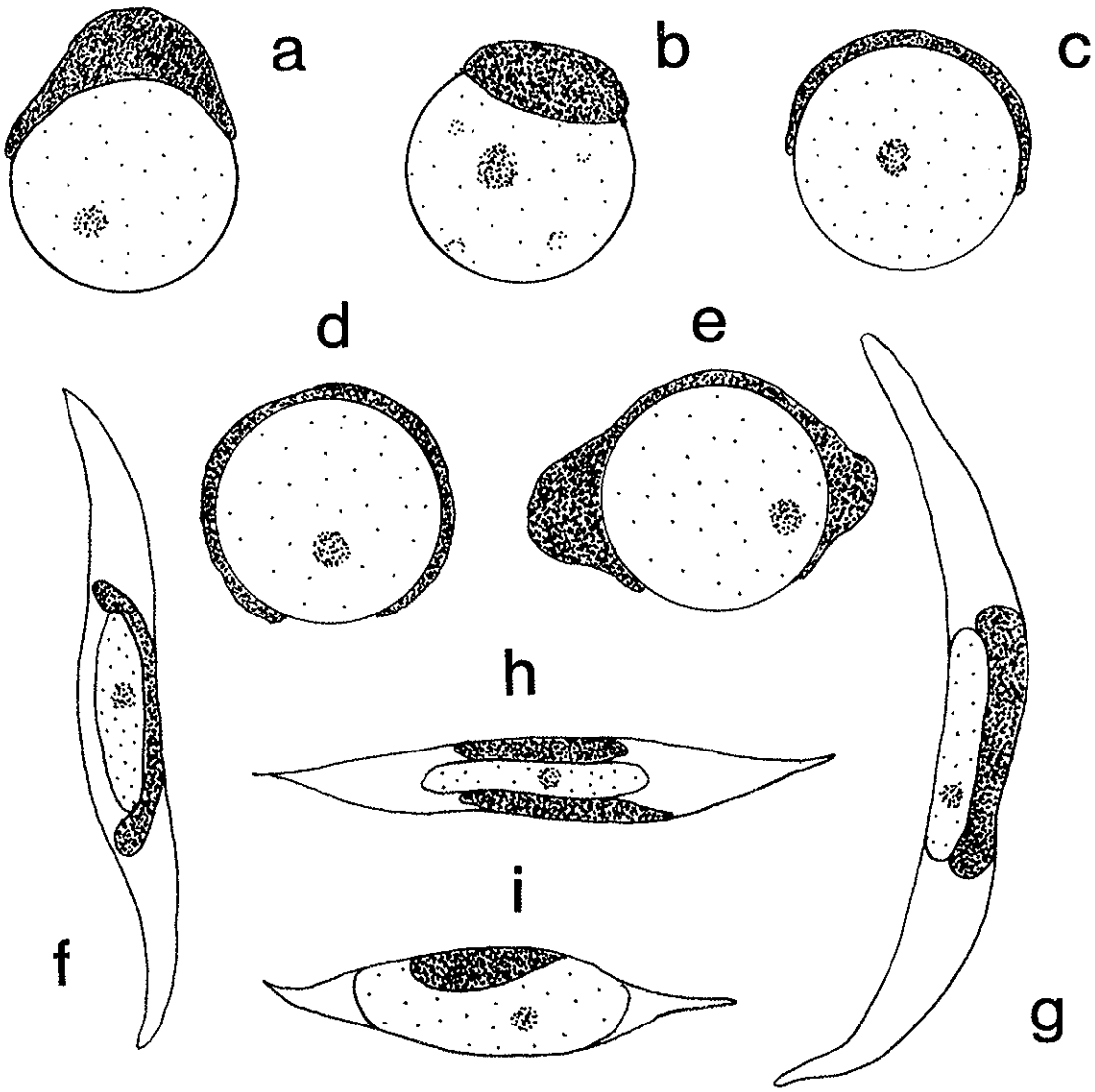
Winter 1989 Vol. 12, No. 4

WILDLIFE JOURNAL

Journal of the International Wildlife Rehabilitation Council



Avian Blood Parasites . . . Page 3

Guidelines for Authors for the *Wildlife Journal*

The *Wildlife Journal* is designed to provide useful information to wildlife rehabilitators and others involved in the care and treatment of native wild species with the purpose of returning them to the wild. All featured articles are subjected to a review process and our staff and reviewers work with authors to produce the most accurate information possible in our ever changing field.

The *Wildlife Journal* has various feature categories. We solicit submissions in the form of feature articles, rehabilitation notes, book reviews, news articles related to major events in the field, and selected case studies. Articles on veterinary medicine as it relates to wildlife rehabilitation are also welcome. We would like to expand the *Wildlife Journal* to include wildlife rehabilitation articles from countries outside the United States and welcome inquiries regarding such articles. We have two short features we use for basic information: "For The New Rehabber" and "Simple Things That Make a Difference". They are intended for basic information or new ideas that are easily demonstrated with a short text and photos. We pay authors a $25 honorarium for each article accepted for publication or provide a one year membership or renewal.

Submission Instructions

1. An original and two copies of the manuscript should be submitted to the Editor, IWRC, 1171 Kellogg St. in Suisun, California. The manuscript should include references, photo legends and footnotes, and should be typewritten and double-spaced if possible. Please put the authors name and address on at least the first page of the article so it can be readily identified.

2. The *Wildlife Journal* is prepared on a Macintosh and if papers are already written and on a disc the author should let us know that at submission what type of hardware and software systems were used.

3. Manuscripts are received with the understanding that they have not been submitted to other publications. It is the authors obligation to obtain permission or reprint rights if the submitted copy has been published elsewhere. All accepted manuscripts are subject to editing. Unused manuscripts will be returned.

4. A separate page should be submitted with a short synopsis of information on the background of the author. Included should be the name, address, rehabilitation organization, and phone number for the author. Where appropriate, please list any academic degrees, or special training that is relevant to the article (i.e., physical therapy—RPT, etc.)

5. Black and white photographs are preferred for use in the *Wildlife Journal*. When no other option is available, color slides and prints can be converted but this is costly and to be avoided when possible. Photo credits should be listed with the photographs.

6. Acknowledgments are limited to people who have contributed to the article in a major way.

7. References should be noted in the text with the last name of the author and the year published (i.e., Leighton, 1983) and listed at the end of the manuscript in alphabetical order in the following style:

LEIGHTON, F.A., Peakall, D.B., and Butler, R.G, 1983. "Heinz-Body Hemolytic Anemia from the Ingestion of Crude Oil: A Primary Toxic Effect in Marine Birds". *Science* 20:871-873.

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• The IWRC Journal is designed so you can use a 3-hole punch and store back issues in a binder for easy reference.

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Editorial. . .

1990—A Year to Examine Wildlife Rehabilitation Ethics

As many of you know, after 10 years of experience as a wildlife rehabilitator, I began a four year long epic to become a veterinarian in 1985. I then spent four years trying to get through a curriculum geared at dogs, horses, cats, sheep and cows. I was fortunate enough to be able to attend the University of California, Davis, which is well known for its innovations and which has some options available for those interested in "odd" creatures such as zoo and laboratory animals. Most veterinary schools offer little training or course work in *native* wildlife medicine with some notable exceptions at Tufts and Minnesota, which do have programs. While more schools are offering training in regards to pet bird medicine (27 of 31 schools recently surveyed), little curriculum development exists at most school in native wildlife medicine. Graduating veterinarians are not trained in wildlife rehabilitation unless they were rehabilitators before or during school and sought training on their own.

Although most practicing veterinarians are responsible enough to acknowledge their limitations of training and experience in this field, lately, we have run into some who assert that their degree in veterinary medicine somehow makes then qualified to do wildlife rehabilitation. The results have not always been in the best interest of the animals.

Take the case of Kent Walker and "Coconino," a tiny black bear cub Dr. Walker acquired with its umbilical cord still attached. While there are a lot of unanswered questions regarding how he came into possession of a bear that young, there are even more questions regarding his delayed notification of the California Department of Fish and Game (CDFG)—some thirty-one days following the acquisition. In California, possession of native species, and many introduced species (except the English Sparrow and Opossum) is illegal and generally known by citizenry to be illegal. People are not allowed to keep pet raccoons or foxes or bears!

When the bear became known to CDFG, they attempted to negotiate peacefully to secure the animal for transfer to a qualified wildlife rehabilitation program where the animal would have a chance for release. The talks failed under bizarre allegations that the animal would be abused by rehabilitators, including the use of electrical shocks. Through all the noise, it became clear that the people holding the bear were emotionally attached to it and wanted to keep it as a pet putting their own wants and needs above the welfare of the animal.

Local articles talk about it as a potential mascot for the area, which is known as Big Bear Lake. On television, we see pickets and protests with slogans like "Euthanize F & G, not Coconino." Well-meaning local folk, many of whom have been allowed to hug, hold, or kiss the bear, are squarely behind the mascot concept. The real sad thing about the whole spectacle is that the very people who are trying to hold onto the bear (past the point at which it could be rehabilitated) may end up condemning it to life in a cage (their goal) or the thing they fear the most, eventual euthanasia!

Another really frightening thing is the insistence of this veterinarian and his wife that "Coco" came in at "too young an age" to be rehabilitated and assert the bear would only have 10% chance of surviving in the wild. The Walkers claim to have 14 years of experience

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Avian Haematozoa

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Introduction

Haematozoans in birds have been recognized for many years, [13,32,33] and have been implicated in mass mortalities [24] and other aspects of economic importance [18,28]. Since the late 1800's over 450 species of haematozoans have been described from nearly 4,000 species of birds [8,10,26].

On admission to a rehabilitation facility the majority of animals will undergo a veterinary examination, including blood tests, to determine their state of health. During routine haematological tests parasitic protozoans (single-celled organisms, haematozoans) may be detected. These parasites, depending on the type, may be located in the plasma, the erythrocytes, or leucocytes, with four major groups being recognized, namely *Haemoproteus, Leucocytozoon, Plasmodium and Trypanosoma.*

Many blood smears will also contain the microscopic larvae (microfilariae) of filarial worms (roundworms, Nematoda). The identity of these larvae is almost impossible to determine in the absence of adult worms [10]. Other parasites that may be seen in the blood on rare occasions are members of the haematozoan genera *Atoxoplasma, Babesia, Haemogregarina, Hepatozoon, Lankesterella, Nuttallia and Toxoplasma.* The taxonomic status, and affinities, of many of these groups are poorly known [5].

