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The impact of surgical timing and intervention on outcome in traumatized dogs and cats

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Abstract

Objective – To review the relevant human and veterinary literature regarding the timing of surgical intervention for trauma patients and the impact on outcome.

Data Sources – Original research, clinical studies, and review articles with no date restrictions from both human and veterinary literature.

Human Data Synthesis – Despite extensive research into the ideal timing of surgical intervention for human trauma victims, debate is ongoing and views are still evolving. Prior to the 1970s, the standard of care consisted of delayed surgical treatment, as these patients were considered too ill to undergo surgery. Beginning in the 1970s, and continuing for nearly 2 decades, early definitive surgical treatment was recommended. The most recent evolution of human trauma management incorporates the concept of damage control surgery, which acknowledges the importance of early skeletal stabilization or laparotomy for reducing morbidity while attempting to avoid complications such as acute respiratory distress syndrome or multiple organ dysfunction syndrome.

Veterinary Data Synthesis – Despite a relatively large amount of literature available regarding veterinary trauma, no evidence exists to provide the clinician guidance as to the ideal timing of surgery for trauma patients. With the exception of diaphragmatic hernia, no studies were identified that attempted to evaluate this variable.

Conclusions – Veterinary-specific studies are needed to evaluate the impact of surgical timing on outcome following trauma. The information that can be obtained from studies in this area can improve veterinary trauma care and may be used as models for human trauma care through translational applications.

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Keywords: damage control surgery, multiple organ failure, trauma, "2-hit theory"

Abbreviations	
ALI ARDS CARS	acute lung injury acute respiratory distress syndrome compensatory anti-inflammatory response syndrome
CRP	C-reactive protein
DAMP	damage-associated molecular pattern
DCS	damage control surgery

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DH	diaphragmatic hernia
ETC	early total care
FAST	focused assessment with sonography for
	trauma
IR	ischemia reperfusion
MODS	multiple organ dysfunction syndrome
MOF	multiple organ failure
PAMP	pathogen-associated molecular pattern
PRR	pattern recognition receptor
SIRS	systemic inflammatory response syndrome
TBI	traumatic brain injury
UP	uroperitoneum

Introduction

Defining the controversy

The optimal timing of definitive surgical intervention for veterinary patients suffering trauma is currently unknown. Although an area of active investigation in the

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management of human trauma victims,¹⁻⁴ a surprisingly small amount of research exists on the topic in veterinary medicine. While interesting in its own right, the evidence from human trauma should be applied to veterinary medicine with great care. Many fundamental differences exist in the types of trauma sustained, the organization of prehospital care, and ultimate management within the hospital system, specifically trauma centers specializing in management of severely traumatized human patients.

Trauma patients requiring surgery can be categorized into 3 categories:

- 1. Patients requiring immediate surgery to prevent imminent death.
- 2. Patients with varying degrees of hemodynamic stability and with injuries that are contributing significantly to morbidity that require surgical intervention.
- 3. Patients that are hemodynamically stable and can be operated on a semielective basis as resources allow.

There is little debate about when to perform surgery on patients in categories 1 and 3. The same cannot be said for patients in category 2, who have suffered severe but not immediately life-threatening injuries such as long bone fractures, pelvic ring fractures, and severe soft tissue wounds that result from polytrauma or high velocity injury. Therefore, determining patient stability is critical and is an area of ongoing investigation in human medicine.⁵⁻⁷ Complications in patients with blunt trauma are often associated with severe tissue injury and development of an inflammatory state. This inflammatory state may not be identified through evaluation of common markers of tissue perfusion.⁸ Surgery-induced trauma contributes to inflammation and if surgery is performed during the phase of pathologically increased systemic inflammation, adverse effects may occur.8,9 The most significant complication with inappropriately timed surgery is the potential to develop multiple organ failure (MOF).

Human perspective

Delayed total care

During the 1950s and 1960s, standard of care consisted of delayed surgical treatment to be performed days after the injury occurred, as these patients were considered too ill to undergo surgery.^{2,10} In this era of delayed total care for polytrauma patients, surgery for definitive stabilization was delayed by up to 10–14 days after the injury for patients with orthopedic injuries since evidence suggested that outcomes were improved despite the seemingly contradictory evidence that early immobilization appeared to further improve outcomes by limiting additional tissue injury at the fracture site.^{2,10,11}

Early total care

Beginning in the early 1970s, studies began to appear that reported immediate fracture fixation reduced the incidence of pulmonary failure and postoperative care when compared to traditional delayed surgical management.² This led to the "early total care" (ETC) era in which early definitive surgery was performed in all patients with polytrauma.¹⁰ The goal of this early surgical intervention was reduction of additional tissue injury and therefore limitation of the proinflammatory consequences associated with the presence of infected or devitalized tissue. Although ETC led to improved outcomes for most,² a subset of patients developed severe complications up to and including death.

Damage control surgery

In the mid-to-late 1990s, the practice of ETC began to be questioned with several studies documenting acute respiratory distress syndrome (ARDS) in patients with no obvious risk of pulmonary complications undergoing ETC for orthopedic injuries.² Most descriptions of damage control surgery (DCS) place an emphasis on avoiding the "Triad of death": acidosis, hypothermia, and coagulopathy.^{3,12–16} During DCS, focus is placed on short operative times with both concurrent and subsequent resuscitation. Once hypothermia, coagulopathy, and acidosis are resolved, definitive surgical repair can be considered.¹⁷

Since its advent, DCS has been accepted as a method for reducing mortality in human victims of trauma and has been extended beyond management of trauma victims to patients with any emergent abdominal surgery.¹² In the context of the abdomen, DCS is intended to prevent exsanguination and contamination of the abdomen while providing simultaneous resuscitation with definitive management after physiologic derangements have been resolved.^{12,18} The phases of DCS are as follows:¹²

- 1. Goal-directed resuscitation.
- 2. Identification of the patient based on injury pattern.
- Abbreviated surgery to control bleeding or contamination.
- 4. Reassessment of the patient on the operating table.
- 5. Continued resuscitation in the ICU.
- 6. Definitive surgical repair.

Veterinary perspective

Aside from general recommendations to proceed with life-saving surgery immediately, there is little veterinary literature regarding the timing of surgical intervention beyond the recommendation that resuscitation and stabilization occur prior to anesthesia.¹⁹ Until recently,

there has been limited success in exploring veterinary trauma patients on a systematic scale. Over the last several years, a concerted effort has been made with the establishment of provisional veterinary trauma centers and an online database for tracking trauma patients (Veterinary Committee on Trauma).^{20,21} The information that is being gathered currently may shed some light on this contentious issue.

Several large retrospective studies and 1 large prospective multi-institutional study have described the epidemiology of veterinary trauma, and have evaluated the ultimate outcome of these patients.^{22–25} Despite the large data set available in these studies, no evaluation of the timing of surgical intervention or the criteria used to evaluate surgical suitability has been described. While not extensively researched by the veterinary community, it is possible that veterinary patients follow a bimodal mortality curve similar to human trauma patients. In this model, early deaths are attributable to initial trauma load and late deaths are attributable to secondary complications (ie, sepsis) or secondary trauma load (ie, surgical trauma).

Human trauma management as a model for veterinary trauma management

Two major questions must be answered before applying human trauma management techniques to veterinary trauma patients, the first of which is: which human victims most closely resemble veterinary trauma patients? It has been noted that discovery of the victim and access to appropriate care significantly impact the outcome of human trauma patients in rural environments where trauma deaths are much more common than urban environments.²⁶ Veterinary patients resemble these human trauma patients for several reasons including the predominance of blunt injury rather than penetrating injury,^{24,25} delays in locating or identifying victims, and lack of a coordinated transportation and triage system that ensures severely traumatized patients preferentially receive care from facilities that are equipped and experienced to handle them.²⁶

The second question that must be answered is: Can techniques from human trauma patients be extrapolated successfully to veterinary patients and still be financially viable? If so, which ones? Similarities exist between human and canine trauma with respect to demographics, patterns and mechanisms of trauma, frequency of polytrauma, development of MOF, and predictive capabilities of injury scores.²⁷ For these reasons, extrapolating some human guidelines to veterinary practice may be appropriate. In fact, dogs have recently been proposed as a model for naturally occurring trauma in people for use in translational medicine.²⁷ Although there are

differences in capabilities and resources between human and veterinary trauma management, certain interventions, such as the timing of surgery following polytrauma, could be compared with some degree of confidence.

