

# Echocardiographic parameters in 14 healthy English Bull Terriers

CA O'LEARY<sup>a</sup>, BM MACKAY<sup>a</sup>, RH TAPLIN<sup>b</sup> and RB ATWELL<sup>a</sup>

**Objective** To determine the range of various cardiac parameters using echocardiography in apparently normal, healthy English Bull Terriers.

**Design** Fourteen English Bull Terriers were selected for study. Cardiac auscultation of the parents of these dogs was normal. Echocardiographic examination of one parent of each animal showed: no mitral or aortic valve abnormalities; no myocardial lesions; no two dimensional evidence of fixed or dynamic left ventricular outflow tract obstruction; and no systolic aortic or left ventricular outflow tract turbulence on colour flow Doppler examination. The 14 selected dogs did not have arrhythmias or murmurs, and on echocardiographic examination had similar findings to their parents. Systolic blood pressure was measured in all dogs and they had no clinical evidence of Bull Terrier polycystic kidney disease or Bull Terrier hereditary nephritis.

**Procedure** All dogs were auscultated and subjected to a sequential global echocardiographic assessment of the heart, including two dimensional long and short axis, and colour flow Doppler interrogation of the mitral and aortic valves. Dimensional measurements, including those from the left atrium, aortic annulus and left ventricle, were taken from a right parasternal window, and derived values such as fractional shortening, stroke volume and left atrial to aortic annulus ratio were calculated. Peak systolic aortic velocity was measured from the left parasternal window using two dimensional-guided pulsed wave Doppler with angle correction. Systolic blood pressure was measured using a Doppler monitor. The absence of Bull Terrier polycystic kidney disease was determined using renal ultrasonography, and of Bull Terrier hereditary nephritis using urinary protein to creatinine ratio.

**Results** These 14 dogs had greater left ventricular wall thickness and smaller aortic root diameters than those reported as normal for other breeds of comparable body size. Left atrial dimensions were also larger, however this may have been due to the "maximising" method of measurement. These apparently normal English Bull Terriers also had higher aortic velocities than those reported for other breeds, possibly due to a smaller aortic root diameter or other anatomic substrate of the left ventricular outflow tract, lower systemic vascular resistance, or breed-specific "normal" left ventricular hypertrophy. While these dogs were selected to be as close to normal as possible, the breed may have a particular anatomy that produces abnormal left ventricular echocardiographic parameters.

**Conclusion** These echocardiographic parameters may be used to diagnose left ventricular outflow tract obstruction and left ventricular hypertrophy, and inaccurate diagnoses may result if breed-specific values are not used.

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<sup>a</sup>School of Veterinary Science, The University of Queensland, Queensland, 4072

<sup>b</sup>Division of Science and Engineering, Murdoch University, Western Australia 6150

English Bull Terriers have a high prevalence of murmurs,<sup>1,2</sup> and echocardiographic and necropsy reports have shown a high prevalence of mitral regurgitation, mitral stenosis and LVOTO.<sup>1-5</sup> These defects are likely to have a genetic basis, as they do in degenerative mitral valve disease in the Cavalier King Charles Spaniel and Dachshund,<sup>6,7</sup> and in LVOTO in the Golden Retriever, Newfoundland and other breeds.<sup>8-10</sup>

Such cardiac disorders can lead to CHF and sudden death, making early detection important in the individual animal.<sup>4</sup> Early detection is also important in breeding animals to facilitate effective disease control, because undiagnosed or mild cardiac defects can be transmitted to offspring before the parent develops clinical signs.<sup>11</sup> Some pups may also express more severe disease than did the parents, confusing breed selection programmes.<sup>9</sup>

While an experienced veterinary practitioner, using careful auscultation, can detect most cardiac diseases,<sup>12</sup> definitive diagnosis of any defects and detection of subclinical disease requires a knowledgeable echocardiographic examination. However, normal echocardiographic parameters vary between breeds, as do body size and somatotype.<sup>13-15</sup> Indeed, echocardiographic reference ranges derived from some dog breeds may be misleading for others.<sup>16</sup> Breed-specific, or at least somatotype-specific, normal echocardiographic parameters, such as left ventricular and atrial measurements and AoV, are required to make more accurate cardiac diagnoses and assess disease severity. This need is particularly so in English Bull Terriers, as LVOTO

Ao	Aortic annular diameter
AoV	Aortic velocity
BP	Blood pressure
BSA	Body surface area
CHF	Congestive heart failure
FS%	Fractional shortening
HR	Heart rate
IVSd	Diastolic interventricular septum thickness
IVSs	Systolic interventricular septum thickness
LA	Left atrial diameter
l/a	Long axis view
LA:Ao	Ratio of long axis diameters of left atrium to aortic root
LVfracd	Myocardial fraction, indicating LVH
LVFWd	Diastolic left ventricular free wall thickness
LVFWs	Systolic left ventricular free wall thickness
LVH	Left ventricular hypertrophy
LVId	Diastolic left ventricular internal dimension
LVIs	Systolic left ventricular internal dimension
LVOT	Left ventricular outflow tract
LVOTO	Left ventricular outflow tract obstruction
PG	Pressure gradient
PI	Prediction interval
s/a	Short axis view
SD	Standard deviation
SV	Stroke volume

appears to be a complex 'end-point' lesion that varies in anatomical expression and dynamic severity, making differentiation of affected and unaffected dogs difficult. Indeed, establishing whether a dog is normal can be very difficult. This study's aim was to determine echocardiographic parameters in apparently normal English Bull Terriers.

## Materials and methods

### Case selection

Fourteen English Bull Terriers were selected after each animal's parents were found to be normal on cardiac auscultation. One parent of each animal was found on echocardiographic examination to be free of: mitral or aortic valve abnormalities; myocardial lesions; lesions indicating the presence of LVOTO including 2D obstruction; and systolic aortic or LVOT turbulence on colour flow Doppler examination. Aortic velocity was not used to diagnose LVOTO. These animals were from eight bloodlines. Two sets of two dogs were full-siblings, and six animals were half-siblings to one other animal. All animals were on oral *Dirofilaria immitis* prevention therapy.

