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## APPROACH TO THE EMERGENCY PATIENT

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All stages of emergency evaluation are important to the positive and successful management of the emergency patients: waiting room triage, primary survey and initial stabilization, secondary survey. Triage and primary survey allow the clinician to identify the most unstable patient and to focus on the treatment of the most life threatening conditions.

**Triage:**
Triage involves a logical examination of patients so that they can be classified according to the severity of their illness/injury. This ensures that the most critical patients/problems are dealt with first [1,2]. Every emergency patient should be examined immediately, whilst still in the in the waiting room. Triage should focus on the rapid evaluation of the ABCD's: airway, breathing, circulation, and disabilities (evaluation of the mental state and gait) [1,2].

1) Airway
   - Be sure the animal has a patent airway.

2) Breathing:
   - Is the animal breathing? What is the animal's respiratory rate and effort?
     Signs of respiratory distress include loud airway sounds, increased rate and effort, flaring of the nares, open mouth breathing, extended head and neck. Apnoeic patients should be intubated and ventilated with 100% oxygen.
     Patients that are breathing but show signs of respiratory distress require immediate stabilisation with supplemental oxygen.

3) Circulation:
   - Is the heart beating effectively? Is there a peripheral pulse? What colour are the mucous membrane?
     Palpate the peripheral pulse and listen to the heart. If no pulse or heartbeat can be detected cardiopulmonary cerebrovascular resuscitation (CPCR) should be initiated.
     Signs of cardiovascular compromise include severe tachycardia or bradycardia, weak peripheral pulses, pale or hyperaemic mucous membrane.

4) Disability:
   - Observe the patient's level of consciousness. Observe the posture of the animal ( head tilt, Shiff-Sherrington's posture) and the ability of the animal to stand walk or the presence of paresis or paralysis.

A very brief or capsule history should be obtained from the owners about the nature of the primary complaints and its progression.
As a result of the triage any unstable animal should be promptly moved to the examination room. Here, the primary survey should be performed to gather further information and first treatment can be provided [2].
It's important to observe the patient quickly for obvious signs of trauma injury (limb or spinal fractures, thoracic wounds). Being aware of such abnormalities helps with the initial approach of the patient: manipulating a broken leg is painful and can provoke aggressive behaviour; suspicion of spinal injury necessitates the immediate need of a backboard before moving the patient to avoid any further damage to the spinal column.

*Primary survey*: Major body system assessment.
The primary survey amplifies the information obtained during the triage and it's focused on the assessment of the three major body system: respiratory, cardiovascular and (central) nervous system [2]. Its purpose is to determine the stability of the patient in more detail and to identify and treat more subtle life-threatening conditions.

*Respiratory system examination:*
Dogs and cats with respiratory distress are prone to sudden deterioration, so careful management is always required.

Further assessment should focus on:
- Respiratory pattern
- Respiratory rate
- Respiratory effort
- Lung sounds on auscultation



The patient's breathing pattern, effort, rate, and rhythm can be evaluated from a distance. The simple observation of the respiratory pattern could help us localise the area of the respiratory tract primarily involved (upper airway, lower airway, pulmonary parenchyma and pleural space).
Loud sounds that can be heard without the aid of the stethoscope (stridor and stertor) should be recognized as they are indicative of upper airways partial obstruction.
The respiratory assessment should proceed with thoracic auscultation. Increased lung sounds and the presence of wheezes and crackles are usually signs of lower airway (wheezes) or parenchymal disease (crackles). In pleural space disease auscultation will identify a marked decrease in, or absence of, breath sounds. With pleural effusion, the breath sounds are decreased ventrally; with pneumothorax they are decreased dorsally.

**Cardiovascular system**
Is the patient in shock? This is the most important question we should be able to answer quickly while performing the assessment of the cardiovascular status of the patient.
Our evaluation should focus on assessment of :
- heart rate and auscultation
- pulse quality
- mucous membrane colour
- capillary refill time (CRT)

*Heart rate*
Seriously ill patients may have heart rates higher than 160-180 bpm, although bradycardia in cats can be a sign of severe illness and hypoperfusion.
When, despite the degree of collapse bradycardia is detected, particularly in dogs, be very vigilant for possible underlying metabolic (Addison disease), electrolyte (hyperkalaemia) neurological disease (increased intracranial pressure) or sinoatrial node disease.

Heart auscultation should be carefully performed to detect murmurs or gallop rhythm.

*Pulse quality*
Peripheral pulse should be palpated or at the femoral or at the metatarsal pulse. A normal pulse should be readily palpable and strong. An arterial pulse stronger or easier to feel than normal is described as 'bounding' or 'snappy' and it is typically identified in early stages of shock and anaemia. An arterial pulse may also be weak, very weak/'thready', or absent. Causes include later stages of shock as well as heart disease resulting in poor cardiac muscle contraction. The presence of pulse deficit is usually indicative of the presence of a dysrhythmia.

