Crotalus adamanteus, one of the more dangerous snakes in the United States and the most dangerous snake in Florida, is indigenous to the southeastern United States (North and South Carolinas, Georgia, Florida, Alabama, Mississippi, and Louisiana). Crotalids characteristically have two elongated canalicular upper maxillary teeth that fold back against the roof of the mouth. When striking a victim with its mouth wide open, the snake's maxilla and fangs are rotated forward about 90 degrees and thus become effective stabbing instruments. Other characteristic identifying features of the crotalids include vertically elliptical pupils, a deep pit (hence, the name pit viper) between the eye and the nostril, which functions as a heat receptor organ, and a somewhat triangular head. Crotalus adamanteus can grow as long as seven feet and can live longer than 22 years.

In general, most snake bites occur between the months of June and October and are rare between December and March; most incidents involving humans occur between 3 PM and 6 PM, with 80% occurring between 9 AM and 9 PM. More specifically, most incidents of rattlesnake bites involving humans occur in the late afternoon during the hot summer months (July and August). From what the author has seen at the University of Florida, much of the same pattern has applied to dogs and cats.

Pit viper bites cause one or two fang puncture wound(s). The snake can strike and envenomate its victim in less than 1 sec. Penetration and envenomation are rapidly followed by the onset of swelling, hemorrhage, and pain around the wound. A deeply penetrating fang puncture and subsequent envenomation can result in shock within minutes.

Not all poisonous snakebites result in envenomation. From one third to one half of all human victims showed little or no evidence of envenomation. Variables that influence the severity
of a snakebite include (1) the location, depth, and number of bites, (2) the amount of venom injected, (3) the species and the size of the snake involved, (4) the age and the size of the victim, (5) the victim's sensitivity to venom, (6) the microbes present in the snake's mouth, and (7) the type of first aid treatment and subsequent medical care. Bite wounds to the tongue or oral cavity are particularly dangerous because of subsequent soft tissue swelling and upper airway obstruction. Other locations of facial bites in the dog are not associated with airway obstruction in the author's experience. In addition, the increased vascularity of the head and neck areas facilitates rapid entry of the venom into the systemic circulation, and tourniquets therefore cannot be applied easily to this area. The after effects of head wounds are particularly relevant for dogs because 80% of the dogs treated by the author involved the face. This incidence differs markedly from statistics on humans, which indicate that the majority of snakebites have involved the distal extremities. Most of the envenomations in cats involve the front limbs and the trunk.

The amount of venom released depends on when the snake last ate and whether or not the snake is threatened (they can control the amount of venom that is released from the venom sac. If a snake has not eaten recently, a large volume of venom will be available for release at the time of the strike. In addition, a large snake can usually inject a greater amount of venom than a smaller snake. Human victims that are either very young or old are particularly susceptible to the toxic effects of envenomation. Furthermore, the toxic effects are inversely proportional to a victim's size.

Venom Toxicity

Familiarity with the components and effects of venom and an understanding of the pathophysiology of poisonous snake venoms are essential for effective treatment. Most poisonous snake venoms have direct or indirect toxic effects on the victim's blood cells, heart, blood vessels, and respiratory and nervous systems. The brain is uniquely resistant to any direct toxic effects of rattlesnake venom. The primary effects of venom include local tissue damage,
edema, hypoalbuminemia, coagulopathy, thrombocytopenia, hypovolemia, and in some pit vipers, neurotoxicity. Rattlesnake venom is a complex mixture of 5-15 enzymes, metal ions, biogenic amines, lipids, free amino acids, large and small proteins, and polypeptides. Lethal proteins and peptides consist of 20-80 amino acids that damage endothelial cells and plasma membranes. Polypeptides are low- molecular weight proteins that lack enzymatic activity and are 5-20 times more lethal in animal models than crude venom. The weight of these polypeptides range from 6000-30,000 daltons, and the concentration of these lethal proteins are higher in cobra (elapidae) than in rattlesnake (crotalinae) venom. Digestive enzymes include the following: 1) phospholipase A2 that hydrolyzes the ester bond of lecithin and damages fatty acid molecules in cell membranes, 2) hyaluronidase that decreases the viscosity of connective tissue, 3) amino acid esterase that promotes fibrin formation, and 4) proteolytic enzymes and 5’ nucleotidase that damage proteins in muscle fibers.

Phospholipase A, B, C, and D hydrolyze lipids with subsequent disruption of神经transmission at both the presynaptic and postsynaptic areas. Phospholipase A penetrates nervous tissue, where it destroys or alters certain phospholipids; it also causes hemolysis and contributes to the cardiotoxic effects of venom. Hyaluronidase allows for the rapid spread of venom through tissue by hydrolyzing connective tissue hyaluronic acid, thereby contributing to the swelling and edema at the site of the bite wound. Amino acid (L-arginine) esterases are common in crotalid venom and cause procoagulant activity and bradykinin release. Proteases produce damage by dissolving. *Crotalus adamanteus* venom has low proteolytic activity, which enhances its procoagulant effect. Hemolysis results from the destruction of lecithin in the cell membranes. L-aminoacid oxidase produces local tissue destruction by catalyzing the oxidation of amino acids. Other enzymes include transaminases, ribonuclease, L-arginine ester hydrolase, deoxyribonuclease, phosphomonoesterase, diphosphodiesterase, DNA-ase, ATP-ase, alkaline phosphatase, acid phosphatase, and endonuclease.
As mentioned earlier poisonous snake venom also contains nonenzymatic polypeptides. They are known as hemorrhagins, cardiotoxin, and neurotoxin, all of which exert profound clinical effects. Hemorrhagins, which are commonly found in crotalid venom, are vasculotoxic and cause rapid hemorrhage and edema at the wound site as well as extensive systemic hemorrhage, which contributes to hypovolemic shock. Damage to the blood vessels results from disruption of the endothelial cell junctions and basement membranes. The marked hemorrhagic edema in the dogs that the author has treated clearly illustrates the combined effects of the hemorrhagins and the enzymatic components of poisonous snake venom.

Cardiac arrhythmias can occur in as many as 50% of dogs that are severely envenomated. Cardiac dysfunction results from the combined effects of cardiotoxin and impaired myocardial perfusion. Most of the arrhythmias are ventricular tachyarrhythmias.

Mental depression is the only one sign in the dog that might reflect the effects of the neurotoxin; other dogs have shown 4 legged weakness which is reversible. In humans, the neurotoxin found in Crotalus adamanteus venom causes paresthesia, tetanic contractions, and fasciculations.

Adverse hematologic side effects commonly occur and include hemolysis, anemia, defibrination without overt hemorrhagic diathesis, and fibrinogenolysis associated with hemorrhagic diathesis. Because of the high incidence of hemolysis and the possible need for transfusion, a snakebite patient should be crossmatched with blood donors as soon as possible after admission. Anemia can result singly or from a combination of hemolysis and extravasation of blood into the soft tissues, which may be massive enough to necessitate whole blood transfusion.

