XXX is an 18 month old black and white Holstein heifer who presented to the XXX on May 16, XXX with obvious signs of dyspnea. As she was escorted from the trailer to a box stall for examination, hospital staff noticed a moderate amount of mucoid sanguineous discharge on her tail at the level of the vulva. The owner was not present, but the transporter had brought a written history which was copied for the medical record and is described below.

HISTORY

The heifer had been purchased at a sale recently and brought to the farm (time frame not reported). She was 7 months pregnant, and appeared normal 12 days ago. Ten days prior to arrival at the hospital the heifer was anorexic and tachypneic. Her rectal temperature was 104 F, and unknown doses of flunixin and tulathromycin were administered. Seven days prior to arrival, she showed no improvement and was treated with tilmicosin and a second dose of flunixin. Five days prior to arrival, the heifer received flunixin and tulathromycin again. Three days prior she looked clinically dehydrated and remained anorexic even after an oral drench of unknown fluid. The day before admission, the oral drench was repeated and there was still no improvement in her appearance or demeanor.

PHYSICAL EXAM

Physical examination of the dyspneic heifer revealed a temperature of 102.1 F, heart rate of 96 beats per minute, and a respiratory rate of 40 breaths per minute. Her lung sounds were absent ventrally, while wheezes and harsh sounds were ausculted dorsally (bilaterally). She stood in the stall with her head and neck extended. Her eyes were sunken with a dull appearance and there was bilateral purulent nasal discharge. Her mucous membranes were pink with a prolonged CRT at 3-4 seconds and extremities were cold. Upon rectal examination, an unresponsive calf was palpated within the pelvic canal. The heifer was estimated at a weight of 500 kg.

The transporter informed us that XXX was of considerable value genetically, and that the owners were willing to make every effort to save her. Because she presented with multiple problems, the admitting veterinarians immediately began to efficiently triage her conditions, so as to stabilize her as quickly as possible.

INITIAL DIAGNOSTICS

The staff worked together to stabilize and make comfortable the dyspneic heifer. After blood was collected for a CBC and plasma biochemical profile, a 14 gauge longterm over-the-wire catheter was placed in the right jugular vein. As the catheter was being placed and sutured, the ultrasound exam began with evaluation of the left lung field revealing pleural effusion and visible fibrin within the fluid. Areas of lung consolidation were present. The heifer was next examined on the right side, and the same results were found. While awaiting bloodwork and the arrival of the theriogenologist, an endoscopic tracheal wash was performed. Present within the upper airway was a moderate amount of sanguinopurulent material that was sterilely aspirated and sent to the lab for cytological and microbiological analysis.

Upon arrival of the theriogenologist, it was decided that XXX should receive 2 liters of 7.2% (hypertonic) saline prior to the vaginal delivery as there is considerable fluid loss during birth and the heifer already appeared to be hypovolemic. A vaginal exam revealed cervical dilation, despite the normal appearance of the vulva. With copious amounts of lube and gentle traction on the obstetrical chains that were placed

proximally to the calf's front fetlocks, a dead heifer calf was extracted. Within minutes, the placenta was also removed. During the delivery, a 20 L carboy of 0.9% NaCl with 20 milliequivalents (mEq) of potassium chloride (KCL)/L was started at 5 L/hour.

The last procedure performed was the bilateral thoracic trochar placement. The optimal sites for fluid drainage were determined ultrasonigraphically, and XXX was then clipped bilaterally at the level of the olecranon and approximately the 6th intercostal spaces. A lidocaine block followed the initial scrub, and finally a sterile prep (betadine and alcohol) preceded the placement of the two 24 french thoracic trochars. Serosanguinous, malodorous fluid was collected from both the right and left chest drains totaling 25 liters. Samples of the drained thoracic fluid were also collected for cytological and microbiologic evaluation, and the chest tubes were then sutured in place. Heimlich one-way valves were placed at the end of each of the chest tubes to allow continuous drainage of thoracic fluid without introducing a pneumothorax to the patient. XXX, now physically looking more comfortable, was allowed to rest. While it would be days before the culture could confirm bacterial growth, the history and gross findings were indicative of a pleural pneumonia caused by *Mannheimia hemolytica*, commonly known as shipping fever.

