumetopias jubatus, O. weddelli* from *Leptonychotes weddelli,* and *O. pygmaeus* from *P. largha* (Odhner, 1906; Nicoll, 1923; Price, 1932; Rausch and Locker, 1951; Beverley-Burton, 1972; Delyamure and Popov, 1975b; Popov, 1975b, c; Yurakhno and Treshchev, 1975; Delyamure and co-authors, 1976; Fay and co-authors, 1978; Goltsev and co-authors, 1978). The life cycles of campulids are unknown. Fish probably function as second intermediate hosts (Beverley-Burton, 1972).

Incidence and intensity of infestation of pinnipeds with campulid trematodes may be high. Fay and co-authors (1978) found Bering Sea walruses to be 100 %, and bearded seals to be 50 % infested with *Orthosplanchnus fraterculus.* Popov (1975c) recovered as many as 170 *O. arcticus* from the bile ducts of a single bearded seal in the Sea of Okhotsk. Delyamure and Popov (1975b) found up to 400 of these worms in *Erignathus barbatus* from Sakhalin Bay, USSR. Stroud and Dailey (1978) found 5 of 9 Steller sea lions from Oregon to be infested with *Zalophotrema hepaticum,* and Sweeney (1973) reported the same parasite from 7 of 20 California sea lions on San Miguel Island, California.

Orthosplanchnus spp. are medium-sized (3 to 7 mm) worms. When present in sufficient numbers, they may produce distinct biliary fibrosis. Three bearded seals from Alaska examined by Fay and co-authors (1978), had greatly thickened bile ducts, and in 2 of these, the fibrous thickening was so extreme that the ducts were readily palpable. In each case, this condition was associated with the abundant presence of *O. fraterculus* and, in 2 cases, with the presence as well of bacterial agents (*Staphylococcus epidermidis* and *Edwardsiella tarda*). The extreme fibrosis, resulting from severe, chronic inflammation, appeared to have been caused by the combined effects of both the parasitic and the microbial agents (Fay and co-authors, 1978). Considerable *O. fraterculus*-produced biliary pathology has also been observed in sea otters (Rausch, 1953; Chapter 4).

Zalophotrema hepaticum causes 'parasitic hepatitis' in California and Steller sea lions (Fleischman and Squires, 1970; Schroeder and co-authors, 1973; Sweeney, 1973; Stroud and Dailey, 1978). Lesions in *Zalophus californianus,* associated with the presence of

these large (11 to 13 mm) worms are characterized grossly by portal thickening and dilatation, and histologically by portal fibrosis with bile-duct proliferation and dilatation. Hepatic changes are not seen with every case of fluke infestation, but with heavy infestation the changes are consistently present. Circulating hepatic cell enzymes are elevated only slightly, even in severe cases. Of the enzymes used as clinical indicators, the ornithine carbamyl transferase test (OCT) was shown to be the most sensitive indicator of liver changes (Sweeney, 1973). Changes in the bile ducts of *Eumetopias jubatus,* associated with *Z. hepaticum,* included mild chronic pericholangitis, biliary fibrosis and bile-duct hyperplasia (Stroud and Dailey, 1978). Similar *Zalophotrema*-caused liver pathology has also been observed in cetaceans (Woodard and co-authors, 1969).

Parasite-induced biliary lesions were observed in an adult male *Erignathus barbatus.* In this individual, most intrahepatic bile ducts were surrounded by thick concentric cuffs of fibrous connective tissue; grossly they appeared as firm, tan anastomosing cords and nodules (Fig. 5-20). The epithelium of some larger ducts was hyperplastic and formed

Fig. 5–20: *Erignathus barbatus.* Extensive parasite-caused bile-duct fibrosis in a liver lobe. (After Bishop, 1979.)

luminal papillary projections (Fig. 5-21, a). Portal fibrosis, associated with acute and chronic inflammation and hyperplasia of bile ductules, was also seen (Fig. 5-21, b). Although no trematodes were observed in liver sections, the lesions were believed to have been caused by either *Orthosplanchnus arcticus* or *O. fraterculus.* Indirect evidence for this was obtained from the presence of characteristic triangular campulid ova in the pancreas, which also showed pathological changes. Numerous firm, grey, finely lobulated nodules in the pancreas, up to 1 cm in diameter, contained large numbers of trematode eggs, which were surrounded by fibrous tissue and acute and chronic inflammation (Fig. 5-22, a). The nodules also contained atrophic acini and exocrine ducts with hyperplastic epithelium (Fig. 5-22, b). Adult trematodes were not seen, possibly because the pancreatic ducts were not carefully examined (Bishop, 1979).

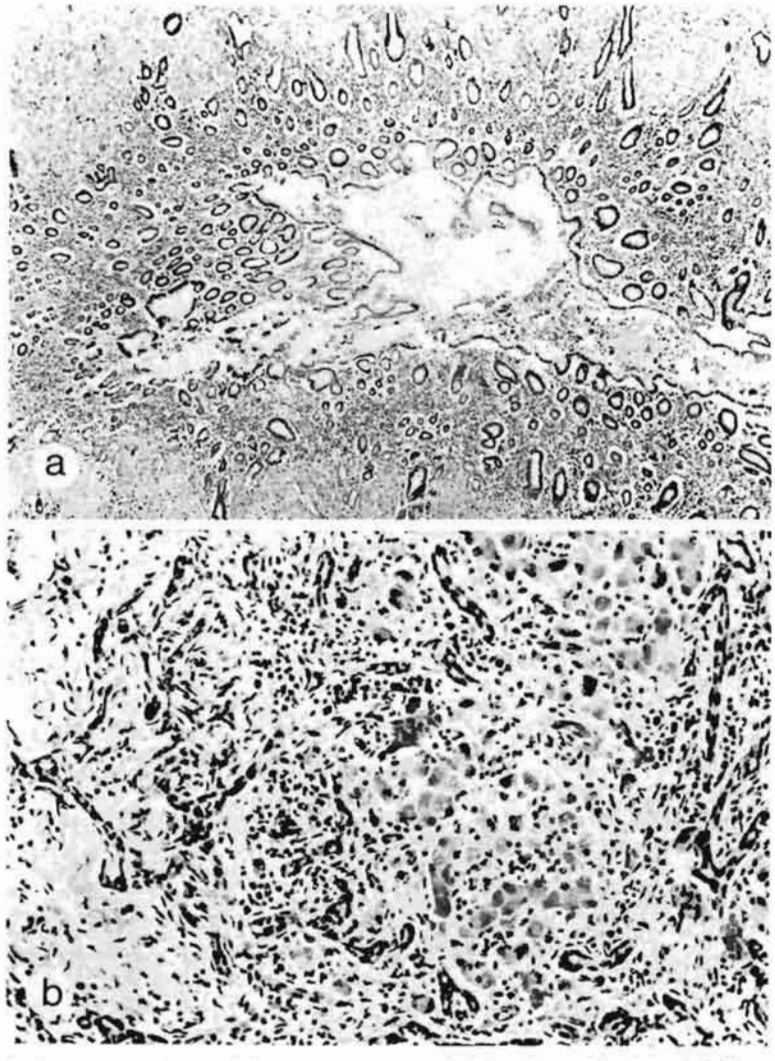

Fig. 5–21: *Erignathus barbatus*. (a) Large intrahepatic bile duct with chronic inflammation and hyperplasia of mucusa; (b) section of liver with proliferation of bile ductules, fibrosis and nonsuppurative inflammation. (After Bishop, 1979.)

In addition to the more common trematodes discussed above, members of the Notocotylidae have been reported from pinnipeds. Trematodes of the genus *Ogmogaster* are known from baleen whales (Dailey and Brownell, 1972). *O. antarcticus* occurs in both baleen whales of the southern hemisphere and in Antarctic lobodont pinnipeds *Leptonychotes weddelli* and *Lobodon carcinophagus* (Leiper and Atkinson, 1914; Johnston, 1931, 1937b; Price, 1932; Rausch and Fay, 1966). As pointed out by Beverley-Burton (1972), notocotylids from the Weddell seal, referred to as *O. plicatus* in the above publications, are probably *O. antarcticus*.

The life cycle of *Ogmogaster antarcticus* is unknown. Notocotylids do not require a second intermediate host for development; their cercariae encyst directly on solid surfaces

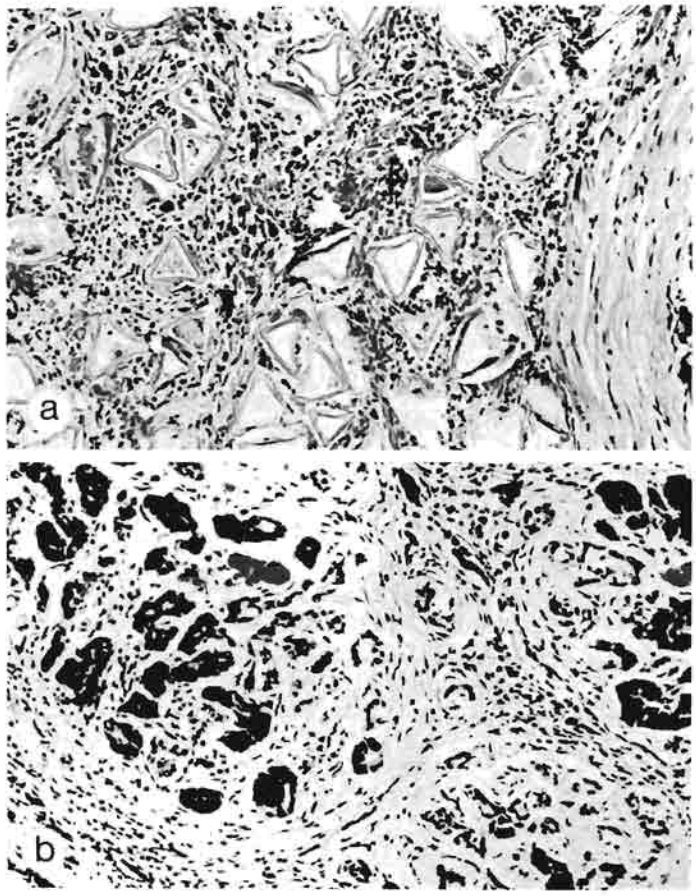

Fig. 5–22: *Erignathus barbatus*. (a) Fibrotic nodule from pancreas with campulid-type trematode ova; (b) nodule from pancreas showing atrophy of acini, interstitial fibrosis and chronic inflammation. (After Bishop, 1979.)

in the open water. Both the Weddell seal and the crabeater seal feed on crustaceans and benthic invertebrates (Lindsay, 1937; King, 1964) and probably acquire their *O. antarcticus* infestations *via* these food organisms on which the cercariae encyst. *Ogmogaster* species possess a well-developed musculature especially suited to firm attachment to the intestinal mucosa. Ridges on the ventral body surface contain glands believed to elaborate proteolytic enzymes. In the intestine of *Leptonychotes weddelli,* mucosal protuberances, exposed by removal of attached *O. antarcticus,* were deeply imprinted by the worms' ventral ridges (Fig. 5-23). There was also evidence of disruption of the mucosal epithelium and the underlying cells in the vicinity of the parasite's ventral ridges (Rausch and Fay, 1966).

Yurakhno (1968) described *Microphallus orientalis* from the intestine of *Odobenus rosmarus* and *Erignathus barbatus* from the Barents Sea. Delyamure and Popov (1975b) and Delyamure and co-authors (1976) found these trematodes in immense ('uncountable') numbers in bearded seals from Sakhalin Bay (Sea of Okhotsk, USSR) and the Gulf of Karaginsk (Bering Sea, USSR). The life cycle of *M. orientalis* is unknown. Most micro-

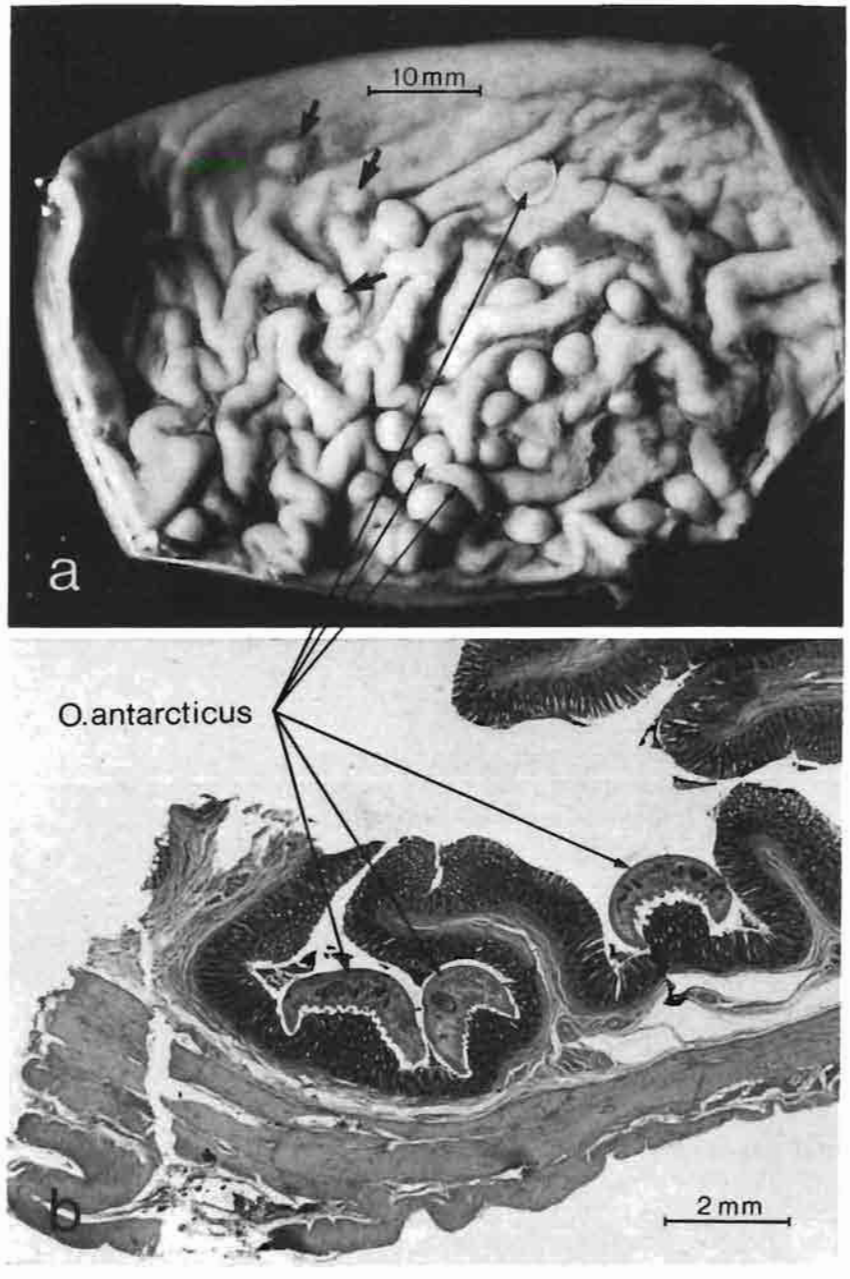

Fig. 5–23: *Ogmogaster antarcticus* preserved *in situ* in large intestine of Weddell seal *Leptonychotes weddelli*. (a) Host intestine, short arrows: mucosal protuberances exposed by removal of trematodes; (b) transverse section of *O. antarcticus in situ*, showing relationship of parasite's ventral ridges to host's intestinal mucosa. (After Rausch and Fay, 1966.)

phallids encyst in benthic crustaceans (Vol. III); some have abbreviated life cycles (Vol. I, Chapter 12). Their adults usually parasitize in the small intestine of birds. Both the walrus and the bearded seal feed on bottom invertebrates (King, 1964). Their *M. orientalis* infestations can, beyond doubt, be traced to the consumption of metacercariae-carrying crustaceans and snails.

Agents: Cestoda

Pinnipeds harbour a rich cestode fauna. Comparatively few species are represented by sometimes enormous numbers of individuals. The overwhelming majority of pinnipedian tapeworms are Pseudophyllidea of the genus *Diphyllobothrium* (family Diphyllobothriidae). The literature on marine-mammal cestodes is voluminous but exceptionally confusing. A vast number of both generic and specific synonyms exist. Thus, species now recognized as members of the genus *Diphyllobothrium* have been listed under the names *Bothriocephalus, Dibothrium, Dibothriocephalus, Cordicephalus, Schistocephalus, Adenocephalus, Clestobothrium* and *Diplogonoporus.* The well-represented southern-hemisphere species *Diphyllobothrium antarcticum,* for instance, has been recorded as *Bothriocephalus antarcticus* Baird, 1853, *Dibothrium antarcticum* Diesing, 1863, *Diplogonoporus antarcticus* Zschokke, 1903, and *Dibothriocephalus antarcticus* Shipley, 1903.

It should be borne in mind, however, that the genus *Diphyllobothrium,* as defined by Lühe (1910), represents a heterogeneous assemblage. Taxonomic revision of these tapeworms is indispensable but difficult if not impossible to conduct on the basis of available literature. In a critical evaluation of the material published by previous workers, Stunkard (1948, p. 219) stated:

> ". . . their descriptions are so incomplete and inadequate that, from their accounts, it is often impossible to recognize the species with which they were concerned. Moreover, descriptions of incorrectly determined specimens have added perplexity and dissension to the taxonomic problem."

For these reasons, no reference will be made here to the papers published before the turn of the century. For more updated parasite-host lists, determinative keys, literature revisions, etc., the reader is referred to Zschokke (1904), Railliet and Henry (1912), Joyeux and Baer (1936), Markowski (1952), Mohr (1952), Margolis (1954), King (1964), Dailey and Brownell (1972) and Margolis and Dailey (1972).

Species of *Diphyllobothrium* reported from various pinnipeds on the northern hemisphere include (in alphabetical order) *alascense, coniceps, cordatum, dubius, elegans, fasciatum, glaciale, hians, krotovi, lanceolatum, macrophallum, pacificum, polycalceolum, pterocephalum, roemeri, schistochilum, scotium, tetrapterum* and *variabile* (Zschokke, 1904; von Linstow, 1905a; Joyeux and Baer, 1936; Lyster, 1940; Krotov and Delyamure, 1952; Mohr, 1952; Margolis, 1956; Rausch and Williamson, 1958; Baer, 1962; King, 1964; Delyamure and Skrjabin, 1966; Sprehn, 1966a,b; Delyamure and Parukhin, 1968; Yurakhno and co-authors, 1968; Machida, 1969; Dailey and Hill, 1970; Dailey and Brownell, 1972; Margolis and Dailey, 1972; Delyamure and Popov, 1974, 1975a,b; Kovalenko, 1975; Treshchev and Popov, 1975; Treshchev and Yurakhno, 1975; Yurakhno and Treshchev, 1975; Delyamure and co-authors, 1976; Goltsev and co-authors, 1978; and others).

Southern-hemisphere species of *Diphyllobothrium* infesting pinnipeds include *antarcticum, arctocephalinum, atlanticum, clavatum, coatsi, lashleyi, mobile, pacificum, perfoliatum, quadratum, resimum, rufum, scotti, tectum* and *wilsoni* (Shipley, 1907; Railliet and Henry, 1912; Rennie and Reid, 1912; Leiper and Atkinson, 1914; Nybelin, 1931; Johnston, 1937a,c; Delyamure and Parukhin, 1968; Baer, 1969; Cattan and co-authors, 1977; and others). It should be emphasized that the above lists include several synonyms and, almost certainly, misidentifications.

Further pseudophyllideans commonly reported from pinnipeds on the northern hemisphere include *Pyramicocephalus phocarum, Diplogonoporus fasciatus, D. tetrapterus* and *Diplogonoporus* sp. (Mohr, 1952; Margolis, 1956; King, 1964; Rausch, 1964; Dailey and Brownell, 1972; Margolis and Dailey, 1972; Delyamure and Popov, 1975b; Treshchev and Popov, 1975; Treshchev and Yurakhno, 1975; Yurakhno and Treshchev, 1975; Delyamure and co-authors, 1976; Goltsev and co-authors, 1978).

Cyclophyllideans *Anophryocephalus anophrys* and *A. skrjabini* have been reported from phocid pinnipeds in Arctic waters (Delyamure and Popov, 1974; Popov, 1975d; Treshchev and Popov, 1975; Treshchev and Yurakhno, 1975; Delyamure and co-authors, 1976; Goltsev and co-authors, 1978).

The above specification shows that members of the genus *Diphyllobothrium* make up the largest component of the cestode fauna of pinnipeds. Provided that specific determinations given in the literature are correct, the tapeworm faunas of northern- and southern-hemisphere pinnipeds appear to have little overlap. Of the species listed, *D. pacificum* occurs in otariid seals throughout their geographic range. Two species, *D. cordatum* and *D. roemeri,* have been recorded from *Odobenus rosmarus.* The largest number of species is known from phocid seals. Some of these cestodes and their hosts form 2 distinctive assemblages, the species of which are restricted distributionally to the Antarctic and to the northern hemisphere, respectively (Rausch, 1969).

Seals of the genus *Monachus* have an intermediate (tropical to temperate) geographic range. Rausch (1969) recorded *Diphyllobothrium elegans* and *D. hians,* and described *D. cameroni,* from *Monachus schauinslandi* on Midway Atoll, Pacific Ocean. The former 2 species are also known from Arctic pinnipeds. Markowski (1952) has reported them from Mediterranean monk seals *Monachus monachus.*

In addition to adult cestodes in the intestine, larval forms may occur encapsulated in various body parts of pinnipeds. Similar plerocercoids have been found in cetaceans and fish (Ridgway and Johnston, 1965; Williams, 1968). Although, beyound doubt, encompassing a complex of species, most of these have been given the collective name *'Phyllobothrium delphini'.* Corresponding forms encountered in cephalopods, and probably identical with *P. delphini,* have been termed *'Phyllobothrium loliginis'.* Their relationships and hypothetical life cycles have been discussed by Southwell and Walker (1936) and Skrjabin (1972). Dollfus (1964) assembled a list of known records from cetaceans and pinnipeds. Linton (1922) presented evidence for the identity of *'P. loliginis'* with *P. tumidum,* a tetraphyllidean phyllobothriid cestode described by him from the mackerel *(Isurus dekayi)* and maneater *(Carcharodon carcharias)* sharks. Some of the forms reported as *'P. delphini'* may have included larval *Monorygma* (Williams, 1968; Dailey and Brownell, 1972). Large numbers of plerocercoids in different stages of development were found in the blubber of Antarctic elephant seals *Mirounga leonina.* The cysts were spread throughout the body but were particularly abundant on the ventral side (Krylov, 1971). What toll

is taken by massive larval *Phyllobothrium* and *Monorygma* infestations remains to be established (Dailey, 1974).

In pinnipeds, *'Phyllobothrium delphini'* plerocercoids occur in cysts up to 2 cm in diameter mainly in the subcutaneous tissues of the ventral abdomen, ventral pelvic area and the medial aspects of the hindlimbs (Cordes and O'Hara, 1979). Whether pinnipeds act as regular intermediate hosts in the life cycle of *'P. delphini'*, is unclear. Although some of the larger species of sharks are known to prey upon smaller cetaceans, it appears more likely that pinnipeds are merely accidental carriers of these plerocercoids. Larval cestodes have a pronounced ability to 'change hosts' by escaping from a prey organism ingested by a predator, and by reestablishing themselves in the tissues of the latter. Adult Tetraphyllidea are all parasitic in the intestine of elasmobranchs. Their complete life cycles are unknown.

The pinniped-invading diphyllobothriids appear to be unspecific with respect to the choice of their final hosts. Claims of some of the early authors of the restriction of certain cestodes to single species of pinnipeds have been disproven by more recent studies. In fact, the host spectrum of most diphyllobothriid cestodes is broad: At least 2 (and probably all) marine species of *Diphyllobothrium* can infest man. Zschokke (1904) reported 1 case of human *D. cordatus* infestation from Greenland. On the South American Pacific coast, endemic human diphyllobothriasis caused by *D. pacificum* can be traced to the traditional consumption of 'cebiche', a tasty dish prepared from raw marine fish (Baer and co-authors, 1967; Baer, 1969).

In spite of the lack of host specificity of diphyllobothriids, striking differences in infestation incidences and intensities of different host species have been noticed. However, these differences are probably attributable to variations in the feeding habits and ecology of the respective pinniped species, rather than to parasite-host incompatibility. Fay and co-authors (1978) found 8 bearded seals from Alaskan waters to be 100 % infested with *Diphyllobothrium cordatum, D. lanceolatum* and *Pyramicocephalus phocarum*. These cestodes were not found in anyone of 128 pinnipeds (representing 6 other species) from the same area. While *Anophryocephalus skrjabini* was fairly equally represented in individuals of both the Anadyr Bay and Karaga Bay (Bering Sea, USSR) populations of *Phoca largha, Anophryocephalus* sp., *Diplogonoporus tetrapterus* and specifically undetermined diphyllobothriids occurred only in the latter, but *Diphyllobothrium* sp. only in the former population. In addition, trematodes *Orthosplanchnus arcticus* were recovered only from Anadyr-Bay seals and *O. pygmaeus* only from Karaga-Bay individuals. Similar (qualitative and quantitative) differences occurred among the nematode parasites, while acanthocephalans *Corynosoma* spp. were equally (statistically not significantly differently) distributed in both seal populations (Goltsev and co-authors, 1978). Similar differences were observed in the composition of the helminth fauna of the 2 bearded-seal subspecies *Erignathus barbatus barbatus* and *E. barbatus nauticus* (Yurakhno and Treshchev, 1975).

Seasonal variations in helminth-parasite infestation have been observed in ringed seals *Pusa hispida ochotensis* from the Sea of Okhotsk. Trematodes *Phocitrema fusiforme* and nematodes *Phocanema (= Pseudoterranova) decipiens* were found only in spring, larval diphyllobothriids and nematodes *Terranova (Phocanema)* sp. only in autumn. Statistically significant differences in seasonal prevalence were observed among 8 of 11 further helminth species parasitizing the seals from that area (Delyamure and Popov, 1974). The observed seasonal variations probably reflect a seasonality of the larval stages of the respective parasites (temperature-dependent emergence of cercariae from mollus-

can intermediate hosts, time required for maturation of invasive stages, etc.). Unfortunately, our knowledge of the ecology of these helminths is by far too incomplete to arrive at even a remote understanding of their population dynamics.

Cestode infestations in pinnipeds may reach enormous levels. As stated above, Alaskan bearded seals, examined by Fay and co-authors (1978), were 100 % infested with *Diphyllobothrium cordatum, D. lanceolatum* and *Pyramicocephalus phocarum*. Of 42 Steller sea lions examined by the same authors, 40 were infested with *Anophryocephalus ochotensis* and 39 with *Diplogonoporus tetrapterus*. More than 1,000 cestodes, comprising *D. cordatum* and *P. phocarum*, were present in the small intestine, mostly in the distal jejunum, of a bearded seal from the Chukchi Sea, Arctic Ocean (Bishop, 1979). Counts of *D. lanceolatum* in 124 *Erignathus barbatus* from the Gulf of Karaginsk (Bering Sea, USSR) ranged from 33 to 28,700 (mean 6,570 ± 3,420) (Delyamure and co-authors, 1976). Popov (1975a) recovered *Anophryocephalus skrjabini* in 'uncountable numbers' (denoted by a ∞ sign in his table) from *Phoca largha* in the Sea of Okhotsk. Uncountable numbers of *D. lanceolatum* were found in 100 % of the bearded seals from the same area (Popov, 1975c).

There exists some disagreement concerning the pathogenicity of tapeworms, and in particular of *Diphyllobothrium* spp. The literature on the pathogenesis of adult cestodes, as reviewed by Rees (1967), reveals considerable discrepancies in the expressed opinions. Pathogenesis, when it occurs, has mostly been attributed to passive obstruction, irritative and inflammatory action, migration to unusual sites, spoliative and toxic action, etc.

Cestodes may cause damage to the intestinal epithelium at their attachment sites, but reports on pathology of this kind in pinnipeds appear to be lacking. Fay and co-authors (1978) state that a dense population of *Pyramicocephalus phocarum* had caused severe inflammation of the upper duodenum in a bearded seal. In general, the bothridia of pseudophyllideans do little damage to the host. However, large intestinal worms like some diphyllobothriids may become so entangled as to provoke acute volume obstruction of the gut. Cordes and O'Hara (1979) diagnosed intestinal obstruction by a large mass (estimated to weigh 900 to 1,350 g) of *Diphyllobothrium* sp. in a captive leopard seal. Clausen (1978) extracted a mass of *Diphyllobothrium cordatum* weighing more than 0.5 kg from the intestine of a common seal from Danish waters. The seal was sick and emaciated. It was thought that the tapeworms may have caused the poor condition.

According to Sweeney (1973), the only specific pathology caused by *Diphyllobothrium pacificum* in California sea lions appears to be intestinal obstruction. Once the tapeworms are removed, weight gains usually increase. The author states that *D. pacificum* occurs in large numbers in sea lions which are debilitated, but does not necessarily cause this condition. In contrast, Wallach (1972) claims that cestodiasis in pinnipeds appears as diarrhoea, anorexia, emaciation and anaemia associated with vitamin B_{12} depletion.

Vitamin B_{12} depletion and resultant anaemia occurs in a certain proportion of human patients infested with *Diphyllobothrium latum*. Other biochemical disturbances may occur concomitantly. Cases are well documented in the literature (von Brand, 1966; Rees, 1967; Cheng, 1973; Noble and Noble, 1976). No authentic records of the existence of similar conditions in pinnipeds have come to the reviewer's attention. As pointed out by Rees (1967), most controlled studies on the pathogenicity of adult cestodes have been made on man, domestic animals and laboratory animals. Morphologically, most of the pinniped-invading *Diphyllobothrium* spp. differ from the typical species, *D. latum*, only in minor

aspects (Zschokke, 1904). In fact, tapeworms causing diphyllobothriasis in humans in Peru, were initially identified as *D. latum* (Miranda and co-authors, 1967), but were later found to represent *D. pacificum* (Baer and co-authors, 1967; Baer, 1969). Although *D. latum* is a ganuine freshwater species, Delyamure (1955), King (1964), Sprehn (1966a,b), Dailey and Brownell (1972), Dailey (1975b), Yurakhno and Treshchev (1975) and others listed it as a parasite of pinnipeds. As the plerocercoids of *D. latum* occur encysted in anadromous fish such as salmon, infestations of marine mammals with this cestode are within the range of possibility.

Conceding physiological similarities between the pinniped-invading diphyllobothriids and *Diphyllobothrium latum,* one may ask whether debilitation of California sea lions infested with *D. pacificum,* as reported by Sweeney (1973), is the *result* or the *cause* of cestode infestation. With respect to captive pinnipeds, this question is purely academic because cestodiasis is easily controlled by the administration of anthelminthics. Diagnosis is made upon finding proglottids or ova in the faeces. Treatment with niclosamide or diphenthane-70 has been very effective (Ridgway, 1965; Wallach, 1972; Sweeney, 1973). Controls should be made at regular intervals because re-infestations are likely to occur if captive pinnipeds are maintained on live fish.

Agents: Nematoda

Roundworms are by far the most deleterious parasites of Pinnipedia. At least 5 superfamilies of nematodes are represented in these mammals (Table 5-3). According to their site in the host, they are commonly termed 'stomachworms', 'lungworms' or 'heartworms'.

The systematics of the ascaridoid nematodes are currently in a state of flux. Thus, the commonest stomach nematode of pinnipeds, *Phocanema decipiens* (also known under the generic names *Terranova* and *Porrocaecum*), has recently been assigned to the genus *Pseudoterranova* Mozgovoi, which now accomodates *Terranova*-like species from marine mammals. *Porrocaecum* Railliet & Henry has been reserved for nematodes of terrestrial mammals and birds. Species of *Contracaecum* Railliet & Henry *(sensu lato)* from fish, which became *Thynnascaris* Dollfus, are now, in part, *Hysterothylacium* Ward & Magath, while *Contracaecum sensu stricto* has been reserved for nematodes maturing in birds and mammals (Deardorff and Overstreet, 1981; Gibson and Colin, 1982; Gibson, 1983).

Similarly, the older literature on the anisakid nematodes of pinnipeds and cetaceans, as summarized by Dollfus (1948), contains gross errors and long arrays of synonyms. As pointed out by Dollfus, worms belonging to *'Porrocaecum decipiens'* (now *Pseudoterranova decipiens*) have even been mistaken for *'Ascaris (= Anisakis) simplex.* According to Gibson (1983, p. 325), gross errors have been introduced into the systematics of ascaridoids by the "erection by inexperienced workers of spurious new genera". Little emphasis will be placed, therefore, on a review of the older literature.

More recently, van Thiel (1966) assigned stomach nematodes from cetaceans and pinnipeds to the newly created species *Anisakis marina,* a designation not upheld by subsequent workers. Pippy and van Banning (1975) clearly identified worms reared *in vitro* from larvae dissected from various species of teleost fish as *A. simplex* and rejected *A. marina.*

In his revision of the genus *Anisakis* Dujardin, 1845, Davey (1971) listed a number of

Table 5-3
Roundworms (nematodes) reported from Pinnipedia (Compiled from the sources indicated)

Superfamily	Nematode species	Site in host	Common designation	Ref. page
Ascaridoidea	*Anisakis simplex*	Stomach	Stomachworms	729
	Contracaecum osculatum	(rarely intestine)		731
	Pseudoterranova decipiens			731
	Phocascaris spp.	Stomach and intestine		731
Ancylostomatoidea	*Uncinaria* spp.	Intestine (adults) Blubber (larvae)	Hookworms	734
Metastrongyloidea	*Parafilaroides* spp.	Lung	Lungworms	738
	Otostrongylus circumlitus	Lung (normally) Right ventricle, pulmonary artery (exceptionally)		744
Filarioidea	*Dipetalonema spirocauda*	Heart (adults)	Heartworms	745
	Dirofilaria immitis	Circulatory system (microfilariae)		748
	Dipetalonema odendhali	Intermuscular fascia (adults)	(Not in heart)	747
		Circulatory system (microfilariae)		
Trichinelloidea	*Trichinella spiralis*	Musculature (larvae)	Trichina worm	749
	Capillaria delamurei	Intestine (adults)		750

more 'exotic' species *(alata, bicolor, pegreffii, rosmari, similis, tridentata)*, described or redescribed from pinnipeds by Baylis (1920, 1929, 1937), Hsü (1933), Kreis (1938) and Campana-Rouget and Biocca (1955), as synonyms of *A. simplex*. The latter species has also been reported from a large number of cetacean species, while *A. typica* (in the literature also reported from pinniped hosts) is restricted to the Cetacea. From the reports of several of the above exotic *Anisakis* spp. from marine mammals in the southern hemisphere the impression may arise of the existence of several true species in that area. Hurst (1979, 1984a,b), however, positively identified larval anisakids from various species of New Zealand fish as *A. simplex*, using the rearing method of van Banning (1971). Poorly described species, such as *A.* (or *'Ascaris') dehiscens, A. patagonica, A. pacificus, A. shchupakovi* and others, have been listed by Mozgovoy and Ryzhikov (1950), Mohr (1952), Delyamure (1961), King (1964), Dailey and Brownell (1972), Dailey (1975b) and Kurochkin (1975). The genus *Stomachus*, found (not only!) in the older literature on pinniped parasites (e.g., Mawson, 1953; Sprehn, 1966a,b), is a synonym of *Anisakis*.

The present state of knowledge about the first intermediate hosts (planktonic crustaceans) and second intermediate hosts (fish, squid) of *Anisakis* spp. has been reviewed by van Thiel (1976). More general reviews of the literature referring to the worms that cause 'anisakiasis', their life cycles, intermediate and final hosts, scientific names and synonyms,

etc., have been presented by Myers (1959, 1960, 1970, 1975, 1976) and Cheng (1976). The best-studied species is *Pseudoterranova decipiens*. Various aspects of its biology — life cycle, pathology, moulting, growth, reproduction and survival — have been described in detail by Scott (1953, 1955) and McClelland (1976, 1980a,b).

Contracaecum osculatum and *Pseudoterranova decipiens* are by far the most frequent stomach nematodes of pinnipeds; they are cosmopolitan in distribution. Both have identical life cycles (aspects of which await, however, further elucidation) using small crustaceans as first, and teleost fish as second intermediate hosts, and both occur frequently in mixed infestations. One of the earliest accounts of the prevalence of these nematodes in seals and walruses from Greenland is that of Krabbe (1878a,b), and both have subsequently been found by almost all authors studying the helminth fauna of pinnipeds. Although apparently most prevalent in hosts from the northern hemisphere, *C. osculatum* has also been recorded from the Antarctic and Subantarctic. According to Hartwich (1975), *C. ogmorhini* and *Phocascaris hydrurgae*, reported from *Hydrurga leptonyx* by Johnston and Mawson (1941), are synonymous with *C. osculatum*. The taxonomic status of *C. radiatum, C. rectangulum, C. corderoi* and *C. miroungae* (Baylis, 1929; Johnston, 1937d; Lent and Teixeira de Freitas, 1948; Nikolskii, 1974b) awaits further evaluation. Similarly, *Pseudoterranova decipiens* and its larvae have been described under a variety of names. *Terranova piscium* and its larva, *Filaria piscium*, reported from southern-hemisphere hosts (Johnston and Mawson, 1945, 1953; Mawson, 1953), are clearly *P. decipiens*. Hurst (1979, 1984a,b) confirmed the life cycle of the latter species in New Zealand waters. The taxonomic status of *Terranova azarasi*, a species frequently recorded by Russian workers, is uncertain.

Another genus of alimentary tract-inhabiting ascaridoids, very close to *Contracaecum*, is *Phocascaris*. *P. phocae* has been described from *Pagophilus groenlandicus* and *P. cystophorae* from *Cystophora cristata* (Höst, 1932; Berland, 1963). Curiously, the latter author emended Höst's (1932) generic diagnosis in order to include *Contracaecum osculatum*(!) in the genus. Both species inhabit the stomach and the duodenum. *Phocascaris* sp. have, together with *C. osculatum* and other nematodes, most frequently been reported by the Russian authors cited in this section.

The ascaridoid stomachworms of pinnipeds are of considerable economic concern because their larvae, commonly known as 'herringworm' *(Anisakis)* or 'codworm' *(Pseudoterranova)* encapsulate, sometimes in enormous numbers, in food fish. In the North Sea, and particularly west of Scotland, high levels of infestation of cod with larval *Pseudoterranova decipiens* and other ascaridoids prevail, especially in those areas where there are major breeding colonies of grey seals *Halichoerus grypus*, and continue to cause costly processing problems (Rae, 1963, 1972; Young, 1972; Anonymous, 1981). The high fecundity of these worms accounts for the large-scale contamination of food fish with the larval forms. A single female of *P. decipiens* contains, on the average, 2.4×10^5 ova (McClelland, 1980b).

The literature on pinniped-invading ascaridoid stomach nematodes and their larval forms is vast. Ascaridoids have been reported by virtually every student of the helminth fauna of marine mammals. Mixed infestations are common, with an overwhelming proportion of *Pseudoterranova* and *Contracaecum* and lesser numbers of *Anisakis*. Popov (1976) found ribbon seals *Histriophoca fasciata* from the Sea of Okhotsk to be infested with an average of 783 (range: 62 to 2,600) *C. osculatum* but with only 12 (5 to 24)

Anisakis sp. Grey seals *Halichoerus grypus* from Nova Scotia (Canada) harboured, on the average, 577 (range: 11 to 1,694; number of seals: 53) *P. decipiens.* Respective counts from 16 harbour seals *Phoca vitulina* were only 62 (5 to 177). Incidences varied with season and host age. Similarly, van Banning and Becker (1978) reported higher intensities of infestation with *P. decipiens, C. osculatum* and *A. simplex* from North Sea grey seals than from harbour seals. Proportions of the latter nematode were lowest. Up to 6,584 *C. osculatum* and up to 1,530 *Anisakis* sp. have been recovered from individual ribbon seals in the Sea of Okhotsk (Popov, 1975d). Scott and Fisher (1958), Kenyon (1962, 1965) and Pálsson (1977) studied the nematode infestation of seals in relation to food and feeding habits.

Incidences of infestation with stomach nematodes usually approach 100 % in adult hosts and almost that level in juveniles. Of 1,355 northern fur seals taken off the Pribilof Islands, Alaska, in 1963, all but 4 were infested (Keyes, 1965). Infestation intensities normally increase with host age; occasionally, very young individuals may already be infested. Grey seals acquire their initial infestations with stomach nematodes within 1 month of weaning and are harbouring mature worms 5 weeks later (Havinga, 1933; Prime, 1973; Reijnders and co-authors, 1981).