*Mucous membrane colour*
A pale to white mucous membrane colour is caused by hypoperfusion (hypovolaemic / cardiogenic shock) or anaemia. A red color suggests an increased volume of blood in the capillary bed because of vasodilation, as in systemic inflammatory diseases or sepsis. The evaluation of the mucous membrane allows also the detection of cyanosis, icterus, petecchiae or ecchimosis

*Capillary refill time*
It is normally 1 to 2. CRT is determined by precapillary sphincter tone. Vasoconstriction caused by an increase in sympathetic tone, such as in response to hypoperfusion in shock, can cause the CRT to be greater than 2 seconds, while vasodilation often characteristic of systemic inflammation or sepsis, can result in a CRT that is shorter than normal (i.e, less than 1 second).

**Neurologic system examination**
A brief neurologic survey is part of the major body system evaluation in the initial assessment and should focus on:
- Mental state
- Posture
- Examination of the spine

*Mental state*
It is the patient's level of consciousness and response to surroundings.
Characterize the patient's mental state as:
- Alert and normally responsive
- Depressed or obtunded
- Semicomatose, stuporous
- Comatose

The last two categories suggest abnormal brain function; a depressed or obtunded state is often caused by extracranial disease, such as decreased tissue perfusion, pain, or metabolic disease.

*Posture:*
It's important to note abnormal posturing, such as hyperextension of the neck with extensor rigidity of the forelegs (Schiff- Sherrington sign); spontaneous nystagmus, strabismus, and head tilt.

*Examination of the spine.*
Palpate the entire spinal column for areas of swelling, crepitation, bony deformity, or pain. Spinal reflexes (when possible) should be elicited and deep pain assessed.

**Additional Evaluation**
Another vital sign which should be measured and recorded during the initial assessment is core body temperature.
The aim of the primary survey is to identify life-threatening respiratory and cardiovascular states and address them before a definitive diagnosis is reached. Once the patient is stable then a more in-depth secondary survey can begin, which should include a full physical examination of the patient and further, in-depth investigations and treatments.

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## APPROACH AND MANAGEMENT OF THE BLEEDING PATIENT

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Bleeding is a common clinical presentation in small animal practice, occurring more commonly in dogs than in cats. Clinical manifestations of bleeding disorders may range from mild and self-limited to life-threatening hemorrhage requiring immediate medical attention.

Hemostasis is the complex physiologic response to bleeding. Classically, three systems (the endothelial vascular cells, the primary and secondary haemostatic systems) are considered to be responsible for this [(1)].

*Vascular wall*
The vascular wall is principally responsible for maintaining an antithrombotic surface in health and alterations in the expression of vascular wall molecules help to promote clotting when the wall is damaged[(1)].

*Primary haemostasis*
It involves the interaction between the damaged vascular wall, Von Willebrand factor (a large plasma protein synthesized by endothelium that facilitates adhesion between platelets and subendothelium) and platelets, leading to the formation of the fragile platelet plug[(1,2)].

*Secondary haemostasis:*
Secondary haemostasis refers to activation of the clotting cascade resulting in formation of fibrin that acts to stabilise the platelet plug.
The clotting cascade is traditionally split into the intrinsic, extrinsic and the common pathways and it involves the activation of different clotting factors. The majorities of the clotting factors are synthesized in the liver and circulate as inactive precursors in the plasma. Vitamin K is needed for the functional synthesis of the coagulation factor II, VII, IX, and X. Calcium (factor IV) is required for most reactions to occur[(1)].

*The cell based model of coagulation*
It has long been recognised that the model of coagulation as described above does not explain all the clinical phenomena associated with blood clotting disorders but it remains a clinically useful distinction in terms of the tests we have available for assessment of secondary haemostatic function. This has led to the development of the cell based model of coagulation which essentially integrates the roles of the vascular endothelium, platelets and clotting cascade[(3)].

*Diagnostic approach:*
Clinical presentations of the bleeding patient may be suggestive of certain haemostatic disorders and this explains the importance of an accurate physical examination. Different diagnostic tests have been developed and are available for clinical practice; their clinical application and correct interpretation, associated with physical examination findings, allow a specific diagno-



sis of the bleeding disorder and prompt and appropriate management of the coagulopathy.

*Clinical signs:*
Clinical signs commonly associated with disorders of the primary haemostatic system include petechiation/ecchymoses, meleana, haematemesis, epistaxis and excessive surgical bleeding. However, von Willebrand disease (vWD) and thrombopathias are causing bleeding at sites of injury (trauma, dental disease, estrus, gastrointestinal) rather than petechiae or ecchymoses[2].
Clinical signs associated with secondary haemostatic disorders include large bleeding into body cavities, haematuria, pulmonary haemorrhage, epistaxis and bleeding from venipuncture sites[1].

*Diagnostic tests:*
When evaluating a patient with a suspected primary haemostatic disorder, an accurate platelet count should be obtained. Although in house haematology machines may be useful they may also be inaccurate and emergency clinicians should be familiar with the assessment of platelet counts on a fresh blood smear. Generally 8-15 platelets per high power field (x100) would be considered normal. Clinical bleeding is unlikely unless the platelet count drops below 50 x 109/L which is approximately 3-4 platelets per high power field. The presence of multiple platelet clumps in the feathered edge is likely to represent numbers adequate for haemostasis.
The function of the primary haemostatic system may be assessed by the buccal mucosal bleeding time (BMBT).

