Stabilization of the Critical Patient

Philip R Judge BVSc, MVS, MACVSc

Animal Emergency Centre, 37 Blackburn Rd, Mt Waverley, VIC 3149

Introduction

Critically ill patients often have multiple life-threatening problems, and can present a challenge, to even the most experienced clinician. Prioritization of treatment in these patients should be directed at the most immediate life-threatening problems, followed by a full body systems review to enable effective treatment to be directed at the underlying cause of the patients illness. When a traumatized, or critically ill patient arrives at the Emergency Centre, the patient is managed in most instances in a manner consistent with the following general principles.

1. Perform and Primary Survey and Triage Classification
2. Emergency Management of Airway
3. Restore Normal Pleural Pressure
4. Restore and Maintain Normal Ventilation
5. Provide Fluid Therapy to Improve Tissue Oxygenation and Cardiovascular Function
6. Provide Supportive Care for the Neurological System
7. Perform Full Body Systems Examination
8. Definitive Management of the Underlying Disorder
9. Supportive Management of Each Body System

- **Perform a primary survey** - a primary survey is a rapid clinical examination designed to assess the patient for the presence of any life-threatening abnormalities. The examination should take no longer than 1 minute to perform. Life threatening abnormalities in patients are most commonly associated with disorders of the airways (obstruction), breathing (airway obstruction, pleural space disease, pulmonary parenchymal disease, thoracic wall disease, and respiratory paralysis) cardiovascular system (shock - hemorrhagic, septic, hypovolemic) and neurological function (seizures, depression, coma, paralysis)

- **Perform triage** - triage is necessary when there is more than one patient presenting to the emergency centre at the same time. Accurate triage will ensure that the patients with the most severe injuries are seen first, while those with less severe injuries are seen at the next earliest opportunity. There are several triage systems in use in emergency rooms, An example is as follows:

    - ***Class I*** *- most urgent, these patients must receive treatment immediately, within seconds. Examples include traumatic respiratory failure, cardiorespiratory arrest, airway obstruction, and ALL unconscious animals*
    - ***Class II*** *- are those patients that require treatment within minutes. Examples include all patients suffering multiple injuries, shock, or bleeding, but have 'adequate' ventilatory function.*
    - ***Class III*** *- are those patients with serious injury requiring attention within an hour - these patients may have fractures, open wounds etc, but without active bleeding, shock, or altered mentation*

- ***Class IV*** *- are those patients that require attention within a few hours and include those patients that present several hours following trauma, with lameness, anorexia etc*

Management of Life-Threatening Abnormalities

Just as the primary survey, and triage classification are performed with systems oriented priorities, so is resuscitation. Airway disruption and blockage are the highest priority. Respiratory system difficulties not directly associated with airway obstruction are the next priority. Life-threatening cardiovascular emergencies are the third priority, and neurological function follows.

Airway Management Priority 1: Secure a Patent Airway

Management of Airway Obstruction

Etiology of airway obstruction

- Brachycephalic upper airway obstruction syndrome
- Pharyngeal trauma, basilar skull fractures, pharyngeal hematoma, or allergic reaction to an insect bite or sting resulting in pharyngeal edema
- Laryngeal edema
- Laryngeal paralysis
- Foreign body in pharynx, larynx, or major airway
- Neurological disorders (central or peripheral) may lead to loss of laryngeal tone and gag and swallow responses, resulting in airway obstruction
- Encroachment on proximal airway lumen by an extra-mural mass or foreign object
- Blood clots or mucus present in the larynx, trachea, main-stem bronchi. The source of bleeding can be the lungs (results in bubbles or foam seen in the trachea), trachea, larynx, or oral cavity. Aspiration of saliva, and/or gastric and esophageal contents may also result in airway obstruction. A liquid aspirate of about .25ml/kg with pH of 2.5 can produce a fatal obstructive bronchospasm, and acute chemical pneumonitis
- Direct trauma to the larynx can result in bronchospasm
- Trauma to airways
- Foreign body

Clinical signs of airway obstruction

- Prolonged inspiratory phase, inspiratory dyspnea. (Note: if obstruction is present in the thoracic trachea or bronchi, there is usually an expiratory component to the dyspnea) extended neck, lips drawn back (accessory muscles of respiration are activated)
- Cyanosis - **is a late and unreliable sign of airway obstruction**. The presence of cyanosis demands immediate action to secure the patient airway and restore ventilation Note: with complete airway obstruction, no breath sounds are heard on thoracic auscultation.

- **Partial obstruction may not give rise to clinical signs until over 75% of the airway is compromised**

Treatment of airway obstruction

- Provide supplemental oxygen therapy at all times while evaluating respiratory function, until it is confirmed that the patient does not require supplemental oxygen
- Gently extend the head and neck, pull the tongue forward, and clear the mouth of blood, mucus and vomitus
- Suction the larynx if required
- Intubation (laryngoscope preferred to minimize damage to the airway during intubation)
- Sedation/anesthesia for intubation – patients with airway obstruction are hypoxic and hypoxemic, and are EXTREMELY sensitive to the effects of anesthetic and sedative agents frequently used in veterinary medicine. In general, the safest anesthetic to use in the emergency is the anesthetic with which you are most familiar. However, some anesthetics are safer than others are. The authors preference is to sedate any patient in which intubation cannot be achieved without chemical restraint, using an intravenous bolus of diazepam at 0.1-0.3 mg/kg. If diazepam alone is insufficient to allow endotracheal intubation, addition of ketamine to effect (1-5 mg/kg IV), or fentanyl (1-4 mg/kg IV) are preferred agents
- If orotracheal intubation is not possible, perform an emergency tracheostomy