The pathology produced by stomachworms is considerable and essentially the same in all 4 genera *(Anisakis, Pseudoterranova, Contracaecum, Phocascaris).* All are considered as being of medical importance. Clinically, all produce gastritis, enteritis, diarrhoea, dehydration and anaemia (Wallach, 1972; Ridgway and co-authors, 1975). Damage produced by these worms (Figs 5-25 to 5-29) has been reported by numerous authors and has been studied in detail in a number of pinnipeds belonging to different species (*Zalophus californianus:* Blessing and Peitz, 1970; Migaki and co-authors, 1971; Sweeney,

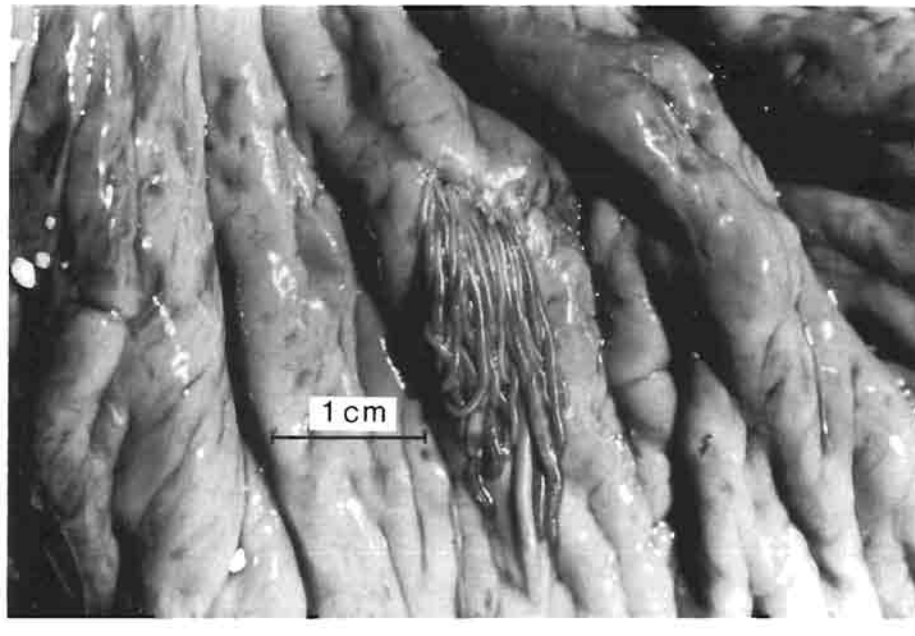

Fig. 5–24: *Monachus schauinslandi.* Cluster of *Contracaecum* sp. attached to gastric mucosa. (After Whittow and co-authors, 1979.)

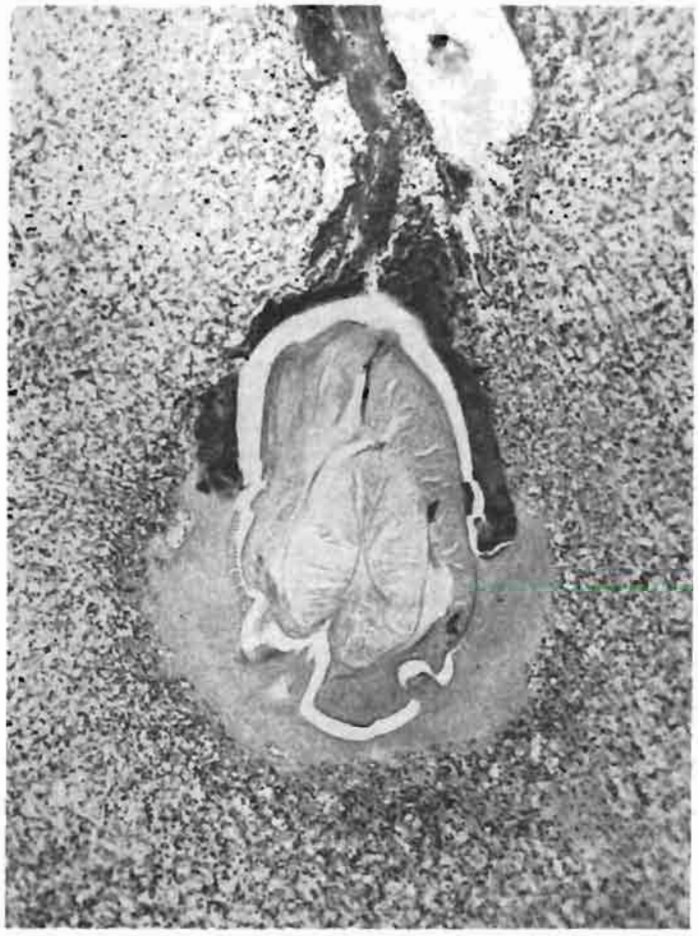

Fig. 5–25: *Eumetopias jubatus.* Section of stomach ulcer with *Contrcaecum* sp. *in situ.* Head of nematode surrounded by hyaline material and immersed in necrotic debris and granulation tissue, bacterial masses trailing behind hyaline material. H & E stain, × 41. (After Liu and Edward, 1971.)

1973; *Eumetopias jubatus:* Liu and Edward, 1971; *Phoca vitulina* and *Mirounga leonina:* Blessing and Peitz, 1970; *Otaria flavescens:* Cattan and co-authors, 1976; *Halichoerus grypus:* Young and Lowe, 1969; *Monachus schauinslandi:* Whittow and co-authors, 1979; *Callorhinus ursinus:* Keyes, 1965; *Pagophilus groenlandicus*: Wilson and Stockdale, 1970).

The nematodes are normally attached to the stomach mucosa, often in clusters of 100 or more (Fig. 5-24). A typical lesion produced by a single worm consists of a small ulcer, 1 to 5 mm in diameter, surrounded by a swelling to form a crater in which the anterior end of the worm is buried (Figs 5-25 and 2-26). Larvae appear to produce more damage than adult worms. Occasionally, nests of larvae may be found within thick-walled gastric carbuncles up to 40 mm across and 10 mm thick (Keyes, 1965). The large granulomas frequently seen in heavy infestations appear to form around a central lesion which, in turn, may be eroded away through either mechanical action or secretory activity of the worms

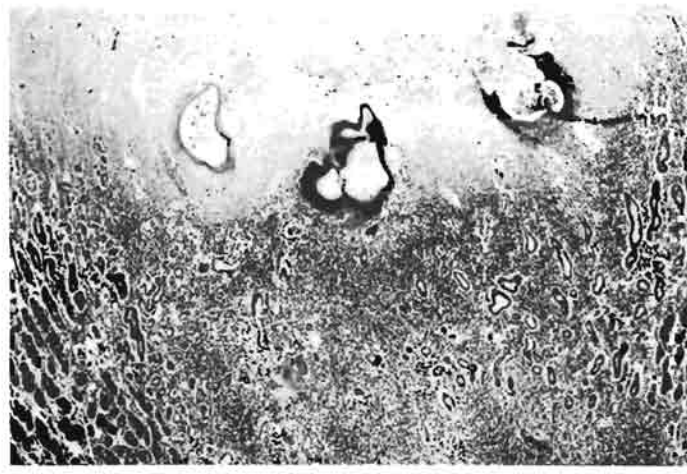

Fig. 5–26: *Erignathus barbatus.* Chronic gastric ulcer with hyaline caps marking site of *Pseudoterranova decipiens* attachment (nematodes were dislodged during tissue processing). Note fibrotic base of ulcer and infiltration of inflammatory cells. No worm penetration evident. (After Bishop, 1979.)

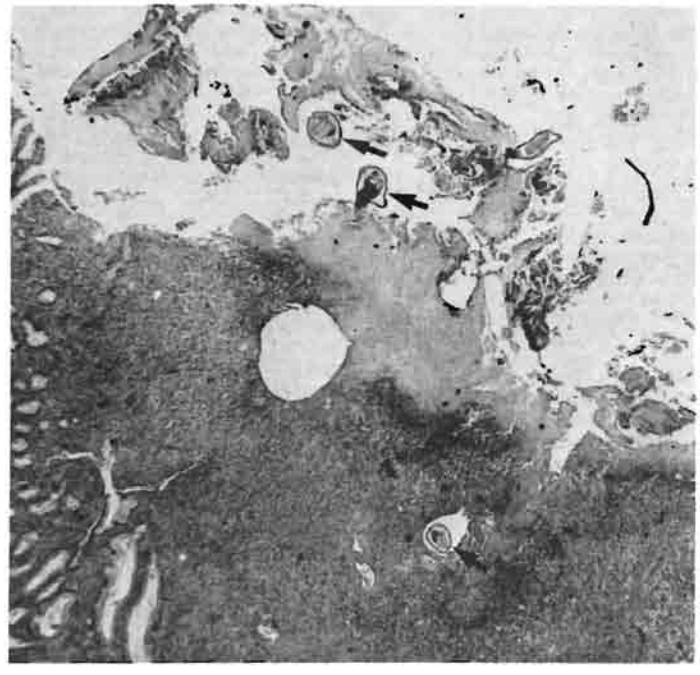

Fig. 5–27: *Eumetopias jubatus.* Gastric ulcer associated with *Contracaecum* infestation. Ulcerated surface coated with necrotic material and fibrin, mucosa replaced by granulation tissue. Sections of nematode (arrows) on surface as well as deep within granulation tissue. H & E stain, × 21. (After Liu and Edward, 1971.)

(Sweeney, 1973; Dailey, 1978). Gastric ulcers may be important debilitating or even lethal factors in feral animals (Fay and co-authors, 1978).

Normally, nematode-produced stomach lesions (Figs 5-25 to 5-29) are confined to

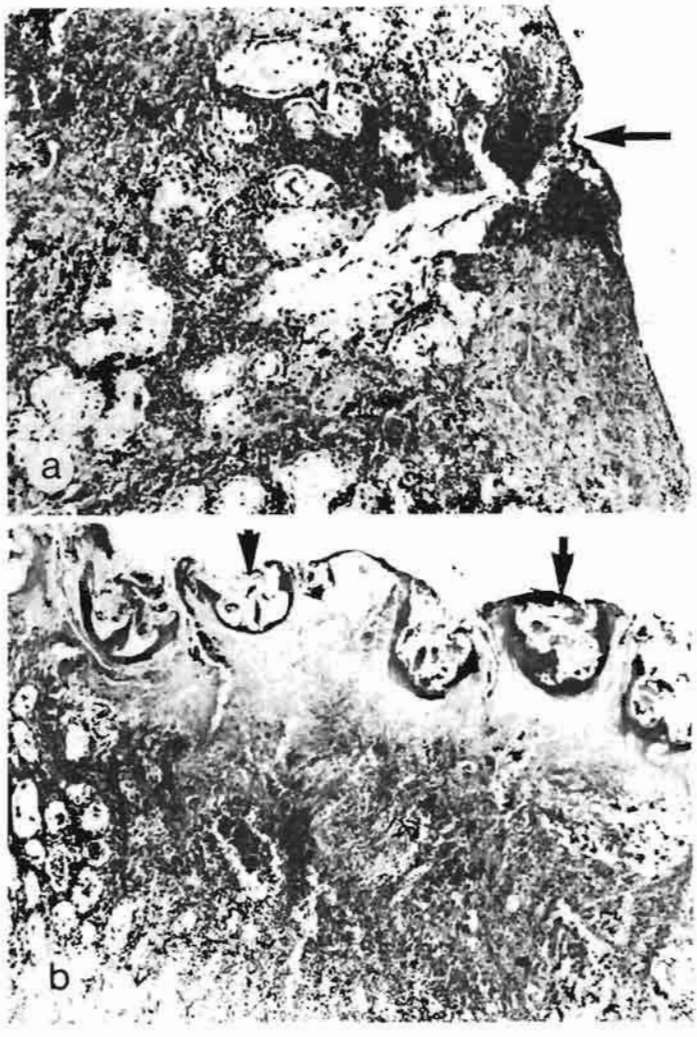

Fig. 5–28: *Pagophilus groenlandicus*. Pathology associated with *Contracaecum* sp. infestation. (a) Oesophageal wall showing loss of epithelium (arrow) and inflammation of submucosa; H & E stain, × 80. (b) Necrosis and inflammation of gastric mucosa associated with nematode (arrows) infiltration; H & E stain, × 33. (After Wilson and Stockdale, 1970.)

the mucosa and submucosa but, occasionally, perforation of the stomach wall occurs, resulting in peritonitis. Several emaciated California sea lions, encountered on the beach, had perforated gastric ulcers containing *Contracaecum osculatum*. The entire abdomen of the sea lions was filled with purulent fluid and there was severe peritonitis. White blood-cell counts were greatly elevated. All individuals had died within 24 h of being taken from the beach (Ridgway and co-authors, 1975).

Aside from the purely mechanical action of stomachworms, possible toxic effects should be considered. *Ascaris* spp. are known to produce toxins that evoke a variety of host responses including 'ascaris allergy' and the production of *Ascaris* antibody (for review consult Matsumura, 1972). Grabda and Felińska (1975) described toxic effects of

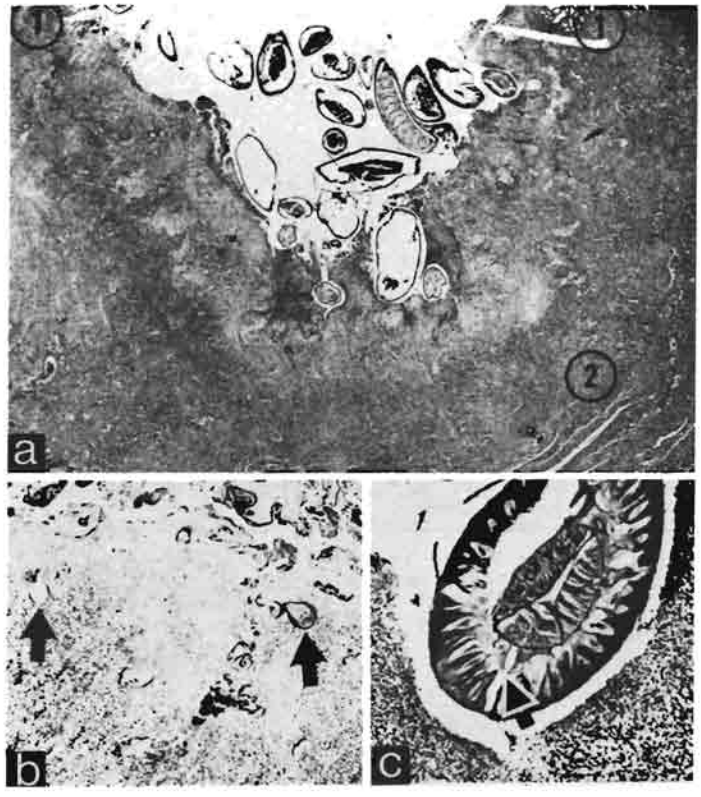

Fig. 5–29: *Zalophus californianus.* Gastric ulcers associated with nematode infestation. (a) Numerous worm sections seen within ulcer (1: mucosa, 2: muscularis of stomach); × 6.5. (b) Eosinophilic cuticular worm remnants (arrows) present within necrotic portion of ulcer; × 41. (c) Embedded nematode in cross section. Y-shaped lateral cord (arrow) suggests that worm belongs to genus *Anisakis*; × 61. (After Migaki and co-authors, 1971.)

Anisakis simplex larvae on laboratory mice.

Therapy for gastric ulcers consists of administration of antibiotics and antacid compounds. Feeding of a 'gruel' made up of magnesium aluminium hydroxide, tetracycline, thiamine, essential fatty acid-vitamin supplement and fish, has also been successful (Sweeney, 1973, 1974a; Ridgway and co-authors, 1975). There are several anthelminthics available for efficient treatment of stomach-nematode infestation. According to Sweeney (1973, 1974a), *Contracaecum osculatum* appears to be most sensitive to levamisole hydrochloride. Piperazine adipate or piperazine tartrate, administered at a rate of 80 to 100 mg kg^{-1} of body weight, is a safe agent against *Pseudoterranova ('Porrocaecum')* and *Contracaecum.* However, it lacks broad-spectrum activity against other nematodes (Hubbard, 1969; Wallach, 1972).

Adult ascaridoids are normally confined to the stomach of pinnipeds. Montreuil and Ronald (1957), however, found that the intestinal tract of seals (*Halichoerus grypus, Pagophilus groenlandicus* and *Phoca vitulina*) may harbour nematodes in numbers sometimes equal to or even in excess of those found in the stomach. The worms were usually firmly attached to the intestinal mucosa. Therefore, they cannot be considered as trans-

ients. The authors concluded that embryonic development observed in these worms suggests that the intestinal phase may be essential to the normal development of the egg and embryo. Adult harp seals may carry nematodes in the intestine even in the absence of stomach nematodes. The worms seen by Montreuil and Ronald (1957) were not identified specifically. Eggs of *Pseudoterranova decipiens* are normally laid in the stomachs of seals and pass out to the sea in the faeces. The larvae hatch as first-stage larvae in the open water or are ingested by a first intermediate host (Scott, 1953, 1955; Platt, 1976). Flores-Barroeta and co-authors (1961) reported on the erratic occurrence of *Contracaecum osculatum* in the brain of 4 *Zalophus californianus* from Asuncion Island, California. "Very many parasites, adults and larvae" were recovered from the tissue close to the median brain fissure. The condition was associated with severe pathology.

Hookworms of the genus *Uncinaria* (superfamily Ancylostomatoidea) are significant parasites of pinnipeds. *U. lucasi* affects northern fur seals *Callorhinus ursinus* on the Pribilof Islands, Alaska (Stiles and Hassall, 1899; Stiles, 1901). It has, thus far, not been recorded in fur seals in the USSR (Delyamure, 1955, 1961). *U. hamiltoni* is known from *Otaria flavescens* and *Mirounga leonina* in the South Atlantic and Subantarctic (Baylis, 1933; Johnston and Mawson, 1945; Botto and Mañé-Garzón, 1975). A specifically undescribed *Uncinaria,* in dimension somewhat intermediate between *U. lucasi* and *U. hamiltoni,* has been recovered from *Zalophus californianus* from the California coast (Dailey and Hill, 1970). All other members of the genus are parasites of terrestrial hosts.

Uncinaria lucasi has a 1-host life cycle consisting of 3 basic stages: (i) The pre-invasive phase including eggs and free-living third-stage larvae in the soil, (ii) the tissue phase with parasitic third-stage larvae occurring in the blubber of seals, as well as in the mammary glands and milk cisterns of the cows, and (iii) the intestinal phase with adult worms parasitizing in the ileacaecal portion of the intestine of fur-seal pups (Olsen and Lyons, 1965). Hence, the development of *U. lucasi* is direct and its life cycle is essentially similar to that of *Ancylostoma duodenale,* a common hookworm of man in the tropics and subtropics.

Lucas (1899), examining dead *Callorhinus ursinus* pups on the Pribilof Islands, found large numbers of hookworms in many young individuals and concluded that these parasites were the cause of death. While hookworms were never found in older seals, they were believed to be present but overlooked. Olsen (1958) postulated that all age classes of seals exposed to third-stage larvae become infested but that in adult individuals the worms were destroyed during their migration within the body. Subsequently, however, Olsen and Lyons (1965) showed that — due to the peculiarity of the life cycle of *Uncinaria lucasi* — adult worms are restricted to pups and that survivors of an infestation never become reinfested following puphood.

Eggs of *Uncinaria lucasi,* passed onto the sandy rookeries crowded with seals, develop into first-stage larvae and, after 2 successive moults, into third-stage larvae still confined within the egg membrane. Upon hatching, the free-living third-stage larvae invade the pinniped host *via* the percutaneous route. Seals of all age classes and both sexes are susceptible and have been found infested. *Per os* infestation has been accomplished experimentally in pups. The parasitic third-stage larvae locate predominantly in the belly blubber of all seals and in the mammary glands and milk of pregnant cows shortly before parturition and in parous cows for a short time after birth of the pups. Only larvae from the mammary gland ingested with the milk by newborn seals develop into adult worms in the

intestine, which takes about 2 weeks. As worms have been found in newborn pups with the placenta still attached, it is evident that the young seals acquire *U. lucasi* infestations with their earliest meal of milk. The dynamics of hookworm infestation in *Callorhinus ursinus* have been studied in great detail by Olsen and Lyons (1965) and Lyons and Keyes (1978).

Hookworm infestation has been the cause of high fur-seal pup mortality on the Pribilof Islands rookeries for many years. Over 72,000 fatalities have been observed on St. Paul Island alone in a single pupping season. Deaths may exceed 1,000 a day during the early summer. During 1949 to 1951, when the St. Paul Island herd had reached its ceiling, the overall mortality rate had increased to about 14.6 %, corresponding to a loss of 60,000 to 80,000 pups per summer. On individual rookeries, mortality rates of up to 39 % have been recorded. During the 2-month stay of the young seals on the rookeries, enormous numbers of carcasses may be observed covering the nursing grounds (Lucas, 1899; Kenyon and co-authors, 1954; Olsen, 1957; Jellison and Milner, 1958).

Immediate cause of death is nematode-associated haemorrhagic enteritis. The mesenteric lymph nodes may also be affected, being hypertrophied to 4 times the size of those of healthy individuals of similar size and weight. Histologically, such affected lymph nodes lack germinal centers. Numerous large cells with a clear halo around them may be abundant. Higher magnification reveals these to be binucleate atypical histiocytes, morphologically similar to the mirror-image type of Reed-Sternberg cells seen in Hodgkin's lymphoma in man, and probably resulting from cellular hypersensitivity associated in some way with the enteric parasitic infestation (Brown and co-authors, 1974b).

In contrast to the intestinal phase of the parasite, the tissue phase appears to pose little problems. *Uncinaria lucasi* larvae in the blubber appear to have a limited survival time, as evidenced by the lack of accumulation of larger numbers of larvae in older animals subjected to repeated exposures (Olsen and Lyons, 1965).

Specifically unidentified hookworms, *Uncinaria* sp., have been recovered from the intestines of all of 3 unweaned *Zalophus californianus* pups examined by Dailey and Hill (1970) from the southern and central California coast. The authors considered it "highly probable that this parasite infects nearly all the pups at the San Nicolas Island rookery" (p. 127). Overt pathology associated with the presence of these worms in California sea lions autopsied by Sweeney (1973, 1974a), was not apparent. Dailey (1978) listed what appears to be the unidentified species of *Uncinaria* from *Z. californianus* as *U. lucasi* and stated that it causes anaemia, "particularly in young animals".

Treatment of uncinariasis appears to be practicable only under controlled research conditions. Disophenol, given subcutaneously at 9.9 mg kg^{-1} (single dose), resulted in decreased faecal egg counts. Single oral doses of dichlorvos (capsules at 29.3 to 32.8 mg kg^{-1}, tablets at 10.5 to 11.5 mg kg^{-1}) removed 99 % of adult *Uncinaria lucasi* from fur-seal pups (Brown and co-authors, 1974b; Lyons and co-authors, 1978).

In addition to nematodes parasitizing in the gastrointestinal tract, pinnipeds may harbour roundworms in their respiratory and cardiovascular systems. *Parafilaroides*, often referred to as 'the small lungworm', inhabits mainly the bronchioli and alveoli, *Otostrongylus* ('the large lungworm') occurs mainly in the bronchi, and *Dipetalonema* (the 'heartworm') mainly in the right ventricle. Occasionally, either of these worms may be found in the arteria pulmonalis.

At least 10 metastrongyloid nematodes are known as lungworms of Pinnipedia. Nine of these belong to the genus *Parafilaroides* and 1 to the genus *Otostrongylus*. Some species

of *Parafilaroides* are lethal parasites of both otariid and phocid pinnipeds. The — less pathogenic — *Otostrongylus* spp. appear to be restricted to phocid hosts.

Parafilaroides gymnurus, the smaller of the 2 lungworms of *Phoca vitulina,* was originally described as *Pseudalius gymnurus* by Railliet (1899). Baylis and Daubney (1925) transferred it to their new genus *Halocercus,* which otherwise contained only lungworms of cetaceans. Dougherty (1946) recognized that the affinities of *H. (Pseudalius) gymnurus* are actually with certain metastrongylids of terrestrial mammals rather than with those of odontocete cetaceans, and assigned it to his new genus *Parafilaroides.* Males of *P. gymnurus* are about 15 to 18 mm long and 120 μm in diameter, females 22 to 23 mm long and 170 μm in diameter (Railliet, 1899).

Parafilaroides decorus, described by Dougherty and Herman (1947), is a common lungworm of *Zalophus californianus* (Dailey, 1970; Dailey and Hill, 1970; Schroeder and co-authors, 1973; Ashizawa and co-authors, 1978; Geraci, 1978b). Males are 6 to 7 mm long and 92 μm wide, females 16 to 21 mm long and 165 μm wide (Dougherty and Herman, 1947). The latter authors described further species, *P. nanus* and *P. prolificus,* from *Eumetopias jubatus. P. kraschenninnikovi* has been reported from *Pusa hispida* and *Phoca largha, P. arcticus* from *Pusa hispida,* and *P. caspicus* from *Pusa caspica* (Delyamure and Alekseiev, 1966; Kurochkin, 1975; Popov, 1975b; Treshchev and Yurakhno, 1975; Delyamure and co-authors, 1976; Goltsev and co-authors, 1978). On the southern hemisphere, *P. hydrurgae* has been described from *Hydrurga leptonyx* (Mawson, 1953).

As shown experimentally by Dailey (1970), the life cycle of *Parafilaroides decorus* involves a small fish, the opaleye *Girella nigricans,* as intermediate host. No other hosts are required; 8 species of molluscs and 1 species of copepod from splash pools contaminated by sea-lion faeces at the San Nicolas Island (California) sea-lion rookery were negative for stages of *P. decorus.* Individuals of *G. nigricans,* fed sea-lion excrement containing first-stage larvae, yielded successive developmental stages of the nematode (Figs 5-30 and 5-31). The first moult occurred in the teleost's intestinal mucosa and submucosa 12 to 15 days post-infestation; the second moult occurred in the intestinal serosa and mesenteric adipose tissue 25 to 36 days post-infestation. Infested fish were fed to a young uninfested *Zalophus californianus.* First-stage larvae appeared in its faeces 21 days later. This was the first demonstration of the full life cycle of a member of the Pseudaliidae and the first metastrongyloid life cycle wherein a vertebrate intermediate host was found (Dailey, 1970). The life cycles of the other species of *Parafilaroides* are unknown.

The adults of *Parafilaroides* spp. occur rolled up in tiny knots in the lung parenchyma (Fig. 5-32). The worms, *in situ,* resemble small irregularly coiled or folded white threads and are readily visible to the naked eye (Dougherty and Herman, 1947; Margolis, 1956; Ashizawa and co-authors, 1978). Massive infestation may lead to the formation of verminous granulomas. Mature females may have their uteri filled with hundreds of tightly packed embryonated eggs. The freely motile first-stage larvae hatching from the eggs are carried up the airways, swallowed, and passed out in the faeces. The respiratory epithelium of pinnipeds is rich in goblet cells, which actively secrete mucus in response to mechanical irritation. The minuscule nematodes induce the cells that line the trachea and bronchi to secrete such large amounts of mucus that the infested individual has difficulty in breathing. Many seals and sea lions, particularly pups, die of asphyxiation, *P. gymnurus* is the most pathogenic parasite of *Phoca vitulina* in the Wadden Sea and is most frequently found in stranded seals (van den Broek and Wensvoort, 1959; van den Broek, 1963; Reijnders and

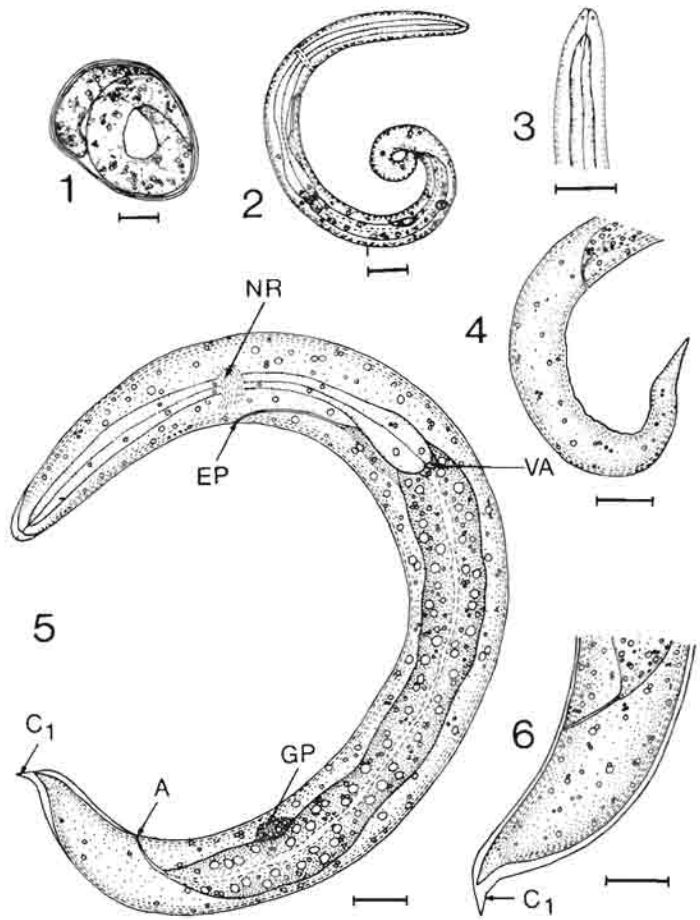

Fig. 5–30: *Parafilaroides decorus*. 1: Egg; 2–4: first-stage larva from bronchial mucus of *Zalophus californianus* (*2* lateral aspect; *3* anterior extremity, dorsal aspect; *4* posterior region showing spike-like tail); 5–6: second-stage larva from *Girella nigricans* (*5* lateral aspect; *6* caudal extremity). *A* anus, $C_{1,2}$ cuticles of first and second stage, *EP* excretory pore, *GP* genital primordium, *LA* lateral alae, *NR* nerve ring, *VA* oesophago-intestinal valve. Bars: 12 μm (1–4), 10 μm (5–6). (After Dailey, 1970.)

co-authors, 1981). Asphyxiation due to heavy *P. decorus* infestation is also the major cause of stranding among young *Zalophus californianus* along the California coast (Geraci, 1978b; Stroud and Dailey, 1978). Within hours following a surge in larvae released by adult worms, or over days of constant irritation, bronchial obstruction may ensue (Sweeney, 1978c). *Parafilaroides* spp. affect mainly young pinnipeds (up to about 2 years of age) and are rare in individuals over 4 years old. *P. gymnurus* may produce chronic infestation in adult harbour seals (van den Broek, 1963).

Early clinical signs of *Parafilaroides* infestation include increased respiratory rate associated with a mildly productive cough. As the condition becomes more severe, anorexia and malaise occur. Dyspnoea, typified by flared nares or an open mouth during periods of inspiration, and forced inspiration indicate severe obstruction. On physical

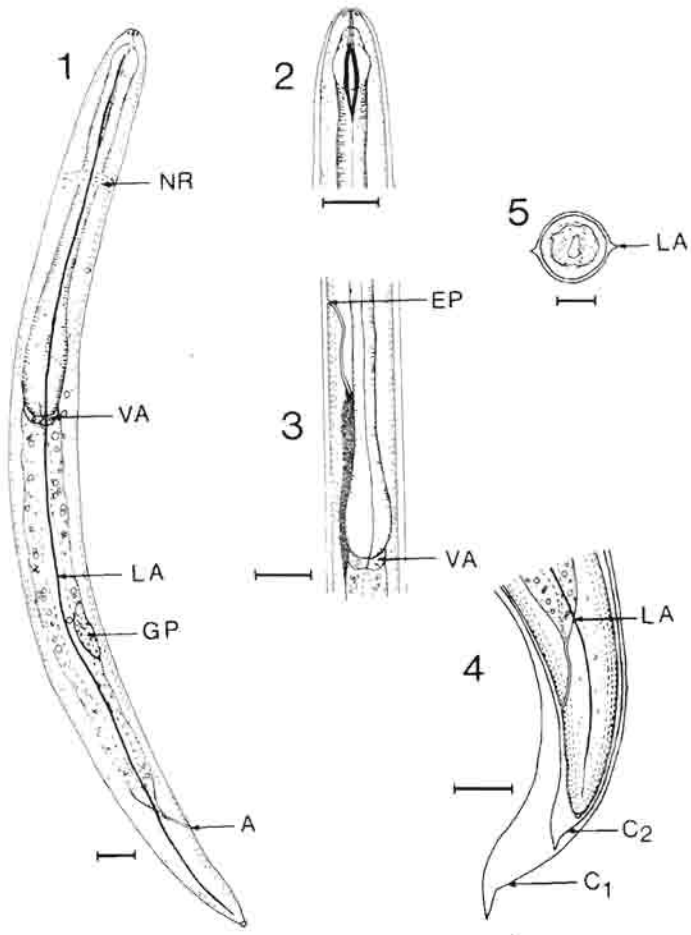

Fig. 5–31: *Parafilaroides decorus*. Third-stage larva from *Girella nigricans*. 1: Lateral aspect; 2: cephalic region, dorsal view; 3: posterior portion of oesophagus with excretory pore, duct and excretory sinus; 4: caudal region, lateral aspect; 5: cross section. For abbreviations see Fig. 5–30. Bars: 10 μm. (After Dailey, 1970.)

examination rales and wheezing sounds are easily auscultated over the thorax. Occasionally, no sounds are heard, suggesting total occlusion of the bronchi. The animal's mucous membranes are often pale and cyanotic. The combination of clinical signs, the finding of first-stage larvae in stool or sputum specimens, together with the absence of haematological evidence of systemic bacterial or fungal infection supports the diagnosis (Sweeney, 1978c).

Histologic examination of the affected lung reveals adult larviparous nematodes together with larvae, lymphocytes, macrophages, proteinaceous fluid and neutrophils in the alveolar spaces and bronchi. The lungs are markedly oedematous and the bronchi contain mucinous froth (Fig. 5-33). Inflammatory reactions presumably occur in response to the larvae because comparable conditions are not observed in lung tissue containing only adult worms. Pneumonia and mucoid bronchiolar obstruction, common grossly identifiable sequelae to lungworm infestation, are apparent in almost all cases. The

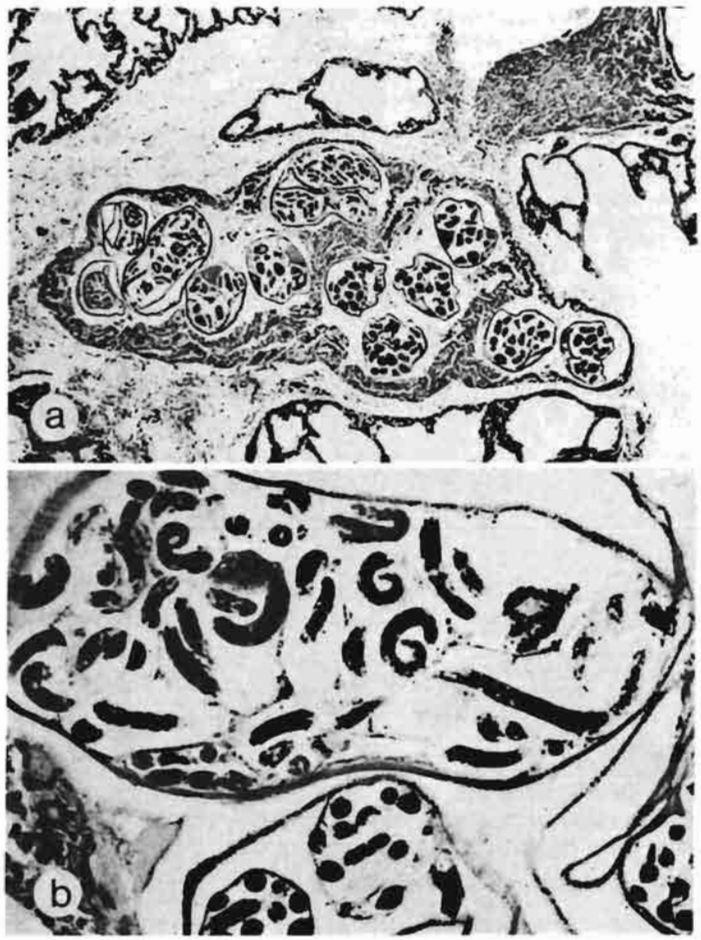

Fig. 5–32: *Parafilaroides decorus.* (a) Well-defined group of female parasites in *Zalophus californianus* lung parenchyma. Note numerous embryos in uteri and slight inflammatory reaction; × 25. (b) Higher magnification (× 390) of (a). (After Morales and Helmboldt, 1971.)

histopathology of *Parafilaroides* infestation has been studied in detail by van den Broek and Wensvoort (1959), Fleischman and Squires (1970), Migaki and co-authors (1971), Morales and Helmboldt (1971), Simpson and Gardner (1972) and Ashizawa and co-authors (1978).

Prevalences of *Parafilaroides* spp. in pinniped populations may be high. Dailey (1970) found 10 of 14 feral *Zalophus californianus* from Point Mugu, San Nicolas Island, and Seal Beach, California, to be infested with *P. decorus,* four of these heavily. Of 43 California sea lions that had stranded on southern California beaches, 33 were infested (Sweeney and Gilmartin, 1974). Van den Broek (1963) reported high *P. gymnurus* prevalences from 43 of 49 *Phoca vitulina* from the Dutch Wadden Sea. In contrast, Popov (1975b) recovered *P. krascheninnikovi* in low numbers (21 to 34) from only 3 of 143 *Phoca largha* from the Sea of Okhotsk.

Treatment for *Parafilaroides* has not been totally successful, and the clinician must

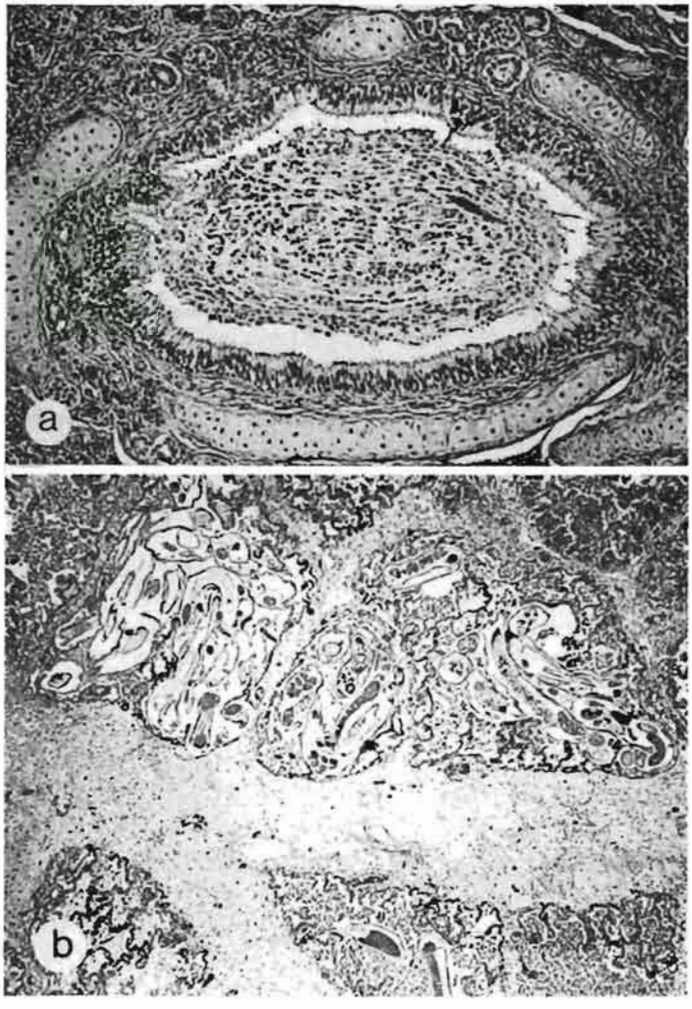

Fig. 5–33: Lung pathology associated with *Parafilaroides decorus* infestation. (a) Bronchus of *Zalophus californianus* with thick fibrinous exudate containing polymorphonuclear cells and a small piece of larva; × 155. (b) Section of sea-lion lung with several groups of nematodes and severe interlobular oedema with fibrinous background; × 25. (After Morales and Helmboldt, 1971.)

decide whether a low level of infestation exists and if treatment is truely indicated considering the potential risk (Wallach, 1972). Information on the longevity of *Parafilaroides* spp. is not available, but in sea lions experimentally infested with *P. decorus*, first-stage larvae were still present in the faeces after 1 year and viable adult worms were still present in large numbers within the pulmonary alveoli. Apparently, the nematode is voided from the host within several years, as very few sea lions over 4 yr of age were found to be infested (Sweeney, 1973).

It appears that at an early stage in the course of a lungworm infestation, the migration of third- and fourth-stage larvae leads to focal pneumonitis characterized by an accumulation of inflammatory cells within the lung parenchyma. Eventually, bacterial infections

occur, leading to microabscess formation and, if the animal lives long enough, into frank abscessation. It is likely that the process begins with focal pneumonitis, but it may be related to the presence of adult worms in the alveoli or possibly to mucus accumulation and resultant ineffective flushing of the bronchiolar villi.

At least 11 different strains of bacteria have been isolated from the lungs of California sea lions suffering from *Parafilaroides decorus* infestation. Of these, *Escherichia coli* and *Klebsiella pneumoniae* were most common. Initial therapy of lungworm disease must, therefore, be directed toward the bacterial component of the disease. Subsequent therapeutic measures must aim at destroying the worm itself and its larvae. No anthelmintic has yet been demonstrated to be completely effective against all stages of the parasite. Levamisole hydrochloride (oral) and Levamisole dihydrogen phosphate (parenteral) have a definite larvicidal effect and probably a partial effect upon the adult worms. Sudden deaths of pinnipeds treated for lungworms may be due to an acute anaphylactic reaction in response to massive worm-die off (Scott, 1971; Wallach, 1972; Sweeney, 1973, 1974a).