Pathophysiology of Tissue Injury

The immune recognition of tissue damage is necessary to initiate healing. This recognition occurs through innate immunity, which is an evolutionarily conserved system important to organism survival and the first line of defense against nonself antigens. The innate immune system recognizes insults from numerous microbial invaders or tissue damage from a traumatic insult.^{28–30} Despite a local focus, inflammation induced by trauma may result in systemic inflammation virtually indistinguishable from an infectious insult.³¹

The 2-hit theory

Following a traumatic episode, 2 main determinants dictate the complex cascade of the host defense response. The first and primary driver of inflammation is tissue damage directly resulting from trauma; the second is sequelae of the inflammatory response.³² Primary trauma force and impact severity determine the scope and significance of solid organ damage, soft tissue injury, and fractures through actual mechanical disruption of traumatized tissue and are referred to as the "first hit."³³ This first hit in turn dictates the severity of the secondary response that can be considered as endogenous or exogenous varieties of so-called "second hits."

Endogenous second hits are direct complications of the initial injury and include hypoxia from respiratory distress, hypovolemia, or hypoperfusion; metabolic acidosis; ischemia and reperfusion injuries; and wound necrosis. One of the postulated major contributing factors to late trauma deaths is ischemia reperfusion (IR) injury secondary to shock and subsequent reperfusion.³³

Exogenous second hits generally result from attempts to treat the underlying disease. Hypotensive episodes that may occur during anesthesia can be a significant contributor of "second hit" injury. The longer an anesthetic episode lasts, the greater the chance for a hypotensive event and further IR injury.³² If definitive repair is performed, the surgical procedures will necessarily be longer than procedures in which definitive care is not attempted. Attempts at surgical repair of the primary injury result in additional tissue trauma with possible unintended consequences such as hypothermia and blood loss exacerbating the impact of the new trauma load. Secondary infections are a significant problem and include hospital-acquired pneumonia, IV catheter site infections, or urinary catheter-related infections that may be multidrug resistant. Other common exogenous second hits include drugs, administered blood, or plasma transfusions, IV fluid administration, and enteral or parenteral nutrition.³⁴

Primary solid organ injuries can vary from contusions to lacerations. Intra-abdominal organ damage is one of the most commonly recognized clinical problems associated with blunt trauma.³⁵ Hemorrhage associated with fractures or avulsions of the liver, spleen, kidneys, and large blood vessels is a major source of pathology of traumatic injuries. Blunt and crushing injuries to the intra-abdominal organs can be particularly severe due to the amount of mechanical tissue injury that occurs at the time of trauma.³⁵ Thoracic injuries such as rib fractures, pulmonary contusions, traumatic pneumothorax, and myocardial contusions are often complicated by associated shock and hypoxia. Systemic inflammation may worsen organ dysfunction by decreasing inotropy, or inducing acute lung injury or ARDS.³²

Fractures of long bones and injuries of the extremities carry with them a large amount of tissue injury.³² Additionally, extremities are sacrificed during shock as blood flow is preferentially diverted to the core and vital organs, contributing to peripheral tissue injury. This may exacerbate crushing injuries, shearing injuries, and fractures of the extremities, increasing the likelihood of IR injury and secondary infections.³⁶ Humeral, femoral, and pelvic fractures can result in significant blood loss, which can contribute significantly to duration and severity of shock. While any form of trauma can induce inflammation, the highest incidence for development of posttraumatic systemic inflammatory response syndrome (SIRS) in people follows traumatic brain injury.³⁷ In veterinary medicine, blunt torso trauma causing thoracic and abdominal trauma is more likely to be the trigger.

The innate immune system

The innate immune system is a highly conserved immune system that does not rely on pre-exposure of antigens to elicit a response. As integral parts of the innate immune system, pattern recognition receptors (PRRs) identify molecular patterns that indicate tissue damage or microbial invasion. Innate immunity and PRRs allow the host to be protected from novel microbes, to stop further tissue damage and blood loss and to begin healing.

When trauma occurs, direct tissue damage (exogenous trauma load) leads to the expression of damage (or danger)-associated molecular patterns (DAMPs) that are then recognized by PRRs.^{38,39} DAMPs are molecules of host origin that are only expressed when tissue damage is present.^{39–45} An identical cellular response is elicited when the stimulating molecular pattern is pathogen-associated. Importantly, innate immunity participates in both noninfectious and infectious systemic inflammation.

It is well recognized that tissue damage and cell death cause inflammation.³¹ DAMP expression is both an active process that occurs when cellular activation or death occurs (eg, the S100A8/S100A9 complex has been shown to be secreted from activated neutrophils), and a passive process (injection of dead cells creates an inflammatory response) that occurs when cell death leads to liberation of intracellular molecules.^{46–48} This infers that cell death passively creates inflammation as DAMPs leak out of the cell cytosol and nucleus.

Pattern recognition receptors

PRRs are present on a large number of immune and nonimmune cell types and are constantly monitoring for pathology. Importantly, the patterns recognized are present only in disease states. PRRs are a relatively small set of receptors capable of recognizing a wide range of pathogens such as viruses, bacteria, and fungi. Cell types that participate in immune function, including macrophages, monocytes, dendritic cells, neutrophils, and epithelial cells, all express PRRs.⁴⁹

Numerous PRRs have been identified with remarkable homology across species and groups of organisms. For instance, the Nod-like receptor gene is very similar to some disease resistance genes in plants.⁴⁸ Additionally, toll-like receptors are similar in shape to toll receptors found in fruit flies. Because this system is so evolutionary, its importance is obvious for the survival of not only individuals but also the species.⁵⁰

The most studied and best known PRRs are the toll-like receptors, but many others, including NOD-like receptors, C-type lectin receptors, RIG receptors, and the RAGE receptors have been defined and are important for the coordinated function of the innate immune system. It is important to note the high degree of redundancy in the system as numerous molecules stimulating different receptors yield similar results. Extensive tissue damage as seen in trauma is one mechanism by which PRRs induce the innate immune response.⁴⁹

As a result of PRR activation, a coordinated response by the immune system recruits cells into the fight against infection and damaged tissue. This recruitment, the actions of the recruited cells, their associated killing mechanisms, and soluble factors that augment the inflammatory process all work together to either kill the invader or begin healing.

Inappropriate inflammation

Hyperinflammatory states create and exaggerate organ damage and dysfunction during systemic inflammation, regardless of the nature of the inciting event (ie, infectious or traumatic). A classic example of organ dysfunction secondary to trauma is ARDS, where increased pulmonary capillary permeability due to the presence of inflammatory mediators leads to the accumulation of pulmonary edema even if the origin of inflammation is at a site distant from the lung. Trauma-induced ARDS has been recognized for decades and became a significant problem as first aid and emergency medical care improved and patients survived their initial injuries. Hyperinflammation alone does not, however, explain all of the recognized clinical manifestations of trauma-induced immune dysfunction, and a so-called compensatory anti-inflammatory response syndrome (CARS) has been recognized in some human trauma victims as well as patients with sepsis.³⁴ Development of CARS can lead to secondary infection of either the primary site of tissue injury or distant organs such as the lungs (ie, pneumonia).

Cross-talk of coagulation and inflammation

The necessary activation of the coagulation system following trauma is obvious to prevent or limit hemorrhage. Unfortunately, activation of the coagulation system is inherently proinflammatory and as the systemic inflammatory response begins to impair immunity, coagulation abnormalities often occur simultaneously.⁵¹ This coactivation of both the innate immune system and coagulation system occurs when pathogen-associated molecular patterns (PAMPs), such as LPS, stimulate the activation of factor XII and generate the expression of tissue factor on cell surfaces.⁵¹ Additionally, proinflammatory cytokines decrease the expression of endogenous anticoagulants on the endothelial surface. This leads to a vicious cycle of inflammation and coagulation that may ultimately result in MOF and death.^{52,53}

In addition to their effects on coagulation, proinflammatory cytokines, PAMPs, and DAMPs activate the compliment cascade as part of the innate immune system response to cellular damage or apoptotic cell death.⁵⁴

Acute phase response

An acute phase response is a systemic response intended to initiate tissue-protective and antimicrobial processes.³² The acute phase response participates in trauma-induced systemic inflammation. It is triggered by inflammatory cells through the release of proinflammatory cytokines and is accomplished by the liver. Production of positive acute phase proteins, such as C-reactive proteins, fibrinogen, and prothrombin is increased. Negative acute phase proteins are proteins whose production is decreased in response to inflammation and include albumin, protein C, protein S, and antitrombin.^{32,55} Each of the acute phase proteins, whether positive or negative, has some systemic effect. Generally, however, the acute phase response is considered proinflammatory and is intended to fight infection or manage tissue trauma.