Tests used for evaluation of secondary haemostasis include
- Prothrombin time (PT) – extrinsic and common pathway
- Activated partial thromboplastin time (APTT) – intrinsic and common pathway
- Activated clotting time (ACT) – intrinsic and common pathway

*Differential diagnosis:*
Thrombocytopenia may be caused by[2]:
- increased platelet destruction (e.g. immune mediated thrombocytopenia)
- decreased platelet production (bone marrow disorders, drug toxicity)
- increased platelet consumption (excessive bleeding and disseminated intravascular coagulation)
- sequestration (commonly in the spleen)

Thrombocytopathia (i.e. abnormal platelet function) may be secondary to hereditary disorders (rare) or to acquired conditions such as in renal or hepatic failure or secondary to certain drug administration ( non steroidal anti-inflammatory drugs) [2].

The differential diagnosis of secondary haemostatic disorders includes[1]:
- decreased clotting factor production (e.g. hepatic failure)
- increased clotting factor consumption (e.g. disseminated intravascular coagulation)
- specific clotting factor decrease (primarily congenital- Haemophilia A (factor VIII deficiency) and Haemophilia B (factor IX deficiency))
- inability to activate clotting factors (vitamin K deficiency/antagonists).

**Common disorders associated with coagulation abnormalities**

*Immune mediated thrombocytopenia:[2]*
Patients present with a wide spectrum of clinical signs: from mild petechiation to hypovolaemic shock secondary severe bleeding. The commonest site for marked blood loss is the gastrointestinal tract.
Typically these patients have very low platelet counts (0-30 x $10^9$/L). Although most cases are idiopathic, patients should always be evaluated for an underlying trigger such as infection, drug reaction or neoplasia.
The most important treatment is appropriate immunosuppressive medication (corticosteroids) although the sicker patients may also need transfusion support in the short term.

*Von Willenbrand disease[2]:*
There are 3 types of vWD. Type 1 is the most commonly recognized in breeds such as Doberman pinschers, Pembroke Welsh Corgis, and Bernese Mountain Dogs. This is considered the mildest of all three types.
Dogs with vWD bleed from mucosal surfaces such as the nares and gingiva. They may have excessive bleeding and bruising with trauma and surgery. It is usually diagnosed in animal with normal thrombocytes count, normal clotting factors and a prolonged BMBT. vWF analysis should be performed.

Fresh whole blood, fresh frozen plasma, and cryoprecipitate are recommended products to replace vW factor. DAVP (desmopression) is a synthetic vasopressor that has been shown to release stores of vWF, increasing plasma vWF.

*Disseminated intravascular coagulation:*
DIC represents a consumptive coagulopathy and manifests with abnormalities of both the primary and secondary haemostatic systems. It is invariably associated with other severe conditions such as sepsis, systemic inflammatory response syndrome or disseminated neoplasia and prognosis is largely dependent on whether this triggering condition can be resolved[1]. Treatment should be targeted at resolving the underlying disease with support of the coagulation system with supplementation of the clotting proteins (fresh frozen plasma transfusions) whilst this is achieved.

*Rodenticide toxicity[1]*
Rodenticides containing vitamin K antagonists (warfarin, brodifacoum etc) are a relatively common cause of clotting problems especially in young dogs. The commonest site for bleeding is the thoracic cavity and respiratory tract although bleeding can occur anywhere. Signs typically develop 3-4 days following ingestion. Both PT and APTT will ultimately be prolonged, however, factor VII has the shortest half-life so PT becomes prolonged first. In the stable patient, the treatment is parenteral or oral vitamin K therapy (5-2.5 mg/kg once a day). In the bleeding patient, besides vitamin K administration, urgent replacement of activated clotting factors is required with plasma transfusions. Vitamin K therapy may need to be continued for 6-8 weeks if a second or third generation rodenticide has been ingested.

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## CARDIOPULMONARY CEREBRAL RESUSCITATION (CPCR)

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Cessation of effective circulating blood flow and ventilation constitutes cardiopulmonary arrest (CPA) that leads to inadequate oxygen delivery to tissue, shock and death. Cardiopulmonary arrest is typically associated with loss of consciousness, collapse, lack of a palpable pulse, pale or cyanotic mucous membranes, lack of effective respirations, and lack of measurable blood pressure[1].

There are many predisposing causes for CPA, including sepsis, cardiac failure, pulmonary disease, neoplasia, coagulopathies, anesthesia, toxicities, multisystem trauma, traumatic brain injury, and systemic inflammatory response syndrome[1]. Additionally abnormally high vagal tone such as may accompany severe vomiting, tenesmus or upper-respiratory tract obstruction might initiate some CPA events.

Closed and careful monitoring for deterioration in critical patients is essential as the most successful CPCR is the one that is avoided. Before a possible episode of CPA the patient may become obtunded, hypothermic, bradycardic or hypotensive, develop dilated, unresponsive pupils and change his respiratory pattern progressing to gasping and agonal breaths at the time of CPA. Unfortunately some cases that require CPCR present after CPA have already occurred.

Cardiopulmonary-cerebral resuscitation refers to re-establishing blood flow to the cerebral and coronary systems in the event of CPA by performing manual cardiac and thoracic compressions and manual ventilation until spontaneous circulation and ventilation occurs[1,2].

