Introduction

Apart from pectus excavatum, there is very little written in the veterinary literature about thoracic wall deformities in kittens yet they are not uncommon occurrences and most large breeders have had kittens that have been affected by thoracic deformities. In the case of Burmese kittens, thoracic wall deformities are estimated to affect 3-4% of kittens born. The purpose of this article is to review the current literature and to highlight sources of further information. Thoracic wall deformities are of clinical significance when their effect is either to reduce thoracic volume to the extent that the kitten is dyspnoeic or vertebral anomalies that lead to neurologic deficits.

Vertebral abnormalities

A variety of vertebral abnormalities are described including block vertebrae (incomplete separation of the body, arches or entire vertebrae) and hemivertebrae. Hemivertebrae occur when half of the vertebral body fails to ossify resulting in unilateral, dorsal or ventral hemivertebra that lead to deviation of the thoracic spine.

Kyphosis and scoliosis have been reported to occur together. A sagittal cleft in the vertebral body leads to a butterfly vertebra. Transitional vertebrae have characteristics of two major divisions of the vertebral column and involve the last vertebra of the group i.e. at the cervicothoracic or thoracolumbar junctions. By counting the vertebrae the abnormality can be described e.g. if it is the last thoracic vertebra that has features of a lumbar vertebra then this would be described as lumbralization of T13.

Spinal dysraphism

Failure of normal neural tube closure can affect the vertebral column or spinal cord. Spina bifida is the incomplete fusion of the vertebral arches and usually affects the lumbar vertebrae. It is common in Manx cats especially ‘rumpies’. It may be associated with a meningocele (protrusion of the meninges) or myelomeningocele (protrusion of the spinal cord and meninges). The meningocele or myelomeningocele may attach to the skin causing a dimple (Figure 3). If the site is open, spinal fluid may leak on to the skin causing ulceration and the associated risk of meningitis. Clinical signs are dependent on the severity of the involvement of the spinal cord.
**Abnormalities Affecting the Rib Cage and Sternum**

A variety of rib deformities are encountered and are not uncommon, including missing ribs (usually T13), fused ribs, extra ribs (usually L1), and malformed ribs. Clinical signs associated with the defect are rare and surgical intervention unnecessary. Pectus carinatum (chicken breast) is a congenital abnormality that results in a laterally compressed thorax secondary to ventral displacement of the caudal aspect of the sternum; it has not been reported in animals.

The two most common congenital defects are flat-chested kitten syndrome (FCKS) and pectus excavatum (PE). These two conditions are sometimes confused in the literature but are distinct and likely to have very different pathogenesis. PE affects the sternum and costal cartilages. FCKS affects the whole rib cage resulting in a dorsoventral flattening of the thoracic cavity. (Figure 4)

**Pectus excavatum**

Of the thoracic wall deformities resulting in a change in rib shape, pectus excavatum is the only one described with any frequency in the veterinary literature (Hoskins 1995). The prevalence of pectus excavatum (funnel chest, chondrosternon, chondrosternal depression, koliosternia, trichterbrust, peito de sepaterio [cobbler’s chest], thorax en
Pectus excavatum is a dorsoventral narrowing of the thoracic cavity beginning around the 5th/6th rib and most severe around the 10th thoracic vertebra (occasionally the sternum will lie immediately ventral to the vertebral column) (McAnulty and Harvey 1989) (Figure 5a & b). This may result in a severe reduction in chest volume and sometimes caudal/distal displacement of the heart (Smallwood & Beaver 1977), which, in man, has been shown to cause demonstrable heart compression. Variable lung changes are seen on radiography generally the left lung is more compressed than the right due to the cardiac displacement. Decreased chest compliance is evident. In some individuals the condition can be progressive.

**Clinical Signs**
The clinical signs in affected cats are exercise intolerance, dyspnoea (inspiratory stridor) cyanosis, coughing, mild upper respiratory tract disease, weight loss / failure to gain weight and vomiting (Boudrieau and others 1990 and Soderstrom and others 1995). On auscultation heart sounds are muffled and displaced; cardiac murmurs are present in some cases.

**Aetiology**
The aetiology of the condition is unclear and may well involve multiple causes. Theories including abnormal pressure gradients in brachycephalic dogs or in upper respiratory tract obstruction, shortening of central tendon of diaphragm, abnormal intrauterine pressure, deficient muscular components derived from the septum transversum of the diaphragm, congenital thickening of the musculature of the cranial portion of the diaphragm, thickening of substernal ligament, failure of osteogenesis/chondrogenesis, arrested sternal development and rachitic influences are reviewed by Smallwood and Beaver (1977). In man pectus excavatum has been associated with other congenital diseases including Marfan’s syndrome, Noonan’s syndrome and mucopolysaccharidosis I. Pectus has also been reported associated with mucopolysaccharidosis (VII) in a cat (Schultheiss and others 2000). PE can occur as an autosomal dominant condition in man and has been reported in litter of setter cross puppies (Pearson 1973). Whether pectus excavatum in cats is an inherited condition is not known.

