

CYTOLOGY

Blood Smear Review: Erythrocyte Inclusions and Integrating Red Blood Cell Changes

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Blood smear review adds crucial complementary information to routine hematology analyzer results. (Normal blood smear findings and erythrocyte association, size, color, and shape changes in dogs and cats are discussed elsewhere.^{1,2}) This article describes how to identify erythrocyte inclusions and organize erythrocyte findings into broad categories to determine differential diagnoses. As a reminder, start with a properly prepared and stained blood smear. Scan the entire smear on low magnification (10× to identify large structures/cells), and then view the monolayer and feathered edge on high magnification (100× to discern smaller changes/structures).¹

ERYTHROCYTE PRECURSORS AND MATURATION

To identify and understand erythrocyte morphology changes, knowing the stages of erythropoiesis is necessary. Erythroid lineage includes the following cells from least to most mature:

- **Rubriblast (FIGURE 1A):** Large cell with scant, deeply basophilic cytoplasm surrounding finely stippled nuclear chromatin and multiple prominent nucleoli.
- **Prorubricyte (FIGURE 1A):** Similar to rubriblast but lacks distinct nucleoli.
- **Rubricyte (FIGURE 1A AND 1B):** Smaller than earlier precursors with similar scant cytoplasm, which can be dark (basophilic) to light (polychromatophilic),

Abstract

Blood smear review is a key component of a CBC and facilitates identification of inclusions (e.g., parasites, Heinz bodies, basophilic stippling) and erythrocyte morphology changes that may not be detected by an automated hematology analyzer. Some infectious agents/infected cells, such as microfilaria and schizont-filled macrophages, can be identified on low magnification (10× for large structures), while detection of others requires high magnification (100× for small structures). Infectious inclusions need to be differentiated from artifacts and noninfectious inclusions that can indicate other physiologic, metabolic, or pathologic processes. Multiple erythrocyte morphology changes (e.g., association, size, shape, color, inclusions) can be grouped by mechanism and correlated with other clinical/diagnostic findings to determine differential diagnoses (e.g., iron deficiency, oxidative injury, immune targeting) and prioritize next clinical steps.



Take-Home Points

- Evaluation of a freshly prepared blood smear can facilitate detection of various erythrocyte morphology changes, including infectious and noninfectious inclusions; therefore, when sending blood to a reference laboratory for analysis, always submit a freshly prepared blood smear along with an anticoagulated sample.
- Manually performing a blood smear review to assess automated hematology instrument flags is recommended because some morphology changes can interfere with automated analytes (e.g., agglutination, nucleated red blood cells).
- Analyzing a blood smear involves conducting a low-magnification scan of an entire smear, including the feathered edge, to look for large extracellular organisms and infected cells and then moving to high magnification to evaluate the feathered edge and monolayer for small erythrocyte inclusions.
- Erythrocyte inclusions and changes must be distinguished from artifact; correlated with clinical findings; and, in most cases, confirmed by molecular testing because light microscopy sensitivity for detecting infectious agents can be limited.
- Evaluating erythrocyte abnormalities along with other blood work data and clinical history may help elucidate differential diagnoses.
- Bone marrow can take up to a week to mount a response to anemia; thus, if the duration of anemia is unknown, it may be difficult to initially distinguish prerenal and nonregenerative anemia and serial monitoring, with or without reticulocyte enumeration, may be helpful.

surrounding coarsely clumped nuclear chromatin and indistinct nucleoli.

- **Metarubricyte (FIGURE 1A):** More cytoplasm than earlier precursors, similar size and cytoplasmic color as later precursors, condensed nuclear chromatin.
- **Polychromatophil (i.e., reticulocyte, FIGURE 1B):** Purple/blue-tinged anucleate cell that is larger than a mature erythrocyte and represents less than 1.5% of circulating erythrocytes in healthy dogs and cats.

- **Mature erythrocyte (FIGURE 1B):** Round anucleate cell with variably prominent central pallor depending on species.

Maturation from rubriblast to mature erythrocyte takes approximately 1 week; however, this process can be accelerated (3 to 5 days) in response to acute peripheral demand or anemia. This results in the release of late-stage precursors (e.g., polychromatophils) and possibly nucleated red blood cells (nRBCs) like