Procedure for Tracheostomy

- Make a ventral midline incision from the manubrium (anterior sternum) to the laryngeal cartilages
- Part the sternohyoid muscles on the midline by blunt dissection
- Continue blunt dissection down to the tracheal rings
- Blunt dissect around the circumference of the trachea, and elevate the trachea using artery forceps placed around the trachea
- Make an 'H' incision through the tracheal rings, or transverse incision between tracheal rings
- Place stay sutures through the tracheal rings - one on each side of the incision
- Insert the tracheostomy tube. The tube should be 2/3 to 3/4 the diameter of the trachea, and should have a high volume/low pressure cuff. Only inflate the cuff if positive pressure ventilation is required, or if it is necessary to prevent aspiration of oropharyngeal contents.
- Fasten the tube to the patient by tying it around the patients' neck with umbilical tape or gauze

Airway Management Priority 2: Restore Normal Intra-pleural Pressure

Pleural Space Disease - pneumothorax, tension pneumothorax, hemothorax and diaphragmatic hernia

Pleural space disease occurs commonly following catastrophic trauma. In addition, intrathoracic neoplasia, congestive heart failure, cardiac tamponade, and emphysematous bulla all lead to the presence of pleural space disease in non-traumatic patients. The presence of pleural space

disease decreases effective pulmonary reserve, and interferes with normal gas exchange and tissue perfusion and oxygenation. Animals suffering from pleural space disease appear anxious, and may have an exaggerated respiratory effort, frequently with prolonged expiration, or biphasic expiration with an abdominal grunt at the end of expiration.

Pneumothorax is the most common pleural space disease encountered in patients with multi-system trauma. A description of the approach to pneumothorax follows

Pneumothorax

Etiology of pneumothorax

- Trauma with rupture of alveoli secondary to increase in intra-thoracic pressure against a closed glottis
- Direct penetration of thoracic wall (sharp objects, rib fractures)
- Rupture of major airway. Note that major airway rupture will also cause pneumomediastinum

Pathophysiology of pneumothorax

- The pleural space is normally at sub-atmospheric pressure, with a small amount of fluid forming a cohesive bond between the lungs and parietal pleura. If air enters the pleural space, the cohesion is lost and the lungs collapse.
- The initial response of the patient is tachypnea, leading to decrease in blood carbon dioxide, and increasing blood pH. Hyperventilation increases the efficiency of gas exchange BUT it does increase patient energy needs, and compounds cellular hypoxia.
- As a pneumothorax becomes worse, compensatory mechanisms fail, and the patients develop hypercapnea, acidosis and death
- It is interesting to note that dogs and cats can increase the degree of chest wall expansion by 2.5-3.5 x normal during compromised pulmonary function

Definitions

- Open pneumothorax – A pneumothorax in the presence of an open chest wound
- Closed pneumothorax – A pneumothorax in the presence of an intact thoracic wall; tears in visceral pleura and pulmonary tissue result in pneumothorax
- Valvular pneumothorax – is a form of closed pneumothorax, in which air enters the pleural cavity chest during inspiration. This causes a tension pneumothorax. Causes include traumatic lung injury, emphysematous bulla rupture, lung granulomas, and lung cysts.
- Tension pneumothorax - results in a progressive increase in intra-pleural pressure, resulting in impaired chest expansion, and collapse of intra-pleural blood vessels, elimination of the thoracic pump of venous return, decreased cardiac output, and rapid patient decompensation and death

Clinical signs

Clinical signs of pneumothorax include some or all of the following

- Tachypnea
- Anxiety

- Restlessness
- Cyanosis
- Pale mucous membranes
- Mouth breathing
- Barrel-shaped thorax
- Inspiratory +/- expiratory effort increased

The approach to the patient with pneumothorax

- A respiratory rate of 45-60 breaths per minute or the presence of supportive clinical signs indicates thoracocentesis is required
- **Thoracocentesis - NOTE: be prepared to evacuate the entire pleural space at the first attempt at thoracocentesis.**

Thoracocentesis – The Procedure

Clip and prep intercostal spaces 5-11 on both sides of the chest, infiltrate lignocaine into intercostal space 6-7. Insert a 22 g needle attached to 3-way stopcock extension set and 10ml - 20ml syringe, into chest cavity, at the level of the rib 6-7 intercostal spaces. If air is suspected in the pleural cavity, insert the needle at the junction of upper and middle 1/3 of the rib cage; if fluid is suspected in the pleural space, the needle should be inserted at the junction of middle and lower 1/3 of the rib cage

ALWAYS, ALWAYS DRAIN BOTH SIDES of the chest cavity, due to compartmentalization of the thoracic cavity with mediastinal anatomy in dogs and cats. Drops of saline or lidocaine placed in hub of needle prior to introducing into chest cavity can serve as indicator of entering chest cavity, and the presence of a tension vs. a closed pneumothorax

- **When to Consider Placement of a Chest Drain**
  - Insert chest drain 10-20fr tube at the 7th intercostal space if indicated using the following criteria
  - If thoracocentesis is required more than 4 times during the first 4 hours of admission to hospital
  - Air +/- blood is being aspirated on repeated thoracocentesis attempts
  - Patients status is not significantly improving despite adequate oxygen therapy, fluid support, and the continued presence of active pleural space disease
  - Vacuum cannot be established using simple aspiration via thoracocentesis
- If pneumothorax is failing to resolve in 36 hrs, consider thoracotomy to evaluate problem

If the patient does not respond to therapy for pneumothorax, or continues to deteriorate despite therapy, a major air leak in a bronchus, or thoracic trachea is suspected; or thoracic or abdominal bleed (indicated by blood in thoracic or abdominal cavity, or failure of improvement in cardiovascular function despite appropriate fluid therapy). Further diagnostic evaluation, including thoracic and abdominal ultrasound, DPL, +/- radiography is warranted. In some instances, emergency surgery may be indicated to locate and rectify the problem so that effective respiration and cardiovascular performance can be established.

