Diagnostic Update

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In this Issue:

An Unusual Cause of Nasal Inflammation in a Cat

By Betsy O'Neil, Veterinary Clinical Pathology Resident and Kimberly Ho, Veterinary Intern

Clinicians and clinical pathologists at the Atlantic Veterinary College recently dealt with an unusual case. A strictly indoor 5-year-old female spayed domestic shorthaired cat presented with a non-ulcerated raised haired lesion on the nasal planum of 1.5 months duration. One week prior to presentation, increasing respiratory noise and effort became evident. The mass was slowly growing and did not appear painful. It was localized to the soft tissue nasal structures and did not have bony involvement, but it partly occluded the left nasal passageway. The cat had a good appetite, was drinking normally and was mildly overweight. A CBC and urinalysis were unremarkable and a mild hyperglobulinemia was the only abnormality on a serum biochemical profile.

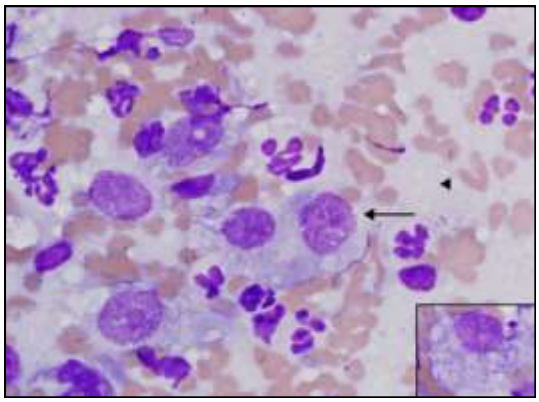

Figure 1: Non-staining *Mycobacteria* spp. noted within macrophages (arrow) and in the background (arrowhead). Wright-Giemsa, x 100 objective. Inset: Macrophage x 100 objective.

Microscopic examination of Wright-Giemsa stained smears of a fine needle aspirate of the mass showed variable numbers of both nucleated cells and erythrocytes on a non-staining to dark blue background containing large numbers of small non-staining rod-shaped slightly refractile structures. The nucleated cells consisted primarily of macrophages and neutrophils. Approximately 60% of the macrophages contained non-staining rod-shaped structures with similar morphology to those seen in the background (Figure 1).

General considerations for this cytological picture included a foreign body reaction or infection with *Mycobacterium* species (spp.). The refractile nature of the structures raised suspicion for a crystalline foreign body particle but the non-staining nature left *Mycobacterium* spp. highest on the list. Positive acid-fast staining confirmed that the structures were *Mycobacterium* spp. with a beaded morphology (Figure 2). Due to financial constraints, the owners opted not to identify the mycobacterial species.

Mycobacterium species are aerobic, non-spore-forming non-motile gram positive acid-fast positive bacteria. Cats can present with localized skin disease or systemic signs related to the gastrointestinal or respiratory tract. Mycobacterial culture is the reference standard for diagnosis and species are divided into slow growing, non-growing and rapid growing mycobacteria.

The slow growing group includes *M. tuberculosis*, *M. bovis* and *M. microti* which are tubercle-producing bacteria. *Mycobacterium avium*, a member of the *M. avium-intracellulare* complex (MAC), is a slow-growing saprophyte also included in this group; it is a non-tuberculous bacterial species. Large numbers of bacteria are typically seen with MAC on cytological examination but this is not routinely seen with the tuberculosis causing agents. These organisms often have a beaded appearance. These species have potential zoonotic risk but reverse zoonoses can also occur. It is this group of bacteria that are noted to produce systemic disease in patients.

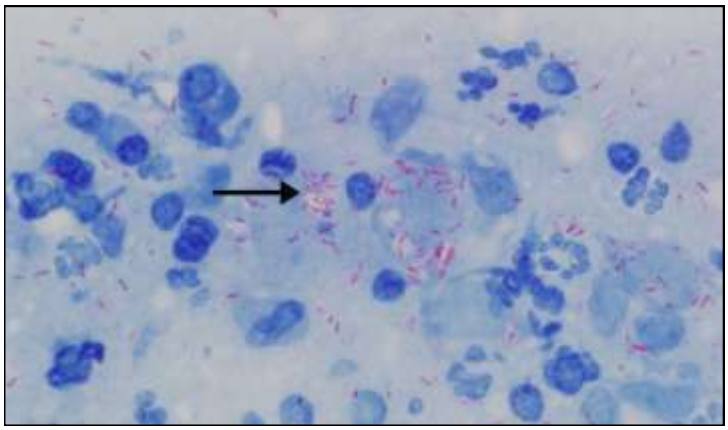

Figure 2: Acid-fast *Mycobacteria* spp. (arrow). Ziehl-Neelsen, x 100 objective.

