Blood pressure changes in dogs with babesiosis

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ABSTRACT

Systemic arterial blood pressures were measured in 30 dogs with acute babesiosis, 10 each with mild uncomplicated, severe uncomplicated and complicated disease. Ten healthy dogs were used as controls. Hypotension was defined as more than 3 standard deviations below the control mean. Normal mean pressures (±SD) were: systolic arterial pressure 151 (±11) mm Hg, diastolic arterial pressure 89 (±8) mm Hg and mean arterial pressure 107 (±10) mm Hg. Hypotension was the most frequent abnormality, and increased strikingly in incidence as disease severity increased, with 5/10 dogs in the complicated group being hypotensive for systolic, diastolic and mean arterial pressures, compared with 2/10 in the severe uncomplicated group and 0/10 in the mild uncomplicated group. Systolic, diastolic and mean arterial pressures in the complicated group and severe uncomplicated group, and systolic pressure in the mild uncomplicated group, were significantly lower than in the controls. There were no significant relationships between arterial pressures and age, pulse rate, respiratory rate, temperature, mucous membrane colour or haematocrit. There was a significant negative correlation between arterial pressures and white cell and immature neutrophil counts. Arterial pressures differed significantly between dogs that were clinically collapsed and those that were not, but not between survivors and non-survivors. Pulse pressure (systolic – diastolic) was low in 7/10 complicated, 1/10 mild uncomplicated, and 1/10 severe uncomplicated cases, and differed significantly between the complicated and control groups. The high incidence of hypotension in clinically severe babesiosis has important implications for therapy.

Key words: *Babesia canis,* babesiosis, blood pressure, canine, dog, hypotension, shock.

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INTRODUCTION

Circulatory failure and shock have long been recognised in severe and terminal babesiosis caused by *Babesia canis*[18], and it has been suggested that virulent babesiosis results in 2 syndromes, one characterised by hypotensive shock and the other by haemolytic anaemia[31]. However, objective evidence of hypotension has been provided in only 1 case[10]. In addition, little consideration has been given to the possibility that clinically mild or inapparent hypotension (as opposed to life-threatening hypotensive shock, *i.e.* hypotension accompanied by failure of capillary perfusion[21]) might occur in canine babesiosis, and that a fall in blood pressure might follow a continuum of severity, rather than being exclusively a fulminant marker of catastrophe.

Wright and Kerr[38] documented arterial hypotension in splenectomised calves infected with *Babesia bovis,* and commented that hypotensive shock was characteristic of the disease. Blood pressure began to fall approximately 3 days after infection, well before the reduction in haematocrit (Ht) that occurred on about day 5, and continued to decrease over the next 6 days, until the study ended. Thus, reduced systemic blood pressure was present long before hypotensive shock occurred. Wright[37,38] attributed hypotension in babesiosis to vasoactive mediators (kallikrein and kinins). However, Gilles demonstrated a marked reduction in blood volume in a dog infected with *B. canis* that developed shock and haemoconcentration[11].

Falciparum malaria in humans is clinically similar to canine babesiosis in many respects[14,18,19]. Orthostatic hypotension is common in uncomplicated malaria[30]. Patients with malaria typically present with increased cardiac output, low systemic vascular resistance and low to normal blood pressure[36]. Hypotensive shock is a relatively rare event, and is termed 'algid malaria'[36].

Sepsis has been redefined in human medicine as the systemic response to infection, regardless of whether that infection is bacterial, viral, fungal or protozoal[6]. The systemic response to severe insults, labelled the systemic inflammatory response syndrome (SIRS) in the absence of infection, and sepsis in its presence, is defined as 2 or more of the following: hypo- or hyperthermia, tachycardia, hyperventilation, and leukocytosis, leukopaenia or neutrophilic left shift. Acute babesiosis is thus a form of sepsis, according to the consensus definition, and would be expected (as predicted[7,8]) to manifest in similar ways. Hypotension is a consistent feature of the haemodynamic response to sepsis in the dog, occurring in natural[16] and experimental[29] bacterial sepsis, as well as after injection of endotoxin and pro-inflammatory cytokines[22,23].

The aims of this study were: (1) to measure systemic arterial pressures in dogs with naturally occurring babesiosis, (2) to establish whether the presence and severity of hypotension increases with increased disease severity; and (3) to establish whether blood pressure is correlated with easily measured clinical and/or laboratory parameters.