The decision to begin CPCR has to be based on clinical signs, consideration on potential outcome and underlying disease process (CPCR should be attempted only



in patients that have a possible treatable disease) and ideally on a previous agreement with the animal owners. The patient's resuscitation code ( "attempt resuscitation" and "do not attempt resuscitation" (DNAR)) should be discussed with the owner at time of hospital admission or in case of deterioration of the clinical condition[1].

There three phases of CPCR:
1) Basic Life Support (BLS)
2) Advanced life support (ALS)
3) Post- resuscitation care

**Basic life support:**
Basic life support involves establishing and maintaining an airway (A) , with supplemental oxygen initiating artificial ventilation (B) and starting manual cardiac and thoracic compression to re-establish circulation (C) [1,2].

*Airway:*
An airway should be rapidly established by placing a well-fitting, cuffed endotracheal tube. When orotracheal intubation cannot be performed an emergency tracheostomy or mouth-to-nose respiration should be considered.

*Breathing:*
Manual ventilation should be instituted with 100% oxygen at a rate of 10-12 breaths per minute. All breaths should be given over 1 second followed by a pause to allow normal relaxation of the chest. High volumes and high pressures (>20 mmHg) must be avoided to prevent iatrogenic barotrauma. An Ambu® bag or an anaesthetic breathing system delivering 100%oxygen should be used.

*Circulation:*
External chest compressions are intended to move blood from the chest to the vital organs and enhance venous return. External chest compressions should be performed at a rate of 80-100 compressions/minute with the patient on a firm surface in lateral recumbency. Compressions should be performed with a 1:1 ratio of compression to relaxation and the chest wall should be allowed to expand fully between compressions. External chest compressions employ either the thoracic pump (the compressor's hand should be placed over the widest part of the chest wall) for animal heavier than 15 kg, or the cardiac pump (the compressor's hand placed directly over the apex of the heart ($4^{th}$ - $6^{th}$ intercostal spaces)) for smaller animals.

In patients with conditions like pneumothorax, hemothorax, flail chest or rib fractures, diaphragmatic hernia, pericardial effusion or unsuccesfull closed chest CPCR ( >5 min), open-chest CPCR (direct cardiac massage through a lateral thoracotomy) should be considered.

If the chest compressions are successful we should be able to detect the presence of a peripheral femoral pulse and the patient should have an end tidal $CO_2$ > 10 mmHg on a capnograph[1,2].

**Advanced Life Support (ALS)**
Following BLS (if possible simultaneously), a venous access should be gained often through cut-down procedure or the placement of an intraosseus catheter should be considered. Alternatively the endotracheal rout offers a simple and rapid approach to drug administration during CPCR. Epinephrine, atropine, vasopressin and most drugs can be administered (not sodium bicarbonate), but with at least 2 to 3 times the intravenous dose, diluted in an appropriate volume (e.g. 5 mL/20 kg) of normal saline and injected via a catheter through the ET tube[1,2].
Fluid therapy is necessary only if the patient was hypovolaemic before the CPA[2]. If a patient was on any medication that is a potential cardiac or respiratory depressant, that drug must be immediately reversed.
An electrocardiogram should be performed to determine the cardiac rhythm. Drugs should be administered based on a particular cardiac rhythm and timing during CPCR[1,2].
The anticholinergic compound atropine (0.02-0.04 mg/kg) can be given at initiation of CPR in cases in which the arrest rhythm is severe bradycardia, pulseless electrical activity or asystole (the majority of feline and canine CPA patients) and repeated every 3-5 minutes. Epinephrine (0.01 mg/kg) repeated every 3-5 min should be used also in case of asystole. Transthoracic defibrillation should be attempted if ventricular fibrillation/tachycardia is the primary arrest rhythm.

The last phase of CPCR consists of **post-resuscitation care**: protection of the heart and brain from the adverse effects of CPA, providing perfusion to vital organ systems, and addressing any underlying condition that caused CPA in the first place[1,2].

Respiratory function should be supported with supplemental oxygen and many patients will need ventilatory support.
Crystalloid or colloid IV fluid therapy should be administered cautiously to restore and then to maintain euvolaemia. It the animal remains hypotensive despite adequate intravascular volume then vasopressors may be required. In patients with post-resuscitation myocardial dysfunction may require a positive inotrope such as dobutamine. Arrhythmias should be treated if they are compromising the patient's haemodynamic status.
Neurologic dysfunction is common after CPCR because of cerebral edema secondary to decreased cerebral perfusion and cerebral hypoxia[2]. Many of these abnormalities might resolve with 1-2 days of return to spontaneous circulation (ROSC). Patients should be allowed a minimum of 48 hours before any judgement on their neurological status is made. Antiepileptic treatment should be administered to any post-CPA patient with seizures and interventions that may decrease intracranial hypertension secondary to brain oedema such as mannitol (0.5-2 gr/kg in 30-45 min) should be considered[2].
Cardiovascular, respiratory parameters and body temperature should be regularly evaluated. Urine output, electrolytes, blood glucose concentration, central venous pressure, electrocardiogram , blood pressure neurologic function, and patient comfort should be monitored.

**Criteria for terminating resuscitation**
Various objective criteria can be used to decide the appropriate time to discontinue resuscitative efforts. Typically the duration of resuscitative efforts is most commonly used and a figure of <20 minutes is suggested.