Airway Management Priority 3: Restore and Maintain Adequate Ventilation and Tidal Volume

- Administer oxygen with a mask, intranasal, or via endotracheal tube, or trans-tracheal (16-20g needle percutaneously into trachea if complete upper airway obstruction is present - prior to tracheostomy if required)
- Initiate positive pressure ventilation if indicated (See Table 1). It is important to note that oxygenation assessment using pulse oximetry can be misleading. Table 2 may be used as a guide to determine when ventilatory assistance is required. We consider ventilation in any patient that is receiving oxygen supplementation that has a reliable pulse oximetry reading of less than 90-94%.

Table 1. Indications for the Provision of Positive Pressure Ventilation (PPV)

Disorders of the Neuromuscular Junction	**Pulmonary Parenchymal Disease**
1. Tick Paralysis 2. Elapid Snake Envenomation 3. Botulism 4. Polyradiculoneuropathy 5. Myasthenia Gravis 6. Muscle relaxants	1. Pneumonia 2. PIE 3. Neoplasia 4. Pulmonary edema 5. Pulmonary interstitial disease
Central Nervous System Disease	**Hypoventilation**
1. CNS disease causing depression of respiratory drive a. Head trauma b. Neoplasia c. Drugs/medications d. Toxicity e. Seizures f. Infection/inflammation g. Cerebral edema, increased intracranial pressure	1. Shock a. Hypovolemic shock b. Hemorrhagic shock c. Septic shock d. Cardiogenic shock e. Non-cardiogenic shock 2. Pleural space disease 3. Sepsis 4. Mediastinal disease 5. Pain

Table 2. Interpretation of Pulse Oximetry Readings

SaO_2	**PaO_2**	**Interpretations**
> 95%	> 80%	Normal
< 89%	< 60%	Serious Hypoxemia
< 75%	< 40%	Lethal Hypoxemia

A Note about Pulmonary Contusions

Pulmonary contusions are detrimental to the patient because they impair the oxygenating ability of the lungs. Contusions result from a compression-decompression insult to the thoracic wall, and lead to direct pulmonary capillary disruption, and alveolar damage.

Pathophysiology

Pulmonary contusions result in intra, and extra pulmonary hemorrhage. Hemorrhage into the alveoli causes interference with the gas exchange unit. This causes hypoxia and increased ventilatory rate and effort which is mediated via chemoreceptors and the respiratory centre in the brain. Bronchospasm occurs due to pulmonary trauma and the presence of blood and mucus in the larger conducting airways. The combination of bronchospasm and fluid in airways reduces airflow within the larger airways and bronchioles. In addition, the presence of blood and cellular debris in the distal airways dilutes surfactant and results in flooding and collapse of alveoli. The net result is areas within the lung of low and no ventilation, ventilation-perfusion mismatching, and a reduced ability of the lungs to oxygenate blood. Concurrent traumatic injury to the myocardium, the presence of circulatory shock, and intra-pleural diseases (hemorrhage, effusion, pneumothorax, fractured ribs, diaphragmatic hernia, and flail chest) may also interfere with gas exchange and respiration. Within the lung tissue, a secondary inflammatory reaction occurs in response to extravasation of blood, concussive trauma to the lungs, and tissue hypoxia. This reaction is progressive over the first 24-48 hours of injury and further impairs the ability of the lungs to oxygenate effectively.

Treatment

The management of pulmonary contusions is based on the principles of improving tissue oxygenation, improving pulmonary function and gas exchange, and general supportive care.

- Fluid therapy, correction of shock – fluid therapy in the patient with pulmonary contusions has long been controversial due to the desire of clinicians not to "flood" the lungs with large quantities of intravenous fluid, which could potentially translocate into the pulmonary parenchyma and airways. To date, there are only limited numbers of studies that have evaluated the ideal fluid resuscitation plan in patients with pulmonary contusions. Two retrospective studies of clinical human patients found no correlation between the nature of fluid resuscitation and the severity of pulmonary lesions found in patients. Currently, there is very little evidence to make a strong recommendation regarding appropriate fluid therapy in patients with pulmonary contusions. However, an understanding of the pathophysiology of pulmonary contusions does justify a conservative approach. We currently recommend a carefully titrated fluid plan to achieve adequate cardiac output and tissue perfusion, while avoiding excessive venous pressures.
- Flow past oxygen, nasal oxygen
- Mechanical ventilation – a discussion of ventilation therapy is beyond the scope of this presentation, however, ventilation therapy using lung-protective ventilation techniques, +/- addition of positive end-expiratory pressure (PEEP) may be recommended in patients that remain hypoxemic despite oxygen therapy.
- Drainage of pleural fluid, stabilization of flail chest

Complications

Complications of pulmonary contusions may include pulmonary or systemic infection, the development of lobar cysts, lung lobe torsion, and spontaneous pneumothorax

For the remainder of this tutorial, we will concentrate on fluid dynamics during shock, and tissue oxygen delivery.

The Diagnosis and Treatment of Shock and Circulatory Dysfunction

Shock is a condition of severe hemodynamic and metabolic dysfunction, characterized by reduced tissue perfusion, impaired oxygen delivery, and inadequate cellular energy production.