**Diagnosis**
The diagnosis of PE is based on the thoracic shape and radiographic changes (Figure 6a & b). More objective parameters have also been suggested including the frontosagittal index (FSI) and vertebral index (VI) (Table 1).
**TREATMENT**

Surgical intervention may be necessary if the defect is severe and associated with significant clinical signs and abnormal physiological parameters (blood gas analysis). The application of an external splint allowing the sternum to be reshaped by using sutures placed around the sternum and through the rigid splint. The technique may also serve to stretch or tear soft tissues that are acting to pull the sternum ventrally. The optimal time for splinting is when the kitten is around 14 days old. The value of surgical intervention if there is an associated cardiac abnormality is unknown.

The kitten is placed in dorsal recumbency, and a piece of mouldable splint made in a U shape with 4-6 pairs of holes positioned so that the distance between adjacent holes is slightly greater than the width of the sternum. Blind sutures are placed around the sternum and may also incorporate the costal cartilage to reduce the risk of the suture pulling through the soft sternebral bone (Figure 7). The needle is kept close to the dorsal surface of the sternum. Once all sutures are placed, the splint is padded, fitted and the sutures passed through the pre-drilled holes. Large (0–2) absorbable or non-absorbable suture material preferably with a swaged-on taper-point needle is recommended. The splint should be left in place for 10–21 days. Suture abscesses, superficial dermatitis and skin abrasions are common. The risk of haemorrhage associated with piercing the lung, heart or internal thoracic vessels can be minimised by careful positioning of the kitten, keeping the needle close to the sternum and paying attention to the phase of respiration. Kittens often exhibit a growth spurt after correction of the defect. It is therefore important to check the kitten weekly and adjust the splint as necessary. If correction is required in a mature cat, partial sternectomy or a wedge osteotomy may be necessary.

### Flat-chested kitten syndrome

FCKS is poorly reported in the veterinary literature (Sturgess and others 1997) but "flatties' are well recognised by breeders. Flat chests are seen in many breeds of cats but are more commonly encountered in Orientals and in particular Burmese cats (T.J.Gruffydd-Jones personal communication). In Burmese, a survey conducted by one of the breed societies (The Burmese Cat Club), suggests that around 3–4% of Burmese kittens born are flat chested. The condition is usually not present at birth and may be accompanied by a cranial thoracic vertebral kyphosis (Figure 8). In those kittens that survive, the deformity will become less obvious as the kitten grows and is frequently unnoticeable in the adult. Little change in the vertebral deformities occurs with age. The condition is variable in its appearance from very mildly affected kittens reported to be flat chested for short periods (hours to days) to severely affected kittens where the prognosis is very guarded. There is a higher incidence of PE in kittens with FCKS than normal kittens (4 of 46 kittens with FCKS also had PE).

### CLINICAL DESCRIPTION

The ventral ribcage is flattened along almost its entire length (Figure 9a & b) and in some more severely affected kittens will curl inwards. Flat chested kittens therefore have a flat rather than S-shaped sternum, the rib deformities beginning more cranially creating a sharp angular deformity at the costochondral junction (Figure 10). A marked dip in the spine over the shoulder blades (thoracic kyphosis) is seen in up to 30% of cases. The defect is not always present at

<table>
<thead>
<tr>
<th></th>
<th>FSI†</th>
<th>VI†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0.7–1.3 (1.00)</td>
<td>12.6–18.8 (15.0)</td>
</tr>
<tr>
<td>Mild P.E.</td>
<td>2.0</td>
<td>&gt;9.0</td>
</tr>
<tr>
<td>Moderate P.E.</td>
<td>2.0–3.0</td>
<td>6.0–9.0</td>
</tr>
<tr>
<td>Severe P.E.</td>
<td>&gt;3.0</td>
<td>&lt;6.0</td>
</tr>
</tbody>
</table>

* Ratio of the thoracic width at T10 as measured on a DV/VD radiograph and the distance from the centre of the ventral surface of T10 or vertebrae overlying the deformity and the nearest point on the sternum.
† Ratio of the distance from the centre of the dorsal surface of the vertebral body overlying the deformity to the nearest point of the sternum and the DV diameter of the centrum of same vertebra.
Birth and may develop during the first week or two of life (Figure 11). If the kitten survives, the flattening usually becomes less noticeable beyond 3-4 months of age, although the kyphosis may remain. Single, multiple or all kittens within a litter may be affected. FCKS occurs unpredictably even when previous litters using the same mating have not shown evidence of a problem. The flattening can occur suddenly over a few hours or worsen over a number of days. On physical examination, the most obvious finding is an angular deformity at the costochondral junction.

