Retrospective Study

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Severe blunt trauma in dogs: 235 cases (1997–2003)

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Abstract

Objective – To evaluate population characteristics, injuries, emergency diagnostic testing, and outcome of dogs with blunt trauma requiring intensive care in an urban hospital.
Design – Retrospective study 1997–2003.
Setting – All data obtained from the University of Pennsylvania – Matthew J. Ryan Veterinary Hospital.
Animals – Dogs admitted to the intensive care unit for treatment following blunt trauma.
Interventions – None.
Measurements and Main results – Of the 235 dogs that met inclusion criteria, 206 (88%) survived and 29 (12%) did not survive. Blunt vehicular trauma accounted for 91.1% of cases. Mild hyperglycemia and hyperlactatemia was common in both survivors and nonsurvivors. The chest was the most common region traumatized and the prevalence of polytrauma was 72.3%. Initial weight, vital signs, PCV, total plasma protein, BUN, glucose, lactate, acid-base status, and electrolytes did not differ between survivors and nonsurvivors. Nonsurvivors were significantly more likely to have had head trauma ($P = 0.008$), cranium fractures ($P < 0.001$), recumbency at admission ($P < 0.001$), development of hematochezia ($P < 0.001$), clinical suspicion of acute respiratory distress syndrome ($P < 0.001$), disseminated intravascular coagulation ($P < 0.001$), multiorgan dysfunction syndrome ($P < 0.001$), development of pneumonia ($P < 0.001$), positive-pressure ventilation ($P < 0.001$), vasopressor use ($P < 0.001$), and cardiopulmonary arrest ($P < 0.001$).
Conclusions – Outcome of severe blunt trauma in dogs treated with intensive care is very good. Despite the high survival rate, several features associated with poor outcome were identified. Neither admission lactate nor glucose was able to predict outcome.

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Introduction

Trauma encompasses physical or psychological injury to a human or animal.[1] Physical injury can be caused by nonpenetrating (blunt) or penetrating trauma. Blunt trauma includes vehicular trauma, crush/compression, or acceleration/deceleration injury. An accident resulting in blunt trauma may include 1 or all of these mechanisms of injury. Traumatic injury can result in acute death due to overwhelming primary damage, initial survival, and subsequent death due to complications (delayed hemorrhage, multiorgan dysfunction, infection, or sepsis), or patient survival to discharge.[2] An epidemiological study of 289 human trauma deaths in an urban area demonstrated a prehospital mortality rate of 34%, and of those admitted to the hospital and subsequently died, 81% died acutely (within 48 h).[2] Prehospital death occurred from CNS injuries in 43% of cases, exsanguination in 36% of cases, or a combination of both in 9% of cases.[2] These statistics are similar for acute in-hospital mortality, while organ failure was most common in late (>7 d) in-hospital mortality (61%).[2]

Physical traumatic injury induces an acute inflammatory response. Following initial trauma or shock there is activation of the complement system.[3] Generation of complement's active components results in chemotaxis of neutrophils, and leukocyte activation.

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This initial response to injury is protective; however, if its activation is in excess or widespread, a systemic inflammatory response may result. Studies have demonstrated an elevation in the chemokine IL-8, early in the posttraumatic injury period,[4–7] as well as consistent elevations in IL-6 following tissue trauma, hemorrhage, and shock.[4–8] Injury disrupts endothelium and blood vessels resulting in exposure of cells that constitutively express tissue factor.[9] This upregulation of tissue factor may result in a systemic inflammatory response and widespread activation of the coagulation cascade, and evolution into hypercoagulable states.[9–14] Derangement of anticoagulant pathways, such as antithrombin depletion, and decreased levels of activated protein C result in further abnormalities of coagulation and may progress to fulminant disseminated intravascular coagulation.[9,10,12] Occult hypoperfusion and tissue hypoxemia may occur secondary to hypovolemia, microvascular thrombosis, and subsequent microvascular collapse, leading to multiorgan dysfunction. The combination of primary injury and host response may be responsible for a significant proportion of in-hospital mortality from multiple organ failure among severe blunt trauma patients who survive their primary injury.

Trauma is the leading cause of death among young adults (ages 15–24) and the fifth leading cause for all age groups nationally.[15] An epidemiological study of traumatic deaths in an urban population over the 1-year study period showed that the incidence penetrating and blunt trauma were similar.[2] The majority of deaths occurred in the hospital (65%) compared with deaths en route or at the scene.[2] In this study 81% of in-hospital deaths from trauma occurred in the first 48 hours, 14% after 7 days, and 6% between 48 hours and 7 days.[2] Acute death (0–48 h postinjury) resulted from exsanguination (42%), CNS injuries (39%), or multiorgan failure (7%).[2] Both exsanguination and CNS injuries, as a cause of in-hospital death, peaked within the first 12 hours after admission.[2] Alcohol abuse and presence of comorbid conditions, which have been shown to be independent predictors of mortality, may confound results in human trauma studies.[16,17]

Traumatic injury is a common occurrence in veterinary medicine; however, no national database for data collection exists for veterinary patients. Scoring systems for veterinary trauma are limited, and only the animal trauma triage scoring system for dogs and cats has had statistical validation.[18] An index of disease severity called the improved survival prediction index (SPI2)[19] has also been created for critically ill dogs. However, the SPI2 is not specific to the trauma patient, and relies on the most severe values for the first 24-hour period of admission to the ICU.[19] Utilization of a large veterinary database for acute severe trauma may allow development of an accurate trauma scoring system that could be used to identify blunt trauma patients at risk for death early in the course of their hospitalization. The National Trauma Data Bank is the largest collection of data regarding human trauma and is overseen by the American College of Surgeons.[20] This sizeable resource has allowed the development of patient scoring systems for adult, adolescent, and pediatric patients. Scoring systems rank patients based on injury severity and degree of physiologic derangement, allowing patient stratification. Clinical application of a trauma scoring system for veterinary patients would allow for early identification of animals at higher risk for death and complications, more focused treatment and care, and may provide prognostic information for the client. Additionally, prospective studies of trauma patients require such scoring systems to help elucidate morbidity and mortality relationships.

There is a paucity of data regarding severe blunt trauma in dogs. A large retrospective analysis of traumatic injury in both dogs and cats was performed in 1975 by Kolata and Johnston.[21] Although this study provided valuable basic information regarding signalment, traumatized region, and outcome, it did not provide focused data on the select population of dogs affected by severe blunt trauma. The Kolata and Johnston study included a large percentage (31%) of animals with minimal injuries, and may not be an accurate reflection of severe blunt trauma cases. A descriptive trauma study generating information on animals that had severe trauma and could not be stabilized shortly after hospital admission is needed. The current veterinary literature lacks information on mortality rate, cause of death, injuries, and morbidity and mortality predictors within this subpopulation of severe trauma.

The regularity with which veterinary patients are afflicted by blunt trauma injury, their lack of comorbid conditions (particularly drug and alcohol abuse), and the nature of the trauma (blunt vehicular trauma) make the dog a particularly useful model for some aspects of human trauma. Additionally, study of the severe canine blunt trauma patient provides an opportunity to collect a large body of critical information that may help in the development of a trauma scoring system and advance the care of these patients. We performed a retrospective study to characterize the injuries and response to severe blunt trauma in dogs.

Materials and Methods

Animals

A search of the computerized database at the Matthew J. Ryan Veterinary Hospital at the University of Pennsylvania for dogs sustaining blunt trauma that received

ongoing intensive care after initial emergency stabilization between January 1, 1997 and December 31, 2003 revealed 305 potential cases. Exclusion criteria included inappropriate coding of the medical record for species, no evidence of trauma, stable postoperative ICU stay, no ICU stay, and penetrating trauma or bite wounds.