INTERPRETATION OF DIAGNOSTIC TESTING

As results of the CBC, chemistry profile, and fluid analysis became available, we were able to piece together a strong differential diagnosis for *M. haemolytica*. The CBC revealed a high fibrinogen at 1200mg/dL (200-800), which is a marker of inflammation and supports an ongoing suppurative infection. The WBC count was markedly elevated at 23,600 cells/uL (4,000-12,000) and the segmented neutrophil count was 19,400 or 82%

(15-45%). The bands were 473 or 2% (0-2%) indicating a mild left shift. These results are suggestive a bacterial infection due to the nature of the resultant neutrophilia caused by the *M. haemolytica* bacteria, and will be discussed later.

The blood chemistry revealed several abnormal values that also were consistent with the bacterial infection. Many of the abnormal values, may however, be explained as unrelated to the respiratory disease. It is known that steroid hormones produce a gluconeogenic effect, and likely that the stress hormone cortisol produced a similar response in the heifer resulting in hyperglycemia with a measured glucose at 198 mg/dL (reference range 50 – 79).

Creatine kinase (CK) is classified as a muscle leakage enzyme that increases with cellular damage or ischemia. It is not surprising that this enzyme was elevated at 966 units/liter (U/L) (reference range 50 - 271), as the heifer had received numerous intramuscular injections over the course of the 10 days prior to admission. Tilmicosin in particular, causes severe tissue reactions if not injected properly (SQ only). Although aspartate aminotransferase (AST) is generally considered a hepatocellular leakage enzyme, it is also present within skeletal muscle and is not organ specific. The AST was elevated at 188 U/L (reference range 57 - 108) and could also be attributed to the IM injections.

The serum level of alkaline phosphatase (AP) was increased at 103 U/L (reference range 26 - 78). Alkaline phosphatase is broadly termed a liver enzyme, as it is considered an indicator of cholestasis, or impaired bile flow. AP is not, however, entirely organ specific, as four isoenzymes of AP exist. One of these 4 isoenzymes is largely present in bone (BAP). Young growing animals will show increased total levels of AP as

a result of increases in BAP produced by osteoblastic activity in the bone, likely explaining the elevation seen in this case.

XXX's chemistry panel also revealed a mild azotemia, with elevations in blood urea nitrogen (BUN) at 32 mg/dL (reference range 8 - 22) and creatinine at 1.7 mg/dL (reference range 0.6 - 1.4). Azotemia can be classified as pre-renal, renal or post-renal. These 3 disturbances can be explained by the following generalizations. Pre-renal disease results from a decreased glomerular filtration rate (GFR), renal disease occurs when greater than 75% of the nephrons become non-functional, and post-renal disease is associated with an outflow obstruction. The best indicator for determining the basis for the azotemia is a response to fluid therapy. It is likely that XXX had a pre-renal azotemia, as she appeared to be hypovolemic. As hypovolemia is resolved with fluid therapy, the GFR will increase, resulting in normalization of the BUN and creatinine levels.

The low cholesterol at 39 mg/dL (reference range 112-331) may be of significance, as it can be explained by the presence of inflammatory cytokines which also play a key role in the pathogenesis of the shipping fever disease. These inflammatory cytokines also act on enzymes that convert very low density lipoproteins (VLDL) to low density lipoproteins (LDL) thus reducing their activity. The result is lower lipoprotein numbers and cholesterol content.

