Felis domesticus:

A Manual of Feline Health 1982–1983

Cornell Feline Health Center
Cornell University College of Veterinary Medicine Ithaca, New York 14853

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A Publication of

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SECTION I

Proceedings of

The Second Annual Feline Health Seminar

at the

Cornell Feline Health Center

July 1982

FELINE BEHAVIOR AND BEHAVIOR PROBLEMS

Thomas R. Wolski, D.V.M.

The cat displays a complex social organization and behavioral repertoire which approaches that of the far more gregarious dog. Cats are not asocial as some have claimed, and as repeated cat fights and other examples of incompatibility testify, but certainly neither are they well adapted for group living. The following material sketches the basics of feline social behavior and ethology as an introduction for the understanding and handling of some of the more common behavior problems seen in cats.

I. Domestication of the Cat.

A. Primary or exclusive ancestor is North African wildcat, Felis lybica (also known as Felis sylvestris-lybica and Felis catus lybica).

B. Process started 6,000 - 10,000 years ago by the cat itself; primary selection criteria was for increased intraspecific (cat-cat) and interspecific (cat-human) tolerance.

1. Little selective breeding in past and even to present.

2. Cat valued almost exclusively as a rodent killer until last several hundred years.

3. Almost no dependence on man for hand-outs until recently.

4. Cat best adapted, therefore, to rural setting and low density situation.

C. Cat found today on every continent except Antarctica, and on most large islands.

1. Traveled extensively with man on ships; geographic barriers insignificant to spread.

2. Sometimes treated as a valuable trading item.

II. Social Behavior and Organization.

A. The farm cat.

1. Relatively closed society; little immigration; replacement of population by births.

2. Peak queening season is March to June. Later births occur if early litters lost or if litter weaned early. Litters

often hidden for 4-6 weeks. Dead food items provided by queen starting at 5-6 weeks; kittens start catching small prey items such as insects at same time. Weaned at 12 weeks or later; highly variable, apparently dependent somewhat on amount of supplemental food available. Queen may provide food intermittently until kittens are 5-6 months.

3. Queen may take 8-16 week old kits on foraging excursions about range. May lose kittens on these trips; possibly deliberate seeding out of kittens in some cases.

4. After weaning queen may move away or stay and share parts of range with kits the following season. Young females likely to emigrate if mother remains at home farm.

5. Young males will usually emigrate at 12-24 months whether mother is present or not.

6. Females will give birth to first litter at 12-24 months of age.

7. Toms have very large ranges often covering several farms. A large portion of this range is covered daily. Urine marking while moving very frequent; up to once every 5 meters. Aggression toward resident females and kittens highly infrequent. Infanticide not documented.

8. Cats have fairly exclusive use of a range once a range has been established. Repeated use, and presumably repeated marking, is necessary to maintain this exclusiveness.

9. Area about farm buildings is communally shared, although some division of space evident here also.

B. The urban and suburban cat.

1. Little has been systematically studied of either.

2. One researcher describes "community" of neighborhood cats with a system of mutual avoidance governed by a relative dominance hierarchy. Each cat is dominant about its own home or sleeping area despite its status away from this area.

3. Males may fight for a week or more to gain acceptance into a male "brotherhood." Young males may spend a year or more gaining ranking. High density, ownership with a reasonably steady supply of food and shelter, and unavailability of area in which to emigrate are most probable causes for this prolonged conflict.

4. Evening gatherings reported, but of uncertain occurrence

or significance. Mix of sexes and ages with little aggression and direct contact.

III. Social Communication.

A. Olfactory (the sense of smell).

1. Urine marking.
   a. Both males and females spray urinate. Mark edges of ranges as they enter, mark intermittently along their route. Males routinely mark as they travel; as frequent as every 5 meters.
   b. Urine has no apparent repelling quality.
   c. Loses most of its communicative significance after 24 hours.
   d. Young males in a farm environment retain the squat urination posture for 12-18 months before commencing spraying.
   e. Females urinate more frequently when in estrus (heat); males spray more in presence of estrous female.

2. Feces.
   a. Uncertain significance.
   b. Cats defecating around the core area of a farm will usually cover their stool. Feces often found uncovered along some pathways frequented by cats on their ranges.

3. Anal Glands.
   a. Uncertain importance.

4. Skin Glands.
   a. Cats have diffuse clumps of sebaceous (oil-excreting) glands along tail (caudal glands), forehead (temporal gland), lips (perioral gland), and chin.
   b. Inanimate objects tend to be rubbed with the caudal and perioral glands.
   c. Friendly head rubbing associated with temporal gland rubbing.
   d. One author claims rubbing is done more to pick up scent/increase social attraction, reassurance, camouflage, than to deposit scent.

5. Scratching.
   a. Foot pad area contains an abundance of sweat glands.
   b. Also visual signal, of hygienic value (removal of loose ungual, or claw, tissue), and a pleasurable activity (seen when cat awakens).

B. Visual.

1. Face and head.
   a. Ears. Alert = erect, pointed toward stimulus.

Offensive threat = directed toward side. Defensive threat = pulled down and/or to rear.

b. Mouth. Usually teeth not bared in aggressive display unless defensive also.

c. Pupil size. Alert, confident = slit pupils. Fear = dilated pupils. Confident cat looks directly at, fearful cat furtively looks away from stimulus.

d. Tail. Alert = tail carried out behind. Relaxed = same or curled up and forward. Offensive threat = pulled down perpendicular to ground. Submission = curled tail around body. Defensive threat = tail curled in inverted V.

e. Body Posture. Relaxed, moderately alert = head up, back straight and level. Offensive threat = legs straightened, hind-quarters elevated. Defensive = crouches, lies in sternal position, head may be held to side on ground.

f. Varying combinations of conflicting moods signaled by combinations of or fluctuations between certain displays.

C. Vocal.

1. Purr. Relaxed cat or mildly anxious, perhaps as a means of self-assurance.

2. Chirr. A rolled meow used by queen to call kittens, or in a friendly approach by two cats.

3. Call. Closed-mouth loud murmur used by female to indicate a readiness to mate.

4. Meow. Basically attention-seeking, announcement or frustration.

5. Growl, snarl, hiss. Open-mouth calls used in combative encounters.

IV. Behavior Problems.

A. Urine spraying.

1. Types.
   a. Tomcat.
   b. Castrated male.
   c. Female.
   d. Non-behavioral - cystitis, nephritis (bladder, kidney disease).

2. Treatment.
   a. Castrate male.
   b. Progesterone therapy - MPA, MA.
   c. Punish.

d. "Booby traps" - Aluminum foil, plastic sheet, mouse traps.
e. Feed in area.
f. Medial preoptic area lesion (brain lesion); males only.

B. Urinating or defecating outside litter.

1. Causes.
   a. Change in environment.
   b. Dirty litter or different brand (chlorophyll).
   c. Too many cats per pan.
   d. Social significance of area: bed, couch, sink, bathtub.

2. Treatment.
   a. Clean litter daily.
   b. One cat per pan, separate areas of the house.
   c. Use sand or change litter brand.
   d. Bed wetting pads; aluminum foil or pie tins.
   e. Covered litter box.
   f. Spend time with cat.
   g. Cage with litter.
   h. Punish if caught in act.
   i. Pan where cat eliminates; gradually move.
   j. Feed where eliminates.
   k. Water in sink or tub.
   l. Train to toilet.
   m. Dominate.

C. Intracat aggression.

1. Treatment.
   a. Introduce slowly.
   b. Time spent with each cat.
   c. Separate cats, fast them; feed together, gradually moving dishes together.
   d. Vaseline on back of both cats.
   e. Catnip.
   f. Anti-anxiety drugs.
   g. Confine if aggression with outdoor cat.
   h. Eliminate one or more cats.

2. Prevention.
   a. Slow introduction.
   b. New cat opposite sex.
   c. New cat a kitten.
   d. Keep only one cat in a house.
   e. Raise kittens in same house together.
   f. Neuter young cats early.
   g. Confine cat to house.

D. Aggression toward people.

1. Types.
   a. Irritable.
   b. Pain induced.
   c. Play induced.
   d. Hypothyroidism (deficiency of thyroid hormones).

2. Treatment.
   a. Relieve pain, hormone deficiency.
   b. Punish, especially by shaking.
   c. Progesterone therapy.

E. Ingestion problems.

1. Types.
   a. Anorexia.
   b. "Finickiness."
   c. Plant eating.
   d. Wool sucking.

2. Treatment.
   a. Anorexia: hand-feeding, highly odiferous foods, specific appetite-stimulating drugs.
   b. "Finickiness": be stubborn.
   c. Plant eating: provide "cat garden" since this is a normal behavior; put pepper or vinegar on house plants; punish cat directly.
   d. Wool sucking: punish, do not breed cat.

3. Prevention.
   a. "Finickiness": provide one or two types of food exclusively; stimulate the bored cat with attention, play items and structures.
   b. Wool sucking: perhaps allow kittens to nurse for 12 weeks or longer.

F. Mourning.

1. Treatment.
   a. Another cat.
   b. Confine when owner away.
   c. Time with cat.

G. Meowing.

1. Treatment.
   a. Do not reward.
   b. Time out, systematic confinement.
   c. Punish.

H. Sexual problems.

1. Types.
   a. Masturbation.

b. "Psychological castration."
c. Nymphomania.
d. Paternal aggression toward kittens.

2. Treatment.
a. Gonadectomy (neuter or spay).
b. Remove dominant animal.
c. Induce ovulation.

I. Maternal problems.

1. Types.
a. Neglect.
b. Adoption.
c. Rejection.

2. Treatment.
a. Hand rear first litter.
b. Do not breed again if multiparous female (cat which has had more than one litter).
c. Keep litters separate from other females.
d. Massage mammary glands to relieve pressure if they seem painful.

FELINE NUTRITION

Francis A. Kallfelz, D.V.M.

I. Nutrition involves the chemical reactions and physiological processes necessary to transform foods into body tissues and activities.

A. Physiological processes for processing nutrients

1. functional gastro-intestinal system
   a. to ingest and digest nutrients
   b. to absorb nutrients
   c. to excrete waste products

2. accessory organs
   a. pancreas (digestive enzymes)
   b. liver (remove toxins, secrete bile acids)
   c. endocrine glands (to put nutrients to use in the body's cells)
   d. kidney (excretion of digestive waste, e.g. ammonia)

B. Functions of nutrients in the body

1. growth

2. tissue repair

3. reproduction/lactation

4. maintenance
   a. cell structure
   b. normal body weight
   c. conversion of energy to useful work

II. The basic nutrient needs of any living being must be supplied in the diet and absorbed. They must be made available in a palatable form.

A. Proteins are composed of nitrogen-containing subunits called amino acids.

1. structural functions of proteins in the body
   a. major component of soft tissues (muscles, organs, skin)
   b. major compoenent of cell membranes (red blood cells, tissue)

2. physiologic functions
   a. tissue repair
   b. growth of new tissues (muscles, fetus)
   c. enzymes (catalyze reactions in the body for food conversion)
   d. antibodies (immunity)

3. energy functions
   a. can be used as a source of energy when fats or carbohydrates are unavailable to meet energy demands

4. specific protein requirements for cats
   a. adults: 2.5-3.0 gm protein/lb. body weight/day
   b. kittens: 8.6 gm protein/lb. body weight/day
   c. requirements depend on:
      --biological value of the protein source (the composition of amino acids - essential vs. non-essential)
      --energy intake and demands (the use of protein as an energy source prevents its use as a structural component)
   d. cats have a specific need for the amino acid known as taurine (only available in animal protein sources)

5. too little protein or protein of poor biological value leads to problems:
   a. poor healing
   b. slow growth (young)
   c. specific deficiency of taurine results in retinal degeneration and blindness.

B. Energy - carbohydrates, fats (lipids) and proteins acts as energy stores because useful energy is released when they are broken down in the body.

1. uses of energy
   a. regulation of body temperature
   b. maintaining functions (activity, growth, reproduction)

2. requirements
   a. measured in kilocalories
      adults: 35-40 kcal./lb. body weight/day
      kittens: 115 kcal./lb. body weight/day
   b. various enzymes, vitamins and minerals are required for the utilization of energy
   c. requirements depend on activity and stage of growth

3. sources
   a. fats: provide 9 kcal/gm of fat
      --cats require certain lipids in the diet (essential fatty acids) which are supplied only in animal fat arachidonic acid, linoleic acid and linolenic acid
      --fats carry fat soluble vitamins (A,D,E,K)
      --improve the palatability of food
   b. carbohydrates (CH_2O): provide 4 kcal/gm of carbohydrate
      --cats have no specific requirement but this is the cheapest form of energy
      --starch may be cooked to increase its digestibility
      --lactose is a major energy source for kittens (milk) but adults are often unable to digest it and develop diarrhea

4. problems with energy imbalances
   a. lack of essential fatty acids results in poor haircoat (severe dandruff, greasy feeling) and poor growth
   b. diarrhea from lactose intolerance in adults

C. Minerals (ash) are needed for structural support and in chemical reactions. They cannot be used as energy sources.

1. Macro-minerals
   a. calcium/phosphorus/magnesium (in milk, bone meal, egg shells)
      --major elements in bones and teeth
      --needed for energy conversion, nerve impulse conduction, muscle contraction, blood coagulation, lactation
      --imbalances can create bone problems (high P, low Ca) and can contribute to urinary tract obstruction in tom cats (high Mg).
   b. potassium/sodium (salt)
      --maintains body fluid balance
      --helps to maintain muscle strength
      --imbalances can result in weakness, edema (abnormal accumulations of fluid)

2. Micro-minerals
   a. iron/copper - blood formation, hair
   b. zinc - insulin, skin, hair
   c. manganese - growth, bones, reproduction
   d. selenium - interacts with viramin E, muscles, reproduction
   e. iodine - thyroid gland function - metabolism

D. Vitamins are not useful as energy sources but are needed in small amounts to allow proper functioning of organs and cells. Some are manufactured in the body, most can only be supplied from food.

1. fat soluble vitamins (A,D,E,K)
   a. vitamin A
      --cats are unable to convert B carotene (plant source of pre-vitamin A) into vitamin A; need source of vitamin A from animal tissues or synthetic vitamin A
      --hair, eyes, bone growth
      --imbalance may result in vision impairment and in bone defects
   b. vitamin D
      --needed for bones and teeth
      --easy to produce excess or toxicity
      --excess may cause soft tissues to mineralize
      --deficiency causes poor bone growth
   c. vitamin E
      --with selenium, important in muscles and reproduction
      --helps to keep fats from oxidizing (becoming rancid)
      --imbalances may be seen in high fat diets and result in steatitis (a painful inflammation of fatty tissue)

d. vitamin K - blood coagulation (D-con)

2. water soluble vitamins (B vitamins and C)
   a. Thiamin (B_1)
      --necessary in the use of carbohydrates and proteins
      --deficiency may cause nerve impairment
      --adequate in most foods, but improper processing may destroy the vitamin - too much heat
      --raw fish contain thiaminase, a substance capable of destroying thiamin: cats lose their appetite, vomit, have convulsions and begin to stagger...thiamin treatment is curative
      --cats need about twice the amount needed by dogs
   b. Riboflavin (B_2)
      --growth, skin, hair
   c. Pantothenic acid
      --skin and hair
   d. Niacin
      --tissues of the mouth, nerves
      --most animals can convert tryptophan (an amino acid) to niacin, cats cannot and must get niacin from the food
   e. Pyridoxine (B_6)
      --protein metabolism and growth
   f. Folic acid
      --growth and blood
   g. Biotin
      --growth, skin, hair
      --deficiency only with ingestion of large quantity of raw egg whites (avidin ties up the biotin)
   h. B_{12}
      --red blood cells
   i. vitamin C
      --cats can produce their own vitamin C, not needed in food
      --needed to maintain tissue integrity
      --may be used clinically to help acidify urine of tom cats who tend to become obstructed

E. Water is the most basic and the most important nutrient. Clean, fresh water should always be available.

1. cats need about 1 oz./lb. body weight/day

2. water is available from drinking water, from ingested food, and made during metabolism

3. requirements depend on environmental temperature, activity and physical condition...several diseases result in excessive water consumption (polydipsia)

III. Commercially available cat foods - probably the best approach

A. Dry cat foods

1. most are complete and balanced
2. almost all are "expanded" products

3. composition

Dry Cat Food

Ingredient	% as is	% dry	Calories (per lb.)	Calories (%)
Crude Protein	30.0	34.0	545	33.5
Crude Fat	8.0	9.0	327	20.1
Crude Fiber	4.5	5.1	---	
Moisture	12.0	---	---	
Ash	6.0	6.8	---	
NFE	41.5	47.1	754	46.4
			1626	

4. advantages
   a. inexpensive
   b. long-term storage
   c. easily used for continuous feeding

5. disadvantages
   a. less palatable than other types

B. Soft moist cat foods

1. all are complete and balanced

2. contain textured soy protein

3. composition

Soft Moist Cat Food

Ingredient	% as is	% dry	Calories (per lb.)	Calories (%)
Crude protein	27.0	40.9	490	33.8
Crude Fat	8.5	12.9	344	23.8
Crude Fiber	3.5	5.3	---	
Moisture	34.0	----	---	
Ash	4.5	6.8	---	
NFE	22.5	34.1	614	42.4
			1448	

4. advantages
   a. more palatable than dry
   b. less expensive than canned
   c. may increase water turnover

5. disadvantages
   a. dries out after package opened
   b. more expensive than dry

C. Canned cat foods

1. much variability - some balanced, many are not - gourmet products

2. contain 70% or more water

3. composition

Canned Cat Food (Gourmet)

Ingredient	% as is	% dry	Calories (per lb.)	(%)
Crude Protein	12.0	46.2	218	31.0
Crude Fat	6.0	23.1	245	35.0
Crude Fiber	1.5	5.7	---	
Moisture	74.0	---	---	
Ash	3.0	11.5	---	
NFE	3.5	13.5	245	34.0
			708	

4. advantages
   a. very palatable
   b. high water content?

5. disadvantages
   a. expense
   b. many are unbalanced
   c. storage and spoilage

IV. Nutritional adequacy of foods: a food must be ingested (palatable), digested and absorbed to be available as a source of nutrients. To ensure the adequacy of commercial rations, the Federal Food and Drug Administration and the U.S. Department of Agriculture require foods to be pure and wholesome without harmful substances and truthfully labelled.

A. Label information

1. "nutritional statement"
   a. the words "Complete, Balanced" can only appear on the label of rations that have been tested and can meet the needs stated.
   b. rations must meet NRC requirements in order to be so labeled.

2. "ingredient list"
   a. ingredients are in order or predominance by weight (an example of adequate protein sources for cats would be an animal protein source appearing as the first or second

ingredient in a canned ration or third or fourth in a semi-moist or dry ration)

3. "guaranteed analysis"
   a. includes crude protein, crude fat, crude fiber, moisture and ash
   b. gives no information on digestibility (an example: hair is high in protein but is not readily digested and is poorly used by the body)

4. cost
   a. helps to evaluate relative quality of different rations

5. net weight
   a. needed to evaluate relative expense and amounts to feed for adequate supply of nutrients

6. manufacturer's address
   a. to obtain information on digestibility

B. Using label information

1. Nutrients on a dry matter basis - different rations have different moisture contents. To compare different products, convert to % dry matter (%DM) for each of the nutrients.

   100% minus % moisture = % total dry matter

   Guaranteed % of nutrient divided by % DM x 100 = % specific nutrient DM

2. Cost per pound of dry matter

   cost/weight (oz.) x 16 = cost/pound of food

   $$\frac{\text{cost/pound of food}}{\text{\% dry matter}} \times 100 = \text{cost/pound of dry matter}$$

3. Cost per pound of protein (protein is the most expensive ingredient)

   $$\frac{\text{cost/pound}}{\text{\% protein}} \times 100 = \text{cost/pound of protein}$$

4. Caloric analysis (calories/pound of food)
   a. determine Nitrogen-free extract (NFE) - this is the carbohydrate source.

      100 minus total % of nutrients in guaranteed analysis= NFE

b. NFE (carbohydrates) and protein provide 4 kcal/gm

NFE % x 4 = kcal/100 gm

Protein % x 4 = kcal/100 gm

c. fat provides 9 kcal/gm

% fat x 9 = kcal/100 gm

d. Kcal/pound = kcal/100 gm x 4.5

V. Feeding cats with special needs may or may not require supplementation. Supplements are usually necessary for cats fed complete commercial rations.

A. Available supplements

1. Brewer's yeast
   a. relatively high in phosphorus
   b. provides some B vitamins (these are usually adequate in the diet)
   c. may not be adding anything useful to an already complete ration

2. Commercial vitamin-mineral supplements
   a. some approach "mega-vitamin" proportions - may cause an imbalance
   b. added to a complete ration may result in toxicity

B. Home formulation of a complete diet

1. difficult to achieve a complete diet

2. must know the nutrient value of the foods used

3. must provide variety and be certain that all is eaten (not just the meat)

4. even the best combination may not meet the cat's needs

C. Special nutritional needs of different stages of life

1. Reproduction/parturition
   a. needs slightly more food at this stage than for maintenance
   b. obesity makes parturition difficult

2. Lactation
   a. lactating queens may need up to three times the maintenance level of nutrients
   b. increased needs for energy, protein, calcium, phosphorus

3. Kittens (growth)
   a. high protein and energy requirements decrease with increasing age
   b. kittens need 10 gm/lb. body weight;
      adults: 2 gm/lb. (protein)
   c. Kittens need 136 kcal/lb. body weight;
      adults: 40 kcal/lb. (E)
   d. complete ration suitable for that stage of life

4. Orphans
   a. queen's milk is usually unavailable, so a replacement is needed
   b. KMR (Kitten Milk Replacer, commercial), Unilact, etc.
   c. require high protein, high fat, lactose
   d. caloric analysis (similar to that for complete ration)

KMR	Protein	7.5% x 4	= 30.0 kcal/100 ml
	Fat	4.5% x 9	= 40.5
	Ash	1.5% -	
	Fiber	0% -	
	Moisture	82.0% -	

100% - 95.5% = 4.5% (NFE) x 4 = 18.0 kcal/100 ml

88.5 kcal/100 ml

0.1 kg kitten x 300 cal/kg = 30 cal (total needed)

30/88.5 x 100 = 34 ml (approx. 2-3 TBS)

34 ml provides .34 x 7.5 (protein) = 2.55 gm protein
Requirement is 15.9 x .1 (kg) = 1.59 gm protein

   e. kittens can be tube fed or bottle fed if active and alert

VI. Current research efforts in nutritional disease

A. Feline Urolithiasis Syndrome is a complex disorder affecting primarily tom cats. It has received much attention due to its tendency to recur in "susceptible" cats.

1. clinical signs
   a. straining to urinate, bloody urine, depression, crying

2. treatment
   a. catheterization to remove obstruction
   b. fluid therapy to correct fluid imbalances
   c. antibiotics

3. suspected causes
   a. viruses
   b. castration
   c. water consumption
   d. high ash in diet (magnesium)
   e. high urine pH

4. possible means of prevention
   a. feed low ash (magnesium) diet, e.g., c/d
   b. acidify urine
   c. increase water consumption (salt food)
   d. feed soft-moist ration

DAILY FOOD REQUIREMENTS OF CATS ACCORDING TO AGE AND WEIGHT

Age (wks)	Wt (lbs)	Caloric Need (cal/lb)	Food Requirements (lbs)		
			Dry	Soft-Moist	Canned
5	1.0	125	0.10 (1.5 oz)	0.10 (1.5 oz)	0.31 (5.0 oz)
10	2.0	100	0.15 (2.4 oz)	0.16 (2.6 oz)	0.50 (8.0 oz)
20	4.5	65	0.15 (3.0 oz)	0.24 (3.8 oz)	0.73 (12.0 oz)
30	6.5	50	0.20 (3.5 oz)	0.26 (4.2 oz)	0.81 (13.0 oz)
Adult	9.0	40	0.25 (4.8 oz)	0.30 (4.7 oz)	0.90 (14.4 oz)

PROTEIN AND ENERGY REQUIREMENTS OF CATS+

	Req.* in Food	Req.** in Cat	
		Kittens (10 weeks)	Adults
Protein (%)	30	8.6	2.9
Energy (Cal/100 gm)	400	115.0	40.0

+Modified from NRC (1978).
*Expressed on a DM basis.
**Expressed as amt/lb body weight/day.

MINERAL REQUIREMENTS OF CATS+

1. Macro-minerals

Mineral	Req.* in Food	Req.** Per Cat	
		Kittens (10 wks)	Adults
Ca	1000	290	90
P	800	230	80
Mg	50	14	5
K	300	90	30
Salt	500	140	50

+Modified from NRC (1978).
*Expressed as mg/100 gm food DM.
**Expressed as mg/lb body weight per day.

MINERAL REQUIREMENTS OF CATS+

2. Micro-minerals

Mineral	Req.* in Food	Req.** Per Cat	
		Kittens (10 wks)	Adults
Iron	100.0	30.0	10.0
Copper	5.0	1.4	0.5
Manganese	10.0	2.8	0.9
Zinc	30.0	8.6	3.2
Iodine	1.0	.3	.09
Se	0.1	.03	.009

+Modified from NRC (1978).
*Expressed as ppm DM.
**Expressed as ug/lb body weight per day.

VITAMIN REQUIREMENTS OF CATS+

Vitamin	Req.* in Food	Req.** Per Cat	
		Kitten (10 wks)	Adult
A (IU)	1000.0	290.00	100.00
D (IU)	100.0	29.00	10.00
E (IU)	10.0	3.00	1.00
Thiamin (mg)	0.5	0.14	0.05
Riboflavin (mg)	0.5	0.14	0.05
PA (mg)	1.0	0.28	0.10
Niacin (mg)	4.5	1.00	0.50
Pyridoxine (ug)	400.0	140.00	45.00
Folic Acid (ug)	100.0	30.00	9.00
Biotin (ug)	5.0	1.00	0.50
B_{12} (ug)	2.0	0.60	0.20
Choline (mg)	200.0	57.00	20.00

+Modified from NRC (1978).
*Expressed as amts per 100 gm food DM.
**Expressed as amts per lb body weight per day.

CONTENT OF COMMON VITAMIN SUPPLEMENTS COMPARED TO BREWER'S YEAST AND NRC REQUIREMENTS

Vitamin	Kitty Bloom*	Forte*	Vita Yeast	Brewer's NRC
A (IU)	5506.00	440.0	-	800.0
D (IU)	413.00	30.0	-	80.0
E (IU)	11.00	8.0	-	8.0
K (IU)	2.00	-	-	-
C (mg)	25.00	6.0	-	-
Thiamin (mg)	2.75	0.5	.25	0.4
Riboflavin (mg)	4.40	0.5	.10	0.4
PA (mg)	23.00	2.0	.30	0.8
Niacin (mg)	34.40	3.8	1.25	4.0
Pyridoxine (mcg)	825.00	506.0	125.00	360.0
Folic Acid (mcg)	275.00	25.3	25.00	4.0
Biotin (mcg)	5.50	31.6	2.50	4.0
B_{12} (mcg)	44.00	1.0	-	1.6
Choline (mg)	165.00	5.6	10.80	160.0

*Amt in 1 teaspoon assuming 1 teaspoon weighs 2.5 gm.
**Daily requirement for adult cat weighing 8 lbs.

FELINE GENETICS

Elizabeth A.B. Oltenacu, Ph.D.

The cat breeder needs a knowledge of how traits are inherited in order to make improvements in the chosen breed. A breeding program should include careful selection of the breeding stock, which can only be achieved if the breeder is aware of the genetic nature of the trait of interest. Traits may be inherited in a simple manner, or may be controlled by many inherited factors, each with a barely measurable effect on the trait it controls. Hence, the breeding program to be used will depend on the breeder's understanding of the inheritance of the trait. A poor understanding of the genetics of the cat will lead to an ineffective breeding program, whereas a clearer knowledge of genetics will enable the breeder to achieve much in improving the breed. This paper discusses the types of inheritance that the breeder is likely to encounter, the goals of breeding programs, and how to choose the most effective breeding and selection scheme to improve the characteristic(s) of interest.

THE PHYSICAL BASIS OF INHERITANCE

The inheritance of any trait has a clearly-defined physical (chemical) basis, in that the elements of genetic material can be seen under the microscope when body cells are correctly prepared, and their chemical nature is understood. Much still remains to be discovered in terms of their precise workings, however.

The cat's body is made up of cells, and within each is a "center of operations," the nucleus, which contains the genetic information -- the "blueprint" that specifies the makeup of that cat. Chemical stains can be used on cells that are arrested during their division process to show the thread-like chromosomes which carry the genetic information. The cat has 38 chromosomes. When the cell is observed during its division process, the chromosomes are seen to have duplicated themselves, and the duplicates then pull apart into the two new daughter cells that result from the division. Hence, each new cell has the same number of chromosomes.

What happens when reproductive cells (sperm in the male and ova in the female) are produced? If each had the full number of chromosomes, then fertilization of an egg by a sperm would produce an offspring with twice as many chromosomes as each parent had. This doubling would occur in each generation. However, when reproductive tissues are studied, the chromosomes are seen to line up in pairs, with one member of each pair going into each reproductive cell. So, a feline sperm cell or an ovum contains 19 chromosomes. When a sperm fertilizes an egg, the progeny has 19 + 19 = 38 chromosomes, just like either parent.

The conclusion to be drawn from this is that the individual units of inheritance, the genes, which are located along the chromosomes, also occur in pairs. This is important in understanding how inheritance operates.

Close examination of the 19 pairs of chromosomes shows that shapes differ from pair to pair, but that the two members of each pair look alike in the female. In the male, there is one pair of unmatched chromosomes. These are the sex chromosomes. The larger one (the X-chromosome) matches a pair of the female's chromosomes, the smaller one (the Y-chromsome) being quite different in size and shape. Females are therefore XX and males XY in sex chromosome makeup. Each egg must contain an X-chromosome, while half the sperm cells will bear an X, half a Y. Hence, it is the male that determines the sex of each kitten. Those produced by a Y-bearing sperm will be males, and those produced by an X-bearing sperm will be females.

		♂ Male (sperm) X	♂ Male (sperm) Y
♀ Female (egg)	X	XX Female	XY Male
	X	XX Female	XY Male

This diagram can be used with any pair of genes, to understand the possible outcomes of a mating. Here, it is used for entire chromosomes, but it works as well for a gene pair.

MENDELIAN GENETICS

An Austrian, Gregor Mendel, was the first person to develop a clear understanding of how traits were inherited, and so the inheritance of simple traits is named after him. He achieved his understanding before the physical basis of inheritance had been observed.

The sex-chromosomes carry genes that determine sex, and the Y-chromosome has few other genes. However, the X-chromosome carries genes for many traits. A well-known example is the sex-linked orange color in cats. Sex-linked traits are those controlled by genes on the sex chromosome. This color gene takes two different forms (called alleles). One allele determines orange color (O), the other allele non-orange (o). So, using the same diagram, mating a black male and an orange female:

		Male (♂) X^o	Male (♂) Y
Female (♀)	X^0	X^0X^o tortoiseshell	X^0Y orange
	X^0	X^0X^o tortoiseshell	X^0Y orange

Female kittens are tortoiseshell, males orange. If the reverse mating had been made (orange male x black female), the result would have been tortoiseshell daughters and black sons.

The outcome is more straightforward for genes that are not on the X-chromosome, which is the majority of genes. For such cases, it is irrelevant which parent supplied which allele. Hence, a mating between a longhair (l) and shorthair (L) gives:

	L	L
l	Ll	Ll
l	Ll	Ll

The progeny are all alike, but they are not intermediate in hair length. They are all shorthairs. The gene for short hair is said to be dominant to that for long hair, which is recessive. The cat with two similar alleles (LL or ll) is said to be homozygous, while the Ll progeny are heterozygous. For these kittens, the phenotype (visual appearance) does not disclose the genotype (genetic makeup for this trait) as it did for the tortoiseshell kittens. This is one of the problems of animal evaluation. Often, only a breeding test will disclose what alleles a cat will transmit.

Interactions between different pairs of genes (called epistasis) can also confuse the picture. The following example shows a dominant white (W) male mated to three females:

			male Ww CC	
females:			WC	wC
1.	wwc^bc^b Burmese	wc^b	$WwCc^b$ white	$wwCc^b$ full color
2.	wwc^sc^s Siamese	wc^s	$WwCc^s$ white	$wwCc^s$ full color
3.	wwc^ac^a blue-eyed white	wc^a	$WwCc^a$ white	$wwCc^a$ full color

Firstly, it is difficult to know whether two white cats are genetically the same color or not without careful test matings. The male is white because of the W allele, female #3 is white because of a totally different gene, c^a. The gene for full color (C) is carried by the male, but he does not show its effect in his phenotype because the W gene prevents any color expression.

This example also illustrates multiple alleles at a locus (the position on the chromosome where specific genes are located). C, c^b, c^s and c^a are all alleles. Mating between cats of all genotypes for this locus has shown C (full color) to be dominanat to all its alleles. The c^b (Burmese) and c^s (Siamese) alleles are codominant, the result being the Tonkanese (c^bc^s) intermediate between Siamese and Burmese. This is comparable to the Oo tortoiseshell. The c^a allele ranks next, and, at the end of the list is c (pink-eyed true albino) which is recessive to all other alleles at this locus. Table 1 gives a listing of the best understood major alleles of the cat.

Table 1. Major alleles of the cat.

Color		Hair type	
A	agouti (banded hair color)	H	normal hair
a	non-agouti	h	hairless
B	black	L	short hair
b	chocolate brown	l	long hair
b^l	red brown		
		R	normal hair
C	full color	r	Cornish rex
c^b	Burmese		
c^s	Siamese	Re	normal hair
c^a	blue-eyed white	re	Devon rex
c	albino (pink eyes)		
		Wh	wire hair
D	intense color	wh	normal hair
d	dilute color		
I	pigment inhibition		
i	normal pigmentation		
O	sex-linked orange		
o	non-orange		
		Other	
S	white spotting (incompletely dominant)		
s	no spotting	FD	folded ear
		fd	normal ear
T^a	Abyssinian tabby		
T	striped tabby	M	Manx tail
t^b	blotched tabby	m	normal tail
W	dominant white	Pd	polydactyly
w	colored	pd	normal # of toes

BREEDING OUT AN UNDESIRED GENE

A recessive allele (h) that causes hairlessness in kittens has a disastrous effect on the individual. A breeding colony will always have some such allele in it, but it is only discovered when particular matings (often those between closely related individuals) are made. How should the owner discover the extent of the problem and eliminate the unwanted allele from the breeding stock?

The first answer is not to use affected animals (e.g. hh hairless cats) in the breeding program, or their parents, which must have been Hh animals. The problem lies in detecting which of the normal-appearing (H_) cats actually carry the h allele. For example, other kittens from the Hh parents that produced the hh kitten must all be suspect. If the homozygous recessive type (hh in this example) is not lethal but able to survive and reproduce, it can be used in test matings with suspect individuals:

	Suspect (H_)	
	H	_
recessive (hh) h	Hh normal	h_

If the blank (_) is an h, half the kittens are expected to be hairless. Remember that it only takes one abnormal kitten to prove that the suspect does indeed have the h allele to transmit!

How many normal kittens are needed before the breeder can be reasonably confident that the suspect is HH and can be safely brought into the breeding program? The chance (probability) of getting an H allele from the suspect is 1/2 each time it produces a kitten, if the suspect is Hh in genotype. Hence, in the mating of the suspect with the hh test animal:

Chance of 1 Hh kitten = 1/2

Chance of 2 Hh kittens = 1/2 x 1/2 = 1/4

Chance of 3 Hh kittens = $(1/2)^3$ = 1/8

Chance of 4 Hh kittens = $(1/2)^4$ = 1/16

etc.

Confidence that the suspect is not Hh increases with each normal kitten born, as it is less and less probable that successive kittens will all be normal from an Hh parent. The breeder stops when the probability level is low enough. Less than 1% is an acceptable level, and is reached when 7 successive normal kittens have been produced. Never forget, however, that just one hh kitten would prove the suspect to be a carrier, and the testing can stop right there.

If the trait in question prevents the homozygous individual from reproducing, then test matings must be made with carrier individuals, i.e., those that have produced affected progeny. This means that more progeny must be obtained before the same certainty that the suspect is not a carrier is reached (only 1 in 4 progeny from the mating of two heterozygotes is expected to be homozygous recessive, so there is a probability of 3/4 for each progeny being normal). Sixteen successive normal progeny are needed to reach the 1% probability level. Again, the test matings can stop the moment one abnormal kitten is produced.

Test matings are mostly done to test males, as it is not always practical to expect to get enough kittens from one female to adequately test her.

CHARACTERISTICS THAT SHOW CONTINUOUS VARIATION

Basic Mendelian genetics is the underlying principle for the inheritance of any trait. However, there are many traits controlled by large numbers of gene pairs, each with a small, not individually measurable, effect on the trait. These are quantitative traits, as opposed to the qualitative traits discussed in the previous two sections. Also, such traits are frequently influenced by the environment, so that the phenotype is the expression of the genotype as modified by the environment. This increases the problems of animal evaluation even further.

To better understand such traits, consider spotting in the cat's coat pattern. Spots are qualitatively controlled by a major allele (S), but the degree of spotting is determined by an unknown number of minor, modifier gene pairs. The net result can be thought of as S determining that the cat will have white spots, and other loci increasing (+) or decreasing (-) the area of the spotting. For example, suppose that there are only 3 minor gene pairs affecting the amount of spotting. If each upper case allele increases the amount of spotting by 10%, and the baseline aabbcc is 10% spotted, then animals will vary from this to 70% spotted (AABBCC). When animals in the whole population are studied, they will be seen to show a continuous variation in amount of spotting with fewest animals at the extremes, and most in the middle of the range (this is a bell-shaped curve, called a Normal distribution).

Important traits such as growth, reproduction, milking ability, etc. are all quantitative traits. Some, such as reproductive traits, have a low genetic component (called the heritability) with a major environmental component (e.g.. feeding, health, etc.). This must invariably influence the breeder in the choice of a breeding program. Selection schemes for such quantitative traits are more difficult than are those for qualitative traits, and the breeder must understand the genetic basis of the trait of interest to understand the potential for making genetic progress.

BREEDING PROGRAMS

The type of program to be undertaken to eliminate an undesirable allele was discussed previously. Breeding schemes to be discussed here

a) Inbreeding
b) Linebreeding
c) Line crossing
d) Crossbreeding

Inbreeding is the mating of closely related individuals, often full sibs (brother to sister matings) or parent to offspring. The intention is to produce progeny that are more homozygous and therefore more uniform than those produced without inbreeding. However, homozygosity of good and detrimental alleles will result, so many inbred lines are lost as a result of a decline in vigor. Sometimes major deleterious alleles will be uncovered, but a steady loss of vigor and reproductive ability is the most likely problem. A good inbred line is very predictable in breeding performance, but, overall, close inbreeding is to be avoided because of the decline in quality often found in inbred stocks.

Linebreeding is a form of moderate inbreeding which attempts to concentrate alleles from an outstanding individual without matings between close relatives. So, when the ancestry of kittens is studied in a pedigree, the same outstanding individual will appear several times in the breeding of the two parents. Hence, the percentage of alleles coming from that individual may be quite high, but the inbreeding of the kittens will not be excessive.

Line crossing is the mating of individuals from different lines in order to put together desirable features from both. Often, line cross or outcross kittens have added vigor. This results from their increased heterozygosity because the two lines will have differed in the alleles at many loci.

Crossbreeding is the mating of cats from totally different breeds. Crossbreds are usually vigorous because of their extensive heterozygosity. This is known as hybrid vigor or heterosis. Crossbreeding is used in the creation of a new breed, or in the introduction of a desired trait into a breed that lacks it. It is followed, in the latter case, by successive matings back to the pure breed (grading up), with stringent selection and culling of breeding stock. First generation crossbreds are genetically and phenotypically uniform, but, if mated among themselves, will produce highly variable offspring. So, if a new breed is being developed, very strong culling of all but the best individuals must be practiced.

COMMON FELINE DERMATOLOGICAL DISORDERS

Charles A. Baldwin, D.V.M., M.S. and Thomas O. Manning, D.V.M.

The skin and hair (integument) is an organ system which is accessible to the senses of sight, touch and smell. Usually any change from the normal condition is readily observed by an owner. The integument can only react in a limited number of ways to a varied number of inciting agents, chemicals, parasites, bacteria, viruses, and mechanical injuries. Unfortunately, though it may be easy to observe a deviation from the normal skin condition, it may be very difficult to diagnose and resolve the problem. For instance, it may be easy and accurate to say that a cat is demonstrating an allergic reaction via the skin, but it may be impossible to detect exactly what the cat is allergic to and also impossible to attain complete remission of signs.

An attempt will be made to categorize the multitude of skin diseases that commonly occur in the cat. The various diseases will be divided into broad categories by the major presenting sign or symptom. This is not to say that the disease will always present with that particular sign. The major complaint by the owner may vary with the length of time that the disease has been present. For instance, persistent scratching may destroy any vesicles that may have been the original lesion or may denude an area of skin that only had a large number of broken hair shafts.

The various major presenting signs include: (1) hair loss or alopecia; (2) scratching or pruritus; (3) crusts or scabbing; (4) ulcers and fistulas; (5) tumors; and (6) miscellaneous (signs that do not fit anywhere else in the scheme). The reader will note as he or she proceeds that there can be a large amount of overlap of categories and diseases. Some diseases may be listed in several of these broad categories, because, as stated before, the length of time the disease has been present alters the progression of the presenting lesion; and, also, there is individual variation and sensitivity to the basic underlying cause of the skin disease.

DISEASES THAT PRESENT AS ALOPECIA

Alopecia is the loss of hair occurring anywhere on the animal's body. There is usually little to no skin involvement and therefore the animal is usually not scratching; the skin is not red and irritated. Upon close examination the affected area may be devoid of all hair or else hair shafts may still be present, but broken off with only a stubble remaining. There are several diseases in the cat which can produce alopecia, but the four common diseases are feline endocrine alopecia, miliary dermatitis, dermatophytosis (commonly called ringworm), and psychogenic alopecia.

Feline endocrine alopecia is seen primarily in male neutered cats, although approximately 10% of the cases may be in spayed females. This condition is not seen in purebred animals. The hair loss is usually bilaterally symmetrical, and usually begins on the ventral abdomen,

perineum, and caudal thigh regions. From these areas, it will usually progress slowly anteriorly. Oddly enough, the dorsal midline along the spine is usually spared. The skin shows no redness or itching, and the hairs are usually very easy to pull out. The cause of this disease is unknown, but it is thought to be due to a deficiency in the sex hormones, since it is seen only in neutered animals. The treatment involves replacement therapy with the sex hormones. Benefit from treatment will last from 6 months to 2 years; but up to 50% of the cases tend to recur.

The second disease involving alopecia is miliary dermatitis, or scabby cat disease. In this syndrome, the cat loses hair along the top of the spine with associated scabs. The list of inciting agents for miliary dermatitis is long, including: flea bite hypersensitivity; Cheyletiella; pediculosis; endoparasites; dermatophytosis; bacterial folliculitis; drug eruption; food allergy; biotin deficiency; fatty acid deficiency; and idiopathic (unknown). Many of these will be discussed in the next category.

The third disease that produces primarily alopecia is dermatophytosis or ringworm. Ringworm is a superficial fungal disease. Ringworm cannot survive in live tissues; it must invade the dead layers of skin and hair. Another important aspect of the disease is that the cat is believed to be a natural host for certain dermatophytes (fungus parasites on the skin). For this pathogen to survive, it must not severely harm its host. The greater the tissue damage, the greater the likelihood the pathogen will be rejected. In a natural host, there is little to no reaction to a parasite or pathogen. Therefore, many cats are asymptomatic carriers of ringworm. Classic ringworm lesions are seen as circular areas with broken hairs, etc., however, dermatophytes can vary greatly in their clinical presentation. In the cat, 98% of the cases are due to one type of dermatophyte, Microsporum canis. Ringworm is most accurately diagnosed with fungal cultures. A Wood's lamp (ultraviolet light) and looking for fluorescence is helpful in 30% of ringworm cases. Skin biopsy can be a helpful adjunct to fungal cultures. Treatment involves the use of griseofulvin (50-80 mg/lb) daily in a fat-laden food and topical 2% lime sulfur dips until cured. But, as mentioned above, it is difficult to tell sometimes in a carrier cat whether a "cure" has taken place. Ringworm lesions classically resolve in 6-8 weeks. The problem, especially for catteries, is that the fungal spores can persist in the environment for over a year. Reinfection is often impossible to prevent. Griseofulvin, when given to a pregnant queen, can cause birth defects and congenital deformities. Ringworm, then, can be a major problem in a cattery situation, since there is a high concentration of cats, which are a natural host for the pathogen and can be asymptomatic carriers. This situation is especially true for long-haired cats. This disease has human health significance, and a physician should be consulted.

The last major disease producing alopecia is psychogenic alopecia. This problem can occur in any breed of cat, but it is especially prevalent among Siamese, Burmese, Himalayans, Abyssinians, and their crosses. The lesions can be found anywhere on the cat. The dorsum of the spine is the most common area affected. The skin is normal, but the hairs are all broken and stubbled. Unlike feline endocrine alopecia, the lesions are usually not bilaterally symmetrical. The actual cause is unknown but, as

the name implies, it is psychogenic in nature. The range of pyschogenic stimuli could include new pets, loss of pets, or loss of a favorite family member, to name but a few. Diagnosis is usually made by exclusion (eliminating other potential diseases) and "backing into" a diagnosis of psychogenic alopecia. No matter what the inciting stimulus is, the only beneficial treatment is correcting the insult or mild sedation with the use of phenobarbitol daily. Bandaging and other treatments have all proved to be of little benefit.

DISEASES PRESENTING AS PRURITIS

The second major classification of diseases includes those which usually present with pruritis, a scratching cat. Since the animal is bothered by the disease process, the skin, in addition to hair loss, will usually be red and may even be bleeding. Therefore, the skin may be quite scabby. The list of common diseases that present with pruritis includes external parasites such as Notoedres, fleas, lice, Otodectes, and Cheyletiella. Also included would be food allergy, psychogenic hyperesthesia and alopecia, flea allergy dermatitis and drug eruption. A good topical treatment coupled with good environmental control and good management will take care of most, if not all, parasitic disease conditions.

The first parasite to be discussed is Notoedric mange. This is caused by a mite and usually involves the head and neck. Lesions appear scabby with crust formations, appearing very similar to sarcoptic mange, as it is seen in dogs. Fortunately, when diagnosis is made by skin scraping and microscopic examination, treatment is straightforward and only involves weekly lime sulfur dips. It is necessary to clean up the environment and to treat all in contact animals, as well as those affected. Human infestation and lesions may occur. A physician should be consulted.

Lice are highly host-specific. Therefore, lice are not transmissible from cats to other species, including man. In cats, signs of lice can vary from no signs whatsoever to hair loss, mild to severe pruritis, to generalized symptoms in severe cases, such as anemia, weight loss, and irritability. The parasite is usually found on the dorsal aspect of the animal. As with the Notoedres, good topical treatment with lime sulfur at weekly intervals for 3-4 weeks coupled with environmental treatment is usually adequate. Other areas to consider in lice control would be correcting predisposing factors such as poor nutrition, poor grooming, overcrowding, filth, cold, and debilitation.

Fleas, like lice, can be seen on the animal without the aid of a microscope. Unlike lice, however, fleas know no species limitations in animals and man. Therefore, methods of flea management in the cat are also applicable to the dog. Fleas spend a lot of time off the animal and can persist in the environment (rugs, chairs, etc.) for long periods of time (4 months to 2 years); therefore environmental control is a must! Fleas are small, dark brown creatures about the size of a gnat. The signs they can produce on the cat vary with the sensitivity of the individual cat. Some show mild irritation; some show a mild allergic dermatitis; while others may show a severe case of self-destruction, perhaps due to the bite from

only one flea. This would be an example of severe hypersensitivity to flea bites and the associated flea saliva. The scabs and severe irritation due to fleas tend to occur along the dorsum (miliary dermatitis), around the tail, and around the head and neck. In severe infestations the whole body can be involved. Diagnosis of fleas is usually obvious, but in cases of severe hypersensitivity combined with mild infestations, definitive diagnosis may be a challenge. Finding a flea or two amongst all the cat's hairs may be like finding the proverbial needle in a haystack. There is, however, usually some evidence of fleas, namely the characteristic flea dirt deposited on the cat. This can be helpful in diagnosis. The flea dirt is the excretion of the flea and looks like dirt scattered over the animal and found directly on the skin. If there is flea dirt present, then there is at least one flea somewhere.

Treatment and control of fleas can be difficult and is definitely long-term. Flea control on the animal must be practiced at least once weekly for up to 4-6 weeks at a minimum. Flea control includes flea shampoos, flea powders, or flea dips. In dusting or shampooing the cat, it is important to avoid products containing lindane or others specifically formulated for dogs. Also products available through a veterinarian usually have greater effectiveness than over-the-counter medications. It is important to treat all contact animals as well as the infected cat; otherwise, as one animal is treated, the fleas will feed upon another animal. Reinfestation of the originally infected cat can occur once therapy is stopped. Treatment must also include the environment. The area must be vacuumed thoroughly to remove fleas, flea larva and eggs. After vacuuming, flea powder may be vacuumed into the vacuum bag. The vacuum bag should be changed at least weekly. Commercial foggers should be considered, although they can be costly. Extermination is another alternative. In flea allergy dermatitis, one may have to resort to steroids to decrease the redness, pain, and scratching while attempts are being made to rid the cat, contacts and environment of fleas. If the lesions are particularly severe or are unmanageable medically, desensitization therapy may be of benefit. Fleas are probably one of the most common ailments of the skin and can be difficult to eliminate. But, with effort, flea control is an attainable goal.

Ear mites (Otodectes) are also a common problem in general small animal practice. Fortunately they are usually reasonable to treat. As the name implies, the condition is caused by a mite. Signs seen with infestation include head shaking, ear scratching, pain, head tilt, and odor (especially if infected). The mites usually infect both ears. They produce a characteristic dark black discharge. Diagnosis is best made by otoscopic exam, although sometimes examination of ear swabs on black paper can demonstrate the small mites. Treatment includes cleaning the ear thoroughly and medicating the ear with a good miticidal preparation. There are many products on the market which are effective. Just as important is to treat the whole animal with a good flea powder at weekly intervals for 4 weeks to kill the mites which have crawled out of the ears and which can reinfect the animals even after treatment is stopped. It is also important to treat all of the contact animals.

The last parasite which can affect the skin is Cheyletiella, or "walking dandruff." Cheyletiella can also infest humans. Signs vary from

asymptomatic carriers to cats showing pruritis with no lesions, to cats showing seborrhea or severe excoriation. Diagnosis is easily made by skin scraping, though it may take several scrapings to find the mite. Occasionally one can see the mites crawling around, hence the name, "walking dandruff." Treatment involves a good dip or shampoo weekly for 4 weeks while treating the environment and contact animals at the same time. Like Notoedres, human lesions clear up in 3 weeks.

Another cause of pruritis in cats is food allergy. It has been estimated that up to 10% of the allergies in dogs and cats are due to food. The problem does not have to be related to anything new in the diet. A substantial proportion of animals affected by food allergy have been on the same food without change for up to 2 years. In cats, the signs can vary from a miliary dermatitis to a pruritic dermatitis of the head and neck which may or may not be accompanied by gastrointestinal signs. Diagnosis of the particular allergen can be difficult and includes putting an animal on a boiled chicken and rice diet, clearing up the lesions, and then gradually adding back ingredients one at a time. The regimen is easier to describe than to implement. Steroids appear to be of little benefit in food allergies. Food allergy can be very difficult to diagnose and treat in humans, and perhaps even more so in cats.

Drug eruptions are just as difficult to deal with as food allergies. Drug eruptions can be reactions to any drug. It does not matter whether the drug is given orally, by injection, by inhalation, or topically. The eruption can mimic any other skin disease. It also can occur any time during the course of treatment by the drug. Treatment involves (obviously) stopping the drug and symptomatic treatment for the lesions.

The last disease to be discussed which can produce pruritis, feline psychogenic alopecia and hyperesthesia, has already been discussed in the alopecia section. The presenting case will be more severe than the simple alopecia case, due to constant licking and/or scabbing, leading to hair loss, redness, ulceration, oozing of serum, and crusting. It is usually a single lesion and can occur anywhere on the body. Again, phenobarbital is necessary to "quiet" the cat.

DISEASES PRODUCING CRUSTS

These are a group of diseases that present usually as scabs all over the body. A few of these diseases, such as miliary dermatitis, dermatophytosis, and Notoedres, have been mentioned previously.

Feline acne can occur in any cat and is thought to be due to poor grooming habits. It occurs under the chin. Inability to wash may be a more accurate cause. Papules, blackhead-like lesions, and pustules are seen. Occasionally the lower lip becomes very swollen and may abcess, due to bacterial infection. Treatment involves cleansing the area with a good cleanser and antibiotics. Once cleared, the chin may have to be cleansed periodically (weekly?) for the duration of the life of the cat. A large number of cases are recurrent or persistent. In general, however, the disease can be controlled.

DISEASES PRODUCING ULCERS AND FISTULAS

There are many diseases that produce ulcers and fistulas or draining tracts. The few to be discussed here are the eosinophilic granuloma complex, nodular panniculitis, pemphigus, and systemic lupus erythematosus.

