Mitral dysplasia in bull terriers

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INTRODUCTION

Mitral dysplasia may be defined as an abnormal development of the mitral valve apparatus. This may affect the mitral valve itself, the papillary muscles or the chordae tendineae. Mitral insufficiency is the usual haemodynamic consequence of the lesion, but occasionally mitral stenosis may be evident. Mitral dysplasia appears to be a significant problem in bull terriers in the UK.

PREVALENCE

Mitral dysplasia appears to be becoming more common in the dog. In a recent Veterinary Cardiovascular Society survey of congenital heart disease in the UK (Matic, 1993), mitral dysplasia was the third most common defect (after aortic stenosis and patent ductus arteriosus), affecting 14% of dogs reported after investigation of congenital heart disease. Reported breeds with mitral dysplasia in this survey were the bull terrier (59%), German Shepherd dog (8%), Cavalier King Charles spaniel (6%) and Springer spaniel (4%). The main breeds affected with mitral dysplasia reported in the literature in the USA, UK and Australia are Great Danes, German Shepherd dogs and bull terriers (Hamlin *et al.*, 1965; Hamlin & Harris, 1969; Dear, 1971; Liu & Tilley, 1975; Lord *et al.*, 1975; Atwell, 1979; Malik & Church, 1988; Dukes, 1991; Fox *et al.*, 1992).

Of bull terriers presented to me in first opinion practice, at shows or as referred cases, 43 out of 91 examined had heart murmurs (47.3%) (Fig. 1). Eight of these dogs have still to be investigated further, but were all felt clinically to have mitral regurgitant murmurs. Of the dogs receiving further investigation, 31 dogs had mitral dysplasia with mitral and tricuspid regurgitation; two dogs had isolated subvalvular aortic stenosis; one puppy had lethal acrodermatitis (an inherited metabolic disease of bull terriers) and mitral dysplasia and stenosis (McEwan, 1993); and one dog has mitral dysplasia with mitral stenosis and aortic stenosis – this latter dog now belongs to the author (Dukes, 1991). From this screening there does not appear to be any sex predisposition or relationship to coat colour for congenital heart disease in bull terriers.

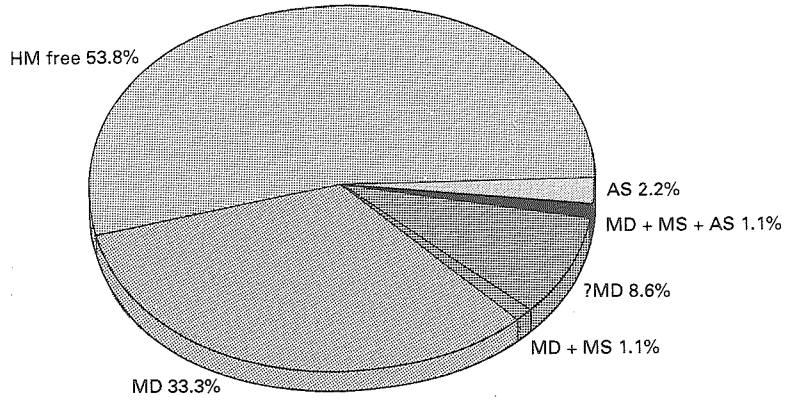


Fig. 1 Heart disease in bull terriers. HM free, heart-murmur free; MD, mitral dysplasia; MD + MS, mitral dysplasia with mitral stenosis; ?MD, probable mitral dysplasia but not confirmed by echocardiography; MD + MS + AS, mitral dysplasia with mitral stenosis and aortic stenosis; AS, aortic stenosis.

AETIOLOGY AND PATHOPHYSIOLOGY

Mitral dysplasia results from a developmental abnormality of the atrioventricular cushions which usually results in valvular incompetence (Noden & DeLahunta, 1985). This also commonly affects the right atrioventricular (tricuspid) valve, although this is less haemodynamically significant clinically (Fox *et al.*, 1992). Mitral regurgitation results in volume overloading of the left atrium and left ventricle and compensatory eccentric hypertrophy and hyperkinesis, exactly as in acquired degenerative mitral valve lesions (valvular endocardiosis) (Keene, 1988). Increased left atrial pressures cause pulmonary venous congestion and pulmonary oedema. If the dysplastic mitral valve is also stenotic, gross left atrial dilatation with elevated pressure is recognized. Persistently increased pulmonary capillary wedge pressure may cause pulmonary arterial hypertension and right-sided heart failure, aggravating the right-sided volume overload associated with any tricuspid regurgitation. The atrial stretch can result in atrial (supraventricular) tachydysrhythmias such as atrial fibrillation. Some patients with long-standing disease may develop myocardial failure.

CLINICAL PRESENTATION

Most bull terriers with mitral dysplasia are identified when a heart murmur is detected at vaccination or health checks, or by cardiologists at regional bull terrier meetings or shows. The breed is not easy to

is required in some cases to detect these murmurs. Many affected dogs are asymptomatic throughout their lives, but may go into left-sided heart failure or have syncopal episodes as they get older – possibly due to superimposed degenerative valvular lesions. Some dogs present at a young age with syncope, exercise intolerance, dysrhythmias and left-sided heart failure. Signs of left-sided heart failure in this breed are usually shortness of breath and reverse sneezing – they appear to cough rarely even with gross left atrial enlargement.