Studies have shown that Crotalus adamanteus venom contains an amino acid esterase that exerts thrombin-like activity. This enzyme acts directly on fibrinogen in vivo (and in vitro) apparently without affecting any other protein or the platelets involved in blood coagulation. The procoagulant effect of this thrombin-like enzyme causes inappropriate fibrinogen cleavage, with
subsequent formation of soft friable microclots, fibrinogen degradation products, and fibrinogen depletion. Fibrinogen degradation products inhibit normal fibrin polymerization, and their identification in clinical patients indicates activation of the fibrinolytic system. Fibrinogenolysis caused by a thrombin-like enzyme aggravates the bleeding produced by the hemorrhagins. In humans, the true syndrome of disseminated intravascular coagulation (DIC) rarely is documented after *Crotalus adamanteus* envenomation because the snake venom esterase seldom causes platelet aggregation, does not activate and consume factors V and VIII, and shows minimal if any response to heparin treatment.

In humans, fibrinogenolysis with grossly anticoagulant blood may persist for days without any signs of bleeding. Dogs can also show abnormal coagulation times in the absence of bleeding.

**First Aid**

Common first aid measures for poisonous snakebites of humans include (1) immobilizing the patient and the affected limb to slow the spread of venom, (2) applying a light-constricting tourniquet, and (3) performing local incision and suction.

A tourniquet should be applied at least 10 cm proximal to the fang marks and cause only light constriction to obstruct the superficial lymphatic and venous flows; the tourniquet should be released every 30 minutes for 60 to 90 seconds. A tourniquet is most effective when applied within 30 minutes of envenomation. Some authors, however, object to the use of a tourniquet because it prevents dilution of the venom and decreases tissue perfusion, thereby promoting ischemia and tissue necrosis. Furthermore, there is no evidence with North American pit viper envenomations supporting a reduction in morbidity or mortality when a tourniquet is used. There are exceptions in Australasia where tourniquets are useful to restrict powerful elapid neurotoxins from entering the human victim’s circulation. Tourniquets are seldom (if ever) useful in dogs because the majority are struck in the head. In addition, the elapsed time between the
strike and the owner's awareness of the incident might exceed 30 minutes. Incision and suction are effective only when done immediately.

**Treatment**

- Establish whether the dog was bitten by a nonpoisonous or a poisonous snake this is often answered by the toxidrome in unwitnessed bites.
- Decide whether envenomation has occurred.
- Institute measures to prevent or limit severe tissue damage.
- Provide life support measures if severe envenomation has occurred.

The most potent venom of the Florida pit vipers is the Eastern Diamondback Rattlesnake followed by the Timber Rattlesnake, Water Moccasin and finally the Pygmy Rattlesnake. Copperhead snakes can be found in the very NW part of the state. Although rare, the Pygmy bite can be fatal. Dogs that are moderately affected by envenomation should receive a thorough medical evaluation and intensive medical treatment, including a hemogram, urinalysis, clotting evaluation, and an electrocardiogram. These tests should be repeated regularly during hospitalization to assess the adequacy of the patient's response to treatment as well as to detect the onset of delayed complications (especially severe anemia and cardiac arrhythmia). Urine output and blood urea nitrogen or serum creatinine levels should also be measured to detect any renal failure resulting from ischemia or hemolysis.

**Patient Severity Assessment: Author’s Practical Guidelines**

1. Mild. Patient acts normal and vitals are normal. Bite site localized and minimally progressive over the first 2 hours.
2. Moderate. Able to stand and walk. Ambulation slow, mentation “quiet”, bite site swelling slowly progressive over first 2 hours. Cardiac rhythm normal and coagulation normal. TPR – stable, but HR and RR mildly increased. Pulses weak to strong and CRT normal to prolonged. Note: can suddenly become severe within short period of time, so observe intensively.

INTRAVENOUS FLUIDS AND BLOOD PRODUCTS

Essential treatments are intravenous fluids and antivenom. Isotonic intravenous crystalloid solutions are essential for providing patient stabilization. This can be accomplished by administering lactated or acetated Ringer’s. These fluids are preferred over 0.9% saline intravenously. Many snakebite victims will be hypotensive and therefore require substantial resuscitative amounts of intravenous fluids. This can be accomplished in dogs and cats by administering 20-25 ml/kg IV and repeating this amount every 15 minutes for the first hour so long as there are no signs of intravenous overload or an accelerated rate of bleeding because of the increased blood pressure. Cats should receive 5-7 ml/kg IV every 15 minutes as described for the dog. Blood pressure monitoring will be the best guide for further treatment with additional crystalloid solution or adding colloid. Any colloids (dextran and hetastarch) that can potentially impair coagulation should not be used because thrombocytopenia is a common consequence to many but not all pit viper envenomations.

Because hypoproteinemia and anemia are common results of soft tissue extravasation, fresh whole blood should be given if the packed cell volume and total protein drop below 20% and 5 g/dl, respectively. Whole blood transfusion should be considered if antivenom does not correct the coagulopathy or if there is imminent risk of serious bleeding. Immediate use in humans is indicated in the setting of active hemorrhaging or if the platelet count drops below 20,000. All blood products can pose a potential risk because of transfusion reactions and their ability to potentially further the coagulopathy caused by the venom. According to some experts, the addition of extra substrate in fresh frozen plasma or cryoprecipitates may only add “fuel to a venom-stoked fire”, especially with procoagulants, unless all venom has been removed. This, in turn, will accelerate the hyperfibrinolytic state that can occur. This increases the risk of further bleeding which can even worsen if fibrin degradation products are released which can further add to the fibrinolytic or fibrinogenolytic crisis. Heparin should not be used for envenomation-induced bleeding because it will not “switch off” the venom-induced coagulopathy and might even cause its own degree of pathologic changes to clotting.

ANALGESICS
Analgesic drugs should be used, if necessary. They are rarely used beyond the first 48 hours. Lidocaine by CRI can be given at a loading dose of 1 mg/kg IV and then a maintenance dose of 50 ug/kg/min. Buprenorphine at 0.02 mg/kg IV push can also be given every 6 hours. Fentanyl is a commonly used analgesic in human snake bite victims. It is best not to pharmacologically alter the victim’s state of consciousness during the acute stages where close monitoring of awareness is essential to assessing the patient’s mental and physical status.

**ANTIVENOM**

The use of polyvalent crotalid antivenom is the mainstay of therapy for moderate to severe envenomation; however, the cost of large quantities of antivenom limits its affordability as a treatment for some dogs. In humans, early intravenous antivenom treatment of victims with moderate to marked symptoms has been stressed repeatedly. Under optimal conditions, antivenom should be given within four hours after the snake bite. Although the full beneficial effect diminishes when antivenom is given after this period, it is still recommended up to 24 hours after envenomation. If the bite results in the injection of a large quantity of venom deep into well-vascularized soft tissues or directly into a vessel, death may occur despite vigorous antivenom treatment.

Anaphylaxis and anaphylactoid reactions are possible complications of antivenom treatment because of its horse serum origin. Any early signs of anaphylaxis (vomiting, salivation, urticarial, defecation, restlessness) should be immediately treated with epinephrine (use 1:1000 concentration and administer 0.01 mg/kg IM; can repeat Q 15-20 min). The continued administration of the antivenom is allowable along with simultaneous epinephrine injections at the above dosage so long as the patient becomes hemodynamically stable. In a study by de Silva HA, Pathmeswaran A, et al published in Science Daily May 11, 2011, they reported on pretreating 1000 human snakebite victims in Sri Lanka with epinephrine and the results showed reduced severe antivenom reactions by 43% at one hour and by 38% over 48
hours. Those people who were pretreated with hydrocortisone and promethazine alone showed no reduction in adverse reactions. Note that that the purity of the antivenom will influence its antigenicity and predispose the patient to anaphylaxis.