MATERIALS AND METHODS

Thirty dogs presented at the Onderstepoort Veterinary Academic Hospital with clinical signs of acute babesiosis were prospectively studied. Inclusion criteria were: *Babesia canis* parasites identified on a thin capillary blood smear, no history of an acute inflammatory or infectious disease in the past 6 weeks, and body mass greater than 5 kg. Dogs with known (smear-positive) concurrent *Ehrlichia canis* infections were excluded. Informed consent of the owner was required to enrol a patient in the study. The dogs were classified into 3 groups, based on WHO criteria for severe falciparum malaria[35] and a classification system for canine babesiosis[14]: (1) Mild uncomplicated babesiosis: Ht ≥0.20 ℓ/ℓ, no complications; (2) severe uncomplicated babesiosis: Ht <0.20 ℓ/ℓ, no complications; (3) complicated babesiosis: acute renal failure, cerebral babesiosis, haemoconcentration ('red biliary'), hepatopathy, hypoglycaemia and/or pulmonary oedema, defined as follows:

- Acute renal failure: serum creatinine

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>150 μmol/ℓ and/or oliguria (<1 mℓ/kg/hour); not resolved by rehydration.
- Cerebral babesiosis: central neurological signs not attributable to any other cause.
- Haemoconcentration: haematocrit >0.37 ℓ/ℓ *in conjunction with* congested mucous membranes and severe haemolysis (severe haemoglobinuria and/or grossly visible haemoglobinaemia).
- Hepatopathy: icterus and/or alanine transaminase and alkaline phosphatase *both* >2× normal upper limit.
- Hypoglycaemia: plasma glucose ≥2.2 mmol/ℓ.
- Pulmonary oedema: dyspnoea with typical frothy nasal discharge, radiographic oedema and/or P_aO_2 <60 mm Hg.

The control group consisted of unmatched healthy dogs from the same catchment area as the dogs with babesiosis, admitted for routine sterilisation. To qualify for inclusion, these dogs had to be clinically normal and aparasitaemic. There were 10 dogs in each group.

A full clinical examination was performed at admission. Blood samples and blood pressure measurements were obtained before treatment was administered. Blood pressure was determined non-invasively by an oscillometric technique, using a Dinamap blood pressure monitor (Critikon, Johnson & Johnson) and neonatal cuffs (Disposa-Cuf, Critikon). Each dog was positioned in right lateral recumbency and the cuff placed on the left distal forelimb immediately proximal to the carpus, over the radial artery. The cuff size was selected so that the width was approximately 40 % of the limb circumference. Five to 6 readings of systolic arterial pressure (SAP), diastolic arterial pressure (DAP) and mean arterial pressure (MAP) were recorded over a 5-minute period. The readings were then averaged. The Dinamap measures SAP and DAP, and calculates MAP. Pulse pressure (PP) was calculated by subtracting DAP from SAP. The laboratory database consisted of haematology and serum biochemistry (urea, creatinine, bilirubin, alanine transaminase, alkaline phosphatase and glucose).

All dogs with babesiosis were treated with diminazene aceturate (Berenil, Hoechst) according to the manufacturer's instructions. Dogs with mild uncomplicated babesiosis did not receive additional supportive treatment. Dogs with Ht <0.15 ℓ/ℓ were given a whole blood transfusion. Other supportive treatment was tailored to individual requirements and was not standardised.

The results were analysed using Excel 97™ (Microsoft) and SigmaStat™ v2.0.3 (Jandel Scientific). Hyper- and hypotension were defined as falling outside 3 standard deviations (SD) of the normal control group[32]. Values greater or less than 2 SD and up to 3 SD from the mean were considered borderline[32]. Nonparametric statistical methods were used for most comparisons, for 2 reasons: (1) the small sample size meant that normal distribution of data could not be established with certainty; and (2) although most blood pressure data in this study was normally distributed, a large study has shown that blood pressures in dogs have a log-normal distribution[5]. The Mann-Whitney rank sum test was used for comparisons between 2 groups, and Kruskal-Wallis ANOVA on ranks, with pairwise comparisons against the control group using Dunnett's method, for comparisons between 3 or more groups. The Student-Neuman-Keuls test was used for pairwise comparisons among the babesiosis groups. The Spearman rank order correlation was used to determine the strength of association between selected variables in the patient groups. Where variables from all 30 patients were used for correlation or regression analysis, data were also analysed using the Pearson product moment correlation or linear regression, after normalisation where required. Univariate analysis was used to measure relationships between grouped variables in the patient groups, using the Mann-Whitney and Kruskal-Wallis tests. The probability value for significance was set at $P < 0.05$.