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## HYPOTHERMIA AND HYPERTHERMIA : WHEN THE BODY LOSES ITS ABILITY TO REGULATE THE TEMPERATURE

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Hypothermia and hyperthermia are relative common condition in the emergency and critical care settings, with significant deleterious cardiovascular, respiratory , neurological and metabolic effects. Evaluation of the body temperature in the initial assessment of the emergency patient will allow to recognize the condition and to initiate the most appropriate treatments.

**Thermoregulation[1,2,3,4]**
The hypothalamus, via thermoregulation, maintains an almost constant core body temperature (37.8-39 C) under varying environmental conditions through the balance of heat dissipation and heat production. The body is also able to retain the heat, by piloerection and peripheral vasoconstriction.
Heat is dissipated by 4 mechanisms:
- Conduction
- Convection
- Radiation
- Evaporation

It's the loss of heat from moisture on the body surface or through the respiratory tract. It's the most effective method of heat dissipation in small animals through panting.

**Hypothermia:**
Hypothermia is defined as a decrease in normal body temperature below 37 C. In mild hypothermia the body temperature is between 32 and 37 C; in moderate hypothermia between 28 and 32 C ; in severe hypothermia below 28 C [1,2]. Hypothermia can be the result of prolonged exposure to extreme cold environment or , more commonly in the emergency patient, the result of trauma, severe disease, surgery or administration of drugs.



As body temperature begins to fall, normal thermoregulation becomes impaired. When body temperature falls below 34 C , vasodilation and the decreased metabolic rate result in diminished heat production. At body temperatures below 31 C , thermoregulation ceases completely [2].

Physiologic effects of hypothermia:

*Cardiovascular effects:*
Mild hypothermia causes increase in catecholamine release, tachycardia and peripheral vasoconstriction that might lead to decrease tissue perfusion. Severe hypothermia decreases the responsiveness to catecholamines and baroreceptor responsiveness, which can result in bradycardia, hypotension, arrhythmias and decreased cardiac output[1,2].

*Respiratory effects:*
Severe hypothermia can lead to reductions in respiratory rate and tidal volume that can contribute to hypoxia and the development of pulmonary oedema[1,2].

*Neurological effects:*
Cerebral metabolism drops 7% for each degree Celsius in decrease of body temperature; this leads to decreased mentation, coma[1].

*Metabolic effects:*
Moderate to severe hypothermia can cause:
Initially increased poliuria ("cold diuresis"), azotaemia followed renal failure
Decrease hepatic function
Impairment of immunological function and increased risk for wound infection
Increased risk of bleeding and coagulopathy
Severe metabolic acidosis and decrease tissue perfusion

*Treatment of Hypothermia*
In mild hypothermia passive rewarming ( wrapping the patient in blanket ) is usually effective.
In moderate hypothermia or unstable mild hypothermia, active surface rewarming is used. The goal is to transfer heat to the patients, using techniques like heat lamp, warm water bottle, forced air warming blankets (i.e. Bair Huggers). Initial rewarming should be directed at the trunk and not at the extremities to avoid "rewarming shock" [1,2]. Patients should be actively rewarmed, while their hypoperfusion is aggressively addressed.
In severe hypothermia active core rewarming is indicated (warm IV fluids; warm water enemas; instillation of warm saline into the bladder; and heated, humidified, inhaled air)

**Hyperthermia:**
Hyperthermia is the elevation of body temperature above the accepted range for that species; when the heat is produced or stored in the body at a rate greater than it is lost, hyperthermia ensues [3].
Hyperthermia in the emergency patients can be secondary to increased muscular activity (working dogs, seizuring animals) or secondary to inadequate heat dissipation ( heatstroke (HS)) [3].

Heatstroke is a life-threatening condition. It is characterized by a temperature above 40C, central nervous system dysfunction and varying degrees of organ dysfunction[4].
Heatstroke is divided in
- Exertional HS: occurs late spring or early summer when dogs are not acclimatised to high environmental temperature and it occurs during working
- Non-exertional HS: when dogs are confined in overheated place or without water in a deprived shade environment

Clinical conditions such as brachycephalic anatomy, laryngeal paralysis, collapsing trachea, cardiovascular disease, obesity may predispose a dog to develop severe hyperthermia[3].

**Effects of severe hyperthermia:**

*Cardiovascular effects:*
Most animals present in hyperdinamic state of distributive shock: tachycardia, weak pulses, hyperaemic mucous membrane and fast capillary refill time[3,4]. Dysrhythmias might be present.

*Neurological effects:*
The majority of these animals present stuporous or comatose, because of poor cerebral perfusion, direct thermal cerebral injuries, CNS haemorrhage or metabolic abnormalities[3,4].

*Gastrointestinal effects:*
The elevated temperature and the poor perfusion causes the disruption of the gastrointestinal mucosa

with possible bacteria translocation. Vomiting, haemorrhagic diarrhea, gastric ulceration are common presentation[3,4].

*Coagulation effects:*
The release of systemic inflammatory mediators, associated to direct heat injury on the vascular endothelial cells, activate the coagulation cascade leading to consumption of coagulation factors, fibrinogen and platelet and the development of an haemorrhagic diathesis.

Initial evaluation of patients should include a minimal database (PCV/TS/BUN/Glu), but a full serum chemistry, complete blood count, urinalysis and assessment of the coagulation system should be performed.