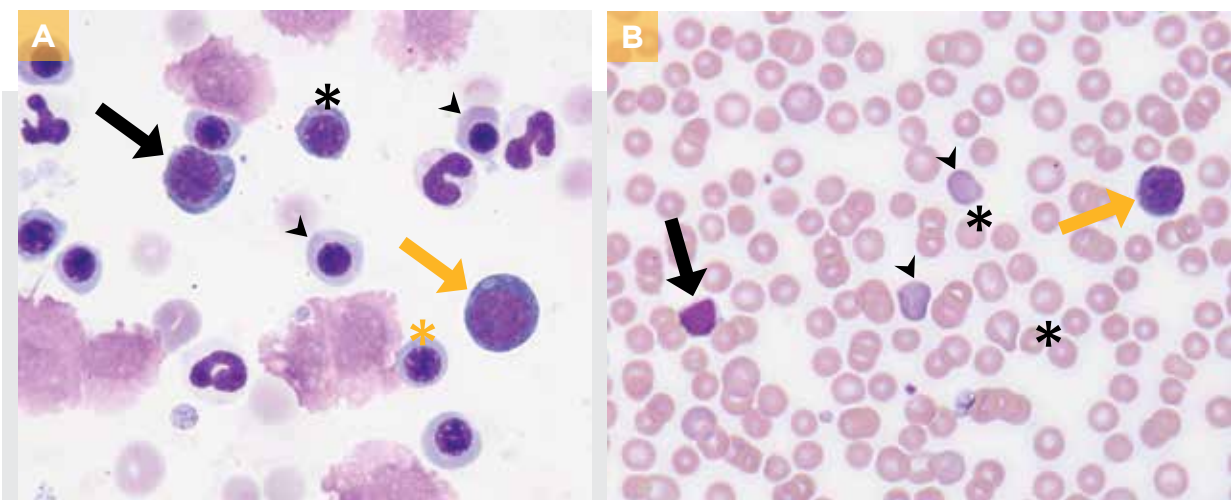


FIGURE 1. (A) Erythroid precursors in a canine bone marrow aspirate: rubriblast (yellow arrow), prorubricyte (black arrow), basophilic (black asterisk) and polychromatophilic (yellow asterisk) rubricytes, and metarubricytes (arrowheads). Wright-Giemsa stain, 100× objective. **(B)** Erythroid precursors in a canine blood smear: basophilic rubricyte (yellow arrow), polychromatophils (arrowheads), mature erythrocytes (asterisks), and intermediate lymphocyte (black arrow). Wright-Giemsa stain, 100× objective.

metarubricytes and rubricytes. The increased circulating nRBCs are considered an appropriate response to anemia. An increased number of nRBCs in the absence of anemia is considered inappropriate; possible causes include heat stroke, lead poisoning, bone marrow disease (e.g., myelofibrosis), splenic disease (e.g., hemangiosarcoma), or splenectomy—all of which cause untimely release or impaired clearance of nRBCs.^{3,4} Atypical morphology features (e.g., enlarged or multiple nuclei) of erythroid precursors could also suggest myelodysplasia/dyserythropoiesis or erythroid leukemia. An increased number of nRBCs or earlier erythroid precursors may be difficult to discern from white blood cells (WBCs; e.g., lymphocytes, other precursor cells) and may be misclassified by automated instruments (**FIGURE 1B**). Therefore, identification and quantification of nRBCs are crucial for determining accurate WBC counts (via correction formula when values exceed 5 nRBCs per 100 WBC).^{5,6}

ERYTHROCYTE INCLUSIONS

Erythrocyte inclusions can be noninfectious (e.g., retained or altered internal organelles/structures) or infectious agents; they are best evaluated using 100× magnification.

Noninfectious Inclusions

Howell-Jolly bodies (i.e., micronuclei) are nuclear remnants and appear as single, round, small to punctate, dark blue structures (**FIGURE 2**). They are typically removed by the spleen but may be seen in low numbers in cats (due to their nonsinusoidal spleen), increased numbers in dogs and cats with regenerative anemia, after splenectomy, or in poodles with benign macrocytosis.

Siderotic inclusions (siderocytes) are focal, granular, often multiple (unlike Howell-Jolly bodies), blue inclusions and represent iron accumulation in the cytoplasm or mitochondria (**FIGURE 2A**). Siderocytes can be seen with conditions that cause increased iron turnover (e.g., hemolytic anemia) or abnormal heme synthesis (e.g., myeloproliferative/dysplastic disorders, lead toxicity, certain drugs).⁷

Basophilic stippling appears as coarse, diffuse (not focal like siderocytes), blue granular inclusions and signifies spontaneous aggregation of ribosomes (i.e., RNA) (**FIGURE 2B**). Basophilic stippling is more common in ruminants with regenerative anemia, yet it

can occur with exuberant regeneration in other species. In the absence of anemia, it can indicate lead poisoning.^{3,4}