Otostrongylus circumlitus is the 'large lungworm' of phocid pinnipeds in circumboreal waters. Railliet (1899) originally placed it in the genus *Strongylus,* but de Bruyn (1933) transferred it to her new genus *Otostrongylus. O. circumlitus* is a common lungworm of *Phoca vitulina* in European waters (Mohr, 1952; van den Broek, 1963; Menschel and co-authors, 1966; Sprehn, 1966a,b; Zimmermann and Nebel, 1975; Clausen, 1978; Reijnders and co-authors, 1981; and others). Sweeney (1973, 1978c), Dunn and Wolke (1976a,b) and Geraci (1978b) recorded it in the same host species from the North American Atlantic and Pacific coasts. Other records are from *Pusa hispida, Histriophoca fasciata, Mirounga angustirostris* and *Erignathus barbatus* from North Pacific and Arctic waters (Delyamure, 1955; Popov, 1975d; Treshchev and Yurakhno, 1975; Yurakhno and Treshchev, 1975; Stroud and Dailey, 1978). *Kutassicaulus andreewoi,* described by Skrjabin (1933) from Okhotsk ringed seals *Pusa hispida ochotensis,* was recognized by Schuurmans Stekhoven (1935) as a species of *Otostrongylus.* Delyamure (1955) showed it to be identical with *O. circumlitus.* Similarly, *Otostrongylus* sp., reported by Schroeder and co-authors (1973) from harbour seals in California, may be attributable to the same species.

Fully grown males of *Otostrongylus circumlitus* are about 10.5 cm in length and 1.3 mm in maximum width; the larviparous females are 14 to 16 cm long and 1.8 to 2 mm wide (Railliet, 1899). Its life cycle is unknown but probably involves an invertebrate as intermediate host. *O. circumlitus* inhabits mainly the lungs (bronchi) and trachea (Fig. 5-34), but may also be found in the right ventricle, the pulmonary artery, and even in the blood vessels of the liver. The large worms commonly cause obstructive lung disease merely by their presence in the major airways. Free larvae are present in the airways and are coughed up with the mucus produced in response to the worms. The 'verminous pneumonia', developing upon partial blockage of the trachea and bronchi, may have a bacteriologic component as in *Parafilaroides* infestations (see above).

Although generally considered less pathogenic than *Parafilaroides* spp., *Otostrongylus circumlitus,* when present in large numbers, can cause severe respiratory disturbances (Menschel and co-authors, 1966; Sweeney, 1973, 1974b, 1978c; Geraci, 1978a). *O. circumlitus* caused most damage to *P. vitulina* in Danish waters. Eight of 65 seals examined actually had died of pneumonia caused by lungworms, and 7 other individuals that had been shot were so heavily infested that they would have died anyway (Clausen, 1978). Of

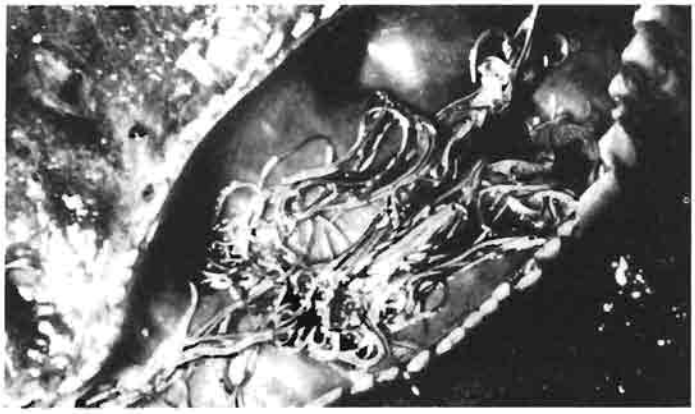

Fig. 5–34: *Phoca vitulina.* Trachea with mass of *Otostrongylus circumlitus.* (After Geraci, 1978b.)

108 harbour seals under 1 yr of age, stranded on the New England (USA) coast, 28 % carried *O. circumlitus* (Geraci, 1978b). Van den Broek and Wensvoort (1959) and van den Broek (1963) examined a total of 73 harbour seals from the Dutch Wadden Sea. Of these, 56 were infested with large lungworms. Seventeen had *O. circumlitus* in the lungs and 6 had worms also in the heart. In 3 cases, more than 80 adult nematodes were recovered from young hosts. Severe vasculitis and bronchopneumonia, associated with the presence of large adult *O. circumlitus,* was believed to have caused the death of a yearling northern elephant seal from Oregon waters (Stroud and Dailey, 1978).

Diagnosis of *Otostrongylus circumlitus* infestation may be made from finding larval worms in sputum or faeces. Clausen (1978) found larvae in faeces or lung-tissue smears of 30 of 65 *P. vitulina* examined from the Danish North Sea, Kattegat and Limfjord areas. Animals from the Wadden Sea were most heavily infested, 70 % being positive for larvae. No specific therapy for *O. circumlitus* infestation has been developed, but drugs used in the treatment of *Parafilaroides* infestation might be effective.

The filarioid 'heartworm' *Dipetalonema spirocauda* affects a considerable number of pinniped species. It is a common parasite of *Phoca vitulina* throughout the seal's distributional range and has been found in both feral and captive individuals (Railliet, 1899; Wülker, 1930; Mohr, 1952; Anderson, 1959; van den Broek and Wensvoort, 1959; Brown and co-authors, 1960; Taylor and co-authors, 1961; King, 1964; Sprehn, 1966a,b; Nakamura and Mikami, 1967; Schroeder and co-authors, 1973; Dunn and Wolke, 1976a,b; Machida, 1977). Delyamure and Popov (1974), Popov (1975b,c,d), Treshchev and Yurakhno (1975), Delyamure and co-authors (1976) and Goltsev and co-authors (1978) reported it from *Phoca largha, Pusa hispida, Erignathus barbatus* and *Histriophoca fasciata* in the Bering Sea and the Sea of Okhotsk; Delyamure and Treshchev (1966), Helle and Blix (1973) and Pouvreau and co-authors (1980) from *Cystophora cristata* in the Greenland Sea and from the French Atlantic coast, respectively; and Brown and co-authors (1960), Taylor and co-authors (1961) and MacDonald and Gilchrist (1969) from *Zalophus californianus.*

The first accounts of the occurrence of *Dipetalonema spirocauda* in *Phoca vitulina* are those by Leidy (1858), who described it under the name *Filaria spirocauda,* and by Joly (1858), who designated it *Filaria cordis phocae.* Railliet and Henry (1911) transferred it,

together with other filarioids, to the newly erected genus *Dipetalonema*. Lubimov (1927) renamed the worm *Skrjabinaria spirocauda* (a designation frequently encountered in the literature) but, according to Anderson (1959), the Russian author's worms were no filaroids at all. Kreis' (1953) *Skrjabinaria heteromorpha*, described from a captive *P. vitulina*, is identical with *D. spirocauda*. Some of the older reports of filarioid heartworms of pinnipeds possibly include other species of *Dipetalonema* or even *Dirofilaria*. In a more recent study, Anderson (1959) elaborated the taxonomy of *D. spirocauda*. The author states that the descriptions of Joly (1858) and Leidy (1858) are so general that they could apply to either *Dipetalonema spirocauda* (as determined by Lubimov's, 1927, description of the male) or *Dirofilaria immitis* (see below).

Adult females of *Dipetalonema spirocauda*, 163 to 170 mm long and 0.64 to 0.76 mm wide, as well as males, 90 to 92 mm long and 0.34 to 0.44 mm wide (Machida, 1977), parasitize in the right ventricle (Fig. 5-35). Microfilariae, 235 ± 10 μm long and 5.4 μm wide (Fig. 5-36; Taylor and co-authors, 1961) circulate in the blood. Measurements given for *D. spirocauda* by other authors show minor differences.

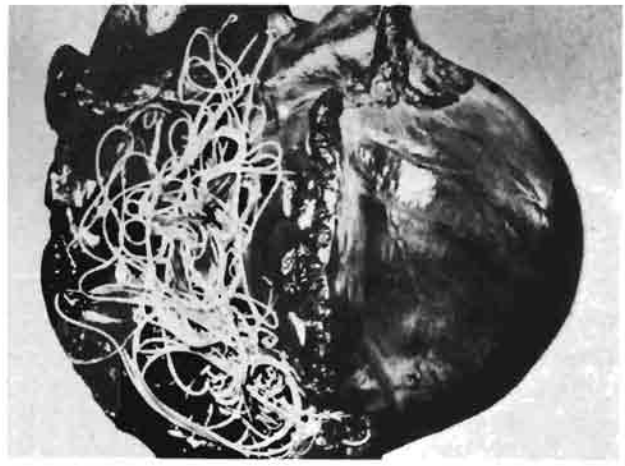

Fig. 5–35: *Dipetalonema spirocauda*. Adult worms in heart of *Phoca vitulina*. (After Geraci, 1978b.)

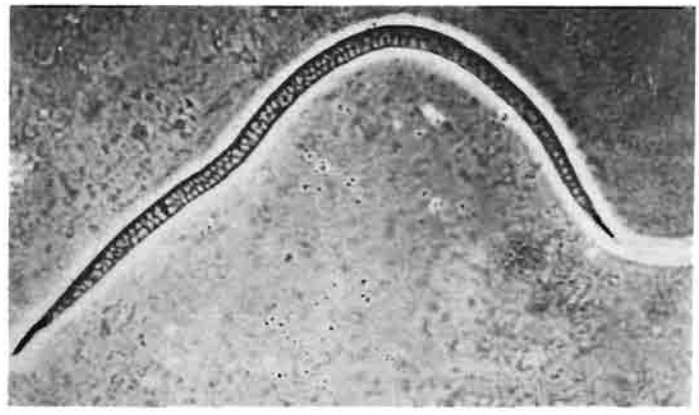

Fig. 5–36: *Dipetalonema spirocauda*. Microfilaria from peripheral blood of *Zalophus californianus*. (After Taylor and co-authors, 1961.)

Perry (1967) described a second filarioid, *Dipetalonema odendhali,* from *Zalophus californianus.* Adult worms were found in the intermuscular fascia of 4 hosts and at additional sites, but never in the heart. Males were 46 to 64 mm, females 100 to 150 mm in length, i.e., distinctly smaller than *D. spirocauda.* Microfilariae, 231 to 249 μm in length, were seen in formalinized blood. Perry and Forrester (1971) and Forrester and co-authors (1973) refound *D. odenthali* in feral northern fur seals and in captive California sea lions, respectively. Machida (1969) reported filarioids from the subfascia of the cervical and thoracic region of feral Japanese *Callorhinus ursinus* as *D. spirocauda,* but later (Machida, 1977) assigned them to *D. odendhali.* Kagei and Oda (1975) detected the same parasite in captive northern fur seals in Japan.

Probably, further — yet unrecognized — species of filarioid worms exist in pinnipeds. Unidentified microfilariae, 245 to 263 μm long and 3.5 to 5.3 μm wide, have been seen in the blood of a captive Steller sea lion, and larger (280 ± 12 × 5.4 μm) forms (probably attributable to *Dirofilaria immitis;* see below) occurred in the blood of a captive California sea lion (Taylor and co-authors, 1961; Perry, 1967). Apparently, *Dipetalonema* spp. have not yet been reported from pinnipeds on the southern hemisphere.

Experimental infestation, which could be helpful in the solution of open questions (e.g., morphological variation of a given species of *Dipetalonema* in relation to host species, etc.), are largely precluded by the fact that the mode of transmission of these filarioids to pinnipeds remains an enigma. Wülker (1929, 1930) raised the question whether lice of the genus *Echinophthirius,* which abound on the skin of *Phoca vitulina,* may serve as intermediate hosts, but found no microfilaria in lice. Mohr (1952) maintains, without supporting evidence, that microfilariae encountered in the blood of pinnipeds are transmitted by lice. Taylor and co-authors (1961) and Dunn and Wolke (1976b) examined large numbers of *E. horridus* taken from seals but found no life-cycle stages of *D. spirocauda* or any other filarioid. The authors feel that, as mosquitoes and simuliids are frequently present in large numbers in haul-out areas, these insects should be examined more closely for their possible role in the transmission of heartworms.

Transplacental transmission or infestation of pups during lactation would be other possible routes of pathogen transfer but have, apparently, not yet been considered in detail. *Dipetalonema spirocauda* infestation can occur at a very early age, as evidenced by reports of the detection of heartworms in the cardiovascular system of harbour seals estimated to be less than 1 month of age, suggesting transplacental transmission (Sweeney, 1974a, 1978c).

Heartworm infestations are a constant finding in pinniped populations. Of 48 *Phoca vitulina,* examined by van den Broek (1963) from Dutch waters, 31 had *D. spirocauda.* One host harboured as many as 230 adult worms. Counts of *D. spirocauda* taken from the heart of *P. largha* in the Bering Sea ranged from 1 to 53 (Delyamure and co-authors, 1976). Clausen (1978) diagnosed heartworm infestation in 16 of 65 common seals from Danish waters. Three common seals stranded on Rhode Island and Connecticut (USA) shores all had *D. spirocauda* and, apparently, had succumbed to their heavy parasite burdens. Of 5 captive seals, 4 were infested (Dunn and Wolke, 1976a,b).

Clinical signs of filariosis are listlessness, anorexia, dyspnoea, coughing and erratic breathing patterns. The caretaker will report that the patient is short of breath after only minimal exercise. Diagnosis is made by finding microfilariae in blood smears. The pathology associated with *Dipetalonema spirocauda* infestation is considerable. The right

ventricle, pulmonary arteries and venae cavae may contain large numbers of adult worms, in some instances nearly occluding these structures. The right side of the heart may show pronounced dilatation and hypertrophy. There may also be an accompanying diffuse pneumonitis. Gross lesions, attributable to either adult worms or microfilariae, are observable in the lungs, liver, kidney, spleen and vascular system. Microfilariae can be seen within the lumen of vessels, as well as in the hepatic and spleenic parenchyma. Hepatic lesions vary from foci of acute eosinophilic necrosis to chronic focal granulomas with foreign-body giant cells. Microfilaria are often present in acute lesions. Many of the histological findings would suggest a septicaemia or toxaemia. Bacterial lung infections range among the sequelae of filariosis. The histopathology of heartworm disease has been studied in detail by Taylor and co-authors (1961), MacDonald and Gilchrist (1969) and Dunn and Wolke (1976a,b). In 3 stranded common seals, studied by the latter authors, the immediate cause of death was determined to be complete occlusion of a branch of the pulmonary artery by a verminous embolus dislodged from the heart.

Clausen (1978) pointed out that heartworms may be an aggravating factor in lungworm *(Otostrongylus)* disease. Of 108 harbour seals stranded on the New England coast (USA), 38 % were infested with *D. spirocauda,* and most of these also had lungworms (Geraci, 1978b).

In contrast to *Dipetalonema spirocauda, D. odendhali* is rather harmless. As stated, it never occurs in the heart but its microfilariae may nevertheless be found in the blood. *D. odendhali* is enzootic in California sea lion herds and its microfilariae are the most common forms encountered in *Zalophus californianus* (Dailey, 1978). According to Sweeney (1973, 1974a), it is common to find up to 50 % of a new arriving group of yearling individuals to be infested with this filarioid. Stroud and Dailey (1978) recovered adult *D. odendhali* from the subcutaneous tissue of the scrotum of 2 California sea lions. Microfilariae were numerous in histologic sections of the mesenteric lymph nodes, liver and an adrenal tumour found in one of the animals.

Dirofilaria immitis is a common heartworm of dogs distantly related to *Dipetalonema spirocauda.* There are several confirmed and some doubtful reports of the occurrence of *D. immitis* in *Zalophus californianus* and *Phoca vitulina.* Some of the doubtful records may be referable to *D. spirocauda* or another, yet unrecognized, filarioid. The somewhat confusing literature has been discussed by Faust (1937), Anderson (1959) and Perry (1967) and will not be considered here in detail.

Dirofilaria immitis has been reported from *Phoca vitulina* (Joest, 1925; Faust, 1937; Bisbocci, 1939; Eriksen, 1962; Larsen, 1962; Sprehn, 1966a,b; Medway and Wieland, 1975) and *Zalophus californianus* (Forrester and co-authors, 1973; White, 1975; Sweeney, 1974a, 1978c). All records are from captive hosts. According to Dailey (1978), *D. immitis* infestation has not been substantiated in wild populations. Transmission of this filarioid is effected by mosquitoes, and its prevalence in pinnipeds may be of significance in areas where dog-heartworm disease is enzootic. Clinical signs of the disease in seals and sea lions, site of the parasite in the host, and the pathology produced are essentially the same as in *Dipetalonema spirocauda.* Severe infestations may be accompanied by loss in body weight, estimated at between 10 and 20 % of the original weight. Carriers may be asymptomatic for years, with the onset of clinical signs occurring only with heavy parasitism or after a sudden increase in work requirements (White, 1975; Sweeney, 1978c).

Differentiation of *Dirofilaria immitis* and *Dipetalonema spirocauda microfilariae* may become of prime importance for the practitioner working in *Dirofilaria*-enzootic areas. Both microfilariae differ in size, those of the former species measuring roughly 285 to 290 μm long by 5.5 μm wide, and those of the latter 225 to 250 μm long and 4.4 μm wide (Forrester and co-authors, 1973; Sweeney, 1973, 1974a). Wallach (1972) points out that both forms also differ morphologically. When viewed in formalinized, methylene blue-stained preparations, *D. immitis* microfilariae show a tapering, well-stained anterior end and a straight-tapering posterior end, while those of *D. spirocauda* have a blunt, lightly stained anterior end and a hook-shaped, blunt and poorly stained posterior end.

With the more recent widespread distribution of *Dirofilaria immitis* in the canine population, this parasite may well become a more common disease problem in California sea lion exhibits. Infestation of captive sea lions has been successfully prevented by daily routine administration of diethylcarbamazine citrate at a rate of 5 mg kg^{-1} of body weight (White, 1975; Beusse and co-authors, 1977). Treatment for adult *Dipetalonema* spp. consists of oral administration of a 1 % solution of thiacentarsamide IV twice each day at a rate of 0.2 ml kg^{-1} of body weight for 2 days. Antihistamines or prednisones may be given if a reaction to the dying filarioids occurs. Treatment for microfilariae consists of administering dithiazine iodide orally for 7 to 10 days at a rate of 10 mg kg^{-1} of body weight. Treatment for microfilariae is only effective after the adult worms have been destroyed (Wallach, 1972).

There are a few scattered reports on the occurrence of *Trichinella spiralis* in pinnipeds. This aphasmidian (adenophorean) nematode is normally a tissue parasite of terrestrial mammals and man. The existence of a natural cycle of trichinosis in the Arctic has long been recognized (Thorborg and co-authors, 1948; Rausch and co-authors, 1956; Myers, 1970; Rausch, 1970; Zimmermann, 1970). Britov (1962) designated the Arctic form *Trichinella nativa*. The major possible participants in the marine cycle are the polar bear *Thalarctos maritimus*, the bearded seal *Erignathus barbatus* and the walrus *Odobenus rosmarus*. Adult *T. spiralis* have been recovered from the intestine of *E. barbatus* and the encapsulated larvae from *O. rosmarus* (Roth, 1950; Rausch and co-authors, 1956; Madsen, 1961; Sprehn, 1966a; Fay, 1968; Gould, 1970; Rausch, 1970; Yurakhno and Treshchev, 1975; Thing and co-authors, 1976).

Relatively high prevalences of *Trichinella spiralis* larvae in walruses throughout the Arctic indicate that infestations are not purely accidental. The highest incidence of trichinosis (17 out of 394 individuals positive = 4.3 %) was reported from *Odobenus rosmarus* in East Canada. Values reported in the literature for North Norway were 9 % of 74, for Alaska 1 % of 104, for Greenland 1 % of 489, and for the USSR 0.6 % of 145 (Gould, 1970). Madsen (1961) found 2 % of 271 walruses from Egedesminde, Greenland, to be positive. A 'walrus disease' known for many years to occur among the local population in Greenland was verified as trichinosis (Roth, 1950). Thing and co-authors (1976) detected 1 to 2 *T. spiralis* larvae per gram of digested muscle tissue in walruses from the Thule, Greenland, district.

As to the mode of infestation of marine hosts with these terrestrial-mammal parasites, various hypotheses have been put forward. Rausch and co-authors (1956) and Fay (1960) suggested that transmission of *Trichinella* larvae from a trichinous source to a pinniped may occur *via* marine crustaceans. Eventually, Fay (1968) established light infestation in a dog by feeding it amphipods *Anonyx* sp., which had previously been fed trichinous bear

meat. Hulebak (1980) demonstrated that various species of amphipods would ingest trichinous rat muscle and hold contained *T. spiralis* larvae, apparently undigested, for as long as 28 h. Thus, mechanical transmission of larval *T. spiralis* to marine mammals *via* marine crustaceans appears likely.

Zablotzky (1971) described adult trichinelloideans *Capillaria delamurei* from the small intestine of *Pusa caspica*. If his generic identification is correct, this is the first record of a capillariid from a pinniped host.

Agents: Acanthocephala

Numerous acanthocephalans or 'thorny-headed worms' — members of the genera *Corynosoma* and *Bolbosoma* — have been reported from pinnipeds but, after correction for synonyms and doubtful records, some 16 (14 valid and 2 dubious) species of *Corynosoma* remain. The genus *Bolbosoma* is well represented in Cetacea (p. 840); its occasional occurrence in Pinnipedia appears to be fortuitous. Of the 16 pinniped-invading *Corynosoma* spp., 12 occur in hosts from the northern hemisphere and only 4 in hosts from the southern hemisphere. As shown in Table 5-4, the 2 circumpolar pinniped faunae have no species of thorny-headed worm in common. On the northern hemisphere, *C. strumosum* is the most widely distributed species, invading at least 13 pinniped species and subspecies. It is followed by *C. semerme* (9 host species), *C. validum* (7 host species), and *C. villosum* and *C. wegeneri* (5 host species each). On the southern hemisphere, *C. australe* and *C. hamanni* have been reported from 4 pinniped species each, *C. bullosum* from 3 host species. *C. falcatum, C. similis, C. obtuscens, C. reductum* and *C. rauschi* (all from northern hemisphere), as well as *C. sipho* (Antarctic), have, thus far, been recovered from single host species.

Only 1 species, *Corynosoma semerme,* has been reported from both Arctic (Table 5–4) and Antarctic waters. Johnston and Edmonds (1953) list it from *Otaria (= Phocarctos) hookeri* at Campbell and Auckland Islands (Southern Ocean), but Golvan (1959) regards the latter record as involving a separate species. Acanthocephala have, thus far, been reported from all but the following pinnipeds: the Baikal seal *Pusa sibirica,* the Mediterranean monk seal *Monachus monachus,* and the Caribbean monk seal *M. tropicalis. C. strumosum* and *C. villosum* also parasitize sea otters (p. 666); several species *(C. strumosum, C. semerme, C. similis, C. wegeneri)* occur in cetaceans (Neiland, 1962; Margolis and Dailey, 1972). Acanthocephala in general display little if any host specificity at the specific, generic or familial levels (Petrochenko, 1956). However, in birds, which have been identified as accidental carriers of *Corynosoma* spp., these acanthocephalans show arrested development (p. 628; Meyer, 1931; Nuorteva, 1965; Schmidt, 1965).

The pinniped-infesting species of *Corynosoma* listed in Table 5-4 very probably include one or several synonyms. Particularly the common *C. strumosum* has been listed under a variety of specific names, i.e., *hystrix, ventricosus, gibbosus, striatus, incrassatus, carchariae,* etc. (Golvan, 1959). To these may have to be added *osmeri* and *ambispinigerum* (Harada, 1935; Lincicome, 1943). Several authors (e.g., Fisher, 1952) have confounded *C. strumosum* and *C. semerme. C. sipho* may be a synonym of *C. hamanni, C. hadweni* almost certainly a synonym of *C. wegeneri* (Margolis, 1955; Edmonds, 1957; Golvan, 1959). Some confusion also exists in the literature with respect to the identification of the pinniped hosts (see p. 683 and Table 5-4).

Table 5-4
Some records of *Corynosoma* spp. in Pinnipedia from the northern (N) and southern (S) hemispheres (Original)

Parasite	*C. strumosum* (N)*)	*C. semerme* (N)	*C. validum* (N)	*C. villosum* (N)	*C. wegeneri* (N)	*C. reductum* (N)	*C. falcatum* (N)	*C. magdaleni* (N)	*C. similis* (N)	*C. osmeri* (N)	*C. obtuscens* (N)	*C. rauschi* (T)**)	*C. hamanni* (S)***)	*C. bullosum* (S)	*C. australe* (S)	*C. sipho* (S)
Parasite body length†)	5 to 9 mm	3 mm	5.4 mm	8.4 mm	up to 10 mm	up to 10 mm	7.3 mm	4.5 mm		6 mm	3 mm	4 mm	3.5 mm	up to 16 mm	up to 3.1 mm	up to 6 mm
Otariidae																
Zalophus californianus (N)	*1, 37*									*31*	*4, 31, 37, 61, 62*					
Eumetopias jubatus (N)	*4, 15, 28*	*37*	*61, 62*	*12, 15, 37, 39, 61, 62*												
Callorhinus ursinus (N)	*15, 37, 44*	*37, 62*		*15, 36, 37, 61, 62*					*45*							
Arctocephalus forsteri (S)															*23*	
Arctocephalus pusillus (S)															*5*	
Neophoca cinerea (S)															*25*	
Phocarctos hookeri (S)															*26*	
Odobenidae																
Odobenus rosmarus (N)		*40, 41, 42, 56, 57*	*7, 15, 37, 61, 62*													
Phocidae																
Phoca vitulina (N)	*2, 15, 16, 29, 37, 39, 40, 41, 42, 56, 57, 62* (~1200[2])**)	*40, 41, 42, 56, 57, 62*		*12*	*21, 38, 43, 61, 62*		*61*	*43*		*20*						
Phoca largha (N)	*10, 15, 18, 49, 50, 51* (5700[10])	*10, 15, 18, 49, 50, 51* (70[49])	*10, 50* (5[10])	*8, 18* (4[8])	*10, 18* (17[10])											
Pusa hispida (N)	*6, 10, 15, 21, 33, 34, 40, 41, 42, 48, 51, 59, 60* (517[10])	*6, 10, 15, 17, 22, 34, 48, 51, 59, 60, 62* (450[51])	*6, 15, 59, 60*	*8, 60*	*10, 15, 21, 60* (33[10])	*32, 33, 56, 59, 60, 62*										
Pusa caspica (N)	*11, 58*															
Halichoerus grypus (N)	*12, 37* (100[11])	*40, 41, 42, 43, 56, 57*			*37, 43, 61, 62*		*61, 62*	*43*								
Mirounga angustirostris (N)														*55*		
Histriophoca fasciata (N)	*10, 15, 52, 53* (177[52])	*10, 15, 52, 53* (9[52])	*10, 15, 52* (21[52])	*8, 12, 52*												
Pagophilus groenlandicus (N)		*40, 41, 42, 56, 57*														
Erignathus barbatus (N)	*8, 10, 35, 51, 53* (39[10])	*61, 62, 63* (14[51])	*8, 10, 15, 27, 51, 63* (530[10])	*8, 63* (18[8])	*38, 61, 62, 63*											
Cystophora cristata (N)	*9*															
Mirounga leonina (S)														*13, 14, 26, 32, 47*		
Hydrurga leptonyx (S)													*32, 46*	*13*	*26*	
Lobodon carcinophagus (S)													*46*	*3, 32*		
Leptonychotes weddelli (S)													*14, 24, 30, 46*			*54*
Monachus schauinslandi (T)												*19*				

Sources: *1* Ball (1928), *2* Ball (1930), *3* Baylis (1929), *4* Dailey and Hill (1970), *5* Delyamure and Parukhin (1968), *6* Delyamure and Popov (1974), *7* Delyamure and Popov (1975a), *8* Delyamure and Popov (1975b), *9* Delyamure and Treshchev (1966), *10* Delyamure and co-authors (1976), *11* Dogiel and co-authors (1963), *12* Duncan (1956), *13* Edmonds (1955), *14* Edmonds (1957), *15* Fay and co-authors (1978), *16* Fisher (1952), *17* Forssell (1904), *18* Goltsev and co-authors (1978), *19* Golvan (1959), *20* Harada (1935), *21* Heinze (1934), *22* Helle and Valtonen (1981), *23* Johnston (1937a), *24* Johnston and Best (1937), *25* Johnston and Best (1942), *26* Johnston and Edmonds (1953), *27* Kenyon (1962), *28* Krotov and Delyamure (1952), *29* Kruidenier (1954), *30* Leiper and Atkinson (1914), *31* Lincicome (1943), *32* von Linstow (1892), *33* von Linstow (1905b), *34* Lundström (1942), *35* Lyster (1940), *36* Machida (1969), *37* Margolis (1954), *38* Margolis (1955), *39* Margolis (1956), *40* Meyer (1931), *41* Meyer (1932), *42* Mohr (1952), *43* Montreuil (1958), *44* Neiland (1961), *45* Neiland (1962), *46* Nickol and Holloway (1968), *47* Nikolskii (1974a), *48* Nuorteva (1965), *49* Popov (1975a), *50* Popov (1975b), *51* Popov (1975c), *52* Popov (1975d), *53* Popov (1976), *54* Railliet and Henry (1907), *55* Schmidt and Dailey (1971), *56* Sprehn (1966a), *57* Sprehn (1966b), *58* Stschupakov (1936), *59* Treshchev and Popov (1975), *60* Treshchev and Yurakhno (1975), *61* Van Cleave (1953a), *62* Van Cleave (1953b), *63* Yurakhno and Treshchev (1975).

*) Host/parasite records from northern hemisphere. – **) Host/parasite records from temperate/tropical waters. – ***) Host/parasite records from southern hemisphere. – †) Parasite-body lengths reported in literature. Pathology produced by different species may be related to parasite body size. – ††) In parentheses: maximum number of individuals found in single hosts; source.

With a single exception, the life cycles of the above Acanthocephala are unknown. In the low-salinity areas of the northern Baltic Sea, as well as in the freshwater of the Finnish lake district, amphipods *Pontoporeia affinis* — which survived there as a 'glacial relict' (Ekman, 1953) — act as intermediate hosts for *Corynosoma strumosum* and *C. semerme*. In that area, *Pusa hispida* is the sole final host of these acanthocephalans (Nuorteva, 1965). In the Caspian Sea, which has a similar geological history, *C. strumosum* occurs as larva in *P. affinis microphthalma* and as adult in *Pusa caspica* (Dogiel and co-authors, 1963). Since, in the Arctic and Subarctic, *P. affinis* is restricted to brackish water (Ekman, 1953), both acanthocephalans must utilize other (yet unknown) crustacean intermediate hosts in the more saline areas of their distributional range in the Arctic Ocean.

Cystacanths of *Corynosoma* spp. occur abundantly in numerous species of fish. Only a few have been identified to species, i.e., *C. strumosum* and *C. semerme* (Meyer, 1931, 1932; Lundström, 1942; Ward and Winter, 1952; Dollfus, 1953; Van Cleave, 1953b; Margolis, 1958, 1963; Moles, 1982), *C. villosum* (Margolis, 1963; Arthur and Arai, 1980), *C. magdaleni* (Montreuil, 1958), *C. similis* (Neiland, 1962), *C. obtuscens* (Ward and Winter, 1952; Van Cleave, 1953a) and *C. wegeneri* (as *C. hadweni;* Van Cleave, 1953b).

On the southern hemisphere, *Corynosoma hamanni* (syn. *C. antarcticum*) has been reported from Antarctic Weddell seals *Leptonychotes weddelli* (von Linstow, 1892; Leiper and Atkinson, 1914). Its cystacanths occur, together with those of *C. bullosum,* in a considerable number of fish species (Baylis, 1929; Edmonds, 1955; Nickol and Holloway, 1968; Holloway and Nickol, 1970; Zdzitowiecki, 1978).

The role of fish hosts in the life cycle of *Corynosoma* is not quite clear. Several authors (Nybelin, 1924; Petrochenko, 1956; Golvan, 1959) maintain that fish are merely paratenic hosts for these acanthocephalans, and that pinnipeds can acquire infestations by devouring amphipods harbouring juvenile worms. With regard to the sometimes considerable incidences and intensities of *Corynosoma* infestation (see below), direct transmission appears unlikely.

There are relatively few quantitative data on the prevalence of *Corynosoma* spp. in pinnipeds. According to Bonner (1972), *C. strumosum* is "regularly" found in the intestine of *Phoca vitulina* and *Halichoerus grypus* from European waters. Helle and Valtonen (1980, 1981) found *C. strumosum* and *C. semerme* in almost all of 42 *Pusa hispida* from the Bothnian Bay (Baltic Sea). High (up to 100 %) *C. strumosum, C. semerme* and *C. villosum* infestation incidences have been observed in Alaskan pinnipeds (Fay and co-authors, 1978; Table 5-5).

One grey seal *Halichoerus grypus* from the Gulf of St. Lawrence was found to harbour 129 male and 195 female *Corynosoma magdaleni* (about 4.5 mm in length), while 4 other grey seals and 1 harbour seal had moderate to low infestations. In all cases, the worms were crowded within the last 2 m of the small intestine or, exceptionally, in the colon (Montreuil, 1958). Individuals of *Phoca vitulina richardi* from the Canadian Pacific coast carried from 1 to more than 300 *C. strumosum* (Margolis, 1956), and 284 specimens of *C. strumosum* and 28 specimens of *C. semerme* have been recovered from a ringed seal *Pusa hispida* living as a glacial relict in the Finnish lake district (Nuorteva, 1965). The numbers of *C. strumosum* (max. 1,698 host^{-1}) and *C. semerme* (max. 1,230 host^{-1}), found in Baltic Sea *P. hispida,* were believed to exceed any previous record (Helle and Valtonen, 1981; however, see Table 5-4!). In a study on seasonal variation in the parasite burden of

Table 5-5
Frequency of *Corynosoma* infestations in Alaskan pinnipeds (After Fay and co-authors, 1978)

Host species	Number of hosts examined	Number of hosts infested with *C. strumosum*	*C. semerme*	*C. villosum*	*C. validum*	*C. wegeneri*
Eumetopias jubatus (Gulf of Alaska)	42	36	–	42	–	–
Eumetopias jubatus (Bering Sea)	7	–	–	7	–	–
Odobenus rosmarus	1	–	–	–	1	–
Erignathus barbatus	8	–	–	–	8	–
Phoca vitulina (Bering Sea)	2	1	–	–	–	–
Phoca vitulina (Gulf of Alaska)	11	11	–	7	–	–
Phoca largha	31	29	27	–	–	–
Histriophoca fasciata	19	17	7	4	2	–
Pusa hispida	15	13	12	–	4	1

Baltic seals, the authors found the ratio of *C. strumosum: C. semerme* to be 1 : 6.6 in spring and 1 : 2.1 in autumn (Table 5-6). Examination of sculpins *Myoxocephalus* spp., the most important food items of ringed seals in the Baltic Sea, revealed high cystacanth prevalences in these teleosts. Incidences of infestation with juvenile *C. strumosum* and *C. semerme* averaged 29 to 13 %, respectively, in *M. quadricornis* from inshore areas and 84 and 33 %, respectively, in four-horned sculpins from offshore areas. Respective figures for the prevalence of both acanthocephalan species in short-horned sculpins *M. scorpius* from the open sea were 100 and 96 % (Helle and Valtonen, 1981).

A similar mass occurrence of juvenile worms in fish has been reported from the Antarctic for *Corynosoma hamanni* (Nickol and Holloway, 1968). Zdzitowiecki (1978)

Table 5-6
Pusa hispida. Seasonal prevalence of acanthocephalans *Corynosoma strumosum* and *C. semerme* (mean number and range of worms host^{-1}) in seals from Bothnian Bay, Baltic Sea (After Helle and Valtonen, 1981)

	Spring (Apr/May)	Autumn (Oct/Nov)
C. strumosum		
Mean	76	66
Range	9–313	1–324
S.D. (N)	79 (13)	83 (28)
C. semerme		
Mean	504	136
Range	90–1,698	1–1,230
S.D. (N)	443 (13)	234 (28)

recovered up to 456 *C. hamanni* cystacanths from individual South Georgian and South Shetland Islands ice fish *Notothenia rossi*. Evidently, seals can accumulate massive acanthocephalan burdens by devouring only a few highly infested fish.

Opinions regarding the pathogenicity of acanthocephalans in homoeothermic marine hosts are not unequivocal (pp. 668 and 841). The magnitude of damage inflicted may depend on the parasite species involved, and may vary with the body size relation of host and parasite. It appears likely that large acanthocephalans in small hosts produce more severe pathology than *vice versa*. As an example, the smaller (~ 3 mm) *Corynosoma semerme* is far less pathogenic to mink *Mustela vison* than the larger (5 to 9 mm) *C. strumosum* (Nuorteva, 1966). For comparison, the maximum body lengths of the pinniped-infesting acanthocephalans, as given by Golvan (1959), have been listed in Table 5-4. Several species display sex dimorphism, the females being larger than the males. Females penetrate the host's intestinal wall more deeply than males and are more pathogenic.

Massive acanthocephalan infestation was found to result in a significant amount of necrosis of the intestinal wall in *Phoca largha* from Sakhalin, USSR (Popov, 1975a). Pinnipeds may acquire acanthocephalans at a very early age, and infestations may be particularly detrimental to such young hosts. Ball (1928) recovered 1,154 individuals of *Corynosoma strumosum* from the intestine of a 90-cm long Californian harbour seal *Phoca vitulina richardi*. The seal still had the umbilical cord present and was believed to have been about 3 weeks old. It was obviously weak and ill at the time of capture, presumably as a result of the exceptionally high infestation intensity. The presence of ripe eggs in some of the *C. strumosum* indicated that a period of about 3 weeks or less is necessary for the worms to reach maturity.

There are a few scattered records of the occurrence of *Bolbosoma* spp. in Pinnipedia. The most frequently encountered species are *B. nipponicum* (Delyamure, 1955; King, 1964; Shustov, 1969; Dailey and Brownell, 1972; Delyamure and Yurakhno, 1974; Dailey, 1975b; Delyamure and co-authors, 1976; Popov, 1976) and *B. bobrovoi* (Krotov and Delyamure, 1952; Dailey and Brownell, 1972; Dailey, 1975b). *Bolbosoma* found by Machida (1969) and Fay and co-authors (1978) in seals and sea lions from Japanese and Alaskan waters have not been identified to species. As stated above, infestation of pinnipeds with these cetacean parasites is merely accidental.

Agents: Insecta

Sucking lice — members of the order Anoplura — are common ectoparasites of Pinnipedia. Eight species (in 4 genera) have been described from this host group, but a large number of synonyms exist (listed by Ferris, 1934, 1951, in his comprehensive monographs).

Echinophthirius horridus (syn. *setosus, phocae, sericans*) is restricted to phocid seals. It has most frequently been reported from *Phoca vitulina* and less frequently from *Pusa hispida, Pagophilus groenlandicus, Erignathus barbatus, Halichoerus grypus* and *Cystophora cristata*. *Antarctophthirus microchir* is common to Otariidae in both the northern and southern hemispheres (Table 5-7).