Data collection

All records were retrospectively reviewed for patient admission characteristics including: signalment, body weight, physical examination findings, temperature, heart rate, respiratory rate, blood pressure, pulse oximetry, and presence of arrhythmias (review of ECG strips, or written medical record documentation). Characteristics of the trauma such as trauma source, time from trauma to hospital presentation, location of injuries (head, chest, abdomen, and extremity), and acquired conditions resulting from trauma were recorded.

An emergency database was collected at presentation (PCV, total plasma protein [TPP], glucose, electrolytes, and venous blood gas) via direct venipuncture or upon placement of an IV catheter, anticoagulated with lithium heparin and typically analyzed[a] within 15 minutes. More extensive diagnostic test results (eg, CBC, chemistry profile, and coagulation profile) were reviewed if samples were drawn in the first 12 hours after admission to the hospital.

Previous treatment (if the dog had been seen initially at a different veterinary practice), fluid type and volume used for resuscitation, blood products used and volume given, analgesic agents used, and type of vasopressor were recorded. Surgical procedures were described by type and duration, anesthesia time, and postoperative temperature. Total hospitalization was divided into total days and days strictly in ICU.

Outcome was defined as survival or nonsurvival to discharge. Nonsurvival was subdivided into in-hospital deaths, euthanasia due to perceived poor prognosis, or euthanasia due to owner's financial constraints. Survivors and nonsurvivors were compared to determine parameters associated with morbidity (eg, hospital duration, development of complications such as disseminated intravascular coagulation [DIC], multiorgan dysfunction syndrome [MODS], acute respiratory distress syndrome [ARDS]) and mortality.

Definitions

For the purposes of syndrome recognition the following definitions were used. In all cases the syndrome must have appeared in the medical record with supporting clinical evidence of the condition. DIC was defined as development of coagulopathy and thrombocytopenia with elevation in fibrin degradation products, or elevation in D-dimer levels, or both, with clinical suspicion of the syndrome by the attending veterinarian.[22,23] ARDS was defined as having bilateral pulmonary infiltrates, severe hypoxemia, and a clinical suspicion of the syndrome by the attending veterinarian.[24] MODS was identified if, in addition to clinical suspicion of the condition by the attending veterinarian, 2 or more of the following developed: creatinine $>106\,\mu mol/L$ ($1.2\,mg/dL$), bilirubin $>22.2\,\mu mol/L$ ($1.3\,mg/dL$), vasopressor use was required, platelet count $<120 \times 10^9/L$, clinical signs of elevated intracranial pressure, or ARDS.[23]

Data analysis

Descriptive data were reported as the mean $\pm$ SD if the distribution satisfied the Kolmogorov-Smirnov test for normality. Nonparametric data were reported as a median with its interquartile range (IQR). For comparisons of nominal data between survivors and nonsurvivors the Student *t*-test was used for normally distributed data and the Mann-Whitney test was use for nonparametric data. For comparison of categorical data between survivors and nonsurvivors, a χ^2 test was used for 2 or more categories as long as there were >5 data points per category. If categories contained <5 data points, a Fisher exact test was used. All statistical analysis was performed using standard statistical software.[b] Results were considered significant if they achieved a value of $P<0.05$.

Results

Of the records reviewed, 235 met inclusion criteria and 70 cases were excluded from further evaluation. Twenty-one cases did not have evidence of trauma, 20 cases were stable postoperative patients, 13 had bite wounds, 6 had no recorded ICU admission, 4 had no emergency stay data, 3 did not have recent trauma, 2 were cats, and 1 record could not be found. Admission characteristics, trauma cause, and trauma distribution are depicted in Table 1 and Figures 1 and 2, respectively.

Injuries and acquired conditions for all patients

The extensive database allowed generation of 67 trauma descriptors for various injuries and acquired conditions. Chest injuries included pulmonary contusions in 58% of all patients, pneumothorax in 47%, hemothorax in 18%, rib fractures in 14%, pneumomediastinum in 8%, diaphragmatic hernia in 6%, pulmonary bullae in 2%, and flail chest in 2%. A clinical diagnosis of head trauma was made in 25% of all patients. Clinical evidence of epistaxis and fractures to bones of the skull were each present in 11% of all patients. The most common abdominal injury was hemo-

Table 1: Canine severe blunt trauma patient admission characteristics

Parameter	*N*	Mean	Median	SD	IQR
Age (y)	235		2.5		0.9–5.1
Weight (kg)	235		21		11.7–28.9
Temperature (°C/°F)	235		38.4/101.1		37.7/99.8–38.8/101.9
Heart rate (beats/min)	235		141		120–168
Respiratory rate (breaths/min)	235		54		36–80
Systolic blood pressure (mm Hg)	108*	114		29	
Diastolic blood pressure (mm Hg)	102	72		22	
Mean blood pressure (mm Hg)	107	86		24	
Pulse oximetry (%)	94		93		91–95

*Doppler blood pressure that could not be detected.
IQR, interquartile range; *N*, number of tests performed.

peritoneum in 23%, followed by abdominal hernias in 5%, and urinary tract rupture in 3% of all cases. Soft tissue injuries in all patients included superficial abrasions in 56%, lacerations in 26%, SC emphysema in 10%, major degloving injury in 8%, and other less common injuries in 7% of all cases. Forelimb appendicular orthopedic injuries included scapular fractures in 7%, elbow luxations in 3%, and radius fractures in 2% of all cases. Hind limb appendicular orthopedic injuries included pelvic fractures in 28% of patients, femur fractures in 16%, hip luxations in 12%, and distal limb fractures in 8% of all cases. Axial orthopedic injuries included spinal fractures in 10%, sacral luxations in 9%, and sacral fractures in 3% of all cases. Acquired conditions for all patients included recumbency at admission in 41%, arrhythmias in 22%, hematuria in 12%, development of a cough in 7%, regurgitation in 6%, hematochezia in 5%, MODS in 4%, development of pneumonia in 4%, hemoptysis in 4%, requirement for mechanical ventilation in 4%, development of ARDS in 3%, respiratory arrest in 2%, cardiopulmonary arrest in 2%, melena in 2%, and DIC in 2% of all cases.

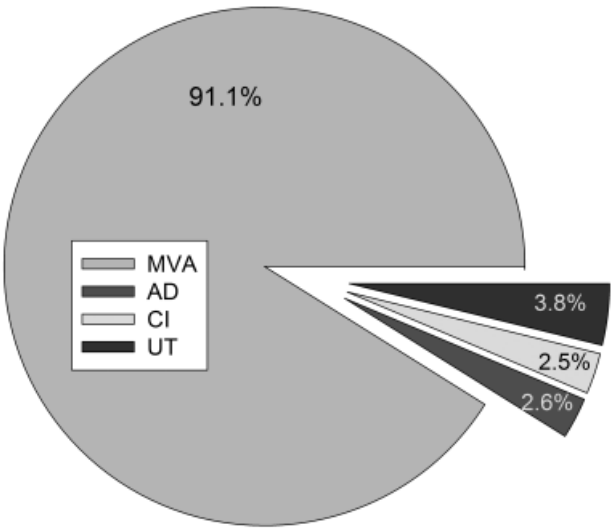

Figure 1: Percentage of dogs with severe trauma from unknown trauma (UT), motor vehicle accidents (MVA), crush/compressive injury (CI), and acceleration/deceleration (AD) for all 235 dogs.