Hyponatremia can be explained by two major mechanisms, either retention of free water or excess losses of sodium. The likely explanation for the mild hyponatremia in this case 127 mmol/L (reference range140 - 151) would be explained by the sequestrum of fluid in an inaccessible third space, also known as pleural effusion. Chloride is a

major anion of extracellular fluid and is necessary for the maintenance of osmolality and acid-base balances. Hypochloremia 77mmol/L (100-109) often results concurrent with hyponatremia because changes in free water alter these ions proportionally and can be explained by the third spacing of fluids. Additionally, ruminants who present as anorexic will often have low chloride levels as secretion of HCl continues into the abomasum, but decreased motility yields decreased amounts of HCl passing through to the small intestine for reabsorption. This sets the stage for a metabolic alkalosis, as excess bicarbonate is left in the small intestine for reabsorption that would otherwise be used for acid neutralization.

Hypokalemia 2.7mmol/L (3.7-5.6) has many predisposing factors including decreased intake, increased loss, and transcellular shifting of this tightly regulated cation. Decreased intake or anorexia was consistent with the heifer's history, while potassium losses, like sodium loss, could be explained by the fluid sequestered in the pleural cavity. Furthermore, shifting of potassium from extracellular fluid to intracellular fluid occurs with alkalosis, and based on the elevated TCO2 at 34 (reference range 22 - 29), we can confirm that the heifer is indeed alkalemic.

The mild hypomagnesemia 1.6 (1.8 - 2.9) can be explained as a result of the heifer's anorexia. Magnesium is a major intracellular cation, second only to potassium in amount. In blood, approximately 20-30% of Magnesium is bound to albumin, while approximately 60% is free or bound to phosphates or citrates.

Albumin is a protein that is synthesized in the liver and plays an important role in the colloid oncotic pressure (COP), as it accounts for approximately 80% of the blood COP and helps to maintain fluid within the vascular space. The causes of

hypoalbuminemia are decreased production, increased loss, and physiologic factors, such as overhydration. While we know that XXX was not overhydrated upon presentation, we could surmise that mild changes in her liver would yield insufficient amounts of amino acids available for albumin production; however, the most reasonable explanation for the heifer's hypoalbuminemia 2.2 g/dL (3.2-4.2) is again, the third spacing of fluids. Additionally, increased catabolism of these proteins due to the septic nature of the infection may play a role in the development of the hypoalbuminemia.

Total carbon (TCO2) is a reasonably accurate means of determining bicarbonate (HCO3) levels in the blood. As approximately 95% of the CO2 is transported as bicarbonate, we find that the high TCO2 (34 mmol/L) indicates a metabolic alkalosis. A metabolic alkalosis occurs when there is an excess of HCO3 in the blood, either from excessive loss of hydrogen ions in the kidneys, or by failure of the small intestine to reabsorb hydrochloric acid (HCl) that is secreted in the abomasum. Results from the blood gas analysis support this finding as well.

Interpretation of the blood gas can be done in a relatively systematic manner, with three key factors are needed for analysis: pH, bicarbonate concentration (HCO3) and the rate of alveolar ventilation (pCO2). For the purposes of analysis, it is helpful to understand that HCO3 is an alkaline (basic) buffer, while pCO2 acts as a respiratory acid. Because HCO3 is primarily regulated by metabolic events and kidney function, any disturbances are labeled metabolic in nature. The pCO2 concentration is determined primarily by changes in respiratory function, consequently acid-base disturbances reflected by abnormal pCO2 would be labeled respiratory in nature. The first step in analysis of the blood gas is done by examining the measured pH. XXX's pH was

measured at 7.508 (reference range 7.31 - 7.53) which falls at the high end of normal, leaning toward the side of alkalemia. Because her HCO3 was elevated at 37.5 mmol/L (17 – 29), we can further substantiate the metabolic alkalosis.