Diphenhydramine (an H₁ blocker) should be given to treat hypersensitivity at a dose of 1.1 mg/kg IM. It is also recommended that H₂ blockers be administered as well because of their ability to decrease the amount of mucous that is secreted into the bronchial lumen and because of their beneficial effect on the vascular smooth muscle. Famotidine can be given at a dose of 0.5 mg/kg IM. Glucocorticoids are unable to act fast enough to counteract the immediate effects of anaphylaxis although they are helpful for the treatment of delayed signs and serum sickness which can occur a few days later. Anaphylaxis is also accompanied by the loss of significant fluid from the vascular space which worsens the hypotension in this disorder. Therefore, a rapid infusion of isotonic crystalloid solution such as lactated Ringer's is indicated for patient stabilization.

Fort Dodge (Boehringer-Ingelheim) Antivenin Crotalidae Polyvalent (ACP). Composed of IgG whole derived from horses and is prepared in a lyophilized form. It has the most extra antigens which makes it more capable of causing immune allergic reactions (>30% incidence). It can distribute through the interstitial space and can remain in circulation for 82 hours thus having a very long half life. Most of the dogs treated with Fort Dodge’s Crotalid Polyvalent Antivenin at the University of Florida have received an average total dose of 42.7 ml; one leading authority recommends administering larger doses similar to those used on humans (80 to 100 ml). In general, the dose will range from 1-2 vials for mild signs (only localized nonprogressive swelling), 2-4 vials for moderate signs (progressive swelling and coagulopathy), and 5-10 vials for severe signs (recumbency, hypotension, neuropathy, coagulopathy). In rare and severe situations, as many as 20-25 vials of various combinations of different pit viper antivenoms have been administered over a 72 hour period. The more vials used, the greater likelihood of Type I and Type II hypersensitivity reactions.
Crofab (made by BTG) is a Fab1 antivenom that is derived from sheep and is more pure than the Fort Dodge product. It is prepared as a lyophilized product. It is the most pure antibody product and it has the widest distribution throughout the body. There is no FcAb (complement fixing) thus sparing the patient from the main cause of allergic reactions (incidence of allergic reactions in humans is 0.8%). Its half life is 18 hours therefore requiring repeat injections in humans. It is supposed to be 5x the potency as the Fort Dodge product thereby allowing for smaller doses. This dose (1-2 vials) is best given initially and again 6 hrs later with additional amounts administered as needed.

Antivipmyn is a Fab2 antivenom produced by Bioclon in Mexico that is very effective against the North American pit vipers. It is prepared as a lyophilized product. It requires a USDA importers’ license and permission from the State veterinarian prior to purchase. Antivipmyn is derived from the horse but it is more pure antibody than ACP. It has a moderate elimination half life of 36 hours and has a good volume of distribution compared to ACP. It has no Fc ab thereby making it less antigenic and having an allergy incidence of 10%. The dose for Antivipmyn calls for 2-4 vials IV with repeat dosage given 4 hrs later. Dogs, unlike humans, are not particularly susceptible to immediate allergic reactions from antivenom treatment, but precautions are always recommended. All drug package inserts provide instructions for hypersensitivity testing, desensitization procedures, and the treatment of allergic reactions. The occurrence of type 1 hypersensitivity reactions is unpredictable despite the results of the testing procedure. The author highly recommends that epinephrine be drawn into a syringe at a dose of 0.01 mg/kg so that it is ready available whenever any antivenom is administered.

VenomVet – An Fab2 antivenim made in Argentina, but sold commercially and readily availble in the USA. It is very similar to Antivipmyn and used similarly.

PoliVet-ICP is prepared in Costa Rica and is prepared as a liquid product thus having a shelf life of only 1 yr as compared with a 5 year shelf life of the other lyophilized products. It is an
IgG whole product derived from horses. It is effective against most North American pit vipers but not effective against the Mohave rattlesnake.

**Rattler Antivenin, MG Biologics, Ames, Iowa**

Crotalidae polyvalent, equine origin. Contains antibodies against Prairie, Mohave, Western diamondback, Eastern diamondback rattlesnakes. No mention of effectiveness against copperhead or water moccasin snakes. This is a full IgG product that contains the Fc component (similar to Ft. Dodge) which might be associated with increased allergic reaction so be prepared accordingly. Manufacturer recommends dose not to exceed 2 packages in most instances.

**GLUCOCORTICOIDS AND NSAIDS**

In humans, the benefits of glucocorticoid treatment of poisonous snakebite victims are controversial. Several authorities find no proof of efficacy and find its use contraindicated. In fact, many experts state that glucocorticoid might even antagonize the effects of antivenin although this is based on old literature which might have been flawed. New studies to help answer this question would be most helpful. Most of the dogs in the author’s experience have done well without steroid treatment.

Nonsteroidal anti-inflammatory drugs are not recommended for pit viper envenomations. They might compound bleeding problems when coupled with the possibility of venom-induced thrombocytopenia.

**ANTIBIOTICS**

Because fang wounds can be accompanied by inoculation of bacteria into the victim's tissues, broad-spectrum antibiotic treatment is sometimes recommended. Most snake bites in dogs and humans are not complicated by bacterial infection therefore not requiring antibiotics. Exceptions do occur when necrosis, abscessation and putrefaction occur which will become
evident by the 3rd day after the bite. In these situations, antibiotics with broad gram negative
cover and debridement are recommended. In humans, the administration of tetanus antitoxin,
tetanus toxoid, or both is routine. Communications over periods of weeks to months between
University of Florida staff and the owners of surviving dogs that did not receive tetanus antitoxin
revealed that these dogs had done well after being discharged from the hospital, with none
showing any signs of tetanus.

**SPECTRUM OF COVERAGE BY VARIOUS ANTIVENOM PRODUCTS**

**CROFAB (Ovine)**

<table>
<thead>
<tr>
<th>Snake Species</th>
<th>Antivenom Coverage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crotalus atrox</td>
<td>Western diamondback rattlesnake</td>
</tr>
<tr>
<td>Crotalus adamanteus</td>
<td>Eastern diamondback rattlesnake</td>
</tr>
<tr>
<td>Crotalus scutulatus</td>
<td>Mohave rattlesnake</td>
</tr>
<tr>
<td>Agkistrodon piscivorus</td>
<td>Cottonmouth (water moccasin)</td>
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*Also has cross reactivity with:*

<table>
<thead>
<tr>
<th>Snake Species</th>
<th>Antivenom Coverage</th>
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</thead>
<tbody>
<tr>
<td>Crotalus horridus</td>
<td>Timber rattlesnake</td>
</tr>
<tr>
<td>Crotalus viridis helleri</td>
<td>Southern Pacific rattlesnake</td>
</tr>
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<td>Crotalus molossus molossus</td>
<td>Blacktail rattlesnake</td>
</tr>
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<td>Crotalus horridus atricaudatus</td>
<td>Canebrake rattlesnake</td>
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<tr>
<td>Agkistrodon contortrix contortrix</td>
<td>Copperhead</td>
</tr>
<tr>
<td>Sistrurus miliarius barbouri</td>
<td>Dusky pygmy rattlesnake</td>
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**FORT DODGE (EQUINE)**