Heinz bodies are oxidized or precipitated hemoglobin and appear as pale, irregularly round, small to large inclusions best visualized with new methylene blue, which stains them dark blue (**FIGURE 3**). Their identification by blood smear review is necessary as some analyzers can erroneously report increased hemoglobin, mean corpuscular hemoglobin concentration (MCHC), and WBCs when these inclusions are present in increased numbers.^{3,8} Heinz bodies reduce erythrocyte deformability, which can result in hemolysis or accelerated removal.⁹ Healthy cats can have low numbers of Heinz bodies because feline erythrocytes are prone to oxidative injury.¹⁰ Heinz bodies can also form in patients—with or without anemia—with hyperthyroidism, diabetes mellitus, and lymphoma.³

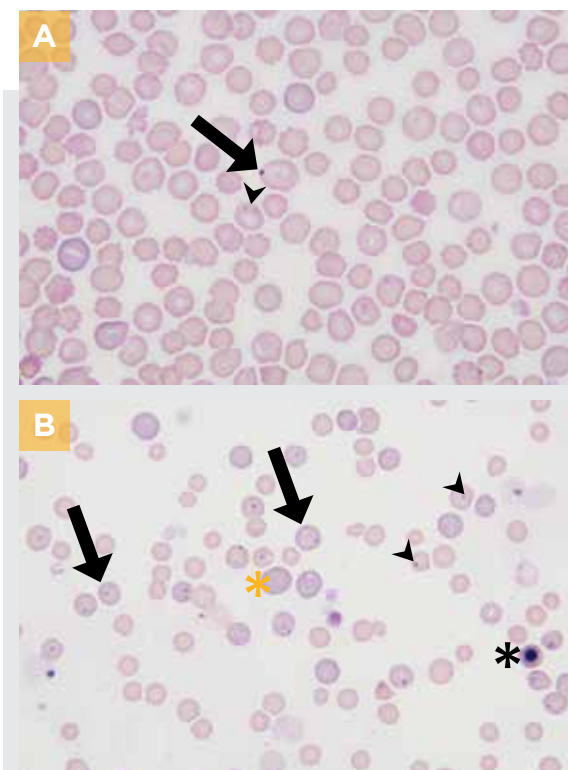


FIGURE 2. (A) Canine blood smear monolayer with a Howell-Jolly body (arrow) and a siderocyte (arrowhead). Wright-Giemsa stain, 100× objective. **(B)** Feline blood smear monolayer featuring marked anisocytosis, several polychromatophils (yellow asterisk), Howell-Jolly bodies (arrowheads), basophilic stippling (arrows), and a single metarubricyte (black asterisk). Wright-Giemsa stain, 100× objective.

TABLE 1 Erythroparasitic Inclusions

INCLUSION SHAPE	DOGS	CATS
Cocci or rod	Hemotropic mycoplasmas (<i>Mycoplasma haemocanis</i>, <i>Candidatus Mycoplasma haematoparvum</i>)	Hemotropic mycoplasmas (<i>Mycoplasma haemofelis</i>, <i>Candidatus Mycoplasma haemominutum</i>, and <i>Candidatus Mycoplasma turicensis</i>)
Ring	Small: <i>Babesia</i> (<i>Babesia gibsoni</i>, <i>Babesia conradae</i>, <i>Babesia vulpes</i>), hemotropic mycoplasmas Large: <i>Babesia</i> (<i>Babesia canis</i>, <i>Babesia rossi</i>, <i>Babesia vogeli</i>)	Small: <i>Babesia</i>* (<i>Babesia felis</i>), <i>Cytauxzoon felis</i>, hemotropic mycoplasmas

*Not commonly found in North America

Infectious Inclusions

Hemotropic mycoplasmas are gram-negative bacteria that appear as small (0.5 to 1.5 μm) blue structures of various shapes (cocci, rods, and rings) on the surface of canine and feline erythrocytes (FIGURE 4).³ Even though several *Mycoplasma* species have been identified in cats, *Mycoplasma haemofelis* is the species most likely to induce clinical disease in immunocompetent cats (TABLE 1). Cats with acute infection exhibit a high parasite load and severe hemolytic anemia, and cats with chronic infections exhibit a low or undetectable parasite load.¹¹ In dogs, *Mycoplasma haemocanis* is the

most common species and typically causes subclinical disease unless the dog is immunocompromised or has had a splenectomy.¹² Given their epicellular location, these organisms can dissociate from erythrocytes over time, particularly during shipping and/or if smear preparation is delayed, which can diminish the sensitivity of organism detection via blood smear review.¹³

Babesia species can infect canine and feline erythrocytes, yet in the United States, infections are much more common in dogs than cats. *Babesia* species have various shapes and sizes, with basophilic cytoplasm and a red to purple nucleus; they have historically been classified as large and small forms (TABLE 1).

