Back to the Basics!

Rebecca Kirby, DVM, DACVIM, DACVECC
Animal Emergency Center
Glendale, WI

The real "art" of medicine lies in the clinician's ability to perform and interpret accurately the findings from the patient history, physical examination (PE) and the minimum data base. The animals are "talking" to us, and we have to be patient and wise enough to understand what they are trying to tell us. The history, PE and assessment of the minimum data base are THE most important diagnostic tools. Additional tests should be done to confirm, deny or better define what the clinician has found from the initial examination. The veterinary team is to be clearly reminded that "tunnel vision", the failure to remain open to other options for the diagnosis and rule out other potentially life-threatening problems, can cause a great deal of harm to the patient. Here is an example of "tunnel vision":

A 4 month old puppy presents with bloody diarrhea. The clinician, after performing the history and PE has decided that the dog has parvovirus diarrhea and treats accordingly. The clinician "knows" that this is the problem and fails to rule out other potentially life-threatening causes. Had the clinician taken a radiograph, the small ball causing total intestinal obstruction would have been found. The intestine perforates and the puppy decompensates.

This does not require that *every* diagnostic test be performed to ensure that we haven't missed something. It does require an excellent history, physical exam and assessment of the data base, as well as an open-mind to reassess the initial diagnosis or treatment plan should the patient not respond as anticipated or decompensate in spite of aggressive treatment.

The History

The art of taking a rapid, but thorough history is dependent upon organization. The receptionist or nurse that is registering the patient should write the initial presenting complaint on the record, along with the signalment (species, breed, age, sex). From the signalment, alone, the clinician is reminded to consider potential breed and age related problems and problems associated with intact reproductive tracts. The nurse or veterinarian then takes the history in the following order.

Last normal

The clients are asked "When was your pet last absolutely normal?" This is meant to determine whether this is truly an acute illness or one that has occurred over a more extended period of time than the presenting complaint might suggest. For example, a pet can present for vomiting for one day (which is an acute sign) but will have started showing signs of illness several days earlier by not eating or acting lethargic (which suggests a more chronic problem).

A history of the animal only having abnormal signs for 24-48 hours (they were absolutely normal prior to this time) directs the clinician to focus on first ruling out problems that typically have an acute onset, such as: trauma, acute toxicities, medication overdose, blood clots, ischemia, foreign bodies (GI, ocular or respiratory), acute hemorrhage, or an acute obstruction (urinary, respiratory, gastrointestinal). When the animal has had a chronic or recurrent illness prior to the onset of the acute signs, then acute decompensation of the chronic illness must be added to the list above. Common examples include congestive heart failure, hepatoencephalopathy, renal failure, and status epilepticus.

When the animal has had several days to weeks of abnormalities (whether obviously related to the presenting complaint or not), then other causes are more likely, such as: metabolic illness (liver, renal, endocrine, gastrointestinal, pancreatic, etc), neoplasia, immune mediated processes, infection, and toxicities with delayed onset of signs (eg. rat poison, ethylene glycol),

Progression

Once it has been determine when the pet was last absolutely normal, the progression of the problems must be detailed on a day-by-day basis. What started first? Is that now better worse or the same? If better or worse, when in the progression of the problem did it change? When an abnormality is identified, it must then be characterized. Important characterizations of some common presenting complaints are listed below and can guide the clinician in formulating a diagnostic and therapeutic plan.

Systems review

Questions should be asked to identify any abnormalities of body or organ system that was not mentioned in the progression. The answers to these queries must be documented in the record. Abbreviations can be used such as:
No v/d/c/s/dc indicates no vomiting, diarrhea, coughing, sneezing, discharges

Past history

Past medical history presumed by the owner to be unrelated to the presenting complaint should be documented here. This will include vaccination history (especially rabies), heartworm preventative, and worming history.

Medications

All medications, to include vitamins, supplements, over the counter drugs, and prescription drugs should be listed. Any past blood transfusions are noted as well as any past problems with anesthesia.

Allergies

Any known contact, inhaled or drug allergies are noted here

Characterization of common presenting complaints

Complaint	Characterization	Localization	Interpretation
Vomiting	white, foam	gastric	saliva, water, acute, inflammatory
	yellow	gastric	digested bile, more chronic
	red streaks	gastric	result of vomiting
	red fluid	gastric	active hemorrhage –acute ulceration
	"coffee grounds"	gastric	digested blood – chronic ulceration
	green	upper duodenum	obstruction, ileus
	brown, fetid	jejunum, ileum	ostruction, ileus
Diarrhea			
	Watery	small bowel	fluid and electrolyte abnormalities
	Mucoid	large bowel	not typically assoc with systemic signs
	Red blood	large bowel	if systemic signs, look at lower ileum
	Melena	stomach Small bowel	digested blood – ulcer, lower platelets, obstruction
Cough			
	Dry, "honking"	large airway	tracheal inflammation, irritation
	Productive	bronchi	fluid – edema or inflammation
	Soft cough	bronchioles	lung fluid – edema, hemorrhage
Collapse			
	Conscious, alert	metabolic, cardiovascular	hypoglycemia, hypokalemia hypoxia – heart disease, arrhythmias
	Unconscious	cerebrum or brainstem	cardiovascular - hypoxia or neurologic – edema, hemorrhage, mass blood clot, hypoglycemia
Lameness			
forelimb	toe touching	foot, shoulder	doesn't want to bear weight
	carries limb	elbow, neck	doesn't want to extend limb
hindlimb	toe touching	foot, hip	doesn't want to bear weight
	carries limb	knee	doesn't want to extend limb
Nasal discharge			
	unilateral	nasal passage	localize to side of discharge
	bilateral	nasal or systemic	both sides of nose or systemic problem such as infectious disease, coagulopathy

The physical examination

Each clinician must develop their own style and routine for the physical examination (PE). The key is to be consistent and thorough. A rapid evaluation of the ABCs has been done with the primary survey. The secondary survey allows a more focused and thorough evaluation. Saving the examination of the body parts most likely related to the presenting problems to the last of the PE can aid the clinician to complete the total examination. A head to tail routine keeps the clinician focused, with equipment dependent examinations done at the end of the exam (to save time).