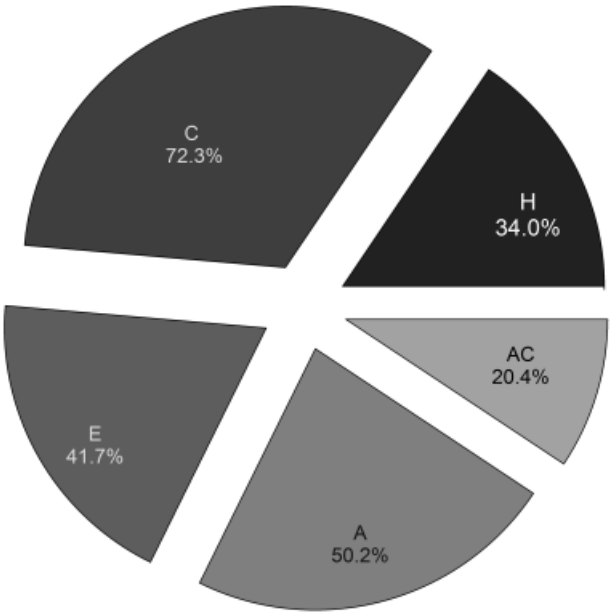

Figure 2: Percentage of anatomic blunt trauma distributions in all 235 dogs for head/neck trauma (H), chest (C), extremity (E), abdomen (A), and abdomen and chest (AC). Other combinations were <9% and trauma in all regions accounted for 2.6%. Because of polytrauma results do not add to 100%.

Hematologic/biochemical findings

Results of analysis of initial PCV/TPP, PCV/TPP after fluid resuscitation, venous blood gas, CBC, and serum biochemistry for all patients were analyzed. Although individuals varied, the overall mean and median values for initial PCV/TPP, pH, $PvCO_2$, bicarbonate, sodium, potassium, chloride, BUN, ionized calcium, ionized magnesium, phosphorous, creatinine, and total bilirubin were within the normal reference interval. Coagulation tests performed in the clinical laboratory (prothrombin time [PT] and activated prothrombin time [aPTT]) were performed in only 53 patients. The PT and aPTT tests were within the reference interval in 79.2% and 52.8% of patients, respectively. Mild prolongation (25–50% greater than control) of PT and aPTT was observed in 13.2% and 30.2% of cases, respectively. Moderate prolongation

Table 2: Abnormal hematologic/biochemical results for all severe blunt trauma dogs

Parameter	*N*	Mean	Median	SD	IQR	Reference interval
PCV* (%)	175	35		9.59		
Total plasma protein† (g/L)	175	50		13		
Total plasma protein† (g/dL)	175	5		1.3		
Blood glucose (mmol/L)	235		11.1		6.4–8.6	3.6–6.2
Blood glucose (mg/dL)	235		131		115–155	65–112
Lactate (mmol/L)	218		3.5		1.9–5.8	<2
Base excess	225	−3		−6		−4
Total WBC count ($\times 10^9$/L)	206		17		12.2–23.8	5.3–19.8
Platelet count ($\times 10^9$/L)	196		171		122–230	177–398
Alanine aminotransferase (U/L)	194		746		229–1857	16–91
Aspartate transaminase (U/L)	191		678		224–1267	23–65
Alkaline phosphatase (U/L)	195	118	118		59–177	24–174
Albumin (g/L)	195	24.4		6.5		25–37
Albumin (g/dL)	195	2.44		0.65		2.5–3.7

*PCV after fluid resuscitation. †After fluid resuscitation.
N, number of tests; IQR, interquartile range.

(50–100% greater than control) of PT and aPTT was observed in 7.5% and 13.2% of patients, respectively. No patient's PT was prolonged >100% of control and aPTT was >100% of control in only 2 patients (3.8%) that were tested. The median and mean values for PCV/TPP after fluid resuscitation, blood glucose, base excess (BE), lactate, total WBC, platelet count, aspartate aminotransferase, and alanine aminotransferase median and mean values were outside the reference interval following severe blunt trauma in all 235 patients (see Table 2).

Treatment

Fifty-five percent of dogs (113/206 survivors and 16/29 nonsurvivors) were referred to our hospital from another veterinarian, with the remaining 45% of dogs presenting directly to our hospital after traumatic injury. An IV catheter was placed in all patients. An initial IV fluid bolus was administered in 114 patients. Isotonic crystalloid solution[c] was used as a sole agent for initial fluid resuscitation in 55 of 114 patients and no patient received an artificial colloid (dextran[d] or hydroxyethyl starch[e]) as a sole agent. A combination of colloid and 7.5% hypertonic saline[f] was used in 14 patients. Initial resuscitation also included the use of mannitol[g] in 18 patients, fresh frozen plasma (FFP) in 12 patients, and packed RBC (pRBC) in 13 patients. A combination of more than 1 fluid type was used in 39 patients. Fluid bolus volume was variable and differed based on patient weight and fluid type. A total of 57 patients received pRBC transfusions during hospitalization (mean of 1.1 U of pRBC and range 0.3–3.0 U, where 1 U represents the red cells obtained from 450 mL of blood). Sixty-six patients received FFP infusions during hospitalization (mean of 1.8 U and range of 0.3–4 U; 1 U is 120 mL). Initial analgesia was provided in 199 patients with opioids being used most frequently (162 patients). Oxygen support (nasal or oxygen cage) was required in 119 patients and the mean duration of oxygen treatment was 40 hours (± 36 h) during course of hospitalization. Pneumothorax was sufficient to warrant chest tube placement in 34 patients. Mechanical ventilation was instituted in 9 of 235 patients that demonstrated worsening respiratory function with increased rate/effort. Corticosteroids were administered to 49 patients (86% of these were referred from local veterinarians). An abdominal compression wrap was placed in 12 of the 53 patients with hemoperitoneum during initial stabilization. Only 3 patients with severe hemoperitoneum required surgery (2 total splenectomies and 1 liver lobectomy). Surgical intervention was required in 119 patients and 19 patients required more than 1 procedure. The type of surgical intervention varied based on injuries incurred; however, 63.5% of procedures were orthopedic and 36.5% involved soft tissue. The mean number of days from admission to surgery was 2.2 days (± 1.7 d). Total surgical time for all surgical patients was a mean of 2.97 hours (± 1.66 h) and total anesthetic time was 4.35 hours (± 2.35 h). Mean postoperative temperature was 36.1°C (97.0°F) ± 1.5°C (2.6°F). Vasopressors were used in 3 patients during their ICU stay. One patient received a combination of dopamine[e] and dobutamine,[e] a second received dobutamine and phenylephrine,[h] and 1 patient received epinephrine.[i] The mean duration of hospitalization was 5.32 days (± 3.07 d) and the mean number of days spent in ICU was 2.98 days (± 2.12 d).

Outcome

Two hundred and six patients were successfully discharged (survivors) and 29 died or were euthanized (nonsurvivors). The overall survival rate of severe blunt trauma injury in dogs requiring intensive care was 88%.

Stratification of nonsurvivors revealed in-hospital death in 1 patient, euthanasia due to perceived poor prognosis in 23 patients, and euthanasia due to financial constraints in 5 patients. When euthanasia due to financial constraints was excluded, mortality rate was 10% and when euthanasia for any reason was excluded mortality rate was 0.005%.