Many common disorders lead to shock and circulatory dysfunction, including those associated with heart failure, hypovolemia, blood loss, blood flow maldistribution, thromboembolism, sepsis, hypoxia, heat stress, hypoglycemia, central nervous system diseases, and toxicity e.g. cyanide, sodium Monofluoroacetate etc.

The diagnosis of circulatory dysfunction has typically been based on the appearance of various clinical signs, including the following

1. Tachycardia, or bradycardia
2. Dry, clammy, pale, cold mucous membranes; mucous membranes may also be red and warm
3. Cyanosis – may be due to low oxygen saturation, or sluggish capillary blood flow
4. Slow capillary refill time - may be due to vasoconstriction, and reduced blood volume
5. Initial euphoria mediated by increased sympathetic tone, followed by mental depression due to tissue hypoxia and systemic hypotension
6. Rapid pulses, becoming weak (decreased cardiac output)
7. ECG changes include S-T segment slurring, ventricular premature depolarization or ventricular tachycardia, especially following blunt chest trauma. Sinus tachycardia progressing to bradycardia is a poor prognostic sign
8. Reduced urine output, reduced urine sodium, acute renal failure
9. Depressed liver blood flow is characterized by centrilobular necrosis, with leakage of liver cytosol enzymes, and increasing blood-clotting times
10. Blood clotting abnormalities
11. Changes in blood pressure – systolic blood pressure may be elevated, normal, or decreased in shock. Interpretation of blood pressure reading should always be made in concert with other clinical parameters of cardiovascular function and tissue perfusion.

The symptoms of shock outlined above, (tachycardia, hypotension, pale mucous membranes, circulatory collapse, dehydration etc) are easily recognized, but they are common to many other conditions. The underlying problem, or inciting event of all causes of shock is a decrease in effective blood flow and oxygen delivery to tissues, which does not meet the demand for oxygen of those tissues. **Simply stated, shock can be defined as an imbalance between oxygen delivery, and oxygen consumption, such that the delivery of oxygen does not meet the needs**

of the tissues. The difficulty in effectively treating circulatory dysfunction is that the presence or absence of symptoms of shock does not correlate well with oxygen delivery to tissues. How do we overcome this problem, and why is it important? To overcome this problem, and to arrive at a rational approach to the management of tissue oxygen delivery, rather than directing our treatment at the symptoms of shock, we must understand the determinants of tissue perfusion and tissue oxygen delivery. We must then arrive at an approach to fluid therapy that meets the demands of the tissue under any given circumstance.

Tissue Perfusion and Oxygen Delivery

Why are concentrating on tissue perfusion and oxygen delivery?

1. Adequate delivery of oxygen to tissues is essential to ensure adequate cellular energy production. Without adequate cellular energy production, cellular function will decline. Stated another way, oxygen is required in aerobic biochemical pathways to produce biological energy from energy substrates. Inadequate tissue oxygenation results in anaerobic metabolism. Anaerobic metabolism leads to reduced cellular energy production (ATP production), and an increase in lactic acid production from pyruvate. A reduction in ATP production leads to a reduction in activity of cell membrane pumps, loss of cell membrane integrity, osmotic stability, and cell lysis. In addition, tissue hypoxia and anaerobic metabolism leads to the development of acidosis, which further blunts normal metabolic pathways within cells and organs.

2. Although cardiac and respiratory functions are directly measurable, tissue perfusion and oxygenation are not quantifiable. However, tissue perfusion and oxygenation are of greater consequence in terms of outcome. Inadequate tissue perfusion with either low of high blood flow leads to tissue hypoxia, which, when extensive in degree or protracted time, produces organ dysfunction, multiple organ failure, and death. When the early manifestations of shock are alleviated by therapy that is insufficient to correct poor tissue oxygenation, the resultant oxygen debt may not be recognized until the appearance of organ failure, including ARDS, sepsis, acute cardiac failure, renal failure, hepatic failure, DIC or coma.

In summary, these two points state that the delivery of oxygen to tissues is required to ensure normal organ function and to prevent mortality. This statement underlies the focus of our treatment and stabilization of the critical patient.

Tissue oxygen delivery is dependant on five (5) factors. These factors and their relationships with each other are summarized below

Oxygen delivery (DO_2) = CO x [Hb] x SaO_2 x 1.3 + 0.03 x PaO_2
= Cardiac output x hemoglobin saturation + partial pressure of oxygen in the blood

From this equation, we can surmise that in order to have adequate tissue oxygenation, we must have a functional respiratory tract, cardiovascular system, appropriate vascular tone and adequate hemoglobin concentrations. These factors are considered below

1. **A functional respiratory tract and gas-exchange unit** (alveolus). The function of the respiratory tract is vital to ensure adequate tissue oxygen delivery to tissues throughout the

body. Continuous assessment of the ability of the patient to ventilate effectively is vital to patient survival. Emergency measures to ensure the patient is able to ventilate effectively have been discussed. Ongoing monitoring of respiratory function through analysis of blood gas analysis, pulse oximetry, and clinical observation and assessment are crucial to the success of management of the critical patient.

2. **Normal Ventilation - perfusion matching within the pulmonary tissue.** The lung may be considered as a three compartment model, with
    a. Ideal matching of ventilation and perfusion
    b. Dead space – where the alveolus is well ventilated but poorly perfused with blood
    c. Venous admixture – where the alveolus is poorly ventilated but well perfused with blood

    Any increase in lung pathology e.g. infiltrative lung disease, pneumonia, neoplasia, congestive heart failure etc, will increase the venous admixture – also called the "shunt fraction" within the pulmonary circulation, and will result in a reduction in oxygen content of the blood available for the systemic circulation. Critical care of the respiratory tract is usually directed at attempting to minimize the shunt fraction and dead space within the pulmonary circulation by means of oxygen supplementation, bronchodilatation, ventilation strategies, and management of any underlying pulmonary condition such as pneumonia or pulmonary hypertension etc.