Other congenital heart disease identified by the author in screening and investigating bull terriers with heart murmurs is aortic stenosis, which is usually subvalvular. Some dogs have mitral dysplasia together with aortic stenosis. Some puppies with lethal acrodermatitis have mitral dysplasia also but it is unlikely that the two conditions are linked (Fig. 2) (McEwan, 1993).

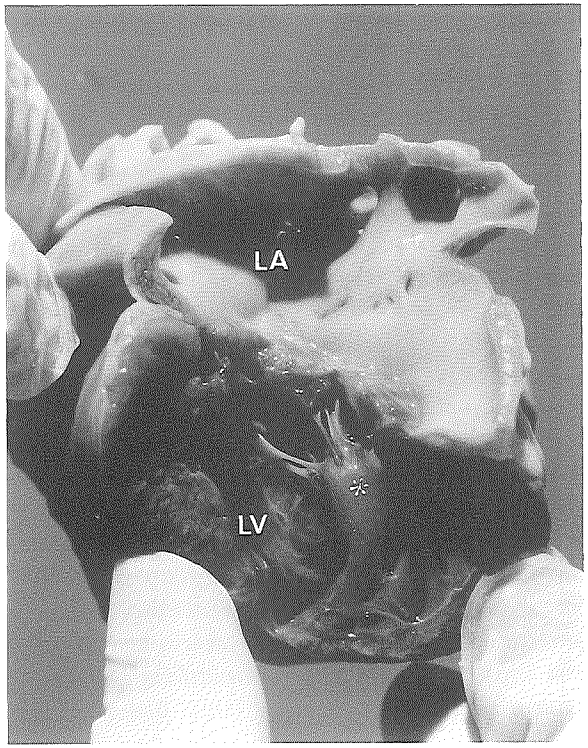
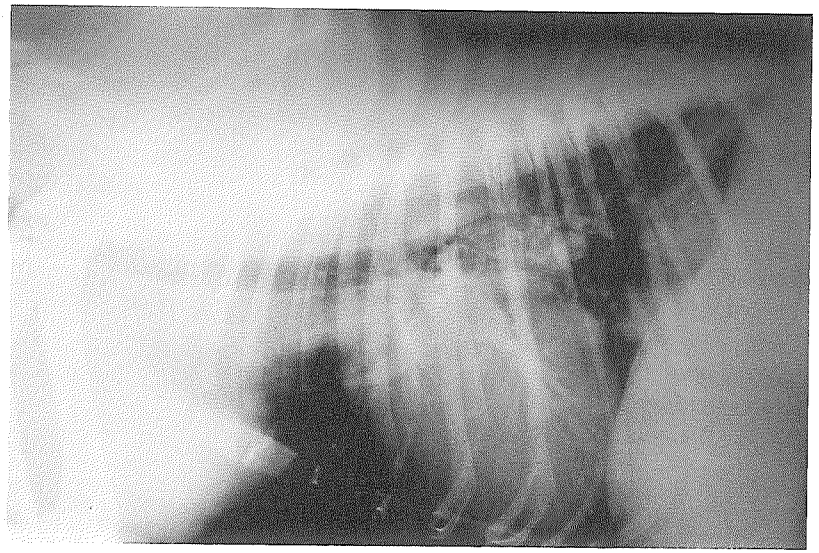
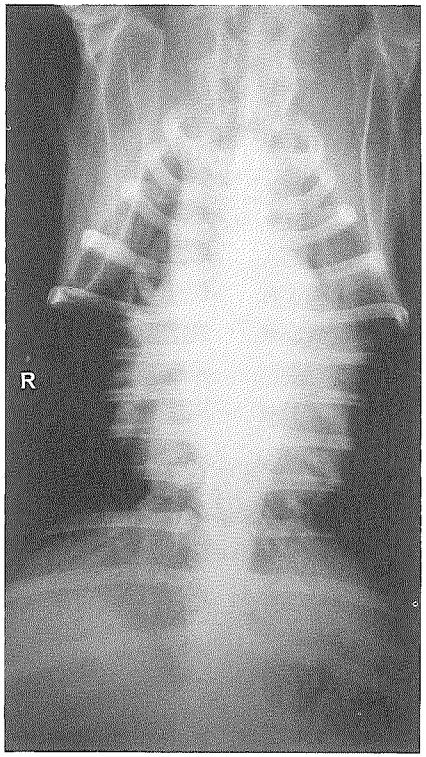


Fig. 2 Heart taken at post mortem following euthanasia of a 14-month-old bull terrier puppy bitch with lethal acrodermatitis and mitral dysplasia with mitral stenosis. The mitral valve leaflets are short, thickened and nodular. Gross papillary muscle hypertrophy (*) is evident with short thick chordae tendineae attaching them to the mitral valve leaflets. LA, left atrium; LV, left ventricle. Acknowledgements:



(a)



(b)

Fig. 3 Lateral (a) and dorsoventral (b) radiographs obtained from a bull terrier with mitral dysplasia. Marked left atrial enlargement with division and compression of the mainstem bronchi and left ventricular enlargement is evident in the lateral view. There is generalized interstitial pulmonary oedema, accentuated in the perihilar area. Pulmonary venous congestion may be appreciated from the lateral view.