While diagnosis of an infection can be made relatively easily, the specific identity of the parasites is much more difficult. Despite the fact that good keys and a series of coloured pictorial guides of the more common haematozoans of North America are available, [21,22,25] identification is best left to the expert. The International Reference Centre for Avian Haematozoa (IRCAH, Department of Biology, Memorial University, St. John's, Newfoundland, Canada, A1B 3X9) has the expertise to confirm specific identifications, and welcomes the opportunity to become involved with individuals and organizations that can supply large numbers of blood smears on a regular basis. The blood films can be made of blood drawn from the brachial artery/vein of small birds, [4] or femoral artery/vein in large birds. The frequently employed technique of toe-clipping to obtain blood is not advised. The toes and claws are often contaminated with fecal and other materials which can be transferred to the smear, causing problems in diagnosis.

Once an infection has been confirmed a decision must be made as to whether or not to treat the bird. In some instances the parasites may be of little concern, even when present in large numbers in both the population of birds and within individuals, while others may be pathogenic in low numbers. It is necessary to understand the life-cycle of these organisms if control is to be undertaken. The design of the rehabilitation facility should be such that transmission which involves a vector (carrier) is cut to a minimum. No adage is more true, especially in the case of parasites, than the one that states that "Prevention is better than the cure."

Genus *Haemoproteus* (Apicomplexa: Haemoproteidae)

This is the most commonly encountered genus of avian haematozoans, with over 140 species and varieties having been found in more than 1,700 bird species (110 families) [9]. Species identification is difficult and is based on the morphology of the gametocytes that locate in the red blood cells (erythrocytes: Figures 1-2). It may well be that many more of the species described to date will prove to be synonyms, and it has been found that many species tend to be host-family specific.

The life-cycle of these organisms involves a vector, either a louse-fly (Hippoboscidae) or a biting midge (*Culicoides*, Ceratopogonidae) [16,17]. The

parasite is ingested by a suitable vector, in which it undergoes a series of developmental changes before being transmitted to another host via a bite. In the bird (definitive) host the infective stage of the parasite (sporozoite) disappears from the blood for a period of 14-21 days (prepatent period). Multiplication of the parasite during this exoerythrocytic stage occurs in the lungs and reticuloendothelial system, with gametocytes (Figures 1-2) then appearing in the blood. The number of erythrocytes infected can range from 60-90 percent, with high intensity infections lasting up to nine months or more. The infection may last for more than one year and show a regression and recrudescence of intensity, with the highest intensity occurring in the breeding season or periods of stress. Immature birds appear to have a higher prevalence, and intensity, of infection than adult birds. [7]

Few signs, symptoms and pathological effects have been associated with *Haemoproteus* infections, even in the most intense infections. GFB noted a 96 percent infection of erythrocytes with *H. velans,* with an average of 3.5 parasites/cell, in a captive, immature, male yellow-bellied sapsucker, *Sphyrapicus varius.* The infection maintained at this level for more than eight months. The bird was one of eight kept in captivity for over three years and showed no discernible ill-effects from the parasitic infection. The only reported signs are of listlessness and anorexia in Muscovy ducks[27].

A decision as to whether or not to treat an infection is one that should be made by a veterinarian or other expert in the the field. Atebrin and Plasmochin have been shown to reduce the number of gametocytes of *H. columbae* circulating in the blood of pigeons, but were ineffective against tissue stages. Evidence gathered to date suggests that treatment is probably not necessary and that the drugs used to cure may cause more problems than the haemoproteids [12]. The best way to control this parasite, and the ones that will be discussed later, is by the exclusion of the vectors, in this case louse-flies and biting midges, from the bird holding quarters.

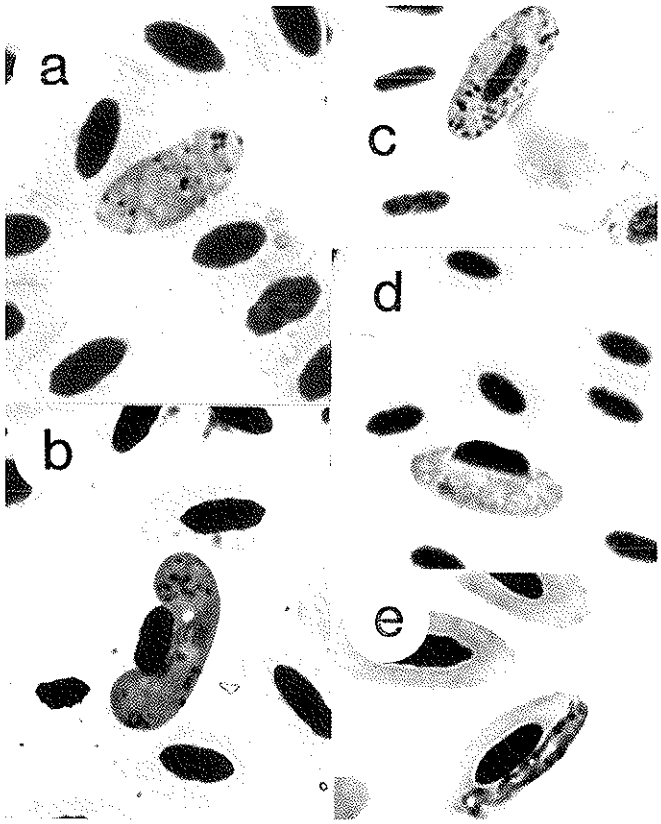


Figure 1. Gametocytes of five species of *Haemoproteus* in red blood cells. (a) *H. enucleator,* (b) *H. halcyonis,* (c) *H. velans,* (d) *H. sacharovi,* (e) *H. balearicae.*

Genus *Leucocytozoon* (Apicomplexa: Leucocytozoidae)

This is a geographically widely spread group, with some 96 species and varieties having been

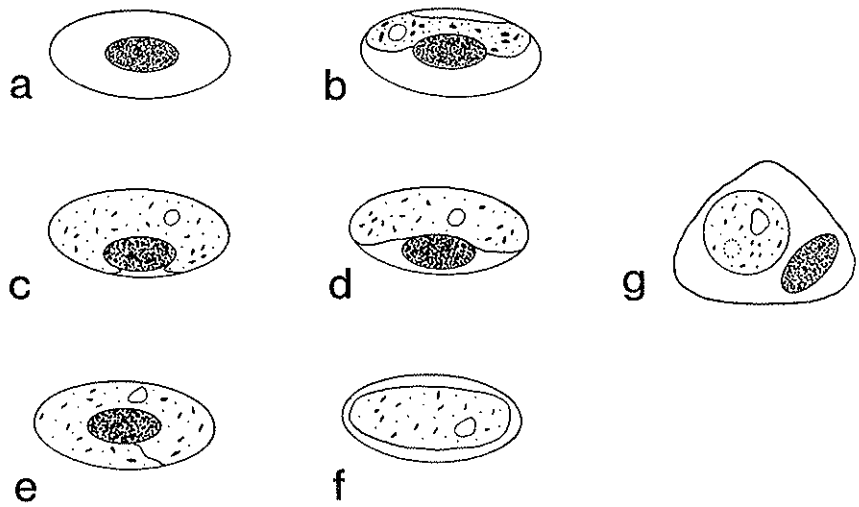


Figure 2. The gametocyte of different species of *Haemoproteus* assume different morphological shapes when in the erythrocyte. Five basic forms are recognized: (a) normal erythrocyte, (b) microhalteridial form, (c,d) halteridial form, (e) circumnuclear form, (f) rhabdosomal form and (g) discosomal form (after Bennett and Peirce, 1988).

found in over 1,000 species of birds (98 families) [10,19]. *Leucocytozoon* spp. appear to be host-family specific, with only one species usually being found in a host family. Once again, despite the availability of coloured pictorial guides, [21,25] specific identification is best left to the expert. The shape of gametocytes in the blood cells will vary according to the species (Figures 3-4), and in which organ the parasite develops, e.g., *L. simondi* produced during a hepatic cycle result in round gametocytes, those from the kidney, fusiform gametocytes [14,23].

The life-cycle of these haematozoans involves blackflies (Simuliidae), in which infective stages develop[19]. Blood containing gametocytes is ingested by the fly during feeding, and in 3-5 days oocytes give rise to sporozoites which migrate to the salivary glands and are then transmitted at the next feeding. In the bird, a 5-9 day prepatent period occurs with cycles initially occurring in the liver, followed by other cycles in a variety of organs. Gametocytes then appear in the peripheral blood with different shapes being indicative of different species and organ origin. Infections can last for more than one year, with highest prevalences and intensities of infection occurring during the breeding season or periods of stress.[7]

Pathological signs attributable to the dividing parasites (schizonts) are often visible in the liver in the form of white dots. Gross enlargement of both the spleen and liver are also seen (up to 10x normal size). Various changes in blood values may be seen including a markedly reduced packed cell volume (PCV) and haemoglobin content, with resultant anaemia. Clinical signs of *Leucocytozoon* infection include listlessness, anorexia, somnolence, watery eye discharge, and in severe infections convulsions followed by death. The onset of leucocytozoonosis is rapid, with many birds succumbing within 24-28 hours. Survivors of the initial crisis usually recover and eventually become "normal" in all respects.