Body surface area was calculated in m<sup>2</sup> using the formula below, where W was the animal's weight in grams;

$$BSA = (10.1 \times W^{2/3}) / 10^4$$

### Renal ultrasonography

Animals were excluded from the study if Bull Terrier polycystic kidney disease was diagnosed, due to its association with cardiac disease in English Bull Terriers, humans and in genetically modified mice with a similar polycystic kidney disease.<sup>17-20</sup> Animals were examined using renal ultrasonography. Three or more cysts distributed between both kidneys and a family history of Bull Terrier polycystic kidney disease was considered diagnostic for this disease.<sup>21</sup>

### Urine testing

A urine sample was collected from all dogs, and all animals had a protein creatinine ratio  $\leq 0.3$ .<sup>21,22</sup> This excluded animals with preclinical Bull Terrier hereditary nephritis, a disease which may be associated with hypertension and LVH, and lead to renal failure.<sup>23</sup>

### Auscultation

Cardiac auscultation was performed for 5 minutes in a quiet room, by one clinician, bilaterally over the apex beat and at the thoracic inlet to ensure absence of murmurs and arrhythmias.

### Echocardiography

Echocardiographic examination was carried out at The University of Queensland Veterinary Teaching Hospital, in a dark, quiet room, with dogs loosely restrained by their owners. An ATL Ultramark 9, and either a 2.25 or 5MHz phased array transducer, were used as appropriate for the dog's size and chest width. A hole in the table allowed the transducer to be placed on the dog's cardiac impulse from below. All images were optimised for image quality, size and symmetry. All dimensional measurements were obtained from a right parasternal window.

All dogs underwent sequential global echocardiographic assessment of the heart, including 2D long and short axis, and colour flow Doppler interrogation of the mitral and aortic valves. This was followed by measurement of the left atrium in 2D mode with the long axis value being the maximal atrial diastolic diameter when measured parallel to a line through the

mitral annulus.<sup>24</sup> The left atrial short axis value was the maximal atrial diastolic diameter measured at the level of the aortic valve. Aortic long and short axis dimensions were taken in 2D mode at the opening of the aortic valve, at the level of the aortic valve annulus. The LA:Ao was calculated from these long axis measurements. Left ventricular measurements were taken from 2D-directed M mode short axis views at the level of the chordae tendinae, with the beam directed perpendicular to the septum. The leading edge method of measurement was used. Derived values such as fractional shortening and stroke volume were computed by the machine from short axis measurements taken at the papillary level, and the Teicholz formula was used to calculate stroke volume. Values recorded were averages from three to five cycles. As the velocity was measured just distal to the aortic root, it is referred to as aortic velocity. The peak systolic AoV was measured using 2D-guided pulsed wave Doppler with angle correction, from the left parasternal window. The effect of ventilation was minimised as peak AoV was recorded from three to five cycles. The Doppler cursor was placed at the point of maximal opening of the aortic valve and aligned with the direction of flow. Evidence of turbulence in the left ventricular and aortic outflow tract was assessed using colour flow Doppler, continuous wave Doppler and pulsed wave spectral Doppler displays, after manipulation to avoid aliasing.

### Systolic blood pressure measurement

Systolic BP was measured using a Doppler blood pressure monitor (model 811-BTS, Parks Medical Electronics Inc), after the animal was placed in lateral recumbency and calm for at least 20 min. A 5cm paediatric cuff, being 40% of the metatarsal circumference, was used,<sup>25</sup> and measurements were taken from the upper (right) distal hindlimb. Once the dog was acclimatised, the average of at least two measurements from each dog was recorded as the final systolic BP.

### Statistical analysis

Statistical analysis was performed using Minitab 12.2 for Windows (Minitab Inc). Regression analysis and Students *t* tests, with a significance level of 5% ( $P < 0.05$ ), were used to determine the relationship between echocardiographic measurements and body surface area or weight. Ninety five percent prediction intervals were calculated for echocardiographic measurements, values derived from these measurements, systolic BP and HR. Statistical analyses of results involving LVH used a mathematically derived indicator of LVH, the myocardial fraction or LVfracd, calculated from diastolic short axis measurements where IVSd was the thickness of the diastolic interventricular septum and LVFWd the diastolic left ventricular free wall, and LVID the diastolic left ventricular internal dimension;  $LVfracd = (IVSd + LVFWd) / (IVSd + LVFWd + LVID)$ .

## Results

### Health status of selected cases

Of the 14 English Bull Terriers selected, ten were entire females and four were entire males. They ranged in age from 9 to 30 months, and 18 to 30.2kg in weight. No murmurs or arrhythmias were detected by auscultation. On echocardiographic examination no mitral or aortic valve abnormalities, myocardial lesions, 2D evidence of fixed or dynamic LVOTO, or systolic aortic or LVOT turbulence on colour flow Doppler examination, were detected.

### Cardiac parameters

In this study, most cardiac measurements had no statistically significant relationship with weight, hence these results are presented as 95% prediction intervals (95% PI, Table 1). The use of gender status did not provide any additional predictive value above weight for calculating cardiac parameters. Data appeared to be normally distributed except LA:Ao, which had one outlying result due to one dog with a large LA. Thirteen animals had LA:Ao values between 1.4 and 1.9, with the outlier's value being 2.4.

Three cardiac parameters measured in this study were affected by weight (Tables 2 and 3). These included the LA diameter measured in the long and short axis, and the left ventricular internal diameter measured in systole (LVIs). Many reports describe results for body surface area rather than weight, however in this study body surface area did not provide any predictive advantage over weight.

### Aortic velocity and flow patterns

On 2D echocardiographic examination no animals had fixed or dynamic LVOTO lesions, mid-systolic closure of the aortic valve, or late systolic fluttering of the aortic valve. Colour flow Doppler echocardiography did not show diastolic aortic regurgitation or systolic aortic or LVOT turbulence.

Pulsed wave Doppler echocardiography was also used to examine blood flow in the LVOT and aorta for evidence of turbulence. At rest all 14 dogs had normally shaped spectral display envelopes. Seven of these 14 dogs also had spectral displays showing laminar flow (Figure 1), however the other seven showed spectral broadening or non-laminar blood flow in the LVOT and aorta, even at rest (Figure 2).

In three of the seven dogs with laminar flow at rest, transition to non-laminar flow was observed once the HR increased from a mean of 118/min to a mean of 161/min, and AoV from a mean of 1.8m/s to a mean of 1.95m/s, respectively. However, a fourth animal (from this group of seven) had laminar flow at a HR of 180/min and AoV of 1.95m/s. Colour flow Doppler did not show turbulence in these four animals, even at elevated HR. The mean resting AoV in these 14 dogs was  $1.9 \pm 0.2$  m/s, with the maximum recorded being 2.3m/s (Figure 3).