Comparison of survivor and nonsurvivor data

The nonsurvivor median age was 3.3 years (IQR 1.0–8.0 y) compared with survivor median age of 2.3 years (IQR 0.9–5.0 y) ($P = 0.17$). The most common breeds were mixed breed dogs (32%), American Pit Bull Terriers (5%), German Shepherd Dogs (4%), and Labrador Retrievers (4%) and proportions were not different between survivors and nonsurvivors. There was no detectable effect of sex or estrogen status on survival. Admission body temperature was 38.4°C (101.1°F) (IQR 37.8–38.8°C [100.0–101.9°F]) in survivors compared with 37.7°C (99.8°F) (IQR 36.7–39.1°C [98.0–102.4°F]) ($P = 0.13$) in nonsurvivors. There were no detectable differences in the following admission data: time to treatment (survivors: median 3.2 h, IQR 1.0–12.0 h; nonsurvivor: median 3.5 h, IQR 1.0–10.1 h; $P = 0.98$), body weight (survivors: median 21.6 kg, IQR 12.0–28.8 kg; nonsurvivor: mean 19.3 ±11.2 kg; $P = 0.35$), heart rate (survivor: median 144/min, IQR 120–168/min; nonsurvivor: median 140/min, IQR 120–163/min; $P = 0.58$), respiratory rate (survivor: median 56/min, IQR 40–80/min; nonsurvivor: median q48/min, IQR 30–77/min; $P = 0.21$), systolic blood pressure (survivor: mean 114 ± 27 mm Hg; nonsurvivor: mean 116 ± 35 mm Hg; $P = 0.74$), diastolic blood pressure (survivor: mean 73 ± 21 mm Hg; nonsurvivor: mean 72 ± 29 mm Hg; $P = 0.96$), mean arterial pressure (survivor: mean 87 ± 23 mm Hg; nonsurvivor: mean 86 ± 27 mm Hg; $P = 0.85$), or pulse oximetry (survivor: mean 93 ± 3%; nonsurvivor: mean 93 ± 2%; $P = 0.72$).

Comparison of survivor and nonsurvivor injuries and acquired conditions

Polytrauma combinations for survivors and nonsurvivors were similar between groups. Patients with a clinical diagnosis of head trauma had significantly decreased survival (45/206 survivors; 14/29 nonsurvivors; $P = 0.008$). Cardiac arrhythmias, while noted in over 22% of all patients, were not associated with an increase in mortality. All 52 cases of documented arrhythmias were ventricular in origin, except for 4 cases with bradyarrhythmias associated with opioid use. All but 2 cases had paroxysmal arrhythmias. Information regarding the specific response to therapy was not available from the medical records. The acquired conditions that were significantly different between survivors and nonsurvivors were grouped into cardiovascular, respiratory, and neurologic and are depicted in Figures 3–5. The small numbers of nonsurvivors and the large number of different injuries limits the power to detect subtle differences for the other injuries and acquired conditions.

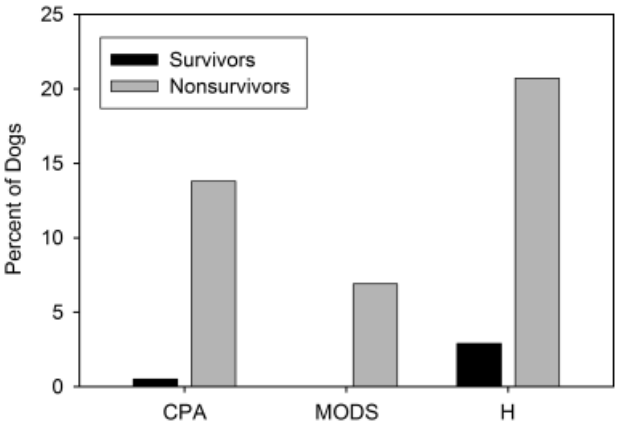

Figure 3: Cardiovascular conditions in dogs resulting from severe blunt trauma (S = 206 survivors; N = 29 nonsurvivors). CPA, cardiopulmonary arrest; MODS, multiorgan dysfunction syndrome (those with cardiovascular dysfunction); H, hematochezia. H was included in this group as it may reflect underlying gastrointestinal perfusion deficits. All were significantly associated with nonsurvival with $P < 0.001$.

Comparison survivor and nonsurvivor emergency database

No detectable difference in initial PCV/TPP and PCV/TPP after volume resuscitation was present between

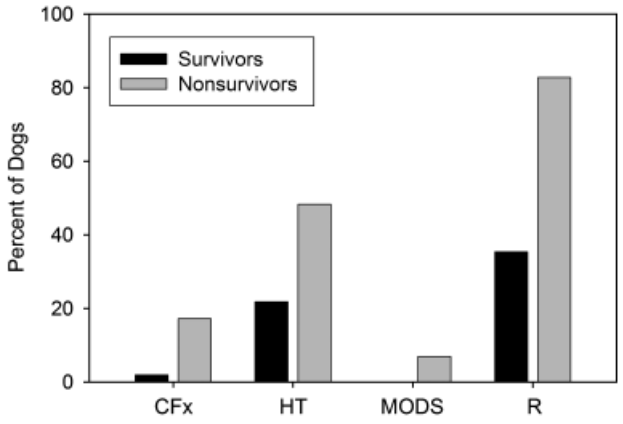

Figure 4: Neurologic conditions in dogs resulting from severe blunt trauma (S = 206 survivors; N = 29 nonsurvivors). CFx, cranium fracture; HT, head trauma; MODS, multiorgan dysfunction syndrome (those with neurologic dysfunction); R, recumbency at admission. R was included in this group; however, the cause may be multifactorial. All were significantly associated with nonsurvival (HT $P = 0.008$; remainder $P < 0.001$).

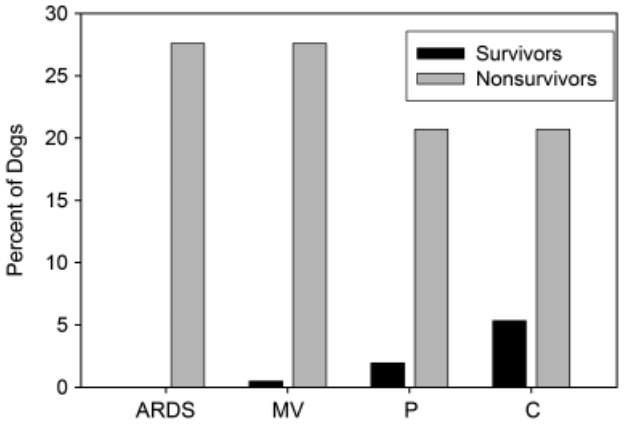

Figure 5: Respiratory conditions in dogs resulting from severe blunt trauma (S = 206 survivors; N = 29 nonsurvivors). ARDS, acute respiratory distress syndrome; MV, mechanical ventilation; P, pneumonia; C, development of a cough. All significantly associated with nonsurvival (C $P < 0.009$; remainder $P < 0.001$).

survivors and nonsurvivors. Likewise initial blood glucose, lactate, BUN, BE, bicarbonate, $PvCO_2$, pH, sodium, chloride, potassium, ionized magnesium, and ionized calcium were not different between groups. Results are depicted in Table 3.

Comparison of survivor and nonsurvivor hematologic, biochemical, and coagulation profiles

CBC analysis and serum biochemical testing were performed in all but 28 cases (24 survivors and 4 nonsurvivors) and all but 40 cases (35 survivors and 5 nonsurvivors), respectively. All samples were obtained within 12 hours of presentation to the hospital, and there were no detectable differences in any of the parameters between survivors and nonsurvivors. Results are depicted in Table 4.

Coagulation testing was only performed in 53 cases (43 survivors and 10 nonsurvivors). Results of PT and aPTT were stratified into groups based on <25%, 25–50%, and 50–100% prolongation from control. There were no detectable differences between survivors and nonsurvivors.