The eosinophilic granuloma complex (EGC) is a complex of diseases of unknown etiology which can occur in any age, breed, or sex. One form of EGC is the eosinophilic ulcer. It is usually an erosion or ulcer occurring on the lips and nostrils, but can occur anywhere on the skin or in the mouth. The lesion can be very disfiguring if allowed to progress. The second form of EGC is an eosinophilic plaque, which is a red-yellow, very pruritic raised plaque, usually occuring on the abdomen and flank; but again, it can occur anywhere on the cat. The final form of EGC is the linear granuloma which is usually a raised, firm, linear lesion on the posterior thighs or along the Achilles tendon. Diagnosis of the EGC complex can be made by physical exam and skin biopsy. The linear granuloma form is self healing. Corticosteroid therapy is the treatment of choice for eosinophilic plaque and ulcer. These lesions may be recurrent. Fifty percent of cats with eosinophilic ulcers are FeLV positive.

The second disease, nodular panniculitis, is also of unknown etiology. It can present as a single or as many subcutaneous nodules all over the cat's body. Usually the condition is first diagnosed as cysts. These nodules will rupture and discharge. They can then progress to a fistula and heal up as a scar. Response to steroids is usually good.

Pemphigus is an auto-immune disease caused by the interaction of antibodies formed against the skin. Vesicles form which ulcerate, drain, and crust. The lesions are very severe and usually appear at the margins of the skin and mucous membranes, ears, eyes and head. Diagnosis is usually made with skin biopsies and immunological testing. Therapy at this time consists of high doses of corticosteroids.

Systemic lupus erythematosis is a multi-system (skin, blood, kidney, joint, etc.) auto-immune disease with skin lesions characterized by vesicles, serum oozing and crusting, and by showing a positive antinuclear antibody test. Again, high dose steroid therapy helps to control the disease. Prognosis is poor for both pemphigus and systemic lupus erythematosus.

DISEASES PRODUCING TUMORS AND NODULES

There are many types of tumors and nodules that can be seen in the cat. These include squamous cell carcinoma, basal cell tumors, mastocytosis, epidermal cysts, fibromas, fibrosarcoma and adenocarcinomas, to name a few. All produce a swelling in the skin. Tumors or nodules can only be accurately diagnosed and treated through skin biopsy and/or surgical removal. Discussion of the individual types of cutaneous masses, therefore, will not be undertaken here. The prognosis obviously varies with the type of nodule involved, where it is located, and how successfully it is surgically removed.

DISEASES PRODUCING MISCELLANEOUS CONDITIONS

Several miscellaneous conditions are mentioned here for completeness. These are stud tail, solar dermatitis, and Ehlers-Danlos Syndrome. Stud tail is a disease seen in sexually active cats of any age. The cause is unknown, but what is seen is comedones (backheads) and seborrheic accumulations on the dorsum of the base of the tail. This is an area analagous to the preen gland of birds. The only "effective" control of this blemish is shampooing to control.

Solar dermatitis is photosensitization seen in any breed, sex, or age of cat. It is especially common in white cats. The condition starts off as a redness, or scaling, progressing to edema and pruritis, leading eventually to ulceration and crusting. Persistent ulceration is very suggestive of transformation of a solar dermatitis condition to a squamous cell carcinoma. Treatment varies from avoidance of sunlight and use of sunblocking creams to amputation of the ear in carcinoma cases.

Ehlers-Danlos Syndrome, or stretching skin disease, is a hereditary (autosomal dominant) connective tissue disease producing very fragile skin and blood vessels. The skin becomes hyperelastic and tears very easily. Treatment consists of avoidance of trauma, and avoiding that breeding line.

CONCLUSION

The purpose of this paper was not to cover all aspects of feline dermatology, but rather to discuss briefly some of the more common skin diseases of the cat. Many less common diseases have not been mentioned. Diagnosis, at times, can be difficult; even when diagnosis is made, correct treatment may be difficult to impossible to maintain. In difficult cases ancillary tests, such as skin biopsy and laboratory work, are necessary to diagnose a condition and to monitor therapy.

SELECTED TOPICS IN FELINE INTERNAL MEDICINE

Robert M. DuFort, D.V.M.

Cats are unique creatures. Although they have diseases also seen in other species, the manifestation or treatment of these diseases in cats is often different. Two examples would be diabetes mellitus and cystitis. Cats also have some diseases that are rarely seen in other small animal species, such as hyperthyroidism. Many diseases, like hyperthyroidism, are of recent discovery, while others have been known for many years. Our knowledge of feline diseases varies greatly in its depth. In general, those diseases also seen in man are best understood.

This discussion will cover some diseases commonly seen in the cat as well as a few diseases that are unique to the cat or are of recent discovery. The talk will be organized by organ systems, i.e. gastrointestinal, cardiovascular, etc. Clinical signs (the observable manifestations of the disease) will be described for each body system so that you may be better able to recognize a diseased animal. The goal of this discussion is not to make you a veterinarian, but to help you recognize a sick animal and understand the disease once the diagnosis is made. Hopefully then we can work together to take better care of our feline friends.

DISEASES OF THE NERVOUS SYSTEM

I. Description
   - A. central nervous system
   - B. peripheral nervous system
   - C. special sense organs

II. Signs of disease of the nervous system
   - A. altered consciousness (depression, stupor, comatose)
   - B. balance problems (falling, circling, rolling, head tilt)
   - C. gait abnormalities (limping, dragging limb)
   - D. behavioral changes (aggression, affection, anxiety, withdrawal)

Feline Vestibular Disease

Feline vestibular disease is an unusual disturbance of the cat's vestibular system. This system includes the inner ear, whose function is proper balance and orientation, and its nerve supply. With vestibular disease, the cat's head is tilted to one side, the eyes move back and forth very rapidly, walking is unsteady and awkward. When walking, the cat drifts to the side on which the head is tilted.

The disease occurs most frequently during the late summer and fall, and its onset is very sudden. At this time, the cause of the disease is not known, but recovery can be expected. Since other conditions such as

severe ear infection or brain injury may cause similar signs, various blood tests and x-ray pictures may be necessary to establish the diagnosis.

Home Care

1. Medication: Medication must be fitted to your pet's particular needs.

2. Diet: Ordinarily, no special diet is required.

3. Activity: It is essential that your pet be kept indoors while his ability to move is impaired. Allowing him to go outdoors could result in severe injury from automobiles, dogs, etc. Confine your pet to an area of the house where he will be safe from stairs and stairwells, open windows, high ledges, etc.

DISEASES OF THE CARDIOVASCULAR SYSTEM

I. Description
   A. The cardiovascular system includes the heart and blood vessels.

II. Signs of disease of the cardiovascular system
   A. weakness, lethargy, collapse, depression, lack of appetite
   B. difficulty breathing, open mouth breathing, rapid breathing
   C. rapid heart rate, pale gums, low body temperature, weak pulse

Congestive Heart Failure

Chronic congestive heart failure is a type of heart trouble in which the heart does not pump enough blood to take care of normal body needs.

Pets with congestive heart disease tire easily, have shortness of breath, and a deep cough due to poor circulation through the lungs. They may lose weight but have an enlarged abdomen due to accumulation of fluid (dropsy). The legs may be swollen and puffy. Often these patients faint or collapse after excitement or exertion, and the tongue appears to be a bluish gray in color.

Congestive heart disease can result from heart-valve disease acquired during life, from heartworms or from congenital heart defects present at birth. Although congestive heart disease cannot be cured, many patients can live a comfortable life with proper medical management.

Aortic Embolism

Aortic embolism means a blood clot in the aorta, the major artery that carries blood from the heart to the smaller vessels that supply the body. In this case, the clot is toward the rear of the body where the aorta divides to supply the rear legs. The blood supply to one or both of the legs is greatly reduced depending on just how far back the clot is.

Signs of aortic embolism are extreme depression, sudden pain and coldness and paralysis of one or both rear legs. The exact cause of the clot formation is unknown, but it may be related to a previous infection in the body.

Heartworm Disease

Heartworm disease is becoming increasingly more common in many parts of the United States. It is caused by the heartworm, Dirofilaria immitis. This parasite lives in the right side of the cat's heart and in the nearby large blood vessels. The female worm produces large numbers of microscopic immature heartworms which circulate in the blood. These young worms, called microfilaria, are ingested by a mosquito feeding on an infected cat. After living in the mosquito for 10 to 14 days, the microfilaria are capable of infecting another cat when the mosquito feeds. They are injected under the cat's skin by the mosquito where they eventually reach the heart and develop into adult worms. It takes about 6 months for the worms to become adults after they are injected by the mosquito.

Diagnosis is made by finding the microfilaria in a blood sample. It will be 6 or 7 months after exposure before microfilaria can be found in the blood. Delayed treatment may result in heart failure and/or permanent damage to the liver and kidneys with eventual death.

Important Points in Treatment

Treatment for heartworms consists of 2 phases: (1) destruction of the adult heartworm and (2) elimination of microfilaria from the blood. Destruction of the adult heartworm is accomplished by intravenous medication given at the hospital. Some degree of risk is always involved, so a thorough physical examination, chest x-ray examination and various blood tests are usually necessary. Microfilaria are eliminated 6 weeks after the initial treatment with an oral medication.

Autoimmune Hemolytic Anemia

Your cat's natural body defense system is probably its greatest asset. This system is responsible for the production of antibodies which help the animal to destroy disease-producing agents such as bacterias and viruses.

In autoimmune hemolytic anemia, the defense system goes completely astray and produces antibodies which are active against the animal's own red blood cells. This results in the destruction of the red blood cells, severely threatening your pet's life. At this time, there is no known cause for this disease.

DISEASES OF THE RESPIRATORY TRACT

I. Description
   A. Upper airways (nose, pharynx, larynx, trachea)
   B. Lower airways (main bronchi, bronchioles, alveoli)

II. Signs of disease of the respiratory system (some in common with cardiovascular)
   A. Difficult breathing (inspiratory, expiratory, both), gasping, open mouth breathing
   B. Lack of appetite, weight loss, depression
   C. Fractious, cyanosis

Feline Bronchial Asthma

Asthma is a condition caused by constriction of the small air passages in the lungs. Affected cats suffer episodes of extremely difficult breathing, and many have periods of severe cough. Although they are bright and alert, they usually avoid exertion since this will cause further distress.

The exact cause is not known, but asthma is believed to be a manifestation of allergy. A few cases have been found to be caused by inhaling dust from cat-box litter. Recurrent attacks of asthma are to be expected and generally are impossible to predict. Many months may pass between attacks.

Important Points in Treatment

Mild cases of bronchial asthma are treated medically at home while the more severe cases require hospitalization and oxygen treatments for 24 to 48 hours. Anti-inflammatory drugs and medications to dilate the air passages are effective in treatment.

DISEASES OF THE URINARY TRACT

I. Description
   A. Upper urinary tract (kidneys, ureters)
   B. Lower urinary tract (bladder, urethra)

II. Signs of disease of the urinary tract
   A. Change in urine character (blood, pus)
   B. Changes in frequency or volume
   C. Straining
   D. Loss of voluntary control

Feline Urologic Syndrome (FUS)

For our purposes here, the term feline urologic syndrome or FUS will be used to refer to urinary obstruction in male cats. Although FUS is currently the subject of much study and research, the exact cause is

unknown. The influence of environment, heredity, diet, infections and stress are being investigated. FUS occurs in all breeds and ages, in castrated and noncastrated, in indoor and outdoor cats and in those fed a variety of diets. It is one of the most serious and distressing cat diseases confronting cat owners and veterinarians.

Cats with FUS cannot pass their urine because of sand or mucus plugs in the urinary canal, generally near the end of the penis. If the obstruction is not relieved promptly, affected cats rapidly become very sick and die. Early signs of trouble are: (a) irritability and restlessness, (b) blood in the urine, (c) frequent trips to the litter box, (d) straining while in a squatting position (often mistaken for constipation) and (e) urinating in unaccustomed places about the house. ANY ONE OF THESE SIGNS SHOULD ALERT YOU TO THE POSSIBILITY OF FUS. Cats with more advanced FUS may emit deep cries of pain, strain constantly, have an enlarged tender abdomen and vomit and drool.

Important Points in Treatment

The presence of FUS is an emergency situation, and prompt treatment is essential. Treatment is directed at relieving the obstruction and controlling pain and spasm with medication.

With early treatment, many FUS patients respond well; however, recurrence within a few hours to a few months is common, and some patients die from complications of the disease. In chronic, recurring cases, surgery may be recommended to enlarge the urinary opening.

Chronic Interstitial Nephritis

Normal, healthy kidneys function as filters for the blood, removing wastes and excreting them in the urine. Kidneys that are permanently damaged by infection or inflammation lose some of their filtering ability, the patient becomes ill, and chronic interstitial nephritis then exists. Continual recirculation of waste in the blood causes vomiting, increased thirst, poor appetite, listlessness and bad breath.

A physical examination and blood and urine tests are used for diagnosis and also to help your doctor advise you about the care your pet will require. Although the disease is not curable, it is controllable. Many pets can live reasonably normal lives when properly managed in a cooperative effort between owner and veterinarian.

Important Points in Treatment

Treatment for chronic interstitial nephritis is directed at maintaining as much functional kidney tissue as possible. Part of the treatment may be done in the hospital. In addition, a balanced diet with a moderate quantity of high quality protein and an adequate vitamin content must be fed. The diet must be one which produces a minimum of undesirable waste products which must be filtered out by the kidneys.

DISEASES OF THE GASTROINTESTINAL TRACT

I. Description
   A. oral cavity, pharynx, esophagus, stomach, intestines
   B. accessory organs (liver, pancreas)

II. Signs of disease of the gastrointestinal tract
   A. lack of appetite, salivation, difficult swallowing
   B. vomition vs. regurgitation
   C. diarrhea, constipation
   D. weight loss, dehydration

Constipation

Constipation is the occurrence of infrequent or difficult bowel movements. Since the stool is delayed in passing through the colon, there is more time for water absorption. This results in dry, hard stools. This type of stool results in straining and painful elimination.

There are three types of causes of constipation: (1) diet, (2) obstruction and (3) abnormalities of the nervous system control of elimination. Within each category there are numerous causes of constipation.

Important Points in Treatment

Treatment for constipation consists in relief of the constipation with laxatives, enemas, or surgery and the prevention of recurrences. Depending upon the cause of your pet's constipation, hospitalization may or may not be necessary.

Foreign Bodies

The variety of non-food materials swallowed by dogs and cats is truly amazing. Foreign objects ranging from coins to articles of clothing have been found in the stomach and intestines of pets. Animals of any age may swallow foreign bodies, but puppies and kittens are the worst offenders. Specifically, a foreign body is any non-food material that remains in the stomach or intestine without necessarily obstructing the digestive flow. The main signs of foreign body ingestion are vomiting, abdominal discomfort and vague uneasiness. Diagnosis is made on the basis of history, physical examination, x-ray examination, and often exploratory surgery. Induced vomiting may retrieve foreign bodies, but surgical removal is usually necessary.

Intestinal Obstruction

Intestinal obstruction is partial or complete blockage of the normal movement of material through the intestine. Obstruction can be caused by lodged foreign objects, by tumors or by telescoping of the intestine. A paralysis of a segment of the intestine can also result in obstruction.

Signs of intestinal obstruction are vomiting, refusal to eat and abdominal pain. Patients become weak and dehydrated. Because the signs are vague, diagnosis may be difficult, and x-ray pictures are often necessary.

Obstruction is considered an emergency situation, and surgery is necessary. Supportive intravenous fluids may be given before and during surgery. Peritonitis, or infection in the abdominal cavity, can be a complication.

Diarrhea

The term diarrhea is used to describe the frequent passage of very fluid stools. It is not a disease; rather, it is the most common symptom of intestinal disorders in pet animals. There are many causes of diarrhea and various diagnostic tests are frequently required to determine the underlying cause. Hospitalization may or may not be necessary.

Enteritis

Enteritis is the inflammation of the intestinal tract due to various causes, including microorganisms, allergies, injuries, foreign matter (bones, wood, etc.), hypersensitivity, emotional upsets, and parasites (worms).

Important Points in Treatment

There are many causes of enteritis, and various laboratory tests may be necessary to determine the cause and also the most effective treatment for your pet's enteritis. In some cases, x-ray studies of the intestinal tract may be needed.

NUTRITION AND GASTROINTESTINAL DISORDERS

Pets with gastrointestinal disorders have special problems. They undoubtedly have been vomiting and have had diarrhea. They certainly need nutrients and fluids. Many foods, however, perpetuate the vomiting and diarrhea. The type of food needed at this time is bland, yet tasty enough to appeal to a pet with a depressed appetite. Not only should the food be bland, but it should be fed in small amounts several times a day.

Water should also be given in small quantities several times daily. In fact, ice cubes work well to supply fluids, yet prevent gulping of water and subsequent vomiting.

Examples of bland foods are cooked eggs, cottage cheese, cooked rice.

Dental Tartar In Dogs and Cats

Dental calculus (Tartar) is composed of various mineral salts, organic material, serum and food particles. In the early stages of accumulation, the material is soft, but it later becomes very hard and adheres to the teeth. Continual accumulation causes pressure and inflammation of the gums and eventually recession of the gum tissue, which loosens the teeth. The breath becomes very odorous and the mouth becomes a dangerous source of infection.

It is currently believed that some of the internal diseases of mature dogs and cats are the result of diseased teeth and gums.

Ascaridiasis (Roundworms)

Ascarids, or roundworms, are the most common intestinal parasite found in dogs and cats. The adult worm lives in the intestinal tract and deposits eggs within the intestines where they pass to the outside in the stool. Diagnosis is accomplished by examining a stool sample for roundworm eggs under the microscope.

Life Cycle

Cats become infected by eating larvae (immature worms) from contaminated soil or feces or by eating infected rodents, birds or some insects. Kittens may be infected by their mother while still developing in the uterus. Consequently, many young animals are born with roundworms.

Ingested larvae travel through the body to the intestine and develop into mature worms. There, adult worms begin to deposit eggs in the intestines. The eggs pass with the stool and develop into infective larvae and the cycle is complete.

Public Health Significance

Human infestation with the larval stage of roundworms is possible but does not occur frequently. Eating contaminated soil or stool material is required for infestation to occur. Children should be instructed concerning the importance of cleanliness when playing with or handling animals. The best insurance against human contamination is keeping your pet free from roundworms by regular periodic stool examination and treatment, if indicated.

Control Measures

1. Stool specimens should be examined microscopically at regular intervals. Your doctor will advise you regarding the correct time to do this.

2. Good sanitation procedures are essential. This includes prompt removal of stools and regular cleaning of any area in which your pet is confined.

3. Eggs can remain infective in soil for years, so any contaminated ground becomes a source of reinfestation. This is particularly true of runs or areas where your pet may be confined. Control measures for these areas are the following.

   a. Soil should be turned over to a depth of 8-12 inches after your pet is free from worms.
   b. Replacing dirt runs with concrete seems to be the best procedure. Stools should be removed daily.
   c. Moving your pet to a different area may be successful.

Cuterebra Larvae

Cuterebra is a group of flies whose larval (small worm) stage of development may infest the skin of rodents, squirrels, rabbits, dogs and cats. The small fly may lay her eggs on the fur or in the immediate area of your pet. After the eggs hatch, the small larvae burrow into the skin and a small abscess appears characterized by a small hole in the center in which the larva's head can be seen. After about one month, they emerge and drop to the ground to continue their life cycle.

Important Points in Treatment

1. A general anesthetic may be required for removal of the larvae and cleaning of the wound.

2. The larva should not be crushed while still embedded in the skin because a serious shocklike reaction could occur.

3. Bacterial infections accompanying the embedded larva constitute a need for further medical treatment after the larva is removed.

Tapeworm

The tapeworm is a parasitic worm found in the intestines of cats. It consists of a head (scolex) attached to the lining of the intestine and a long body made up of segments called proglottids. The segments may be shed and passed in the feces, leaving the head still attached to produce new segments.

Tapeworm infection may not be noticeable, or it can produce digestive upsets, variation in appetite, poor hair coat and skin, weight loss and vague signs of abdominal discomfort.

Diagnosis of tapeworms is made by finding the segments in your pet's feces, in his bed or clinging to hair around the anal area. Unlike most

other worms, the eggs are not generally found in a microscopic examination of the feces. When first passed, segments will be yellowish to white, about 1/4 inch long and may expand and contract. When dry, segments resemble cucumber seeds or grains of rice.

Tapeworms are not passed directly from pet to pet, but require another animal called an intermediate host in which to develop. Common intermediate hosts are fleas and small rodents such as mice, rats, squirrels and rabbits. In some areas, fish are the intermediate host for one type of tapeworm.

Important Points in Treatment

Treatment must destroy the tapeworms already infecting your pet and control reinfection by eliminating or reducing contact with the intermediate hosts. Several treatments may be necessary to eliminate tapeworms. Your veterinarian may treat your pet on either an inpatient or outpatient basis, depending on his professional judgment.

Coccidiosis

Coccidiosis is a parasitic disease of the intestinal tract caused by a small organism (protozoa) visible only with the aid of a microscope. The disease generally spreads from one animal to another by contact with feces (stool) from infected individuals. It is more severe in younger or weakened animals and usually causes bloody diarrhea. There is no definite evidence that human coccidiosis is acquired from cats.

Important Points in Treatment

1. Clean and dry bedding should be provided.
2. Provide a high quality, balanced diet.
3. Keep infected animals away from noninfected animals.

FELINE INFECTIOUS ANEMIA (Hemobartonella felis)

Feline infectious anemia is a blood diseae of cats in which a microscopic parasite affects the red blood cells.

The disease can be very mild with no obvious signs of illness, or it can be extremely severe with fever, weakness, emaciation, loss of appetite, pale membranes and occasional yellow jaundice. High fevers occur early in the disease, later falling to below normal.

Feline infectious anemia is transmitted to healthy cats by the bite of a blood-sucking insect such as the flea. It may also be possible for kittens to become infected in utero (before birth), but this is not definitely known. Stress such as other illnesses or injury can trigger the onset of the disease in mildly affected cats. Recurrence of feline infectious anemia is fairly common, even after treatment. Recovered cats

can be carriers; therefore, control of external parasites such as fleas and lice is necessary to prevent the spread of infection to other cats.

FELINE NEOPLASIA (Cancer)

I. Description
   A. Neoplasia or cancer is an uncontrolled growth of a particular cell type of the body. There are potentially as many types of cancer as there are types of cells in the body. Cancers are named after their cell type of origin. As an example, the cancer lymphosarcoma is derived from a cell called lymphocytes.

II. Signs of Cancer
   A. Specific signs will depend upon the organ system affected.
   B. General signs include: depression, malaise, weight loss, inappetence, fever.

Malignant Lymphoma

Malignant lymphoma is a type of cancer of the lymphatic system. Several different types are included under this one name. The disease may involve certain blood cells only, or there may be noticeable glandular enlargement. The disease is progressive, and is fatal after an undetermined and variable period of time.

Mammary Cancer

Mammary cancer is fairly uncommon in the cat. Unfortunately, almost always it is malignant (has a high potential to spread to distant sites in the body). Mammary cancer must be distinguished from normal lactating glands and mastitis (infection of inflammation of the gland).

DISEASES OF THE ENDOCRINE ORGANS

Endocrine organs are very small glands located in specific sites in the body. They secrete hormones (small protein substances) that have specific actions on other tissues at different sites.

Diabetes Mellitus

Diabetes mellitus is a disease caused by an insulin deficiency. Insulin is a hormone produced by special cells of the pancreas and is necessary to convert blood sugar into a more useful form for body tissues to utilize. Without insulin replacement, further serious changes occur within the body that lead to diabetic coma and death. There is no known cure. Therefore, control of diabetes mellitus will be the prime objective.

Hyperthyroidism

Hyperthyroidism is a disease caused by overproduction of thyroid hormone (thyroxin). It is usually caused by a small benign tumor of the thyroid gland. Treatment is surgical removal of the tumor. Medical treatments are being tried.

INTOXICATION (POISONING)

Ethylene Glycol Poisoning

Ethylene glycol is used as an automotive anti-freeze. If it is left uncovered or drained on the floor, cats often drink it because of the sweet taste. Dangerous and often fatal poisoning results.

Prompt treatment by your veterinarian is essential, or else ethylene glycol may cause permanent kidney damage. In spite of treatment, many pets still may die from the poisoning. Though seldom possible, the ideal situation is to begin treatment before symptoms occur.

Warfarin Poisoning

Warfarin is used as a poison for rodents. It functions by interfering with the clotting ability of the animal and results in excessive, fatal bleeding. Since the poison is in the form of a bait, cats may consume some of the drug. They may also eat a rodent that has been poisoned with warfarin.

Although many pets may eat a small amount of the bait, actual poisoning is not common. Poisoning in pets requires several exposures to the poison over a period of time. Only then will absorption of the poison be sufficient to affect the clotting ability of the blood.

A pet that has been poisoned by warfarin will show signs of bleeding. There will be a bloody diarrhea, breathing will be labored, and your pet will become very depressed. Your veterinarian will attempt to reverse the signs of the poisoning by blood transfusions and administration of drugs to improve clotting.

Strychnine Poisoning

Strychnine sulfate is a powerful poison used in the control of predators, rodents, pigeons and ground moles. It is often placed in bait such as meat, cereal, grain and peanuts, accounting for the frequent accidental poisoning of pets. Deliberate strychnine poisonings also occur. Poisoned peanuts buried in the ground keep their potency indefinitely and are extremely hazardous to pets and children.

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FELINE RESPIRATORY DISEASES

Fredric W. Scott, D.V.M., Ph.D.

Originally, all infectious feline respiratory diseases were thought to be "pneumonitis" caused by Chlamydia psittaci. Extensive research in several countries has shown that C. psittaci is not the main cause of respiratory disease of cats but in fact there are numerous agents that produce clinical diseases that may be indistinguishable from one another. Feline viral rhinotracheitis and feline calicivirus infection are approximately equal in incidence and account for the majority of feline respiratory diseases. It is important to realize that multiple infection can occur in the same cat; thus, an agent that normally does not produce clinical disease will exacerbate an infection caused by another agent of respiratory disease.

CAUSES

Feline viral rhinotracheitis (FVR, "Rhino", feline herpes). Feline viral rhinotracheitis is an acute respiratory disease of cats caused by feline herpesvirus-1, a DNA virus that is quite labile, being sensitive to acid, heat, and most disinfectants. FVR virus will survive for only 18 to 24 hours at room temperature. There is only one FVR virus; all isolates of FVR virus that have been studied belong to a single serotype.

Feline calicivirus (picornavirus) infection (FCI). Feline calicivirus infection is an acute respiratory infection of cats caused by one of a number of strains of calicivirus. This virus is a RNA virus similar to the human cold viruses. The calicivirus is more resistant than the herpesvirus, surviving for one or 2 weeks at room temperature.

Feline pneumonitis (FPn). This acute respiratory disease is caused by the psittacoid agent, Chlamydia psittaci (Miyagawanella felis, Bedsonia felis). It is labile, being sensitive to heat, ether, acids, and broad spectrum antibiotics. There may be two or more serotypes of this agent, although this point is not entirely clear.

Reovirus infection. Reovirus infection is caused by feline reovirus, which is similar to reoviruses of many other species. The prefix "reo" stands for "Respiratory Enteric Orphan," indicating that this virus replicates in the respiratory and enteric tracts of various animals, often without producing disease. The virus is a resistant RNA virus, and more than one serotype may exist.

Chronic upper respiratory infection. Chronic, mild upper respiratory infection has become a relatively common problem, especially in catteries. While some of these infections are due to herpesvirus or calicivirus, the cause of many of the outbreaks remains unknown.

Feline infectious peritonitis (FIP). FIP is a severe, usually fatal generalized disease of cats caused by a coronavirus. In the initial infection, virus may enter through and replicate in the respiratory system. While FIP and feline leukemia are not normally included in the respiratory disease complex, one should be aware that these viruses may affect the respiratory system.

Mycoplasma infection. Mycoplasma can frequently be isolated from cats with respiratory disease, and probably play an important role as secondary invaders. These are labile organisms and may be susceptible to antibiotics.

Bacteria. Numerous bacteria may be involved as secondary invaders in respiratory diseases. The role of bacteria in feline respiratory disease has not been fully determined, but Bordetella appears to cause severe bronchopneumonia as a concurrent infection with some of the above viruses. It is not known whether or not this bacterium, or any of the numerous other bacteria that are common secondary invaders, can produce respiratory disease without initial infection by a virus.

SPREAD OF DISEASE

The route of viral infection is either oral or intranasal. The acute disease develops after an incubation period of from one to several days after infection, depending upon the severity of exposure and the virus involved. The clinical disease lasts from one to several days or may linger for weeks in some cases, again depending upon the severity of the infection and the virus involved.

Infected cats will discharge virus or other agents in the saliva, the nasal and ocular discharges, the feces (cats with calicivirus and reovirus infections), and the urine (cats with calicivirus infections). Infection of susceptible cats occurs by direct contact, by contact with cages, food dishes, water dishes, or litter pans that have been contaminated with virus or other agents, and by aerosol exposure from infected cats. These viruses may also be transmitted several feet by aerosol droplets. Hands, clothing, or shoes of persons handling and caring for infected cats frequently become contaminated and can serve as a vehicle for transmitting these respiratory disease-producing agents to susceptible cats.

An important aspect of the spread of the respiratory diseases of cats is the presence of a carrier state. After recovery from FVR cats will continue to shed herpesvirus intermittently from the oropharynx for many months. Cats infected with calicivirus have been shown to shed virus continuously from the throat and occasionally in the feces for long periods of time. Mother cats that had FVR or FCI as kittens may pass the virus to their young. Severe stress or another viral or bacterial infection may cause those animals carrying virus to become infectious to other cats.

METHOD OF DISEASE PRODUCTION

Oral, ocular, or intranasal infection results in a local infection of the epithelium of these regions, which then spreads to involve the remainder of upper respiratory epithelial cells; the infection may even spread to the lungs. The infection generally remains a superficial infection but may spread through the blood to produce a generalized infection (viremia). Feline viral rhinotracheitis generally does not produce a viremia; but in certain instances viremia may occur, and infection of the osteoblasts (bone-forming cells) may result. Viremia in pregnant cats may result in infection of the fetuses and abortion.

Although the clinical disease in feline calicivirus infections is often milder than FVR, the spread of virus is generally greater. Infection occurs not only in the respiratory mucosa but may also occur in the intestine and the urinary tract. Severe pneumonia may occur in calicivirus infection, and ulcerations of the tongue and/or the hard palate are common.

Studies have shown that FVR infection may produce severe disease in germ-free cats. Thus, secondary bacterial infections are not necessary to produce the disease. In any respiratory infection, however, the disease may be complicated by secondary bacterial or other viral infections.

DIAGNOSIS

Feline respiratory disease is easily diagnosed, but the exact cause is extremely difficult to determine clinically. The diagnosis is made on the basis of clinical signs and can be confirmed by special laboratory tests.

Clinical signs. Several of the agents involved in the feline respiratory disease complex can produce signs that are essentially identical in any given case. This makes it extremely difficult to determine exactly which of the agents is the cause of the specific infection. For the most part, the agents can be determined only by laboratory tests.

Sneezing and coughing are the first clinical signs observed, especially in FVR, and are followed by a sensitivity of the eyes to light (squinting), red and swollen eyes, and a watery discharge from the eyes. Frequently, only one eye is involved initially, but involvement of the second eye occurs in a few hours. The eye discharge usually becomes thicker and contains pus. Infection of the nose frequently causes a runny nose and subsequently, in one or two days, a thick nasal discharge which may dry and form crusts. These crusts block the nostrils and force the cat to breathe through the mouth. Excess salivation may occur, especially if tongue ulcers are developing. The animal is usually depressed and may stop eating. There may be a fever, especially early in the infection.

There are some generalities that may help in determining the agent involved. FVR usually is a severe infection, especially in young kittens. Ulcers of the eye may develop, followed only rarely by a severe infection of the entire eye and total blindness. Severe sneezing is usually indicative of FVR.

Calicivirus infections generally are milder than FVR. If pneumonia develops, however, the mortality may be high (30%), especially in young kittens in colonies or catteries. Ulcers of the tongue and palate generally can be attributed to calicivirus, and pneumonia is most likely due to calicivirus.

Reovirus infection is mild, with signs usually restricted to a mild eye infection.

Chronic upper respiratory infection usually presents as intermittent mild ocular and/or nasal watery discharge which persists for months to years.

Laboratory tests. Diagnosis can be confirmed by viral isolation in the laboratory. Pharyngeal, ocular, or nasal swabs may be submitted to a laboratory where the swabs are placed in cell cultures or embryonated eggs for viral isolation. In most cases, the pharyngeal swab produces the best chance of viral isolation. Unfortunately, few diagnostic laboratories are equipped to run these viral isolation tests.

TREATMENT

Treatment for feline respiratory disease is for the most part symptomatic. Broad-spectrum antibiotics are indicated to prevent secondary bacterial infections. In pneumonitis, broad-spectrum antibiotics are specifically indicated since this agent is susceptible to antibiotics. With chronic upper respiratory infection, broad spectrum antibiotics usually result in transient improvement but not a cure. Eye ointments containing antibiotics are indicated for relief of the conjunctivitis. The routine use of ointments containing corticosteroids, however, is contraindicated unless there is specific need. In human herpes infection there is definite evidence that corticosteroids may lead to the development of ulcerative keratitis, and there is some indication that a similar problem may exist with FVR.

With severe infections, fluids are indicated to overcome dehydration. Oxygen therapy is also indicated if the animal is severely distressed from lack of respiratory function. Systemic vitamin injections may be indicated, since many diseased cats have a low vitamin level, especially vitamins A and B. Some clinicians have indicated that vitamin C is valuable, while others dispute the value of vitamin C in treatment of respiratory disease. A controlled study showed that large doses of injectable ascorbic acid had no beneficial effects on preventing or reducing the severity of FVR.

Good nursing care is extremely important in treating respiratory diseases of the cat. It is important to clean the dry, crusted material from the nose in order to allow drainage of the nasal passages. A cat that cannot smell does not eat. Vaporization may be helpful in decreasing the swelling in the membranes and removing the nasal discharges. Baby foods are beneficial; cats tend to eat baby foods before they will eat regular cat foods.

There are some specific products that may be beneficial in treatment of herpes infection in the cat. Idoxuridine (IDU) and adenine arabinoside (Ara-A) are specific antiviral compounds that are beneficial in treatment of ocular herpesvirus infection. There are other specific antiviral agents being developed that may, in time, prove beneficial.

PREVENTION

Prevention of feline respiratory disease depends upon (1) identification and restriction of the source of virus (infected cats); (2) reduction of the concentration of virus in the environment; and (3) immunization of cats by vaccination.

Since the major source of infection is direct contact of susceptible and infected cats, any means of reducing or preventing direct contact of these cats will greatly reduce the chance of infection. The use of isolation or quarantine areas to house new cats, cats that are on the show circuit, or cats that have been sent away for mating is widely recognized. These cats should be isolated for a period of at least 2 weeks and observed for signs of illness. Even with this precaution, these animals may be chronic carriers of infection and may introduce infection when they enter the colony.

Because these viruses are also transmitted by aerosol, it is very important to have proper air-flow conditions within catteries. The humidity should be kept at a reasonably low level, and the ventilation should be good, with 10 air changes per hour within the room. Infected cats or cats that are starting to sneeze or cough should be immediately isolated in a distant corner of the room or preferably in a distinct isolation ward.

Many queens are carriers of virus and transfer this virus to the kittens after the kittens have lost their temporary protection acquired from their mother's milk. It may be beneficial to wean kittens at a relatively early age (4 to 5 weeks) when the kittens still have some protection from passive maternal antibodies. These kittens can be removed and raised away from the adults and thus break the line of transmission.

One should be constantly aware of the methods to prevent indirect spread of infectious agents. Dishes and other utensils that cats may come in contact with should be disinfected between uses. It is preferable to use disposable dishes. Persons handling or caring for infected cats should wash their hands between cats. If rubber gloves are worn, these can be disinfected and washed between cats. The weak link in any control measure is usually the people involved.

Household bleach (sodium hypochlorite, Clorox, diluted one to 30 or 4 oz. per gallon of water) is excellent for disinfection of food and water dishes, litter pans, cages, and floors.

The most effective way of preventing infection is immunization with vaccines. Extensive research in recent years has resulted in the development and marketing of several vaccines to protect cats against FVR and

FCI. These, in addition to the pneumonitis vaccine, provide considerable protection against the most important respiratory diseases.

The most effective way of preventing infection is immunization with vaccines. Extensive research in recent years has resulted in the development and marketing of several vaccines to protect cats against FVR and FCI. These, in addition to the pneumonitis vaccine, provide considerable protection against the most important respiratory diseases.

Numerous respiratory vaccines are available in a variety of combinations. Many are also combined with panleukopenia (enteritis, "distemper") and some also include rabies vaccine. The 3 basic types of respiratory vaccines are (1) modified-live-virus (MLV or attenuated) vaccines for injection, (2) inactivated or "killed" vaccines for injection, and (3) MLV vaccines for intranasal administration.

Controlled laboratory studies have shown that the injectable vaccines are safe and do not produce any signs of illness. The intranasal vaccines may cause the kitten to sneeze for a few days, and runny eyes or runny nose may occur briefly. A few kittens given this vaccine have developed tongue ulcers due to the calicivirus components. Reactions produced by the intranasal vaccine are mild and transient.

These vaccines provide reasonably good protection against severe infection. Vaccinated cats may still become infected but they usually do not show any signs of illness. Virus may replicate in small amounts in the superficial cells of the upper respiratory tract, which are protected from the antibodies or immunity of the blood stream. An occasional vaccinated cat (approximately 10 percent) may have a watery eye for one or two days after exposure to FVR virus, and some cats may sneeze a few times. These adverse reactions are mild compared to the severe disease normally seen with FVR.

Kittens should be vaccinated against FVR and FCI (and possibly pneumonitis, if it is a problem in that area) at 8 to 10 weeks of age and again 3 to 4 weeks later. These vaccinations can be given at the same time as those for panleukopenia. If problems with infection in younger kittens are encountered in a cattery, kittens should receive their first FVR-FCI vaccine at 4-5 weeks of age and their second vaccine at 8 to 9 weeks of age.

All cats should receive annual booster vaccinations for FVR and FCI. Pregnant cats should not be vaccinated with the MLV vaccines; rather, breeding queens should be vaccinated at least one month prior to breeding.

There is as yet no need for a vaccine for the remaining agents in the respiratory disease complex. The reovirus has not been shown to be a significant cause of disease in the cat. The information presently available indicates that mycoplasma and bacteria are secondary invaders and, as such, do not warrant the development of vaccines.

Chronic upper respiratory infection is a persistent problem in some catteries. Since the exact cause(s) of this condition has not been established, no vaccine is available.

SUMMARY

Feline viral rhinotracheitis (FVR) and feline caliciviral infection (FCI) are the two most important respiratory infections of cats, with pneumonitis, reovirus, mycoplasma, and bacterial infections of less significance. These agents are spread by direct contact of susceptible cats with infected cats, or by contact with cages, feed dishes, litter pans, or persons contaminated with infectious agents.

Feline respiratory disease is easily diagnosed, but the exact cause is extremely difficult to determine clinically. Laboratory tests may be required to establish the specific cause of the disease.

Prevention of infection is the key to reducing or eliminating feline respiratory disease. This can be done by (1) keeping infected cats away from susceptible cats; (2) eliminating the virus in the environment; and (3) establishing an immune population by vaccination.

All cats should be vaccinated against FVR and FCI, and possibly against pneumonitis, with one of the respiratory disease vaccines. Kittens should be vaccinated starting at 8-10 weeks of age (or possibly at 4-5 weeks of age if the risk of exposure is great). Cats should receive annual revaccinations.

FELINE PANLEUKOPENIA

Fredric W. Scott, D.V.M., Ph.D.

Feline panleukopenia (FP, feline parvovirus, feline infectious enteritis, feline "distemper," feline ataxia) is a highly contagious viral disease of cats characterized by sudden onset, fever, anorexia, depression, marked leukopenia (decreased white blood cell count), vomition, dehydration, and often a high mortality. All members of the cat family (Felidae) are susceptible to panleukopenia virus as well as the raccoon, coati mundi and ringtail in the family Procyonidae. The gastrointestinal disease of mink known as mink viral enteritis is caused by a virus antigenically identical to panleukopenia virus. An apparent mutation of FP virus resulted in canine parvovirus, a new and severe enteric disease of dogs. Intrauterine infection with FP virus may result in abortions, stillbirths, early neonatal deaths, cerebellar hypoplasia manifested by a symmetrical incoordination (feline ataxia) first seen at 2 to 3 weeks of age when the kittens begin to walk.

While numerous excellent vaccines are available to immunize cats against panleukopenia, the disease is the most severe and destructive disease of cats in unvaccinated populations.

ETIOLOGY

Feline panleukopenia virus (FPV) is a very small, stable DNA virus classified in the parvovirus group. The virus is resistant to ether, chloroform, acid, alcohol, and temperature (56° C for 30 minutes), but is susceptible to Clorox and the aldehydes (glutaraldehyde, formaldehyde). Replication of the virus is dependent upon cells that are in active mitosis or cell division. All strains of the virus that have been studied to date are antigenically identical.

EPIDEMIOLOGY

Feline panleukopenia is primarily a disease of young kittens; however, it can affect cats of all ages. The incidence, morbidity, and mortality may vary considerably from population to population and outbreak to outbreak. In susceptible populations, the incidence may approach 100%, whereas in other populations occasional cats will undergo disease during an outbreak.

Subclinical or very mild clinical infection occurs in certain outbreaks of FP. As many as 75% of the unvaccinated cats in the area of Ithaca, New York (home of the Cornell Feline Health Center) had circulating antibodies against FP, indicating previous infection with this virus. Many of these cats never exhibited any clinical disease. The incidence of antibodies against panleukopenia is much higher in cats over one year of age.

There is a seasonal incidence of FP which usually coincides with the development of a population of susceptible young kittens. In Northeastern United States this is during the summer and early fall, but the seasonal incidence may vary according to location. However, outbreaks of FP occur at any time of the year.

Transmission of the virus is usually by direct contact of susceptible cats with infected cats since during the early stages of the infection the virus is excreted in feces, urine, saliva, and vomitus. Mechanical transmission from infected animals to susceptible animals may occur via inanimate objects such as food dishes, bedding, infected cages, or by persons. Evidence suggests that certain recovered cats may shed virus in their feces and urine for considerable periods of time, and thus provide a source of infection. The virus has been demonstrated for several weeks in the kidney of recovered cats and neonatally infected cats may contain virus in their kidneys for periods of at least one year. Mink that have recovered from mink viral enteritis may shed infected virus in their feces for at least one year.

During the acute disease, fleas may transmit the disease from infected to susceptible cats. It seems likely that biting and flying insects may also transmit the disease.

The extreme stability of this virus necessitates that infected premises must be thoroughly cleaned and disinfected before introduction of susceptible cats. Once an infection with FPV occurs on a premise, infectious virus will remain for years on that property. New cats should be vaccinated at least two weeks prior to introduction to an infected premise.

PATHOGENESIS

In newborn, infected kittens nearly every tissue in the body contains virus. By 18 hours after infection the virus has established itself first in the oral pharynx (tonsils), and then via a viremia the virus is present in the thymus, heart, mesenteric lymph nodes, kidney, small intestine, and cerebellum. By 2 days after infection essentially every tissue has significant amounts of virus. High titers of virus remain in most tissues through 7 days after infection. As circulating antibodies appear, titers gradually decrease until 14 days when most tissues lose their virus. However, small quantities of virus may persist up to one year in certain tissues, but the strong immunity usually neutralizes the virus as it is shed so that most of these kittens are not infectious to other cats.

The most severely affected tissues in the newborn cat are those undergoing rapid mitosis, namely the thymus and the external granular layer of the cerebellum. The small intestine, which has a slow mitotic activity in the neonate, is not grossly or histologically involved, although virus is present. At approximately 9 days of age the cerebellum is no longer involved since the mitotic activity of its granular layer cells is reduced.

In older kittens the pathogenesis also depends upon the state of mitotic activity of the various tissues within the body. Virus enters via

the oral route and infection occurs primarily in the lymphoid tissues of the oral pharynx and the intestine. With 24 hours after ingesting virus the animal is viremic, and the virus is distributed throughout the body via the blood. The epithelial cells of the crypts of the intestine are severely involved as are the lymphoid tissues and bone marrow.

Virus persists in the blood of the infected cat until approximately 7 days after exposure (approximately the third day of illness) at which time circulating neutralizing antibodies appear. The antibody titer increases rapidly and reaches its maximum by about 14 days after exposure. With the appearance of antibody the virus in the various tissues gradually disappears. The virus may persist intracellularly where it is protected from antibodies for periods of several weeks in certain tissues such as the kidney.

If infection occurs in a pregnant cat the virus readily infects the uterus and crosses the placenta to infect the fetus. Infection then occurs throughout the fetus and crosses the blood brain barrier to infect the cerebellum and other tissues within the central nervous system. The congenital birth defects that result will depend upon the stage of gestation at the time of infection. Intrauterine infection may result in abortion, stillbirths, early neonatal deaths, cerebellar hypoplasia or other birth defects.

DIAGNOSIS

A presumptive diagnosis of FP can be made by the veterinarian on the basis of history, clinical signs and the presence of leukopenia. The diagnosis can be confirmed by pathological changes, viral isolations, by the demonstration of virus in infected tissues by immunofluorescence, or by the demonstration of an increase in circulating antibodies by testing paired serum samples.

The severity of clinical signs exhibited varies tremendously. Many cats undergo subclinical infection and the only method of diagnosis would be viral isolation or serology. Other cats may show a very mild clinical infection in which a slightly elevated temperature, a mild anorexia, and a mild leukopenia are observed. In the typical case of FP there is a sudden onset of clinical signs, the animal may have a temperature of 104° F or higher, and it may show severe depression and complete anorexia. Vomition usually occurs and severe diarrhea may develop in 24 to 48 hours. If the vomiting and diarrhea continue, severe dehydration and electrolyte imbalances occur.

Cats infected with FP often assume a typical posture, hunched with the head between the paws, and often hanging the head over a water dish or feed dish. They often act as if they would like to drink, and may even take a lap or two of milk or water, but are unable or reluctant to swallow. The hair coat becomes rough and dull and there is a loss of elasticity of the skin due to the dehydration. The third eyelid often appears prominent. The abdomen is painful and abdominal palpation elicits signs of pain. The mesenteric nodes are enlarged and the gastrointestinal tract contains excess gas and liquid.

Terminally, a subnormal temperature is observed indicating a grave prognosis. Coma and death usually occur in a few hours.

The mortality may vary from 25 to 75% in various outbreaks of FP. Death may be acute without previous signs (the owner often suspecting poisoning), or death may occur within the first five days of illness in uncomplicated cases, or after 5 days in complicated cases. If the animal survives approximately 5 days of illness and secondary complications such as bacterial infections, dehydration, or chronic enteritis do not occur, then recovery should be fairly rapid. It usually takes several weeks for the animal to regain its lost weight and condition.

The clinical signs observed in kittens infected in utero or neonatally are either sudden death without any particular signs, or the development of ataxia at about 2 weeks of age when the kittens begin walking. The ataxia is a symmetrical incoordination, exemplified by rolling or tumbling as the cat tries to walk, by an involuntary twitching of the head, or by swaying of the body. These ataxic kittens are alert and strong. If they are coordinated enough to obtain food they will survive, but the ataxia will persist throughout life with little, if any, improvement or compensation in the ataxia as the animal becomes older.

The most characteristic finding in FP is the leukopenia (low white blood cell count). This is present in almost all infected cats, even if they do not show clinical signs. There is usually a direct correlation between the severity of the leukopenia and the severity of the disease. A progressive drop in circulating leukocytes occurs 1 to 2 days prior to the development of clinical signs with a precipitious drop on the day of the crisis. The leukocyte count usually is less than 4,000 in clinically ill cats and may be less than 1,000. Due to the extreme reduction in neutrophils, a relative lymphocytosis may occur, but as the disease progresses the lymphocytes may disappear also. A count of 100 to 200 leukocytes per cubic millimeter of blood is not unusual. If the cat survives for approximately 5 days after the onset of signs there is a marked rebound in the leukocyte count, often exceeding the upper normal limit of approximately 24,000 in another 3 to 4 days. This leukocytosis is a neutrophilia with a shift to the left.

For viral isolation, swabs can be taken by the veterinarian from the pharynx or the rectum and placed in transport medium, frozen, and submitted to a diagnostic laboratory that is equipped to do feline viral isolations. The best tissues to submit for viral isolation from necropsied animals are spleen, thymus, ileum, or mesenteric lymph node. These samples should be placed in sterile vials and either transmitted directly to the laboratory or frozen and submitted under dry ice refrigeration. For immunofluorescence, tissues from necropsied animals should be snap frozen in liquid nitrogen and submitted for sectioning and staining. Impression smears can also be taken of the spleen or mesenteric lymph node, fixed in cold acetone, and the dried slide then submitted to the diagnostic laboratory.

For the serologic diagnosis of FP paired serum samples are required. One blood specimen is taken during the acute phase of the disease and a second sample taken 2 weeks later. The serum samples are frozen until

submitted to the laboratory but they may be shipped by regular mail without refrigeration. Paired serum samples are required since results of a single sample are meaningless in establishing a diagnosis.

TREATMENT

Panleukopenia normally has a high mortality but with diligent treatment and good nursing care the mortality rate can be reduced. The main object in treatment is to keep the animal alive and in reasonably good health until the natural defenses take over; that is, the rebound in leukocytes and the appearance of antibody. Serum antibodies usually appear about 3 to 4 days after the first signs of the disease, and 2 to 3 days later a sharp rebound in total leukocytes occur. If the animal can be maintained without complications for 5 to 7 days the chances of recovery are usually good.

More specifically, the object of treatment is to correct and prevent the symptomatic abnormalities such as vomiting, diarrhea, and dehydration, and secondly to prevent secondary bacterial infections. The only specific treatment that is available is the use of antiserum, and there are conflicting opinions as to its benefit once clinical signs have appeared. Some clinicians feel that early in the disease the use of antiserum is beneficial.

The most important aspect of treatment is maintenance of fluid and electrolyte balances. To prevent the loss of fluids, drugs to counteract the vomiting and diarrhea are indicated. For replacement of fluids, 5% dextrose in saline, or 5% dextrose in an electrolyte replacer solution formulated for replacement of electrolytes lost in vomiting and diarrhea, is beneficial. Whole blood is also excellent.

Broad spectrum antibiotics are used in all cases since secondary bacterial infections are common. These antibiotics are administered by injection until the gastroenteritis is controlled. Then they may be administered orally for a total of 5 days.

Other supportive therapy should be administered as indicated, such as liver injections or B vitamins. Cats should be kept warm and not be placed in a drafty location during recovery.

All food should be withheld until the gastroenteritis is controlled, then strained baby foods can be fed in small quantities several times a day.

Secondary viral respiratory infections are common complications of FP. The FP infection may trigger a latent respiratory virus infection such as feline viral rhinotracheitis or feline calicivirus infection. This dual viral infection is more severe than if either of the viruses had infected the cat alone. The secondary viral infections should be treated as indicated.

There is no treatment that is beneficial for cats showing ataxia.

PREVENTION

There are several excellent vaccines that are available to immunize cats against FP. These vaccines are effective and produce long-lasting immunity. Since this disease is entirely preventable through proper immunization, one cannot overemphasize the importance of proper vaccination. Considerable information has been published concerning the evaluation of the types of vaccines and various vaccination recommendations. Immunization against panleukopenia should be started at 8 to 10 weeks of age using tissue culture origin vaccines, either inactivated or modified live. A second vaccination should be given 4 weeks following the first vaccination. In areas of high incidence of virus infection and for maximum protection a third vaccination may be indicated at 16 weeks of age. If the kitten was 12 weeks of age or older at the time the first vaccination with modified live virus vaccine is given, a second vaccination is not necessary until at least one year of age.

These vaccines produce immunity for long periods of time. Since the exact duration of immunity is not known, annual revaccinations are recommended for maximum protection. This is especially pertinent for apartment cats that usually are not exposed to street virus.

Maternally derived immunity must be considered in establishing a vaccination program. It is the most common cause of vaccine failure. There is a direct correlation between the titer of the queen at the time of parturition and the duration of passive immunity in the kitten. This passive immunity, if present in sufficient quantities, will not only protect the kitten against virulent virus but will also interfere with vaccination. Thus vaccination must be done after the kitten has lost all or the majority of its maternally derived immunity.

The use of antiserum to passively immunize cats against FP is indicated in unvaccinated cats that have been exposed or probably will be exposed to virus before there is sufficient time to develop an immune response following vaccination. Antiserum is also indicated in colostrum deprived or orphan kittens as soon after birth as possible. The routine use of antiserum in unexposed kittens is not recommended. Instead, these kittens should be vaccinated at their first visit to the veterinarian's office, regardless of age, and revaccinated as indicated.

FELINE IMMUNIZATION

Fredric W. Scott, D.V.M., Ph.D.

The incidence of infectious diseases in cats has been reduced greatly through the use of vaccines. While no vaccine is 100% effective, the proper use of the currently available vaccines will allow kittens the best opportunity to grow up as healthy, robust cats.