Analysis did not demonstrate a significant relationship between AoV and most of the measured cardiac parameters. Only AoV was negatively related to LVFWd ( $P = 0.048$ ,  $r = -0.536$ ). While Ao l/a was negatively associated with LVFWd ( $P = 0.045$ ,  $r = -0.542$ ), other relationships between Ao diameter and various cardiac parameters were not significant. There were no significant relationships between systolic BP and other measured cardiac parameters.

## Discussion

### Left atrial dimensions

Left atrial dimensions in this study were measured using 2D echocardiography through the main body of the chamber. This method is more applicable than measuring the LA at the level of the aortic root using M mode, as this measures through the right auricle.<sup>24,26</sup> However, M mode LA measurement has been used in many other studies, and this methodological difference may explain the larger LA found in English Bull Terriers in this study compared to those in breeds with similar or larger adult weights.<sup>14,15,27</sup> Perhaps more significantly, measurements in these English Bull Terriers were similar to those reported in the general dog population using the 2D method.<sup>28</sup>

### Aortic annular diameter

Aortic annular diameter in this study was also measured in 2D mode, whereas most other reports have measured this parameter in M mode.<sup>29</sup> As both these methods maximise Ao measurements, the results should be comparable. Despite this, the Ao in these 18 to 30 kg English Bull Terriers was smaller than expected for the range of weights. Indeed the Ao in this study were comparable in size with the Ao in the 8 to 19 kg Welsh Pembroke Corgi,<sup>14</sup> and smaller than Ao in dogs of the same weight in the general population.<sup>27,28</sup> A narrow aortic annulus and aortic sinotubular junction have also been reported in normal Boxers and Golden Retrievers, breeds with a high prevalence of LVOTO.<sup>11,30,31</sup> Recently, Boxers with AoS and subvalvular LVOTO have also been reported with narrow aortic diameters.<sup>30,32</sup> Thus, the smaller Ao in the English Bull Terriers in this study could be normal for this group of dogs or could reflect the preclinical LVOTO substrate in this breed.

### Ratio of left atrial to aortic annular diameter

In this study, the prediction interval for LA:Ao calculated from 2D long axis measurements in English Bull Terriers was 1.3 to 2.1. While another study using the 2D method of measurement reported a mean LA:Ao ratio of 2.4,<sup>28</sup> others using 2D measurement reported values of 1 to 1.6 in normal Cavalier King Charles Spaniels and other breeds, and values above 2.1 in dogs with decompensated heart failure.<sup>24,26,33</sup> In one of these latter studies, the Ao was measured at various points between the aortic annulus and the wider proximal part of the aortic sinus, and another used an oblique view of the LA and Ao.<sup>24,33</sup> Studies using M mode measurements that maximise the aortic root at the expense of the LA reported LA:Ao ratios in normal dogs were  $< 1.3$ , and often approximately 1.<sup>26,27</sup> Hence, differences in measurement methods may account for the increased LA:Ao in English Bull Terriers compared with varying breeds of similar and larger body weights,<sup>15,29,34,35</sup> or the smaller Ao found in these English Bull Terriers may contribute to the increase in LA:Ao.

### Diastolic left ventricular measurements

Compared to other breeds and dogs in the general population of similar weight, English Bull Terriers in this study had thicker left ventricular wall measurements and similar left ventricular chamber widths when assessed in diastole.<sup>14,15,27,34,35</sup>

The statistically significant negative correlation between the LVFWd and the AoV ( $r = -0.536$ ,  $P = 0.048$ ), was unexpected. An increase in AoV due to LVOTO would, if anything, be expected to be associated with an increase in left ventricle wall thickness, and a positive correlation between AoV and left ventricle wall thickness. The small negative correlation in this study does not support this expectation and suggests the cause of elevated AoV was not associated with secondary left ventricle wall thickening due to LVOTO in these dogs. The association was weak however, and may have been of spurious significance. The lack of a positive relationship between left ventricular measurements and AoV does not rule out the presence of secondary mild LVH due to LVOTO, as LVOTO does not always cause LVH until later in the disease, when obstruction has progressed to a critical stimulatory level.

In contrast, LVFWd was negatively correlated with Ao l/a ( $P = 0.045$ ). This finding suggests a narrowed aortic root may contribute to increased afterload and thicker left ventricle walls, possibly constitutes some degree of obstruction to blood leaving

the heart in dogs in this study, and may contribute to the elevated AoV observed in these dogs. This association was also weak, and requires further study for confirmation. While both these associations were weak, they could be further tested by stressed measurements where the outflow tract is under pharmacologically induced sympathetic loads.

Ratios of IVSd/LVId and IVSd/LVFWd were used to determine whether asymmetric septal hypertrophy is present, or to assess the extent of compensatory hypertrophy resulting from LVOTO.<sup>27,36</sup> The IVSd/LVId and IVSd/LVFWd in English Bull Terriers in this study were similar to those of normal dogs,<sup>27</sup> suggesting asymmetric septal hypertrophy did not occur, or that the walls were equally normal or altered in width.

*Fractional shortening*

Fractional shortening is an index of systolic function and is calculated from the LVId and LVIs. It assumes normal ventricular morphology, afterload, preload and ventricular contractility,<sup>29</sup> varies between breeds, and is negatively correlated with weight.<sup>13,37</sup> Fractional shortening was lower than the normal range in dogs from the general population of similar body weight.<sup>14,15,27</sup> The FS% in these English Bull Terriers was similar to that in the Newfoundland, but higher than the normal range for the Irish Wolfhound, Great Dane and Spanish Mastiff.<sup>34,35</sup> The lower than expected FS% may have been due to a smaller LVId or larger LVIs than other dogs of comparable size. Assuming normal preload, it suggests these dogs may have increased afterload (despite the lower systolic BP), abnormal ventricular geometry, or decreased contractility. Alternatively, these values maybe normal for the breed.

*Systolic blood pressure*

Systolic BP in normal dogs may be up to 180mmHg in a clinical setting, however normal mean systolic BP in young dogs is more likely to be 120 to 130mmHg.<sup>38</sup> In this study, systolic BP measurements were lower than expected. However, measurements were taken after a 20 minute adjustment period, dogs were handled by their owners, and were passive (but not asleep). Systolic BP may also vary according to breed.<sup>39,40</sup> No association was found between the relatively low systolic BP and the relatively high AoV observed in this study, suggesting that the AoV is associated with some other factor.

