Diaphragmatic hernias

It is thought that up to 85% of diaphragmatic hernias (DH) are traumatic in origin and a significant proportion of dogs and cats that sustain major trauma can sustain DH. The theorized mechanism by which these injuries arise is by major blunt trauma to the abdomen with the glottis open causing a sudden increase in the pressure gradient between the chest and abdomen. Rupture of the diaphragmatic costal muscle is the most common consequence due to the inherent weakness of these tissues in comparison to the stronger central tendon. When costal muscles are involved tearing most commonly occurs parallel to the orientation of the costal muscle fibers and these are usually termed radial tears. Another common defect configuration is circumferential where the diaphragm is avulsed from its attachment to the body wall. Circumferential tears occur commonly in both dogs and cats but proportionally occur with greater frequency in cats.1,2 Almost any non-fixed organ within the abdominal cavity can become herniated in a DH but the most common include stomach, small intestine, liver, spleen.

Of great importance prior to surgical intervention in DH cases is global assessment of the patient. Many cases of DH present with concurrent musculoskeletal or other organ system injuries. Recognition and treatment of comorbidities is essential in these cases and in some cases distract the clinician from recognizing DH as well as other internal injuries. In one study only 57% of DHs were diagnosed within 30 days of trauma.3 Animals with DH should be carefully evaluated. In some cases no or few clinical signs referable to the respiratory system will be detected. In other cases mild to severe respiratory distress may be evident. Diagnostic assessment usually starts with thoracic radiography which should be performed with the least amount of physical restraint necessary. Classical signs consistent with DH include loss of a clear diaphragmatic outline and cranial displacement of abdominal organs or the presence of abdominal viscera within the thoracic cavity. Pleural effusion is evident in approximately 20% of cases.2,3 Ultrasonography has also been assessed as a diagnostic modality in these cases and was shown to have 93% accuracy in one report.4

Currently, the recommendation is that surgical intervention is warranted without delay after hemodynamic stabilization and treatment of other life-threatening injuries has been performed. Chronic diaphragmatic hernias are diagnosed frequently due to the sometimes delayed diagnosis in these cases.5,6 Despite concerns over these cases having a greater incidence of adhesion formation and re-expansion pulmonary injury, survival in chronic DH cases has not been shown to be different compared to those diagnosed shortly after injury. In one large study of 1674 cases mortality rates in dogs with acute and chronic DH were 27.8 and 26.2% whereas in cats those figures were 20% and 11.8% respectively.5

The surgical approach of choice for DH repair is a ventral celiotomy. Despite this, the possibility of having to perform a caudal sternotomy if significant adhesions exist in the chest should always be anticipated and so a wide clip and aseptic preparation of the entire thoracic area should be performed. Upon inspection of the diaphragmatic area, the defect is usually obvious. Herniated organs should be very gently grasped and caudal traction applied. In many cases organs will move easily out of the thoracic cavity back into the abdomen. In others, adhesions within the thorax or to the edges of the diaphragmatic defect may exist and can be broken down digitally or using electrocautery. In cases where traction does not result in abdominal organs retreating back into the abdomen two choices can be made. In some cases enlargement of the hernial defect may allow the hernial contents to become more mobile or may allow greater access to the adhesions. The other option is to perform a caudal sternotomy to provide the necessary visualization of intra-thoracic structures to allow hernial content reduction. Sternotomy was necessary in 28% of dogs and cats with chronic DH in one study.6 Once all hernial contents have been replaced in their correct anatomical location, closure of the defect is performed. In simple radial or circumferential tears where apposition of the edges can be performed without undue tension, primary apposition with either a simple interrupted or continuous line of 2-0 to 3-0 absorbable or non-absorbable suture is reasonable. The author favors not trimming the edges of the defect to minimize hemorrhage and maximize the suture holding power on tissue, which might be improved by anchorage around the scar tissue at the edge of the defect. In rare cases the defect in the diaphragm cannot be easily apposed in a tension-free manner and a reconstructive technique is required. This has been done using a large variety of different materials. The author usually favors an omentalized polypropylene mesh closure that is simple to perform and usually quite reliable. The peripheral 0.5cm of the mesh is turned over on itself and sutured to the muscular rim at the periphery of the defect using simple interrupted 3-0 non-absorbable monofilament sutures that pass through the double thickness of the mesh that has been turned over. The free cut end of the mesh is oriented towards the abdominal side to avoid the potentially sharp cut ends of the mesh damaging lung parenchyma. The abdominal component can then be covered with omentum to cushion the sharp edges as well as to cover the defect. Another elegant method of defect closure in chronic DHs with large defects is the use of a pedicled abdominal wall muscle flap from the tranversus abdominis.7 Just prior to final defect closure a catheter or red rubber tube can be inserted into the thoracic cavity and an acute drainage of air from the thorax can be performed making placement of an indwelling thoracic tube unnecessary in most cases. In cases where
significant hemorrhage occurred during dissection or lung injury occurred or is suspected to have occurred, placement of an indwelling thoracic drain may be warranted. Some authors like to re-expand the lungs very slowly after DH repair to minimize the risk of re-expansion pulmonary edema. Placement of a thoracic drain that allows slow sequential thoracic drainage over time may be advantageous if this is of great concern. Re-expansion pulmonary edema appears to occur relatively rarely in these cases with few credible reports in the literature. However, if it occurs it can be life-threatening and so should be monitored for carefully in the post-operative period.

