Temperature - 100.5 -102.5 F
Heart rate - 180 beats/minute for puppies
60-160 beats/ minute for most adult dogs
180 beats a minute for toy breeds
Respiration - 10 to 30 breaths/min
Pulse - 60 to 120
Gestation - 62 days
Estrous Cycle - 4 to 6 months
Estrus - 9 days
Average lifespan - 10 to 14 years
Pet Health Topics

• http://www.vetmed.wsu.edu/ClientED/problems_diagnoses.asp
GIVING ORAL MEDICATION
Vaccination on cats
The vaccines

- Rabies
- Panleukopenia (Distemper)
- Rhinotracheitis
- Calicivirus
- Feline Leukemia
- Feline Infectious Peritonitis
## Feline vaccination guidelines

<table>
<thead>
<tr>
<th>Vaccine type</th>
<th>Age at vaccination</th>
<th>Booster</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVR</td>
<td>6-8 wks</td>
<td>12 weeks</td>
</tr>
<tr>
<td>FCV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FeLV, FIP, clamydia*</td>
<td>12 wks</td>
<td>1 yr</td>
</tr>
<tr>
<td>Rabies</td>
<td>16 wks</td>
<td>1 yr</td>
</tr>
</tbody>
</table>

* non-core vaccination, use only risk cat
The changes were aimed at standardizing where different vaccines were injected on every cat.

The new vaccination site protocols would:

- reduce the risk of vaccine-associated fibrosarcomas
- aid in the study of any vaccination-site related problems by making it easier to trace problems to the use of a specific vaccine
Protocols

Vaccines containing antigens panleukopenia, feline herpes I, feline calicivirus (+/- chlamydia) should be administered over the right shoulder (RF) as distally as practical according to the manufacturer's recommendations.

Vaccines containing leukemia virus antigen (+/- other antigen except rabies) should be administered in the left rear leg (LR) as distally as practical according to the manufacturer's recommendations. (Leukemia = Left)
Vaccination sites on the cat
Panleukopenia
Rhinotracheitis
Calicivirus
Chlamydia (intranasal or right fore limb – vaccination in scapular region no longer recommended.)

Any Rabies Vaccine Antigen

Any Leukemia Virus Antigen
Vaccines containing rabies antigen (+any other antigen) should be administered in the right rear leg (RR) as distally as practical according to the manufacturer's recommendations (Rabies = Right)

Other intramuscular injections should be administered avoiding the right rear leg. Other subcutaneous injections should be administered on a side of the body or over the left shoulder (LF) as distally as practical. The interscapular and dorsal spinous regions should be avoided for all injections.
Perhaps the most obvious change the cat breeder must incorporate into their vaccinating routine of kittens is to no longer give SQ injections right between the shoulder blades. Instead, move the injection site as far down the right shoulder as is practical. This may seem like a small difference but it is significantly important.
• Feline panleukopenia, rhinotracheitis, calicivirus, and chlamydia vaccines should be administered subcutaneously (SQ) on the right shoulder. These vaccines are usually combined in a 3 or 4 way single vaccine injection. Rabies vaccinations should be administered SQ on the right rear limb, as far down on the limb as possible.
• FeLV vaccines should be administered SQ on the left rear limb, as far down on the limb as possible.

• Any time you need to give your cat a subcutaneous injection other than the above mentioned vaccines, it should be administered on the side of the body or over the left shoulder as low on the leg as is practical.
• The right hind leg has been "reserved" for rabies vaccinations. That means that other intramuscular injections (antibiotics, oxytocin etc.) should always be administered in the left hind leg, as far down the leg as is practical

• No injections should be given directly between the shoulder blades or along the backbone
## Dog vaccines

<table>
<thead>
<tr>
<th>Core vaccine</th>
<th>Non-core vaccine</th>
<th>Not recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabies</td>
<td>Bordetella</td>
<td>Adenovirus</td>
</tr>
<tr>
<td>Distemper</td>
<td>Distemper-measles</td>
<td>Coronavirus</td>
</tr>
<tr>
<td>Parvovirus</td>
<td>Lyme disease</td>
<td>Giardia</td>
</tr>
<tr>
<td>Adenovirus-2</td>
<td>Leptospirosis</td>
<td></td>
</tr>
</tbody>
</table>
Progeneration & Heat Cycle

Copyright Jack Vanderwyk (c)
A variety of estrogens have previously been recommended and used as treatments for mismating.

