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Health Care and Diseases of Captive-Reared Loggerhead and Kemp's Ridley Sea Turtles

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During 1977 to 1983, Kemp's ridley (Lepidochelys kempi) and loggerhead (Caretta caretta) sea turtles were reared for one year or less at the National Marine Fisheries Service, Southeast Fisheries Center, Galveston Laboratory. Many of the diseases, malformations and injuries observed in captive-reared sea turtles were named and classified into 27 major categories. The categories were: sudden hatchling death syndrome, papillary dermatitis, emaciation, focal erosive dermatitis, injuries from aggressive biting, focal dermal granulosis, scolecobasidiosis, white-suture syndrome, yolk sac mycosis, internal nodular mycosis, hypernecrotic warts, malabsorption of yolk sac, urolithiasis, duodenal ulceration, hemorrhagic bacteriosis, mycobacterial pneumonia, swollen-eye, intussusception, curved-back, soft shell, coelomic edema, lung aplasia, flipper malformation, cross-beak, congenital blindness, intestinal prolapse, and prolapse of the urinary bladder. Each category of ailment is described with respect to etiology, symptomatology, occurrence and suggested remedy if known. Current levels of our knowledge of diagnosis and control of diseases during captive-rearing of these two species of turtle are discussed. Recommendations are made on perspectives and needs in sea turtle pathology research.

Numerous kinds of diseases and physical injuries occurred in Kemp's ridley (*Lepidochelys kempi*) and loggerhead (*Caretta caretta*) sea turtles reared for one year or less at the National Marine Fisheries Service (NMFS), Southeast Fisheries Centers Laboratory in Galveston, Tex. Rearing Kemp's ridleys from hatchlings to yearlings is a feasibility study of the head starting concept, part of a broader conservation program aimed at preventing extinction of this critically endangered species (Klima and McVey, 1982).

Information on nesting sites and population decline in Kemp's ridley has been summarized in several papers (Klima and McVey, 1982; Mrosovsky, 1983; Caillouet, 1984; Caillouet *et al.*, 1986; Fontaine *et al.*, 1985). To recapitulate, there is only one known primary nesting beach located near the village of Rancho Nuevo, in the State of Tamaulipas, Mexico. The number of nesters at that beach has declined from an estimated 40,000 reported to have nested in a single day in June 1947 to some 1,200 in 1974 and then to 500 or so in 1977. It is hoped that head starting will prove useful as one among several methods to help preserve and augment the population. In head starting, the turtles are reared in captivity from hatchlings to about one year or less of age, then tagged and released into the Gulf of Mexico or adjacent estuaries.

To gain experience before attempting to rear Kemp's ridleys, the Galveston Laboratory staff obtained 1,160 loggerhead hatchlings, a species which had not yet been listed as threatened under the Endangered Species Act of 1973. Experimenting first with the loggerheads proved beneficial, because almost all of the loggerhead hatchlings became ill at some time during head starting, so the staff at the Galveston Laboratory was able to derive important information on sea turtle pathology and health care methods without jeopardizing Kemp's ridleys. Only about 9 percent of the loggerheads survived after a 10-month rearing period. The knowledge, experience and information gained on prophylaxes, diagnoses and therapeutics for loggerheads were applicable in part to Kemp's ridleys in subsequent years. Pathology studies and observations on loggerheads and Kemp's ridleys contributed greatly to the high survival rates of six year-classes (1978-1983) of head started Kemp's ridleys: 68 percent, 83 percent, 95 percent, 88 percent, 89 percent and 77 percent, respectively (Fontaine *et al.*, 1985).

This paper is a review of some of the diseases and injuries suffered by captive loggerhead and Kemp's ridley sea turtles at the Galveston Laboratory. It also describes remedies or prophylaxes that we have found useful in resolving or preventing some of the problems. The diagnostic, prophylactic, therapeutic and health-care methods that we adopted represent the best approaches that we could undertake, given the constraints on funding, personnel and other resources. In addition, we were constrained by our U.S. Fish and Wildlife Service Permit, which prohibited deliberate sacrificing or injuring of live, normal sea turtles. To secure baseline, in-depth pathological information essential to development and refinement of diagnostic, prophylactic and therapeutic methods will require controlled experiments necessitating sacrificing or injuring some test animals.

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With few exceptions, there was a paucity of documented information on diseases and causes of death in sea turtles and efficient remedies, especially those applicable to mass-culture situations such as ours. The information in this paper may be useful both to those interested in rearing sea turtles from hatchling to yearling stages and those concerned with their morbidity and mortality.

Materials and Methods

Sources of Hatchlings

In September 1977, 1,160 loggerhead hatchlings were supplied by the Florida Department of Natural Resources, Jensen Beach, Fla. (Clary and Leong, 1984). Approximately 200 loggerhead hatchlings were received from the same source in July 1978. Kemp's ridley hatchlings were obtained each summer, beginning in 1978, through the joint efforts of the Instituto Nacional de la Pesca of Mexico, U.S. Fish and Wildlife Service, National Park Service, Texas Parks and Wildlife Department and Gladys Porter Zoo in Brownsville, Tex. (Fontaine *et al.*, 1985). The number of Kemp's ridley hatchlings received annually at the Galveston Laboratory for 1978-1983 were 3,081, 1,846, 1,822, 1,865, 1,524 and 250, respectively (Fontaine *et al.*, 1985).

Rearing of Turtles

Turtles were reared at the Head Start Research Project facilities of the Galveston Laboratory until they were 7 to 12 months old. They were then tagged and released offshore at preselected locations off Texas, Florida and Mexico (Klima and McVey, 1982; McVey and Wibbels, 1984; Wibbels, 1984; Fontaine *et al.*, 1985).

Two major concepts of maintenance and rearing were tried by the Galveston Laboratory: (1) free-contact by group-rearing in raceways or tanks, and (2) individual-rearing in isolation (Clary and Leong 1984; Caillouet, 1984). The first concept was implemented in 1977 and 1978. Hatchlings were divided into groups of about 50 to more than 550 turtles, depending on the size of rearing tanks, and the hatchlings were allowed to swim freely in contact with one another.

A variety of holding vessels was used, including: concrete raceways, fiberglass-lined wooden raceways and fiberglass tanks. Seawater in the concrete raceways was either recycled (non-flow-through) to remove wastes via a biodisc (Mock, Ross and Salser, 1977), or was constantly replaced by flow-through. Oyster-shell filters were used to remove wastes from recycled seawater in the fiberglass-lined, wooden raceways. Static water in the fiberglass raceways was replaced three times per week.

Initially, the incidence of disease, traumatic injuries and death was very high, indicating inadequacy of the group-rearing method. Laboratory and tankside observations revealed that hatchlings fared better when isolated from one another in small containers and when the culture water was replaced frequently with clean seawater. Not only were traumatic injuries caused by mutual biting and scratching eliminated, but morbidity and mortality were also greatly reduced. Based on these findings, isolation-rearing was implemented in January 1979, after the 1978 year-class of Kemp's ridleys had been reared for six months.

The isolation-rearing system (Klima and McVey, 1982; Fontaine *et al.*, 1985) was employed in the same kinds of holding tanks already described. Each hatchling was raised in isolation from others in a 10-liter plastic bucket, with holes in the bottom to allow seawater exchange and removal of feces and surplus food. The waste materials would fall through the holes to the tank floor and would be eliminated when the tanks were drained during routine cleaning operations. The seawater was completely changed daily for rearing the 1978 year-class, but the frequency was reduced to two to three times a week for subsequent year-classes.

Raw fish flesh was the primary food used for rearing the 1977 year-class of loggerheads. Feeding of the Kemp's ridleys has been described by Fontaine *et al.* (1985) and McVey, Leong, Wheeler and Harris (unpublished manuscript on the culture of young Kemp's ridley sea turtles). Kemp's ridleys were fed a synthetic, floating, pelleted diet (Fontaine *et al.*, 1985). In general, two meals per day were provided, one in early morning and one in late afternoon. The daily amount of pelleted feed ranged from roughly five percent of wet body weight for hatchlings to roughly 1.5 percent of body weight for yearlings.

Maintenance of Sick or Injured Turtles

In general, sick or injured turtles were removed from their rearing tanks and maintained separately for observation and medication until recovery. Two different systems, collective isolation and individual isolation, were used for holding sick or injured turtles.

Collective isolation was used for some of the sick loggerhead turtles of the 1977 year-class. Two or more turtles having similar signs or symptoms of illness were held together in one container. The number of animals placed in each container was dependent on the sizes of container and animals. In general, no less than 300 square cm of seawater surface area were provided for every 100 square cm of turtle carapace area. Water depth was at least twice the thickness of the body of the turtle, except in the specific situation in which an animal was unable to raise its head above the water due to physical weakness. In the latter cases, shallower water was used to prevent drowning. The kinds of

containers used for collective isolation of sick or injured turtles included 2,100-liter circular fiberglass tanks (1.83 m inside diameter, I.D.), 190-liter rectangular fiberglass tanks 0.83 m x 0.5 m (upper-rim dimensions) x 0.5 m deep, and 19-liter circular plastic tubs (0.46 m I.D.).

In individual isolation, used for both loggerhead and Kemp's ridley turtles, each sick or injured turtle was held singly in a separate container. Containers included 4-liter glass jars, 10-liter plastic buckets, 19-liter tubs, and 190-liter rectangular tanks. When there were only a few sick turtles, the containers were placed on the floor or bench tops, and seawater was manually replaced daily. However, within the 1978 year-class of Kemp's ridleys, there were routinely from 800 to 1,000 sick or injured turtles to be cared for at a time. Under these circumstances, 10-liter buckets with holes in their bottoms were suspended in a concrete raceway, and each contained a sick or injured turtle. Each day the seawater was drained, the buckets and turtles were washed with fresh water from a hose, the turtles were given the appropriate medication, and the raceway was refilled with clean seawater to the appropriate level. This method also reduced the labor required to clean individual buckets manually.

Feeding sick turtles varied according to the nature of the illness. The animals were either fed food similar to that given to healthy turtles, or they were given a special diet such as a dry breakfast cereal (rice or wheat) or commercial baby food (used for human infants), supplemented in some instances with multivitamins. When semi-solid food was used, it was fed to the turtle via intraesophageal intubation by means of plastic tubing attached to a graduated medical syringe. In general, sick animals had reduced appetites. Therefore, the amount of food was about one-half to one-third of normal feeding rate. On many occasions, sick turtles were not fed at all because of the nature of certain illnesses such as suspected intestinal perforation. To sustain the life of non-feeding or anorexic turtles, subcutaneous injection of a sterile dextrose-multivitamin solution (10 percent dextrose + 10 percent multivitamin infusion concentrate, U.S. Pharmaceutical) was administered at the rate of 0.25 ml per 50 mg wet body weight per day. Daily injection continued until the animal regained its appetite or for as long as seemed necessary.

Drug Administration

At least four methods of administering therapeutic chemicals to sick or injured turtles were studied as follows:

1. For medicated baths the calculated dose of a water-soluble drug or antibiotic was added to the seawater. The turtle was then placed in the medicated seawater for a period of time, either predetermined or on an as-needed basis, depending on the nature of the illness or injury, the progress of recovery, and the kind of medication used. Examples of medicated baths were formalin (50 to 100 ppm), potassium permanganate (2 to 5 ppm), malachite green (0.1 to 0.2 ppm), Cutrin-Plus (Applied Biochemist, Inc., Wisconsin; 0.3 ppm copper), minocyclin hydrochloride (0.3 to 0.6 ppm), erythromycin (2 to 4 ppm), and furanace (Dainippon Pharmaceutical Co., Osaka, Japan; 0.05 to 0.1 ppm).
2. For oral administration a drug was fed to the turtle either through intraesophageal intubation or by incorporating it into the feed or agar carrier. For the latter, two methods were tried. The first was to spray by means of an atomizer a predetermined volume of solution of the drug onto a premeasured quantity of dry pelleted food. The second was to mix the drug with a one percent solution of heat-melted Bacto agar (DIFCO) which had cooled to 45-50°C. After solidification, the medicated agar was diced and offered to the sick turtles. It was necessary that the agar pieces floated on the surface for the turtle to bite and swallow them. If they did not float, the quantity of agar in the solution was reduced until the diced pieces would float. In a liquid medium of a given salinity, diced pieces having a lower concentration of Bacto agar will float more readily than those having a higher concentration. Nevertheless, both methods of incorporating drugs into the food were discontinued in favor of intraesophageal intubation, because effectiveness of drugs in the feed or agar matrix was unknown.