3. **Adequate cardiac output - Cardiac output (CO)** is defined by the following equation

    Cardiac output = stroke volume x heart rate

    Stroke volume is further defined by the following equation

    Stroke volume = end diastolic volume - end systolic volume

    Improving end diastolic volume is achieved by improving the patients' volume loading. Improving (reducing) end systolic volume is achieved by improving contractility of the heart muscle through the use of positive inotropes. It is possible to increase cardiac output by as much as 50% by using fluid therapy and positive inotropes. Appropriate use of fluid therapy and positive inotropes is discussed below in "Effective Fluid Therapy".

4. **Appropriate vascular tone** - Critical illness results in alteration of blood vessel tone. Initially, the effects of the sympathetic nervous system and adrenal axis stimulation are observed, and result in vasoconstriction on the vascular beds of the gastrointestinal tract, liver, kidneys and skin. Vasoconstriction results in progressive decreases in oxygen delivery to these tissues, and results in cellular hypoxia, destruction of capillary beds, and activation of the coagulation system. This results in a subsequent reduction in vascular tone in these tissues and diversion of blood flow away from vital organs such as the heart, lungs and brain. In addition, many diseases resulting in critical illness including trauma, neoplasia inflammatory diseases etc., release vasoactive mediators into the systemic circulation that produce abnormal vascular tone throughout the systemic circulation. Appropriate vascular tone is achieved through provision of oxygen supplementation, appropriate and timely intravenous fluid support, and with the use of positive inotropes and vasopressors such as dobutamine and dopamine, following correction of intravenous fluid deficits**.**

5. **Adequate oxygen extraction from the blood by the tissues.** Adequate oxygen extraction from blood is dependant on four (4) factors.

a. Oxygen content of whole blood. Oxygen in blood is either bound to hemoglobin or

dissolved in plasma. The sum of both of these is called the oxygen content, or CaO_2. The CaO_2 is calculated using the following formula

$$CaO_2 = (1.3 \times Hb\ (g/dl) \times SaO_2) + (0.003 \times PaO_2)$$
$$= \text{Oxygen carried by Hb} + \text{oxygen dissolved in blood}$$

The hemoglobin content and percent saturation of hemoglobin by oxygen in arterial blood (SaO_2) are the most important variables for determining oxygenation of arterial blood. PaO_2 is the partial pressure of oxygen in arterial blood. PaO_2 becomes very important in blood oxygen content in circumstances when either blood hemoglobin levels are low, or when hemoglobin is not fully saturated, for example in smoke inhalation, acetaminophen toxicity etc. PaO_2 levels are useful for evaluating efficiency of gas exchange (diffusion of oxygen across the alveoli) in the lungs.

b. Oxygen delivery to the tissues. Tissue oxygen delivery is the *rate* of oxygen transport in arterial blood and is defined as the product of the cardiac output (Q) and the arterial oxygen content as described above (CaO_2). Cardiac output is defined as heart rate x stroke volume. **Therefore, tissue oxygen delivery is dependent on heart rate, stroke volume, and hemoglobin concentration**.

c. Oxygen uptake. Oxygen uptake represents the oxygen supplied to the tissue for tissue metabolism, and is represented by the equation below

$$VO2 = Q\ (\text{cardiac output}) \times (CaO_2 - CvO_2)$$
$$= \text{Cardiac output x arteriovenous difference in oxygen content}$$

d. Fractional extraction of oxygen from capillary blood. Under normal circumstances, oxygen uptake from the capillaries is equivalent to the metabolic rate or consumption of oxygen. However, if there is a defect in the ability of the tissue to extract oxygen, the VO_2 (oxygen uptake) will underestimate the actual rate of tissue metabolism. This situation is common in critically ill patients and is a well described phenomenon in sepsis, burns, and multiple trauma.

The Control of Oxygen Uptake

The rate of oxygen delivery to a tissue normally exceeds the rate of oxygen uptake by that tissue by a wide margin. This allows tissues to adjust to decreases in blood flow by increasing the oxygen extraction from the blood. The ability of capillary beds to adjust oxygen extraction in the face of decreasing blood flow is a feature of all capillary beds except those in the coronary and diaphragmatic capillary beds - these capillary beds extract the maximum amount of oxygen under normal circumstances, rendering these tissues very sensitive to even small changes in blood flow. The ability to adjust oxygen extraction however is impaired in critical illness.

Effective Fluid Therapy

Having considered the determinants of tissue oxygen delivery, a rational approach to fluid therapy can be made with the knowledge that

1. The patient requires a functional respiratory tract
2. The patient requires adequate cardiac output
3. The patient requires adequate hemoglobin concentrations

4. The patient requires appropriate vascular tone to ensure oxygenated blood is received by the tissues
5. The patient requires adequate blood flow through capillary beds to enable oxygenated blood to be extracted into the tissues

Fluid therapy in small animal practice is usually directed at correcting a maldistribution of blood flow, and the improvement of tissue perfusion so that tissue oxygen delivery can be optimized.

The three major causes of inadequate tissue perfusion in small animals are hypovolemia, sepsis, and inadequate cardiac function. Therapeutic objectives of fluid therapy are therefore correction of hypovolemia, and sepsis, and improving cardiac function.

Therapeutic objectives in the therapy of shock are outlined below in order of temporal priority. At all times, the goals of fluid therapy are the achievement of supra-normal values of cardiac output, and oxygen delivery to tissues.

