



Manual of Trauma Management in the Dog and Cat

Edited by

Kenneth J. Drobatz, Matthew W. Beal
and Rebecca S. Syring

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MANUAL OF TRAUMA MANAGEMENT IN THE DOG AND CAT

EDITORS

KENNETH J. DROBATZ, DVM, MSCE, DACVIM, DACVECC

*Professor, Section of Critical Care
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, PA 19104*

MATTHEW W. BEAL, DVM, DACVECC

*Associate Professor, Emergency and Critical Care Medicine
Director of Interventional Radiology Services
College of Veterinary Medicine
Michigan State University
East Lansing, MI 48824-1314*

REBECCA S. SYRING, DVM, DACVECC

*Staff Veterinarian, Section of Critical Care
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, PA 19104*

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Editorial offices: 2121 State Avenue, Ames, Iowa 50014-8300, USA 9600 Garsington Road, Oxford, OX4 2DQ, UK

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I would like to dedicate this book to my mother and father, who taught me to have a strong work ethic, to be kind to others, and to have a strong loyalty and love for friends and family. Their level of generosity and caring is certainly a goal to strive for but one that will likely be too high for me to ever achieve.

Ken Drobotz

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And to my family for their love and support.

Matt Beal

To the doctors and veterinary nurses who I have worked with over the years, who have provided me immeasurable mentorship and assistance.

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Rebecca Syring

Preface

Trauma is one of the most common problems that dogs and cats encounter. Traumatic injuries range in severity from the simple breaking of a toenail to massive, life-threatening blunt or penetrating injuries involving multiple organ systems. Emergency veterinarians and criticalists are truly specialists in trauma. Traditionally, trauma has always fallen into the surgeon's arena since they induce tissue trauma on a daily basis through their surgical manipulations. As the field of traumatology has evolved, there is greater understanding of the systemic physiologic affects of massive trauma that may contribute as much, if not more, to the clinical consequences experienced by the animal than just the inciting incident. As this greater understanding of trauma developed, so in parallel did the development of the specialty of Emergency and Critical Care Medicine. These parallel developments resulted in veterinary Emergency and Critical Care specialists taking a major interest in the treatment of trauma patients. The field has developed rapidly, yet we are only beginning to understand the full ramifications of trauma and what it can do. As specialists in this area, we find the full understanding of this field of medicine daunting despite our clear focus on it. Yet, emergency and critical care specialists are not the only veterinarians to see these patients. General practitioners see traumatized patients on a daily basis. General practitioners are truly the veterinarians who "work in the trenches" and have the best global understanding of all conditions affecting the health of animals. The requirement for global understanding is very challenging, and hence, it is impossible to have an intimate understanding of every condition---trauma being one of

them. With this realization, we have developed a manual that applies a practical approach to traumatized patients. We have produced it in a logical and easily accessible organization that provides rapid but adequately detailed information about the general approach to the trauma patient as well information on specific traumatic injuries. Our goal was to make this manual something that the practicing veterinarian can use on the clinic floor to assist in the quick assessment and treatment of these very diverse and challenging patients. We have asked our chapter authors to make their portions clinically useful and as practical as possible, and yet provide an understanding of the underlying principles of what is being written. To this end, we have organized this book into an outline format that provides the essential facts that are necessary to understand and implement the principles in the clinical approach to trauma patients. The initial chapters provide a background in the global assessment of the traumatized patients followed by chapters dealing with very specific types of traumas. It is our hope that the practitioner will find this not an "Ivory Tower Manual" but one that will be used in the "trenches", one that will get dirty and worn out due to its frequent use on the clinic floor.