Examples of drugs used in oral administration (the quantity of active ingredient for each 50 g wet body weight of turtle per day is indicated in parentheses) were ampicillin (sodium, 0.5 mg), chloramphenicol palmitate (oral suspension, 2.5 mg), quinacrine hydrochloride (0.35 mg), chloroquine phosphate (1.5 to 3 mg), minocyclin hydrochloride (0.2 to 0.4 mg), neomycin (0.75 mg), and ketoconazole (2 mg). The daily drug dose was divided into two equal portions, one given in the morning and the other in the afternoon. In intraesophageal intubation, the drug solution was delivered by means of a hypodermic syringe fitted with plastic tubing inserted into the esophagus of the turtle.

3. Parenteral injection of small loggerhead and Kemp's ridley turtles was accomplished by subcutaneous injection of the drug. Until 1980, the site of injection was the suprafemoral pouch area immediately above the hind femur on the ventral side of the torso. Thereafter, the dorsal side of the neck was favored because it was discovered that sodium diatrizoate, a commonly used excretory, urographic, iodinated contrast material, was absorbed more rapidly in the dorsal neck than in the suprafemoral pouch area in subcutaneous injection (McLellan and Leong, 1982). These findings implied a similar differential absorption of therapeutic drugs in

the two locations. Examples of drugs (quantity of active ingredient of drug for each 25 grams wet body weight of turtle per day is indicated within the parentheses) used in parenteral administration were ampicillin (sodium, 0.25 mg), chloramphenicol sodium succinate (0.63-1.25 mg), penicillin G (500-2,500 units), gentamicin (0.06 mg), terramycin (0.6 mg), and amphotericin B. Administration of amphotericin B was started at 0.0125 mg per 100 g body weight per day, with weekly increments of 0.0125 mg per 100 g per day.

4. Topical application of certain drugs or chemicals to minor surface wounds or lesions was accomplished with sterile cotton swab. Examples were neosporin ointment (Burroughs Wellcome), fungizone lotion (Squibb), tincture of iodine, merthiolate (Lilly) and gentian violet. In our experience, gentian violet was ineffective against many external lesions, and after it leached into the seawater it appeared to be toxic to small hatchlings, especially when applied too often. Neosporin appeared to be effective against certain presumptive bacterial lesions. The effects of the other topical agents have not been determined.

Diseases, Maladies and Injuries

Sudden Hatchling Death Syndrome

Sudden hatchling death (SHD) syndrome involves death of hatchlings, usually overnight, without forewarning signs or symptoms. On rare occasions, lethargy in hatchlings up to three weeks old may be observed prior to death. This disease, which affects both loggerhead and Kemp's ridley hatchlings, appeared in the former about one week after they arrived at the Galveston Laboratory in September 1977. The disease was so devastating that within four weeks after the first hatchling died, about 40 percent of the entire captive population was lost.

Etiology — Our current hypothesis assumes that SHD syndrome is the result of microbial infection magnified by polluted culture seawater. Pollution could have occurred in the closed (non-flow-through), recycled seawater system due to the gradual build-up of soluble organics from excess food and turtle wastes. Such conditions probably favored multiplication of pathogenic microorganisms, and at the same time created undue stresses on the turtles, thereby lowering their resistance to infection. Evidence to support the above hypothesis came from laboratory analyses and tankside observations. Bacteria were isolated from blood taken from moribund and freshly dead Kemp's ridley and loggerhead hatchlings, and the incidence of SHD syndrome was found to decline sharply (from about four percent to about 0.3 percent or less per day) and almost immediately after the hatchlings were transferred from polluted seawater to clean seawater.

Bacteria isolated from hatchlings showing SHD syndrome were of many varieties. One of them, *Clostridium bifermentans*, was derived from blood cultures of one moribund and one freshly dead loggerhead. This same bacterium has been isolated from cases of gas gangrene in humans (Smith, Conant and Overman, 1964). Another isolate, *Vibrio alginolyticus*, which is a common marine organism frequently associated with diseases in marine animals, was also obtained from blood culture of the same freshly dead loggerhead. The presence of these two bacteria in moribund or freshly dead turtles has suggested their possible role in causing SHD syndrome. However, more experimental studies will be required to confirm their true pathogenicity.

Other bacteria isolated and identified from post-mortem cultures were *Achromobacter pinnatum*, *Ac. delmarvae*, *Aeromonas formicans*, *Aer. punctata*, *Aer. shigelloides*, *Citrobacter (Escherichia) freundii*, *C. intermedius*, *Escherichia aerogenes*, *Pseudomonas* sp. (a pathogen of the Atlantic croaker, *Micropogonias undulatus*), gamma *Streptococcus* sp. (not group A, B or D), *Proteus mirabilis* and *Vibrio alginolyticus*. All of these except *Escherichia aerogenes*, *C. intermedius*, *Achromobacter pinnatum*, *Ac. delmarvae* and *Vibrio alginolyticus* have been associated with infections in humans, terrestrial animals or aquatic animals (Smith *et al.*, 1964; Jawetz, Melnick and Adelberg, 1972; Buchanan and Gibbons, 1974). Moreover, *C. freundii* has been associated with a fetal infection, known as septicemic cutaneous ulceration disease (SCUD) in turtles, Kaplan, 1957; Campbell and Busack, 1979). However, the symptomatology described for SCUD bears little resemblance to SHD syndrome. Determination of pathogenicity of bacterial isolates in relation to SHD syndrome will require more study.

At one time during the 1977 epizootic in loggerhead hatchlings, poisoning from the following sources was considered a possible mortality factor due to: remnants of stale, uneaten fish flesh left in the raceways, and dense blooms of a variety of algae in the closed system raceways. Stomachs of dead loggerhead hatchlings were frequently packed with fish flesh and algae. However, experimental feeding of healthy hatchlings with stale fish flesh and algal masses collected from the raceways and with food-packed stomachs of dead turtles produced no ill effects.

Signs and Symptoms — Although there are usually no observable external signs and symptoms associated with SHD syndrome, careful observations in an epizootic may show a few very weak hatchlings up to three weeks of age that float passively on the water surface with their front flippers hanging loosely downward. In normal hatchlings, the front flippers are folded back over the carapace during rest. Lethargic animals usually die overnight. Slow-dying animals display no external abnormal signs, so the lethargic condition is considered part of the SHD syndrome.

Necropsy of hatchlings with SHD syndrome frequently reveals a feed-packed stomach and fecal impaction in the lower bowel. Paralytic ileus is indicated.

Occurrence — An epizootic of SHD syndrome occurred in September 1977 when newly arrived loggerheads were about one week old. The loggerheads were maintained in concrete raceways with recycled (non-flow-through) seawater. Within four weeks, more than 400 hatchlings (about 40 percent) had died. Peak mortality reached about four percent per day toward the end of the fourth week.

In 1978, Kemp's ridley hatchlings were introduced and reared in fiberglass tanks and basins with daily changes of seawater (non-flow-through). In January 1979, due to outbreaks of various diseases and biting injuries, the ridleys were transferred to individual buckets suspended in the tanks, and seawater was completely replaced at least three times a week. About two percent of the 3,081 ridleys died from SHD syndrome during the 10 months of rearing. In all subsequent annual batches of Kemp's ridleys reared singly in suspended buckets, mortality attributable to SHD syndrome was negligible, estimated at less than one percent for the 1979 year-class and virtually non-existent in year-classes 1980-1983.

A condition designated as "early hatchling mortality" in green sea turtle (*Chelonia mydas*) hatchlings is thought to be an early manifestation of the grey-patch disease (Haines, Rywlin and Rebel, 1974). This latter disease has been attributed to a herpes virus infection (Haines *et al.*, 1974; Rebel, Rywlin and Haines, 1975; Koment and Haines, 1977). The sudden death nature of "early hatchling mortality" appears to resemble that of SHD syndrome. However, we have not observed signs of grey-patch disease in ridley or loggerhead hatchlings. Therefore, we do not consider "early hatchling mortality" and SHD syndrome to be the same.

Remedy — There is no established treatment for SHD syndrome. The key to its prevention seems to be use of non-polluted culture seawater. Frequent exchange or replacement with fresh seawater by manual draining and refilling of tanks is required to ensure good water quality in the rearing tank. Experiments have been conducted toward developing flow-through and recycled water systems, but none of these has proved practical for rearing Kemp's ridleys in our situation.

Chlorination (5 ppm chlorine) of seawater in the reservoir tanks before transferring the seawater to the turtle holding tanks was tried in early 1978 for prophylactic purposes. The results were not encouraging. Incidence of SHD syndrome and many other kinds of diseases in the chlorinated seawater did not appear appreciably different from those in control (non-chlorinated) seawater. Therefore, chlorination is not considered an effective method of prophylaxis. These observations suggest that the causal organisms of many infectious diseases were introduced into the seawater after it was transferred to the raceways and to other rearing tanks from the reservoir tanks. Apparently, the pathogens multiplied under favorable conditions in the culture seawater after the residual chlorine disappeared.

Papillary Dermatitis

Papillary dermatitis (PD), formerly called papillary eruption (Leong, 1979), affects skin tissues. Lesions occur around the eyes and the anus and on the limbs and the plastron. Although hatchlings of both Kemp's ridley and loggerhead can be affected, the disease was more prevalent in the latter species than in the former when reared in our laboratory.

Etiology — PD is responsive to antimicrobial chemicals, and is therefore presumed to be a microbial infection. Bacteria isolated from eye and anal lesions have included *Aeromonas formicans*, *Vibrio alginolyticus*, *V. alginosus* and *Pseudomonas* sp.

Rebel *et al.* (1975) mentioned the presence of papillar tissues in virus-caused grey-patch disease of green sea turtles. Electron microscopic work performed in our laboratory on eye lesions of PD in loggerhead hatchlings did not show the presence of viruses.

Signs and Symptoms — Small, papilla-shaped projections that develop around the eyes and anus and on the skin and the plastronic suture are off-white, yellowish-tan or light-tan in color. When occurring around the eye, multiple papillae usually protrude from underneath the upper and lower eyelids and from the rear corner of the eye-socket. In advanced cases, the papillae may fuse into a crust which may spread to cover the whole eye, blocking eyesight. At the anus, the area surrounding the anal aperture usually becomes pale and swollen and assumes the shape of a ring bearing rows of small papillae. In advanced cases, crusts will form in the affected area.

Localized, small papillae also occur singly on the limbs, protruding through gaps between adjacent scales on the surface of the flippers. These flipper papillae do not form crusts. Similar single papillae can develop on the ventral side of the turtle's body, projecting through sutures between plastronic scutes. Currently eye, anal, flipper and plastronic eruptions are collectively considered under the name of one disease, PD.

PD is frequently accompanied by progressive emaciation of the turtle. Since only one or the other condition alone also is found in some turtles, it is not known yet whether PD and emaciation are related. Emaciation is discussed in more detail as a separate malady later in this paper.

PD progresses slowly. It takes many days for minor eruptions around the eyes and cloaca to become serious, as manifested by crustiness. Papillae on the flippers and the plastronic sutures usually remain solitary and non-fusive.

Occurrences —PD was first observed in one-month-old loggerhead hatchlings in early October 1977 after they were transferred from non-flow-through raceways to flow-through concrete tanks at the Galveston Laboratory's East Lagoon facility on the northeast tip of Galveston Island. The disease spread relatively fast. Within two months, practically every one of the more than 600 juvenile loggerheads held at the East Lagoon facility had contracted the disease. During the same period, 120 similar loggerhead juveniles were maintained individually in the Galveston Laboratory in 4-liter glass vessels for experimental purposes. Despite daily replacement of the culture seawater, approximately 15 percent of them developed PD.

In 1978, when new crops of loggerhead and Kemp's ridley hatchlings were group-reared in closed raceways in which seawater was replaced daily, less than two percent of each species developed PD, although other diseases flared.

Remedy — The prognosis in PD is good when not complicated by emaciation. Although PD alone does not seem fatal, the blocking of eye-sight by necrotic tissues (crust) may affect the turtle's ability to find food, a point not yet proven. However, when PD is complicated with emaciation, the survival rate for affected young turtles is estimated as 20 percent or less, even with medications.

Formalin baths (50 to 100 ppm for seven days with daily replacement of medicated seawater) combined with daily subcutaneous injections of a one percent (w/v) sodium ampicillin solution for 10 to 21 days has proven to be an effective treatment for PD. The lesions usually clear up within four weeks after medication. For unknown reasons, neither formalin bath nor ampicillin injection alone seems as effective as when combined in treating PD.

Ampicillin for injection is prepared in either sterile distilled water or a sterile solution containing 10 percent (v/v) Multiple Vitamin Infusion Concentrate (U.S. Pharmaceutical) and 10 percent (w/v) dextrose. The latter preparation is intended primarily for an emaciation complication. The dosage for injection is 0.25 mg ampicillin, or 0.025 ml of the ampicillin solution per 25 g wet body weight per day, preferably divided into two equal doses, one given in early morning and the other in late afternoon. The whole daily dose may be administered in a single injection, although this may be less effective.

