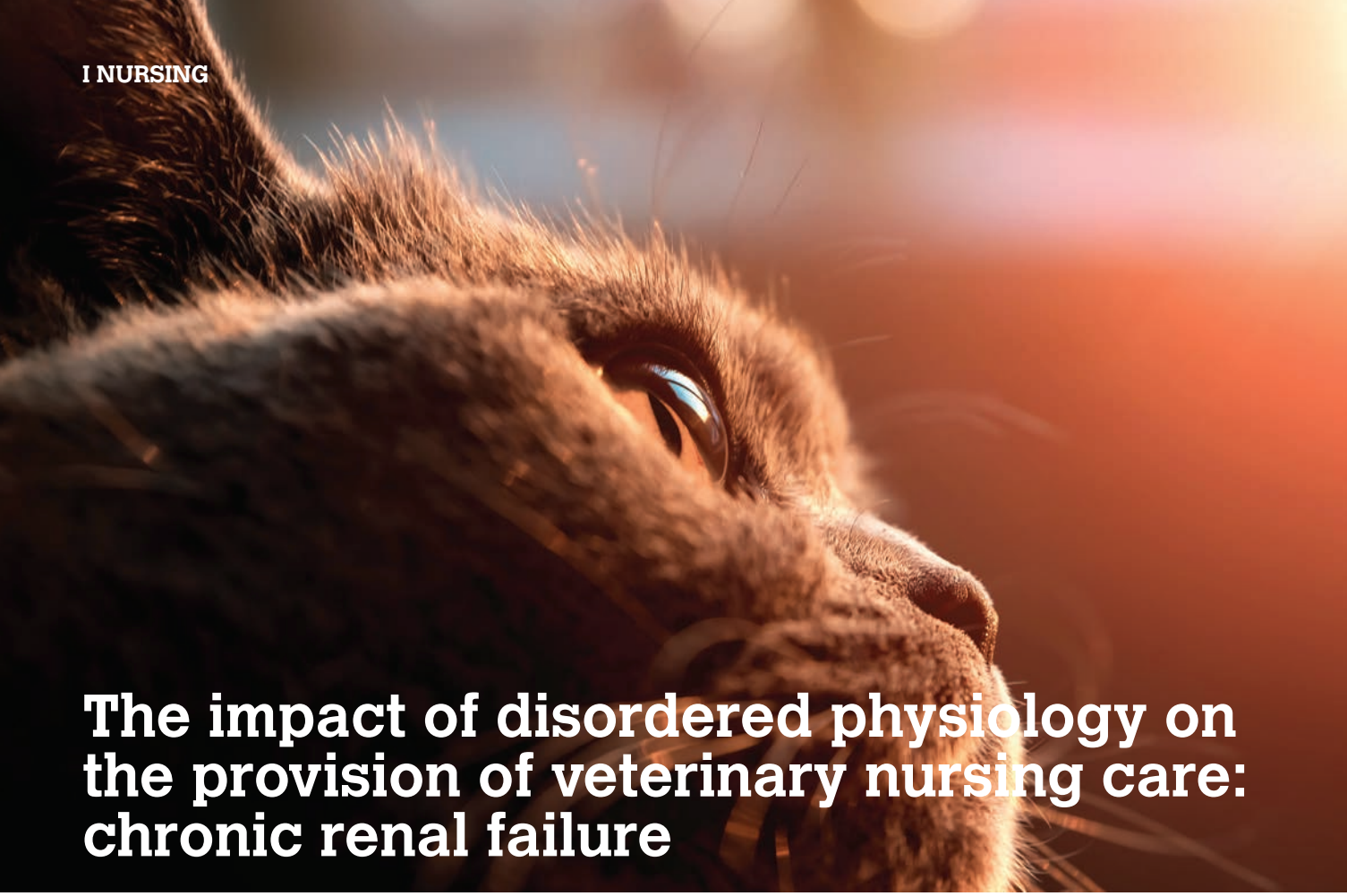

The impact of disordered physiology on the provision of veterinary nursing care: chronic renal failure

Patients are hospitalised for many reasons and it is essential VNs have knowledge and understanding of common disorders and the associated clinical signs in order to justify their chosen nursing interventions, writes Liane Henry RVN

Veterinary nurses are commonly the main care-givers for hospitalised patients. Branscombe (2010) discussed how up-to-date knowledge and skills are relevant to veterinary nurses to provide the highest standards of nursing care to their patients disorders and the associated clinical signs in order to justify their chosen nursing interventions. This article will discuss chronic renal failure (CRF) and how the disruption of normal physiology produces abnormal clinical signs requiring nursing care.

