Chronic kidney disease (CKD) remains an important and significant cause of morbidity and mortality in dogs and cats. The understanding, assessment, and management of CKD in veterinary practice is evolving rapidly from its foundations in serum creatinine and therapeutic diets to sophisticated diagnostic testing, novel therapies, and changing clinical paradigms. Yet, in some cases, the new advances represent merely a reunderstanding of established concepts: “old tricks for new dogs.” In other cases, there are directional shifts and emerging technologies redirecting our previous understanding: “new tricks for old dogs.”

In the area of evolving diagnostic assessments for CKD, novel biomarkers exemplified by symmetric dimethylarginine now provide diagnostic extension and increased sensitivity to the time-honored value of serum creatinine for the detection and monitoring of CKD. Equally exciting is the development of diagnostic markers of active kidney injury with the potential and sensitivity to detect the presence and persistence of kidney injury before it is suspected clinically or detectable by conventional diagnostics.

Deeper probing into cellular function and regulatory processes defining kidney health and kidney disease suggests the same molecular processes that control and regulate normal kidney function also may participate in the responses to kidney injury. These cellular pathways can become dysregulated by even subtle pathologic “stresses” and promote maladaptive responses that signal progressive erosion of kidney mass and function that remains subclinical in the early and evolving stages of progression. These new insights provide potential for strategic therapeutic targets to prevent progressive kidney disease. The detection and characterization of early and asymptomatic kidney disease must become a higher diagnostic priority. Logically, advanced CKD recognized at a point when it is clinically evident must have extended from a milder, less evident, and less advanced stage of CKD. Going forward, clinical practice patterns must be modified to incorporate emerging diagnostics to detect and characterize asymptomatic kidney disease (IRIS CKD stage 1). When detected early, therapeutic strategies will be more effective to slow or halt progression of CKD to more advanced stages associated with progressive morbidity and less therapeutic potential.
The concept of spontaneous progression of CKD has been recognized for many decades, and current evidence supports that this concept is applicable to dogs and cats with CKD. Extensive research has led to a more sophisticated understanding of some of the mechanisms underlying progression of CKD, particularly in cats. The combination of improved diagnostics for recognizing early kidney disease as well as ongoing kidney injuries is potentially synergistic with the expanding understanding of progression of CKD. These exciting findings provide opportunities for intervening earlier in the course of CKD, thereby allowing us to provide a period of extending high quality of life, possibly even stopping progression of CKD before clinical signs develop.

An important controversy in Veterinary Nephrology has developed in recent years: what should be fed to cats with CKD. Although renal diets have been recommended for decades with the expectation that they will be effective in mitigating clinical signs of uremia and slowing progression of CKD, their clinical effectiveness and nutritional impact recently have been challenged. Some veterinarians have even suggested that cats with CKD should be fed high-protein diets. Three views of the current status of recommending renal diet therapy for cats are presented in this issue: the evidence supporting current feline therapeutic renal diets, the evidence questioning the current formulations of renal diets, and an overview of the disagreements concerning limiting protein content in renal diets. In addition to the impact of diet formulation, assuring adequate food ingestion is an important component of maintaining adequate nutrition in cats with CKD. The role of feeding tubes in preventing nutritional deficiencies in cats with CKD also is presented.

Two critical therapies linked to slowing progression of CKD are minimalizing proteinuria and limiting phosphorus intake. New approaches to managing proteinuria appear to enhance the effectiveness of reducing the magnitude of proteinuria, thereby potentially slowing progression of kidney disease. There are exciting new insights into the pathophysiologic events that maintain phosphorus balance in patients with CKD. Not surprising, our old construct of renal secondary hyperparathyroidism has been found lacking. Nonetheless, controlling phosphorus intake remains pivotal. An increasing variety of intestinal phosphate binders have become available for managing phosphorus balance. These have expanded our options for therapy and vary in effectiveness, side effects, and cost. In some patients, ionized hypercalcemia or ionized hypocalcemia may indicate the need for pharmacologic modification of serum calcium concentration.

Renal osteopathy is a classic and consistent feature of CKD documented and managed in human patients for decades but rarely recognized or specifically managed as a feature in dogs and cats. However, with utilization of sophisticated imaging and biomechanical techniques, reviewed in this issue, there now is compelling evidence these classic features of CKD also are pathologic features of naturally occurring CKD in dogs and cats. These new revelations require an updating of our concepts of mineral and bone disease in animal patients and reprogramming of our diagnostic approaches and holistic approach to the management of CKD. Similarly, CKD must be recognized as a polysystemic syndrome that necessitates a broad-reaching and comprehensive therapeutic approach. To be effective, the therapy must be global to ameliorate all identifiable consequences of CKD to targeted endpoints, not merely a ritualistic or uniform approach that fails to address all manifestations.

Renal transplantation, once the advanced standard for the surgical management of CKD in cats, has nearly vanished as an option for animals despite its persisting role in human nephrology. The transition in surgical interests and expertise from microvascular techniques to interventional technologies likely has contributed to this change in status. There is still opportunity and need to resurrect renal transplantation as an
ongoing and future strategy for the correction of CKD in cats (and perhaps dogs) as newer advances in graph-versus-host prevention or development of genetically engineered kidneys evolves. Despite its potential impact on renal transplantation, the advent of interventional surgical expertise and devices emerged fortuitously with the emergence of ureteral obstruction as a major cause for both acute and chronic kidney disease in cats. The use of stents and subcutaneous ureteral bypass systems for the management of ureteral obstruction has provided a treatment option that is more effective and less morbid than their predecessor medical or surgical approaches.

In this issue, we have attempted to update the current and topical issues surrounding CKD in dogs and cats. We have chosen to review old issues that require conceptual updating and the conventional understanding of CKD through the lens of new technologies and insights. We hope introduction of these new concepts and therapies will catalyze future developments in the practice of veterinary nephrology.

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