PRECAUTIONS FOR VACCINATION

While vaccination may appear to be a simple, innocuous procedure, there are several adverse effects or precautions concerning vaccination of cats that must be kept in mind. Because of these it is recommended that vaccines be adminstered only by licensed veterinarians, or by licensed animal health technicians or animal owners under the supervision of a licensed veterinarian. Only vaccines obtained from reputable companies where adequate quality control is maintained should be used.

The following are specific precautions or possible adverse effects that might be encountered when vaccinating cats.

Production of disease from vaccine - If the vaccine virus is not modified sufficiently, clinical disease can result following vaccination. Examples are mild respiratory disease after intranasal vaccination, or clinical rabies following vaccination with a living vaccine not licensed for use in the cat. Improperly prepared vaccines may contain virulent virus. The stress of vaccination may trigger a latent infection into a clinical disease.

Effect on fetus - Developing fetuses are much more susceptible to damage by vaccine viruses than are adults. Fetal death, abortions, resorbtions, or congenital birth defects can be the result of vaccination of a pregnant cat with certain vaccines.

Allergic reactions - While the cat is not overly prone to allergic reactions, occasional severe and even fatal allergic reactions can occur after vaccination. With appropriate knowledge and medication these reactions can be counteracted.

Infection - Improperly handled equipment and vaccines can become contaminated with bacteria resulting in abcess formation and/or generalized bacterial infections.

Nerve injury - An improperly placed injection can result in injury to a peripheral nerve with resulting lameness or paralysis.

Failure of vaccine to immunize - The use of vaccines which have lost their immunizing ability, the vaccination of kittens that still have maternal immunity, and the vaccination of immunocompromised cats are examples

which may result in vaccine failure. Exposure to virulent virus later will result in severe clinical disease.

NATURE OF THE VACCINE

Both inactivated and modified live virus (MLV) vaccines are available. The vaccines must be handled, stored, and administered according to the manufacturer's instructions in order to maintain potency. MLV vaccines should not be administered to pregnant cats.

ROUTE OF VACCINATION

The route by which the vaccine is administered may affect the degree of protection provided. Feline panleukopenia (FP) vaccine can be given intramuscularly (IM) or subcutaneously (SC) with equal effect. The MLV-FP vaccines can also be given by the intranasal or aerosol route, but they will not result in immunization if administered orally.

Rabies vaccine must be given by the IM route. Although extensive studies on the route of rabies vaccination in cats have not been reported, studies in dogs with a MLV vaccine have shown that the IM route is at least 100 times more effective than the SC route. The same should hold true for the cat.

The MLV respiratory vaccines appear to be slightly more effective by the IM route, but they can be given SC. Aerosol vaccination with injectable vaccines may result in mild signs of illness.

The intranasal (IN) respiratory vaccines are administered by allowing the cat to inhale drops of recently reconstituted lyophilized FVR-FCV or FVR-FCV-FP vaccine into the nostrils. One or two drops are also placed in each eye (conjunctival sac). These vaccines produce rapid local as well as systemic immunity. Owners should be aware that vaccinated cats may sneeze and develop mild ocular and/or nasal discharge 4 to 7 days after IN vaccination. Occasionally, ulcers may develop on the tongue following vaccination. Vaccinated cats shed FVR and FCV viruses for long periods after IN vaccination.

AGE OF THE CAT

The most frequent cause of vaccine failure with FP vaccines is interference caused by maternally derived immunity. These cats become susceptible later, after the passive immunity wanes. The level and duration of passive immunity following nursing are determined by the antibody titer of the queen at parturition, assuming that the kitten nurses. Although the majority of cats can be immunized successfully at 8 to 10 weeks of age, occasional kittens may not be susceptible to vaccination until 12 weeks of age. Therefore, if FP vaccines are given at ages less than 12 weeks, they should be repeated at three- to four-week intervals until the cat is at least 12 weeks old.

Little is known about maternal antibody interference in feline viral rhinotracheitis (FVR) and feline calicivirus (FCV) disease vaccines. The same principles of colostral transfer, antibody half-life, and vaccine virus neutralization should apply to these viruses as well as FP. Therefore, we can predict that there will be interference if the maternal titers are high enough. Generally the FVR and FCV titers are much lower than the FP titer, and therefore the duration of interference (and passive protection) should be much shorter. It is doubtful that this will be longer than 5 to 6 weeks for the FVR and 7 to 8 weeks for the FCV. By 9 to 10 weeks of age, the vast majority of cats should be susceptible to FVR and FCV vaccination.

FELINE PANLEUKOPENIA VACCINES

There are many excellent vaccines available for immunization of cats against panleukopenia. If these are used correctly and at the proper age, cats should be completely protected against this very severe viral infection.

Several slightly different programs for the immunization of cats against panleukopenia have been presented during the past few years. Most recommendations indicate that the kittens should be vaccinated starting at 8, 9, or 10 weeks of age, and the vaccination repeated at 3- to 4-week intervals until the kittens are at least 12 weeks old.

If there is any question about exposure to FPV prior to 8 weeks of age, kittens can be vaccinated as early as 4 weeks of age. Vaccination is then repeated at 4-week intervals until the kitten is at least 12 weeks old.

FELINE VIRAL RHINOTRACHEITIS (FVR) VACCINES

FVR vaccines may be obtained in combination with calicivirus vaccine, as a triple FP-FVR-FCV vaccine, or as a 4-way vaccine with pneumonitis. The FVR vaccines produce significant protection following vaccination and, as such, should be part of the routine vaccination program, as outlined in Table 1. As a result of local viral replication, vaccinated cats develop a rapid anamnestic response when exposed to virulent virus. Some of these exposed vaccinated cats may sneeze, and an occasional one may have watery eyes for one to two days, but severe systemic disease does not occur in properly immunized cats as it does in unvaccinated susceptible cats.

FELINE CALICIVIRUS (FCV) VACCINE

Originally, it was thought that multiple serotypes of FCV existed and thus that an effective FCV vaccine would not be possible. Studies have shown that most outbreaks of FCV infection are caused by a single serotype of virus. Most strains of FCV tested exhibit good protection against other strains of FCV. However, recently 2 new serotypes of FCV have been identified from cases of "limping kittens." It would appear that current

vaccines will not protect against these new viruses. The same parameters (i.e., route of vaccination, anamnestic response when challenged, and good clinical protection against virulent virus exposure but not protection against local viral replication) apply to FCV vaccines as to FVR vaccines. These vaccines are produced in combination with FVR vaccine and recommendations are the same as for FVR (see Table 1).

FELINE PNEUMONITIS (FPn) VACCINE

Although FPn is not as prevalent as FVR or FCV disease, it is evident that in some cat populations a severe, chronic respiratory disease is produced by the FPn agent, a chlamydial organism. Vaccines currently available appear to produce significant protection following a single IM vaccination. As with other respiratory vaccines, complete protection is not afforded, but clinical signs, if they do occur, are restricted to a very short course and are mild and local. Chronic disease (characteristic of natural infection in susceptible cats) apparently does not occur in vaccinated cats.

FPn vaccines are available in combination with other vaccines. The vaccines now can be produced from infected cell cultures instead of eggs, thus eliminating some of the side effects occassionally seen when egg-origin vaccines are used.

Although there are many basic parameters concerning immunity to FPn that are not known, it appears that, if FPn is a problem in a particular area, the FPn vaccine should be part of the routine vaccination program. The age at which to vaccinate is not critical, since there appears to be little interference with maternal antibody by the time kittens are old enough to be vaccinated. A single injection appears to afford adequate protection.

RABIES VACCINES

Dogs but not cats are routinely vaccinated in many parts of the United States. However, in 1982 for the first time in history, there were more cases of rabies in cats than there were in dogs in the United States. Cats should be routinely vaccinated for rabies in any area where rabies in wildlife (skunks, raccoons, bats) is endemic. There is a high correlation between skunk rabies and cat rabies. Skunk rabies is endemic throughout the central United States from Canada to Mexico. Raccoon rabies is endemic along the east coast from Florida to Pennsylvania, and it is rapidly moving northward. Some states have legislation requiring rabies vaccination and licensure of cats.

Inactivated rabies vaccines should be used in cats. Most MLV rabies vaccines have been withdrawn for use in cats because of the tendency to produce clinical rabies. All rabies vaccines must be administered by the IM route.

FELINE LEUKEMIA VIRUS

No vaccines for feline leukemia virus (FeLV) infections are licensed at this time. Several commercial companies are studying experimental FeLV vaccines, and hopefully one or more of these vaccines will prove to be both effective and safe for immunizing cats against FeLV.

FELINE INFECTIOUS PERITONITIS

Feline infectious peritonitis (FIP) defies immunization at present. Attempts to develop vaccines against FIP have only sensitized vaccinated cats to a more acute infection upon exposure to virulent FIP virus, rather than providing protective immunity. FIP is being investigated actively in several laboratories.

Table 1. Feline Vaccine Recommendations

DISEASE	TYPE OF VACCINE	AGE AT FIRST VACCINATION (weeks)	AGE AT SECOND VACCINATION (weeks)	REVACCINATION	ROUTE OF ADMINI-STRATION
Panleukopenia (FP)	(1) Inactivated	8-10	12-14	Annual	SC or IM
	(2) MLV	8-10	12-14	Annual	SC or IM
	(3) MLV-IN	8-10	12-14	Annual	IN
Viral rhino-tracheitis (FVR)	(1) MLV	8-10	12-14	Annual	SC or IM
	(2) MLV-IN	8-10	-	Annual	IN
	(3) Inactivated	8-10	12-14	Annual	SC or IM
Caliciviral disease (FCV)	(1) MLV	8-10	12-14	Annual	SC or IM
	(2) MLV-IN	8-10	-	Annual	IN
	(3) Inactivated	8-10	12-14	Annual	SC or IM
Pneumonitis	(1) Live Attenuated	8-10	-	Annual	SC or IM
Rabies	(1) Inactivated	12	-	Annual	IM
	(2) MLV*	12	-	Annual	IM

*Approved for use in cats (only one vaccine as of 5/83). Use of non-approved MLV vaccines in cats can result in vaccine-induced rabies.

MLV = modified-live-virus
IM = intramuscular
IN = intranasal
SC = subcutaneous

CORONAVIRUS INFECTIONS OF CATS

Jeffrey E. Barlough, D.V.M.

In the mid-1960s, several viruses which had been isolated from man, mice, and swine were found to have a common morphology with avian infectious bronchitis virus (IBV). "Coronavirus" was the name proposed for this group of viruses on the basis of certain common and distinctive characteristics.

The morphologic characteristic from which the name is derived is a fringe of club-shaped viral surface projections which resembles the rays or corona of the solar disk.

Coronaviruses cause infections in animals, birds, and humans. They tend to have a predilection for cells of the intestinal and respiratory tracts. In humans, they are one of the major etiologic agents of upper respiratory tract disease, often resulting in lost time due to sick leave.

Intestinal infections in swine, cattle, dogs, mice, and turkeys can cause high mortality and great economic loss. In chickens, the economic loss from IBV is appreciable from the mortality due to bronchitis in chicks, decreased egg production, and vaccination costs.

FELINE INFECTIOUS PERITONITIS (FIP)

FIP is an important and complex coronavirus disease of domestic and exotic cats which is worldwide in occurence.

Currently 3 forms of the disease are recognized:

(1) Effusive or "wet" FIP
   a) Abdominal and/or thoracic effusion.

(2) Non-effusive or "dry" FIP
   a) Minimal effusion.
   b) Characteristic lesions involving especially the kidneys, liver, central nervous system (CNS), and/or eyes.

(3) Or: Combinations of the two

EFFUSIVE FIP: COMMON CLINICAL SIGNS

1. Anorexia, weight loss, depression
2. Dehydration
3. Fever non-responsive to antibiotics
4. Progressive fluid accumulation
5. Respiratory insufficiency (thoracic form)

NON-EFFUSIVE FIP: POSSIBLE CLINICAL SIGNS

1. Signs of kidney and/or liver insufficiency
2. Central nervous system involvement: posterior paresis, ataxia, behavioral changes (BEWARE OF RABIES!), increased muscle rigidity, paralysis, seizures
3. Ocular involvement: inflammation, hemorrhage, white "specks" on cornea

FIP: DIAGNOSIS OF THE DISEASE

1. History and clinical signs
2. Hemogram
3. Analysis of effusion (if present)
4. Serum protein analysis
5. Clinical chemistry profiles
6. Serum coronaviral antibody titer
7. Histopathology - the definitive method for diagnosis

FIP: SEROLOGY

1. In the general cat population, about 10-40% of cats are seropositive for coronaviral antibodies.
2. In breeding catteries and multiple-cat households, either 0% or 80-90% of animals are seropositive.
3. In specific pathogen-free (SPF) cat colonies, coronaviral antibodies are usually absent.
4. Virtually 100% of cats with FIP are seropositive.
5. The presence of coronaviral antibodies in a cat indicates prior contact with a **coronavirus in the FIPV group...**
   - ...could be FIPV
   - ...could be TGEV
   - ...could be CCV
   - ...could be one of the FECVs
   - ...could be 229E??? (human pathogen)
   - ...others???
6. A POSITIVE CORONAVIRAL ANTIBODY TITER DOES **NOT** DIAGNOSE FIP.
7. Considering that FIP occurs sporadically in the general cat population and that most cats in FIP-problem catteries are seropositive and yet do not contract FIP, it seems that a certain percentage of cats with coronaviral antibodies are protected against the development of FIP following natural exposure. The immune mechanism responsible for this immunity is not known.
8. Some Seropositive cats may shed virus from either the GI tract or respiratory tract, or both. Those with persistently high titers apparently shed more virus than do cats with persistently low titers.
9. Presently there is no way to differentiate the cat with immunity to FIP from the asymptomatic chronic carrier, nor is there any way of predicting whether a seropositive cat will ever develop FIP.
10. Asymptomatic seropositive cats in the general population usually have antibody titers ranging from 1:25 to 1:400 or greater.

11. Titers in these cats may vary with time; some cats with titers of 1:1600 have shown a decrease in titer to less than 1:25 within one year.
12. Antibody titers of 1:400 and above are consistent with but not diagnostic of FIP; effusive FIP may on occasion be associated with lower titers.
13. The magnitude of the antibody titer is proportional to the chronicity of the disease; i.e., cats with non-effusive FIP generally have higher titers than cats with the effusive form.

FIP: WHEN TO USE THE IMMUNOFLUORESCENT CORONAVIRAL ANTIBODY (IFA) TEST:

1. As an aid in the diagnosis of FIP in a symptomatic cat with signs suggestive of FIP.
2. To determine the presence or absence of antibodies (i.e., coronavirus exposure) in a previously untested household.
3. To detect potential virus carriers when introducing new cats into FIPV-free households or catteries.
4. To monitor treatment of FIP. Decreasing titers (except in the terminal stages of the disease) are indicative of clinical remission.

FIP: ALTERNATIVE SEROLOGIC TECHNIQUES

1. Virus neutralization: Results are often conflicting. Recent research suggests that FIPV-neutralizing antibodies will **not** protect against FIPV infection.
2. ELISA and KELA apparently detect the same antibody population as the IFA.
3. Passive hemagglutination: inconclusive.
4. Cell-mediated immunity?? - fertile area for future research.

FIP: IMMUNOPATHOGENESIS OF THE DISEASE

1. Initial exposure to FIPV may result in localized upper respiratory disease in about 25% of cats from 2-6 weeks later. This "primary" stage of the disease manifests clinically as a mild to severe conjunctivitis and/or rhinitis, which may persist for 1-4 weeks. During this period, exposed cats develop low to moderate coronaviral antibody titers.
2. Although the vast majority of cats undergoing the primary form of FIP recover, many will probably remain chronically infected (i.e., they will be potential virus shedders).
3. A very small number of exposed cats will develop the lethal disseminated ("secondary") form of FIP weeks to months (or years) later after their primary infection.
4. The factors involved in susceptibility to fatal FIP are probably multiple:
   a) Dose and strain of virus
   b) Route of exposure
   c) Age at time of exposure

d) Concurrent viral infections (e.g., FeLV)
e) Genetic predisposition
f) Administration of immunosuppressive drugs
g) Adverse environmental influences (stress, overcrowding, etc.)

5. Although mechanisms of disease in FIP have been discussed for many years, recent evidence indicates that clinical FIP is the result of Arthus-like antigen-antibody-complement reactions across blood vessel walls, accompanied by virus persistence within mononuclear phagocytes.
6. Kittens with coronaviral antibody titers are more susceptible to FIP than seronegative kittens.
7. Thus, coronaviral antibodies apparently contribute to an enhancement of the FIP disease process, probably by facilitating uptake of virus into mononuclear phagocytes.

FIP: TREATMENT AND PREVENTION

1. Presently there is no curative therapy for FIP, nor is there an effective prophylactic vaccine.
2. However, some treatment regimens may induce temporary remissions in a very small percentage of carefully-selected patients.
3. The basic aim of therapy in FIP at present is to alleviate the disseminated inflammatory reactions which are the hallmark of the disease.
4. The most effective treatment protocols combine high levels of corticosteroids, cytotoxic drugs, broad-spectrum antibiotics, and maintenance of nutrient intake and fluid and electrolyte balance.
5. Co-infection with FeLV is a significant complicating factor and the FeLV status of all suspected FIP cats should be determined prior to commencing treatment.
6. Routine vaccination against FIP is not currently possible. Due to the immunologic nature of the disease, immunization with existing strains of FIPV may paradoxically predispose cats to lethal FIP.
7. Further research using tissue culture-adapted FIPV strains may eventually yield information necessary for development of a safe and effective vaccine. However, results reported thus far have not been encouraging.

FELINE ENTERIC CORONAVIRUS(ES)

1. Recently reports of coronaviruses localized to the gastrointestinal tract have appeared. A virus morphologically similar to FIPV has been reported from cats reared in a conventional feline breeding colony at the University of California, Davis. This virus was detected in feces of healthy cats as well as in feces from cats with enteric disease. A coronavirus has been isolated in cell culture at Washington State University from an adult cat with fatal hemorrhagic enteritis, and has been shown to be closely related to both FIPV and CCV. More recently, a case of enteritis apparently caused by a coronavirus was reported in a young kitten from Japan. Both the small and large intestines were involved in this animal. Several naturally-occurring cases of FIP have shown similar intestinal involvement. Very recently another report of coronaviral particles in feces of a cat with diarrhea has appeared in Canada.

2. Presently it is unclear whether coronaviruses localized to the gastrointestinal tract represent FIPV itself, modified FIP strains, or new coronaviruses originating either in the cat or in another species.

FELINE ENTERIC CORONAVIRUS-LIKE PARTICLES

1. Coronavirus-like particles, morphologically distinct from FIPV, have been observed in feces of healthy kittens and adult cats raised in the Cornell minimal-disease feline breeding colony. Both coronaviral antibody-positive and negative cats can excrete these particles. Recent experimental studies at Cornell have failed to produce coronaviral antibodies in cats given coronavirus-like particles by a variety of routes, suggesting that they are distinct from coronaviruses and may represent a separate virus group. Coronavirus-like particles have also been detected in fecal samples from a number of hospitalized cats (approximately 10%) at the Cornell Small Animal Clinic. Both clinically healthy cats and cats with enteric disease were found which shed these particles.

KITTEN MORTALITY COMPLEX

Cheryl A. Stoddart, B.S.

During 1977 and 1978, many cat breeders and their veterinarians consulted the Cornell Feline Health Center reporting alarming reproductive failure and high kitten mortality. These and other secondhand reports were strikingly similar. Everyone's story was the same: convincing evidence that a specific disease complex, termed Kitten Mortality Complex (KMC), was occurring throughout the country. Kitten mortality rates in certain extensively studied catteries ranged from 40% to 80% during peak periods of kitten loss. It was apparently a new disease whose etiology was and still is unknown.

KMC is characterized by 3 main problems: reproductive failure, kitten mortality, and various diseases in the adult. Reproductive failures include repeat breeders, fetal resorption between 4 and 6 weeks of gestation, abortion (usually during the last 2 weeks), stillbirths, and congenital malformations. These malformations have included skull defects ("open top fontanelles"), cleft palates, "open stomachs," heart defects, and atresia ani (incomplete extension of the colon to the anus).

Kitten mortality is usually exhibited by the "fading kitten syndrome" where the kittens either are born weak and die within a few hours or seem healthy and then become depressed and anorectic, eventually dying of starvation or secondary bacterial infections. Perhaps the most dramatic expression of kitten mortality is in acute congestive cardiomyopathy. These kittens suddenly are unable to breathe, become cyanotic, and die within a few hours. Postmortem and histologic examinations reveal hugely dilated and thin-walled hearts with acute muscle fiber degeneration, usually accompanied by fluid-filled thoracic cavities and lungs. A small percentage of kitten mortality is due to feline infectious peritonitis (FIP), usually the granulomatous (dry) form as opposed to the more typical effusive (wet) form.

In the adult cats, respiratory disease and uterine infections (endometritis and pyometritis) are both common and highly consistent findings in the catteries experiencing KMC. The respiratory disease is usually chronic and mild, involving the upper respiratory tract and with sneezing as its most common symptom. Watery ocular and nasal discharges may also occur, and the cat seldom becomes seriously ill. Many queens (up to 40% of the queens in some catteries) have vaginal discharges; further examination usually reveals endometritis or pyometra. Other reported problems include adults and older kittens with intermittent and usually low grade fevers, acute congestive cardiomyopathy, and cardiovascular disease.

Although the exact cause of KMC is not known, many feline viruses can cause reproductive failure, fetal malformations, neonatal kitten death, and various other diseases in the adult. These include feline viral rhinotracheitis (FVR), feline panleukopenia (FPL), feline calicivirus (FCV), and feline leukemia virus (FeLV). Most catteries affected by KMC routinely

vaccinate against FVR, FPL, and FCV. It must be remembered that kittens should be vaccinated twice, preferably at 8 and 12-16 weeks of age, to provide full protective immunity. Although most catteries experiencing KMC have been found negative for FeLV, any cattery having reproductive and kitten mortality problems should be FeLV-tested. If cats are found to be positive, a proper course of action must be quickly followed to either isolate or eliminate those FeLV-positive cats. Many other factors, such as uterine bacterial infections, toxoplasmosis, genetics, toxic chemicals, and nutrition, might also play roles in this disease complex.

Many, if not all, catteries have cats which are FIP antibody-positive, and therefore FIP virus (FIPV) has been incriminated as the etiologic agent of KMC. My research has involved studying the transmission of FIPV from queens to their kittens, particularly to determine whether or not the virus can be transmitted in utero to the developing fetus. If it can be transmitted, this would be strong evidence that FIPV is somehow involved in KMC. At the present time, however, we do not completely understand the relationship between FIPV and KMC. It is also important to note that the great majority of catteries have FIP antibody-positive cats, both those which are experiencing KMC and those which have had no problems whatsoever.

Based on the number of cases of KMC reported to us, KMC seems to have peaked in incidence in 1978. There are catteries which still experience these same reproductive and kitten mortality problems and we continue to receive reports of new cases of KMC in previously unaffected catteries. However, KMC seems to have run its course for the most part. In many catteries, the problems disappeared as fast as they appeared. In the absence of any cures for KMC, this may offer some consolation for cat breeders. In the meantime, the Cornell Feline Health Center continues to explore this extremely frustrating disease.

KITTEN MORTALITY SURVEY 1975 - 1980

Cheryl A. Stoddart, B.S.

Yearly surveys conducted by the Cornell Feline Health Center in conjunction with the Research Committee of the Cat Fanciers Association (CFA) from 1975 to 1980 revealed that kitten mortality was a serious problem in many catteries throughout the United States. Over the 6-year period, a total of 9,517 kittens born in 2,309 litters from 28 breeds was surveyed, 9.3% of which were stillborn. Of the 8,630 kittens born alive, 5.8% died within 24 hours of birth, 2.8% died the second day, 13.2% had died by the first week, and 24.2% failed to reach one year of age. Stillbirths and deaths by one year of age considered together resulted in a total mortality rate of 31.1% for those kittens surveyed over the 6-year period. A brief summary of the survey data for each year from 1975 to 1980 and the totals for the entire period are presented in Table 1.

The most interesting aspect of this survey is that while kitten mortality rates are alarmingly high, they did not change appreciably from 1975 to 1980. (We have received the 1981 surveys but have not as yet compiled the results.) It was assumed that kitten mortality would noticeably decrease from 1975 to 1980 with the advent and widespread use of the feline respiratory virus vaccines. There was a slight yet statistically insignificant decrease in mortality from 1975 to 1976. Kitten mortality rates did not significantly increase during the precipitous outbreak of kitten mortality and reproductive failure, termed Kitten Mortality Complex (KMC), in 1977 and 1978.

A possible explanation for these unexpected results is that while kitten deaths due to the feline respiratory viruses had indeed decreased, this decrease was masked by increasing rates of kitten deaths related to KMC. It is very difficult to determine the validity of this hypothesis based on the data collected so far. However, by continuing our study of kitten mortality with improved yearly surveys, we hope to find the underlying causes of such alarmingly high rates of kitten deaths in catteries throughout this country. The Cornell Feline Health Center will continue to seek answers to this puzzling, high incidence of kitten mortality.

Table 1. CFA Kitten Mortality Survey 1975-1980.

	1975	1976	1977	1978	1979	1980	TOTAL 1975-1980
Litters surveyed	341	449	438	455	340	286	2,309
Total kits born	1,528	1,940	1,763	1,833	1,327	1,126	9,517
Average kits/litter	4.48	4.32	4.03	4.03	3.90	3.94	4.12
Kits born alive	1,363	1,753	1,576	1,701	1,209	1,027	8,630
% kits born alive	89.2	90.4	89.4	92.9	91.1	91.2	90.7
Average live kits/litter	4.00	3.90	3.60	3.74	3.56	3.59	3.74
Kits born dead	165	187	187	131	118	99	887
% kits born dead	10.8	9.6	10.6	7.1	8.9	8.8	9.3
% malformations (in total kits born)	7.7	6.2	4.9	4.5	7.3	8.3	6.3
% kits born alive, dying in:							
less than 24 hours	6.8	6.6	4.9	4.3	5.8	7.2	5.8
24-48 hours	4.1	3.7	2.1	2.5	1.8	2.0	2.8
48-72 hours	2.7	2.6	2.8	1.4	1.1	2.6	2.2
3-6 days	1.4	2.5	3.2	1.8	2.9	3.0	2.4
1-2 weeks	4.3	1.5	2.1	2.8	1.7	1.5	2.3
3-6 weeks	4.4	4.7	3.6	3.6	3.8	2.8	3.9
6 weeks - 6 months	4.3	3.5	3.6	4.3	6.5	3.4	4.2
6 months - 1 year	0.7	0.7	0.2	0.5	0.3	0.7	0.5
% kits dying within 1 year	**28.7**	**25.8**	**22.8**	**21.2**	**24.0**	**23.3**	**24.2**
% kits born dead or dying within 1 year	**36.4**	**33.0**	**30.7**	**26.8**	**30.7**	**30.0**	**31.1**

FELINE LEUKEMIA VIRUS (FeLV)

Jeffrey E. Barlough, D.V.M. and Carol E. Pepper, B.S.

FeLV is the most important viral agent of fatal infectious disease of American domestic cats today. It was first visualized by EM in 1964 in sections of mesenteric lymphoid tissue from a cat with lymphosarcoma. The results of recent molecular studies suggest that an ancient rodent retrovirus, transferred in some manner to a North American ancestor of the domestic cat, may have served as the origin of FeLV millions of years in antiquity.

An antigenically similar group of viruses, the feline sarcoma viruses (FeSVs), is known to cause multicentric fibrosarcomas in susceptible neonatal kittens, and, when injected into neonates of other species, including dogs, sheep, goats, rats, rabbits, and non-human primates, to induce similar tumors. In addition, intracutaneous inoculation may produce locally invasive melanomas in young kittens. In the natural state, FeSVs apparently occur infrequently and are generally not contagious. Evidence suggests that these sarcoma viruses have arisen by genetic recombination between FeLV and normal gene sequences from feline host cells. Thus they are also replication-defective, requiring "helper" virus (FeLV) in order to replicate.

A third feline retrovirus, designated RD-114, is an endogenous, vertically-transmitted virus, whose genome is represented within the genetic material of all domestic cat cells. Expression of its genetic information is usually repressed in feline cells, and replication is best demonstrated in cells of other species. Molecular studies suggest that RD-114 represents an endogenous primate virus which was horizontally transmitted to an ancestral cat in ancient times, and has been maintained within the feline cellular genome for millions of years. RD-114 appears to be unrelated to FeLV/FeSV, is generally non-infectious and non-immunogenic, and is not oncogenic per se for any known species.

THE FeLV-ASSOCIATED DISEASES

1. Lymphosarcoma (LSA) - an uncontrolled proliferation of lymphocytes, a cell type found in the blood and lymphatic system which serves in the immune system to destroy invading organisms or foreign matter.
   a) Thymic form - the proliferating cells form a mass in the chest, leading to difficulty in breathing. The cat may cough often and try to breathe through its mouth. These symptoms can arise quickly. This form affects mainly kittens and young cats.
   b) Alimentary form - this form is characterized by the proliferation of cells into masses in the abdomen. It affects the various organs found there, such as the intestines, liver, kidney, etc. Cats with this form of the disease will lose appetite, become weak, and lose weight. Diarrhea or difficulty in defecation may occur. This is the most common form of lymphosarcoma seen in the U.S.

c) Multicentric form - this is characterized by lymphocytes proliferating within the lymphatic system so that many lymph nodes are enlarged. These cats will eventually lose their appetite and become extremely emaciated.

d) Leukemic form - characterized by the presence of abnormal cells circulating in the blood. The abnormal numbers circulating in the blood interfere with the normal function of the blood and prevent gas and nutrient exchange with tissues.

e) Unclassified forms - a miscellaneous category for lymphosarcoma. Solitary masses of uncontrolled proliferating cells can be found anywhere throughout the body, including the skin, eye, kidney, and central nervous system (brain and spinal cord). Symptoms may vary depending on the location of the tumor.

2. Myeloproliferative Disorders (MPDs) - an abnormal proliferation of cells in the bone marrow which spill out into the blood. (The marrow is responsible for the production of all blood cell types such as red and white blood cells, platelets, etc). A blood test can help diagnose this condition.
3. Non-regenerative Anemia - A type of anemia (deficiency of red blood cells or RBCs) where the animal is incapable of responding with an increase in production to replace the lost RBCs. In other words, the bone marrow fails to regenerate a supply of RBCs. The cause may be due to the uncontrolled growth of lymphocytes in the bone marrow, which occupy space needed by RBC-producing cells.
4. Leukoerythroblastic Anemia - a disease of the bone marrow where two cell types are both proliferating out of control. These are the cell types necessary for both red and white blood cell formation. An anemia develops because these proliferating cells usually do not mature correctly so there is a lack of normal RBCs. This disease is seen in only some cases of lymphosarcoma.
5. Thymic Atrophy - the thymus gland is present only in kittens and young cats and normally atrophies as the cat gets older. It functions in the immune response and is responsible for the maturation of a type of lymphocyte. Very young cats infected with FeLV tend to have virus remain in the bloodstream for a longer period of time. These animals have a greater susceptibility to various diseases, and the thymus can atrophy prematurely in these cases. This usually occurs before the virus has produced lymphosarcoma and is therefore a pre-neoplastic condition.
6. Reproductive Disorders - A FeLV-infected queen has difficulty raising a healthy litter, for various reasons. Conception may be prevented, abortion or fetal death may occur anytime throughout pregnancy, or kittens may be born unhealthy and die soon thereafter. This can be due either to the presence of a tumor in the reproductive tract, an unthrifty, sick queen which is unable to supply nutrients to the fetuses, or because the kittens themselves were infected with the virus while developing in utero.
7. Panleukopenia-like Syndrome - Some cats with FeLV will develop symptoms similar to panleukopenia (feline distemper) with sneezing and diarrhea. Circulating white blood cells in the blood are very low. Unlike panleukopenia, however, all of these cats will die.
8. Glomerular Disease - In some FeLV cases, death can be mainly due to kidney failure. A mass of lymphocytes may have invaded the kidney,

thereby interfering with its function; or an immune reaction between the cat's own immune cells and the virus may produce a complex which "plugs up" the kidney, preventing normal function.

9. Chronic progressive polyarthritis (CPP) - The joints are eroded by an immune complex reaction and become unstable. This disease appears to occur mostly in cats infected with both FeLV and the feline syncytium-forming virus (FeSFV). Glomerular disease can occur simultaneously.

TRANSMISSION OF FeLV:

1. Routes of Virus Excretion
   a) Saliva - most important
   b) Respiratory secretions
   c) Feces, urine
   d) Placental transfer
2. Importance of Immunologic Maturity
   a) Kittens vs. adult cats
   b) Pathogenesis of FeLV infection
      1) Minor viremia - mononuclear cell infection
      2) Major viremia - bone marrow, intestinal crypt epithelium, systemic lymphoid tissue - essentially irreversible (in most cases)
3. Survival of FeLV in the Environment
   a) Very labile
   b) Susceptible to lipid solvents

MAJOR COMPONENTS OF FeLV VIRIONS:

1. gp70
   Envelope glycoprotein, present in the viral peplomers; responsible for attachment and penetration of cells.
2. p27
   Group-specific viral core protein; provides the antigenic basis for both the IFA and ELISA tests for FeLV.
3. p15(E)
   Envelope-associated protein; partially responsible for FeLV-mediated immunosuppression.
4. RNA
   Diploid, single stranded; positive polarity.
5. Reverse Transcriptase

ANTIBODY RESPONSES TO FeLV PROTEINS:

1. gp70
   Virus-neutralizing antibodies (protection against viremia).
2. p27
   Possible immune-complex disease; antibodies not protective.
3. p15(E)
   Antibody response not yet fully characterized.

4. Reverse Transcriptase
   Problems in interpretation of RT antibodies.
5. Feline Oncornavirus-associated Cell Membrane Antigen (FOCMA)
   Not an FeLV structural protein; protection against development of LSA (in many cases).

DISEASES ASSOCIATED WITH FeLV-MEDIATED IMMUNOSUPPRESSION:

1. Hemobartonellosis (feline infectious anemia).
2. Toxoplasmosis - public health aspects.
3. Bacterial septicemias.
4. Chronic oral ulceration, stomatitis, gingivitis.
5. Poorly-healing, recurrent abscesses.
6. Pyodermas.
7. Chronic respiratory infections (sinusitis, pyothorax, pneumonias).
8. Intermittent enteritis, peracute enterocolitis.
9. Severe purulent otitis externa.
10. Feline infectious peritonitis (FIP) - 45-50% are FeLV+

DIAGNOSTIC AIDS FOR FeLV:

1. Detection of viremia
   a) IFA
   b) ELISA
2. Detection of virus-neutralizing antibodies
   a) Protective titer of 1:10 or above
3. Detection of FOCMA antibodies
   a) Protective IFA titers either 1:8 or 1:32, depending upon laboratory

DETECTION OF FeLV VIREMIA:

1. Immunofluorescence Assay (IFA)
   a) Hardy test, slide test, FeLeuk™ test
   b) Detects FeLV p27 in infected leukocytes and platelets
2. Enzyme-linked Immunosorbent Assay (ELISA)
   a) Kit test, Leukassay™
   b) Detects soluble FeLV p27
3. Persistent vs. transient viremia
4. Prevalence of persistent viremia:
   a) Multiple-cat FeLV households: 25-35%
   b) Single-cat urban households: Probably less than 1%

INTERPRETATION OF FeLV TEST RESULTS:

1. Detection of p27 in leukocytes and platelets (slide test)
   a) Positive: Almost always indicates lifelong viremia
   b) Negative: Infection is absent or has not reached bone marrow stage

2. Detection of soluble p27 (ELISA test kit)
   a) Positive: Indicates presence of circulating p27 (either intact virus or p27 alone); viremia may be transient or persistent
   b) Negative: Infection is absent or has not reached primary viremia stage
3. CAUTIONARY NOTE ON "NEGATIVE" FeLV TESTS!
   a) A negative test, either by IFA or ELISA, in no way indicates whether past infection has occurred!
   b) Integration of DNA copy of FeLV genome?
   c) Future FeLV-related disease in the presence of neutralizing antibodies?

INTERPRETATION OF ANTIBODY TEST RESULTS:

1. Virus-neutralizing antibodies
   a) Titers of 1:10 or above indicate protection against persistent anemia
   b) Protective titer does not imply freedom from proviral integration!
2. FOCMA antibodies
   a) Constellation of antibodies against a myriad of FOCMAs
   b) Do not always indicate protection against LSA development
   c) "Protective titers": 1:8 or 1:32, depending upon laboratory

PREVALENCE OF ANTIBODIES TO FeLV PROTEINS IN THE GENERAL CAT POPULATION:

1. Virus-neutralizing antibodies
   a) Multiple-cat FeLV households: 40-50%
   b) Single cat urban households: 5-20%
2. FOCMA antibodies
   a) Multiple-cat FeLV households: 50-60%
   b) Single-cat urban households: 30-50%

FELINE LSA: TREATMENT

1. Chemotherapy - drug combinations
   a) Prednisolone
   b) Vincristine (Oncovin™)
   c) Cyclophosphamide (Cytoxan™)
   d) Cytosine arabinoside (Cytosar™, Ara-C™)
2. Alternate chemotherapeutic agents
   a) Vinblastine (Velban™)
   b) Chlorambucil (Leukeran™)
   c) Adriamycin
3. Irradiation
4. Surgery
5. Patient monitoring
   a) Hemogram
   b) Kidney and liver function tests
6. Ethical issue: To treat or not to treat?

FELINE LEUKEMIA VIRUS INFECTION: TREATMENT

1. Plasmapheresis
   a) Removal of "blocking" antibodies in patient serum
   b) Staph Protein A column
   c) Reversal of FeLV+ status in a number of cases

CONTROL OF FeLV INFECTIONS:

1. All cats in household should be tested; all positive cats should be removed.
2. Clean household premises with sodium hypochlorite (Clorox™; 1:32 in water) or other common household product.
3. Replace all feeding bowls and litter pans.
4. Re-test negative cats several times over 8-12 months, and remove all cats which have become positive.
5. Test all new cats prior to entry; quarantine all such cats for 3-5 months (2 or 3 negative tests) prior to intermixing.
6. Routine yearly or twice-yearly testing is recommended.
7. Never use viremic cats for breeding purposes.

FeLV VACCINATION:

1. Soluble Tumor Cell Antigen Vaccine (STAV)
   a) Contains only surface antigens, no infectious viral nucleic acid, no p15(E).
2. Killed Vaccines
3. Live, attenuated vaccines
4. Tumor cell vaccines
   a) Can be highly effective
   b) Live cells, live attenuated cells, killed cells
5. Importance of intact p15(E) to vaccination

INFECTIOUS DIARRHEAS

Charles A. Baldwin, D.V.M., M.S.

Diarrhea can be described as an increase in the amount of water present in the feces. The increase in fecal water could be due to water retention or water secretion or malabsorption. No matter what increases the fecal water content, the condition is noticed by the owner of the pet very readily.

The normal intestine contains tall, fingerlike projections called villi, which project into the lumen or the center of the bowel and are responsible for absorbing water and nutrients.

At the base of these villi are the immature cells, which divide and produce many more cells to replace those mature cells at the top of the villus tips. In these basal areas or crypts, as they are known, secretion of water into the lumen usually occurs. Thus, anything that affects these cells may produce hypersecretion and diarrhea. At the top of the villus, as mentioned, are the mature absorbing cells. Any loss of these cells means that no absorption can take place, and diarrhea also results. In one way or another, most of what will be discussed affects one of these two areas.

I. Causes of diarrhea

A. Functional

1. Drugs
   a. Direct toxic effect - organophosphates, arsenic, thallium, lead.
   b. Microflora change - changes that occur in the normal bacterial microflora such as seen with antibiotic use.

2. Dietary Overload

3. Malabsorption
   a. Bile deficiency - blockage of the bile duct.
   b. Pancreatic enzyme deficiency
   c. Small intestinal mucosal disease
      1. Disaccharidase deficiency
      2. Glucose malabsorption

4. Intraluminal obstruction

B. Anatomic

1. Intramural infiltration of gut wall
   a. Lymphosarcoma
   b. Histoplasmosis
   c. Eosinophils and immunocytes

2. Strictures
    a. Congenital
    b. Secondary to peritonitis
    c. Neoplasia
        1. Adenocarcinoma
        2. Leiomyosarcoma
    d. Healing ulceration

C. Systemic problems

1. Adrenocortical insufficiency

2. Heavy metal poisoning

3. Rodent and insecticide poisoning

4. Canine distemper

5. Feline distemper

D. Inflammatory

1. Intestinal parasites
    a. Coccidia
    b. Giardia
    c. Trichomonas (?)
    d. Strongyloides
    e. Hookworms
    f. Whipworms
    g. Ascarids (?)

2. Allergic enterocolitis

One cause of diarrhea in the cat is bacteria. Many bacteria can cause diarrhea, but I would like to briefly discuss only one, Salmonella. Salmonella can cause a severe diarrhea in most animal species, and it can be contagious to you, the pet owner. The diagnosis of Salmonella can be obtained by culturing the feces of animals with diarrhea. However, Salmonella organisms can be found in normal cats suffering no disease. If your cat has diarrhea due to this bacteria, all that is needed for treatment is good nursing care and isolation. Antibiotics should only be used if your pet is suffering from systemic Salmonellosis. In fact, antibiotics can make an intestinal disease become a systemic disease. Systemic disease is very rare and can be fatal. Therefore, your veterinarian should be consulted if your cat has Salmonellosis, and a rational plan of therapy can be formulated.

The discussion will now turn to the smallest pathogenic organisms, namely viruses. There are 6 to 7 viruses either known or suspected to cause diarrhea in the cat. These include feline parvovirus, feline leukemia virus, feline enteric coronavirus, feline calicivirus, feline astrovirus, and feline rotavirus.

Feline parvovirus disease has many synonyms: feline panleukopenia, feline infectious enteritis, feline distemper, and feline ataxia. The virus infects rapidly dividing cells which, in the cat, are found in the intestine. In the unborn kitten, the affected area would be the cerebellum, the portion of the brain so important for coordinated walking (hence the name feline ataxia, the inability to walk in a coordinated fashion). In the cat, the rapidly dividing cells of the intestine produce new cells on the villi daily. In the disease state, these villi cells are lost, no absorption takes place, and diarrhea results. The cat will have a profuse watery diarrhea and no appetite. He will be very depressed and will die if untreated.

A good vaccination program, however, will virtually eliminate feline parvovirus disease from a cattery. By vaccinating all kittens at 6 to 8 weeks and giving a booster at 12 weeks of age, the kittens will be protected in about 95-99% of cases. (No vaccine regimen can be considered 100% effective, including those for humans.) To keep the adult cat protected, all that is necessary is an annual booster vaccination. Once the disease hits a cattery, good supportive therapy such as warmth, fluids, and food is all that can be done; there is no effective antiviral compound at this time, and antibiotics are ineffective. One other necessary step in prevention and control is good disinfection of the premises and eating utensils because the virus can survive up to a year in the environment. A proper dilution of sodium hypochlorite (Clorox) is an excellent disinfectant.

Feline leukemia virus can also occasionally infect the same rapidly dividing cell and produce a disease that exactly mimics feline parvovirus. The prognosis, however, is vastly different. Whereas with good supportive therapy one might successfully treat up to 60 or 70% of cats with parvovirus, very few if any animals with feline leukemia virus can be treated successfully.

Feline enteric coronavirus has been described as causing diarrhea. At a recent American Animal Hospital Association meeting, it was stated that this virus can cause anything from an inapparent infection to a mild diarrhea in kittens 4-12 weeks of age. It appears in one-quarter of all outdoor cats and probably can be found in all catteries. It has been isolated from both normal cats and those that show a diarrhea.

Rotaviruses are known to produce a diarrheal disease in many other species. In some species, the significance of the disease can be quite serious (cows, pigs, human infants). In the cat, however, the role is currently under investigation. It is known when cats contract the virus they will shed it in the feces for up to 12 days. Colostrum may provide necessary protection for newborns.

The last two feline viruses, astrovirus and calicivirus, have also been isolated from cats with diarrhea. The exact role of these viruses in producing the diarrhea is not known at this time. These viruses do produce diarrhea in other species, but the nature of their role in the cat awaits further investigation.

FELINE SURGERY

Jim Flanders, D.V.M.

Because of the size of the patient, feline surgery is definitely challenging. Cats in general are very good at healing if they are given a little surgical help.

SOFT TISSUE SURGERY

Surgery of soft tissues includes anything from plastic surgery of the skin to surgery on the great vessels of the heart. The most common soft tissue surgeries performed on the cat are those involved with removal of the reproductive organs: the castration and the ovariohysterectomy.

The best age for castrating a cat depends on the desired use of the cat. Castration at 7-9 months of age tends to result in an adult male with more female structural characteristrics; often such a cat will become an adult of slight build and lack the large jowls characteristic of a "tom cat." A cat castrated before one year of age is less likely to urine-mark his surroundings, although some early-neutered males will still do so. Castrating a fully mature "tom cat" will decrease the odor of the urine and often causes some behavioral changes such as reduced urine-marking and less night-roaming.

The main indication for ovariohysterectomy is sterilization of the female cat. Removal of the ovaries without removal of the uterus will provide sterilization and prevent demonstration of estrus. However, leaving the uterine horns in the abdominal cavity provides a potential space for infection should there be any ascending infection from the vagina through the cervix.

Spaying a cat before its first heat has not been shown to reduce the incidence of mammary cancer in the feline as it has been shown in the dog. Mammary cancer in the cat is much less common than in the dog; however, when a mammary tumor does appear in a cat, it is much more likely to be malignant.

Pyometra, or pus-filled uterine horns, is another indication for ovariohysterectomy in the cat. A pyometra is the result of chronic hypertrophy of the glandular and vascular elements within the uterus. There is a normal proliferation of the lining of the uterine horns in response to progesterone and estrogen increases during the estrous cycle. If the progesterone stage is prolonged, however, the uterine lining becomes cystic. Repeated cycles of this hyperplasia can predispose to infection and the eventual filling of the horns with pus. Pyometra is a very debilitating condition in the cat and can lead to death if not treated. Medical management of pyometra by giving prostaglandins to cause uterine contractions and pus expulsion has been attempted. It is not without considerable risk of causing rupture of a diseased, pus-filled uterus within the abdomen. Flushing the horns with antibiotic solution through an open

cervix is infrequently effective, and requires several days of treatment when a cat is critically ill. The treatment of choice for pyometra is ovariohysterectomy. The source of the infection, the uterine horns, is removed from the abdomen, and recovery with supportive care is usually relatively rapid.

THE FELINE UROLOGICAL SYNDROME (FUS)

FUS has resulted in the very common performance of perineal urethrostomy. Both male and female cats are plagued with the formation of small crystals of calcium and phosphorus within the bladder (struvite crystals). These crystals usually form in the absence of bacterial infection, but may be the by-product of a virus residing in the bladder wall or a metabolic abnormality in the cat.

Female cats rarely suffer from obstruction of the urethra as a result of crystal formation. This is because the diameter of the female urethra is large enough to allow expulsion of most of these small crystals. The male cat's urethra at the level of the penis becomes much narrower than that of the female, and it is in this area that urethral obstruction occurs. Urine backs up within the bladder as a result of urethral obstruction and eventually kidney function is impaired. The accumulation of various waste products within the cat's blood stream can cause death.

The surgical answer to the male cat's dilemma is to "convert" him into a female. This metamorphosis is done by removing the penis and its small-diameter urethra and making a new opening for the remaining portion of the urethra below the anus. The remaining portion of the male urethra is similar in diameter to that of the female and crystals can be passed more easily. It is important to note that the urethrostomy does not alter the production of crystals, it only changes the route of their expulsion.

ORTHOPEDIC SURGERY

Orthopedic surgery most frequently involves the repair of fractured bones. Cats are rarely treated for congenital bone deformities, so orthopedics usually involves a patient that has been traumatized. Before any fracture can be repaired, the injured cat must be examined carefully so that concurrent soft tissue injuries are not overlooked. Trauma to the forelimbs may also result in damage in the chest or skull. Rear limb injuries may be accompanied by fractures of the pelvis, rupture of the bladder or urethra or internal bleeding due to spleen or liver hemorrhage. The patient needs to be stabilized before orthopedic surgery can be undertaken.

Perhaps the most common skeletal injury in the cat is a fractured femur. The most simple femur fracture to repair is a simple fracture at the midshaft level. The method of choice would be the placement of an intramedullary stainless steel pin in the central canal of the bone extending from end to end. With good fixation, bone fractures in cats heal quite rapidly.

If a femur is fractured into many pieces, an intramedullary pin is not

as effective and a better choice for fixation is the placement of a metal bone plate on the femur. This plate is held in place with small screws and may be contoured to match the shape of the bone.

Bone plates usually allow a quicker return to function than intramedullary pins, however they are more expensive and require specialized equipment to be applied. Sometimes a bone plate will offer such good protection to the underlying bone that the plate must be removed before the bone begins to atrophy underneath it. This is not much of a problem however, in lightweight animals such as the cat.

Another common area to be fractured in cats is the pelvis. Because of the light weight and relatively great muscle strength of the cat, most cats can compensate for small deviations in the pelvis produced by trauma. Within weeks callus forms and helps to stabilize the fracture. This form of repair (bed-rest) often results in secondary problems such as arthritis or a narrow pelvic canal and also causes the cat to undergo prolonged discomfort while waiting for nature to provide stabilization. In many cases it is indicated to surgically stabilize pelvic fractures with pins or plates to provide a more rapid return to function and a more normal anatomic result.

SECTION II

Proceedings of

The Third Annual Feline Health Seminar

at the

Cornell Feline Health Center

June 1983

ANATOMY OF THE CAT

Howard E. Evans, Ph.D.

The cat (Felis domestica) is in the family Felidae of the order Carnivora which includes a large assemblage of intelligent, mostly flesh-eating animals. The felids are all carnivorous. Close relatives of the cats are the civets (Viverridae) and the hyaenas (Hyaenidae) of Africa. More distant relatives of cats are the raccoons, weasels, bears, dogs, seals, and walruses. All of these carnivores are descended from a cat-like ancestor called Cynodictus that lived some 20 million years ago in the Oligocene during which time small horses were already present. The natural history, behavior, and structure of cats and their relatives have been well described by Ruth Ewer in "The Carnivores" published by the Cornell University Press.

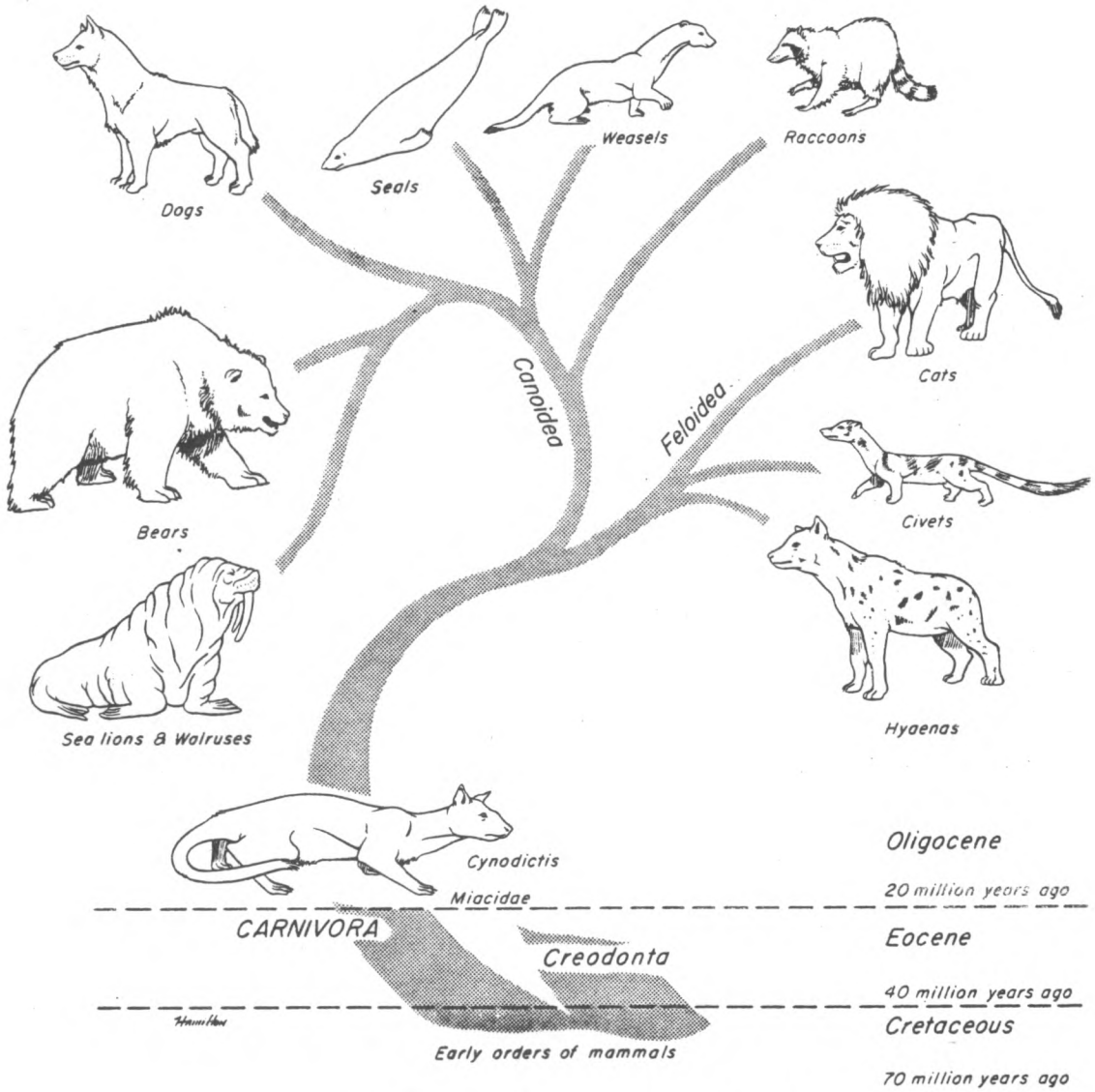

Figure 1. The family tree of the order Carnivora. from Evans and DeLahunta

Period	Epoch	Beginning of Interval (Millions of Yrs. Ago)	Important Events
Quaternary	Recent	.01	Modern man spreads worldwide
	Pleistocene	1.5 - 2	Many mammals vanish
Tertiary	Pliocene	5	Oldest-known hominids
	Miocene	22 - 23	Mammals reach their maximum diversity Grasslands - grazing mammals
	Oligocene	37 - 38	
	Eocene	53 - 54	Modern types of flowering plants appear
	Paleocene	65	Spread and diversification of mammals
Cretaceous		136	Dinosaurs and many other organisms become extinct Peak of dinosaur diversity Flowering plants appear
Jurassic		190 - 195	First birds. Dinosaurs increasingly abundant
Triassic		225	First dinosaurs, first mammals Abundant cycads and conifers

Domestication of the cat from Felis lybica and/or Felis sylvestris, the sand cat of Africa, has led to the development of many breeds which include: Abyssinian, Burmese, American Shorthair, Himalayan, Persian, Manx, Rex, Russian Blue, and Siamese. Each of the breeds has a distinctive hair-coat and body conformation that is judged to be desirable by the breeders. The hair may be wavy, plush, or double coat, short or long, in a solid, ticked, patched, barred, ringed, pointed, or tortoise-shell pattern.