CHRONIC RENAL FAILURE

CRF is a condition that causes pathophysiological and irreversible damage to the parenchyma of the renal anatomy, inhibiting normal function and causing the patient to exhibit abnormal clinical signs. Sparkes (2016) et al stated that renal disease is considered chronic when there is evidence of reduced renal function for a period of three months or more. The International Renal Interest Society published a set of algorithms in 2006 (which have since been modified) to assist the staging of CRF from stages 1-4, in both canines and felines. Patients are classified in the later stages (stages 3-4) of CRF when azotemia is diagnosed, due creatinine levels >140 umol/L in felines and >125 umol/L in canines. Clinical signs and uraemia appear in the later stages of CRF.

Hewitson (2009) reported that kidneys react similarly to other body systems when injured. Inflammation occurs in the nephron causing inflammatory cells to populate the area and McLeland et al (2015) reported that these cells are found at all stages of CRF, with incidence increasing with progression of the disease. Cytokines are produced, which activate local mesenchymal cells to proliferate and synthesise extracellular matrix, which contracts and increases in density, causing interstitial fibrosis (Hewitson 2012). Brown et al (2016) reported that these changes are commonly identified in the tubulointerstitial region of the nephron and this continuous accumulation of fibrotic tissue causes reduced function, including tubular degeneration and atrophy. Reduced function in the nephron tubules limits their permeability, resulting in decreased tubular absorption and secretion of materials from the filtrate. Brown et al (2016) stated that pathophysiological changes also occur in the glomerulus of the nephron and McLeland et al (2015) identified global glomerulosclerosis as present when >75% of the capillary tuft was irreversibly damaged, resulting in blood being redirected to remaining viable nephrons causing hypertrophy.

Podocytes in the visceral layer of the Bowman's capsule respond to hypertrophy by adapting in size to cover a larger surface area, which Chakrabarti et al (2012) reported

inhibits sufficient filtration through the glomerulus. Affected glomeruli exhibit thickened and wrinkled basement membranes, collapsed capillary tufts and interstitial fibrosis (Brown et al 2016), allowing normally filtered macromolecules to pass through the membrane and enter the Bowman's capsule, convoluted tubules, loop of Henle and collecting ducts. Hewitson (2012) described how interstitial fibrosis in the nephron affects the vasculature, causing sclerosis in the capillaries, then ischemia and hypoxia. Chakrabarti et al (2012) noted it may be a combination of these factors that contributes to the obsolescence of affected glomeruli. As discussed, this pathogenic process leads to the malfunction of the renal parenchyma causing irreversible nephron loss and clinical signs seen in the affected patient. The kidneys play a vital role in acid-base regulation through the generation of bicarbonate and excretion of acids, such as hydrogen ions, in urine. Damage to the nephron reduces the kidney's ability to carry out this function, causing a decreased pH in the blood, resulting in acidosis (Welsh and Girling 2010). Korman and White (2013) noted that increased concentrations of gastrin in blood due to poor excretion via the kidneys results in gastric acidity and ulceration. Hypokalaemia is common in cats with CRF due to increased potassium loss and, according to Reynolds and Lefebvre (2013), can induce metabolic acidosis leading to hypergastrinemia, resulting in adverse clinical signs such as ulceration, stomatitis, nausea, inappetence and vomiting. Further clinical signs associated with CRF include hypertension, polyuria, polydipsia, anaemia, and dehydration.

An understanding of CRF pathophysiology and clinical signs assists the VN when planning and implementing chosen nursing care and optimises the care plan specific to the patient's needs.

THE HYPERTENSION LINK

Systemic hypertension and CRF are strongly linked, with a high incidence of patients presenting with hypertension also showing evidence of CRF. Despite this, there is little evidence to determine whether hypertension causes CRF or vice versa (Jepson 2011). Auto regulation of renal blood pressure may be compromised by systemic blood pressure causing increased glomerular permeability and damage to the convoluted tubules (Jepson 2011). The renin-angiotensin-aldosterone system (RAAS) is noteworthy in CRF. Reynolds and Lefebrve (2013) summarised that activation of the RAAS induces vasoconstriction of the efferent arteriole, contributing to glomerular hypertension, increased cellular matrix, tubular and interstitial inflammation and, ultimately, CRF. Angiotensin II is particularly concerning, as discussed by Rüster and Wolfe (2006), due to its stimulation and proliferation of mesangial, glomerular endothelial and fibroblast cells, causing tubular hypertrophy. The VN should assess hospitalised patients regularly for hypertension using a blood pressure monitoring device. Maltman (2003) advised the readings should be taken in a quiet environment where the patient has acclimatised, with the aim of recording accurate readings unaltered by high stress levels.