- **Large forms (FIGURE 5A):** 2.5 to 5 μm with piriform-shaped, paired or multiple merozoites. *Babesia canis* is more common in Europe, and *Babesia vogeli* is more common in the United States, with greyhounds overrepresented.
- **Small forms (FIGURE 5B):** 1 to 2.5 μm with signet ring appearance, singular or paired. *Babesia gibsoni* is

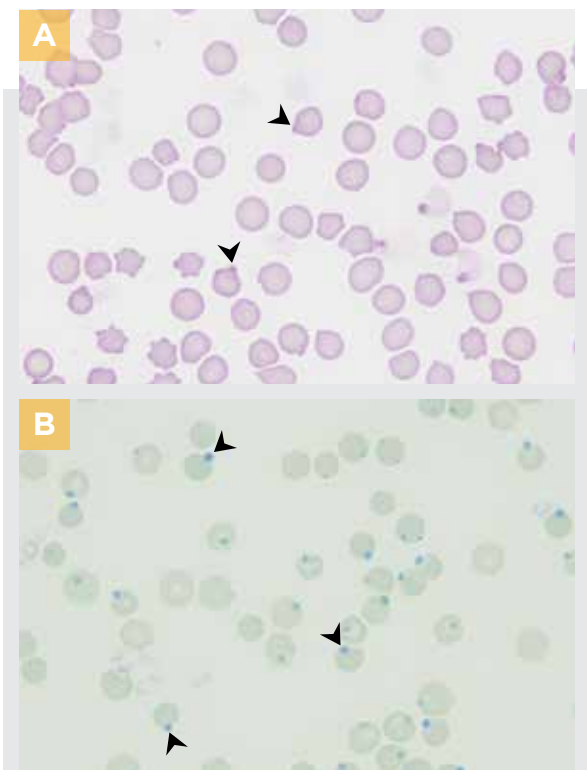


FIGURE 3. Heinz bodies (arrowheads) in the monolayer of a feline blood smear. (A) Wright-Giemsa stain, 100 $\times$ objective. (B) Methylene blue stain, 100 $\times$ objective.

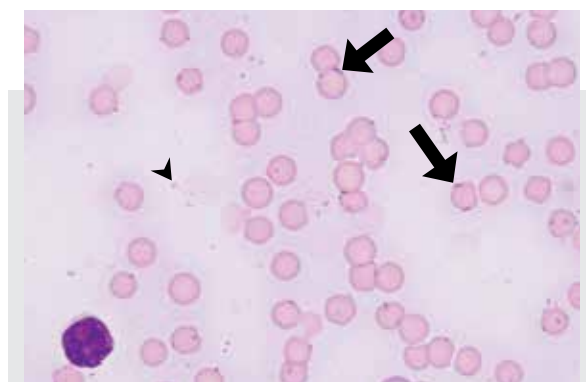


FIGURE 4. *Mycoplasma haemofelis* on the surface of erythrocytes (arrows) and in the background (arrowhead) of a feline blood smear monolayer. Wright-Giemsa stain, 100 $\times$ objective.

the most common worldwide; prevalence is increased in American pit bull terriers. *Babesia conradae* is particularly prevalent in dogs used to hunt coyotes in California and Oklahoma.¹⁴ *Babesia felis* is reported to cause hemolytic anemia in cats in South Africa.¹⁵

Babesiosis is typically characterized by thrombocytopenia and hemolytic anemia, but some dogs may not have clinical signs. Geographic distribution is somewhat restricted by tick vectors; other transmission modes have been reported.¹⁴

Cytauxzoon felis is a feline-specific intraerythrocytic protozoal agent. Because it requires a tick vector, *C felis* is restricted to the Americas with greater prevalence in the southern United States. Sporozoites from an infected tick invade the cat's mononuclear cells that enlarge with schizonts and become thrombi with the potential to cause multiorgan failure. For this reason, thrombocytopenia is a common finding, though patients may also exhibit pancytopenia. The schizont-laden macrophages (15 to 250 μ m) can occasionally be detected by blood smear review and cytology from

spleen and lymph node aspirates (FIGURE 6A). Schizonts divide into merozoites that rupture the macrophage and invade erythrocytes. *C felis* piroplasms resemble small-form *Babesia*, appearing as 1- to 2- μ m signet ring-shaped structures that rarely form bipolar or tetrad shapes (FIGURE 6B). The erythroparasitemia results in hemolysis during the acute phase of infection but can be more benign in the chronic phase.