Ampicillin, if unavailable, may be substituted with the less effective chloramphenicol sodium succinate. The recommended dosage is 1.25 mg chloramphenicol per 25 g wet body weight per day. Inconclusive results were obtained with the following chemicals and antibiotics in clinical tests against PD in loggerheads: malachite green, methylene blue (may be toxic as a bath), gentian violet (may be toxic to very young hatchlings), penicillin G, oxytetracycline, minocycline, erythromycin, and furacin. The routes of delivery of these drugs included subcutaneous injection, medicated bath and topical swabs.

Emaciation

Emaciation is a wasting syndrome with various underlying causes in young Kemp's ridleys and loggerheads. Affected turtles are weakened and their health deteriorates progressively until death. From the first discernible signs of emaciation until death, the whole process may last up to two months or more. In general, it appears that the younger the turtle, the more susceptible it is to emaciation and the more difficult is its recovery. Circumstantial evidence has indicated that loggerhead hatchlings are more vulnerable to emaciation than ridley hatchlings.

Emaciation in animals is the net result of dehydration or depletion of body tissues or both. There are many possible causes. Examples are microbial infection, metabolic or catabolic dysfunctions, inappropriate diet and involuntary reduction in intake of food, water or both. The last cause can be dismissed as a factor contributing to emaciation of turtles at the Galveston Laboratory, because plentiful food and sea water were always available to the animals, but all the other causes have been considered as potential contributors to emaciation.

A bacterium, *Mycobacterium marinum*, was isolated from the lungs of two dead and severely emaciated loggerhead hatchlings, the carcasses of which were kept frozen for over one month at 0 °C prior to necropsy. It was possible that emaciation in those turtles was a result of lung infection by the bacillus. In necropsy, small fecal impaction was frequently observed in the lower bowel of emaciated specimens indicative of constipation prior to death.

Reichenbach-Klinke and Elkan (1965) suggested that faulty nourishment combined with lack of exercise may produce constipation in captive turtles. Thus, faulty diet could have been a possible cause of constipation in loggerheads of the 1978 year-class, leading to a form of emaciation which gave the hatchlings a shrivelled but hardened or block-like appearance. These turtle hatchlings were fed a diet composed exclusively of commercial feed (Master Mix S. S. Turtle Feed 9349) which might have been unsuitable for them, because within five months 95 percent of the head started loggerheads became emaciated and many died. Loggerhead hatchlings of the 1977 year-class, which were fed raw fish flesh during their early stage of life, did not contract a similar form of emaciation. Neither Kemp's ridleys at the Galveston Laboratory nor olive ridleys (*L. olivacea*) (Lyle Kochinsky, Nova University, Dania,

Fla., personal communication), appeared to have a similar problem when fed with the same pelleted food.

Lack of exercise was an unlikely cause of constipation in the Galveston turtles. Fecal impaction of the bowel was observed in necropsy whether the hatchlings had been allowed to swim freely in large rearing tanks with ample opportunities to exercise or had been confined in individual buckets.

There have been reports of emaciation and its causes in other turtles and reptiles. An epizootic among 2,000 captive-reared green sea turtle hatchlings at Cayman Turtle Farm (1983), Ltd., Grand Cayman, British West Indies, resulted in typically flat, weak and emaciated hatchlings, and was caused by a coccidian parasite, *Caryospora* sp. (Rebel, Rywlin and Ulrich, 1974). Marcus (1977) reported that reptiles infected with intestinal amoebiasis exhibited non-specific signs of decreased activity, progressive weight loss and anorexia although they also exhibited diarrhea, vomiting and excretion of bloody stools. Although emaciation has not been mentioned, it is reasonable to expect that in prolonged amoebiasis, emaciation eventually would follow. No evidence of similar parasitic infections has been found in the Kemp's ridleys and loggerheads head started at the Galveston Laboratory.

Weight loss and emaciation in turtles at the Penrose Laboratory of the Zoological Society of Philadelphia have been attributed to inanition (Cowan 1968), which is physiological dysfunction due to insufficiency of nutritional factors necessary for health and well-being. Berklow (1977) classified nutritional deficiency as either primary or secondary. Primary deficiency is due to inadequate nutrient intake, and secondary deficiency is a result of failure to absorb or utilize nutrients, increased nutritional requirements or excessive excretion. Digestion and absorption may be disturbed by gastrointestinal disease, and utilization and storage of nutrients may be impaired due to endocrine dysfunction, inborn errors of metabolism, severe infection or degenerative disease. Experiments will be required to determine whether the turtles reared at the Galveston Laboratory suffered from primary or secondary deficiency or both.

Captive Kemp's ridley hatchlings suffered a fatal form of systemic mycosis with the fungus *Paecilomyces* isolated as a presumptive causative organism (see *Yolk-sac Mycosis*). At first glance, such affected hatchlings appear emaciated, but careful examination will show that they do not possess the typical lean-and-wrinkled-neck characteristic of the emaciation syndrome as defined in this section. Instead, the infected turtles usually exhibit taut cervical skin with good muscle tone. Therefore, mycosis is not considered a cause of emaciation.

Signs and Symptoms — Sick turtles gradually lose weight and have a sunken plastron and a wrinkled and lean-looking neck. In advanced cases, anorexia is common, the vertical plane of the body trunk becomes thin and looks flattened due to severe sinking of the plastron, and the turtle becomes very weak and lethargic. Quite often the pliable skin of the suprafemoral pouch between the base of the hind femur and the shell sags and fails to assume a taut, domed shape as in normal turtles. In some cases, the body shell may become softer and more pliable than normal. On other occasions, the whole torso may appear relatively hard, solid and shrivelled as in constipated loggerheads of the 1978 year-class, which were suspected of having been fed a faulty diet. X-radiography shows that in "soft-shelled" individuals, the ribs in the carapace are poorly developed.

Necropsy shows that emaciation could be associated with one or more kinds of internal disorder such as fecal impaction of bowel (most common), mycobacterial pneumonia, intestinal perforation and peritonitis. In fecal obstruction of the bowel, the lower colon is packed with hard feces, while the rectum is usually void.

Growth of hatchlings after recovery from emaciation is often stunted, the animals retaining a dwarfed physique and having a weak and unhealthy appearance. A few recovering turtles also maintain a rather soft and pliable shell.

Occurrence — Emaciation first appeared in early October 1977 in one-month-old loggerhead hatchlings maintained in outdoor, flow-through raceways. The condition was frequently concurrent with papillary dermatitis (PD) described earlier in this paper. Emaciation, either uncomplicated or complicated with PD, continued to spread when the loggerheads were transferred to an indoor, non-flow-through raceway in December 1977 in anticipation of colder weather. The seawater in the raceway was recycled through an oyster-shell filter bed. Emaciation soon reached epizootic scale, affecting more than 300 of about 600 hatchlings within two months. By April 1978, about 80 percent of the turtles had exhibited emaciation at one time or another, and many of them died. Meticulous clinical care and the development of new therapeutic methods helped some (about nine percent) emaciated turtles recover.

Kemp's ridleys and loggerheads of the 1978 year-class were mass-cultured in non-flow-through raceways. An estimated 10 to 15 percent of the ridleys became emaciated, often in association with PD, and died within the first nine months. The loggerheads did well in the first five months, but later 95 percent of them developed the hardened and "block" form of emaciation with no contemporary PD. Necropsy showed that most of the emaciated loggerheads had hard feces in the colon. Nevertheless, these data should not be taken to imply that Kemp's ridleys were less susceptible to emaciation than loggerheads until more conclusive data from controlled experiments are obtained, because the ridleys of the 1978 year-class were reared in solitude in individual buckets in the raceways starting from the eighth month, while the loggerheads continued to be kept under conditions of non-isolated, group rearing. The isolation culture of sea turtle hatchlings had been found to play an extremely important role in disease prevention, although

the reason is unknown. Emaciation occurred only sporadically in Kemp's ridley hatchlings of subsequent year-classes kept under isolation rearing.

Conditions resembling emaciation have been reported in other turtles and reptiles. At the Penrose Research Laboratory, among 1,249 cases of reptilian mortality including 92 turtles and tortoises, 60 percent were characterized by a wasted condition, depletion of fat depots and fragile tissue and skin (Cowan, 1968). In the spring of 1973, 2,000 captive green turtle hatchlings at the Cayman Turtle Farm were ill and described as typically flat, weak and emaciated (Rebel *et al.*, 1974).

Although no emaciated turtle hatchlings have been noted among those shipped to us, other workers have reported that on occasion newly acquired turtles may be received in a severely starved and dehydrated condition (Campbell and Busack, 1979). The age of these turtles was not specified. The emaciation syndrome encountered in the Galveston Laboratory was not shipment-related.

Remedy — Current prognosis in emaciation is grave because of difficulty in diagnosis and lack of fundamental understanding of the etiology. Despite treatment of symptoms and general clinical care given to sick turtles, the recovery rate has been less than 20 percent.

To a large extent, emaciation seems preventable by isolation-rearing. For example, in Kemp's ridley hatchlings of the 1978 year-class, an estimated 10 to 15 percent of the animals contracted fatal emaciation while being held in free-swimming groups in raceways. Since then, solitary rearing has been the standard, and incidence of emaciation has decreased correspondingly. In the 1980 year-class, the incidence of emaciation was less than two percent. For loggerheads, there have been no comparable data for both isolation and non-isolation rearing within raceways, but in 1977 loggerhead hatchlings that were isolated in individual containers under laboratory conditions were rarely afflicted with emaciation, while about 80 percent of those group-reared in raceways suffered from the syndrome.

In clinical care, emaciated turtles should be individually isolated as soon as possible. The culture seawater should be completely replaced every day. When only a few animals have to be cared for, and staff is sufficient, it may be wise to disinfect the container with sodium hypochlorite every time the used seawater is replaced. This disinfection procedure may not be practical, and therefore omitted, when a large number of sick turtles is involved. Resources permitting, non-corrosive and autoclavable containers may be used, so that they can be steam-sterilized.

It is preferable to feed sick hatchlings thin slices of frozen-and-thawed raw fish flesh in lieu of pelleted commercial feed. Freezing and thawing can destroy some potential pathogens, and freezing can preserve the nutrients in the fish flesh in storage. The daily ration should not be excessive: three percent of body weight or an amount which the sick turtles can totally consume in an eight-hour work day. Animals which do not eat may be fed a semisolid cereal diet by means of intraesophageal intubation. Occasional starving of anorexic animals for 24 hours does not appear to harm them. Either independently or in combination with intubation feeding, subcutaneous injection of life-sustaining doses of a sterile dextrose-multivitamin solution is also recommended. Intubation feeding, injection or both may be continued until the animals regain appetite.

In clinical tests, injections of antibiotics such as chloramphenicol, ampicillin, oxytetracycline and gentamicin did not produce significant therapeutic effects, nor did bath treatments with formalin, methylene blue, chloramphenicol, minocycline (either alone or in combination with erythromycin) or gentamicin. The recovery rate from emaciation was no better in medicated turtles than in non-medicated ones.

A number of cathartics including warm water, light mineral oil, milk of magnesia and Metamucil (psyllium hydrophilic mucilloid; Searle Laboratories) have been tested in attempts to correct constipation in emaciated turtles. These cathartics were administered either orally by intraesophageal intubation or as an enema as appropriate. Only milk of magnesia at 0.025 ml per 100 g body weight per day *per os* occasionally induced a slight level of defecation in two out of six juvenile Kemp's ridley patients. There are unconfirmed reports that castor oil appears to be effective in inducing defecation in some constipated Kemp's ridley hatchlings.

Focal Erosive Dermatitis

Focal erosive dermatitis (FED) is characterized by the progressive disintegration of cutaneous tissues and formation of shallow, erosive lesions which are sometimes covered with crusts of necrotic tissues. The disease may affect the skin or surface tissues of many body parts, such as the eyelids (blepharal FED), head (cranial FED), carapace (carapacial FED), flippers (flipper FED) and nose (nasal FED). An additional form which erodes the edge of a flipper is named flipper-edge focal erosive dermatitis (Flipper-edge FED).

Although the lesions of FED are sometimes rather unsightly and alarming, there has been no evidence that the disease by itself is fatal.

Etiology — FED is presumably caused by bacterial infection. Several bacteria have been isolated from lesions on the eyes and carapace of Kemp's ridleys, and these lesions can be cured by the use of antibacterial drugs.

Injury to the skin is perhaps a predisposing factor for bacterial invasion. Skin injuries may be caused by mutual

scratching or biting among hatchlings that are placed in close contact (group-rearing) with each other. Also, human handling of the turtles during routine maintenance or transshipment may contribute to skin injuries. The supposition of an injury-infection is supported by the observation that there was a rapid decline in incidence of FED when the group-rearing approach was replaced by solitary rearing.