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<tr>
<th>Snake Species</th>
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<tr>
<td>Crotalus adamanteus</td>
<td>Eastern diamondback rattlesnake</td>
</tr>
<tr>
<td>Species</td>
<td>Common Name</td>
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<td>---------------------------------------</td>
</tr>
<tr>
<td>Crotalus atrox</td>
<td>Western diamondback rattlesnake</td>
</tr>
<tr>
<td>Crotalus durissus terrificus</td>
<td>Tropical rattlesnake, Cascable</td>
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<tr>
<td>Bothrops atrox</td>
<td>Fer-de-lance</td>
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**Additional cover for Fort Dodge**

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<th>Species</th>
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<tr>
<td>Crotalus and Sisrurus</td>
<td>Rattlesnakes</td>
</tr>
<tr>
<td>Agkistrodon</td>
<td>Copperheads and cottonmouths</td>
</tr>
<tr>
<td>Agkistrodon halys</td>
<td>of Korea and Japan</td>
</tr>
<tr>
<td>Bothrops</td>
<td>and sub-species</td>
</tr>
<tr>
<td>Crotalus durissus</td>
<td>and subspecies</td>
</tr>
<tr>
<td>Agkistrodon bilineatus</td>
<td>Cantril</td>
</tr>
<tr>
<td>Lachesis mutus</td>
<td>Bushmaster of South and Central America.</td>
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**ANTIVIPMYN (BIOCLON- MEXICO)**

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<tr>
<td>Crotalus basiliscus</td>
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<td>Crotalus durissus durissus</td>
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<td>Crotalus durissus terrificus</td>
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<td>Crotalus simus</td>
<td>Shunu (Middle American Rattlesnake)</td>
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<tr>
<td>Crotalus polystictus</td>
<td>Hocico de Puerco (Twin spotted)</td>
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**Also covers:**

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<tr>
<td>Akistrodon bilneatus</td>
<td>Common Cantril</td>
</tr>
<tr>
<td>Akistrodon b. taylori</td>
<td>Taylor’s Cantril</td>
</tr>
<tr>
<td>Crotalus scutulatus</td>
<td>Mohave rattlesnake</td>
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May also have x-reactivity with:

**Agkistrodon**

<table>
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<th>Species</th>
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<tbody>
<tr>
<td>A. contortrix contortrix</td>
<td>Southern copperhead</td>
</tr>
<tr>
<td>A. c. laticinctus</td>
<td>Broad-banded copperhead</td>
</tr>
<tr>
<td>A. c. mokeson</td>
<td>Northern copperhead</td>
</tr>
<tr>
<td>A. c. phaeogaster</td>
<td>Osage copperhead</td>
</tr>
<tr>
<td>A. c. pictigaster</td>
<td>Trans-Pecos copperhead</td>
</tr>
<tr>
<td>A. piscivoros piscivoros</td>
<td>Eastern cottonmouth</td>
</tr>
<tr>
<td>A. p. conanti</td>
<td>Florida cottonmouth</td>
</tr>
<tr>
<td>A. p. leucostoma</td>
<td>Western cottonmouth</td>
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**Sistrurus**

<table>
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</tr>
</thead>
<tbody>
<tr>
<td>S. miliarias ravus</td>
<td>Cascabel de neve places (Mex.)</td>
</tr>
</tbody>
</table>

**Also cross reactivity**

<table>
<thead>
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<th>Species</th>
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</tr>
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<tbody>
<tr>
<td>S. cantenatus</td>
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<tr>
<td>S. edwarsi</td>
<td>Desert Massasauga</td>
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<td>S. c. turgeminus</td>
<td>Western Massasauga</td>
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<td>Carolina pigmy</td>
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<tr>
<td>S. m. barbouri</td>
<td>Southeastern (dusky) pygmy</td>
</tr>
<tr>
<td>S. m. strekeri</td>
<td>Western pygmy</td>
</tr>
</tbody>
</table>

**POLIVET-ICP – COSTA RICA**

Antivenom derived from:

Bushmaster
Fer de Lance
Neotropical rattlesnake

Rattler Antivenin- MG Biologics, Ames, Iowa
Western and Eastern diamondback
Mohave, Prairie
INTRODUCTION

Fluid therapy in clinical medicine is used to fulfill the following objectives: (1) to replace dehydration and plasma volume deficits, (2) to maintain normal hydration, (3) to replace essential electrolytes and nutrients, and (4) to serve as a vehicle for the infusions of certain intravenous medications. Except for the urgency of treatment, the same objectives apply in the critically ill animal. The methods for providing fluids often influence the eventual outcome of the case. The clinician and support staff should therefore familiarize themselves with the pathophysiology of the diseases they are treating and how these conditions relate to the various types of fluids that are available for general use.

BODY WATER DISTRIBUTION

Total body water (TBW in liter volume) accounts for approximately 60% of the body weight in kilograms (where 1 L H\text{2}O weighs 1.0 kg). Approximately two thirds of TBW is intracellular fluid (ICF) and one third is extracellular fluid (ECF). Three quarters of the ECF is interstitial fluid, and the remaining one quarter is intravascular fluid. The interstitial fluid space is three times the intravascular volume, and the intracellular space is 2.5-3 times the interstitial volume. The intracellular volume is therefore 7-9 times the intravascular volume. Fluid administered intravenously is distributed between the intravascular and extravascular spaces in fractions determined by the compartments’ protein and sodium contents.

Under "normal" circumstances isotonic crystalloid solutions such as lactated Ringer's solution or 0.9% sodium chloride will distribute between the intravascular and interstitial spaces at a 1:3 ratio by 30 minutes after intravenous infusion. So, if two liters of LRS are given IV, 500 ml will remain in the intravascular space while 1500 ml will end up in the interstitial space after thirty minutes. Because it is iso-osmolar, no osmotic gradient is created causing no net movement of water from the intracellular space.

FLUID VOLUME REPLACEMENT

The patient requires fluids for (1) rehydration, (2) maintenance, (3) replacement of insensible loss volumes, and (4) replacement of ongoing loss volumes. Clinically, the amount of fluid needed to correct dehydration deficits can be assessed from the degree of skin turgor, capillary refill time, and pulse rate and quality. The degree of dehydration ranges from 5% to 12% (Table 3) of the body weight, remembering that 1 liter of water weighs 1 kilogram or 2 pounds. Skin turgor assessment can sometimes be misleading in the obese animal, because adipose tissue replaces subcutaneous interstitial water thus allowing the skin to maintain its elasticity despite negative water balance. Also, old, cachectic patients that have lost skin resiliency may give a false impression of marked dehydration; this can make them victims of overestimation of fluid deficits. To clarify the assessment in such questionable situations, the patient’s packed red cell volume and plasma total solids levels can be determined, taking any anemia and hypoproteinemia into consideration.