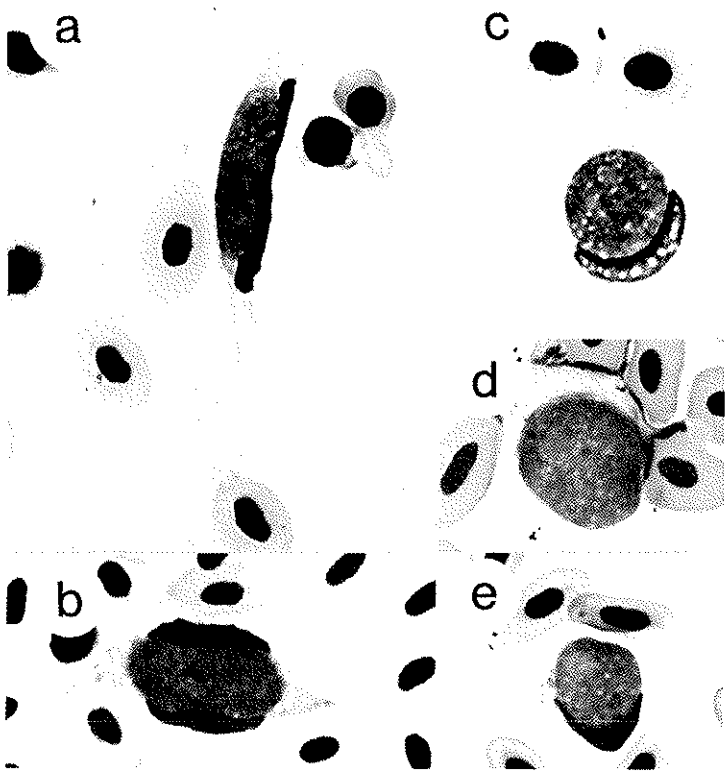


Figure 3. Gametocytes of five species of *Leucocytozoon*. (a) *L. simondi*, (b) *L. smithi*, (c) *L. simondi* (d) *L. tawaki*, (e) *L. fringillinarum*.

The genus possesses two species that have been implicated in mass mortality (up to 100 percent) of ducks and geese *(L. simondi)* and turkeys *(L. smithi)* [20]. Several other species of

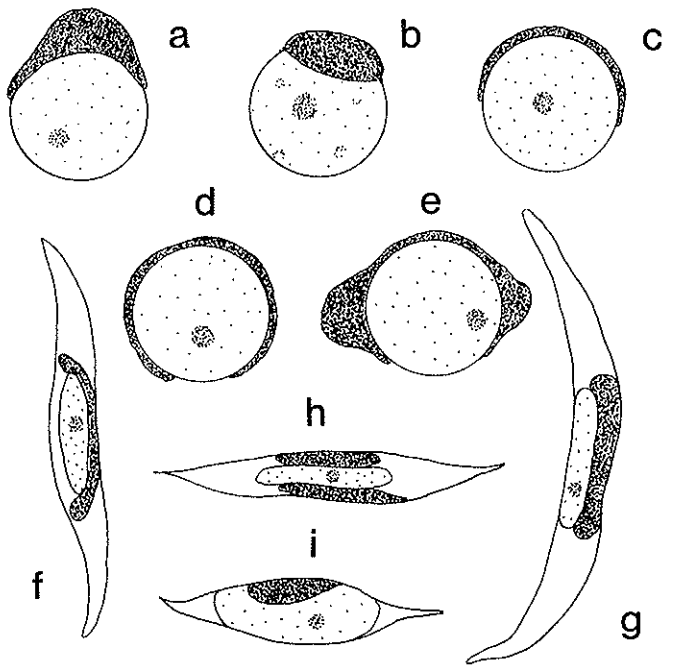


Figure 4. Typical morphological forms of *Leucocytozoon*. (a) *L. fringillinarum* of passerines, (b) *L. vandenbrandeni* of anhingid screamers, (c) *L. majoris* of parids, (d) *L. sakharoffi* of corvids, (e) *L. dubreuili* of thrushes, (f) *L. ziemanni* of owls, (g) *L. simondi* of ducks and geese, (h) *L. smithi* of turkeys, (i) *L. toddi* of hawks (after Bennett, 1987).

Leucocytozoon are reported to have killed a variety of bird species[19].

Again, the best method of control is by prevention of infection by excluding vectors from the rehabilitation facility. No drugs are available for treatment of the condition, with Atebrin, Paludrin and sulfamerazine proving ineffective[15].

Akiba is a closely related genus, containing just one species, *A. caulleryi.* This species occurs in domestic fowl in southeast Asia and Japan. It differs from *Leucocytozoon* in having a ceratopogonid midge as a vector, and gametocytes that may circulate freely (intercellular) in the blood. It causes severe mortality and economic loss in domestic poultry.

Genus *Plasmodium* (Apicomplexa: Plasmodidae)

Bird malarias are cosmopolitan in distribution, with about 35 species having been recorded from over 1,000 bird species (97 families)[10]. This genus occurs in highest prevalence where vector populations are large. It is difficult to comment on host-specificity in this group, with some species being highly host-specific (*P.durae* and *P.hermani* in turkeys; *P.kempi* in quail), while others are less specific and occur in a wide variety of bird species, e.g., *P. circumflexum, P.relictum* and *P.vaughani* [10]. Thin stained blood smears are the main diagnostic tool (Figure 5) with specific identification best being left to the expert, despite the availability of species keys[20,22]. In the majority of cage birds, and particularly passerines, the species most likely to be encountered are the three named above.

Vectors of these parasites are mosquitoes, principally of the genera *Aedes, Culex* and *Culiseta* and development in the vector is similar to that described for

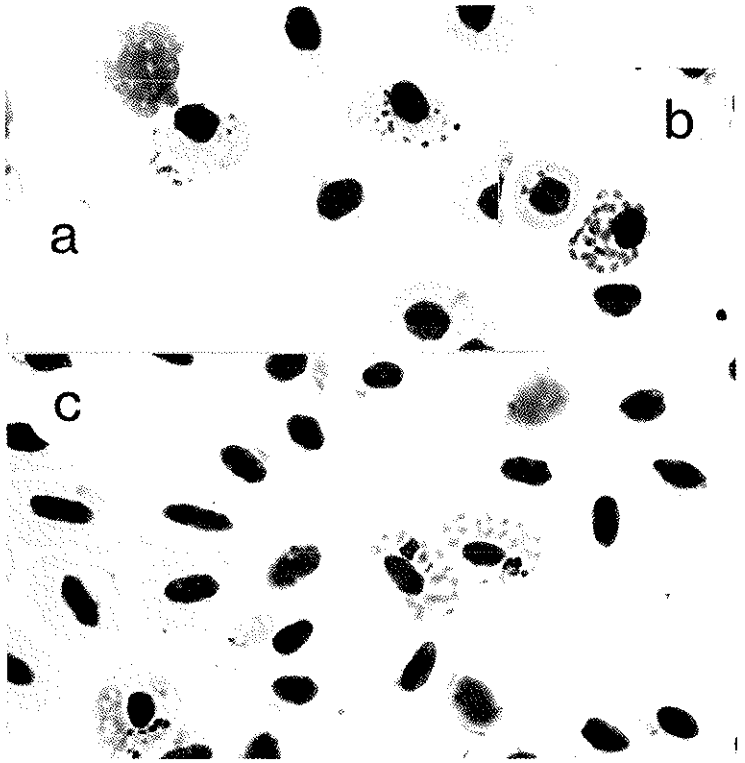


Figure 5. Two common species of *Plasmodium.* (a)*P. gallinaceum,* a species restricted to gallinaceous birds, showing developing gametocytes, (b) *P. gallinaceum* schizont that segments into 30-40 merozoites, (c) *P. circumflexum,* a parasite that occurs in many avian families, mature schizonts that divide to give 6-26 merozoites. Note the pigment granules. *P.gallinaceum* frequently causes mortality in poultry; *P.circumflexum* is known to cause mortality in a variety of birds.

Haemoproteus and *Leucocytozoon.* Transmission is via the bite of an infected vector.

A prepatent, hepatic stage occurs on injection of sporozoites into the bird. This is followed by an erythrocytic phase, and the invasion of other organs e.g., spleen, lungs and kidneys. The feature that sets this genus aside from other avian haematozoans is the fact that the parasites within the erythrocytes grow and divide (Figure 5). The number of merozoites formed during schizogony varies between species and is a species characteristic, (e.g., *P.juxtanucleare,* 3-5 merozoites, *P.gallinaceum,* 30-40 merozoites.

Pathological signs include

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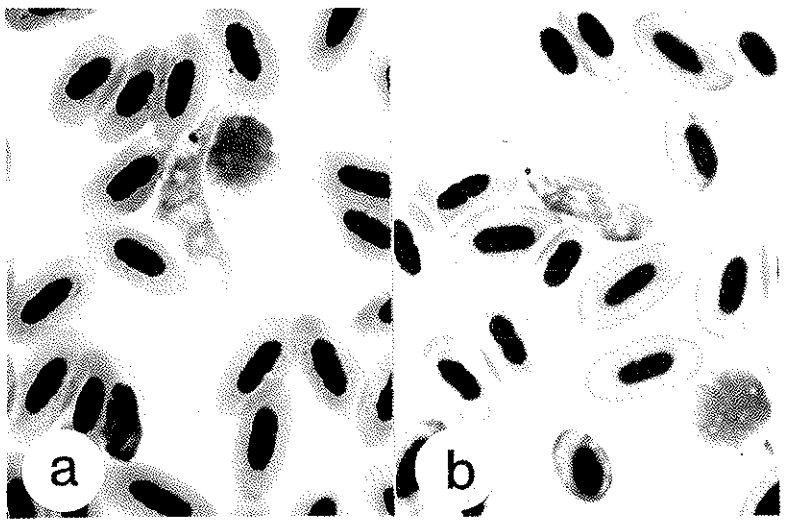


Figure 6. Two species of *Trypanosoma.* (a) *T. avium* (b) *T. bouffardi.*

Fluid Therapy: The Science Behind The Miracle

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Introduction

The rehabilitator all too often is presented a severely compromised patient for treatment with only one thing known about his charge's immediate past: the unfortunate creature was too sick, injured or debilitated to elude capture. Many times the identity of the primary or original problem of the patient is known or suspected (gunshot wound, fight wound, toxicity, infectious disease or other metabolic disease) but, unfortunately, by the time the animal reaches the rehabilitator's hands secondary problems of exposure, infection, emaciation and dehydration may have combined to overshadow the urgency for treatment of the primary problem.

The rehabilitator must first tabulate all of the recognized problems of the patient and assess them according to their immediacy in order to formulate a treatment plan. Each problem can then be assigned a place in the time-flow of treatment events according to its severity. The requirements for and benefits of fluids in the sick or debilitated wild patient frequently place fluid therapy at the top of the list of things to do on the rehabilitator's initial treatment plan.

Assessment

The initial physical exam will provide the necessary observations that will enable the rehabilitator to classify the patient as shocky, dehydrated, or as a candidate for supportive therapy. These observations guide the rehabilitator in the appropriate choice of fluid type and route of administration.