Leukocyte recruitment

The movement of WBC into organs is key in development of systemic inflammation.^{56,57} Many of the aforementioned proinflammatory mediators induce rolling, adhesion, activation, and migration of neutrophils from the circulation into the tissues through the upregulation of adhesion molecules.⁵⁸ Once within the tissues, these cells move toward the site of inflammation or tissue damage by following a trail of chemoattractants.

After injury, reactive oxygen species are produced as a result of IR injury and are a potent attractor for neutrophil migration.^{33,56} This can be an important pathological turning point where the inflammation that is necessary for tissue debridement and healing could worsen tissue trauma if not titrated appropriately.^{33,59} As neutrophils move through the endothelium, tight junctions between endothelial cells are disrupted. Soluble mediators, given off by neutrophils, also influence endothelial permeability and function. These soluble mediators are not released by the neutrophils in circulation, but when they are activated following migration.^{60,61}

Importance of inflammation and surgical timing

Extrapolating from human data, it appears that the most inopportune time to operate a patient with polytrauma is in the 2–4 days postinjury timeframe.² It is during this period that SIRS and immunologic changes are sustained and the immune system is primed to respond to any additional trauma load. In a review of 4,314 human polytrauma patients undergoing initial DCS, it was determined that upon definitive fixation an operative duration of greater than 3 hours was related with development of MOF.⁶² A further relationship to the timing of the definitive surgery was also identified.⁶² Pape et al found that patients operated between days 2 and 4 were more likely to develop MOF than patients operated between days 6 and 8.62 It has been further demonstrated that the inflammatory response was greater in patients who received definitive orthopedic surgery at days 1-4 when compared to patients operated on days 6-8.63 Given this evidence, it appears that a certain delay between initial DCS and definitive care improves the likelihood of a good outcome.²

Current human trauma management strategies appear to be rethinking the long held recommendation for early exploratory laparotomy for all trauma patients; there is growing evidence that many blunt and penetrating trauma patients can be managed nonsurgically.^{4-6,18,26} The ideal time to perform definitive surgical management for trauma is likely highly patient-dependent. Every effort should be made to find the balance between allowing for preoperative patient resuscitation and preventing complications associated with delayed surgical intervention (ie, sepsis, SIRS, multiple organ dysfunction syndrome [MODS]), which contributes to morbidity and possibly mortality. Identifying the best time for initiating definitive surgical intervention in veterinary patients therefore is of particular importance. It is possible that the best time for intervention is different for animals suffering primarily orthopedic injuries than for animals suffering primarily soft tissue injuries; similarly, the time for intervention may also be different for animals suffering high velocity injuries versus low velocity or crushing injuries.

Specific Traumatic Disease Categories

Historically, determining which patients are capable of withstanding surgical intervention has focused on evaluation of specific parameters. Indications for consideration of DCS have been proposed and are currently being used in clinical practice in human trauma patients.¹⁷ Over the course of 30 years, many variables have been evaluated as potentially useful for determining surgical suitability.^{7,8} While many parameters have been found to aid in determining surgical suitability, the evidence is conflicting. One recent example of these efforts evaluated 10 specific perioperative parameters to determine which patients were at risk to develop MOF following definitive surgical correction of orthopedic injuries in noncritically injured people with polytrauma and found no relationship.⁸

Blunt abdominal trauma

Many injuries can occur after blunt trauma including diaphragmatic hernia (DH), hemoperitoneum, and appendicular or axial fractures.^{22,25,64-78} The forces created during these injuries can cause devastating injuries and death, with an overall mortality rate of 10%-12% in canine patients.^{22,79} The varying physiologic derangements that can occur during blunt trauma make it difficult to discuss as a single entity but prognostic variables and recommendations for initial diagnostics do exist. Common diagnostic findings include peritoneal effusion, pneumoperitoneum from a ruptured viscus, body wall hernias, and orthopedic injuries. Currently in veterinary medicine, any diagnosis of pneumoperitoneum is considered cause for immediate surgical exploration, which contrasts with human trauma management, where 15% of cases can be managed nonsurgically.^{80,81} Unfortunately, human and

veterinary studies show physical exam findings and blood work results to be unreliable and insensitive for evaluating the severity of abdominal trauma.^{80–82}

No recommendations exist about the best timing of exploratory laparotomy following blunt abdominal trauma in veterinary medicine but diagnostic tests that may impact the decision of surgical intervention have been evaluated.^{79,80,83-86} In people suffering blunt abdominal trauma, focused assessment with sonography for trauma (FAST) is preferred compared to penetrating trauma, when computed tomography is the imaging modality of choice.⁸⁰ FAST scans are usually performed within 10 minutes of presentation in human trauma centers, and are accurate for detection of free fluid with a specificity of 86%-100% and a sensitivity of 99%–100%.⁸³ The use of FAST scans has been reported in veterinary medicine and can be performed by veterinarians without extensive ultrasound experience. Boysen et al reported the average time to perform this test was 6 minutes (range 2-15 minutes), with free abdominal fluid found in 45% of patients.⁷⁹ A scoring system developed from 101 dogs with vehicular trauma concluded that abdominal FAST (AFAST) was more reliable for detection of intra-abdominal injury than traditional radiography.⁸⁵ The value in preoperative AFAST scoring lies in its ability to identify major intra-abdominal injury, and thereby guide surgical decision making.

The most common etiology of blunt trauma in dogs are motor vehicle accidents, accounting for up to 90% of cases.²² One large, retrospective study combined all blunt trauma cases, analyzing data from 200 dogs and found 50% of these patients required some type of surgical intervention, with 8% requiring multiple surgeries.²² Polytrauma was seen in 72% of these cases, illustrating how difficult it is to characterize these patients into only 1 trauma subtype (eg, open fracture vs. hemoperitoneum). The most common surgical procedures were orthopedic (63.5%) followed by soft tissue (36.5%). Hemoperitoneum was present in 23% of cases with only 5% of those dogs undergoing emergency surgery. Hernias were present in 5% of cases and all required surgical intervention at some point during hospitalization. The mean number of days from admission to surgery in this study was 2.2 days (\pm 1.7 days). Unfortunately, the timing of surgery in relation to the time of trauma or admission was not analyzed for outcome. There were no significant associations with mortality and the need for surgical intervention, length of surgery, length of anesthesia, or postoperative temperature. Poor prognostic indicators included head trauma, cranium fractures; recumbency on admission, hematochezia, suspicion ARDS, disseminated intravascular coagulation, of MODS, development of pneumonia, positive-pressure ventilation, and vasopressor use.²²

Diaphragmatic hernia

In the veterinary literature, only DH has been evaluated for the effect of surgical timing on outcome. DH is created when an opening in the diaphragm allows any of the abdominal organs access to the thoracic cavity. Results of a PubMed search reveal that over the last 13 years, 22 articles pertaining to DH in dogs and cats have been published. Of these, 3 were specifically related to chronic DH, 6 are primarily imaging studies, and 3 are related to specific treatment modalities.87-98 Of the remaining 10 reports, 7 are case reports, 1 is a case series with 3 cats, and 2 are retrospective studies.^{69-72,98-105} These 2 studies include data from 126 cases (63 cats and 63 dogs).72,104 Forty-five cats and 45 dogs were described as suffering from acute trauma leading to DH. The other 18 animals of each species were either missing for some period of time, had an injury that was never witnessed but assumed, or were adopted or purchased with evidence of DH at the time.

The overall mortality rate for acute DH ranges from 6.3% to 20%.72,104 Historically, associations were presented that suggested mortality rate was higher for traumatic DH repaired within 24 hours of injury or more than 1 year after injury. Some authors have stated that DH is only a surgical emergency if evidence of gastrothorax is present; otherwise surgery should be delayed 24-48 hours to allow for sufficient stabilization.⁷³ Schmiedt et al reported a feline mortality rate of 17.6%, with younger cats more likely to survive.⁷² The mean duration of DH in this study was 17.6 days, with DH known to be present for 1-7 days in half of the cats. Despite small numbers, no significant differences among groups were found, suggesting timing of surgical repair is not related to outcome. Factors that were related to outcome included age, respiratory rate on admission, and the presence of multiple concurrent injuries.