The volume of fluid needed to correct dehydration is calculated from either of the following formulas:

1. Volume (ml) of fluid needed = % dehydration x body weight (lb) x 500
2. Volume (ml) of fluid needed = % dehydration x body weight (kg) x 1000
The maintenance volume is that amount normally required in a 24-hour period by a well hydrated patient. Taking insensible fluid loss into consideration, which amounts to 13-20 ml/kg/day, the 24-hour maintenance volume for a dog or cat whose urine output is normal is approximately 50 to 60 ml/kg (25 to 30 ml/lb) per day. The total 24-hour fluid requirement for the dehydrated animal is the sum of maintenance volume and volume required to correct dehydration plus any estimated ongoing losses. When on-going losses can only be approximated, some clinicians will double the maintenance volume and then re-assess the patient’s fluid balance status.

The initial rate and route of fluid delivery depend on the patient's status. Mildly to moderately dehydrated small dogs and cats (<10 kg) that require short-term (1-2 days) fluid treatment can be adequately managed with subcutaneous fluids. Any severely (>10%) dehydrated patient must initially receive fluids intravenously (IV). Because of any co-existing hypotension, medications injected subcutaneously may not be adequately absorbed into the systemic circulation. For the mildly to moderately hypovolemic patient, one fourth to one half of the estimated dehydration deficit should be replaced with isotonic crystalloid solutions IV over the first two to four hours with the remaining dehydration deficit and maintenance isotonic volumes administered over the subsequent 20 to 22-hour period. The amount of subsequent fluid infusion will depend on the patient's response to treatment. If the patient is markedly hypovolemic ("thready" pulses, prolonged capillary refill time, recumbent and mentally depressed, blood pressure <100mm Hg), the amount of administered intravenous fluids over the first hour should equal one whole blood volume which is approximately 70-90 ml/kg body weight for the dog and approximately 35-45 ml/kg body weight for the cat. This amount of fluid can be divided into boluses with dog’s receiving 15 ml/kg every 15minutes and cats receiving 7.0 ml/kg Q15 min with repeat doses given depending on re-assessment of vital signs. Vital signs in such patients must be monitored every 15 minutes and the fluids adjusted accordingly when signs of hypervolemia or interstitial edema occur. Intravenous colloid solutions might also be necessary.

Exceptions to the recommended maintenance doses of fluids occur under the following circumstances:

1. Oliguria and anuria. After dehydration deficits are replaced, the patient's maintenance needs depend on urinary output, which should be quantitated. Providing full normal maintenance fluid volumes to oliguric and anuric patients can lead to fatal pulmonary edema or pleural effusion because of iatrogenic intravascular fluid overload. Specific treatment for low output renal failure is provided in the next section.
2. Polyuria. Polyuric animals require fluid volumes in excess of normal maintenance needs. Failure to provide these volumes can result in a sustained negative water balance if the patient is unable to drink. The maintenance needs for polyuria consist of exact urinary losses plus insensible and ongoing losses. Assurance of adequate treatment is made by weighing the patient each day as well as by assessing the physical and laboratory parameters for hydration. An acute loss of 1 kg of body weight suggests a 1 L fluid deficit while a 1.0 kg weight gain reflects a 1.0 liter increase in total body water.
3. Rapid internal shifts of fluid (third spacing) can occur in pancreatitis, extensive burns, enteritis, and gastrointestinal obstructions. In these conditions, the fluid needs of the patient will exceed the usual maintenance volumes by as much as three times.
The assessment of the animal's fluid requirements must always be made within the pathophysiologic context of the underlying disorder and its predisposition to pulmonary edema as described in the following section.

The Microvascular Fluid Exchange in the Lung – Normal, Cardiogenic Pulmonary Edema, and Non-Cardiogenic Pulmonary Edema

The accurate diagnosis of acute pulmonary edema requires an understanding of the normal physiology involved with microvascular fluid exchange in the lung. In the normal lung, fluid and protein leakage is thought to occur primarily through small gaps between capillary endothelial cells. Fluid and solutes that are filtered from the circulation into the alveolar interstitial space normally do not enter the alveoli because the alveolar epithelium is composed of very tight junctions. Once the filtered fluid enters the alveolar interstitial space, it moves proximately into the peribronchovascular space. Under normal conditions, the lymphatics remove most of this filtered fluid from the interstitium and return it to the systemic circulation. Movement of larger plasma proteins is restricted. The hydrostatic force for fluid filtration across the lung microcirculation is approximately equal to the hydrostatic pressure in the pulmonary capillaries which is partially offset by a protein osmotic pressure gradient.

The inter-relationship between capillary hydrostatic and oncotic pressures and interstitial pressures as to their roles in net fluid fluxes across the alveolar membrane are described in the Starling equation.

**The Starling Equation**

\[ JV = Kf \left( P_c - P_is - \sigma (\pi_c - \pi_is) \right) \]

Where:
- **JV** = net fluid flux across the alveolar capillary membrane
- **Kf** = the filtration coefficient, or measure of the ease (conductance) of fluid (solvent or water) movement across the alveolar capillary membrane
- **P_c** = alveolar capillary hydrostatic pressure
- **P_is** = interstitial pressure
- **σ** = reflection coefficient with a value of 0 to 1 (normally 0.7 to 0.8) indicates the alveolar capillary membrane resistance to protein (solute) movement across the alveolar capillary membrane. A value of 1 indicates total resistance and 0 indicates zero resistance to transmembrane protein flux.
- **π_c** = capillary (blood) oncotic pressure
- **π_is** = interstitial oncotic pressure

The main factors contributing to the escape of fluid from the vascular space include vascular tone, cardiac tone, vascular endothelial integrity, and plasma oncotic pressure.

**Cardiogenic Pulmonary Edema**

A rapid increase in hydrostatic pressure in the pulmonary capillary leading to increased transvascular fluid filtration is the hallmark of acute cardiogenic or volume-overload edema. Increased hydrostatic pressure in the pulmonary
capillaries is usually due to elevated pulmonary venous pressure from increased left ventricular end-diastolic pressure and left atrial pressure. Mild elevations of left atrial pressure (18-25 mmHg) cause edema in the perimicrovascular and peribronchovascular interstitial spaces. As left atrial pressure rises further (> 25 mm Hg), edema fluid breaks through the lung epithelium, flooding the alveoli with protein-poor fluid. When lung interstitial pressure exceeds pleural pressure, fluid moves across the visceral pleura, creating pleural effusion.

Non-Cardiogenic Pulmonary Edema

By contrast, non-cardiogenic pulmonary edema is caused purely by an increase in the vascular permeability of the lung, resulting in an increased flux of fluid and protein into the lung interstitium and air spaces. Non-cardiogenic pulmonary edema has an increased protein content because the vascular membrane is more permeable to the outward movement of plasma proteins. The net quantity of accumulated pulmonary edema is determined by the balance between the rate at which the fluid is filtered into the lung, the integrity of the alveolar epithelium, and the rate at which fluid is removed from the air spaces and lung interstitium. At necropsy, the lungs appear heavy and the fluid is more of a serosanguineous type because of the plasma and blood cells present in the fluid.