Safety is always first! Protect yourself, your staff and your clients from injury by asking the owner if the pet has every nipped or bitten anyone. Do not hesitate to ask your staff to help with restraint, to apply a muzzle, or to insist on chemical restraint with an aggressive or fractious pet. Know the vaccination history.

Physical examination tips

While you are taking the history, encourage the owners to allow their pet to walk freely around the enclosed examination room. Note the animal's gait, posture, mentation, response to verbal stimuli, and behavior/aggression. Does the pet avoid movement of the neck or show a "root signature"?

As the clinician progresses from head to tail, neurologic and orthopedic exams are done along with the general PE. The clinician progresses down the body, the hands are used to feel the skin, muscle and bones for lumps, bumps, swelling, pain, symmetry or any other irregularities. The hairs are parted to look for bumps, redness, swelling, or discharge. Body parts are gently manipulated to detect pain and range of motion. Should an area of pain be identified, finish the general PE, then return to the painful region, with adequate restraint, to better characterize the site and cause of the pain. It is helpful if the pet can stand during the examination.

Head

An overall look at the head is made from a slight distance to detect any asymmetry or discharges from the eyes, nose, ears or mouth. The skin of face is tapped with the finger all over to get an impression of their ability to feel and move their facial muscles. Mucous membrane and corneal moisture are assessed to estimate the hydration status. The eyes are checked for discharge, color of the conjunctiva, corneal clarity and moisture, pupil size and shape, response to menace (do they blink and retract their eye), third eyelid protrusion, and pupil symmetry. Closing the pet's eyelid for a moment can demonstrate the direct pupil response to light. When the eyelid is opened again, the clinician can see if the pupil had dilated in the dark and can now constrict when returned to light. Penlight testing of pupils (direct and consensual) and retinal examination are done during the equipment phase of the examination. The nose is checked for discharges. The lips are lightly pinches to ensure that they can be retracted. The mouth is opened, assessing jaw tone, and tongue movement and position. Oral mucous membranes are examined for color, capillary refill time, petechiation, ulcerations, bleeding, infection or other problems. The teeth are evaluated and can be tapped with the finger to see if there is any sensitivity. If the animal allows, a finger can be placed at the back of the throat to check for gag and swallow reflex and to see if that area is free of obstruction. The ear flaps are checked for swelling or hematomas, and the ear canals grossly examined for discharge, debris and foul odor. The otoscopic exam is done during the equipment phase of the PE.

Important

Any animal that has had head trauma, loss of consciousness, prolonged seizures, or other indication of intracranial edema or hemorrhage must maintain a normal head position throughout the examination.

Neck

Below the chin, the neck is palpated to detect abnormalities of salivary glands and lymph nodes. The larynx is palpated to see if the pet swallows and the trachea palpated to see if a cough is initiated. Muscle tone of the neck muscles is noted. The neck is moved right and left, then up and down, just below the head to detect high neck pain. This is repeated just anterior to the shoulders to detect lower neck pain. Skin elasticity is examined over the neck region as part of the estimation of hydration status. The head is elevated and the jugular veins evaluated for distension or pulse, a reflection of pericardial effusion.

Important

Do not manipulate the neck of any animal with intracranial problems or at risk for high cervical fracture (may present conscious and recumbent with constant extension of all four limbs).

Body posture and tone

At this time, the body posture and tone are evaluated. Is the pet's back arched? Are the elbows abducted? Are the hind limbs and pelvis dropping down? Press down gently on the back between the shoulder blades to determine the tone in the forelimbs, and then on the pelvis. If the animal does not resist, there is likely forelimb or hindlimb weakness.

Forelimbs

The hands are moved up and down each forelimb to feel for any changes in the skin, muscles and bones. The toes are parted and the skin, pads, nails and hairs are examined. Each joint is lightly tapped with a finger to check for joint swelling. Then each joint is flexed and extended to detect pain, crepitance, or abnormal range of motion. The whole forelimb is adducted, abducted and moved front and back to check for range of motion and pain in the shoulder.

Thorax

At this time, the hands are moved down the sides of the thorax, feeling for abnormalities. The hairs are parted and the skin examined. The ribs are palpated for fracture or pain.

Hind limbs

Same procedures as for the forelimbs

Spine

The muscles are palpated up and down the spine, with some downward compression to detect pain, hypersensitivity, or any weakness initiated by the exam. Any deviations of the spine are noted.

Abdomen

Palpation of the abdomen is saved for the last part of the initial examination. The hands should first be run down the sides of the abdomen from dorsal to ventral, then cranial to caudal, to assess the hair, skin, and muscles. The abdomen is then gently balloted to see if there are any large masses or fluid waves present. A routine should be developed for deep palpation of the abdomen. Starting a the cranial abdomen, the examiner is positioned behind the rump of the animal and reaches to the rib cage with one hand on each side of the pet's body. The hands are placed behind the last rib and deep palpation begins, moving cranially under the rib cage. Any obvious palpation of the liver or spleen is noted, as is any pain on palpation. If the stomach can be palpated or percussed, this is abnormal. For deep or barrel chests or obese animals, it may be helpful to have someone elevate the front limbs while you palpating to shift the anterior abdominal contents caudally.