The non-growing group cannot be cultured using standard methods; they cause a condition termed feline leprosy. On cytological examination, there are usually large numbers of bacteria observed. If immune status is compromised, the predominant cell population typically consists of foamy macrophages with the absence of lymphocytes and plasma cells. The causative agent is reported to be *M. lepraemurium* but studies suggest a larger number of different organisms are involved. Feline leprosy is thought to occur in cats following bite injuries from infected rodents. Lesions are most common on the head, limbs and trunk. Regional lymphadenopathy may occur but systemic disease is rare. Although culture should still be attempted to rule out other species, polymerase chain reaction (PCR) testing is required for diagnosis.

The rapidly growing group of *Mycobacterium* spp. are ubiquitous organisms in the environment. A breakdown in the natural defenses allows them to gain entry and produce disease in animals. These species have a predilection for replication in tissues rich in lipids. Therefore, obese cats are considered to be at greater risk following contamination of penetrating wounds. The inguinal area is a common location for disease to occur. On cytological examination, the bacteria may have a beaded appearance. Speckled structures or non-staining ghosts may be observed but are much harder to appreciate than those noted in other groups. This group has rarely been reported to cause systemic disease.

Treatment and success of treatment is variable. Both depend on the species of bacteria and the extent and severity of the disease. Treatment may include surgery and long-term antibiotic administration. Euthanasia is often recommended because of the potential zoonotic risk with those species in the slow growing group.

Cytological examination was rewarding in this case and acid-fast staining confirmed the diagnosis of mycobacteriosis. Even if organisms are not observed, mycobacterial infections should still be considered in lesions with histiocytic-neutrophilic inflammation, especially in non-healing wounds. Handling of tissue suspect for tuberculosis should follow strict biosecurity guidelines. If there is suspicion of a mycobacterial infection, it is vital to inform the laboratory so special procedures for specimen processing are adhered to. Submission to a specialized laboratory is often required. The following is a summary of recent submission recommendations for biopsied tissue if mycobacteriosis is suspected (Gunn-Moore et al., 2010):

1. Cut the biopsy sample(s) into three or four pieces. Place one in formalin for routine histopathological examination and Ziehl-Neelsen (stain for acid-fast bacteria) staining. One or two samples can be placed in a sterile container and frozen. If acid-fast organisms are present, one of the frozen samples can then be sent for a special culture.
2. If other infections are suspected, a fourth sample that has been placed on saline soaked gauze can be submitted unfixed for routine bacterial/fungal culture.

Although PCR cannot identify all species, it is preferred over culture for rapid turn-around time and avoiding potential zoonotic risk.

References:

1. Gunn-Moore D, Dean R, Shaw S. Mycobacterial infections in cats and dogs. *In Practice.* 2010;32:444-452.
2. Greene CE, ed. Mycobacterial Infections. In: *Infectious Diseases of the Dog and Cat.* 3rd ed. Saunders Elsevier, St. Louis, MO. 2006:462-488.

Dilated Cardiomyopathy in the Juvenile Toy Manchester Terrier

By Carolyn Legge, Veterinary Anatomic Pathology Resident

In most dogs, dilated cardiomyopathy (DCM) is a progressive disease causing gradual cardiac decompensation; this eventually leads to death due to congestive heart failure or fatal arrhythmias. It occurs most commonly in giant and large breed dogs and typically has an adult onset. Dilated cardiomyopathy is reported much less frequently in young dogs, and may occur as a familial disease or as a result of canine parvovirus myocarditis.

Recently, a rapidly progressive form of DCM has been recognized in juvenile Toy Manchester Terriers (TMTs). The clinical signs and lesions associated with this disease process are currently being investigated. To date, we have examined heart tissue and reviewed post-mortem reports, including the available clinical historical information, provided from necropsies performed on 12 TMTs. The ages of the affected animals ranged from approximately 10 - 40 weeks, with males and females represented in approximately equal numbers (7 males and 5 females). Interestingly, most of the affected males were cryptorchid (5 of 7).