**Treatment of severe hyperthermia**

Immediate cooling techniques should be implemented. Gradual but aggressive cooling with IV fluid therapy, spraying cool water, fans should be done and it should stop once the temperature of 39-39.5 C is reached[3,4].
Hypovolaemic shock is treated by aggressive intravenous fluid therapy. If large volume of crystalloid solution are unable to reverse hypoperfusion and hypotension colloid solution and the use of vasopressor ( dopamine 5-15 µg/kg/min) should be considered.
Oxygen should always be administered.
Dextrose should be supplemented IV (0.5-1ml of a 20% glucose solution diluted 1:2 with 0.9% NaCl) in cases of hypoglycaemia.
If the patient is in haemorrhagic diathesis, fresh frozen plasma (20ml/kg), or whole blood transfusions are indicated.
The GI tract should be protected with histamine$_2$ blocker, proton pump inhibitor, sucralfate, enteral nutrition if tolerated, and antiemetics.

If neurological deficits are severe and/or progressive despite the correction of perfusion deficit and hypoglycaemia , mannitol (0.5-2 gr/kg slow IV bolus over 20-30 minutes) and seizure control drugs are indicated.

Broad spectrum antibiotics are frequently administered to treat sepsis but their routine use in all cases is controversial due to the potential to induce resistance[4]. Systemic steroid and non steroidal anti-inflammatory drugs ( NSAIDs) should be avoided[4].

During hospitalization, cardiovascular, respiratory parameters and body temperature should be regularly evaluated. Urine output, electrolytes, blood glucose concentration, renal and liver function central venous pressure, electrocardiogram , blood pressure neurologic function, and patient comfort should be closely monitored.

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## APPROACH AND MANAGEMENT OF THE MULTITRAUMA PATIENTS

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Traumatic events, such as motor vehicle accidents, penetrating injuries, animal bites are common presentations in small animal emergency medicine.

**Initial Assessment**
Every trauma patient that is brought to your practice require proper and immediate triage to determine the urgency and severity of the problems[1]. Traumatic injuries will often affect more than one body system[2]. Common problems in multitrauma patients that require immediate attention and treatment include shock, respiratory distress, acute abdomen, brain and spinal trauma, bleeding[2]. It is important not to get distracted by less life-threatening abnormalities that have to be addressed in second instance, such as an open fracture, open wounds on



the limb, or proptosis of an eye. They will not affect the survival of the patient in the acute phase.

**Diagnostic testing**

In multitrauma, choosing and timing additional diagnostic examinations is difficult and controversial. With proper physical examination, the clinician should be able to initiate treatment and stabilise the animal. Further diagnostics such as radiographs of the thorax, ultrasound of the abdomen, CT of the head and spine, should only be considered in a stable animal.

An early obtained blood sample can be useful to detect early internal haemorrhage and electrolytes abnormalities. A low packed cell volume (PCV)and total solids (TS) is very suggestive of blood loss; however, the initial presence of a normal or elevated PCV with concurrent low TS also may be the consequence of acute bleeding with splenic contraction and release of sequestered red blood cells.

*Shock:*

Multitrauma patients often present in a state of generalised hypoperfusion and they can suffer from different types of shock[1,2,3]. Most patients in traumatic shock will be suffering from hypovolaemia secondary to external or internal haemorrhage or haemorrhage associated to long bone fractures and/or severe tissue trauma. Pain and severe injury to the spinal cord can contribute to the inability of the body to compensate to shock. Cardiogenic or arrhythmogenic shock may result as consequence of myocardial contusion and hypoxia. Septic shock may develop in later stages, because of wound infection or bacterial translocation from the gastrointestinal tract.

Therapy for hypovolaemia revolves around replacement of the intravascular volume deficit with intravascular fluid therapy and should never be delayed[3]. It is preferable to consider fluid administration in resuscitation from hypovolaemia as a fluid dose or bolus (and the dose will vary depending on the type of fluid being used) that needs to be administered over a short period of time (usually 15-20 minutes).

Different fluid types can be used in these patients[3]:

- isotonic crystalloid replacement solutions;
- hypertonic saline solution;
- colloid administration;
- blood products.

**Respiratory distress:**

Oxygen should always be administered in patients that are dyspnoeic. Multitrauma patients can suffer commonly from chest injuries (pulmonary contusions, pneumothorax and haemothorax ) or penetrating chest injury [2]. A careful examination of the respiratory system should help us identify the presence of these abnormalities.

Penetrating chest injury should be initially cleaned and covered. Pneumothorax and haemothorax that cause severe dyspnoea should be addressed immediately with a thoracocentesis. If pulmonary contusions are suspected we should be more careful in administering fluids in the resuscitation phase.

*Neurologic system:*

Signs of head injury[4]include bleeding or wounds involving the head, altered mentation(stupor or coma) associated to cranial nerve deficits and abnormalities in the pupil size. Scleral haemorrhage or blood in ear canals or from the nose could be signs of cranium fracture. When signs of head injury exist, then the first order of priority includes promoting cerebral blood flow by maintaining good tissue perfusion and mean arterial pressure between 80-100 mmHg. Hypertonic saline can be used as first line resuscitation fluid[4]; otherwise isotonic crystalloid or colloid can be used. In case of worsening mental status mannitol infusion can also be considered (0.5-1 g/kg IV). Care is taken to minimize increases in intracranial pressure [4]. The head and neck are kept in a slightly raised position (~30 degrees) with little manipulation and the jugular veins are not occluded. The use of steroid in head trauma is contraindicated!