The examiner may not opt to move to the side of the animal. One hand is placed on each side of the abdomen dorsally, just behind the rib cage to palpate the kidneys The right kidney is more posterior than the left and not as tightly adhered. The size, shape and texture of the kidney should be noted, as well as any pain on palpation. Moving ventrally from here, the examiner will try to palpate or ballot the spleen.

Then placing the hands near the paralumbar muscles, deep palpation (in a circular massaging type motion) is done of the bowel in the mid abdominal region - moving from dorsal to ventral. The bowels are allowed to slip between the fingers of the hands. It should be noted whether or not the bowels contain fluid, feces or gas, if they are mobile, and if there is any pain on palpation. Any masses associated with bowel should be gently compressed with the fingers to see if they indent or not. Fecal material will indent, but tumors, lymph nodes and most foreign bodies will not.

At the pelvic inlet, the urinary bladder is palpated. It can be determined how much urine is present and if the bladder is easily expressed.

Should the pet retch or vomit with palpation of any abdominal organ, this suggests that there is peripheral receptor stimulation (stretch or inflammation) as one of the causes of vomiting. This can become important in selecting anti-emetics.

Equipment examinations

Rectal temperature

Stimulation of the anal sphincter can initiate a vasovagal reflex and severe bradycardia. The unstable cardiovascular patient should be closely monitored for decompensation while taking a rectal temperature. An alternative would be to use an ear thermometer, recognizing that the ear temperature can be 1-5 degrees Fahrenheit below the rectal temperature. High temperatures suggest inflammation or heat exposure and an accelerated rate of metabolism. Additional crystalloid fluid replacement may be required and close monitoring of blood glucose. Hypothermia can reflect cold exposure, medications that reset the thermoregulatory center (non-steroidal anti-inflammatory drugs) or poor perfusion. Hypothermia is anticipated in the cat in shock, with warming the cat a vital part of the resuscitation plan (see shock and resuscitation). Hypothermia in the dog resulting from poor perfusion is very significant, warning the clinician that this dog will likely have a third body fluid space and will have a difficult time of fluid resuscitation.

Ophthalmoscope

The direct and consensual papillary light response is assessed for each eye. The retinas are examined for chorioretinitis (infectious diseases), hemorrhage (coagulopathy or hypertension, edema of the optic disc, (elevated intracranial pressure), engorged blood vessels (hypertension), or retinal detachment (trauma, infectious diseases).

Otoscope

The ear canals are examined for debris, foreign bodies, exudates, wax, polyps, or any other abnormalities.

Stethescope

It is critical to block out everything all other sights and sounds while you are ausculting the heart and lungs. Form the habit of listening to the lungs prior to the heart or airways. Our ears better attuned to soft sounds initially. [Its hard to appreciated the fine points of classical music immediately after you've heard hard rock!] Harsh lung sounds heard in the periphery can reflect pleural friction rub, or in cats may be the only abnormality heard in the lungs with pulmonary edema. High pitches wheezes generally reflect fluid or constriction of the bronchioles. Moving from the periphery to the center of the chest, air sounds will become louder and lower pitched as the airway diameter increases.

As you move to the center of the chest, the heart is now ausculted. The peripheral pulse, usually femoral, should be palpated simultaneously to cardiac auscultation ensure that there are no pulse deficits suggestive of an arrhythmia. The heart rate and rhythm are determined. The heart is carefully ausculted for murmurs and/or gallops. Murmurs in the dog are typically loudest on the left mitral region, though the right should also be evaluated.

Remember that in the cat, most murmurs or gallops are intermittent, not continuous. These are best detected on the left side, ventral, just off of the sternum, at the level of the 3rd or 4th rib. You need to listen for at least 2 minutes before determining that there are no murmurs or gallops. In addition, a murmur is the sound made as fluid regurgitates through an incompetent valve. If there is little fluid, the murmur may not be heard. Therefore, it is critical that the clinician reassess the heart of the dog and cat for any murmurs or gallops after fluid resuscitation has occurred.

Pleximeter

Limb reflexes can be checked, if there is any indication of gait abnormalities or limb dysfunction.

Minimum data base

As the intravenous catheter is being placed for resuscitation, whole blood is collected in 2 or 3 heparinized microhematocrit tube from the hub of catheter for the initial minimum data base (MDB). This blood will be used to determine the packed cell volume (PCV), plasma total solids or proteins (TS), blood glucose and blood urea nitrogen (BUN). Blood is then drawn into a lithium heparin tube fore venous blood gases and electrolytes during resuscitation. A blood smear is made to estimate platelet numbers and blood draw for an initial activated clotting time (ACT) or prothrombin time (PT) and partial thromboplastin time (PTT). Below are values that are of great concern when found on the initial MDB, referred to as red check mark items to alert the clinician and nursing staff of potential problems.

Minimum data base values of concern

PCV
- ✓ **> 60% or < 20%**

TS
- ✓ **> 9.0 or < 5.0**

Dextrose
- ✓ **< 60 mg/dl**
- ✓ **> 400 mg/dl**

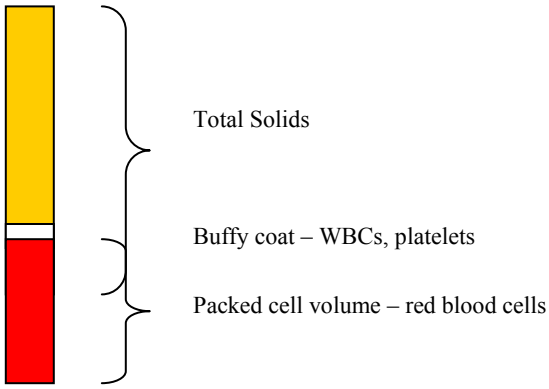
BUN
- ✓ **> 40-50 mg/dl**
- ✓ **< 5 mg/dl**

Lactate
- ✓ **> 2 mmol/L**

Electrolytes
- ✓ **Sodium > 170 or < 135**
- ✓ **Potassium < 3.0**
- ✓ **Calcium > 6.0 or < 3.0**

The Microhematocrit Tube – More than just a hematocrit!