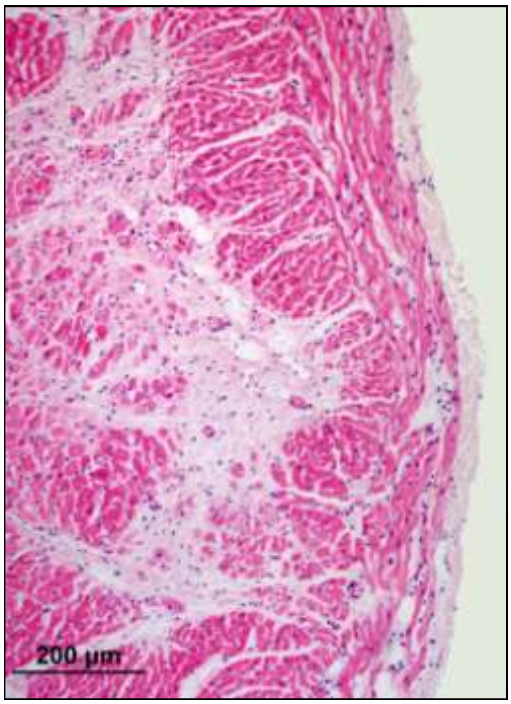

Figure 1: Right ventricular myocardium from a 40-week-old female TMT with DCM. The pale areas represent extensive myofiber loss and replacement with fibrous stroma. H&E.

Our investigation revealed similar histories for most affected puppies; they typically included sudden death without premonitory signs or a brief history (hours) of listlessness and/or respiratory distress followed by death. Therefore, DCM in affected TMTs may go undetected prior to death, similar to that reported in the Doberman Pinscher with occult myocardial disease. Collapse and respiratory distress may be related to tissue hypoperfusion, pulmonary edema and hypoxemia associated with decreased cardiac output as well as profound systemic blood pressure changes resulting from acute heart failure or cardiac arrhythmias. In 5 of the 12 TMTs, terminal events occurred during or following anesthesia/surgery or light exercise. These findings suggest that these events may trigger arrhythmias and potential sudden death in susceptible animals. Among male TMTs, the association of cryptorchism with DCM could relate to the longer duration of anesthesia required for cryptorchid surgical sterilization (allowing greater myocardial depression) or could reflect a genetic association between cryptorchidism and cardiomyopathy.

The gross cardiac changes ranged from no obvious change to moderate cardiac dilation. Microscopically, the changes were striking and consisted of myofiber attenuation, loss and replacement by fibrous connective tissue (Figure 1). Less prominent features included foci of acute myocardial degeneration, necrosis (with or without mineralization) and mild to moderate inflammation. The lesions appeared to be most severe in the right ventricle when compared to the left ventricle and interventricular septum. Because parvovirus is a known cause of DCM in puppies, all heart sections were tested for canine parvovirus antigen using immunohistochemistry; all samples tested negative.

Overall, this work describes a previously unrecognized form of juvenile onset DCM in TMTs. The fact that most of these animals appeared healthy prior to death suggests an occult form of disease in this breed. With the hearts from affected animals having minimal to mild gross changes, submission of heart tissue for the histological identification of lesions is essential for diagnosis. Cardiac lesions may interfere with proper electrical conduction through the myocardium leading to severe arrhythmias. So far, affected TMTs have not been evaluated for evidence of arrhythmias or electrical disturbances. Therefore, electrocardiographic and/or echocardiographic evaluation of juvenile TMTs may help to detect conduction abnormalities or asymptomatic cardiac dysfunction in normal appearing animals. The presence of DCM in this breed, the young age of onset and the similarity of histological lesions are suggestive of a heritable condition. However, pedigree analysis is necessary to determine if it is hereditary and if so, to investigate the mode of inheritance. Currently,

pedigree analysis is underway and we are collecting frozen tissue from affected puppies at necropsy for future genetic testing. Interest and cooperation by owners, breeders and veterinarians is essential to expanding our knowledge about this disease. If you wish to learn more about our research project, if you would like to discuss clinical evaluation of TMT patients, or if you wish to submit deceased dogs for post-mortem examination, we encourage you to contact our research group directly: Dr. Shannon Martinson (smartinson@upei.ca), Dr. Etienne Côté (ecote@upei.ca) or Dr. Carolyn Legge (clegge@upei.ca).