Canine distemper virus differs from the parasitic inclusions mentioned above in that anemia is not the main clinical finding. Virus inclusions can be seen in the blood of acutely infected dogs, appearing as variably sized (2 to 4 μ m), round to irregular, red or blue structures (depending on the stain used) in erythrocytes and leukocytes (FIGURE 7).¹⁶ Because the inclusions may be present in the acute phase of infection only,¹⁷ molecular assays are advised for greater diagnostic sensitivity if clinical concern is high.¹⁸

In all, the best method for erythrocyte infectious inclusion confirmatory testing, particularly for animals

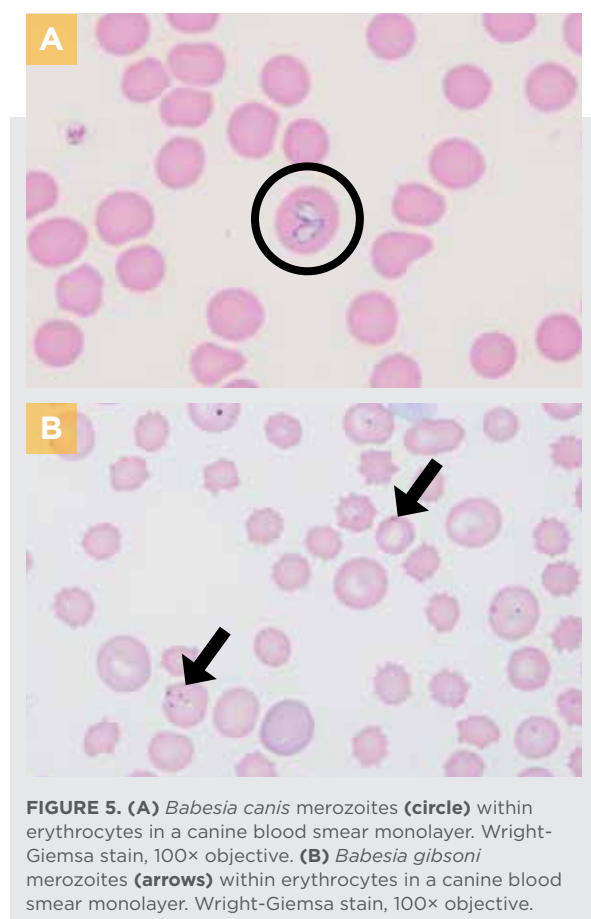


FIGURE 5. (A) *Babesia canis* merozoites (circle) within erythrocytes in a canine blood smear monolayer. Wright-Giemsa stain, 100 $\times$ objective. **(B)** *Babesia gibsoni* merozoites (arrows) within erythrocytes in a canine blood smear monolayer. Wright-Giemsa stain, 100 $\times$ objective.

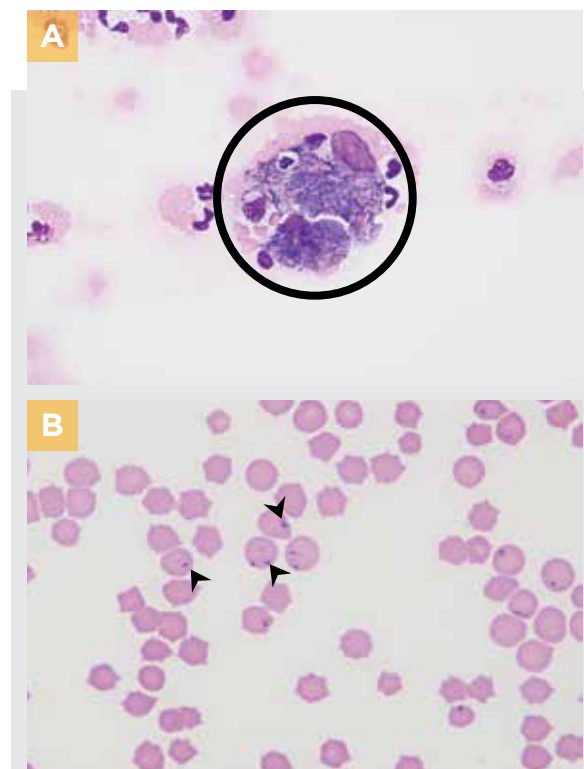


FIGURE 6. (A) *Cytauxzoon felis* schizont-laden macrophage (circle) on the feathered edge of a feline blood smear monolayer. Wright-Giemsa stain, 50 $\times$ objective. **(B)** *Cytauxzoon felis* merozoites (arrowheads) within erythrocytes in a feline blood smear monolayer. Wright-Giemsa stain, 100 $\times$ objective.



with fitting travel histories and/or clinical findings, is PCR due to the overlap of the morphology features of these parasitic agents, variable sensitivity of cytology for detection (depending on stage of disease), and potential for coinfections with vector transmission.¹⁹ Information on infection specifics can be found elsewhere.²⁰