A characteristic feature of the cat is the round head with the forwardly directed orbits. The jaws are short and this results in a reduced number of teeth, a total of 30 in the adult. The canine teeth are prominent and they are used for piercing and holding. The cheek teeth, consisting of premolars and molars, are adapted for cutting and crushing. The digestive tract is relatively short.

The limbs are long and the toes are very extensible, which allows them to function in the capture of prey. All cats except the cheetah have retractable claws that are provided with strong elastic ligaments which fold the second and third phalanges of the digit in such a manner that they lie parallel to each other when at rest.

The clavicle or "collar bone" is well developed in the cat as a slender bar at the shoulder which usually appears on a radiograph. It may look like a foreign body in the trachea.

A peculiarity of the circulatory system is the atrophy of the internal carotid artery, whose function of supplying the brain is taken over by the ascending pharyngeal, vertebral, and maxillary arteries.

Various features of the cat's anatomy will be discussed using the figures which follow.

MORPHOLOGICAL FEATURES TO BE CONSIDERED

Integument
- skin
- hair
- glands

Skeleton
- skull
- vertebrae
- ribs
- sternum
- girdles
- limbs
- claw

Muscles
- head
- trunk
- limbs

Nervous System
- brain
- cranial nerves
- spinal cord
- spinal nerves
- sense organs
  - eye
  - ear
  - nose
  - taste buds

Digestive System
- mouth
- oral cavity
- pharynx
- tonsils
- tongue
- teeth
- esophagus
- stomach
- intestine
- cecum
- anus

Reproductive System
- Male
  - testis
  - accessory glands
  - penis
- Female
  - ovary
  - oviduct
  - uterus
  - vagina

Circulatory System
- heart
- arteries
- veins
- lymphatics

Excretory System
- kidneys
- bladder
- urethra

Respiratory System
- nasal cavity
- sinuses
- larynx
- trachea
- lungs
- diaphragm
- thorax

Endocrine System
- adrenals
- gonads
- pituitary
- pancreas
- pineal
- thymus
- thyroid
- parathyroid

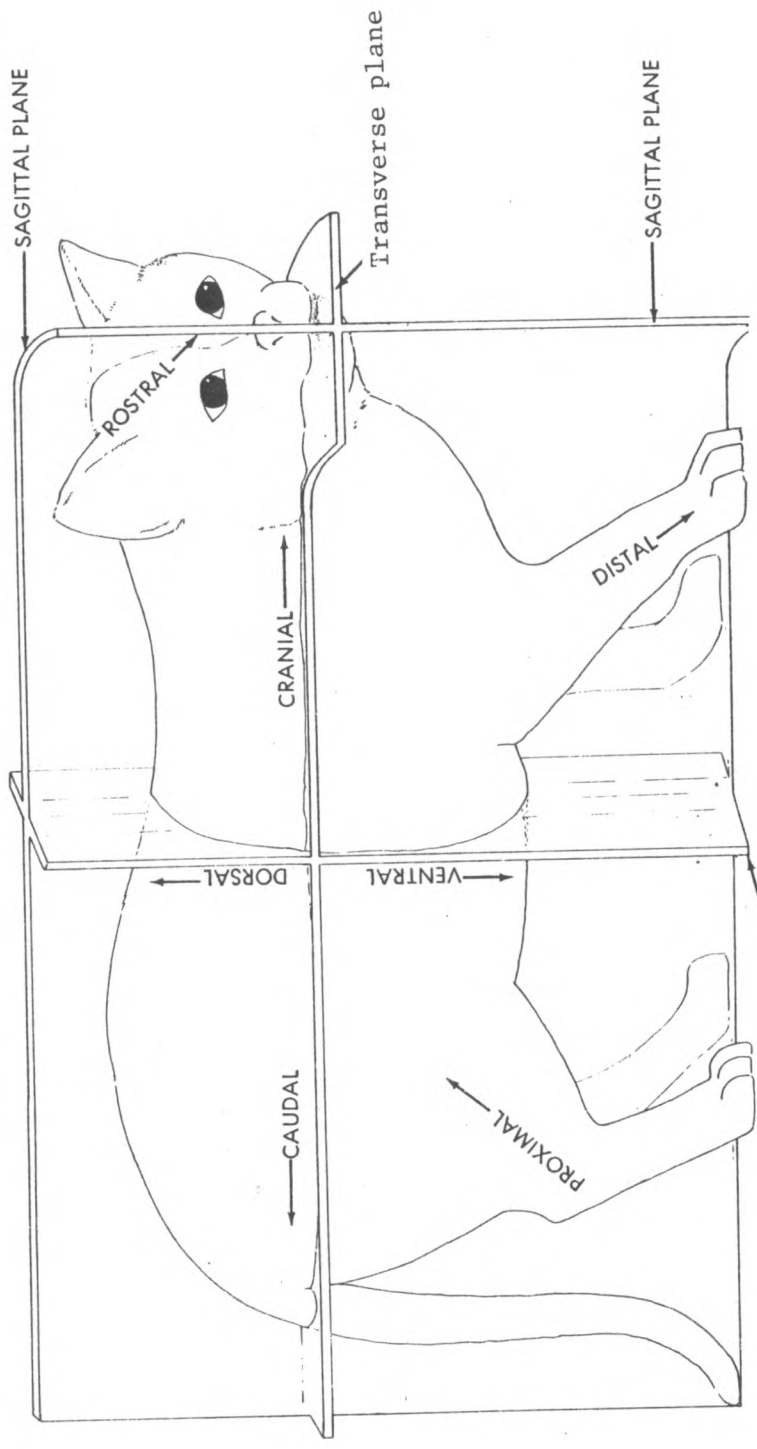

Figure 2. Diagram of anatomical planes and directions of the cat. From Chiasson, Robert B. and Ernest S. Booth, Laboratory Anatomy of the Cat (7th Ed.). copyright 1967, 1972, 1977, 1982. William C. Brown Co. Publishers, Dubuque, Iowa. All rights reserved. Reprint is by special permission.

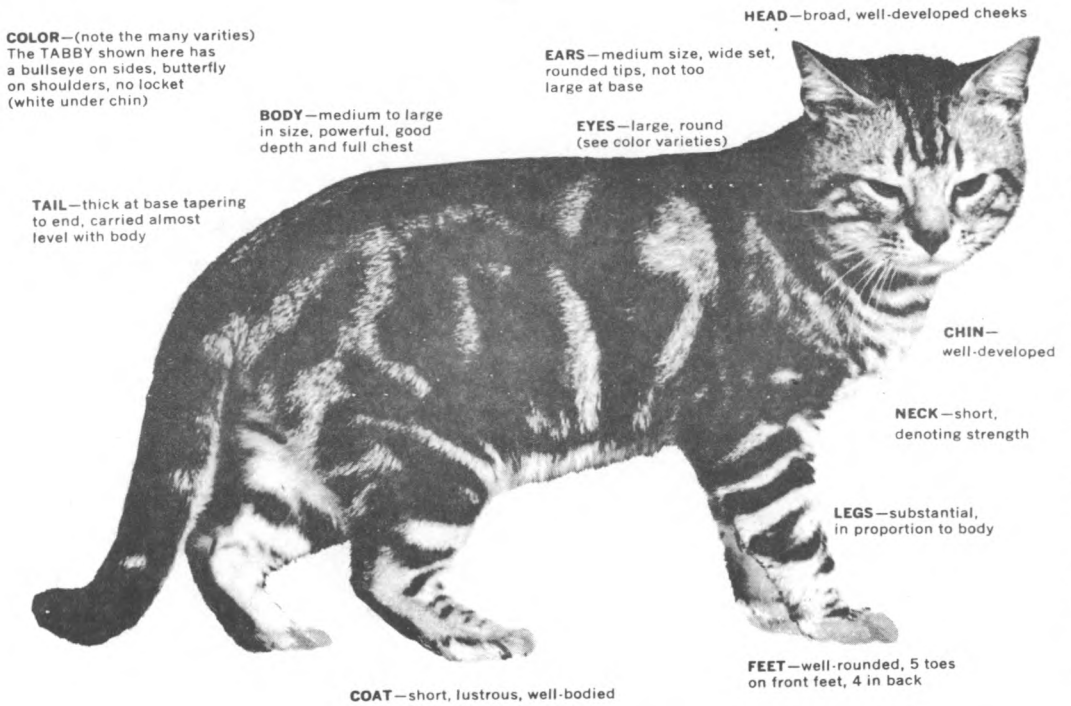

Figure 3. External features. from Ralston Purina Co.

AMERICAN SHORTHAIR

The most well-known breed of all! The tabby is believed to be the earliest domesticated cat in history. Its complicated markings make it most difficult to breed as an ideal show specimen. Great effort, by breeders, has been made to preserve the "true colors and types" which include both single-color and multi-color varieties. (Note: eye color important feature in judging).

SOLID COLOR: WHITE—blue-eyed, copper-eyed, odd-eyed; BLUE; BLACK; RED; CREAM—all copper or orange eyed.

SILVER: CHINCHILLA; SHADED SILVER—both blue-green eyed; BLUE SMOKE; BLACK SMOKE—both copper or orange eyed.

TABBY & TORTIE: BLUE TABBY; CREAM TABBY—both copper or orange eyed. RED TABBY; BROWN TABBY—both orange eyed; SILVER TABBY—green, hazel or lemon eyed; BLUE CREAM—orange eyed; TORTOISESHELL—copper or orange eyed.

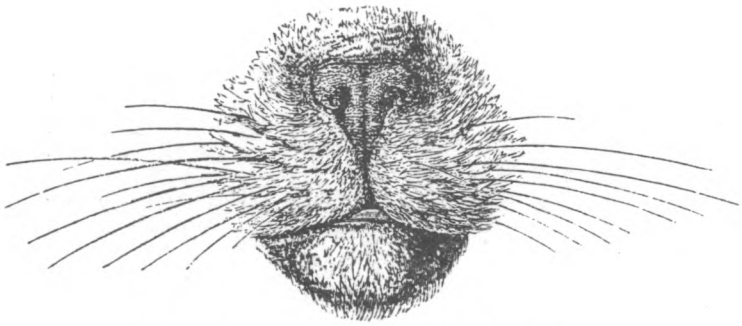

Figure 4. Tactile hairs or Vibrissae

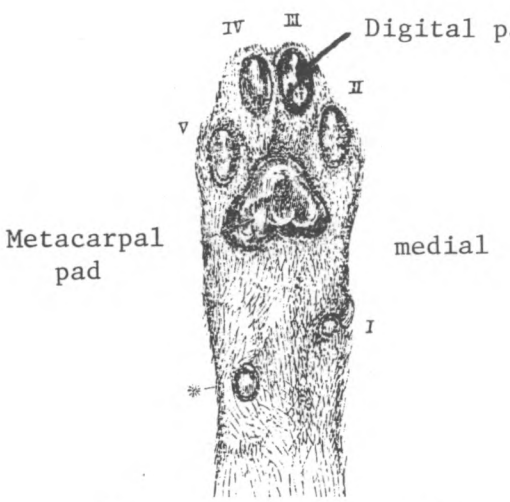

Figure 5. Palmar surface of forepaw. Digits I to V

Figures 4 - 7 from Mivart.

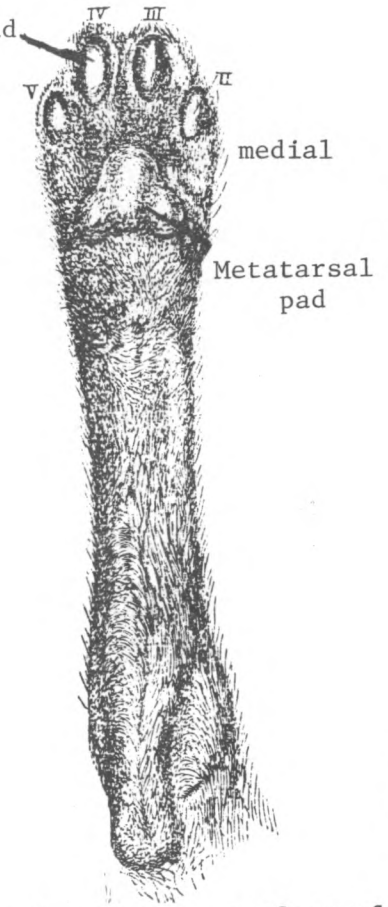

Figure 6. Plantar surface of hind paw.

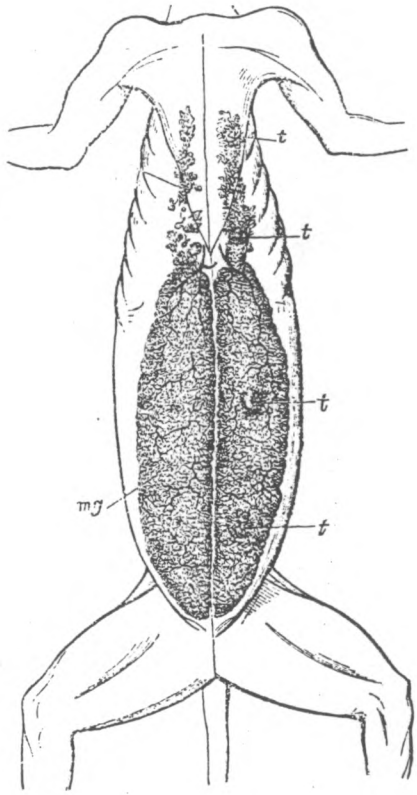

Figure 7. Ventral view of mamma.

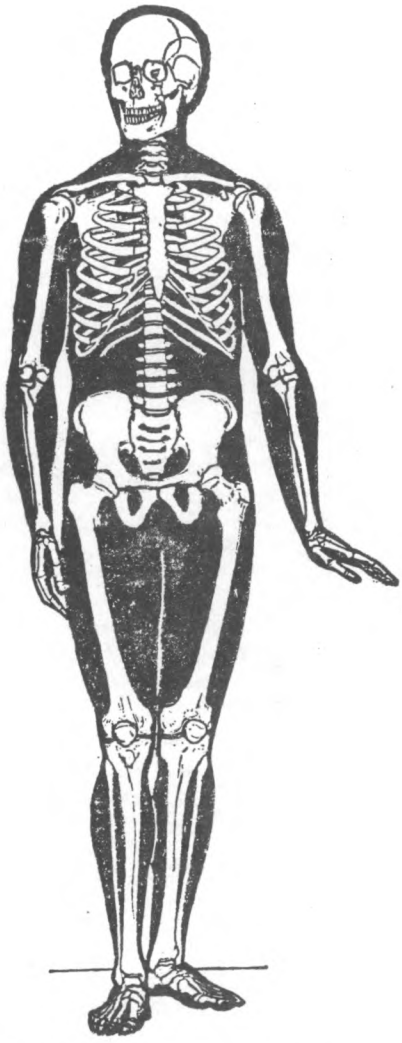

Figure 8. Human skeleton.

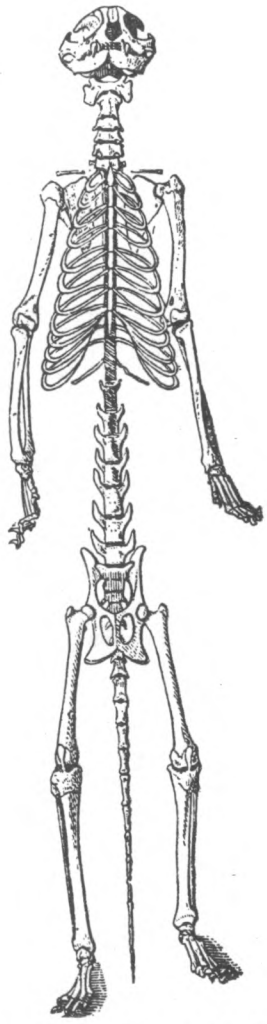

Figure 9. The skeleton of the cat, as if a human.

Figures 8 - 10 from Jayne.

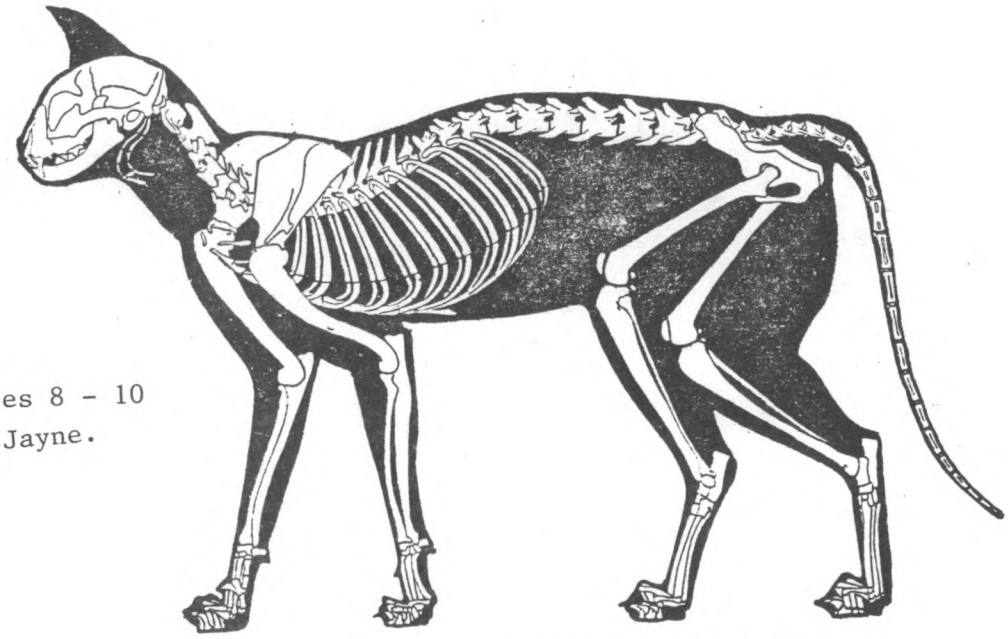

Figure 10. The skeleton of the cat.

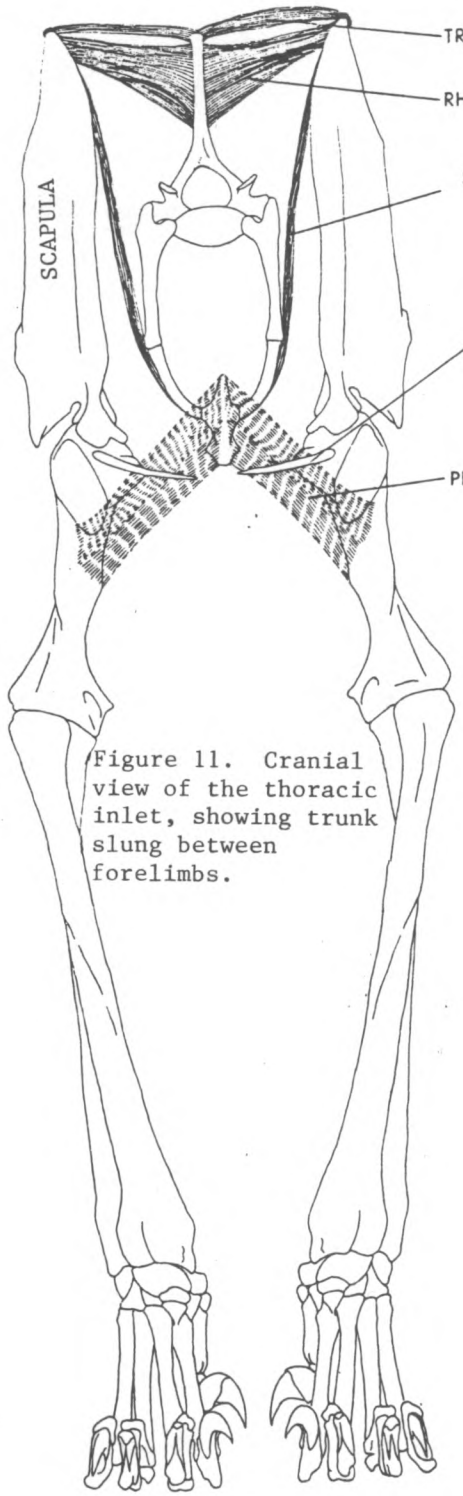

Figure 11. Cranial view of the thoracic inlet, showing trunk slung between forelimbs.

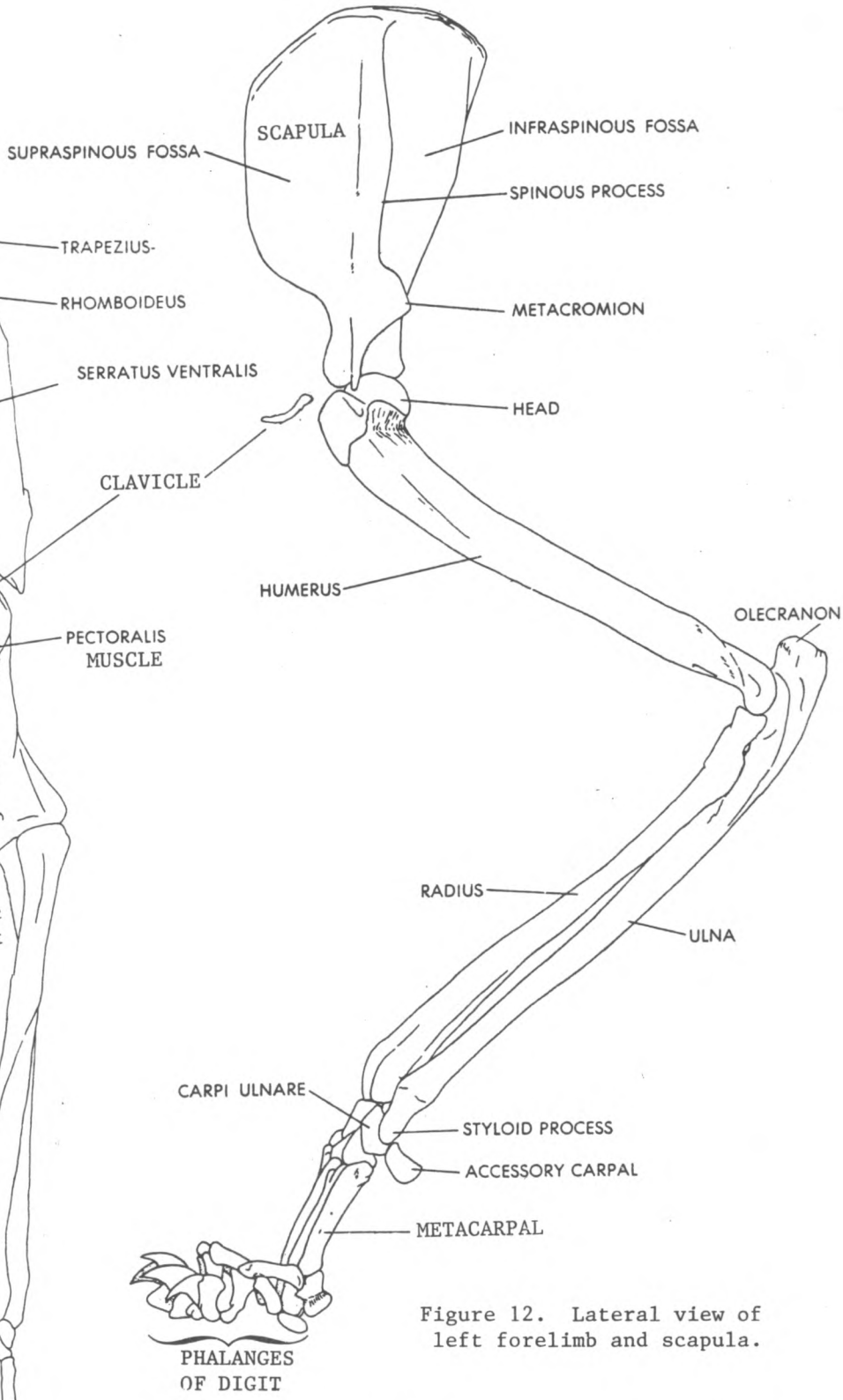

Figure 12. Lateral view of left forelimb and scapula.

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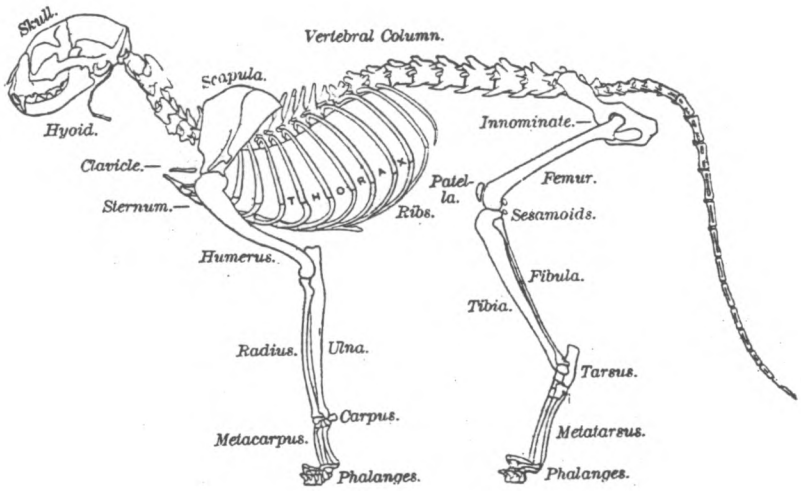


Figure 13. Lateral view.

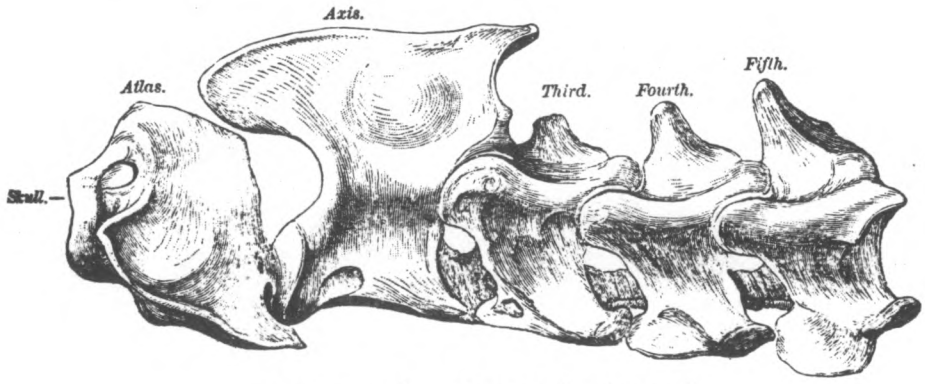


Figure 14. Lateral view of the first 5 of 7 cervical vertebrae.

Figures 13 and 14 from Jayne.

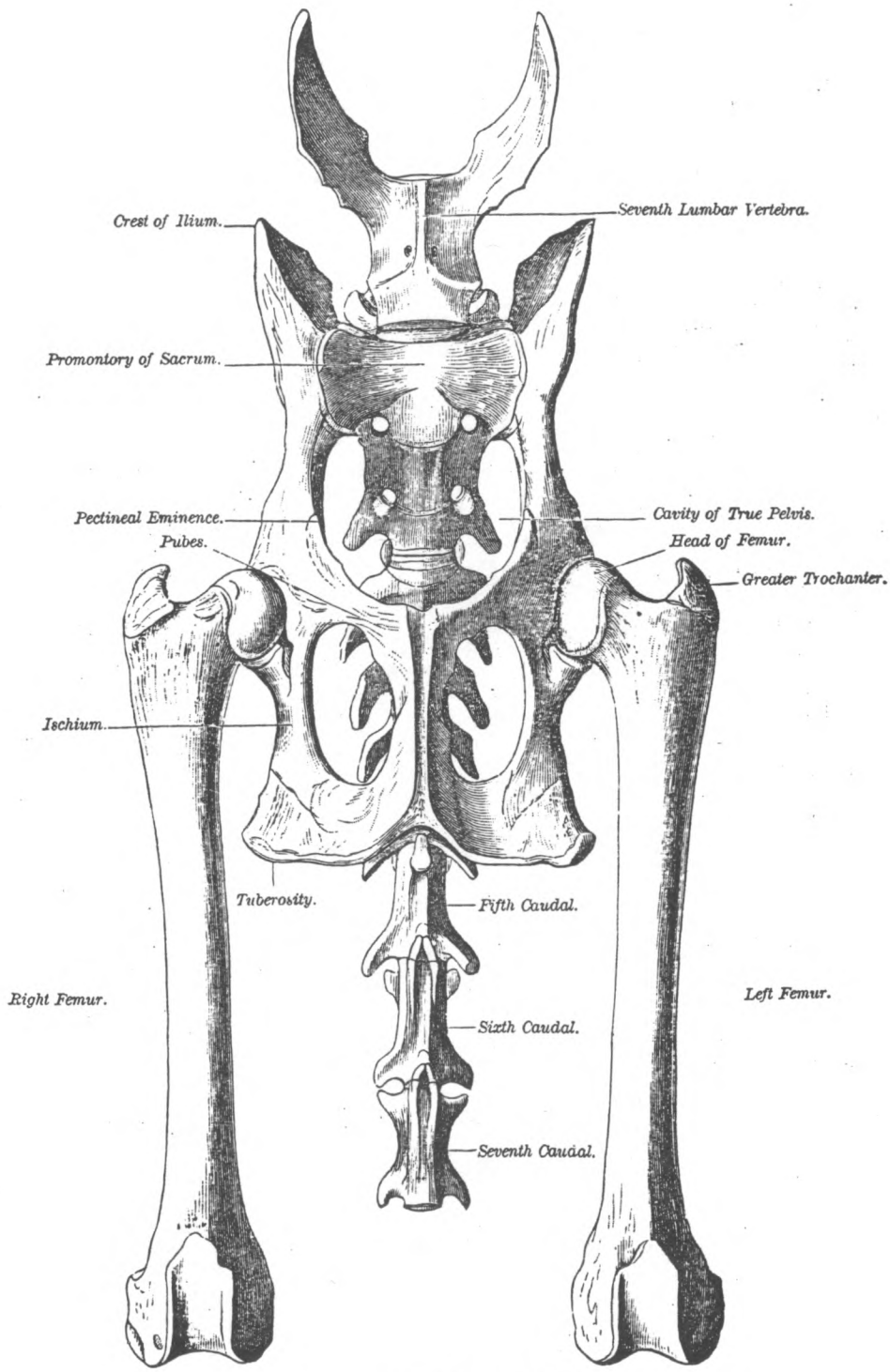


Figure 15. Ventral view of the pelvis, sacrum, and femur.

from Jayne

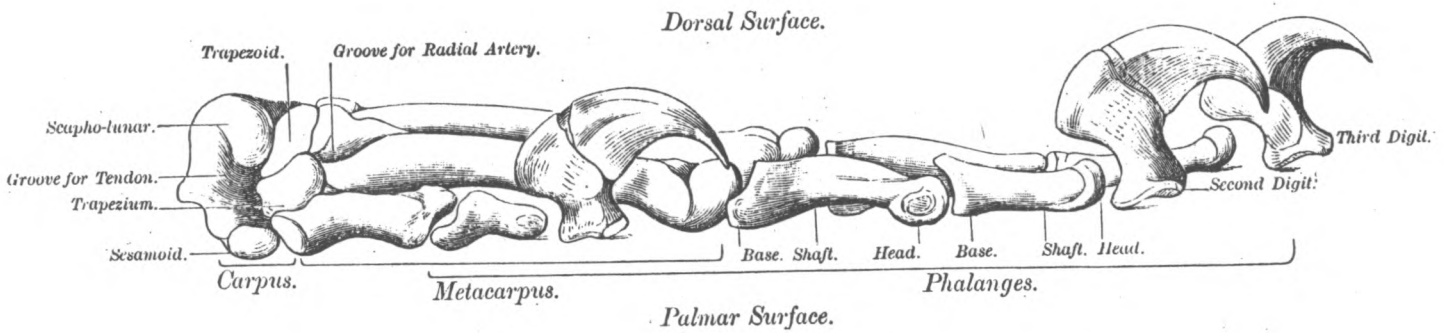


Figure 16. Left paw of cat, medial. from Jayne.

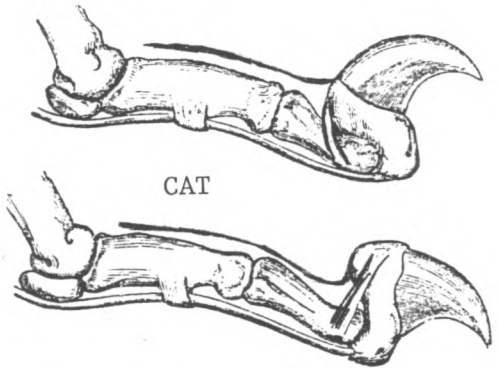


Figure 17. Elastic ligaments between the 2nd and 3rd phalanges which retract the claw. from Mivart

The digital pads of the dog are large and the elastic ligaments are small.

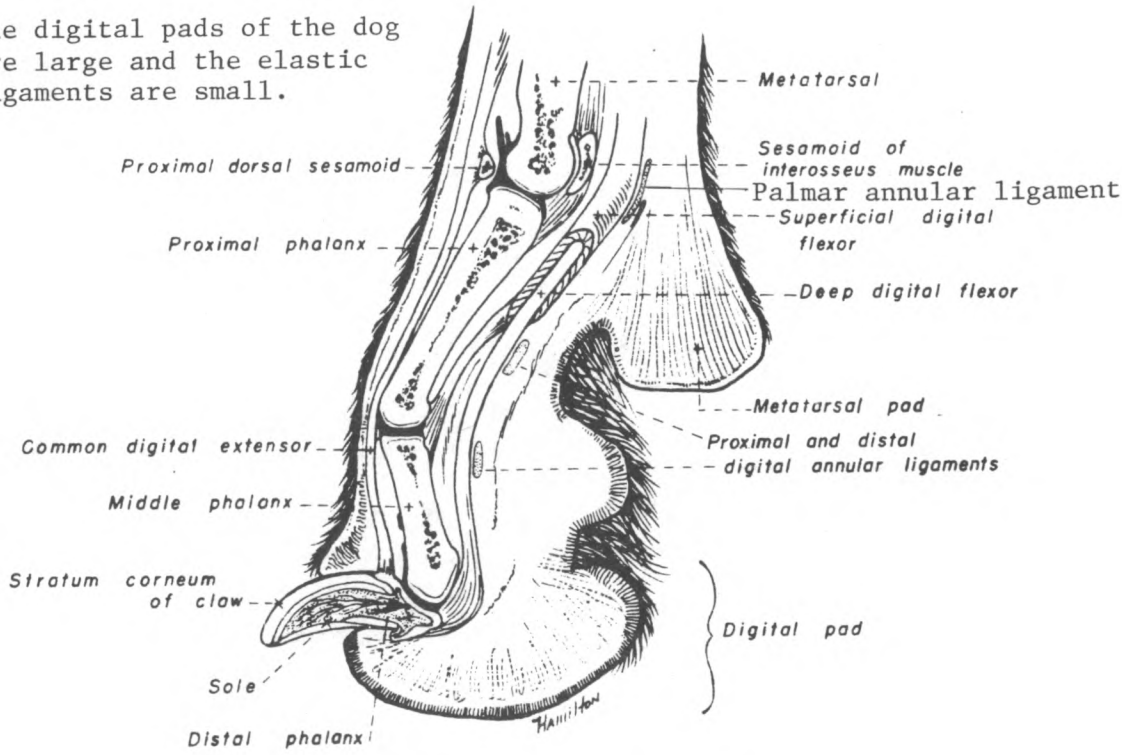


Figure 18. Median section of the third digit of the hind paw. (Dog.)

from Evans and DeLahunta

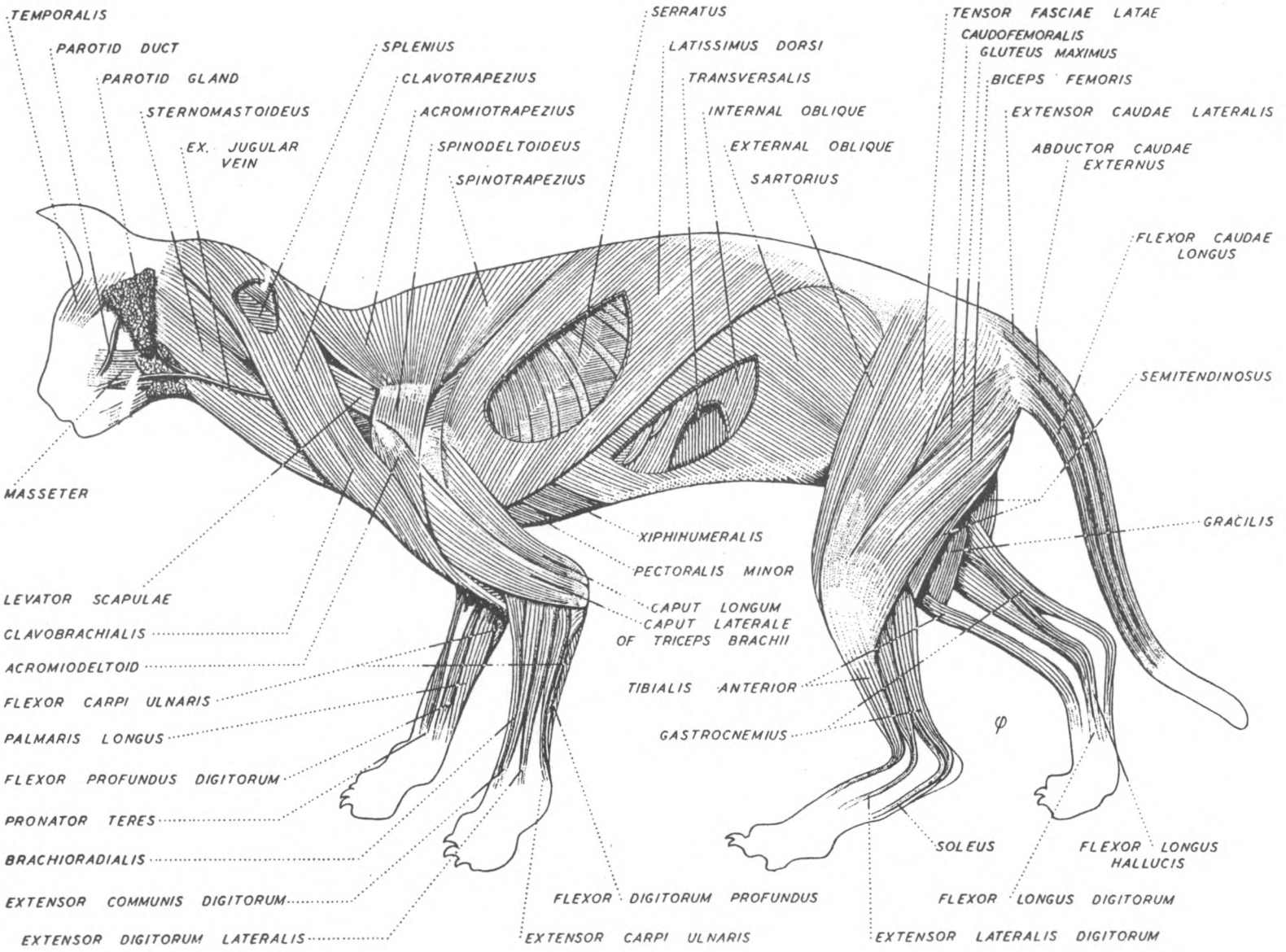


Figure 19. Superficial muscles. from Booth, Ernest S. Laboratory Anatomy of the Cat. (3rd edition) copyright 1948, Ernest S. Booth. Reprint is by special permission.

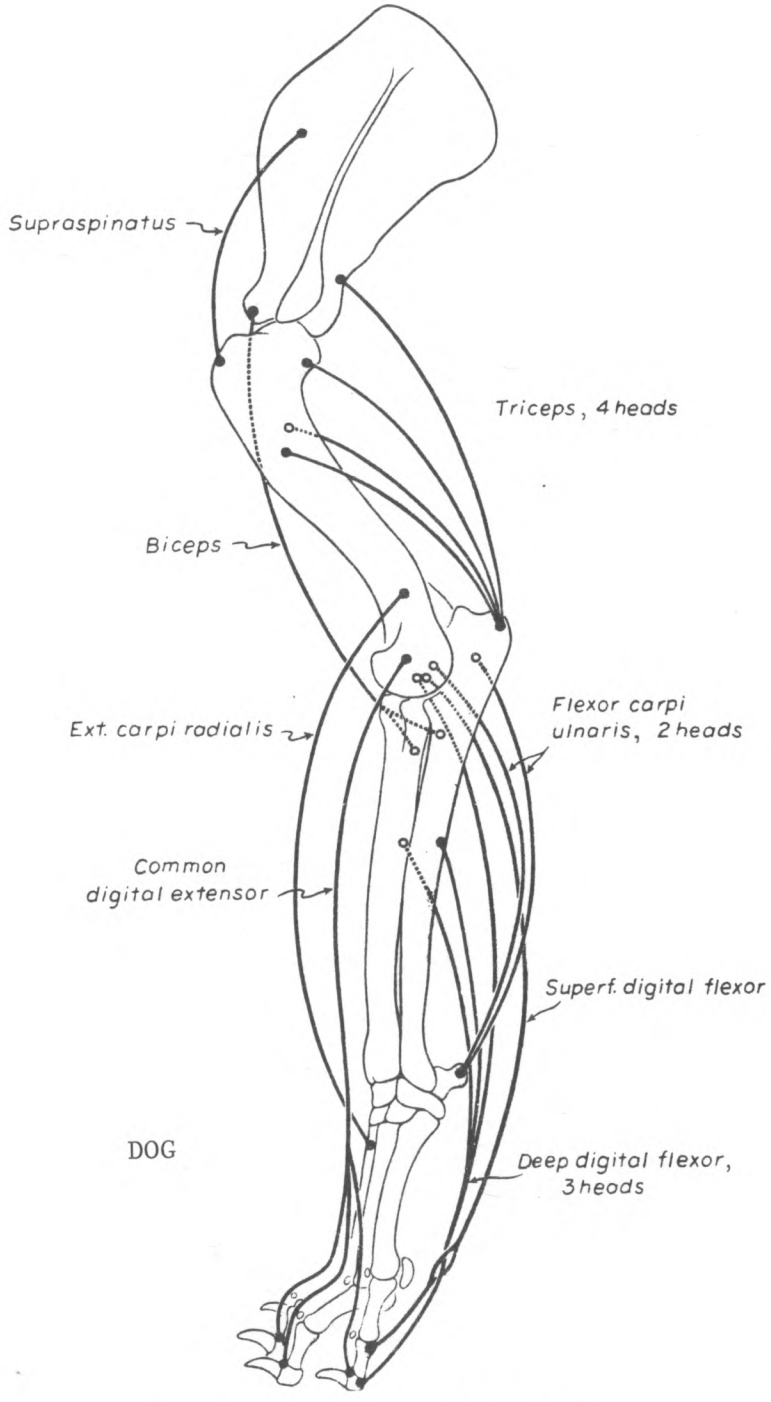


Figure 20. Major extensors and flexors of left forelimb.

Note that some muscles such as the biceps and long head of the triceps span two joints.

from Evans and DeLahunta

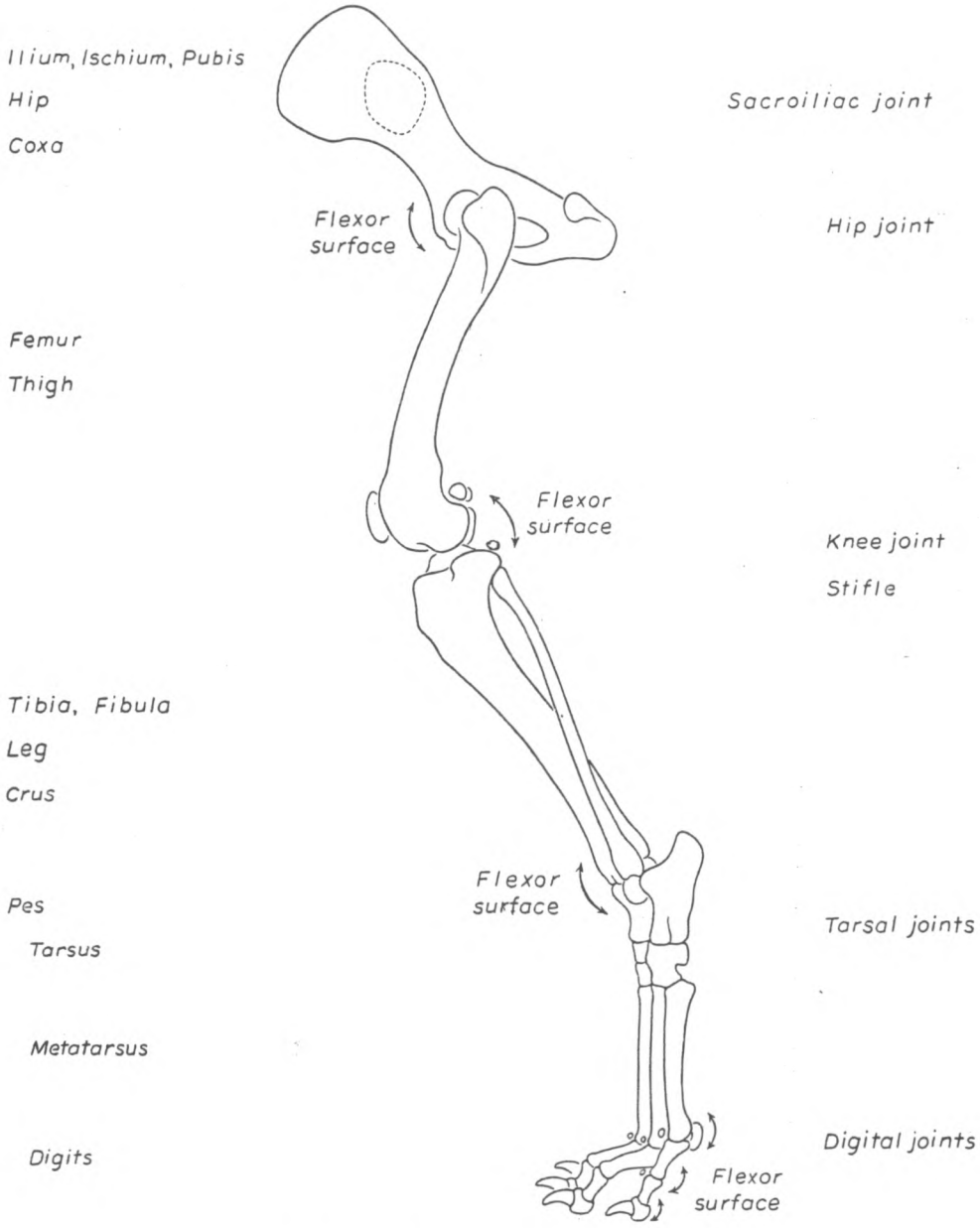
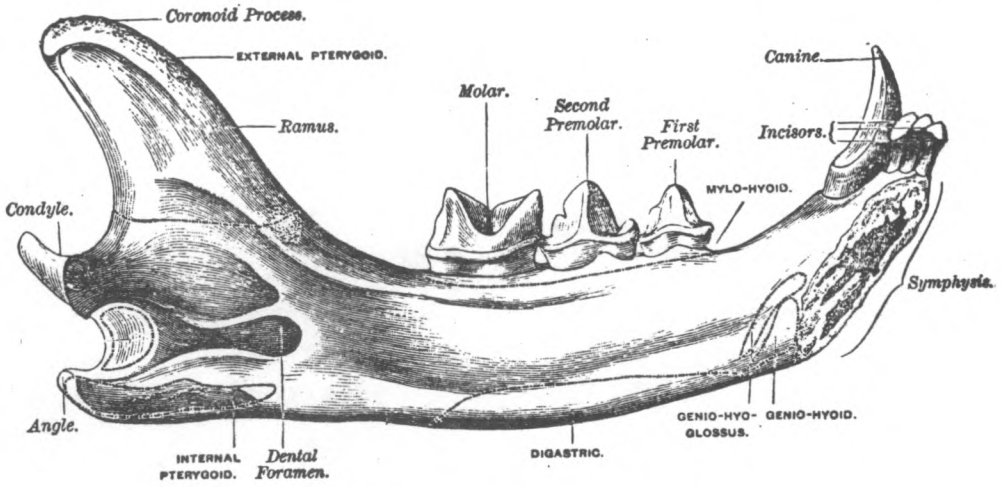


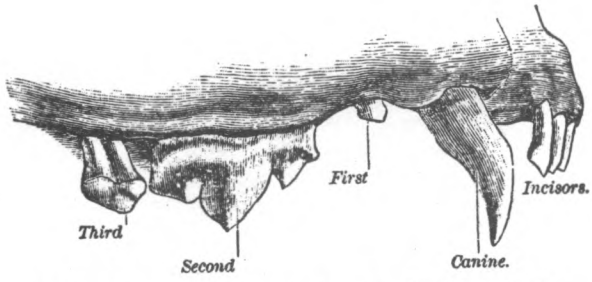
Figure 21. Parts of left pelvic limb.

from Evans and DeLahunta



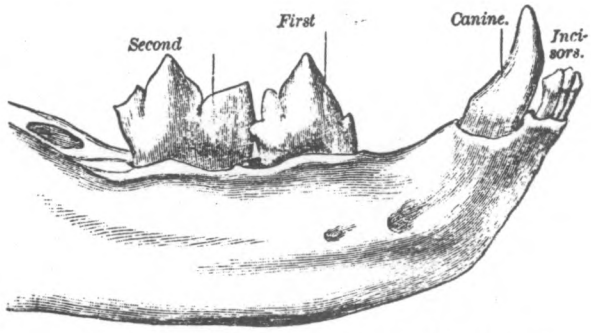
LOWER JAW, LEFT SIDE, INNER SURFACE.

Figure 22a. Lower jaw, left side, inner surface.



RIGHT UPPER DECIDUOUS TEETH. OUTER ASPECT.

Figure 22b. Right upper deciduous teeth. Outer aspect. (Kitten)



RIGHT LOWER DECIDUOUS TEETH. OUTER ASPECT.

Figure 22c. Right lower deciduous teeth. Outer aspect. (Kitten)

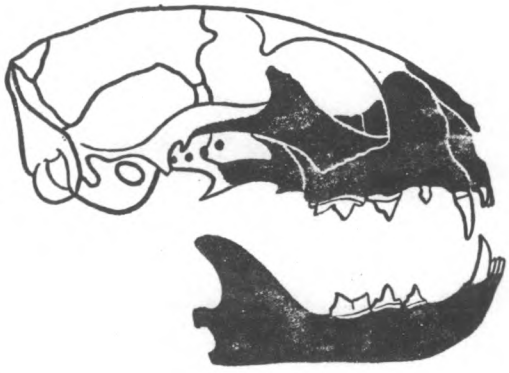


Figure 22d. Lateral aspect of skull, showing bones of maxilla and mandible.

Figure 22. Teeth.

	I	C	PM	M	
upper	3	1	3	1	= 30
lower	3	1	2	1	

From Jayne.