Shipping Glass Slides – Getting Them to the Laboratory in 1 Piece!

By Shelley Burton, Veterinary Clinical Pathologist

Have you ever been contacted by our laboratory with the disappointing news that a blood smear or fine needle aspirate from one of your patients has been damaged in transit and cannot be evaluated? Here are a few handy tips to protect slides:

1. Use rectangular styrofoam (Figure 1) or plastic (Figure 2) containers. Avoid cardboard holders (Figure 3), as they do not protect slides well.
2. Tape the end of the container shut before shipping.
3. Do not refrigerate or freeze the slides or ship adjacent to cold packs. When they warm, condensation can affect cell morphology.
4. Avoid exposure of the slides to formalin fumes as this can affect staining quality. If a container with formalin is going in the same shipment, be sure to protect it well from breakage and seal it in a ziplock bag.

Figure 1: Styrofoam slide holder container.

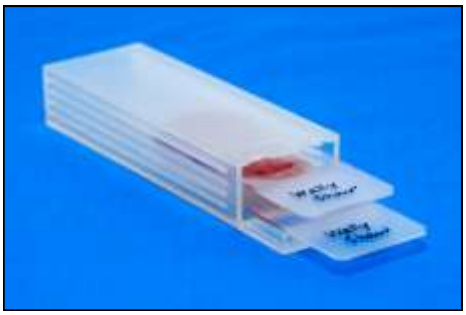

Figure 2: Plastic slide holder container.

Figure 3: Cardboard slide holders should be avoided.

Interpreting Serum Protein Electrophoresis Results

By Noel Clancey, Veterinary Clinical Pathologist

Serum protein electrophoresis (SPE) is a laboratory test for determining serum protein fractions. These separate into bands based on their ability to migrate through agarose gel or cellulose acetate when an electrical current is applied. The number of bands produced can range from 5 to 15 depending on the material used, the species and the individual. These bands may reflect a single protein or a collection of proteins that have migrated to the same point. The distance each protein migrates is defined by its electrical charge, shape and size. For example, albumin tends to migrate farthest toward the positively charged terminal (anode) because it is very anionic and small. Most immunoglobulins are large and cationic, thus they migrate toward the negatively charged terminal (cathode) or do not migrate at all.

Protein bands that represent globulins are defined into electrophoretic regions. In dogs, cats and horses, these regions typically include alpha-1 (α_1), alpha-2 (α_2), beta-1 (β_1), beta-2 (β_2) and gamma (γ) regions. Cattle tend to have only three observable regions; α, β and γ. A densitometer is used to scan the stained gel or strip to result in an electrophoretogram pattern.

Protein electrophoresis can be performed using plasma but serum is preferred since it lacks fibrinogen. When plasma is used, fibrinogen migrates to the β-γ region which can interfere with interpretation. Evaluating the results of a SPE is useful for characterizing hypoproteinemias, differentiating causes of hyperproteinemia and providing a more accurate estimation of albumin concentration when globulins interfere with

other methods for determining albumin. However, SPE is most commonly used to determine if a monoclonal versus a polyclonal pattern exists in patients with a gammopathy. These patients have a hyperproteinemia characterized by a hyperglobulinemia and a low albumin:globulin (A:G) ratio. The albumin concentration can vary but it is frequently low.

When many clones of B-lymphocytes and plasma cells increase synthesis of a variety of immunoglobulins in response to antigenic stimulation, a polyclonal gammopathy is produced. The resulting SPE tracing has a broad-based, single or multi-peaked