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Contributor List

Matthew W. Beal, DVM, DACVECC

Associate Professor, Emergency and Critical Care
Medicine

Director of Interventional Radiology Services

College of Veterinary Medicine

Michigan State University

East Lansing, Michigan

Benjamin M. Brainard, VMD, DACVA, DACVECC

Assistant Professor, Critical Care

Department of Small Animal Medicine and Surgery

University of Georgia

Athens, Georgia

Andrew J. Brown, MA, VetMB, DACVECC, MRCVS

VetsNow Referral Hospital

123-145 North Street

Glasgow

G3 7DA

SCOTLAND

Dana L. Clarke, VMD, DACVECC

Lecturer in Emergency and Critical Care

Fellow in Interventional Radiology

Department of Small Animal Clinical Sciences

University of Pennsylvania

Philadelphia, Pennsylvania

Merilee F. Costello, DVM, DACVECC

Staff Criticalist

Boston Road Animal Hospital

Springfield, MA

William T. Culp, VMD, DACVS

Assistant Professor, Small Animal Surgery
Department of Surgical and Radiological Sciences
School of Veterinary Medicine
University of California
Davis, California

Kenneth J. Drobatz, DVM, MSCE, DACVIM, DACVECC

Professor, Section of Critical Care
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, Pennsylvania

Daniel J. Fletcher, PhD, DVM, DACVECC

Assistant Professor of Emergency and Critical Care
Cornell University College of Veterinary Medicine
Ithaca, New York

David E. Holt, BVSc, DACVS

Professor of Surgery
Department of Clinical Studies
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, Pennsylvania

John R. Lewis, VMD, FAVD, DAVDC

Assistant Professor of Dentistry and Oral Surgery
Department of Clinical Studies
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, Pennsylvania

Deborah C. Mandell, VMD, DACVECC

Staff Criticalist, Emergency Service
Adjunct Assistant Professor
Department of Clinical Studies
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, Pennsylvania

Lauren R. May, VMD, DACVS

Staff Surgeon
Department of Surgery
Veterinary Specialists of Rochester
Rochester, New York

Philipp D. Mayhew, BVM&S, MRCVS, DACVS

Assistant Professor, Small Animal Surgery
Department of Surgical and Radiological Sciences
School of Veterinary Medicine
University of California-Davis
Davis, California

Charles S. McBrien Jr., DVM, MS

Northeast Veterinary Referral Hospital
Plains, PA

Stephen J. Mehler, DVM, DACVS

Staff Surgeon
Department of Surgery
Veterinary Specialists of Rochester
Rochester, New York

**Alexander M. Reiter, Dipl. Tzt., Dr. med. vet.,
DAVDC, DEVDC**

Associate Professor of Dentistry and Oral Surgery
Department of Clinical Studies
School of Veterinary Medicine

University of Pennsylvania
Philadelphia, Pennsylvania

Jessica M. Snyder, DVM, DACVIM (Neurology)

Staff Neurologist
Department of Neurology
VCA Veterinary Specialty Center of Seattle
Lynnwood, Washington

Lindsey Beth Culp Snyder, DVM, MS, DACVA

Clinical Assistant Professor, Anesthesia and Pain
Management
Department of Surgical Sciences
University of Wisconsin—Madison
Madison, Wisconsin

Rebecca S. Syring, DVM, DACVECC

Staff Criticalist, Section of Critical Care
School of Veterinary Medicine
University of Pennsylvania
Philadelphia, Pennsylvania

Chapter 1

GLOBAL APPROACH TO THE TRAUMA PATIENT

Kenneth J. Drobatz

1. GLOBAL APPROACH TO THE TRAUMA PATIENT

a. Trauma is defined as a “wound or injury” and may occur secondary to motor vehicle accidents, fall from heights, animal interactions, human-animal interactions, etc. Severity may range from mild to fatal. Trauma may affect only one organ system or multiple organ systems, either directly or indirectly. Therefore, a global and thorough approach is required to improve survival and decrease morbidity in trauma patients.

b. Initial assessment of the trauma patient occurs quickly but can theoretically be broken down into two major phases; primary survey and secondary survey:

i. Primary survey is the assessment of the respiratory and cardiovascular systems, followed by assessment of the central nervous system and urinary tract system.

ii. Secondary survey is assessment of all other systems once the animal's most immediately life-threatening problems, identified during the primary survey, are dealt with.

c. The primary goal in attending to a critically injured trauma patient is to optimize oxygen delivery to the tissues. In fact, during the initial approach and resuscitation of these patients, all procedures are

oriented toward this goal. Oxygen delivery is dependent upon cardiac output, hemoglobin concentration, and oxygen saturation of hemoglobin. It is important to optimize these variables at all times.