If spinal injury is suspected the spine should be stabilised and supported on a blackboard and lateral radiograph of the spine performed before attempting to move the animal. As for the head trauma, maintaining good perfusion to the spinal cord is fundamental, so general perfusion and blood pressure should be maintained with fluid therapy. The role of corticosteroids in the treatment of acute spinal injury is becoming less controversial, as there is little evidence supporting improved outcome when corticosteroids are used and the potential risks of corticosteroid therapy (gastrointestinal ulceration) are considered to outweigh the potential benefits.

*Abdominal evaluation*

The abdomen should be palpated for painful response, focal lesions and the presence of a fluid thrill; the bladder should be identified. Frequent re-evaluation of the abdomen for progressive distension is important

A significant volume (40 ml/kg) of free peritoneal fluid is necessary for a fluid thrill to be detected and palpation with the patient standing is most likely to be successful[5]. If available, ultrasonography provides a much more sensitive and patient-friendly means of detecting peritoneal fluid[5].
Occasionally umbilical and peri-testicular skin discoloration may be observed when significant intra abdominal hemorrhage dissects through the abdominal muscle planes and subcutis.
If abdominal effusion is suspected abdominocentesis should be performed. The fluid collected should be evaluated and analysed for haematocrit, urea, creatinine potassium and cytology performed.
Traumatic haemoabdomen does not often require surgical management. During initial stabilization of the patient it is possible to apply an abdominal counterpressure bandage to help in stopping minor venous bleeding from parenchymatous organ laceration[5]. Uroabdomen should be considered a surgical emergency after the stabilization of the animal's major body system and electrolytes.

**Pain assessment**
Dogs that have suffered from trauma are usually in considerable pain. Pain should always be addressed during the initial stabilisation. Non steroidal anti-inflammatory drugs are contraindicated in animals that are in shock. A pure μ-opioid should be used in these patients.

*Additional Care*
All wounds should be covered to prevent desiccation followed by placement of some form of dressing (preferably sterile). Once the patient is stable the wounds should be clipped and cleaned and ALL penetrating wounds should be surgically explored.

Animal that do not stabilize after fluid resuscitation, pain management and abdominal counterpressure bandage should be suspected of ongoing haemorrhage and should be considered candidates for emergency surgical intervention.

*Monitoring:*
Careful monitoring of the physical examination parameters is the most reliable indicators of change in perfusion status. Evaluation of the respiratory pattern and neurological status should be frequently repeated. Further parameters that can provide information on perfusion status include arterial blood pressure, central venous pressure, oximetry (pulse and central venous), and urine output [1]. Recording and identifying trends of change illuminates the need for intervention.

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## PAIN MANAGEMENT IN THE CRITICALLY ILL PATIENT

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"The inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain relieving treatment" (International Association for the Study of Pain 1994).

Accurate recognition and effective management of pain are an extremely important part of our patient management and as clinician we have both an ethical reason towards our patients and a medical reason to address pain.

Pain is usually classified as physiologic (protective pain) or pathologic, acute or chronic. The type of pain most commonly recognized in an emergency setting is the pathologic and acute pain. Pathologic pain is a painful stimuli that occur when tissues have already been damaged. Acute pain is exemplified by traumatic or postoperative pain and it's often associated with inflammatory disease. Many diseases that require



emergency and critical care management are painful: trauma, organ distension, pleuritis and peritonitis, immobility and especially most of the diagnostic and therapeutic procedures that are often required ( IV catheter placement, position of an indwelling urine catheter, position of a chest drain etc) can be associated with a painful experience[1,2].

**Pain pathways:**
Nociception involves a series of electrochemical processes that start at the site of injury and that result in the perception of pain. Nociception consists of three distinct physiologic processes that are important to recognize as they can be targeted by analgesic drugs.

- *Transduction:* translation of physical energy (noxious stimuli) into electrical activity at the peripheral nociceptor.
- *Transmission:* propagation of nerve impulses through the nervous system.
- *Modulation*: amplification o inhibition of the stimuli processing within spinal dorsal horn cells.
- *Perception:* not considered part of the nociceptive process, results from integration of the noxious stimuli in the thalamocortical, reticular, and limbic region to produce the final conscious subjective and emotional experience of pain.

**Consequences of pain:**
Pain when left untreated, besides inducing unnecessary suffering, will also generate a stress response and catabolic state with resultant increased metabolic and energy demands[1]. Other negative effects of pain are delayed wound healing; reduced immune function; weight loss; lack of sleep; decreased mobility, with an increase in conditions associated with prolonged recumbency (e.g. urine and faecal retention, pneumonia, nosocomial infections).

**Pain assessment:**
Assessing pain is quite challenging[1,3]. The ability to assess and treat pain is influenced by knowledge of the specific animal's normal behaviour, of normal species behaviour and the clinician's observational skills and attitude toward pain[3]. It's important to remember that often, patients presented as emergency are too weak or obtunded to be able to respond to pain with dramatic behaviour[1,3].
Assessment of pain should include careful observation of behaviour, the evaluation of some clinical parameters and the use of pain scales.