The ultrasound exam of the chest was very remarkable. Fluid is ultrasonically anechoic, meaning that it does not reflect back the ultrasound waves. As a result, a dark or black image is formed on the screen. This fact makes the ultrasound very useful in locating pockets of fluid, particularly in the pleural space. The presence of adhesions, fibrin, pleural thickening and irregularity can also be detected with the ultrasound. When XXX was examined, pleural fluid was observed as a black area on the screen, with finger-like debris of increased echogenicity (grey) interpreted as visible fibrin within that fluid (Figure 1). Lung consolidation is a process that occurs whereby the normal, usually collapsible lung tissue becomes filled with inflammatory cellular exudates within the alveoli resulting in solidification of the tissue into a firm dense mass. Examination of normal lung tissue with the ultrasound will reveal only the white lines reflecting the pleural surface, as normal lung tissue is filled with gas which is impenetrable with the instrument. Figure 2 shows not only fluid in the pleural space between the two cross marks, but also reveals evidence of consolidation as the bright white line reflecting off the lung disappears into blackness. Visualization of the lung is possible because it is now filled with infiltrative debris and not just gas. The triangular shape below the bottom cross mark is consolidated lung tip.

During drainage of the pleural fluid, a sample was obtained for analysis. The results of the fluid analysis performed revealed an elevated total protein of 4.8 grams (g)/dL (reference range < 2.5), with WBC present at 1.41 x 10^3 cells/microliter (uL).

Eighty-nine percent (89%) of these cells were neutrophils, many of which were degenerate. The laboratory technician also commented that most of the neutrophils contained short to medium sized rods, and the interpretation was a septic neutrophilic inflammation.

Pleural fluid can be classified as a transudate, modified transudate, or an exudate. Normal pleural fluid in large animals should have a total nucleated cell count (TNCC) less than 5,000 cells/uL with a protein less than 2.5 g/dL. It is possible to differentiate between the types of pleural fluid by comparing these values. A transudate is often a clear fluid with protein levels <2.5 g/dL and cell counts < 30,000 cells/uL. A modified transudate has an increased protein > 3 g/dL but with normal cell counts <30,000 cells/uL. An exudate often appears as a cloudy fluid and will have an elevated protein at > 3 g/dL and also increased cell counts > 30,000 cells/uL. While bacterial pneumonias tend to produce exudative pleural fluid, XXX's cell count was not increased but the protein was elevated, classifying her fluid as a modified transudate. The TNCC often varies with sampling sites, and while the particular classification of this fluid is not of great significance, the presence of rod shaped bacteria in the pleural fluid was of great significance, as it determined antibiotic therapy.

The endoscopic tracheal wash results, while not finalized, also supported a bacterial infection as many nondegenerate neutrophils were seen along with occasional extracellular bacterial rods. Macrophages and inflammatory giant cells were also seen yielding an interpretation of a suppurative, possibly septic infection. While it would be days before the culture of the tracheal wash fluid would be finalized, cultures of both pleural fluid and tracheal wash fluid supported an infection with the gram negative, rod

shaped bacteria, Mannheimia haemolytica.

TREATMENT PLAN

In treatment of an animal with pleural effusion and bronchopneumonia, both medical treatment and nursing care are of importance. After all diagnostics were completed, XXX was allowed to rest in her stall and offered a variety of forages including fresh grass, in an attempt to stimulate her appetite. The plan for the remainder of the first day of hospitalization included the following:

- Physical exam
- 0.9% Sodium Chloride (NaCl) + 20 milliequivalents (mEq) of Potassium Chloride (KCl)/L
- Enrofloxacin 5 mg/kg IV once daily
- Monitor chest tube placement/Heimlich valves q 1 h
- Offer feed / try to get heifer to eat
- Oral electrolyte water free choice
- PCV/TP

The hospital's standard treatment protocol for adult dairy cattle providing broad spectrum antibiotic activity consists of potassium penicillin (22,000 IU/kg IV QID) and ceftiofur (2.2 mg/kg IV BID). Enrofloxacin, however, was recently approved for use in dairy animals under 20 months of age, and was prescribed in place of the penicillin/ceftiofur combination. Enrofloxacin is a bactericidal fluoroquinolone which has good spectrum of activity specifically against gram negative respiratory pathogens.