Comparison of survivor and nonsurvivor treatments

Oxygen therapy provided by nasal device or oxygen cage lasted a median of 53 hours in nonsurvivors compared with a median of 32 hours in survivors ($P = 0.09$). Overall, the use of FFP or pRBC was not different between groups. In a subgroup analysis of only the dogs that received pRBC transfusions, the number of required units of pRBC was higher in nonsurvivors (nonsurvivor: median 1.5 U, IQR 1.0–2.0 U; survivor: median

Table 3: Emergency database comparison between survivors and nonsurvivors

	Survivors				Nonsurvivors				
Parameter	**Mean**	**SD**	**Median**	**IQR**	**Mean**	**SD**	**Median**	**IQR**	***P* value**
PCV(%)	43.4	9.2			41.9	11.5			0.44†
PCV(%)*	34.4	9.4			33.9	9.8			0.81†
Total plasma protein (g/L)			60	52–68			58	47–70	0.53
Total plasma protein (g/dL)			6.0	5.2–6.8			5.8	4.7–7.0	0.53
Total plasma protein (g/L)*	50	13			45	13			0.13†
Total plasma protein (g/dL)*	5.0	1.3			4.5	1.3			0.13†
Blood glucose (mmol/L)			7.3	6.3–8.4			7.4	6.7–11.4	0.16
Blood glucose (mg/dL)			131	114–151			134	120–205	0.16
Lactate (mmol/L)			3.5	1.7–5.4			4.3	21.0–7.13	0.29
Blood urea nitrogen (mmol/L)			5.4	3.6–6.4			6.4	3.6–8.9	0.86
Blood urea nitrogen (mg/dL)			15.0	10.0–18.0			18.0	10.0–25.0	0.86
pH			7.355	7.296–7.392			7.307	7.245–7.394	0.09
$PvcO_2$ (mm Hg)			41	35.4–46.2			45.1	36.7–54.5	0.07
Base excess	−3.0	−5.2			−3.7	−6.8			0.39
Bicarbonate (mmol/L)	22.0	3.8			22.0	3.8			0.95†
Sodium (mmol/L)			148	145–149			147	146–150	0.56
Chloride (mmol/L)			113	111–115			114	111–116	0.88
Potassium (mmol/L)	3.90	0.45			3.96	0.56			0.48†
Ionized calcium (mmol/L)			0.60	0.57–0.63			0.60	0.55–0.65	0.44
Ionized calcium (mEq/L)			1.20	1.14–1.25			1.19	1.09–1.29	0.44
Ionized magnesium (mmol/L)			0.18	0.15–0.22			0.18	0.18–0.23	0.06
Ionized magnesium (mEq/L)			0.35	0.30–0.44			0.35	0.35–0.46	0.06

*After volume resuscitation.
†Insufficient statistical power.
IQR, interquartile range.

Table 4: Hematologic and biochemical profile comparison between survivors and nonsurvivors

	Survivors		Nonsurvivors		
Parameter	**Median**	**IQR**	**Median**	**IQR**	***P* value**
Total WBC count ($\times 10^9$/L)	17.2	13.6–23.8	13.1	7.6–23.3	0.07
Neutrophils ($\times 10^9$/L)	15.7	11.9–22.1	12.6	6.3–20.9	0.07
Platelets ($\times 10^9$/L)	169	122.0–222.5	203	109.3–296.5	0.28
Alanine aminotransferase (U/L)	828	285.5–2048.3	236	84.3–1321.8	0.01
Aspartate transaminase (U/L)	690	268.3–1293.3	439.5	103.0–946.0	0.12
Alkaline phosphatase (U/L)	113	61.0–173.5	130	53.5–204.0	0.79
Total bilirubin (μmol/L)	8.6	5.1–12.0	8.6	6.8–12.0	0.45
Total bilirubin (mg/dL)	0.5	0.3–0.7	0.5	0.4–0.7	0.45
Albumin (g/L)	24	20–28	22	18–29	0.36
Albumin (g/dL)	2.4	2.0–2.8	2.2	1.8–2.9	0.36
Creatinine (μmol/L)	70.7	53.0–97.2	79.6	53.0–114.9	0.87
Creatinine (mg/dL)	0.8	0.6–1.1	0.9	0.6–1.3	0.87
Phosphorous (mmol/L)	1.5	1.2–1.8	1.5	1.3–1.9	0.86
Phosphorous (mg/dL)	4.7	3.7–5.7	4.5	3.9–6.0	0.86

IQR, interquartile range.

1.0 U, IQR 0.5–1.0 U; $P = 0.02$). For the same subgroup, nonsurvivors received a median dose of 0.095 U/kg (IQR 0.06–0.17 U/kg) compared with survivors that received a median dose of 0.060 U/kg (IQR 0.04–0.09 U/kg) ($P = 0.07$). In the subgroup of dogs receiving FFP, there was no detectable difference in volume given between survivors and nonsurvivors (nonsurvivors: median 2.3 U, IQR 1.5–3.3 U; survivors: median 2.0 U, IQR 1.0–2.0 U; $P = 0.20$). The dose of FFP was not different between groups (nonsurvivor: median 0.11 U/kg, IQR 0.08–0.24 U/kg; survivor: median 0.08 U/kg, IQR 0.05–0.12 U/kg; $P = 0.12$). Mechanical ventilation was associated with nonsurvival ($P < 0.001$). All 3 patients that required vasopressor support were nonsurvivors. There were no detectable differences in corticosteroid use between groups. Likewise, no detectable association was found between mortality and surgical intervention, surgery duration, anesthesia duration, or postoperative temperature.

Discussion

Most dogs within this blunt trauma population were young, medium-sized, and victims of motor vehicle trauma. The chest was the most commonly affected individual location. Patients suffering severe blunt trauma might be expected to have multiple injuries and indeed polytrauma was noted in 72.3% of patients, with the abdomen and chest being the most common regions affected. Tachycardia and tachypnea were common physical examination findings.

At admission, mild hyperglycemia, mild hyperlactatemia, mild uncompensated metabolic acidosis, mild hypoalbuminemia, mild thrombocytopenia, and moderate increases in serum alanine aminotransferase were common. The remainder of the presenting venous blood gas, CBC, and serum chemistry profile results were unremarkable.

Recumbency at admission, development of hematochezia, pneumonia, DIC, ARDS, or MODS, requirement for mechanical ventilation, use of vasopressors, and cardiopulmonary arrest were all associated with nonsurvival. Furthermore, nonsurvivors had a significantly higher incidence of cough and head trauma ($P = 0.009$ and 0.008, respectively).

Neither the initial blood parameters (venous blood gas and electrolytes) nor the CBC and chemistry profile results, obtained within 12 hours of admission, predicted morbidity or mortality in this group of severely traumatized dogs. In the 22% of cases in which coagulation testing was performed, no significant difference between survivors and nonsurvivors could be detected. Of the treatments used in the management of these dogs, only mechanical ventilation ($P < 0.001$) and use of vasopressors ($P < 0.001$) were significantly associated with nonsurvival.

Survival for severe blunt trauma was excellent. Only 29 of the 235 dogs in this study died or were euthanized. One patient died in the hospital, 23 were euthanized due to perceived poor prognosis, and 5 patients were euthanized due to financial limitations. When euthanasia due to financial limitations was excluded the mortality rate in this group of dogs with severe blunt trauma was 10%.