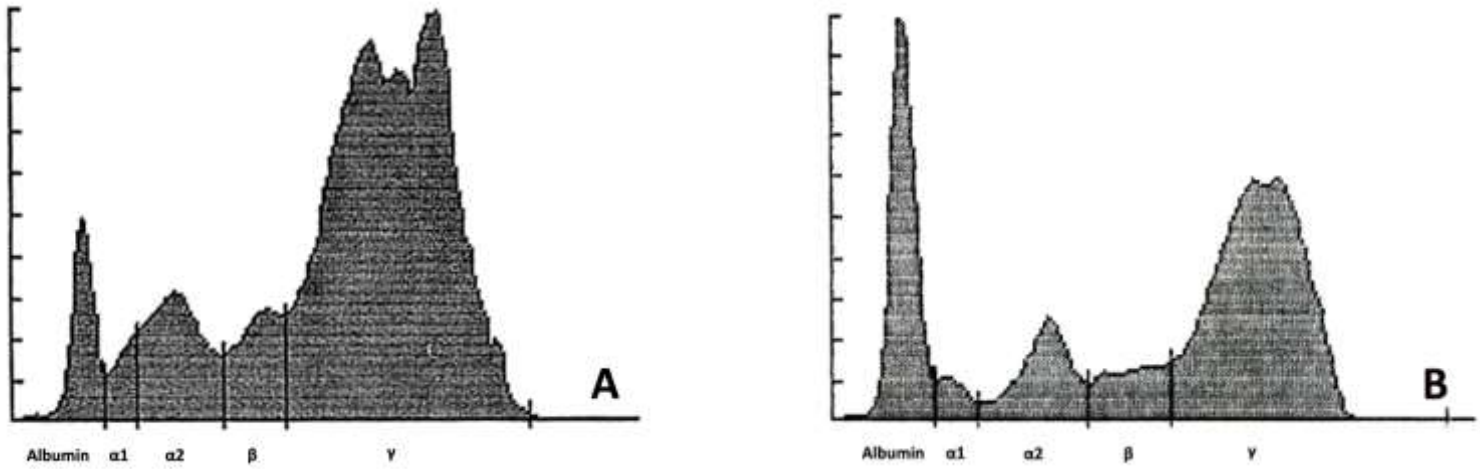

Figure 1: Polyclonal gammopathy with a multi-peak (A) or single smooth peak (B).

pattern located in the γ-globulin region which sometimes overlaps with the β region (Figure 1). This occurs most logically with infectious disease but immune-mediated disease, non-infectious inflammatory disease and necrosis can produce similar electrophoretic results. Monoclonal gammopathies usually result in a single, large, narrow-based peak in the γ region or less often in the β region (Figure 2). These are most often due to paraprotein production by a single clone of B-lymphocytes or plasma cells, usually due to a B-lymphoid or plasma cell tumor (multiple myeloma). However, similar results can infrequently be produced by inflammatory disease. In these situations, a polyclonal gammopathy is truly present but the proteins have migrated to a restricted area, appearing as a monoclonal gammopathy. These are referred to as oligoclonal gammopathies. Therefore, when a monoclonal gammopathy is seen, other features which support B cell or plasma cell neoplasia need to be sought. These include punctate lytic bone lesions on radiographs, a B cell or plasma cell tumor population identified cytologically or histologically and a monoclonal peak on urine electrophoresis (Bence-Jones proteinuria) if proteinuria is present. While it is theoretically possible that any inflammatory disease can result in an oligoclonal gammopathy, these are most commonly reported in canine monocytic

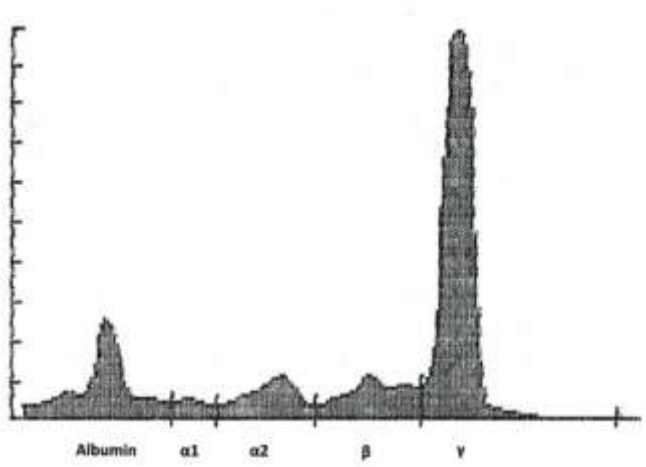

Figure 2: Monoclonal gammopathy.