*Diagnosis of left ventricular outflow tract obstruction*

LVOTO is likely to be prevalent in English Bull Terriers,<sup>1,3</sup> and diagnosis can be difficult, particularly as this is a difficult breed to auscultate and to examine echocardiographically. Murmurs are not always present in cases of LVOTO and several echocardiographic criteria may be used to diagnose the disorder, making it difficult to identify animals of this breed that could be unequivocally defined as 'normal'.<sup>3,10</sup>

LVOTO may be characterised by fixed obstruction, where a structural narrowing is present in the LVOT below the aortic valve. Alternatively, obstruction may be dynamic and characterised by systolic bulging of the interventricular septum, elevated and variable outflow tract blood flow velocities and pressure gradients, and by systolic anterior motion of the mitral valve.<sup>11</sup> In this study, fixed and dynamic lesions and abnormal mitral valve motion were not observed.

A fixed or dynamic lesion is not always visible on echocardiographic examination.<sup>41</sup> Obstruction may be characterised by diffuse, subtle, non-focal narrowing of the outflow tract,<sup>11,41</sup> and be difficult to detect. One report imaged LVOTO lesions in about one third of affected dogs, though the method used to confirm which dogs were affected was not stated.<sup>42</sup> Hence, while in no animals in the current study was a fixed or dynamic LVOTO lesion detected, the presence of undetectable lesions could not be excluded.

Another criterion used to diagnose LVOTO includes diastolic aortic regurgitation and an elevated AoV.<sup>41</sup> Aortic regurgitation is rare in young dogs, and because it occurs in most cases of LVOTO, is a sensitive indicator of mild obstructive pathology.<sup>43-45</sup> Aortic regurgitation in LVOTO is due to the high velocity jet from the obstruction causing secondary cusp thickening, stiffening, and subsequent regurgitation, extension of the LVOTO lesion to involve the aortic valve, post-stenotic

**Table 1. 95% prediction intervals for echocardiographic parameters that were significantly related to weight (kg) in the 14 English Bull Terriers in this study.**

Parameter <sup>a</sup>	Mean	Standard Deviation	Significance	95% Prediction Interval
Ao l/a (cm)	1.9	0.3	0.496	1.3-2.5
Ao s/a (cm)	2	0.2	0.105	1.6-2.4
LA l/a:Ao l/a	1.7	0.2	0.271	1.3-2.1
LVId (cm)	3.8	0.3	0.096	3.2-4.4
IVSs (cm)	1.3	0.2	0.951	0.9-1.7
IVSd (cm)	1	0.2	0.715	0.6-1.4
LVFWs (cm)	1.2	0.1	0.394	1.0-1.4
LVFWd (cm)	1	0.1	0.391	0.8-1.2
IVSd/LVFWd	1.1	0.2	0.549	0.7-1.5
IVSd/LVId	0.3	0.04	0.598	0.2-0.4
LVfracd	0.3	0.03	0.675	0.2-0.4
FS%	32.5	4.5	0.086	24-41
SV (mL)	38.2	7.3	0.573	24-53
Systolic BP (mmHg)	105	14	0.581	77-133
HR (beats/min)	130.9	22.5	0.747	86-176
Weight (kg)	22.9	3.7	*	*
AoV (m/s)	1.9	0.2	0.373	1.5-2.3

<sup>a</sup>Ao aortic annular diameter, l/a long axis, s/a short axis, LA left atrial diameter, LVI left ventricular internal dimension, d diastolic, IVS interventricular septum, s systolic, LVFW left ventricular free wall thickness, LVfracd left ventricular fraction and indicates LVH, FS% fractional shortening, SV stroke volume, BP blood pressure, HR heart rate, AoV aortic velocity.

**Table 2. Descriptive statistics and formulae for the range of echocardiographic parameters significantly related to weight (kg) in the 14 English Bull Terriers in this study.**

Parameter <sup>a</sup>	Mean	P value	Standard Deviation	95% Prediction Interval
LA l/a (cm)	kg x (1.75 + 0.0606)	0.003	0.22	= kg (1.31 + 0.0606) to kg (2.19 + 0.0606)
LA s/a (cm)	kg x (1.64 + 0.0605)	0.029	0.32	= kg (0.99 + 0.0605) to kg (2.29 + 0.0605)
LVIs (cm)	kg x (1.46 + 0.05)	0.028	0.26	= kg (0.93 + 0.05) to kg (1.99 + 0.05)

<sup>a</sup>LA left atrium diameter, l/a long axis view, s/a short axis view, LVIs left ventricular internal dimension during systole

**Table 3. Range of echocardiographic parameters significantly related to weight in the 14 English Bull Terriers in this study.**

Parameter <sup>a</sup>	Weight (kg)		
	20	25	30
LA l/a (cm)	2.5-3.4	2.8-3.7	3.1-4.0
LA s/a (cm)	2.2-3.5	2.5-3.8	2.8-4.1
LVIs (cm)	1.9-3.0	2.2-3.2	2.4-3.5

<sup>a</sup>LA is left atrium diameter, l/a is long axis view, s/a is short axis view, LVIs is left ventricular internal dimension during systole

dilation of the aorta, or aortic valvular endocarditis.<sup>11,45,46</sup> Aortic regurgitation was not observed in dogs in this study. Thickened aortic valves and early systolic partial aortic valve closure also occur in LVOTO,<sup>47</sup> but neither was observed.