**Pericardio-peritoneal diaphragmatic hernias**

PPDH results from the embryological failure of fusion within the most ventral aspects of the diaphragm. Communication between the pericardial sac and abdomen results although there is no communication into the thoracic cavity in these cases. Decision-making in PPDH management is controversial as many cases are asymptomatic and only detected on work-up of other conditions. In one study 41% of feline cases were detected incidentally and the abnormality occurs much more frequently in cats than dogs. Clinical signs that have been attributed to PPDH include signs of gastrointestinal and respiratory dysfunction as well as non-specific signs such as weight loss, anorexia and lethargy. Similarly to DH, organs most commonly herniated into the pericardial sac in PPDH cases include small intestine, liver and gall bladder. Thoracic radiography is sufficient for establishing the diagnosis in the vast majority of cats with classical signs including enlargement of the cardiac silhouette and visible abdominal viscera within the peritoneal sac. Cats with PPDH should always be evaluated for other congenital abnormalities such as pectus excavatum and other cardiac abnormalities which can be present in a proportion of cases.

Decision making on surgical management has been controversial as many cats can experience long-term survival with medical management. Post-operative mortality in larger studies ranges from 5.1-14% of cases. However, left untreated progression of clinical signs or acute death have been documented. In one large study outcomes of the surgical versus conservative management appeared to be similar. However it is very likely that selection bias may affect outcomes in retrospective studies of this nature as the more clinically affected cases may be more likely to be treated surgically.

Surgical treatment of PPDH involves reduction of the hernial contents back into the abdomen with subsequent closure of the diaphragmatic hernial defect. Adhesions of the liver and/or omentum to the epicardium can be encountered and these need to be very carefully broken down. The necessity for liver lobectomy to be performed needs to be considered if there is significant damage to liver lobes during reduction. Primary suturing of PPDH defects is possible in most cases.

**Hiatal herniation**

In many cases the clinical signs of hiatal herniation are both vague and non-specific and diagnostic tests are plagued with the difficulties of imaging what is a dynamic and intermittent pathology. Hiatal herniation can occur in a number of different forms with four common types being recognized in veterinary patients. Type 1 is the sliding hiatal hernia and is the classical form seen in most patients who present just with intermittent regurgitation. It is associated with cranial movement of the gastroesophageal junction (GEJ) into the thoracic cavity and affects patients to varying degrees. Type 2 is much less common and is a paraesophageal hernia where part of the stomach moves into the thorax adjacent to the normal GEJ. Type 3 is a combination of the abnormalities seen in Type 1 and 2. Gastroesophageal intussusception is another abnormality that is not a true hiatal hernia but needs to be considered as a differential diagnosis in animals where these defects are suspected.

In dogs it is known that the LES is a naturally lax structure compared to humans and that some gastro-esophageal reflux is normal in this species. What is not known is the relative contributions of muscular tone within the esophageal wall at the LES and the support afforded to the GEJ by the gastroesophageal ligament and crural muscles. It is known that in dogs with congenital or acquired sliding hiatal hernia the GEJ moves cranially and that in some normal dogs there may actually be no intra-abdominal component to the esophagus at all, meaning that the GEJ is located cranial to the diaphragm in those cases. Because of this inherent variation of normal in dogs as well as the lack of a complete understanding of the pathophysiology, diagnosis of the condition and interpretation of normal from abnormal can be challenging. The most common diagnostic test for HH used in clinical practice today is the positive contrast esophagram. Because of the dynamic nature of the disease these studies are best performed with the aid of fluoroscopy and even then there is probably a high rate of false-negative results. In our institution it is common practice to perform a second positive contrast esophagram if the first study is negative and the index of suspicion remains high for a diagnosis of HH based on signalment and clinical signs.

Treatment of hiatal herniation is controversial as medical and surgical options exist. One study has suggested that 30 days of medical management may alleviate signs in many patients and should be pursued prior to surgical management. Surgical management using a combination of hiatal plication, esophagopexy and left fundic gastropexy is the classical combination of techniques used to treat HH but has never been extensively and objectively evaluated. Response rates in the range of 80% have been reported for this surgical regimen in the small numbers of cases reported.
References