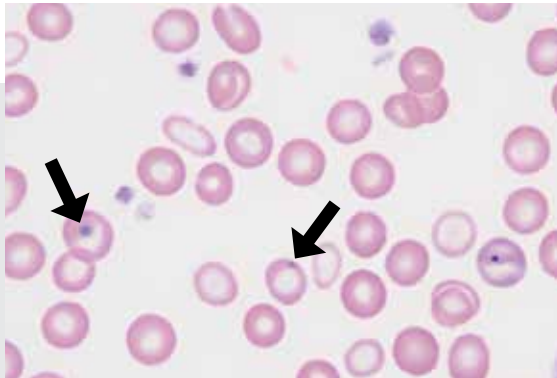


FIGURE 7. Canine distemper virus inclusions (**arrows**) within erythrocytes in a canine blood smear monolayer. Wright-Giemsa stain, 100× objective.

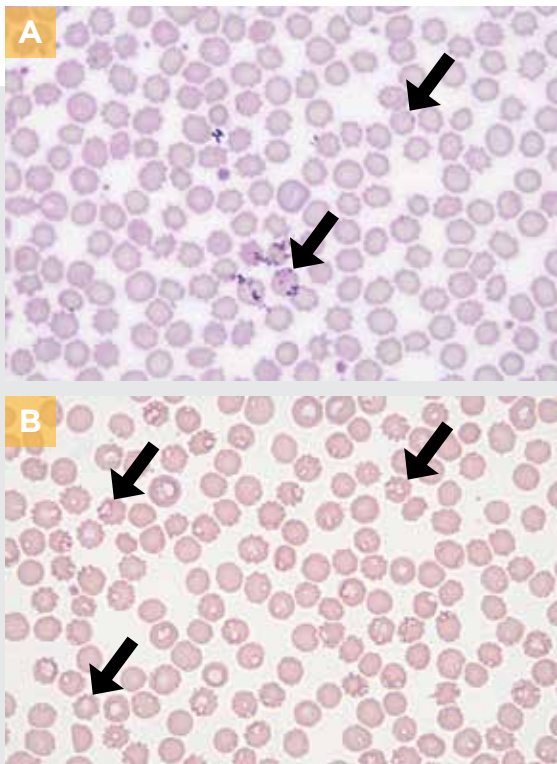


FIGURE 8. (A) Stain precipitate (**arrows**) artifacts in the monolayer of a canine blood smear. Wright-Giemsa stain, 100× objective. **(B)** Water artifact (**arrows**) on erythrocytes in the monolayer of a canine blood smear. Diff-Quik stain, 100× objective.

ARTIFACTS AND EXTRACELLULAR HEMOPARASITES

Differentiating inclusions from artifacts is crucial.⁴ Stain precipitate can be distinguished by its pink color, variable size, and refractility, often overlying cells when viewed in fine focus (**FIGURE 8A**). Its presence may indicate that the stain should be filtered, staining time was too long, or washing time was too brief. Drying or fixation artifacts (appearing as clear irregular refractile inclusions on erythrocytes) can also mimic erythrocyte inclusions (**FIGURE 8B**).²¹

Although this article focuses on erythrocytes, it would be remiss not to mention the most common extracellular hemoparasites of dogs and cats:

- **Microfilariae** (nematode larvae) are most frequently encountered in dogs and include pathogenic *Dirofilaria immitis* (**FIGURE 9A**) and nonpathogenic *Acanthocheilonema reconditum* (**FIGURE 9B**), which can be identified on blood smear review (particularly in the feathered edge or buffy coat) or by modified

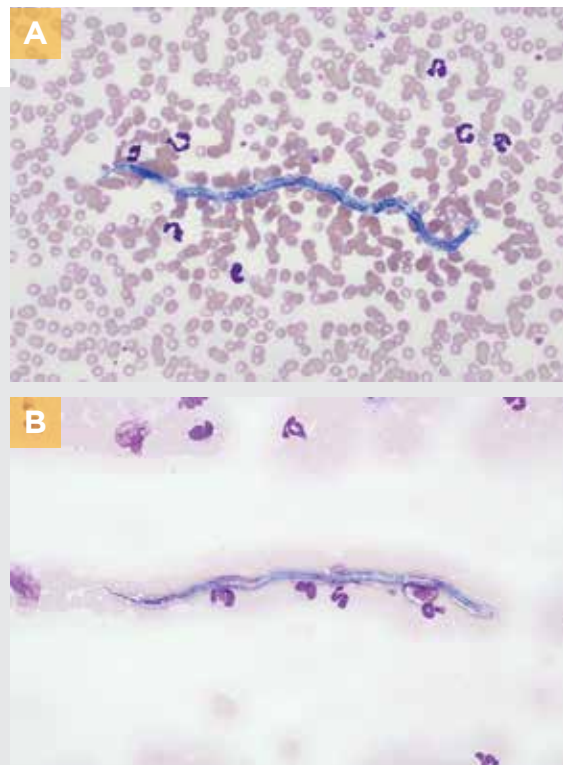


FIGURE 9. (A) *Dirofilaria immitis* microfilaria with a conical front end and typically straight caudal end in the feathered edge of a canine blood smear. Wright-Giemsa stain, 50× objective. **(B)** *Acanthocheilonema reconditum* microfilaria with a blunt front end and hooked caudal end in the feathered edge of a canine blood smear. Wright-Giemsa stain, 50× objective.