1. Circulatory support begins by control of internal and external bleeding. Thereafter, the primary method of circulatory support is fluid therapy (except in cardiogenic shock). Intravenous fluid therapy is typically administered through a large bore venous catheter. Initially, a cephalic or saphenous catheter is used. However, if the patient is expected to remain hospitalized for longer than 24 hours, a jugular catheter may be placed at the earliest opportunity.

   Intravenous fluid therapy and circulatory support is aimed at achieving the following
   I. Immediate intravascular volume resuscitation
   II. Immediate restoration of normal blood hemoglobin concentration
   III. Immediate restoration of colloid oncotic pressure
   IV. Rehydration
   V. Maintenance of fluid balance

   Fluids available for resuscitation and support of the circulatory system include isotonic crystalloid solutions (Lactated Ringers Solution, PlasmaLyte A), hypertonic saline (administered as a 7-7.5% solution), and colloids (plasma, whole blood, dextran 70, pentaspan). Several comparisons between crystalloids and synthetic colloids have shown no difference in survival in human patients suffering from hypovolemic shock. However, colloids do provide superior intravascular volume support and may lead to a decrease in the production of pro-inflammatory cytokines. Interestingly, in experimental models of hemorrhagic shock, resuscitation with colloids and hypertonic saline has been shown to result in reduced oxygen tension and delivery to intestinal and hepatic tissues when compared to resuscitation with isotonic crystalloid fluids, either alone or in combination with dextran 70.
   Combinations of fluids appear to be the most effective method of providing fluid therapy, especially in early decompensatory (stage II) shock, end stage (stage III) shock, and shock secondary to dehydration and third space losses of fluids. The use of pentaspan or dextran 70 lowers the amount of isotonic crystalloid required by 40-60%.

   So, how much fluid are we going to administer to our patients? This depends largely on the clinical state of the patient, the type of fluid lost, and the presence of shock.

a. Fluid therapy for shock: Traditionally, it was suggested that one blood volume of isotonic crystalloid be administered by rapid intravenous infusion to the patient showing clinical signs of shock within the first hour of presentation. More recently, the practice of 'small volume resuscitation' with fluids has been advocated - that is, titrating the volume of fluid a patient receives, whether it be crystalloid, colloid, blood products, or a combination of these, to achieve a set of end-points. In the patient showing clinical signs of shock, these end-points include
    i. Normal mucous membrane color
    ii. Normal heart rate, normal respiratory rate
    iii. Return of normal pulse pressures
    iv. Central venous pressure of 5-10cm water
    v. Normal blood gas analysis
    vi. Establishment of normal or supra-normal urine output
b. Fluid therapy for rehydration – administer isotonic crystalloid solutions such as Hartman's solution to replace hydration deficits over 6-12 hours
c. Fluid therapy for hospital maintenance – is dictated by the clinical status of the patient. Typically, most critically ill patients require between 1.5 and 4 times their normal daily intake of fluids in order to cope with fluid losses resulting from their illness

2. **Maintenance of optimum hemoglobin concentration.** The "ideal" packed red cell mass in critically ill patients is 25-27%. This level of red cell mass provides adequate blood hemoglobin concentrations, while producing a reduction in blood viscosity. In humans, the incidence of thromboembolism in critical patients is lower when patients are mildly anemic. In critically ill animals, packed red blood cells or whole blood should be administered to maintain a hematocrit of approximately 27%. Transfusion to a higher hematocrit does not improve tissue oxygen delivery significantly. The rate of infusion of whole blood or packed red cells should not exceed 20ml/kg/hr unless the clinical state of the patient dictates a faster rate of infusion is required e.g. during exsanguination following arterial laceration. Blood products should not be administered concurrently with calcium-containing fluids as calcium may cause in-line clotting of the blood product.

3. **Maintenance of colloid oncotic pressure** may be achieved by using plasma products such as fresh frozen plasma, or by using synthetic colloids such as dextran 70 or pentaspan. Administration of synthetic or naturally occurring colloids aids in the maintenance of an effective colloid oncotic pressure within the blood vessel lumen, which, in turn influences circulating blood volume and blood flow, venous return, and cardiac output.

4. **Maintenance of cardiac output and tissue blood flow.** This is achieved through adequate intravascular volume resuscitation using crystalloids and colloids, and by the use of positive inotropic support after the maximum effect of intravenous fluid administration has been obtained. How do we know when the maximal effect of intravenous fluid therapy has been reached? In most emergency patients, we use an assessment of the presence or absence of the clinical signs of shock to determine if we have given sufficient fluid to a patient to restore normal tissue blood flow. In critical patients that have a jugular catheter in place, measurement of central venous pressure provides a useful index as to the relative "fullness" of the vascular system. Normal central venous pressure in the dog is between –2 and 2 cm water. In most critically ill patients, we aim to provide mild hyper-volemia, and a central venous pressure of +5-+10 cm water. Central venous pressure should also be interpreted in conjunction with mixed venous lactate concentrations. Lactate is a bi-product of the

anaerobic metabolism of pyruvate. Serial measurements of venous blood lactate can be used to assess a return of body tissues to aerobic metabolism – this provides a more accurate measure of the success of out fluid therapy in achieving cardiac output and tissue oxygenation. Regardless of the monitoring technique used, failure of the patient to show signs of improving tissue perfusion despite seemingly adequate amounts of intravenous fluid support indicate that the cardiovascular system requires assistance to improve cardiac output and blood vessel tone. The most effective drug therapy if poor cardiac output is suspected despite adequate fluid therapy is the use of the positive inotropic agent dobutamine. The starting dose is 2 μg/kg/min – this dose is titrated according to the patient status. Dobutamine produces a marked increase in cardiac output and stroke volume, as well as a decrease in systemic and pulmonary vascular resistance, and venous flow pressures. Hypotension can occur in patients that are inadequately volume resuscitated prior to commencement of therapy. If this occurs, the dobutamine infusion should be stopped, and the patient given a bolus of intravenous fluids. Dopamine also has positive inotropic properties as well as being a potent vasopressor. Administration of a vasopressor such as dopamine will produce greater increases in blood pressure than dobutamine; however, dopamine does not improve tissue oxygen delivery to the same extent as dobutamine. For this reason, dobutamine is preferred over dopamine in the therapy of shock and circulatory dysfunction.