RESULTS

Signalment, clinical and laboratory data for the study population are summarised in Table 1 and blood pressure data shown in Table 2. The signalment of the control group was similar to that of the babesiosis groups. Laboratory values were frequently abnormal in the babesiosis groups, and often differed from controls and between groups (see Table 2). The abnormalities were consistent with the disease classification. In the complicated group, 3 dogs had acute renal failure (Dogs 3, 7 and 9); 4 had haemoconcentration (Dogs 3, 5, 6 and 7); 2 had icterus (Dogs 4 and 8); 3 had hypoglycaemia (Dogs 1, 2 and 10) and 2 had pulmonary oedema (Dogs 3 and 5). Dog 9 also had rhabdomyolysis. No dog had cerebral babesiosis. The dogs with visible icterus had extremely high bilirubin concentrations (142.6 and 165.6 μmol/ℓ, respectively), but no dogs had both liver enzymes elevated above twice normal. Hypoglycaemia has not previously been recognised in canine babesiosis, and will be reported in more detail separately, together with other data on carbohydrate metabolism.

Normal ranges for blood pressures, obtained from the control group, were as follows (mean (±2 SD)): SAP 151 (129–173) mm Hg; DAP 89 (73–105) mm Hg; MAP 107 (86–128) mm Hg; and PP 62 (48–76) mm Hg. The cut-off values for hypertension were SAP >184 mm Hg; DAP >113 mm Hg; and MAP >138 mm Hg, and for hypotension SAP <118 mm Hg; DAP <65 mm Hg; and MAP <76 mm Hg. Cut-off values for elevated and low PP were, respectively, >83 mm Hg and <41 mm Hg.

Hypotension was the most common abnormality, and increased strikingly in incidence as disease severity increased (Table 2A–D). Five of 10 dogs in the complicated group had hypotensive values for SAP, DAP and MAP, compared with 2/10 in the severe uncomplicated group and 0/10 in the mild uncomplicated group. Eight of the 10 dogs in the complicated group had at least 1 hypotensive value, compared with 6/10 in the severe uncomplicated group and 3/10 in the mild uncomplicated group. As a rule, changes in a particular pressure measurement were paralled by changes in the others, although the degree of change frequently varied. Pulse pressures were abnormally low in 7/10 dogs in the complicated group, 1/10 in the mild uncomplicated group and 1/10 in the severe complicated group. The changes in PP indicated that SAP and DAP did not tend to change by the same amount. Elevated arterial pressures were uncommon, with only 1 dog (mild uncomplicated group Dog 8) showing hypertension (elevated SAP, DAP and MAP). DAP and MAP were elevated in Dog 5 in the complicated group, and borderline elevated SAP was present in Dog 5 in the mild uncomplicated group.

There was a significant difference in SAP ($P < 0.05$) between all 3 patient groups and the control group (Fig. 1A). The DAP and MAP of the severe uncomplicated and complicated groups, but not of the mild uncomplicated group, differed significantly from the controls ($P < 0.05$) (Fig. 1B–C). There was a significant difference in PP between complicated cases and controls (Fig. 1D).

There were no significant relationships between SAP, DAP or MAP and age, pulse rate, respiratory rate, rectal temperature and Ht. There was no correlation between PP and Ht. A significant negative correlation was found between white cell count and SAP, DAP and MAP (SAP: $r^2 = 0.252$, $P = 0.0049$; DAP: $r^2 = 0.297$, $P = 0.00195$; MAP: $r^2 = 0.2959$, $P = 0.0020$). Results were similar using normalised data and

Table 1: **Signalment, clinical data and laboratory data for dogs with babesiosis and healthy controls. Summary data is shown as mean (SD).**