The pinniped-invading sucking lice are fairly uniform in size, the females (range: 2.75 to 3.5 mm) being slightly larger than the males (range: 2.5 to 3 mm). The insects go

Table 5-7
Occurrence (×) of sucking lice (order Anoplura) on Pinnipedia (compiled from the sources indicated)

Louse species	*Echinophthirius horridus*	*Proechinophthirus fluctus*	*Antarctophthirus trichechi*	*Antarctophthirus microchir*	*Antarctophthirus callorhini*	*Antarctophthirus ogmorhini*	*Antarctophthirus lobodontis*	*Lepidophthirus macrorhini*	Sources
Otariidae									
Zalophus californianus				×					*10, 11, 12, 13, 20*
Eumetopias jubatus		×		×					*9, 10, 11, 12, 13, 19, 20*
Callorhinus ursinus		×			×				*9, 11, 12, 13, 14, 15, 20, 25*
Phocarctos hookeri				×					*11, 12*
Otaria flavescens				×					*7, 8*
Neophoca cinerea				×					*7, 8*
Odobenidae									
Odobenus rosmarus			×						*3, 11, 12, 20, 21, 28*
Phocidae									
Phoca vitulina	×			×					*1, 2, 4, 5, 7, 10, 11, 12, 16, 18, 20, 21, 26, 27, 28*
Pusa hispida	×								*7, 11, 12, 16, 18, 20, 24*
Halichoerus grypus	×								*11, 12, 16*
Pagophilus groenlandicus	×								*11, 12, 16*
Erignathus barbatus	×								*7, 16*
Cystophora cristata	×								*7, 16*
Hydrurga leptonyx						×			*7, 8, 16*
Leptonychotes weddelli						×			*7, 11, 12, 16, 17, 23*
Mirounga leonina								×	*11, 12, 16, 22*
Lobodon carcinophagus							×		*11, 12, 16*

Sources: *1* Anderson and co-authors (1974); *2* van den Broek and Wensvoort (1959); *3* Brown (1962); *4* Brown and co-authors (1960); *5* Caldwell and Caldwell (1969); *6* Cansdale (1970); *7* Dailey and Brownell (1972); *8* Enderlein (1906); *9* Ewing (1923); *10* Ferris (1916); *11* Ferris (1934); *12* Ferris (1951); *13* Jellison (1952); *14* Kim (1972); *15* Kim (1975); *16* King (1964); *17* Lugg (1966); *18* Luther (1910); *19* Margolis (1956); *20* Margolis and Dailey (1972); *21* Mohr (1952); *22* Murray and Nicholls (1965); *23* Murray and co-authors (1965); *24* Nuorteva (1965); *25* Osborn (1899); *26* Railliet (1899); *27* Reijnders and co-authors (1981); *28* Scherf (1963)

through 3 nymphal stages (Fig. 5-37) before becoming adults, and all instars require blood meals. Various aspects of the morphology and life history of *Echinophthirius horridus* and *Antarctophthirus trichechi* have been described in detail by Scherf (1963). Murray and Nicholls (1965), Murray and co-authors (1965) and Murray (1967) studied the ecology of *Lepidophthirus macrorhini* parasitic on *Mirounga leonina* and of *A. ogmorhini* parasitic on *Leptonychotes weddelli,* respectively (Figs 5-38 and 5-39). Kim (1972, 1975) made a similar study on *A. callorhinus ursinus.*

Approximately 92 % of the northern fur seals examined by Kim (1975) from St. Paul Island, Alaska, were infested with either *Antarctophthirus callorhini* or *Proechinophthirus fluctus,* or both. About 83 % of the seals harboured both lice simultaneously. All pups, but only 88 % of the adult seals, were infested. The mean population size was 250 lice per

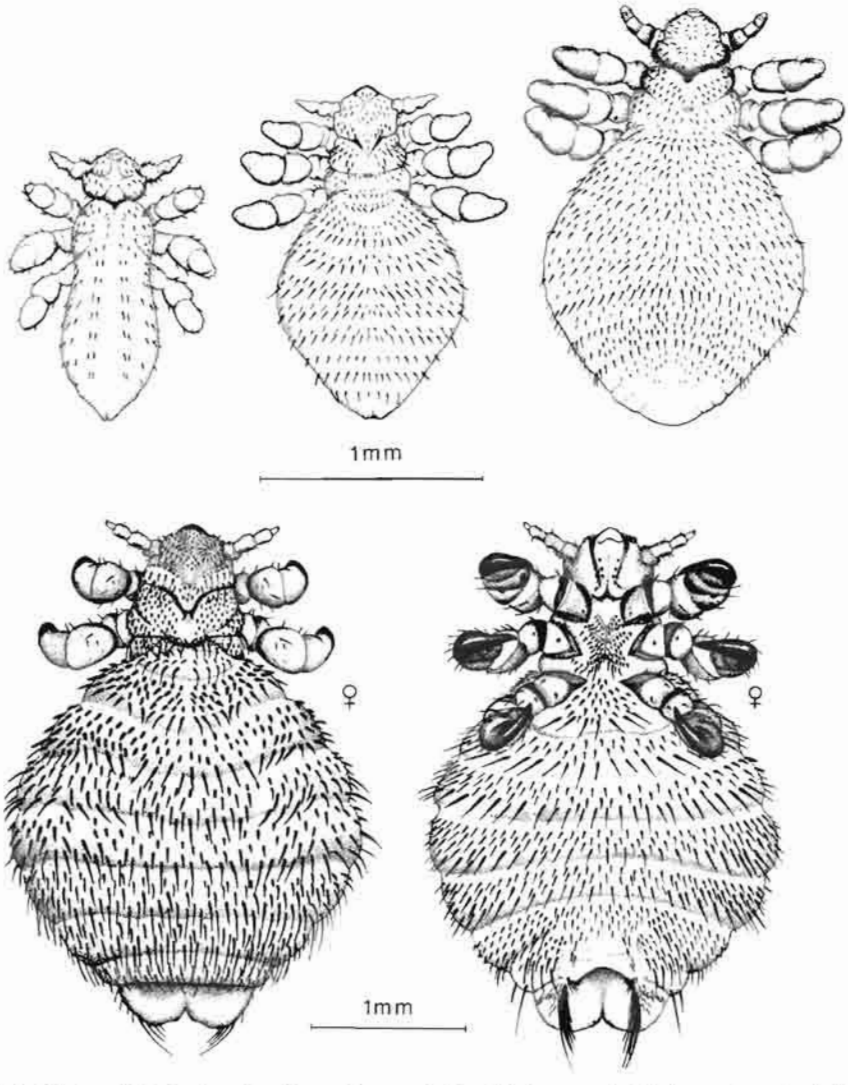

Fig. 5–37: *Echinophthirius horridus* from *Phoca vitulina*. First, second, third nymph and adult female (dorsal/ventral aspect). (After Scherf, 1963.)

pelagic adult seal and 2.1 lice per seal on the St. Paul rookeries. Dailey and Hill (1970) found 4 of 14 *Zalophus californianus* and 5 of 9 *Eumetopias jubatus* from central California to be infested with *A. microchir.*

Seal lice have perfectly adapted to the mode of life of their carriers in feeding when the host is at sea and reproducing while it is ashore. *Antarctophthirus ogmorhini* becomes active and reproduces on *Leptonychotes weddelli* at 5 to 15 °C. Eggs can develop and hatch at constant temperatures as low as 0 to 4 °C (Murray and co-authors, 1965). *Lepidophthirus macrorhini* burrows into the stratum corneum, thus reducing losses to the population when its host, *Mirounga leonina* annually sheds the outer layers of the stratum corneum attached to the hair, because only the roof of the burrow is lost (Fig. 5-40). Lice

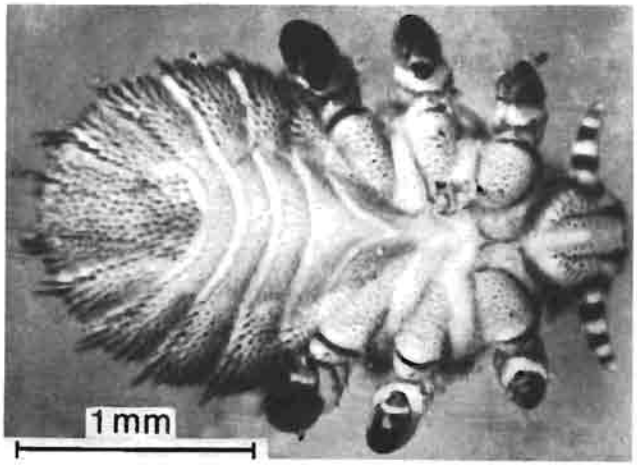

Fig. 5–38: *Lepidophthirus macrorhini.* Male, ventral aspect. Abdomen covered with many stout spines, intersegmental regions of thorax and abdomen more invaginated than in lice from terrestrial mammals. (After Murray and Nicholls, 1965.)

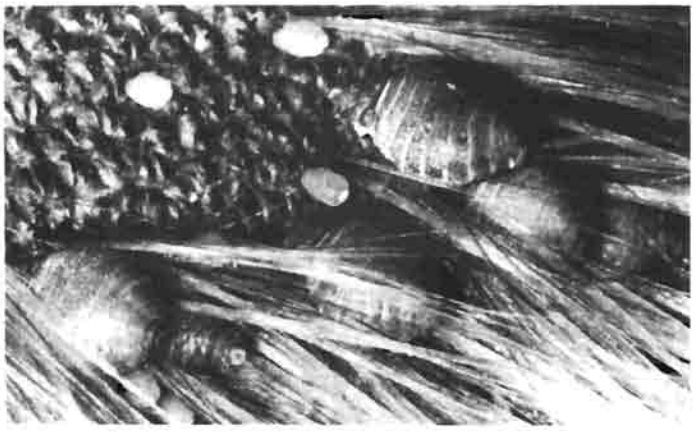

Fig. 5–39: *Acanthophthirus ogmorhini* on skin of *Leptonychotes weddelli.* Hair removed from upper left portion to show uneven skin surface and to reveal eggs and lice. The 4 largest individuals are females, the other 2 are males. (After Murray and co-authors, 1965.)

do not reproduce on the older seals that moult in muddy wallows, and consequently fewer parasites are found on these individuals (Murray and Nicholls, 1965).

The presence of large numbers of lice on the skin beyound doubt causes serious irritation and, in young hosts, probably debilitation. High lice burdens are believed to render young seals less resistant to disease. Heavy infestations, on the other hand, are believed to reflect a debilitated condition which may have other causes (van den Broek and Wensvoort, 1959). 'Seal lice' *Echinophthirius horridus* are rarely found on healthy *Phoca vitulina*, but seem to flourish on weakened pups, "sometimes covering the whole head area with their drab, unsightly forms" (Geraci, 1978b, p. 40). Clausen (1978) found 1 young common seal from Danish waters to be so heavily infested with *E. horridus* that the lice appeared to have caused its death. Densities of more than 3 lice cm^{-2} of body surface have

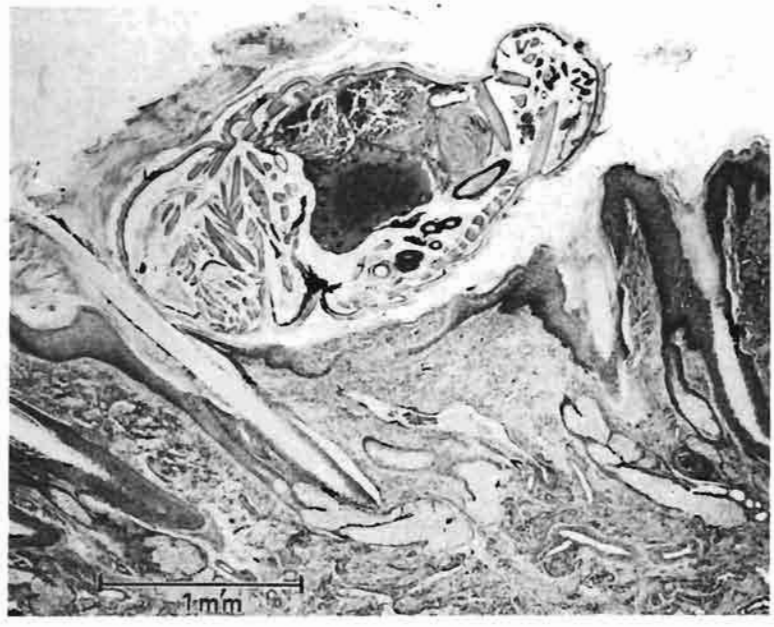

Fig. 5–40: *Mirounga leonina*. Longitudinal section of *Lepidophthirus macrorhini* nymph in burrow in stratum corneum of skin. At moult, stratum corneum and hair are shed, which removes roof of burrow. Note inflammatory reaction to louse and host blood in stomach of nymph undergoing digestion. (After Murray and Nicholls, 1965.)

been observed. Such heavy infestations often lead to severe anaemia. 'Pediculosis' is a problem of most free-living pinnipeds. Since lice are highly transmissible, careful examination of new arrivals in parks and exhibits is desirable (Cansdale, 1970; Sweeney, 1978c).

Both juvenile and adult Weddell seals *Leptonychotes weddelli* from Vestfold Hills, Antarctica, were infested with *Antarctophthirus ogmorhini*. However, although pups were in close contact with louse-infested parents, they remained free of lice until they were about 5 weeks old, when their newborn coat had been replaced by the adult type. Twenty-five percent of the pups were infested in the early weeks of December (Lugg, 1966). In contrast, *Mirounga leonina* pups become infested with *Lepidophthirus macrorhini* within a few days of birth, and the gregarious habits of the elephant seal rapidly spread the infestation through the host population. Lice transfer to all parts of the body, but it is the multiplication of those on the flippers that maintains the louse population (Murray and Nicholls, 1965). *A. ogmorhini* numbers are greatest on yearling and immature individuals but are lower on mature seals. Few mature bulls are infested. In general, those age groups of Weddell seals, which haul out of the sea most frequently throughout the year yield the highest louse counts (Murray and co-authors, 1965).

Lice are well-recognized vectors of a number of serious homoeothermic-animal diseases (Noble and Noble, 1976). Jellison and Milner (1958) suspect that sucking lice may play a role in spreading *Salmonella enteritidis* infections through fur-seal populations. Wülker (1929, 1930) considered the possibility that *Echinophthirius horridus* may serve as intermediate host for filarioid nematodes *Dipetalonema spirocauda* (p. 747), but failed to find microfilariae. Mohr (1952) maintains, without experimental proof, that microfilariae

are transmitted by anoplurans. Like Wülker (1929, 1930), Taylor and co-authors (1961) and Dunn and Wolke (1976b) found no evidence substantiating this conjecture.

Although sucking lice are easily transmitted to captive pinnipeds from new arrivals, pediculosis is easily controlled. Lice are very sensitive to chlorinated hydrocarbons. Rotenone louse powder is also very effective (Sweeney, 1978c). Two anthelminthics, dichlorvos and disophenol, have also used with good success (Lyons and co-authors, 1978).

Agents: Acarina

Mites of the family Halarachnidae are the causative agents of pulmonary acariasis, and representatives of the Sarcoptidae and Demodicidae produce mange in Pinnipedia.

'Nasal mites' and 'lung mites' of pinnipeds were originally all assigned to the genus *Halarachne*. Newell (1947) erected the genus *Orthohalarachne* to include species invading otariids and odobenids, while members of the genus *Halarachne* are believed to be restricted to phocids. Definite conclusions should be drawn with caution because of the existence of numerous synonyms and misidentifications at both the generic and specific levels. In his monograph of the family Halarachnidae, Newell (1947) listed 3 named species of *Halarachne* and 6 species of *Orthohalarachne*, some of which, however, were considered to be of dubious validity. In describing *O. letalis* from *Zalophus californianus*, Popp (1961) added another species to the list. In a rigorous revision, Domrow (1974) reduced the number of recognized species of *Orthohalarachne* from 7 to 2: *O. diminuata* Doetschman, 1944, and *O. attenuata* Banks, 1910. In a similar revison of the genus *Halarachne*, Furman and Dailey (1980) confirmed the validity of *H. halichoeri* (from *Halichoerus grypus*), *H. miroungae* (from *Mirounga angustirostris*) and *H. americana* (from *Monachus tropicalis*), declared *H. taita* (described by Eichler, 1958, from *Mirounga leonina*) a synonym of *H. halichoeri*, and added *H. laysanae* (from *Monachus schauinslandi*) as a new species.

Different species of halarachnids have originally been described from northern- and southern-hemisphere pinnipeds. However, after the revisions discussed above it becomes evident that at least *Orthohalarachne diminuata* and *O. attenuata* occur on fur seals from the Antarctic and Subantarctic.

Most species of *Halarachne*, as well as *Orthohalarachne attenuata*, inhabit the nasopharyngeal region. *O. diminuata* occurs deeper down in the respiratory system. The development of halarachnids includes 1 larval stage, a characteristic hexapod larva (Fig. 5-41,3). Females are larviparous. There appears to be no nymphal instar. The 'nymphes octopodes' of Blanchard (1906) must have been adults; the 'nymph' of Ferris (1925; Fig. 4-12) is the larva (Newell, 1947). Both adults are short-legged and engorge heavily; they are sluggish. Transmission is, therefore, almost certainly effected by the slender-legged, and often long-haired, larvae (Domrow, 1974). Furman and Smith (1973) studied the development of *Orthohalarachne* spp. *in vitro*.

Orthohalarachne diminuata (Fig. 5-41), the smallest member of the genus, is the common 'lung mite' of the California sea lion. It resides in the bronchi and trachea. Females average 920 μm in length and 500 μm in width; males are 600 μm long and 364 μm wide (Doetschman, 1944). Clinical signs of pulmonary acariasis are sneezing, productive coughing, nasal itching, mouth odour and tenacious sputum. Diagnosis can be made by administration of an organophosphorus insecticide through the nose. Mites can

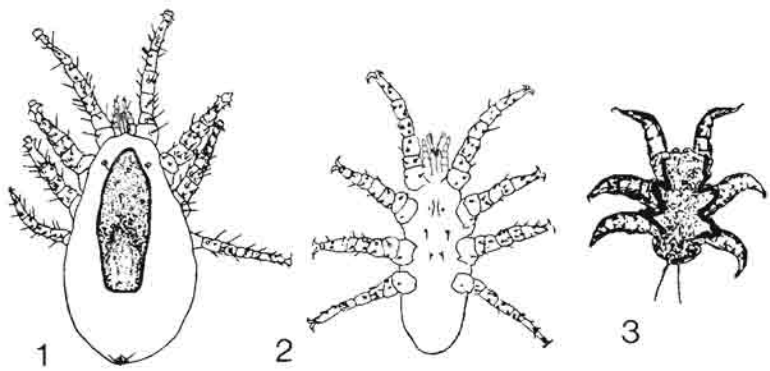

Fig. 5–41: *Orthohalarachne diminuata.* 1: Female, dorsal view (size 900 μm); 2: male, ventral view (size 685 μm); 3: hexapod larva (size 620 μm). (After Doetschman, 1944.)

subsequently be collected from sputum and from around the nostrils (Ogata and co-authors, 1979).

Seawright (1964) studied the pathology of pulmonary acariasis in the Tasmanian fur seal *Gypsophoca tasmanica,* now recognized as *Arctocephalus doriferus.* The parasite was believed to be *Orthohalarachne attenuata.* However, *O. attenuata* has, thus far, only been reported from the nasopharyngeal region, not from the lung, of pinnipeds. The species dealt with by Seawright is more likely to have been *O. diminuata* because the mites resided in the trachea and bronchi, and not in the nasal cavity. Domrow (1974) has reported *O. diminuata* from that host in Victoria, Australia. The seal examined by Seawright had been held in captivity for 17 days when it became 'quiet' and depressed and died within 24 h.

At necropsy, the lungs were found to display regions of emphysema (Fig. 5-42,a). The trachea and bronchi revealed copious amounts of translucent, thick yellow mucus and slightly hyperaemic mucous membranes. Some 43 pale-coloured mites were recovered from the mucus in the distal third of the trachea and in the proximal 10 cm of both bronchi (Fig. 5-42,b). From this region, the effusion of mucus completely filled the lumen of most smaller bronchial branches. Histologically, the lungs showed extensive alveolar collapse affecting 9/10 of the total lung volume, and occlusion of small bronchi and bronchioles with an exudate consisting of mucus and desquamated epithelial cells. Lymphatics everywhere in the lung were greatly distended (Fig. 5-43,a). In many areas of collapsed lung, proteinaceous fluid containing few red blood cells filled bronchioles, atri and alveoli. Sections of the trachea and bronchi associated with the mites showed the presence of exudate consisting of mucus and desquamated epithelium on the mucosa. No inflammatory reaction was apparent. Distension of lymphatic vessels in the tracheal and bronchial walls was marked (Fig. 5-43,a). Death of the seal was attributed to asphyxiation since a large amount of lung tissue was found to be collapsed (Seawright, 1964). Dahme and Popp (1963) reported on deaths of captive *Zalophus californianus,* caused by heavy *'O. letalis' (= O. diminuata)* infestations.

Orthohalarachne attenuata infestations are confined to the nasopharyngeal region. Associated lesions are normally less severe than pulmonary lesions with *O. diminuata.* The larvae reside on the turbinate mucosa; the adults are found attached to the nasopharyngeal mucosa. In northern fur seals examined by Dunlap and co-authors (1976), there was little

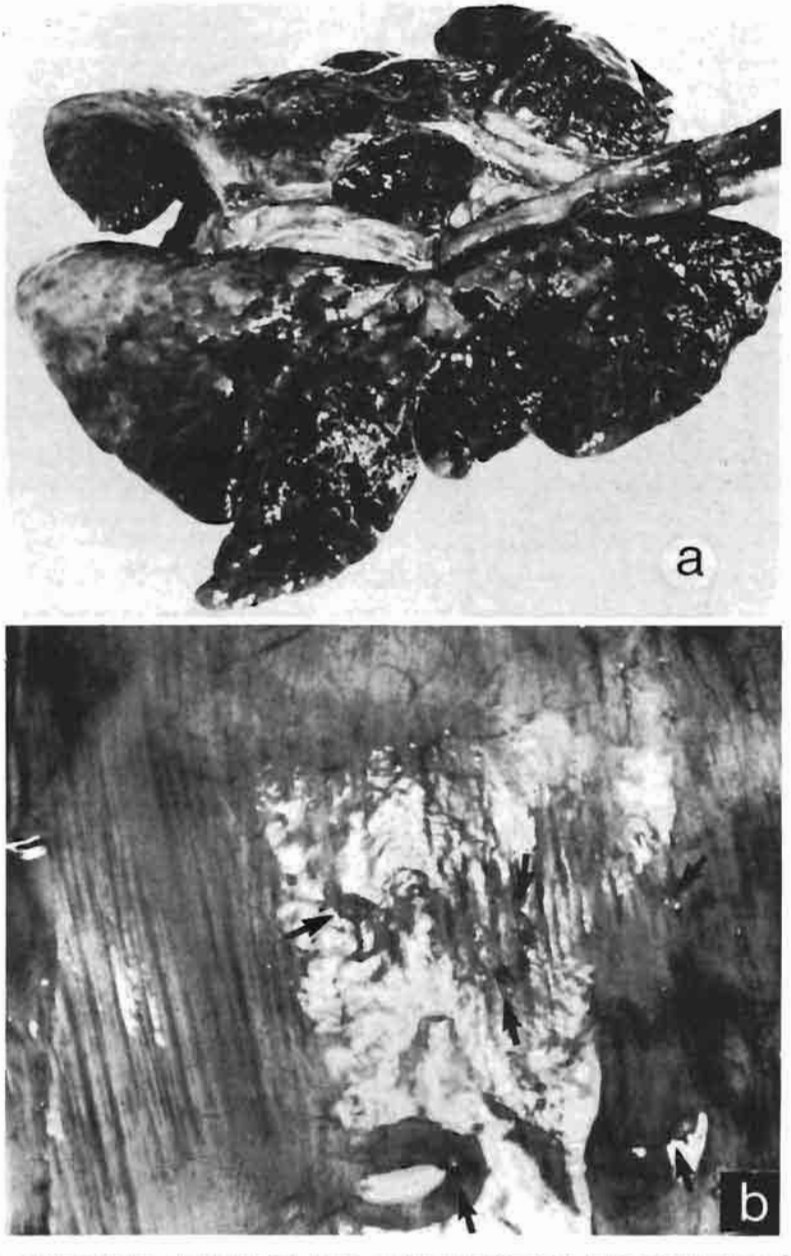

Fig. 5–42: *Arctocephalus doriferus*. (a) Lung pathology caused by halarachnid mites. Dark areas represent collapse and pale areas compensatory emphysema. (b) Tracheal mucosa. Mites *in situ* indicated by arrows. Presence of copious mucus visualized by uneven exposure and high-lights on photograph. Injected blood vessels indicate hyperaemia. (After Seawright, 1964.)

gross evidence of an inflammatory response to adult mites attached to the mucous membranes. Histological examination of the nasal turbinates revealed variable degrees of mucosal erosion adjacent to the larvae, with an accumulation of cellular debris and

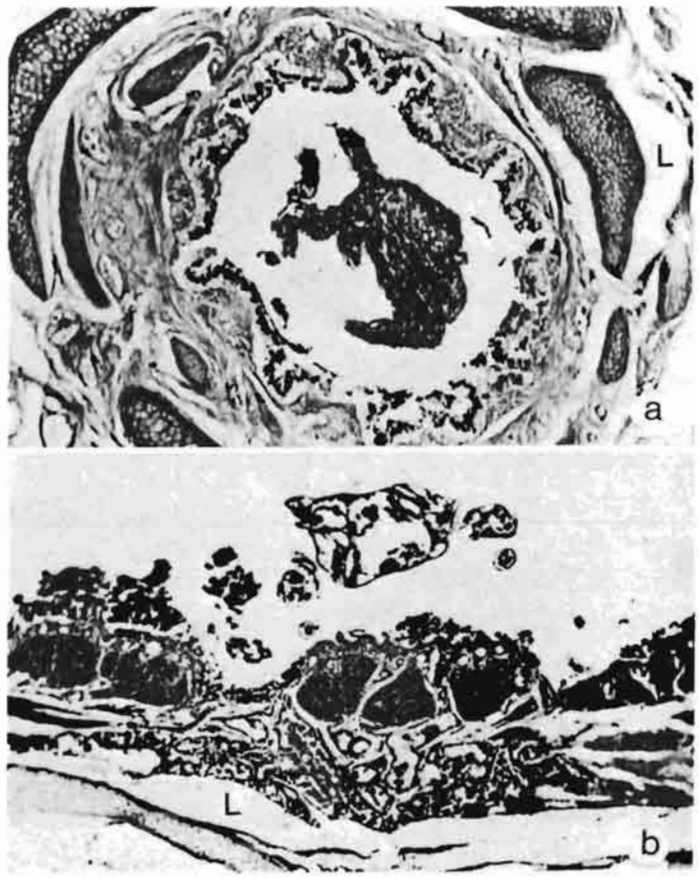

Fig. 5–43: *Arctocephalus doriferus*. Lung pathology caused by halarachnid mites. (a) Small bronchus plugged with mucus. Extent of goblet-cell activity of bronchial epithelium is apparent. Note distended lymphatics (L) of peribronchial connective tissue; PAS stain, × 130. (b) Trachea showing relationship of mite to mucosa. Epithelium near mite desquamated, but little submucosal reaction present. Note distended lymphatics (L). H & E stain, × 65. (After Seawright, 1964.)

effusion of neutrophils in the eroded areas in some cases (Fig. 5-44,a). Inflammatory oedema and mild to moderate infiltration with neutrophils was often present in the subadjacent lamina propria. Squamous metaplasia of the epithelium was frequent around the periphery of eroded areas. Well-preserved or partially haemolyzed red blood cells were occasionally seen within the mites. In non-eroded areas adjacent to larvae, the epithelium varied from normal to atrophic, disorganized, devoid of goblet cells or slightly hyperplastic, and the mucosa appeared compressed in some instances.

At the attachment points of the adult mites in the nasopharynx, the mucosa was often compressed and eroded or ulcerated, with necrosis of the superficial lamina propria and effusion of inflammatory cells (Fig. 5-44,b). In the mucosa and submucosa there was heavy local infiltration of inflammatory cells, mainly lymphocytes, with occasional histiocytes and plasma cells (Dunlap and co-authors, 1976).

Although, in the above-mentioned study, virtually all of 200 subadult *Callorhinus*

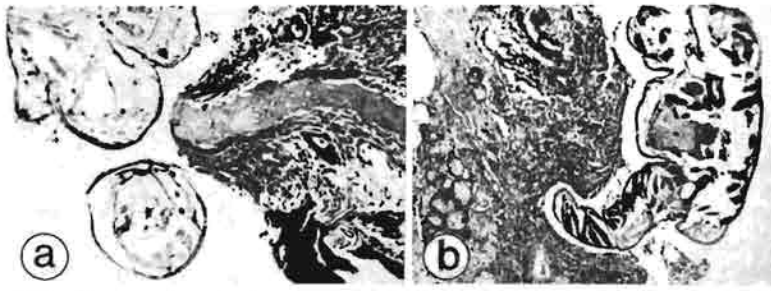

Fig. 5–44: *Callorhinus ursinus.* Lesions associated with *Orthohalarachne attenuata* infestation. (a) Section of turbinates showing erosion of epithelium and lamina propria with moderate infiltration of neutrophils and hexapod larvae *in situ*, × 350. (b) Section of nasopharynx showing mucosa with erosion and ulceration of surface. Adult *O. attenuata in situ*, with necrotic cellurlar debris around leg, × 140. (After Dunlap and co-authors, 1976.)

ursinus examined on the Pribilof Islands had *Orthohalarachne attenuata,* infestations were normally moderate to low. One seal had 670 hexapod larvae; the number of adult mites recovered from a single host was 65 (Dunlap and co-authors, 1976). Kurochkin and Sobolevsky (1971) recovered *O. attenuata* from 45 out of 52 fur seals and 1 out of 10 Steller sea lions in the Northwest Pacific. Mite counts ranged from 1 to 197 in the fur seals and amounted to 21 in the single infested sea lion.

In a subsequent study, Kurochkin and Sobolevsky (1975) found 66 % of 136 *Callorhinus ursinus* from the Komandorski Islands to be infested with an average of 19 (range: 1 to 217) *Orthohalarachne attenuata.* In these hosts, the mites were often found crowded together in groups of 10 or more per cm^2. Noticeable pathology was associated with such heavy infestations and was aggravated by the presence and activities of large numbers of mites belonging to other species. The total number of mites present in the nasal passages of a single seal often exceeded 1,500. Sneezing and coughing, frequently observed among the seals, was attributed to irritation of the nasal mucosa by large numbers of halarachnid parasites.

Nasal and pulmonary mite infestations can pose some problems in captive hosts. Pinnipeds autopsied at the San Diego Zoo showed fairly high prevalences of *Orthohalarachne diminuata* and additional species of mites. Infestation of the lungs was found in 60.8 % and of the nasal passages in 21.8 % of 74 fatalities; a total of 70 % were infested (Doetschman, 1941). Double infestation with 2 species of mites have also been reported from feral populations. Thus, Dailey and Hill (1970) reported *Orthohalarachne diminuata* from the trachea and bronchi of 13 of 14 feral *Zalophus californianus* and 6 of 9 *Eumetopias jubatus* from the southern and central California coast. *O. attenuata* was recovered from the nasopharynx of 4 of the California sea lions and 5 of the Steller sea lions.

As pointed out by Dailey (1975a), mite infestations — found nearly 100 % of the time in captive pinnipeds — can build up to uncommon massive numbers under conditions of confinement, but what detrimental effect these parasites have in nature is conjecture. Keyes (1965) considers mite infestation a significant factor for pinnipeds during their pelagic phase. He points out that heavy infestation of the nasal turbinates of *Callorhinus ursinus* with *Orthohalarachne diminuata* and *O. attenuata* can critically impair respiration, especially at sea.

No specific therapy against nasal and pulmonary mite infestation has as yet been developed (Sweeney, 1973, 1974a).

Mange mites — representatives of the families Sarcoptidae and Demodicidae — affect the skin. The first brief report on mange in Pinnipedia appears to be that of Jacobsen (1966). Large ulcerative skin lesions in a harbour seal from the German North Sea coast were initially believed to be of fungal etiology. Microscopic examination, however, revealed the presence of numerous mites close to the stratum germinativum. The epidermis at the margins of the wounds was greatly hypertrophied and keratotic; the neighbouring corium layers showed cellular infiltration. In the center of some of the lesions, the epidermis was entirely destroyed, exposing the subdermal tissue. In the absence of any detectable microbial agent, the wounds were believed to be the result of a primary parasitosis. From their roundish shape, the mites were tentatively identified as a species of *Sarcoptes* (Jacobsen, 1966). Similar lesions had previously been seen in seals from the Dutch coast but were not examined more closely. It was estimated that over 10 % of the seal population in that area (about 1,000 individuals) were infested (van Haaften, 1962). Hubbard (1969) briefly mentioned 1 case of "sarcoptic-like mange caused by an as yet unidentified mite" on an (unspecified) pinniped.

Demodectic mange in *Zalophus californianus* has first been observed by Kenny (in Dailey and Brownell, 1972), but the causative agent, which resides in the hair follicles, was not studied in detail. Dailey and Nutting (1980) described it as *Demodex zalophi*. The mites, which are difficult to remove from the skin and are best obtained by long-term (8 to 10 days at 20 °C) digestion in 5 % pepsin solution, are typically vermiform demodicids. Adult males measure, on the average, 202 × 29 μm, adult females 258 × 33 μm. The spindle-shaped ova are 85 μm long and 25 μm wide, the fusiform larvae 119 × 24 μm in dimension. The protonymph (171 × 27 μm) and nymph (237 × 33 μm) stages are much like the larva. *D. zalophi* is most similar, although specifically different from, *D. canis*, the causative agent of canine mange.

All life-cycle stages of *Demodex zalophi* reside in the hair follicle usually above the duct of the sebaceous gland. The mites have their mouthparts closely apposed to the follicular epithelium. Cell destruction and undercutting near the mouthparts indicates cytophagy. Thickened keratin, especially in the area of the podosoma and opisthoma, suggests induced hyperkeratinization, possibly by the strong leg claws (Dailey and Nutting, 1980).

In *Zalophus californianus*, the mangelike lesions, which are characterized by alopecia, hyperkeratosis, scaling and excoriation, are limited to the webbing and dorsal surface of the flippers (Fig. 5-45) and posterior ventral surface near the genital area. They are normally not pruritic, but tend to become chronic if not treated early. Diagnosis of demodicosis is made by finding mites in deep scrapings from affected areas. Secondary bacterial infection may occur, causing pyoderma most commonly of staphylococcal origin. In at least 1 instance, the lesions spread into a generalized form of the disease (Sweeney, 1973, 1974a; Nutting and Dailey, 1980).

Histological examination reveals that, in areas of deep erosion, the entire skin had been sloughed. Areas of shallow erosion show Gram-positive bacterial invasion, free blood cells (pus and normal blood elements) and an inflammatory response. In other adjacent areas, mites are found in the hair follicles oriented in typical demodicid fashion, i.e., anterior end down and legs against the follicular epithelium. In contrast to normal hair

Fig. 5–45: *Zalophus californianus*. Gross lesion produced by *Demodex zalophi* on anterior flipper. (After Nutting and Dailey, 1980).

follicles, the epithelium of infested follicles shows severe cellular undercutting near mite mouthparts and marked hyperkeratinization near mite claws. Follicles with multiple invasions are distinctly distended, the keratin is sheathlike and the hair is wanting. Dense mite populations cause coalescence of adjacent follicles to form hairless, distended lesions. These apparently rupture, either mechanically or through mite activity, so that the parasites invade the dermis. Under such conditions, a foreign-body reaction occurs with the formation of giant cells and phagocytosis of the parasites. Occasionally, mites may be found in venules, the walls of which had presumably been punctured. No mites occur in sebaceous glands or in the fascia beneath the skin, but a few dead or defunct parasites may be found in thick-walled keratinoid cysts (Fig. 5-46; Nutting and Dailey, 1980).

Effective treatment of demodicosis in *Zalophus californianus* has been reported with ronnel (Ectoral, Pitman-Moore) (Sweeney, 1973, 1974a). Benzyl benzoate (20 %) and 0.9 % gamma isomer of benzene hexachloride (Mulzyl, Pitman-Moore) was effective in 1 case of mange caused by an unidentified sarcoptid mite (Hubbard, 1969).

An interesting feature of the occurrence of a species of *Demodex* on a marine mammal is the fact that demodicids can obviously adapt to survival and transfer between hosts under moist conditions and high salt concentrations. Demodicosis in *Zalophus californianus* is apparently not a product of captivity, as first thought, but captivity may be an exacerbating factor (Nutting and Dailey, 1980). Some yet unknown predisposing factor(s) appear to be required for infestation, since the parasitosis is not readily transmitted in captivity (Sweeney, 1978c). As demodicid mites are normally host-specific (Nutting, 1968), *Demodex zalophi* is likely to be unique to the California sea lion. However, demodectic mange has not yet been observed in the feral population. The similarity between *D. zalophi* and *D. canis* suggests that the sea-lion parasite may serve as an adjunct discriminator (biological tag) in pinniped phylogeny, and that demodicids in general may be used as phylogenetic markers for all mammals (Dailey and Nutting, 1980; Nutting and Dailey, 1980).

Fig. 5–46: *Zalophus californianus*. Section through edge of a keratinoid cyst (K) showing sections of mites *Demodex zalophi* (M), × 300. Tissue stains indicate that mites are moribund or defunct. (After Nutting and Dailey, 1980.)

Agents: Cirripedia

'Whale barnacles' are of common occurrence on the skin of cetaceans. Although seemingly improbable, barnacle 'infestations' also exist in pinnipeds.

According to Bonner (1968), individuals of *Arctocephalus tropicalis* from South Georgia are seen, from time to time, with varying 'infestations' of the pedunculate barnacle *Lepas australis*. The cirripeds are attached to the guard hairs or, less frequently, directly to the skin of the naked ear pinna. When attached to the guard hairs, the barnacles may occur anywhere on the dorsal surface of the seal except the ventral portions. They vary in length from less than 3 to over 30 mm. The largest specimen seen measured 44 mm. So far, only breeding cows have been found to be infested.

Stirling (1971b) saw barnacles on females of *Arctocephalus forsteri* from South Neptune Island, Australia. The cows became infested during the period of feeding at sea prior to parturition. Baldridge (1977) found a yearling northern elephant seal *Mirounga angustirostris* with 8 goose barnacles *Lepas pacifica* attached to the skin of the dorsal surface. Upon closer inspection of 'many' northern elephant seals, Baldridge found only 1 other individual similarly affected.

The seals do not seem to suffer any detriment from the presence of these epizoites. In 1 instance, however, a very heavy infestation of an individual of *Arctocephalus tropicalis* was associated with a patch of mange. There appeared to be no causal relationship between both conditions (Bonner, 1968).

NEOPLASIA

Information on neoplastic diseases in Pinnipedia is scanty. Tumours of various origins have been reported infrequently and mostly characterized inadequately. There are only a few detailed studies. In his survey of tumours in feral marine animals, Waddell (1968) does not mention a single case of neoplasia in pinnipeds. Most reports on neoplastic conditions appear as incidental findings in captive individuals. They are difficult to diagnose, they do not occur with any degree of regularity, and they are either innocuous or incurable without surgery. Therefore, tumours are generally considered to be of academic rather than practical interest in feral pinnipeds (Ridgway and co-authors, 1975).

The reported cases of neoplastic disorders in pinnipeds are listed in Table 5-8. With the exception of leukaemic lymphomas found in 2 harbour seals (Griner, 1971), all other reports refer to single observations. Although virtually any organ can be affected by neoplasia, tumours of the urogenital and reproductive systems have been encountered more frequently than others.

Mawdesley-Thomas (1971) described an ovarian tumour in an adult southern elephant seal *Mirounga leonina* shot in the Bay of Isles, South Georgia, about 2 to 3 weeks post-partum. The right ovary measured 40 × 21 × 14 mm and weighed 8.5 g, while a yellowish nodular mass, measuring 9 × 9 × 11 cm and weighing 340 g, apparently replaced the left ovary. The tumour (Fig. 5-47) represents the first recorded case of a granulosa-cell neoplasm in the Pinnipedia.

Several uterine tumours have been observed in a grey seal *Halichoerus grypus* shot at Shunni Wick, Shetland, and estimated (from counts of the number of dental layers of a canine tooth) to be at least 44 yr old. The cervix of the animal contained several well-defined tumours consisting of elongated spindle-shaped cells, typical of the uterine leiomyomas. No mitotic figures were seen, and the growths were not considered to be malignant. Two of these tumours, which measured 2.5 × 3.5 cm and 4.0 × 4.5 cm, were seen partly obstructing the cervical canal. In addition, there was a well-differentiated squamous-cell carcinoma in a uterine horn. Invasion of the uterine musculature was seen in many sections (Mawdesley-Thomas and Bonner, 1971).

A retro-peritoneal tumour, histologically appearing as a malignant mesenchymoma, was found to occupy most of the abdominal cavity of a California sea lion. The growth weighed 7.3 kg (Nakajima and Fujimoto, 1971).

A captive 16 yr-old female *Zalophus californianus* died suddenly following a gradual weight loss over a 6-month period. At necropsy, gross lesions were observed in the small intestine and pancreas. About two-thirds of the small intestine were haemorrhagic and distended with fluid and gas. This portion had herniated through a fibrous ring joining a pancreatic mass and the mesentery. The mass was located in the proximal portion of the pancreas, had a slightly irregular surface, and measured 8 × 5 × 4 cm (Fig. 5-48). Mitotic figures in the tumour were rare and there was no evidence of invasiveness, nor were metastatic cells present in draining lymph nodes. Histopathologic diagnosis of the growth (Fig. 5-49) was pancreatic duct adenoma (Moore and Stackhouse, 1978).