The wild patient in shock is one who is often presented quiet, often somnolent or moribund and usually has pale mucous membranes, slower than normal capillary refill time, shallow, oftentimes rapid respiration and weak, rapid pulse rate (except in the deeper stages when even heart rate slows). Frequently the shock may have been the result of trauma, blood loss, emaciation, dehydration, hypothermia, sepsis or the stress of handling. Regardless of the cause of shock, the primary physiologic abnormality is decreased cardiac output, venous dilation, pooling of body fluids and general circulatory collapse. While there is no single best treatment for shock and the most successful handling of the problem will often combine corticosteroids, antibiotics, and other supportive care, the need for fluid therapy is of paramount importance. Fluids, appropriately and expediently administered, can quickly help expand the circulating blood volume, increase cardiac output and restore blood flow to vital compromised organs including the kidney and liver. The intravenous route of administration is preferred in the treatment of shock due to the immediate response to therapy that the IV route affords and because IV administration avoids the pitfalls of slow or poor absorption of fluids by other routes.

Fluid therapy is also mandated in the primary treatment plan for any patient suffering from emaciation or dehydration. Here, too, the intravenous route is often considered the initial route of choice although subcutaneous and gavage fluid therapy of balanced (not hypertonic) solutions is often very satisfactory.

Debilitated wild patients, surgical patients, or those who for one reason or another cannot meet all of their fluid needs through self-feeding and watering, are often supported over long periods with supplemental **maintenance** fluid therapy. Maintenance fluids, according to the requirements of the patient and the composition of the fluid, are given either intravenously, subcutaneously, or by gavage. A rule-of-thumb calculation for supply of maintenance fluids to a patient is 50 ml/kg/day, although smaller animals, some water birds, anesthetized patients and those where natural diet provides high fluid volumes per body weight will require higher rates.

Fluid Therapy Basics

The sick, debilitated or shocky patient is often one whose electrolyte balance as well as fluid balance is compromised. Standard replacement, supplemental or maintenance fluids ideally will match closely the composition and concentration of dissolved solutes in the patient's own extracellular fluid. Besides providing necessary **volumes** of fluid and at the appropriately calculated **rate** and **route of administration** the goal of fluid therapy is to meet the patient's needs for re-establishing homeostasis by providing the required **composition** of fluid as well.

Normal extracellular fluid (defined as that fluid found **outside** the animal's individual cells), including interstitial fluid and extravascular fluid (fluid not within the veins and exterior to the circulatory system) is primarily composed of the basic elements of:

1) water
2) sodium
3) bicarbonate
4) chloride
5) (calcium)
6) (potassium)
7) (magnesium)

Normal extracellular fluid also has a specified **tonicity** and specified pH. Most mammals and birds (including creatures of the sea) have remarkably similar compositions of extracellular fluid. Minor disruption in total extracellular fluid volume, tonicity, pH or variations of individual electrolyte components in the composition of the circulatory blood volume can be disastrous to the patient. These disruptions in extracellular fluids often result in the wild patient coming into the hands of the rehabilitator in the first place; the rehabilitator would be wise to avoid any therapy that might further disrupt the composition of the extracellular fluid and further compromise the patient.

Replacement fluid therapy begins with a calculation of the patient's **fluid deficit**. This calculation is accomplished using observations gleaned from the physical exam and can be aided using simple, readily available laboratory tests.

Dehydration levels of less than 5% are difficult to detect. An animal 5-7% dehydrated, however, will exhibit a decrease in skin elasticity, early signs of "tenting" of skin, loss of brightness in the eyes and perhaps dry mucous membranes. These signs become more dramatic at dehydration levels of 10-12% when additional signs such as depression, wrinkling of the eyelids in some raptors and "muddy" color changes to the scales of the feet of other birds are also seen.

The severely dehydrated patient (12% or greater) will suffer from greatly compromised circulation, coolness to the extremities, severe depression and shock. A simple laboratory measurement of the bird's red blood cell packed cell volume (PCV) and plasma protein (PP) can aid in initial evaluation of dehydration and can serve as a tool for monitoring response to fluid therapy. Greater levels of dehydration will concentrate the animal's serum and increase both the PCV and PP. Care must be shown in cases of severe emaciation however, because anemia and decreased serum protein levels accompany the emaciated state and reduce the value of PCV and PP in the evaluation of dehydration.

Calculation of the approximate amount of fluids required to rehydrate the patient is simple once an estimate of percentage dehydration is made. (In practice debilitated or emaciated patients are often estimated 10% dehydrated.) An estimate of the animal's **normal** metric weight in health multiplied by the calculated percentage dehydration will yield the weight (metric volume) of fluids required to rehydrate the animal. Adding to this the normal daily maintenance requirement will enable the rehabilitator to calculate the amount of fluids necessary for rehydration. Generally the dehydrated animal is rehydrated in steps: with 50% of the calculated volume administered over the first 12 hours and the remaining volume given over the next 48 hours. (See calculations below).

Let's say an animal is presented (emaciated and 10% dehydrated) who weighs 1200 grams. In health you would expect an animal of this species and sex to weigh 1600 grams. The goal is to correct the dehydration within 48 hours **and** provide for daily maintenance requirements as well.

Presented weight - 1200 grams
Normal weight - 1600 grams
Percent dehydration - 10%
Maintenance requirement - 50 cc/kg/day

Fluid deficit 1600 g. X .1 = 160 cc's
Maintenance 50 cc's X 1.6 = 80 cc's
240 cc's

1st 24 hours: Give 80 cc's to replace one-half calculated deficit and 80 cc's to provide maintenance (total = 160 cc's).

2nd 24 hours:Provide 40 cc's to replace deficit and 80 cc's to provide maintenance (total = 120 cc's).

3rd 24 hours: Provide 40 cc's to replace deficit and 80 cc's to provide maintenance (total = 120 cc's).

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Rehabilitation Notes:

The Raccoon *(Procyon lotor)*

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Berkeley, CA 94710

Introduction

The North American raccoon is classified in the order Carnivora, an order that is comprised of mammals which primarily consume meat, although many of them also eat vegetable matter (fruit, berries, or nuts). All members of this order have five toes and large canine teeth.

The family *Procyonidae* includes raccoons, coatis, cacomistle, potto, and the kinkajou. Members of this family walk in a plantigrade (on their soles) or semi-plantagrade manner. Raccoons are fully plantigrade, and the soles of their feet are naked or partially hair-covered (Ewer, 1973).

The name raccoon, is derived from the Algonquin Indian language and translates "he scratches with his hands". Raccoons have a high degree of tactile sensitivity in those hands. In fact, researchers have found them to have discriminatory tactile abilities comparable to those of humans.

Typically, procyonids are forest dwellers and usually omnivorous. They are found throughout the continental United States in their preferred habitats. Those areas are often near ponds, lakes, streams, or marshes. Raccoons are not found above 2500 meters (8200 feet), in pure evergreen forests, or in arid regions lacking water.

Description

•ADULT: Size varies depending on age, sex, time of year, and latitude. In northern climates, the size is large, ranging in height from 305 to 356 mm (12-13") at the shoulder with weights from 9-14 Kg (20-30 pounds). Individual variation in weights are substantial and some individuals have weighed in at 59-62 pounds. In southern climates, such as Florida, the raccoon is much smaller, weighing as little as 1.8 to 2.7 Kg (4 to 6 pounds) (MacClintock, 1981).

•Characteristic markings include the black face mask which often connects just below eye level with the brown-black streak that extends from the nose to the forehead (MacClintock, 1981). They have forty teeth, including four canines which can be used as weapons. They have six mamae.

•Normal body temperature is 100-102° F. (Fowler, 1979).

•Activity patterns: nocturnal.

•Lifespan: While they can live to be ten or twelve years old, in the wild most die by the age of five (MacClintock, 1981).

•Age weaned in the wild: weaning starts at 6-9 weeks and is completed by 16 weeks.

Natural Diet

Omnivorous (i.e., meat and vegetable matter). Diet depends on locality and season. They eat more animal matter in the spring when insects, snails, frogs, worms, and young fish, birds, and mammals are available. In the summer, more carbohydrates are consumed (i.e., corn, acorns, fruits, berries) (Evans, 1985). One favorite food item is the meadow mouse (microtus).

Birth and Young

•Gestation: 54-65 days; usually 63. Birth periods vary by latitude and climate with northern births generally in April or May and southern births as early as February.

•Litter size: 1-7 with four being the mode.

•Breeding ages: First breeding cycle is at about 10 months of age. While males are able to breed in their first year, they usually are unable to do so due to

competition. Average age of first breeding in the male is 22 months.

•Birth size/weight: The average size is 100 mm (4" long) and weight is about 62 g.

•Appearance of newborns: The head seems large in comparison to the rest of the body. The ears are sealed closed. They may have pigmented tail rings or those may not appear until the age of one week. The color of the fur varies from black to shades of gray to tan. Litter mates may be colored differently.

Formula Feeding

•Raccoon milk is high in fat, protein, and calories. Based on available milk analysis for this species, KMR™ is the recommended formula. Esbilac can be used but is not as close in composition to raccoon milk and does not produce as good a growth curve.

•Upon initial intake, check skin turgor for signs of dehydration. If skin is slow to return to the normal position (greater than 1-2 seconds), rehydrate with an electrolyte solution orally prior to starting on formula. Commonly available electrolyte solutions include Pedialyte™ or similar veterinary solutions.

•Use a pet nurser or a nipple attached to a 3 cc syringe for tiny infants or human bottle for larger sized cubs. Use a small nipple aperture (preemie or water bottle size) to prevent aspiration problems. They are very adept at sucking on nipples.

•Feeding intervals depend on age and weight and the individual animal. Raccoons under 150 g may need five or six feedings per day. From 150-300 g., they will likely require four feedings a day; from 300-600 g., three feedings daily may be sufficient.

•Amounts fed will vary by individual animal. A comfortable rule of thumb is 50 cc/kg. Feed with the animal on its stomach, not flipped on its back.

•Stimulation and elimination: On a milk-based diet, feces should be formed and yellow in color. Gently rub the genital area after each feeding in the young raccoon to encourage elimination. The stools should be soft but well formed and will be yellow while they are on formula. Discontinue this practice when it is obvious that they no longer require it.