Flunixin is a non-steroidal anti-inflammatory drug (NSAID) which is recommended for use in cases of pleural pneumonia to help reduce both pain and the production of fluid hopefully resulting in increased feed intake. Flunixin is used at the hospital in 3 different dose regimes. While commonly used for relief of visceral pain, a low dose of 0.25mg/kg given TID was prescribed for 'anti-endotoxic' purposes due to the presence of bacteria in all cytological samples. Flunixin was not administered on the first day of hospitalization because the heifer had received 3 doses within 5 days prior to admission. Because flunixin is implicated as a cause of abomasal ulcers in ruminants, use of this drug is always carefully considered by veterinarians at the hospital.

The fluid selection of 0.9% NaCl was made based on the low chloride and sodium values from the chemistry analysis, and because it is an acidifying solution which helped to correct the metabolic alkalosis. Potassium chloride was added to the fluids in order to correct the hypokalemia. The rate of administration was based on an adult maintenance rate of 50 ml/kg/day. XXX's maintenance rate was 1041 ml/hour (50x500/24), or approximately 1 L/hour. A rate of twice maintenance was used initially to correct the hypovolemia and azotemia present.

The chest tubes were monitored for patency and placement. Should the one-way valves, or the trochar itself become displaced, it is possible to introduce a pneumothorax to the patient, making ventilation extremely difficult. Decision to drain the 25 L of fluid was based primarily on patient comfort and the presumption of bacteria present within the fluid.

DAILY PROGRESSION

Day 2

On May 17, XXXX, the heifer was reported to have eaten little to nothing overnight, but was drinking. Her eyes looked less sunken, but she remained tachypneic

with a heart rate of 96. Rumen motility was weak, with 1 contraction present per minute. Wheezes were still present upon auscultation of the lungs, and the heifer continued to extend head and neck to breathe, especially when recumbent. All other physical exam parameters were within normal limits. Treatment orders remained the same with the addition of the following:

- Flunixin 0.25 mg/kg IV TID
- Transfaunate with 2 gallons from hospital donor and alfalfa meal
- Recheck albumin and electrolytes

Results of the repeated chemistry values revealed the heifer to be normonatremic, normochloremic, and while she was still hypokalemic, her plasma potassium had increased from 2.7 mmol/L to 3.3 mmol/L. The TCO2 remained elevated at 31 mmol/L, while the albumin dropped to 1.9 g/dL from 2.2 g/dL the day before. The most likely explanation of the drop in albumin can be explained by rehydration and the continuous loss of protein into the pleural space. The fluid rate was decreased from 2L/hour to 1 L/hour (maintenance rate).

Transfaunation is a procedure that is done on a regular basis when bovines present to the hospital off feed for various reasons. Our donor cow has a rumen cannula, which when opened, allows space for a large bore Kingman tube to be manually placed down into the fluid layer of the rumen. A hose is attached to the other end, and the desired volume of fluid is obtained via siphoning action. The strained fluid, rich in cellulose digesting bacteria and protozoa is then administered to the sick patient with the use of a frick speculum and stomach tube.

Day 3

On May 18, XXXX, physical examination of the heifer revealed a temperature of 102.4 F, heart rate 100 beats/minute, and respiratory rate 36 breaths/minute. She looked brighter, was more active and appeared to show more interest in her feed. Her rumen motility was evaluated at 2 contractions per minute. Auscultation of the lungs revealed breath sounds on both sides of the thorax, with less intensity in specific areas and ventrally. A repeat ultrasound exam revealed less than 2 centimeters (cm) of fluid and areas of lung consolidation present on the left side of the thorax. Examination of the right side revealed approximately 3.8 cm of fluid that appeared fibrinous, loculated and caudal to the chest tube. The ultrasound exam confirmed that the chest tubes were not draining fluid and were removed. All orders remained the same, and XXX was taken outside to the hospital's paddock for exercise and to encourage grazing. Overnight, the jugular catheter was not flowing well and was replaced with another 14-gauge long term over-the-wire catheter in the right jugular vein.