A northern fur seal female pup, about 2 weeks old, was found dead on the Pribilof Islands, Alaska. Necropsy revealed the presence of an irregular mass at the anterior pole of the right kidney. The tumour did not protrude nor extend to a noticeable degree above the contour of the kidney. On cut section, the mass was greyish white and about 8 × 4 mm in

Table 5-8
Tumours in Pinnipedia

Species	Tumour diagnosis	Source
Zalophus californianus	Adenocarcinoma and myosarcoma in lung	Fox (1941)
	Retro-peritoneal tumour (malignant mesenchymoma)	Nakajima and Fujimoto (1971)
	Fibroma in neck musculature; adenocarcinoma in neck and lung	Simpson and Gardner (1972)
	Adrenal adenoma	Sweeney (1973)
	Bile-duct carcinoma	Schroeder and co-authors (1973)
	Bladder carcinoma with metastasis	Sweeney (1974a)
	Benign nephroblastoma; squamous-cell carcinoma with lymphatic metastasis	Sweeney and Gilmartin (1974)
	Adenocarcinoma of eccrine gland	Simpson and Ridgway (in Ridgway and co-authors, 1975)
	Pancreatic-duct adenoma	Moore and Stackhouse (1978)
	Necrotic endocrine adenocarcinoma	Stroud and Roffe (1979)
Phoca vitulina	Leukaemic lymphoma	Griner (1971)
	Lymphosarcoma	Stroud and Roffe (1979)
Halichoerus grypus	Uterine leiomyoma and squamous-cell carcinoma	Mawdesley-Thomas and Bonner (1971)
Callorhinus ursinus	Renal fibrosarcoma	Brown and co-authors (1975)
	Lymphosarcoma	Stedham and co-authors (1977)
Mirounga leonina	Malignant ovarian (granulosa-cell) tumour	Mawdesley-Thomas (1971)
	Neoplastic mass associated with an eye; subcutaneous neoplastic mass on posterior thorax	Tierney (1977)
Hydrurga leptonyx	Tumour obstructing bronchus; stomach carcinoma	King (1964)
'Seal'	Lymphadenoma	Eriksen (1962), Larsen (1962)
Odobenus rosmarus	Osteosarcoma (?)	Piérard and co-authors (1977)
	Myelogenous leukaemia	Eriksen (1962), Larsen (1962)

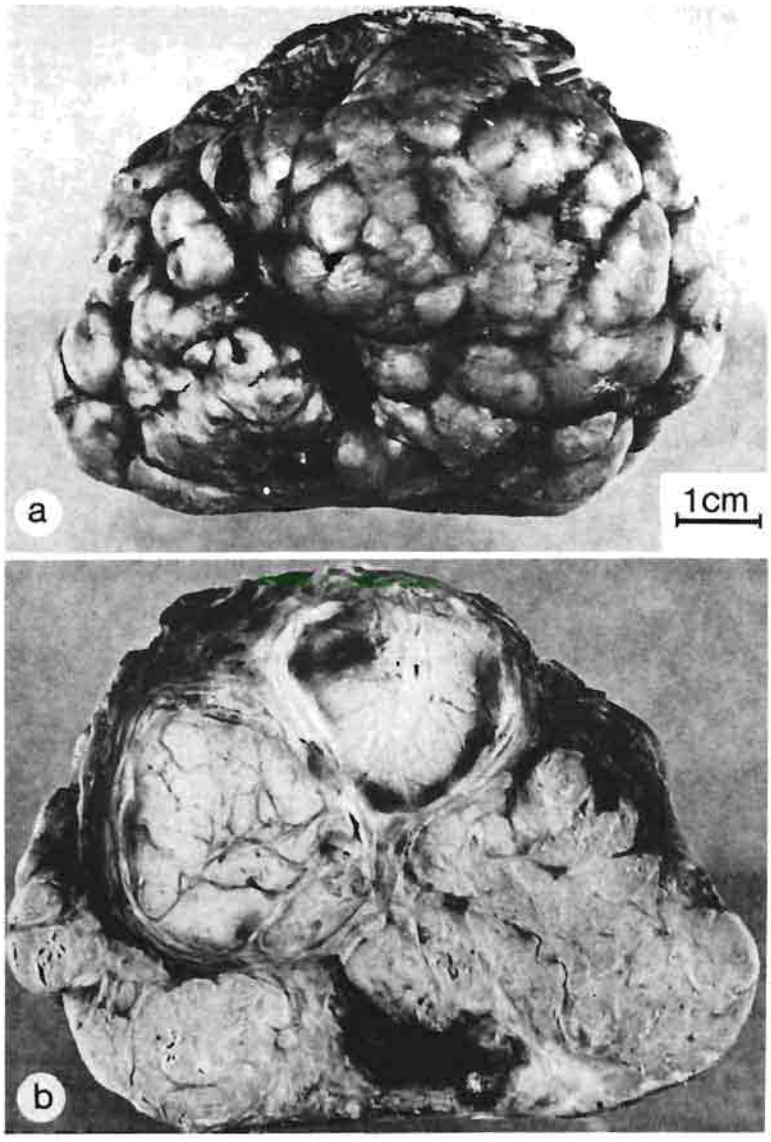

Fig. 5–47: *Mirounga leonina*. Granulosa-cell neoplasm. (a) External appearance of ovarian tumour; (b) cut surface of growth. (After Mawdesley-Thomas, 1971.)

dimension. No other lesions were seen. Histopathological examination revealed a renal fibrosarcoma displaying numerous mitotic figures (Brown and co-authors, 1975).

A neoplastic disease of the lymphoid system was diagnosed in another northern fur seal pup found dead in a rookery on St. Paul Island, Pribilof Islands, and estimated to be less than 1 month of age. Grossly, slight enlargement of the mesenteric lymph nodes was noted. Microscopically, a lymphosarcoma composed of sheets of monomorphic lymphoid cells was seen in sections of lymph nodes and tonsil. Numerous mitotic figures were present (up to 5 per high-power field). Necrotic lymphoid cells or histiocytes were scattered

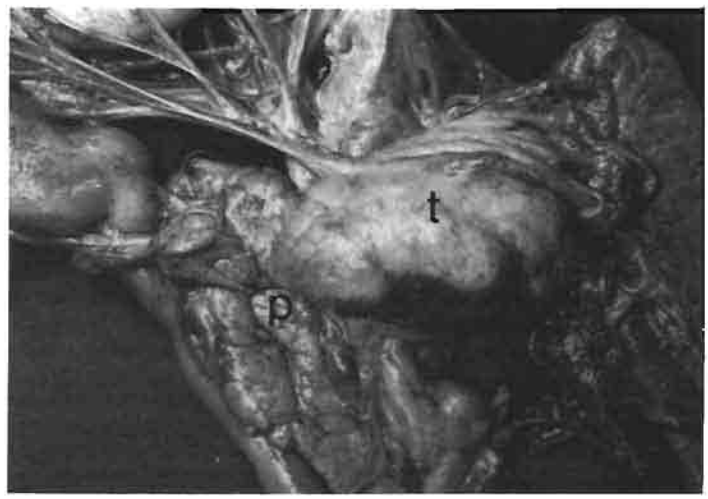

Fig. 5–48: *Zalophus californianus.* Gross appearance of pancreatic tumour (t) adjacent to normal pancreas (p). (After Moore and Stackhouse, 1978.)

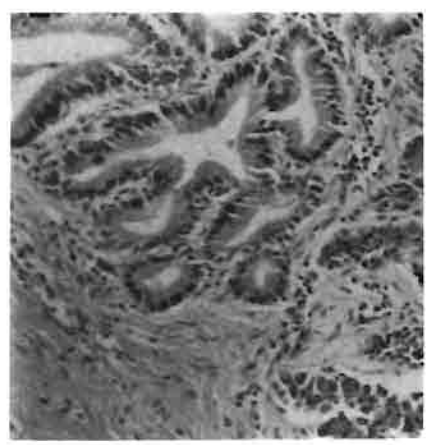

Fig. 5–49: *Zalophus californianus.* Pancreatic duct adenoma surrounded by fibrous connective tissue and mononuclear inflammatory infiltrates. Note normal acinar cells in lower right; × 375. (After Moore and Stackhouse, 1978.)

through the tissue, imparting a 'starry sky' appearance. Electron microscopy of poorly preserved material revealed the presence of unidentified round structures in the cytoplasm of some of the neoplastic cells. These structures, which measured from 60 to 90 nm in diameter, had dense, sometimes granular outer margins and variably less dense cores. Morphologically, they resembled viruses, possibly oncornaviruses, but positive identification was not possible (Stedham and co-authors, 1977).

Malignant leukaemic lymphoma was diagnosed in 2 harbour seals that died within a 4-day period in the San Diego Zoo. The individuals involved were young males, $1^1/_2$ and $2^1/_2$ years of age. Microscopic examination of tissues of the older seal indicated a neoplastic process which involved most of the reticuloendothelial system. The pathologic changes

were characterized by proliferation of lymphoblasts in the spleen, lymph nodes and thymus. Numerous mitotic figures were seen in neoplastic cells, which were large, immature mononuclear cells of the histiocytic type. Neoplastic infiltration and proliferation were also observed in sinusoids of the liver, subserosa of the stomach, pancreas, interstitial tissues of the lungs and bone marrow. The process in the peripheral blood was evidenced as lymphoblastic leukaemia. Histopathological changes in the younger seal were similar. The almost simultaneous appearance of the disease in 2 seals maintained in the same pool suggested that it might have been of viral origin (Griner, 1971).

Sweeney (1973, 1974a) briefly mentioned 3 fatal cases of neoplasia in *Zalophus californianus* — a squamous-cell carcinoma with metastasis in an adult female, a transitional cell carcinoma of the bladder with metastasis in another adult female, and a nephroblastoma in a yearling male sea lion.

ABNORMALITIES

Observations of congenital disorders and abnormalities among the Pinnipedia appear to be extremely scanty; reports in the literature mostly reflect incidental findings. King (1964) mentioned a curiously hairless and toothless harbour seal. At 2 weeks of age, when the orphaned pup was taken into captivity, it had almost no hair and no sign of teeth, and remained unchanged in this condition for 3 yr, although otherwise developing normally.

A pseudopersistent urachus was diagnosed by Cornell and co-authors (1975) in a baby walrus from St. Lawrence Island, Alaska. Upon capture, the individual exhibited a pattern of initial weight gain consistent with that of other pups and appeared to be completely normal. After about 1 month in captivity, the pup's appetite waned and it died about 2 weeks later. Pathologic examination revealed a pseudopersistent urachus, along with subsequent hydronephrosis and pyelonephritis, accompanied by secondary pneumonia. Although the occurrence of persistent urachus, whether true or pseudo, in domestic mammals is well documented, this is the first report from Pinnipedia.

Debilitation and emaciation resulting from multiple proliferative and degenerative bone lesions, severe tooth wear, peridontal disease and a poorly-healed fracture of the lower jaw have been diagnosed as the cause of death in an adult male harbour seal autopsied by Stroud and Roffe (1979).

Blood-vessel diseases and abnormalities (arteriosclerosis, aortitis, spontaneous arterial lesions, etc.) are another cause of debilitation, and possibly death, in pinnipeds (Kelly and Jensen, 1960; Prathap and co-authors, 1966; Stout, 1969).

Other examples of structural abnormalities in pinnipeds include oesophageal dilation (a neuromuscular disease) and diffuse hyaline muscular degeneration of unknown cause (Wilson, 1972; Stroud, 1978). Both conditions have been reported from captive harbour seals and are known to occur in a number of terrestrial animals.

Vertebral-column deformity and osteonecrosis of pelvis and femur has been observed in an adult male California sea lion found in extremely poor condition on Año Nuevo Island, California (USA). In the absence of typical tubercular lesions in the surrounding tissues and evidence of a primary lesion, the etiology of the abnormality remained undiscovered (Morejohn, 1969).

Huey (1924) observed a deformity in the posterior parts of the parietals of a northern elephant seal, consisting of a 17-mm variation in length. It was not clear whether this

abnormality was acquired congenitally or in an accident during juvenile life. Beyond doubt, serious disturbances may result from traumatic insult (e.g., Jortner, 1974).

References to the older literature on abnormalities etc. of wild animals, including pinnipeds, have been compiled by Halloran (1955).

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6. DISEASES OF MAMMALIA: SIRENIA

G. LAUCKNER

The order Sirenia contains only 2 recent families (including 4 valid species) of sea cows or 'mermaids' — the monotypic Dugongidae or dugongs and the Trichechidae or manatees, each of which is represented by a single genus. *Dugong dugon* inhabits tropical bays and estuaries of the Indian and Pacific Oceans and the Red Sea; the 3 *Trichechus* species are restricted to similar habitats on both sides of the Atlantic Ocean. Several geographic races or subspecies have been recognized (Scheffer and Rice, 1963; Rice and Scheffer, 1968). Both *D. dugon* and *Trichechus* spp. have been listed under a variety of generic *(Halicore, Manatus)* and specific names. The confused taxonomic situation in the Sirenia has recently been reviewed by Husar (1978a, b, c).

The Sirenia are almost strictly vegetarians — a fact which may be important when considering their parasites and the life cycles of these. Reports on microbial or protistan diseases of sirenians are scarce and based almost exclusively on necropsies of individuals which had died in captivity. Only a few metazoan (helminth) parasites have been reported from wild and captive sea cows. The 'Annotated Bibliography of Sirenia' of Whitfield and Farrington (1975), the only one of its kind, contains a total of 488 references to the general biology of these mammals, but only 5 papers referring to parasites and commensals. The literature on the physiology of *Dugong dugon* has been reviewed by Marsh and co-authors (1978), the general scientific literature on this species by Husar (1975). The latter summary shows that the available information is widely scattered and is largely the result of incidental observation or is frequently anecdotal. According to Husar (1978a), no diseases have been reported from *Dugong dugon*. Pertinent publications on the biology and medicine of marine mammals including sirenians (Ridgway, 1972; Dailey, 1978; Geraci, 1978; Sweeney, 1978a, b, c, d) make no reference to specific diseases of Sirenia. However, methods of disease diagnosis and treatment, as outlined, in these publications, for other marine mammals, may possibly be applied successfully also to sirenians.

DISEASES CAUSED BY MICROORGANISMS

Agents: Virales and Bacteria

Viral diseases have apparently not yet been diagnosed in sirenians, although in all probability such diseases do exist. Manatees appear to be highly susceptible to pneumonia and other bronchial disorders (Husar, 1978c). An individual of *Trichechus senegalensis*, held captive in the Antwerpen (Belgium) Zoo, died of acute enteritis (Derscheid, 1926). The causative agent(s) of such affections have not been identified.

Granulomatous lesions in the skin and lungs of *Trichechus inunguis* may be caused by infection with acid-fast organisms. In the first case reported (Boever and co-authors, 1976), a 1-year-old captive female developed pyoderma. Initially, the condition responded

to systemic antibiotic treatment with lincomycin, but subsequently the lesions became more severe. Each pustule was filled with purulent exudate. The back and flanks of the manatee were most severely affected, but the head, back, flippers and tail fluke were also involved. Various antibiotics, administered orally and intramuscularly, as well as topical application of various antifungal, antiparasitic, salt and iodine baths gave no apparent signs of improvement. Exposure to ultraviolet radiation seemed to control the condition, although complete healing was never accomplished.

When the diseased manatee eventually died at 5 years of age, necropsy revealed half a dozen abscesses, 1 to 3 cm in diameter, in each lobe of the lungs. The abscesses were filled with a caseous exudate. Cultures from these lesions were negative for pathogens when incubated for 24 h at 37 °C on blood agar. However, acid-fast bacilli, isolated from the skin lesions and incubated at 30 and 37 °C on culture media, were identified as *Mycobacterium cheloniae*, a micro-organism characterized by resistance to antimycobacterial drugs (Boever and co-authors, 1976). Boever (1978) reported 2 further cases suggestive of mycobacterial infection in zoo-held *Trichechus inunguis*. Since *M. cheloniae* (previously called *M. abscessus*) is a common pathogen of poikilothermic animals and has also been isolated from the water of public aquaria (Pattyn and co-authors, 1971), it appears likely that the manatees have acquired their infections in captivity.

Bartmann (1972) described skin lesions in a zoo-held individual of *Trichechus manatus*. The picture of the disease, which did not respond to topical treatment with various therapeutical ointments or intramuscular chloramphenicol administration, is highly suggestive of a *Mycobacterium* infection, although unidentified streptococci, staphylococci and *Pseudomonas morgani* were believed to be the causative agents.

Agents: Fungi

A captive manatee developed raw-appearing erosions of the skin of the nose, face, flippers and tail. Skin scrapings revealed *Epidermophyton floccosum*. Topical treatment with nystatin ointment plus oral administration of 1 g of griseofulvin twice a day for 21 days resulted in satisfactory recovery (Dilbone, 1965). *E. floccosum* normally infects only man (McCarty, 1973).

A dermatosis, characterized by a floccular growth of filamentous fungi and the formation of purulent abscesses 5 to 10 mm in diameter, was found to affect 2 captive male manatees *Trichechus manatus*. Presumably saprophytic *Cephalosporium* sp. and *Mucor* sp. were isolated from the lesions and grown in culture (Tabuchi and co-authors, 1974). Mok and Best (1979) described a saprophytic hyphomycete infection of *T. inunguis*.

DISEASES CAUSED BY PROTISTANS

Florida manatees living in freshwater may become coated with algae *(Lyngbya martensiana, Compsopogon coeruleus)*, which provide a substrate for a fouling community comprising amphipods, isopods, dipteran larvae, nematodes, copepods, ostracods and protozoans. Individuals spending time in sea water may become covered with marine diatoms *(Zygnema, Navicula)*. However, these associates tend to be free-living and phoretic rather than parasitic (Hartman, 1971). No reports of protistan diseases in Sirenia have come to the reviewer's attention.

DISEASES CAUSED BY METAZOANS

Agents: Trematoda

Relatively few digenetic trematodes are known to infest sirenians. All known species are monostomes, mainly of the family Paramphistomatidae. The others are members of the Pronocephalidae, Opisthotrematidae, Rhabdiopoeidae and Labicolidae. The majority inhabit the intestinal tract, while others parasitize in the nasal passages, oesophagus or lungs. The monostomes of the Sirenia manifest striking morphological agreement with the monostomes of marine turtles (Stunkard, 1970, see Chapter 2).

Paramphistomatids of the genera *Zygocotyle, Chiorchis, Solenorchis, Indosolenorchis* (Fig. 6–1) and *Schizamphistoma* inhabit, sometimes in large numbers, the intestinal tract and particularly the pyloric caeca of *Dugong dugon* and *Trichechus senegalensis* from the Red Sea and the Indian Ocean (Stunkard, 1929; Price, 1932; Sokoloff and Caballero y Caballero, 1932; Baylis, 1936; Hilmy, 1949; Dollfus, 1950, 1955; Crusz, 1951; Crusz and Fernand, 1954; Boever and co-authors, 1977). Dollfus (1955) believes the species of *Solenorchis* and *Indosolenorchis*, described by Hilmy (1949) and Crusz (1951), to be identical with *Zygocotyle* sp., described by him (Dollfus, 1950).

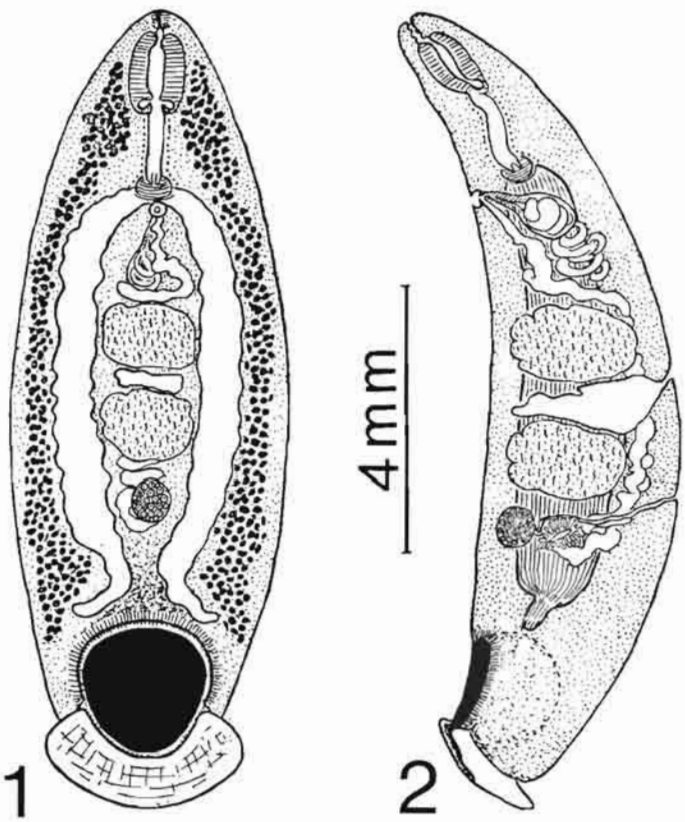

Fig. 6-1: *Indosolenorchis hirudinaceus* from caecum of *Dugong dugon*. 1: Ventral view; 2: lateral view, omitting left intestinal caecum and vitellaria. (After Crusz, 1951.)

Pronocephalids of the genera *Lankatrema* and *Taprobanella* (Fig. 6–2) and rhabdiopoeids *Rhabdiopoeus* inhabit the stomach and pyloric caeca of *Dugong dugon* from Ceylon and the intestine of *D. dugon* from Queensland (Australia), respectively (Johnston, 1913; Crusz and Fernand, 1954). Opisthotrematids *Opisthotrema (Cochleotrema)* occur in the respiratory tract (nasal cavity, oesophagus, Eustachian tubes). *Pulmonicola*

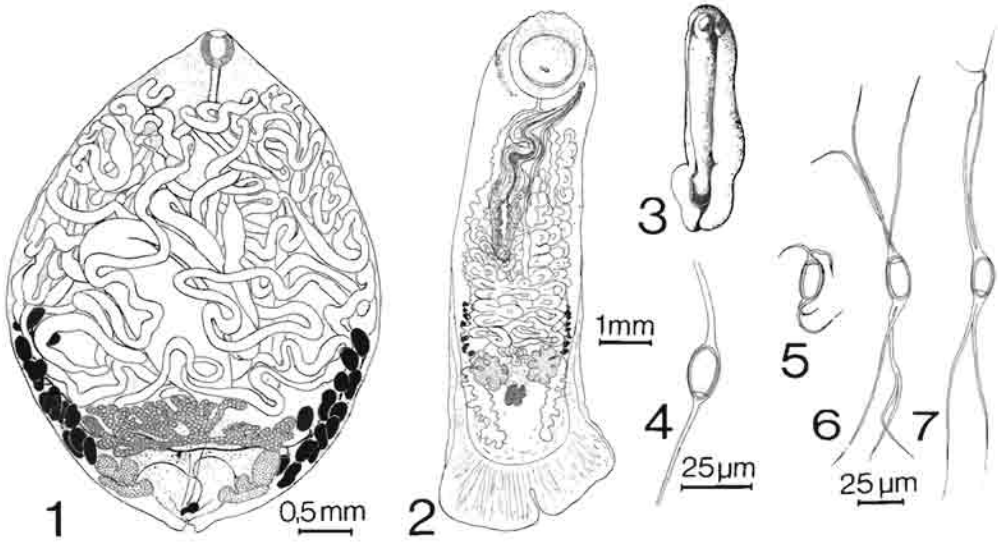

Fig. 6-2: Stomach trematodes from *Dugong dugon*. 1: *Lankatrema mannarense*, ventral view; 2. *Taprobanella bicaudata*, ventral view; 3: *T. bicaudata*, freshly fixed specimen showing ventral concavity; 4: egg of *L. mannarense;* 5-7: eggs of *T. bicaudata*. (After Crusz and Fernand, 1954.)

occur in the lungs of sirenians from the Indo-Pacific and the tropical Americas (Johnston, 1913; Poche, 1925; Travassos and Vogelsang, 1931; Price, 1932; Ruiz, 1946; Budiarso and co-authors, 1979). Labicolids *Labicola* inhabit abscesses in the upper lip of Australian *D. dugon* (Blair, 1979). The life cycles of all of these are unknown, and very little has been reported on their pathology.

Lankatrema mannarense (Figs 6–2 and 6–4) produces lesions in various parts of the stomach (glandular pouch, cardiac stomach, pyloric stomach, pyloric caeca; Fig. 6–3) of Ceylonese dugongs. The worms, which are relatively small (2.8 to 5.3 × 1.4 to 3.9 mm), occur either free in the stomach lumen or within cystic cavities in the mucosa (Fig. 6–4), recognizable by the presence of raised nodules on the mucous membrane. The nodules contain caseous matter enclosing worm debris, as indicated by numerous operculated eggs with polar filaments. In some cases it was possible to shell out whole worms from their cystic cavities. The capsules enclosing the trematodes consisted of invaginated mucous epithelium, below which the muscularis mucosae and submucosa were considerably thinned out (Fig. 6–4b,c). Each cavity was lined by cuboidal epithelium continuous with the gastric epithelium. It appeared that the worms when quite young had invaded a gastric gland and grown there, enlarging it to cystic proportions. The histology of the trematodes themselves indicated that they were alive at this stage. In some instances, worms were seen protruding from a minute opening in the mucosa (Fig. 6–4b, arrow). There were no signs

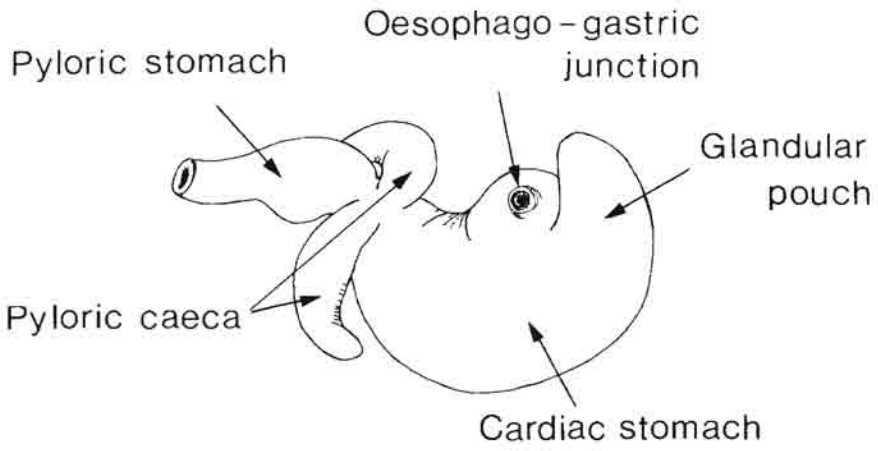

Fig. 6-3: *Dugong dugon*. Stomach regions. (After Crusz and Fernand, 1954.)

of host-cellular reaction to the living parasites comparable to those typical of foreign-body invasion. However, cysts containing dead and disintegrating *L. mannarense* evoked a strong foreign-body reaction (Fig. 6–4, d) (Crusz and Fernand, 1954).

Large numbers of nasal trematodes *Opisthotrema* spp. were recoverd from 1 female and 2 male Indonesian dugongs, which had died after several months in captivity. Three hundred and three *O. nasalis* were found attached to the mucosal layer of the nasal septa (102, 147 and 54 worms, respectively), and 37 *O. dujonis* adhered to the mucosal layer of the Eustachian tubes (17, 5 and 9 worms, respectively).

Gross pathological changes, produced by *Opisthotrema nasalis* and consisting of patchy erosions 3 to 10 cm in diameter in the nasal mucosa, were observed in the nasal passages beginning 5 cm from the opening of the external nares of the margin of the nasopharynx. The surfaces of the wounds were covered by a thin film of fibrinopurulent exudate, and their bases were reddish grey in colour and granular in appearance. In contrast, *O. dujonis*, found throughout the Eustachian tubes, produced no grossly observable damage to the mucosa.

Microscopically, the *Opisthotrema nasalis*-caused inflammatory tissue changes were found to be minimal to mild and consisted of papillary epithelial hyperplasia and areas of epithelial erosion. There was no ulceration, but some mucosal glands were dilated. Plasma cells and lymphocytes were present in the lamina propria especially aggregated around the mucosal glands. Blood vessels were found to be congested, numerous iron-bearing macrophages indicating previous haemorrhage.

The observed lesions in the host-mucous membranes were considered to be due to mechanical contact and/or enzymatic action, followed by secondary microbial infection. As the body of *Opisthotrema nasalis* is convex-shaped and supported by a strong muscular rim inundated with numerous gland cells, the pathological host-tissue changes may be the result of vacuum-cupping by the muscular rim causing abrasion or ischaemia, lysis of tissue by gland-cell secretions, or both (Budiarso and co-authors, 1979).

Individuals of *Dugong dugon* from Northern Australia were found to have abscesses

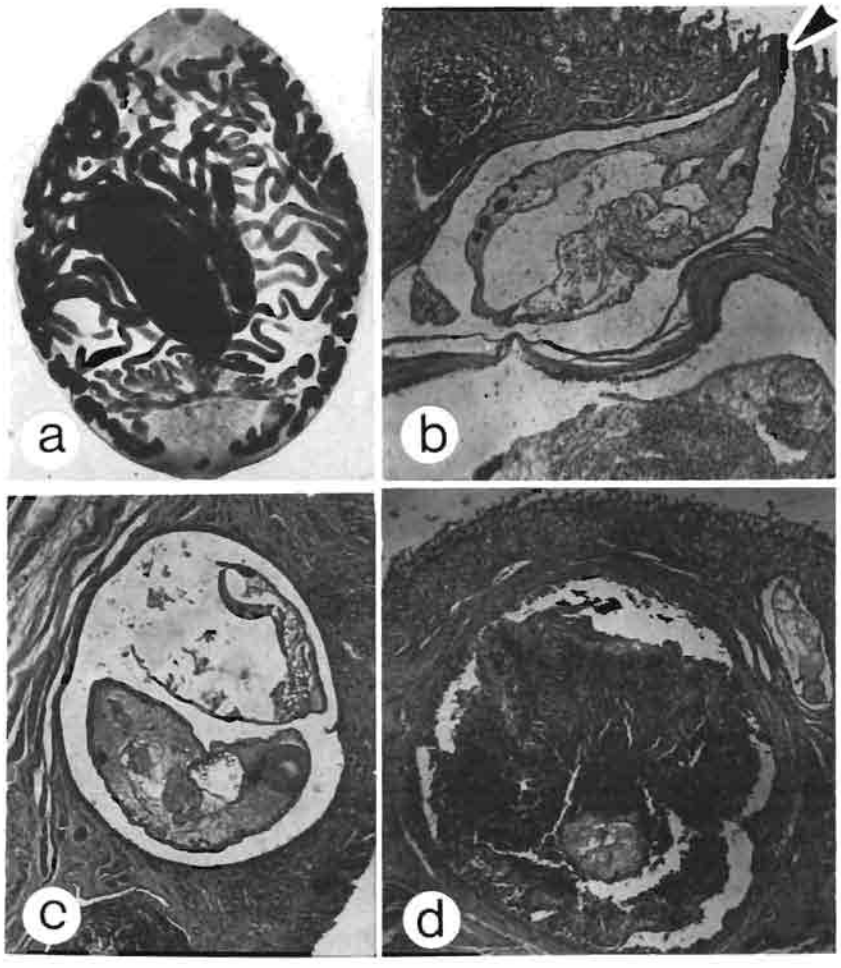

Fig. 6-4: *Lankatrema mannarense* from stomach of *Dugong dugon*. (a) Stained, mounted specimen; × 12. (b) Section of stomach wall, showing *L. mannarense* in cystic cavity presumably formed of invaginated gastric epithelium. Note anterior end of worm protruding into stomach lumen (arrow) through opening in cystic cavity. Note also absence of cellular reaction adjacent to cavity surrounding living worm; × 32. (c) Section of stomach wall, showing 2 worms within same cystic cavity; × 32. (d) Section showing dense cellular reaction around dead, encapsulated individual. Large worm fragment containing eggs, in center of mass; × 20. (After Crusz and Fernand, 1954.)

in the upper lip, which could easily be felt as a series of lumps, generally 5 or 6, along each side of the lip. None were present towards the midline. The abscesses were about 1 cm in diameter, but several may run together in heavy infestations. They communicated with the exterior by pores arranged in a line just inside the upper lip and below the line of the vibrissae, and contained pus and variable numbers of worms identified as monostome flukes. These were found to represent a new family, designated Labicolidae, and named *Labicola elongata* because of their slender, considerably elongated body. Intact worms, 19.1 to 31.1 × 0.41 to 0.78 mm (n = 13) in dimension, could be excised from the abscesses. Similar to the other sirenian monostomes, *L elongata* has eggs (about

22 × 12.5 μm) with conspicuous polar filaments up to 0.4 mm in length. Of 12 dugongs, which were autopsied and examined for *L. elongata*, all were found to be infested, and 9 further individuals observed in the field had abscesses suggestive of *L. elongata* infestation (Blair, 1979). Unfortunately, the pathology produced by these trematodes has not been studied in detail.

Agents: Nematoda

Paradujardinia (Dujardinia) halicoris, an anisakine nematode, appears to be the only roundworm thus far reported as a parasite of sirenians. It has been recovered from the intestine of *Dugong dugon* in the Red Sea and the Indo-Pacific (Johnston and Mawson, 1941; Hilmy, 1949; Jueco, 1977). According to Dollfus (1955), *P. halicoris* is the most common helminth of the dugong.

The female of *Paradujardinia halicoris* measures 110 to 115 mm in length and 4 mm in width, the male 90 to 100 × 4 mm. The worms occur free in the lumen of the stomach and the intestine. Nothing has been reported on their pathology, and their life cycle is unknown. Jueco (1977) hypothesized that infestation of the vertebrate host may be direct, i. e., without intervention of an invertebrate intermediate host.

Agents: Crustacea

Humes (1964) described copepods *Harpacticus pulex* from sloughed skin tissue of a porpoise, *Tursiops truncatus*, and a manatee, *Trichechus manatus*, both confined in the Seaquarium at Miami, Florida. Both hosts had skin ulcerations, which may have attracted the harpacticoids under the unnatural conditions of confinement. *H. pulex* has not yet been recovered from wild mammals, and other species of *Harpacticus* are free-living.

Cirripedia — mainly barnacles of the genera *Chelonibia* and *Platylepas* and rarely of *Balanus* — may occur attached to, or embedded in, the skin of sirenians, sometimes in individuals captured a considerable distance from the open ocean (Darwin, 1854; Dexler and Freund, 1906; Pilsbry, 1916; Daniel, 1953; Marlow, 1962; Stubbings, 1965, 1967; Ross and Newman, 1967; Zann, 1975; Newman in Killingley and Lutcavage, 1983). They appear to have no adverse effects on their carriers.

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7. DISEASES OF MAMMALIA: CETACEA

M. D. DAILEY

Some background information on cetaceans is appropriate before reviewing their diseases. Cetaceans evolved from land dwelling ancestors over 55 million yr ago. Except for *Pakicetus inachus* (Gingerich and co-authors, 1983), no fossils of precetaceans have been found, yet paleontologists assume they resembled large pig-like creatures able to feed on both plants and animals. According to anatomical and serum-protein studies, cetacean ancestors are close relatives of the even-toed ungulates (artiodactyls) which include cattle, sheep and camels. It is not surprising, therefore, that we find cetaceans susceptible to some diseases common to this group of animals.

The oceans, rivers and lakes of this planet provide habitats for approximately 80 species of cetaceans. Two major groups of cetaceans are formally recognized: the Odontocetes, or toothed whales, and the Mysticetes, or baleen whales. Due to the availability of information on the smaller Odontocetes, both wild and captive, most of the information presented will pertain to this group.

DISEASES CAUSED BY MICROORGANISMS

Agents: Virales

In cetaceans, several types of viruses have been reported in the literature. Greenwood and Taylor (1978b, 1979) report viruses as the cause of lesions for 2 of 22 and 0 of 18 dolphins necropsied during those respective years. The 2 deaths were listed as viral skin lesions and were questionable as to their cause of mortality. Skin lesions, or dolphin pox virus, appear often in dolphins (Dailey, own observation). These lesions are known as 'tattoos' and their etiology has been the source of speculation for years. The lesion itself consists of hyperpigmentation in the epidermis as well as pinhole lesions that appear in various patterns resembling a tattoo (Figs 7-1 and 7-2). Britt and Howard (1983) state that these lesions consist of cytoplasmic vacuolar degeneration of cells in the *stratum intermedium*, the thickest layer of the porpoise epidermis. The change in pigmentation is not fully understood at this time. The dolphin 'pox virus lesion' does not conform to the usual pox lesion seen in other animals because it lacks nodular proliferation of the infected epidermis. The virus has not been grown in culture, but has been seen in negative stains by electron microscopic examination from the bottlenose dolphin *Tursiops truncatus* and Pacific white-sided dolphin *Lagenorhynchus obliquidens* (Flom and Houk, 1979; Geraci and co-authors, 1979).

Britt and Howard (1983) have observed tattoo lesions on common dolphins *(Delphinus delphis)*, but have not been able to find the virus by electron microscopy. They suggest that the virus may not always be present, or numerous, in these lesions.

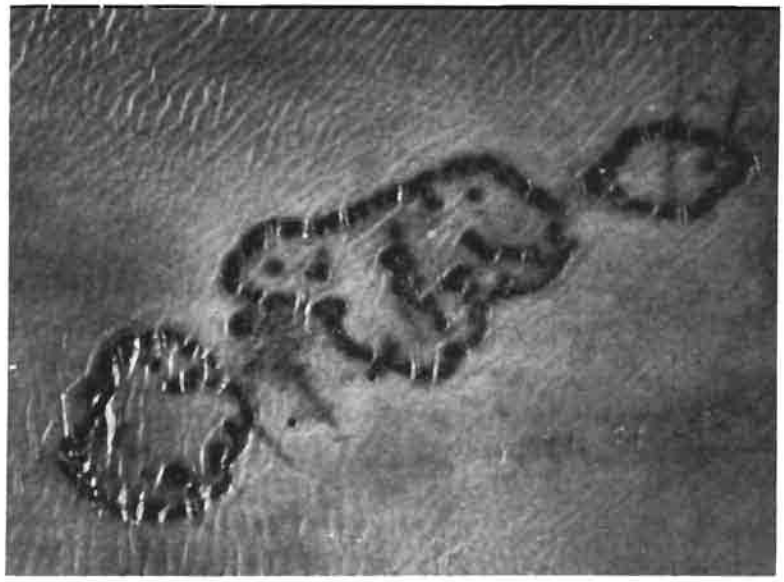

Fig. 7-1: *Lagenorhynchus acutus.* Tattoo lesion showing hyperpigmentation. (After Geraci and co-authors, 1979.)

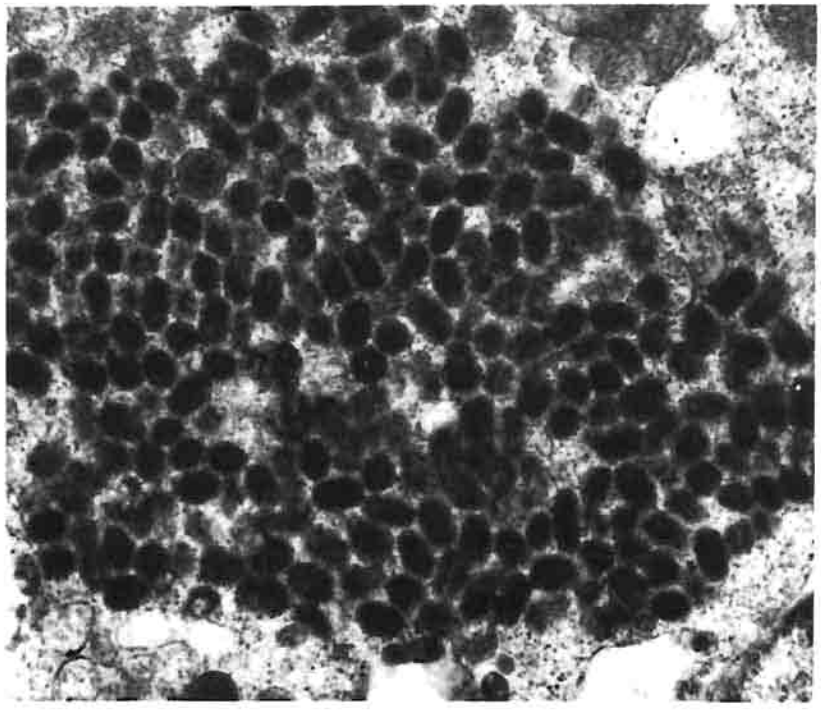

Fig. 7-2: *Tursiops truncatus.* Electron micrograph of tattoo lesion with pox virions in epidermal cells. (After Geraci and co-authors, 1979.)

Smith and Skilling (1979) report an enterovirus and an adenovirus from gray whale *Eschrichtius robustus* and sei whale *Balaenoptera borealis* respectively. These viruses are known to cause a variety of diseases in other mammals but their significance in cetaceans is unsure at this time.

A viral agent has also been suggested to be involved in hepatitis of porpoises (Ridgway, 1972a). He based his conclusions on the histologic nature of the non-supportive inflammation. Britt and Howard (1983) reported similar cases histologically but did not find viral inclusion bodies. This led them to question whether the lesions were due to a virus or simply part of a septicemic process.

Agents: Bacteria

Bacterial problems appear to be the cause of most of the serious diseases found in cetaceans. These diseases can occur alone or with infections by a larger parasite. Bacterial pathogens are a problem particularly in captive animals held in recirculating or closed water systems.

Howard and co-authors (1983a) list bacterial infections of integument, respiratory system, genito-urinary system, and as the cause of septicemia and abscesses. In their study the largest number of isolates of bacteria from cetaceans was 27 from pneumonic lungs, followed by 13 from septicemias, and 3 from abscesses (Table 7-1). The most common bacterial type isolated from cetaceans with bacterial pneumonia was *Pseudomonas* sp. (Table 7-1). The lungs may also be affected in animals with septicemia, which can result in death from mellioidiosis. In Hong Kong, an epidemic of mellioidiosis resulted in the loss of 24 *Tursiops gilli* and 4 pilot whales (Medway, 1980). In those cases the infectious organism, *Pseudomonas pseudomallei*, probably gained entry via the respiratory tract.

Pseudomonas aeruginosa was isolated from a *Tursiops truncatus* which also died of broncho-pneumonia and probable septicemia (Diamond and co-authors, 1979).

Greenwood and Taylor (1977, 1978b, 1979) reviewed the causes of death in captive, small cetaceans from Europe and South Africa in 1976, 1977, and 1978. They list bacteria as cause of death in 72, 27 and 67% respectively in 63 dolphins. *Staphylococcus pyogenes* was the most numerous organism isolated (7) followed by *S. aureus* (4).

Schroeder (pers. comm.) sampled 10 *Tursiops truncatus* and 2 *T. gilli* in Hawaii and isolated 3 species of *Vibrio* from both fecal and blowhole swabs in all animals examined (Table 7-1). This finding is disturbing to those who come in close contact with dolphins in captivity since bacteria of the genus *Vibrio* are known to cause disease in man, thus *Vibrio parahaemolyticus* has long been known to cause foodborne gastroenteritis associated with seafood, and *V. alginolyticus* has been implicated in septicemia and death (Spark, 1979; Tangredi and Medway, 1980).