The bacteria isolated from eye and carapace lesions have included *Aeromonas formicans*, *Vibrio anguillarum*, *V. alginolyticus* and *Citrobacter freundii*. One or more of these bacteria may be the causative agent(s) contingent upon further pathogenicity studies. Stickney, White and Perlmutter (1973) reported isolation of an *Aeromonas* sp. from superficial lesions in three and a half-month-old loggerhead hatchlings. In reference to this findings, Sinderman (1977) commented that *Aeromonas* spp. were common inhabitants of aquatic environments, and might be facultatively pathogenic to animals living under conditions of environmental stress.

An anaerobic bacterium, *Bacteroides* sp., has been considered as the causal organism of a skin disorder in one- or two-month-old loggerhead hatchlings (Witham, 1973a). The disease has been described to produce necrotic, spreading, nonwalled skin lesions causing most of the hatchlings to die within a week of the first appearance of the malady. It is dubious that this disease is the same as FED, since FED does not seem to be fatal by itself.

Signs and Symptoms — Except in carapacial FED, freshly formed lesions have not been observed. Relatively fresh lesions of carapacial FED have an ivory color due to exposure of underlying ground substance of the shell after the dark-colored epidermis erodes away. Older lesions are greyish-tan or yellowish-tan, usually shallow, localized erosions of the skin. In blepharal FED, it appears that the eyelids become puffy and discolored in a pre-erosion phase, but this observation remains to be confirmed.

Typically FED forms shallow lesions below the skin level due to the loss of surface tissues. The lesions are usually irregular in shape, spreading out in all directions and producing an uneven margin. They are variable in size, ranging in diameter from about 2 mm, when first noticeable on hatchlings in the raceway, to more than 1.5 cm. Often as neighboring lesions spread, they become merged, producing a large necrotic patch.

Besides the typical FED lesions on the skin surface of the flippers, there is a condition in which the entire thickness of part of the edge of a limb is eroded away, giving a worm-eaten appearance. Because the condition involves the invasion of deeper rather than just surface tissues, it may be a different form of disease. However, until further studies, it is included under FED for the purpose of this paper. The name flipper-edge FED is given to this abnormal form of FED to distinguish it from the typical, skin-surface type of flipper FED.

Usually there is little if any build-up of crust in cranial, carapacial and flipper FED lesions or in flipper-edge FED lesions. In blepharal and nasal FED, crusts may be present. In severe cases, an overabundance of crusts may cause either total or partial closure of the eyelids or blocking of the nostrils.

Occurrence — In early October 1977, approximately 600 four-week-old loggerhead hatchlings of the 1977 year-class were transferred from closed concrete raceways to flow-through concrete tanks. Within two weeks, many of the young turtles began to exhibit signs of cranial FED. Within the following two months, an estimated 80 percent of the population contracted cranial FED of either severe or limited extent.

During the summer of 1978, 3,081 newly emerged Kemp's ridley hatchlings were acquired and reared in close contact in closed-system, fiberglass raceways.

These turtles were extremely aggressive, chasing and biting each other vigorously. Within three months, practically every turtle was afflicted with either one or more forms of FED at one time or another.

Immediate isolation of sick hatchlings into individual vessels coupled with drug medication generated excellent therapeutic effects. However, when about 2,000 cured turtles were returned to the mass populations in the raceways, they were re-infected. Beginning in January 1979, the isolation-rearing method (see section Rearing of Turtles) was implemented. The incidence of focal erosive dermatitis rapidly fell. To this date, FED no longer constitutes a health threat to captive Kemp's ridleys reared in isolation from each other during their first year of life.

Remedy — Isolation of individual sick turtles and daily replacement of culture seawater coupled with subcutaneous injection of ampicillin (sodium) at 0.25 mg per 25 g wet body weight per day is effective treatment for FED, particularly the blepharal and flipper forms. A seven-day formalin bath (50 to 100 ppm) is also effective against the flipper-edge form of FED. Lesions are usually healed in about four weeks following onset of medication.

The best prophylaxis against FED is to raise the hatchlings by the isolation-rearing method as described in the section Rearing of Turtles.

Self-limiting or spontaneous healing of lesions frequently occurs in carapacial FED when the afflicted turtles are individually isolated and provided with clean seawater daily. The healing process may last up to two months or more. Administration of ampicillin does not seem to shorten the wound-healing time in carapacial FED, as it does in blepharal FED, or have harmful effects from ampicillin been observed. Since blepharal, flipper and carapacial FED often occur simultaneously in a single Kemp's ridley, both ampicillin and isolation treatments are given to such turtles having a mixture of different forms of FED.

Cranial and nasal FED do not respond to medication and isolation as well as do blepharal and carapacial FED. Cranial and nasal lesions can be very persistent. Some cases have failed to respond to clinical treatments for four months or more. Occasionally, a lesion may even expand while the turtle is under clinical care.

A condition that appears to resemble FED and that is designated as focal necrosis of skin has been reported in young green and loggerhead sea turtles (Witham, 1973b). However, unlike FED, focal necrosis of skin has been described as causing death of the affected animals unless treated with potassium permanganate (1 g per 220 liters in bath treatment). Therefore, FED and focal necrosis of skin are probably diseases of a different nature. In short-term observations, application of potassium permanganate bath does not cure FED in Kemp's ridleys.

Aggressive Biting

Aggressive biting is included in this paper because it happens extensively among hatchlings of Kemp's ridley and frequently causes serious physical damage in the bitten turtles. There is no evidence that aggressive biting contributed directly to the death of a turtle at the Galveston Laboratory, but apparently the inflicted wounds provide a convenient portal of entry for pathogenic microorganisms. Further, it may be reasoned that if a turtle with a missing eye or part of a flipper were released into the sea, chances of survival would be reduced, because such a handicapped turtle would be easy prey for larger marine animals. Therefore, a standard practice in the head start project has been not to release such injured sea turtles.

It has not been conclusively established that Kemp's ridleys older than one year do not bite each other, but evidence has indicated that they at least are not as aggressive as individuals one year or less in age.

Etiology — Aggressive biting apparently is an intrinsic behavior in young Kemp's ridleys. Why such aggressive behavior is so intense in captive Kemp's ridley hatchlings but not in loggerhead hatchlings under similar environmental conditions is unknown. What actually stimulates Kemp's ridley hatchlings and juveniles to bite one another is also not understood. Color and movement are two presumptive stimuli, but controlled experiments are needed to evaluate this hypothesis.

Signs and Symptoms — Almost every part of the body of a Kemp's ridley turtle that is accessible to the mouth of another Kemp's ridley is vulnerable to injury through biting. The most vulnerable sites are the flippers, the edge of the shell, the neck and the head. In serious cases, a large piece of body tissue or a portion of a limb may be torn off, leaving behind large fleshy wounds.

Occurrence — Aggressive biting began when captive Kemp's ridleys were about three weeks old. Among the 3,081 ridley hatchlings acquired in late July 1978, more than 800 (27 percent) showed signs of bite wounds by mid-August. Bite-wound incidence continued to mount and affected more than 80 percent of the turtles by January 1979. Afterwards, all the hatchlings were individually confined in plastic buckets in isolation-rearing and therefore were prevented from biting each other. Surprisingly, when solitarily confined ridleys were returned to free-contact and group-rearing raceways upon recovery from disease or injury, they showed a higher level of aggressive biting behavior than before they were isolated.

Minor chasing and biting were observed in hatchling loggerheads, but the turtles suffered no apparent injuries. Hatchlings of the green sea turtle seem even more docile. In summer 1978, no aggressive biting was observed by the senior author among approximately 100 young green sea turtles averaging 250 g each, held in an outdoor concrete tank at Miami Seaquarium, Miami, Florida.

Remedy — Individual isolation-rearing is the best protection against aggressive biting and wounding in Kemp's ridley, but it is labor-intensive, especially in a mass-culture situation such as that at the Galveston Laboratory. Unfortunately, no better rearing system has been developed to date for Kemp's ridleys.

Injured hatchlings are immediately isolated in individual vessels that receive daily changes of seawater. For wound treatment, a piece of sterile surgical sponge or a cotton swab is used to absorb the water from the injured area, followed by cleansing with hydrogen peroxide (three percent) on a cotton swab. When the wound is dry, it may be treated topically with a general antiseptic such as neosporin ointment or merthiolate, and the turtle is then returned to the holding vessel. In serious injuries, ampicillin (sodium) may be administered subcutaneously at a dose of 0.25 mg per 25 g body weight per day. Wounds usually heal in four to eight weeks. The healed surface will resume the normal dark color, but the bulk of the missing tissue is not replaced.

Focal Dermal Granulosis

Focal dermal granulosis (FDG) produces localized lesions in the form of discolored and often grainy patches in the skin of young sea turtles. The disease is not fatal, and the affected turtles are active and continue their normal growth.

Etiology — A bacterium, *Pseudomonas alcaligenes*, was isolated from the deeper tissues in focal dermal granulosis lesions which had been surface-sterilized with 70 percent alcohol and tincture of iodine. Further studies are needed to determine the pathogenicity of that organism.

Signs and Symptoms — Discolored, localized lesions that have a granular appearance can be found on the surface

of the shoulders and the axilla of the front flippers. Lesion color ranges from light grey to greyish-tan, often overlaid by a thin network of bright yellow substance.

The lesions are flat and slightly elevated. They are usually more or less oval-shaped in the neck, but irregularly shaped in other affected skin areas. When first noticed on hatchlings in the raceway FDG lesions are usually about 1 cm or more in length or diameter, depending on their shape. Such lesions continue to expand in time. An axillary lesion may spread to cover an irregular, area 2 cm or more in diameter.

Occurrence — An estimated 2 to 3 percent of loggerhead and Kemp's ridley hatchlings contracted FDG in 1977 and 1978 under group-rearing conditions. However, no deaths were attributed to the disease.

Witham (1973b) reported a fatal condition called focal necrosis of skin in tank-reared green and loggerhead turtles. Since FDG is non-fatal, we assume that it is a different disease from focal necrosis of skin.

Remedy — Static formalin bath (50 to 100 ppm) continued for five to seven days is effective. The medicated bath water is replaced completely on a daily basis. Occasionally, signs of healing appear after only three days of medication. Care should be taken not to over-extend the treatment period. Over-treatment will delay instead of promote healing. Potassium permanganate bath (5 ppm) was tried, but the results were inconclusive.

Occasionally, FDG is self-healing when the affected turtle is kept isolated in clean seawater. The FDG lesion sheds a scab and leaves behind a discolored scar on the skin. The healing and scab-shedding processes are promoted with formalin bath treatments.

Scolecobasidiosis

Scolecobasidiosis is an infection caused by a fungus belonging to the genus *Scolecobasidium*. At least two kinds of scolecobasidial infection have been observed: scolecobasidial pneumonia (SP), an infection of the lung, and scolecobasidial osteomyelitis (SO), an infection of the bone. An infected turtle may live a long time before it dies. In some cases of SP, progress of the disease may arrest spontaneously under the right environmental conditions, and the animal continues to live with only one functional lung and with abnormal swimming and floating patterns; i.e. tilted swimming and side-floating. Both SP and SO have been encountered only in young Kemp's ridleys and not in loggerhead hatchlings.

Etiology — The fungus *Scolecobasidium constrictum* has been observed repeatedly in and isolated from tumor-like or cyst-like spherical bodies in affected lungs and rear flippers of Kemp's ridleys. The same fungus has been observed or isolated in a limited number of cases of scoliosis and inflamed shoulder joints. Cultures of synovial fluid taken from inflamed shoulders were negative for bacterial infections.

Sometimes other fungi such as *Paecilomyces* sp., *Penicillium* sp. and *Cephalosporium* sp. are also recovered in cases of SP. Their role in such infection has not been determined. *Penicillium* and *Cephalosporium* are probably contaminants. *Paecilomyces* sp. is also often isolated from smaller tissue nodules as described under Internal Nodular Mycosis.

Signs and Symptoms — In the majority (estimated 97 percent) of SP cases, an infected turtle swims or rests on the seawater surface with its body tilted to one side. This side-floating syndrome is usually the first sign that the animal may have contracted SP. Another frequently observed gross sign is bulging of the dorsal front half of the carapace of the buoyant side of a tilted turtle into a minor hump. Side-floating and humping are usually not observed until the animals are four months old or older, when the disease is in a relatively advanced stage. Methods for early diagnosis are not known. It should be noted that side-floating alone is not specific for SP. Turtles having lung aplasia (described separately in this paper) also exhibit a similar sign. Jacobson *et al.* (1979) reported isolation of three fungi, *Sporotrichum* sp., *Cladosporium* sp. and *Paecilomyces* sp., from infected lungs in green turtles which exhibited tilted swimming behavior.