Acute Respiratory Distress Syndrome (ARDS) /Acute Lung injury

The main defect associated with this condition is increased vascular permeability. Since the hallmark of the physiologic defects is this increase in vascular permeability to fluid and protein and the mediators of inflammation (cytokines, chemokines, etc), pulmonary microvascular hydrostatic pressure emerges as the major determinant for fluid movement across the microvascular membrane. The threshold pressure for edema is reduced when permeability is altered. It is therefore important in such patients to carefully monitor parenteral fluid administration as to avoid creating excessive amounts of hydrostatic pressure. The choice between crystalloid or colloid fluids remains controversial. The advocates of colloids stress the importance of maintaining intravascular oncotic pressure which will help deter fluid leak. The advocates of crystalloid stress that there are several safety factors that prevent fluid leakage where the colloid oncotic pressure is moderately decreased.

Common Causes of Cardiogenic and Non-Cardiogenic Pulmonary Edema

<table>
<thead>
<tr>
<th>Cardiogenic</th>
<th>Non-cardiogenic</th>
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<tr>
<td>- Myocardial ischemia</td>
<td>- Pneumonia</td>
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<td>- Congestive heart failure</td>
<td>- Sepsis</td>
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<tr>
<td>- Volume overload from excessive IV fluid</td>
<td>- Aspiration of gastric contents</td>
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<td>administration in patients with hypoproteinemia</td>
<td>- Acute pancreatitis</td>
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<td>and anemia</td>
<td>- Smoke inhalation</td>
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<td>- Administering IV fluids too much and too fast</td>
<td>- Trauma (strangulation)</td>
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<td>&quot;normal&quot; patients</td>
<td>- Electrocution</td>
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<td>- Hyponatremia</td>
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<td>- Administering too much fluid to patients with</td>
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<td>oliguria and anuria</td>
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Anemia

Intravenous fluids are sometimes used excessively in the anemic patient when the decrease in red blood cell mass is misinterpreted as total blood volume depletion, when in fact the plasma volume might even be expanded. To compensate for decreased tissue oxygen delivery, the heart rate increases, and if these patients are subjected to large fluid volumes over a short period of time, pulmonary edema can occur.

Anemic cats, in particular, are susceptible to intravenous overload from crystalloid infusions. The dehydration deficit and maintenance fluid volumes should be gradually replaced over a 24-hour period with an isotonic crystalloid solution, while packed red blood cells are administered to correct the anemia. The volume of whole blood infused should be considered when calculating the volume of crystalloid for infusion (give 25-50% of the computed crystalloid volume and adjust according to further need).

Other Disorders Affecting Extracellular Fluid Volume Excess

These conditions are associated with an increase in total body salt and water and occur in a variety of clinical settings including congestive heart failure, glomerulopathies, liver fibrosis, and protein-losing enteropathy. They are characterized with a decrease in "effective arterial volume," which stimulates the renin-angiotensin-aldosterone cycle and the release of antidiuretic hormone to promote renal salt and water retention, respectively. Because of increased venous pressure from heart failure and cirrhosis or because of decreased plasma oncotic pressure associated with hypoalbuminemia, the retained salt and water move into the interstitial and other body spaces, causing edema, ascites, or pleural effusion. Hypervolemia amounting to 20-30% in water excess can cause pulmonary edema.

Vascular compliance (tone) is an uncontrolled factor affecting the response to parenteral fluid infusions. In cases where vascular compliance is low, as might be seen with atheromatous (as might occur with severe hypothyroidism in the dog) or mineral deposits covering the endothelium (as might occur with hyperphosphatemia and secondary renal hyperparathyroidism), a fluid load of colloids especially can lead to dramatic increases in blood pressure. This effect is further exaggerated in the presence of excess circulating catecholamines. The clinical effect can be either right or left ventricular volume overload with systemic and/or pulmonary edema.

Patients with any of these conditions are extremely sensitive to intravenous overload with crystalloid solutions. Treatment should be directed toward improving the underlying primary pathologic process. Fresh or fresh frozen plasma should be used to volume expand animals with hypoalbuminemia, although in glomerulopathies and protein-losing enteropathy (PLE), beneficial effects are usually temporary at best because of continued protein losses, especially with PLE.

Cardiac compliance also plays an important role in the response to fluids. A number of clinical settings including sepsis, myocardial ischemia, and the use of positive end expiratory pressure (PEEP) may affect the relationship between end diastolic cardiac volumes and measured cardiac filling pressures. Hence isolated measurements of cardiac filling pressures may fail to correlate with other indices of cardiac function.

Heart failure patients receiving intravenous fluids should be closely monitored for weight gain and respiratory distress caused by intravascular fluid overload. More precise parameters include dynamic changes in cardiac filling pressures, arterial pressure, and central venous pressure (CVP), although CVP changes can provide its warning too late). Other "cageside" monitoring techniques should include urine output, blood lactate levels, and mental status. Close patient observation can often detect the earliest signs of pulmonary pathology as seen with the detection of a rapid respiratory rate or the auscultation of pulmonary rales. Under optimal conditions, monitoring of central venous and pulmonary wedge pressures is helpful for avoiding
this potentially fatal complication, but the latter technique is hardly ever used in veterinary patients. The reader is referred to other sources for details regarding these techniques.

For conditions where vascular permeability is altered, the debate over the choice of fluids remains unsettled. When parenteral fluid therapy is indicated in the cardiac patient, even the slightest amounts can cause overload. Either 0.45% saline alternated with 5% dextrose solution (D-5-W) can be used for maintenance once blood pressure is restored to normal. Efforts should be made to avoid hypokalemia by adding potassium chloride solution to the fluids at a dose of 7 to 10 mEq/250 ml. Periodic monitoring of serum electrolytes is necessary for accurate and timely treatment adjustments.

**Oliguric and Anuric Renal Failure**

The urine output of all critically ill patients should be monitored, especially during periods of intensive fluid therapy. Fortunately, many oliguric patients will begin producing urine after they receive one half of their estimated dehydration deficit values during the first one to two hours of treatment. If urine production is inadequate, the following protocol is recommended:

1. Insert an indwelling urethral catheter and empty the urinary bladder of any residual urine.
2. Administer the calculated dehydration deficit fluid volume over the first two to four hours of treatment where conditions allow.
3. Once rehydration has occurred and blood pressure is restored to a safe level (>100 mm Hg), administer furosemide (4 mg/kg IV push) or mannitol (0.5 gm/kg IV) over a 10-minute period. Do not use mannitol if the patient is already hyperosmolar as seen in diabetes mellitus.
4. If no urine flow occurs, re-administer furosemide (8 mg/kg IV push) or administer dopamine (1 to 2 ug/kg/min IV). The use of dopamine is no longer recommended in humans according to some sources.
5. If oliguria or anuria persist, the amount of fluids infused per day will consist of the sum of the measured urine output, the insensible water loss (13-20 ml/kg/day), and the extra losses caused by vomiting or diarrhea. Intravenous fluids administered in quantities that exceed the insensible and extra losses will accumulate to cause intravenous fluid overload. The withholding of all fluids and the institution of hemodialysis will be required to rid the body of uremic toxins.

Plasma volume expansion should be accomplished with lactated Ringer’s or 0.9% saline; the latter is preferred if hyponatremia is present. Maintenance fluids can initially consist of Ringer’s lactate or acetate but can eventually be reduced in concentration to one-half strength in the absence of any renal sodium-losing disorder.