There are few veterinary studies that have characterized blunt trauma in dogs and none included a focus on dogs with severe trauma, defined in humans[25] by presence of mortality, an injury severity score >15, need for

in-hospital fluid resuscitation, invasive CNS monitoring, or as in our study, ICU admission. With the advent of ICU care and the establishment of the American College of Veterinary Emergency and Critical Care, the level of care offered to veterinary trauma patients has improved over the last 30 years, making new data particularly useful. In 1 study evaluating cases from 2 large urban veterinary hospitals, trauma accounted for 13% of all cases, with 35% of these cases considered to involve serious injuries.[21] Thirty-one percent of cases sustained mild injuries (eg superficial wounds). Multiregion trauma was noted in 36% of patients in Kolata and Johnston's[21] study and the skeletal system was the most common site, accounting for 87% of those injuries. CNS injuries occurred in 50% of cases. The overall fatality rate was 12.5%. The reported cause of death or euthanasia was directly related to the primary injury (intrathoracic and intra-abdominal injury).[21] Euthanasia was associated with primary injury of the chest or abdomen in Kolata and Johnston's study; therefore, data from more severely traumatized dogs was not available for comparison with our results. As might be expected, in the population we studied that sustained severe trauma and required an ICU stay, the incidence of polytrauma doubled. In contrast to the general trauma population,[21] the most commonly traumatized region in these severely traumatized dogs was the chest. Head trauma and hemoperitoneum accounted for 25.1% and 22.6% of injuries in our study. This combination of injuries may explain why these patients required ongoing intensive care, as they represent injury to and dysfunction of vital organs.

Although the chest was the most commonly traumatized region in our study, the presence of thoracic trauma was not associated with outcome. The high incidence of pulmonary contusions and pneumothorax in our study is consistent with a retrospective study of dogs and cats with mild to moderate blunt trauma.[26] The most common thoracic injuries were pulmonary contusions (44%) and pneumothorax/pneumomediastinum (30%).[26] Another retrospective study reporting blunt vehicular trauma in dogs, also showed chest wall injury and pulmonary trauma in 38% of cases.[27] The study by Spackman et al[27] documented a higher incidence of chest trauma with each subsequent orthopedic fracture. In contrast, our study documented isolated thoracic trauma in 72% of cases and extremity trauma in 36% of cases. Two prospective studies evaluating thoracic ultrasound in the general trauma population reported a 17.5%[28] and 21%[29] incidence of pneumothorax. The higher incidence of pneumothorax in our study is consistent with the higher incidence of thoracic trauma in the severely traumatized patient.

In contrast to blunt trauma from an animal being struck by a motor vehicle, in dogs that sustained trauma from jumping or falling out of a moving vehicle, pulmonary contusions (2/70), and head trauma (1/70) were uncommon.[30] The orthopedic injuries, however, were similar, despite the differences in the mechanism of injury, with pelvic and femoral fractures representing the most common orthopedic injuries.[30]

Blunt trauma to the spine resulting in spinal fractures and luxations has been previously reported in dogs and cats.[31] The study by Bruce and colleagues illustrated that the most common cause of spinal injury was blunt vehicular trauma accounting for 63% of cases. Thirty-six percent of Bruce et al's[31] patient cohort were treated surgically due to the severity of neurologic signs. In the current study 17 of 24 cases with spinal injury (sacrum excluded) required surgical intervention due to the severity of neurologic signs (8/17 were splinted; 9/17 surgically treated) and similar to Bruce et al's study, 50% had fractures or luxations involving the T3-L3 region. Seventeen percent of nonsurvivors had spinal fractures in the current study compared with 9% of the survivors ($P = 0.13$). Information regarding outcome and neurologic recovery was unavailable due to the retrospective nature of this study.

Cardiac arrhythmias indicating possible blunt cardiac injury were not uncommon in our study group and may reflect the high percentage of thoracic trauma. The cause of arrhythmias following blunt trauma is likely multifactorial especially in the polytrauma patient who may be suffering from a variety of conditions such as thoracic trauma, hypoxemia, and hypoperfusion. The incidence of possible blunt cardiac injury and subsequent cardiac arrhythmias in our study was 21.3% in the first 24 hours, but may be under reported due to the retrospective nature of our investigation. There was no association between vasopressor use and development of arrhythmias. ECG abnormalities may not be seen at initial screening or may be sporadic in occurrence. Significant arrhythmias developed in 7–23% of humans with blunt chest trauma.[32] In a large veterinary study, screening ECG (ie, 2-min ECG recording) at presentation only identified ventricular ectopy in 16% of dogs with motor vehicle-related blunt trauma[33] compared with 96% when Holter monitoring was applied. Of the patients with Holter recordings, 16% developed significant arrhythmias (ie, $>4{,}000$ ventricular ectopic beats per day) but none had overt clinical signs. The occurrence of arrhythmias appeared to be sporadic, did not correlate to their animal trauma triage score, and onset was as long as 72–96 hours postinjury. All of these dogs had major trauma in 1 or more anatomic regions and the most common region affected was an extremity. Fifty percent of the dogs had normal thoracic

radiographs and only 1 patient died or was euthanized, suggesting that this study underrepresented severe trauma.[33] The high incidence of chest trauma in our study as well as the high incidence of polytrauma may help explain the higher rate of cardiac arrhythmias observed (21.3%), although, lack of Holter monitoring may have led to underreporting of arrhythmias. There was no difference with respect to arrhythmias between survivors and nonsurvivors. More intensive monitoring may help elucidate the true incidence and clinical significance of blunt cardiac injury.

The incidence of hemoperitoneum in this study was 22.6%, which is lower than the 38% hemoperitoneum incidence reported in a prospective ultrasound-based study of focused assessment with sonography for trauma in dogs.[29] The current study was retrospective in nature and did not use focused assessment with sonography for trauma for every case, therefore, effusions that were clinically silent were unlikely to be documented. Although ultrasound is a sensitive tool, presence of abdominal fluid may not indicate severe trauma. Interestingly, only 3 of 53 severe trauma dogs required surgical intervention in our study and in Boysen et al's study,[29] no dogs required surgery, demonstrating that many cases of hemoperitoneum secondary to blunt trauma injury can be medically managed. This concept is supported by a retrospective study in which medical management of traumatic hemoperitoneum did not decrease survival overall and significantly improved survival in larger breeds.[34] The incidence of severe trauma leading to acute and life-threatening hemorrhage in veterinary blunt trauma patients is unknown. It appears that for patients that initially survive their trauma, surgical intervention for hemoperitoneum is rarely required.

Pulmonary contusions were the most common injury in our study. Mechanical ventilation was only required in 9 of 235 cases and the reasons for ventilation included pneumonia, ARDS, or pulmonary contusions. The survival rate in a group of dogs with pulmonary contusions and mechanical ventilation was previously reported as 30%.[35] This is higher than the rate observed in our study where only 1 of 9 ventilated patients survived. One ventilated patient in our study was euthanized due to financial reasons as the need for mechanical ventilation has major financial consequences. Additionally, differences in patient morbidity such as development of MODS, ARDS, and ventilator-associated pneumonia, compared with patients selected for pulmonary contusions alone, may help explain the mortality difference.[35]

Initial blood values obtained at admission in severe blunt trauma dogs have not been previously reported. Though hyperglycemia has been previously associated with increased morbidity in head trauma patients,[36] no relationship between glucose and morbidity or mortality could be documented in our blunt trauma patients. The trends toward increased age, lower admission temperature, and higher admission blood glucose in nonsurvivors, will need to be documented in a large prospective study with strict inclusion criteria to determine if these parameters are valid predictors.