• Weaning: Introduce blended puppy kibble (a reputable brand, not generic) into the formula by four weeks of age. Offer moist puppy kibble ad libitum from 6 weeks of age onward. Wean as early as possible. Certainly, there should be no bottle feeding by the age of 9 weeks.

•Post-weaning: Offer dry kibble at all times. Two to three times daily, offer moist foods including natural foods such as chicks, rats, mice, crayfish, fish, insects, mollusks, clams, oysters, and fruits. Offer live foods only to pre-fasted animals. Usually the first kill is not efficient (they may bite the tail end of the animal). Do not start with a prey animal capable of mounting a major defense to the attack (i.e., large rat). Instead, start with a crayfish or fish and work up to mice and then rats. Following the first kill, they usually learn to go after the head or neck and quickly kill their prey provided they are hungry.

Special Infant Problems

•Changing diets abruptly or feeding certain solid foods too early may be enough stress to induce bacterial enteritis. Gram negative bacteria, such as Salmonella or E. coli, are often the culprits. Treatment starts with adjusting the diet first. Antibiotics are used only when they are shown effective against the bacteria that is causing the problem. Otherwise, they may further *exacerbate* the problem.

Housing

•Newborn to three weeks: These animals can be housed in a cardboard box or carrier with a heating pad set on low beneath the box, or in an incubator, or other small mammal housing with appropriate heat sources. They are not capable of self-thermoregulation until they are about three weeks old. If they develop dry skin from the heating pad, a source of moisture can be added. Some rehabilitators place a moistened washcloth in the cage, others put moist cotton in a small baby jar inside the cage. Toweling should not have raveling edges that can be stringy and get wrapped around the tiny toes of the raccoon.

•Four weeks to eight weeks: At this time the raccoons are able to ambulate and need more space within which to live. They are being weaned and will need to have food inside the caging necessitating a larger space requirement. Generally a stainless steel cage unit or sealed wooden caging will provide adequate housing. The caging must be cleanable and seams or sharp corners should be avoided as they are ideal spots for parasite eggs, bacterial, and debris to build up and cause problems. Suggested sizes are at least 2'W x 2'L x 1.5'H. One or two kits can be housed in a unit this size. Litters of three or four need more space (i.e., 3'W x 3'L x 3'H). During feeding time, it is very handy to place the kits in a large bathtub or veterinary treatment table tub until they get better at eating. Initially, more food is smeared in their environment than gets into their mouths. Kits and the tub are washed after feeding and they can be placed into a clean cage.

•Eight weeks to sixteen weeks: Once they are weaned, they should go into an outside caging unit providing the climate is not severe as can be the case with real late season babies. In the case of the latter, a heat source to provide supplemental

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Session II - Restraint of Wild Animals by Dave Jessup, DVM, William Clark, and Brian Hunter, California Department of Fish & Game. These materials cover basic considerations and responsibilities for restraint procedures, pharmacology, equipment, medical concerns and safety considerations for the rehabilitator handling wild animals. **Price $5.00**		
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Immobilization of Simple & Compound Fractures in Songbirds & Raptors by Victoria Welsh 1981 This booklet covers many aspects of dealing with fractures in birds. Areas covered in this paper include cleaning and disinfecting wounds, fractures of long bones, their treatment and post-treatment rehabilitation. **Price $3.50**		
Immobilization of Simple & Compound Fractures in Mammals by Victoria Welsh 1981 This booklet covers many areas of dealing with fractures in mammals. Rescue, transport, initial care, treatments for specific breaks, proper confinement, healing time, and rehabilitation. This information is complemented by illustrations for splinting and wrapping methods of fractures. **Price $3.50**		
Hand-Rearing of Non-Domestic Mammals by Debbie Marcum 1988 An informative paper in six parts coving subjects such as initial care, medical problems and their treatment, feeding and nutrition, housing, behavior and release. This paper includes a medical and dietary products list and a biological data sheet. Also added in the updated version are two papers on oral tube-feeding the Virginia Opossum and weaning the Virginia Opossum. **Price $2.00**		
Raccoon Rehabilitation by Jan White and Debbie Marcum 1979 This 20-page booklet covers the care, handling, and release of raccoons. It includes details on infant and adult diet, medical problems, vaccinations, caging, growth charts, and much more. **Price $3.50**		
A New Method for Exercising & Rehabilitating Raptors by Terry Schulz, Russel Tucker, and Judy Klingman, 1984. This paper introduces a new method of exercising raptors on a shoulder flight harness with a minimum amount of human contact. This method is ideal for the rehabilitation program that does not have room for a large flight aviary. Complete instruction to build a fixed flight line and harness are included. **Price $1.50**		
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Wildlife Restraint Handbook Edited by the Wildlife Investigations Staff, California Department of Fish & Game, 1986. This 160-page book is designed for the wildlife rehabilitator and field biologist. It has sections on mechanical capture of animals, transportation of captured animals, restraint equipment (ropes, gloves, hobbles, Ketch-All Pole ™, pole syringes, dart guns, etc.), pharmacology of restraint drugs, medical concerns in restraint, safety, and specific information on various species (including drug dosages) such as Black Bear, Mountain Lion, Bobcat, Coyote, Fox, Skunks, Badgers, Fishers, River Otters, Beavers, Raccoons, and Sea Otters. **Price $16.00**		
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heat may be required. The caging should have a sheltered area that allows the animals to hide from bad weather and feel secure. Provide them with a stimulating environment. They love to climb and cherish hammocks. They will swing on hanging tires and play with rocks in a small pool. The cage should be used only for this species due to the problems associated with *Baylisascaris procyonis*, the roundworm of the raccoon. Ideally, it should have flooring that would allow for thorough cleaning with a blow torch or very strong disinfectants. Cage sizes are typically 6' W x 8' L x 8' H for three or four raccoons.

Adult Housing

•Raccoons have been housed in fairly large groups (15-20 raccoons) due to their numbers and the amount of caging available. However, this practice may lead to outbreaks of distemper or raccoon parvo, especially if unvaccinated animals are present in these groups. In-coming animals should be quarantined for two weeks prior to being introduced into these groups. Once a group is established, if some are released or transferred, no new ones should enter the pen until all have been released. This is a concept called "all in and all out" and is a valuable one in disease prevention.

•Pens for this type of housing are typically 20' W x 40' L x 8' H. They are made of strong wire (i.e., chain link or heavy 1" x 2" welded wire) and have shelters provided within to allow the animals to get out of the elements. The cage wiring should be buried into the ground in such a way as to preclude digging out. Shelters should be constructed in such a way as to allow for cleaning. This involves having a hinged door for cleaning access. Double doors are essential due to the creativeness of the species involved. Flooring should be cleanable, but instead is often dirt or gravel. In the case of the latter, flooring is scraped up and changed annually. Care must be exercised in disposal of such flooring as the raccoon roundworm can be spread if such dirt or gravel is placed in contact with prey species of mammals or birds, therefore, it should be buried. Environmental stimulation can be provided with hammocks, hollow logs, and small pools and even educational children's toys.

•Raccoons like to establish a specific area for defecation called latrines. Given a litter box, they may elect to establish that as the area in which they all defecate. This makes gathering up and removing feces much simpler.

Disease Problems

•Among the Common disease problems encountered is canine distemper, a virus that is spread though the aerosols of nasal discharges. Common signs include neurological (convulsions), respiratory signs (pneumonia), and diarrhea. This disease is so often fatal in raccoons, that once diagnosed, they are usually euthanized. Raccoons should be vaccinated with an avianized vaccine such as Fromm-D™ every two to three weeks from their arrival until sixteen weeks of age.

•Raccoon parvoviral enteritis, an apparent mutant of the feline distemper virus, is also a major problem. Unfortunately, there is no vaccine for this disease at this time. The signs include depression, dehydration, and major diarrhea that may have mucous or blood in it. Raccoons show low white blood cell counts with this disease, as is seen in cats. It can be treated with massive amounts of supportive care (i.e., fluids, baby foods, antibiotics such as gentocin or amikacin and amoxicillin, acidophilus). It is very contagious and treatment should be tempered on the basis of the raccoons presently not affected and whether the sick ones can be isolated and adequately treated.

•Rabies is a zoonotic disease that has been seen in epidemic proportions on the east coast. The signs are very similar to canine distemper and include paralysis, paresis, head-tilt, and facial twiches. They may also be unafraid of humans. Raccoons with central nervous signs may have rabies or canine distemper, neither of which are treatable. Unless we are really sure we are treating a simple hit by car with neurological signs, euthanasia is the best option for raccoons with neurological signs. Raccoons can be vaccinated with **killed** rabies vaccines, such as Trimune™ which is of murine origin. They can be vaccinated at 16 weeks, or at release if earlier. ***Never*** use a modified live rabies vaccine in a wild animal.

•Raccoons carry *Baylisascaris procyonis*, their own roundworm. It can be controlled with regular worming with fenbendazole or pyrantel poamate. They should be treated at 6-8 weeks of age and the treatment should be repeated every 3-4 weeks until the raccoons are released.

•Other parasites include: tapeworms, flukes, and protozoa such as cryptosporidia. The latter is zoonotic and is not treatable. It will cause diarrhea that will stabilize over a period of time with good supportive care. Protozoa outbreaks are more common during times of stress (i.e., weaning, initial entry into captivity). Care should be taken to not expose humans (i.e., wash your hands!)

Release

•Raccoons are released at about 14 weeks of age, depending on the individual and the way in which they are released. Those that are hacked out (or released slowly) can be release earlier (10-12 weeks). Post-release feeding can be

provided for several weeks or until not longer utilized by the raccoon. Feeding stations should not be permanent!

•Raccoons that are released without back-up food (via relocation) should be really self-sufficient and good hunters and foragers. They are typically 14-16 weeks of age.

•They should be released in their proper habitat, near a source of water, late in the day or in the evening. Care should be taken not to place them in areas where prey species are of special concern (i.e., near an Audubon heron rookery or endangered species nesting area). The numbers of raccoons released in a given area should be tightly controlled so that their impact on the area is minimized.

•They should have had no *positive* interaction with dogs!