**References**

Acute pancreatitis usually affects the middle-aged and older dog and cat. The severity of this condition can range from mild as seen in some forms of edematous pancreatitis to life-threatening as seen in the hemorrhagic necrotic form. Some of the known or suspected causes of this condition in the dog and cat include accidental or surgical trauma, pancreatic ischemia, and hypercalcemia (dog). Postprandial hyperlipidemia can stimulate pancreatic secretion in the dog and perhaps this explains why the ingestion of a fatty meal is often suspected as a predisposing cause of acute pancreatitis. The use of certain drugs such as glucocorticoids and azathioprine have been implicated as causative factors as well. In most cases, the cause of acute pancreatitis in the dog and cat is listed as idiopathic. The basic pathophysiologic change involves a premature activation and release of pancreatic hydrolases followed by inflammation and autodigestion of the gland and the surrounding tissues.

**History and Physical Examination Findings**

The usual signs are acute vomiting, anorexia, and depression. Rarely, vomiting will be absent, especially in cats. Diarrhea can occasionally occur. The duration of these complaints can range from fulminating with severe forms of pancreatitis to mild and intermittent with the more mild form.

The physical examination findings again will vary depending on the severity of the problem. The abnormalities accompanying mild pancreatitis might be restricted to slight mental depression and equivocal abdominal tenderness. Those associated with hemorrhagic necrotic pancreatitis can include marked mental depression, fever, hypotension, tachypnea and sinus tachycardia, abdominal pain, and variable amounts of dehydration. Clinically detectable icterus does not accompany the peracute phase of this disorder, but it can appear in the subacute stages due to cholangiostasis with or without bile duct obstruction that occurs from fibrous adhesions. A reddish-brown colored ascitic fluid can sometimes accumulate with acute hemorrhagic necrotic pancreatitis.

**Radiographic and Clinicopathological Findings**

Abdominal radiographic findings are normal in mild pancreatitis. However, those accompanying the severe forms include: (1) a regional peritonitis involving the anterior and midabdominal regions and (2) lateral displacement of the duodenum. Patients with hemorrhagic necrotic pancreatitis can rarely also show pleural effusion and pulmonary fluid accumulation on their thoracic radiographs. Life threatening acute respiratory distress syndrome can also complicate the
clinical picture. Ultrasonography can reveal a heterogeneous pancreatic tissue pattern reflecting edema and inflammation. Peripancreatic mineralization can be seen as a bright hyperechoic density. Free abdominal fluid can also be evident.

The hemogram characteristically shows hemoconcentration and an elevated total protein due to intravascular volume depletion. The serum protein levels might decrease after plasma volume expansion with crystalloid fluid administration. Third spacing will also cause lowering of the plasma protein. The leukogram has a leukocytosis characterized as a neutrophilia. A left shift is commonly seen with the more severe forms of pancreatitis.

The BUN and serum creatinine levels can be mildly elevated and reflect pre-renal azotemia due to the dehydration associated with mild pancreatitis. However, with the more severe forms, these elevated parameters can reflect acute renal failure as well which can lead to the patient’s demise.

The serum electrolyte levels will vary. Mild hypernatremia (serum sodium > 147 mEq/l) reflects dehydration, while hyponatremia might be spuriously due to hyperlipidemia and/or hyperglycemia. Decreased serum sodium levels can also represent actual depletion from gastrointestinal loss. Hypochloremia reflects gastric chloride ion loss. Serum potassium levels are usually normal initially; however, hypokalemia might occur by the second or third day if the patient does not receive intravenous potassium ion supplementation. A low serum potassium level can also be due to metabolic alkalosis. Hyperkalemia suggests oliguric or anuric renal failure. Hypocalcemia is seen in hemorrhagic necrotic pancreatitis and is due to a decrease in the amount of protein bound calcium and saponification of the abdominal fat.

Elevated serum liver enzyme levels (AST, ALT, and alkaline phosphatase) are due to cholangiostasis and hepatocellular damage. These changes are usually self-limiting and of no clinical significance so long as there is no common bile duct obstruction. Hyperbilirubinemia and bilirubinuria can accompany cholangiostasis with or without extrahepatic biliary tract obstruction.

Serum glucose levels will range from low to marked elevations. Hyperglycemia may be transient due to temporary serum increases in glucagon, cortisol, and catecholamine hormones along with simultaneous impaired insulin secretion. If a substantial destruction of pancreatic B cells occurs, the animal might acquire permanent diabetes mellitus. Hypoglycemia can occur with hemorrhagic pancreatitis because of the accompanying enterotoxemia or sepsis.

Hyperlipemia due to hypertriglyceridemia can occur with acute pancreatitis. It can be due to an underlying metabolic disorder such as hypothyroidism or primary hyperlipidemia.

Hyperamylasemia frequently occurs with acute pancreatitis. It is important to remember that the degree of elevation does not necessarily parallel the severity of the disease and that decreased
glomerular filtration will elevate and sustain the serum amylase level. Furthermore, hyperamylasemia in the absence of acute pancreatitis can occur with other abdominal abnormalities such as bowel obstruction or perforation and liver inflammation.

Hyperlipasemia is a more dependable laboratory test for the diagnosis of acute pancreatitis according to several authors. This author essentially agrees with this observation, but several cases of acute pancreatitis have occurred where the animal had marked hyperamylasemia but an absent or only minimally elevated serum lipase level. Serum amylase levels that are 5x normal are suggestive of acute pancreatitis. Therefore, both of these parameters are recommended, with the test results interpreted in light of the patient’s other clinical findings. A pancreas-specific isoenzyme for lipase is available for the dog and cat. It is important to use this test in light of the fact that it is only one of the several pieces of the puzzle that are used to diagnose this condition. Several situations have already occurred when the CPLI was abnormally elevated while the dog showed no clinical signs of pancreatitis which was verified at surgery. The only definitive test for diagnosing acute pancreatitis is by direct visualization +/- biopsy.

**Some of the Newer Diagnostic Tests for Pancreatitis in the Dog and Cat**

CPLI - Canine pancreatic lipase immunoreactivity - 80% sensitivity when > 200 ug/L.
FPLI - Feline pancreatic lipase immunoreactivity - > 10 ug/L. Suggestive.
TLI - Trypsinogen like immunoreactivity. Sensitivity is 33% in dogs and 30-60% in cats; this test is best used for diagnosing exocrine pancreatic insufficiency..
Abdominal ultrasound - 65% sensitivity.

**Treatment**

Patients who present with mild historical and physical signs with unremarkable laboratory test results can often be treated conservatively with food intake restriction for 1-2 days and periodic offerings of water. If vomiting continues, complete oral intake restriction should be extended for 3-5 more days, and the animal’s fluid requirements should be met with the administration of parenteral fluids.

The most important aspect of treatment with the more severe types of pancreatitis is appropriate parenteral fluid therapy. Hypotension should be treated with rapid volume expansion consisting of lactated or acetated Ringer’s, Plasma-Lyte, or 0.9% saline solutions at an initial dosage rate of 70-90 ml/kg over the first 1-2 hours of treatment (administered in divided portions IV every 15-20 minutes) along with closer monitoring of the cardiopulmonary status of the patient in order to avoid pulmonary edema from endothelial leak. Once the vital signs are stabilized, a maintenance fluid rate of 60 ml/kg can be given over the remaining 24-hour period; further adjust as needed. The fluid dose for the cat should be one-half that for the dog. Maintenance IV fluid volume infusions
usually consist of 2.5-5% dextrose in 0.45% saline solution; these should be supplemented with potassium chloride (3-5 mEq/kg BW/day) and soluble vitamin B complex. Any acid-base abnormalities should be recognized and appropriately treated. Hypoproteinemic animals should receive fresh plasma or plasma substitutes as needed. This will increase the plasma oncotic pressure and consequently help prevent edema formation, pleural effusion, pulmonary edema, and renal failure.