The available research and data regarding human blunt trauma injury is substantial compared with the veterinary literature. Both anatomic data (injury location and description) as well as physiologic data (objective data) are used in a variety of trauma scoring systems. The use of scoring systems allows survival prediction that not only benefits grieving families but may help direct and concentrate hospital staff and resources. Additionally, scoring systems allow comparison between trauma centers for performance purposes, and allow for treatment effects in prospective studies to be adjusted for injury severity. There are many human trauma scoring systems described in the literature, with Trauma and Injury Severity Score being the most widely accepted and utilized. Though useful, these scoring systems collectively are unable to determine patient status within the first hour of presentation as some physiologic data may not yet be known, and some anatomic injuries may not yet be immediately noted during the primary patient survey. Investigation of admission blood work may help identify at-risk patients in a prehospital (or immediate presentation) setting, allowing subsequent follow-up with validated scoring systems in the next 12- to 24-hour period.

The mechanism of injury (blunt or penetrating) in humans varies depending on country, age group, and demographic differences. Adolescents have a higher incidence of vehicular trauma than adults.[25,37–40] Motor vehicle trauma, which may act as a study model for adolescent pedestrian trauma, was the cause of injury in 91.1% of our dogs. In addition to the source of trauma, physical pattern of trauma influences severity. Prehospital death is associated with a higher prevalence of head and thoracic trauma.[37,41] The incidence of thoracic trauma in our study was 72.3% and head/neck trauma was present in 34% of cases; prehospital death from blunt trauma in dogs has not yet been described. There was no association between trauma region (or combination) and outcome; however, head trauma as a clinical diagnosis was significantly associated with nonsurvival ($P < 0.008$).

For trauma patients who do survive to admission, one of the major treatment goals is restoration of effective circulating volume and subsequent improved oxygen delivery. Despite their limitations, lactate and BE are still utilized as a point of care monitoring param-

eter, and used to guide resuscitation efforts. Information regarding prognosis obtained from serial lactate determination is possible. Comparison of a venous lactate level of ≥ 2 mmol/L at admission to a standard triage protocol at a Level 1 trauma center showed a significant improvement in sensitivity and specificity for identification of severe blunt trauma patients. This correlation was not observed among penetrating trauma patients.[38] Similar results were observed in a smaller blunt trauma patient pool where admission lactate had good correlation to standard injury severity scores.[42] This more rapid identification of high-risk patients may allow for better resource utilization and more focused resuscitation efforts during the first hour of presentation. Standard scoring systems such as Trauma and Injury Severity Score may allow for similar patient severity recognition; however, they take more time and require more data than a simple venous lactate level. The use of presenting lactate level to identify at-risk blunt trauma veterinary patients is practical. In our study, the blunt trauma patients that required ICU admission had an initial median lactate level of 3.5 mmol/L. This finding is consistent with perfusion deficits and likely increased severity of injury, but failed to identify a mortality risk despite a higher lactate among nonsurvivors. Serial lactate monitoring was not reviewed retrospectively in this study and differences in time to presentation, prior treatment at a local veterinary hospital, and the relatively small number of nonsurvivors, may be responsible for the lack of statistical significance. Human trauma literature suggests delays in lactate clearance are associated with higher infection risk and mortality.[43] Lactate as a single measure may be influenced by other factors; therefore, a multifactorial scoring system may be required to most accurately score the veterinary trauma patient.

Measurement of systolic blood pressure upon arrival in the emergency department as well as at the trauma scene offers some information regarding mortality prediction in human trauma patients. Hypotension, both at the scene and in the emergency department is correlated with a worse outcome.[44] In 1 study involving 19,409 patients (84% blunt trauma), a decline in blood pressure from values obtained in the field was significantly associated with higher mortality (25.7% compared with 9.7%, respectively).[44] In our study, only admission blood pressure was evaluated, and it was not available in every case due to the retrospective nature of the study. Serial blood pressure measurements were not routinely available, making comparative observations difficult. Additionally, many patients may have received initial fluid resuscitation before an attempt at blood pressure determination. There was no detectable difference in mortality when initial blood pressure values between survivors and nonsurvivors were compared. The presence of hypotension refractory to fluid therapy as evident by vasopressor use was, however, associated with nonsurvival.

A large multicenter study documented that carbon dioxide, glucose, PT, and aPTT were most likely to be abnormal in blunt trauma patients especially if hypotensive,[45] making routine initial biochemical and hematologic testing potentially useful in veterinary trauma. Coagulation was not routinely monitored within 12 hours of presentation in this group of patients. Although there were no statistically significant differences between outcome and presence of, or degree of, prolongation in PT and aPTT, in this small group of dogs, a growing body of evidence suggests that coagulation derangements might predict outcome. Both hypercoagulability and hypocoagulability have been described in trauma patients.[13,46] Hypercoagulability may initially be a physiologic response early in trauma to assist in coagulation. Potentiation of a hypercoagulable state may promote microvascular thrombi and tissue hypoxia or more devastating thrombosis of larger vessels. Up to 85% of human blunt trauma patients exhibit a hypercoagulable state.[13] Admission coagulopathy has been associated with a higher degree of injury, and higher incidence of multiorgan dysfunction, as well as increased mortality.[46] No veterinary data currently exist documenting hypercoagulability or hypocoagulability among blunt trauma veterinary patients. Routine monitoring of coagulation parameters including PT, aPTT, D-dimers, antithrombin, and thromboelastography may be useful. Prospective studies are required to elucidate the role of dysregulated coagulation in veterinary trauma.

Hyperglycemia following major trauma likely results from the anti-insulin effects from circulating cortisol and catecholamines. Admission hyperglycemia has been associated with increased morbidity and mortality, and increased incidence of infection among trauma patients in 2 separate trauma studies of humans.[47,48] This increased risk in patients with hyperglycemia was noted in both mild (>7.5 mmol/L [135 mg/dL] glucose) and moderate (>11.1 mmol/L [200 mg/dL] glucose) hyperglycemia.[47,48] The median glucose of our canine trauma patients was 7.3 mmol/L (131 mg/dL), demonstrating that mild hyperglycemia was common. However, blood glucose did not predict mortality in our study. Future studies that include a wider range of trauma severity (mild, moderate, and severe) may help determine the ability of admission blood glucose to predict survival. Additionally, stricter inclusion criteria regarding time to presentation, exclusion of patients receiving initial care at the regular veterinarian before transfer, as well as inclusion of patients that die in the

first few hours of presentation, may help determine the true significance of admission blood glucose in canine blunt trauma.

Traumatic brain injury, evident by a low Glasgow Coma Scale, has been shown to be associated with increased mortality in both penetrating and blunt trauma.[44] Although the veterinary-modified Glasgow coma scale could not be applied to the patients in this retrospective study, nonsurvival was associated with patients with traumatic brain injury as well as a fractured cranium. The latter finding supports the recommendation for routine computed tomography or magnetic resonance imaging for head trauma patients that do not improve in the first 12 hours of presentation. Not all patients in this study had computed tomographic evaluation of the head and this may introduce some bias regarding this observation. It is now recognized that corticosteroids are contraindicated in cases of head trauma in humans.[49] The landmark placebo-controlled Corticosteroid Randomization After Significant Head Injury study documented a higher relative risk of death in patients that received corticosteroids.[49] It is unknown why 49 patients in our study received corticosteroids; however, given the overwhelming evidence of the negative effect of corticosteroids in the aforementioned human study, routine corticosteroid use should be avoided in trauma patients.

Older age and trauma type (motor vehicle accidents, pedestrian accidents, and motorcycle accidents) have been associated with a higher incidence of ARDS development.[50] In a large 4,020 patient trauma study, in which the majority of patients were victims of blunt trauma, the incidence of ARDS was 12%, and development of ARDS increased mortality from 11.9% to 20%.[50] The incidence of ARDS in our study was 3.4% and development of ARDS was associated with nonsurvival.