Two of the most popular formulations have been diethylstilbestrol (DES) and estradiol cypionate (ECP) within 5 days of breeding.

Interestingly, there is very little objective data to support either the safety or efficacy of these drugs for treating mismating in dogs, and essentially none in cats.
ECP

0.125-0.25 mg/kg in 40 hrs after mating

0.022 mg/kg IM within 3 days after mating
• estrogen therapy in bitches is associated with a high risk of inducing uterine disease such as pyometra and some risk of causing a lethal aplastic anemia
• Additionally, the dosages of estrogen and timing of treatment that appear to minimize risk of these disease are poorly effective in preventing pregnancy
• estrogen therapy for mismating in bitches is not only unsafe, but often ineffective in preventing pregnancy
Termination of Pregnancy with Prostaglandin F\textsubscript{2alpha}

- Prostaglandin F\textsubscript{2alpha} (PGF, Lutalyse(R), dinoprost tromethamine) is a hormone that induces luteolysis in many species, including dogs.
- Because progesterone is necessary throughout gestation for maintenance of pregnancy, PGF-induced death of the corpus luteum leads to termination of pregnancy.
- PGF also has the ability to stimulate uterine contractions, which may contribute to its abortifacient activity.
• The canine corpus luteum is essentially unresponsive to PGF prior to diestrus day 5, then becomes progressively more susceptible to luteolysis through gestation. As a consequence, lower doses of PGF are required to induce abortion later in gestation.

• PGF bid, either for 4 days (less than 4 weeks of gestation) or until abortion is complete (after 4 weeks). In the later case, the bitch should be monitored daily by palpation or ultrasound to evaluate whether abortion has taken place.
PGF treatment has a number of unpleasant side effects in dogs, including vomition, panting, cramps excessive salivation and defecation. These effects can be ameliorated to some extent by walking the animal immediately after treatment. Because of these adverse effects of PGF, treatment should be conducted in a veterinary clinic.
Two important precautions should be recognized with respect to use of PGF:

1. Women of childbearing age and people with asthma or other respiratory problems should use extreme caution in handling PGF solutions. This drug is readily absorbed through the skin and can cause uterine contractions and bronchospasm in exposed persons.
2. PGF analogs such as cloprostenol are not approved for termination of canine pregnancy. They are very much more potent than PGF, and using the analog at the same dosage as PGF can be lethal.

**PGF treatment is an effective treatment for termination of pregnancy in bitches.**

Properly administered, it is also safe and does not appear to have adverse effects on future reproductive performance of the bitch.
• PGF can also be used to terminate pregnancy in cats, at least after day 33 of gestation
Other Methods for Terminating Canine Pregnancy

- These treatments either are not currently available or cannot yet be recommended due to lack of data from clinical trials

**Dopamine agonists** (bromocryptine and cabergoline):

- **Prolactin** is necessary to support function of the canine corpus luteum, and secretion is inhibited by dopamine. The drugs, which bind to dopamine receptors in the pituitary gland, suppress prolactin secretion and can terminate pregnancy in dogs by suppressing progesterone secretion from the corpus luteum

- effective only after about 25 days of gestation
• **Epostane**: This drug inhibits steroid hormone synthesis by inhibiting the enzyme that converts pregnenolone to progesterone. It has been shown to terminate pregnancy in dogs after a 7 day treatment and appears to have this effect throughout gestation. Further, adverse side effects in dogs have not been reported.

• **Mifepristone**: This well-known drug acts as a progesterone **antagonist**. Small clinical trials have demonstrated that his drug is very effective in terminating canine pregnancy after 25-30 days of gestation without adverse effects.
CONTRACEPTION
Ovariohysterectomy (spaying)
Medroxyprogesterone

Covinan
PSEUDOPREGNANCY
Probable and proposed causes of clinical pseudopregnancy in female dogs

- Idiopathic occurrence of a more extensive increase in prolactin than occurs in normal diestrus
- Idiopathic increase in sensitivity to the endocrine changes that normally occur in late diestrus, including the normal progressive decline in progesterone and modest elevation in prolactin
- Pseudo-luteal phase induced by administration of exogenous progestins
• Progesterone withdrawal caused by:
  • ovarietomy during diestrous,
  • termination of long-term or short-term progestin therapy
  • idiopathic or prostaglandin-induced abrupt luteolysis
  • antiprogestin therapy
• Idiopathic hyperprolactinemia potentially associated with pituitary microadenomas
• Psychogenic or reflexive hyper-prolactinemia occurring in response to stimulation by surrogate neonates or other visual, physical or social stimulation
CLINICAL SIGNS (most common)

