Blood pressure changes in dogs with babesiosis

L S Jacobson, R G Lobetti and T Vaughan-Scott

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 8/10 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.

INTRODUCTION
Circulatory failure and shock have long been recognised in severe and terminal babesiosis caused by Babesia canis, and it has been suggested that virulent babesiosis results in 2 syndromes, one characterised by hypotensive shock and the other by haemolytic anaemia. However, objective evidence of hypotension has been provided in only 1 case. 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) 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 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 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.

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

Sepsis has been redefined in human medicine as the systemic response to infection, regardless of whether that infection is bacterial, viral, fungal or protozoal. 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, leukaopenia or neutrophilic left shift. Acute babesiosis is thus a form of sepsis, according to the consensus definition, and would be expected (as predicted) to manifest in similar ways. Hypotension is a consistent feature of the haemodynamic response to sepsis in the dog, occurring in natural and experimental bacterial sepsis, as well as after injection of endotoxin and pro-inflammatory cytokines.

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 and a classification system for canine babesiosis: (1) Mild uncomplicated babesiosis: Ht ≥0.20, no complications; (2) severe uncomplicated babesiosis: Ht <0.20, no complications; (3) complicated babesiosis: acute renal failure, cerebral babesiosis, haemoconcentration (‘red biliary’), hepato-pathy, hypoglycaemia and/or pulmonary oedema, defined as follows:

• Acute renal failure: serum creatinine...
Cerebral babesiosis: central neurological signs not attributable to any other cause.

Haemoconcentration: haematocrit >0.37 \(\text{lt} \) in conjunction with congested mucous membranes and severe haemolysis (severe haemoglobinuria and/or grossly visible haemoglobinemia).

Hepatopathy: icterus and/or alanine transaminase and alkaline phosphatase both >2\( \times \) normal upper limit.

Hypoglycaemia: plasma glucose >2.2 mmol/l.

Pulmonary oedema: dyspnoea with typical frothy nasal discharge, radiographic oedema and/or \( \text{P}_{2} \text{O}_{2} < 60 \) 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 \(\text{lt} \) were given a whole blood transfusion. Other supportive treatment was tailored to individual requirements and was not standardised.

The results were analysed using Excel \(97^\text{TM} \) (Microsoft) and SigmaStat \(\text{TM} v2.0.3 \) (Jandel Scientific). Hyper- and hypotension were defined as falling outside 3 standard deviations (SD) of the normal control group\(^5\). Values greater or less than 2 SD and up to 3 SD from the mean were considered borderline\(^6\). 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\(^7\). 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 haemocoagulation (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 (125–173) mm Hg; DAP 99 (73–105) mm Hg; MAP 107 (86–128) mm Hg. The cut-off values for hypotension 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 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 paralleled 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). SAP 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
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 (<20 \( \mu l \)/l and >20 \( \mu l \)/l); rectal temperature (<37.5 °C; 38.5–40 °C; and >40 °C); or age group (<0.5 years; 0.5–5 years; and >5 years). 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 73 mm Hg (60–89); non-pale 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 haemocoelosis. 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. SAP 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\(^1\) identified substantially more dogs as hypoten-sive than a more conservative textbook definition\(^7\). Using the textbook definition of hypotension (SAP <80 mm Hg or MAP <60 mm Hg\(^7\)), 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\(^7\), 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\(^2,3,4\). 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 1: Signalment, clinical data and laboratory data for dogs with babesiosis and healthy controls. Summary data is shown as mean (SD).