2. BRIEF PATHOPHYSIOLOGY

a. Traumatic shock results in a maldistribution of blood flow because of increased circulating catecholamines, hypovolemia, and increased vasoactive hormones.

b. Persistent microcirculatory perfusion failure may lead to sludging of blood and increased cellular and platelet aggregation.

c. Endothelial injury due to trauma may result in release of inflammatory mediators and stimulation of coagulation.

d. Damaged cellular membranes, due to poor perfusion or direct trauma, result in release of phospholipids. The enzymes phospholipase, cyclooxygenase, and lipoxygenase produce thromboxane and leukotrienes:

i. Thromboxane causes vasoconstriction, further compromising tissue perfusion and stimulating platelet aggregation.

ii. Leukotrienes and activation of the complement cascade propagates the inflammatory response with mobilization and activation of neutrophils.

iii. Neutrophil release of lysosomal enzymes and toxic oxygen metabolites cause further cellular damage, leading to edema with subsequent increase of oxygen diffusion distance to the cells from the capillaries.

iv. Optimizing tissue oxygen delivery will minimize the perpetuation of these inflammation-producing cascades.

3. ASSESSING AND MAINTAINING OXYGEN SATURATION OF HEMOGLOBIN—THE RESPIRATORY

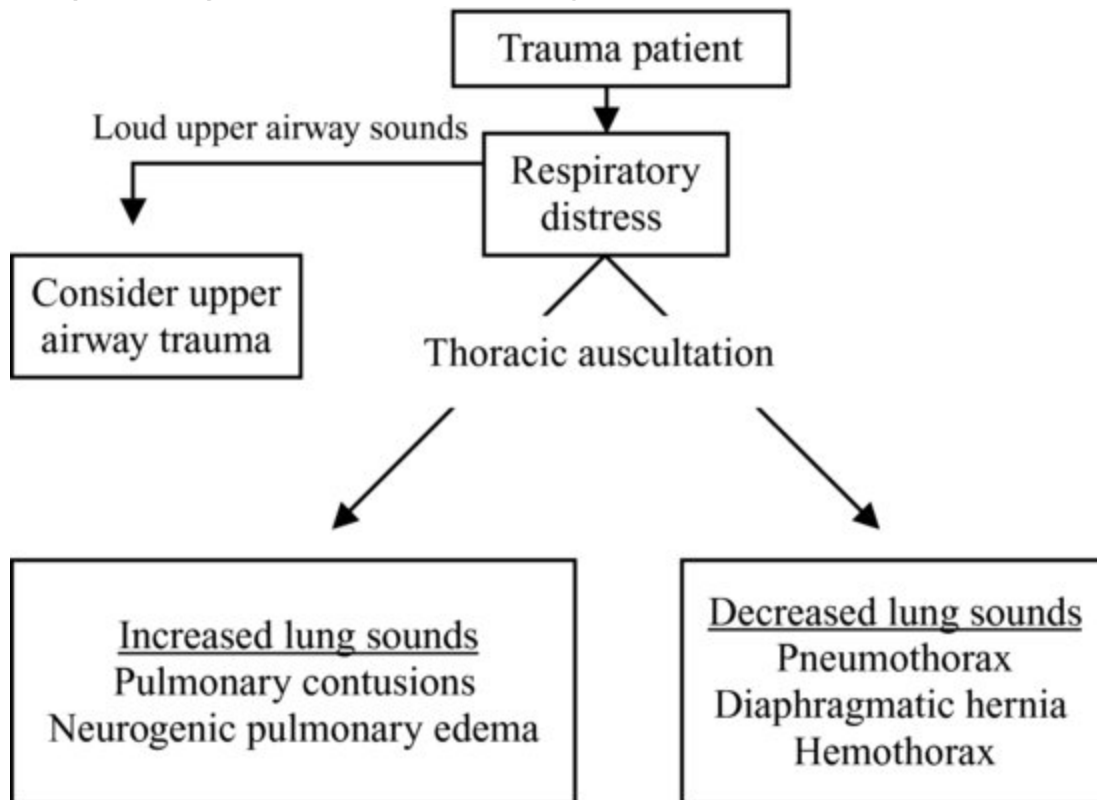
SYSTEM

a. At presentation, supplemental oxygen should be provided to the severely affected trauma patient until assessment of an arterial blood gas or measurement of hemoglobin saturation confirms that oxygen supplementation is not required:

i. Clinical signs of respiratory compromise include increased respiratory rate or effort, flail chest, pale or cyanotic mucous membranes, increased heart rate, increased upper airway sounds, or altered lower airway sounds (increased or decreased airways sounds). If these signs are present, oxygen supplementation is essential.

b. Physical assessment of the respiratory system ([Figure 1.1](#)):

FIGURE 1.1 Algorithm for assessment of the patient with respiratory distress following trauma.



i. Look:

1. Signs of respiratory compromise include increased respiratory rate and effort, restlessness, extended head and neck, abducted elbows, and paradoxical movement of the chest and abdominal walls (normally should move in and out together but with paradoxical movement they move in opposite directions).

2. Remember: animals in severe respiratory distress do not have to have all of these signs (especially cats), *but* if they do have all of these signs they have severe respiratory compromise.

ii. Listen:

1. Loud upper airway sounds heard without the stethoscope indicate upper airway compromise (from the carina forward).

2. Traumatic causes of upper airway problems include cervical tracheal crush injury (bite wounds are most common), pharyngeal injury (swelling/bleeding), and tracheal avulsion (see Chapter 6: Trauma-Associated Thoracic Injury):

a. Note that nasal airway injuries and frontal sinus injuries can result in bleeding into the nasal passages, causing upper airway sounds. Despite this problem, most animals do not suffer respiratory compromise from these injuries.

3. Decreased air sounds on auscultation:

a. Indicates pleural space disease.

b. Traumatic causes of pleural space disease:

i. Pneumothorax (most common)

ii. Diaphragmatic hernia

iii. Severe hemothorax (least common)

c. Thoracocentesis:

i. If the patient is in respiratory distress and you cannot hear the lung sounds well, then

thoracocentesis is warranted.

4. Increased lung sounds on auscultation (not the upper airway sounds referred to earlier):

a. Indicate lower airway/pulmonary parenchymal injury:

i. Pulmonary contusions:

1. Therapy for pulmonary contusions is largely supportive with caution on overzealous fluid therapy.

5. Open chest wound/“sucking” chest wound:

a. Open wounds connecting to the pleural space should be immediately covered with a sterile bandage, which will create a seal, and thoracocentesis should then be performed to remove the residual air.

b. If respiratory distress persists, intubation and positive pressure ventilation should be provided until the wounds can be surgically repaired.

6. Flail chest:

a. Recognized by paradoxical movement of the chest wall. The flail segment moves in on inspiration and is a result of a “floating” segment of the chest wall because consecutive ribs are broken in more than one place.

b. Primary physiologic consequences are pain and underlying lung damage and pleural space injury in most instances.

4. ASSESSING CARDIAC OUTPUT/TISSUE PERFUSION

a. Initial assessment of adequate cardiac output and circulating volume includes mucous membrane color, capillary refill time, pulse rate, and quality:

i. Abnormalities indicating poor tissue perfusion

1. Pale or gray mucous membranes

2. Prolonged capillary refill time (>1-2 seconds)

3. Weak and rapid pulses

b. Measurement of arterial blood pressure directly or indirectly by Doppler or oscillometric techniques should be performed when possible:

i. A Doppler systolic blood pressure of <80 is suggestive of decreased tissue perfusion.

ii. A mean arterial pressure of <60 mm Hg is suggestive of decreased tissue perfusion.