- Prepartum-like and maternal-like behaviors
- Nesting, digging, over-affection, over-protectiveness, over-defensiveness, aggression, licking, mothering of inanimate objects
- Mammary enlargement and distension
- Lactation and milk release
- Weight gain, Anorexia
Less common sign

- Emesis
- Abdominal enlargement
- Abdominal contractions
- Diarrhea
- Polyuria
- Polydipsia
- Polyphagia
Approved and extra-label drug use reported for the treatment of clinical pseudopregnancy in dogs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Name</th>
<th>Action</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Contraceptive Androgens</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mibolerone</td>
<td>Cheque Drops®</td>
<td>Not known</td>
<td>Contraceptive. Extra-label use</td>
</tr>
<tr>
<td><strong>Dopamine Receptor Agonists</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bromocriptine</td>
<td>Parlodel®</td>
<td>Reduces prolactin</td>
<td>Human drug. Extra-label use</td>
</tr>
<tr>
<td>Cabergoline</td>
<td>Galastop®</td>
<td>Reduces prolactin</td>
<td>Veterinary drug marketed in Europe but not North A.</td>
</tr>
<tr>
<td><strong>Serotonin Antagonists</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metergoline</td>
<td>Contralac®</td>
<td>Reduces prolactin</td>
<td>Veterinary drug marketed in Europe and some South American countries</td>
</tr>
</tbody>
</table>
Poisoning
• Paracetamol
• Warfarin
• Zinc phosphide
• Organophosphate
• organochlorine
• Strychnine
Paracetamol poisoning

source:  http://www.vetinfo.com/cptoxic.html
• Cats are more susceptible to paracetamol poisoning which occurs at concentrations of only 45mg/kg body weight - because they:

  have a deficiency of liver enzymes to combine the drug with glucuronide

  feline haemoglobin is susceptible to oxidative damage and readily forms methaemoglobin
• In dogs much higher doses (250mg/kg body weight) are needed to induce toxicity - which results in liver and kidney failure
Methaemoglobin formation that interferes with ability of red blood cells to carry oxygen

The formation of Heinz bodies that make red blood cells more fragile and susceptible to haemolysis resulting in destruction of the red blood cells

Hepatic necrosis which will ultimately cause liver failure
What happen?

- Paracetamol is rapidly absorbed from the small intestine and signs of poisoning are usually seen within 4-12 hours.
- Initially signs include vomiting, dullness, difficulty breathing, excessive drooling and dark discoloured gums. Accompanying swelling of the face and paws is often seen.
- Later after 2 days evidence of liver damage may become apparent such as jaundice.
- Coma, convulsions and pulmonary oedema have also been reported.
• Intravenous fluid administration
• Induce vomiting if presented early Gastric lavage
• minimise the effects of the methaemaglobin using various drugs as antidotes

**Acetylcysteine :**

Dogs - give intravenously at a dose rate of 140mg/kg as soon as possible, then give 70mg/kg after 30 minutes, and after 1 hour

Cats - give 140 mg/kg IV initially, then 70mg/kg by mouth qid for 5 days
Treatment

**Ascorbic acid** - in cats - give by IV 30mg/kg every 6 hours to reduce methaemoglobinemia as well as acetylcysteine

- Supportive care and oxygen therapy are the major management tools. Occasionally a blood transfusion may be necessary if large numbers of the cat’s red blood cells have been destroyed.
YELLOW FAT DISEASE

- Pansteatitis = generalized inflammation of the adipose tissue
- related to an excessive consumption of unsaturated fatty acids, with or without a deficiency in Vitamin E
- It is usually associated with the consumption of fat fish
Mesenteric adipose tissue; marmoset No. 1. Steatitis with rings of inflammatory cells around degenerating adipocytes (stars) and increased interstitial fibrous tissue. HE. Bar = 39 µm.
Poisoning

Induce vomiting: 3% of hydrogen peroxide 25-30 CC if not vomit in 10 min, repeated

Get Antidote

Fluid therapy, supportive therapy
• Warfarin
• organophosphorous
• organochlorine
• Zinc phosphide
• Strychnine
• ethylene glycol
• Chocolate
Zinc Phosphide