How do we as clinicians decide when we have restored adequate cardiac output, tissue perfusion, and oxygen delivery? Without the use of pulmonary arterial catheters as are used in human medicine, we as veterinarians rely on measurements of clinical parameters such as heart rate, respiratory rate, neurological function, blood lactate levels, blood gas analysis, urine output and central venous and arterial blood pressure.

5. **Maintenance of pulmonary function and adequate gas exchange** involves the provision of oxygen supplementation by nasal catheter or oxygen-enriched air. Ensuring the patient has an optimal hemoglobin level is also critical in ensuring adequacy of gas exchange in the lungs. Mechanical ventilation is indicated in those patients where oxygen supplementation fails to increase SpO_2 above 90-95%, or in patients where excessive work of breathing is present. Strenuous use of the accessory muscles of respiration can increase oxygen consumption by 50-100%, and decrease cerebral blood flow by as much as 50%. In addition, any increase in the work of breathing creates a greater negative pressure within the thorax during inspiration, resulting in an increase in impedance to ventricular ejection. Ventilation of these patients is critical in reducing oxygen demand and improving cardiac output – it could make the difference between life and death.

6. **Maintenance of adequate mean arterial blood pressure**. Hypotension is defined as a mean arterial pressure below 70 mm Hg, and diastolic pressures less than 50 mm Hg. How do we treat hypotension in the critical patient? The short answer is to administer intravenous fluid therapy until the patient is volume replete, and to administer a vasopressor if hypotension persists in the face of adequate volume resuscitation. However, alpha-adrenergic vasopressors must be used with caution because they may intensify the uneven vasoconstriction produced by neural mechanisms, sepsis and critical illness. Vasoconstriction produced by vasopressors does raise blood pressure, but may further exacerbate the uneven microcirculatory flow present in patients with shock and circulatory dysfunction. The effect of vasopressors such as dopamine isoproterenol, and epinephrine, because they also have inotropic actions that improve cardiac performance, is a balance between favorable increase in blood pressure, and unfavorable uneven maldistribution of

blood flow. If the decision is taken to use a vasopressor, the smallest dose needed to maintain satisfactory blood pressure should be used, because no amount of vasopressor can make up for inadequate blood volume. Dopamine is used at a starting dose of 1-3 μg/kg/min.

7. **Maintenance of cardiac rhythm and the synergy of cardiac conduction and contraction**. Cardiac dysrrhythmias are common in emergency and critically ill patients. Cardiac rhythm may be abnormal in patients due to a wide variety of causes. These may include the presence of cardiac disease, myocardial contusions, hypovolemia, pain, electrolyte and acid-base balance abnormalities, systemic circulation of mediators of inflammation and infectious organisms. In all cases, searching for and managing the underlying disease process is the most effective means of managing the abnormal rhythm. Many anti-arrhythmic drugs have toxic or undesirable side effects if they are administered inappropriately to patients. Prior to starting anti-arrhythmic therapy, it is therefore recommended that all patients have normal intravascular volume and hydration, electrolyte and acid-base status, analgesia, and adequate management of the underlying disease process (e.g. sepsis, infection, heart failure etc.). In addition, it is wise to document that the abnormal cardiac rhythm is causing hemodynamic compromise to the patient prior to starting anti-arrhythmic therapy, so that an assessment of the effectiveness of therapy can be made using clinical parameters as well as ECG parameters.

8. **Maintenance of adequate urine volume** is achieved through management of hypovolemia and maldistribution of blood flow as outlined above. Oliguria or anuria are managed by the addition of furosemide at 2-4 mg/kg IV, mannitol at 0.5 – 1.0 gm/kg IV over 10 minutes, and dopamine at 1-3 μg/kg/min IV. The goal is urine output of 2-4 ml/kg/hr.

9. **Body temperature control** is achieved through normal tissue perfusion, the provision of warm humidified air, and the warming of intravenous fluids. The goal is a normal rectal temperature of 38.0-39.2^{0}C.

10. **Manage sepsis** through ensuring adequate tissue perfusion and tissue oxygen delivery as outlined above. Selection of antibiotics should be based on culture and sensitivity from isolated organisms.

11. **Maintain normal blood glucose, electrolyte, and acid-base balance –** electrolyte balance is essential to ensure normal tissue metabolism, cell function, normal cardiac rhythm and vascular tone. Supplementation of intravenous fluids with electrolytes such as potassium magnesium, and glucose is usually based on measurement of serum levels. However, because most body potassium and magnesium is located within the intracellular space, serum measurements poorly reflect total body levels. Supplementation of potassium and magnesium may be based on expected urinary losses, or based on urinary electrolyte measurement.

The Patient That Does Not Stabilize – What to Do?

