Dear Sir:

Regarding the Editorial Review (Red Cell Fragmentation in the Dog) by Rebar et al., in Vol. 18, No. 4, p. 415, 1981, it would be advantageous if the authors could elucidate why fragmentation results in such different shapes. For example, the typical schistocyte of microangiopathy is easily conceptualized, but microangiopathy is also purportedly the cause of burr cells in uremia. How can spherocytes, spheroechinocytes, and acanthocytes be such diverse morphologic forms resulting from the same mechanism, i.e., loss of red cell membrane. It is easy to see how these different abnormalities can lead to fragmentation, but how can they be called examples of fragmented red cells? One would expect a fragmented cell to be smaller and irregular shaped, such as a schistocyte.

Also, I would like to point out an unfortunate omission in the extensive literature review. Since the number of references reporting these abnormalities in animals is small, I think the only reported case of spontaneous spur cell anemia in the dog (to my knowledge) should have been included (Spur Cell Anemia in a Dog, Robert M. Shull, et al., JAVMA 173:978, 1978).

Sincerely,
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College of Veterinary Medicine
University of Tennessee
Knoxville, Tenn.

Dear Sir:

Traditionally, red cell fragmentation has been associated primarily with microangiopathic hemolytic anemia and schistocytosis; the present editorial review suggests that a variety of other pathogenic mechanisms also may be associated with abnormal red cell shapes and red cell fragmentation.

Dr. Easley has asked the authors to explain why fragmentation results in such a variety of red cell morphologies. In answer, the authors would emphasize that only some of the poikilocytes discussed in the editorial review represent the product of fragmentation; others represent altered red cells which are more susceptible to subsequent fragmentation. We believe that this distinction is clearly identified in the text of the review. For example, the formation of spherocytes as end products of partial phagocytosis of antibody-coated red cells is outlined on pages 417-418 and represents a true fragmentation phenomenon. On page 419 the spherocytes of immune-mediated disease are compared to the echinospherocytes of the intrinsic hemolytic disorder associated with pyruvate kinase deficiency. At no time do the authors suggest that echinospherocytes necessarily represent fragmented red cells; indeed, in paragraph 5, page 418, it is suggested that spherocyte formation associated with intrinsic hemolytic disorders “may not represent a true fragmentation syndrome”. In the section entitled “Red cell fragmentation in association with abnormal membrane physiology due to systemic disease”, acanthocytes are described as red cells with abnormal plasma membranes and resultant increased cellular rigidity. The relationship of acanthocytosis and cellular rigidity to
fragmentation is discussed on page 421 where the pathogenesis of acanthocyte formation is addressed in detail. The pathogenesis of burr cell formation and fragmentation in uremia is fairly obscure and the review is purposefully vague in this regard.

Dr. Easley rightfully points to the omission of the paper “Spur Cell Anemia in a Dog”, JAVMA 173:978, 1978. The authors were aware of this manuscript and its omission was an oversight. However, the occurrence of spontaneous acanthocytic anemias in dogs is mentioned in at least three of the other cited references (54, 55, 60). In addition the authors would add the following very current reference to the list:


Respectfully,
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