- Poisoning occurs by the liberation and rapid absorption of phosphine gas (PH$_3$) into the bloodstream when the zinc phosphide comes into contact with the dilute acids in the stomach.
- This results in damage to the blood vessels and erythrocyte membranes and eventual cardiovascular collapse and irritation of the alimentary tract.
- Toxicosis usually is evident in 15 minutes to 4 hours following ingestion of a toxic dose.
• There are no definitive clinical signs
• Clinical signs include anorexia, nausea, vomiting (often containing blood), abdominal pain, colic, diarrhea, prostration, lethargy, ataxia, chest tightness, dyspnea, salivation, excitement, convulsions, paralysis, rigor, and coma
• In fatal cases there is liver, kidney, heart, and brain damage
• Death is usually due to anoxia
• Microscopic examination reveals congestion of the liver and kidney. Renal tubular necrosis can be seen in many instances and cloudy swelling and fatty degeneration can be seen in the liver
Diagnosis

- Detection of phosphine gas
- Two fractions of the gas in zinc phosphide, one which degrades rapidly and one which degrades slowly in the alimentary tract
• no specific antidotes for zinc phosphide
• Sodium bicarbonate can be given orally to neutralize the stomach acidity
• calcium gluconate and sodium lactate can be given intravenously to combat systemic acidosis
• the stomach and intestinal tract can be evacuated, oxygen administered and cardiac and circulatory stimulants given
Organophosphate Toxicity
• Organophosphate compounds include some of the most toxic chemicals used in agriculture

• Disulfoton, phorate, dimethoate, cirodin, dichlorvos, dioxathion, ruelene, carbophenothion, supona, TEPP, EPN, HETP, parathion, malathion, ronnel, coumaphos, diazinon, trichlorfon, paraoxon, potasan, dimefox, mipafox, schradan, sevin, chlorpyrifos and dimeton
• Organophosphate toxicity is due to the ability of these compounds to inhibit acetylcholinesterase at cholinergic junctions of the nervous system.

• These junctions include postganglionic parasympathetic neuroeffector junctions, autonomic ganglia and the neuromuscular junctions and certain synapses in the CNS.

• Acetylcholine is the neurohumoral mediator at these junctions. Since acetylcholinesterase is the enzyme that degrades acetylcholine following stimulation of a nerve, its inhibition allows acetylcholine to accumulate and result in initial excessive stimulation followed by depression.
• Acute signs can result within 1-12 hours following inhalation or cutaneous absorption and more rapidly following ingestion

• The clinical signs of organophosphate poisoning occur as a result of excess acetylcholine at nerve endings, which mimics hyperactivity of the parasympathetic nervous system
• Signs relative to the alimentary tract include excess salivation, lacrimation, abdominal pain, vomiting, intestinal hypermotility, and diarrhea

• Muscarinic effects of acetylcholine cause bronchoconstriction and an increase in bronchial secretions

• Nicotinic effects of acetylcholine consist of involuntary irregular, violent muscle contractions and weakness of voluntary muscles

• Death occurs as a result of respiratory failure
No definite postmortem changes are seen and when present, are usually secondary to the symptoms and include pulmonary edema, asphyxia, gastroenteritis, and rarely kidney and liver degeneration.
Diagnosis

- As postmortem findings are usually not revealing, diagnosis is usually made by laboratory analysis.
- The most reliable diagnostic test is the determination of the acetylcholinesterase level in red blood cells, but it must be performed on fresh samples.
- Acetylcholinesterase levels can be determined on red blood cells, whole blood or plasma.
- Analysis of brain tissue for decreased acetylcholinesterase levels is also good if done within a few days following death.
Treatment

- atropine and 2-PAM (2-pyridine aldoxime methiodide) can alleviate some of the symptoms

- Decontamination of the skin, stomach and eyes of the animal may be necessary, along with symptomatic treatment and respiratory support.
Gross lesions

• venous congestion, capillary breakdown, pulmonary congestion, interlobular lung edema, liver and kidney congestion, and gastroenteritis

• When the stomach is opened, an odor of carbide (acetylene) may be apparent

• Yellow mottling liver in animals that live long enough for liver damage to occur
Warfarin toxicity
• Coumarins inhibit hepatic synthesis of the vitamin K–dependent coagulation factors II, VII, IX, and X and the anticoagulant proteins C and S