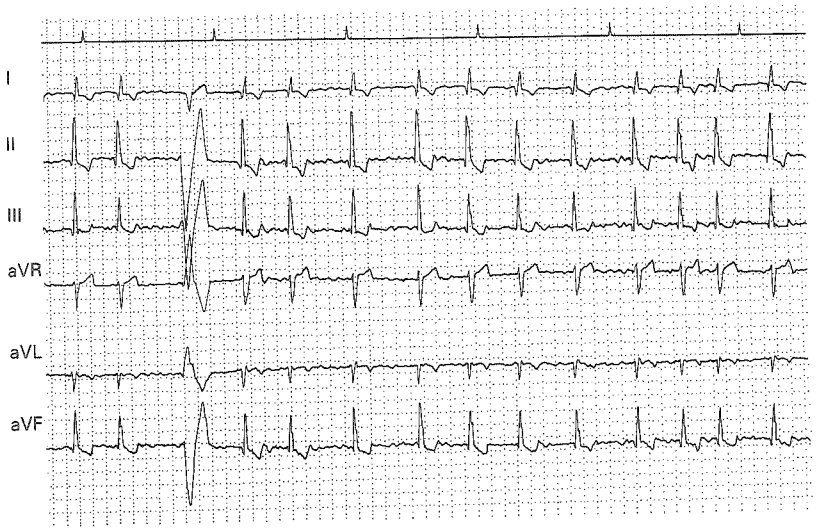


Fig. 4 A 6-lead simultaneous ECG obtained from the author's own dog which has mitral dysplasia with mitral stenosis and aortic stenosis. Atrial fibrillation with coarse fibrillation waves are apparent. The ventricular ectopia was related to digitoxicity at the time of this ECG. At the onset of this dog's atrial fibrillation, marked clinical deterioration was noted. Because of the adverse nature of this rhythm in mitral stenosis, an attempt to convert him back to normal sinus rhythm was initially made, with quinidine sulphate. He became hypotensive, tachycardic and generally unwell on this drug. His ventricular rate is instead controlled with digoxin–he has survived for 5 years with left-sided heart failure and 3.5 years with atrial fibrillation, defying his prognosis! He is, however, markedly exercise and stress intolerant with syncopal episodes.

Clinical signs

Usually, cardiac output signs are good, with pink mucous membranes, brisk capillary refill and good volume femoral pulse. A holosystolic or pansystolic murmur is detected over the mitral valve area which appears to radiate to the apex and to the right side. It is rare for a diastolic murmur of mitral stenosis to be detected, although this has been described (Tashjian & McCoy, 1960). Atrial fibrillation or other dysrhythmias may be detected clinically. Auscultatory evidence of pulmonary oedema may be detected in patients with congestive cardiac failure.

Radiography

Lateral and dorsoventral views should be obtained. This is the best method of ascertaining the haemodynamic significance of any lesion,

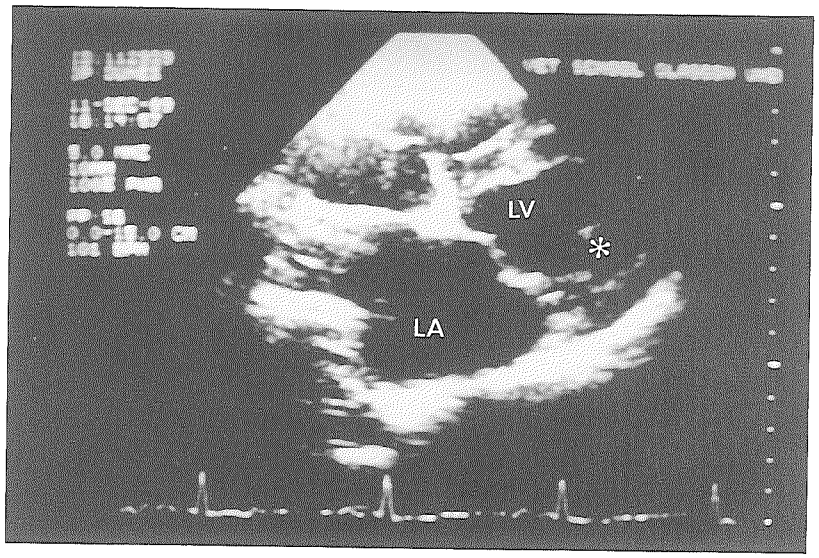


Fig. 5 A right parasternal long-axis view of a bull terrier with mitral dysplasia. Gross left atrial enlargement is apparent, with dilated pulmonary veins. The mitral valve is thick, irregular and echogenic. A prominent hypertrophied papillary muscle (*) and short thick bright chordae tendineae are also apparent. The short chordae tendineae may allow mitral regurgitation by restricting upward systolic motion of the valve, restricting closure (Liu & Tilley, 1975). LA, left atrium; LV, left ventricle.