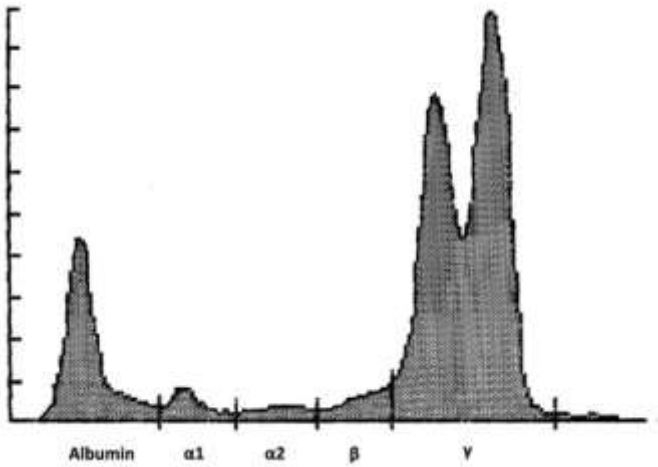

Figure 3: Bi-clonal gammopathy.

ehrlichiosis, canine visceral leishmaniasis, feline infectious peritonitis and feline lymphoplasmacytic stomatitis. Recently, heartworm infection in a dog resulting in a monoclonal gammopathy has also been reported.

While most electrophoretic tracings fit with a polyclonal or monoclonal pattern, rare cases of biclonal patterns are seen. This pattern has two narrow monoclonal peaks in the γ region or sometimes can also involve the β region (Figure 3). A biclonal gammopathy is usually associated with hematopoietic neoplasms, such as multiple myeloma, extramedullary plasmacytoma and other lymphoproliferative disorders. The biclonal term applies to the electrophoretic pattern and can indicate either an expansion of 2 cell clones or a single clone with different dimerization patterns, each of which migrate to different locations. The biclonal pattern may also occur from production of two different classes of immunoglobulins, usually IgG and IgA, by two separate B-cell clones. As with a monoclonal gammopathy, further support for a possible B cell or plasma cell tumor should be sought if this rare double-peaked pattern is seen.

Peaks can be present in the α or β region which represent acute phase proteins. These increase in concentration due to production by hepatocytes in response to select cytokines produced during inflammatory conditions.

References:

1. Ramaiah SK, Seguin MA, Carwile HF, Raskin RE. Biclonal gammopathy associated with immunoglobulin A in a dog with multiple myeloma. *Vet Clin Pathol*. 2002;31:83-89.
2. Stockham SL, Scott MA. Proteins. In: *Fundamentals of Veterinary Clinical Pathology*. 2nd ed. Blackwell Publishing, Ames, IA. 2008:369-413.
3. Antognoni MT, Birettoni F, Miglio A, Lalli P, Porciello F, Mangili Pecci V. Monoclonal gammopathy associated with multiple myeloma and visceral leishmaniasis in the dog: a comparison of two cases. *Vet Res Commun*. 2010;34 (Suppl 1):S97-101.
4. de Caprariis D, Sasanelli M, Paradies P, Otranto D, Lia R. Monoclonal gammopathy associated with heartworm disease in a dog. *J Am Anim Hosp Assoc*. 2009;45:296-300.

Nucleated Erythrocytes on Blood Smears: What Do They Mean?

By Cora Gilroy, Veterinary Clinical Pathologist

Metarubricytosis or rubricytosis is the term used to denote the presence of nucleated red blood cells (nRBCs) in peripheral blood. Typically metarubricytes account for the majority of the nRBCs, with fewer rubricytes and rare earlier erythroid precursor cells noted. An occasional nRBC (< 1-2 per 100 white blood cells) can be noted in the peripheral blood from any patient. When higher numbers of nRBCs are observed, it needs to be determined if their presence is appropriate or inappropriate.

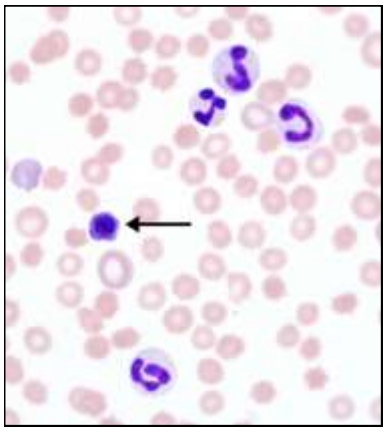

Figure 1: Nucleated RBC (arrow) in a canine blood smear. Wright-Giemsa, x 100 objective.

Appropriate metarubricytosis is noted in conjunction with a regenerative anemia during a period of increased erythropoiesis. This is the most common cause of metarubricytosis in domestic veterinary species. It is, however, important to note that the presence of the nRBCs is not considered part of the regenerative response and is only an accompanying feature in some cases. Only increased reticulocyte numbers allow an anemia to be classified as regenerative.