The microhematocrit tube provides a great amount of valuable information. In addition to the PCV and TS, the character of the serum is assessed and the buffy coat smeared out for microscopic evaluation. The microhematocrit tube is spun down in a hematocrit centrifuge.



PCV

The PCV or hematocrit measures what percentage of whole blood is composed of red blood cell (RBCs). The RBCs are the heaviest components of whole blood and are centrifuged to the lowest (outermost) portion of the tube. A hematocrit scale is utilized to determine the PCV. The PCV should be assessed in conjunction with the TS.

Variable	Cause	Interpretation	Plan
PCV			
> 60%*	general concern:	hyperviscosity	O2,fluids
	hypoxia	pulmonary disease	treat cause
	hemoconcentration	loss of plasma water	fluids
	overproduction	polycythemia vera	± bleed
< 20%	general concern:	tissue hypoxia	± transfusion
	blood loss	hemorrhage	hemostasis
	lack of production	bone marrow problem	treat cause
	RBC destruction	immune mediated	treat cause

* PCV between 60-70% can be normal for sight hounds, ferrets, and animals living at a high altitude.

TS

The TS are measured on a refractometer and are a reflection of the proteins in the serum or plasma. The TS result from the plasma includes fibrinogen while the result from serum does not. The TS should be assessed in conjunction with the PCV and eventually, a serum albumin.

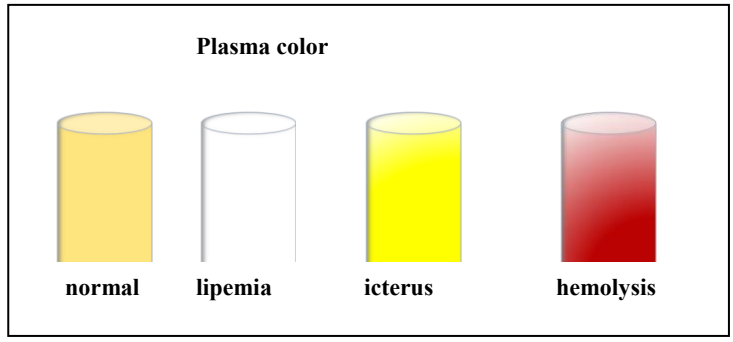
Variable	*Cause*	*Interpretation*	*Plan*
TS			
> 9.0 g/dl	general concern:	hyperviscosity	promote flow
	loss of fluids	hemoconcentration	fluids
	over production	inflammation, cancer	treat cause
< 5.0 g/dl	general concern:	loss of oncotic pull	give colloids
	dilution of plasma	excessive water	adjust fluids
	lack of production	GI or liver disease	treat cause
	loss of proteins	vasculitis, glomerular hemorrhage	treat cause hemostasis
PCV /TS			
⇧ ⇧	hemoconcentration	loss of plasma water	fluids
⇧N or ⇩	blood loss (dog)	trauma; splenic contract	hemostasis
	hemoconcentration w/ protein loss or not made	SIRS, liver, glomerular	fluids, colloid
⇩ ⇩	blood loss	acute hemorrhage	hemostasis ± transfuse
	chronic disease	liver, glomerular	treat cause ± transfuse
N ⇩	protein loss/not made	liver, glomerular GI	treat cause olloids

Buffy coat

The white layer in the hematocrit tube, between the plasma and the RBCs consists of the white blood cells WBCs and platelets, called the buffy coat. When this is thick, it suggests high WBC counts and when thin, low counts. A slide can be made of this layer and the cells examined for morphology and any inclusion bodies or parasites. Platelet estimates are best made from a drop of whole heparined blood rather than the buffy coat. However, if few or no platelets are seen in the buffy coat, further investigation is warranted.

Plasma/serum coloration

The color of the plasma or serum is illustrated below should always be reported. Normal plasma or serum should be yellow-brown, or "straw" colored. White coloration is due to lipids in the plasma/serum. These lipids can cause an elevation of TS values on the refractometer that are not reflective of serum proteins, Serum sodium results may be lower than would occur without lipemia and may not reflect the true sodium status of the blood. Icterus is seen in the serum (total bilirubin $\geq$ 3.0) before it is detected in the mucous membranes (tital bilirubin $\geq$ 5.0) of dogs and cats. Hemolytic serum can be a result of RBC disruption during sample collection or due to a toxic or immune mediate process causing intravascular lysis of RBCs.



Blood glucose

Whole blood, serum or plasma can be measured for glucose content. When a test strip is used with whole blood, the result is evaluated in light of the PCV. If the animal has a PCV greater than 55-60%, the whole blood glucose tested by labstick or meter may be lower than what is truly available in the body. With an elevated PCV, there is less plasma or serum in the drop of blood being tested, creating a lower than real glucose concentration result. If the animal is showing signs, glucose is administered. If not, then a correction of the PCV is done and the blood retested, or the plasma or serum are tested.