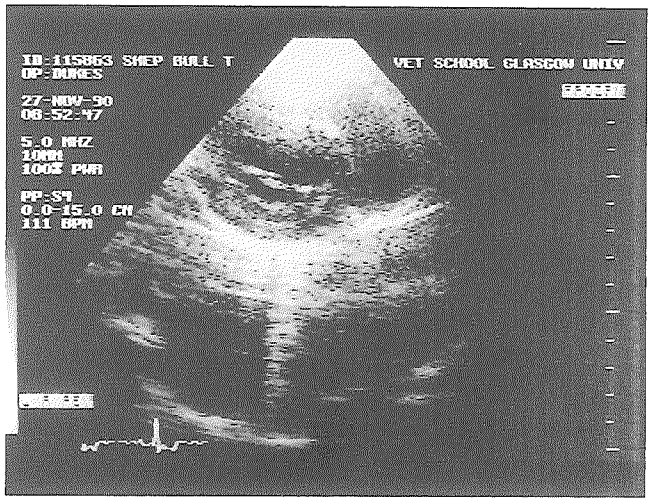
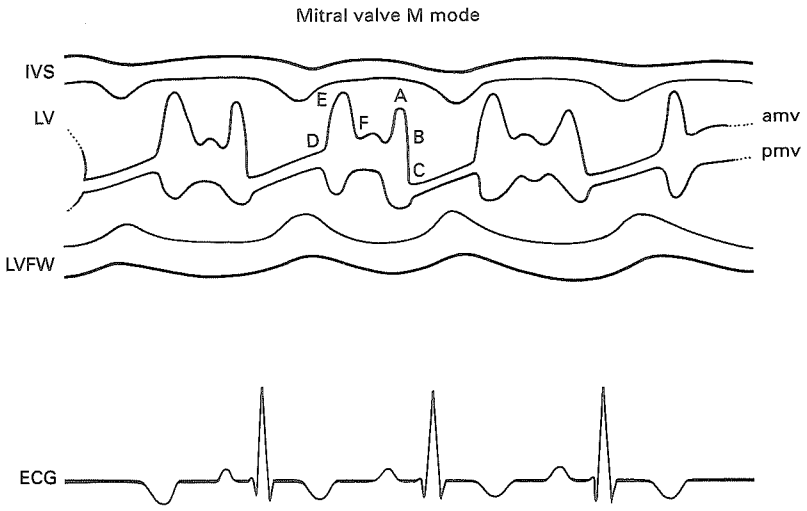
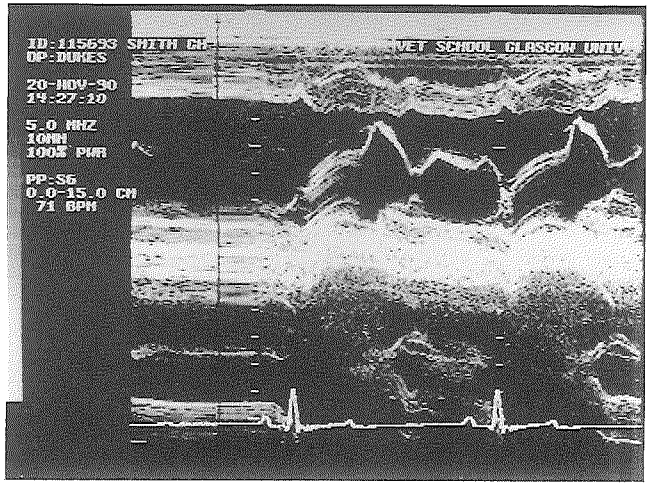


Fig. 6 A right parasternal short-axis view of a bull terrier with mitral dysplasia at the level of the mitral valve – this appearance (dynamic in real time) is the 'fish mouth view' of the mitral valve. The leaflets are thick, nodular and bright. In mitral



(a)

Fig. 7 (a) Labelled diagram of mitral valve motion in a normal M-mode echocardiogram. Normally the anterior mitral valve inscribes an M pattern reflecting the two phases of left ventricular diastolic filling (the E peak corresponding to early passive filling (after the T wave on the ECG) and the A peak to active atrial contraction (after the P wave on ECG)). Patients in atrial fibrillation show no A peak. Normal passive diastolic filling initiates between the D–E points as the valve opens. Normally, the mitral valve partially closes in mid-diastole, with the E–F slope. Atrial contraction augments left ventricular filling as the valve opens again at the A point, and closes at the onset of systole (A–C slope). Normal anterior systolic motion of the closed mitral valve apparatus is appreciated between the C–D points (Feigenbaum, 1981a; Pollick & Sutton, 1989). The posterior leaflet of the mitral valve normally mirrors this motion, transcribing more of a W pattern as it moves posteriorly, maximally separated from the anterior leaflet during the two diastolic filling phases at the E and A points. IVS, interventricular septum; LV, left ventricular lumen; LVFW, left ventricular freewall; amv, anterior leaflet of the mitral valve; pmv, posterior leaflet of the mitral valve. (b) Mitral valve M-mode obtained from a normal dog without any mitral valve disease illustrating the points detailed. (c) In mitral stenosis, there is delayed or no partial closure (less steep E–F slope), and often an increased dependence on atrial contraction in patients in normal sinus rhythm, so the A peaks dominate the E peaks (altered E:A ratio). The posterior mitral valve (pmv) motion usually inscribes a W motion, reflecting the M of the anterior leaflet. Patients with mitral stenosis appear to have the posterior leaflet (arrowed) also moving anteriorly, concordantly with the anterior leaflet, as the two leaflets are fused, unable to move independently. This is classical of human patients with mitral stenosis (Feigenbaum, 1981a,b; Braunwald, 1992; Weisfeldt *et al.*, 1992).