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For The New Rehabber: Nipple Size

Jan White, Editor
IWRC

Taking care of infant mammals can be a real challenge to new rehabilitators. One important aspect of feeding infant mammals is the size of the aperture (hole) in the feeding nipple. If the aperture is to small, the infant will suckle fretfully and receive little for its efforts. After a few minutes, they often will give up feeding leaving the rehabilitator frustrated and often resulting in failure of the animal to thrive. On the other hand, if the nipple aperture is to large, the animal will take in too much formula and often it will bubble out the nose. This situation has the potential to cause aspiration pneumonia should the animal inhale the formula. Additionally, the correct size for a tiny infant is too small for an older infant.

So what's a new rehabilitator to do? What we suggest is that a set of similar sized nipples be given graduated aperture sizes. This helps prevent the problem of taking a nipple with a small aperture and making it larger and then not being able to use it on the next small infant. Finding nipples with small holes can be accomplished by purchasing preemie or juice nipples.

Nipples should be kept very clean by washing with dish soap and hot water after each use. Old nipples that are frayed should be discarded. The size of the aperture on that nipple is too large for most infant mammals as well.

Many other types of nipples have been used for wildlife rehabilitation. Deer and moose are often raised using elongated goat nipples. Small mammals are often raised using "pet nurser" nipples. Some people have gotten creative and adapted other things to work. For example, a 3 cc syringe with a pet nurser pressed over the top of the syringe allows for the use of a nipple in a system where positive pressure can applied and milk can be dripped onto the tongue to tempt them to feed.

In summary, the size of the aperture of the nipple is very important in raising infant mammals. The opening must be adjusted (made larger) as the animal grows. New rehabilitators should monitor feedings for signs that the animal is not getting enough formula or is getting too much. Adjustments need to be made on an immediate basis.

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The **rate** of fluid administration varies according to the route chosen and condition of the patient. In the unique requirements for rapid expansion of blood volumes and increase in cardiac output in shock states **intravenous** fluid therapy at the **rate** of 100 cc/kg/hour or even greater is often necessary to stabilize the patient. In other states the total daily requirement is divided into 3-4 administrations (or supplied continuously) if the animal is connected to an intravenous drip.

Intravenous bolus therapy (administration of high volumes of fluids through venipuncture several times each day) is often of great value with critically ill avian patients. Once again the calculated daily volume is divided by the number of times in a day that the fluids can be administered. The size of the avian patient does determine the amount of fluids that can be given at any one time, however.

INTRAVENOUS BOLUS THERAPY*

Patient Weight	AMOUNT FLUIDS Per Bolus Dose	Needle Size
15-20 g	.5 cc	30 ga
30-40 g	1.0 cc	30 ga
80-100 g	2.5 cc	27 ga
150-250 g	8.0-10.0 cc	25 ga
800-1000 g	12.0-14.0 cc	25 ga
1200-1600 g	14.0-16.0 cc	25 ga

*IV fluids are best warmed to 96° -102° F

Within 48 hours after the initiation of intravenous bolus fluid therapy many avian patients are sufficiently stabilized to better tolerate other forms of supportive therapy such as gavage therapy. Birds of different species differ in their tolerances for gavage therapy and in the relative amounts of fluids that can be administered orally in a single treatment, but below are rough guidelines for volume administration of oral fluids to avian patients by weight of the patient.

GAVAGE THERAPY FLUID VOLUMES*

Weight of Bird	Volume Per Dose
20-30 g	.5-1.0 cc
30-40 g	1.5-2.5 cc
80-100 g	6-8 cc
300-500 g	15-20 cc
800-1500 g	30-40 cc

*Gavage fluids are best warmed to 96° -102° F

Nutritional supplements, vitamins, simple foodstuffs (early low protein, high carbohydrate and later, more complete foods in liquid form) may be added to fluids intended for maintenance gavage therapy. Although supplementation can begin with initial **replacement** therapy regimens, care must be taken not to administer hypertonic (potentially dehydrating) fluids by any route in **replacement** therapy. The rehabilitator must also realize that the emaciated or dehydrated patient is also one who often lacks the energy and abilities to absorb, digest, and utilize even basic nutrients through the gastrointestinal tract. Doing too much too soon for these patients can be disastrous to them. (White & Emanuelson, 1983)

There are a large number of fluid types readily available for administration to compromised wild patients. Although many fluids have been used for **maintenance** of the stable, rehydrated patient (Kool-Aid, Gatorade, 1 tsp. salt/quart warm water, Pedialyte, boiled Coca-Cola, etc.) none of those is designed for **replacement** therapy. Any fluid such as the fluids mentioned above that differs in any significant way from the patient's own extracellular fluid makeup may serve only to further disrupt an already compromised fluid and electrolyte balance and should not be used in **replacement** therapy.

Intravenous fluids frequently used in veterinary practice and of value in the treatment of compromised patients include:

LACTATED RINGER'S

NON-LACTATED RINGER'S

5% DEXTROSE

2.5% DEXTROSE IN .45%SALINE

.9% SALINE

The all-around fluid choice for the rehabilitator in the treatment of wild patients is Lactated Ringer's. Mammalian Lactated Ringer's solution, although it varies in small ways from the avian variety, is generally considered adequate for treating both avian and mammalian patients. Lactated Ringer's comes very close to duplicating the elements in the normal patient's own extracellular fluid, both qualitatively and quantitatively. Since many compromised patients are presented in mild to moderate states of metabolic acidosis, the addition of lactate to Ringer's solution provides a way of increasing the uptake and excretion of hydrogen ions by the liver, therefore serving to adjust the patient's acid pH toward a more normal range. Lactated Ringer's serves as a reasonable choice in gavage fluid

therapy replacement as well as by the intravenous and subcutaneous route.

There are a number of special circumstances that would indicate the use of other than standard replacement and maintenance parenteral fluid therapy. It is beyond the scope of this paper to discuss each in detail, but several special needs for fluid therapy do warrant discussion: acidosis, hypernatremia, hyperkalemia, and hypokalemia.

Acidosis is a state in which the animal's normal extracellular fluid pH is altered downward (to the acid side of the pH scale). This occurs for various reasons but chiefly through severe wasting (muscle and fat catabolism for energy leads to the production of acidic by-products), severe metabolic diseases (including liver, kidney disease and diabetes) as well as through respiratory compromise (retention of carbon dioxide which combines with water to produce an acid by-product). Although the lactate in Lactated Ringer's serves to correct acidosis through the removal of hydrogen ions from the extracellular fluid, other molecules such as acetate can help do the same. In practice, however, the most expedient way to correct acidosis in compromised patients is through the addition of sodium bicarbonate ($NaHCO_3$) to the fluids. Measurement of serum bicarbonate is a readily available test to evaluate the body's acid/base status. The measurement of serum bicarbonate unfortunately requires that a laboratory be near or that the equipment to measure bicarbonate be in the hands of the rehabilitator or technician to achieve accurate results (time is of the essence). If serum bicarbonate measurements are unavailable, acidotic patients are often empirically given 3-4 meQ/kg of $NaHCO_3$ slowly in the initial few hours of corrective fluid therapy. Injectable sodium bicarbonate (always administered with other fluids due to the hypertonic nature of the additive) is inexpensive and easily obtainable. A typical solution contains 1 mEq/cc.

Hypernatremia (high serum sodium) occurs in states of dehydration or in cases (iatrogenic?) where the animal has experienced a salt overload. High serum sodium can result in dehydration of essential intracellular tissues (i.e., brain tissue) and can lead to convulsions. Early fluid replacement to severely dehydrated patients should be **low sodium** such as half-strength physiologic saline with 2.5% dextrose. As hydration continues other sodium-containing fluids such as Lactated Ringer's can be instituted.

Hyperkalemia (high serum potassium) can occur in severe wasting and other metabolic disease states (particulatly renal disease). Hyperkalemia is aggravated further by the presence of acidosis. High serum potassium levels can result in cardiovascular insult (electrical conduction abnormalities of the heart) which can be minimized with the administration of calcium gluconate to the patient and reduced through the concurrent administration of glucose to the patient when hyperkalemia is suspected or doctmented, the clinician avoids the administration of potassium-containing fluids. Lactated Ringer's contains potassium, although in small enough quantities to enable its use in any but the most severely muscle wasted patient or in cases of kidney failure.

Hypokalemia (low serum potassium) occurs in prolonged vomiting and diarrheic states. Hyperkalemia is aggravated by alkalosis (alkaline serum pH). Low serum potassium levels can lead to depression, cardiovascular abnormalities and tetany. Laboratory measurement of serum potassium levels aid the clinician in potassium replacement therapy. In cases of extreme/persistent vomiting and diarrhea, potassium chloride is often supplemented in fluids at the rate of .1-.3 mEq/kg of patient body weight.

In summary, the rehabilitator, given the responsibility of caring for the debilitated, injured or emaciated patient has an awesome responsibility for the patient. Early treatment decisions that are made often determine the eventual chances for recovery of the patient. The decision to administer fluids to the patient and the choice of fluids and/or additives are best made from experience and consultation with a veterinarian. Proper fluid therapy with balanced electrolyte fluids such as Lactated Ringer's at the appropriate volume, interval and route of administration can do wonders in the nutritional care of the patient. We all should adhere to a basic tenet of rehabilitative wild animal care: Do nothing that could harm.

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Definitions

Homeostasis: a state of equilibrium with regards to chemical composition of fluids and tissues.

Iatrogenic: an unfavorable response to therapy caused by the therapy itself.

Tonicity: the relative chemical and particulate compositions of a fluid.

About the author:

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hepato-splenomegaly and anaemia, while clinical signs vary from listlessness, anorexia, cessation of normal behavior, to sudden death. If a bird survives the initial attack, which seems to be the most severe, a chronic, low-level parasitemia may persist. Recrudescence of the infection may occur, particularly when the host is stressed.

Treatment may be advisable, but the efficacy of available drugs may be questioned. Some species, e.g., *P. gallinaceum*, are susceptible to antimalarials such as chloroquine (5mg/kg body wt.) and proguanil (7.5mg/kg body wt.), while others are refractory [31]. Control of the vectors (mosquitoes) in the rehabilitation facility should reduce and/or eliminate this pathogen from bird stocks.