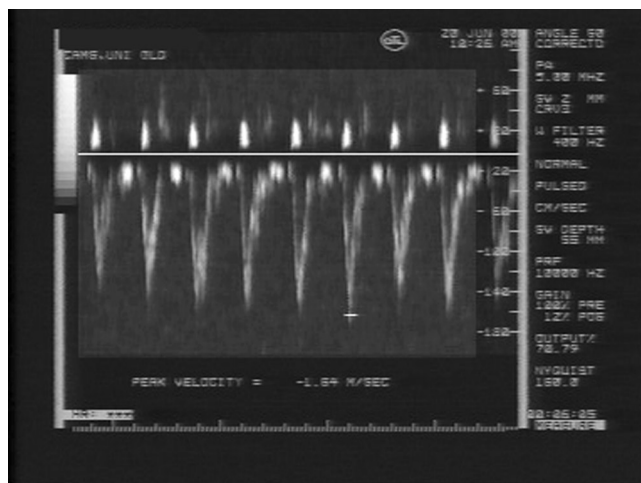
Further criteria used to diagnose LVOTO are systolic LVOT turbulence distal to the obstruction and an elevated AoV in a calm dog.<sup>11,41</sup> Turbulent flow is characterised by disappearance of the normally clear region within the Doppler spectral envelope,<sup>4</sup> and by appearance of a multiple coloured mosaic pattern when using colour flow Doppler.<sup>26</sup>

In this study, evidence of apparent LVOT turbulence was present on pulsed wave Doppler examination but not on colour flow Doppler examination. The apparent turbulence on pulsed wave Doppler may have been echocardiographic artifact, as these sources of information are qualitative. Alternative verification is needed to diagnose LVOTO or an incorrect diagnosis may be made.<sup>48</sup> Echocardiographic artifacts may occur in normal dogs and appear similar to patterns produced by turbulent flow.<sup>48</sup> Mild spectral disturbances may also appear if the beam is not properly aligned with blood flow or an excessive angle of interrogation (greater than 20 degrees to the flow) is used.<sup>41,49</sup> Hence, if the angle of interrogation exceeds 20 degrees, as may occur in English Bull Terriers due to their unique conformation, spectral dispersion may appear in some apparently normal dogs.

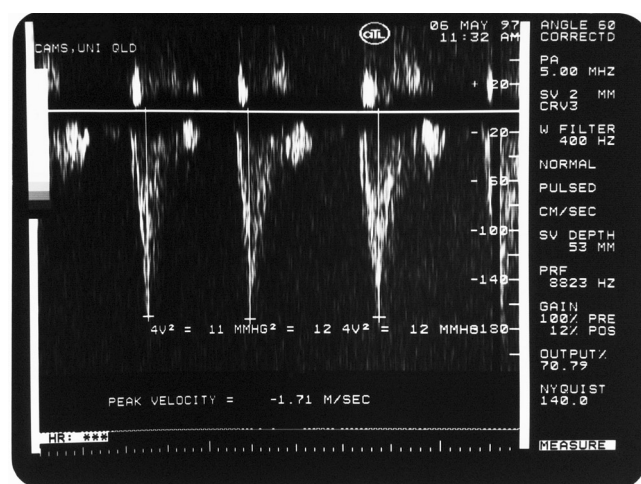
Physiological turbulence may occur in normal dogs, especially if large and excited.<sup>50</sup> An alternative explanation for the apparent turbulence found in some animals in this study may be physiological non-laminar flow occurring due to breed-specific characteristics such as the increased AoV or the narrow Ao found in this study. However, some of the dogs in this study may have had very mild LVOTO and LVOT turbulence, only detectable using pulsed wave spectral Doppler displays.

A final method for diagnosing LVOTO in unexcited dogs is an elevated AoV without LVOT turbulence or other flow or valve abnormalities,<sup>41,51</sup> however, no maximal upper limit for AoV has been categorically established in the "so-called" non-excited normal dog.

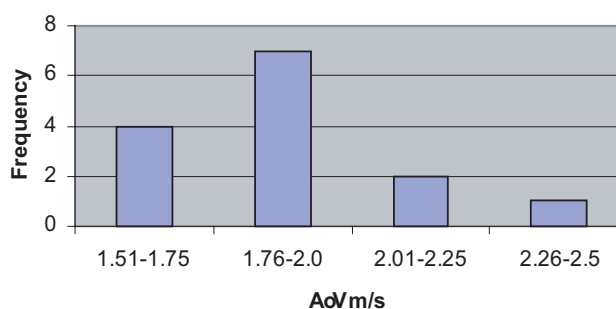
Previous studies in normal unsedated dogs have reported mean peak systolic AoV (measured using pulsed wave Doppler from a left parasternal window and not always stating whether angle correction was used) to be 1.15 to 1.7m/s, with values greater than 2m/s uncommon.<sup>41,51,52</sup> Hence, studies have generally agreed that AoV above 2 to 2.5m/s without LVOT turbulence or other flow or valve abnormalities indicate LVOTO.<sup>41,51</sup> Similarly, AoV between 1.7 to 2.5m/s have been reported to be equivocal for LVOTO.<sup>41,46</sup> English Bull Terriers in this study were equivocal for LVOTO using these criteria, derived from multiple breeds and authors.



**Figure 1. Pulsed wave spectral Doppler echocardiograph (video download) showing normal laminar blood flow in the aorta, with red blood cells in each region moving in the same direction at similar velocities. A thin acceleration envelope and relatively sharp peak are seen. The deceleration limb is approximately twice the thickness of the acceleration limb and less steep.**



**Figure 2. Pulsed wave spectral Doppler echocardiograph (hard copy download) showing spectral broadening as a result of non-laminar or turbulent flow in the aorta, with red blood cells showing a wide distribution of velocities in different directions. The thin acceleration envelope has also been lost.**



**Figure 3. Frequency and range of aortic velocity (AoV) in the 14 English Bull Terriers in this study.**

These studies may have underestimated AoV, as the accuracy of AoV measurement using Doppler echocardiography depends on the intercept angle between the ultrasound beam and the moving red blood cells.<sup>50</sup> Ideally, the angle of intercept is close to zero degrees. Angles less than 20 degrees underestimate velocity by less than 6%, but larger angles significantly underestimate velocity.<sup>50</sup> Angle correction allows for the intercept angle and prevents underestimation of velocity, particularly when AoV is measured from the left parasternal window. Some echocardiographers discourage the use of angle correction, as overestimation of velocity may occur,<sup>41</sup> but one study using normal German Shepherds and Beagles reported peak AoV from the left parasternal window using angle correction was 2.3m/s.<sup>53</sup>

Recently the subcostal window has been used to measure AoV in some breeds, because the intercept angle is closest to zero from this view.<sup>54</sup> Unfortunately, obtaining parallel alignment from the subcostal view in large unsedated dogs that can generate significant abdominal resistance can be difficult.<sup>54</sup> In this study, it was not possible to measure AoV from the subcostal view in unsedated muscular English Bull Terriers due to their unique chest conformation. AoV was measured from the left parasternal window and angle correction was used to provide the closest true maximum value.