iii. It should be kept in mind that blood pressure may be normal or even high and peripheral tissue perfusion could still be inadequate for the body's requirements.

c. More objective evaluation is obtained by pulmonary artery catheter placement and actual measurement of cardiac output, oxygen delivery, and oxygen consumption; although this is uncommonly done in veterinary emergency medicine and carries its own complications.

d. The most common cause of poor tissue perfusion in the trauma patient is hypovolemia from blood loss:

i. Most common areas for hemorrhage causing hypovolemia are in order of frequency; the peritoneal space, retroperitoneal space, thigh musculature, pleural space, external, or a combination of spaces (e.g., retroperitoneal and peritoneal spaces).

e. The less common causes of poor tissue perfusion are cardiac arrhythmias, tension pneumothorax, diaphragmatic hernia, peritoneopericardial diaphragmatic hernia, and cardiac tamponade due to hemopericardium from traumatic atrial rupture.

f. Therapy of poor tissue perfusion:

i. Thoracocentesis if a tension pneumothorax is present (this is extremely rare).

ii. Intravenous fluids/colloids/blood products/hemoglobin substitutes:

1. Caution with fluids when pulmonary contusions are present.

5. ASSESSMENT OF THE NEUROLOGIC SYSTEM

a. It is important to assess brain function at presentation to obtain a baseline for potential dynamic changes that may occur.

b. Conclusions regarding brain and neurologic function (brain and spinal cord) should be withheld until perfusion is adequate and function is reassessed:

i. Profound neurologic changes may be found with poor tissue perfusion and corrected once tissue perfusion is improved.

c. Assessment of brain function:

i. Mentation:

1. Grades of mentation:

a. Excited/agitated

b. Normal

c. Depressed

d. Obtunded (markedly decreased level of consciousness, but arousable with auditory or tactile stimulation)

e. Stupor (unconscious but responds to noxious stimuli)

f. Coma (unconscious and does not respond to noxious stimuli)

2. Cranial nerve assessment:

a. Assessment of facial sensation, jaw tone (be careful with jaw fractures), eye position and movement, papillary light responses, menace reflex, eye blink, and gag reflex.

3. Evidence of potential brain injury:

a. Obvious head trauma (skull fractures, bleeding from ear, jaw fractures, scleral, or ocular hemorrhage, etc.)

b. Altered mentation

c. Altered cranial nerve function

4. General principles of treating brain injury:

a. Primary injury (direct trauma to tissues)—no specific treatment for this.

b. Secondary injury (swelling, inflammation, reperfusion injury, increased intracranial pressure):

i. Maintain good perfusion/blood pressure (MAP at least 80 mm Hg, systolic at least 90 mm Hg).

ii. Maintain good oxygenation via oxygen supplementation, ventilation, etc. ($\text{PaO}_2 > 80$ mm Hg, $\text{SpO}_2 > 95\%$).

iii. Hyperosmotics:

1. Mannitol

2. Hypertonic saline/colloid combination to treat decreased blood pressure and poor tissue perfusion

iv. Elevate head 30° from horizontal if increased intracranial pressure is suspected.

d. Assessing spinal cord function:

i. Ensure that tissue perfusion is adequate.

ii. Palpate full length of the spine and pelvis.

iii. Note the voluntary movement of all legs.

iv. Assess the ability to stand/ambulate.

v. Check anal tone and anal reflex.

vi. Check proprioception of limbs.

vii. Assess limb reflexes.

viii. Evidence of spinal cord injury:

1. Spinal or sacral fractures/displacement

2. Spinal pain

3. Changes in limb function or sensation.

4. Change in tail tone, bladder, or anal sphincter function

ix. General principles of treating spinal cord injury:

1. Stabilization of fractures/luxations
2. Decompression surgery (e.g., hemilaminectomy for ruptured intervertebral disk)
3. Corticosteroids (controversial)
4. Time