Low blood glucose (glucose < 60 mg/dl) can be an immediate life-threatening problem. The cause and the effect of the hypoglycemia must be considered. Causes can include liver disease, prolonged muscle activity (e.g. status epilepticus, exercise), increased metabolic rate (e.g. sepsis, SIRS diseases), or an insulin secreting tumor (insulinoma). All cells required glucose to produce energy, However, the brain required glucose at a fairly constant rate. The effect is then generalized weakness with neurologic abnormalities as a critical sign. The clinical signs will range from mental depression, twitching, seizures, non-responsive hypotension, loss of consciousness and eventually death. Should hypoglycemia occur, glucose or dextrose is administered intravenously (0.5 g/kg IV). The blood glucose is rechecked and additional glucose given as indicated. Causes such as insulinoma or sepsis will likely require continuous glucose supplementation until the underlying problem can be stabilized. This can be accomplished by adding glucose to the intravenous fluids, at the lowest concentration possible to maintain the blood glucose consistently over 60 mg/dl. It is best to correct perfusion and hydration prior to continuous supplementation, when possible.

Hyperglycemia (glucose > 250 mg/dl) will result in glycosuria by exceeding the renal threshold for resorption. Mild elevations like this can result from stress or mild insulin deficiency or resistance. Blood glucose > 400 mg/dl presents an on-going concern for the emergency or critical care patient. The causes will include insulin resistance or deficiency (diabetes mellitus), an unusual stress response, or over supplementation of fluids. When it is due to diabetes mellitus, the animal is immediately checked for ketones, The hyperosmolarity of the fluids leads to polyuria and the resultant hypovolemia. Acidosis can occur with ketone production and hypernatremia is possible due to excessive loss of water with the glucose in the urine. Hypokalemia is also likely due to the polyuria. Careful initial fluid administration is required to avoid rapid osmotic shifts of water into the brain cells. The use of colloids during initial resuscitation can help prevent this shift. In addition, once the perfusion has been addressed, an insulin CRI can begin.

It is also important to avoid or correct hyperglycemia in head trauma patients. Studies in humans have shown that maintaining the blood glucose between 80-120 mg/dl provides better neurologic recovery. This is due to the vasodilation of intracranial blood vessels that occurs from the hyperglycemia. At this time, it has not been shown that this is the same in dogs and cats.

Blood urea nitrogen

The BUN content of the blood can be determined by test strip. It must be remembered that the test strips have as their high normal >60 mg/dl. This leaves a wide range of elevation in BUN. For example, the test strip would be same when the BUN = 60 mg/gl or 250 mg/dl. Therefore it is used as a screening test. Low BUN (<5 mg/gl) should direct the clinician to look for liver disease or liver shunt. Mild to maximal test strip results can occur from dehydration (pre-renal azotemia), renal disease, post-renal BUN should be retested after perfusion and hydration have been corrected to ensure that it has returned to within normal limits. If this has not occurred, causes other than pre-renal azotemia should be investigated.

Electrolytes

Whole blood, serum or plasma electrolytes can provide life-saving information for emergency and critical patients. Sodium (Na+), potassium (K+), chloride (Cl-), ionized calcium (Cai++) and phosphorous (PO4-) should be determined, with the cause and effect assessed and appropriate therapeutic intervention initiated.

Variable	Cause	Interpretation	Plan
Na+			
< 130 mEq/L	excess free water	hypo-osmolar plasma water shift into cells	colloids, NaCl fluids; correct over 24-48 h; ± diuretic
>170 mEq/L	free water deficit	hyperosmolar plasma water shift out of cells perfusion/hydration;	colloids, NaCl fluids; restore replace free water deficit
K+			
< 3.0 mEq/L	diuresis starvation GI loss	loss of potassium greater than intake; muscle weakness	supplement IV fluid enteral feeding ± support ventilation
> 6.0 mEq/L	over dose; kidney failure; muscle breakdown	slow nerve conduction leads to bradycardia severe arrhythmias, hypotension, ± cardiac arrest	IV regular insulin and glucose and/ or calcium gluconate; ensure urine output
Cl-			
< 110 mEq/L	pyloric outflow obstruction; ileus; vasculitis;	metabolic alkalosis GI loss common	use 0.9% saline
>115 mEq/L	iatrogenicmetabolic acidosis renal disease		use low Cl- fluids ensure urine output promote blood flow
Cai++			
< 3.5 mmol/L	milk prod; from bone: kidney failure; toxins	eclampsia; pancreatitis; high PO4- diet; diuresis, diuretics; ethylene glycol	Ca++ gluconate IV treat cause
>6.0 mmol/L	mobilizedneoplasia; from bone; kidney failure; ingestion ; toxin	hyperparathyroidism; fungal disease; hypoadrenocorticism; cholecalciferol ingestion	0.9% NaCl diuresis ± furosemide ± glucocorticoids ± calcitonin treat cause

Sodium

Hyponatremia (Na+ < 13- mEq/L) can cause mild signs such as generalized weakness and mental depression. Severe signs can include abnormal mentation, seizures, stupor, coma and eventually death, I t is important to assess the perfusion and resuscitate utilizing hetastarch in saline and 0.9% saline crystalloids to minimize sudden shifts of water and electrolytes as intravascular volume is being restored. A search for causes of hypoperfusion is done and identified problems managed. Diuretics can then be used if needed

to promote free water excretion. Water excess unresponsive to diuretic therapy can be treated with peritoneal dialysis using 2.5-4% glucose dialysate. Excessive water intake or renal retention occurring in an animal with normal perfusion and hydration can be treated with water restriction.

Hyponatremia (<115 mEq/L) in a patient with severe neurologic alterations may require sodium supplementation after restoration of perfusion and hydration. Hypertonic saline (3%) can be used and the sodium deficit calculated using the following formula:

(140 – measured Na+) x 0.3 x kg body weight = mEq/L Na+ deficit.

This should be replaced slowly, over 12-24 hours. Rapid replacment has resulted in central pontine myelinolysis.

Underlying disease entities such as nephritic syndrome, liver cirrhosis, congestive heart failure, and renal failure can lead to hypo-osmolar hyponatrmia and peripheral edema. Other causes include hypothyroidism. Glucocorticosteroid deficiency, mineralocorticoid deficiency, and inappropriate release of ADH and typically do not present with edema. It is important to identify the underlying disease and treat accordingly.