Subcostal measurement of mean peak AoV in unsedated healthy Beagles has been reported to be  $1.55 \pm 0.04$ m/s (SEM),<sup>55</sup> and another recent study also recommended the cut-off value for normal velocity measured subcostally to be 2.25m/s.<sup>30</sup> Interestingly, this value is also similar to that measured using the left parasternal window and angle correction in the apparently normal English Bull Terriers in this study.

Other factors may also affect the AoV recorded and may have also contributed to the high AoV in this study. Aortic velocity depends on where the value is measured, with higher values reported from a site just distal to the opened aortic valve cusps (as was done in this study), rather than more proximally.<sup>41</sup> This may be particularly relevant as English Bull Terriers in this study also had an apparently narrower Ao dimension compared with other dog breeds of similar weight, and measurement of AoV just distal to an apparent narrowing of anatomical outflow may significantly affect the recorded velocity values.

Aortic velocity also depends on SV, stage of ventilation, sympathetic tone, and outlet cross sectional area.<sup>41</sup> SV and ventilation were not likely to have affected AoV measured in this study as calculated SV values were within the range expected for their body size,<sup>56</sup> and the effect of ventilation was minimised because peak AoV was taken after examination over three to five cycles. Sympathetic tone was also unlikely to have contributed as it was presumed to be low, based on handling and mean systolic BP values (105mmHg),<sup>38</sup> and no association was found between BP and AoV. In contrast, while the outlet cross-section area was not measured in this study, a narrow aortic root was observed in these English Bull Terriers compared with other dog breeds of similar body size. Despite the fact that no statistically significant association was observed between Ao/la and AoV ( $P=0.556$ ), IVSd ( $P=0.867$ ) or LVfracd ( $P=0.142$ ), this narrowed outlet based on 2D measurements could contribute to an elevated AoV.<sup>48</sup>

The significance of an elevated AoV is not only important in diagnosis of LVOTO, but also in the prognosis for the dog, because a higher pressure gradient across the obstruction is associated with an increased risk of clinical heart disease.<sup>11</sup> In clin-

ical practice the PG is generally calculated from the value for AoV, determined using Doppler echocardiography, and the modified Bernoulli equation.<sup>54</sup> This method may overestimate the PG, as this equation only considers convective acceleration, and ignores other factors such as flow acceleration and viscous friction, which may be important with a long tunnel-like obstruction.<sup>26,54,57</sup> This equation is also affected in high flow states by the velocity proximal to the obstruction. This value is very difficult to measure, and its effect on the pressure gradient is ignored.<sup>29,57</sup> This may lead to calculation of a falsely high PG when elevated AoV and left ventricular pressures occur.<sup>58</sup> Hence, the PG across the English Bull Terrier LVOT may be less than calculated from the AoV using the modified Bernoulli equation, and an apparently elevated AoV may have a more benign prognosis than initially indicated.

The elevated AoV and nonlaminar aortic flow seen only with pulsed wave Doppler in some of the English Bull Terriers in this study would indicate one of two possibilities. Firstly, these features were nonpathogenic and were a breed-specific characteristic of English Bull Terriers, with elevated AoV due to a different but nonpathogenic cardiac substrate. The abnormal substrate was most likely a narrow aortic root, however a narrow LVOT, lowered systemic vascular resistance, LVH, or unusual ejection mechanisms may have also been involved. Normal secondary physiological responses to a narrowed outflow tract may have produced increased LA and LV wall thickness values compared to other dogs. Alternatively, by definition, most English Bull Terriers have mild LVOTO as their AoV tends to be higher and their Ao abnormally small, is associated with a decreased FS%, and with thicker left ventricular walls.

The animals in this study, and their parents, were apparently healthy.<sup>a</sup> It does not seem helpful to the breed to euthanase or prevent breeding in dogs with no murmur and a normal echocardiographic examination other than an AoV  $\leq 2.3$ m/s, and mild LVOT turbulence on pulsed spectral wave Doppler examination. This is particularly so when these findings may result from a nonpathogenic anatomic or functional variant of the LVOT in this breed or may be associated with echocardiographic recording difficulties.

The applicability of the findings of this study are illustrated by a recent abstract which reported elevated AoV in 23 Miniature English Bull Terriers that were examined using echocardiography.<sup>59</sup> The mean AoV in these dogs measured from an apical view was  $1.75 \pm 0.33$ m/s, and  $2.1 \pm 0.35$ m/s from a subcostal view. This study suggested the high AoV was due to LVOTO in these dogs, and 81% did have murmurs. However, 82% of the dogs with murmurs also had mitral regurgitation, which could have contributed to or caused these murmurs. Further, the number of animals with LVOT turbulence, and the method used to determine it, were not stated. While LVOTO may have been common in this group of Miniature English Bull Terriers, some may have resembled the English Bull Terriers in our study and had an elevated AoV, possibly due to a breed specific, potentially nonpathogenic, LVOT. Diagnostic interpretation in this breed needs to be objective and conservative.

In summary, this study has described cardiac parameters in young English Bull Terriers believed to be clinically normal.

<sup>a</sup>Of these 14 English Bull Terriers, 11 are still alive 2 years after this study. Two were euthanased with noncardiac complaints and one was believed killed by a snake. Thirteen of these dogs were auscultated by local veterinarians within the last 12 months, with no murmurs detected.

The dogs in this study had increased LA diameter, LV wall thickness and AoV, and decreased aortic root diameter compared with other breeds of similar body size. In addition, FS% was lower than in dogs of comparable size, and more similar to the values reported from giant breeds. Further experiments are needed to differentiate whether the echocardiographic features of English Bull Terriers in this study were early or mild signs of LVOTO that will progress to obvious clinical LVOTO, or aberrant findings in one breed that may lead to confusion in diagnosing LVOTO.