The larger of these studies specifically evaluated surgical timing with regard to survival.¹⁰⁴ The majority (92.6%) of cases of acute DH were taken to surgery within 24 hours of admission to the hospital, with a median time of admission from trauma of 18 hours (range 4 hours to 2.3 years) for cats and 48 hours (range 1 hour to 10 years) for dogs, and 93.7% of those cases survived to discharge. Approximately half (42.6%) of the patients suffering from acute DH were taken to surgery within 24 hours of the actual trauma event, and 89.7% of those patients survived to discharge. For cats, the median time from admission to surgery was 3 hours (range 1 hour to 216 hours) but 79% had surgery within 12 hours of admission and 93% had surgery within 24 hours. For dogs, the median time from admission to surgery was 3 hours (range 1 hour to 14 days), with 90.5% having surgery before 24 hours. Even though the data from acute cases (68) were analyzed separately, a formal test

for the effect on early versus late intervention between cats and dogs failed to show significance so the authors combined the species for statistical analysis. The odds ratio for perioperative survival was not significantly associated with time from trauma to admission, admission to surgery, or trauma to surgery. These findings suggest that surgical intervention within 24 hours of DH may not have an adverse effect on survival contradicting older recommendations.¹⁰⁴ Importantly, most of the patients in this report were stabilized at a primary care practice before being referred to the specialty center.

The literature regarding DH appears to support the practice of appropriately stabilizing each patient before surgery and once this is accomplished, regardless of the length of time from trauma, surgery can be performed without increasing risk of mortality. Paradoxically, it may be necessary to perform surgery to stabilize some patients with acute traumatic DH.

Uroperitoneum

Uroperitoneum (UP) results from rupture of the urinary tract with subsequent accumulation of urine in the peritoneal or retroperitoneal cavity. This injury can occur from blunt or penetrating trauma and can be difficult to discover as up to 69% of animals can urinate or have a palpable urinary bladder during exam.^{68,105,106} Most reports of UP are retrospective studies or case series and very few discuss surgical timing. The most common cause for UP is blunt trauma (85%), often associated with motor vehicle accidents.¹⁰⁶

In people, the decision between surgery and conservative treatment is based on the site and extent of the lesion as well as any concomitant injuries. Surgery in animals is generally recommended for UP but reports of conservative therapy with catheterization exist.¹⁰⁷ Conservative management has been reported in veterinary medicine with bladder rupture or partial ure-thral tears but length of hospitalization is extensive (2–4 weeks). Most authors recommend urinary diversion (cystostomy tubes/urinary catheters/intra-abdominal drainage) before definitive surgery in uremic patients, as uremia has been associated with a high risk of anesthetic complications.^{106–109}

In a retrospective study on 26 cats, no correlation between time of injury, time of presentation, and outcome could be established.¹⁰⁶ Surgical timing was not evaluated. Mortality rates of 38.5% and up to 42% are reported in cats and dogs, respectively, and mortality is associated with concurrent injuries in a large number of cases.¹⁰⁶ Delay in diagnosis and treatment has been associated with an increase in mortality in dogs and people.^{106,107} Within the remaining veterinary literature, no recommendations of timing for surgery are found. Ureteral rupture is an indication for immediate repair in people, contingent on patient stability as ureteral structures are extremely fragile and the longer they remain damaged, the more difficult they are to repair.¹⁰⁷ In 1 human study of 164 bladder ruptures in trauma patients, associated injuries were the most common cause of death.¹⁰⁷ With regard to UP management in people, the overwhelming majority of practitioners appear to consider that stabilization of many patients includes surgery and that some patients will only be definitively stabilized after a surgical procedure has been performed.

Hemoperitoneum

Hemoperitoneum is defined as an accumulation of blood within the abdominal cavity.¹¹⁰ Despite a large amount of literature about hemoperitoneum, very little has been published related to traumatic causes in veterinary medicine. Blunt or penetrating injuries associated with motor vehicle accidents are the number 1 etiology when traumatic hemoperitoneum does occur.64,67,74,75,79,111,112 Four articles pertaining directly to traumatic hemoperitoneum were found, including 2 case reports and 2 retrospective studies, the largest of which describes 83 dogs with 6 cases due to trauma.^{67,74,75,112} In 1 review, arresting ongoing hemorrhage is fourth on a list of initial stabilization goals and can in some cases be managed without the need for surgery (abdominal counterpressure techniques). If a patient cannot be stabilized with volume expansion, blood products, and counterpressure, then emergency surgery is warranted.¹¹¹ The timing of surgical intervention with regard to traumatic hemoabdomen has not been analyzed, perhaps because the need for surgical intervention appears to be rare.^{22,74}

Mongil et al reported 28 patients (27 dogs and 1 cat) who suffered blunt trauma with hemoperitoneum.⁷⁵ Overall survival in this study was 57%. Of the nonsurvivors, half died despite interventions and the others were euthanized. Only 6 of the 28 patients (21%) were treated surgically compared to 16 treated medically, with survival rates of 67% and 75%, respectively. Twentyseven of 28 cases were presented to the hospital within 24 hours of trauma; however, timing from trauma or admission to surgery was not evaluated. Interestingly, the decision to perform an abdominal exploratory was evaluated and was not found to correlate with survival. Body weight was the only variable associated with survival in this study.⁷⁵

Lux et al reported 6 of 83 dogs with hemoperitoneum occurring secondary to trauma, all of which were managed operatively.⁷⁴ The overall mortality rate in this study of 83 dogs was 15.7%, with at least 1 of the 13 fatalities being a trauma patient. Every patient in this study underwent FAST scan or complete abdominal

ultrasound and every patient received a blood transfusion. A retrospective study reporting massive transfusion in 15 dogs included 3 dogs that had traumatic hemoperitoneum.¹¹³ Of the 12 dogs in that study treated surgically, 2 were trauma patients. The timing of surgical intervention or the rationale for electing surgical management was not discussed. Two case reports of traumatic hemoperitoneum report surgery days after the traumatic event. Surgery was performed in these cases not due to life-threatening hemorrhage but for management of concurrent injuries.^{67,112}

Very little information exists about hemoperitoneum in cats, leading to even lower-quality evidence to build upon for recommendations of surgical timing. This may be due to their smaller size and higher likelihood of suffering fatal injuries at the time of trauma but potential species differences should not be overlooked.

Surgical readiness of the patient and the facility is vitally important in cases of hemoperitoneum, especially in trauma patients. Specific staffing needs for a decompensating patient (eg, anesthetist, primary surgeon, assistant) must be considered and met before attempting surgical resolution of a hemoperitoneum. Blood products should be available in the operating suite and operative times kept short. This may include rapid clip and prep if catastrophic hemorrhage is occurring. All emergency and pain-related drug dosages should be calculated before induction and the need for ventilation, blood pressure support, and intensive anesthetic monitoring assumed.¹¹¹

Penetrating trauma

Penetrating trauma has been frequently reported in veterinary medicine, with multiple retrospective studies examining oropharyngeal trauma, gunshot wounds, and bite wounds with survival rates ranging from 38% to 100%.^{76,114–126} Examining reports on penetrating trauma is made difficult by the vast differences in the type and severity of trauma created by different mechanisms (eg, gunshot wounds vs. bite wounds) and specific recommendations for surgical timing should be considered separately when possible. Risselda et al reported a survival rate of 87.5% in 16 cases of penetrating wounds from multiple causes,¹²³ while Baker et al reported a much lower survival of 38% in actively serving military dogs with gunshot wounds.¹²⁴ Neither provided information regarding the timing or any apparent effect of timing of surgical intervention. One retrospective study of major abdominal evisceration injuries did report time from admission to surgery but did not include discussion as to whether these variables affected outcome.⁶⁴

Two retrospective studies of oropharyngeal trauma fail to report impact of surgical timing on outcome.^{121,122}

Nonetheless, both recommend early exploration due to the high complication rate associated with chronic cases. Abdominal impalement injuries have been described and may warrant special consideration for emergency laparotomy as extent of injuries, particularly with sticks, may not be immediately evident due to the potential for these foreign bodies to migrate.^{118,120} Pratschke and Kirby reported that all cats suffering impalement following falls from height underwent surgery on the day of trauma.¹¹⁸ The likelihood of polytrauma in these cases should lead to careful consideration prior to operating these patients.^{118,123}