• Typically, vitamin K is a cofactor in the postribosomal synthesis of the clotting factors mentioned above

• The vitamin K–dependent step involves carboxylation of glutamic acid residues and requires regeneration of vitamin K to
Clinical signs

Massive GI bleeding and intracranial hemorrhage

More common findings of excessive anticoagulation are ecchymoses, subconjunctival hemorrhage, epistaxis, vaginal bleeding, bleeding gums, or hematuria
Treatment

- **GI decontaminants**: Activated charcoal -- Emergency treatment in poisoning caused by drugs and chemicals. Network of pores present in activated charcoal adsorbs 100-1000 mg of drug per gram of charcoal.
- **Antidote**: Vit K1
- **Bile sequestrants**: Decreases warfarin absorption by interference with enterohepatic recirculation -- Cholestyramine (Questran) -- Forms a nonabsorbable complex with bile acids in the intestine, which, in turn, inhibits enterohepatic reuptake of intestinal bile salts.
Organochlorine toxicity
• Toxaphene and related organochlorine compounds (eg, endrin, dieldrin, aldrin, endosulfan, chlordane, heptachlor, dichlorodiphenyltrichloroethane [DDT], lindane, chlordecone)
Clinical signs

convulsion
Treatment

- Activated charcoal
- Sedative: Diazepam or Nembutal
Anti-freeze (ethylene glycol)

- sweet, so given the opportunity, dogs will drink it.
- Anti-freeze causes kidney failure, very quickly
- Often, once the animal is showing signs of anti-freeze poisoning, it is too late to save them. Signs include staggering, depression, nausea, vomiting, drinking lots of water, urinating a lot, seizures, coma and death
Treatment

• aggressive intravenous fluid therapy and intravenous ethanol

• Just 2cc, less than half of a teaspoonful, is enough anti-freeze to kill a small dog
Chocolate toxicity
Chocolate toxicity

- Chocolate contains both theobromine and caffeine, which are stimulants to the nervous system.
- While a human system can metabolize theobromine efficiently, the half life in a dog or cat is 17.5 hours.
- As the animal struggles to excrete the substance, it affects CNS, the cardiovascular system and BP.
The toxic dose is generally 100-150 milligrams per kilogram **White chocolate:** 200 ounces/lbs. It takes 250 pounds of white chocolate to cause signs of poisoning in a 20-lbs dog, 125 pounds for a 10-lbs dog

**Milk chocolate:** 1 ounce/lbs. Approximately one lbs of milk chocolate is poisonous to a 20-lbs dog; one-half pound for a 10-lbs dog. The average chocolate bar contains 2 to 3 ounces of milk chocolate. It would take 2-3 candy bars to poison a 10 lbs dog. Semi-sweet chocolate has a similar toxic level
**Sweet cocoa:** 0.3 ounces/lbs. One-third of a pound of sweet cocoa is toxic to a 20-pound dog; 1/6 pound for a 10-pound dog

**Baking chocolate:** 0.1 ounce/lbs. Two one-ounce squares of bakers' chocolate is toxic to a 20 lbs dog; one ounce/10-lbs dog
Chocolate toxicity is dependent on several factors: the type of chocolate, the size of the dog, and the amount ingested. The most toxic form of chocolate is unsweetened baking chocolate. It is almost ten times as potent as milk chocolate, which is the least toxic form of chocolate.
Symptoms

- Signs will normally show up within 12 hours
- Vomiting, diarrhea, increased urination, hyperactivity, increased heart rate and respiratory rate, abnormal heart rhythms, restlessness, tremors, staggering, seizures, hyperthermia, coma and death
Treatment

• IV fluids may be introduced to prevent dehydration and help flush the system
• Emetics or activated charcoal
• Anti-seizure
• Cardiac medications may be administered, for pets showing signs of irregular heart rhythms or rates
Strychnine
• Strychnine is an alkaloid molecule found in the seeds of the plant *Strychnos Nux Vomica*, native to India, Sri Lanka and Australia, and a few related species.