Hyponatremia associated with normal serum osmolality (pseudonyponatremia) is due to hyperlipemia, hyperglycemia or hyperproteinemia which provide a dilutional factor. The underlying disease is identified and treated. ubstanced n tlucose, urea nitrogen, and toxins,. Disorders such as diabetes mellitus, toxins such as ethylene glycol, and renal failure can drawn additional water into the serum and dilute serum sodium. It is important to diagnose and manage the underlying disorder.

Hypernatremia (na+ > 170 meq/l)

The most common mechanisms for hypernatremia involve the loss of water greater than the loss of Na+ from the intravascular and interstitial fluids. Routes of substantial water loss include: gastrointestinal – vomiting and diarrhea; renal – diuretic therapy, glycosuria, post-renal obstruction, and acute or chronic renal failure; respiratory – hyperventilation or chronic rhinitis, and loss of fluids into a third body fluid space – uterus, peritoneal cavity, pleural cavity, or muscle tissues. Other causes include of high salt intake, severe hyperthermia, adipsia or hypodipsia and or sudden loss of response to ADH in head trauma and diabetes insipidus.

An acute elevation of serum Na+ concentration leads to intracellular dehydration. High sodium concentration occurs in the cerebral spinal fluid (CSF), interfering with the Na+-K+-ATPase pump. Sodium is trapped within the CSF and profound neurologic signs (depression, weakness, confusion, seizures, coma, and finally death) can occur due to dehydration of neurons.

When sodium elevation is chronic, the nervous system is initially protected by production of intracellular idiogenic osmoles which counterbalance the hyperosmolarity of the serum. This protective mechanism can be overwhelmed and, in time, neurologic abnormalities become apparent.

Severe dehydration and poor perfusion should be anticipated with hypernatremia. The use of hetastarch in saline with normal saline as the crystalloid will minimize extravasation of fluids into the interstitium or cell.

Free water deficits should be replaced slowly over 12-24 hours using normal saline, a balanced isotonic crystalloid such as Normosol-R® or Plasmsalyte-A®, or 1/2 strength saline *once adequate perfusion has been restored*.

Sodium diuresis can be promoted using furosemide, a lower sodium containing fluid, and treating the underlying disease process.. Once rehydratied, the sodium is measured and the new sodium value is used to determine the remaining extracellular water deficits through the following formula:

$$\frac{(\text{new Na+} - 140)}{140} \times \text{kg body weight} \times 0.6 = \text{water deficit (L)}$$

The free water deficit can be is replaced first using 1/2 strength maintenance solution, such as 1/2 strength saline, 1/2 strength lactated ringers, or 3% amino acid solutions such as Procalamine® or Freeamine®. This supplementation will occur over 12-24 hours. Maintenance fluid requirements are provided utilizing a balanced electrolyte solution with potassium is supplemented as needed.

Diabetes insipus and head trauma related ADH alterations will result in a very low urine specific gravity due to the loss of the effects of ADH on the renal distal tubules and collecting ducts. These patients will benefit from administration of ADH once their perfusion and hydration are restored.

Potassium

Because K+ is the principal intracellular electrolyte, serum K+ values may not accurately reflect the total body potassium stores. In addition, alteration in blood pH , massive cell injury such as crush injuries, burns, or heat stroke, and disorders altering renal K+ regulation and urine output can affect serum K+ concentration.

Hypokalemia (k+ < 3.0 meq/l)

Diuretic therapy, starvation, vomiting, diarrhea, polyuria, and medications (e.g.insulin) are important historical findings. Clinical signs are generalized muscle weakness, fladdic paralysis, ileus, impaired ventilation, and arrhythmias

An arterial blood gas is done and the serum K+ corrected for alkalosis. For each increase in pH of 0.1 above normal, there is a compensatory decrease in serum potassium of 0.6 mEq/L. Should hypokalemia persist, potassium supplementation can be required.

Hypokalemia becomes a life-threatening problem when serum values <2.5 mEqL are associated with ECG changes (T wave depression, prolonged QT interval, U waves, and ST segment depression), severe weakness or ventilatory compromise. Potassium

should be administered with IV fluids in a peripheral vein and infused at a rate less than 0.2 mEq/kg/hr. Extreme caution must be taken when renal insufficiency is present.

When potassium supplementation is not urgent, a slower infusion given according to the following guidelines is desireable:

Serum Potassium	**Potassium added/250 ml fluids**
< 2.0 mEq/L	20 mEq/250 ml
2.0 - 2.5 mEq/L	15 mEq/250 ml
2.4 – 3.0 mEq/L	10 mEq/250 ml
3.0 – 3.5 mEq/L	5 mEq/250 ml

It becomes important to identify the site of potassium loss. Gastrointestinal loss occurs through vomiting, diarrhea or malabsorption and inadequate potassium intake suring starvation. Diuresis and renal tubular acidosis result in renal loss. Medications such as sodium bicarbonate and insulin promote potassium shifts between the cytosol and plasma. Extensive soft tissue damage can result in hypokalemia due to seepage of electrolytes from the affected areas.

Hyperkalemia (k+ > 6.0 meq/l)

Hyperkalemia should be suspected in any small animal patient with renal compromise, urinary obstruction, bradycardia, arrhythmias, poor perfusion, massive tissue destruction, or severe generalized weakness. Hyperkalemia affects electrical conduction within the myocardium, prolonging repolarization and eventually depolarization. ECG changes consistent with hyperkalemia include bradycardia, tall spiked T waves, prolongation of the P-R interval, flattening of the P wave, widening of the QRS complex, and eventually sine wave formation.