Genus *Trypanosoma* (Trypanosomatidae: Protomonada)

The haematozoans described thus far are intracellular organisms. In some blood smears small flagellates (*Trypanosoma* spp.) may be seen lying between the blood cells (intercellular organisms: Figure 6). More than 90 species have been described from birds (almost 800 species in 93 families)[10], with some showing little host-specificity. Parasites from a single bird may be transferred to, and cause an infection in, a variety of bird species from other families and orders[2]. These parasites usually occur in low numbers, making detection difficult. This problem may be alleviated somewhat by using concentration techniques, such as the haematocrit centrifuge technique [3,34]. Identification is even more difficult than in the previously described groups due to the fact that they are pleiomorphic, i.e., they occur in a variety of body shapes and sizes.

The life-cycle of these organisms has received less attention than those of the foregoing parasites, but involves a vector that may be a blackfly, mosquito, biting midge, tabanid fly, reduviid bug or louse fly, depending on species. Infective trypanosomes are ingested during a blood meal, reproduction (binary fission) occurs in the insect's stomach and then infective stages are deposited on the host in feces as the insect feeds and subsequently penetrate the skin, possibly through the bite wound. In the bird host the intercellular parasites are seen in the peripheral blood. To date, no tissue-oriented stage has been found.

No signs, symptoms or clinical effects have been attributed to those organisms, which is in contrast to those found in mammals[29]. As these parasites seem to cause little, if any, damage to the bird host, treatment is unnecessary.

A series of other parasites may be found in the blood, [1,11,29,30] on rare occasions. The biology of these organisms is poorly understood, and until such time as data are available there is little that can be done to alleviate any problems they may cause.

Conclusion

In conclusion, it is obvious that many haematozoans have the potential to cause great damage to individual birds and whole populations, yet few have been implicated in disease-related die-offs [6]. The rehabilitator must strive to prevent bird/vector interactions where possible, and design facilities that are not conducive to such interactions. Many rehabilitation centers tend to concentrate on raptors which play host to several pathogenic species of *Leucocytozoon* and *Plasmodium,* and the majority carry at least one species of *Haemoproteus.* Data gathered by the IRCAH over the past several years has shown that the prevalence of parasites in this host group obtained from zoos and rehabilitation centers is extremely high. The chances are good that any raptor admitted to a rehabilitation center will harbour at least one species of blood parasite. Therefore it is important that efforts be made to properly screen any cages in which these birds may be kept. Such mitigative measures, particularly in the southern parts of the continent where vector breeding occurs virtually the year around, are extremely important to prevent acquisition of new infections whilst in captivity in a stressed condition, and to prevent spread of any pre-existing conditions within the avian population of the center.

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State Representative Reminder

Anyone interested in becoming a state or province representative for the IWRC should send of letter of interest and resume to Jay Holcomb, IWRC President, 4437 Central Place, Suite B-4, Suisun, CA 94585.

State/province representatives will help connect rehabbers, local events and concerns to the IWRC leadership. Annual meetings for the reps are planned in conjunction with the IWRC conference.

Book Review

Care of the Wild Feathered and Furred

Maxine Guy and Mae Hickman
Michael Kesend Publishing Ltd.
1025 Fifth Ave.
New York, New York
143 pp. 1973; Revised 1978
$10.95

This was a difficult book to review. In summary, we found it to be, above all, a dangerous book, outdated, inaccurate, and inconsistent. Although there is some good information, it is buried among misleading and downright frightening advice, given that it is a mass-market book and not one directed at licensed rehabilitators. There is no doubt that at the time of its writing, this book represented a lot of information and experience collected over many years by its authors. However, wildlife rehabilitation has changed enormously since 1973 and it is doing any wild animal a serious disservice to base its care upon this book.

Although mentioning in various contexts that expert help might be needed, the authors do not hesitate to provide information that implies that the finder of an injured wild creature can deal with almost any problem alone. In the introduction, it is stated that periodic weighing of an animal is an important monitor but that it is too difficult (!) for the inexperienced, while Chapter 5 suggests that anyone should feel free to attempt suturing or amputation or even euthanasia. Although veterinary supervision may have been scarce during the "combined 60 years" of the author's experience, that is rarely the case today. When wildlife rehabilitation is receiving public recognition and wildlife medicine is becoming of more interest to veterinarians, it is difficult to imagine a situation where veterinary care and supervision in some degree would not be available for such cases.

The book's cover attests to a powerful following among humane and wildlife groups - yet none of these testimonials is from a wildlife rehabilitation group. Although the book talks of legalities and the fact that it is against the law to possess native wildlife, it is obviously addressed to the casual or accidental care-giver, not the licensed and trained rehabilitator, who now has access to a variety of literature, conferences, seminars, and workshops where updated information is readily available. There are probably 5,000 licensed wildlife rehabilitators nation-wide who are committed to providing quality care to untold thousands of wild creatures each year - these are the people pushing back the frontiers of wildlife rehabilitation. This book does them, and the animals in their care, no justice.

No attempt is made to explain the importance of raising young birds or mammals in conspecific groups - without which they will not learn appropriate behaviors and vocalizations so important to survival in the wild. Most examples given in the book indicate that the animals were raised singly. Added to the problem of improperly socialized animals surviving, we are in an increasingly urbanized society and what one person loves as a backyard visitor another person won't. This sets up death or mistreatment for an innocent animal at the hands of an irate neighbor, or the risk of being caged for life with potentially inappropriate care by someone who recognizes it as tame. The authors repeatedly use the example of releasing animals on their personal property, where they continue to feed them.

This book was written almost twenty years ago and times, as well as laws, have changed. If rehabilitation is to continue moving toward attaining a professional status with regulatory authorities, the public, and educational facilities, rehabilitators must show that animals released exhibit wild behavior. This does not occur when wild animals are raised singly and then released in human habitat.

The text is accurate in regards to certain things, such as bandaging. However, without experienced help and practice under professional guidance, this information is simply dangerous. Every rehabilitation center has received animals that have had amateur wing wraps or leg splints applied, with the end result being that the animal often must be euthanized: wings/legs with compound fractures beautifully wrapped, but when the dressing is removed, the wings/legs flails because the fracture was not reduced and no healing has taken place or worse yet, the limb has necrosed and falls off. Upon further examination it is found that the wrong tape

was used or it was wrapped too tight or the wound was not cleaned before the wrap was applied, etc., etc., etc.

Almost every rehabilitation group/ center we know has the following rules regarding injured, sick, or orphaned animals found: (1) put in a dark environment; (2) keep it warm; and (3) get it in to the center/licensed rehabilitator as quickly as possible. No food (it uses needed energy in what might be an emaciated animal and most people do not know enough about the species or food habits to offer appropriate food anyway); no liquid (too much potential for aspiration); no amateur fixits (too many mistakes); no keeping it (it's against the law anyway, the public doesn't know what kind of care to give, and they don't know to what they are exposing themselves in regard to zoonoses or personal injury, not to mention that the welfare of the animal dictates getting it to experienced care as soon as possible.) Most groups/centers do not give out care instructions over the phone either, for all the same reasons.

This book is a see-saw. The authors mention not giving pet names to wild animals in their care and yet many of the case histories concern animals who were given a name. Although there is some good advice on bandaging, wound management, diseases, or oil spills, to what audience is such advice offered? There is inaccurate information on rabies, bumblefoot, and diets. Experienced rehabilitators cringe every time milk - with or without bread - is mentioned. We all know, statistically, what toll those products have taken on wildlife which has wandered into inexperienced, though sometimes well-meaning, hands. Cow's milk or any inappropriate formula can kill. In a time when caloric and other nutritional requirements are known, the components of wildlife mammal milk have been broken down, when growth rates of numerous species have been charted, feeding bread or sugar in milk is simple cruelty. Honey and molasses should definitely not be used in hummingbird formulas. When the lay public is offered choices among substitute diets, they may choose the one which is least desirable or make substitutions which they assume are 'good enough'. That may be why rehabilitation centers/groups lost young raptors each year to rickets because people think hamburger or bacon is 'close enough' to their natural diet. There are a host of other problems in the book, of which only a few examples are feeding orphaned rabbits way too often, the lack of quantity guidelines for feeding orphaned mammals which could lead to either ruptured stomachs or stunted growth, and inaccurate anatomical names (ignoring the elbow in a bird's wing, they move it to the ankle!)

It is clear that this review goes against a formidable arena; however, the field of wildlife care has come of age since this book was written. *Care of the Wild Feathered and Furred* has no place in either the friendly neighborhood bookstore or on wildlife rehabilitation resource lists. Either the authors should considerably revise the book and limit its marketing to wildlife rehabilitators, or the book should be withdrawn from the market. This book made an enormous contribution towards care of wildlife when it first appeared. Today, there is probably no way to write the perfect wildlife rehabilitation book, but if it were written, it should be updated in less than twenty years.

This book is doing a considerable disservice in the hands of inexperienced people who, thinking they have a valid guidebook, try to care for an animal themselves. Clearly the authors acquired a lot of experience at a time when there was not much hard knowledge available. This has changed. Members of the public relying on *Care of the Wild Feathered and Furred* instead of finding professional care for a wild creature are doing far more harm than good.

Diana Granados
Louise Shimmel

Diana Granados is the facilities manager at Alexander Lindsay Museum, one of the oldest and largest wildlife rehabilitation operations in the country, caring for over 8,000 animals each year.

Louis Shimmel, is the Executive Director of Willamette Wildlife Rescue and Rehabilitation in Eugene, Oregon, which handles some 2,000 wild animals annually.

IWRC Moves!

The office of the International Wildlife Rehabilitation Council has moved to larger quarters. We have been growing steadily for several years now and we really needed the additional space. We have added another phone line so that move of you can reach us with fewer attempts. We are also moving the wildlife hotline into the office this year and will be handling those calls from that location as well. The old phone number (707) 429-IWRC will automatically connect to the new lines for the time being. Make a note of our new information. New new phone number is (707) 864-1761.

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rehabilitating wildlife and yet have no wildlife rehabilitation permit (unusual in California), and are not members of either national wildlife rehabilitation group (NWRA or IWRC). Having reviewed documents relating to their qualifications, what I see is a veterinarian with experience in a small zoo, not a trained wildlife rehabilitator. It is clear that someone with that background is far more likely to have an orientation towards keeping wildlife in captivity—not release. However, it is impossible to know why they are doing what they are—their motives are anyones guess.