In the largest retrospective on gunshot wound injuries, specifically in military working dogs, evacuation times and initial stabilization were discussed but time from injury or evaluation by a veterinarian to surgery was not analyzed.¹²⁴ A case report outlining a cat with multiple gunshot wounds, bile peritonitis, and bilothorax illustrated surgical success 5 days after the original injury.¹¹⁶ Human patients with gunshot wounds to the abdomen are often managed operatively but can be successfully managed nonoperatively. Recent work suggests that an initial nonoperative approach to patients meeting specific criteria leads to fewer nontherapeutic surgeries without affecting outcome.^{6,127}

Three large retrospective studies evaluating penetrating trauma, most commonly caused by bite wounds, reported survival rates of 73%-83%.76,125,126 The patients in these reports suffered a variety of injuries including body wall hernias, soft tissue wounds, flail chest, pulmonary contusions, and rib fractures. The degree of tissue damage, especially with bite wounds, is commonly underappreciated by visual exam and traditional radiography.¹²³ Although surgical timing was not discussed or analyzed in any of these studies, recommendations were made to take patients to surgery based on finding injuries to the thorax or abdomen because of the high potential for intracavitary trauma.¹²³ All authors emphasized the importance of a thorough physical exam and diagnostic work up and recommended early exploratory procedures to rule out more serious internal injury. In some cases, body wall hernias were not recognized for days after initial exam. These patients did not appear to suffer negative consequences from this delay although the number of cases is small.⁷⁶

With regard to thoracic trauma, some authors recommend exploratory surgery for any patient with a flail chest, rib fractures, lung contusion, or pneumothorax but the optimal timing of surgery for these potentially unstable patients is unknown.¹²⁵ Anecdotally, many clinicians treat these injuries conservatively with great success, and 1 report showed no significant difference in outcome between cases of flail chest stabilized surgically versus those that were not.¹²⁶ Penetrating trauma into the cranium arising from bite wounds has also been reported.^{115,117} In these studies, diagnosis of intracranial disease and therefore surgery was delayed and consequently no recommendations were made regarding timing of surgery.

Spinal trauma

Compared to other traumatic etiologies, little clinical work has been done regarding spinal trauma in veterinary medicine.^{65,78,128,129} This may be due to actual or perceived poor outcomes causing many owners to opt for euthanasia in this subset of patients. Most reports detail acute spinal cord injuries caused by motor vehicle accidents or traumatic bite wounds.^{78,128–130} A thorough review on the physiologic effects and mechanisms underlying acute spinal cord injury only superficially explored the topic of surgery and focused on initial stabilization, examination findings, and postsurgical care.¹³⁰

Two large retrospective studies on spinal fractures in dogs and cats list motor vehicle accidents as the most common cause. The most common region affected in dogs was the T3-L3 region (20%–55%) with up to 45% of patients having concomitant injuries.78,129 Bruce et al reported approximately one-third of patients were euthanized without treatment, one-third were treated conservatively, and one-third underwent surgery.⁷⁸ Surgical patients regained more neurologic function but required longer hospitalization. Bali et al reported poor outcomes in 61% of cats and 56% of dogs and although time from injury to presentation was evaluated between the species, it was not analyzed as it pertained to eventual outcome.¹²⁹ The necessity of surgical intervention was determined by: spinal instability, compression of the spinal cord, continued pain past 48-72 hours of medical treatment, and deterioration in neurologic status. Neither study discussed the timing of surgery, instead focusing on initial exam findings as they related to outcome. In human medicine, few spinal injuries are considered surgical emergencies but in these specific cases, early spinal cord decompression has been shown to improve neurologic recovery.⁷⁸ Nonsurgical traction devices allow for stabilization of the patient prior to definitive correction and are often the treatment of choice in people. Unfortunately, most of the literature describing stabilization techniques for spinal fractures does not comment on surgical timing.^{65,131,132}

Open fractures

Traumatic long bone and pelvic fractures from motor vehicle accidents are one of the most commonly encountered problems in blunt trauma patients.^{77,84,133–137} Despite a large amount of research in this area, the optimal timing of definitive surgical stabilization is

controversial. In veterinary medicine, no guidelines with respect to fracture management in traumatized patients have been proposed, so clinicians are left to rely on their experience to determine when the patient is sufficiently stable for a potentially long anesthetic procedure.

In a thorough review of complex and open fractures in cats, Corr detailed an approach to diagnostic investigation, outlined fracture management options, and described postoperative care measures and reported on outcome.77 This review maintains the commonly held belief that open fractures are an orthopedic emergency requiring immediate action in order to increase the chances of a good postoperative outcome.^{138,139} This report stated that 59%-72% of animals with long bone fractures will have concurrent injuries and that identification and management of these potentially life-threatening problems should be the initial focus.⁷⁷ Emergency treatments that must be performed on open fractures include: providing sedation/anesthesia for patient comfort, removal of gross debris, clipping, flushing of wounds, culture procurement, administration of antimicrobials, and application of sterile dressings on open wounds.¹³⁹

Unfortunately, no specific guidelines as to when these fractures should be definitively repaired were found in an exhaustive literature review. In one author's opinion,⁷⁷ distal tibial fractures and tarsocrural (sub)luxations should be stabilized as quickly as possible due to the high potential for vascular compromise to the foot associated with these injuries. A review of radiographic findings in 100 feline trauma patients reported surgical intervention in 51% of cases but did not analyze whether the need for surgery or the timing of the procedures related to outcome.⁸⁴ Overall survival in this paper was 73%, and was negatively correlated with age, free abdominal air, soft tissue injuries, and abdominal trauma.⁸⁴ Available retrospective reviews on open fractures do not outline the timing of surgery but rather focus on previous recommendations of staged wound management and discuss the lengthy recovery seen in most patients.^{140,141} Species differences may exist in terms of outcomes between dogs and cats.¹⁴¹

Conclusion

A DCS approach appears reasonable for veterinary patients, with attention being focused on eliminating immediately life-threatening conditions while making every effort to minimize operative time and additional tissue trauma. The application of DCS principles to veterinary patients is an area that does not appear to have been explored based on the results of our literature search. While there may be a subset of patients that may benefit from a DCS approach, this is far from proven, and if attempted, this limitation should be kept in mind. Whether DCS has been performed or not, the optimal time to operate a trauma patient that required aggressive hemodynamic stabilization is not known.

No published reports with statistical data specific to the timing of surgery in trauma patients, with the exception of DH, exist in veterinary medicine in contrast to the human trauma literature in which the topic is widely researched. Guidelines in people have been published and are still an area of active investigation.¹⁴² While veterinary recommendations exist, they are based on anecdotal or personal experience. The lack of current veterinary guidelines or consensus on the appropriate timing of surgical intervention following polytrauma suggests that this should be further investigated. The ongoing Veterinary Committee on Trauma (VetCOT) and Veterinary Trauma Center initiatives¹⁴³ instigated by the American College of Veterinary Emergency and Critical Care may provide an avenue to begin this process through the use of an online database allowing evaluation of very large numbers of trauma cases in a multi-institutional manner.