Necropsies of turtles that have died from SP generally show consolidation of tissues of one of the lungs into a relatively large spherical cyst. This tumor measures as much as 2 cm or more in diameter and may be empty or filled with blood or a clear fluid. Tissue consolidation appears to occur more frequently at the frontal portion of the lung. The interior wall of an empty sphere may be dark-green, but if the sphere is filled with fluid, the wall is lined with coagulated blood. Also residing in the wall are fungal mycelia and spores from which the fungus *S. constrictum* is invariably isolated. Another fungus *Paecilomyces* sp. is also sometimes isolated.

Generally, only one of the lungs develops a large spherical body (2 cm or more in diameter). Smaller solid tissue spheres or nodules ranging from pin-head size to 1 cm in diameter may also be present in one or both lungs, in the liver and occasionally in the cardiac auricles. These smaller nodules cannot be distinguished from those caused by other fungal organisms such as *Paecilomyces* sp. Therefore, unless they are confirmed as scolecobasidial nodules through the actual isolation of *Scolecobasidium* they are categorized as Internal Nodular Mycosis for the purpose of this paper.

Images of infected lungs can be seen in X-radiographs, but the diagnosis is non-specific. Also, X-ray can reveal that a non-affected lung may expand over to the other side of the spinal column.

SO affects the bones of the hind flippers and perhaps the vertebrae and the shoulder joints. Tissues adjacent to infected bones in a flipper swell to form a tumor. X-ray shows that the phalanges, the metatarsals, the tarsals and the tibia may be eroded to a greater or lesser extent.

There are indications that SO can cause scoliosis of the vertebral column as well as swollen and stiffened shoulder joints. X-ray shows the formation of a curvature in the spine and sometimes there is erosion in some of the vertebrae. *S. constrictum* has been recovered by culturing tissues taken by biopsy adjacent to the affected vertebrae. In correspondence to curvature of the internal spinal column, the ridge of the carapace is also curved, thus affording an external means of detecting scoliosis. The specific relationship between scoliosis and scolebasidiosis remains to be determined.

When a turtle experiences an inflamed shoulder joint, the affected front flipper shows stiffness and difficulty in swimming. On closer examination, the shoulder joint is swollen, and X-ray may show erosion of the head of the humerus at the glenoid fossa. *S. constrictum* sometimes can be recovered through culturing the affected tissues as in scoliosis.

Occurrence — With the exception of one unconfirmed case in a young loggerhead in 1981, all cases of scolebasidiosis have been observed in Kemp's ridleys. SP was by far the most frequent of the two forms of scolebasidiosis in Kemp's ridleys with 7, 18, 4, 23, 8 and 6 cases in the 1978 to 1983 year-classes, respectively. Since SP is confirmed only through isolation of the causal fungus or by actual observation of the characteristic tumefaction of the lung at necropsy, and since we could not perform detailed laboratory analysis on every turtle, there may have been cases of SP that escaped our detection.

There were three cases of rear-flipper SO and two cases of scoliosis in the 1979 year-class of Kemp's ridleys. One of the scoliotic cases was associated with arrested SP in a Kemp's ridley that has been kept in captivity for three years because of fungal pneumonia, but there was no sign that the infection had spread to the vertebrae. In the other case of scoliosis, X-ray showed lesions in a few vertebrae. There were at least two cases of inflamed shoulder joint diagnosed in the 1979 year-class of Kemp's ridleys.

Casual observations suggested a direct relationship between the incidence of SP and lower environmental temperatures, especially when there were frequent fluctuations between cold and warm temperatures during winter.

Remedy — In clinical trials, fungizone (Squibb; containing amphotericin B as the active ingredient) was injected subcutaneously into six young ridleys, averaging about 300 g in body weight and suffering from advanced SP. The starting dosage was 0.0125 mg amphotericin B per 100 g wet body weight per day, with weekly increments of 0.0125 mg per 100 g. Four turtles died after one to two months of medication indicating that the therapy was ineffective. Therefore, treatment was terminated.

Other forms of chemotherapy were also tried, but found to be ineffective. These methods included formalin bath, malachite green bath, and oral ketoconazole (dissolved in dimethyl sulfoxide). In general, evaluation of chemotherapy for SP was difficult because there was lack of specific internal monitoring and diagnostic methods. It is possible that some of the antifungal drugs are effective against scolebasidiosis if applied at an early stage of infection. Therefore, research should be done to discover and develop early diagnostic and monitoring methods.

In 1978 and 1979, most ridley turtles that were afflicted with SP eventually died. Occasionally, the infection in some turtles seemed to be arrested spontaneously without medication. Upon discovery of turtles with signs of SP, these turtles were isolated quickly and were given a warm (26° to 28°C) clean seawater environment. In 1982, eight live Kemp's ridleys of the 1981 year-class were diagnosed as having presumptive SP. After receiving more than three months, non-medication, clinical care, six (75 percent) of the animals survived, and the SP infection in them appeared to have been arrested. This 75 percent recovery rate was an exception rather than the norm. In our experience, the rate of spontaneous recovery of SP infection does not exceed 20 percent. Nevertheless, in the absence of effective chemotherapy, the key to spontaneous recovery of SP-stricken Kemp's ridleys is immediate isolation and provision of a clean, stable and warm (26° to 28°C) seawater environment. It is not necessary to reduce the feeding level unless the animal becomes anorexic.

The isolation-rearing method was also effective against inflamed shoulder joints. After about three months solitary rearing in two turtles with SO, the swelling and stiffness at the shoulder joints subsided and the turtles were able to use the flippers freely again in swimming.

There is no effective treatment for scolebasidial infection of flipper bones and scoliosis. Surgical removal of infected soft tissues in two cases of rear-flipper SO followed by daily oral administration of ketoconazole (4 mg in dimethyl sulfoxide per 100 g wet body weight per day) was ineffective. Both turtles died within one month. The two turtles with scoliosis also died.

At this point, the best strategy seems to be prevention. Based on our experience, it appears that avoidance of sudden changes of ambient temperature from warm to cold would reduce incidence of scolebasidiosis.

White-suture Syndrome

White-suture (WS) syndrome involves the whitening and broadening into a narrow ribbon shape of the suture lines between scutes of the carapace. There are two kinds of WS syndrome: (1) dull white-suture (DWS) syndrome and (2) shiny white-suture (SWS) syndrome. These two kinds of WS syndrome not only are morphologically different but also respond differently to the same medication. It is most likely that their etiology, which remains to be determined, is also not the same. In this paper, they are grouped under the same heading for convenience.

Etiology — The etiology has not been determined for either form of WS syndrome. *Fusarium*-like fungal spores have been observed in the white ribbon on the suture line in the DWS syndrome. Confirmation of the pathogenicity of this fungus will require further studies.

Signs and Symptoms — In DWS syndrome, the carapace sutures widen into a narrow ribbon shape and assume a dull white or greyish-white color. The edges of the "ribbon" are smooth and non-undulate. Transformation and discoloration of the sutures are gradual, starting out with a few sutures and slowly spreading to the others. In severe cases, all sutures are affected. Microscopic examinations of ribbon materials reveal a mixture of debris, bacteria, protozoans and *Fusarium*-like fungal spores.

In SWS syndrome, the white ribbon over the transformed suture is shiny or glistening with a bluish tint and appears slimy. The edges of the ribbon are uneven and undulate. Microscopically, the ribbon material is composed of debris, bacteria and protozoans.

Occurrence — DWS syndrome occurred very commonly in young loggerheads of the 1977 year-class. In static seawater in raceways, more than 70 percent of the turtles contracted DWS syndrome. In loggerheads and Kemp's ridleys of the 1978 year-class, less than 10 percent of the young turtles contracted either DWS or SWS syndrome, and since then either syndrome was encountered only sporadically. The decrease in WS syndrome could have been due to the progressive modification of turtle rearing methods, resulting in a significant improvement of seawater quality in the culture tanks.

Remedy — DWS syndrome responds well to formalin bath (50-100 ppm) treatments carried out for three to seven days with daily replacement of medicated seawater. One week or more after termination of the bath treatment, the ribbon-like white material, which by then has turned grey, detaches from the affected sutures. The healed sutures on the carapace resume a normal appearance. Formalin bath is not effective against SWS syndrome.

From all indications, both DWS and SWS syndromes seem to be preventable through the provision of a sanitary seawater environment.

Yolk-sac Mycosis

This disease is called yolk-sac mycosis (YSM) because the causal fungus invades the yolk-sac inside the body cavity of neonate Kemp's ridleys. Most infected hatchlings die young, usually within the first month after hatching.

Etiology — A fungus, *Paecilomyces* sp., has been frequently observed in or isolated from infected yolk-sac tissues. This fungus is a presumptive causal organism for yolk-sac mycosis.

Signs and Symptoms — Hatchlings usually die very young with little external manifestation of disease. Therefore, it is very hard to detect infected live hatchlings in a mass-culture situation such as that at the Galveston Laboratory.

At necropsy, the infected yolk-sac is usually found to be hardened into a block with the internal yolk material turning into a friable mass. Vascular congestion is often prominent on the yolk sac surface. Microscopic examination of diseased yolk materials show the presence of abundant fungal hyphae and spores characteristic of *Paecilomyces*. The fungus which can be isolated from infected tissues will grow on Sabouraud dextrose agar supplemented with a three-salt solution at 26°C.

Occurrence — YSM was first observed in hatchlings of the 1980 year-class of Kemp's ridleys. Since then, the disease occurred in every year-class through 1983. There were 7, 59, 5, and 3 cases in the 1980 to 1983 year-classes, respectively. Younger hatchlings appeared to succumb more readily to the disease than older ones.

Remedy — There is no known therapy for YSM. Currently, the disease is only discovered at necropsy, too late to provide a remedy even if one were available. Methods are needed for early detection and diagnosis. Moreover, information is needed on the source and mode of infection to guide possible development of prophylaxis.

Internal Nodular Mycosis

Internal nodular mycosis (INM) refers to formation of nodules in an internal organ as a consequence of mycotic infection. The nodules are solid and relatively small in contrast to the larger, spherical, hollow cysts formed in the lungs in advanced stages of SP. It is possible that some of the nodules in INM are early lesions of SP, and some are caused by *Paecilomyces* infection. Grossly, INM nodules are indistinguishable with respect to etiological agent. The only way to distinguish them is detection of the specific fungus in laboratory analyses such as culturing the nodular tissue to isolate the microorganism or direct microscopic examinations of tissue specimens. Unfortunately, such diagnostic procedures are time-consuming and labor-intensive, especially when the nodules are numerous and

several organs are infected. To compound the problem, sometimes *Scolecobasidium constrictum*, *Paecilomyces* sp. and perhaps other fungi such as *Cephalosporium* sp. and *Penicillium* sp., which are thought to be contaminants, are cultured from the same nodule. Therefore, precise diagnosis is not easy when many turtles are infected and when resources are limited. In light of such difficulties, we do not attempt at this time to distinguish the different kinds of mycotic nodules, but group them as one disease category, INM.

INM appears to progress slowly in a hatchling host after initial infection. The net result of INM infection is usually death of the host.

Etiology — The fungi *S. constrictum* and *Paecilomyces* sp. have been frequently isolated from nodular tissue specimens. These two fungi are presumptive causal organisms. Other fungi such as *Cephalosporium* sp., *Penicillium* sp. and *Aspergillus* sp. are found occasionally. Pending further studies, we regard them as either secondary pathogens or contaminants.

Signs and Symptoms — Hatchlings afflicted with INM do not always display external manifestations. Those that do usually but not always show an atrophied body trunk, which resembles a solid hard block and is thickened at the horizontal plane (i.e., from carapace to plastron), so that the whole trunk appears bloated. The neck, however, is not affected as in emaciation syndrome; i.e., it remains relatively full with good muscle tone and its skin is taut, not wrinkled. An afflicted hatchling often, but not always, floats higher in the seawater than a normal one, and its activity ranges from normal to lethargic. Appetite for food may be reduced. Infected turtles usually continue to feed until they become very weak or approach death.

External signs are nonspecific for INM. Definitive diagnosis is dependent upon observing fungus-infested nodules in internal organs. Unfortunately, techniques for such observations have not yet been firmly established for live hatchlings. X-radiography coupled with *in vitro* culture and isolation of fungus from biopsied tissues provides a promising approach to diagnosis of this mycosis.