There are numerous pain scales available but the only pain scale that has been validated for assessment of acute pain in dogs is the Glasgow Composite Measure Pain Scale (GCMPS) [4]. This multifactorial pain scale is able to assess pain in all its complexity ( intensity, sensory, motivational and behavioural aspects of pain) [4].

**Approach to pain management:**
Non pharmacologic approaches
Careful and compassionate approach of the injured animal, minimal restraint techniques are very important to limit further pain experience during medical procedures and physical examinations.

**Pharmacological approach:**
Drug therapy might be directed to one or more steps in the nociceptive pathway. Multiple drugs with synergic effects and targeting nociception at different steps, can be combined in a multimodal analgesic protocol[1].
Analgesic drugs available are: opioids, non-steroidal anti inflammatory drugs (NSAIDs). In addition local anaesthetics, ketamine, medetomidine, tramadol, have analgesic properties, which may be beneficial in a multimodal protocol.

**Opioids :**
Drugs of choice for moderate to severe pain; commonly used in critically ill patients because of potent analgesic effect , rapid onset of action, large safety and their reversal is possible[1,2].
Opioids have been classified as agonists, partial agonists, or agonist antagonists depending on the dose-response relation of the drug at the different opioid receptors.

**μ-receptor agonist:**
Gold standard analgesic for moderate to severe pain in dogs and in cats and their analgesic effect is more efficient as there is no ceiling limiting effect to the analgesia provided[1,2].

- *Morphine:*
  It confers sedation and optimal analgesia and they are both dose dependant, reliable in different clinical settings. Used at 0.1-0.4 mg/kg IV, SC. IM or as constant rate infusion (CRI) at 0.1-0.4 mg/kg/h. Rapid morhine intravenous administration has been associated to:
  - histamine release, so it should always be administered very slowly;
  - vomiting.

- *Methadone (0.1-0.4 mg/kg IV, IM, SC):*
  It acts very similar to morphine but it has been associated to less sedation and vomiting.
- *Fentanyl*
  It has a rapid onset ( 3 minutes) of action but a short duration ( 10 min) of its analgesic effects so it has limited use when used as a bolus (1-3 µg/kg), but it provides great analgesia when used a CRI (3-5 µg/kg/h)

Agonist-antagonist:

- *Butorphanol (0.1-0.4 mg/kg IV,SC,IM):*
  Minimal analgesic effect and of short duration (30 min) but adequate sedative effects.

Partial agonists:

- *Buprenoprhine:*
  Well recognized analgesic effect for mild to moderate pain. Long onset of action (45 min) but long duration of action ( 6 hours).

Different side effects have been described with the use mainly of pure µ-opioid ( respiratory depression, dysphoria, vomiting etc), but these are mainly recognized when an high dose is used in minimally painful animals.

**NSAIDs**

Non steroidal anti inflammatory drugs (NSAIDs) are potent analgesic, anti-pyretic and anti-inflammatory drugs. NSAIDs have a slow onset of action (up to 45 to 60 min), but they provide analgesia for an extended amount of time.

The most common types of NSAIDs available for use in dogs and cats are:

- *Carprofen*
  Licensed in dogs for acute and chronic use and in cats for a single pre-operative use of 4mg/kg.
- *Meloxicam*
  Licensed in dogs and cats for acute and chronic pain. Metacam at a lower dosage (0.05 mg meloxicam/kg body weight) can be used as a follow up dose in cats for acute pain to be administered once daily (at 24-hour intervals) for up to five days.

NSAIDs block the enzyme cyclo-oxygenase decreasing the production of prostaglandins. Prostaglandins are necessary to maintain gastro-intestinal and kidney perfusion in animals that are in shock, severely dehydrated, in patients with previous history of gastro-intestinal ulcerative disease[1,2]. For these reasons NSAIDs should never be used in unstable emergency patients as they might place these patients at severe risk of developing[1,2]:

- Gastrointestinal ulceration
- Acute renal failure
- Worsening bleeding because of decreased platelet function

**Other analgesic drugs:**

Ketamine is a competitive N-methyl-D-aspartate receptor antagonist. Ketamine causes minimal cardiovascular and respiratory depression. A small bolus of ketamine (0.3-0.5 mg/kg IV) , followed by low dose ketamine CRI (3-5 µg/kg/min) can be used for intra and post operative multimodal analgesia and at this dosage the dissociative and dysphoric effects are usually minimal [1].

Tramadol (2-4mg/kg IV, PO q 8 h)provide a mild opioid like analgesic effect. It's useful for the treatment of acute and chronic pain of moderate intensity associated with a variety of conditions

Medetomidine or dexmedetomidine administered as a low dose CRI (1-2 µg/kg/h) in stable patients are used to provide sedation, supplement analgesia, and reduce the stress response in agitated animals[1,2].

**Local anaesthetic**

Lidocaine, like other sodium channel–blocking drugs (bupivacaine, chirocaine) is usually classified as a local anaesthetic. Lidocaine has also been shown to have mild analgesic effects when administered as a low dose CRI (50 µg/kg/min) in a multimodal protocol [1].

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