**Clinical Signs**

Clinical signs will depend on the severity of the defect but are associated with a kitten that has a reduced thoracic capacity, hence dyspnoea is common. Kittens will be less active and often fail to thrive (Figure 12) as they have increased energy demands and are reluctant to suckle due to their dyspnoea or lose out in competition for the queen’s milk supply.

**Aetiology**

Statistical analysis performed on the BCC surveys (M.Rehahn, R.Robinson, and A.Seville personal communications) has also provided strong evidence that there is a heritable component to the condition. The initial statistical evidence pointed to a simple, autosomal recessive mode of inheritance. This is likely to be an over simplification as other factors, including non-heritable, environmental influences, also appear important in the phenotypic expression of flat chestedness. The speed at which changes in the thoracic shape occur, its transient nature (in some kittens) and flexibility of skeletal system in the new-born make it unlikely that this condition is a primary skeletal deformity or connective tissue abnormality. This would tend to suggest that some myopathy involving the intercostal and diaphragmatic muscles may well be involved. The role of taurine in FCKS has been investigated in Burmese kittens; affected kittens showed high whole blood and skeletal muscle taurine than non-affected Burmese kittens. The role of taurine in FCKS, however, remains unclear.
**Diagnosis**

Diagnosis is based on physical examination. No objective radiographic parameters have been reported, however, the FSI and VI are likely to be outside the normal range.

**Treatment**

A variety of treatments have been suggested but there have been no good studies to assess the efficacy. Maintaining growth rate does seem to be of crucial importance, affected kittens should be given supplementary feeding by bottle or stomach tube. Medical therapy has focused on dietary supplementation. Some kittens do appear to respond to potassium supplementation but the response is not consistent. Splinting techniques have also been advocated and anecdotal reports suggest good success rate using cardboard toilet rolls or plastic drinks bottles to provide lateral thoracic compression forcing the sternum ventrally (Figure 13). It is, however, essential that there is no inward curling of the ribs as lateral splinting could worsen the defect. A recent report purporting to be the treatment of a Siamese cat with PE (Crigel and Moissonnier 2005) involving a sternal realignment with a pin and external splint actually looks likely correction of a kitten with FCKS and PE.

**Prognosis**

Kittens that have minor defects and those that maintain their body weights generally have a good prognosis. Regardless of the original severity, kittens that are alive beyond 3 weeks of age will generally survive and thrive.

**Advice to breeders**

Thoracic wall deformities will occur sporadically as congenital defects in kittens of all breeds. Whether some forms of PE in cats are inherited, as in man, is unknown. It would be prudent to consider whether to continue breeding from a queen or stud where there is a history of PE occurring in kittens from more than one litter. FCKS is likely to have a hereditary component but inheritance may well be complex with significant environmental influences. Because of the prevalence of FCKS in some oriental breeds, it would be impractical to stop breeding from stud cats or queens.
that have single episodes of FCKS. If a queen has had more than one litter affected by FCKS, particularly if several members of the litter are affected, retiring the queen should be considered. Whether to continue breeding from a stud cat that has had multiple litters with kittens affected by FCKS is more difficult as surveys have indicated that the majority of Burmese stud cats, for example, have sired affected litters. Currently the best advice to breeders is to
- Avoid using any medications including herbal and alternative remedies and food supplements during pregnancy or nursing unless they have been specifically shown to be safe, or the severity of the disease necessitates their use.
- Avoid inbreeding (close line breeding), since FCKS seems to appear more often in lines with limited genetic diversity.
- Avoid breeding from a sire or queen that has had a number of litters affected with FCKS.
- Never breed from a cat that is a recovered flat-chested kitten.
- Ensure good nutrition during pregnancy and nursing.

Conclusions

Accurate information on the types, causes, incidence, therapy and prognosis for kittens affected by thoracic wall deformities is lacking. A group has been set up to try and answer some of these questions with a focus on FCKS due to its prevalence. As the first task, veterinary health care professionals are encouraged to report thoracic wall deformities that they encounter in their practice in order to generate a body of data that will begin to answer some of these questions.

References and Further Reading