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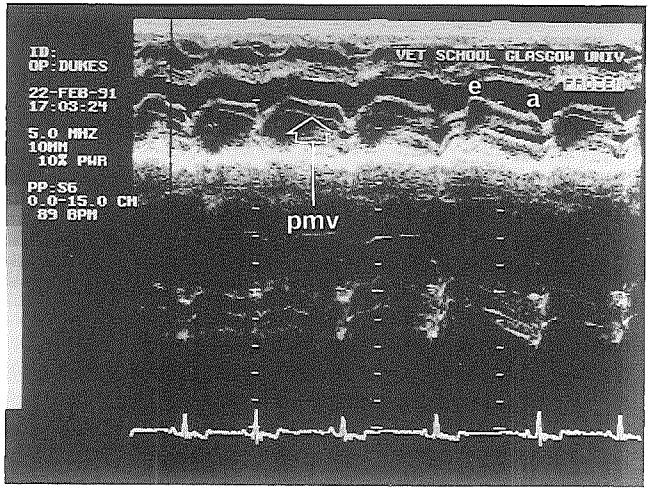


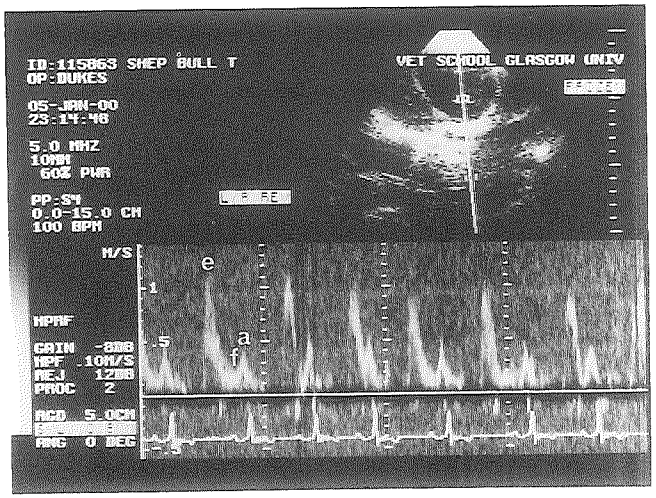
(c)

Fig. 7 *Continued*

terriers with mitral dysplasia, there is left atrial and left ventricular enlargement. The main stem bronchi may be divided and compressed by the left atrial enlargement (Fig. 3). If tricuspid regurgitation is also haemodynamically significant, right atrial and right ventricular enlargement may also be identified.

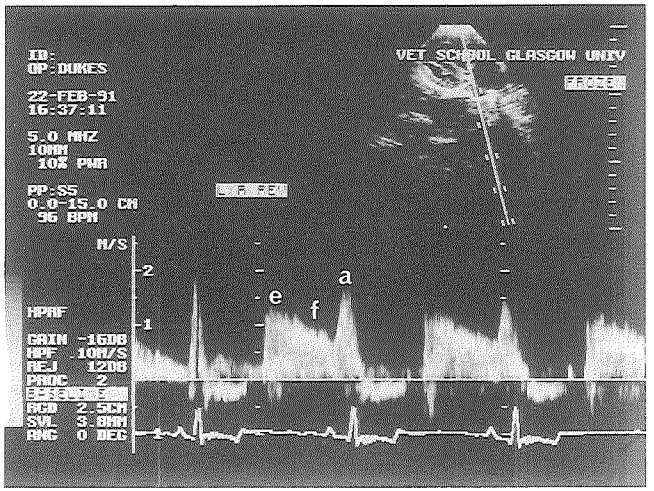
Electrocardiography

An ECG is important if any dysrhythmia is identified. Supraventricular tachydysrhythmias, particularly atrial fibrillation, are secondary to atrial



(a)

Fig. 8 (a) Mitral M-mode and pulsed wave or continuous wave spectral Doppler studies of mitral inflow provide much information not only about mitral valve function but about left ventricular function, especially in diastole, in human and veterinary patients (Feigenbaum, 1981c, d; Borow, 1989; Yellin, 1990; Dukes 1991). M-mode illustrates the classical concordant motion of the posterior mitral valve leaflet with the anterior mitral valve leaflet seen in mitral stenosis, distinguishing this condition from those of left ventricular diastolic dysfunction (Braunwald, 1992). Doppler provides more haemodynamic information across the mitral valve. There is normally biphasic diastolic filling of the left ventricle, and the passive diastolic filling phase (the E peak) is usually dominant in individuals with normal sinus rhythm at normal heart rates. The A peak is the flow subsequent to active atrial contraction. Obviously patients in atrial fibrillation lose this contribution. At very fast heart rates, E and A peaks may be summated (recognized frequently in cats). Patients under the influence of catecholamines will show increasing dominance of the A peaks. Patients with any diastolic dysfunction with reduced left ventricular compliance will have dominant A peaks. Dogs with mitral regurgitation with volume-overloaded left atrium and left ventricle will have a normal E:A peak ratio, but high velocity mitral inflow – this could be considered as a relative mitral stenosis, but there is no prolongation of pressure half-time – the E–F slope (deceleration) is steeper than normal, the pressure half-time is short (Goldberg *et al.*, 1988a) indicating no anatomical mitral stenosis. This bull terrier with mitral dysplasia manifested as solely mitral regurgitation illustrates this point. a, A peak (after P wave of ECG); e, E peak (>1.0 m/s); f, mid-diastolic closure of valve: shows E–F slope as indicator of pressure half-time. (b) The spectral Doppler findings with mitral stenosis show high velocity inflow affecting both the E and A peaks. The E:A peak ratio may be reversed as increased dependence on atrial contraction is required to achieve adequate ventricular filling (Braunwald, 1992). The pressure half-time is prolonged (Goldberg *et al.*, 1988b), with shallow E–F slope and prolonged deceleration. The pressure half-time can be defined as the time taken for the pressure gradient across the valve to halve. These criteria of mitral stenosis are taken from the human literature (Feigenbaum, 1981a,b; Kisslo *et al.*, 1988b; Pollick & Sutton, 1989; Braunwald, 1992) and appear to extrapolate to bull terriers with mitral stenosis. a, A peak (not abnormal E:A ratio – greater dependence on atrial contraction for ventricular filling); e, E peak; f, mid-diastolic closure of mitral valve (not closed with mitral