Organ failure and death is typically a leading cause of late phase mortality among trauma patients.[2] In a large study by a Level 1 trauma center involving 77% blunt trauma patients, single organ failure was noted in 18.7% of patients, and multiple organ failure occurred in 5.1% of patients.[51] As expected, mortality in this human blunt trauma cohort increased with each subsequent organ failure.[51] Based on strict organ failure definitions and exclusion of CNS dysfunction, the lungs were the most common organ to fail.[51] The incidence of MODS in our study population was 4.3% and all MODS patients were in the nonsurvivor group. Similar to the human study, ARDS was the most common problem, affecting 6 of 8 dogs with MODS. The CNS (6/8) followed by hematologic (5/8) were the next most common organ systems that had signs of dysfunction or failure in these severely traumatized dogs. Two dogs had 2 failing/dysfunctional organ systems, and the remaining 8 patients had more than 3 organ systems affected.

Hematochezia was observed during hospitalization and was associated with nonsurvival. Interestingly 3 of 6 nonsurvivors who developed hematochezia had $<75 \times 10^9$/L at admission and 2 of 6 had $<50 \times 10^9$/L. All patients who developed hematochezia were recumbent and 4 patients had received corticosteroids. Corticosteroid use was not significantly different between survivors and nonsurvivors. However, the small number of nonsurvivors limited the power of the analysis. Bloody diarrhea is often observed after major shock, and may be a sequel of severe shock, and a manifestation of organ dysfunction. Additionally, alterations in mucosal blood flow from use of nonsteroidal anti-inflammatories or corticosteroids may predispose the gastrointestinal tract to injury or ulceration following shock. The routine use of corticosteroids after major trauma should be avoided. The most recent human definitions of multiorgan dysfunction have excluded gastrointestinal dysfunction as it lacks objective measure and tends to be more subjective.[52] Development of hematochezia in the context of the severe blunt trauma patient is likely multifactorial, and may be attributable to the severity of shock, alterations in splanchnic blood flow, changes to primary hemostasis, or changes in microcirculation. The development of hematochezia may serve as an important prognostic indicator in dogs with blunt trauma.

Recumbency at presentation was also associated with nonsurvival. CNS trauma, severity of shock, and the presence of hypoxemia may be responsible for the recumbent nature of some of these trauma patients. Of the 73 recumbent survivors, only 11 had evidence of head trauma, and 13 had evidence of spinal cord injury indicating non-CNS factors were responsible for the recumbent nature of these trauma patients. Of the 13 survivors with spinal cord injury, 6 also had head trauma. Similarly, of the 24 recumbent nonsurvivors, 12 had evidence of head trauma and 4 had evidence of spinal cord injury, again indicating other factors promoting recumbency. Of these 4 nonsurvivors with spinal cord injury 3 had concurrent head trauma. The apparent CNS dysfunction within these recumbent trauma subgroups does not account for over 50% of patients in the nonsurvivor group who were recumbent, and 75% of the patients in the survivor group who were recumbent. It is possible that other associated sequelae of major trauma such as shock and hypoxemia are likely contributing factors. Recumbency from shock when other more obvious causes (orthopedic and neurologic injury) have been ruled out may help stratify canine trauma into a severe trauma subgroup. Prospective evaluation of recumbency at admission may help

elucidate its possible role as an indicator of injury severity.

A large body of research exists clearly demonstrating a survival benefit among women compared with men and postmenopausal women in both head trauma and also blunt trauma injury.[4,53,54] Although our study did not show any significant sex trends with respect to mortality or morbidity, evidence in human medicine suggests a protective effect of estrogen. Larger prospective studies may be necessary as spayed females outnumbered intact females by nearly a ratio of 2:1 in our study group.

Mortality in human medicine differs when prehospital and hospital mortality is compared. The rate of prehospital mortality in traumatic injury ranges from 34% to 60.3%.[2,55] However, these large epidemiological studies encompass many causes of traumatic injury. The mortality rate upon admission to hospital peaks at 48 hours and declines thereafter, with early posttraumatic mortality resulting from severe primary injury.[2,56] Subsequent late posttraumatic mortality can be attributed to multiple organ failure and infection.[2,39] Various reports from many Level 1 trauma centers report blunt trauma mortality ranges from 3% to 12.3%.[38,43,56] Our study of blunt trauma in dogs had a similar mortality of 10%. Euthanasia and the inclusion criteria (admission to ICU) are major confounding factors however. This study excluded severe blunt trauma dogs that died at home as well as in the first few hours upon presentation to the emergency service and therefore the mortality is likely higher than reported.

The retrospective nature of this study makes cause and effect relationships impossible to determine. Although bias was limited to data entry by a single researcher there was a degree of subjectivity in the interpretation of some aspects of the medical record. The data obtained is from a single urban institution and thus may introduce some institutional bias, but, given the size of our study these results are likely to mirror trauma cases in other urban regions. The clinical diagnosis of syndromes such as DIC, MODS, and ARDS were made based on the expertise of the clinician in charge of the case rather than strict uniform criteria that could be applied in a prospective study. Additionally, diagnostic testing obtained at presentation may have been performed after some degree of medical intervention (such as fluid therapy) warranting careful interpretation of study results. Furthermore, the variability in time from injury to patient presentation as well as prehospital treatment by local veterinarians introduces confounding factors. The group of dogs that die at the scene or shortly thereafter were not included in this database and important data regarding this cohort were not available. By human research standards, the study size was relatively small. Comparatively, it is a relatively large study in veterinary medicine. Inclusion of a larger study population over a longer period may help evaluate some of the underpowered associations in this study. Given how rapidly the field of veterinary emergency and critical care has grown, allowing for continued improvement in the quality and standard of care, new data obtained in the next decade may help strengthen observations made from the current retrospective study. A prospective study with inclusion of a large number of patients with a focus on admission blood glucose, lactate, and blood pressure, degree of neurologic injury, and with documentation of injuries and trauma sequelae, will help support some of the observations made in this study.

Conclusions

Canine blunt trauma patients that survive the initial insult but require intensive care have an excellent prognosis for full recovery to discharge. Possible causes of mortality among blunt trauma canine patients are development of MODS, ARDS, DIC, and infection (pneumonia). Dogs that present in lateral recumbency may be at risk for increased mortality, and in particular, the recumbent patient with hyperlactatemia and hyperglycemia may be at highest risk. Advanced imaging modalities such as CT and MRI may be useful in prognostication of head trauma patients that do not stabilize in the first 12 hours after admission. Given the similarities between human and canine blunt traumatic injury with respect to injury mechanism, and trauma pattern, the dog is an excellent model for the study of many aspects of blunt trauma injury and its sequelae. Development of an accurate scoring system from a large veterinary trauma database is feasible and may allow for injury severity correction in future research.

Footnotes

[a] NovaStat 9, Nova Biomedical, Waltham, MA.
[b] Sigmastat 3.5, Systat Software, Chicago, IL.
[c] Isotonic crystalloid solution, Normosol R, Abbott Laboratories, Abbott Park, IL.
[d] Dextran 70, Kendall McGaw Laboratories Inc, Oklahoma City, OK.
[e] Hetastarch, Dopamine, Dobutamine, Hospira Inc, Lake Forest, IL.
[f] Hypertonic saline (23%), Lyphomed Inc, Des Plaines, IL.
[g] Mannitol, ELDN Health Care Co Ltd, Mount Holly, NC.
[h] Phenylephrine, Taylor Pharmaceuticals, Decatur, IL.
[i] Epinephrine, American Regent Inc, Shirley, NY.

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