REHYDRATION

Damage to convoluted tubules and the loop of Henle causes reduced permeability, resulting in several clinical signs for the CRF patient. Polyuria occurs when excess water is excreted in urine due to reduced reabsorption of water from the glomerular filtrate in the convoluted tubules, leading to dehydration then polydipsia in an effort to rehydrate (Korman and White 2013). Breton (2013) discussed diuresis as the gold standard of care for CRF. The VN's role in the administration and monitoring of intravenous fluid therapy (IVFT) was discussed by Bloor (2015) and care should be taken when calculating fluid requirements to include any fluid losses due to dehydration, vomiting and diarrhoea. In addition, Breton (2013) advised regular reassessment of fluid requirements as the patient becomes rehydrated. Where patients are receiving IVFT for a number of days, Bloor (2015) advised the VN should include daily inspection of the catheter insertion site for signs of infection in the patient care plan. In addition to IVFT, polyuria indicates ample access to litter trays with absorbent litter for cats and numerous toilet walks for dogs. Urine output volumes

should be recorded and accounted for in fluid-deficit IVFT requirements and bedding should remain clean and dry to prevent urine scalding (Almond 2017). Proteinuria occurs due to decreased reabsorption in the tubules in combination with systemic hypertension, causing reduced filtration in the glomerulus, leading to proteins being excreted in urine (Beetham and Cattell 1993). Almond (2017) suggested the VN should monitor for proteinuria at regular intervals during hospitalisation and at subsequent visits to the clinic using a urinalysis dipstick, with results recorded. This also provides the opportunity to screen for haematuria or infection, which is a risk for patients with CRF due to dilute urine and some medications (Almond 2017).

ANAEMIA

Reduced production of erythropoietin in the peritubular cells results in anaemia for 30-60% of felines with CRF (Reynolds and Lefebvre 2013) and is worsened by shortened red blood cell lifespan and gastrointestinal haemorrhage (Chalhoub et al 2011). Anaemic patients may present with pale mucus membranes and also display clinical signs such as weakness and lethargy (Chalhoub et al 2011). Breton (2013) recommended care when blood sampling these patients including taking the minimum amount of blood required for blood screens or packed cell volume count and suggests applying a pressure bandage to the venipuncture site to prevent haemorrhage.

Gastrointestinal signs are major indicators of uraemia and are often the first signs of CFR noted by the owner. Nursing care for the hospitalised CRF patient should include a plan for responding to these clinical signs. Vomiting and nausea can cause inappetence and the attending veterinary surgeon (VS) should be notified where anti-emetics may be required. Clinical signs, such as stomatitis, may be painful and Cherry (2014) suggests the VN pain scores CRF patients regularly. Where the patient is deemed painful, the VS should be notified and analgesics prescribed and administered. Almond (2017) suggested care should be taken when choosing a feed in hospital to reduce the likelihood of diarrhoea and to avoid food aversion due to a quick change in diet. Other clinical signs must also be considered when feeding. Ackerman (2015) noted the effect uraemia has on a patient's appetite, including reduced sense of smell and taste, and suggested small, warmed, frequent meals may be better accepted. Despite IVFT, fresh water should be available at all times to satisfy polydipsia and a wet-food diet is usually preferable for these patients as it will increase overall water intake (Almond 2017). Newly diagnosed patients should be allowed a slow transition onto a renal diet once discharged from hospital. A study by Plantinga et al (2005) showed renal-specific diets increase longevity in cats with CRF. A thorough clinical history and a holistic approach to nursing these patients will allow the VN to identify the patient's likes and dislikes to select a diet for use in hospital, as well as recommending an appropriate commercial diet for long-term management. Almond (2017) noted the importance of the VNs continuing role after the patient has been discharged, providing support and advice to owners during the initial weeks following diagnosis, as well as being a familiar face for routine blood and sampling and blood pressure monitoring where a VS consult is not required.

Another area for VNs to consider is the patient's emotional wellbeing during hospitalisation. Cherry (2014) advised the VN should attend to this aspect of patient's wellbeing to provide holistic nursing care to the patient and suggested low-stress handling, hidey-holes in kennels and the use of pheromones may be beneficial to the feline CRF patient. Cherry (2014) also mentioned the unrivalled benefits of touch and tender-loving-care, for example grooming, which benefits the patient's mental health and in the case of CRF also provides an appropriate nursing intervention for managing reduced grooming due to stomatitis.

CRF is a multifactorial pathophysiological process, causing irreversible loss of nephrons in the kidneys. Chronic and cumulative inflammation, alongside increased production of extracellular matrix production causes tubulointerstitial fibrosis, glomerular sclerosis and vascular sclerosis resulting in abnormal clinical signs, which are thought to further contribute to the disease pathway and to end-stage renal failure. Nursing care for these patients requires a combination of knowledge and understanding of the disease pathway along with a holistic nursing approach, considering all related clinical signs in order to manage the disease, both in hospital and at home.