Grasping at straws, the Walkers are invoking an 1896 law which, if upheld, would mean that any wild animal brought into captivity is considered "domestic" and can be held as captive. This would be a devastating blow to the hundreds of wildlife rehabilitators in California, who have fought for so long to establish qualified care centers or organizations to remove wildlife from the hands of the untrained public and return them to the wild. Besides the obvious issues regarding appropriate care from the animals' point of view, wildlife can be dangerous and can transmit disease to people—the more untrained the person, the higher the risk factor for injuries or illnesses thru bites or other methods of transmission. Yet, for reasons unfathomable to us in wildlife rehabilitation, the Walkers and their supporters are pushing us closer to this dangerous precedent. In whose interests are they really operating? If they "save" Coco, what happens to all the other animals that will be taken and held by people who are emotionally attached?

Recent oil spill coverage has gone a long way towards establishing wildlife rehabilitation in the minds of the public as a professional and recognized field. The hoopla surrounding this unfortunate animal represents a major step backwards into emotionalism and away from an animal's right to a free and wild life and the role of a qualified rehabilitator to preserve those rights through appropriate care. Bear rehabilitation is being done successfully in various parts of the United States. This information is published and available in numerous professional journals (drop me a note and I will send the citations). The Walkers' position seems to be why take any risks with an animal that is the state's official symbol? My response is — life is a risk. When a child welfare agency interviews prospective adoptive parents, the parents are asked only that they do their best to raise the child and prepare it for adulthood. The child could die six months later in a school bus accident — but a guarantee is not demanded from adoptive parents that all risks to life and limb be prevented. That would be tantamount to requiring them to incarcerate the child for life in a protective bubble. The same would be true for rehabilitators.

In the wake of the Exxon Valdez, wildlife rehabilitation is in the public eye and I have some bad news for rehabilitators. Public expectations for wildlife care have risen so high that we are all going to have trouble meeting them. Some people expect full-service functioning hospitals within two hours after a disaster. Operating under a public microscope is not fun and if we have to add to that guarantees of 100% survivability on release, we will all fail. Besides, life in captivity is also fraught with complications and does not in and of itself guarantee a longer life span than in the wildlife, especially in the hands of untrained caretakers. I would like to think that the general public, given an opportunity to make an informed choice, would vote for rehabilitation and release! And what about the quality of life issues? If this bear could make her own choice, what would she want to do? One thing is for sure, as this case drags on, her choices become more limited as she is now over two months old.

Jay Holcomb, IWRC President, myself, and an experienced team of oiled-bird wildlife rehabilitators from the International Bird Rescue and Research Center have been on the road a lot in the last year responding to spills in Alaska, San Francisco Bay, and Huntington Beach. We have encountered some severe problems with wildlife ethics, especially as it relates to veterinary involvement. It is time we start working on these — or someone besides qualified wildlife rehabilitators will be calling the shots in our centers and disaster response arenas. More on this later. Meanwhile, lets all hope the judge does not uphold a law that would undo the existing wildlife protection regulations. As for Coco, with recent court stays and delays, I fear that she will spend so long as a pet, there is little hope she could be rehabilitated. As for Dr. Walker, I think he should be made to live with the bear for the next 30 years and not be able to shuttle it off into someone else's care. Only time will tell.

-Jan White-

In Memorium: Doug Morris

Jan White, Editor
IWRC

Doug Morris, one of IWRC's original founders, was killed on January 28, 1990 while working as a special agent for the United States Fish & Wildlife Service near Spring, Texas where he lived. He was a very special person with a sense of vision and purpose and we will all miss him.

Doug helped create the Wildlife Rehabilitation Council in 1974 when he and other bay area rehabilitators recognized the need for networking amongst those in the field. Working with Gary Bogue (ALJM, Walnut Creek), Alice Berkner (IBBRC, Berkeley) and Sheila Shannon, DVM (PHS, San Mateo), James Harris, DVM (Montclair Vet Hospital, Oakland), Paul Maxwell (Corte Madera), Jim Barbieri (ALJM, Walnut Creek), and Dave Smith (IBRRC, Berkeley) he helped create an organization that would go on to serve wildlife rehabilitators across the country.

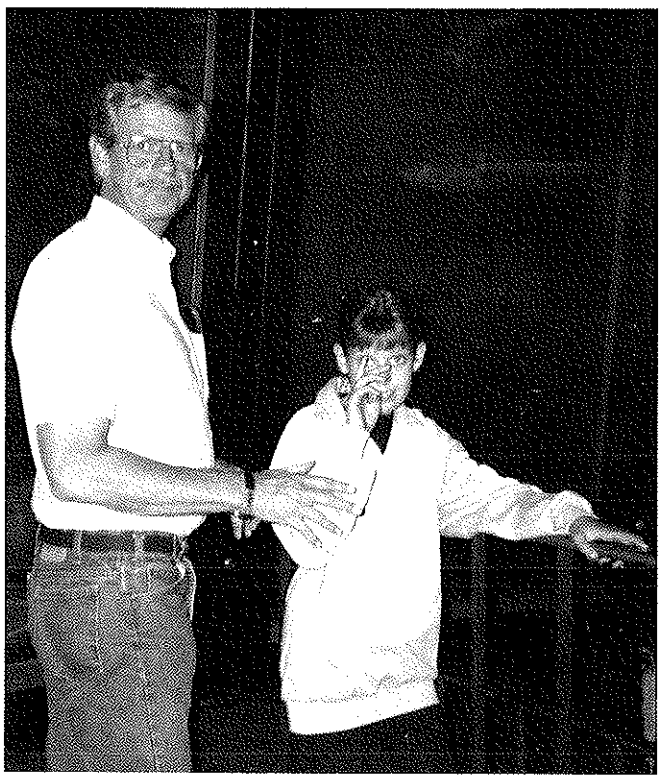
Doug had a BS in wildlife management and used that training to establish a large wildlife care facility at Peninsula Humane Society (PHS) in San Mateo, California. He was an early pioneer in the area of finding suitable release sites in urban areas for rehabilitated wildlife. Always willing to share what he knew, he acted as a resource person to many of us as we entered this field in the mid-1970's. I personally remember spending whole days in his facility seeing how it worked and was funded as I struggled to develop a similar program in an nearby county. His imprint on PHS still remains today even though it has continued to grow and evolve through the years.

Doug left PHS in 1977 to join the United States Fish & Wildlife Service. He worked assignments with the Special Task Force on the Klamath River in California as well as other assignments in Oklahoma City and Houston, Texas. Known as a dedicated conservationist, he received numerous letters of commendation for his performance on key projects such as Operation Eagle in 1983 and Operation Trophy Kill in 1984.

Even though he continued his career in law enforcement, he never lost his affinity for wildlife rehabilitation. Doug was an advisor to a wildlife care organization called Eden's Reserve (P.O. Box 2761) in Spring, Texas at the time of his death. Serving in that capacity, he was instrumental in the initial conceptual stages of developing the wildlife center there. The center has started a memorial fund in his name to construct a large raptor aviary. When completed, they plan to place a memorial plaque on the aviary to commemorate the passing of such a good friend to wildlife rehabilitation.

Doug is survived by his wife, Amy, a son, Sean, and Kate, pictured with him in the photo below. He holds a very special place in the history and development of wildlife rehabilitation and he will be missed by all that knew and worked with him.

-Jan White-



Special Event!! Two-day Oil Symposium on the Effects of Oil on Wildlife

October 17-18, 1990 Held in Conjunction with the IWRC Annual Conference

Sponsored by Int.Bird Rescue Research Center, Tri-State Bird Rescue, & Int. Wildlife Research, Inc.

Tenative Program Topics

Research Topics

Review Presentation on the Effects of Oil
Toxic Effects of Ingested Oil
Detergent Efficacy
Water Repellency & Feather Structure
Nutrition in Oiled Birds/Mammals
Oils & Avian Reproduction

General Topics

Oil Spills & Wildlife– Environmental Ethics
Ethics & Profession Conduct
Case Reports from Countries outside the USA
Basics of Contingency Planning for WL Rehabilitators
Damage Assessment
Ethics of Research Design
Mortality Determinations

Alaska Topics

USFWS Outreach Efforts (Monitoring the Rescue)
Overview of the Tanker Accident
Otter Rehabilitation Overview
Otter Facilities in Alaska
Otter Triage, Cleaning, & Rehabilitation
Rearing Otter Pups
Bird Rescue Overview & Operations
Sea Bird Anemias
Blood Transfusions & Treatment
Eagle Capture Program
Eagle Washing & Short-term Rehabilitation

Rehabilitation

Presidente Rivera Spill
Zoonotic Concerns in Oiled Wildlife Rehabilitation
Water Hardness and its effects on Oiled Wildlife
Pathology as a Treatment Guide & Research Tool

"BEYOND A BIRD IN THE HAND"

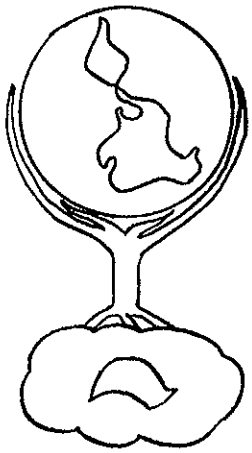
2-Day Oil Symposium Added

The Partnership of Wildlife Rehabilitation and Conservation Education

International Wildlife Rehabilitation Council

Conference, 1990

Washington, D.C. October 19-21st



Papers are in the works on:
*Raccoon, Deer, Javelina, Fox, Bobcat, Otter
*Goldfinch, Roadrunner, and Dove
*Avian Hematology and Clinical Pathology
*1989 Alaskan Oil Spill: Short & Long-term Effects
*Reptiles and Their Rehabilitation
*The Expanding Need for New Sources of Funds
*The Ever-Present Problems of Day to Day Operation

The Conference Committee would like to expand the focus on a subject only peripherally covered before — Conservation Education.

§ When people leave an animal with you, do they leave your facility committed to preserving the habitat that supports animal life?

§ How, within your limited time frame, do you impress upon people that, more important than the grebe or warbler they have brought you is the preservation of the wetlands or tropical forest that species **must** have to survive?

§ If you have the answer to these questions, we want to hear from you! You may contact the IWRC Conference Chair, Jonolyn McCusker at 11631 E. Calle Javelina, Tucson, AZ 85748 (602) 296-5920 or call our national office (707) 428-4972 for further information. If you don't have the answers, but have suggestions or questions, we still want to hear from you!