Bacterial agents reported from cetaceans obviously play a major role in disease proliferation within a given population. Although very little is mentioned in the literature about enterotoxemia infections, it would be surprising to find that they do not play an important part in future findings concerning cetaceans. However, to date, no definitive studies have been carried out on anaerobic bacteria.

Table 7-1
Bacterial isolates from Cetacea (Compiled from the sources indicated)

Cetacean	Bacterial isolates	# of isolates	Source
Killer whale	*Pseudomonas* sp.	1	Howard and co-authors (1983a)
	Beta-hemolytic streptococcus	1	Howard and co-authors (1983a)
	Edwardsilla sp.	1	Howard and co-authors (1983a)
	Salmonella sp.	1	Howard and co-authors (1983a)
	Salmonella sp.	1	Ridgway (1979)
Pilot whale	*Pseudomonas* sp.	1	Howard and co-authors (1983a)
	Erysipelothrix rhusiopathiae	1	Howard and co-authors (1983a)
	Klebsiella pneumoniae	1	Howard and co-authors (1983a)
	Edwardsiella sp.	1	Howard and co-authors (1983a)
	Salmonella sp.	1	Howard and co-authors (1983a)
	Vibrio parahaemolyticus	1	Hall and co-authors (1971)
	Pseudomonas pseudomallei	4	Medway (1980)
Pacifice white-sided dolphin	*Pseudomonas* sp.	7	Medway (1980)
	Klebsiella pneumoniae	1	Medway (1980)
	Beta-hemolytic streptococcus	2	Medway (1980)
	Hemolytic staphylococcus	1	Medway (1980)
	Edwardsiella sp.	2	Medway (1980)
	Pseudomonas sp.	1	Medway (1980)
	Aeromonas sp.	1	Medway (1980)
	Aeromonas sp.	1	Medway (1980)
	Neisseria mucosa	2	Vedros and co-authors (1973)
Atlantic white-sided dolphin	*Vibrio alginolyticus*		Tangredi and Medway (1980)
Pacific bottlenose dolphin	*Pseudomonas pseudomallei*	24	Medway (1980)
	Vibrio parahaemolyticus	2	Schroeder (pers. comm.)
	V. alginolyticus	2	Schroeder (pers. comm.)
	Vibrio sp.	2	Schroeder (pers. comm.)
Atlantic bottlenose dolphin	*Salmonella* sp.	2	Howard and co-authors (1983a)
	Hemolytic staphylococcus	2	Howard and co-authors (1983a)
	Erysipelothrix rhusiopathiae	1	Howard and co-authors (1983a)
	Vibrio alginolyticus	1	Colgrove and co-authors (1975)
	V. parahaemolyticus	12	Schroeder (pers. comm.)
	V. alginolyticus	12	Schroeder (pers. comm.)
	Vibrio sp.	12	Schroeder (pers. comm.)
	Aeromonas hydrophila	2	Schroeder (pers. comm.)
	Citrobacter sp.	1	Schroeder (pers. comm.)
	Pseudomonas aeruginosa	1	Diamond and co-authors (1979)
	Staphylococcus pyogenes	7	Greenwood and Taylor (1977, 1978b, 1979)
	S. aureus	4	Greenwood and Taylor (1977, 1978b, 1979)
Common dolphin	*Pseudomonas* sp.	9	Howard and co-authors (1983a)
	Klebsiella sp.	3	Howard and co-authors (1983a)
	Edwardsiella sp.	1	Howard and co-authors (1983a)
	Neisseria mucosa	3	Vedros and co-authors (1973)

Agents: Fungi

Reports of systemic mycotic infections from cetaceans are not unusual. Sweeney and co-authors (1976) list 27 cases represented by 7 genera of fungi. All cases were characterized by pulmonary involvement. Numerous reports by other workers (Pier and co-authors, 1970; Migaki and co-authors, 1971; de Vries and Laarman, 1973; Greenwood and Taylor, 1978b, 1979; Ridgway, 1979; Buck, 1980; Dunn and co-authors, 1982) add additional host species as well as another genus of fungi (Table 7-2). Systemic fungal disease has been reported from wild, stranded (single) and captive animals. The fungi

Table 7-2
Fungal reports from cetacea (Compiled from the sources indicated)

Fungus	Host	Source
Nocardia		
N. braziliensis	*Tursiops gilli*	Sweeney and co-authors (1976)
N. caviae	*Tursiops gilli*	Sweeney and co-authors (1976)
N. asteroides	*Globicephala macrorhyncha*	Sweeney and co-authors (1976)
	Orcinus orca	Sweeney and co-authors (1976)
	Pseudorca crassidens	Sweeney and co-authors (1976)
	Stenella longirostris	Sweeney and co-authors (1976)
	Phocoena phocoena	Sweeney and co-authors (1976)
N. paraguayensis	*Tursiops truncatus*	Sweeney and co-authors (1976)
N. sp.	*Kogia breviceps*	Sweeney and co-authors (1976)
Actinomyces		
A. bovis	*Tursiops truncatus*	Sweeney and co-authors (1976)
Candida		
C. albicans	*Steno bredanensis*	Sweeney and co-authors (1976)
	Tursiops truncatus	Sweeney and co-authors (1976)
	Orcinus orca	Sweeney and co-authors (1976)
	Globicephala malaena	Dunn and co-authors (1982)
	Delphinapterus lucas	Dunn and co-authors (1982)
C. humicola	*Tursiops truncatus*	Buck (1980)
C. parapsilosis	*Tursiops truncatus*	Buck (1980)
C. pelliculosa (var.)	*Tursiops truncatus*	Buck (1980)
C. cylindrica	*Tursiops truncatus*	Buck (1980)
C. tropicalis	*Tursiops truncatus*	Buck (1980)
Aspergillus		
A. flavis	*Tursiops truncatus*	Buck (1980)
A. fumigatus	*Tursiops truncatus*	Buck (1980)
A. sp.	*Monodon monoceros*	Buck (1980)
	Kogia breviceps	Buck (1980)
Zygomycetes		
Entomophthora coronata	*Tursiops truncatus*	Buck (1980)
Mucor sp.	*Tursiops truncatus*	Buck (1980)
Blastomyces		
B. dermatitidis	*Tursiops truncatus*	Buck (1980)
Lobomycosis		
Loboa loboi	*Sotalia guianensis*	de Vries & Laarman (1973)
	Tursiops truncatus	Migaki and co-authors (1971)

appear to be introduced into the aquatic environment through natural means such as soil runoff. They are known opportunists that become symptomatic when the host is compromised. This infectivity is usually related to a variety of causes such as suppression of immunity within hosts that are debilitated from chronic primary disease or from stress, prolonged stress that occurs under various conditions of confinement, repeated movement and handling, exposure to unfamiliar environments, and inferior husbandry management (Sweeney and co-authors, 1976).

Given the above factors, one would suspect that systemic mycotic diseases play a minor role in their affect on populations. The organisms appear to be a normal part of the flora in ocean waters throughout the world. Sweeney and co-authors (1976) list 6 cases of nocardiosis from 5 species of Cetacea all from the island of Oahu, Hawaii (Figs 7-3 and 7-4). They state that between 1957 and 1961 the disease was enzootic in dairy cattle on the

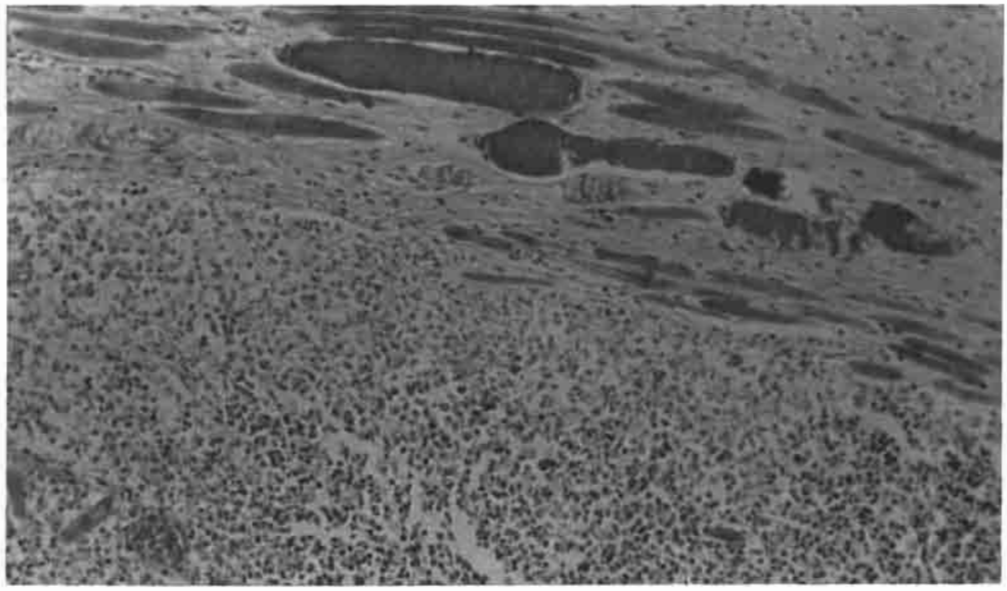

Fig. 7-3: *Globicephala macrorhynchus. Nocardia* sp. in muscle. (Photo: Dr. Jay Sweeney.)

island. The sample of 6 included 4 captured animals that had been kept locally from 7 months to 4 yr. The other 2 animals had beached and died. The authors do not list other disease complications found at necropsy. If the 2 stranded animals were typical, they were probably suffering from a combination of other problems.

Candidasis is a primary or secondary infection usually involving *Candida albicans.* Clinical manifestations of this disease are extremely varied, ranging from acute, subacute, and chronic, to episodic. Yeasts of the genus *Candida* have been implicated in the deaths of several species of cetaceans in captivity, including the harbor porpoise *Phocoena phocoena* (Spotte and co-authors, 1978); Atlantic bottlenose dolphin *Tursiops truncatus* (Nakeeb and co-authors, 1977); long finned pilot whale *Globicephala melaena* (Buck, 1980); and killer whale *Orcinus orca* (Ridgway, 1979). Dunn and co-authors (1982) describe the successful treatment of disseminated candidasis in an Atlantic bottlenose dolphin and the

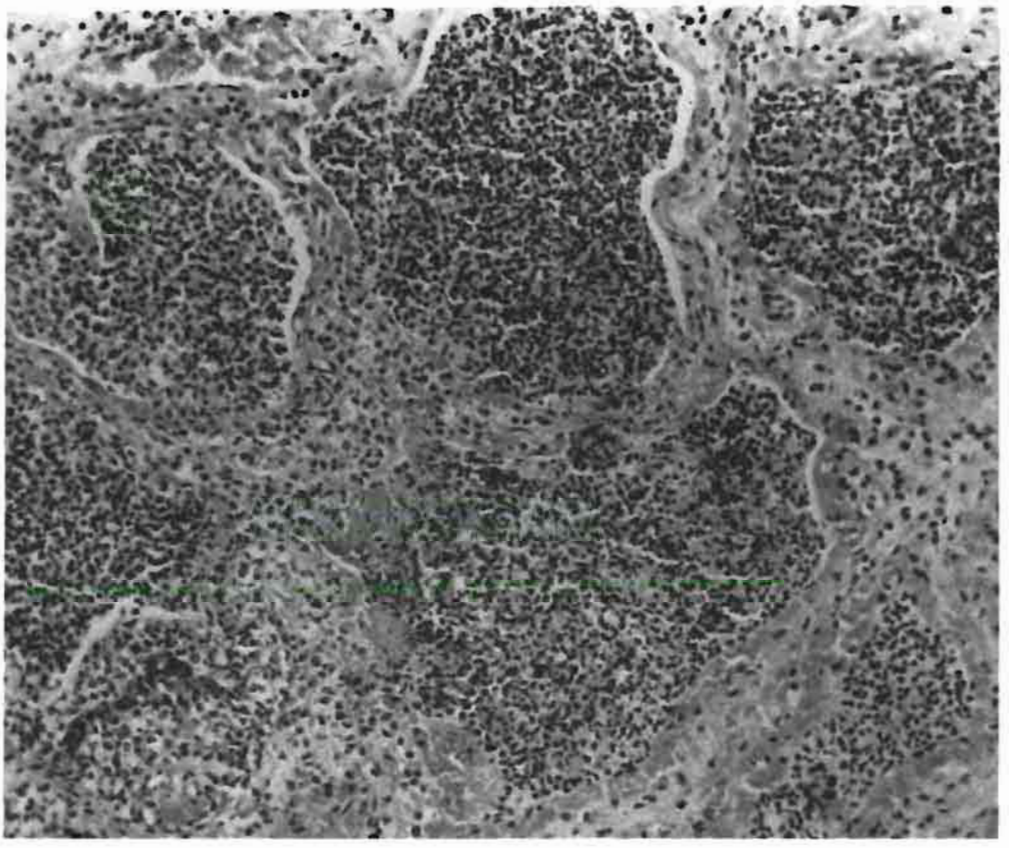

Fig. 7-4: *Globicephala macrorhynchus. Nocardia* sp. in lung. (Photo: Dr. Jay Sweeney.)

cessation of *C. albicans* shedding by a beluk-ha whale *Delphinapterus leucas.* They observed that the gastrointestinal tract of healthy captive cetaceans contain few or no *C. albicans* cells and further suggest that most of *C. albicans* infections in captive cetaceans begin in the oral cavity and esophagus (although this has not been confirmed by controlled studies). The combination of sequences leading to symptoms presumably start with the oral cavity being bathed in chlorinated pool water. The normal bacterial flora of the oral cavity are more sensitive to the effects of chlorine than *C. albicans.* The decreased bacterial numbers allow the proliferation of *Candida.* Damage to the oral or esophageal mucosa by fish spines may initiate rapid dissemination of the yeast throughout the body. A review of the behavior of infected animals shows that lesions in the upper digestive tract are responsible for some of the clinical signs, such as anorexia, headshaking, and apparent abdominal cramping.

Esophageal lesions are a useful diagnostic tool because when the lining of the esophagus sloughs, leaving the underlying tissue raw and sensitive, the animal may stop eating abruptly, or may just mouth the food due to pain in swallowing.

Cutaneous lesions appear initially as small, slightly raised discolorations of the skin (Nakeeb and co-authors, 1977). The lesions are warmer to the touch than surrounding areas, and often lack the slick texture of normal cetacean skin. Successful treatment of

Fig. 7-5: *Tursiops truncatus*. Lobo's disease or keloidal blastomycosis on external surface. (Photo: Dr. Sam Ridgway.)

candidasis is reported with Ketoconazole*, given in food b.i.d. at a dosage of 5 mg kg^{-1} of body weight. Treatment continued over approximately 3 months returned a dolphin to a healthy state with no evidence of regression in clinical status (Bossart and Bossart, 1982).

Lobomycosis, or Lobo's disease (Keloidal blastomycosis), is a chronic granulomatous infection of the skin caused by the fungus *Loboa loboi* (Fig. 7-5). Originally known only from man, this disease was reported in 1971 from a feral *Tursiops truncatus* in Florida by Migaki and co-authors. An additional case was described in 1973 by de Vries and Laarman from a river dolphin (*Sotalia* sp.) caught in the estuary of the Surinam River in South

* Ketoconazole (introduced in 1981 as Nizoral by Janssen Pharmaceutics, Inc.), Janssen R & D Inc., New Brunswick, N.J., USA

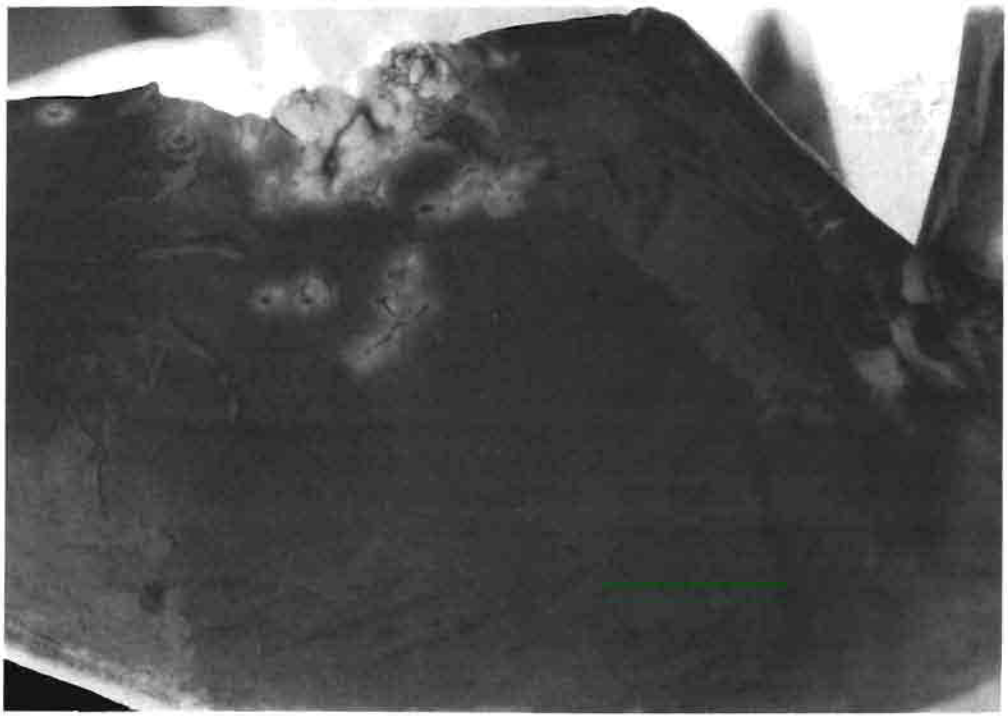

Fig. 7-6: *Tursiops truncatus*. Lobo's disease on flipper showing typical white patches. (Photo: Dr. J. P. Schroeder.)

America. Also, Woodard (1972), Caldwell and co-authors (1975), Van Heel (1976) and Schroeder (1983, pers. comm.) have reported lobomycosis. The cutaneous lesions usually appear as extensive white patches or raised bumps (Fig. 7-6). These are large, histiocytic granulomas in the dermis resulting in severe acanthosis. If unchecked the disease causes loss of mobility and probably death. Van Heel (1976) reports successful treatment of the disease in *T. truncatus* with Miconazole** given at 18 mg kg^{-1} of body weight.

In general, mycotic diseases can be viewed clinically as a single class of disease, the specific organism involved being more of geographical, than medical, relevance. All disease manifestations have a similar clinical appearance with lesions of candidiasis being by far the most commonly encountered symptoms, and the only ones commonly associated with cutaneous manifestations. The disease condition is nearly always chronic with progressive wasting and often the absence of a specific clinico-pathologic diagnosis. A diagnosis can be made either with radiographic visualization of pulmonary granulomatous disease, or by cytologic examination of skin scrapings, sputum or gastric lavage specimens.

As mentioned above, events leading to suppression of immunity are the cause/effect basis for infection; thus good historical information is helpful in the diagnosis and necessary for subsequent prevention. Neonatal cetaceans are particularly high-risk candidates, especially when maternal feeding is impaired.

** Janssen Pharmaceutics, Inc USA

DISEASES CAUSED BY PROTISTANS

Little is known of protozoan populations in cetaceans. To date no enteric forms have been reported. Whether this is due to lack of adequate study or absence of organisms is not clear at this point. Ciliates of the class Holotricha have been reported by Woodard and co-authors (1969) from *Tursiops truncatus*. Blowhole swabs from 5% of newly captured individuals contained large, ciliated protozoans in addition to a dark mucoid exudate. At necropsy (1 of 24) Woodard and co-authors found 'large quantities of yellowish, solidified puriform material in the larynx, trachea and stem bronchi'. Histologically, the same authors reported focal areas of suppurative pneumonia and hemorrhage attributed to the presence of innumerable ciliates in the surrounding parenchyma tissue. However, most of the parasites were found free within the alveolar spaces. This report is similar to that from a captive *T. truncatus* in Hawaii and San Diego, USA. During exhalation large pieces of mucus lining were expelled from the blowhole; microscopic examination of the tissue

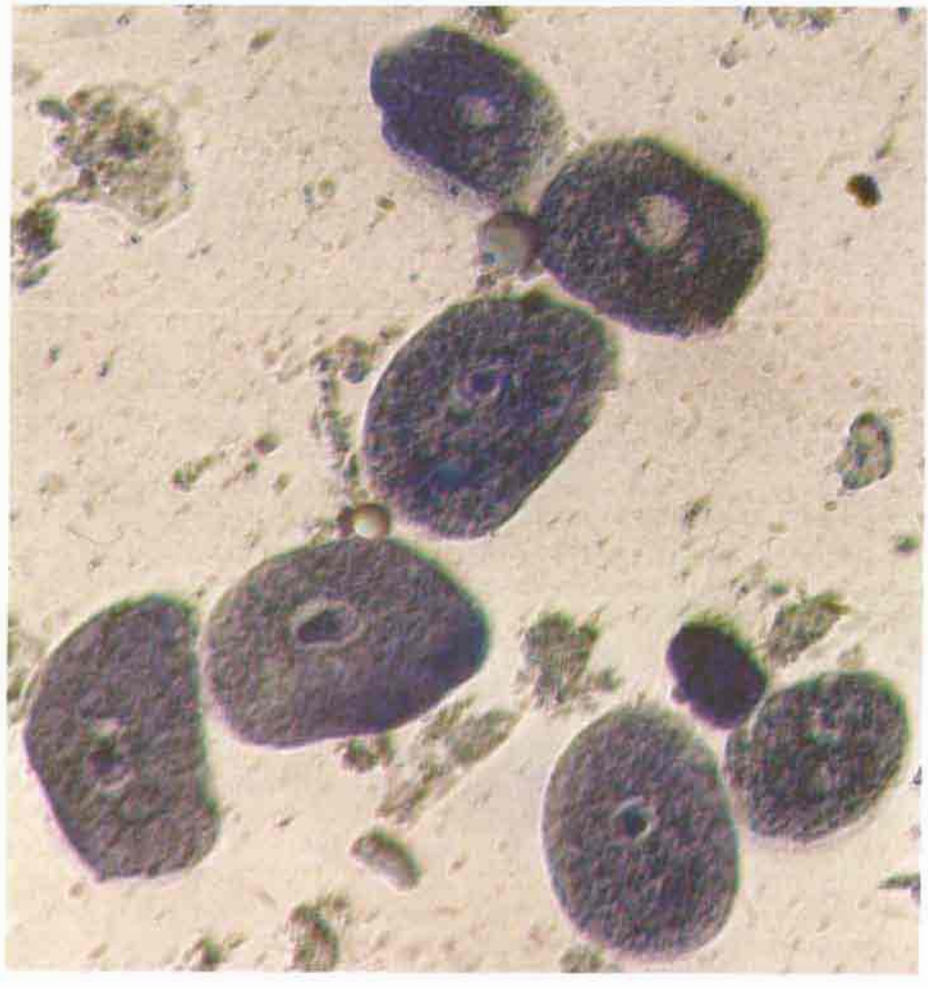

Fig. 7-7: *Tursiops truncatus*. Ciliates from blowhole exhalent. (Photo: Dr. C. Fenner.)

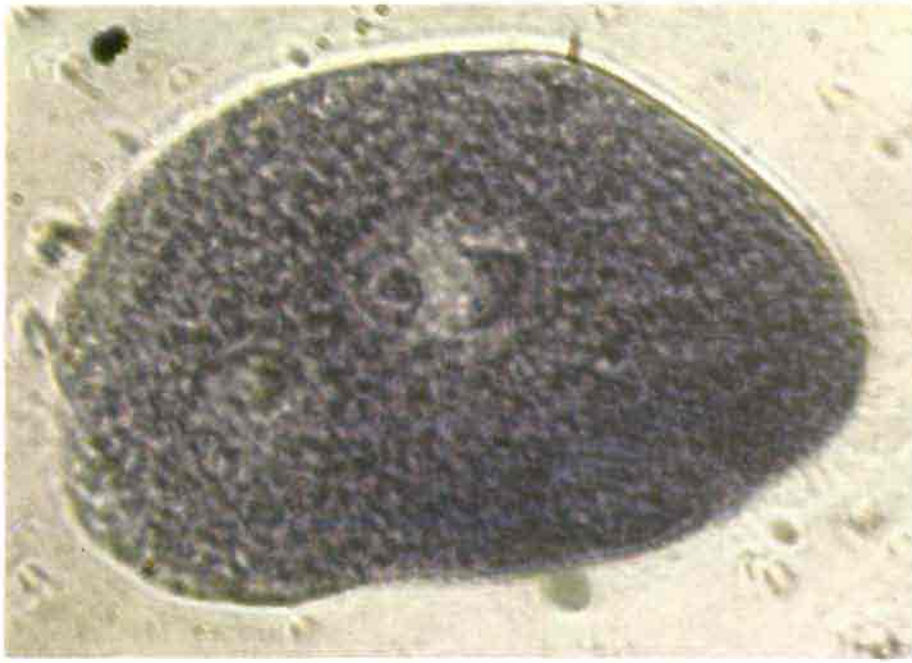

Fig. 7-8: *Tursiops truncatus.* Higher magnification of ciliated protozoans. (Photo: Dr. C. Fenner.)

revealed massive numbers of holotrich ciliates (Figs 7-7 and 7-8) (Fenner, pers. comm.). An unidentified ciliate from a skin lesion of a single bottlenose dolphin in Florida was reported by Howard and co-authors (1983a). The ciliates were large (40 to 80 mm in diameter) and found primarily in the upper dermis where they caused a sloughing of the epidermis. Found only in ulcerated tissue, these ciliates are probably opportunistic rather than active tissue invaders.

Another protozoan, a species of *Sarcocystis,* has been reported from the skeletal muscle of striped dolphin *Stenella coeruleoalba* (Dailey and Stroud, 1978), pilot whale *Globicephala melaena* and sperm whale *Physeter macrocephalus* (Fig. 7-9) (Cowan, 1966; Owen and Kakulas, 1967). These are probably incidental findings with little or no effect on the cetacean hosts.

It is apparent that additional work needs to be carried out on protozoans infecting cetaceans before a conclusion can be drawn regarding their role in marine mammal diseases.

DISEASES CAUSED BY METAZOANS

Agents: Trematoda

Digentic trematodes, reported from cetaceans, number approximately 9 families and 37 species. They cause disease in 3 body areas: air sinuses and head, digestive tract, and internal organs.

Digenea in air sinuses and head region are represented by the genus *Nasitrema* (Ozaki, 1935). Currently, the genus contains 9 species (Neiland and co-authors, 1970) all

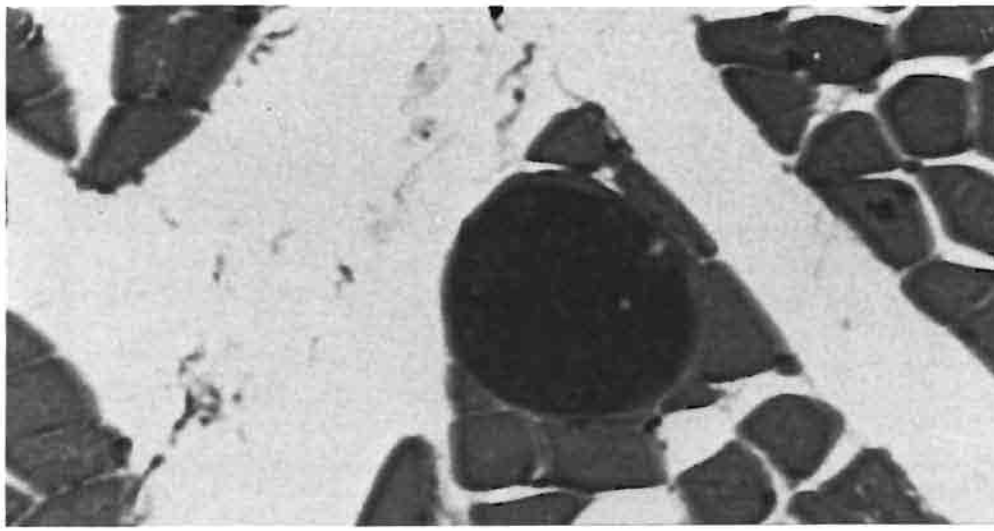

Fig. 7-9: *Stenella coeruleoalba*. Sarcocyst in skeletal muscle. (After Dailey and Stroud, 1978.)

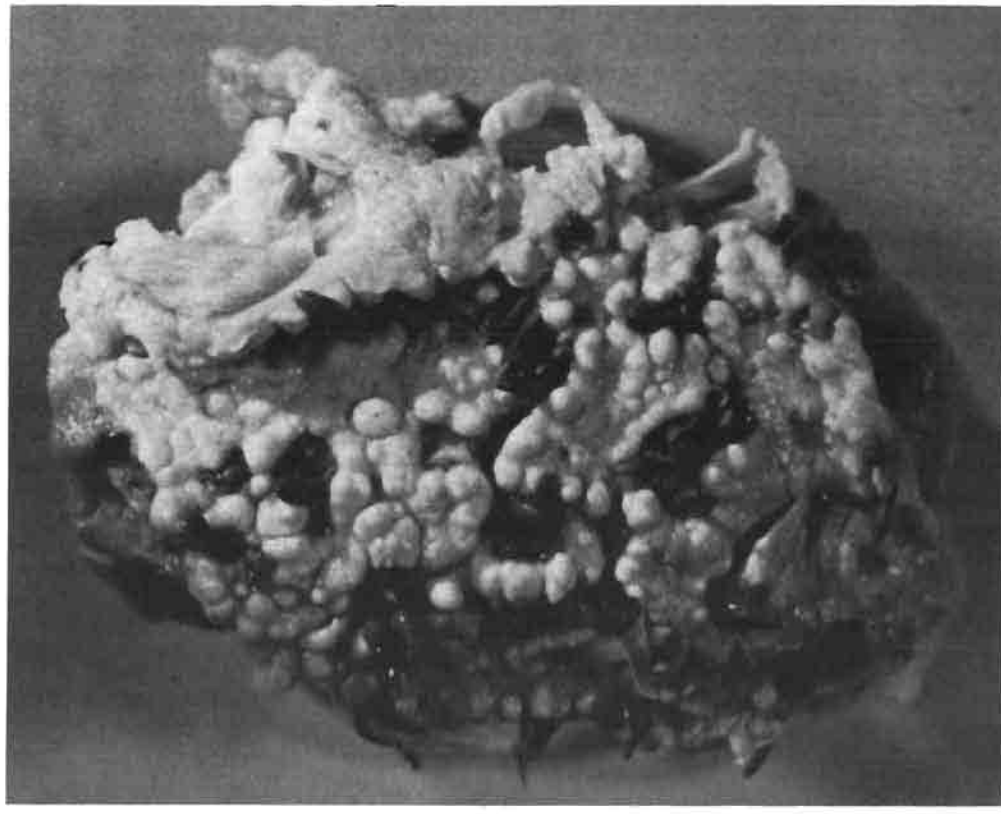

Fig. 7-10: *Delphinus delphis*. *Nasitrema* sp. in tissue of air sinuses. (Original.)

of which infest the head of small Odontocetes. Once in the cetacean, the worms are apparently highly mobile. Dailey and Walker (1978) found the worms in a high percentage of stranded cetaceans surveyed along the Southern California coastline. Massive infestations were seen with worms completely occluding the Eustachian tube, as well as invading tissue of the air sinuses (Fig. 7-10). Worms had also been sucked into the lung, causing large abscesses around the parasite (Fig. 7-11). In that same report, 60 stranded *Delphinus delphis* collected over a 100-mile coastline were compared with 31 *D. delphis* taken at sea. The results clearly indicated that trematodes of the genus *Nasitrema* were the primary cause of single strandings of cetaceans in the area. Adult *Nasitrema* were recovered from brain tissue in 26 of 40 individuals. The worms were abnormally small but gravid. Brain lesions occurred as gross discolored areas, most commonly on and in the cerebral hemispheres (Fig. 7-12). In microscopic sections the lesions revealed a fatty necrotic nature with massive numbers of fluke eggs (Fig. 7-13). Additional mechanical damage to

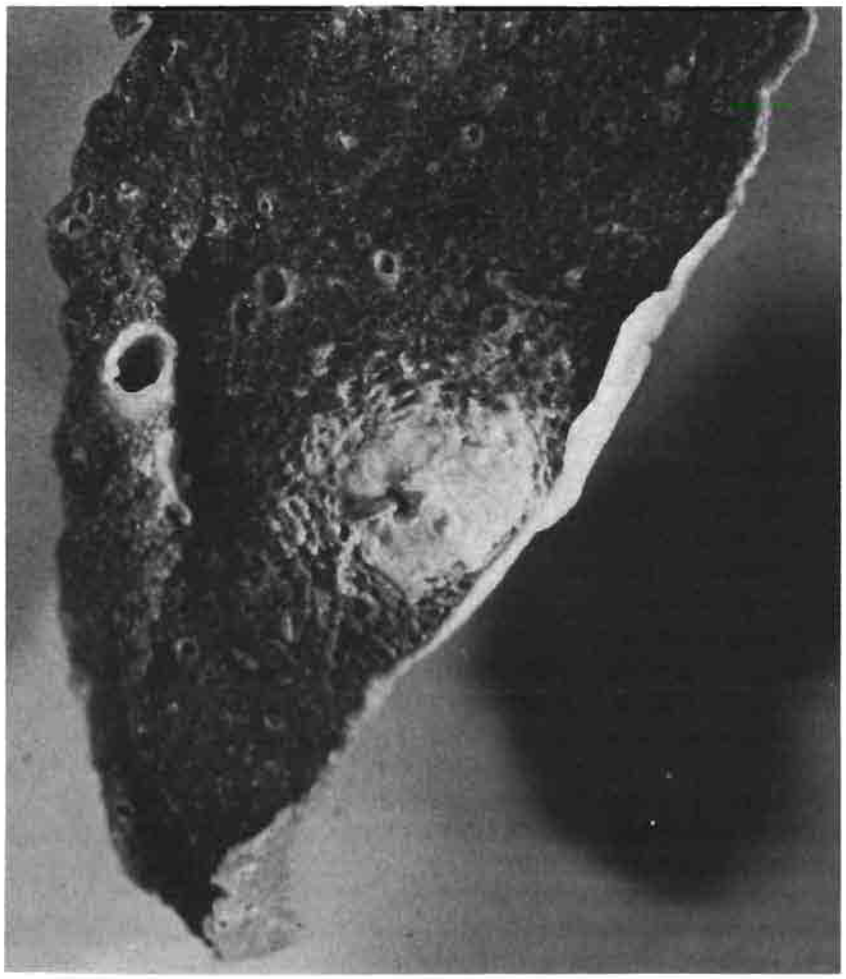

Fig. 7-11: *Delphinus delphis. Nasitrema* sp. in lung with surrounding abscess. (Original.)

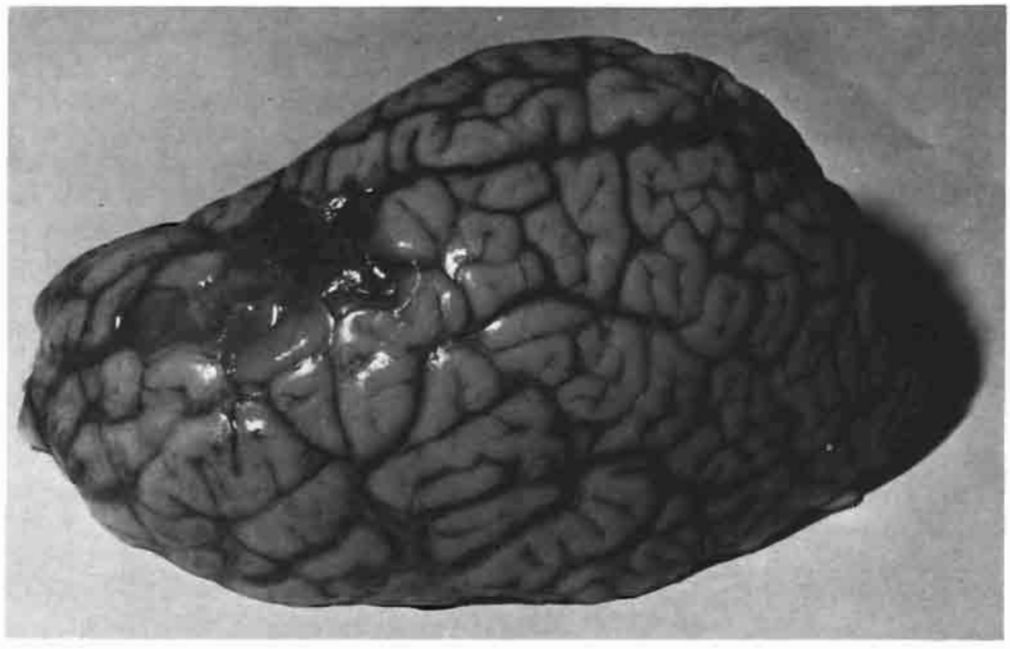

Fig. 7-12: *Delphinus delphis*. Necrotic lesion caused by *Nasitrema* sp. in brain. (Photo: William Walker.)

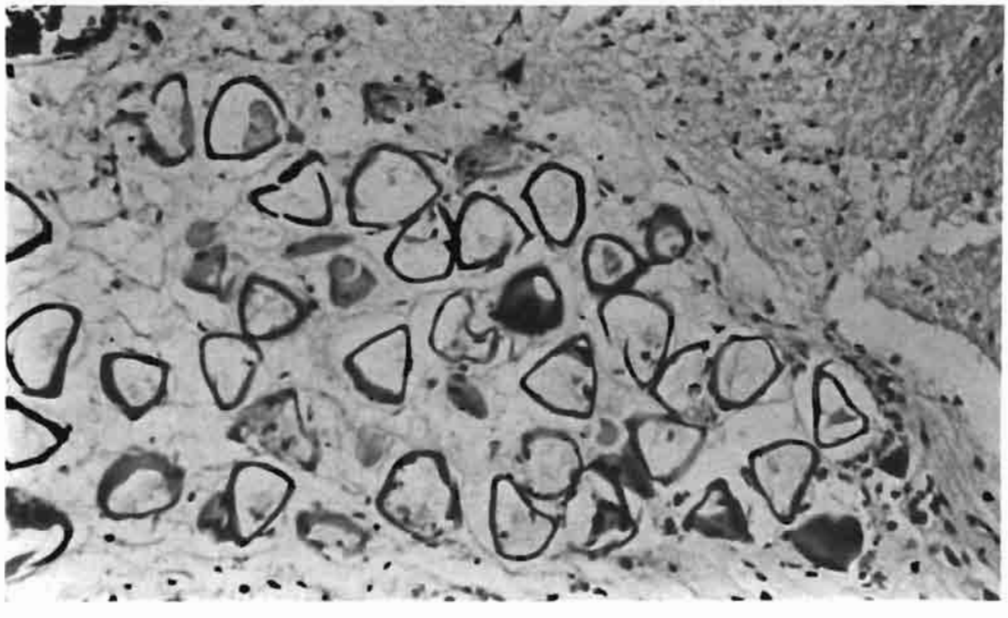

Fig. 7-13: *Delphinus delphis*. Eggs of *Nasitrema* in brain lesion. (Photo: William Walker.)

the brain resulted as the worms migrated through the tissue (Fig. 7-14). Such lesions were found in the brains of the common dolphin *Delphinus delphis*, Pacific white-sided dolphin *Lagenorhynchus obliquidens*, Dall porpoise *Phocoenoides dalli*, and northern right whale dolphin *Lissodelphis borealis* (Dailey and Walker, 1978). *Nasitrema globicephalae* was found in the umbilical artery of a term fetus removed from mass-stranded pilot whales on

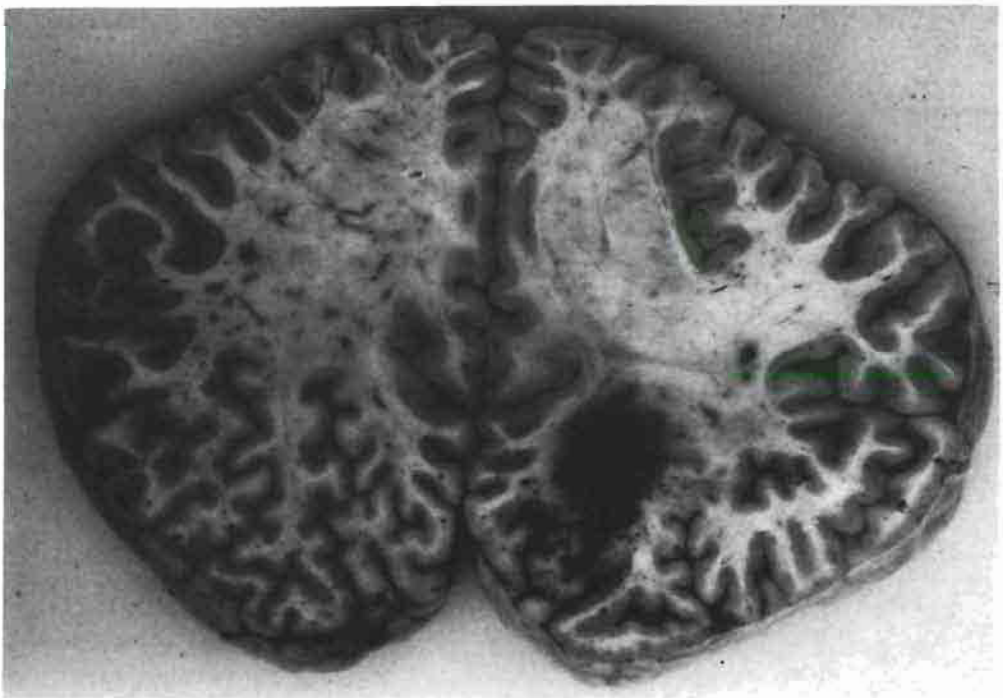

Fig. 7-14: *Delphinus delphis*. Gross lesion caused by migrating *Nasitrema* sp. in brain. (Photo: William Walker.)