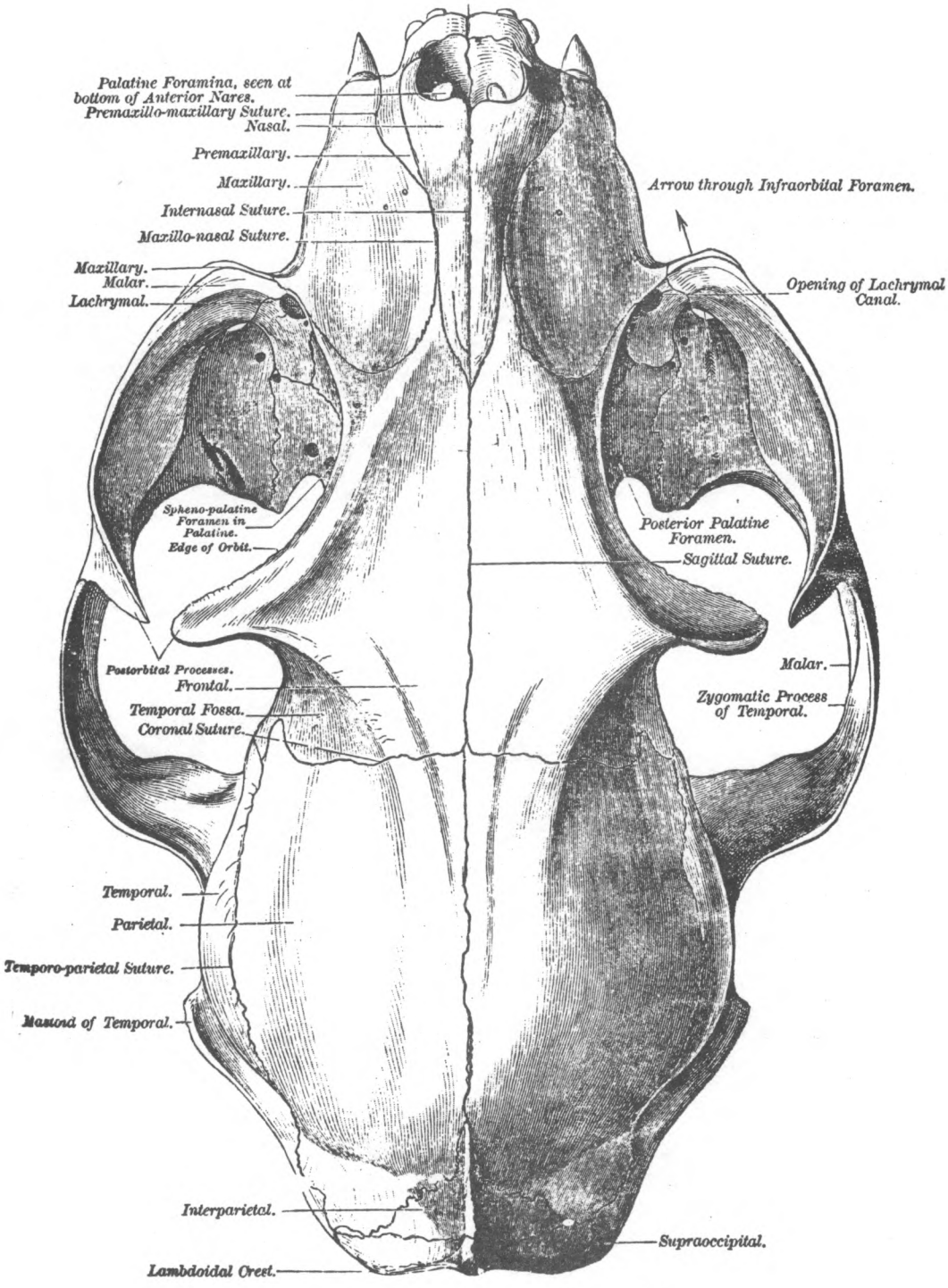


Figure 23. Dorsal view of skull.

from Jayne

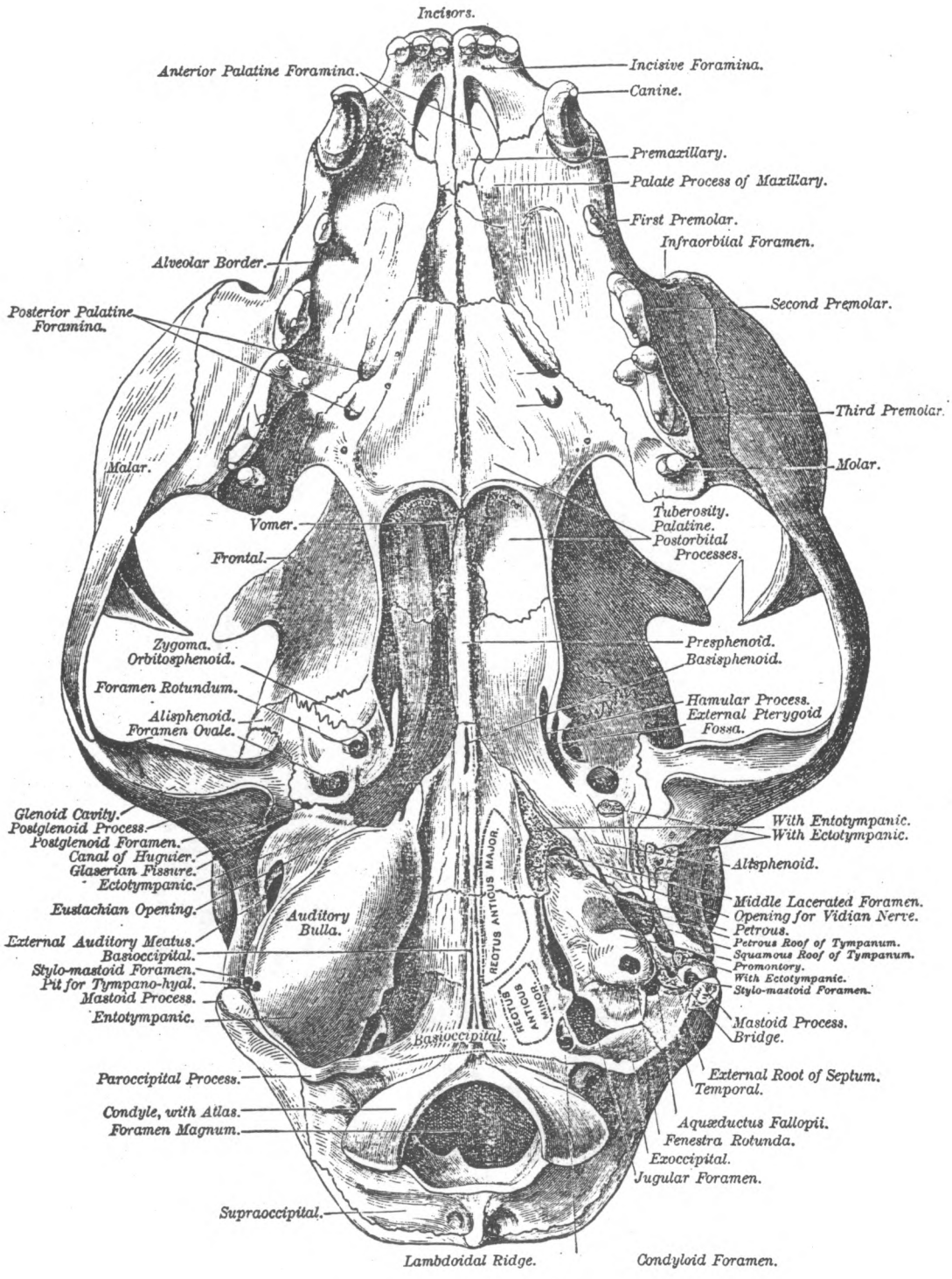


Figure 24. Ventral view of skull.

from Jayne

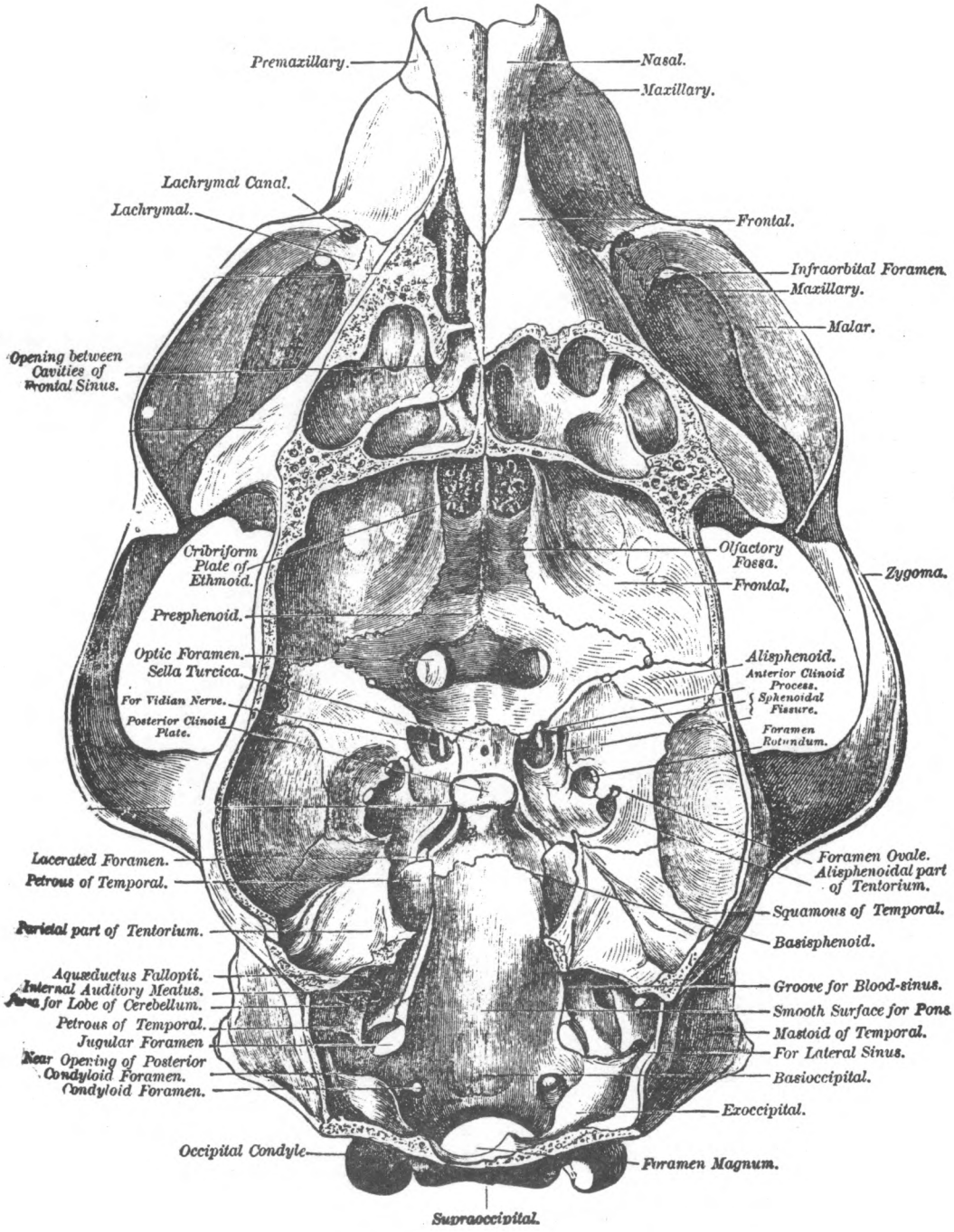


Figure 25. Floor of brain case (calvaria removed).

from Jayne

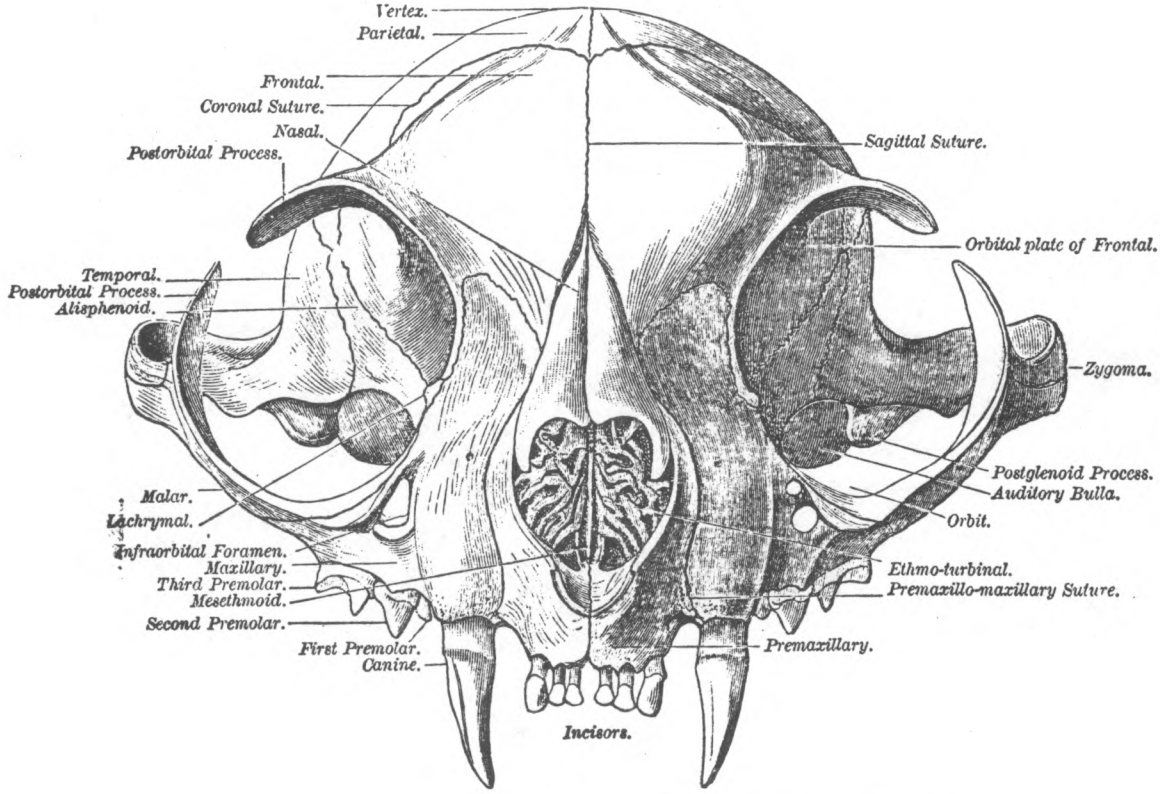


Figure 26. Cranial view of skull.

from Jayne

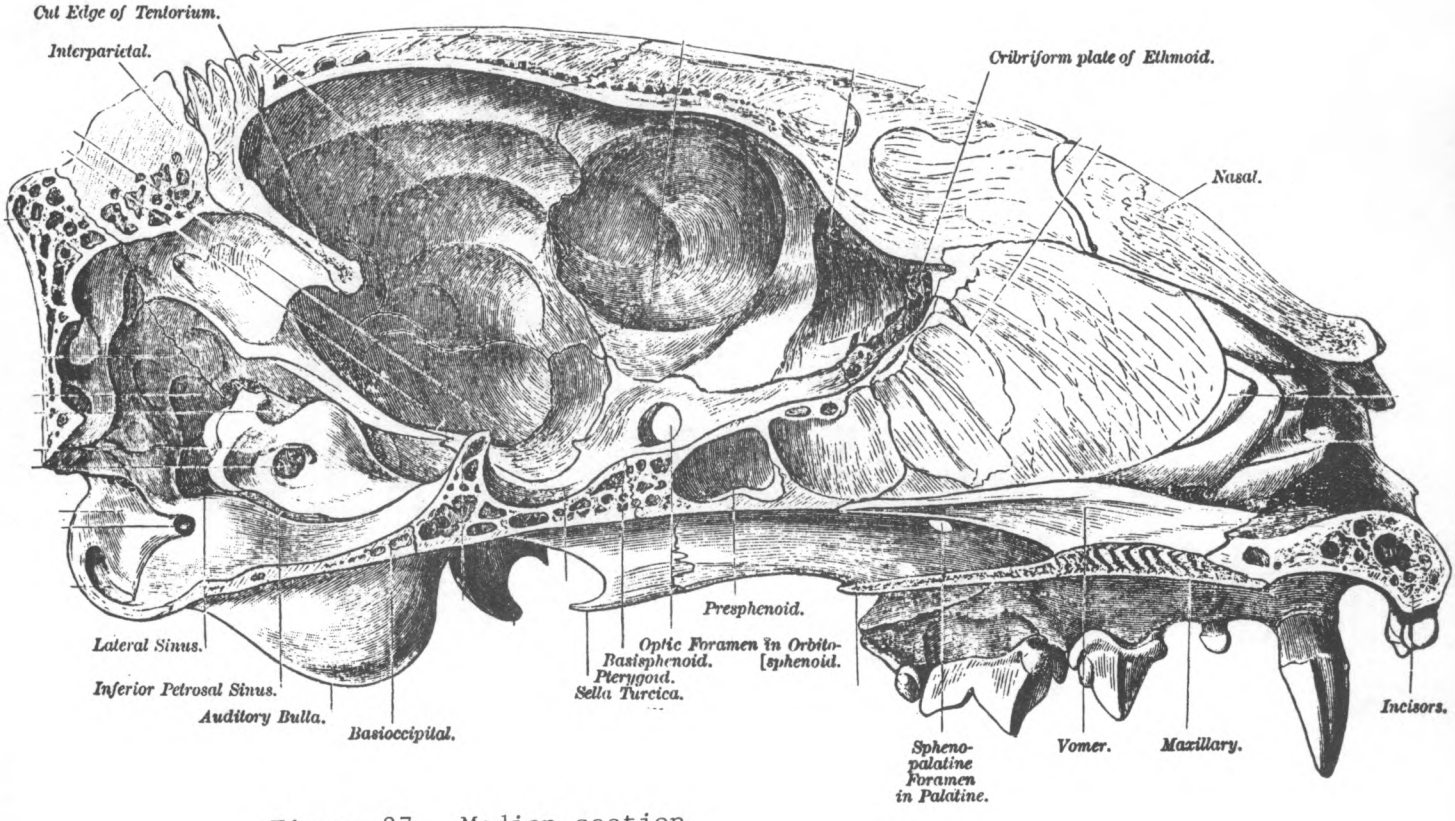


Figure 27. Median section, inner surface.

from Jayne

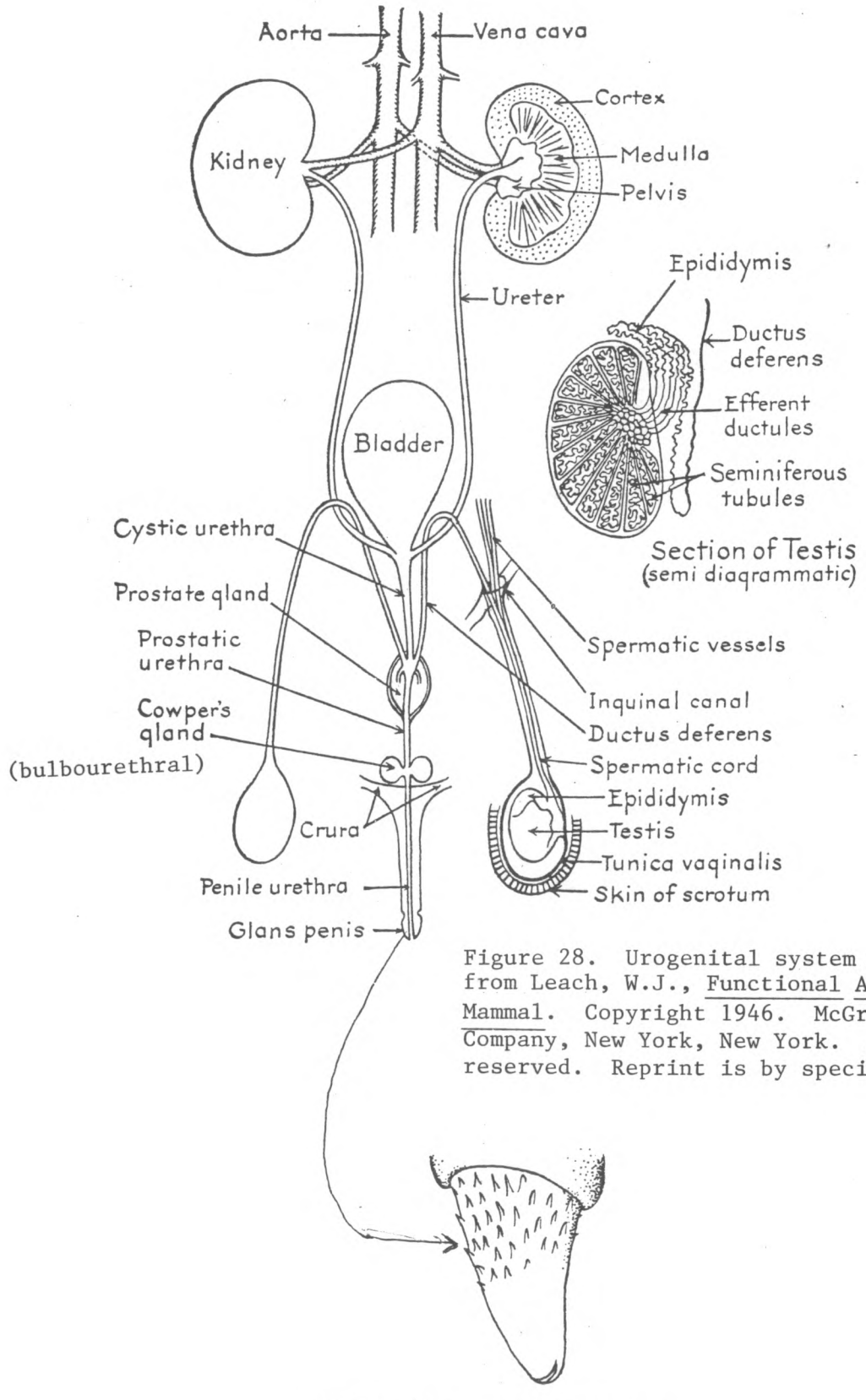


Figure 28. Urogenital system of the male cat. from Leach, W.J., Functional Anatomy of the Mammal. Copyright 1946. McGraw-Hill Book Company, New York, New York. All rights reserved. Reprint is by special permission.

Figure 28a. Spines on the glans are androgen dependent.

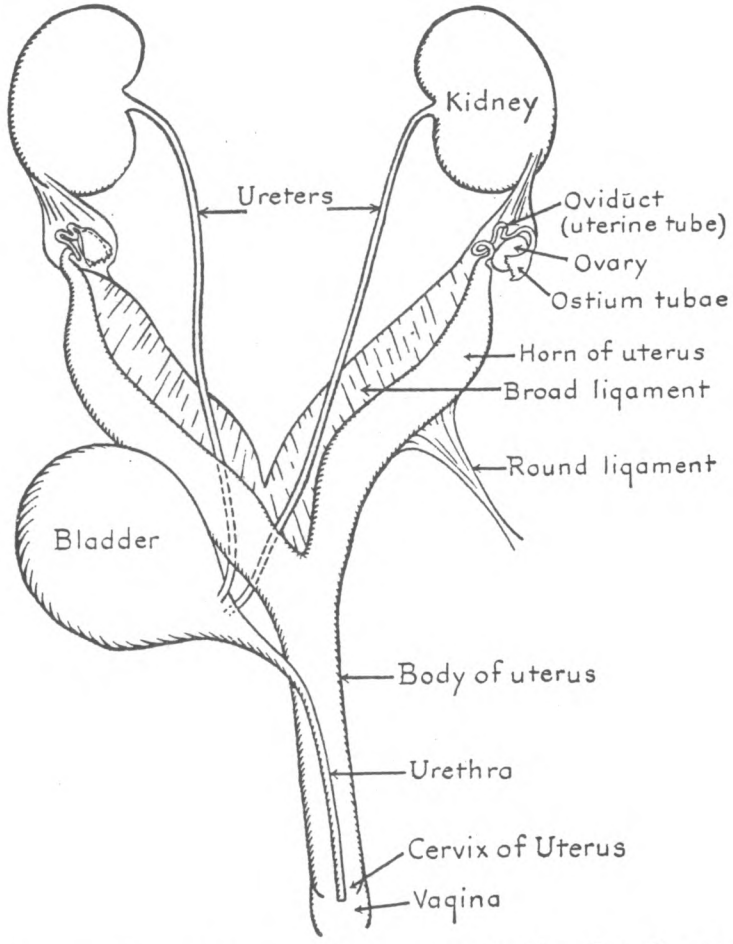


Figure 29. Urogenital system of the female cat. from Leach, W.J., Functional Anatomy of the Mammal. Copyright 1946. McGraw-Hill Book Company, New York, New York. All rights reserved. Reprint is by special permission.

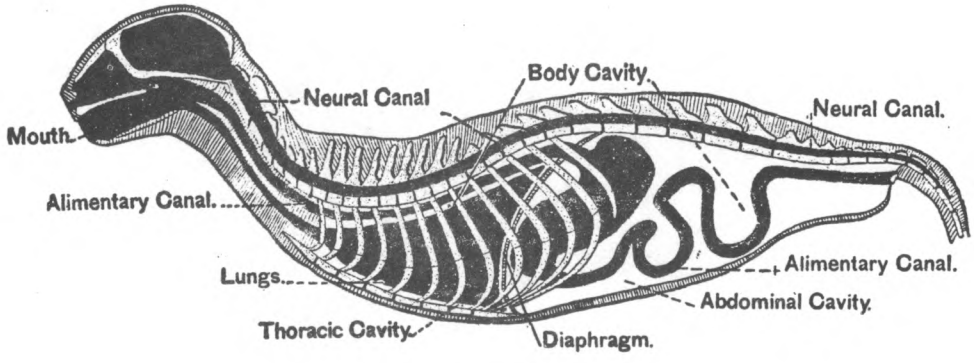


Figure 30. General Body Plan.

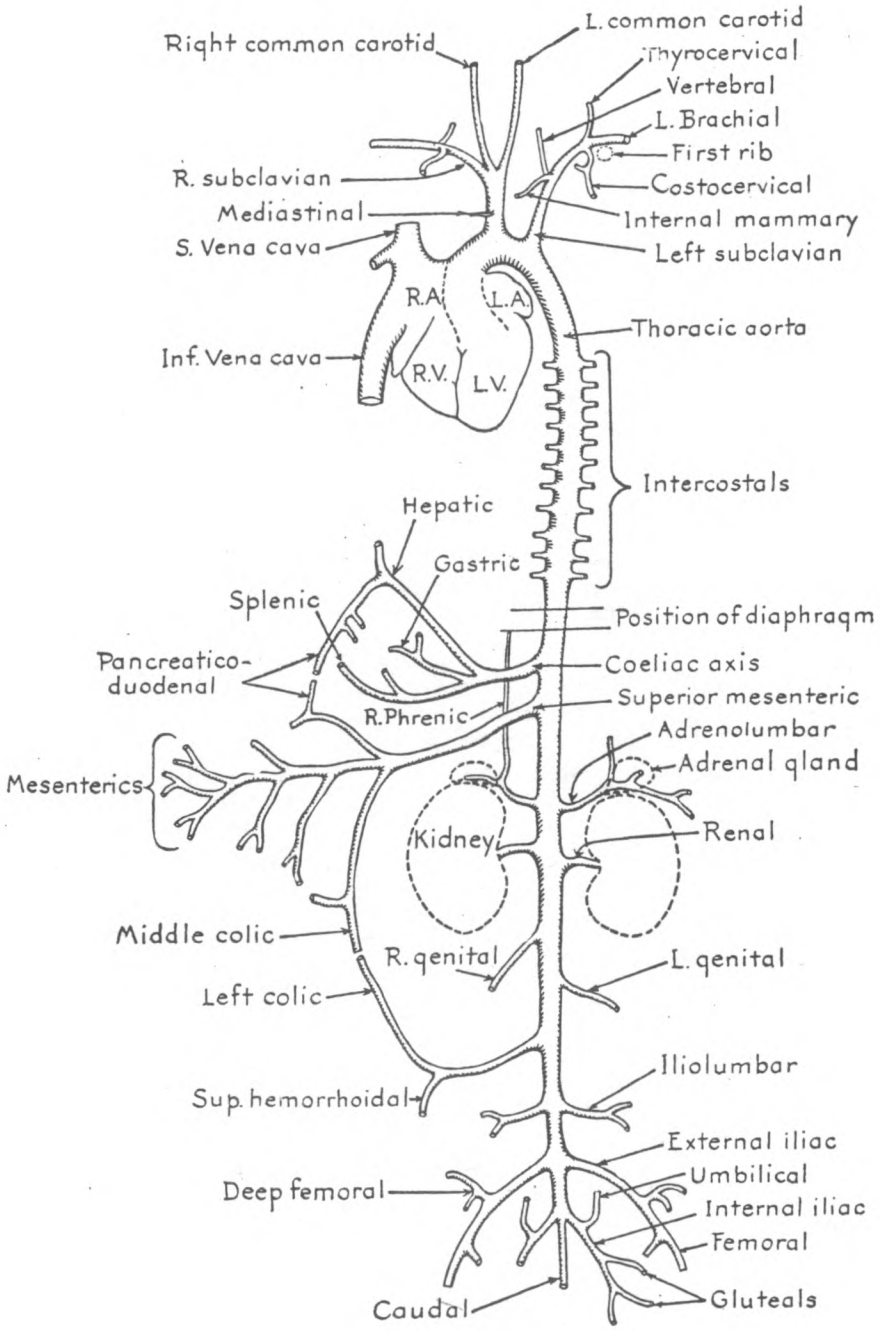


Figure 31. Principal systemic arteries of the cat (somewhat diagrammatic). from Leach, W.J., Functional Anatomy of the Mammal. Copyright 1946. McGraw-Hill Book Company, New York, New York. All rights reserved. Reprint is by special permission.

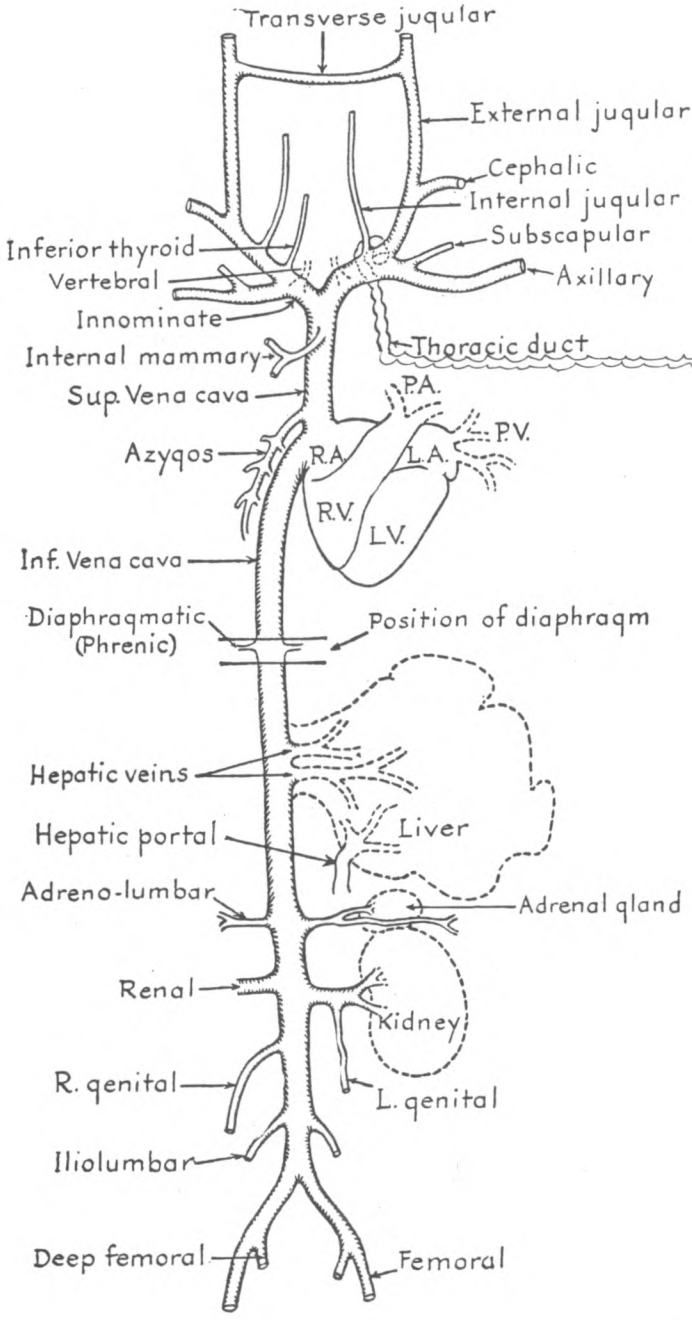


Figure 32. Principal systemic veins of the cat (somewhat diagrammatic). from Leach, W.J., Functional Anatomy of the Mammal. Copyright 1946. McGraw-Hill Book Company, New York, New York. All rights reserved. Reprint is by special permission.

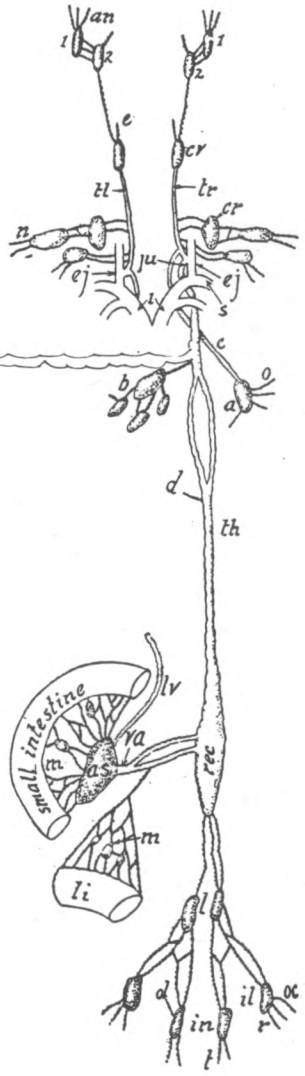


Figure 32a. Chief lymphatic vessels of the cat. Note that the thoracic duct enters the venous system.

from Davison

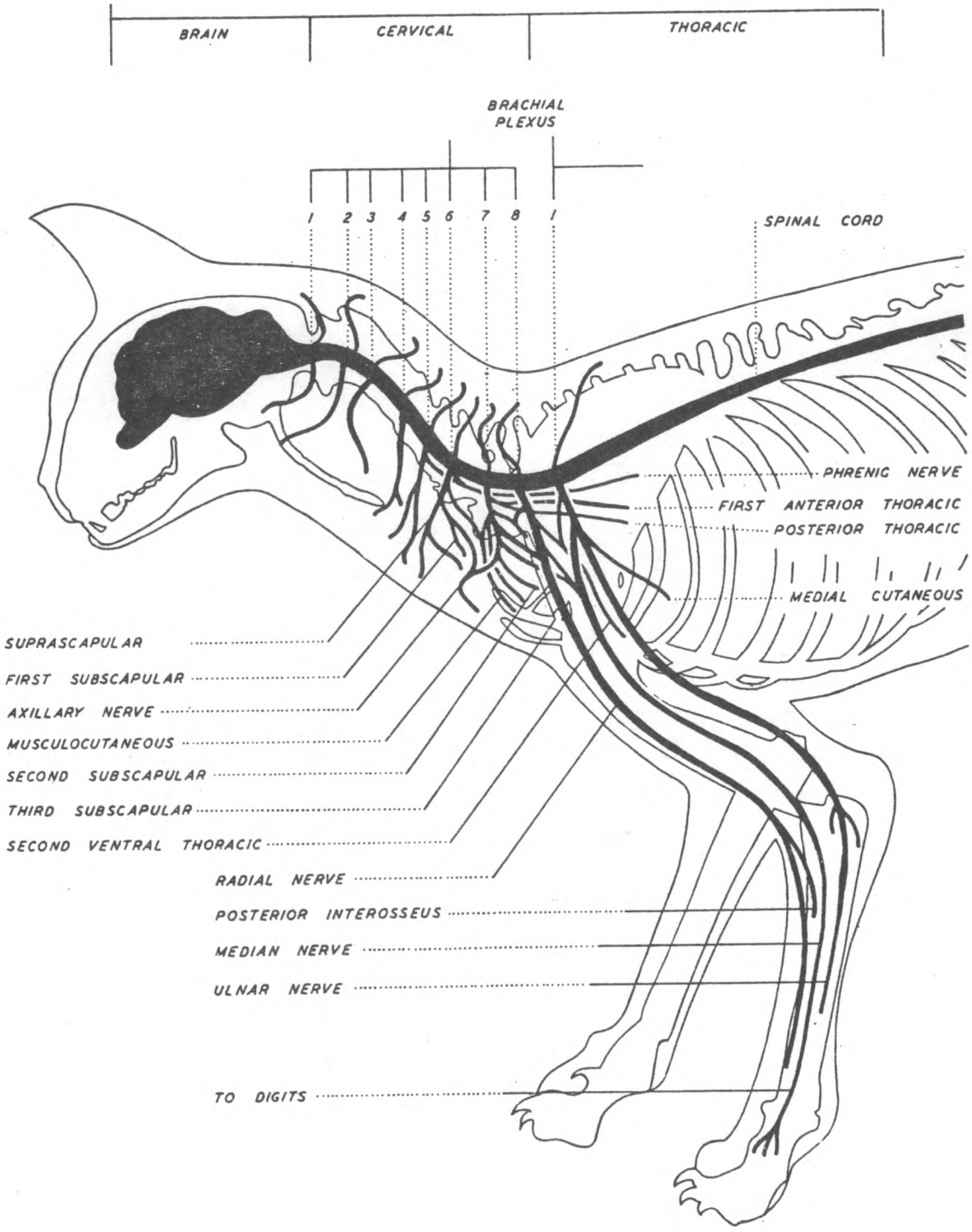


Figure 33a. The brain, spinal cord and brachial plexus. from Booth, Ernest S. Laboratory Anatomy of the Cat (3rd edition). Copyright 1948. Ernest S. Booth. Reprint is by special permission.

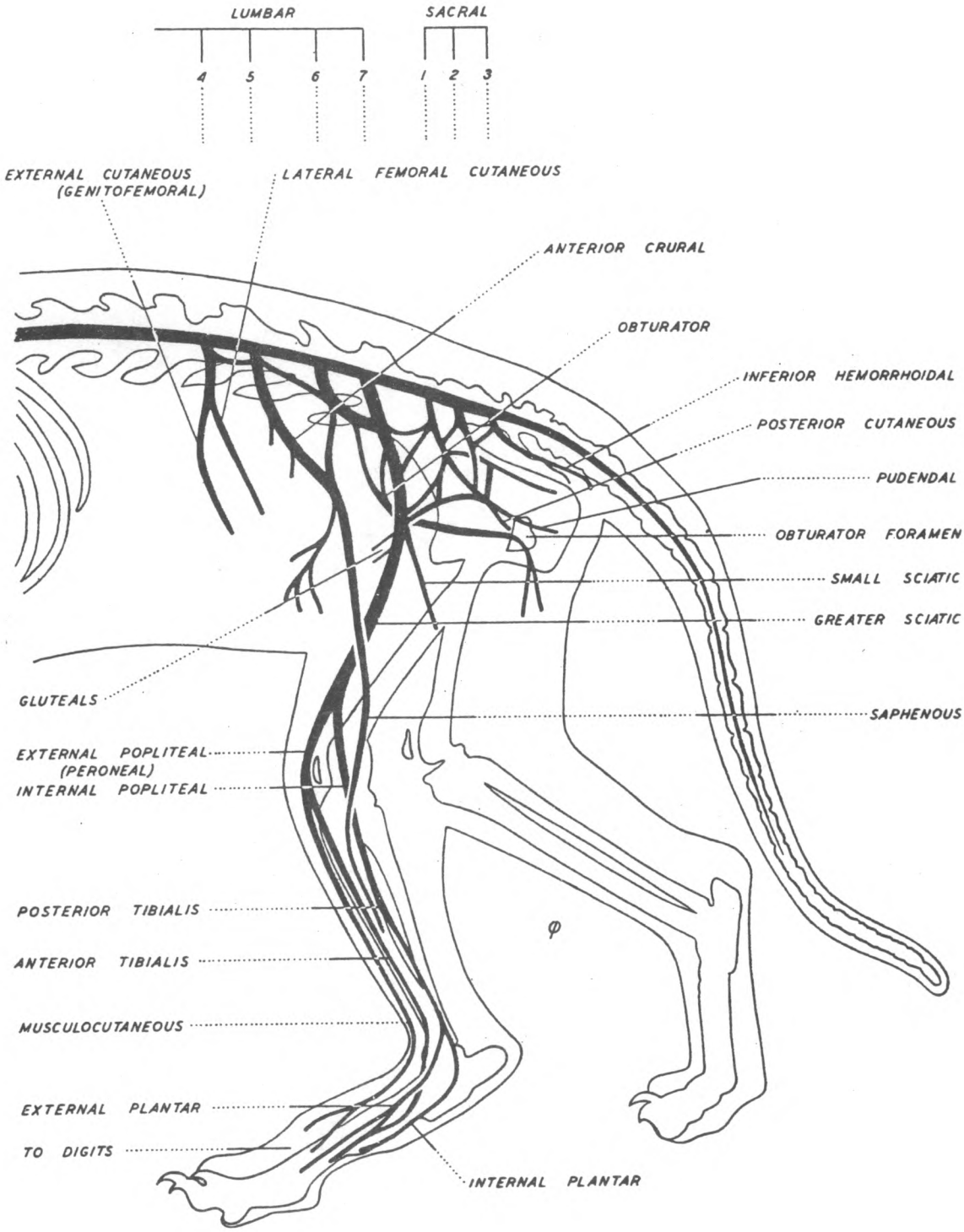


Figure 33b. The spinal cord and lumbosacral plexus. from Booth, Ernest S. Laboratory Anatomy of the Cat (3rd edition). Copyright 1948. Ernest S. Booth. Reprint is by special permission.

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FELINE REPRODUCTION:
PHYSIOLOGY AND REPRODUCTIVE DISEASES

Donald H. Lein, D.V.M., Ph.D.

FELINE REPRODUCTIVE PHYSIOLOGY

The queen attains puberty at 4-12 months of age with a minimum body weight of usually 2.3 to 2.5 kg. Puberty depends on growth rate and season of birth. Adequate nutrition, freedom from disease, late winter or early spring birth, the companionship of other sexually active queens and toms are necessary for early puberty. The tom cat is usually fertile at 6-8 months with a minimal body weight of 3.5 kg. The reproductive life may be 14 or more years, with a period of 8 to 10 years most suitable for continuous breeding of a queen. The litter size is reduced if the queen conceives on the first estrus or is an aged cat. The average litter size is about 4 kittens (1-8). Siamese cats average about 6 kittens.

The queen is seasonally polyestrous with a breeding season that usually starts in January and continues to September or October in the Northern Hemisphere. The queen is known as a "long day" breeder and is influenced by the lengthening of daylight during this period of the year. Twelve to 14 hours per day of continuous artificial light may cause continuous polyestrus with no anestrus period noted in the fall and winter. Less than 12 hours of artificial light per day in a cat that is not exposed to daylight may lead to anestrus regardless of the season. The tom also has depressed sexual activity in the fall months.

The queen is an induced ovulator, requiring coital or pseudocoital stimulation of the vulvovaginal area to ovulate. Spontaneous ovulation has been reported but appears to be an individual characteristic. Proestrus, which is exhibited by attraction of tom cats without sexual acceptance, takes place for 1 or 2 days. Estrus or sexual reception, expressed by the queen's change in personality, constant calling, rolling, rubbing against objects and exhibiting the crouched mating stance with treading of the hind legs and tail twitching, lordosis of the back and tipping of the pelvis upward, occurs for 3 to 6 days in mated queens and a variable period, 10 to 21 days, in unmated queens. Serum estradiol levels range from 5 to 20 pg/ml; however, peaks of 40 to 100 pg/ml are attained by the onset of estrus. This is followed by degeneration of the ovarian follicles in the unmated queen, resulting in an anestrus period that normally lasts for 3 to 21 days but may be as long as 30 days. Pseudopregnancy following a sterile mating, luteinizing hormone injection, or pseudocopulation, lasts from 20 to 70 days (average 45 days), followed by 7 to 10 days of anestrus before ovarian activity and a new cycle are initiated. The gestation period is 64 to 69 days. Lactational anestrus is common in queens until the kittens are weaned. A few will show estrus 7 to 10 days postpartum and may conceive.

A mating sequence involves the male neck biting, mounting, and pelvic thrusting, until intromission takes place. Intromission is brief, 15-20

seconds, with ejaculation taking place immediately. Neck biting to intromission can vary from 0.5 to 5 minutes. During intromission the queen screams, rolls and strikes at the male. The queen's after reaction may last from 1 to 9 minutes and consists of rolling and thrashing and obsessive licking of the vulvar area. Another mating sequence may occur immediately following the after reaction, or the female may refuse for several minutes to hours. Intervals are usually brief (5 to 30 minutes) and become longer as time increases. Queens have been reported to copulate up to 30 times in a day or 36 times in 36 hours.

Most queens will ovulate after 4 or more copulations, but less than 50% of fully estrous queens ovulate after a single copulation. The initial copulation causes LH release, although repeated copulations for one or 2 days or more cause negligible LH release after the first 2 to 4 hours of mating.

Corpora lutea form after the follicles rupture (ovulate) and luteinize within 1 to 3 days. Progesterone levels rise over 1 ng/ml. by day 3 after ovulation and reach peak levels of 15 to 90 ng/ml. by day 15 to 25. Progesterone levels in early pregnancy prior to implantation are not different from pseudopregnant levels, but by day 20, pregnant queens on the average have higher levels. By day 50, placental production of progesterone is adequate to maintain pregnancy and the ovaries can be removed.

Following copulation and ovulation, sperm and released ova (eggs) are transported to the uterine tube where fertilization takes place and remain here for about 4 days before entering the uterus. The embryos are usually evenly spaced in both uterine horns. Implantation occurs about day 12 to 14 of gestation with a "band" or zonary placenta formed.

Estrus behavior is estimated to occur in about 10% of pregnant queens with acceptance of a male, copulation and postcoital rolling occurring at about 21 or 42 days of pregnancy. It is believed that some queens may conceive on this breeding, resulting in a second litter (a phenomenon called superfetation).

Queening or parturition is accompanied with a decline in progesterone to basal levels (less than 1 ng/ml). Prolactin levels remain basal during the first half of pregnancy, (day 35), increase to reach 3 to 5 fold higher levels by day 63, and rise to peak levels just before parturition. Prolactin levels are elevated in response to suckling during lactation and decline to basal levels 2 weeks after weaning.

Estrus control in the queen using progestins has been successful in Europe and Australia. Megestrol acetate (ovaban) has been released for use only in dogs in the United States, but has been used successfully in the queen at 5 mg/day for 3 days, then 2.5 to 5 mg/week. Therapy should be only in young healthy queens. Progestin therapy may cause increasing appetite, a calming effect, increase body weight and, with prolonged treatment, cushingoid cats. Progestins should not be used in eats with genital tract infections, cystic endometrial hyperplasia - pyometra complex, or hyperplastic or neoplastic mammary gland problems.

MISMATING

Mismating of queens has been successful with estrogen therapy given within the first 6 days following copulation. Single low doses of estradiol cypionate, 0.125 to 0.25 mg. I.M, retarded egg transport through the oviduct. Queens may exhibit signs of estrus following estrogen treatment. Indiscriminate estrogen therapy may lead to bone marrow depression, thrombocytopenia, hemorrhagic shock and rapid death.

INDUCTION OF ESTRUS

Estrus has been induced in queens by daily injections of pregnant mare's serum gonadotropin (PMSG) for eight days or follicle stimulating hormone-pituitary (FSH-P) daily for 5 days. Luteining hormone may be given to enhance ovulation during mating. This procedure is usually followed by successful matings.

PYOMETRA

Pyometritis in the queen can be the result of septic conditions, post-breeding or post-parturition infections, or following pseudopregnancies resulting in cystic endometrial hyperplasia-endometritis-pyometritis complex. The affected queen may appear normal or may show various degrees of clinical signs, from simple vaginal discharges to possible fulminating septic acute endometritis, bloody discharges, depression, toxic bone marrow depression, and septicemia and death. Usually antibiotic therapy, fluid and supportive therapy and ovariohysterectomy are indicated. In queens intended only for further breeding use, supportive antibiotic and fluid therapy and prostaglandin F_2 alpha therapy have been beneficial in returning queens to reproductive soundness.

SELECTION OF QUEENS FOR BREEDING

Queens and tom cats for breeding should not be selected only by pedigree, conformation or show performance. Both sexes should also be selected from parents that show good sexual aggression, normal sexual cycles, good litter size, queen with ease and rear a healthy litter. These desirable characteristics are genetically based and can be inherited. Individuals selected for breeding should have a complete physical examination including the reproductive tract, mammary glands and all other body systems for evidence of any abnormalities. Records of all heats, breeding dates, queenings, litter size and rearing information should be strictly maintained and utilized when making decisions.

PREGNANCY DIAGNOSIS

Pregnancy should be determined as early as possible following mating. Early diagnosis will insure proper care and use of the queen through the gestation period and also will allow plans to be made for

queening and rearing the kittens. Pregnancy diagnosis can be accomplished by the following methods: (a) abdominal palpation of the pregnant uterus, individual fetuses and their fetal membranes from 20 to 30 days. As early as 16 days in a well relaxed queen, the beaded effect of the pregnant uterus can be detected; (b) x-ray of the abdomen from about 43 days to term; (c) ultrasonic Doppler detection of placental circulation and fetal heart beat from about the 4th week to term; (d) enlargement and pink color of the teats and mammary glands from about day 18 to term.

CARE DURING PREGNANCY

The pregnant queen needs a well balanced diet, with food provided so that as fetal growth causes increased fetal weight, maternal food intake increases automatically. Overfeeding and excessive weight gain should be avoided. Feeding recommendations of commercial cat food suppliers for pregnant and nursing queens should be followed. Advice and discretion should be sought before supplementation of vitamins, minerals, high proteins or fats is used.

Nonstrenuous daily exercise to keep good muscle tone is necessary for easy queening. Obesity and poor muscle tone can result in low conception rates and difficult queening.

Medication, vaccination and worming should be avoided during pregnancy with treatment being planned and performed prior to breeding if possible. A clean, warm, dry, secluded comfortable area with a large nesting box should be provided at least 10 days before queening. Near term the enlarged abdomen may cause restricted physical activity. A few days prior to queening, the mammary glands enlarge further and milk is present. The excessive hair in long haired breeds should be removed from around the teats and vulva prior to queening. The vulvar area, if soiled, should be cleansed just prior to queening.

Evidence of abnormal vulvar discharges, fetal resorption, abortions, premature kittens, stillborns or mummified fetuses or weak kittens with neonatal death should be carefully examined by a veterinarian and treated. Specimens of the vulvar discharge, blood samples and any fetal or placental tissue or dead kittens should be carefully handled and submitted to a veterinary diagnostic laboratory for examination and hopefully a definitive diagnosis so that meaningful treatment, control, and/or preventive measures can be instituted. The queen and any remaining kittens should be strictly isolated from other cats, especially pregnant queens, since a contagious agent may be involved.

Feline viral rhinotracheitis, panleukopenia, toxoplasmosis and bacterial agents can cause the above types of fetal loss. Feline leukemia virus and feline infectious peritonitis virus have also been associated with this syndrome.

Diseases that primarily affect other body systems may secondarily affect the pregnant uterus. Any sickness should be examined by a veterinarian, treated and recorded.

QUEENING

Nesting may be exhibited 12 to 24 hours before queening. The rectal temperature falls in the first stage of labor. Normal presentation of the fetuses can be either forward or backward as they enter the birth canal. Thirty to sixty minute intervals are frequently seen between the delivery of 1 or 2 kittens. During this time the queen is removing the placenta and cord from the kitten, cleaning and stimulating the kitten to breathe and move. The placentas are ingested by the queen and the vulvar area is cleansed. The kittens may be nursed before delivery of the next. Occasionally, there may be a 12 to 24 hour delay following the delivery of 2 or 3 kittens before the rest are born. This is especially seen when a queen is interrupted and, if free, she may move her litter to another secluded spot.

The first litter of a queen with only one or two large kittens may be difficult and assistance may be needed. Healthy queens seldom have difficulty with delivery. Queens that are inbred, have nutritional deficiencies or the stress of a disease may have poor uterine contractions (inertia) and require assistance. Trauma or a nutritional deficiency may result in a pelvic deformity causing difficult delivery. Veterinary assistance for surgical intervention or medication with uterine muscle stimulating and contracting agents will be needed for difficult births.

Stillborn kittens and placentas are usually eaten by the queen. Cannibalism is seen, but it is more likely in the first queening, especially in highly nervous queens. These queens should not be used for breeding.

Postpartum hemorrhage, retained fetal membranes and/or dead kittens, and uterine inversion are uncommon in the queen. Oxytocin, a uterine contracting agent, may be used to control hemorrhage. Following uterine prolapse, treatment of shock or abdominal surgery with possible removal of the reproductive tract may be needed. Systemic antibiotics may also be indicated. A brown vaginal discharge and enlarged segments of the uterine horns indicate a retained fetus and/or placenta(s). Medical or surgical removal will be needed to correct this condition.