<table>
<thead>
<tr>
<th></th>
<th>Normal values(^1)</th>
<th>Mild uncomplicated</th>
<th>Severe uncomplicated</th>
<th>Complicated</th>
<th>Control</th>
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<tr>
<td><strong>Age (years)</strong></td>
<td>2.9 (3.0)</td>
<td>3.5 (4.2)</td>
<td>2.5 (2.7)</td>
<td>2.1 (1.3)</td>
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<td><strong>Sex (M:F)</strong></td>
<td>6:4</td>
<td>3.7</td>
<td>6:4</td>
<td>3.7</td>
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<td><strong>Weight (kg)</strong></td>
<td>24.6 (14.5)</td>
<td>17.3 (8.7)</td>
<td>24.0 (15.6)</td>
<td>18.2 (8.3)</td>
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<td><strong>Rectal temperature (°C)</strong></td>
<td>38.4–39.4(^1)</td>
<td>40.0 (0.8)</td>
<td>39.8 (0.6)</td>
<td>39.4 (1.3)</td>
<td>NR</td>
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<tr>
<td><strong>Pulse rate (per min)</strong></td>
<td>70–120(^1)</td>
<td>118 (27)</td>
<td>132 (23)</td>
<td>119 (42)</td>
<td>NR</td>
</tr>
<tr>
<td><strong>Respiratory rate (per min)</strong></td>
<td>18–34(^1)</td>
<td>53 (24)</td>
<td>49 (21)</td>
<td>41 (10)</td>
<td>NR</td>
</tr>
<tr>
<td><strong>Mucous membrane colour</strong></td>
<td>3:6:1:0</td>
<td>0:10:0:0</td>
<td>0:3:5:2</td>
<td>10:0:0:0</td>
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<tr>
<td><strong>Haematocrit (t/l)</strong></td>
<td>0.37–0.55</td>
<td>0.34 (0.08)(^a)</td>
<td>0.13 (0.03)(^ab)</td>
<td>0.27 (0.20)(^b)</td>
<td>0.50 (0.04)</td>
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<td><strong>White cell count (×10^9/l)</strong></td>
<td>6.0–15.0</td>
<td>5.5 (1.9)(^a)</td>
<td>8.6 (4.3)(^a)</td>
<td>13.2 (7.3)(^a)</td>
<td>13.3 (6.7)</td>
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<tr>
<td><strong>Immature neutrophil count (×10^9/l)</strong></td>
<td>0–0.30</td>
<td>0.29 (0.54)(^ab)</td>
<td>1.27 (1.53)(^a)</td>
<td>1.88 (2.84)(^b)</td>
<td>0.28 (0.41)</td>
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<td><strong>Urea (mmol/l)</strong></td>
<td>3.6–8.9</td>
<td>6.8 (3.5)(^ab)</td>
<td>13.3 (6.5)(^a)</td>
<td>18.7 (6.4)(^b)</td>
<td>6.7 (2.6)</td>
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<td><strong>Creatinine (µmol/l)</strong></td>
<td>&lt;133</td>
<td>104 (30)(^ab)</td>
<td>84 (25)(^ac)</td>
<td>146 (61)(^bc)</td>
<td>123 (16)</td>
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<tr>
<td><strong>Total bilirubin (µmol/l)</strong></td>
<td>&lt;6.8</td>
<td>10.0 (11.8)</td>
<td>20.5 (23.8)</td>
<td>49.3 (59.6)</td>
<td>14.4 (13.8)</td>
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<td><strong>Alanine transaminase (U/l)</strong></td>
<td>&lt;20</td>
<td>41 (21)</td>
<td>48 (74)</td>
<td>50 (43)</td>
<td>23 (6)</td>
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<tr>
<td><strong>Alkaline phosphatase (U/l)</strong></td>
<td>&lt;190</td>
<td>77 (40)(^ab)</td>
<td>178 (295)(^*)</td>
<td>84 (59)(^a)</td>
<td>44 (23)</td>
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<td><strong>Glucose (mmol/l)</strong></td>
<td>3.3–5.5</td>
<td>5.1 (0.6)(^a)</td>
<td>5.1 (0.8)(^b)</td>
<td>3.5 (2.0)(^ab)</td>
<td>5.2 (0.7)</td>
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<td><strong>Collapsed at presentation</strong></td>
<td>0</td>
<td>2</td>
<td>7</td>
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<td><strong>Died</strong></td>
<td>0</td>
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\(^1\)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 \((P < 0.05)\).
Table 2: Systemic arterial pressures in dogs with babesiosis.

<table>
<thead>
<tr>
<th>Dog</th>
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<td>A: systolic arterial pressure (mm Hg)</td>
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*Significantly different from control group (P < 0.05).

○ = 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.
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 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.

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. Increased systemic capillary permeability occurs in malaria, and its severity is associated with disease severity. 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. This is consistent with recent evidence of increased plasma volume in canine babesiosis, possibly due to movement of interstitial fluid into the vascular bed secondary to hypotension, and with evidence of increased blood volume in the presence of reduced MAP in some patients with malaria. 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. 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’
effector of many inflammatory media-
tors, and a potent and ubiquitous
vasodilator, might play an important
role.

The lack of correlation between blood pressures and most of the clinical para-

teters tested was unexpected. In a large
study, Bodey and Michell 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 pres-
sure dropped, but this did not occur, nor
was it a consistent finding in hypotensive
dogs with endotoxaemia or hypotensive
calves with babesiosis. Surprisingly, PP
and Ht were not related to one another. It
was expected that the ‘bounding’ or ‘waterhammer’ pulse frequently encoun-
tered in severely anaemic dogs with
babesiosis would be caused by a widen-
ing in PP, but the results of this study did
not support this. Pulse pressure was
narrowed in hypotensive dogs, reflect-
ning, overall, a greater drop in SAP than
DAP. A relationship between mucus-
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 with-
out 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 pres-
sure overall. The negative correlation
between blood pressure and white cell
count/immatute neutrophil count might
reflect a relationship between systemic
inflammation and hypotension. An ele-
vated white cell count and neutrophilic
left shift were the most sensitive indica-
tors of sepsis in a recent canine study.

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. As in this study,
pressures were lower in dogs that died
than in those that survived, but the differ-
ences 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, how-
ever, 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 prognos-
tic indicators. The presence or absence of
clinical collapse was the only clinical
factor for which blood pressure measure-
ments differed significantly. However,
evaluation of collapse is subjective, and its
presence or absence was not 100 % sensi-
tive or specific for hypotension (see Fig. 2).

The endpoint of hypotension is hypo-
tensive shock. Shock is essentially a
failure of capillary perfusion. Treatment
of shock in the dog has been reviewed, and
and options for fluid therapy in canine
babesiosis patients with shock have been
suggested. 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, reduc-
ning their beneficial effect on blood pres-
sure and perfusion, and increasing the
likelihood of intermittent oedema. Central
venous pressure is a good indicator of
overhydration and should be monitored if
possible. Serial arterial pressure mea-
surements are useful indicators of the
efficacy of fluid therapy in the absence of
more invasive techniques. Other indica-
tors of the effectiveness of fluid therapy
and resuscitation are normalisation of
urine output, serum lactate concentration
and base deficit.

CONCLUSIONS

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

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