References

- Nahm NJ, Vallier HA. Timing of definitive treatment of femoral shaft fractures in patients with multiple injuries: a systematic review of randomized and nonrandomized trials. J Trauma 2012; 73(5):1046–1063.
- Pape HC, Giannoudia P, Krettek C. The timing of fracture treatment in polytrauma patients: relevance of damage control orthopedic surgery. Am J Surg 2002; 183:622–629.
- 3. Cirocchi R, Montedori A, Farinella E, et al. Damage control surgery for abdominal trauma (review). Cochrane 2013; 3:1–14.
- Olthaf DC, van Delden OM, Luitse JSK, et al. Time to intervention in patients with splenic injury in a Dutch level 1 trauma center. Injury 2014; 45:95–100.
- McKenney LK, McKenney MG, Cohn SM, et al. Hemoperitoneum score helps determine need for therapeutic laparotomy. J Trauma 2001; 50:650–656.
- Inaba K, Barmpara G, Foster A, et al. Selective nonoperative management of torso gunshot wounds: when is it safe to discharge. J Trauma 2010; 68(6):1301–1304.
- Asensio JA, McDuffie L, Petrone P, et al. Reliable variables in the exsanguinated patient which indicate damage control and predict outcome. Am J Surg 2001; 82(6):743–751.
- Dienstknecht T, Rixen D, Giannoudis P, et al. Do parameters used to clear noncritically injured polytrauma patients for extremity surgery predict complications. Clin Ortho Relat Res 2013; 471:2878– 2884.
- 9. Waydhas C, Nast-Kolb D, Trupka A, et al. Posttraumatic inflammatory response, secondary operations, and late multiple organ failure. J Trauma Inj Infect Crit Care 1996; 40(4):624–631.
- 10. Bose D, Tejwani NC. Evolving trends in the care of polytrauma patients. Injury 2006; 37:20–28.
- Smith JEM. The results of early and delayed internal fixation of fractures of the shaft of the femur. J Bone Joint Surg 1964; 46B(1); 28–31.
- 12. Weber DG, Bendinelli C, Balogh ZJ. Damage control surgery for abdominal emergencies. Br J Surg 2014; 101:e109-e118.
- Chovanes J, Cannon JQ, Nunez TC. The evolution of damage control surgery. Surg Clin N Am 2012; 92:859–875.
- Rotondo MF, Zonies DH. The damage control sequence and underlying logic. Surg Clin N Am 1997; 77(4):761–777.
- 15. Mathieu L, Bazile F, Barthelemy R, et al. Damage control orthopaedics in the context of battlefield injuries: the use of

temporary external fixation on combat trauma soldiers. Ortho Trauma Surg Res 2011; 97:852-859.

- Schreiber MA. Damage control surgery. Crit Care Clin 2004; 20:101–118.
- 17. Waibel BH, Rotondo MM. Damage control surgery: its evolution over the last 20 years. Rev Col Bras Cir 2012; 39(4):314–321.
- Higa G, Friese R, O'Keefe T, et al. Damage control laparotomy: a vital tool once overused. J Trauma 2010; 69(1):53–59.
- 19. Devey JJ. Surgical considerations in the emergent small animal patient. Vet Clin Small Anim 2013; 43:899–914.
- Hall K, deLaforcade AM. Veterinary trauma centers (editorial). J Vet Emerg Crit Care 2013; 23(4):373–375.
- 21. Hall K, Sharp C. The veterinary trauma initiative: why bother? (editorial). J Vet Emerg Crit Care 2014; 24(6):639–641.
- 22. Simpson SA, Syring R, Otto CM. Severe blunt trauma in dogs: 235 cases (1997–2003). J Vet Emerg Crit Care 2009; 19(6):588–602.
- Streeter EM, Rozanski EA, de LaForcade-Buress A, et al. Evaluation of vehicular trauma in dogs: 239 cases (January-December 2001). J Am Vet Med Assoc 2009; 235(4):405–408.
- 24. Kolata RJ, Kraut NH, Johnston DE, et al. Patterns of trauma in urban dogs and cats: a study of 1000 cases. J Am Vet Med Assoc 1974; 164(5):499–502.
- Hall KE, Holowaychuk MK, Sharp CR, et al. Multicenter prospective evaluation of dogs with trauma. J Am Vet Med Assoc 2014; 244:300–308.
- 26. Rinker CF, Sanddal ND. Rural trauma, In: Mattox KL. ed. Trauma, 7th edn. New York: McGraw Hill; 2013, pp. 140–153.
- Hall KE, Sharp CR, Adams CR, et al. A novel trauma model: naturally occurring canine trauma. Shock 2014; 41(1):25–32.
- Neher MD, Weckbach S, Flierl MA, et al. Molecular mechanisms of inflammation and tissue injury after major trauma-is complement the "bad guy"? J Biomed Sci 2011; 18:90–127, 18–90.
- Lippross Š, Klueter T, Steubesand N, et al. Multiple trauma induces serum production of host defence peptides. Injury 2012; 43(2):137– 142.
- Stahel PF, Smith WR, Moore EE. Role of biological modifiers regulating the immune response after trauma. Injury 2007; 38(12):1409–1422.
- Rock KL, Kono H. The inflammatory response to cell death. Annu Rev Pathol 2008; 3:99–126.
- 32. Keel M, Trentz O. Pathophysiology of polytrauma. Injury 2005; 36(6):691–709.
- Lenz A, Franklin GA, Cheadle WG. Systemic inflammation after trauma. Injury 2007; 38(12):1336–1345.
- 34. Smith MA, Hibino M, Falcione BA, et al. Immunosuppressive aspects of analgesics and sedatives used in mechanically ventilated patients: an underappreciated risk factor for the development of ventilator-associated pneumonia in critically ill patients. Ann Pharmacother 2014; 48(1):77–85.
- Jones EL, Stovall RT, Jones TS, et al. Intra-abdominal injury following blunt trauma becomes clinically apparent within 9 hours. J Trauma Acute Care Surg 2014; 76(4):1020–1023.
- Flohe S, Flohe SB, Schade FU, et al. Immune response of severely injured patients-influence of surgical intervention and therapeutic impact. Langenbecks Arch Surg 2007; 392(5):639–648.
- Lou M, Chen X, Wang K, et al. Increased intracranial pressure is associated with the development of acute lung injury following severe traumatic brain injury. Clin Neurol Neurosurg 2013; 115(7):904–908.
- Hirsiger S, Simmen HP, Werner CM, et al. Danger signals activating the immune response after trauma. Mediators Inflamm 2012; 1–10 (doi: 10.1155/2012/315941).
- Bianchi ME, Manfredi AA. Immunology. Dangers in and out. Science 2009; 323(5922):1683–1684.
- Yu DH, Nho DH, Song RH, et al. High-mobility group box 1 as a surrogate prognostic marker in dogs with systemic inflammatory response syndrome. J Vet Emerg Crit Care 2010; 20(3):298–302.
- Ishida A, Ohno K, Fukushima K, et al. Plasma high-mobility group box 1 (HMGB1) in dogs with various diseases: comparison with C-reactive protein. J Vet Med Sci 2011; 73(9):1127–1132.

- Karlsson I, Wernersson S, Ambrosen A, et al. Increased concentrations of C-reactive protein but not high-mobility group box 1 in dogs with naturally occurring sepsis. Vet Immunol Immunopathol 2013; 156(1–2):64–72.
- 43. Osterloh A, Geisinger F, Piedavent M, et al. Heat shock protein 60 (HSP60) stimulates neutrophil effector functions. J Leukoc Biol 2009; 86(2):423–434.
- 44. Pugin J. How tissue injury alarms the immune system and causes a systemic inflammatory response syndrome. Ann Intensive Care 2012; 2(1):27–5820, 2–27.
- 45. Manson J, Thiemermann C, Brohi K. Trauma alarmins as activators of damage-induced inflammation. Br J Surg 2012;suppl 99:12–20.
- Kerkhoff C, Klempt M, Sorg C. Novel insights into structure and function of MRP8 (S100A8) and MRP14 (S100A9). Biochim Biophys Acta 1998; 1448(2):200–211.
- Donato R. Intracellular and extracellular roles of S100 proteins. Microsc Res Tech 2003; 60(6):540–551.
- Chen CJ, Kono H, Golenbock D, et al. Identification of a key pathway required for the sterile inflammatory response triggered by dying cells. Nat Med 2007; 13(7):851–856.
- Takeuchi O, Akira S. Pattern recognition receptors and inflammation. Cell 2010; 140(6):805–820.
- 50. Takeda K, Kaisho T, Akira S. Toll-like receptors. Annu Rev Immunol 2003; 21:335–376.
- 51. Levi M, van der Poll T. Inflammation and coagulation. Crit Care Med 2010;Suppl 38:S26–S34.
- 52. Weiss. In: Feldman. ed. Schalm's Veterinary Hematology, 6th edn. Hoboken, NJ: John Wiley & Sons; 2010.
- Dewar D, Moore FA, Moore EE, et al. Postinjury multiple organ failure. Injury 2009; 40(9):912–918.
- 54. Tizard I. Veterinary Immunology. 9th ed. St. Louis: Saunders; 2013.
- Dunham CM, Damiano AM, Wiles CE, et al. Post-traumatic multiple organ dysfunction syndrome—infection is an uncommon antecedent risk factor. Injury 1995; 26(6):373–378.
- Botha AJ, Moore FA, Moore EE, et al. Postinjury neutrophil priming and activation: an early vulnerable window. Surgery 1995; 118(2):358–364; discussion 364–365.
- 57. Fujishima S, Aikawa N. Neutrophil-mediated tissue injury and its modulation. Intensive Care Med 1995; 21(3):277–285.
- Ley K, Laudanna C, Cybulsky MI, et al. Getting to the site of inflammation: the leukocyte adhesion cascade updated. Nat Rev Immunol 2007; 7(9):678–689.
- 59. Bhatia R, Dent C, Topley N, Pallister I. Neutrophil priming for elastase release in adult blunt trauma patients. J Trauma 2006; 60(3):590–596.
- Zemans RL, Colgan SP, Downey GP. Transepithelial migration of neutrophils: mechanisms and implications for acute lung injury. Am J Respir Cell Mol Biol 2009; 40(5):519–535.
- 61. Downey GP, Dong Q, Kruger J, et al. Regulation of neutrophil activation in acute lung injury. Chest 1999; Suppl 116:46S–54S.
- Pape HC, Stalp M, v Griensven M, et al. Optimal timing for secondary surgery in polytrauma patients: an evaluation of 4314 serious-injury cases (German). Chirurg 1999; 71(11):1287–1293.
- Pape HC, v Griensven M, Hildebrand FF, et al. Systemic inflammatory response after extremity or truncal fracture operations. J Trauma 2008; 65:1379–1384.
- 64. Gower SB, Weisse CW, Brown DC. Major abdominal evisceration injuries in dogs and cats: 12 cases (1998–2008). J Am Vet Med Assoc 2009; 234:1566–1572.
- Voss K, Montavon PM. Tension band stabilization of fractures and luxations of the thoracolumbar vertebrae in dogs and cats: 38 cases (1993–2002). J Am Vet Med Assoc 2004; 225:78–83.
- 66. Hamilton MH, Sissener TR, Baines SJ. Traumatic bilateral ureteric rupture in 2 dogs. J Small Anim Pract 2006; 47:737–740.
- Millward IR. Avulsion of the left renal artery following blunt abdominal trauma in a dog. J Small Anim Pract 2009; 50:38–43.
- 68. Klainbart S, Merchav R, Ohad DG. Traumatic urothorax in a dog: a case report. J Small Anim Pract 2011; 52:544–546.
- 69. Hodinott K. Traumatic diaphragmatic hernia in a 5 month old Boxer dog. Can Vet J 2013; 54:507–509.