Many major visceral organs or tissues can be afflicted with the mycotic nodules. Nodular formation is most frequently (about 85 percent of the cases) encountered in the lungs. It is also found in the kidneys, liver, and mesentery, and occasionally in the alimentary system (stomach, intestine and esophagus), muscle, inner surface of the carapace and in the yolk-sac. In about 20 percent of the cases, more than one organ or tissue in the same turtle have nodules on them.

The nodules are solid, spherical objects that usually protrude on the surface of an afflicted organ. In a few cases, some nodules have been found embedded below the surface of an organ. The nodules are either white or creamy white and range in size from about 1 to 8 mm in diameter. Their solid core distinguishes these nodules from the hollow, often fluid-filled cysts in the advanced stages of SP, although *S. constrictum*, which is a presumptive causal organism of SP, is also one of the fungi isolated from the smaller, solid nodules.

Occurrence — Based on necropsy records, the annual occurrence of INM in Kemp's ridley hatchlings maintained in the Galveston Laboratory was 19, 8, 13, 138, 73 and 23 cases in year-classes 1978 to 1983, respectively.

Remedy — Clinical trials with prolonged, daily intraesophageal incubation of potassium iodide in hatchlings that exhibited outward signs of INM succeeded in reversing or reducing some of the abnormal signs in a few turtles. These turtles continued to live. In other similarly infected hatchlings, potassium iodide treatment was ineffective and the animals died. Nevertheless, potassium iodide treatment appears promising and more research should be done to explore its usefulness as an anti-INM agent in sea turtles.

Hypernecrotic Warts

Hypernecrotic warts (HW) are tumor-like or swollen lesions that occur on the skin, front flippers, carapace, plastron and head and that usually become heavily encrusted with continuous formation of necrotic tissues. The disease appears to be fatal, although sometimes an afflicted hatchling can live a relatively long time (e.g., two months or more) before it dies. Necropsies reveal that hypernecrotic warts are often accompanied by INM, but not necessarily vice versa. Whether death occurs as a direct result of hypernecrotic outgrowth, INM or both has not been determined.

Etiology — Two fungi, *Scolecobasidium constrictum* and *Paecilomyces* sp., have been isolated from hypernecrotic lesions. They are the presumptive pathogens. Although *S. constrictum* has been cultured from flipper and carapace lesions and *Paecilomyces* sp. from cranial and skin nodules, the specific affinity of individual fungi to specific tissue types has not been established.

Signs and Symptoms — Basically there are two forms of HW lesions: a conical form and a round (non-conical) form. Conical lesions appear to be limited to occurrences on soft skin parts such as the neck and the suprafemoral pouch. They are cone-shaped projections with pointed ends distal from the skin. The nodule is about 3 to 5 mm high and about 2 to 4 mm in diameter at the base. The color of the nodule is grey or dark-grey, similar to the color of the skin. Necrotic tissues that appear on older lesions continue to form as laminated layers on the nodule.

A rounded HW is basically represented by elevated, localized swollen tissues. The lesion is relatively round or flat at the top, rather than cone-shaped. Such lesions may be found on a variety of external body parts.

Formation of the lesions differs according to the site of infection. On the front flipper, a lesion arises from either the elbow or the base of the lateral claw. In the latter cases, the tissues adjacent to the claw swell to form a localized and irregularly oval-shaped lesion visible from both the upper side and underside of the flipper. The lesion, when measured from either face of the flipper, usually grows to about 5 x 10 mm. At a yet undetermined time, necrotic tissues begin to appear on the swollen lesion, and they continue to grow and accumulate in laminated layers following the contour of the lesion.

On the head, a round hypernecrotic lesion begins as one or more small elevated hard nodules or bumps in the crown area above the brain. The nodule(s) continue to grow and usually, although not always, the skin of a nodule breaks open to expose an accumulation of necrotic tissues. These necrotic tissues continue to multiply gradually to form a necrotic mass outside the skull. Surgical excavation shows that the crust can reach the soft tissues underneath the skull.

A round HW on the carapace perhaps begins as a small vesicle or bleb. Later, this bleb breaks open and necrotic tissues begin to develop into a small necrotic nodule of approximately 3 to 4 mm in diameter above the carapace surface. In the limited number of cases of carapacial HW observed in Kemp's ridley hatchlings, all the lesions were located on the front margin of the carapace close to the neck or shoulder of the animal. Round hypernecrotic lesions have also been encountered in the plastron and the neck.

Hatchlings or juveniles afflicted with HW usually remain active and eat well. They may become lethargic shortly before they die.

Occurrence — There were 3, 6, 32 and 10 cases of HW in the 1978, 1979, 1981 and 1982 year-classes of Kemp's ridleys, respectively.

Remedy — There is no established drug therapy for HW. Experimentally, long-term oral administration with a saturated solution of potassium iodide (SSPI) plus daily topical application of three percent hydrogen peroxide has produced positive responses from warty outgrowths on the flippers and carapace. After more than four weeks of treatment the lesions begin to show signs of regression and one or two weeks later black pigmentation returns. Diarrhea can be a side-effect of SSPI treatment. When diarrhea occurs, oral SSPI should be discontinued for a few days then resumed when diarrhea stops.

The cranial form of HW responded well to sunlight treatment. After about two months of ineffective treatments with formalin and malachite-green baths inside the laboratory, the turtles were taken outdoors daily for a 30 to 60 minute exposure to sunlight. After about five days, the cranial lesions began to heal. While the turtles were outdoors, the water temperature in the holding vessels was carefully monitored to ensure that the turtles were not overheated. If the water temperature exceeded 28 °C, the turtles were moved to a shady place or returned to the laboratory. Sunlight therapy has not been used for the other forms of HW.

In formalin bath treatment, the nodule of the conical form may shed but is regenerated after the treatment is terminated.

Malabsorption of Yolk Sac

Malabsorption of yolk sac (MYS) refers to a condition in which a hatchling, after a reasonable period of posthatching time, fails to absorb either the entire or the bulk of the embryonic yolk sac, presumably due to physiological dysfunction. The length of posthatching period within which a Kemp's ridley neonate is expected to complete the yolk-sac absorption process is unknown. According to F. Wood [Cayman Turtle Farm (1983), Ltd., Grand Canyon, BWI, personal communication, January 1985], the yolk sac in hatchling green sea turtles probably has been absorbed by the time the hatchlings start to eat, which is about five to six days after hatching. Observations at necropsy suggest that in many Kemp's ridley hatchlings, by the seventh day after hatching, a large portion of the yolk sac has been absorbed. Therefore, if a week-old or older Kemp's ridley is found to bear a relatively large yolk sac, such as one that fills a large area of the abdominal cavity, it might be considered a case of malabsorption. [Editors' note: initiation of feeding of pelletized diets to hatchling Kemp's ridleys is now postponed for one to two weeks after emergence.]

Etiology — The cause for malabsorption of yolk sac in Kemp's ridleys is unknown. It is probably an organic dysfunction. G. Harwell, Houston Zoological Gardens, Houston, Tex., suggested that certain avian neonates would die from malabsorption of yolk sac if fed too soon. Malabsorption of yolk sac has also been observed in young alligators with undetermined cause (E. Jacobson, University of Florida, Gainesville, personal communication, January 1985). It is uncertain if results with birds and alligators could be applicable to sea turtles, but some biologists believe that sea turtle hatchlings will not eat until the yolk sac is absorbed (F.E. Wood, Cayman Turtle Farm, Grand Cayman, BWI, personal communication, January 1985).

Signs and Symptoms — There are no external signs or symptoms in Kemp's ridley hatchlings suffering from MYS. The only evidence comes from week-old or older hatchlings that die in captivity, and which are shown through necropsy to retain relatively large yolk sacs 15 mm or larger in diameter. In many instances, the unabsorbed yolk sac almost completely fills that portion of the abdominal space not occupied by other viscera. The unabsorbed yolk sac

is soft and creamy-yellow in color and its surface is frequently lined with congested blood vessels. The liver is usually pale and mottled, indicating anemia.

Occurrence — The occurrence of MYS in the head started Kemp's ridley hatchlings was 1, 4, 25 and 4 cases in the 1978, 1980, 1981 and 1983 year-classes, respectively.

Remedy — There is no known treatment or prophylactic method against MYS. If the hypothesis of too-early-feeding is proven as a cause of this anomaly, then the obvious remedy is prevention by postponing initial feeding. The current standard practice at the Galveston Laboratory is no feeding of newly hatched Kemp's ridleys until they are one to two weeks old (Fontaine *et al.*, 1989).

Urolithiasis

Urolithiasis is the formation of calculi or crystals in the urinary system. The disease is fatal in Kemp's ridley hatchlings.

Etiology — The crystalline deposits found in the urinary systems of affected Kemp's ridley hatchlings have been identified by X-ray crystallography as struvite or ammonium magnesium-phosphate (E. Czerwinski, The University of Texas Medical Branch, Galveston, Tex., personal communication, 1980). The cause of such calculus formations has not been determined. It may be a consequence of an infectious disease, because urolithiasis is frequently accompanied by Internal Nodular Mycosis (INM) of the kidney, the lung, the liver, or a combination of these organs, as shown by necropsy. However, urolithiasis is not always present in all cases of such fungal infections. Delineation of the possible relationship between urolithiasis and INM will require more study.

Signs and Symptoms — Externally, a Kemp's ridley having severe urolithiasis displays a shriveled and stunted trunk, much like that in INM. The animal can be very weak and may refuse to eat shortly before death. At necropsy, crystalline calculi are present in the urinary bladder and ureters. The bladder may be so packed with crystals that it is distended up to five times or more its original size and becomes a solid block. The kidneys are probably also impregnated with crystals, because when they are sliced across with a scalpel blade a sandy texture in the tissues is sensed.

Occurrence — Urolithiasis was first observed in Kemp's ridleys of the 1982 year-class in which about 43 cases were recorded. In the 1983 year-class, only two cases were confirmed.

Remedy — There is no known treatment or therapy for urolithiasis.

Duodenal Ulceration

Ulceration of the duodenum can occur suddenly in otherwise healthy-looking hatchlings with few warning signs. Ulcers can cause perforation of the duodenum and result in death of the animals.

Etiology — The cause of duodenal ulcers is not known. There has been evidence that it could be food-related. For instance, in a serious epizootic caused by duodenal ulceration in 1979 in Kemp's ridley hatchlings, necropsies showed that along with an ulcerated duodenum, the stomach was packed with undigested, pelleted feed. Most of the hatchlings (about 350) had contracted signs of duodenal ulceration. Either the turtles were being overfed or something was wrong with the pelleted feed which caused gastrointestinal paralysis. Various modified feeding regimens were tested including feeding a total daily ration of no more than about seven percent of body weight, reduced frequency of feeding from four meals per day to either one or two meals per day, and either partial or complete substitution of the pelleted feed with thawed fish flesh which had been frozen. If a turtle became sick or stopped eating, the ration was greatly reduced or withheld. Within a month, new incidence of duodenal ulceration dropped to practically zero. There appeared to be no difference among the three tested feeding regimens with respect to effectiveness in suppression of the disease.

After successful implementation of the modified feeding regimens, the manufacturer of the pelleted feed informed us that analyses of feed samples showed contamination by a fungal toxin, aflatoxin (James McVey, National Oceanic and Atmospheric Administration, Sea Grant Program Office, Washington, D.C., personal communication, 1980). It is possible, though not conclusive, that aflatoxin may have been responsible for the ulceration.

Signs and Symptoms — Live Kemp's ridley hatchlings that are afflicted with duodenal ulceration usually are much bloated in the body trunk. In severe cases, the intestine partially protrudes outside the body through a suture between plastronic scutes.

In dead hatchlings, the stomach is usually distended by packed pelleted feed, as shown by necropsy. The duodenum bears an ulcer, usually in the portion that is proximal to the pyloric valve. In some cases (3 out of 13 necropsies in the 1979 year-class), duodenal ulcers have occurred concomitantly with ulcers in the large intestine. The intestine is frequently highly distended due to gas formation in the tract, probably the cause of bloating of the body.

Occurrence — A major outbreak of duodenal ulceration occurred in the 1979 year-class of Kemp's ridley in October 1979. Suddenly, about 350 hatchlings were discovered ill, and many died within one week. Both morbid and dead turtles showed signs and symptoms of duodenal ulceration. Morbidity and mortality continued to rise daily until

modified feeding regimens were adopted for the surviving hatchlings in the raceways. About 150 hatchlings died before the feeding changed. About 250 surviving hatchlings that were obviously ill with duodenal ulceration recovered after they were isolated from those remaining in the raceways and placed on either a fasting or a restrained diet.

Thirty-two cases of duodenal ulceration, frequently concomitant with ulceration, or tissue necrosis, or both in the stomach and other parts of the intestine, were also recorded at necropsy in Kemp's ridley hatchlings of the 1978 year-class. However, unlike the epizootic in the 1979 year-class during which cases of duodenal ulceration surged suddenly to more than 400 within a few weeks, the 32 cases of the 1978 year-class were spread out throughout the 11 months or so of rearing period. Bloating sometimes occurred in the 1978 year-class cases, but not nearly as frequently as in the 1979 year-class.