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## References

- Dukes McEwan J. Mitral dysplasia in Bull Terriers. *Vet Annal* 1995;35:130-146.
- Malik R, Church DB. Congenital mitral insufficiency in Bull Terriers. *J Small Anim Pract* 1988;29:549-557.
- Pyle RL. Interpreting low-intensity cardiac murmurs in dogs predisposed to subaortic stenosis. *J Am Anim Hosp Assoc* 2000;36:379-382.
- Bonagura JD, Lehmkuhl LB. Congenital heart disease. In: Fox PR, Sisson D, Moise NS, editors. *Textbook of canine and feline cardiology: principles and clinical practice*. 2nd edn. Saunders, Philadelphia, 1999:471-535.
- Fox PR, Miller MW, Liu S-K. Clinical, echocardiographic, and Doppler imaging characteristics of mitral valve stenosis in two dogs. *J Am Vet Med Assoc* 1992;201:1575-1579.
- Olsen LH, Fredholm M, Pedersen HD. Epidemiology and inheritance of mitral valve prolapse in Dachshunds. *J Vet Intern Med* 1999;13:448-456.
- Swenson L, Haggstrom J, Kvarn C, Juneja RK. Relationship between parental cardiac status in Cavalier King Charles Spaniels and prevalence and severity of chronic valvular disease in offspring. *J Am Vet Med Assoc* 1996;208:2009-2012.
- Patterson DF. The genetics of canine congenital heart disease. *Proc Am Coll Vet Intern Med Forum* 1996;14:225-227.
- Patterson DF. Genes and the heart: congenital heart disease. In: Harpster NK, Jones CL, editors. *Proceedings of the American Academy of Veterinary Cardiology*, 1991:13-21.
- Pyle RL, Patterson DF, Chacko S. The genetics and pathology of discrete subaortic stenosis in the Newfoundland dog. *Am Heart J* 1976;92:324-334.
- Sisson D. Fixed and dynamic subvalvular aortic stenosis in dogs. In: Kirk RW, Bonagura JD, editors. *Kirk's Current Veterinary Therapy: Small Animal Practice*. Saunders, Philadelphia, 1992:760-766.
- Pedersen HD, Haggstrom J, Falk T et al. Auscultation in mild mitral regurgitation in dogs: observer variation, effects of physical maneuvers, and agreement with color Doppler echocardiography and phonocardiography. *J Vet Intern Med* 1999;13:56-64.
- Della Torre PK, Kirby AC, Church DB, Malik R. Echocardiographic measurements in Greyhounds, Whippets and Italian Greyhounds-dogs with similar conformation but different size. *Aust Vet J* 2000;78:49-55.
- Morrison SA, Mohammed H, Yeager AE. Effect of breed and body weight on echocardiographic values in four breeds of dogs of differing somatotype. *J Vet Intern Med* 1992;6:220-224.
- Sisson D, Schaeffer D. Changes in linear dimensions of the heart, relative to body weight, as measured by M-mode echocardiography in growing dogs. *Am J Vet Res* 1991;52:1591-1596.
- Snyder PS, Sato T, Atkins CE. A comparison of echocardiographic indices of the nonracing healthy Greyhound to reference values from other breeds. *Vet Radiol Ultrasound* 1995;36:387-392.
- Wu G, Markowitz GS, Li L et al. Cardiac defects and renal failure in mice with targeted mutations in Pkd2. *Nat Genet* 2000;24:75-78.
- Burrows AK, Malik R, Hunt GB et al. Familial polycystic kidney disease in Bull Terriers. *J Small Anim Pract* 1994;35:364-369.
- Hossack KF, Leddy CL, Johnson AM, Schreier RW, Gabow PA. Echocardiographic findings in autosomal dominant polycystic kidney disease. *New Engl J Med* 1988;319:907-912.
- Boulter C, Mulroy S, Webb S et al. Cardiovascular, skeletal, and renal defects in mice with targeted disruption of the Pkd1 gene. *Proc Natl Acad Sci USA* 2001;98:12174-12179.
- O'Leary CA, MacKay BM, Malik R et al. Polycystic kidney disease in Bull Terriers: an autosomal dominant inherited disorder. *Aust Vet J* 1999;77:361-366.
- Hood JC, Robinson WF, Clark WT et al. Proteinuria as an indicator of early renal disease in Bull Terriers with hereditary nephritis. *J Small Anim Pract* 1991;32:241-248.
- Ross LA. Pathophysiology and management of systemic hypertension associated with renal dysfunction. In: Osborne CA, Finco DR, editors. *Canine and feline nephrology and urology*. Williams and Wilkins, Baltimore, 1995:392-399.
- Rishniw M, Erb HN. Evaluation of four 2-dimensional echocardiographic methods of assessing left atrial size in dogs. *J Vet Intern Med* 2000;14:429-435.
- Kallet A, Cowgill LD. Hypertensive states in the dog. *Proc Am Coll Vet Med Forum* 1982:70.
- Kienle RD. Echocardiography. In: Kittleson MD, Kienle RD, editors. *Small animal cardiovascular medicine*. Mosby, St Louis, 1998:95-117.
- Boon JA. Appendix IV. In: *Manual of veterinary echocardiography*. Williams and Wilkins, Baltimore, 1998:453-473.
- O'Grady MR, Bonagura JD, Powers JD, Herring D. Quantitative cross-sectional echocardiography in the normal dog. *Vet Radiol* 1986;27:34-49.
- Boon JA. Evaluation of size, function, and hemodynamics. In: Boon JA, editor. *Manual of veterinary echocardiography*. Williams and Wilkins, Baltimore, 1998:151-260.
- Bussadori C, Amberger CN, Le Bobinac G, Lombard CW. Guidelines for the echocardiographic studies of suspected subaortic and pulmonic stenosis. *J Vet Cardiol* 2000;2:17-24.
- Bussadori C. Echo patterns in Boxers with subaortic stenosis. *Proc Am Coll Vet Intern Med Forum* 2000;18:86-87.
- Abbott JA, Duncan R, Clark EG, Pyle RL. Aortic valve disease in Boxers with physical and echocardiographic findings of aortic stenosis. *Proc Am Coll Vet Intern Med Forum* 2001;19:844.
- Haggstrom J, Hamlin RL, Hansson K, Kvarn C. Heart rate variability in relation to severity of mitral regurgitation in Cavalier King Charles Spaniels. *J Small Anim Pract* 1996;37:69-75.
- Koch J, Pedersen HD, Jensen AL, Flagstad A. M-mode echocardiographic diagnosis of dilated cardiomyopathy in giant breed dogs. *J Small Anim Pract* 1996;43:297-304.
- Bayon A, Fernandez del Palacio A, Montes AM, Gutierrez Panzico C. M-mode echocardiographic study in growing Spanish Mastiffs. *J Small Anim Med* 1994;35:473-479.
- Henik R. Echocardiography and Doppler ultrasound. In: Miller MS, Tilley LP, editors. *Manual of canine and feline cardiology*. 2nd edn. Saunders, Philadelphia, 1995:75-107.
- Vollmar AC. Echocardiographic measurements in the Irish Wolfhound: reference values for the breed. *J Am Anim Hosp Assoc* 1999;35:271-277.
- Moise NS, Fox PR. Echocardiography and Doppler imaging. In: Fox PR, Sisson D, Moise NS, editors. *Textbook of canine and feline cardiology: principles and clinical practice*. 2nd edn. Saunders, Philadelphia, 1999:130-171.
- Littman MP, Fox PR. Acquired valvular heart disease in dogs and cats. In: Fox PR, Sisson D, Moise NS, editors. *Textbook of canine and feline cardiology: principles and clinical practice*. 2nd edn. Saunders, Philadelphia, 1999: 795-813.
- Cox RH, Peterson LH, Detweiler DK. Comparison of arterial hemodynamics in the mongrel dog and the racing greyhound. *Am J Physiol* 1976;230:211-218.
- Bodey AR, Michell AR. Epidemiological study of blood pressure in domestic dogs. *J Small Anim Pract* 1996;37:116-125.
- Bonagura JD, Miller MW, Darke PGG. Doppler echocardiography I: pulsed-wave and continuous-wave examinations. *Vet Clin N Am: Small Anim Pract* 1998;28:1325-1359.
- Luis Fuentes V, Darke DGG, Cattanach BM. Aortic stenosis in Boxer dogs. *Proc Am Coll Vet Intern Med Forum* 1994;12:309-311.
- Buchanan JW. Prevalence of cardiovascular disorders. In: Fox PR, Sisson D, Moise NS, editors. *Textbook of canine and feline cardiology: principles and clinical practice*. 2nd edn. Saunders, Philadelphia, 1999:457-470.
- Kienle RD. Aortic stenosis. In: Kittleson MD, Kienle RD, editors. *Small animal cardiovascular medicine*. Mosby, St Louis. 1998:260-272.
- O'Grady MR. The incidence of aortic valve insufficiency in canine congenital aortic stenosis: a Doppler echocardiographic study. *J Vet Intern Med* 1990;4:129.
- Lehmkuhl LB, Bonagura JD. CVT update: canine subvalvular aortic stenosis. In: Bonagura JD, Kirk RW, editors. *Kirk's Current Veterinary Therapy: Small Animal Practice*. Saunders, Philadelphia, 1995:822-827.
- Wingfield WE, Boon JA, Miller CW. Echocardiographic assessment of congenital aortic stenosis in dogs. *J Am Vet Med Assoc* 1983;183:673-676.
- Bonagura JD, Miller MS. Case studies for internists. *Proc Am Coll Vet Intern Med Forum* 1997;15:143-146.
- Luis Fuentes V, Schober KE, Bonagura J. Current issues in echocardiography.