Inappropriate metarubricytosis occurs when nRBCs are noted without reticulocytosis, as can be seen in a non-regenerative anemia or in a patient that is not anemic. One of the more important causes is damage to the bone marrow, allowing disruption of the controlled release of nRBCs. Bone marrow damage can occur with necrosis, inflammation, endotoxemia, hypoxia, myelodysplasia or neoplasia (hemic or non-hemic). Other conditions that have been associated with inappropriate metarubricytosis include extramedullary hematopoiesis, splenic contraction, splenectomy, altered splenic function, lead poisoning in dogs, increased concentrations of corticosteroids and trauma. An uncommon cause is inherited dyserythropoiesis, such as that seen in Poodles with familial macrocytosis and dyshematopoiesis.

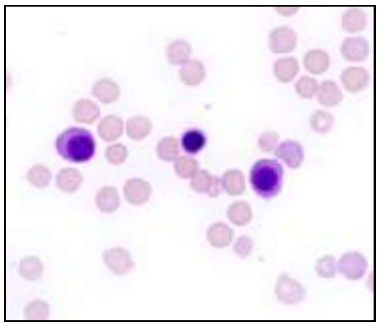

Figure 2: Feline blood smear containing several nucleated RBCs. Wright-Giemsa, x 100 objective.

If the metarubricytosis is appropriate, the nRBCs should not persist and typically are not seen once the anemia resolves. With an inappropriate metarubricytosis, the presence of the nRBCs is a non-specific indicator to prompt further investig-

ation as to why these cells are present. Occasionally the presence of nRBCs may be of use clinically; a recent article (Aroch et al., 2009) reported that monitoring for peripheral nucleated RBCs can be used as a prognostic indicator in dogs with heatstroke.

If nRBCs are present in the blood, it is important to remember that these cells are typically included in the total WBC count performed by many automated hematology analyzers. When they are included, it is important to calculate a corrected WBC count which excludes the nRBCs, as this can impact on the final interpretation of the leukogram results. The nRBCs should be counted separately as the 100 leukocyte differential is performed. The following WBC correction formula is used:

$$\text{Corrected [WBC]} = \text{measured [WBC]} \times \frac{100}{100 + \#\text{nRBCs per 100 WBCs}}$$

Sample Calculation:

Total WBC count = 18 x 10^9/L

25 nRBCs counted per 100 WBCs counted

$$\text{Corrected [WBC]} = 18 \times 10^9/\text{L} \times \frac{100}{100 + 25}$$

$$\text{Corrected [WBC]} = 14.4 \times 10^9/\text{L}$$

The corrected WBC count is then used for calculating the concentrations of different leukocytes in the absolute differential count. As the presence of nRBCs can prompt further disease investigation and can interfere with WBC counts, it is important to always note their presence by microscopically evaluating all blood smears.

References:

1. Aroch I, Segev G, Loeb E, Bruchim Y. Peripheral nucleated red blood cells as a prognostic indicator in heatstroke in dogs. *J Vet Intern Med.* 2009;23:544-551.
2. Meyer D, Harvey J, eds. Evaluation of erythrocyte disorders. In: *Veterinary laboratory Medicine: Interpretation and Diagnosis.* 3rd ed. Elsevier, St. Louis, MO. 2004:47-81.
3. Stockham S, Scott M. *Fundamentals of Veterinary Clinical Pathology*. 2nd ed. Blackwell Publishing, Ames, IA. 2008:53-221.

Laboratory News

By Shelley Burton, Veterinary Clinical Pathologist

Here are some recent happenings in Diagnostic Services:

- We congratulated Dr. Noel Clancey when he passed the American College of Veterinary Pathologists (ACVP) board certifying examination in clinical pathology. He is now a certified Diplomate of the ACVP!
- Dr. Shelley Burton started a one year term as chair of the clinical pathology section of the ACVP Examination Committee. This busy 8 member committee oversees the organization, writing, proctoring and marking the board certifying examination.
- Drs. Maria Forzan, Carolyn Legge and Shannon Martinson attended a training session on forensic pathology given at the annual meeting of the ACVP in Baltimore in October 2010. At the same conference, Dr. Andrea Battison from the Lobster Science Centre gave an interesting presentation on the clinical pathology of lobsters. We were proud to see that Dr. Carolyn Legge, our senior anatomic pathology resident, received a Young Investigator's Award for her poster on cardiac disease in Toy Manchester Terriers.
- Dr. Betsy O'Neil, our resident in clinical pathology, passed her Master of Veterinary Science defense examination. Her work involved improvement of urinalysis techniques for dogs and cats. You will note her name on many diagnostic reports over the next few months as her duty time increases.
- Drs. Sandra McConkey and Shelley Burton, along with Dr. Hans Gelens and Ms. Andrea Chisholm Jack, are excited to prepare sessions for the Atlantic Provinces Veterinary Conference (APVC) in April 2011. Sessions will include lectures on leukemia and immune-mediated hemolytic anemia as well as a blood typing and transfusion laboratory. We hope to see many of you there!