A blood gas is done to derermine the effects of acidosis on K+ concentration Acidosis will cause 0.6 mEq/L rise in serum potassium for each 0.1 decrease in pH. When hyperkalemia persists in the face of severe metabolic acidosis (pH< 7.1), conservative bicarbonate therapy should be considered.

Underlying disease processes to consider include acute renal failure, end-stage chronic renal failure, renal tubular acidosis, urinary obstruction, hypoadrenocorticism, massive tissue trauma, (burns, heat stroke, crush injuries), increased potassium administration or ingestion, severe acidosis, and thrombocytosis.

When poor perfusion is due to volume deficiency, low potassium fluids should be rapidly infused. When inadequate perfusion is due to the myocardial effects of high K+, therapy to shift potassium into the intracellular space is required until the underlying disease can be identified and treated. Regular insulin (0.2 units/kg body weight IV) followed by dextrose (2 grams/unit of insulin given, followed by 2.5% dextrose in the fluids) will decrease serum potassium within 5 minutes and lasts between 20-45 minutes. Glucose can be given alone (0.1-0.5 mg/kg IV) causing endogenous insulin release, but onset is slow. Other alternatives are to administer calcium gluconate (10% solution at 0.5-1.5 ml/kg IV) or sodium bicarbonate (0.2 to 0.5 mEq/kg slowly or diluted in fluids). Calcium gluconate may offset the myocardial effects of hyperkalemia. Sodium bicarbonate changes serum pH and drives potassium into the cell. But sodium bicarbonate has other side effects and should be used cautiously in critical patients.

The mainstay of therapy for hyperkalemia is fluid diuresis and, when indicated, furosemide to promote kaliuresis. Should oliguric or anuric renal failure be the inciting cause, diuresis is not possible and dialysis is required. Severe hyperkalemia that is refractory to therapy, mineralocorticoids such as deoxycorticoacetate (DOCA) or K+ exchange resin (sodium polustyrene sulfonate: Kayexalate® orally or by enema) can be administered or peritoneal dialysis instituted.

Ionized calcium

Calcium is present in the blood boud to albumin for transport, or ina an ionized physiologically active form. Most veterinary clinical pathology laboratories measure total calcium. Because total calcium is affected by the albumin concentration, rough estimate of corrected calcium can be made. Adding or subtracting 0.8 mg/dl for each g/dl of albumin above or below normal to the total calcium value does this. It is preferred to evaluate ionized calcium values to eliminated the many variables that affect serum proteins levels. Most in-hospital electrolyte monitors provide ionized calcium values.

Hypocalcemia (cai++ < 3.5 mg/dl)

Hypoccalcemia can cause signs of severe generalized weakness, seizures, tremors, tonic-clonic muscle activity, facial pawing and scratching; or hyperexcitability. Historical information of significance includes diet (e.g. all meat high phosphorous diet), current blood tranfusion, medications, toxin exposure (e.g. ethylene glycol), and reproductive history. Underlying disorders to consider include pre or post partum eclampsia, acute or chronic renal failure, urinary obstruction, ingestion of toxins, malabsorption with vitamin D deficiency, hypoparathyroidism, pancreatities, high phosphate diet, alkalosis, and drug induced hypocalcemia (e.g. sodium bicarbonate or loop diuretics).

When severe generalized weakness, tetany or seizures is suspected to be caused by hypocalcemia, a blood sample is for Cai++ levels and 10% calcium gluconate (0.5 – 1.5 ml/kg IV slowly) is administered. Chronic hypocalcemia may not be associated with

acute life-threatening problems allowing the calcium gluconate to be given orally or as a CRI in the IV fluids. Definitive therapy is aimed at the underlying cause.

Hypercalcemia (cai++ > 6.0 mg/dl)

Small elevations in total calcium can be attributed to comparable elevations in serum protein. Hypercalcemia is anticipated in patients with tumor masses, ingested toxins know to affect serum calcium, severe arrhythmias, or acute renal failure of unexplained etiology. Clinical signs of polyuria and polydipsia, unexplained shock, heart failure, oliguria, abdominal pain, constipation, vomiting, restlessness, altered mentation, and tachypnea can be seen. Inciting causes to be ruled out include: cholecalciferol rodenticide intoxication, adrenocortical insufficiency, primary or secondary hyperparathyroidism, neoplasia (eg. lymphoma, perianal adenocarcinoma, or metastatic lesions to bone), chronic thiazide administration, and iatrogenic causes (e.g. excessive calcium supplementation).

Perfusion is assessed and an ECG done. Shock treated with normal saline as the crystalloid and hetastarch, as needed. Any arrhythmias affecting perfusion are treated according to ECG findings. When heart failure is attributed to hypercalcemia, calcium channel blockers can be administered. Rapid lowering of the serum Cai++ can be accomplished by administering sodium bicarbonate or sodium phosphate. This does, however, result in precipitation of calcium into the soft tissues with major organ compromise possible. Avoid this therapeutic intervention if possible.

Hypokalemia, hyomagnesemia and hypophosphatemia can result and should be corrected by adding the appropriate supplement to the IV fluids. It is important to maintain the calcium phosphorous product below 55 to avoid soft tissue calcification. Urine output, central venous rpessure, PCV, TS, ionized calcium, phosphorous, potassium, and magnesium, and blood pressure should be monitored.

It has been demonstrated experimentally in the dog that total serum calcium levels > 16 mg/dl are associated with vasomotor spasms of the afferent glomerular arteriole. This results in poor glomerular and tubular perfusion and renal failure. Serum creatinine and urine output are monitored as a reflection of glomerular blood flow and filtration rate. Volume deficits are quickly restored with IV isotonic saline. Calciuresis is promoted by using furosemide (1 mg/kg/hr CRI). Dopamine (2-3 ug/kg/min) can be considered to offset preglomerular arteriolar spasms.