• Symptoms usually begin within about **20 minutes** of ingestion of the poison.
• Strychnine binds to Glycine receptors (one of the main types of inhibitory receptor) resulting in huge over-transmission of signals, resulting in reflex arcs, then, producing sensory stimulus produces powerful muscular contraction
• initial violent convulsion, but frequently symptoms begin with restlessness, apprehension, heightened perception (hearing, vision, etc.), abrupt movements, exaggerated reflexes, muscular stiffness of face and legs, and rarely vomiting

• Movements may be intermittent at first, but then there is hyperextension, with the body arched convexly, resting on head and heels, the legs extended, arms flexed over chest or rigidly extended
• The muscular contractions caused by strychnine produce characteristic contortions of the body, arched backwards so that only the heels and the top of the head touch the ground, and of the face, a fixed grin known as the *risus sardonicus*

• Death occurs as a result of respiratory arrest, due to spasm and paralysis of the respiratory muscles
The poison is metabolised in the liver and has a half-life of about 10 hours in humans.
Treatment

Barbiturate sedatives and muscle relaxants  e.g. Diazepam, Pentobarbitol
FLUID THERAPY
<table>
<thead>
<tr>
<th>Estimated Percentage Dehydration</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5</td>
<td>History of fluid loss but no findings on physical examination</td>
</tr>
<tr>
<td>5</td>
<td>Dry oral mucous membranes but no panting or pathological tachycardia</td>
</tr>
<tr>
<td>7</td>
<td>Mild to moderate decreased skin turgor, dry oral mucous membranes, slight tachycardia, and normal pulse pressure</td>
</tr>
<tr>
<td>10</td>
<td>Moderate to marked degree of decreased skin turgor, dry oral mucous membranes, tachycardia, and decreased pulse pressure.</td>
</tr>
<tr>
<td>12</td>
<td>Marked loss of skin turgor, dry oral mucous membranes, and significant signs of shock</td>
</tr>
<tr>
<td>Percentage</td>
<td>Description</td>
</tr>
<tr>
<td>------------</td>
<td>-------------</td>
</tr>
<tr>
<td>4-5%</td>
<td>Semidry oral mucous membranes, normal skin turgor, and eyes maintaining normal moisture</td>
</tr>
<tr>
<td>6-7%</td>
<td>Dry oral mucous membranes, mild loss of skin turgor, and eyes still moist</td>
</tr>
<tr>
<td>8-10%</td>
<td>Dry mucous membranes, considerable loss of skin turgor, eyes retracted, and weak rapid pulses</td>
</tr>
<tr>
<td>12%</td>
<td>Very dry oral mucous membranes, complete loss of skin turgor, severe retraction of the eyes, dull eyes, possible alteration of consciousness, and thready weak pulses</td>
</tr>
</tbody>
</table>
Intravenous fluid administration is indicated in dogs and cats with 7% or greater dehydration.

There are numerous potential routes for intravenous fluid administration:

- Peripheral veins
- Jugular veins
- Intraosseous
Daily Water Requirements for the Cat

ml Water / Day

Body Weight (kg)

Recommended Formula

(30XKg) + 70

66 ml/kg
Client/Professional Relations

JIRAPORN SUKSAWAT
DVM, MS, Ph.D

KKU. VET. TEACHING HOSPITAL
## Differences between customers and clients

<table>
<thead>
<tr>
<th>IN BUSINESS</th>
<th>IN HEALTHCARE</th>
</tr>
</thead>
<tbody>
<tr>
<td>The customer can say &quot;no&quot; and go elsewhere for a quote.</td>
<td>The client feels at the mercy of the healthcare provider.</td>
</tr>
<tr>
<td>Most people are there voluntarily.</td>
<td>Wellness concerns force access efforts by the client.</td>
</tr>
<tr>
<td>Most people are in a good mood.</td>
<td>Most clients are anxious, scared, or feel exposed.</td>
</tr>
<tr>
<td>Many people feel they can bargain for a deal.</td>
<td>Most people are panicked about the required costs.</td>
</tr>
<tr>
<td>Customers expect to be pampered and served, often by staff members who are</td>
<td>Clients are often confused with high technology, but expect know-how</td>
</tr>
<tr>
<td>inexperienced but try to please.</td>
<td>and compassion from the professionals.</td>
</tr>
<tr>
<td>Employees must try to be courteous and responsive to the customers desires,</td>
<td>The client must be responsive to the professional's case assessment,</td>
</tr>
<tr>
<td>regardless of the demand.</td>
<td>while the staff must be safe, accurate, kind, skilled, alert, and much more.</td>
</tr>
</tbody>
</table>
• Ability to interact positive way
• Promote positive images
• Accept person, maintain attitude of warmth and goodwill whether or not his/her way of thinking is socially acceptable or to your personal liking
• Client have right to self determination
• Listen, try to understand
• Maintain a position of unconditional positive
• refrain from returning anger
Basic principles of effective understanding