6. ASSESSING THE RENAL SYSTEM

- a. Physical assessment of the urinary tract is limited to palpation of the urinary bladder and kidneys.
- b. Urinary tract injury seems to occur more commonly with pelvic fractures.
- c. BUN and serum creatinine may not have substantial increases until several hours after urinary tract rupture or injury.
- d. Hematuria indicates urinary tract injury.
- e. Manifestations of renal damage may not be immediately evident at presentation and may not be detected until several hours later, after continuous monitoring.
- f. Potential trauma-induced urinary tract injuries:
 - i. Kidney contusions, rupture, or avulsion
 - ii. Renal pelvis disruption
 - iii. Urethral disruption
 - iv. Urinary bladder rupture
 - v. Urethral trauma/rupture
- g. General therapy for urinary tract trauma:
 - i. IV fluid diuresis
 - ii. Stabilization of cardiovascular manifestations of urinary tract injury (hyperkalemia)
 - iii. Surgical repair and/or urinary diversion of injuries if necessary

7. SECONDARY SURVEY

- a. Full physical examination.
- b. Particular attention should be paid to the following:
 - i. Musculoskeletal system:

- 1.** Observe limb function of all four legs.
 - 2.** Observe ambulation.
 - 3.** Complete palpation of appendicular and axial skeleton including rectal examination, palpation of skull, and manipulation of jaw.
- ii.** Peripheral nervous system:
Full evaluation of peripheral nerves.
 - iii.** Examine the animal from the tip of the nose to the tip of the tail.

8. MONITORING

- a.** Monitoring of all the above systems should be done for at least 24-48 hours for animals that have had significant trauma.
- b.** Generally, if significant injury has occurred, clinical signs will usually develop within 24-48 hours of the traumatic incident:
 - i.** Biliary tract rupture can sometimes result in clinical signs several days after the trauma.
 - ii.** Patients with diaphragmatic hernias may not develop respiratory distress for months to years.
- c.** The intensity of the monitoring should be proportional to the degree of compromise of the patient.

Chapter 2

TRIAGE AND PRIMARY SURVEY

Matthew W. Beal

1. INTRODUCTION

a. Success in the management of small animal emergency patients or dynamic critically ill patients will largely be based on the ability of the veterinary health care team to rapidly identify and manage immediately life-threatening problems.

b. The following pages will focus on facility readiness, triage and major body systems assessment, and the primary survey of the traumatized dog or cat.

2. FACILITY READINESS

a. Infrastructure:

i. Preparedness for small animals presented with potentially life-threatening problems should not just be reserved for emergency hospitals and large referral centers. Through a minimal amount of staff training in the art of triage (see the following), attention to physical examination findings, delegation of responsibilities to the team, and minimal financial outlay for necessary equipment, any veterinary facility can be equipped to handle immediate stabilization of life-threatening problems.

ii. There is no substitute for practice or “mock” emergencies for honing the skills of the team.

b. Initial contact:

i. Emergency management begins at the front desk / receptionist, whether a client is calling on the phone regarding a crisis with a pet, or walking through the front door with an ill or injured pet. Of critical importance to client satisfaction is that the reception personnel display a “can-do” attitude.

ii. Emergencies are not scheduled for appointments. When the client brings a patient through the door, an *immediate* series of events should be triggered beginning with a call to the doctor(s), technicians, or veterinary assistants so that a triage and major body systems assessment can be performed (see the following).

c. Equipment:

i. If, based on triage, the pet is judged to have potentially life-threatening problems, it is immediately moved to the treatment area.

ii. The treatment area should provide ready access for oxygen supplementation and ideally monitoring equipment including an ECG, Doppler blood pressure monitor, and pulse-oximeter. A defibrillator is desirable.

iii. Crash cart versus crash box:

1. A fully stocked “crash cart” is not likely to be practical for the small private practice; however, a large tackle box that is ONLY used for emergencies is an appropriate substitute.

2. The crash box should be restocked after every usage and a seal placed over the latch to document this. The crash box should be opened weekly to check drug expiration dates and to ensure adequate stock of required supplies. These tasks can easily be delegated to a technician or veterinary assistant.

3. A crash box should contain the equipment listed in [Table 2.1](#). This equipment will allow the