	Normal values† (where applicable)	Mild uncomplicated	Severe uncomplicated	Complicated	Control
Age (years)		2.9 (3.0)	3.5 (4.2)	2.5 (2.7)	2.1 (1.3)
Sex (M:F)		6:4	3:7	6:4	3:7
Weight (kg)		24.6 (14.5)	17.3 (8.7)	24.0 (15.6)	18.2 (8.3)
Rectal temperature (°C)	38.4–39.4[1]	40.0 (0.8)	39.8 (0.6)	39.4 (1.3)	NR
Pulse rate (per min)	70–120[1]	118 (27)	132 (23)	119 (42)	NR
Respiratory rate (per min)	18–34[1]	53 (24)	49 (21)	41 (10)	NR
Mucous membrane colour (normal:pale:congested:icteric)		3:6:1:0	0:10:0:0	0:3:5:2	10:0:0:0
Haematocrit (ℓ/ℓ)	0.37–0.55	0.34 (0.08)* [a]	0.13 (0.03)* [ab]	0.27 (0.20)* [b]	0.50 (0.04)
White cell count (×10^9/ℓ)	6.0–15.0	5.5 (1.9)* [a]	8.6 (4.3)* [a]	13.2 (7.3)[a]	13.3 (6.7)
Immature neutrophil count (×10^9/ℓ)	0–0.30	0.29 (0.54)[ab]	1.27 (1.53)* [a]	1.88 (2.84)* [b]	0.28 (0.41)
Urea (mmol/ℓ)	3.6–8.9	6.8 (3.5)[ab]	13.3 (6.5)* [a]	18.7 (6.4)* [b]	6.7 (2.6)
Creatinine (µmol/ℓ	<133	104 (30)* [ab]	84 (25)* [ac]	146 (61)[bc]	123 (16)
Total bilirubin (µmol/ℓ)	<6.8	10.0 (11.8)	20.5 (23.8)	49.3 (59.6)	14.4 (13.8)
Alanine transaminase (U/ℓ)	<40	21 (6)	48 (74)	50 (43)	23 (6)
Alkaline phosphatase (U/ℓ)	<190	77 (40)*	178 (295)*	84 (59)*	44 (23)
Glucose (mmol/ℓ)	3.3–5.5	5.1 (0.6)[a]	5.1 (0.8)[b]	3.5 (2.0)* [ab]	5.2 (0.7)
Collapsed at presentation		0	2	7	0
Died		0	1	4	0

†Normal values are those used at the Onderstepoort Veterinary Academic Hospital, except where indicated otherwise.
*Significantly different from control group. Patient groups with the same superscripts (a, b or c) differ significantly from one another ($P < 0.05$).

parametric methods. A significant negative correlation was found between SAP, DAP and MAP and immature neutrophil count using non-parametric statistics (SAP: $r^2 = 0.1354$, $P = 0.0455$; DAP: $r^2 = 0.1918$, $P = 0.0157$; MAP: $r^2 = 0.1998$; $P = 0.0135$), but the significance was lost when parametric methods and normalised data were used, although in all cases P still tended towards significance.

Blood pressure variables were compared between groupings based on clinical variables, to establish whether any of these might be predictive of hypotension. There were no significant differences in blood pressures between patients grouped according to respiratory abnormalities (≤40 breaths per minute and >40 breaths per minute/panting/ dyspnoea); Ht (<0.20 ℓ/ℓ and >0.20 ℓ/ℓ); rectal temperature (<37.5 °C; 38.5–40 °C; and >40 °C); or age group (<0.5 years; 0.5–5years; and >5years). Blood pressure did not differ according to mucous membrane colour, whether this was grouped as normal *vs* pale *vs* icteric *vs* congested; or pale *vs* non-pale; or congested *vs* non-congested. There was a trend towards a significant difference between pale and non-pale mucous membranes for SAP (median (25th–75th percentile): Pale 135 mm Hg (112–148); non-pale 107 mm Hg (87–122); $P = 0.055$). There was no relationship between PP and Ht when dogs were divided into severely anaemic (Ht <0.20 ℓ/ℓ), moderately or mildly anaemic (Ht 0.20–0.37 ℓ/ℓ) and non-anaemic (Ht >0.37 ℓ/ℓ) groups.