(b)

Fig. 8 *Continued*

electrocardiographic criteria of left atrial enlargement and left ventricular enlargement. QRS complexes may be notched if the ventricular hypertrophy or arteriosclerosis causes microscopic intramural infarction.

Echocardiography and Doppler

Two-dimensional echocardiography reveals a number of anatomical abnormalities of the mitral valve apparatus in mitral dysplasia in bull terriers. The papillary muscles are large and hypertrophied. The mitral valve leaflets are thick, bright and sometimes irregularly nodular. The chordae tendineae are short and thick (Fig. 5). These changes are supported by necropsy findings (Fig. 2). Observation of mitral valve movement in long-axis view may detect mitral prolapse and subjectively may give the impression of mitral stenosis with poorly opening leaflets – this is usually more apparent in the short-axis view of the mitral valve – the so-called 'fish mouth' view (Fig. 6). Left atrial and left ventricular enlargement with eccentric hypertrophy is evident and confirmed by M-mode echocardiography with comparison to reference dimensions for body weight (Bonagura *et al.*, 1985). Myocardial function and contractility (fractional shortening) may be subjectively assessed and measured. Careful M-mode study of the mitral valve motion should indicate any mitral stenosis (Fig. 7a–c).

Pulsed or continuous-wave spectral Doppler or colour flow Doppler echocardiography of all the valves in bull terriers with mitral dysplasia frequently shows incompetence of all valves, usually more than the trivial physiological insufficiency commonly seen in normal animals.

flow or spectral mapping within the atria and assessing the regurgitant jet areas (Blumlein *et al.*, 1986; Goldberg *et al.*, 1988a; Kisslo *et al.*, 1988a). Colour flow Doppler echocardiography will indicate probable mitral stenosis with the diastolic mitral inflow showing a bright aliased core (where the Nyquist limit for colour is exceeded) (Kisslo *et al.*, 1988b; Pollick & Sutton, 1989). Pulsed or continuous wave Doppler of mitral inflow is diagnostic of mitral stenosis (Fig. 8a, b).

MANAGEMENT AND TREATMENT OF MITRAL DYSPLASIA (Table 1)

Many bull terriers with mitral dysplasia are asymptomatic with class I heart disease. No specific treatment or management measures are necessary in these patients, although they should be monitored every 3–6 months by the veterinary surgeon. Patients with heart failure should be exercise restricted depending on their class of heart failure and dietary sodium restriction may reduce the excessive renal retention of sodium and water.

Patients with mitral regurgitation and left-sided congestive cardiac failure may be managed in the same way as those with acquired mitral incompetence (valvular endocardiosis). Diuretics are important in controlling pulmonary oedema. Patients with mitral stenosis will often require aggressive diuresis – rather than very high doses of frusemide, it is often better to use combination diuresis, such as potassium-sparing diuretics (e.g. spironolactone or amiloride) with frusemide. Renal function and electrolytes (especially potassium) should be monitored in patients requiring high doses of diuretics.

Vasodilators are also important in mitral regurgitation. Arteriodilatation reduces the afterload on the left ventricle, improves the forward stroke volume and reduces the mitral regurgitant fraction – this in turn decreases left atrial pressure and helps to control pulmonary oedema (Kittleson, 1988). Arteriodilators are *contra-indicated* in mitral stenosis and aortic stenosis – this is why careful Doppler study is necessary to exclude these conditions prior to therapeutic decision making. In aortic or mitral stenosis, arteriodilatation increases the pressure gradient across the stenotic valves, exacerbating the severity of the stenosis. Venodilators (such as nitroglycerine ointment (Percutol, Cusi)) should not have this deleterious effect; by increasing the capacitance vessels, blood is directed away from the cardiopulmonary circuit and this helps alleviate critical pulmonary oedema with class IV congestive failure.