In addition, at necropsy, ulceration or tissue necrosis, or both have been observed at sites in the gastrointestinal tract without involving the duodenum. There were eight and two such cases in the 1979 and 1981 year-classes, respectively. Etiology of this pathological condition is unknown and its relation to duodenal ulceration is uncertain.

Remedy — Morbid turtles with bloating should be isolated and maintained in clean seawater without feeding. The key factor in successful treatment of the illness appears to be fasting, which probably provides an opportunity for the affected turtle to purge stale food from its gastrointestinal system.

Treatment of duodenal ulceration with Maalox (aluminum and magnesium hydroxides; Rorer, Inc., Fort Washington, Pa.) did not appear to be effective. Death continued among turtles under such treatment. Parenteral administration of kanamycin also did not help control the disease. Currently, prophylaxis for duodenal ulceration involves avoidance of overfeeding and proper storage of the pelleted feed to prevent growth of microorganisms which may release toxins into the food. In feed manufacture, precautionary steps should be taken to prevent potential contamination of the turtle feed with toxin-producing microorganisms.

Hemorrhagic Bacteriosis

Hemorrhagic bacteriosis is a bacterial infection in which there is significant bleeding from tissues and organs of the infected hatchling. The word hemorrhagic is used to distinguish this type of bacterial infection from those in which there is no primary, profuse bleeding involved, either externally or internally. Primary bleeding here refers to bleeding directly attributable to the infection.

Etiology — With use of sterile techniques, a bacterium, *Vibrio parahemolyticus*, was isolated from the blood of a few recently dead, 11-month-old Kemp's ridley juveniles of the 1982 year-class during an epizootic in May 1983. Septicemia was suspected. The outbreak could have been triggered by stresses caused by tagging with monel flipper tags. Secondary infection through tag wounds was also possible.

On another occasion, Gram-negative bacteria were observed in blood smears prepared from a five-month-old Kemp's ridley. The bacteria were not identified.

Signs and Symptoms — In hemorrhagic bacteriosis attributable to *V. parahemolyticus*, live Kemp's ridleys often vomit blood before death. Even after death, blood frequently flows from the mouth. At necropsy, the body cavity is typically filled with bloody fluid, and the lungs and other internal organs, such as the stomach, liver and kidneys, are either hemorrhagic or congestive and dark-purple in color. The liver often displays a mottling pattern in a purple cast. The lungs are usually not inflated. The deflated lungs probably serve to explain why freshly dead animals are frequently found submerged under water on the bottoms of their buckets.

Besides vomiting blood, live turtles exhibit few external signs or symptoms indicative of the infection. Occasionally, the plastron is more or less depressed (scaphoid) and bruise spots may be observed on it, but these signs have not been unequivocally correlated with morbidity. Similarly unconfirmed as a sign of this disease is a depressed appearance of the carapace on each side of the carapacial ridge. This condition is sometimes observed in postmortem examination.

It was almost impossible to identify infected turtles with hemorrhagic vibriosis through external gross examinations. Infected turtles usually looked healthy with perfectly good muscle tone. Attempts to recover bacteria from the blood of a few live Kemp's ridleys taken from the same groups in which death occurred in the epizootic in May 1983 were unsuccessful. The blood samples were sterile.

Occurrence — Hemorrhagic bacteriosis appears to be enzootic in sea turtle juveniles, as evidenced by occasional sporadic incidences. The disease assumes epizootic scale probably when the turtles are subjected to stress or when the environment becomes favorable for spreading of the disease.

On May 7, 1983, 13 Kemp's ridleys of the 1982 year-class suddenly died in the raceways. Another eight turtles died the next day. The turtles had recently been tagged with monel flipper tags. Postmortem examinations showed signs and symptoms of hemorrhagic bacteriosis, and a Gram-negative rod-shaped bacterium, later identified as *V. parahemolyticus*, was cultured from blood taken from freshly dead hatchlings. Chemotherapy with ampicillin (sodium) was applied, prior to positive identification of the bacterium, to more than 400 live turtles maintained in the same raceways in which deaths had occurred and presumed, therefore, to have been contaminated. By May 27, the

epizootic had tapered off following seven more deaths, for a total of 28 killed by the disease.

Remedy — Hemorrhagic bacteriosis, apparently an acute infection, is unpredictable. Since it displays limited outward signs until it is too late, attempts to identify infected live turtles for isolation and treatment have been unsuccessful. Such lack of diagnostic criteria to identify infected turtles rapidly is a serious handicap, and leads to emergencies. In an outbreak in which the turtles are fast-dying, as in the epizootic of May 1983, infected live turtles cannot be quickly identified for therapeutic treatments. An alternative is to medicate all survivors, whether actually infected or not. However, when antibiotic injection is the method to be used, and when there are hundreds of captive juveniles, it is difficult, labor-intensive and unrealistic if not impossible to complete treatment on all the turtles in a short time. Under this condition, those live turtles that are reared in the same sea water as those that die should be given the highest priority for medication.

The kind of drug or antibiotic to be used for treatment depends on the species of bacterium, the sensitivity of the bacterium to the drug and the tolerance of the juvenile turtles to the drug. In an emergency situation, those three factors cannot be determined immediately, so a drug must be selected on a best-guess approach. However, the situation should be closely monitored and drug susceptibility tests conducted on the bacterial isolates if possible. If the resulting information indicates resistance to the drug in use, then changes should be made. In the hemorrhagic vibriosis outbreak in May 1983, ampicillin (sodium) was used with success. The dosage was 0.5 mg per 25 g wet body weight per day, for the first day, and half of that amount for each of the subsequent six days. The total daily dosage was divided into two equal doses injected subcutaneously in the neck at about 8 a.m. and 3 p.m. The epizootic subsided by the end of the second week.

Mycobacterial Pneumonia

Mycobacterial pneumonia (MP) is an infection of the lungs by bacteria belonging to the genus *Mycobacterium*. It is a wasting disease and probably fatal.

Etiology — Acid-fast, rod-shaped bacteria were seen in impression smears and paraffin sections of lung lesions taken from a three-month-old loggerhead hatchling of the 1977 year-class and stored near 0°C for about one month following the turtle's death. Similar bacteria were cultured on synthetic media from the affected lung tissues of the same turtle. They were identified as *Mycobacterium marinum*.

Signs and Symptoms — Observations on the three-month-old morbid loggerhead hatchling showed stunted growth, emaciation of the body and weakened condition before it died. Postmortem examination revealed a 5-mm long, cylindrical, greyish-colored nodule in the left lung. *M. marinum* was isolated from tissues of the left lung.

Occurrence — Only one confirmed case of invasion of lungs by *M. marinum* was observed as described above. A second presumptive case was observed in an eight-month-old loggerhead of the same year-class. Postmortem examination of the latter turtle within 16 hours after death showed numerous greyish-white small nodules (1 mm or smaller in diameter) in the lungs. Unidentified acid-fast bacteria were cultured from the lung tissues. The animal intermittently exhibited a tilted-swimming behavior while still alive.

Mycobacterial infections in turtles involving other species of *Mycobacterium* have been reported in the literature. *M. chelonae* (*M. friedmannii*) has been isolated from extensive lesions in two turtles (Stanford and Beck, 1969). Brock *et al.* (1976) described six cases of tuberculosis attributable to *Mycobacterium avium* in captive green turtles. Reichenback-Klinke and Elkan (1965) discussed occurrence of tuberculosis caused by mycobacteria in reptiles and suggested that "the clinical picture is that of typical tuberculosis with pulmonary tubercles (in tortoises and turtles) and analogous lesions in skin, liver and spleen (in snakes and crocodiles)."

Remedy — No treatment has been established for MP in loggerhead turtles. Murphy (1975) reported that antibiotic treatment for MP has been ineffective, despite recommended use of streptomycin.

Swollen-eye

Swollen-eye (SE) is a chronic inflammation in the eye of sea turtle hatchlings. Usually only one eye is afflicted, and the disease can be fatal.

Etiology — The cause of SE can be mycotic infection. On one occasion, a fungus, tentatively identified as *Paecilomyces* sp., was cultured from necrotic tissues in the swollen eye of a Kemp's ridley. Whether all cases of SE involve mycotic invasion is not known.

Signs and Symptoms — The tissues surrounding the eye are swollen. In serious cases, the eyelids may be totally closed. Swimming and feeding activities of the hatchlings generally are not affected, except in the terminal stage when such activities become very weak.

In one case, at necropsy, necrotic tissues were present adjacent to an eye-gland behind the lower corner of the swollen eye of a Kemp's ridley. A fungus, *Paecilomyces* sp., was recovered from these tissues.

Occurrence — SE has occurred sporadically in captive populations of Kemp's ridley hatchlings. Incidence has been very low. There was one case each in the 1978, 1979, 1981 and 1982 year-classes.

A swollen eye disease has been reported in land turtles (Reichenbach-Klinke and Elkan, 1965). Both eyes were affected and the disease was attributed to vitamin deficiency. This disease probably is not the same as that in Kemp's ridleys, since in the latter, usually only one eye was affected. It seems reasonable that if the SE syndrome in Kemp's ridley were caused by vitamin deficiency, then both eyes instead of only one eye would have been affected most of the time, and that more turtles would have been affected since they were all fed with the same food.

Remedy — There is no known effective treatment despite various attempts at chemotherapy with a variety of general germicides and antibacterial, antifungal and antiviral drugs. Afflicted turtles may die after a relatively long period of bearing an inflamed eye. Spontaneous remission of swelling of the eye occurred in two five-month-old Kemp's ridleys after they had been held in isolation for about two months.

Intussusception

In a few Kemp's ridley hatchlings of the 1978 year-class, intussusception was observed at necropsy. One segment of the small intestine sloughed into an adjacent distal segment, resulting in a telescopic appearance in that part of the bowel.

Etiology — What caused intussusception and whether this anomaly contributed directly to the death of the hatchlings have not been determined.

Signs and Symptoms — In one hatchling, localized small patches with a charred appearance were present in several areas of an intussuscepted intestine. The significance of such blemishes and their possible relationship with intussusception is unknown.

In human pathology, intussusception in time may lead to infarction, as mesenteric blood supply becomes progressively compressed due to the entrapment of the mesentery in the fold (Robins and Angell, 1981).

Occurrence — Rare.

Remedy — Remedy for intussusception in human infants is through corrective surgery. This procedure is not practical for turtle hatchlings because intussusception in them is not detected until after death.

Curved-back (Lordosis)

In curved back, the carapace is curved with the rear end turning upward.

Etiology — The cause for curved-back is unknown. Death has not been reported in hatchlings with this anomaly.

Signs and Symptoms — The carapace is curved with the rear end turning upward.

Occurrences — Curved-back syndrome (lordosis) occurred in about 16 (0.1 percent) of the 1977 year-class of loggerhead hatchlings. The condition was observed in both neonates and hatchlings which had been reared for some time. On one occasion, a loggerhead began to develop a curved back when it was about 10 months old.

Remedy — Unknown.

Soft-shell

In soft-shell, the shell (especially that of the carapace) in a loggerhead or Kemp's ridley hatchlings may become relatively soft.

Etiology — Softening of the shell occasionally occurs after the turtle has recovered from certain kinds of illness such as emaciation syndrome. X-radiographs show that the ribs in the carapace of a soft-shelled turtle are underdeveloped. It is possible that soft-shell is the result of impaired calcium metabolism.

Signs and Symptoms — The shell of the turtle is soft to the touch. The turtle displays retarded growth. For example, a one-year-old turtle affected by soft-shell may be only about one-third the normal size of a turtle of that age.

Occurrence — Between 0.2 and 0.5 percent of each year-class of hatchlings contracted the soft-shell syndrome.

Remedy — Unknown.

Coelomic Edema

Etiology — A few Kemp's ridley hatchlings of the 1978 year-class that exhibited a tilted-swimming behavior were indicated by X-rays to contain fluid-like substances in one side of the pleural cavity. In collaboration with G. L. McLellan, The University of Texas Medical Branch, Galveston, we withdrew a clear fluid via the carapace, using thoranthetic techniques. As much as 7 ml of fluid were obtained from a single turtle. The fluid was sterile with no growth of bacteria when cultured on synthetic media. The cause of the syndrome is unknown.

Signs and Symptoms — Coelomic edema is a sign of illness rather than a disease in itself. Peritonitis and hepatitis have been observed at necropsy in hatchlings having the edemic condition.

Occurrence — Only three tilted-swimming ridleys of the 1978 year-class were examined for and found to have coelomic edema. The other tilted-swimmers were not examined.