Although there were no significant differences in blood pressures between dogs that survived ($n = 25$) and non-survivors ($n = 5$), a trend towards significance was present for DAP (median (25th–75th percentile): survivors 73 mm Hg (60–89); non-survivors 58 mm Hg (49–67); $P = 0.080$). Five dogs died (Dog 2 in the severe uncomplicated group and Dogs 1, 2, 4 and 6 in the complicated group). Four had severe anaemia and 1 had haemoconcentration. Two of these dogs (severe uncomplicated group Dog 2 and complicated group Dog 2) had abnormally low SAP, DAP and MAP, while the other 3 each had at least 1 hypotensive value. DAP was low in 4/5 non-survivors.

There was a significant difference between patients described as collapsed on clinical examination ($n = 9$) and those not considered to be collapsed ($n = 21$) for SAP, DAP and MAP ($P < 0.001$), as well as for PP ($P < 0.05$) (Fig. 2). Most, but not all, collapsed dogs had hypotension.

DISCUSSION

The results of this study support the usefulness of indirect blood pressure measurement as a clinical tool. The cut-off points used for abnormal values[32] identified substantially more dogs as hypotensive than a more conservative textbook definition[17]. Using the textbook definition of hypotension (SAP <80 mm Hg or MAP <60 mm Hg[17]), the number of dogs with hypotension dropped substantially, with only 3, all in the complicated group (Dogs 2, 7 and 8), fitting the criteria. Since hypotension is only clinically significant when it is associated with poor tissue perfusion[17], the conservative definition might be more correct; however, tissue perfusion cannot be measured in the routine clinical environment and if hypotension is identified early and treated, perfusion failure and shock might be avoided. There is thus much to commend the less conservative definition. It is advisable for each institution to establish its own normal values, since values differ between studies, even those using the same equipment[5,32,34].

The changes in blood pressure in the severely ill dogs with babesiosis were not surprising, given the clinical evidence of collapse and shock in many of these animals. It is notable, however, that even in the mild uncomplicated group, SAP was significantly lower than that of the control group. The low blood pressures in the severely anaemic group were somewhat unexpected, since severe anaemia should result in increased cardiac output and stroke volume in order to maintain tissue oxygenation. It therefore appears that, at least in some dogs, these compensatory mechanisms falter, possibly as a

Table 2: **Systemic arterial pressures in dogs with babesiosis.**

Dog	Mild uncomplicated		Severe uncomplicated		Complicated		Control	
A: **systolic arterial pressure** (mm Hg)								
1	147	❍	133	❍	135	▼	140	❍
2	123	❑	105	▼	84	▼	150	❍
3	147	❍	135	❍	87	▼	160	❍
4	101	▼	110	▼	119	❑	160	❍
5	176	■	80	▼	156	❍	155	❍
6	143	❍	116	▼	107	▼	170	❍
7	102	▼	158	❍	80	▼	155	❍
8	191	▲	148	❍	82	▼	140	❍
9	111	▼	118	❑	88	▼	138	❍
10	152	❍	148	❍	123	❍	139	❍
Median	145*		126*		98*		153	
25th percentile	111		110		84		140	
75th percentile	152		148		123		160	
B: **diastolic arterial pressure** (mm Hg)								
1	92	❍	78	❍	64	▼	85	❍
2	66	❑	50	▼	47	▼	90	❍
3	93	❍	64	▼	50	▼	95	❍
4	54	▼	69	❑	58	▼	85	❍
5	87	❍	58	▼	134	▲	90	❍
6	76	❍	73	❍	74	❍	100	❍
7	77	❍	66	❑	45	▼	100	❍
8	134	▲	98	❍	60	▼	75	❍
9	69	❑	51	▼	46	▼	84	❍
10	87	❍	90	❍	88	❍	83	❍
Median	82		68*		59*		88	
25th percentile	69		58		47		84	
75th percentile	92		78		74		95	
C: **mean arterial pressure** (mm Hg)								
1.	118	❍	100	❑	84	❑	90	❍
2	89	❍	75	▼	57	▼	100	❍
3	118	❍	89	❍	64	▼	110	❍
4	80	❑	87	❍	85	❑	105	❍
5	117	❍	68	▼	149	▲	105	❍
6	99	❍	92	❍	89	❍	120	❍
7	84	❑	89	❍	58	▼	127	❍
8	163	▲	102	❍	54	▼	100	❍
9	86	❍	69	▼	61	▼	109	❍
10	117	❍	110	❍	111	❍	106	❍
Median	108		89*		74*		106	
25th percentile	86		75		58		100	
75th percentile	118		100		89		110	
D: **pulse pressure** (mm Hg)								
1	55	❍	55	❍	71	❍	55	❍
2	57	❍	55	❍	37	▼	60	❍
3	54	❍	71	❍	37	▼	65	❍
4	47	❑	41	❑	61	❍	75	❍
5	89	▲	22	▼	22	▼	65	❍
6	67	❍	43	❑	33	▼	70	❍
7	25	▼	92	▲	35	▼	55	❍
8	57	❍	50	❍	22	▼	65	❍
9	42	❑	67	❍	42	❑	54	❍
10	65	❍	58	❍	35	▼	56	❍
Median	56		55		36*		63	
25th percentile	47		43		33		55	
75th percentile	65		67		42		65	