- Litman LM. Traumatic diaphragmatic hernia in a clinically normal dog. Can Vet J 2001; 42:564–566.
- Katic N, Bartolomaeus E, Bohler A, et al. Trauamtic diaphragmatic rupture in a cat with partial kidney displacement into the thorax. J Small Anim Pract 2007; 48:705–708.
- Schmiedt CW, Tobias KM, McCrackin SMA. Traumatic diaphragmatic hernia in cats: 34 cases (1991–2001). J Am Vet Med Assoc 2003; 222:1237–1240.
- Sullivan M, Reid J. Management of 60 cases of diaphragmatic rupture. J Small Anim Pract 1990; 31:425–430.
- 74. Lux CN, Culp WT, Mayhew PD, et al. Perioperative outcome in dogs with hemoperitoneum: 83 cases (2005–2010). J Am Vet Med 2013; 242:1385–1391.
- Mongil CM, Drobatz KJ, Hendricks JC. Traumatic hemoperitoneum in 28 cases: a retrospective review. J Am Anim Hosp Assoc 1995; 31:217–222.
- 76. Shaw SP, Rozanski EA, Rush JE. Traumatic body wall herniation in 36 dogs and cats. J Am Anim Hosp Assoc 2003; 39:35–46.
- 77. Corr S. Complex and open fractures: a straightforward approach to management in the cat. J Feline Med Surg 2012; 14:55–64.
- Bruce CW, Brisson BA, Gyselink K. Spinal fracture and luxation in dogs and cats: a retrospective evaluation of 95 cases. Vet Comp Orthop Trauamtol 2008; 21:280–284.
- 79. Boysen SR, Rozanski EA, Tidwell AS, et al. Evaluation of a focused assessment with sonography for trauma protocol to detect free abdominal fluid in dogs involved in motor vehicle accidents. J Am Vet Med Assoc 2004; 225:1198–1204.
- Simmonds SL, Whelan MF, Basseches J. Non surgical pneumoperitoneum in a dog secondary to blunt force trauma to the chest. J Vet Emerg Crit Care 2011; 21:552–557.
- 81. Mularski RA, Sippel JM, Osborne ML. Pneumoperitoneum: a review of nonsurgical causes. Crit Care Med 2000; 28:2638–2644.
- Schurink GW, Bode PJ, van Luijt PA, et al. The value of physical examination in the diagnosis of patients with blunt abdominal trauma: a retrospective study. Injury 1997; 28(4):261–265.
- Dempsey SM, Ewing PJ. A review of the pathophysiology, classification and analysis of canine and feline cavitary effusions. J Am Anim Hosp Assoc 2011; 47:1–11
- Zulauf D, Kaser-Hotz B, Hassig M, et al. Radiographic examination and outcome in consecutive feline trauma patients. Vet Comp Orthop Trauamtol 2008; 21:36–40.
- 85. Lisciandro GR, Lagutchik MS, Mann KA, et al. Evaluation of an abdominal fluid scoring system determined using abdominal focused assessment with sonography for trauma in 101 dogs with motor vehicle trauma. J Vet Emerg Crit Care 2009; 19:426–437.
- Lisciandro GR. Abdominal and thoracic focused assessment with sonography for trauma, triage, and monitoring in small animals. J Vet Emerg Crit Care 2011; 21:104–122.
- 87. Minihan AC, Berg J, Evans KL. Chronic diaphragmatic hernia in 34 dogs and 16 cats. J Am Anim Hosp Assoc 2004; 40:51–63.
- Witsberger TH, Dismukes DI, Kelmer EY. Situs inversus totalis in a dog with a chronic diaphragmatic hernia. J Am Anim Hosp Assoc 2009; 45:245–248.
- Hambrook LE, Kudnig ST. Lung lobe torsion in association with a chronic diaphragmatic hernia and hemorrhagic pleural effusion in a cat. J Feline Med Surg 2012; 14:219–223.
- 90. Hyun C. Radiographic diagnosis of diaphragmatic hernia: review of 60 cases in dogs and cats. J Vet Sci 2004; 5:157–162.
- 91. Kumar A, Saini NS. Reliability of ultrasonography at the fifth intercostal space in the diagnosis of reticular diaphragmatic hernia. Vet Rec 2011; 169:391b (doi: 10.1136/vr.d4694).
- Parry A. Positive contrast peritoneography in the diagnosis of a pleuroperitoneal diaphragmatic hernia in a cat. J Feline Med Surg 2010; 12:141–143.
- Choi J, Kim H, Kim M, et al. Imaging diagnosis–positive contrast peritoneographic features of true diaphragmatic hernia. Vet Radiol Ultrasound 2009; 50:185–187.
- Lamb CR. Radiology corner: loss of the diaphragmatic line as a sign of ruptured diaphragm. Vet Radiol Ultrasound 2004; 45:305–306.
- Ricco CH, Graham L. Undiagnosed diaphragmatic hernia-the importance of preanesthetic evaluation. Can Vet J 2007; 48:615–618.