It is desirable to observe the vulva and surrounding areas of the queen daily for evidence of abnormal discharges. A bloody discharge is usually present for 7 to 10 days post-queening. Persistent discharges that are odorous or bloody indicate uterine infections. The mammary glands should also be examined for evidence of mastitis.

Queens with the above postpartum problems may be listless and unable to nurse or care for their kittens. Immediate diagnosis, treatment and supplemental care for the kittens are required or loss of both the queen and kittens may occur. Both the queen and frequently the kittens need antibiotic and supportive therapy. Antibiotic sensitivity tests for organisms cultured from vulvar discharges or mastitic milk are important. Separation of the queen and kittens will depend on the severity of the condition and her ability to care for the kittens and the contagiousness of her infection.

MATERNAL CARE AND LACTATION

The queen usually remains continuously with her kittens for a period of 24 to 48 hours. About 2 to 3 ml. of milk are taken by the kittens 3 times an hour. Kittens double their weight in 7 days and open their eyes. They are able to take 4 to 7 ml. of milk at a feeding in the second week. At this time, the queen will leave the nest for several hours. In her absence the kittens will sleep quietly if normal. After feeding, the queen washes each kitten, especially around its head and anal region. She consumes the urine and feces voided in response to grooming. By 3 weeks of age the kittens have increased their activity to exploring and playing. The queen then teaches the kittens to urinate and defecate away from the nest. Lactation is then supplemented by solid food beginning the 4th week and weaning is completed by the 7th or 8th week.

Queens with strong maternal instinct and good milk supply are capable of fostering orphaned kittens or will allow kittens from their previous litters to nurse. Orphan kittens can be raised on commercial replacement diets (follow manufacturer's instructions for amount and frequency of feeding). Stimulation and cleaning for urine voiding and defecation with warm wet cotton swabs must follow each feeding and a warm area must be provided for very young kittens to maintain body temperature. Daily weights should be recorded. Kittens that are not gaining daily or that are losing weight should be examined and treated immediately by a veterinarian.

REPRODUCTIVE PHYSIOLOGY AND INFERTILITY OF THE TOM

The male is born with descended testes. Cryptorchid or ectopic testes should not be corrected, but removed when the tom has reached appropriate age, size and maturity, since this condition may be inherited.

Spermatozoa are present in the seminiferous tubules of well grown toms by 24 to 36 weeks of age. The average volume of an ejaculate is about 0.05 ml. (0.03 -0.3 ml.), and contains from 60 x 10^6 to 12 or 15 x 10^8 sperm/ml. Aproximately 80 to 90% motility is present. The pH of fresh semen is 7.4. less than 10% abnormal sperm are seen in normal ejaculates.

Semen can be collected with an artificial vagina or by electroejaculation while the tom is anesthetized. The tom must be trained (3 weeks +) to collect with an artificial vagina, which consists of a 2 ml. rubber bulb with the end cut off and fitted over a 3 x 44 mm. test tube. The unit is placed into a small polyethylene bottle with warm water to produce a temperature at collection of 45°C. A female in estrus or an ovariectomized female which has been estrogenized should be used as a teaser and mount. Collection takes 1 to 4 minutes. Semen can be obtained by flushing the vagina of a queen immediately following copulation, but the quality of the specimen is poor.

A tom will defend and mark a territory. The territory will be marked by spraying a mixture of urine and a secretion containing valeric acid

from the anal glands that produces the characteristic penetrating "catty" urine odor. Urine spraying may be inhibited by castration in the complete male or controlled by progesterone therapy in the complete or castrated tom.

Infertility in the tom is infrequently seen or reported, although purebred or inbred toms should be examined closely. The role of the tom in possible venereal spread of infectious diseases is unknown. A negative status for toxoplasmosis, feline leukemia and feline infectious peritonitis would be important when obtaining a breeding tom. Adequate vaccination should be carried out prior to breeding in order to control disease.

Bite wounds of the scrotum and testes are frequently found following territorial fights. A febrile response and scrotal abcessation should be treated by a veterinarian and could result in the removal of the affected testis to save the nonaffected testis from heat degeneration and extension of the infection from the affected testis.

A hair ring may form around the base of the glans penis following several matings, resulting in difficult intromission and mating. If the tom does not remove this ring during routine cleaning, it must be removed by retracting the preputial sheath and sliding the hair ring over the penis. Successful mating is immediately possible.

Psychological infertility may be seen in tom cats undergoing environmental changes, territorial changes or in young novice males placed with aggressive experienced queens. Time and acclimation may result in acceptance and a return of fertility. If not, the tom may have to be removed to a less hostile environment.

Congenital testicular hypoplasia may be a consequence of fetal or neonatal panleukopenia. Poor libido and variable testicular degeneration leading to aspermatogenesis has been associated with malnutrition, obesity, hypervitaminosis A caused by diets containing excessive liver, and hypothyroidism. Proper diagnosis of the condition and treatment of the cause usually results in normal fertility with time.

The tortoiseshell male cat is reportedly sterile, but a few are reported fertile. The orange and black color genes are within the X chromosome and are sex-linked. The white gene is a dominant gene S for piebald and may mix with orange and black producing the "calico" or "tricolor" pattern. Tortoiseshell cats therefore should be heterozygous for two sex-linked alleles and should therefore have two X chromosomes. They are usually females, but exceptional male torties occur. Blood, bone marrow and skin cultures of these males show a karyotype of XXY or XX/XY/XXY mosaicism. These male cats have small testicles, lack libido, are sterile, and contain a Barr body at the nuclear membrane in smears from buccal cells or drumstick polymorphonuclear leucocytes (expressions of extra X chromosome). This syndrome is homologous to the Klinefelter's syndrome in man. Fertile tortoiseshell males may have XX/XY mosaic karyotypes or possibly a normal XY component with somatic mutation occurring in the X cells so that some express orange, others black.

FELINE GENETICS

Judith Kinnear, Ph.D.

Cats appear to have shared human households for several thousands of years, as testified by art work from ancient Egypt. For example, the wall painting "Fowling in the Marshes" from the tomb of Nebamun at Thebes (3400 B.P.*) shows a cat with a family boating on the Nile; as the man beats the papyrus reeds, the cat is adeptly catching birds. A painting on parchment now held in the British Museum shows a cat under a chair eating a bone. This cat of ancient Egypt was a brownish mackerel tabby.

From Egypt, the cat spread to other regions: Greece (2500 B.P.), China (2200 B.P.), Italy (1900 B.P.), Britain (1600 B.P.), and it reached the New World through the voyages of European seafarers.

Changes have occurred in the genetic makeup of the cat throughout its history. Some of these changes or gene mutations have produced visible alterations in coat color and pattern, and in quality and composition of the coat. Some mutations do not markedly reduce survival and reproductive capability. The time of occurrence of those mutations may be inferred from their frequencies and distributions throughout the world.

"Ancient" mutations include:

1. *A* (agouti) ⟶ *a* (non-agouti)

   The gene mutation from *A* to *a* led to the appearance of cats lacking tabby patterning. Such cats are typified by Mr. Mistoffelees, who...

   "is quiet and small, he is black
   From his ears to the tip of his tail;
   He can creep through the tiniest crack
   He can walk on the narrowest rail."[1]

2. *D* (dense coloring) ⟶ *d* (dilute)

   The mutation from *D* to *d* led to the appearance of cats with dilute coloring, such as blues and creams, and later the lilacs.

3. *s* (non spotted) ⟶ *S* (spotted)

   This gene mutation from *s* to *S* produced cats with areas of white on their coats, such as Jellicle cats:

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*Before the Present time.

[1]T.S. Eliot, "Mr. Mistoffelees," in Old Possum's Book of Practical Cats. London: Faber & Faber, 1939.

"Jellicle Cats are black and white
Jellicle Cats are of moderate size
Jellicles jump like a jumping-jack
Jellicle Cats have moonlit eyes."[2]

These ancient mutations which are widespread through cat populations provide a contrast with recent gene mutations which are restricted in their occurrence.

Recent mutations include:

1. *R* (normal coat) ⟶ *r* (Cornish rex)

   This mutation was first detected in Cornwall in 1950; it is responsible for the soft and curly coat seen in Cornish rex cats.

2. *wh* (normal coat) ⟶ *Wh* (wirehaired)

   This mutation produces the wire coated trait and it was first detected in New York in 1966.

The many mutations which have occurred in the history of the cat are responsible for the inherited variation in this species. It is this genetic variation that differentiates the distinct breeds of cats and permits the range of color variants within a breed. So, from the prototypic brownish mackerel tabby of ancient Egypt we now have a diversity of appearances.

Some people recognize a very restricted range of variation in cats. One of the most succinct statements is:

"As far as I'm concerned there are two kinds of cats:
city cats and others."[3]

Others see a great diversity:

"I have consorted with practically all kinds (of cats), of every color and description: short haired, long haired, alley cat, Siamese, Abyssinian, tabby, tiger, black, white, orange, and every combination you can imagine; good cat, bad cat, sweet cat, sour cat, dull ones, bright ones, pompous and stuffy cats and gay ones with an enchanting sense of humor. For cats vary, as do people, in their characters and characteristics..."[4]

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[2] T.S. Eliot, "The Song of the Jellicles," in Old Possum's Book of Practical Cats. London: Faber & Faber, 1939.

[3] L.J. Camuti, All My Patients Are Under the Bed. New York: Simon & Schuster, 1980.

[4] P. Gallico, Honourable Cat. London: Heinemann, 1972.

At a more official level, the variation recognized by the Cat Fancy has increased from 25 classes at the first Cat Show held at Crystal Palace in 1871 to approximately 75 today.

The concept of cat breeds was first recognized in the mid-nineteenth century, when names and standards were first adopted. Since that time, breeders have been concerned to refine and perfect each breed, and their efforts have been directed towards the attainment of excellence in physical appearance - as exhibited in coat color and quality, head and body conformation - and in temperament, general health, vigor, and fertility.

The character and characteristics of a cat are the product of its genotype, that is the genetic endowment inherited from its sire and dam, an of its environment. Environmental factors - such as nutrition, both ante- and post-natal, disease, etc. - influence how fully the genetic potential will be attained.

To achieve the goal of excellence in a breed, breeders are faced with the challenge of manipulating and recombining combinations of major genes, modifier genes and polygenes. This process involves:

- selection of appropriate stock for breeding;
- choice of a suitable breeding program which may be a form of inbreeding or may involve outcrossing;
- culling of offspring;

and must be carried out under conditions which maximize the positive effects of environmental factors.

SOME BASIC PRINCIPLES

The information which controls inherited traits - physical, biochemical and physiological - is encoded in genes. Genes are composed of the chemical substance called deoxyribonucleic acid (DNA). This genetic material makes up the chromosomes which can be seen under certain conditions in the cells of a cat. Any one chromosome contains many hundreds or even thousands of different genes. An examination of the chromosomes from cells, such as skin cells or white blood cells of a normal cat, reveals that there are 38 chromosomes present in each cell. These 38 chromosomes consist of 18 pairs of autosomes and 1 pair of sex chromosomes. In the case of a normal male cat, the pair of sex chromosomes is distinctive in that the two members of the pair are dissimilar, and consist of one longer X-chromosome and one shorter Y-chromosome. In a normal female cat, the pair of sex chromosomes consists of 2 X-chromosomes.

The genetic endowment is transmitted from a parent to its offspring via the chromosomes of the gamete which it contributes to its offspring. The gamete contributed by the female parent (dam) is the egg; that contributed by the male parent (sire) is the sperm. Gametes are produced in the ovary or the testis by a special process of cell formation known as meiosis. This process results in a precise halving of the chromosome number so that

the gametes that are produced by this process contain one member only of each pair of chromosomes.

For each pair of chromosomes, it is equally likely that either member of the pair will be incorporated into a gamete. In consequence, each gamete contains 19 chromosomes; each egg contains 18 different autosomes plus one X-chromosome, while each sperm has 19 chromosomes, consisting of 18 different autosomes and either an X-chromosome or Y-chromosome. Different offspring from the same parents will receive different combinations of parental chromosomes.

Genes have specific functions; for example, there is a gene which controls fur length; another gene controls the pattern of tabby striping. Each gene may have several alternative forms, which are known as alleles. The gene which controls tabby striping has the alternative forms or alleles:

Abyssinian tabby, denoted T^A
mackerel tabby, denoted T
blotched tabby, denoted t^b

Since chromosomes occur in pairs, and because genes occur on chromosomes, each cat has two copies of every gene (except for genes carried on the X-chromosome in normal males).

If a cat has two identical alleles, e.g., T^AT^A or t^bt^b, it is said to be homozygous.

If a cat has two different alleles, e.g., T^AT^b or $T\ t^b$, it is said to be heterozygous.

A heterozygous cat with the genetic information or genotype $T\ t^b$ has two conflicting sets of information:

T : "produce mackerel stripes"
and t^b : "produce blotched stripes"

In this heterozygote, only one of the messages is expressed, namely, mackerel stripes. The trait that is expressed in the heterozygote is said to be dominant; the trait that is "hidden" in the heterozygote is said to be recessive.

From this example, it can be seen that if a cat displays a mackerel pattern, its genotype may be either homozygous TT or heterozygous $T\ t^b$. However, if one of the parents of a mackerel tabby was known to be blotched, then the genotype of the mackerel offspring could be assigned with certainty as $T\ t^b$.

When gametes are formed by meiosis, only one of each pair of chromosomes passes to a particular gamete. In consequence, for each gene, only one of each pair of alleles is found in a particular gamete. A cat that is homozygous for a given gene, e.g., TT, will produce gametes of one kind only with regard to the trait under consideration: T. A cat that is heter-

ozygous, e.g., $T\ t^b$, can produce two kinds of gametes with respect to that gene: T and t^b. Each type of gamete is equally likely to be formed.

The genotype or genetic makeup of a cat cannot be directly observed. However, it is possible to observe, measure or detect the expression of that genotype in the visible appearance or phenotype of a cat. So we cannot see the genotype (the two $t^b t^b$ alleles in the chromosomes of a blotched tabby cat) but we can observe its blotched tabby pattern. From the observed phenotype, we can infer or deduce possible genotype(s).

A LITANY OF MAJOR GENES

This section will introduce some of the major genes of the cat in order to illustrate aspects of gene action and interaction.

A Gene for Color

One gene in cats has the alleles

B	black
b	brown
b^l	light brown

The order of dominance is black > brown > light brown. A cat can be one of six different genotypes. If a cat's genotype includes at least one copy of the B allele (e.g., BB or Bb), it has the potential to show black coloring. The B allele is responsible for the black color of the all-black cat, for the black areas of the black and white bicolor, for the dark color of the mask, ears, legs and tail of the seal-point siamese cat, and for the color of the black stripes of the tabby cat.

The phenotypic expression of the bb genotype is seen in the brown color of the points of the chocolate-point siamese and in the warm, rich color of the havana brown cat.

The phenotypic expression of the $b^l b^l$ can be seen in the sorrel Abyssinian cat.

Consider a cat, genotype BB. What will it look like?

That information alone is insufficient to allow a decision. Such a cat may be, *inter alia*,

a solid black cat
or a seal point siamese
or a brown burmese
or a bicolored black and white cat
or a mackerel tabby with black stripes

In each case, the B-genotype results in black coloring on all or part of the coat. But:

(1) where the dark coloring appears,

(2) how intensely it is expressed,
(3) whether it appears alone or in combination with other colors

depend on other genetic information carried by the cat.

Observations of this nature lead to the general rule:

> Genes do not act in isolation; rather the phenotypic expression of a genotype depends on other genetic information also present.

Another general rule to recall here is that:

> A cat which displays a dominant trait, e.g., black, may also be carrying the allele which determines the recessive trait, e.g., brown. This allele can be "hidden" for several generations. It is by test crossing that status in terms of carrier or noncarrier can be investigated:
>
> e.g., *B?* X *bb*
>
> Carrier status (*Bb*) is identified with certainty if even one kitten showing the recessive trait is produced. Carrier status can be rendered less likely if only kittens showing the dominant trait are produced; there is a probability of error which decreases as the number of kittens produced increases. This testing may also involve the crossing of a suspected carrier with a known carrier for the recessive trait.

An X-Linked Gene for Color

The color of a cat's coat is basically due to the presence/absence of two pigments:

eumelanin
and phaeomelanin

Eumelanin is a dark pigment found in black or brown areas of the coat; phaeomelanin is a yellow-red pigment found in orange areas. If neither pigment is present the coat will be white.

There is a gene which controls the production of orange pigment. The gene is carried on the X-chromosome, and is said to be X-linked. This gene has the alternative alleles:

O^o orange
O^+ non-orange

(The color of a "non-orange" cat will be determined by other elements of the cat's genotype, e.g., $B/b/b^l$ alleles.)

A female cat with her two X-chromosomes may be one of three possible genotypes; a normal male cat has one X-chromosome only and may be one of two possible genotypes. These possibilities are shown in the following table:

Sex	Sex Chromosome Constitution	Possible Genotypes	Phenotypes
female	XX	$O^{o}O^{o}$	orange
		$O^{o}O^{+}$	orange and non-orange (e.g., black) tortie
		$O^{+}O^{+}$	black
male	XY	O^{o}	orange
		O^{+}	non-orange (e.g., black)

The tortoiseshell phenotype cannot occur in a normal male cat. The rare male tortoiseshell cats which are occasionally observed are found to have a chromosomal anomaly. Most commonly, the males are found to have a total of 39 chromosomes per cell including an extra X-chromosome. These XXY tortie males are as a rule sterile.

The asymmetric mosaic distribution of orange and non-orange patches seen in the tortie female is brought about by the process of X-chromosome inactivation, a mechanism which results in the "switching-off" of the genetic information carried on one of the two X-chromosomes present in each somatic cell. The process of X-inactivation will not be described further here.

Consider the mating of a tortie female (genotype $O^{+}O^{o}$) and a black male (genotype O^{+}). The tortie female can produce two kinds of eggs, namely eggs carrying O^{+} and eggs carrying O^{o}. The black male will produce two kinds of sperm, namely sperm carrying O^{+} and sperm containing a Y-chromosome. Random combination of eggs with sperm can produce four kinds of offspring, with equal probability of occurrence:

tortie female	$O^{+}O^{o}$
orange male	$O^{o}(Y)$
black female	$O^{+}O^{+}$
black male	$O^{+}(Y)$

It may be confidently predicted that any orange kitten produced by this mating must be male. Similarly any black kittens produced from the mating of a tortie female and and orange male must be male.

The $O^{o}O^{o}$ genotype of a female cat or the $O^{o}(Y)$ genotype of a male cat masks the expression of the *B/b* gene which controls black or brown pigmentation. An orange cat, then, displays no evidence in its phenotype as to whether it is genetically black *(B-)* or brown *(bb)*.

A Gene Which Controls Density

Another gene in cats controls the density of color. This gene has the alternative alleles:

D non-dilute
d dilute

In cats of genotype *dd*, the color is diluted; non dilution occurs in cats of genotype *DD* or *Dd* (i.e. genotype *D*-).

There are many examples of the action of the *d* allele in both pedigreed and unpedigreed cats. For example, a potentially black cat, genotype *BB*, which is also carrying the genetic information *dd* will be blue. In summary then *dd* dilutes black to blue (e.g., Russian blue, Korat); *dd* dilutes orange to cream; *dd* dilutes brown to lilac or lavender (e.g., self lilac).

In siamese cats, the *dd* genotype acts as follows:

seal point $\xrightarrow{dd}$ blue point

chocolate point $\xrightarrow{dd}$ lilac point

red point $\xrightarrow{dd}$ cream point

seal red tortie point $\xrightarrow{dd}$ blue cream tortie point

chocolate red tortie point $\xrightarrow{dd}$ lavender cream tortie point

The *D/d* gene illustrates the rule mentioned earlier that genes do not act in isolation, but act in conjunction with other genes in the genetic makeup of a cat. The *D/d* gene is strictly not a "color" gene since its action is not to produce a pigment, but rather to act on the expression of pigment produced by other genes.

A "Fading" Gene

The first siamese cats arrived in England from Bangkok in 1880. This introduced the c^s allele into breeding stocks in England. The first Tonkanese cat was brought from Burma to the United States in 1930 and this introduced the c^b allele into breeding populations there.

The siamese and burmese alleles are alternative forms of a gene which affects coloring. The complete series of alleles for this gene are:

C full color
c^b burmese
c^s siamese
c^a blue-eyed albino
c pink-eyed albino

The cat with at least one copy of the *C* allele has normal or full intensity of coloring. The genotype of such a cat may be represented as *C*-.

The effect of the $c^b c^b$ genotype is to reduce pigment intensity. This effect may be seen in the brown burmese in which potentially black coloring is reduced to dark brown. The brown of a burmese cat has a different genetic basis from the brown of the chestnut (havana) brown cat.

The $c^s c^s$ genotype also reduces pigmentation. This particular genotype, which is the essential genetic basis of cats of the siamese breed, reduces pigmentation over the body such that coloring is largely confined to the points (i.e., mask, ears, legs and tail). The extent of pigmentation in siamese cats ($c^s c^s$) is temperature-dependent, with color development being restricted to the relatively cooler areas of the body. Siamese kittens which develop in warm conditions *in utero* have light fur at birth; color first appears on the fur overlying the coolest area of the body - the ears margins. The temperature-sensitivity also means that fur regrowing on a shaved area of the trunk of a siamese cat will be pigmented.

The $c^s c^s$ genotype does not confer any color on the cat; the action of this genotype is to restrict color development to the points. Other genes govern the actual color of the points.

Genotypes of female siamese cats are shown in the table below, along with their corresponding phenotypes. The dash (-) is used to denote any allelic form of that particular gene.

Genotype	Phenotype
$B\text{-}\ D\text{-}\ c^s c^s\ O^+ O^+$	seal point
$bb\ D\text{-}\ c^s c^s\ O^+ O^+$	chocolate point
$--\ D\text{-}\ c^s c^s\ O^o O^o$	red point
$B\text{-}\ D\text{-}\ c^s c^s\ O^+ O^o$	seal-red tortie point
$bb\ D\text{-}\ c^s c^s\ O^+ O^o$	chocolate-red tortie point
$B\text{-}\ dd\ c^s c^s\ O^+ O^+$	blue point
$bb\ dd\ c^s c^s\ O^+ O^+$	lilac point

Cats carrying c^a and c alleles have been reported only very rarely. The cat of genotype cc is a true oculocutaneous albino with no pigment in either the fur or in the iris of the eye. The cat genotype $c^a c^a$ is a white cat with blue eyes. Neither phenotype forms the basis of any pedigreed breeds or variants.

The Gene That Hides Everything

The question I most fear when consulted by a breeder is: "If I mate two white cats what colored kittens could I get?" Why? Because a white

cat is a cat in disguise. Beneath that white coat there may lurk (for breeding purposes) a chocolate point siamese or a black mackerel tabby...or anything. It is only by careful analysis of the pedigree of a white cat that one might obtain some information as to what lies beneath that white exterior. In genetic terms, then, it may be said that the *W*– genotype masks the phentoypic expression of all other genes for color and pattern.

The gene responsible for the all-white coat has the alleles:

W all-white
w non-white

All-white coat color is the dominant characteristic. All-white cats may be homozygous *(WW)* or heterozygous *(Ww)* while cats which lack this characteristic are genotype *ww*. The mating of two non-white cats would be expected to produce non-white kittens only; an all-white kitten must have at least one all-white parent.

The all-white coat condition, particularly in combination with unilateral or bilateral blue eye color, is associated with deafness. Studies of part of the inner ear of deaf white cats have shown that this deafness is related to a failure of nerve cells to migrate to the inner ear.[5] This observation is of interest since the appearance of color in the coat is dependent on the migration of potential pigment-producing cells to those areas during embryonic development, and these cells originate from the neural crest.

PREDICTING OUTCOMES

Matings of cats produce litters of small size. Mendelian expectations and Punnett squares are often used to predict the outcomes of matings and the relative proportions of each type of offspring. Within the context of matings which produce large numbers of offspring, predictions based on Mendelian ratios provide a reliable guide and there is good agreement between the actual and the predicted outcomes. However, litter sizes in the cat are small, and even the cumulative total of offspring from repeat matings involving the same pair of parents is generally low.

The use of classic Mendelian expectations to predict the outcome of a particular mating in cats is not reliable if the prediciton is expressed in terms such as:

"You will get 3 : 1 black : blue."
"You should expect half siamese and half foreign."
"The expected result is 9 black : 3 blue : 3 brown : 1 lilac."

Mendelian ratios, such as the 3 : 1 expectation from a heterozygote *(Bb)* x heterozygote *(Bb)* mating, can only be used as an expected ratio when

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[5]D.R. Bergsma and K.S. Brown, "White fur, blue eyes and deafness in the domestic cat," J. Hered. 62:171 (1971).

the number of offspring is very high. It is difficult to translate a 9 : 3 : 3 : 1 ratio to a litter of 5 kittens!

The classic expectation from the mating of two heterozygotes is 3 : 1 showing the dominant and recessive traits respectively. In fact, if two black cats, each heterozygous *Bb*, were mated and produced a litter of 4 kittens, then the chance that the litter will comprise 3 black kittens and 1 brown kitten is less than the chance that the litter will consist of some other possible combination such as 2 black and 2 brown, or 4 black.

The classic ratios and Punnett squares of Mendelian genetics are of use to breeders in several ways:

1. to identify what kinds of kittens are possible from a particular mating;
2. to obtain an estimate of the chance of occurrence of each type.

For example, consider the outcome of the mating of a black cat, heterozygous *Bb*, with a brown cat, homozygous *bb*. The classic expectation is 1 black : 1 brown. However, this should not be interpreted as meaning that half of the kittens in the litter will be black and half will be brown! This expectation should be interpreted by the breeder as indicating that:

black kittens are possible;
brown kittens are possible;
the chance of a black kitten is $^1/_2$;
the chance of a brown kitten is $^1/_2$.

Now, consider the outcome of a mating between two black cats, each heterozygous Bb Dd. The classic expectation of 9 : 3 : 3 : 1 is shown below:

Bb Dd X *Bb Dd*

↓

9 B- D-	:	*3 B- dd*	:	*3 bb D-*	:	*1 bb dd*
black		blue		brown		lilac

The breeder should interpret this expectation as indicating that from this mating it is possible to obtain black, blue, brown and lilac kittens. The chance of each type is $^9/_{16}$, $^3/_{16}$, $^3/_{16}$ and $^1/_{16}$ respectively. From such a mating it is of course possible that the litter will consist of 5 black kittens only!

"MINOR" GENES: THE LITTLE ONES THAT MATTER!

The discussion of genes has so far dealt with the so-called major genes, and some detail has been given of major genes that control aspects of color and pattern. Major genes also control other traits, such as:

coat texture	*Wh* wirehaired	>	*wh* normal texture
coat composition	*R* normal coat	>	*r* Cornish rex
	Re normal coat	>	*re* Devon rex
fur length	*L* short	>	*l* long
tail formation	*M* manx	>	*m* normal tail
digit formation	*Pd* polydactylous	>	*pd* normal

The major genes are fairly easy to detect and manipulate. It is not the major genes which make the difference between the ordinary pedigreed cat and the pedigreed cat of championship status; rather it is the so-called minor genes: polygenes, and a special group of polygenes, called modifiers.

Polygenes

In cats, inherited traits which are under the control of a single major gene show discontinuous variation. Cats may be readily classified into one of two or three discrete and non-overlapping classes with respect to such a trait. For example, any cat may be classified as either "siamese" or "non-siamese"; likewise, any tabby cat may be classified as "Abyssinian" or "mackerel" or "blotched."

In contrast to those traits which vary discontinuously, other characteristics in cats show continuous variation. If a large group of cats was measured for a continuously varying or quantitative trait (e.g., body size), a spread of values across a range from minimum to maximum would be found, with slight gradations between individual cats.

Inherited traits which show continuous variation are controlled by many genes, the effect of each gene being small, but cumulative. These genes are called polygenes. Each polygene is thought to have two alternative forms or alleles:

a contributing or plus (+) allele
a non-contributing or minus (-) allele

Other inherited traits in cats which vary continuously and are under the control of polygenes include aspects of body conformation. The head shape of cats ranges from the broad, flat face of the longhairs, the round face of the British and American shorthairs, the heart-shaped face of the korats, the long wedge-shaped face of the orientals. Individual cats within each of these groups also show a range of variation with regard to head shape. Head shape, then, is inherited and under the control of several polygenes.

A comparison of drawings of the face of the siamese cat in 1924 with that in 1977 shows the change from an earlier short and round face to the later longer wedge-shaped face.[6] This change would have been achieved only slowly as the + form of the polygenes contributing to an elongation of head shape were concentrated by selection breeding.

The difference in head shape of the American Burmese cat and the English Burmese is due to a different selection of polygenes for the breeds in the two countries.

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[6]M. Eustace, 100 Years of the Siamese Cat. London: Research Publish., 1978.

Polygenes are far less readily manipulated than major genes, and, in consequence, desirable combinations of plus or minus polygenes can be attained less rapidly than a given combination of alleles of major genes. However, by careful selection of parental stock and choice of breeding program, a desired target can be reached or approached.

Modifier Genes

Modifier genes are a special group of polygenes which alter the expression of a simple trait. The modifiers act on the phenotypic expression of a particular allele, and may intensify its phenotypic expression. These modifiers are sometimes termed "enhancers" and may be denoted by "+". Modifiers may suppress phenotypic expression, and such modifiers may be denoted by "-".

The action of modifier genes which enhance phenotypic expression is exemplified by the difference in fur length between "a long haired moggie" and a pedigreed chinchilla. Both cats have the same basic genotype for long fur, *ll*. However, the pedigreed cat has a far greater accumulation of modifier genes which enhance the expression of its genotype for the long haired condition. This accumulation of "+" modifiers has been achieved by selective breeding.

The variation seen in the orange coloring of cats also provides an example of the action of modifier genes. The orange coloring of cats can vary from a nondescript yellow to a rich red. The enhancement of the expression of the O^o allele in rich red cats is due to the action of a group of modifier genes, known as the rufous group.

Several modifier genes act on a particular trait, so it is not one, but many "enhancers" that intensify a specific trait. Consequently, manipulation of the modifier genes that affect a trait is far more difficult than manipulation of a major gene. The fact that several modifiers affect a given trait helps to explain why two ordinary parents may produce offspring which show far better (or worse) expression of that trait than is seen in either parent. For example, consider a long haired sire *(ll)* with the following modifiers that affect fur length: + - + - - -, and a long haired dam *(ll)* with modifiers: + - + - + -. Neither parent would have maximum enhancement of fur length. A kitten from these parents, however, might inherit a combinaton of modifiers such as + + + - + +, and consequently would be expected to show a better expression of the long haired trait than either parent. Another kitten from these same parents might inherit a combination of modifiers, such as - - - - + -, and would be expected to show a lesser expression of the long haired trait.

All cats possess modifier genes affecting fur length, but the effect of the modifiers is seen only in cats which have the appropriate ll genotype and hence have the long haired phenotype which is subject to modification. It follows, then, that a short haired cat can transmit to its long haired offspring modifiers which enhance their long haired trait.

INHERITED ANOMALITIES

Genes control a variety of structures and functions in the cat. We are seeing a gene in action when we watch a cat ecstatically rolling and rubbing in a patch of catnip; we see another gene in action when we observe the unusually shaped ear of the Scottish fold cat. We are seeing a gene in action when we become aware of the deafness of a blue-eyed white cat.

The message encoded by allelic forms of some genes leads to observable defects in a cat, which may be transmitted to its offspring.

Some defects are termed "congenital." The term "congenital defect" does not imply anything about causation. It simply indicates that a defect was present at the time of birth, regardless of whether that defect has a genetic and/or environmental cause or is of known etiology.

The appearance of a defect in an animal, taken as an observation in isolation, does not provide sufficient evidence to assign an underlying cause with certainty or to distinguish between alternative causes. Even the occurrence of the same defect in related individuals can be interpreted in several ways; this observation may point to an underlying inherited basis, be it monogenic, polygenic, or multifactorial, but it may also be consistent with an environmental cause since related individuals are likely to share or to have shared a common environment. However, controlled breeding experiments may lead to an identification of the cause, and certainly will where the defect is due to a single major gene.

Inherited defects in cats believed to be due to major genes include: hairlessness *(h);* split foot *(Sp);* porphyria *(Po);* hydrocephaly *(hy);* deafness, associated with white coat *(W).* The assignment of a mode of inheritance as dominant or recessive may be complicated by factors such as variable expressivity and/or reduced penetrance.

In addition to these single-gene defects, other anomalies show a familial tendency and may be due to the action of polygenes, sometimes with a threshhold effect operating. Defects of this nature are possibly: anophthalmia; umbilical hernia, squint, cleft palate.

It should be noted that the same defective morphological expression in different cats does not imply a uniformity or homogeneity of underlying cause. For example, the lack of tail in one cat may reflect its Manx heritage while the lack of tail in another cat may reflect its tardiness in moving from the path of a circular saw! A deformity of the foot may indicate the action of the *Sp* allele or may reflect the action of drugs *in utero*.

NORMAL FETAL DEVELOPMENT AND CONGENITAL BIRTH DEFECTS IN THE CAT

Drew M. Noden, Ph.D.

It requires 9-10 weeks for a fertilized, one-celled egg measuring approximately 1/250 inch to develop into a newborn kitten. In addition to the tremendous growth that occurs during gestation, the developing organism undergoes major transformations that bring about the formation of unique tissues and organs, each in their proper location and with appropriate functions. The objectives of this presentation are to first discuss when and how these changes occur in the embryo, and then use this information to explain the basis for some of the common inherited, induced, and spontaneous congenital malformations (birth defects) in cats.

NORMAL FELINE DEVELOPMENT

The development of the kitten can be divided into three phases:

1) pre-implantation, days 0-12;
2) embryogenesis, days 12-24;
3) fetal growth, days 24-term.

During the pre-implantation stage the organism undergoes cell division, travels down the oviduct and enters the body of the uterus on about the 6th day of development. It forms a blastocyst (Figure 1), which is a hollow spherical organism containing about 250 cells and covered by a thin layer of cells specialized to make contact with the wall of the uterus. The cells that will form the embryo cluster at one pole of the blastocyst; most of the remaining cells will contribute to the placenta.

Embryogenesis is the most critical phase of mammalian development. It is during this period that the clustered embryonic cell population becomes totally re-organized and forms the earliest discernible forms, or primordia, of every organ system in the body. The first stage of embryogenesis is called gastrulation (gaster = belly). At this time the embryonic cells form three concentric layers of tissues. More importantly, these cells begin to undergo programming for specific developmental pathways. In other words, selective activation of genes is occurring.

Immediately thereafter the organism enters the neurula stage, during which the primordia of the nervous system, the heart, and the vertebral column are established. This is accomplished by rearrangements of all three cell layers, a process called morphogenesis (morphos = form or shape; genesis = beginning). Also, blood vessels are formed both within the embryo and between the embryo and the immature placenta during this period. As indicated in Table 1, these first two embryonic stages occur very rapidly, especially in cats and dogs.

Neurulation is followed by a period of embryonic organogenesis, during which are formed the primordia of most of the other organs, including the liver and digestive tract, respiratory system, limbs, sense organs, skull,

Table 1. EARLY DEVELOPMENTAL STAGES OF MAMMALIAN EMBRYOS

SPECIES	2 CELLS	8 CELLS	BLASTOCYST	GASTRULA	NEURULA	4 mm (1/8 in)	6 mm (1/4 in)	9 mm (3/8 in)	16 mm (5/8 in)
RABBIT	2 days	2.5 days	3 days	5 days	8 days	10 days	11 days	13 days	16 days
CAT	3 DAYS	3.5 DAYS	5 DAYS	12 DAYS	13 DAYS	15 DAYS	18 DAYS	21 DAYS	24 DAYS
PIG	1 day	2.5 days	5 days	9 days	14 days	17 days	19 days	21 days	24 days
SHEEP	1 day	2.5 days	6 days	10 days	15 days	18 days	20 days	24 days	29 days
DOG	4 days	6 days	8 days	16 days	17 days	22 days	23 days	25 days	30 days
HORSE	1 day	3 days	6 days	14 days	18 days	21 days	23 days	26 days	34 days
COW	1 day	3 days	7 days	14 days	19 days	23 days	25 days	28 days	35 days
HUMAN	1.5 days	2.5 days	4 days	14 days	20 days	28 days	32 days	37 days	43 days

note: These are approximate times of gestation. Individual differences of several days in the mating-fertilization interval of some species and also breed-specific differences in the rate of early embryonic development have been reported.

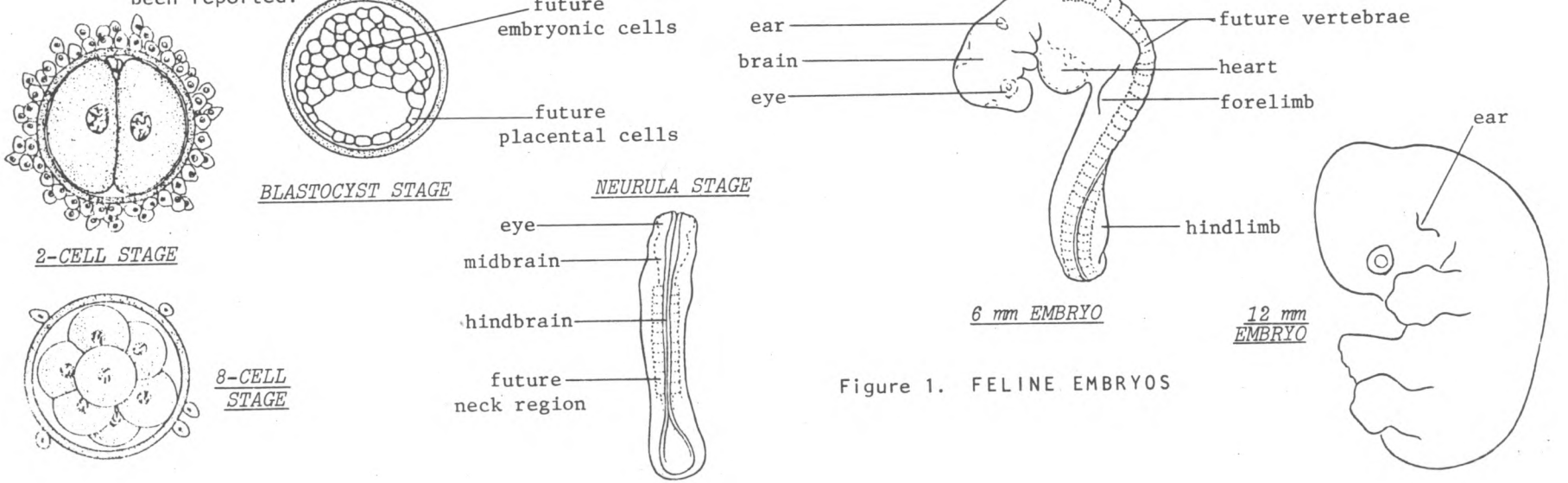
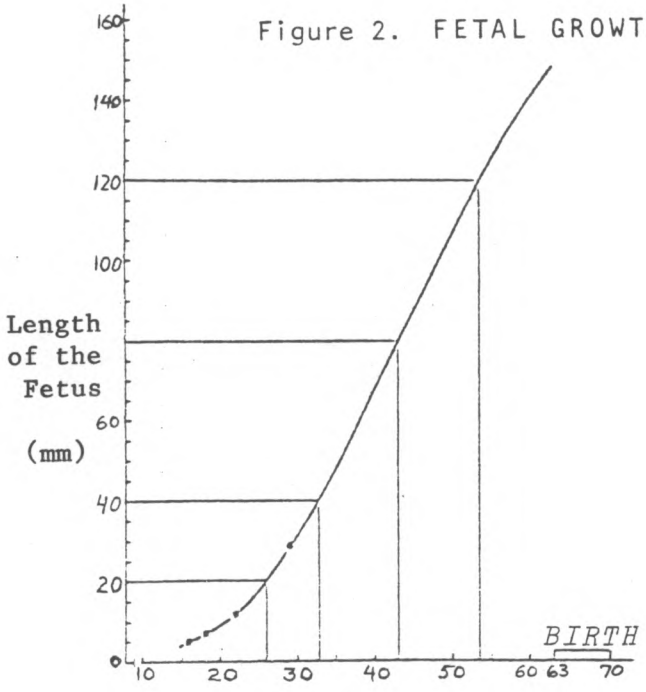


Figure 1. FELINE EMBRYOS

and urogenital structures. This stage is completed by about 3 1/2 weeks of gestation in the cat (6 weeks in humans), by which time the embryo is slightly over 1/2 inch in length.

Fetal development is characterized by rapid growth of the organism (Figure 2). The primordia established earlier assume their correct shapes and configurations, many nerves develop their projections, and the endocrine and secretory glands of the organism begin to function and control many physiological processes. Cats are born in a very immature condition, and all these processes continue after birth. For example, full development of the visual system is not accomplished until 5-6 weeks after birth, and neurons in the cerebellum are not all present for several months in the cat (3 years in humans).

Figure 2. FETAL GROWTH OF THE CAT



A young fetus at 4 weeks of gestation.

29 mm (1 1/8") long.

DEVELOPMENTAL MECHANISMS

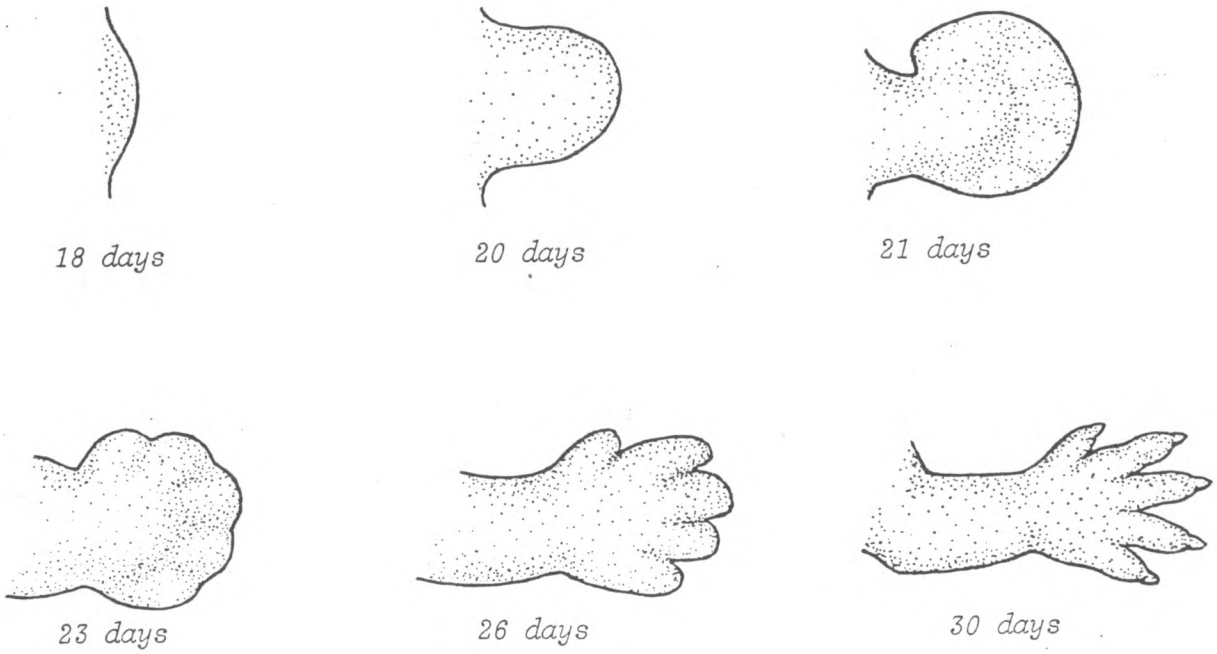
Understanding how populations of embryonic cells become transformed from homogeneous clusters to highly organized, patterned arrays of tissues, each with the correct biochemical characteristics and in the proper location, is a major frontier in biology today. This is not simply a matter of scientific curiosity. Rather, locked in the embryo are the secrets to mechanisms of tissue repair and organ regeneration, genetic programming, and the control of cell proliferation which, when unchecked in adult tissues, can result in tumor formation and metastasis.

The problems confronting the embryo can best be illustrated by focusing on the developing forelimb (Figure 3). Here a few hundred cells must proliferate and form a structure with muscles, cartilages and bones, tendons and ligaments, blood vessels, nerves, claws, fur, etc. Moreover, each of these tissues must have a precise shape and relation to the others, and constitute a mirror image of the contralateral (opposite) forelimb. Finally, both limbs must be the same size, and grow synchronously after birth. This example illustrates the four basic processes of embryonic development:

GROWTH
MORPHOGENESIS
CYTODIFFERENTIATION (the formation of unique cell types)
PATTERNING

Although none of these processes is fully understood, developmental biologists have learned several general features about them. It is known, for example, that all four are interdependent and inseparable. Also, each of these processes requires that adjacent cells and tissues interact with one another. Sometimes this is by direct contact between them; more often the intercellular dialogue is facilitated by chemicals released by the cells. Often the same extracellular molecules will have different effects, depending upon the type of receptors a target cell has, or upon subtle differences in the particular combination of molecules present.

Figure 3. STAGES IN THE DEVELOPMENT OF THE FORELIMB OF THE CAT



CRITICAL PERIODS

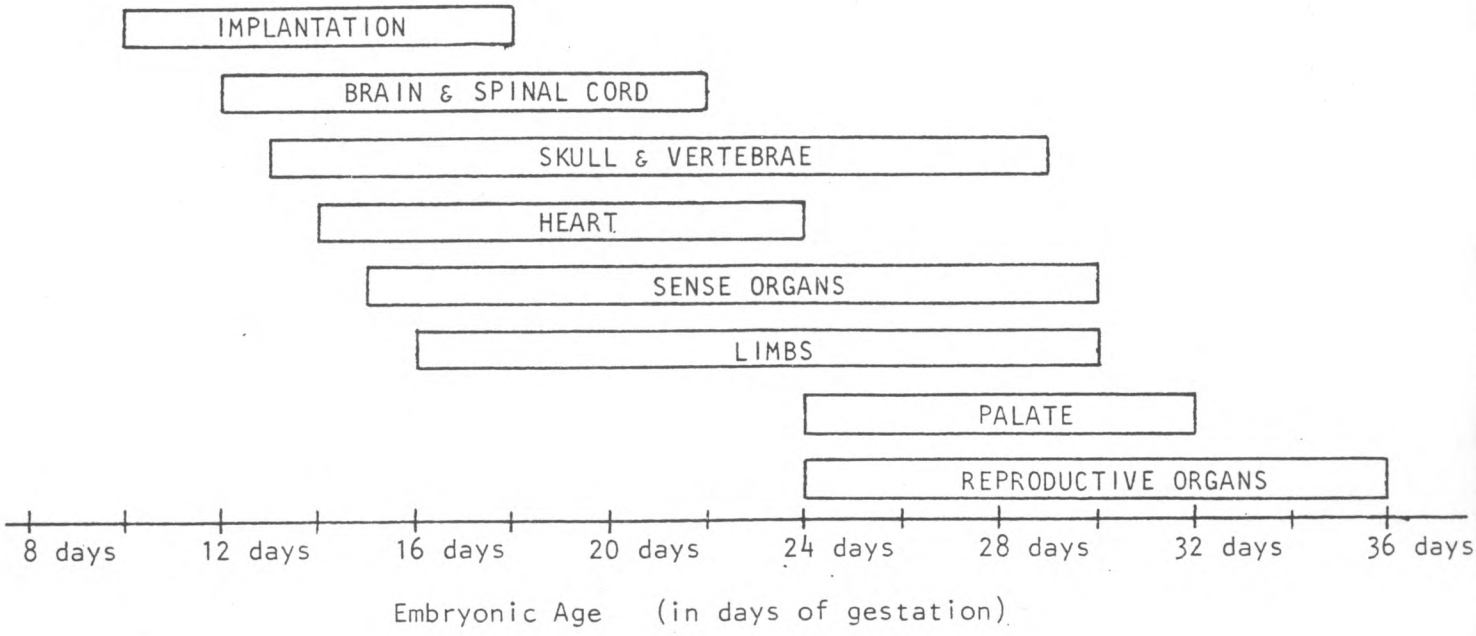
The kinds of interactions described above for limb development occur in every tissue and organ in the kitten embryo. These interactions are extremely sensitive to any kind of chemical or genetic disruption. Also, the complex spatial and temporal relationships that must be established for a structure to develop normally will on occasion spontaneously go awry, resulting in a congenital malformation.

The stage during which each organ is most sensitive to disruption is called the critical period in its development. As indicated in Figure 4, the critical period for most structures occurs in the embryonic stage of development, during the third and fourth weeks of gestation in the cat, the third through sixth weeks in the human. Disruption of development at earlier stages is usually lethal, resulting either in the immediate death of the embryo or in failure to establish normal relations with the uterine wall. Statistically, this is the time of greatest loss of embryos in all mammals. Also, the end of the critical period does not mean that a particular developing structure is no longer susceptible to disruption. Rather, its sensitivity declines, and abnormalities are more likely to be very localized.

The final general point to be made about embryonic malformations is that in many cases a defect in one system will result in abnormal development of others. For example, some cardiovascular defects deprive peripheral tissues of a sufficient blood supply for their normal growth. Similarly, a compromise of the nerve supply to any target tissue will result in secondary atrophy of the muscles and immobilization of the joints in that region.

Figure 4.

CRITICAL PERIODS IN THE DEVELOPMENT OF FELINE ORGAN SYSTEMS



CAUSES OF CONGENITAL MALFORMATIONS

Many malformations, especially those associated with highly inbred breeds, are inherited. Occasionally this may be due to a simple mutation, as is the case for certain enzyme deficiencies (mucopolysaccharidosis, gangliosidosis). More often the precise genetic basis is less obvious, because many genes regulate or are affected by the products of other genes. Problems associated with pigmentation (including defects in vision and hearing in Siamese and Persian cats), with the spinal cord and vertebral column (spina bifida in the Manx), with the limb (polydactylism, ectrodactyly and hemimelia), and with the head (hydrocephalus, anencephaly, diprosopus in Burmese) fall into this broad category. Finally, genetic heterogeneity may create a situation in which no single gene product is abnormal, but the collective effects of these genes is inadequate to support the normal development of an organ.

Induced congenital defects are no easier to deal with. Any agent which disrupts a normal developmental process is called a teratogen. It can be a chemical (dioxin, alcohol, heavy metals, many complex organic hydrocarbons), a drug (aspirin, griseofulvin), a normal body product delivered at the wrong time or in excess amounts (most steroid hormones), or a pathogen (panleukopenia virus). In many cases a chemical entering the mother is converted by her into other metabolites which cross the placenta. This feature makes comparative teratology such a difficult scientific discipline, because each species and often every individual has its own unique metabolic processes and rates. The picture is further complicated by the varied structures and transport mechanisms present in the placentas of various mammals.

COMMON CONGENITAL MALFORMATIONS IN CATS

Axial Duplications

Cojoined twins are an anomaly in which two embryos or parts of embryos are attached to one another. In some cases both may be fully formed and attached only at the head, thorax or abdomen (often called "Siamese" twins). In other situations only one part will be duplicated, for example kittens with two faces (diprosopus), two heads (dicephalus) or two tails have been reported. These anomalies result from duplications that occur between the blastocyst and neurula stages, with those occurring earlier being more complete.

Axial Defects

Malformations of the brain and spinal cord and/or the skeletal tissues surrounding them (vertebral column and skull) are the most common in all domestic animals and humans. Most early defects in neurogenesis will cause abnormalities in development of the surrounding skeletal structures, and vice versa.

Spina bifida is a broad term that includes all failures of the vertebrae to close normally. If, as usually occurs, the spinal cord is

abnormal or secondarily compromised by malformed vertebrae, there will be motor and sensory deficits in structures innervated by nerves whose roots are at or caudal (to the rear of) to the site of the lesion. The Manx line of cats is an example of inherited spinal bifida. It is caused by a dominant gene on an autosomal chromosome and shows incomplete penetrance, meaning that other genes modify the severity of expression.

Failure of the roof of the skull to form is correlated with an increased, abnormal expansion of the brain (exencephaly) or in severe cases with failure of the brain to form a closed tube (anencephaly). In the latter situation, brain tissue secondarily degenerates before birth leaving an empty crater in the top of the head. These lethal conditions are frequently caused by teratogens, but may occur spontaneously or as a result of genetic factors.

Hydrocephalus is a swelling of the fluid-filled ventricles within the brain that causes an abnormal expansion of the roof of the skull. This condition can result from heritable factors (in Siamese cats), pathogenic organisms, or spontaneous developmental errors.

Cardiovascular Malformations

The heart begins as a simple, straight tube formed on the 14th day of development and in the following two weeks becomes transformed into a 4-chambered organ with separate pulmonary and systemic channels. Throughout this period of change the heart is functional and supplies blood to all parts of the embryo without interruption. This is a remarkable feat of biological engineering.

Not surprisingly, congenital heart defects are common in all animals, and represent about 10% of diagnosed cardiovascular problems in young animals. Included in this category are failures of the four chambers to become fully separated (septal defects), constrictions of a major blood vessel (aortic or pulmonary stenosis), and abnormal retention of an embryonic blood vessel. The latter includes patent ductus arteriosus (PDA), vascular ring defects and portocaval shunts.