❍ = normotensive; ❑ = borderline hypotensive; ▼ = hypotensive; ■ = borderline hypertensive; ▲ = hypertensive. Borderline was defined as greater or less than 2 and up to 3 SD from control group mean, and hyper- and hypotensive as greater or less than 3 SD from the mean. Blood pressure values for control group: systolic arterial pressure 151 (±11) mm Hg; diastolic arterial pressure 89 (±8) mm Hg; mean arterial pressure 107 (±10) mm Hg; pulse pressure 62 (±7) mm Hg.
*Significantly different from control group ($P < 0.05$).

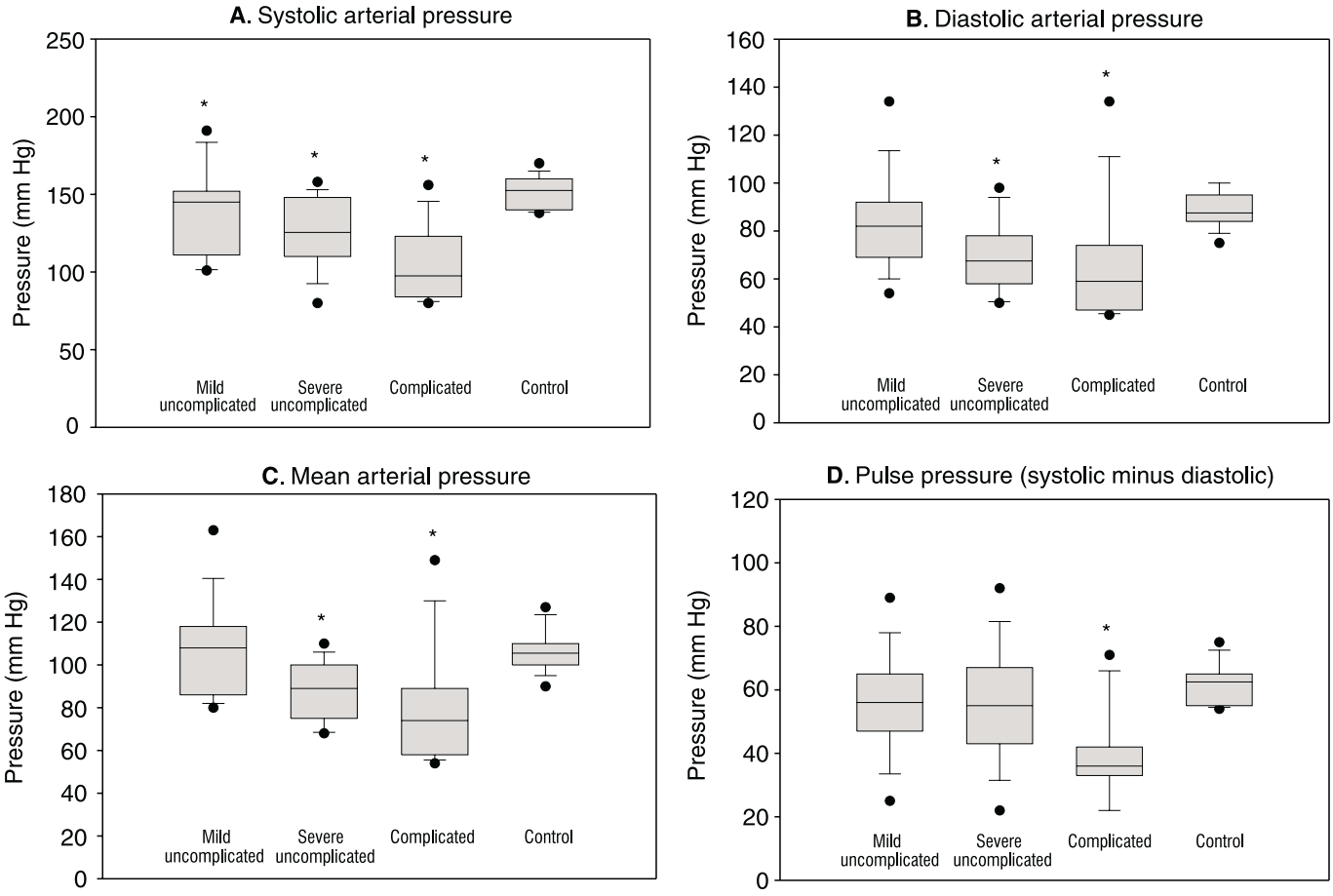

Fig 1: **Systemic arterial pressures in dogs with babesiosis and healthy controls. Data are shown as median** (horizontal line within box)**, 25th and 75th percentiles** (horizontal ends of boxes)**, 10th and 90th percentiles** (T-bars) **and outliers** (solid circles)**.** * = significantly different from control group ($P < 0.05$).

combined result of the acuteness of onset and severity of anaemia, and the inflammatory mechanisms associated with babesiosis. Viewing haemolytic anaemia and hypotensive shock as separate syndromes in babesiosis[31] does not seem appropriate, given the overlap between the 2 syndromes. The presence of hypotension in a large proportion of dogs with complicated babesiosis is consistent with the hypothesis that inflammatory mechanisms play a major role in this syndrome, resulting in a sepsis-like state[7,14,18,25].

What is the cause of hypotension in babesiosis? From a cardiovascular viewpoint, the most likely mechanisms are increased capillary permeability with movement of fluid to the interstitium and/or reduced vascular tone with venous pooling. Both occur in bacterial sepsis, and can be exacerbated by myocardial depression[12]. Increased systemic capillary permeability occurs in malaria[2,3], and its severity is associated with disease severity[9]. Capillary leakage in malaria is not necessarily associated with hypovolaemia, since macromolecules are returned to plasma *via* lymph; in fact, increased plasma volume has been reported[3,28]. This is consistent with recent evidence of increased plasma volume in canine babesiosis[26], possibly due to movement of interstitial fluid into the vasculature secondary to hypotension[27], and with evidence of increased blood volume in the presence of reduced MAP in some patients with malaria[28]. The pathogenesis of this phenomenon is thought to be reduction of effective blood volume through peripheral vasodilation, followed by release of vasopressors, noradrenaline, renin activation and reduced renal haemodynamics[28]. Based on the above, it is likely that reduced vascular tone is the predominant mechanism of hypotension in babesiosis. As in other septic states, nitric oxide, a 'downstream'