SNAP® cPL™ Test Now Available at AVC Diagnostic Services

By Shelley Burton and Cora Gilroy, Veterinary Clinical Pathologists

Severe pancreatitis can be life threatening, painful and have a harmful effect on many organs. Pancreatitis can be challenging to diagnose in dogs based solely on routine blood tests and results from diagnostic imaging.

A helpful blood test to diagnose pancreatitis measures the concentration of canine pancreatic lipase (Spec cPL™). This test, also called quantitative pancreatic lipase immunoreactivity (PLI), is available through the Atlantic Veterinary College (AVC) Diagnostic Services Laboratory; we forward the sample to a laboratory in Texas and the results are usually available ~7-10 days later. Although it is the best diagnostic test for pancreatitis, the delay in receiving results makes it not optimal for same day diagnosis and treatment of dogs potentially suffering from pancreatitis. We are therefore pleased to report that the SNAP® cPL™*, a rapid test for measuring canine pancreatic lipase, is now available at AVC Diagnostic Services for a cost of $25.00. This test offers a positive or negative result to guide immediate diagnosis and therapy. Serum that is fresh or has been stored in the refrigerator for up to one week can be used. Further evaluation or monitoring of a dog with equivocal results or with a diagnosis of pancreatitis can then be done with the quantitative pancreatic lipase test if desired. Please call if you have any questions concerning this test.

*IDEXX Laboratories, Inc., Westbrook, Maine, USA

Staff Focus

Nicole Murphy

By Andrea Bourque, Veterinary Anatomic Pathologist

Do you wake up and find that your work life revolves around Fluffy's irregular bowel movements? Well, this is now true for Nicole Murphy, whose job as a parasitology technologist has been a strangely interesting and rewarding position.

Nicole grew up in Charlottetown and eastern PEI. She received her Bachelor of Science degree from the University of Prince Edward Island in 1996. After a brief stint with the provincial government's Plant Health bacteriology laboratory, Nicole landed a job in the virology laboratory in Diagnostic Services at the Atlantic Veterinary College (AVC). After three years, she took a research technologist position with the Department of Pathology and Microbiology at AVC working with faculty parasitologists. In addition to assisting with research projects, Nicole began working closely on diagnostic cases with Bob Maloney, the now-retired highly experienced parasitology technologist in Diagnostic Services. Bob proved to be an excellent mentor, providing Nicole with a quality of training she would have been hard-pressed to obtain otherwise.

Since Bob's retirement, Nicole has risen to the challenges of working in a busy diagnostic laboratory. Always pleasant, conscientious, sharply dressed and willing to help, Nicole has become very knowledgeable. Along with her professional colleagues, she routinely processes samples and helps to identify parasites from an incredible variety of animal species, including farm animals, fish, domestic and wild birds, whales, snakes, seals, dogs, cats, foxes, bobcats, bears and rodents. She also greatly enjoys participating in the education of veterinary students, where she is integral in their training in first year parasitology laboratories and in fourth year rotations.

When not juggling a busy diagnostic and teaching schedule, Nicole enjoys a full home life with her husband, Philip, and their two Standard Poodles. They take great pleasure in country living in PEI, enjoying hiking, canoeing and gardening. Nicole loves a good red wine and is an avid movie-buff and reader. Her vices include watching "The Bachelor" and "America's Next Top Model"! Given her current vocation, she hears an amazing number of feces-related jokes as an unavoidable consequence of her job, which she endures quite admirably. Thanks Nicole, for always taking all the @#$@# we send your way with a smile!

Reader Feedback: The *Diagnostic Update* group invites comments or suggestions for future topics in the newsletter. Please submit your comments to *Dr. Cora Gilroy* (cgilroy@upei.ca), Diagnostic Services, Atlantic Veterinary College, UPEI, Charlottetown, PE, C1A 4P3 and they will be forwarded appropriately.