PDA is the most common of these. In the embryo and fetus there is a vessel, the ductus arteriosus, that connects the pulmonary artery directly to the aorta. This permits oxygen-rich blood from the placenta to by-pass the nonfunctional lungs and directly enter the systemic circulation. Normally the ductus arteriosus closes a few days after birth. If it remains open (patent), pulmonary and systemic blood streams mix, which causes the heart to work harder and can lead to many secondary complications.

Similarly, a portocaval shunt is the persistence of an embryonic vein joining the major systemic venous channel of the trunk, the vena cava, with portal veins carrying blood from the intestines to the liver tissues, where it will be filtered and detoxified. If the shunt persists postnatally, this permits nonfiltered blood to enter the systemic venous channel, creating serious metabolic inbalances in the animal. Both PDA and portocaval shunts are medically and surgically treatable if they are diagnosed early and are not severe. PDA shows an increased familial frequency in some species.

Malformations of the Face and Mouth

Cleft palate and cleft lip are less common in cats than in most other domesticated animals. Both result from failure of embryonic facial tissues to grow and fuse together. Either condition reduces the ability of the neonate to suckle, and the animals often die of choking. In humans and dogs, cleft palate sometimes shows a familial pattern and may be accompanied by malformations of other systems. Many unrelated teratogens can cause cleft palate, also.

Cat and dog breeders frequently select for animals having particular facial profiles. However, they must recognize that selecting for any extreme, especially a shortening of the snout (called brachycephaly), INEVITABLY increases the incidence of craniofacial malformations and problems of tooth eruption. This is true especially in the new-look Burmese cats, and in Bulldogs, Pugs and some of the toy breeds of dogs.

Limb Defects

Malformations involving the absence of some (hemi-) or all (ectro-) structures of the digits (-dactyly) or limb (-melia) are common. Radial hemimelia, congenital absence of the radius (the long, median bone of the wrist), and ectrodactyly and ectromelia, absence of the digits and of an entire limb, are the most frequently seen in this category. Syndactyly is the condition in which only a single digit is present. This malformation is debilitating in the dog, cat or human, but is the normal developmental program for the horse.

Congenital limb duplication can include an entire limb (dimelia) or only the digits (polydactyly).

There are cases in which each of these has been suspected of being inherited in cats, with polydactyly being the most common (an autosomal dominant) and also the least detrimental. In most cases of limb reduction the cause is not known, nor do we understand why forelimbs are usually more likely to be affected than hindlimbs. The drug Thalidomide causes a reduction in proximal limb structures in humans such that the hands or feet develop close to the shoulder or hip, a condition known as phocomelia (seal-shaped limb). Despite many years of intensive study, the exact mechanisms underlying this disastrous teratogenic insult are not known.

Other Common Feline Congenital Malformations

Gastroschisis is a lethal condition in which the kitten is born with its intestines protruding outside of the ventral abdominal wall. It results from a failure of the abdominal wall to form a narrow constriction around the base of the umbilical stalk.

A congenital diaphragmatic hernia is an opening in the diaphragm that permits abdominal organs to enter the heart or lung cavities and impinge upon the normal functioning of these thoracic organs. This condition, which arises during the early stages in the development of the liver and heart, is unusually common in cats and has been estimated to occur once in every 1000

live births as a result of an autosomal recessive gene.

PERSPECTIVES

In order to eliminate congenital malformations in the domesticated cat there are three problems that must be resolved. First, we must continue research using laboratory animals, tissues and cells grown in culture, and other model systems to better understand the normal mechanisms of embryonic development. Next, research on the physiology, pathology and reproduction of cats must be increased to more accurately reflect the common place of this species in our society. These two tasks are the proper responsibility of the scientific and veterinary communities.

A third and equally important area in which data are needed concerns the results of current and past cat breedings. Except in the cases of a few popular breeds, there have not been thorough, systematic studies of congenital problems in cats. Most of the available data represent isolated case reports.

The type of study needed requires that the incidence and types of malformations present in a breeding line be accurately documented, and careful analysis of the pedigree and all relatives of affected kittens be made. Often it is necessary to perform test matings to establish the exact mode of genetic transmission.

These critical analyses will only occur if cat breeders and fanciers familiarize themselves with the problems, keep accurate records of all litters, being especially watchful for the appearance of similar anomalies in more than one litter, and bring all specimens and data to their veterinarian for examination. It is only through such cooperative efforts that the accumulation of harmful genes or genetic factors can be reduced in many strains and a healthy future for these lines assured.

RAISING KITTENS

R.W. Kirk, D.V.M.

Producing normal healthy kittens begins with planning many months before the expected breeding time. Cats are seasonally polyestrous and although some breeds and some individuals may cycle all year the majority do not. In the northeast, breeding activity begins in January and continues in cycles of about 14 days until late spring. At that time about half the females stop cycling or have prolonged intervals between estrus. By September almost all queens cease cycling until January. Most young queens have their first litter between 12 and 24 months of age. Before the breeding season it is desirable to have all breeders thin, well-exercised, free of intestinal parasites, and with recent vaccinations for all feline infectious diseases. They should be fully nourished and in a relative state of "gain."

The social hierarchy of the cat world must be catered to in planning and carrying out the actual breedings. Multiple breedings with the male are desirable. Queens ovulate only after copulation, and sperm must be in the uterus 24 hours to become capacitated and capable of fertilization. Pregnancy can often be diagnosed by careful abdominal palpation about 25-30 days into gestation. Radiographs are diagnostic at 40 days. Do not overfeed pregnant queens. If they usually are housed with many cats they should be isolated for the last 6 weeks of pregnancy. Queens may have false pregnancy or may resorb foeti, especially when old; also if overcrowded, disturbed, unable to make a nest, or if a strange male is present. They may abort and ingest the foeti. Do not handle pregnant or lactating queens unless it is imperative!

Provide a nest box for the queen and if she feels secure, "queening" is usually fast and easy. The first kitten usually appears within 1 hour of labor onset and the whole litter may be delivered within 2 hours (25 minutes to 4-6 hours). A dark vaginal discharge indicates placental separation. This only persists 2-3 days after delivery unless there is a problem.

The new family should be left alone in warmth, quiet and solitude. Constant crying, kittens squirming around the nest box, and restlessness of the queen are signs of trouble. Young kittens have a normal rectal temperature of 92°-96°F the first few days. By one week it is up to 98°F (when they have the ability to shiver). Umbilical cords drop off at 2-3 days and eyes open around 12 days, ears 2 days later. They crawl around pretty well by 18-20 days and become visually oriented about then, too. Sound orientation follows quickly. Female kittens weigh about 100 gms at birth and gain 10 gm/day for 7-8 weeks. Males weigh 100 gms but gain 15 gm/day. Thus they double their birth weight by 10 days and triple it by 21 days (males even faster). Lots of "formula" is needed and this is a real stress to "Mom." If outside food can be started by three weeks this lessens the need for milk production and may shorten the time to weaning.

Queens' milk is 18% solid, (82% water), and the solids are approximately 25% fat, 42% protein, and 26% CHO. It contains approximately 140 cal/100 ml. Commercial replacement formulas (KMR, Unilact) are best for supplements, but P. Scotts formula [20 gm skim milk powder + 90 ml water + 10 ml corn oil] is better than evaporated milk, which has too much lactose. These formulas have about 90 cal/100 ml and kittens need about 140 cal/lb/day. They must be fed frequently - as compared to young puppies. May feed by bottle or by gavage. Avoid eyedroppers!!

Feeding Routine for Hand-Rearing Kittens

Week Post-Partum	Feed	Number of meals in 24 hours	Volume per meal (ml)	Caloric requirement for 24 hours	Expected body weigh (gm)
1	milk mixture in bottle	12-9	2-7	40-80	100-200
2	milk mixture in bottle	9	7-9	80-100	200-300
3	milk mixture in bottle	9	10	112	300-360
4	milk mixture in bottle	7	10	115	350-420
5	reduce bottle; increase solids	7		120	400-500
6	milk in bowl; solids	6		125	450-600
7	weaning completed	3		130	550-700

RAISING ORPHAN KITTENS

1. Heat is paramount. They cannot regulate their body temperature so thermostatically controlled heat is needed. The first 5 days 85-90°F is needed, then 80°F until 3-4 weeks, and 75°F from 4 weeks on. If the kittens are weak, extra heat is good even when they are with the queen.

2. Feeding - See table on previous page.

3. Sanitation - wipe abdomens and anus with warm water swabs after each feeding to stimulate urination and defecation. Use Domebro soaks if skin becomes irritated. Baby oil may be useful too.

4. Isolation - avoid handling and contacts with people or animals to prevent infectious diseases.

PROBLEMS WITH NEONATAL KITTENS

Overall mortality averages 24-30% to 1 year.

Stillborn 10%, Malformations 6%.

Of those dying, 6% die 1st 24 hours.
8% die between 1-7 days.
5% die between 7-42 days.
4% die between 42-180 days.
<1% die between 180-360 days.

Fading Kitten Syndrome

Kitten Mortality Complex

Bacterial Infections

Parasitisms

Other Infectious Diseases

FIRST AID FOR CATS

R.W. Kirk, D.V.M.

DEFINITION AND OBJECTIVES

First aid is the immediate care given an animal that is suffering from the effects of an accident or sudden illness. First aid also covers the initial treatment of diseases that have "just been discovered" by the owner but that have in reality been present for some time.

When we administer first aid to an animal, we are handicapped by the patient's inability to communicate verbally; we may have difficulty telling when, how, or where an injury occurred or an illness started. Fortunately with experience we can learn to "read" an animal's reactions, which convey a great deal of information. But we still cannot explain our motives to the injured patient when applying first aid.

An injured animal will be frightened and in pain - and thus uncooperative. It will usually attempt to run away and hide (especially a cat) and must often be captured and forcibly restrained. We may be bitten, scratched, or otherwise harmed by the frantic patient. Our use of muzzles, blankets, bags, and other protective measures, together with firm but gentle handling, often "tells" the patient that we are in control but compassionate. Talking quietly to the animal and using its name frequently often has a calming and reassuring effect.

Knowledge of successful methods of handling animals, as well as a recognition of normal structures and functions of the animal body, is a great help in understanding and dealing with problems as they arise.

The key objectives of first aid are:

1. To preserve life.
2. To alleviate suffering.
3. To promote recovery.
4. To prevent aggravation of the injury or illness until we can obtain veterinary assistance.

FIRST AID PROCEDURES IN LIFE-THREATENING EMERGENCIES

1. **Remove the cause** of the injury if possible.

2. **Clear airways** so that the animal can breathe. Remove the collar and any foreign material, blood, or fluids from the nose and throat. Place the animal in the position that makes breathing as easy as possible.

3. **Give artificial respiration** if the patient is not breathing.

4. **Treat cardiac arrest** immediately. Often a sharp blow on the side of the chest, just behind the shoulder, will suffice. Repeat every 15 seconds until the patient responds. Failure after 5 minutes of effort means the patient has died.

5. **Stop or control bleeding** as soon as possible. Use pressure bandages, pressure points, or tourniquets as needed.

6. **Cover any wounds** with clean, dry dressings.

7. **Keep the patient warm** (blankets, box, warm car).

8. **Do not move** or manipulate the patient unnecessarily. An injured animal will usually assume the least painful position, with the injured part uppermost. When it is necessary to move the patient, support and protect the injured area to prevent further damage and pain. Use blankets, rugs, and boards or boxes to support an animal being transported.

9. **Treat for shock.** If the animal is unconscious, place its head slightly lower than the rest of its body to treat shock and to prevent the patient from inhaling fluids or materials in the mouth. **Do not give anything by mouth.** If the animal is conscious and not seriously injured, you may give small amounts of drinking water.

10. **Promptly transport the animal to a veterinary facility** for professional care.

11. If possible, **have someone phone the veterinary hospital or surgery** (while you are on your way) to alert the staff of your need for emergency care. The person who calls should give a brief description of the injuries, so the hospital personnel can make preparations for your arrival.

12. **Don't speed on the way!** A patient that won't survive a few minutes probably can't be saved - and the rough ride to the hospital may aggravate injuries. Risking an additional accident is not justified by any emergency.

THE FIRST AID KIT

The drugs and equipment needed vary. The list here is appropriate for dogs or cats, but it includes only the essentials. It is assumed that common household items such as scissors, pliers, blankets, soap, bicarbonate of soda, and mineral oil are readily available.

The contents of this first aid kit should provide material to help restrain a hysterical patient and to treat major problems such as bleeding, wounds, shock, heatstroke, poisoning, and eye injuries. The parenthetical numbers in the following list indicate the quantity.

Contents for a Pet First Aid Kit

Materials and Equipment

Gauze bandages, 1" and 2" rolls (1 each)
Gauze dressing pads, 3" x 3" (8)
Adhesive tape, 1" roll (1)
Roll of cottonwool (1)
Triangular bandage (1)
Rectal thermometer (1)
Cotton buds (6)
Tweezers (1)

Medications

3% hydrogen peroxide (2 oz.)
Milk of magnesia tablets, 5 gm (10)
Activated charcoal tablets (20)
Kaolin mixture (2 oz.)
Antibacterial ointment
For eye 1/8 oz. tube (1)
For skin 1/8 oz. tube (1)

Be sure to keep replacing materials and medications as you use them.

RESTRAINT

Anyone who reads this article loves animals or at least is vitally interested in their welfare. In most instances the first-aider will be faced with a patient that does not recognize such love and concern. Injured animals hurt. They are afraid and do not understand what has happened to make them hurt. Thus, their master or friend, who may have unintentionally caused the injury, no longer seems trustworthy and their natural instincts tell them to escape and hide. Failing this, they tend to strike out at anyone who approaches, for they fear their painful experience will be repeated.

If the first-aider understands this instinctive behavior and knows how deal with it psychologically, as well as physically, he/she can usually help prevent further pain and avoid personal injury.

In veterinary medicine, tranquilizers, sedatives, and anaesthetics make care of injured animals easier and more humane than it was just a few years ago. However, one must get the patient to the veterinary surgery or hospital for proper selection and administration of one of these medications. In the time beween the injury and arrival at the surgery or hospital, the first-aider must use his/her best efforts to alleviate the animal's suffering.

Principles of Restraint

1. Approach the patient with a firm but kind and quiet manner. Use its name if you know it and allow the animal to sniff the back of your closed fist.
2. Do nothing to further injure the patient.
3. Restrain the patient in a way that will not allow it to injure itself.
4. Protect yourself from injury that may be inflicted upon you by the patient.

Principles of Restraint (Cont.)

5. Place the animal in a situation different from its usual secure environment (e.g., on the top of a table instead of on the floor). The newness will create uncertainty but not pain, and the patient will be more cooperative and thus easier to treat.

In general, the cat is a very independent creature. It has a personality that differs markedly from a typical dog's. For this reason cats must either be restrained very securely and almost forcibly or confined in a way or to an area where they think they are free but in reality are not. The cat also has five points that may cause the first-aider harm: the mouth, which may bite, and the four feet, which can cause painful scratches. It is almost impossible to muzzle a cat, so restraint of the feet is the paramount goal.

To humanely "disarm" the cat's claws, secure the two front legs together, just above the feet, by winding adhesive tape around them two or three times. Then fasten the rear legs together in a similar manner. Hold the head firmly. The cat can now be examined and treated carefully and effectively.

Actually, the cat is usually a bluffer and it will respond well to confident but firm handling. It is also forgetful and forgiving. If a cat becomes angry at forced treatment, release it. Within a few minutes it will once again be docile and approachable.

Picking Up or Carrying a Cat

1. Reassure the cat. Talk to it quietly, gently stroke its head and ears, and run your hand down its back.

2. Place one hand over the cat's back and pick it up by grasping it under the chest (sternum). With your other hand, either grasp the scruff of the neck or cradle the cat under the neck. Rest the cat's lower body on your forearm and snuggle the cat against your body.

3. Transport a cat in a cat basket or carrier, or cloth bag, such as a pillow case or canvas or burlap bag, tied closed. The cat cannot see out and will feel snug and secure. If the head is to be examined or oral medication given, loosen the bag and expose only the head; this effectively restrains the patient in the bag.

Restraining Unruly or Vicious Cats

1. In most instances an unruly or vicious cat can be controlled with a folded blanket or thick towel. Drop the blanket over the cat in its box or cage and pick up the animal inside the blanket. With the cat rolled in the blanket and only a foot or the head protruding, you can apply medication or bandages without danger to either the patient or the handler.

2. Use thick leather or canvas work gloves when handling a truly aggressive cat. Remember, cats will scratch and bite severely, and they will almost always attack your face or hands. Place these obstreperous patients in a strong bag or cat box and take them to the veterinary surgery or hospital, where drugs can be used for restraint. This is safer, wiser, and more humane.

FIRST AID FOR POISONING

Cats are fastidious and will lick substances on their feet or coat. This presents special problems but they can be resolved by washing off external contaminants; or by giving laxatives, emetics, or compounds to prevent absorption of poisons that are ingested.

FIRST AID FOR HEAT STROKE

Usually this is produced in cats confined to closed cars, buildings, or animal carriers. The treatment is to remove the cat to a cool environment and rapidly reduce the body temperature by cool water soaks and gentle massage to stimulate circulation.

FIRST AID FOR FROSTBITE

The tips of the ears are easily frozen because there is only a sparse hair coat. Hold the tips of the ears in the fingers to gradually warm them and apply a thin film of antibacterial ointment.

FIRST AID FOR INJURY, SHOCK AND BLEEDING

Each injury must be assessed on an individual basis, but keeping the cat warm and protected applies to all cases. Bleeding must be stopped promptly. It is important to get severely injured patients to a veterinary hospital promptly. More details of handling these patients will be discussed in the lecture.

100+ HAPPY, HEALTHY FELINES UNDER ONE ROOF

Ellen Yanow

Since its founding in 1971, Tree House Animal Foundation in Chicago, Illinois has initiated new programs which have a direct impact on the stray animal and pet ownership problems which exist in the country today. The 100-year-old accepted ways of simply caging or exterminating animals have neither stemmed the flood of animal overpopulation nor enlightened the public about this plight. Tree House Animal Foundation believes that education, communication and innovation are the means to help animals in the next 100 years.

One of Tree House's initial concerns was the routine housing of many stray dogs -- but very few cats -- at animal shelters. It was commonly thought that "cats could take care of themselves", and stray cats were not given the same attention by animal control workers or even the general public. Out of this existing situation, our plan to open a cageless, non-killing, homelike environment for stray cats was formulated. It began with a unique idea and culminated with the opening of the Tree House Adoption Center, a facility unlike anything Chicagoans had ever seen before, and now a model for the nation.

Innovation is very much in evidence at Tree House Adoption Center, a 15-room house which is licensed by both the city of Chicago and state of Illinois. As many as a couple of hundred calls a week are received from people who want to give up animals -- either their own pets or stray animals they have rescued or wish to rescue. Our policy of focusing on the most downtrodden and helpless strays is unusual in the sheltering world. Almost always the opposite is true and only young, healthy housepets are maintained by shelters. Instead, we counsel people on how to find homes for their own pets and concentrate on the never-ending stream of destitute cases.

The severity of the situation is determined in the telephone conversation with each caller. A cat will be admitted to Tree House if it is injured, abused, sick, pregnant -- and a stray (unowned and homeless). Most are in such piteous condition, it is apparent this is the only chance they will ever have to receive relief from a struggle to survive.

Approximately 99% of incoming cats are brought directly to our facility; 1% may have injuries so extensive that they are rushed directly to an animal hospital for emergency treatment, and transferred later to Tree House.

Upon arrival, the person admitting the cat is asked to sign a Receiving Agreement, bestowing legal custody of the animal to Tree House. Then, a basic physical work-up is begun.

The following criteria are evaluated and actions taken:

- General state of health or extent of obvious injury or illness.
- Age ("mature" or more specific) determined.
- Temperature is taken.
- Weight and body tone are noted.
- Condition of teeth and gums. If the teeth show a lot of tartar build-up, they are scaled or major dental work will be scheduled later.
- Blood is drawn for FeLV, Hematocrit and Total Protein tests.
- Presence or absence of fleas. If flea evidence is found, the cat is powdered or, depending on the time of year, the cat may be powdered routinely.
- Condition of ears. If mites or an infection are present, the ears are cleaned and appropriate medication begun.
- Condition of skin. Abcesses are cleaned and treated. Bare or scaly patches may indicate ringworm or other fungus. A skin culture may be taken and treatment begun.
- If obvious illness, supportive therapy is offered (fluids and antibiotics).
- If healthy and temperature is normal, vaccines are given. Both the intranasal and injectible are used. (The intranasal provides immediate protection against upper respiratory viruses; the injectible includes panleukopenia.)
- Urine may be tested if necessary.
- Nails are clipped.
- A name is determined and a name collar is placed on the cat.
- A file is started to compile all information.

At this point, arrangements are made to transfer certain cases to specialists for further treatment. These would include: cats experiencing serious respiratory distress; orthopedic injuries and possible emergency surgeries; traumatic eye injuries; pregnancies (for spay/abortions); major dental work; other special surgery cases.

All other new arrivals, after the initial exam, are placed in an isolation ward, individually caged. A card is placed on the cage identifying the cat, and daily information regarding fluid or food intake and eliminations is recorded. A treatment sheet is posted to record daily medication. Each cat is individually monitored during this time.

As soon as a cat's condition will allow, a regular bath is given if necessary. A male cat is neutered, if mature. A stool sample is checked for parasites. If negative, the cat is wormed once. If positive, an appropriate drug schedule is followed, depending on the parasite found. A special diet is planned if necessary to the cat's health and recovery.

After a negative FeLV result is received, the cat is vaccinated against rabies to protect the current residents. A female cat will now be spayed. If a positive FeLV result is received, the cat is humanely euthanized by intravenous injection of a euthanasia solution. Dr. William Hardy's complete test and removal program is followed to control the incidence of feline leukemia virus.

If a cat is not in need of continuing special treatment for illness or injury, it will still usually spend at least seven days under observation and until all test results are received and evaluated. If a cat has a medical problem that is not improving, it is taken to a specialist for further evaluation. A cat recovering from any upper respiratory condition must show no signs of sneezing, nasal or ocular discharge, or elevated temperature for at least two to three days before being allowed to leave the isolation area.

Complete information about each cat is continuously recorded in a cat's permanent file.

No expense is spared to give a cat whatever treatment is necessary. If a cat has a chance to survive, we will try to save its life. No cat is euthanized unless in the opinion of our veterinarian it is terminally ill or suffering too greatly from the extent of injuries to survive. We will provide the same excellent medical care and concern as for our own pets.

After the admitting and isolation periods, a now healthy and contented cat is transferred to the adoption area.

The introduction/socializing process begins. Eight cages in the adoption area are used to introduce a new cat to its new "temporary" home. After all, it just wouldn't be correct in terms of cat etiquette to expect a new arrival to assimilate immediately into the general population. It needs a quiet period of observation from a safe distance where it can see the regular activity without feeling threatened. During this period, which can range from several days to several weeks, our staff and volunteers make a special efort to pet and comfort the cats and let each one develop its own courage and curiosity. Eventually, it will begin to come to the front of the cage and demand attention. At this point, the cage door is left open and the cat may either stay inside or venture out. Usually, at the opening of the cage door, one or two other feline visitors will drop in and the new cat will go out to the various playrooms to explore its new surroundings.

What will the cat find? A happy, cageless, interesting and colorful environment developed especially for the comfort and interaction of the cats and the people who will come to adopt them. There are climbing trees for fun and exercise, window perches to gaze from, tunnels to burrow into, cubes and benches to sit on, beds to snooze in. And a separate room for litter pans, 24 in the adoption area alone. A nursery houses young kittens, away from the adult population. Nutritious, high-quality, brand-name canned cat food is fed morning and evening, with a lunchtime snack in the nursery. Special diets are observed if necessary for medical reasons. Ceiling fans circulate warmer air down to the cat's level in the winter and

bring a refreshing breeze in the summer. Humidifiers are added in the dry season and air purifiers operate continuously. An abundance of windows provides natural light and sunshine.

A friendly manner on the part of both volunteers and staff sets a cheerful tone. No one is ever too busy to stop and pay attention to each cat, offering a playful hug, a soothing scratch or listening to the plaintive cries of an orphan. If a cat requires extensive therapy because of a permanent condition such as a missing limb or eye, or just a nervous disposition, it receives special attention on a regular basis. Through such close contact, a personality profile is developed on each cat, and volunteers and staff are trained to match potential pet owners with compatible animals -- the beginning, hopefully, of a successful adoption.

Although a cat may live in our Adoption Center forever, our goal is to find a good and loving home for each of the orphans. Tree House Animal Foundation employs an effective plan: An extensive advertising campaign is aimed at the potential pet adopter. Display and classified ads are placed regularly in the newspapers urging people who are looking for a pet to consider adopting from Tree House. This message is also brought forth in many other ways, traveling on buses, through 10-second television commercials and public service announcements on radio. Satisfied adopters spread the word to friends and relatives who are interested in a pet. Veterinarians also refer clients.

Once the potential adopter makes the decision to come to Tree House, he will be asked to fill out an Adoption Application to determine whether he has had a good history of pet ownership, or is willing to learn how to care for a pet. Some personal and reference information is also asked. An adoption counselor will interview the applicant and discuss our important standards, including but not limited to the following:

- Adopter must be a minimum of 21 years of age.
- Adopter must evidence ability to be contacted by telephone.
- Cats may not be declawed.
- A kitten may not be left alone without human or animal companionship for more than four hours at a time.
- Kittens may not be placed in a home where there are children under six years of age.
- Cats may not be permitted out of doors unless on a hand-held leash or contained in a screened area.
- A cat may not be adopted to a home where there is an adult animal that is not spayed or neutered.

When it is determined that the applicant is sincerely interested in the adoption of a cat that will become a member of the family, he is taken to the adoption area to make a choice.

A person may spend as long as he likes in selecting a cat; the average time devoted is an hour. Sometimes the experience becomes so enjoyable, it

lasts longer! Specific questions are answered by staff and volunteers who know the individual cats' personalities and this is supplemented with information from the cats' files. When a selection is made, the adopter must sign a legally-binding Adoption Contract assuring safe and humane treatment of the new pet. A $25 donation is requested. An additional $10 deposit is required for a kitten as insurance against future spaying or neutering. (If the applicant does not choose a cat, the Application form is held for a three-month period. This is also true of Applications that are not approved.)

To get the new pet safely home, a cardboard carrier box is given to each adopter, as well as our own pet-care literature and a 30-day Health Insurance Policy. If a cat becomes ill during the first 30 days after adoption, arrangements are made for the cat to be treated by our veterinarian or other cooperating animal hospital at no expense to the adopter. If a condition that begins during this period lasts longer than 30 days, the medical bills continue to be covered.

Basic pet supplies are sold at the Tree House boutique, such as litter pans, litter, litter scoops, grooming combs, nail clippers, food and water dishes and scratching posts. An adopter is encouraged to buy supplies on the premises so as not to have to make a stop at a grocery or pet store with the cat in the car.

Adopters are encouraged to call with any questions that might arise regarding their new pets. Plant eating, excessive scratching at furniture, or how to make the introduction to Fido are but a few of the problems that, when solved through proven-effective animal care techniques, can make the difference between a happy or impossible pet/owner relationship.

A telephone follow-up call is made to the home of the adopter during the first month after adoption. A questionnaire form is mailed at the six-month point and, after that, it is assumed that all is well.

Sometimes, though, even the best, most well-informed counseling can't save a pet-and-owner relationship. In such cases, Tree House offers the adopter a special solution. Any pet adopted from Tree House is always returnable for any reason. Concern for the animal's health and safety continues to be a prime consideration.

BEHIND THE SCENES - KEEPING CLEAN

Clean, fresh surroundings are paramount to maintaining a disease-free and odor-free environment, and Tree House workers take it seriously. In the adoption area every morning, all floors are swept and washed with a 1:20 bleach and water solution, then rinsed with clear water. Litter pans are washed with soap and water, and fresh litter is used daily. All towels and rugs are washed daily. All carpeted cat furniture is thoroughly vacuumed daily. Every evening, the floors are again swept and disinfected. The litter pans are scooped. Soiled linens are removed and brought to the laundry area. Once a week, all walls, doors, windowsills, ceilings, light fixtures and fans are washed with the bleach/water solution. All transport

carriers are disinfected after use with the same solution. (Bleach is an inexpensive disinfectant that is effective in killing bacteria, viruses and funguses. Although the recommended solution is 1:32, we use a stronger 1:20 solution for more effectiveness in preventing the spread of ringworm.) A cleaning schedule in the form of a checklist has been developed and each work shift carefully attends to all duties listed.

The same cleaning procedures are in effect for the isolation wards, including additional rules:

- An apron or smock must be worn when medicating or cleaning. A garment worn in one isolation ward must not be worn in any other ward.
- Hands and arms must be washed before going from one ward to the next, or before leaving the isolation area.
- Disposable gloves are worn when handling contagious cats, and changed between cats.
- Cleaning supplies must be kept in their assigned wards, not transferred between wards.
- Disposable litter pans, food dishes and water bowls are always used.
- All isolation garbage is removed from the building via the back door leading from the isolation area, never carried through the building.
- Weekly, a special clean-up of the isolation wards is done. Walls, doors, ceilings, cage tops, cage outsides and cage backs are disinfected. Shelves are emptied and cleaned. Humidifier filters are cleaned and the motors are vacuumed with a wet/dry vacuum (winter season only). Exhaust fans are cleaned.

In all areas, as soon as the morning cleaning schedule is finished, it is almost time to start all over again!

HOW TO "REMEMBER" EVERYTHING - RECORD KEEPING

A permanent file is created for each cat entering Tree House. This file contains the cat's name, the date admitted, Receiving Agreement, color, breed, vaccination dates, weight, examination conclusions, medication, treatment, surgery, prognosis, test results, reports from outside veterinarians, all major information or changes, and a color photo of the cat (taken after several weeks of TLC). Later, the adoption date, Adoption Application and Adoption Contract will be included, along with any correspondence or telephone information received from the adopter. This file system is arranged alphabetically by cat's name and divided by year according to current residents and adopted cats.

An index of cats by admitters' names is used to reply to questions from people who have brought cats to be admitted but have no way of knowing what the cats were named. Often the admitting person will call to ask about the cat's condition and adjustment.

A cross-reference index of cats by adopters' names is of great help when a call is received from an adopter who may not remember the cat's original Tree House name.

Laboratory notebooks contain results of all lab tests performed daily, along with any special remarks. In this way, tests of many cats can easily be compared at a glance.

Treatment sheets posted on individual clipboards show daily and continuing medications for cats in the isolation wards.

Index cards in a treatment box list daily and continuing medications for cats in the adoption area (such as dietary supplements, ear treatments or remaining antibiotics for an abcess or condition).

Special treatment procedures scheduled for the current week are written on an information board in the Treatment Center, easily viewed by the staff.

Another information board in the kitchen area reflects monthly adoptions.

A magnetic board, also in the kitchen, shows locations of all cats in residence, by area: Adoption Center, Treatment Center, Isolation Wards (1, 2, or 3), at a Veterinarian, or in a Foster Home (usually kittens too young to be admitted). This information is offered for everyone's information -- to know "who" is "where".

A monthly treatment tickler file reminds the staff of all follow-up procedures and treatments to be performed in future months. One month after admission, a second 3-in-1 vaccination is given, and all feline residents are flea-powdered monthly as a preventive measure. A second FeLV test is done three months after admission and every six months thereafter. Yearly examinations and vaccinations are given to all cats, as well as any medical attention needed from time to time. (Cats who become ill are temporarily transferred to the isolation area.) These important details would be impossible to remember without a good follow-up system.

An index file of medications is maintained, showing the name of the medication, quantity last ordered, total cost, cost per unit, the expiration date of the medication, and name of supplying company. This information is useful when comparing prices or determining where a specific drug was last obtained.

All transport carriers are numbered, and a log must be signed before a carrier is used and again when it is returned. The Tree House name also appears on each carrier.

All grooming kits are numbered and a log is also used to keep track of their whereabouts.

All lab instruments and equipment are engraved with the Tree House name to discourage loss of these expensive items.

An inventory and order control record is used when ordering food and cleaning supplies, so that the exact amount of inventory can be maintained, without experiencing shortages or overstock. This record also provides an easy comparison week-to-week.

An annual budget is developed in advance of the operating year from the experience of preceding years and an anticipation of what may occur in the forthcoming budget year. Are there any areas where income can be increased? Are there any areas in which expenses can be decreased? Are there any anticipated expenses for repair or replacement forthcoming in the near future that did not occur in the past? Are there any expenses that occurred in the past that will not occur in this budget year? Will supply cost decrease, stay the same, or increase? These are a few of the questions that must be asked before completing the budget -- and then it must be reviewed once again to determine if it is realistic.

A monthly operating statement is issued to monitor the income and expenses and give a clear picture of where we are at and where we are going financially. Each line item on the statement is determined carefully, assigned a code number for income and expense and logged with care. Any deviation from what is expected is immediately apparent. With sufficient information contained on the monthly operating statement, a roadmap of our financial future is evident.

WHO DOES ALL THE WORK? - STAFF AND VOLUNTEER STRUCTURE

If you are wondering who are the Tree House workers that accomplish all these tasks, the structure is as follows:

An on-site Veterinarian spends two days per week at the Tree House Treatment Center, evaluating both incoming cats and current residents. A network of area animal hositals is available for emergency or surgical work that our facility cannot accommodate.

An Adoption Center Manager, who is also a licensed veterinary technician, works closely with the veterinarians. It is her responsibility to supervise the entire feline program, including animal admissions, medical treatment and laboratory evaluation, adoptions, ordering food and supplies, and scheduling staff and volunteers.

Two Assistants for the Adoption Center Manager are also trained in all animal handling, medicating and routine laboratory procedures. They interview prospective adopters, approve Adoption Applications, and do adoption follow-ups.

Three part-time Kennel Workers are responsible for cleaning and feeding, as well as special maintenance projects that arise. They also watch for and report abnormal behavior or suspected illness in the general population, and are responsible for recording information regarding cats' appetites and eliminations on the cage cards in the isolation wards.

Twenty-five dedicated Volunteers work on a regular weekly schedule of either morning or evening shift. They assist in cleaning, feeding, grooming, giving TLC to the feline residents, and adoption counseling. A program of giving extra TLC to cats who are extremely shy or need to learn how to socialize is a special function of this group. A Volunteer Coordinator supervises the volunteer effort, as well as conducts monthly volunteer meetings and other volunteer projects.

A special note: All evaluation and treatment of animals is done under the supervision of a veterinarian. There is no substitute for professional knowledge. Tree House also believes it is important that the same veterinarian evaluate the cats whenever possible. It is of great assistance in treating a sick cat to have been familiar with it in a healthy condition.

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While the Tree House feline Adoption Center may be the most noticeable of our programs, our dedication toward helping animals goes much farther. Tree House provides assistance through other programs, many of which include dogs, such as:

- Pet-Care Hotline - a telephone counseling service through which detailed counseling in the form of a friendly conversation saves many animals' lives before the owners feel there is an insolvable problem.

- Emergency Assistance - A program to help fixed-income pet owners cope with periods of temporary financial distress by supplying pet food or veterinary care.

- Education and Information - Work to educate the public about responsible pet ownership continues through the development of informational brochures and a traveling display staffed by Tree House personnel at pet shows. Tree House pet-care literature is free to individuals and sold to humane agencies and animal hospitals throughout the country.

- Low-Cost Spay/Neuter/Vaccination Program - Arrangements for low-cost spaying, neutering and vaccinations are made with area animal hospitals for pet owners who cannot afford standard rates.

- Pet-Facilitated Therapy - Pets can help people get well, and our therapy programs with emotionally troubled youths from two major Chicago hospitals are conducted three times weekly.

Tree House Animal Foundation is a not-for-profit humane charity which receives no government support. All programs are financed by contributions.

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DESIGN AND MANAGEMENT OF SHELTERS AND CATTERIES FOR PREVENTION OF INFECTIOUS DISEASES

Fredric W. Scott, D.V.M., Ph.D.

The design and operation of a shelter or cattery can have a significant influence on the spread of infectious diseases within that facility. One should try to minimize the risk of spread of infectious agents as much as possible while still maintaining a practical operation. To do this, one should be aware of the degree of "isolation" or "prevention" being exercised in the daily operation of the shelter or cattery. The following are some thoughts on the general principles of spread of infectious agents, and some ideas on facility design and operation to reduce this spread between cats.

I. MODE OF SPREAD OF INFECTIOUS DISEASES

A. Cat-to-Cat (Direct)

The direct spread of infectious disease from cat to cat occurs in several ways. Direct contact, such as licking, rubbing, and breeding, is a primary source of infection. Aerosols, or movement of infectious particles in the air currents, occur after sneezing, coughing, sweeping, or overactive use of litter, and also can produce direct cat-to-cat spread. The use of common feed and water dishes, litter pans, runs and cages is an important method of direct spread.

There is also vertical transmission in utero and between queens and newborn kittens. Virus carrier cats, not necessarily showing symptoms but shedding virus, are a common source of direct cat-to-cat spread of disease.

Use of corticosteroids and certain other drugs in some cases like feline viral rhinotracheitis, can produce an iatrogenic disease (a disease caused by medical progress) by immunosuppression and subsequent recrudescence of viral shed.

B. Indirect

Indirect spread of infectious agents can be produced by contaminated fomites (i.e., any substance other than food) such as feed dishes, uncleaned cages, and contaminated brushes. Contaminated vaccines, drugs, syringes and other equipment and material are another potential source of indirect spread. A very important source of infectious agents is the hands and clothing of people in contact with cats that are infected with virus. Contaminated feed can also result in indirect spread.

II. WAYS OR FACTORS USED TO PREVENT SPREAD OF INFECTIOUS AGENTS

Immunization with effective vaccines is the best way to control spread of disease. This is discussed elsewhere in these proceedings.

Other ways or factors to consider in order to prevent the spread of infectious agents are distance, physical barriers, controlling the flow of air, personnel and animals, the control of rodents, insects, and parasites which might carry infectious agents, and appropriate decontamination procedures.

A. Distance

Distance between cats causes dilution of infectious agents and reduces both chance of exposure and the number of organisms in an exposure. The greater the distance the better. Separate cages, separate rooms, separate buildings and the ultimate, separate facilities, are ways to increase the distance factor.

B. Physical Barriers

Physical barriers consist of cages, fences and walls. Open cages are only of value as a barrier if there is no aerosol spread, or if the cages are sufficiently separated to prevent "nose-to-nose" contact of the caged cats. Closed filter cages which limit air circulation are a good barrier for use in isolation areas. Fiberglass filter cages are effective, practical, and not prohibitively expensive. Horsfall-type filter stainless steel cages are very effective but expensive and not practical for a shelter or cattery.

Fences restrict both animal and human flow but have no effect on airflow.

Walls obviously are excellent barriers and prevent animal contact and prevent air and water flow as well as control insect and rodent vectors.

C. Air Flow

Air flow is a very important factor in the spread of disease, especially respiratory diseases of cats. A room containing numerous cats where there is no air exhange and a high humidity is a disaster waiting to happen. Ideally it is best to use only fresh outside air, with 10-15 air changes per hour. For economic reasons to reduce heat loss or air conditioning costs one may have to recirculate and filter the air.

Gradient air pressure is frequently used to control the spread of disease within isolation facilities, but in most cases this will not be practical for cattery operation. In facilities where it is used, positive pressure (i.e., a higher atmospheric pressure than the surrounding areas) is used in cages relative to the room for protection of the animals, while negative pressure (i.e., lower atmospheric pressure inside the cage compared to the animal room) is used for protection of personnel and to prevent spread of agents into the environment. Isolation wards should have negative pressure. The more contaminated the area, the greater the negative pressure should be.

Filtration of air is used to prevent entrance, spread or escape of agents and is especially important when recirculating air into animal areas. While Hepa filters are excellent for removing viruses they are fragile and not practical for shelters or catteries. Other effective filters are available which remove dust particles and bacteria. Since viruses ride on dust and water droplets, these filters will also remove viruses. Electronically charged filters also are helpful in removing infectious agents since these agents have a surface charge.

D. Personnel Flow

Personnel working with potentially infected cats should be aware that they can spread infectious agents from infected to healthy cats. Healthy, non-infected cats should be cared for before infected or quarantined cats are tended to. Separate outer clothes (lab coats, coveralls) and shoes should be worn when caring for infected cats. Shoes are most important since viruses settle to the floor and are picked up and transported on shoes. Keep one pair of shoes or boots for the isolation or quarantine area. Disposable shoe covers also can be worn while in the isolation area. It is worthwhile to wear rubber gloves, and always wash hands after handling infected or quarantined cats.

E. Animal Flow

Control of animal flow or contact is an important factor in limiting the spread of infectious disease. All cats should be quarantined for a minimum of 2 weeks before being introduced into the general cattery population. A system must be devised to prevent contact between infected and healthy cats at all times. It should be pointed out, however, that many of the important viral diseases of cats have carrier situations whereby a "healthy" cat will carry and shed virus for longer than this 2 week quarantine period.

In shelters with a rapid turnover of cats, it is often impossible to provide adequate isolation areas or procedures. However, newly arrived cats should be placed in decontaminated cages which are separated from healthy, longer term cats.

F. Control of Rodents, Insects, and Parasites

Rodents, insects and parasites can transmit infectious agents mechanically and sometimes biologically. Control of these vectors will eliminate this method of spread.

G. Decontamination

Second to immunization, proper decontamination procedures are most important in controlling infectious diseases within shelters and catteries. Washing affected areas with soap and water is quite helpful as this dilutes the concentration of infectious

agents. Sterilization by autoclaving, boiling, and dry heat is most effective against all infectious agents, while chemical disinfectants vary greatly in their effectiveness. A product advertised as virucidal will not necessarily inactivate all viruses.

Recently it has been shown that a 1/32 solution (4 oz./gal.) of ordinary household bleach (Clorox®) is an effective disinfectant against all known cat viruses, including the very resistant parvovirus or panleukopenia virus. If disposable food and water dishes are not used, dishes should be soaked for 10 minutes in a Clorox® solution daily, then rinsed with water. Cages should be washed with detergent to remove organic debris, then wiped with disinfectant.

Ultraviolet (UV) lights have been promoted to kill viruses and bacteria within wards or isolation areas. Unfortunately these lights are often more of a show than a practical addition to animal areas since a distance of a few inches from the light source quickly eliminates the effectiveness of UV lights. However, UV lights can be effective when used within air return ducts to decontaminate recirculated air. Also, UV light can be harmful to animals (and people) if direct exposure is great enough.

III. Isolation or Quarantine Facilities

All shelters and most catteries should have an isolation or quarantine facility to hold new cats, or cats exhibiting signs of infectious diseases.

A. Aims of Isolation Facility

The aims of an isolation facility are (1) to prevent the entrance of infectious agents, (2) to prevent their spread within the facility, (3) to prevent their escape, and (4) to protect personnel involved. If an isolation or quarantine area is maintained within a cattery, these principles should be kept in mind.

B. Types of Isolation or Quarantine Facilities

1. Maximum Security

There are several types of isolation or quarantine facilities that are used. The first could be classified as "maximum security" facility where absolute and uncompromising procedures must be utilized. These are very effective but are not practical for a shelter or cattery. This type of facility is utilized with exotic diseases, when housing germ-free or specific pathogen-free cats, and when working with animals that are infected with severe human pathogens such as rabies.

2. Moderate Security

A moderate security facility is reasonably effective and could be used in a large cattery, but probably is too sophisticated for the average cattery. The principles, however, are important to understand, and appropriate modifications of this should be considered for adoption shelters and small catteries.

Each must evaluate the risk that one is willing to take and know where to take shortcuts and where not to. Each modification or short cut taken reduces the effectiveness of the isolation and increases the risk of spread within the cattery or shelter.

Advantages of the moderate security facility are that it prevents escape of infectious agents and prevents their spread within the facility in most cases. It is more workable than maximum security facility and lends itself to better observation of cats, but does require some special construction and equipment.

There are several specific physical requirements for operation of a moderate security facility. It necessitates a separate room with a foyer or change area where outer clothes and shoes or shoe covers can be changed. Isolation cages are needed, as is a glass observation wall or window for easy visualization of cats from outside the isolation area. An autoclave is desirable but not essential since litter and cage papers can be disposed of in an incinerator or bagged for disposal, and dishes and cages can be chemically sterilized. A separate, negative pressure air system with filtered air should be utilized, or at least a separate exhaust for that area.

Keeping the forementioned requirements in mind, a multitude of designs are possible, with the operation there-in being thought of as a contaminated area much like a surgeon considers the surgical field as a sterile field. Any time a person or object enters the contaminated area, that person or object must be decontaminated or treated as infectious. When animals are brought into the area, the carrier must be decontaminated before it is utilized again, or disposable carriers should be used.

There are specific steps that personnel should follow routinely in the moderate security area. They enter the change area, remove outer clothing (lab coats, smocks, coveralls) in the clean change area, put on gloves, put on special shoes, boots, or disposable shoe covers as they step into "dirty" area, put on coveralls or other outer wear, then enter the isolation unit. Gloves should be disinfected between cages, watching out for contamination of coveralls or

other objects that could spread infectious agents between cages. When exiting, personnel should decontaminate gloves, remove coveralls, remove foot covers or special footwear as one steps to "clean" area, remove gloves and wash hands before leaving the change area.

Food, cage papers, kitty litter are to be passed in directly. It is best to use disposable dishes filled in clean area and passed in.

Wastes such as paper, feces, kitty litter, and disposable items are placed in plastic garbage bags, sealed, and the outside of the bag is decontaminated as it is passed out of the unit.

Dirty clothes are placed in a closed container, the outside decontaminated, then moved to the wash area to be washed with hot water. Bleaching is recommended where practical.

Isolation cages that are effective and practical are some type of fiberglass cages with Plexiglas® doors. These should have a filtered air intake, and a filtered exhaust with an electric motor for sufficient air flow. Filter cages without exhaust fans are available, but these are less desirable because of odor and humidity build-up. Regular cages are not recommended since cross infection will occur within the isolation facility.

3. Minimum Security

Minimum security facilities are less than ideal and often give a false sense of security. These consist of a separate room with conventional open cages where no special precautions are taken. The effectiveness usually is minimal against the spread of infectious diseases unless appropriate procedures, especially decontamination as outlined above, are practiced.

IV. OTHER CONSIDERATIONS FOR CONTROL OF INFECTIOUS DISEASES WITHIN CATTERY

In the main cattery, good hygiene including proper cleaning and disinfection should be practiced. Adequate air exchange with at least 10 air changes per hour should be available.

The best designed isolation or quarantine facility is useless unless personnel use the the facility properly. Personnel are usually the weak link.

In catteries, vaccinations should be kept up to date for panleukopenia (parvovirus, "distemper," enteritis, FPL), viral rhinotracheitis (FVR), calicivirus (picornavirus) infection (FCI), pneumonitis and rabies. In shelters, cats should be revaccinated as soon as possible after entering the shelter.

Temperature is an important consideration since exposure to sudden temperature changes can stress a cat that is not accustomed to those changes. Nutrition is also important since a healthy, well-nourished cat is more resistant to infectious diseases.

Cats recovered from FVR and FCI usually are virus carriers and can shed virus from their throats persistently or intermittently for months. Stress such as nursing, certain drugs, or an exposure to a sudden drop in environmental temperature can cause a recrudescence of shed of FVR virus. Since only small quantities of virus are shed, transmission of virus from carrier cats requires direct contact and not aerosol. There are ways of reducing the carrier problem, such as separate cages, weaning kittens early and isolating them from their mother, and vaccinations kept up to date.

The breeding season should be spread out to reduce the number of young susceptible kittens at any one time. Estrus in the cat is daylight dependent, normally being stimulated by increasing hours of daylight. Constant 24-hour-a-day light, or continued controlled light of 12 hours on and 12 hours off, result in year-round breeding.

V. SUMMARY

In summary, it is important to have an understanding of how infectious agents are spread within a cattery or shelter, and how practical isolation techniques can be adapted to each situation or facility.

CAN YOU CATCH IT FROM YOUR CAT?

Dorothy F. Holmes, D.V.M., Ph.D.

Society is reevaluating the role of pets. That this is true, is clear from the increasing prevalence of signs excluding animals from public places and by public outcry over the esthetic and health aspects of animal excrement in parks and streets. New concerns about pet involvement in human disease have arisen from such well-publicized findings that cats are a definitive host for the parasite causing toxoplasmosis and from unproven assertions that feline leukemia virus may be involved in human cancer or that the ownership of small dogs may be related to the development of multiple sclerosis. It is difficult for me and I expect for you to recognize that there is a significant portion of the population which does not enjoy the companionship of dogs and cats and may be all to ready to implicate them as the cause of real or imagined human ills.

It is the purpose of this paper to review current knowledge of pet-associated human disease and provide a basis for scientifically acceptable recommendations for pet management. Some generalizations are appropriate to introduce the topic.

People contract most infectious diseases from other people. However, they can acquire some diseases from pets and sometimes these have tragic outcomes. There are over 200 zoonoses (animal diseases transmissible to man). Of these, only a few are associated with pets. The magnitude of the problem is largely unappreciated because only the tip of the zoonotic disease iceberg is visible, and because most pet-borne zoonoses are "dead-end" situations with little subsequent person-to-person transmission. In addition, many pet-borne diseases have nondescript and subtle signs which make an accurate diagnosis difficult. Unless animal association is obvious, the case is unusually severe, or a unique clustering of cases results in an investigation, pet-associated diseases are usually underreported.

RABIES

Rabies is probably the most feared of the zoonotic diseases in spite of the low number of human cases reported in the United States over the past several years.

Although rabies is prevalent in the wildlife population of the country (foxes, skunks, raccoons and bats are the primary wildlife reservoirs in the United States), man is at a greater risk of acquiring rabies from pet animals because of his closer association with them.

When a rabid animal bites a victim, rabies virus is introduced into the wound. It first grows locally in the muscle cells at the site and after a variable period of time invades the peripheral nerve(s) supplying the area. Virus travels along the nerve until it reaches the central

nervous system (brain and spinal cord) where its presence causes the clinical signs associated with rabies. From the central nervous system virus next extends to certain peripheral tissues such as the salivary glands where it is shed in the saliva and can introduce the disease into a new host.

In most animals, rabies is characterized by behavioral changes (agitation, restlessness, excitability progressing to aggression) and unexplained paralysis. Sometimes only paralysis is seen.

Cats are highly susceptible to rabies and about 75% of rabid cats show a pronounced furious phase before progressive paralysis sets in. This makes them particularly dangerous transmitters of rabies as their attacks may be exceptionally vicious, inflicting deep puncture wounds.

In 1981, for the first time, the number of rabid cats in the United States outnumbered the number of rabid dogs, by approximately 30%. Investigation of 64 rabid cats in Iowa in 1981 revealed that none had a history of rabies vaccination. (Only dogs are required by many state and county health codes to be vaccinated). This is an unfortunate state of affairs since cats, especially young males during their nighttime prowling activities, are frequently exposed by rabid wildlife (especially skunks and bats). Reports of documented animal rabies have more than doubled in the United States in the three years between 1978 and 1981. Vaccination of pets and livestock is the most effective control measure in preventing disease and subsequent human exposure.

There are a number of effective rabies vaccines available for use in cats. Care must be exercised to use only a product that is licensed for use in cats as rabies has actually been produced by vaccinating cats with modified live virus vaccines intended for use in dogs. Kittens may be vaccinated at three months of age and should receive annual boosters.

ANIMAL BITES

Animal bites are the most common pet-associated human health problem in the United States. Approximately one of every 170 people in the country is bitten each year. Because of their small size and greater tendency to engage in bite-provoking activity, children are the most frequent victims. While rarely fatal, animal bites are painful and expensive. (The Maryland State Depeartment of Health and Hygiene reports an average cost to the victim of $150 per bite.)

Pasteurella wound infection is a frequent consequence of bite wounds. Caused by a bacterium that is normally found in the mouths of cats and dogs, the infection causes swelling, inflammation, and intense pain within a few hours after the bite.

Other diseases, less common, but still occasionally associated with bites include tetanus, staphylococcal infection, tularemia, and erysipeloid and bubonic plague.

PLAGUE

We usually think of plague in association with epidemics of "black death" in residents of medieval cities, but the disease also exists in the wild rodent population in western United States where sporadic cases of bubonic plague in man are associated with exposure to rabbits, rodents or their fleas. Cats also can acquire plague under natural circumstances through contact with infected rodents or their fleas, and have transmitted the disease to man on a number of occasions. Infected cats appear acutely ill with loss of appetite, lethargy and fever, and frequently a draining abscess or lymph node. A pneumonia may also be present. Cats can be involved in spreading the disease to man in a number of ways. They may mechanically transport infected rodent fleas to their owner's home or additionally, may carry infected rodents from the wild to human residences. If the cat itself becomes infected it can infect man by direct contact. Case reports would indicate that man acquires the disease either following a bite wound from an infected cat or from non-bite contact with cats having plague pneumonia. Both humans and cats respond to treatment with broad-spectrum antibiotics if it is initiated early in the course of the disease. While not a disease of national importance, owners and veterinarians should keep plague in mind when handling cats from areas where the disease exists in rodents.