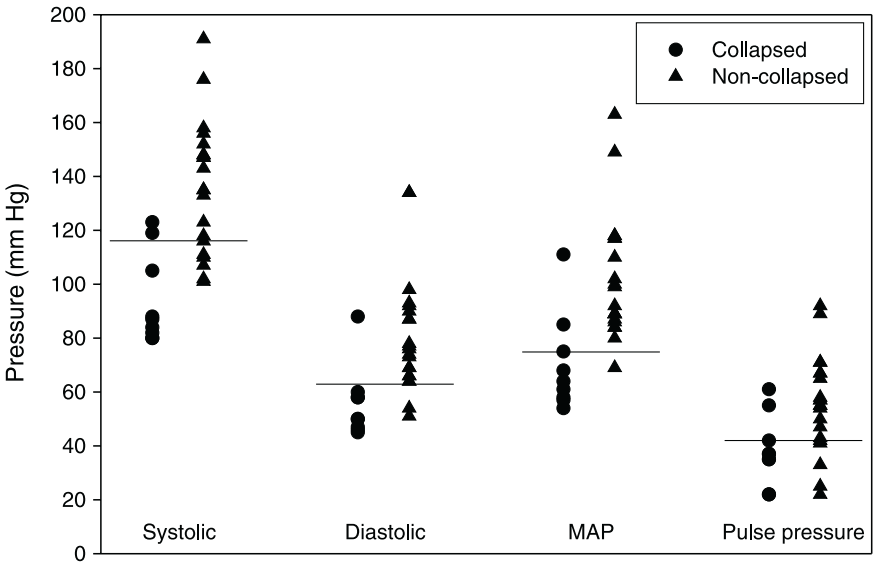

Fig 2: **Systemic arterial pressures in collapsed versus non-collapsed dogs with babesiosis. Horizontal lines show lower cut-off values** (3 standard deviations below the mean) **for each pressure. MAP = mean arterial pressure.**

effector of many inflammatory mediators, and a potent and ubiquitous vasodilator[23], might play an important role.

The lack of correlation between blood pressures and most of the clinical parameters tested was unexpected. In a large study, Bodey and Michell[5] showed that age accounted for almost 60 % of variation in blood pressure in normal dogs, with dogs under 6 months having the lowest pressures, but age did not correlate with blood pressure in our study. Heart rate was expected to increase as blood pressure dropped, but this did not occur, nor was it a consistent finding in hypotensive dogs with endotoxaemia[29] or hypotensive calves with babesiosis[38]. Surprisingly, PP and Ht were not related to one another. It was expected that the 'bounding' or 'waterhammer' pulse frequently encountered in severely anaemic dogs with babesiosis[14] would be caused by a widening in PP, but the results of this study did not support this. Pulse pressure was narrowed in hypotensive dogs, reflecting, overall, a greater drop in SAP than DAP. A relationship between mucous membrane colour and blood pressure was also expected, but was not found. The slightly higher SAP in dogs with pale mucous membranes than in those without mucosal pallor could be attributed to compensatory mechanisms associated with severe anaemia. This was clearly not a uniform phenomenon, given the lack of correlation between Ht and blood pressure overall. The negative correlation between blood pressure and white cell count/immature neutrophil count might reflect a relationship between systemic inflammation and hypotension. An elevated white cell count and neutrophilic left shift were the most sensitive indicators of sepsis in a recent canine study[13].

Blood pressures were not significantly different between dogs that survived and those that died. This is consistent with findings in dogs with septic peritonitis, in which MAP following surgery was not of prognostic value[16]. As in this study, pressures were lower in dogs that died than in those that survived, but the differences were not statistically significant. It is possible that blood pressure is not a predictor of mortality in babesiosis, but larger numbers would be needed to establish this. It is also possible, however, that changes in pressures over time, as opposed to a single measure, would be more useful. In humans with septic shock, MAP at admission did not differ between survivors and non-survivors, but MAP at 24 hours and the change in MAP over 24 hours were useful prognostic indicators[4]. The presence or absence of clinical collapse was the only clinical factor for which blood pressure measurements differed significantly. However, evaluation of collapse is subjective, and its presence or absence was not 100 % sensitive or specific for hypotension (see Fig. 2).

The endpoint of hypotension is hypotensive shock. Shock is essentially a failure of capillary perfusion[21]. Treatment of shock in the dog has been reviewed[12,20], and options for fluid therapy in canine babesiosis patients with shock have been suggested[15]. In severely anaemic dogs, packed red blood cells or whole blood are the fluids of choice, while in non-anaemic hypotensive dogs, colloids are preferred to crystalloids, as crystalloids move rapidly into the interstitial space, reducing their beneficial effect on blood pressure and perfusion, and increasing the likelihood of interstitial oedema[20]. Central venous pressure is a good indicator of overhydration and should be monitored if possible[20]. Serial arterial pressure measurements are useful indicators of the efficacy of fluid therapy in the absence of more invasive techniques. Other indicators of the effectiveness of fluid therapy and resuscitation are normalisation of urine output, serum lactate concentration and base deficit[20,24].

CONCLUSIONS

Hypotension, with the potential sequelae of collapse and/or hypotensive shock, should be suspected in dogs with severe uncomplicated and complicated babesiosis. The presence of clinical collapse is a good indicator of hypotension, but does not replace arterial blood pressure measurement. Prevention of shock by early monitoring of blood pressure and appropriate fluid therapy is a rational strategy in dogs with severe uncomplicated and complicated babesiosis.

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