Knott's test.^{22,23} Detection of circulating microfilaria in cats is uncommon, which most likely contributes to underdiagnosis. These 2 microfilariae have slightly different morphology features, and antigen and/or molecular testing should be used for differentiation.²⁴

- **Trypomastigotes** of *Trypanosoma cruzi* are rarely seen in blood smears of acutely infected dogs and cats and appear as elongated, flagellated protozoa with a central nucleus and posterior dense kinetoplast (a specialized region of mitochondrial DNA) (FIGURE 10).²¹ Trypomastigotes are the etiologic agent of Chagas disease, and its transmission by the reduviid bug limits its geographic presence to the southern United States, including Texas, with prevalence similar to that of *D. immitis*.²⁵ Travel history and clinical signs—including anemia, lymphadenopathy, diarrhea, and especially cardiac and neurologic signs—should prompt serologic/molecular testing even if no organisms are found via blood smear review.²⁶

INTEGRATING ERYTHROCYTE CHANGES

Understanding erythrocyte morphology changes is essential, especially when evaluating anemia, and the constellation of certain findings can elucidate underlying mechanisms (TABLE 2). A regenerative response can take up to 7 days, and an increased mean cell volume (MCV) and decreased MCHC can suggest regeneration; if the duration of anemia is unknown, prerenal anemia (versus truly nonregenerative) cannot be excluded without serial assessment. In addition, degree of regeneration can vary based on acuteness of insult. Of note, hematocrit and MCV can be falsely increased with delayed sample processing due

to erythrocyte swelling in EDTA. Comparing calculated (hematocrit) and manual (packed red cell volume) values can help detect some artifacts.²⁷

Regenerative Anemia

Regenerative anemia indicates that the bone marrow is responding appropriately and is best assessed in dogs and cats by absolute reticulocyte concentration (via manual or automated method). Because reticulocytes are larger and less hemoglobin-rich than mature erythrocytes, their MCV is increased and MCHC may be normal or decreased. On blood smear review, increased polychromatophils with or without increased nRBCs, Howell-Jolly bodies, or basophilic stippling can be seen.

Reasons for regenerative anemia generally include either erythrocyte destruction (hemolysis) or blood loss.³ Because hemolytic anemia may be initially nonregenerative, other evidence of a hemolytic process should be evaluated via blood smear, serum biochemical profile, or urinalysis (e.g., hemoglobinemia/hemoglobinuria, hyperbilirubinemia/bilirubinuria in the absence of primary hepatic disease). Yellow (icteric) or red (hemolytic) plasma can also indicate hemolysis.³ If anemia is regenerative but evidence of hemolysis is lacking, blood loss anemia may be suspected, particularly in conjunction with normal or low plasma protein. Specifically, hypoproteinemia suggests external rather than internal blood loss. Initially regenerative, blood-loss anemia can become less regenerative with chronicity due to losses in iron and protein.²⁷

General differentials for hemolytic processes include the following:

- **Immune-mediated hemolytic anemia (IMHA)** can be primary/nonassociative or secondary/associative (e.g., infectious agents, neoplasia, blood transfusions, drugs/toxins); the former is a diagnosis of exclusion. IMHA is more common in dogs than cats; nonassociative IMHA is more common among dogs and associative IMHA is more common among cats.²⁸ Hemotropic agents (e.g., *Babesia*, *Cytauxzoon*, *Mycoplasma*) and venom from snakes, bees, and spiders can induce hemolytic anemia.³ Signs of immune-mediated destruction include spherocytes, agglutination, and antierythrocyte antibodies noted by a positive Coombs test.²⁸ Agglutination can artifactually increase MCV and thus the hematocrit; therefore, evaluation of the packed cell volume may be more accurate in these cases.²⁷

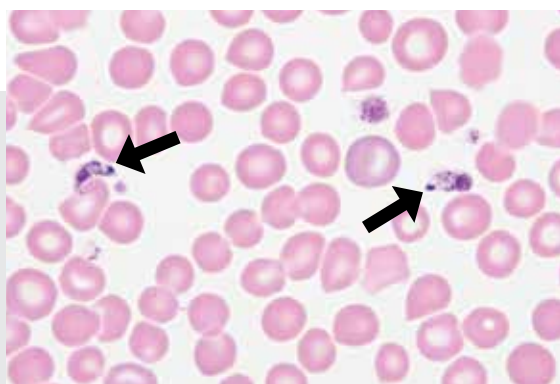


FIGURE 10. *Trypanosoma cruzi* trypomastigotes with kinetoplast (arrows) in the monolayer of a canine blood smear. Wright-Giemsa stain, 100× objective.