Digoxin may be used for control of the ventricular rate in dogs with atrial fibrillation or supraventricular tachydysrhythmias and for myocardial failure. The dose should be titrated carefully, based on body

Table 1 Drug dose rates for mitral dysplasia in dogs

Drug	Dose rate	Indications and contra-indications
Diuretics		
Frusemide (generic) 40 mg tablets	2–4 mg/kg divided bid	Pulmonary oedema. Control of sodium and water retention
Spironolactone (generic) 50 mg, 100 mg tablets	2–4 mg/kg divided bid	Additional diuresis. Potassium sparing. Do not use with ACE inhibitor drugs
Amiloride (Midamor, Morsen) 5 mg tablets	0.1–0.2 mg/kg sid or divided bid	Used with other diuretics for additional diuresis. Potassium sparing. May possibly be used with caution with ACE inhibitors
Hydrochlorothiazide (Vetidrex, Ciba) 25 mg tablets	2–4 mg/kg divided bid	Mild diuretic – may be effective in early congestive failure. May use in addition to a potassium sparing diuretic
Digoxin		
Digoxin (Lanoxin, Wellcome) 0.0625 mg, 0.125 mg and 0.25 mg tablets	0.01–0.02 mg/kg divided bid ($0.22\ mg/m^2$)	Atrial fibrillation and supraventricular tachydysrhythmias to reduce ventricular rate. Myocardial failure (mild positive inotrope). Reduce dose (or use digitoxin) if renal dysfunction. Reduce dose for cachexic and hypoproteinaemic patients. Monitor for efficacy/digitoxicity
Vasodilators: Angiotensin Converting Enzyme (ACE) Inhibitors		
Enalapril Cardiovet, Intever 1, 2–5, 5, 10, 20 mg tablets	0.3–0.5 mg bid	Mitral regurgitation and congestive cardiac failure. Myocardial failure. Monitor renal function. Contra-indicated for mitral/aortic stenosis
Captopril (Acepril, Squibb) 12.5 mg, 25 mg tablets	0.25–2 mg/kg divided tid	Mitral regurgitation and congestive cardiac failure. Myocardial failure. Monitor renal function. Contra-indicated for mitral/aortic stenosis
Vasodilators		
Hydralazine (Apresoline, Ciba) 25 mg tablets	1–3 mg/kg every 8 hours	Mitral regurgitation and congestive cardiac failure. Titrate from low doses. Contr-aindicated for mitral/aortic stenosis
Prazosin (Hypovase, Invicta) 0.5 mg, 1 mg, 2 mg tablets	<15 kg: 0.5–1 mg every 8–12 hours. >15 kg: 1–2 mg every 8–12 hours	Mitral regurgitation and congestive cardiac failure. Contra-indicated for mitral/aortic stenosis
Nitroglycerine (Percutol, Cusi) ointment	1 inch on hairless skin per 20 kg body weight every 4–6 hours. Use for <5 days	Class IV congestive failure/ fulminant pulmonary oedema. Used with intravenous frusemide (Lasix, Hoechst)

digoxin assay should be measured 8 hours post-pill after day 7 of digitalization to ensure levels are within the therapeutic range without digitoxicity.

DISCUSSION

Mitral dysplasia is described in bull terriers by a number of authors (Hamlin *et al.*, 1965; Atwell, 1979; Malik & Church, 1988; Fox *et al.*, 1992). Most cases described have mitral regurgitation without stenosis. There appears to be some controversy about whether the mitral valve apparatus was grossly abnormal in some reports or whether it was a dilated mitral valve annulus with secondary mitral incompetence (differentiated from mitral insufficiency by Hamlin and Harris (1969)). The only one of the six Great Danes reported by Hamlin and Harris (1969) to die and be necropsied was said to have normal mitral valve apparatus and the other five dogs were said to have 'outgrown' the lesion. The cases described by Hamlin *et al.* (1965) were said to have reduced surface area of the mitral valve leaflets but the surviving cases were hypothesized as having a temporary mitral incompetence secondary to idiopathic dilatation of the left ventricle. However, these authors state that the three dogs which died (including one bull terrier) had 'permanent mitral regurgitation' although necropsy details were not provided. In the series of mitral incompetence cases described by Dear (1971), the only abnormality of the mitral valve apparatus was dilatation of the mitral valve annulus. Abnormal mitral valve apparatus was described by Liu and Tilley (1975). It may be that some of the earlier cases were mitral incompetence secondary to dilated cardiomyopathy, mentioned by Dear (1971). Lord *et al.* (1975) used left ventricular angiography to exclude myocardial failure and these cases were confirmed by necropsy to have pathology of the mitral valve complex. The bull terrier cross described by Atwell (1979) had grossly abnormal mitral valve apparatus with one normal and one vestigial papillary muscle.

The ready availability of echocardiography now allows non-invasive assessment of the left side of the heart and the mitral valve apparatus. Conditions with myocardial failure with secondary papillary muscle dysfunction such as idiopathic dilated cardiomyopathy or endocardial fibroelastosis (Fox, 1988) can be readily excluded and in many cases, gross abnormalities of the mitral valve apparatus can be imaged, as recognized in bull terriers (Fig. 5).