Day 4

On May 19, XXXX the heifer appeared clinically the same as on day 3. Her respiratory rate was increased at 52 breaths/minute, with wheezes heard bilaterally upon auscultation and intermittent coughing present. An ultrasound exam was repeated which showed that a large amount of fluid had reaccumulated within the thorax. A second trocharization was performed, draining 4 liters of serosanguinous fluid from the right side only. The chest tube was pulled immediately and the incision closed with a purse string suture pattern. That afternoon, the heifer remained bright with episodes of coughing, but continued to eat and chew her cud.

Day 5

On Tuesday, May 20, XXXX culture results confirmed growth of both *Pasteurella species*, and *Mannheimia haemolytica* in both the tracheal wash fluid and the thoracic fluid. Growth was moderate in the tracheal fluid and heavy in the thoracic fluid. The susceptibility testing performed revealed that the organisms were sensitive to enrofloxacin; consequently the antibiotic therapy was unchanged. Clinically, the heifer remained the same, with tachycardia still present, and periods of variability in her appetite and comfort level. Her respiratory rates were variable, with rates averaging in the mid 40's. Thoracic radiographs were taken and revealed normal looking lung lobes dorsally. The presence of a fluid line due to pleural effusion caused border effacement of the heart and lungs whereby clear margins of these two structures were lost.

XXX broke with diarrhea later in the day. The diarrhea became profuse and watery, but was considered related to the antibiotics, or stress due to infection. Her temperature increased to 103.6 F and was attributed to recrudescence of the bacteria. The IV fluids were discontinued, but the heifer remained on her course of antibiotic and NSAID therapy.

Days 6 – 16

XXX's condition appeared to wax and wane with the reaccumulation of fluid within the pleural space, but it became evident that overall, she was improving. The diarrhea and fever that had started on the 5th day of her hospitalization resolved within 4 days. Her stay was finalized on 5/31/XX, when she was discharged to her owner. During her 2 week stay at the hospital, the heifer was trocharized 4 times (5/16/XX, 5/19/XX, 5/23/XX, and 5/28/XX) removing a total of 41 liters of pleural fluid that continued to accumulate within the thorax. As the hospital is a temperature controlled environment remaining at approximately 65 F year-round, it was made clear to the owner upon discharge that the heat stress of the upcoming summer months could certainly bring about complications and or death, and long-term prognosis could not be determined. XXX was discharged with orders to continue the antibiotic therapy with a prescription of 5 grams of enrofloxacin to be given SQ every 3 days, for a minimum of 30 days.

DISCUSSION

Mannheimia haemolytica, the most common cause of shipping fever pneumonia, colonizes the nasopharynx of normal cattle. It is a Gram negative, rod-shaped opportunistic pathogen that will infect the lower respiratory tract to cause a severe fibrinous pleuropneumonia during times of stress, adverse climatic conditions, and respiratory viral infection.

As a brief review of immunologic function, we know that T-cells (and more specifically T-helper cells) are a type of lymphocyte produced by the Thymus gland. These T-helper cells produce cytokines, which are soluble products that coordinate and regulate a specific immune response. Interleukin-8 (IL-8) is one such cytokine whose function is to cause neutrophil infiltration to the area. During an infection, the *Mannheimia* bacteria release a leukotoxin that up regulates IL-8. As the neutrophils are rapidly recruited to the lung as a result of the increased levels of IL-8, they phagocytize and kill the bacteria using oxygen radicals, antimicrobial peptides and proteolytic enzymes. The presence of neutrophils in the alveoli and bronchioles act as a physical barrier to prevent ventilation of the alveoli, while the secretion of proteases and oxygen radicals often cause pulmonary injury.

Reviewing the anatomy of the bovine lung, we find that there are 3 bronchial

stems emanating from the distal portion of the trachea leading to the right lung, the left lung, and the cranial lobe of the right lung. As the bronchioles continue to branch off, they terminate ultimately at the alveoli, or the true respiratory structures in which gas exchange occurs. The capillaries surrounding each alveolus exchange oxygen and carbon dioxide by the means of diffusion through a series of membranes that are both respiratory and vascular in nature. Also involved are two pulmonary lymphatic systems that function to drain the pleural tissues. Each of the 4-5 lung lobes in cattle is lined by the visceral pleural membrane, while the inside of the thoracic cavity is lined by the parietal pleural membrane. Pleural effusion occurs when excessive fluid accumulates in the area inside the pleural cavity, also known as a third space. Excessive amounts of such fluid can impair breathing by limiting the expansion of the lungs during inhalation.