TABLE 2 Erythrocyte Changes by Condition*

CONDITION	SIZE	COLOR	SHAPE/ASSOCIATION	INCLUSIONS
Erythrocyte regeneration (e.g., acute blood loss, hemolysis)	- Anisocytosis - Macrocytic (↑ MCV)	- Polychromasia (reticulocytosis) - Hypochromic (↓ MCHC)	Depends on underlying cause	- nRBCs - Howell-Jolly bodies ± Basophilic stippling
Immune-mediated destruction (e.g., hemolysis)	Macrocytic (↑ MCV)	Polychromasia (reticulocytosis)	- Spherocytes - Ghost erythrocytes - Agglutination	- See “Erythrocyte regeneration” ± Siderocytes ± Erythroparasite
Oxidant-induced injury (e.g., hemolysis)	Macrocytic (↑ MCV)	Polychromasia (reticulocytosis)	- Eccentricocytes - Pyknocytes - Ghost erythrocytes - Keratocytes	Heinz bodies
Erythrocyte fragmentation	Variable	Variable	- Acanthocytes - Keratocytes - Schistocytes	
Iron deficiency (e.g., chronic blood loss)	Microcytic (↓ MCV)	Hypochromic (↓ MCHC)	- Codocytes - See “Erythrocyte fragmentation”	

MCHC = mean corpuscular hemoglobin concentration; MCV = mean cell volume; nRBCs = nucleated red blood cells.

*These are general findings that may occur; however, not all of these may be present and should be correlated with other clinical and diagnostic findings to discern cause(s) for anemia. Refer elsewhere for additional information regarding erythrocyte size, color, and shape changes.²

- **Oxidant injury** can result from ingestion of onions, garlic, acetaminophen, and methylene blue by cats and dogs, zinc-containing objects (e.g., pennies, naphthalene) by dogs, and methionine and phenazopyridine by cats. Pathologic conditions more likely to generate endogenous oxidants include diabetes mellitus, hyperthyroidism, and lymphoma; these patients may exhibit mild anemia only, unlike in cases from exogenous sources.³
- **Erythrocyte fragmentation** can occur when erythrocytes move through altered vasculature, experience turbulent blood flow (e.g., hemangiosarcoma, disseminated intravascular coagulation, vasculitis, caval syndrome, glomerulonephritis), or have increased fragility (e.g., iron deficiency anemia). The severity of anemia/regeneration varies with the underlying cause. Shape changes indicating erythrocyte fragmentation include schistocytes, acanthocytes, and keratocytes.³

Nonregenerative Anemia

Differentials for nonregenerative anemia include sequelae to other conditions such as iron deficiency, inflammation, or neoplasia as well as renal, hepatic, or endocrine disease. Nonregenerative anemia can also suggest a primary pathology at the level of the bone marrow, particularly when bicytopenia or pancytopenia is present. Pathology at the level of the bone marrow can be secondary to a bacterial infection (e.g., *Ehrlichia canis*), viral infection, or drug/toxin exposure; bone

marrow evaluation may provide insight as to what is inhibiting a marrow response. For example, evaluation of the marrow may indicate erythroid hypoplasia, pure red cell aplasia, or leukemia/neoplasia. If there is erythroid hyperplasia, the marrow may demonstrate ineffective erythropoiesis, yet erythrocyte release is inhibited, which can occur in cases of infections, drug reactions, myelodysplastic syndrome, iron deficiency, or precursor-directed immune-mediated anemia (demonstrating phagocytosis of precursor cells).²⁷ Peripheral hypochromic and microcytic erythrocytes can indicate iron deficiency anemia resulting from a lack of iron that is needed for hemoglobin production. Codocytes, keratocytes, and schistocytes may also be seen due to increased erythrocyte fragility. These changes can be seen in blood from young animals or animals with chronic external blood loss or nutritional deficiency.³

SUMMARY

Blood smear review is an excellent preliminary diagnostic for evaluating erythrocyte morphology changes that can provide insight into the mechanism/cause of anemia or underlying disease and inform diagnostic and therapeutic plans. **TVP**

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