Other therapies available for patients that do not respond to diuresis include; glucocorticosteroids to decrease intestinal calcium absorption and promote calciuresis; and calcitonin or mithramycin to promote calcium deposition in bone. Mithramycin has potential side-effects of blood dyscrasia and is reserved for patients with non-responsive hypercalcemia associated with cancer. Peritoneal dialysis is an option should these treatments fail or severe renal insufficiency is present. A calcium free dialysate is chosen.

Phosphorous

Phosphorus is an important intracellular ion necessary for the generation of adenosine triphosphate (ATP), the principle intracellular energy source. It is absorbed through the small intestines and excreted by the kidney High concentrations are found in bone. Acidosis causes phosphorus to shift from the cell into the plasma and alkalosis results in movement from the plasma into the cell. Disorders in serum phosphorus can present life-threatening complications and should be assessed in relation to serum cooncenetration of potassium, sodium, magnesium, and calcium.

Hypophosphatemia (po4- < 3.0 mg/dl)

Acute hyophosphatemia rarely causes immediate problems but prolonged deficiency results in ATP energy store depletion. It occurs when a severe catabolic state is rapidly changed to an anabolic state, such as in diabetic keoacidosis, burns, or severe emaciation. Leukemia causes excess utilization of phosphorus. Hyperparathryoidism, sodium bicarbonate therapy, steroid administration, insulin administration, hypomagnesemia, starvation, and recovery from hypothermia are reported causes. Severe respiratory alkalosis can cause transient hyophosphatemia but generally corrects when the pH is normal.

Severe hypophosphatemia (<1.0 mg/dl) has effects related to energy depletion. Red blood cell lysis, impaired white blood cell phagocytic and bacteriocidal capabilities, platelet dysfunction, muscle disease, cardiomyopathy, and central nervous system signs have been reported.

Mild asymptomatic hypophosphatemia can be treated by oral supplementation utilizing skim mild or phyeralimentation fluids. Diarrhea is a common complication. Parenteral administration is indicated for severly affected patients and canbe accomplished by administration of sodium or potassium phosphate (3.0 mmol/ml) at a dosage of 0.01 – 0.03 mmol of phosphate/kg body weight/hr for 3 to 6 hours in a calcium free fluid. Serum phosphorous is then re-evaluated. Hypocalcemia, hypernatremia, hypotension, hyperkalemia, and metastatic calcification are complications.

Hyperphosphatemia (po4- > 6.0 mg/dl)

There are no specific clinical signs to suggest hyperphosphatemia. Historical or clinical findings can reveal acute renal failure, chronic renal failure, urinary obstruction, hypoparathyroidism, massive cell lysis, phosphate enemas, or ingestion of phosphate or vitamin D.

Hypocalcemia occurs due to soft tissue deposition of calcium phosphate. This occurs when the calcium-phosphorus product is greater than 55.

Therapy involves reducing gut phosphate absorption by administering phosphate-binding antacids such as aluminum hydroxide get. Volume expansion with isotonic saline will promote phosphate excretion by the kidneys when renal function is normal. When renal failure is present, dialysis is required.

Lactate

Normal lactate in whole blood, serum or plasma lactate is below 2.0 mmol/L. Elevations in lactate suggest either over production or impaired excretion. Overproduction occurs during tissue hypoxia when anaerobic metabolism is required, such as during extreme exercise (muscle production) or shock (all tissues). Lactate is cleared by the liver.

Lactate values can be a monitor of tissue oxygenation and capillary flow during shock resuscitation. A reduction in blood lactate is a reflection of a reduced need for anaerobic metabolism. When the lactate does not decline with fluid resuscitation, inadequate resuscitation, liver disease and organ ischemia are ruled out as critical complications.

Coagulation parameters

Quick evaluation of the coagulation system can be done at the time of the MDB. A drop of whole blood is placed on a slide and a blood smear made, stained and dried. The number of platelets is counted per a representative oil immersion field. Each platelet seen under oil emersion represents approximately 15,000/cumm of the total count. Even though an acceptable platelet count may be found at presentation, a repeated estimate should be made after resuscitation. A declining trend in platelet numbers may be one of the first suggestions of disseminated intravascular coagulation (DIC). This is to be anticipated in dogs and cats with systemic inflammation.

The coagulation cascade can be evaluated, as well. Bedside commercial units are available to test PT and PTT. An ACT can be done with an ACT blood tube and simple heating block warming the tube to 37 degrees centigrade. A shortened clotting may be an early sign of DIC when coupled with a decline in platelet numbers. Anticoagulant rodenticide toxicity will show a prolongation of PT prior to PTT.

Urinalysis

Urine should be collected prior to fluid resuscitation, when possible, especially for patients with likely infectious or metabolic problems. The ability of the kidneys to concentrate urine is reflected by the specific gravity. Glycosuria without hyperglycemia reflects proximal tubular cell damage, a complication of nephrotoxic drugs or renal hypoxia. Urine sediment is evaluated for casts in animals on nephrotoxic drugs or having experienced severe shock. The newest, freshest casts are renal tubular casts, followed by coarse granular casts, fine granular casts, and then hyaline casts. Renal tubular and coarse casts may appear before significant elevations in BUN and creatinine. Should these be seen, any nephrotoxic drugs are is stopped and renal blood flow is promoted.

Urine output is assessed on an ongoing basis as a reflection of renal function, blood pressure and fluid balance. Normal urine output should be $\geq$ 2-4 mg/kg/hr. In addition, the sediment is monitored for signs of infection, protein, blood, ketones, hemoglobin, bilirubin, or myoglobin.