San Clemente Island (Dailey, own observ.). This was an adult gravid worm active and healthy at the time of recovery. The explanation for this finding is still unclear. The possibility of a direct prenatal infection is very doubtful, given the traditional trematode life cycle. A more likely conclusion would be the active migratory habits of the members of this genus.

Preliminary work on the transmission of *Nasitrema* sp. indicates that the cycle is probably not direct. Miracidial development lasts from 16 to 58 days, depending on the individual egg. Samples collected at the same time and maintained in identical conditions vary widely in developmental time. This could possibly be an adaptation for dispersion, but at present this is speculation. The miracidia have eye spots, are ciliated, and are active swimmers (Fig. 7-15). These attributes would indicate that the cycle is not direct but involves an intermediate host or hosts (Dailey, unpubl.).

Treatment of *Nasitrema* in the bottlenose dolphin *Tursiops truncatus* has been most successful with Bithional®. This drug is used for treating human lungfluke *(Paragoneous westermanni)* infections in the Orient.

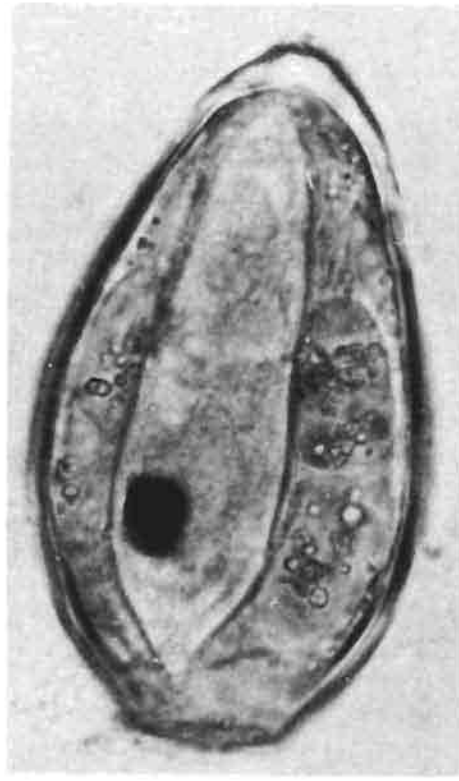

Fig. 7-15: *Nasitrema* sp. Ciliated miracidium. (Original.)

Digenea in the digestive tract of cetaceans are represented by 10 genera *(Leucasiella, Onthosplanchnus, Odhnerilla, Synthesium, Pholeter, Echinochasmus, Hadwenius, Galactosonum, Ogmogaster, Braunina)* (Dailey and Brownell, 1972). Disease problems caused by these parasites are not referred to frequently in the literature. Although the flukes are widespread in many species of cetaceans, reports on pathogenicity vary. Worms found in the lumen of the gut (all but *Pholeter* and *Braunina,* which occur primarily in the stomach) do not seem to affect the health of the host. *Pholeter gastrophilus* has been listed from *Tursiops truncatus* (Zam and co-authors, 1971). The pathology of gastric trematodiasis resulting from *P. gastrophilus* was described by Woodard and co-authors (1969). Effects are similar to those described for *Braunina cordiformis,* a common parasite in fundic stomach, pyloric stomach, and/or ampulla of the duodenum (Delyamure, 1955; Schryver and co-authors, 1967; Ridgway, 1968; Johnston and Ridgway, 1969; Zam and co-authors, 1971). However, Woodard and co-authors (1969) did not believe that the parasite caused any serious pathological consequences. The parasites occur in large numbers, with some being walled off by a fibrous capsule (Simpson and Gardner, 1972).

A mixed infection of *Hadwenius mironovi* and *H. nipponicus* was reported from the harbor porpoise *Phocoena phocoena* by Dailey and Stroud (1978). As in previous infections, the worms were deeply embedded in the mucosa of the pyloric stomach and anterior duodenum, causing mild irritation characterized by hyperemia and hemorrhage

(Fig. 7-16). Although these trematodes cause some irritation, they cannot be considered a major disease problem in terms of causing large population losses. Digenea of internal organs are located almost exclusively in the liver, pancreas, or hepatopancreatic duct (11 genera), with the exception of 1 example from the lungs *(Hunterotrema)*. The family Campulidae contains 4 genera *(Campula, Lecithodesmus, Odhneriella, Zalophotrema)* found in the liver and pancreas. The diseases caused by these worms, and described most often, are those due to the genus *Campula* (Woodard and co-authors, 1969; Zam and co-authors, 1971; Dailey and Stroud, 1978). These parasites cause severe chronic cholangitis. Eggs, when free within the hepatic parenchyma, lead to severe chronic hepatitis. Fibrosis and chronic inflammation were found in the pancreatic ducts. Focal areas of both acute and chronic pancreatitis were also due to these trematodes and their eggs (Dailey and Stroud, 1978). Diseases caused by this group of parasites have the potential to affect various cetacean populations. They apparently are very successful parasites. A total of 7 species of *Campula* have been reported from species of *Phocoenoides, Stenella, Delphinus, Tursiops, Grampus, Phocoena, Neophocoena,* and *Globicephala.* In the larger whales 3 genera of liver flukes have been found in 7 genera: *Zalophotrema* and *Oschimarinella* in *Pyseter macrocephalus* and *Berardius bairdi* respectively; the baleen

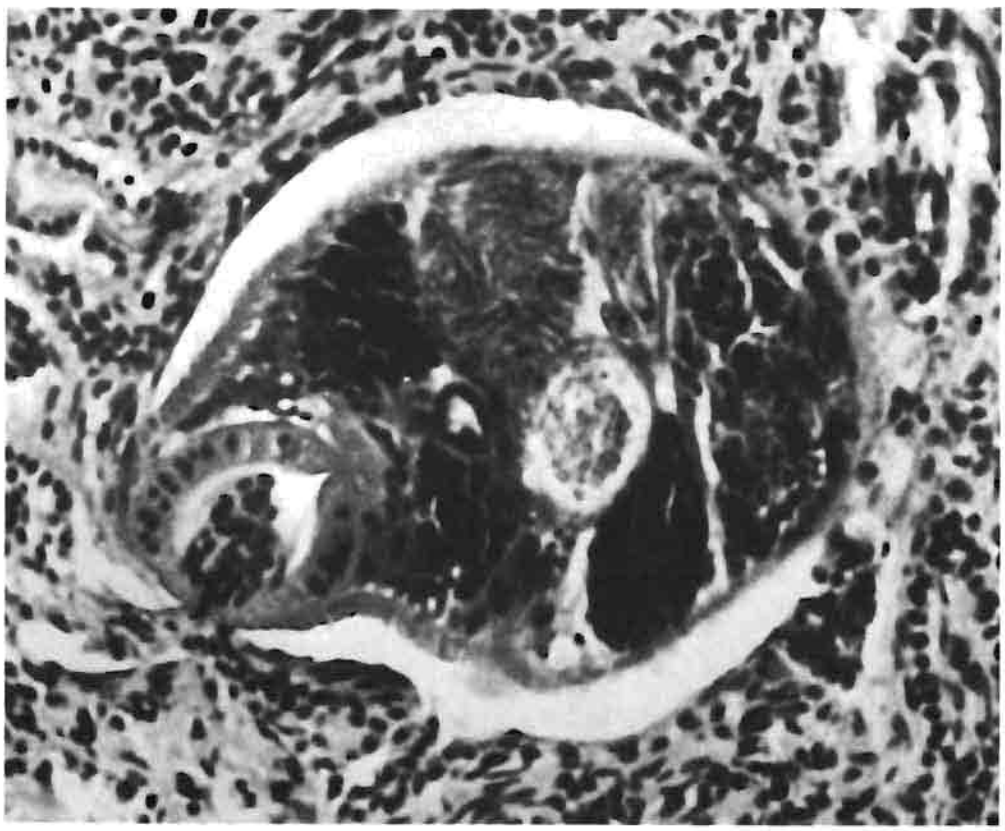

Fig. 7-16: *Phocoena phocoena. Hedwenius nipponicus* embedded in duodenal mucosa. (After Dailey and Stroud, 1978.)

whales *Balaena mysticetus, Balaenoptera acutorostrata, B. borealis, B. phipolus* and *Megaptera novaeangliae* are all infected by members of the genus *Lecithodesmus* (Dailey and Brownell, 1972). Zam and co-authors (1971) found 23 of 53 *T. truncatus* (43%) examined to have or have had hepatic and/or pancreatic trematodiasis. Liver flukes were also found in 2 of 17 *Stenella plagiodon* and 1 of 3 *Grampus griseus.* Although the histopathology has not been reported in the larger whales, we can probably assume the numbers infected and the type of pathology to be somewhat similar.

Transmission of these parasites, as with all other cetacean trematodes, has not been studied in detail. Experimental hatching of *Campula rochebruni* eggs (Dailey, unpubl.) show a ciliated miracidium without eyespots. Lack of eyespots may indicate a deep-water cycle with the intermediate host entering the feeding cycle through the deep scattering layer.

Pulmonary trematodiasis in cetaceans is restricted to the genus *Hunterotrema* found in the Amazon River dolphin *Inia geoffrensis.* The worms occur in the large main stem bronchi, where they may completely obstruct the passages with a mucoid exudate. As in *Nasitrema* infections, areas of cerebral necrosis, which caused hemiparesis and inability to maintain proper balance in the water, were found in connection with trematode ova in the brain (Woodard and co-authors, 1969).

Agents: Cestoda

Cestodes infesting Cetacea are from 3 families: Tetrabothriidae, Diphyllobothriidae and Phyllobothriidae. Literature research reveals that only a few tapeworms can be considered disease problems. Larval phyllobothriid worms are the most common tissue invaders found in cetaceans. *Phyllobothrium delphini* (Bosc, 1802) has been reported from nearly every species of Cetacea in all regions of the World's Oceans. In Odontocetes these are usually found in the subcutaneous fat where they appear as small white fluid-filled bladders, representing several species of worms (Delyamure, 1955; Testa and Dailey, 1977) (Fig. 7-17). No tissue response is noted surrounding the cysts in the blubber; however, just what damage may occur during migration from gut to blubber is unknown. Whether the cetacean represents a 'dead end' to the worm, which grows to adulthood in elasmobranch fishes, or whether it is the natural intermediate host, is an interesting, still unanswered question. If the cetaceans were the natural hosts, this would represent the only cycle where a helminth uses a warm-blooded animal to infect a cold-blooded definitive host.

The other common larval phyllobothriid cestode in cetaceans is *Monorygma grimaldii* (Moniez, 1889; Baylis, 1919). *M. grimaldii* has been recorded from the body cavity of numerous cetaceans (Dailey and Brownell, 1972). Delyamure (1955) lists the definitive hosts of this worm as sharks, probably of the genus *Scyllium.* He assumes that the cetacean is a reservoir host, with the intermediate and supplementary hosts unknown. *Monorygma* appears to represent only 1 type or species of larval worm. These cysts are found singly or in groups on the outer surfaces of mesentery membranes in the body cavity (Fig. 7-18). No information is available on the migration route from gut to infection site. Host reaction to this parasite is minimal in the body cavity; apparently little harm results to the cetacean.

Adult cestodes infecting the gut of both Odontocetes and Mysticetes are represented by 8 genera from 2 families: *Tetrabothrius, Anophryocephalus, Priapocephalus, Strobilo-*

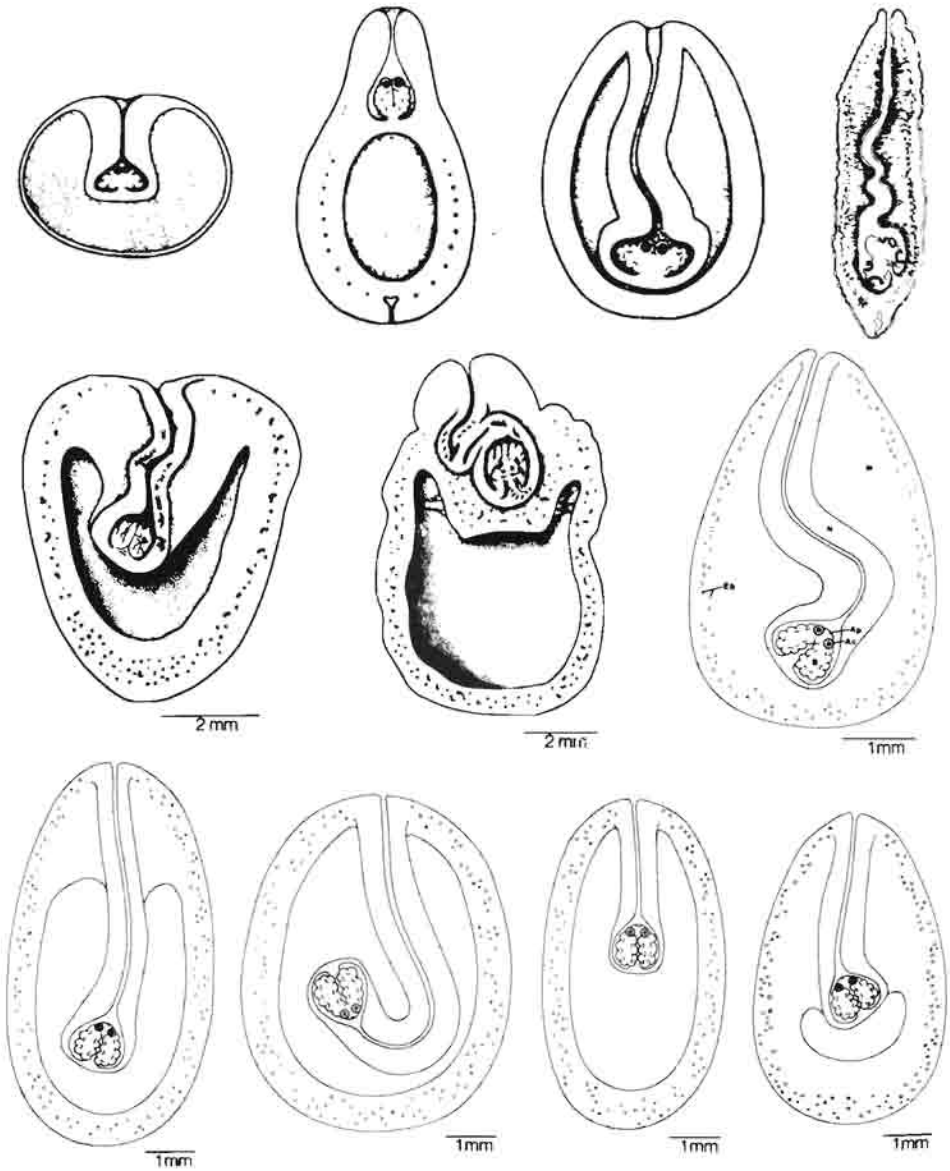

Fig. 7-17: *Phyllobothrium delphini.* Bladder types. (After Testa and Dailey, 1977.)

cephalus, Trigonocotyle from Tetrabothriidae; *Diphyllobothrium, Diplogonoporus* and *Hexagonoporus* from Diphyllobothriidae.

The prime site of infestation for these worms is the intestine, except for *Strobilocephalus,* which occurs in the rectum. Due to the large size of some of these cestodes infecting the whales, they cause most of the damage to the host by mechanical blockage of the gut lumen. *Balaenoptera borealis* taken in the Antarctic were found to be 93.3% infected with *Tetrabothrius wilsoni* Leiper and Atkinson in some regions (Vogelbein, 1981). These worms occurred in such massive numbers that it would seem impossible for

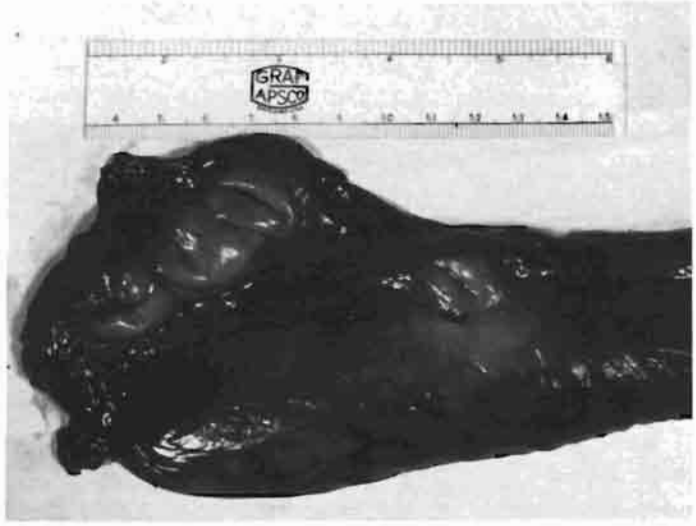

Fig. 7-18: *Delphinus delphis. Monorygma grimaldii* cysts in tissue. (Original.)

food to pass through the intestine (Dailey, own observ.) (Fig. 7-19). What effect these massive infestations have on the host is not clear. However, it was noted by the reviewer that, during the 1976–77 Japanese whaling season in the Antarctic, sei whales chased and ultimately harpooned were those that lagged in the pod. It is possible (although no controls are available) that these whales were energy-deprived due to heavy worm burdens. The tapeworms caused little or no inflammatory response at the attachment site (Dailey, own observ.).

In small Odontocetes of the genera *Stenella, Steno, Delphinus, Hyperodon, Mesoplodon,* and *Lagenorhynchus,* the rectal worm *Strobilocephalus triangularis* has been reported from both Atlantic and Pacific Oceans (Baer, 1955; Delyamure, 1955; Dailey and Brownell, 1972; Dailey and Perrin, 1973). This parasite apparently anchors its scolex in the colon wall when small and grows inside a fibrotic capsule formed by the host (Figs. 7-20 and 7-21). In the genus *Stenella,* Dailey and Perrin (1973) found extremely heavy infections in the last meter of gut. In 2 calves and 2 subadults examined, the lumen was nearly occluded and swollen to 3 or 4 times its normal diameter. The sample of *S. graffmani* was divided into 4 age classes on the basis of total length, developmental pattern and degree of sexual maturity (Fig. 7-22) (Perrin, 1970). The extent of bowel occlusion found indicates impaction of fecal movement. This speculation was born out by the fact that no adult *S. graffmani* were found infected with *Strobilocephalus triangularis* (Table 7-3). The effect of this parasite on populations of infected cetaceans through mortality is unknown but could be significant. To date we have no information on the life cycle of this cestode.

Agents: Nematoda

Nematodes are the most numerous helminth parasites infesting cetaceans. Three families (Pseudaliidae, Heterocheilidae, Crassicaudidae) containing 15 genera have been reported from both cetacean suborders in all oceans. The disease problems caused by

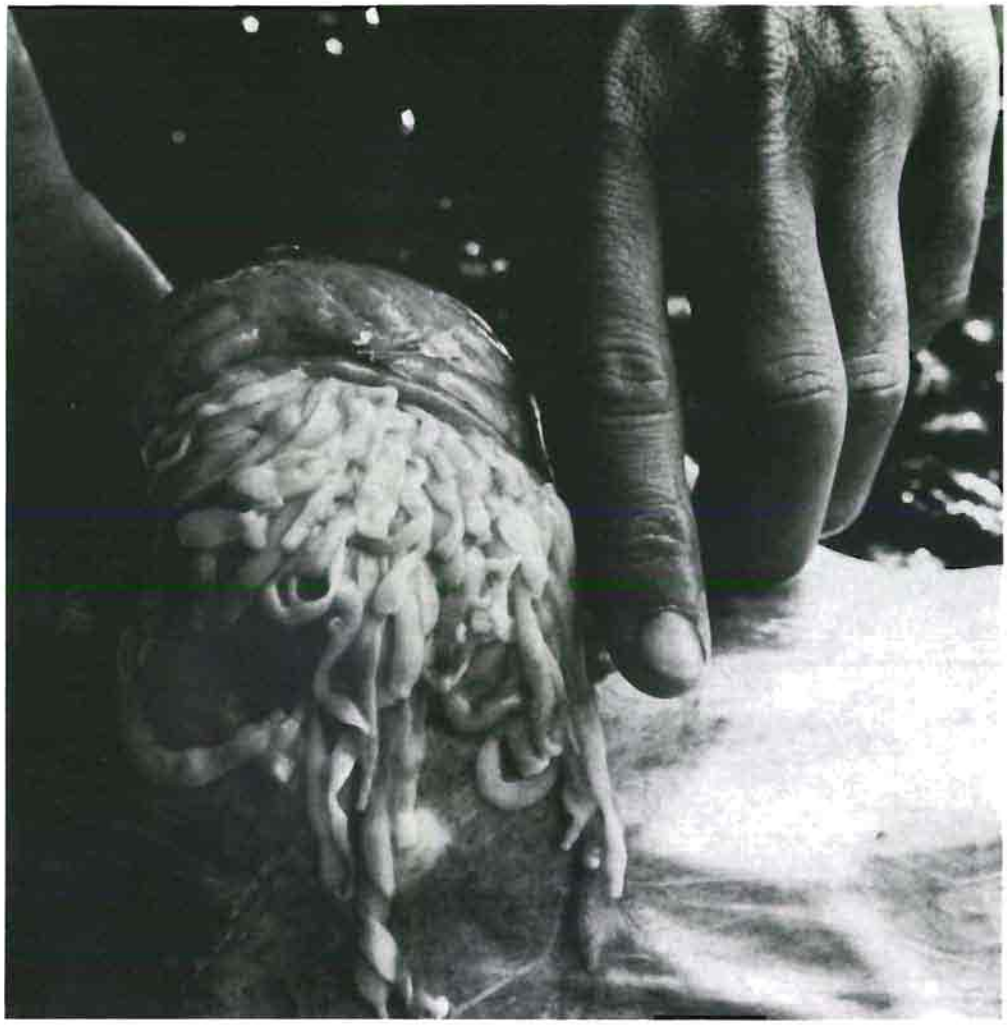

Fig. 7-19: *Balaenoptera borealis. Tetrabothrius wilsoni* filling the intestine. (Original.)

Table 7-3
Strobilocephalus triangularis in *Stenella graffmani* (After Dailey and Perrin, 1973)

Age class	Number examined	Number infected
Neonatals	5	0
Calves	14	3 (21.4 %)
Subadults	26	3 (11.5 %)
Adults	23	0
Total	68	6 (8.8 %)

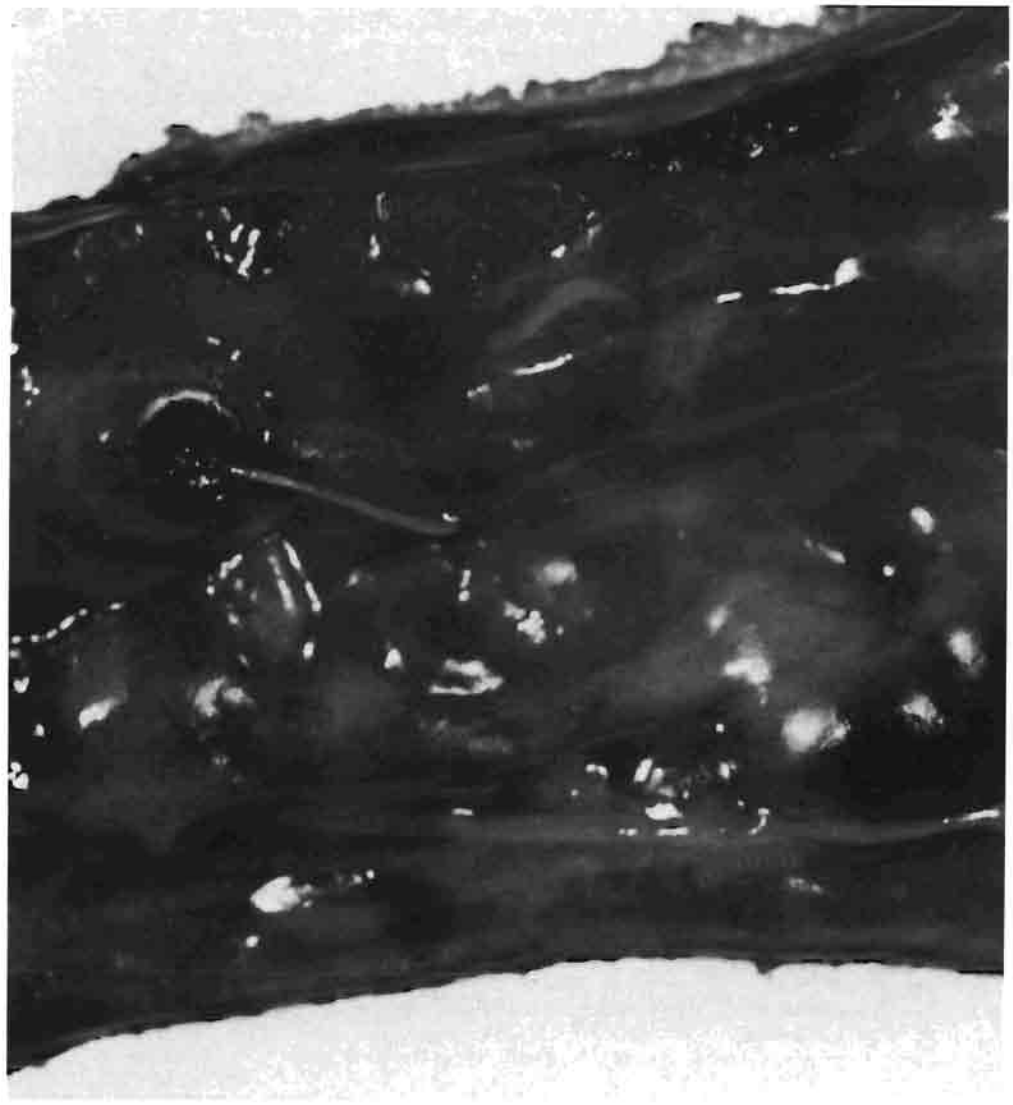

Fig. 7-20: *Stenella coeruleoalba. Strobilocephalus triangularis* with necrotic ulcer in colon. (After Dailey and Perrin, 1973.)

nematodes have to be considered a major factor in the mortality of cetacean stocks. The worms occur in 3 primary infestation sites in the host: thoracic cavity-head, stomach-intestine, tissues and organs.

According to Dailey and Brownell (1972), nematodes infesting lungs, pulmonary artery, auditory spaces and air sinuses of cetaceans comprise 8 genera of the family Pseudaliidae (*Delamurella, Halocercus, Otophocaenurus, Pharurus* [syn. *Torynurus*], *Pseudalius, Pseudostenurus, Skrjabinalius, Stenurus*). Lungworm disease and its associated pneumonia is particularly important. Pseudaliids cause granulomatous inflammation

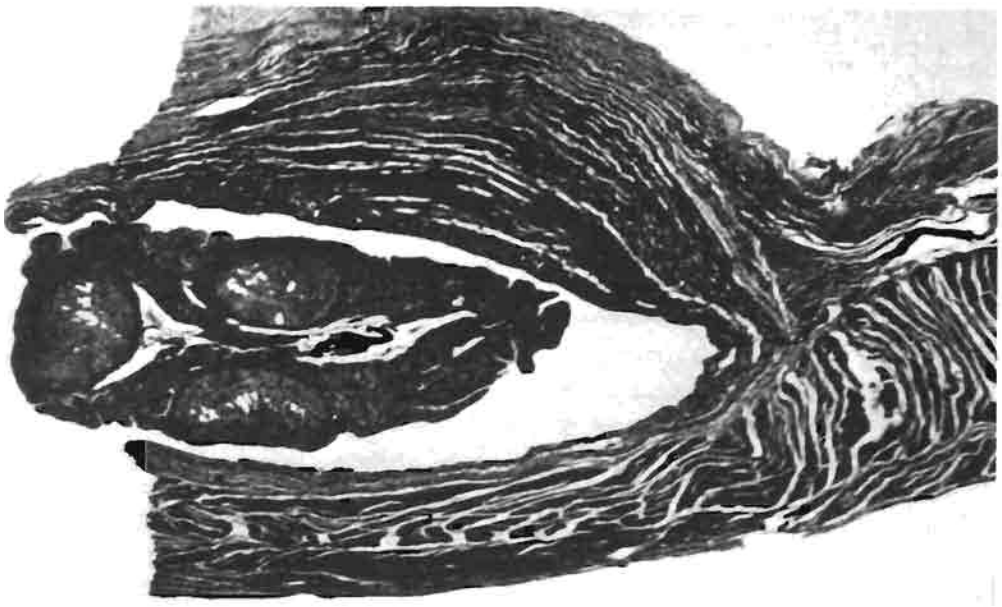

Fig. 7-21: *Stenella graffmani.* Section of rectal tissue showing scolex of *Strobilocephalus triangularis* in capsule. (After Dailey and Perrin, 1973.)

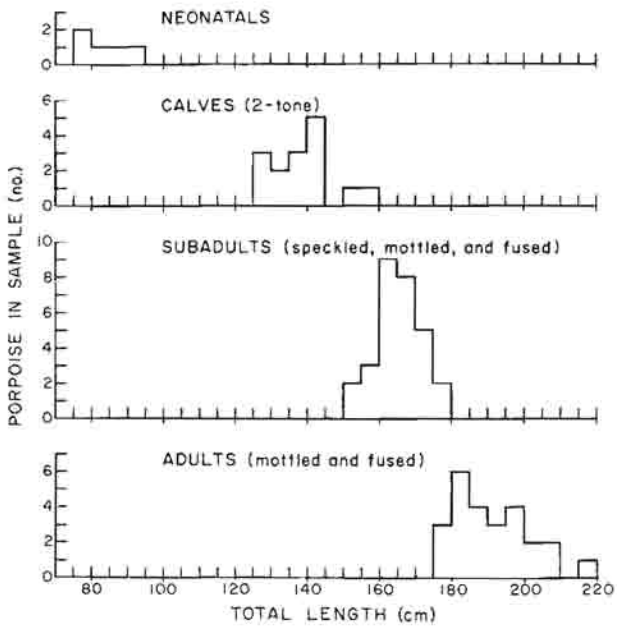

Fig. 7-22: *Stenella graffmani.* Frequency distribution of total length of age class included in sample examined for parasites. (After Dailey and Perrin, 1973.)

in the lungs of cetaceans (Cowan, 1967). Woodard and co-authors (1969) found 19 of 24 *Tursiops truncatus* with evidence of infestation by the lungworm *Halocercus lagenorhynchi*. Both captive and wild dolphins were heavily infested with parasites, generally without clinical evidence of being diseased. *Halocercus* infections occur in bunches with the anterior ends of the worm embedded in capsules found outside the bronchial walls (Fig. 7-23). This orientation has been considered by Delyamure (1955) as an adaptation for

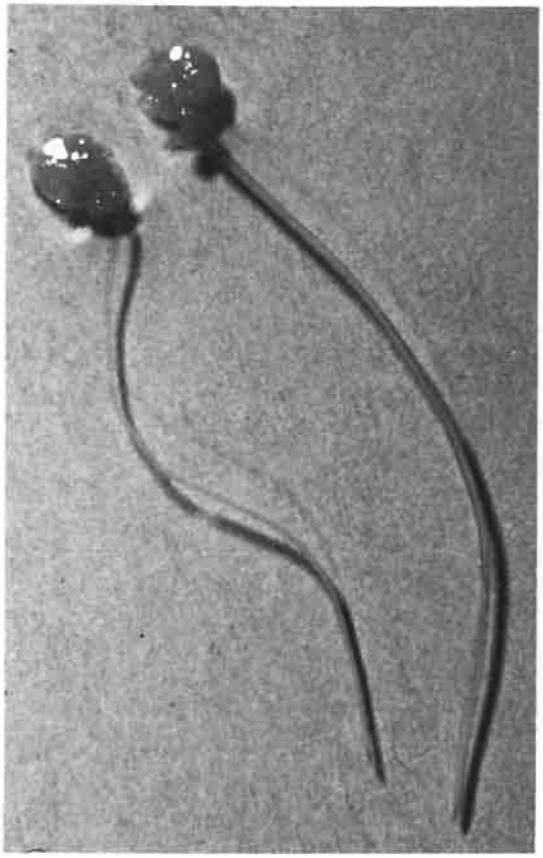

Fig. 7-23: Lungworm *Halocercus delphini* with anterior end embedded in capsule. (After Dailey and Perrin, 1973.)

parasite survival in the cetacean lung. Due to the sharp expirations (in contrast to terrestrial mammals) by cetaceans, the worms must invade the tissue to form 'anchors' through capsule formation. Worms living free in the bronchi would tend to be blown from the host. The posterior portions of the worms are free to mate and discharge larvae. Parasite infection leads to mucopurulent bronchiolitis and pneumonia (Fig. 7-24). Woodard and co-authors (1969) described the pneumonic process as being characterized by exudation of neutrophilic and eosinophilic polymorphonuclear leukocytes and macrophages. The pneumonia was generally limited to the area immediately surrounding the parasitized air passage. Stroud and Roffe (1979) listed parasitism as the primary, or

Fig. 7-24: *Phocoena phocoena*. Mixed infection of *Pharurus convolutus* and *Stenurus minor* from bronchi of a subadult. (After Dailey and Stroud, 1978.)

contributing, cause of death in 9 cetaceans examined. Verminous pneumonia with secondary bacterial branchopneumonia was responsible for the death of 3 subadult *Phocoena phocoena* infested with *H. invaginatus*. This same nematode was found by Dailey and Stroud (1978) throughout the lung parenchyma of a *P. phocoena*, resulting in fibrinous interstitial pneumonia with focal abscesses, and parasites in various stages of calcification (Fig. 7-25).

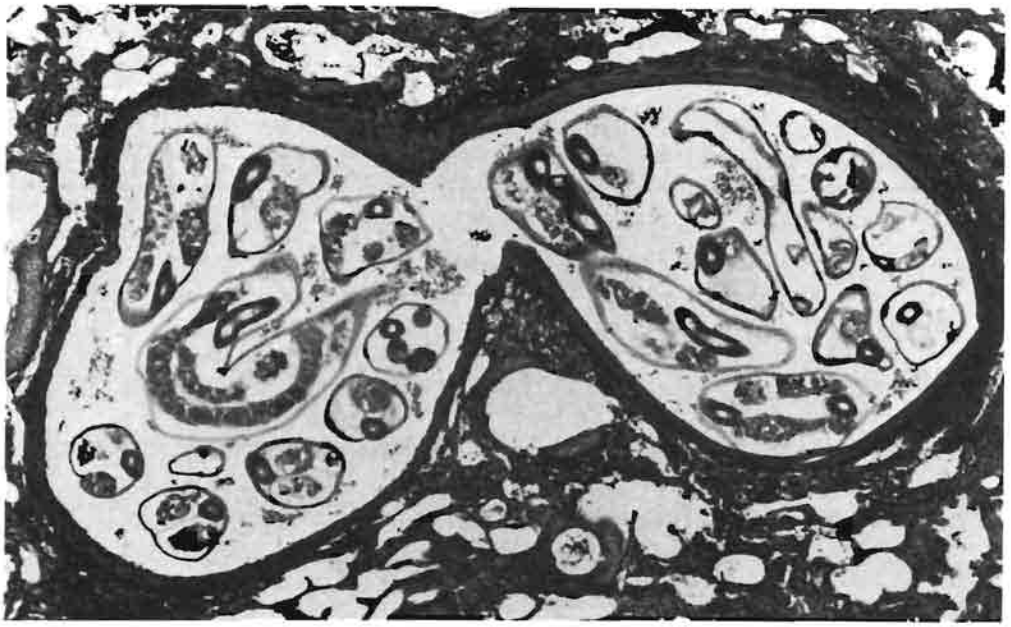

Fig. 7-25: *Phocoena phocoena*. Section of lung nodule containing *Halocercus invaginatus*. (After Dailey and Stroud, 1973.)

The changes caused by *Halocercus* in the cetacean lung differ from those seen in terrestrial animals with verminous pneumonia due to anatomical changes. In cetaceans a series of muscular sphincters are located between bronchus and respiratory complex. These valves reportedly represent an anatomical modification to cope with the changes in intra-alveolar pressure associated with diving (Wislocki, 1929; Slijper, 1962).

Hypertrophy of the muscular sphincters which accompanies lungworm infestation would insure closure of exudate-filled or parasite-laden bronchioles at the end of inspiration. This would prevent possible atelectasis during increased outside pressure through submersion (Woodward and co-authors, 1969). Additional problems may result from lungworm infections in cetaceans due to the fact that they lack a cough-reflex mechanism. Hence, exudate and particles tend to accumulate rather than being expelled by coughing. Woodward and co-authors felt that such material becomes inspissated, undergoes dystrophic calcification and is later incorporated into the bronchial wall. The same authors found a high incidence of pulmonary calcification in the infected *Tursiops truncatus* they examined. Despite the reports of pathogenicity caused by *Halocercus* sp. there is no consensus by authors on host mortality. Tomilin and Smyshlyayly (1968) thought the parasites capable of causing death in small cetaceans. Anderson (1966) was in agreement, attributing 8 out of 12 deaths in captive porpoises to parasitic bronchitis. As previously stated, Woodard and co-authors (1969) felt both wild and captive dolphins, even though heavily infested, were generally asymptomatic. Cowan (1966) originally agreed with the latter authors in suggesting that the resulting subacute and chronic inflammatory process did not cause functional impairment. However, in a later paper (Cowan, 1968), he states that the infestation could cause problems through destructive secondary emphysema. It appears that, as with other parasitic infections, the cetaceans can handle a lungworm

infestation under normal circumstances. However, when burdened with a massive number of parasites, or placed under stress, either in the field or in captivity, the host's immune system fails. In these instances the host succumbs to a combination of events formerly held in check by its defense system.

The genus *Stenurus* comprises another common group of pseudaliids reported from lungs, ears and sinuses of Odontocetes throughout the world (Dailey and Brownell, 1972; Arnold and Gaskin, 1975; Cannon, 1977; Dailey and Stroud, 1978; Dailey and Walker, 1978). Possible problems in echo disruption caused by this parasite due to its location in the head sinuses, eustachian tubes and middle ears were considered by Delyamure (1955). He states that fishermen in the Crimean Sea refer to harbor porpoises as 'deaf Azouka', since, unlike other species, they do not respond to the sound-producing device used to drive the dolphins into net enclosures. Delyamure found all Crimean *Phocoena phocoena* examined to be infected with *Stenurus* located in their auditory organs. Geraci (1978b), discussing potential causes of strandings, cites Dr. F. C. Fraser, an eminent cetologist at the British Museum, as the first to suggest (1966) that *Stenurus* might account for some strandings. During a study on a large number of Atlantic white-sided dolphins *Lagenorhynchus acutus* stranded near Edmunds, Maine, Geraci and co-authors (1976) report massive numbers of *S. globicephalae*. They state that the worms first appear in late-weaned calves with their numbers increasing with host age. By the time the dolphin reaches maturity it may contain as many as 3,300 worms. However, even high frequencies of infection and massive numbers of parasites do not appear to cause much harm to the dolphins. Histologic examination of worm-infested adults has revealed only chronic, low-grade inflammation of the mucus membranes, which Geraci (1978b) felt in itself would not result in impaired hearing. Other workers report similar histologic findings in *Peponocephala electra* (Cannon, 1977) and *Phocoena phocoena* (Dailey and Stroud, 1978) for *S. globicephalae* and *S. minor* respectively (Fig. 7-26). Although to date the sensitive inner ear has not been found to be inhabited by *Stenurus,* its close proximity (separated by only a

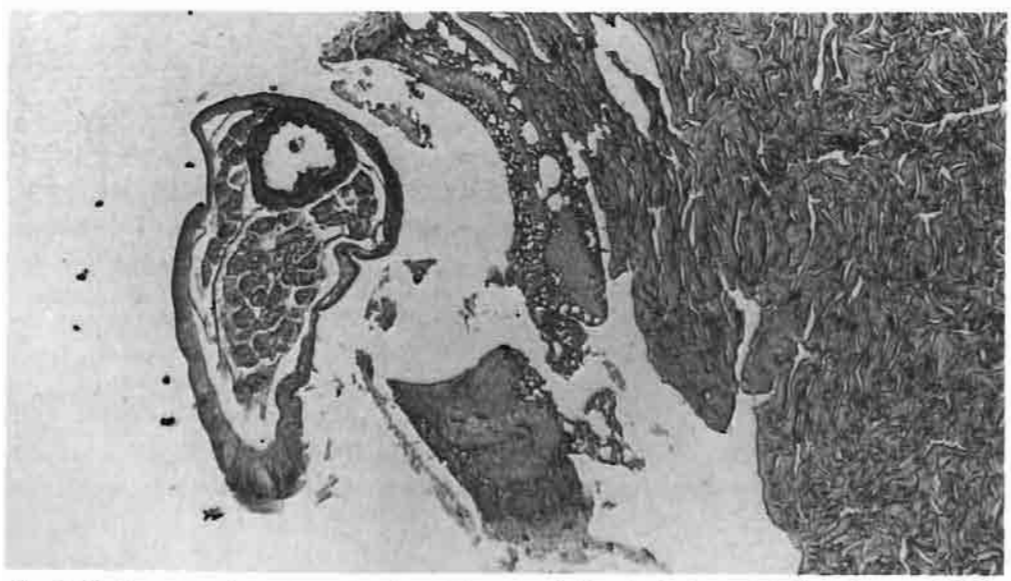

Fig. 7-26: *Peponocephala electra*. Section of *Stenurus globicephalae* in canal. (After Cannon, 1977.)

thick membrane) to the middle ear could expose it to various toxic secretions or other products of the parasite; in such case the dolphin's hearing and positional equilibrium could be seriously threatened (Geraci, 1978b).