Urine output should be closely observed after the patient is adequately volume expanded. Oliguria or anuria should prompt a furosemide-induced diuresis so long as the patient is rehydrated. Osmotic diuresis should be avoided if the animal's plasma is already hyperosmotic. Maintenance parenteral fluid volumes during the impaired urine output period should consist of the volume of urine produced plus any insensible fluid losses. Unsuccessful forced fluid diuresis attempts during anuria will cause potentially fatal pulmonary edema.

Historically, various gastrointestinal drugs such as atropine and propantheline bromide were commonly used. However their adverse side effects often exceeded the benefits. Therefore, the current recommendation calls for withholding parasympatholytic drug treatment so long as restriction of food and water suppresses vomiting. Metoclopramide (0.2-0.4 mg/kg iv or SQ tid), ondansetron (0.11-0.17 mg/kg iv q6-12 h), the newer agent maropitant (Cerenia) (1.0 mg/kg SQ once daily) can be used as antiemetics. The H2 blockers or the H+ pump blockers might be beneficial because of their antacid effects. Although there are theoretical justifications for their use, there are no well controlled clinical trials that substantiate any proven benefit. Acute pancreatitis commonly causes a paralytic ileus which can be corrected with cisapride given orally (0.5 mg/kg) or by J-tube bid-tid.

Antibiotics are usually reserved for the moderately or severely sick patient, and their use is still controversial in the absence of proven infection. A bactericidal antimicrobial effective against gram-negative bacteria is preferred. Aminoglycoside antibiotics are not recommended due to their potential nephrotoxicity in a clinical setting where renal function might already be impaired.

Providing adequate nourishment is probably the most difficult aspect of treatment. Although 5% dextrose solutions will provide a small amount of calories that might suffice for the first few days of treatment, this form of treatment falls far short of providing the patient's caloric needs over the one to two week period of complete oral intake restriction that is sometimes required to inhibit pancreatic proteolytic enzyme secretion. Hopefully most animals will be able to resume the intake of liquids and then solids after the first 5-7 days of "nothing per os" (NPO).

Intravenous parenteral nutrition should be considered when the patient resumes vomiting after oral feedings are begun. This procedure is risky because there is evidence that the intravenous infusion of amino acid and lipid solutions can stimulate pancreatic secretion in the dog. Some of the
problems associated with intravenous hyperalimentation include catheter-induced phlebitis, septicemia, plasma hyperosmolarity, meticulous preparation requirements, and expense. This feeding technique is therefore usually reserved for large medical facilities that can afford the expense and the manpower requirements. A tube jejunostomy (J-tube) and the infusion of elemental nutrients is an efficacious way of nourishing an animal that has protracted acute pancreatitis, but abdominal surgery is necessary for the correct placement of the J-tube. E-tube and G-tube feeding are also successful ways to provide for enteral nutrition with the E-tube becoming especially popular. The administration of the food solution is done by small volume “trickle” tube infusion. Vivonex and Clinicare can be used to provide enteral nutrition, but other products are available.

Analgesic treatment should be reserved for the animal that has severe and intractable pain. Buprenorphine (0.01-0.02 mg/kg SQ q8h) is very safe and effective for dogs and cats. Fentanyl patches can be used as can epidural morphine. Butorphanol (cats 0.4-0.8 mg/kg SQ q6h; dogs 0.05-0.4 mg/kg IV, SQ q6h) can also be used.

Insulin treatment is indicated when the blood glucose level exceeds 300 mg/dl. Regular crystalline zinc insulin (2 unit/kg) is preferred because its short duration of action is advantageous if the hyperglycemia is transient. If the patient shows a continued need for insulin treatment, it should then be managed similar to other diabetics. Do not wait for a sustained hyperglycemic condition to disappear spontaneously because of the catabolic consequences that can occur.

Surgical peritoneal lavage was a recommended treatment for acute hemorrhagic necrotic pancreatitis in the 1970's. Because pancreatic exudate contains many potentially harmful substances that can be absorbed into the circulation and cause cardiovascular and respiratory instability, peritoneal lavage was advocated as a logical way to rid the body of these toxic substances. Although some patients appear to benefit from this treatment, several studies in humans have failed to statistically support the recommendation for its routine use. This option can be done if the animal has to go to surgery for other complications which usually occur after the first week of medical treatment. Lavage-induced hypoproteinemia and electrolyte deficiencies should be replaced with plasma and balanced electrolyte solutions. The surgical exploratory is also helpful for debriding necrotic debris, removing accumulations of pus, and allowing for the insertion of a J-tube.

**Complications and Long-Termed Medical Management**

There are several complications of acute pancreatitis. These include: diabetes mellitus, pancreatic abscess and pseudocyst, bowel infarction and perforation, bowel obstruction, renal failure, bile-duct obstruction, septicemia, pancreatic fibrosis with exocrine insufficiency, consumption coagulopathy, relapsing acute pancreatitis, and death.
**Returning the Patient to Feeding**

Feeding and drinking will be resumed only when the patient has not shown any evidence of vomiting, regurgitation, or nausea for five consecutive days.

The protocol that has worked well for me is:

Day 1: Offer free choice ice cubes to lick. If no vomiting...

Day 2: Offer 5-10 laps of water every 2 hours. If no vomiting...

Day 3: Offer water free choice and begin offering 10 laps of beef or chicken bouillon every 2-4 hours. If no vomiting...

Day 4: Add 3-4 saltine crackers to the bouillon and feed every 4-6 hours. If no vomiting...

Day 5: Feed low fat reducing diet and send home on long-term reducing diet.

The long-terms medical management in the dog should include a low fat diet; the effect of this recommendation is not known in the cat. The animal's total daily food intake should be divided into 2-3 small feedings. Diabetes mellitus and pancreatic exocrine insufficiency should be treated according to standard recommended protocols.
A PICTURE IS WORTH 1000 WORDS - 2017

Michael Schaer, DVM
University of Florida
College of Veterinary Medicine
Post-Trauma Trigeminal Motor Palsy

Acute Vomiting – Sublingual Neg
Look Again!

Positive Linear FB
Negative Lap – No Linear FB

Comparing

What is Your Diagnosis?
Dx Hemorrhagic Cystitis; then PU; Cat Postop

Where is the Urinary Bladder?
Colonic Volvulus Through Diaphragmatic Hernia
Hair Strangulation!

Leishmania – Ocala FL.
Toxic Strep Syndrome
Where is the E Tube?

Why Did This Dog Go Apneic?
Angioneurotic Edema – Pre-Post (24 hrs) Glucocorticoids
Uremic Glossitis

Pancreatic pseudocyst

Aspergillus Uveitis
Classic Acute Pancreatitis Radiographs

Lymphangiectasia

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One Hurting Mandible

Staphylococcus