CAT SCRATCH FEVER

Associated with the scratch of a cat, the agent causing this disease of man is still unknown. Seven to 20 days following the injury the local lymph nodes swell, become painful and may abscess. Many patients have mild fever, loss of appetite, generalized pains and other systemic signs. The enlargement of the lymph nodes may last from a few weeks to several months and may be confused with other diseases such as infectious mononucleosis or lymphosarcoma. Most patients recover with no complications. While 90% of cat scratch fever cases report contact with cats, there are some where the initial injury was produced by objects such as wood splinters or thorns. Most students of the disease believe that cats are only mechanical transmitters of cat scratch fever and are not themselves infected. Until a causative agent is isolated it is impossible to fully understand the mechanism whereby man becomes infected.

TOXOPLASMOSIS

Almost all carnivorous species can become infected with Toxoplasma gondii by eating raw meat containing infective forms of the organism. Cats are unusual in that they are the only animals in which the organism can complete its complex life cycle and be excreted in the feces. Depending on environmental conditions the form of the organism shed in the feces becomes infective in the soil in 1-4 days and is a potential source of infection for carnivores, noncarnivores and man.

In the cat, signs of toxoplasma infection vary from inapparent to such clinical manifestations as weight loss, fever, diarrhea, pneumonia, enceph-

alitis and ocular disease. The infection may be fatal in young kittens. Older cats usually survive.

In man, toxoplasma infection is usually inapparent. Sometimes primary infection is associated with fever, lethargy and lymph node enlargement. Infection of the eye with subsequent loss of sight may occur. The most severe consequence of human toxoplasmosis is fetal infection, acquired transplacentally when a pregnant woman undergoes primary infection. Following transplacental infection, babies can be normal and healthy at birth. Sometimes however, fetal death or abortion occurs. In addition, children can be born at term with ocular disorders, mental retardation, cleft palate or neurological defects.

Discovery that the cat is the natural host of toxoplasmosis aroused concern about cats as a hazard to human health. For a time controversial and confusing recommendations were forthcoming but further research has put the hazard of cats as an immediate source of Toxoplasma infection for man in a much less alarming perspective. In controlling transmission of toxoplasmosis to man, the first concern should be the most probable source of the infection, which is ingestion of incompletely cooked meat. The second concern would be hand washing after handling raw meat. Cat feces cannot be totally disregarded as a hazard. If they contain Toxoplasma oocysts and have incubated under appropriate conditions for 1-4 days they can be infective for man and remain so for more than a year. Areas known to be contaminated with cat feces should be avoided. Sand boxes should be covered when not in use. Pregnant women should be particularly cautious; not cleaning litter pans or sand boxes and wearing gloves while gardening. Since the Toxoplasma oocysts require 1 to 4 days to become infective after being shed in the feces, daily emptying of litter pans and proper disposal of their contents is an excellent control measure.

FELINE LEUKEMIA VIRUS

The feline leukemia virus (FeLV) is the cause of feline lymphosarcoma and has been shown to be readily transmitted from cat to cat. FeLV has been found in many tissues and in the urine and saliva of infected cats. The most likely routes of transmission are the saliva and urine, since cats groom each other and in many households use communal feeding dishes and litter pans. Infected cats may be sick or healthy and the presence of the virus can only be determined by appropriate laboratory testing.

There is no substantive evidence that FeLV infects man or causes human neoplasms, although numerous surveys have been carried out in search of FeLV in humans. The virus, however, can grow in human cells cultured in the laboratory and has been experimentally transmitted to other species of neonatal animals. These findings make it difficult to make unequivocal statements dispelling concern about its potential for human infection. In the light of present knowledge it is reasonable to recommend that pregnant women and infants avoid intimate contact with cats (especially known FeLV-carrier cats) and to observe sound hygienic practice when handling animals.

CAMPYLOBACTER ENTERITIS

It has only been within the last few years that the bacterial organism Campylobacter jejuni has been recognized as an important cause of enteric disease in humans. Patients have symptoms of fever, headache, abdominal pain and other body aches followed by severe diarrhea which becomes watery and often bloody. The illness is reported to last from a few days to three weeks.

Information is still emerging on the sources of infection for man. Domestic poultry have been shown to be carriers of the organism and recent work has implicated contact with infected household pets as another source of infection for man. Isolation rates of Campylobacter jejuni from domestic cats have varied from 2-45% depending on the age and location of the population studied. Most isolations have been made from kittens and disease in man has been reported after contact with kittens with diarrhea. However, a case in a young woman was recently reported in which her infection was acquired from an apparently healthy adult cat. When the patient changed brands of cat litter, the cat ceased to use its litter pan and began soiling the floor. The patient evidently became infected while cleaning up after her pet. Since litter boxes are commonly used for cats, infected animals are an unlikely source of infection for man but it is obvious from this case that the potential is there.

RINGWORM

Ringworm is a superficial infection of the keratinized areas of the body (hair, skin and nails) caused by a variety of fungi. The two most important species affecting the dog and cat are Trichophyton mentagrophytes and Microsporum canis. Both species can infect man. M. canis is very well adapted to cats and in approximately 90% of infected animals, no evident lesions can be seen. When lesions do occur they are most often on the face and claws.

The disease is transmitted to man by direct contact with infected animals or indirectly by spores in the hair and scales shed from such animals. Ringworm acquired from cats commonly infects the scalp or the body. On the scalp it begins with a small papule, the hair becomes brittle and the infection spreads in a wider circle, leaving scaly bald patches. On the body there are similar lesions with a tendency to form rings with reddish borders.

Since infected cats are so frequently without any clinical signs, this can be a difficult disease to avoid. Apparently healthy cats can be examined with an ultraviolet light which gives a brilliant greenish-blue fluorescence with hairs infected with Microsporum.

VISCERAL LARVA MIGRANS

Eggs of the dog roundworm (Toxocara canis) and less frequently the cat roundworm (Toxocara cati) reach the soil in the feces of infected animals.

After an appropriate incubation period they become infective for both animals and humans and remain so for a prolonged period. Children may ingest the eggs while eating soil or playing in feces-soiled areas. In man the eggs may be rejected or hatch in the intestine and migrate to various parts of the body (liver, lung, muscle, eye or central nervous system) via the lymphatic vessels. Depending on the site of migration the symptoms range from pneumonitis, swollen liver with abdominal pain, muscle and joint aches, cough, rashes and skin nodules. Convulsions may occur and if larvae reach the eye, visual deficiencies may result.

It is crucial to keep children from areas likely to be contaminated with animal feces. Worming of puppies and kittens also contributes to the control of visceral larva migrans.

SUMMARY

To summarize the subject of disease transmission from pets to man I will repeat that people are more likely to contract transmissible diseases from one another than they are from their pets. Nevertheless there are some diseases that are transmitted directly or indirectly by pets. This transmission is usually complex and generally requires close contact between susceptible people and animals or their excretions. Such contact often involves lack of common sense and gross violations of sound hygienic practice. Thus it is most common to see pet-borne zoonoses in children.

Responsible pet owners can participate in controlling zoonotic diseases by vaccinating their pets for rabies, limiting excessive contact of infants and pregnant women with pets, seeing to it that pet feces and urine are handled in a hygienic manner and supporting efforts to avoid the unrestricted presence of pets in parks, swimming areas, beaches, sand boxes and gardens.

COMPUTERIZED DIAGNOSTICS

A FELINE MEDICINE INFORMATION SYSTEM FOR THE '80s

Roy V.H. Pollock, D.V.M., Ph.D.

THE PROBLEM

Knowledge about veterinary medicine in general, and about feline diseases in particular, is growing at an ever-expanding rate. New diseases are being discovered, new tests developed, new treatments perfected. It is becoming increasingly difficult to keep up.

Practicing veterinarians and concerned cat owners spend a significant proportion of their time continuing their education. They read journals and textbooks, listen to recorded lectures and attend meetings and conferences. Is this enough? Probably not. The volume of information is now so great that it surpasses the recall capacity of the human mind.

For example, a recent textbook on feline medicine contains almost 700 pages describing over 400 diseases and conditions. For each of these there are a number of associated tests and therapies. Simply remembering all of this would be an overwhelming task even if the information remained static. But it doesn't. New information is continually added and old concepts are revised.

How does this affect medical care? It makes it difficult to ensure that every patient receives the very best of current thinking. Presently, we rely on the clinician's memory to link medical knowledge to patient care. The diseases that are considered, the tests that are ordered, and the treatments that are applied are limited to those the clinician knows and can recall. Uncommon ailments may be overlooked.

Textbooks and other references help. But these are arranged by disease, rather than by clinical sign; the information they contain can only be accessed after a potential diagnosis has been recalled from memory. Telephone consultation with experts can help broaden the search for diseases or assist with test interpretation and therapy, but these consultants cannot be contacted for every case.

What is needed is a practical, constantly available means to provide up-to-date information on diseases, tests and therapies according to clinical problem.

THE SOLUTION

The extraordinary capabilities of modern microcomputers offer the means to resolve the medical information problem. Indeed, a computer-based diagnostic information system is presently being developed at the Cornell Feline Health Center.

How would such a system work? First, the computer would be programmed to store all of the current information on a particular clinical problem, for example, coughing or loss of hair (alopecia). The source of each piece of information would be recorded so that it could be located and updated as new information became available.

When this is done rigorously, it turns out that there are 50 or more possible causes for any single clinical manifestation, say, diarrhea. The diagnostic challenge is deciding among them.

The problem may be compared to trying to identify a single child from among many on a school playground. A single characteristic (say, red hair in a child, or fever in a disease) seldom uniquely identifies an individual. But a collection of 5 or 6 attributes is often quite unique; there are very few children who are boys with red hair, cowboy boots, blue jeans, and a green sweater. Similarly, there are very few diseases manifested by simultaneous diarrhea, vomiting, fever, and low white blood cell count. However, not every patient with feline panleukopenia displays all of these classic findings. Likewise, the child may have taken off his sweater. So a diagnostic scheme must take into account the unique nature of patients; any one may not have all the classic findings, while another has some unexpected manifestations.

This is handled in the following way: When all diseases that could cause a problem have been identified, a search is made for characteristics that distinguish them. Some findings are much more helpful than others. For example, every sick cat will probably be depressed and lethargic, regardless of the causal agent; every child in the playground will have two legs. On the other hand, certain findings are quite helpful; some diseases typically cause fever, while others do not, so the presence or absence of fever is a useful criterion.

From a list of these discriminatory findings a series of questions is composed. The veterinarian and owner would work through these questions together. They would then ask the computer to match the findings in this unique patient against all known causes of the problem. The result would be a list of diseases to be considered, arranged in order of those that seem to fit best. For each disease, current information would be available "on-line" about probable outcomes, available tests and their interpretation, and potential treatments.

Doesn't this eliminate the need for the veterinarian? Not at all. First, a skilled clinician is needed to make critical observations. The presence or absence of a subtle heart murmur, the size of the spleen, or enlargements of lymph nodes can only be assessed by an expert. Furthermore, the resultant list of potential diagnoses must still be reviewed by a skilled clinician to determine which diseases should be ruled out first. The selection of appropriate diagnostic tests and therapies depends as much on the clinician's unique knowledge of this patient and client as on the disease condition.

But then why use the computer? The computer is simply a tool. It extends the clinician's memory in the same way the stethoscope extends his

ears or the x-ray, his eyes. Since its memory is prodigious and permanent, it can assure that no potential diagnosis is overlooked. Since its memory can be readily updated, it can deliver up-to-date information on diseases, tests and treatments. But like any tool, it is useless without a skilled master.

Computer-based diagnostic information systems have the potential to improve veterinary care. We are dedicated to making them a reality.

UPDATE ON INFECTIOUS DIARRHEAS

Sandy Baldwin, D.V.M., M.S.

Diarrhea is the result of any abnormality that leads to an increase in the amount of water in the feces. The increase could be due to a greater retention of water in the intestine, such as is seen with malabsorptive diseases. Alternatively, the increase could be due to a greater secretion of fluids by the intestinal epithelial cells.

In the normal small intestine, nature has tried to increase the area available for absorption of fluids, electrolytes, and nutrients by creating many folds in the surface. These folds are tall, fingerlike structures called villi, which project into the center of the intestine (the lumen). Covering the surface of the villi is a single layer of epithelial cells, the enterocytes, which are responsible for all of the absorption that takes place in the bowel. Therefore, any agent, infectious or otherwise, that damages these epithelial cells can lead to diarrhea.

The enterocytes are produced at the base of the villi in the crypt area. Due to the force of cells migrating out and up from the crypt, enterocytes are pushed up the side of the villus to the tip. Once they reach the tip they have reached full maturity and, due to the passage of intestinal contents, they are gradually eroded or abraded off, only to be replaced by the new cells migrating up from the crypts. In the crypts, the immature, rapidly-dividing cells are capable of secreting water into the intestinal lumen, but they are totally incapable of any nutrient absorption. Therefore, any agent that affects the crypt cells could lead initially to hypersecretion and watery diarrhea.

As the cells migrate up to the tips of the villi, they gradually lose their ability to secrete. But these cells also differentiate and maturate, becoming enzymatically active and therefore fully capable of nutrient digestion and absorption. Premature loss of the villus tip cells, thus, can lead to diarrhea in one of several ways. First, the loss of the mature cells means that nutrients can no longer be absorbed. Nutrients remain in the lumen and, by their mere presence, retain water. Second, as these nutrients pass further down the intestinal tract to the colon, they are digested by bacteria into more particles, which further retain water osmotically. Third, the cells that replace the mature cells at the tips of the villi are slightly less mature, still have some residual secretory function, and therefore secrete more water. A profuse, watery diarrhea is the result.

Diarrhea can be caused by many things, including either inherited or acquired anatomical changes. Strictures may be present at the kitten's birth, or may occur after peritonitis or pancreatitis. Functional changes may occur as side effects of prescription drugs. Other possible causes include dietary changes or overloads, malabsorptions and obstructions, chemical causes, allergic responses, and infectious causes (viruses, bacteria, fungi, protozoans, and endoparasites).

The following is a brief discussion of only some of the many causes of diarrhea. It is by no means intended to be all inclusive, since there are a myriad of factors involved in any one incident of diarrhea. The emphasis will be on the infectious causes found in cats, including the common bacterial causes. The order of presentation is not necessarily an indicator of the importance of each factor as a cause of diarrhea.

ENDOPARASITES

Endoparasites are probably the most common cause of diarrhea. These include roundworms, hookworms, and whipworms. Rarely does tapeworm infestation produce much in the way of clinical signs, unless masses of segments occlude the whole bowel. Endoparasites are seldom a severe problem except in the very young, very old, and debilitated animals, or in cases of very heavy or severe infestations. Also in this category would be included the protozoans. Coccidia is a problem primarily of the young. Giardia is a parasite that can often be difficult to diagnose. Toxoplasma rarely can produce diarrhea but the intestine is not the primary site of infection. Little more needs to be said concerning endoparasites, for proper diagnosis and specific administration of anthelmintics (worming treatments) will usually eliminate the parasites.

BACTERIA

Bacteria form another very common category of diarrhea-causing agents. Whereas it is theoretically possible that almost any bacteria could cause diarrhea, in all likelihood E. coli, Salmonella, and Campylobacter are the three most important ones involved. E. coli is probably one of the more common causes. It produces severe, watery diarrhea in many animal species including man, but is very amenable to treatment (see Treatment section).

Salmonella and Campylobacter are significant due to their human health relationship. Salmonella can cause a severe diarrhea in most animal species and can also be contagious to the pet owner. The sign of a local infection would be diarrhea. A systemic infection of Salmonella, a more serious problem, can produce the following signs: diarrhea, fever, lack of appetite, depression, and an increase in white blood cells (though a severe decrease, mimicking panleukopenia, could be seen initially). Diagnosis of Salmonella can be obtained by culturing the feces of animals with diarrhea. However, Salmonella organisms can be found in 5-20% of normal cats and dogs suffering no disease.

If your cat has diarrhea due to Salmonella, all that is needed for treatment is good nursing care and isolation. Antibiotics should only be used if your pet is suffering from systemic salmonellosis. In fact, antibiotics can make an intestinal disease become a systemic disease. The administration of antibiotics also prolongs the shedding of Salmonella and increases the carrier period. Systemic disease is very rare but it can be fatal; therefore, your veterinarian should be consulted if your cat has salmonellosis, and a rational plan of therapy can be formulated.

Campylobacter is an emerging disease in most species of animals. It can be passed from animals to man and vice versa. Its exact role as a serious cause of diarrhea in cats is still speculative, but cases have been reported in which both the owner and cat have had bouts with Campylobacter enteritis. In these reports, however, it was not known which species was infected first. (In addition, there have been many reports citing a high incidence of Campylobacter isolations from poultry in processing plants, so improper cooking of poultry products is a big factor to consider in human cases of enteritis.)

Signs of Campylobacter in most animals include fever, chills, nausea, diarrhea with and without blood, and occasionally vomiting. None of these signs are specific for Campylobacter alone. As in Salmonella, there appears to be a carrier state; anywhere from 4-45% of healthy cats have been shown to harbor the organism. Diagnosis can be difficult unless specific culture media is used. Treatment is usually straightforward and simple, consisting of fluids and sanitation precautions.

VIRUSES

Viruses are the smallest pathogenic organisms. At least 6 or 7 viruses are either known or suspected to cause diarrhea in the cat. These include feline parvovirus, feline leukemia virus, feline enteric coronavirus, feline calicivirus, feline astrovirus, and feline rotavirus.

Feline Parvovirus

There are many synonyms for feline parvovirus disease: feline panleukopenia, feline infectious enteritis, feline distemper, and feline ataxia. The virus attacks rapidly dividing cells which, in the cat, are found in the intestinal crypts. In the unborn kitten, the affected area would be the cerebellum, the portion of the brain so important for coordinated walking (hence the name feline ataxia, the inability to walk in a coordinated fashion). In the cat, the rapidly dividing crypt cells of the intestine produce new cells for the villi daily. In the disease state, these crypt cells are lost, the villi cells are then lost, no absorption takes place, and diarrhea results. The cat will have a profuse, watery diarrhea, fever, and no appetite. It will be very depressed and will die if untreated.

A good vaccination program, however, will virtually eliminate feline parvovirus disease from a cattery. By vaccinating all kittens at 6 to 8 weeks and giving a booster at 12 weeks of age, the kittens will be protected in about 95-99% of cases. (No vaccine regimen can be considered 100% effective, including those for humans.) To keep the adult cat protected, all that is necessary is an annual booster vaccination. Once the disease hits a cattery, good supportive therapy such as warmth, fluids, and food is all that can be done; there is no effective antiviral compound at this time, and antibiotics are ineffective. One other necessary step in prevention and control is good disinfection of the premises and eating utensils because the virus can survive up to a year in the environment. A

proper dilution of sodium hypochlorite (Clorox® 4 oz./gal. water) is an excellent disinfectant.

Feline Leukemia Virus

Feline leukemia virus can occasionally infect the same rapidly dividing cells and produce a disease that exactly mimics feline parvovirus. The prognosis, however, is vastly different. Whereas with good supportive therapy one might successfully treat 60 to 70% or more of cats with parvovirus, very few, if any, animals with feline leukemia virus can be treated successfully. It also must be remembered that a second manifestation of feline leukemia infection is lymphosarcoma. Infiltration of a tumor into the wall of the intestine prevents absorption of nutrients, and hence diarrhea occurs. Alternatively, a growing tumor could occlude the lumen of the bowel, allowing only the fluid portion of the feces to pass. This in turn gives an impression of diarrhea to the owner, when in fact the consequences are much more serious. This last type of diarrhea can also be seen with other tumors, namely adenocarcinomas and leiomyosarcomas.

Feline Enteric Coronavirus

Feline enteric coronavirus has been described as causing diarrhea. At a recent meeting of the American Animal Hospital Association, it was stated that this virus can cause anything from an inapparent infection to a mild diarrhea in kittens 4-12 weeks of age. It was stated to appear in 25% of all outdoor cats and probably would be found in all catteries. It has been isolated from both normal cats and those that show a diarrhea.

Feline Rotavirus

Rotaviruses are known to produce a diarrheal disease in many species. In some species, the significance fo the disease can be quite serious (cows, pigs, human infants). In the cat, however, the role is currently under investigation. It is known when cats contract the virus they will shed it in the feces for up to 12 days. The virus also has been shown to produce severe lesions on the tips of the villi in specific pathogen-free cats, but without any signs of disease. It is thought that infection of very young or newborn kittens could lead to diarrhea. Colostrum may provide necessary protection for most newborns that nurse soon after birth.

Feline Astrovirus and Feline Calicivirus

Feline astrovirus and feline calicivirus have also been isolated from cats with diarrhea, but their exact role in producing the diarrhea is not known at this time. In other species, rotavirus, astrovirus, and calicivirus infect the mature cells on the villus tip, little or no nutrient absorption takes place, some increased secretion occurs, and diarrhea results. In general, damage is less severe than what is seen with feline panleukopenia, and therefore the disease can usually be remedied with good supportive therapy.

TREATMENT

In general, treatment for all diarrheas, regardless of cause, consists of supportive therapy (also known as "tender loving care"). Most of the deaths that occur are due to dehydration. Dehydration can easily be tested by skin turgor. Pull a fold of skin gently away from the body, then release it. Rapid return is normal. If the fold remains, the pet is dehydrated. The lowest level detectable is approximately 5% dehydration. Death usually occurs around 12-15% dehydration. The range of detection therefore is quite narrow. Strong fluid therapy is usually all that is necessary for a successful outcome. Other very beneficial supportive care includes warmth, good nutrition, and common-sense nursing care.

Small amounts of warm water should be fed to the cat repeatedly unless it is vomiting or suffering from pancreatitis. (In that case, neither food nor water should be given.) If the water is retained for several hours, it should be followed with multiple feedings of a bland diet. The warm water aids in passage of material, approaching normal peristaltic (digestive) contraction. The multiple feedings of a bland diet (chicken and rice or mutton and rice) are particularly beneficial if allergy is the cause of the diarrhea.

Allergic responses are probably just as important as the infectious agents (bacterial and viral) as a cause of diarrhea, once endoparasites are eliminated. Occasionally, if the diarrhea stops while the cat is on the bland diet, gradual addition of the normal diet will allow the identification of the offending allergen. More than likely, however, the actual allergen causing the diarrhea may not be completely identified; control of diarrhea means altering the diet.

Antibiotics have a place in the therapy for diarrhea, but their role is very specific. Only in cases of <u>systemic bacterial</u> disease should antibiotics be given, and then they should be given systemically. The choice of antibiotics should be made only after culture and sensitivity testing. As mentioned, systemic disease is manifested by signs such as fever, anorexia, and depression.

Too often, antibiotics are given as a supportive measure when there is little indication of need. In fact, overuse of antibiotics can actually lead to diarrhea. What occurs is that the antibiotics destroy the natural balance or niches of the bacteria in the intestine. This allows normal commensal bacteria to overgrow or to occupy a new niche in the intestine. Both of these conditions can lead to diarrhea. Alternatively, the destruction of the normal intestinal flora allows pathogens to gain a foothold where they normally cannot, and diarrhea results. The absence of the protective flora also allows yeast organisms such as <u>Candida</u> sp. to colonize the intestine. This phenomenon of gut sterilization by antibiotics is particularly prevalent when oral antibiotics, especially nonabsorbable ones such as neomycin, are used. In addition, overuse of antibiotics kills off the most susceptible species of bacteria but leaves those species that are particularly resistant. Therefore, with long-term overuse of antibiotics, the pathogens become resistant to multiple drugs and are consequently very difficult to treat. Finally, it must be remembered that antibiotics have

no effectiveness against viruses and should not be used to treat viruses. To date, there are no antiviral compounds.

Another common class of drugs used to treat diarrhea are the motility modifiers. It is commonly assumed that in most or all cases of diarrhea, the intestines are hypermotile (i.e., their normal, spontaneous movement is accelerated), and this accounts for the diarrhea. In fact, motility is often reduced in diarrhea. By giving an antimotility drug, the intestinal motion is further slowed, thus allowing increased absorption of bacteria and their toxins, leading to increased severity of systemic signs. Movement of the intestinal contents is a protective mechanism, and to block this is very counterproductive. The only time antimotility drugs are indicated is when intestinal contractions are so rapid that an intussusception may occur, that is, when one section of the intestine enters into another section. The blood supply of the inside piece is shut off, the section dies, and a very serious condition results. Surgery to remove the dead tissue is the only correction once this devitalization or death and necrosis of the gut has occurred.

One of the few over-the-counter drugs that may be of any benefit for diarrhea appears to be Pepto-Bismol®, with its active ingredient bismuth subsalicylate. This is very effective in helping to absorb bacterial toxins and may have other side effects which are beneficial in treating diarrhea.

To summarize, give plenty of liquids (parenterally or intravenously if there is severe dehydration and/or vomiting), give antibiotics if systemic bacterial involvement is present (choosing the antibiotic on the basis of culture and sensitivity tests), give Pepto-Bismol® to aid in diarrheal and toxin control, and give frequent small amounts of warm water followed in time by small amounts of a bland diet.

It is hoped that this article will provide the cat owner an overview of the scope of potential infectious agents involved with diarrhea. It must be remembered that this is just the tip of the iceberg of myriad possible causes, and does not include noninfectious causes such as rodenticides, lead poisoning, insecticides, tumors other than feline leukemia, and foreign bodies swallowed by the cat. Even simple things, such as changing the diet too rapidly, may lead to a transient diarrhea. But by following the simple guidelines above, treatment can be successful.

SUGGESTED READINGS

Lora Miller, B.A.

HINTS ON WHAT TO LOOK FOR IN A BOOK ON CAT CARE:

1. Quality of information presented: Does the book give specific recommendations for specific problems (e.g., "Induce vomiting by giving 1/2 tsp. of salt if the cat has eaten A, B, or C, but not if it has eaten D, E, or F") or is it too general to be useful ("You might wish to induce vomiting")?

2. Quality and quantity of photographs and drawings: Are these clear and informative? A picture of a thin kitten with a caption on roundworms does not tell the reader very much; a detailed description of how to administer a pill is greatly helped by a simple drawing or photograph.

3. Background information: Does the book provide adequate background information so that the reader can understand what's going on, or does it immediately jump into a highly technical discussion?

4. Is the information arranged in a clear, well-organized manner? And is it useful? A person who has kept cats for fifteen years will not need a rambling dissertation on how to house and care for a new kitten.

5. If the book is to be used as an emergency reference, is the information arranged so as to be immediately and clearly accessible, or does the reader have to search the index and then wade through paragraphs and paragraphs of background material in order to locate the vital directions on what to do for the cat? The binding of the book is also of some concern here: a tiny paperback may cost much less than the hardcover edition, but if it requires the use of both hands to keep it from closing, it will be next to impossible to restrain an injured cat and follow directions to administer emergency treatment.

6. A few common-sense points to look for: What does the book suggest in the way of toys? (Wholehearted endorsement of string and wire toys, without mention of the possible dangers of swallowing these objects, may be an indication of an author who has not fully considered the consequences of his or her recommendations.) Are the breed standards up to date? (Some books are reissued every few years and yet retain the same old photographs of long-since retired champions.) Does the book mention the specific nutritional requirements of the cat? (Some economy-minded owners have tried feeding dog food, which lacks taurine, with resulting degeneration of the retina of the eye; a good reference book should warn against this sort of thing.)

7. Where was the book written? The British love their cats, and have published much information about them, but some of their cat care suggestions (such as age at which to spay, whether or not to declaw, etc.) are contrary to those of American veterinarians. The person who goes to see a veterinarian in the United States with a set of British ideas on cat care will probably run into stiff opposition.

Anatomy

Gilbert, Stephen G. Pictorial Anatomy of the Cat. Seattle: University of Washington Press, 1979. 120 p.

A dissection guide with well-labeled drawings; level of detail is probably adequate for most cattery owners, and this paperback should be readily available in bookstores.

Matthews, Valerie. Feline Anatomy series. Cat Fancy, May 1982 (vol. 25 #1) through November 1982 (Vol. 25 #11).

This seven-part series covers the digestive, musculoskeletal, cardiovascular, urological, respiratory, nervous, and reproductive systems of the cat. Articles cover normal anatomy and physiology of these systems, as well as common medical problems involving each one. The articles are concise and aimed at the cat owner or cattery owner. Back issues should be available at your local library.

Normal Fetal Development and Congenital Birth Defects

Foley, C.W., Lasley, J.F., and Osweiler, G.D. Abnormalities of Companion Animals: Analysis of Heritability. Ames, Iowa: Iowa State University Press, 1979. 270 p.

This book is arranged in two parts: first, an introduction to feline genetics, and second, descriptions of the known and suspected genetic abnormalities of the cat. Other species are also discussed. Birth defects are arranged by organ system affected (cardiovascular, bones and joints, etc.), so it is relatively easy to find a particular one of interest. The breeder may be displeased to find that folded-over ears (as in Scottish folds) and lack of tail (as in Manx) are regarded as birth defects, with the admonition to not breed affected animals; however, these features are undeniably a departure from "normal" cat development.

Saperstein, G., Harris, S., and Leipold, H.W. "Congenital Defects in Domestic Cats." Reprinted by Ralston Purina, Checkerboard Square, St. Louis, MO 63188; copies on request.

This booklet, written for veterinarians, summarizes known birth defects of cats, their causes, frequency of diagnosis, and means of identification. The terminology is more concise and technical than that used in the previous reference.

Feline Genetics

Jude, A.C. Cat Genetics. Neptune City, N.J.: TFH Publications, 1967. 126 p.

An introduction to basic principles of genetics and their applications to

cat breeding. The genetics of breeding as presented here are still sound, though the fancier will recognize the cats in the photographs as examples of an outdated British standard. This book should be readily available, either through your local pet store or direct from the publishers.

Robinson, Roy. Genetics for Cat Breeders (2nd Edition). Oxford and New York: Pergamon Press Ltd., 1977. 202 p.

This book is more technical and detailed than the one listed above. It begins with a concise introduction to the principles of genetics, with their relation to feline reproduction; later chapters demonstrate how these principles should be applied to breeding for specific traits. A chapter on birth defects helps to alert the breeder to those abnormalities which may be inherited.

Feline Reproduction

Stapley, Ronald. "Factors Affecting the Reproductive Behavior of the Feline. I. Photoperiodicity and Controlled Exposure to Light." Friskies Research Digest 14 (1):8. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

This brief report indicates that a controlled light cycle (14 hours light, 10 hours dark) may eliminate winter anestrus, allowing queens to cycle and be bred successfully throughout the year.

Nutrition

Committee on Animal Nutrition, National Research Council. Nutritional Requirements of Cats. Washington: National Academy of Sciences, 1978.

This pamphlet summarizes what is known of feline nutritional requirements. The standards for evaluating nutritional value, such as "metabolizeable energy," may be unfamiliar to the reader, and the approach is more theoretical than practical; readers will discover, for example, that energy requirements for an adult cat are about 65 kcal/kg of body weight, but not how much of what sort of cat food this requirement represents. Despite these limitations, the work is still the most basic research available on feline nutrition.

Morris, James G. "New Research in Feline Nutrition." Friskies Research Digest (1976) 12 (1):1-3. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

This article discusses the ability of the cat to digest carbohydrates and ways to maximize digestibility of grains as carbohydrate sources.

Morris, James G. "Nutritional Peculiarities of the Cat." Friskies Research Digest (1977) 13 (3):1-4. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

Discusses the cat's requirements for vitamin A, niacin, fats, and taurine.

Morris, James G. "Arginine: An Essential Amino Acid for the Cat." Friskies Research Digest (1978) 14 (3):6-7. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

Discusses the vital role of arginine and the rapidly severe-to-fatal reactions to arginine deficiency.

Morris, James G. and Rogers, O.R. "The Cat's High Protein Requirement." Friskies Research Digest (1978) 14 (2):1-4. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

Explains the function of protein in normal body maintenance and examines several theories as to why the cat has a relatively high protein requirement.

Kronfield, D.S. "Feeding Cats and Feline Nutrition." (Continuing Education Article #6) Compendium of Continuing Education for the Practicing Veterinarian. 5 (5):419-423.

This article discusses specialized nutritional requirements of the cat. The author gives a recipe for a homemade cat food, and also discusses the shortcomings of the major brands of cat food (without identifying those brands by name). Your veterinarian should have this article or be able to obtain it for you.

First Aid

Gerstenfeld, S.L. Taking Care of Your Cat. Reading, Mass.: Addison-Wesley Publishing Co., 1979. 250 p.

This manual for cat owners helps advise which problems are veterinary emergencies and which are more minor problems that can be handled at home. Home treatments for minor injuries are detailed, along with instructions on what to do for the seriously-ill cat until it can be taken to the veterinarian. The first section of the book also has a good review of feline anatomy.

Kirk, R.W. First Aid for Pets. New York: E.P. Dutton (A Sunrise Book.), 1978. 278 p.

Dr. Kirk's text has boldly-labeled instructions on how to proceed in a given emergency, as well as more detailed descriptions of the causes of

or any other book on veterinary first aid, before the actual emergency arises, so as to be familiar with the format and avoid wasting crucial time searching for the necessary information.

Carlson, Delbert G. and Giffin, J.M. Cat Owner's Home Veterinary Handbook. New York: Howell Book House, Inc., 1983. 391 p.

A very thorough text on feline medical problems for the cat owner. Specialized terminology is defined, though the owner may have to search for the definition in a cross-referenced portion of the book. Photographs showing how to apply bandages or restraint, how to clean ears, etc., are arranged in a useful sequence so that the owner can see just what to do. Veterinary emergencies and how to handle them are described in the very first chapter, making the information readily accessible, but the cross-referencing there may cause the owner to waste valuable time in an emergency; the person trying to stop a severely bleeding wound will not appreciate finding that the emergency section on wounds says, "See Circulatory System" for a detailed discussion of how to control bleeding.

Behavior

Beaver, Bonnie. Veterinary Aspects of Feline Behavior. St. Louis: C.V. Mosby Co., 1980. 217 p.

This book is oriented to the behavior and behavioral problems of the pet cat. Written by and for veterinarians, some sections (such as the one on feline behavior of sensory and neural origin) may contain too much medical terminology for the average reader; later chapters are much more descriptive and do not require a specialized vocabulary to be comprehensible. Numerous photographs and drawings supplement the text, though cat owners will not derive a great deal of information from photographs of cats in the various postures of stalking prey.

Hart, B.L. Feline Behavior. Santa Barbara, Calif.: Veterinary Practice Publishing Co. (P.O. Box 4457, zip code 93103), 1978. 110 p.

This book is a collection of Dr. Hart's columns on behavior from Feline Practice. Normal behaviors and their underlying genetic, hormonal, environmental, and other causes are discussed, as well as behavioral problems and suggested therapies. The book is practically oriented and avoids the overuse of confusing, technical terminology.

Leyhausen, P. Cat Behavior. New York: Garland STPM Press, 1979. 340 p.

This is the recent English translation of a classic German work on behavior of domestic and wild cats. It is written from the viewpoint of a behavioral scientist rather than a veterinarian, and so is primarily concerned with normal, natural behavior patterns rather than how to handle specific behavior problems of pets. Very detailed, but not overly technical.

Respiratory Diseases

Cotter, S.M. "Feline Respiratory Diseases." Friskies Research Digest. (1978) 14 (4):4-5, 15. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

An overview of some of the common causes of respiratory tract dysfunction in the cat. The article should not be used as a guide to home diagnosis and treatment; it merely serves to inform the curious reader of some of the possible respiratory problems of the cat and their relative frequency of occurrence.

Feline Leukemia

Hardy, W.D. "Current Status of FeLV Diseases." (Parts I, II.) Friskies Research Digest (1979) 15 (2):1-3, 15; 15 (3):1-3, 14. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

Discusses disease syndromes associated with FeLV and tests used in FeLV detection. Also included is a discussion of the signs and diagnosis of FIP (feline infectious peritonitis). The owner should read the section on FeLV tests and the meaning of positive and negative results with special care, as understanding what the tests do and do not mean is vital to understanding a cat's prognosis.

Feline Infectious Peritonitis

Feline Infectious Peritonitis. (Client Information Series.) Newly revised by Dr. Jeffrey Barlough. Your veterinarian can order copies from Veterinary Practice Publishing Co., P.O. Box 4457, Santa Barbara, CA 93103.

Scott, F.W., Hoshino, Y. and Weiss, R.C. Feline Infectious Peritonitis. Feline Information Bulletin No. 4, Ithaca, N.Y.: Cornell Feline Research Laboratory, 1978.

Zoonotic Diseases - General

Acha, P.N. and Szyfres, B. Zoonoses and Communicable Diseases Common to Man and Animals. Scientific Publication No. 354. 1980. Pan American Health Organization, Pan American Sanitary Bureau, Regional Office of the World Health Organization, 525 Twenty-third St. N.W., Washington, DC 20037.

This book provides a description of diseases communicable to man from animals, arranged by the type of disease-causing organism: bacteria, virus, fungus, etc. Topics covered include signs of the disease in man and animals, method of transmission, and steps required for diagnosis. The reader without a scientific background may wish to keep a dictionary at hand while reading this book.

Schnurrenberger, P.R. and Hubbert, W.T. An Outline of the Zoonoses. Ames, IA: Iowa State University Press, 1981. 157 p.

A more brief coverage of zoonotic diseases than the above, also arranged by causative agent. This text is more concise and technical than the previous one; a medical professional would find it useful.

Both of the books described above concern diseases of domestic animals in general, not just diseases of cats, and so do not emphasize the growing importance of cats in the transmission of certain zoonotic diseases such as rabies and plague.

Zoonotic Diseases - Specific

Baldwin, S. "Feline Toxoplasmosis: The Disease and its Public Health Significance." Perspectives on Cats, Spring 1983. Ithaca, N.Y.: Cornell Feline Health Center.

Written for the lay reader, this article explains the life cycle of the Toxoplasma organism, the disease as it affects the cat, and the consequences of human infection, with a list of guidelines for prevention of human infection.

Frenkel, J.K. "Toxoplasmosis in Cats and Man." Feline Practice (1975) 5 (1):28-41.

This article is written for veterinarians, so some of the terminology may be unfamiliar to the cat owner; however, the section on public health aspects of toxoplasmosis is quite readable and provides useful guidelines for minimizing the risk of transmission to man.

Other Topics of Interest

Ettinger, S.J. "Adult Feline Cardiac Disease." Friskies Research Digest (1980) 16 (2):1-3, 14-15. (Carnation Co., P.O. Box 128, Pico Rivera, CA 90660.)

Discusses the causes of feline heart disease, signs of illness in affected cats, and physical and laboratory tests for heart disease.

McDonough, Susan, and Lawson, Bryna. The Complete Book of Questions Cat Owners Ask Their Vet. Philadelphia: Running Press, 1980.

A guide for cat owners, to answer all those questions that are remembered every time you get home from a trip to the veterinarian's office.

Siegmund, O.H. et al. (ed.) Merck Veterinary Manual. Rahway, N.J.: Merck and Co., Inc., 1979. 1680 p.

A pocket reference for veterinarians. Concise information on a wide variety of topics, including diseases (organized by body systems), as well as toxicology, nutrition, etc. Some terminology may be confusing to the person with no background in biological sciences.

Wright, M. and Walters, S. (ed.) The Book of the Cat. New York: Summit Books, 1980. 256 p.

This British book discusses all aspects of feline biology, care, medical treatment, breeds, and breeding. The editors note British-American differences of opinion on cat care and management (such as age at which to castrate or spay, whether or not to declaw) without overemphasizing the English viewpoint. Information is concise but adequately detailed, and designed to be read by the average cat owner. The sections on cat genetics, anatomy, and behavior are especially good. The chapter on first aid is less complete; an owner should have another, more detailed first aid reference in addition to this one. The list of common feline poisons is somewhat useful, though poisonous houseplants and Tylenol (acetominophen) have been omitted. Breeders in the United States will disagree with the standards and descriptions given for purebreds, as these are British.

Client Information Series.

These pamphlets provide concise information on a wide variety of topics, aimed at the cat owner with no previous medical training. Your veterinarian may have these available, or may order them from Veterinary Practice Publishing Co., P.O. Box 4457, Santa Barbara, CA 93103. Cost is $10/50 copies. Topics covered include:

Feline Abscesses
Heart Disease in Cats
Castration of the Cat
Dental Care of Cats
Skin Diseases of Cats
Ear Diseases of Cats
Euthanasia
Feline Infectious Anemia
Feline Infectious Peritonitis
Feline Leukemia
Feline Urologic Syndrome
Care of Mother Cats and Kittens
Medical Care of Old Cats
Nutrition in the Cat
Medicating and Force-Feeding a Cat at Home
Eye Diseases in Cats
External Parasites of the Cat
Internal Parasites of the Cat
Breeding and Reproduction in Cats
Upper Respiratory Infections in Cats

GLOSSARY

anamnestic: pertaining to memory. When the immune system is exposed to an antigen, some immune cells are produced which "remember" this antigen. Upon a second (or third) introduction, the immune system is therefore ready to respond more quickly and in a more pronounced manner.

ancillary: additional. An ancillary test is an additional test to help confirm a diagnosis.

anorectic: suffering from anorexia, the loss of appetite.

anorexia: lack of an appetite.

anteriorly: pertaining to a position of the body towards the head.

antigen-antibody-complement reaction: occurs in the body when the FIP virus(or any foreign antigen) is attacked by antibody. A group of proteins collectively known as complement are then activated. These aid in the destruction of the virus and also help to recruit other cell types to aid in the destruction. These cells can also injure body tissues and lead to the signs of FIP disease.

Arthus-like reaction: a reaction occuring in the body when the FIP virus is attacked by antibody near blood vessels, resulting in damage to the vessels, which then leak fluid. It can appear as swellings in the skin or fluid build-up in the chest and abdomen.

ash: the residue left after burning off the organic matter of feed; made up of the mineral elements.

ataxia: incoordination. An inability to control the muscles correctly.

auto-immune: a disease state where the body builds up an immune reaction against its own cells and tissues, causing their destruction.

B carotene: precursor of Vitamin A, found in plant sources.

bacterial folliculitis: a reaction produced by hair follicles when injured by a bacterial infection, resulting in pain, swelling, redness and heat.

bilaterally symmetrical: of the same size and dimension, occurring on both sides of the body.

biological value: the ability of a protein source to supply amino acids in the relative amounts needed to form tissues and nitrogen-containing compounds; a measure of the efficiency of the use of a protein source.

biotin: a member of the B vitamin group.

caloric density: energy supplied per volume of feed; fat has high caloric density.

calorie: basic unit of energy - the heat needed to raise the temperature of 1 gm of water 1° C. 1000 cal = 1 kcal.

caudal: pertaining to a position of the body towards the tail.

cell-mediated immunity: one of the cat's defense mechanisms against invading infection. It involves many cell types that work together in order to destroy foreign material.

CHO or carbohydrates: sugar, starch, cellulose; digestible and non-digestible nitrogen-free substances.

cutaneous: pertaining to the skin.

cyanotic: having a blue tinge to the skin due to inability to get enough oxygen to the tissues. This can be due to an inability to breathe correctly or due to poor circulation of the blood.

desensitization: a treatment to control allergies where the particular substance the animal is allergic to is injected into the skin in a very small amount and then gradually increased in amount until high doses are injected. If successful, the animal's immune system loses the ability to respond to this substance.

digestion: series of processes in the gastro-intestinal tract in which food is broken down in size, solubilized and absorbed.

dorsal: an area of an animal pertaining to the back, or to a position more towards the back than another stated point of reference.

dorsum: the back or upper surface of an animal corresponding to the backbone.

dry matter: 100% of matter minus % total moisture; dry ingredients of foods assuming the complete absence of water.

effusion: a condition where fluid escapes a body cavity such as the chest or abdomen. This fluid can come from blood vessels, lymph, etc.

ELISA: Enzyme Linked Immunosorbent Assay. A very sensitive procedure done on a cat's blood sample to detect the presence of antibodies in the blood. A series of different steps results in a color change so that a visual estimate of the amount of antibody can be taken.

endemic: a disease that is always present in a certain area, but due to the presence of antibodies, not many animals succumb to it.

endogenous: of internal origin.

endometritis: a reaction to injury by the tissues lining the uterus which the fetus normally implants. The injury can be due to infection, poisons, traumatic injury, a dead fetus, etc.

endoparasites: parasites found on the external surface of the body.

energy: factor which allows cells of the body to perform work; sources include carbohydrates and protein.

essential amino acids: amino acids (subunits which combine to form proteins) which cannot be synthesized in the body fast enough for normal function (arginine and taurine are examples); must be supplied in the diet.

ethology: the scientific study of animal behavior.

etiologic: pertaining to the causes of disease.

excoriation: a loss of skin or part of skin due to the animal's own biting or scratching.

fibrosarcoma: disease in which fibrous and connective tissue cell types proliferate beyond control. Tumors arise in many locations in the body (multicentric) as a result of FeSV infection.

fistula: an abnormal opening in a tissue or organ.

FOCMA: feline oncornavirus-associated cell membrane antigen. A protein produced by cells in response to FeLV infection, and found on their surfaces.

genome: the complete set of hereditary factors found in the chromosomes.

glomerular: pertaining to the glomerulus, a tuft of small blood vessels found throughout the kidney, where blood is filtered in order to remove wastes.

granuloma: a firm, rounded lesion found anywhere throughout the body resulting from the body's attempt to destroy a very resistent foreign agent. The causative organism or foreign matter exists in the center of this lump and is surrounded by the immune system's reacting cells trying to digest this matter.

growth: process including multiplication of cells, increase in cell size and differentiation of function.

hemogram: a procedure done on blood in order to determine the type and numbers of the different blood cell types. Since cell types or their concentrations can change as a result of various infections, the hemogram can be used to help diagnose different diseases.

heritability: the proportion of the total observed variation in a population that is due to genetic causes.

heterosis: see hybrid vigor.

heterozygous (noun: heterozygote): having an unmatched pair of alleles.

histopathology: microscopic examination of tissues removed from the body. Diseased tissue reacts in various ways to different diseases, and its microscopic appearance can be used to help diagnose a disease.

homozygous (noun: homozygote): having a matched pair of alleles.

horizontally transmitted: transmission between two animals, other than dam and offspring. It may occur through direct contact between the animals or by being in an area where an infected animal has been.

hybrid vigor: the added vigor found in crossbred progeny, also called heterosis; the result of high levels of heterozygosity.

hyperesthesia: a condition where the animal overreacts to certain stimuli such as touch, pain, noise, etc., or is excessively sensitive.

immunocompromised: a condition where the cat's immune system is not working sufficiently to produce the antibodies, etc. necessary to defend itself against invading organisms. These cats will not respond favorably to vaccination by producing antibodies. They are also much more susceptible to many illnesses. There are various causes including genetics, a prolonged illness, and feline leukemia.

Immunofluorescent Coronaviral Antibody Test: a precedure done on a cat's blood sample to detect the presence of antibodies in the blood. Materials are used that fluoresce or produce light under ultraviolet light. Samples from cats that do not have antibodies will show little or no fluorescence while those with antibodies will show more fluorescence.

KELA: Kinetics-based Enzyme-Linked Assay. A new procedure done on a cat's blood sample to detect the presence of antibodies in the blood.

labile: unstable. A labile virus cannot survive for long outside of its natural habitat (i.e. in a cat), and therefore dies quickly and will not infect other cats for a long period of time.

lesions: any abnormality in a tissue or part of the body.

leukopenia: a deficiency of white blood cells in the blood.

lipids: fats, insoluble in water (fat soluble vitamins, cholesterol, fatty acids), condensed energy reserves.

locus (plural: loci): the physical location on the chromosome where partic ular alleles are situated.

lymph nodes: numerous oval or round glands which contain lymph, a fluid which "washes" bacteria and foreign particles from the tissues, transports fat from the intestines, and supplies lymphocytes (a type of white cell) to the blood.

lymphosarcoma: disease in which lymphocytes, cells normally found within lymph nodes and in blood, proliferate beyond control. They produce tumors and interfere with the functions of various organs.

maintenance: state in which there is neither gain nor loss of nutrients by the body; equilibrium.

melanoma: disease in which pigmented cells in the body and most commonly found in skin proliferate out of control.

mesenteric: pertaining to the mesentery, a fold of tissue attaching the intestines to the abdominal wall. It contains blood and lymph vessels supplying the gut, along with lymph nodes which help to prevent bacteria and other foreign agents from invading the body.

mononuclear phagocytes: a type of white blood cells whose function is to eat and digest any foreign material they find in the body. FIP virus is ingested, but the mononuclear phagocyte cannot digest and destroy it.

morphology: the study of structure and form.

nitrogen balance: an equilibrium of nitrogen gained from the diet and that lost from the body; a measure of protein and energy sufficiency.

nodule: a small, firm lump that can be detected by touch.

NRC or National Research Council: member of the National Academy of Science, which updates animal dietary requirements.

nucleus: the location of the chromosomes within a body cell.

nutritional secondary hyperparathyroidism: a syndrome seen in animals maintained on a high phosphorus-low calcium diet. In an attempt to maintain sufficient calcium in the blood and soft tissues, bone is resorbed. Fractures often occur.

otoscopic exam: examination of the ear with the use of an instrument known as an otoscope.

ovum (plural: ova): the female reproductive cell or egg.

papule: a small, firm bump on the skin surface due to an accumulation of any cell type immediately beneath the skin surface.

paresis: weakness. Posterior paresis is characterized by a weakness in the hind legs which can range from a slight incoordination to an inability to move the legs very well at all.

passive hemagglutination: clumping of red blood cells in a blood sample due to the presence of antibodies linking the cells together.

pediculosis: lice infestation.

pedigree: the "family tree" of an individual, listing its ancestors back through several generations.

perineum: an area of the outer surface of the body between the thighs and below the tail.

peripheral nerve: a nerve outside the central nervous system, i.e. not in the brain or spinal cord. It can be any nerve found in the legs, trunk, head, etc.

phenotype: the physical appearance of the animal.

plasmapheresis: a process whereby the blood is separated into 2 fractions, the blood cells and the liquid plasma which contains proteins, nutrients, antibodies, etc.

preneoplastic: a condition which is not yet out of control, but which exhibits many signs indicating it may become so.

protein: organic compound composed of amino acids, makes up major component of soft tissues and organs; necessary in the diet for tissue growth and repair.

proviral integration: attachment of the viral genome (provirus) into the host's genome.

psychogenic: having an emotional or psychological cause.

purified diet: feed consisting of purified, often synthetic, sources of various nutrients. Diets used in studying essential dietary elements may withdraw a specific nutrient to determine its function.

purulent otitis externa: pus-forming infection of the ear.

pyoderma: a pus-forming skin lesion.
pyometra; pyometritis: a condition of pus in the uterus.
pyothorax: pus in the chest cavity.
qualitative trait: one governed by a few major genes, each with an obvious effect.
quantitative trait: one governed by many gene pairs, each with a small, often immeasurable, effect.
recessive: referring to an allele whose effect is concealed by a dominant allele when the two are present together.
seborrhea: a disease condition of the skin where the sebaceous glands, which produce an oily substance, become overproductive. This results in a greasy, scaly skin.
septicemias: presence of bacteria in the blood. Natural immune defenses would normally destroy these bacteria before or soon after reaching the bloodstream.
serology: a test done on a blood sample collected from a cat to determine if antibodies are present to a specific infectious particle. A seropositive result indicates that the animal has been exposed to an agent that resulted in the cat producing antibodies against it.
sex chromosomes: the pair of chromosomes that determine the sex of the individual (called X- and Y-chromosomes in mammals).
sex-influenced trait: one whose expression varies for males and females of the same genotype.
sex-limited trait: one which can be expressed in only one sex, e.g., milk yield in females.
sex-linked trait: one controlled by alleles on the sex (usually X) chromosome.
sibs: brothers and sisters. Full sibs share both parents; half sibs have one parent only in common.
sperm (spermatozoon; plural: spermatozoa): the male reproductive cell.
SPF or specific-pathogen-free: animals obtained by C-section and raised in an environment free of certain viruses and bacteria.
squamous cell carcinoma: a type of cancer resulting in uncontrolled growth of cells in the skin. It is commonly seen as a red oozing sore around the ears of white cats.
Staph Protein A column: part of the plasmapheresis process (see plasmapheresis). The Staph Protein A is adhered to the column. Plasma is then passed through the column, and antibodies "stick" to the protein. With this method, antibodies can be removed from blood and then returned to the animal.
steatitis: inflammation of fat. A painful condition resulting from insufficient vitamin E intake on a relatively high fat diet. Cats lose their appetite, develop a fever, refuse to move and may die if untreated.
subcutaneous: an area underneath the skin.
taurine: an essential amino acid needed by the cat; deficiency results in retinal damage and vision impairment.
test mating or test cross: a mating specifically designed to determine the genetic makeup of an individual; usually done using the recessive homozygote for the trait.
thoracic: pertaining to the chest.
urolithiasis: urinary stones (calculi); result in disease in cats from blockage of the urethra and obstructing the flow of urine.

ventral: the area of an animal pertaining to the belly or in a position more towards the belly than another stated reference point.

vertically-transmitted: transmission from dam to offspring, either via the placenta to the fetus or through the milk.

vesicle: a fluid-filled swelling occurring in the skin or mouth.

virus neutralization: a procedure done on a cat's blood sample to detect the presence of specific antibodies in the blood. Some antibodies will not prevent a virus from infecting the cat's cells. Those that do prevent infection are called neutralizing antibodies. In this test a laboratory virus is mixed with a portion of the blood sample and then added to a cell culture. Any antibodies in the blood will neutralize the virus and thereby result in a reduced cell infection.