Mitral stenosis occurs infrequently as a congenital abnormality in children, with a variety of deformities described (Friedman, 1992). The most common cause of mitral stenosis in humans is rheumatic fever

(Weisfeldt *et al.*, 1992). The first mention of mitral stenosis in the veterinary literature is documented by Buchanan (1965) who mentioned one case diagnosed in 1957 by D. K. Detweiler (unpublished observations) and quoted Tashjian and McCoy (1960) with a case of mitral stenosis as a presumed acquired degenerative lesion. Mitral stenosis may be more common than appreciated, however, and with increased use of echocardiography and Doppler and greater experience of veterinary echocardiographers, this condition may become more frequently recognized (Fox *et al.*, 1992). The M-mode echocardiographic features of mitral stenosis in humans are well described in the literature (Feigenbaum, 1981a,b; Pollick & Sutton, 1989; Braunwald, 1992). The M-mode echocardiographic features of mitral stenosis are described in a dog as an acquired post-inflammatory lesion (Bonagura & Herring, 1985) and in presumed congenital mitral stenosis (Pipers *et al.*, 1981).

Doppler echocardiography reflects the haemodynamic effects of mitral stenosis (Pollick & Sutton, 1989). The characteristic Doppler findings of mitral stenosis are well described in the human literature (Kisslo *et al.*, 1988b; Pollick & Sutton, 1989; Braunwald, 1992). The first veterinary report of mitral stenosis Doppler findings was of the author's own dog (Dukes, 1991) which was initially diagnosed by the author in 1989 and is the Scottish dog alluded to by Fox *et al.* (1992) together with details of two other cases of bull terriers with mitral stenosis with characteristic Doppler and M-mode findings. One other case of mitral stenosis with mitral dysplasia in a pup with lethal acrodermatitis is included in this last paper. Other than these case reports, it appears that most cases of canine congenital mitral valve disease are associated primarily with mitral regurgitation, which is true for humans also (Friedman, 1992).

It is important to exclude mitral stenosis in a patient with mitral dysplasia, as vasodilators are contra-indicated in this condition, whereas they are a mainstay of the management of mitral regurgitation (Kittleson, 1988). Careful echocardiographic and Doppler studies should be conducted and mitral stenosis looked for particularly in this breed. Patients with mitral stenosis have greater dependence on atrial contraction to achieve ventricular filling – loss of atrial kick in human patients reduces cardiac output by 20% (Braunwald, 1992). Development of atrial fibrillation is associated with sudden clinical deterioration and a grave prognosis (Fig. 4).

Although there is some evidence that Great Danes may 'outgrow' the mitral insufficiency (Hamlin & Harris, 1969), this is refuted by Dear (1971), with a grave prognosis given for this condition. Bull terriers, even if initially asymptomatic, appear to develop problems later in

the mitral insufficiency. Once dogs develop clinical signs of left-sided heart failure, there appears to be a progressively deteriorating course.

Further screening of large numbers of dogs throughout the country, probably by veterinary cardiologists at bull terrier shows, is required to ascertain the full extent of the problem. If the bull terrier breed club recognizes that mitral dysplasia is a problem and members decide that attempting to eliminate it is a priority, then a form of certification of dogs free from heart disease (possibly published) and analysis of pedigrees of affected dogs (confidential) may determine the mode of inheritance and outline a breeding programme which would attempt to reduce the problem within the breed. Such control measures have been instituted by the Boxer Breed Council for aortic stenosis (Luis Fuentes, 1993) and the Cavalier King Charles Spaniel Club for premature mitral valve disease (endocardiosis).

CONCLUSIONS

From the limited screening of bull terriers so far, mitral dysplasia does appear to be a significant problem within the breed in the UK. Colleagues are urged to carefully auscultate young bull terriers presenting to them and to investigate any heart murmur further.

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The differential diagnosis of non-gastric vomiting in the dog

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Vomiting is a common presenting complaint in canine practice and represents one of the consequences of a large number of conditions. These may be broadly divided into conditions of the stomach, conditions of the remainder of the gastrointestinal tract, and extragastrointestinal conditions. A good understanding of the differential diagnosis and pathophysiology of vomiting, good history taking, a thorough physical examination, and appropriate use of other tests will ensure optimal management of each case. The purpose of this paper is to review the investigation of vomiting in the dog where the underlying disease process does not primarily involve the stomach.

CENTRAL CONTROL AND PHYSIOLOGY OF THE VOMITING REFLEX

Vomiting is a reflex activity under both conscious and unconscious control. Initiation of vomiting occurs within the vomiting centre which is located in the reticular formation of the medulla oblongata. It receives afferent supply from the chemoreceptor trigger zone (CRTZ), the vestibular nuclei, the higher brain and, via the vagi and sympathetic afferents, from the mucosal and serosal surfaces of the gastrointestinal tract peritoneum (Fig. 1). The CRTZ lies in the medulla oblongata, on the ventrolateral surface of the fourth ventricle. The capillaries in the area of the CRTZ are fenestrated, therefore the cells lie outside the blood–brain barrier, and are sensitive to emetic substances within plasma. It is through these inputs that a number of conditions that do not involve the stomach can cause vomiting (Table 1).

Triggering of the vomiting reflex leads to a sequence of events terminating in the act of vomition. There is hypersalivation, breathing deepens, the hyoid bone moves rostrally to open the cranial oesophagus, the glottis closes and the soft palate lifts to close the nasopharynx. Then the diaphragm contracts together with the abdominal muscles to increase intra-abdominal pressure. Finally the cardiac sphincter opens and gastric contents are expelled via the oesophagus and mouth.

SIGNALMENT AND HISTORY

The age, breed and sex of an animal are critical points of initial in-