XXX was diagnosed with pleuropneumonia as a result of infection with *Pasteurella* and *Mannheimia* bacteria as confirmed by culture of the thoracic fluid and tracheal wash fluid. Stages of bacterial pleuropneumonia begin with an exudative phase whereby a rapid release of sterile pleural fluid into the pleural space occurs as a response to pleural inflammation. Next is a fibropurulent stage resulting from bacterial invasion. This stage is characterized by a pleural fluid high in neutrophils and other cellular debris, fibrin deposition on the pleural surfaces, and loculation of the fluid which prevents the spread of infection, but makes drainage difficult. Third is the organization phase whereby fibroblasts grow into the exudate to cause consolidation and render the tissue functionless.

XXX's presentation of the disease was typical of an infection with *M*. *haemolytica*, as her CBC showed the characteristic neutrophilia present with an infection

of this type. Not typical of ruminants, however, was the fact that the pleural fluid was able to be drained multiple times over the course of her hospitalization. Drainage in ruminants is not always effective due to the propensity of the bovine to form fibrin and loculate the fluid. The fact that we were able to keep XXX comfortable with excellent nursing care, appropriate antibiotic therapy, NSAID management of endotoxemia, and repeated trocharization attributed to the favorable outcome of this case. Subsequent conversation with the farmers who are taking care of XXX as recent as September XX, XXXX, reveal that she is doing well at home and has been bred back, but not yet confirmed pregnant.

TEST	RESULT	RESULT	RESULT	UNITS	REF. RANGE
Date	5/16/08				
Specimen Appearance	Normal				-
Plasma Appearance	Normal				-
Total Plasma Protein	8.0			g/dL	6-8
Fibrinogen	1200 H			mg/dL	200-600
RBC	6.77			X 10^6/ uL	5-10
Hemoglobin	11.7			g/dL	8-15
RBC Hemoglobin	12.0			g/dL	8-15
Hematocrit	28			%	24-46
Packed Cell Volume	35			%	24-46
MCV	41.4			fL	40-60
МСН	17.3 H			Pg	11-17
MCHC	41.7 H			g/dL	30-36
RDW	21.2			%	
Platelet	433			X 10^3/uL	200-800
MPV	6.3			fL	-
Platelet Estimate	Appears WRI			-	-
Acanthocytes	1+			-	-
Comments	Approximately 2	2-4 polychromato	philic cells noted p	ber HPF	
WDC	22.62.11			- V 1042/11	-
WDC SEC	23.03 П 10.27 Ц (820/.)			X 10 ⁻⁵ /uL	4-12
DANDS	$19.37 \Pi (82\%)$			X 10^3/uL	.0-4
	$0.437 \Pi (2\%)$			X 10^3/uL	012
	5.072(15%)			X 10 ⁻⁵ /uL	2.3-7.3
MONU	0.475(2%)			X 10^3/uL	.02584
EUS	0.236 (1%)			X 10^3/uL	0-2
BASU	-			X 10^3/uL	-
Dohle Bodies				-	-
Comments	basophilic cytop	ononuclear cells se lasm.	een on scanning ha	ive multiple nuc	cleoli and