Remedy — Unknown.

Lung Aplasia

Lung aplasia was observed in Kemp's ridley hatchlings of the 1978 year-class. Afflicted turtles had one of the lungs

missing. When alive, these turtles exhibited a frequent tilting of the body to one side during swimming.

Etiology — The cause of lung aplasia is not known. It is probably congenital.

Signs and Symptoms — Side-floating behavior was not specific to lung aplasia. Other diseases (e.g., SP which damages one of the lungs) can also manifest a similar swimming pattern.

Occurrence — The turtles with lung aplasia ranged from two to seven months in age. The incidence was about 0.15 percent as determined from dead turtles.

Remedy — Unknown.

Congenital Flipper Malformation

Two conditions of congenital malformation of the front flippers of Kemp's ridleys were observed in the 1978 and 1979 year-classes. They are hypoplasia and multibranch-flipper.

Etiology — Congenital flipper malformation is a congenital abnormality.

Signs and Symptoms — In hypoplasia, one or both front flippers are not fully developed at hatching. The animal cannot resurface after it has submerged in the water in the raceway. To prevent suffocation, the turtle needs to be maintained in shallow water.

In multibranch-flipper, the forearm of a front flipper branches to form one or two additional arms. The animals otherwise look normal and survive well.

Occurrence— Less than 0.3 percent of the 1978 and 1979 year-classes of Kemp's ridleys had congenital flipper malformation.

Remedy — None.

Cross-beak

Etiology — Cross-beak is a congenital abnormality.

Signs and Symptoms — In cross-beak, the front part of the lower beak of the mouth is bent sideways. Afflicted hatchlings usually cannot eat and have to be fed via intraesophageal intubation to survive.

Occurrence — Available records show that as many as 0.2 percent of cross-beak occur in a given year-class of Kemp's ridleys. In some year-classes, such as 1978, 1980 and 1981, there was no incidence of cross-beak.

Remedy — None.

Congenital Blindness

Two Kemp's ridleys of the 1978 year-class were hatched without eyesight. One died. The survivor was donated in 1980 to Texas A&M University for use in research. Since the blind survivor was able to locate turtle feed in the holding tank, eat and grow, it indicated that Kemp's ridleys are able to find food via the sense of smell alone.

Etiology — Congenital.

Occurrence — Two Kemp's ridleys of the 1978 year-class were hatched without eyesight.

Remedy — None.

Intestinal Prolapse

Etiology — Unknown.

Signs and Symptoms — The lower part of the intestine protruded from the anus, and one of two turtles died. The other received corrective surgery from a local veterinarian but died later.

Occurrence — Intestinal prolapse occurred in two Kemp's ridley hatchlings of the 1981 year-class.

Remedy — Surgery could be a potentially useful corrective measure, although past attempts were unsuccessful.

Urinary Bladder Prolapse

Etiology — The urinary bladder of a Kemp's ridley hatchling of the 1983 year-class protruded from the anus, and the turtle died. Necropsy showed that the urinary system of the animal was seriously enlarged due to mycotic infection. The pressure from that swollen system had forced the urinary bladder to protrude from the anus. While the turtle was still alive, attempts to restore the protruded bladder to its original place by pushing it back into the body with a small lubricated, cotton swab were not successful. The bladder re-evaginated after a short period of time.

Signs and Symptoms — Protrusion of the urinary bladder and lethargy are the typical signs.

Occurrence — Less than 0.1 percent of the 1983 year-class contracted urinary bladder prolapse.

Remedy — Unknown.

Discussion and Recommendations

Out of necessity, we adopted a two-phase strategy to address the diseases, injuries and associated mortality problems of head starting Kemp's ridley and loggerhead sea turtles at the Galveston Laboratory. Many animals that died rapidly, especially during the early years of the rearing operation, were afflicted with a great variety of diseases of little known nature, or they suffered severe traumatic injuries caused by intraspecific biting. If these diseases and

injuries had continued unabated, catastrophic losses of turtles would have continued to occur, seriously hampering progress of the head start project.

The first phase of our strategy was aimed at stopping, as quickly as possible, on-going mortality and reversing any observed conditions of disease or injury even though the reversals might have been accomplished through treatment only of the symptoms. Due to the paucity of information on diseases and injuries of sea turtle hatchlings at that time, we often had to employ therapeutic methods, skills and knowledge that originated from other scientific disciplines or that were developed as a result of our own observations or short-term experiments. Prescriptions were frequently, if not always, based on educated guesses. Our objective was to save as many of the head started turtles as possible, disregarding means, so that the head start project could progress through its rearing, tagging and release stages. This first phase of our strategy involved clinical practices without much baseline information.

Phase two of our strategy, which was engaged based upon the availability of resources and only after most of the needs of phase one has been met, was intended to involve elaborate and in-depth studies designed to understand better the fundamental nature and causes of sea turtle diseases and injuries. The objective was to provide essential baseline information for improvement and development of techniques and methodologies for diagnosis, therapy and prophylaxis. Such information could lead to more confidence and surety in clinical practices and to a higher degree of predictability of health conditions and prognosis. The net result would be more and healthier sea turtle yearlings for release.

Initially, the objective of phase one was rapidly achieved through discovery and subsequent implementation of the isolation-rearing and use of clean sea water for culturing the turtles from hatchling to yearling stages. While the latter requirement of good hygiene and sanitation was an obvious one, the need for isolation rearing was not. These methods and innovations, coupled with appropriate medications on an as-needed basis, quickly brought under control many significant and wide-spread health problems which plagued the earlier year-classes. Examples of controllable health problems were sudden hatchling death syndrome, papillary dermatitis, focal erosive dermatitis and aggressive biting.

Isolation-rearing and seawater cleanliness became cardinal requirements for maintaining health of captive-reared Kemp's ridleys, and laid the cornerstone for subsequent successes in head starting this species. General statements concerning importance of culture water to rearing aquatic turtles (Pope, 1950) were published before 1977, the year when the Galveston Laboratory initiated loggerhead rearing, but there were few specifics described concerning the desirable level of cleanliness or water quality. It was indeed a surprise to us to find that the quality of the recycled seawater suitable for the culture of marine shrimp (*Penaeus* spp.) in closed raceways was unsuitable for rearing loggerhead hatchlings of the 1977 year-class. Nearly 40 percent of the animals died from sudden hatchling death syndrome within about four weeks. Biodisc filter systems used to remove wastes from the recycled seawater had maintained a seawater quality sufficient for intensive shrimp culture in these raceways (Mock, Ross and Salsler, 1977).

Despite the considerable progress and success in phase one of our strategy, certain earlier maladies such as emaciation syndrome remain little understood, incurable and unpredictable. In addition, other forms of disease and injuries emerged, and some of them had the potential of killing large numbers of turtles. Examples were the various forms of scolecobasidiosis, internal nodular mycosis, malabsorption of yolk sac and urolithiasis. Some of the new maladies (e.g., duodenal ulceration in October 1979 and hemorrhagic bacteriosis in May 1982), suddenly erupted into serious epizootics, creating emergencies of potentially alarming proportions. Under such circumstances, we devoted our entire efforts to addressing these new problems, trying to find immediate answers to prevent potential catastrophes. Thus, we remained for the most part engaged in phase one and inevitably had to interrupt all phase two work, either on-going or scheduled, and to postpone or forego parts of the mission-oriented and fundamental pathological studies.

Despite unforeseen difficulties, we have been able to lay much groundwork for future pursuits of phase two work. Some of our accomplishments included identifying and categorizing various major forms of disorders afflicting captive sea turtles, isolating and identifying many presumptive pathogens (e.g., *Scolecobasidium constrictum* and *Paecilomyces* sp. in mycoses, and *Vibrio parahaemolyticus* in hemorrhagic bacteriosis), providing X-radiography as a potential tool for disease diagnosis in the laboratory, and obtaining evidence that potassium iodide is a potentially effective drug against sea turtle mycoses.

Pathology information is the backbone for research and development of techniques and knowledge essential for the practice of good medicine on any living system, whether it be man, other animals (including sea turtles) or plants. Correct approaches to diagnosis, therapy and prophylaxis and the development of techniques and methodologies for such require fundamental understanding of the nature and cause of disease, injury or death. Such basic information can be acquired through pathological observations and studies. Once the basic knowledge and skills are established, clinical and laboratory examinations and testing can be evolved from these basics and employed to obtain data in specific patients to effect correct medical judgements and decisions.

Because of practicality, it was inevitable that many minor illnesses in head started Kemp's ridleys would have to be handled medically on a "best-guess" basis without going through extensive and costly pathology research. However, for diseases which potentially can cause serious debilitation or death in a significant portion of a captive population and which may lead to catastrophic losses, reliance on best-guesses alone is risky. The spread of these diseases could result in loss of the entire captive population. Such a price could be high for a seriously endangered species such as Kemp's ridley, where each surviving female could provide a substantial contribution to the conservation and augmentation of the declining wild population. It is fortuitous that we were able to resolve all major disease or injury problems that arose during the head start project up to the 1983 year-class, but there were no guarantees that such luck would persist.

For pragmatic reasons, we recommend that more pathological studies on Kemp's ridleys be conducted. These studies, should focus on certain fatal diseases which presently are incurable and unpreventable. Examples are emaciation syndrome, scolecobasidiosis, internal nodular mycosis, yolk-sac mycosis, urolithiasis, hemorrhagic bacteriosis and malabsorption of yolk sac. Research should be directed toward determining causes of these illnesses (e.g., through pathogenicity experiments), sources of the problems, how the diseases are transmitted and spread, the nature and biology of the etiological agents, histopathology (both basic and clinical), hematology (both basic and clinical hematology for diagnosis), X-radiography and other similar techniques for specific diagnosis and monitoring of internal ailments, and *in vitro* responses of pathogens and host turtles to potential therapeutic agents and techniques.

A good understanding of the microflora and chemistry of the culture seawater is also of paramount importance. Baseline profiles of microflora and chemical constituents in the culture seawater should be determined and the potential role of these entities in disease development should be delineated. With such information on hand, environmentally-oriented prophylactic measures, which in the long run are perhaps the least expensive in disease control, can be developed.

Aggressive biting can be contained through isolation-rearing, but other methods might be developed to accomplish the same end. Among other things, isolation-rearing is costly in labor and materials and many turtles outgrow their buckets during the typical head start period of 9 to 11 months. One obvious alternative is to provide larger containers, but they would take up more space in a given raceway or tank and reduce the numbers of turtles reared per raceway or tank. Along with adding more raceways or tanks to accommodate greater numbers of turtles, other needs such as more space, seawater, electric power, labor and supplies will arise. In light of the costs of isolation rearing, studies are needed to determine the underlying mechanism of the aggressive behavior in hopes of developing procedures for alleviating it.

Finally, since the methods by which the sea turtles are maintained during head starting have great impact on their diseases and traumatic injuries, pathologists should be involved in the design of culture systems and modes of operation. What may appeal to the culturist as useful or practical may turn out to be unacceptable from a turtle health point of view, as was demonstrated in our early experiences with both loggerheads and Kemp's ridleys reared in groups. Effective compromises can be worked out through better understanding of pathology in relation to rearing methods. We encourage more studies to provide relevant information needed to optimize the culture and operation systems in relation to health maintenance and disease control while assuring their cost-effectiveness.

Despite the continuing need for improvement, the culture and health care procedures that have evolved during the rearing of 11 year-classes of Kemp's ridley sea turtles have been successful (Caillouet, 1984; Fontaine, Leong and Harris, 1984; Fontaine *et al.*, 1989). Each year additional knowledge is gained and methods are improved. In all, as of October 31, 1988, about 14,060 Kemp's ridleys of the 1978-1988 year-classes had been successfully reared out of 16,538 live hatchlings received, representing 85 percent survival during head starting (Fontaine *et al.*, 1989). Of these, 13,572 had been tagged and released into the Gulf of Mexico (this does not include the 1988 year-class). The remaining normal and healthy turtles were transferred to other locations for extended head starting and captive propagation, and a few that were abnormal, sick or injured were transferred to other laboratories for research or humanely disposed of.

In conclusion, sea turtle pathology research has barely started. Since 1978, there have been good successes in containing diseases and injuries in head started sea turtles at the Galveston Laboratory, but some difficult disease problems remain to be solved. Carefully planned and selected research can shed light on some of these difficult problems which if left unexplored have the potential of developing into uncontrollable epizootics. Research should include in-depth studies on basic and applied pathology of the host turtle, the etiological agents and the culturing environment as well as their inter-relationships. Such information will provide the prerequisite data for development of sound and practical health care methodologies, within the constraints of established guidelines and regulations controlling the possession, handling and care of endangered sea turtles.

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