raphy. *Proc Am Coll Vet Intern Med Forum* 2000;18:88-93.

51. Boon JA. Congenital heart disease. In: Boon JA, editor. *Manual of veterinary echocardiography*. Williams and Wilkins, Baltimore, 1998:383-445.
52. Gaber CE. Normal pulsed Doppler flow velocities in adult dogs. *Proc Am Coll Vet Intern Med Forum* 1987;5:69.
53. Kirberger RM, Bland-van den Berg P, Darazs B. Doppler echocardiography in the normal dog: part 1-velocity findings and flow patterns. *Vet Radiol Ultrasound* 1992;33:370-379.
54. Lehmkuhl LB, Bonagura JD. Comparison of transducer placement sites for Doppler echocardiography in dogs with subaortic stenosis. *Am J Vet Res* 1994;55:192-198.
55. McEntee K, Clercx C, Amory H et al. Doppler echocardiographic study of left and right ventricular function during dobutamine stress testing in conscious

healthy dogs. *Am J Vet Res* 1999;60:865-871.

56. Mashiro I, Nelson RR, Cohn JN, Franciosa JA. Ventricular dimensions measured noninvasively by echocardiography in the awake dog. *J Appl Physiol* 1976;41:953-959.
57. Moise NS. Doppler echocardiographic evaluation of congenital cardiac disease. *J Vet Intern Med* 1989;3:195-207.
58. Lehmkuhl LB, Bonagura JD, Jones DE, Stepien RL. Comparison of catheterization and Doppler-derived pressure gradients in a canine model of subaortic stenosis. *J Am Soc Echo* 1995;8:611-620.
59. Lefborn BK, Rosenthal SL. Left ventricular outflow tract velocity in 23 Miniature Bull Terriers-variation from other breeds. *Proc Am Coll Vet Intern Med Forum* 1998;16:739.

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### Evidence-based physicians' dressing : a message for the veterinary profession?

A group of doctors from the Newcastle area has conducted an important clinical trial. The study, involving 12 male doctors and 1680 patients, assessed the effect of physicians' dress codes on levels of confidence in their medical ability amongst patients. Traditionally the male physicians' attire has included a white coat and tie with the stethoscope used as an accessory. While ties may increase the level of patient confidence, they have also been shown to be sources of contamination, thus creating a dilemma - should doctors wear items known to be dangerous to their patients' health but which inspire confidence in their abilities? The authors set out to analyse the importance of various items of clothing on patient confidence, by means of a multiple crossover dressing trial. (This, the authors stress, should not be confused with a cross-dressing trial.)

Over 7 months, one item of clothing was removed, changed or added at 1 month intervals in a set sequence: shedding the white coat, losing the tie, changing from dress pants to flared jeans, from dress shirt to Hawaiian shirt, moussing and highlighting the hair, and wearing a nose ring. The completed sequence was termed the 'respectable' to 'retro' transformation and each doctor acted as his own control.

Predictably, patient confidence was highest with a 'respectable' dress protocol, although confidence levels did not fall significantly with the loss of the white coat or tie. Confidence levels plummeted in the presence of a nose ring, and "retro" dressing (flared jeans, Hawaiian shirt) was regarded as an affront. Dress pants and shirt accounted for most of the patients' confidence, and the presence of a nose ring was the most deleterious.

The authors concluded that further study is needed to define the effect of specific fashions on specific patient groups, and emphasise the potential of a new specialty called PCAM, or physicians' complementary accessories medicine, that recognises fashion accessories as adjuvant therapies.

Is there a message here for the veterinary profession?

Nair BR, Mears SR, Hitchcock KI, Attia JR. Evidence-based physicians' dressing : a crossover trial. *Med J Aust* 2002;177:681-682.

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