• Observing
• Listening
• Empathizing
• Interpreting
• Hospitals require routines, uniformity, and standardization

• impersonal, complex, frustrating
• Honestly and openly share personal feeling
• I rarely feel frustrate that everything we do seem wrong, can we help you better?
• I want to listen to you but it is hard to do when you are yelling at me
Money disputes

• Before doing any procedure, fee should be discussed with owner
• should have written itemized estimate posted at reception/billing area
• Do not criticize, nonjustimental, supportive
COPING WITH CLIENT GRIEF

JIRAPORN SUKSAWAT, DVM. MS, Ph.D
Stages of grief

I. Numbness and denial
II. Anger
III. Bargaining
IV. Depression and grief
V. Acceptance or resolution
What to do to grief client

• Send sympathy card in 3 days and 3 week after pet loss
• Staff should reassure that hopelessness, helplessness, irregular sleeping and eating habits, mental confusion, hallucination are normal
• If grief happen more than 3 weeks seek medical help
To every thing there is a season, and a
time to every purpose under the heaven: a
time to be born and a time to die.

-Eccles. 3:1
Malpractice..before treatment

• No guarantee of result
• Risk is advised
• Owner written’s consent before surgery
Malpractice..before treatment

- Treatment should not begin without proper equipments
- Not present yourself as specialist
- Inquiries should be made if animals has been under the care of other vet
Malpractice... during treatment

- Detailed accurate record must be kept
- Avoid owner assist you particularly when restraining animal
Malpractice..after treatment

- Avoid mentioning other treatment if original treatment is unsuccessful
- Necropsy should be performed when the cause of death is in question with owner permission
- Avoid apologetic statements or excuses
Malpractice..after treatment

- Do not release original records or radiographs to the owners or their representatives
- Label all dispensed products
Heat Stroke = Sun + humidity

an excessive elevation in body temperature such that there is direct thermal injury to body tissues

Humidity interferes with animals' ability to rid themselves of excess body heat

heat gain > heat loss

- Overexercice
- Obesity, too much hair, stay in bad ventilated areas
- Too long exposure to sunlight
- Dehydration, Destruction of hypothalamus
They had left their dog in the car for only a short time. But they returned to heartbreak.
Heat stroke

- Factors that increase an animal's risk of developing heat stroke include:
  - water deprivation
  - excessive humidity
  - obesity
  - exercise
  - cardiovascular disease
  - lack of acclimatization
Heat stroke

- Brachycephalic breeds
- Upper airway disease
- Laryngeal paralysis
- Antihistamines
- Phenothiazines
- Increased age
Pathophysiology

- hypothalamus, a thermostat, guiding the body through mechanisms of heat production or heat dissipation
- thermosensors in the skin, muscles, and spinal cord send information to the anterior hypothalamus
- Excessive heat denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to cardiovascular collapse, multiorgan failure, and, ultimately, death
Signs of heatstroke

- Panting, Sweating, Salivating, Difficulty in breathing
- Vomiting, Bloody diarrhea, T= above 40 C or 104 F
- Increased HR RR
- Mucous membranes bright red
- Capillary refill time very fast (less than 1 sec)
- Dehydration
- Depression, lethargic (acting drunk)
- Shock, Seizure, Collapse, or coma
Clinical signs

• Increased RR, HR
• Sweating, hypersalivation, thirsty, hyperesthesia
• No sweating, abnormal gait
• When t=105-panting, arrhythmia, tremor and subconscious
Diagnosis

- Diff diag between heat stroke and septicemia
- If Septicemia-hemorrhage found
- History
Findings

• ARF may occur because of direct thermal injury of the kidney, myoglobinuria, hypotension, and/or shock

• Pulmonary edema is a common complication of heat stroke and may be due to a number of factors, including fluid overload from aggressive rehydration, fluid overload from renal failure, congestive heart failure
Treatment

- Good ventilation and cold pack
- Reduce temperature by fluid infusion, insert cold solution via rectum
- Sedative
- Broad spectrum ABO
- Corticosteroid for shock treatment
- Alkalinizing agents
- Diuretic drug
First Aid for Heat Stroke