- 96. Chantawong P, Komin K, Banlunara W, et al. Diaphragmatic hernia repair using a rectus abdominis muscle flap in three dogs. Vet Comp Orthop Traumatol 2013; 26:135–139.
- Merbl Y, Kelmer E, Shipov A, et al. Resolution of persistent pneumothorax by use of blood pleurodesis in a dog after surgical correction of a diaphragmatic hernia. J Am Vet Med Assoc 2010; 237:299– 303.
- Loqman MY, Wong CM, Hair-Bejo M, et al. The use of freezedry bovine pericardium (FDBP) in diaphragmatic herniorrhaphy in dogs. Med J Malaysia 2004; 59:113–114.
- 99. Lojszczyk-Szczepaniak A, Komsta R, Debiak P. Retrosternal (Morgagni) diaphragmatic hernia. Can Vet J 2011; 52:878–883.
- Cariou MP, Shihab N, Kenny P, et al. Surgical management of an incidentally diagnosed true pleuroperitoneal hernia in a cat. J Feline Med Surg 2009; 11:873–877.
- 101. Joseph R, Kuzi S, Lavy E, et al. Transient megaoesophagus and oesophagitis following diaphragmatic rupture repair in a cat. J Feline Med Surg 2008; 10:284–290.
- Lin JL, Lee CS, Chen PW, et al. Complications during labour in a Chihuahua due to diaphragmatic hernia. Vet Rec 2007; 161:103– 104.
- 103. Formaggini L, Schmidt K, De Lorenzi D. Gastric dilatationvolvulus associated with diaphragmatic hernia in three cats: clinical presentation, surgical treatment and presumptive aetiology. J Feline Med Surg 2008; 10:198–201.
- 104. Gibson TWG, Prisson BA, Sears W. Perioperative survival rates after surgery for diaphragmatic hernia in dogs and cats: 92 cases (1990–2002). J Am Vet Med Assoc 2005; 227:105–109.
- 105. Hamilton MH, Sissener TR, Baines SJ. Traumatic bilateral ureteric rupture in 2 dogs. J Small Anim Pract 2006; 47:737–740.
- Aumann M, Worth LT, Drobatz KJ. Uroperitoneum in cats: 26 cases (1986–1995). J Am Anim Hosp Assoc 1998; 34:315–324.
- 107. Stafford JR, Bartges JW. A clinical review of pathophysiology, diagnosis and treatment of uroabdomen in the dog and cat. J Vet Emerg Crit Care 2013; 23:216–229.
- 108. Hunt GB, Culp WTN, Epstein S, et al. Complications of Stamey percutaneous loop cystostomy catheters in three cats.
- 109. Stiffler KS, McKrackin Stevenson MA, Cornell KK, et al. Clinical use of low-profile cystostomy tubes in four dogs and a cat. J Am Vet Med Assoc 2003; 223:325–329.
- 110. Kirby. In: Slatter. ed. Textbook of Small Animal Sugery, 3rd ed. Philadelphia: Elsevier Science (USA); 2003, pp. 414–445.
- Herold LV, Devey JJ, Kirby R. Clinical evaluation and management of hemoperitoneum in dogs. J Vet Emerg Crit Care 2008; 18:40–53.
- 112. Peddle GD, Carberry CA, Goggin JM. Hemorrhagic bile pleuritis and peritonitis secondary to traumatic common bile duct rupture, diaphragmatic tear, and rupture of the spleen in a dog. J Vet Emerg Crit Care 2008; 18:631–638.
- Jutkowitz LA, Rozanski EA, Moreau JA, et al. Massive transfusions in dogs: 15 cases (1997–2001). J Am Vet Med Assoc 2002; 220:1664– 1669.
- Green PD. Protocols in medicolegal veterinary medicine II. Cases involving death due to gunshot and arrow wounds. Can Vet J 1980; 21:343–346.
- Haley AC, Abramson C. Traumatic pneumocephalus in a dog. J Am Vet Med Assoc 2009; 234:1295–1298.
- Murgia D. A case of combined bilothorax and bile peritonitis secondary to gunshot wounds in a cat. J Feline Med Surg 2013; 15:513– 516.
- 117. Costanzo C, Garosi LS, Glass EN, et al. Brain abscess in seven cats due to a bite wound: MRI findings, surgical management and outcome. J Feline Med Surg 2011; 13:672–680.
- 118. Pratschke KM, Kirby BM. Highrise syndrome with impalement in three cats. J Small Anim Pract 2002; 43:261–264.
- 119. Rayward RM. Acute onset quadriparesis as a sequel to an oropharyngeal stick injury. J Small Anim Pract 2002; 43:295–298.
- Menard J, Schoeffler GL. Colonic, ureteral, and vascular injuries secondary to stick impalement in a dog. J Vet Emerg Crit Care 2011; 21:387–394.
- 121. Doran IP, Wright CA, Hotston MA. Acute oropharyngeal and esophageal stick injury in 41 dogs. Vet Surg 2008; 37:781–785.

- 122. Griffiths LG, Tiruneh R, Sullivan M, et al. Oropharyngeal penetrating injuries in 50 dogs: a retrospective study. Vet Surg 2000; 29:383–388.
- 123. Risselada M, de Rooster H, Taeymans O, et al. Penetrating injuries in dogs and cats: a study of 16 cases. Vet Comp Orthop Trauamtol 2008; 21:434–439.
- 124. Baker JL, Havas KA, Miller LA, et al. Gunshot wounds in military working dogs in Operation Enduring Freedom and Operation Iraqi Freedom: 29 cases (2003–2009). J Vet Emerg Crit Care 2013; 23:47– 52.
- 125. Scheepens ETF, Peeters ME, L'Eplattenier HF, et al. Thoracic bite trauma in dogs: a comparison of clinical and radiological parameters with surgical results. J Small Anim Pract 2006; 47:721–726.
- 126. Olsen D, Renberg W, Perrett J, et al. Clinical management of flail chest in dogs and cats: a retrospective study of 24 cases (1989–1999). J Am Anim Hosp Assoc 2002; 38:315–320.
- 127. Oyo-Ita A, Ugare UG, Ikpeme IA. Surgical versus non-surgical management of abdominal injury (review). Cochrane 2012; 11:1–18.
- 128. Tatton B, Jeffery N, Holmes M. Predicting recovery of urination control in cats after sacrocaudal injury: a prospective study. J Small Anim Pract 2009; 50:593–596.
- 129. Bali MS, Lang J, Jaggy A, et al. Comparative study of vertebral fractures and luxations in dogs and cats. Vet Comp Orthop Trauamtol 2009; 22:47–53.
- 130. Park EH, White GA, Tieber LM. Mechanisms of injury and emergency care of acute spinal cord injury in dogs and cats. J Vet Emerg Crit Care 2012; 22:160–178.
- 131. Krauss MW, Theyse LFH, Tryfonidou MA, et al. Treatment of spinal fractures using Lubra plates: a retrospective clinical and radiological evaluation of 15 cases. Vet Comp Orthop Trauamtol 2012; 25:326–331.
- 132. Nishida H, Nakayama M, Tanaka H, et al. Safety of autologous bone marrow stromal cell transplantation in dogs with acute spinal cord injury. Vet Surg 2012; 41:437–442.

- 133. Puerto DA, Aronson LR. Use of a semitendinosus myocutaneous flap for soft tissue reconstruction of a grade IIIb open tibial fracture in a dog. Vet Surg 2004; 33:629–635.
- Langley-Hobbs ŠJ, Brown G, Matis U. Traumatic fracture of the patella in 11 cats. Vet Comp Orthop Trauamtol 2008; 21:427– 433.
- 135. Nolte DM, Fusco JV, Peterson ME. Incidence of and predisposing factors for nonunion of fractures involving the appendicular skeleton in cats: 18 cases (1998–2002). J Am Vet Med Assoc 2005; 226:77–82.
- 136. Boero Baroncelli A, Peirone B, Winter MD, et al. Retrospective comparison between minimally invasive plate osteosynthesis and open plating for tibial fractures in dogs. Vet Comp Orthop Trauamtol 2012; 25:410–417.
- 137. Pozzi A, Hudson CC, Gauthier CM, et al. Retrospective comparison of minimally invasive plate osteosynthesis and open reduction and internal fixation of radius-ulna fractures in dogs. Vet Surg 2013; 42:19–27.
- Brinker, Piermatti, Flo. In: Piermatti, Flo. eds. Brinker, Piermatti, and Flo's Handbook of Small Animal Orthopedics and Fracture Repair, 3rd ed. Philadelphia: Elsevier Science; 1997, pp. 24–146.
- 139. Grant, Olds. In: Slatter. ed. Textbook of Small Animal Sugery, 3rd ed. Philadelphia: Elsevier Science (USA); 2003, pp. 1793–1798.
- 140. Ness MG. Treatment of inherently unstable open or infected fractures by open wound management and external skeletal fixation. J Small Anim Pract 2006; 47:83–88.
- 141. Wallace AM, De La Puerta B, Trayhorn D, et al. Feline combined diaphyseal radial and ulnar fractures: a retrospective study of 28 cases. Vet Comp Orthop Trauamtol 2009; 22:38–46.
- 142. Wyrzykowski AD, Feliciano DV. Trauma damage control. In: Mattox KL. ed. Trauma, 7th ed. New York: McGraw Hill; 2013, pp. 725–746.
- 143. Veterinary Committee on Trauma (VetCOT). Available at: https://sites.google.com/a/umn.edu/vetcot/. Accessed November 12, 2014.