Transmission of pseudaliids in a cetacean has yet to be proven experimentally. However, Caldwell and Caldwell (1968) and Woodard and co-authors (1969) have made observations on the age at which a *Tursiops truncatus* was infected with *Halocercus* sp. They report a heavy lungworm infestation in a 2.5 month old calf born in captivity to a field-caught female. Prior to its death the calf received only milk through nursing in a pool with copper-treated water. No lungworm larvae were recovered from milk, blood, fecal or blowhole samples taken from the female following death of the calf. No colostrum had been available for examination. Hence, Caldwell and Caldwell (1968) felt transmission could have been direct by inhalation of infected spray or taken in from infected water. Woodard and co-authors (1969) support the direct-transmission theory. They found numerous *Halocercus* sp. in the lungs of a 21 yr old dolphin born in captivity. Reviewing the report by Caldwell and Caldwell (1968), Arnold (1973) suggested that the parasite may have been transmitted transplacentally but qualified his conclusions in stating that an intermediate host was the usual mode of transmission. Woodard and co-authors (1979) also state the possibility of transplacental or transmammary passage to the young. At the time of this writing, the only pseudaliid cycle completed under experimental conditions is that of *Parafilaroides decorus* from the California sea lion *Zalophus californianus* (Dailey, 1970). In this case the intermediate host was the opal eye fish *Girella nigricans* which inhabited the pools on the sea lion's breeding grounds. The larvae of this worm had a short survival time once shed into ambient sea water. If this was also true for worms parasitizing cetaceans, copper treatment of ambient water would almost certainly preclude larval survival in captivity. Therefore, in view of the data available, a colostrom or transplacental route of infestation seems highly possible in members of the genus *Halocercus*. No such information is available for other genera of cetacean lungworms. According to Geraci and co-authors (1976), *Stenurus globicephalae* did not appear in nursing *Lagenorhynchus acutus* but were present in late-weaned calves and older individuals. This indicates that an intermediate host may be necessary for parasite transmission. Until experimental proof becomes available, one can only speculate on the mechanisms involved. It seems, however, that the pseudaliids have remained flexible enough to modify their means of transmission in order to account for the conditions prevailing in host and environment.

All nematodes found in stomach and intestine of Cetacea belong to the family Heterocheilidae (Anisakidae, Skrjabin and Karochkin, 1945). In this family the genus *Anisakis* by far accounts for nearly all infestations where tissue damage is sustained by the host. *Anisakis* has been reported from large numbers of both Odontocetes and Mysticetes (Delyamure, 1955; Dailey and Brownell, 1972) throughout the world. Roundworm attachment to the mucosa of the parietal stomach with associated inflammation was noted in all adult *Globicephala melaena* examined by Cowan (1966). Dailey and Stroud (1978) report 2 of 4 stranded *Phocoena phocoena* with *Anisakis* sp. and *A. simplex* infestations. The worms occurred in circumscribed, granulomatous nodules. Such reaction appears to be a common response to tissue invasion by these parasites (Fig. 7-27). Simpson and Gardner (1972) found stomach worm 'infestations' in captive cetaceans only on occasion. They attribute this lack of abundance to routine use of anthelminthics as well as the feeding of frozen fish limiting the chance of reinfection.

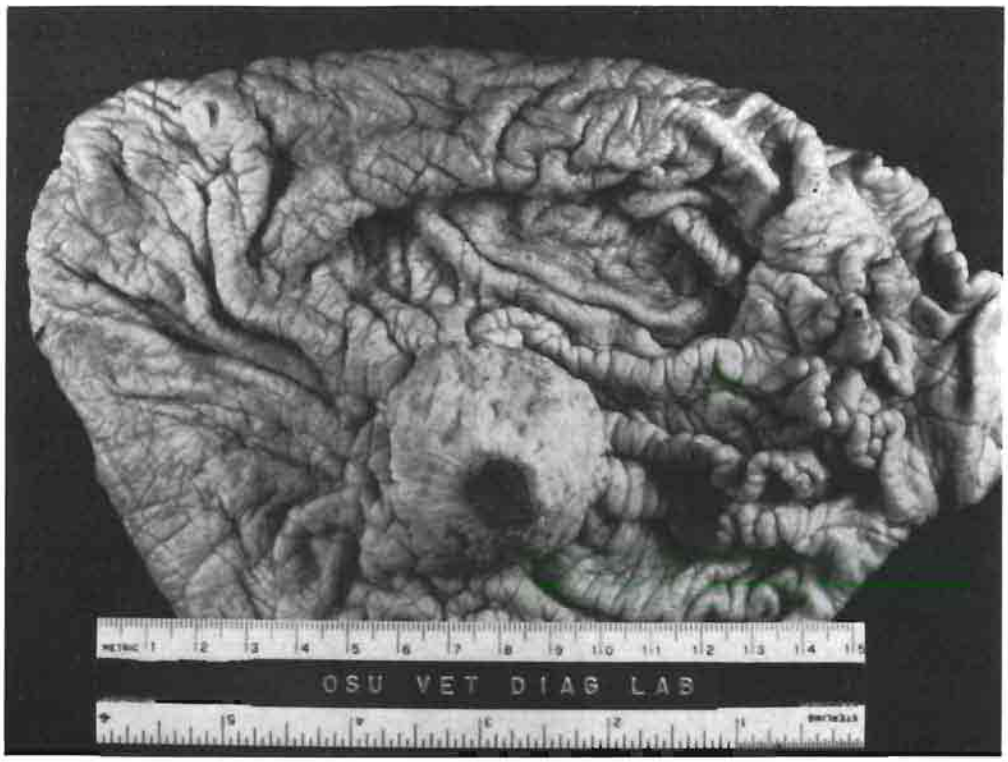

Fig. 7-27: *Phocoena phocoena.* Nematode *(Anisakis simplex)* induced granuloma in first stomach. (After Dailey and Stroud, 1978.)

While the genus *Anisakis* was originally subdivided into numerous species, Davey (1971) in a thorough revision reduced the number to 3 common species *(A. simplex, A. typica, A. physeteris).* He also indicated that *A. simplex* was found more commonly in temperate waters, while *A. typica* frequently occurred in cetaceans from more tropical seas. Roundworms of the genus *Anisakis* have gained attention in the last several decades as cause of anisakiasis in man. Van Thiel (1962) found these worms to be responsible for eosinophilic granulomas in humans who acquired the parasite by eating larval (infective third-stage) worms with their fish host. In areas with large concentrations of marine mammals, high percentages of fishes have been found infected (Dailey and co-authors, 1981). These works have resulted in more awareness of the disease.

Nematodes infesting tissues and organs of cetaceans are most commonly members of the family Crassicaudidae. In this family 2 genera *(Crassicauda, Placentonema)* are frequently listed as cause of major diseases in both Odontocetes and Mysticetes (Howell, 1927; Cockrill, 1960; Dailey and Walker, 1978; Geraci and co-authors, 1978).

Delyamure (1955) lists 6 species of the genus *Crassicauda (C. crassicauda, C. giliakiana, C. bennetti, C. grampicola, C. boopis, C. magna)* from a variety of hosts. Skrjabin (1969) lists 19 species for the same genus. These parasites are difficult to study during necropsy because of their extreme length and delicate body. Consequently, many reports refer to '*Crassicauda* sp'. The species described are almost entirely based on body

pieces rather than entire worms. One common site of infestation appears to be near or in the mammary tissue. Dailey and Perrin (1973) found *Crassicauda* sp. in the abdominal muscle adjacent to the mammary gland in *Stenella longirostris* (Fig. 7-28). A large abdominal cyst containing numerous *Crassicauda* sp. (eggs and fragments of the female) was also found in the same area. Geraci and co-authors (1978) reported that 47% of 30

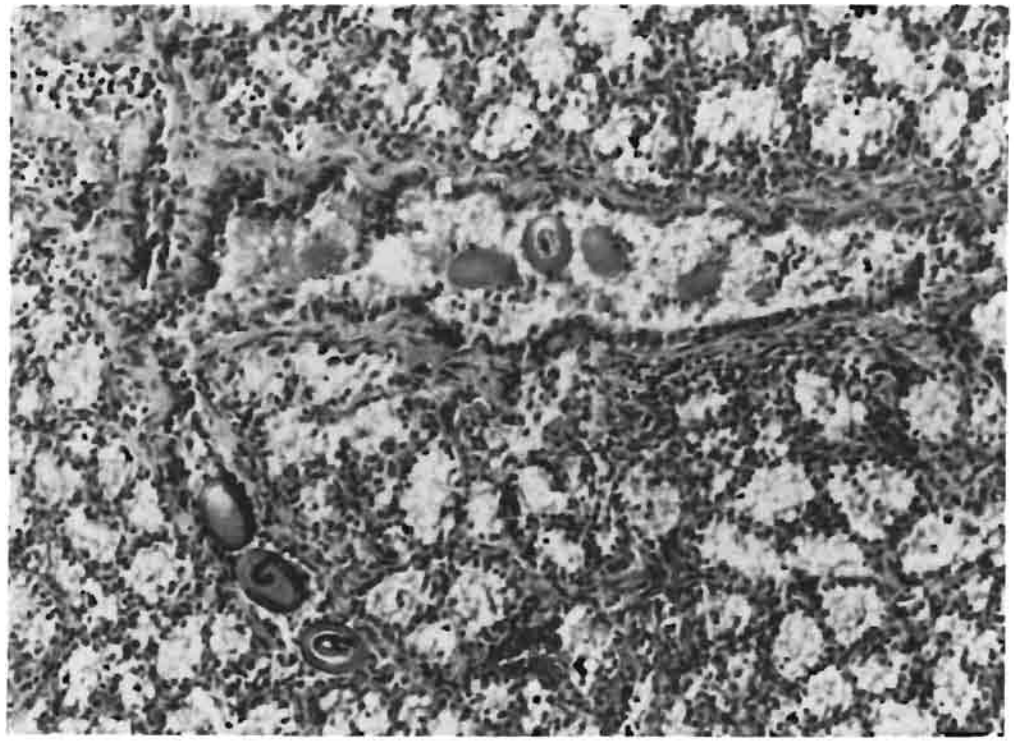

Fig. 7-28: *Phocoena phocoena. Crassicauda* sp. eggs in ducts of mammary gland. (After Dailey and Perrin, 1978.)

stranded female Atlantic white-sided dolphins *(Lagenorhynchus acutus)* were infested with *Crassicauda grampicola* in their mammary glands (Figs 7-29 and 7-30). They observed that (i) the parasites may affect the total milk production of *L. acutus* since the damage recorded exceeded the functional reserve of the gland; (ii) fibroplasia, necrosis, inflammation and squamous metaplasia of alveolar and ductal epithelium represent a replacement of functional parenchyma by non-secretory tissue in which milk cannot be produced. In addition, the quality of milk may suffer by the presence of parasite fragments, tissue debris, ova, and inflammatory cells in the liquid. In the group studied, high prevalence and severity of mammary lesions within the *L. acutus* herd must be viewed with regard to the possible influence on reproductive success. Impairment of the abdominal muscle by severe infestations might inhibit the forced ejection of milk necessary during nursing in Cetacea. In the extreme, these factors may result in nutrient insufficiency leading to malnutrition and reduced growth rate, which in turn could reduce calf survival and thereby influence herd productivity.

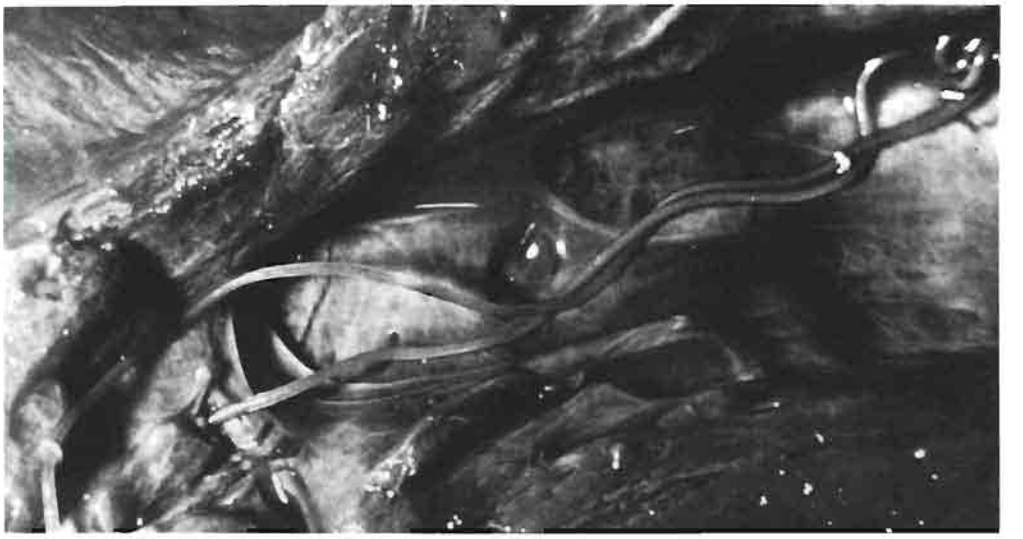

Fig. 7-29: *Lagenorhynchus acutus*. *Crassicauda grampicola* in lactiferous canals. (After Geraci and co-authors, 1978.)

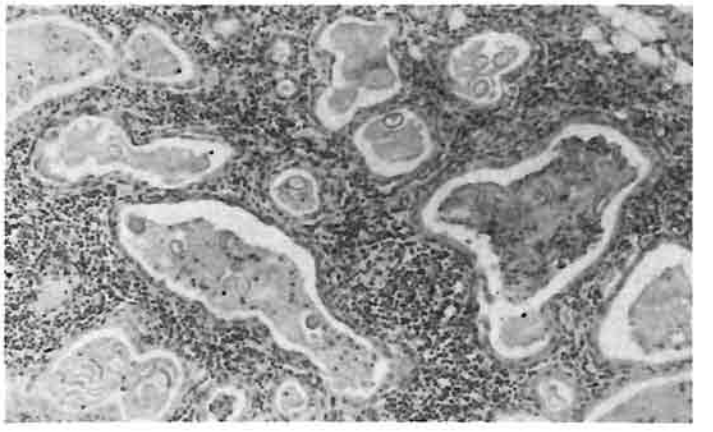

Fig. 7-30: *Lagenorhynchus acutus*. Mastitis in mammary gland caused by *Crassicauda grampicola* infection. (Photo: New England Aquarium and J. R. Geraci.)

The life cycle of *Crassicauda* spp. has not been investigated to date. However, the findings of mature female parasites in the lactiferous canals, and ova recovered from the milk support speculations of direct transmission between cow and calf. However, such a possibility has yet to be proven. The taxonomic classification of crassicaudids is uncertain. They are usually referred to the order Spirurida (Chitwood, 1933) which contains a large number of unrelated families. However, members of the family Crassicaudidae have many affinities to the Dracunculiodea or 'Guinea worm' group. In this group the known cycle involves larval development in a small crustacean, usually a copepod, prior to infecting the final host via accidental ingestion (Crichton and Beverly-Burton, 1975). It is possible that the egg uses a calf only as an exit from the adult female. This hypothesis is supported by the

finding that the youngest infested dolphin studied by Geraci and co-authors (1978) was only 2 yr old. The egg may use the passage through the calf as a period of maturation to the first-stage larval form. This larval stage is shed by female dracunculids during its cycle in fresh-water and marine fishes (Dailey, 1967; Platzer and Adams, 1967). The accidental consumption of these small, infested crustaceans during normal feeding by cetaceans is very probable. Delayed maturation is also common in dracunculids. In the case of mammary-gland infestations the worm may depend on a hormonal triggering mechanism to stimulate continued growth and maturity. This mechanism is known from other life cycles of parasites (Rothschild, 1965; Smyth, 1966).

Crassicaudids have also caused functional disturbances in the pterygoid sinuses and urogenital system of cetaceans. In numerous cases *Crassicauda* species have produced lesions in skulls of cetaceans, primarily in members of Delphinidae (Dailey and Perrin, 1973; Robineau, 1975; Dailey and Stroud, 1978; Dailey and Walker, 1978; Perrin and Power, 1980; Raga and co-authors, 1982a). *Crassicauda* have been found in *Stenella longirostris, S. graffmani, S. attenuata, Tursiops truncatus, Grampus griseus, Lagenorhynchus obliquidens, Lissodelphis borealis,* and *Phocoenoides dalli.* Additional reports involve *Delphinapterus leucas, Pseudorca crassidens* and *Globicephala* sp. (Yamada, 1956; Brodie, 1971). Yamada found 'basket-like' lesions in the pterygoid region in 50% (40 of 80) of *Pseudorca crassidens* skulls examined. He ascribed the damage to parasitic infection, 'most probably by nematodes'. The bone lesions (Figs 7-31 and 7-32) occur over a wide geographical range (Atlantic and Pacific Oceans, Mediterranean Sea) in both stranded and caught dolphins (Dailey and Perrin, 1973; Raga and co-authors, 1982a). Because most of these lesions are seen as eroded areas in prepared museum skulls, identification of the etiologic agent is not always possible. In cases where identifiable worms were recovered, *C. grampicola* was named as the organism involved (Dailey and Stroud, 1978; Raga and co-authors, 1982a; Raga and co-authors, 1982b). Although other

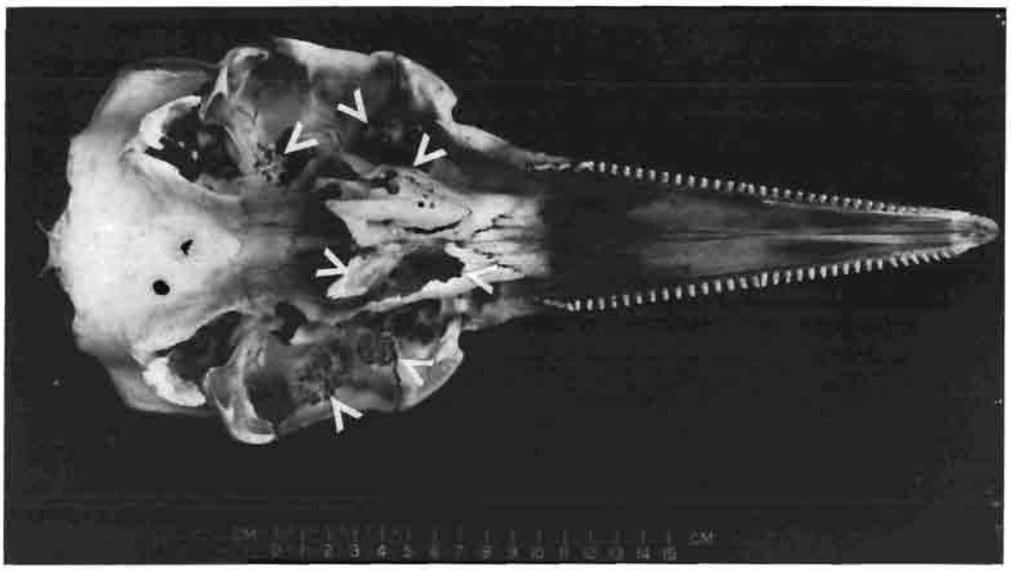

Fig. 7-31: *Stenella longirostris.* Bone lesions (arrows) in skull. (After Dailey and Perrin, 1973.)

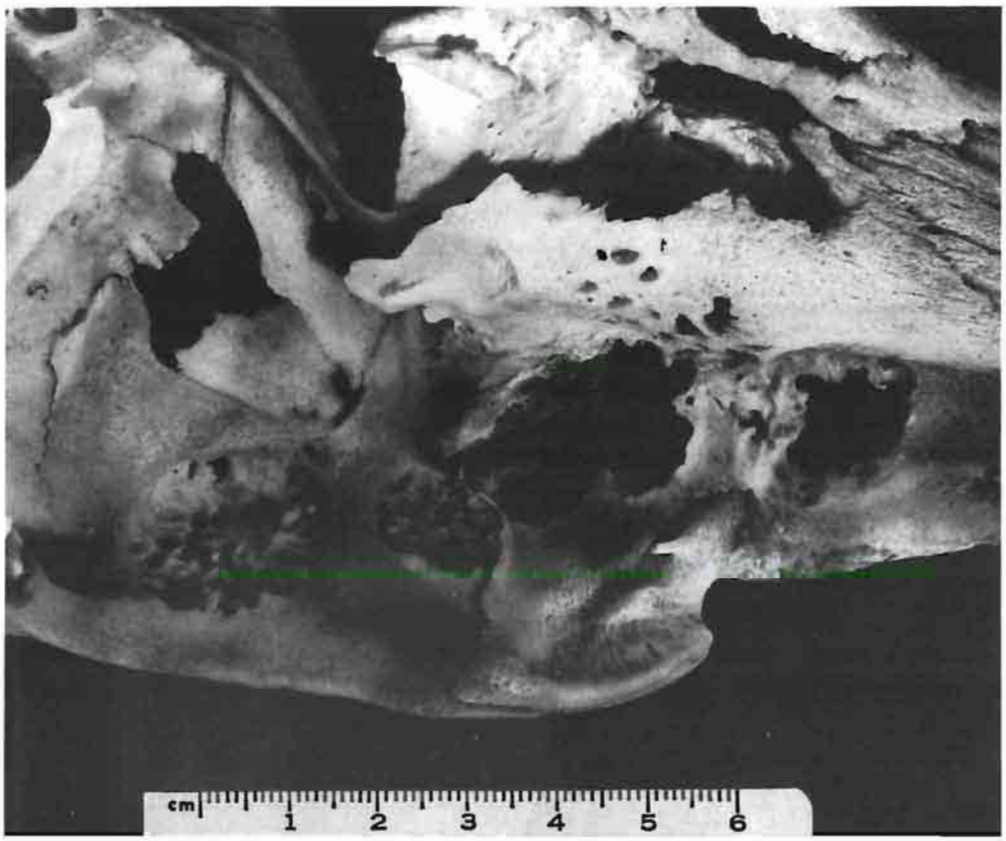

Fig. 7-32: *Stenella longirostris*. Enlarged view of 'basket-like' bone lesions in skull. (Photo: William Perrin.)

nematodes (*Stenurus* sp.) have been found in air sinuses, the similarity of lesions due to *C. grampicola* makes it very likely that the disease was caused by the presence of a crassicaudid nematode. The erosion in the skull appears to involve inflammation of the mucosa, purulent sinusitis and, finally, osteitis (Raga and co-authors, 1982a). The role played by crassicaudid nematodes in mortality of dolphins was analyzed by Perrin and Powers (1980). Skulls of 704 spotted dolphins *Stenella attenuata* were examined and aged by number of dentinal layers, indicating that if dentinal layers are deposited at the rate of 1 yr^{-1}, nematode-induced annual mortality may be estimated at 1% yr^{1} for dolphins with 8 or more dentinal layers, and 3% yr^{1} for those with more than 5 layers. According to Perrin and Powers, higher rates of layer deposition (e.g., 2 yr^{-1}) indicate even higher parasite-caused annual mortalities. Their estimates amount to 11 to 14% of field mortality of the population thus indicating that parasitism may constitute 'a major factor in natural mortality of small cetaceans'.

Crassicaudids also occupy other cetacean organs including sinus cavities and tubes. Reports of worms found in kidney, ureters, and urethra of baleen and toothed whales are common (e.g., Baylis, 1916; Cockrill, 1960; Arvy, 1973–74; Raga and co-authors, 1982b; Dailey, own observ.). Delyamure (1955) lists 4 species specifically from the urogenital

system *(Crassicauda crassicauda, C. bennetti, C. boopis, C. giliakiana)*. Tissue damage in this organ system is similar to that in mammary glands. The worms are tightly coiled in the center of a large crater-like lesion surrounded by a sheath of partially saponified and calcified fat. In the penis of *Balaenoptera borealis* the worms form large abscesses in the tissue with the remainder of their body winding its way to the urethra. It is common to see so many 'free ends' of the parasite in the urinary passage, that the lumen appears impassable (Fig. 7-33) (Dailey, own observ.). Of 35 *B. borealis* taken in the Antarctic from 1976 to 1977, 54.3% of the males were infected with *C. crassicauda* (Vogelbein, 1981).

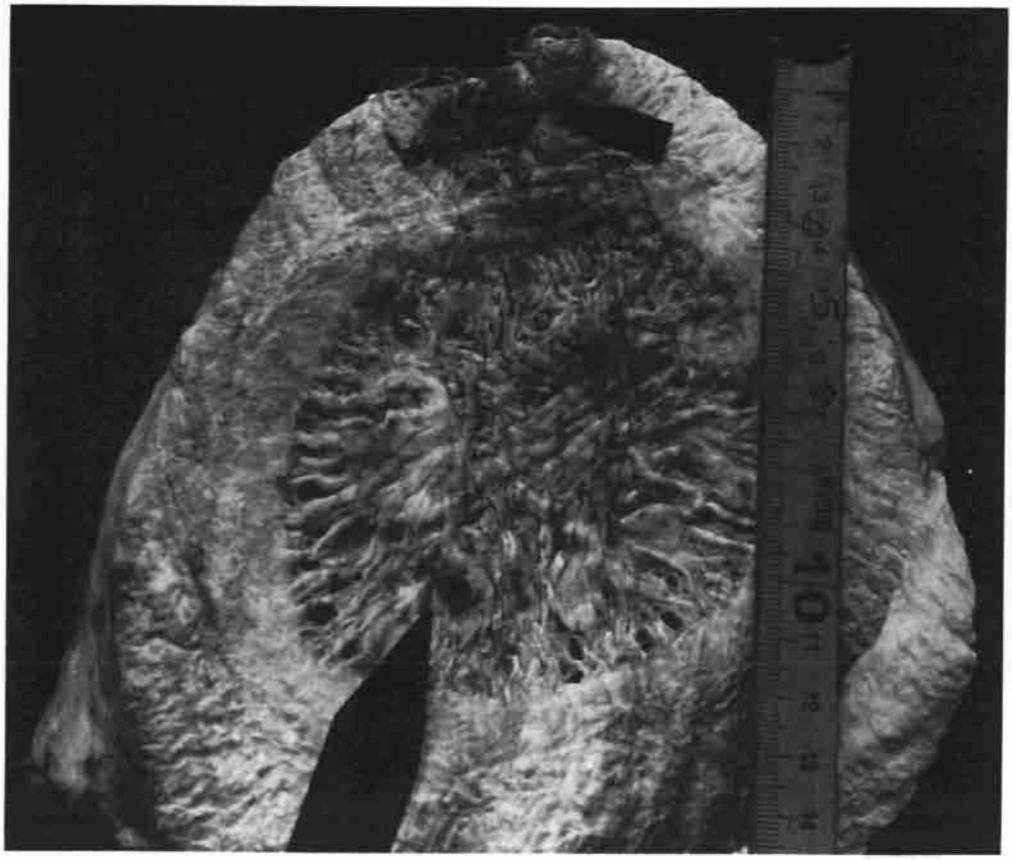

Fig. 7-33: *Balaenoptera borealis*. Free ends of *Crassicauda crassicauda* in lumen of urethra of penis. (Original.)

Transmission of crassicaudid worms inhabiting the urogenital system of cetaceans would most probably follow the same pattern as seen for mammary-gland inhabitants. Eggs and developing larvae are voided in the urine for dissemination and subsequent consumption by the next host. Possibility exists for direct transmission to the calf *via* contamination of the milk during nursing, due to the position of the mammary outlet near the genital slit.

The genus *Placentonema* is monotypic with the single species being *gigantisma*. This worm was originally described by Gubanov in 1951 from a *Physeter macrocephalus*

placenta. To date, with one exception, this very large nematode (females reaching 9 m in length and 2.5 cm in width) has only been reported from the sperm whale uterus and placenta (Delyamure, 1955; Skrjabin, 1965; Dailey and Brownell, 1972; Gibson and Harris, 1979; Vogelbein, 1981). Ridgway (1966) found a number of worms in the mammary glands of a *Phocoenoides dalli* which were identified as *Placentonema* sp. This case was redescribed together with another *P. dalli* in which similar parasites were reported by Johnston and Ridgway (1969). In light of subsequent findings and without specimens at hand for confirmation, it is probable that the *P. dalli* parasites from these reports were species of *Crassicauda.*

Although *Placentonema* species appear not uncommon in pregnant female sperm whales, very little is known of this parasite, except distribution, size and shape. Skrjabin (1965), working with material from 23 pregnant females taken in the Kurile Islands region, found immature worms in 20 individuals. Since the size of the fetus was not given, a correlation between growth of worm and embryo cannot be determined. However, according to Skrjabin, male worms were only 57 to 86 mm long by 0.61 to 0.68 mm wide and females measured 66 to 103 mm in length and 0.66 to 0.99 mm in width. In all female specimens a developing reproductive system was evident. However, Skrjabin does not say if all or some of the immature worms he recovered were from the uterus or from the placenta. Possibly, the immature worms infect the female sperm whale prior to sexual maturity and remain dormant until implantation of the embryo. At that time, increased hormone levels may act as a triggering mechanism for continued maturation of the parasite. Adult *Placentonema* sp. are found in the transparent placental lining, extending throughout the tissue and possessing a transparent body cuticle (Fig. 7-34). During a study of sperm whale parasites conducted in the Antarctic in 1977, 2 of 11 females examined were pregnant and both infected with *Placentonema* (Vogelbein, 1981). In both cases the fluid surrounding the fetus had become turbid, presumably due to the parasite (Dailey,

Fig. 7-34: *Physeter macrocephalus. Placentonema gigantisma* in placenta. (Original.)

own observ.). The role that *P. gigantisma* plays in the number of stillborn term fetuses each year in the sperm whale population can only be speculated on at this time. It seems plausible that it could constitute a major factor in total stock reduction within certain geographical populations. Transmission of *P. gigantisma* would most probably be facilitated through expulsion of the placenta at birth, followed by the release of larvalated eggs through decomposition or rupturing of the female worm.

Agents: Acanthocephala

Delyamure (1955) lists 21 species of Acanthocephala from marine mammals, 11 belonging to the genus *Bolbosoma* and 10 to *Corynosoma*. In his key to the genera of the subfamily Polymorphinae he states that the genus *Bolbosoma* is a parasite of cetaceans, 'rarely pinnipeds' and *vice versa* for *Corynosoma*. Also, it appears that pelagic forms are primarily infested with *Bolbosoma* and off-shore forms with *Corynosoma*. This would probably depend on the number of pinnipeds feeding in the same area and the amount of fish in the diet of the cetacean. Forms feeding primarily on krill were not found to be infested with *Corynosoma*.

Acanthocephalans occur in 16 species of cetaceans and are ubiquitous (Dailey and Brownell, 1972). Members of the genus *Bolbosoma* have been reported in very high incidences (up to 100%) and intensities (11,200 worms m^{-1} of gut) in *Balaenoptera borealis* throughout the Antarctic (Mathews, 1938; Skrjabin, 1968; Skrjabin, 1975;

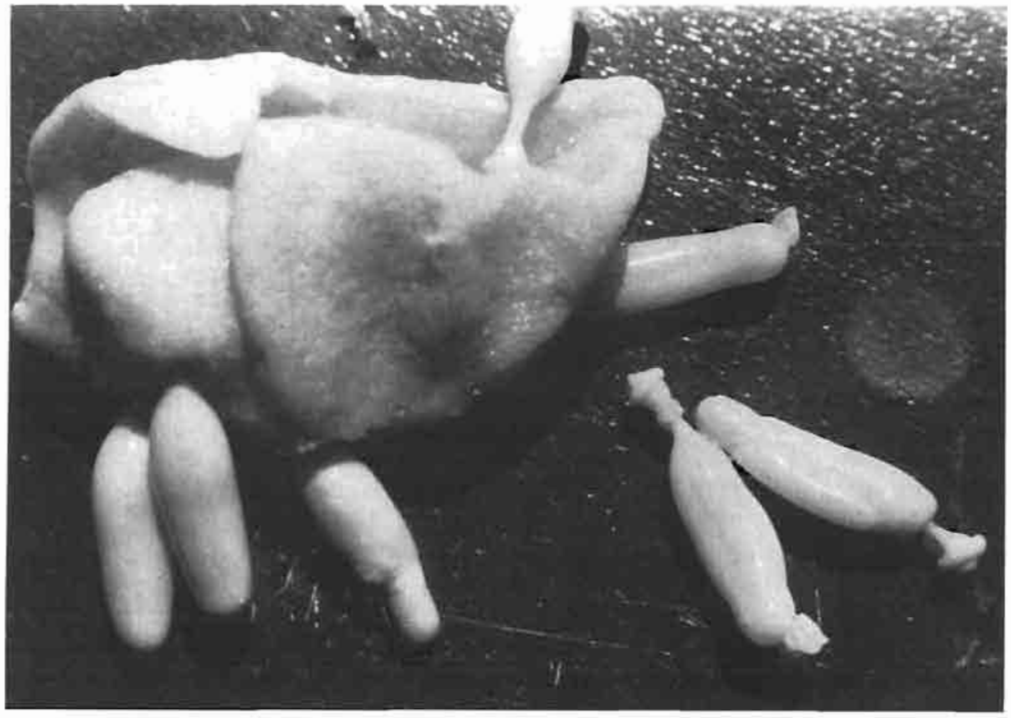

Fig. 7-35: *Balaenoptera borealis*. Acanthocephalans of the genus *Bolbosoma* from intestine. (Original.)

Vogelbein, 1981). Although *Bolbosoma* penetrate the intestinal wall with their proboscis and bulbose head for attachment (Fig. 7-35), they do not cause serious disease problems. A light inflammatory response and some fibrosis is usually all that is seen in infected areas. Occasionally secondary bacterial infections or penetration through intestinal muscle layers may occur. Acanthocephalans probably cannot be considered a prime factor in the mortality of cetaceans.

Agents: Ectoparasites and Epizoites

A wide variety of organisms attach to cetaceans, ranging from diatoms to lampreys and remoras (e.g., Leung, 1965, 1967; Dailey and Brownell, 1972; Margolis and Dailey, 1972). These organisms, with the exception of copepods (Fig. 7-36), are superficial and apparently constitute only an irritant to the cetacean host. While copepods such as *Penella* sp. anchor in the muscle beneath the blubber layer, they appear to cause no major disease problem (Dailey, own observ.).

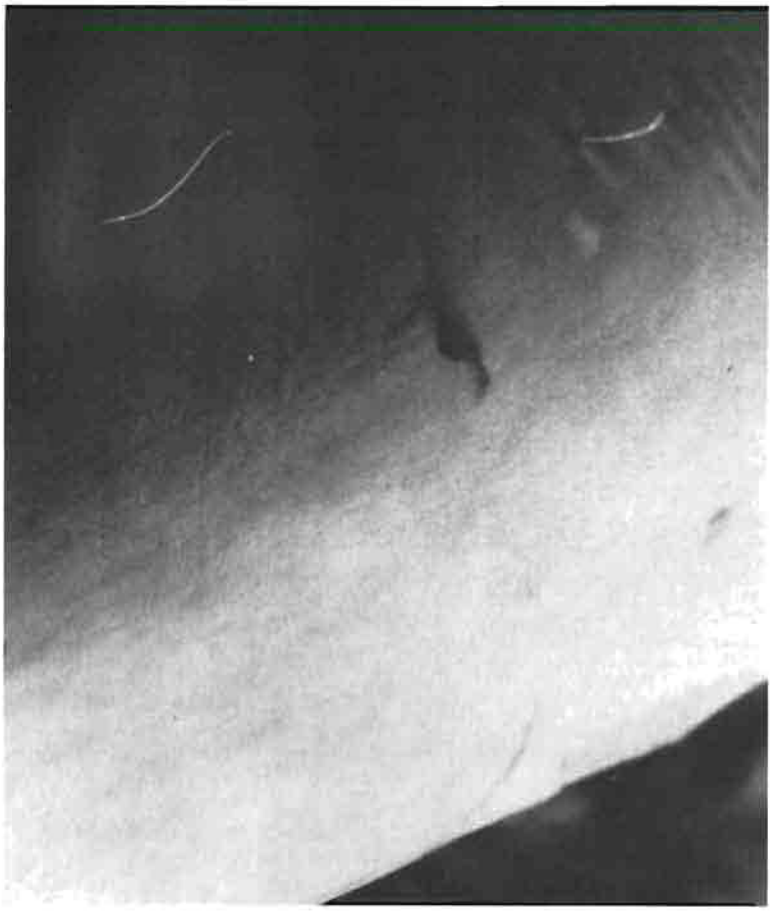

Fig. 7-36: *Balaenoptera acutorostrata*. *Penella* sp. imbedded in blubber. (Original.)

Tumors and Abnormalities

Neoplastic disease was thought to be uncommon in cetaceans until the last decade. Since that time 14 types of tumors have been reported from a variety of sites from 9 species of cetaceans (Table 7-4) (Howard and co-authors, 1983b). Their assessment of just over a 1.9% tumor-related death rate in all marine mammals considered (approximately 1,500 pinnipeds and cetaceans) was probably misleadingly low. Neoplasms primarily cause disease in older animals which are less apt to become beached, or are destroyed by

Table 7-4
Tumors of cetaceans (After Howard and co-authors, 1983b)

Cetacean	Age (yr)	Sex	Tumor site	Tumor type
Common porpoise	Adult	M	Penis	Papilloma
Bottle-nose dolphin	Adult	M	Testicle	–
	–	M	Liver	Adenoma
	10–15	M	Pancreas	Carcinoma
	–	F	Lung, liver	Reticuloendotheliosis
	20+	F	Spleen	Lymphosarcoma
	–	–	Kidney	Adenoma
	–	–	Adrenal	–
	10–15	M	Thyroid	Adenoma
Killer whale	6–7	M	Penis	Papilloma
Pilot whale	31	F	Uterus	Leiomyoma
Sperm whale	19	F	Uterus	Fibromyoma
	–	–	Jaw	Fibroma
	–	–	Liver	Hemangioma
	–	M	Skin	Fibroma
Beaked whale	Adult	F	Vagina	Fibromas (2)
Blue whale	–	F	Uterus	Fibromyoma
	–	F	Ovary	Cystadenoma
	–	F	Ovary	Granulosa cell tumor
	–	–	Mediastinum	Ganglioneuroma
	–	–	Tongue	Papilloma
	–	F	Stomach serosa	Lipoma
	–	–	Pleura	Fibroma
	–	F	Liver surface	Lipoma
	–	–	Intestine serosa	Lipoma
Fin whale	–	F	Ovary	Granulosa cell tumor
	–	F	Ovary	Granulosa cell tumor
	–	F	Ovary	Carcinoma
	Adult	–	Cerebellum	Neurofibroma
	–	F	Tongue	Fibroma
	–	M	Multicentric	Hodgkin's disease
	–	–	Pleura	Fibroma
	–	M	Subcutis	Fibroma
	–	M	Skin	Fibroma
	–	–	Dorsal muscle	Lipoma
Bowhead whale	Immature	M	Liver	Lipoma

– No information available

predators at sea. According to Howard and co-authors there is evidence for an increased incidence of certain types of neoplasia due to chemical water and air pollution.

Congenital abnormalities have been noted primarily in captive marine mammals. In a comprehensive review Leipold (1980) reported ventricular septal defects, transposition of pulmonary artery and aorta, and Hageman factor deficiency in *Tursiops truncatus*. Howard (1983) observed bilateral polycystic kidneys in *Delphinus delphis* which he believed to be a congenital anomaly. Several cases of deformed pectoral flippers were seen on minke whales *Balaenoptera acutorostrata* during an Antarctic whale harvest (Fig. 7-37) as well as a deformed spine on sperm whales (Dailey, own observ.). These whales

Fig. 7-37: *Balaenoptera acutorostrata*. Deformed pectoral fin. (Original.)

appeared to be in good flesh and functioning in a normal manner. It can be assumed that these types of congenital anomalies occur in very low numbers and do not affect cetacean populations.

Acknowledgements. I thank Dr. David St. Aubin, Dr. Joe Geraci, Dr. B. D. Hicks (Univ. of Guelph), Dr. Jay Sweeney, Dr. Pete Schroeder, Jim Runningen, Dr. Richard Stroud, Dr. L. G. Cannon, and Dr. William Perrin for help with photographs and figures. I also express my appreciation to Dr. James Britt Jr. and John Hunter for making material available from 'Pathobiology of Marine Mammal Diseases', Vol. I & II. Sincere thanks also to Dr. Neylan Vedros, Dr. Pete Schroeder and Dr. Tom Deardorff for providing helpful suggestions and comments.

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