Appendix 1 : Complete Blood Count

TEST	RESULT	RESULT	RESULT	UNITS	REFERENCE
					RANGE
Date	5/16/08	5/17/08			
Sodium	127.0 L	143.0		mmol/L	140-151
Potassium	2.7 L	3.3 L		mmol/L	3.7-5.6
Chloride	77 L	100		mmol/L	100-109
TCO2	34 H	31 H		mmol/L	22-29
Anion Gap	19	15			-
Calcium	8.8			mg/dL	7.9-10.5
Phosphorus	5.1			mg/dL	4.4-9.2
Magnesium	1.6 L			mg/dL	1.8-2.9
Glucose	198 H			mg/dL	50-79
Urea Nitrogen	32 H			mg/dL	8-22
Creatinine	1.7 H	1.0		mg/dL	.6-1.4
Total Protein	8.4			g/dL	6.3-8.5
Albumin	2.2 L	1.9 L		g/dL	3.2-4.3
Globulin	6.2			g/dL	-
Alkaline				U/L	26-78
Phosphatase	103 H				
Creatine Kinase	966 H			U/L	50-271
AST	188 H			U/L	57-108
Gamma GT	23			U/L	12-30
Cholesterol	39 L			mg/dL	112-331
Total Bilirubin	1.6 H			mg/dL	.14

Appendix 3 : Arterial Blood Gas Analysis

TEST	RESULT -	RESULT-	RESULT-	UNITS	REFERENCE
	CORRECTED	CORRECTED	CORRECTED		RANGE
Date	5/16/08				
pН	7.508				-
pCO2	47.5			mmHg	-
pO2	77.2			mmHg	-
SO2 %	94.1			%	-
Hct	38			%	-
Hb	12.6			g/dL	-
BEecf	14.7			mmol/L	-
BEb	14.0			mmol/L	-
SBC	37.8			mmol/L	-
HCO3	37.5			mmol/L	-
TCO2	38.8			mmol/L	-
А	84.8			mmHg	-
A-a DO2	7.6			mmHg	-
P50	23.0			mmHg	-
O2Cap	17.6			mL/dL	-
O2Ct	16.7			mg/dL	-

Appendix 4	:	Thoracic	Fluid	Analysis
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DATE	TEST	RESULT	UNITS	REFERENCE RANGE	
5/16/XX	Color	Amber		-	
	Appearance	Cloudy		-	
	Specific Gravity	1.029			
	Total Protein	4.8	g/dL	-	
	RBC	0.02 x 10^6	Cells/uL	-	
	TNCC	1.41 x 10^6	Cells/uL	-	
	Neutropils	89	%	-	
	Lymphocytes	10	%	-	
	Macrophages	1	%	-	
	Macrophages 1 % - Thoracic Fluid Description: - - The majority of cells are neutrophils, many of which are degenerate. Most of the neutrophils contain bacterial rods. The rods are short to medium sized rods. Some are found extracellularly. - Interpretation: Neutrophilic inflammation, septic -				

Appendix 5 : Tracheal Wash Fluid

DATE	TEST	RESULT	UNITS	REFERENCE RANGE		
11/10/XX	Color	Blood Tinged		-		
	Appearance	Clear with		-		
		clumps				
	Tracheal Wash Description: Specimen is moderately cellular and contains					
	abundant mucus. The majority of cells are nondegenerate neutrophils. Low					
	numbers of macrophages and inflammatory giant cells are present. There are					
	occasional bacterial rods found extracellularly. One group of yeast and bacteria					
	is seen, and these organisms are likely contaminants.					
	Interpretation:					
	Suppurative inflammation, possibly septic					



Figure 1 : Ultrasound image of fibrin within the pleural fluid of the lung

Figure 2 : Ultrasound image of pleural fluid and consolidated lung



References:

- 1. Young, K. Fluid, Electrolyte and Acid Base Balance. Lecture Notes, 2007. pp. 1-20
- 2. Thrall, Mary. Veterinary Hematology and Clinical Chemistry. Blackwell, 2006. pp. 345-353
- 3. Cunningham, James. Textbook of Veterinary Physiology, 4th edition. Saunders, 2007. various pages
- 4. Duncan, Robert, et.al. Veterinary Laboratory Medicine 3rd Edition. Iowa State Press, 1994. pp.94-110
- 5. Smith, Bradford. Large Animal Internal Medicine, 3rd Edition. Mosby, 2002. pp.589-590, 501-503, 551-

570 and various other pages.