Failure of a patient to show clinical signs of improvement following adequate intravenous fluid therapy, or stabilization of the patients clinical signs for only a short period of time indicates the presence of one or more of the following, and warrants immediate attention

- Greater than 40% blood loss
- Undetected ongoing hemorrhage – e.g. into fracture sites, pleural cavity, abdominal cavity, fascial planes etc
- Pneumothorax/hemothorax
- Aspiration pneumonia
- Pericardial effusion
- Cardiomyopathy
- Cardiac dysrrhythmias
- Hypothermia
- Acidosis
- Hypocalcemia
- Myocardial contusion
- Severe Sepsis
- Hypoglycemia

The clinician should immediately mount a systematic search for the cause of the poor response to therapy. Use a systematic body-systems approach, beginning with the respiratory system, cardiovascular system, neurological system, urinary, gastrointestinal, hematologic and musculoskeletal systems, in accordance with the patients clinical signs and underlying disease.

Catastrophic hemorrhage is an immediate life-threatening abnormality and results in vascular collapse, decreased oxygen delivery to the tissues, and loss of blood into an anatomical area where space occupation by blood causes secondary cardiovascular or neurological malfunction (e.g. cardiac tamponade, intracranial bleeding). Cardiovascular collapse from exsanguination hemorrhage results in insufficient blood flow to the brain, and profound vasodilatation from persistent hypoxemia and hypercapnea, decreased cellular energy production, and metabolic acidosis. Animals with catastrophic hemorrhage may rapidly develop hypotension, bradycardia, coma, and death.

The compensatory response to catastrophic hemorrhage depends on the rate of bleeding. Rapid hemorrhage in a short period of time leads to a 'blunting' of the normal compensatory response. The normal response to hemorrhage is a centrally mediated sympathetic nervous system stimulation of contraction of venules and splenic vessels, allowing 'mobilization' of red blood cells pooled in these areas. This response is depressed with rapid hemorrhage due to hypoxia of the respiratory and vasomotor centers in the brain. In addition, species differences in splenic capacity also impact on the extent of the compensatory response to catastrophic hemorrhage. Dogs are able to store up to 10-20 ml/kg of blood in the spleen, vs. 5 ml/kg in the cat.

Irrespective of the degree of compensation present, ongoing hemorrhage in traumatized patients will manifest itself in the following manner:

- Progressive delay in capillary refill time
- Increased heart rate and respiratory rate (early hemorrhage)
- Decreased heart rate and respiratory rate (late hemorrhage)
- Apprehension, fright
- Progressive decrease in body temperature
- Progressive decrease in patient mentation
- Severe abdominal pain if hemorrhage is occurring into the peritoneal cavity
- Dyspnea and respiratory distress with both intrapleural of intra-abdominal hemorrhage.

A clinically useful rule of thumb in patients with severe trauma is as follows

"If hemorrhage is unapparent in animals presented following a history of recent trauma, it should be assumed that these animals have serious ongoing internal hemorrhage until proven otherwise"

Obviously, external hemorrhage is easily diagnosed. However, internal hemorrhage is hidden from sight and may occur within the thorax, peritoneum, retro-peritoneum, osseofascial compartments of the cervical area, or at fracture sites. In traumatized patients manifesting shock without evidence of severe external hemorrhage, these areas must be investigated for evidence of blood accumulation.

The management of catastrophic hemorrhage and the shock syndrome that accompanies it is outlined below using four basic principles

- **Volume resuscitation** - using blood, plasma, synthetic colloids, and hypertonic or isotonic crystalloids. The volume of fluid administered will vary depending on the individual patient requirements. Most authors currently recommend low volume resuscitation with a combination of blood products, synthetic colloids, and crystalloid solutions in order to reduce the chances of further bleeding from these patients, as their blood pressure increases following fluid therapy. Following definitive control of hemorrhage, patients are resuscitated to a normo or slightly hypertensive state. In patients with severe blood loss, restoration of intravascular blood volume is ideally obtained with whole blood transfusions, auto-transfusion of pooled thoracic or abdominal blood, or packed red blood cells and plasma/synthetic colloid.
- **Rapid surgical exploration of the thorax, abdomen, or limbs** - and internal control of hemorrhage by occlusion of the arterial blood supply leading to the site of hemorrhage
- **Identification, ligation/repair of bleeding vessels**. A brief outline of a suggested approach to the patient with an acute hemabdomen is presented below

Surgery of the Patient with Acute Hemabdomen

A thorough and systematic approach to exploration of the abdominal cavity should be performed. Techniques of various surgeons vary - the following is a guide

- Uncontrollable arterial bleeding can temporarily be stopped by compressing the aorta cranial to the celiac artery. During suctioning, the entire abdominal cavity should be packed with surgical towels or laparotomy pads. This will control venous hemorrhage.
- The towels or pads are removed one at a time until the source of the bleeding is located. Once located, the source can be ligated, or affected organ, or segment of affected organ removed. It is best to preserve as much of a bleeding organ as possible unless it is severely injured, is infected, or potentially neoplastic
- Once hemorrhage is controlled, each quadrant of the abdomen is carefully examined. Tissues found to be injured should be isolated with moist laparotomy pads prior to definitive surgical repair.

- Tissue and fluid samples should be obtained for both aerobic and anaerobic culture and sensitivity, and biopsies taken from liver, pancreas, kidney, stomach, small intestine, mesenteric lymph node, and abdominal muscle as indicated by the patients condition
- Once surgery is complete, lavage the abdomen with copious amounts of warmed 0.9% NaCl. Ensure complete suctioning of lavage fluid
- Placement of a jejunostomy tube, or gastrotomy tube to allow post operative feeding if a prolonged convalescence is anticipated, or if the patient has sepsis, or was malnourished prior to presentation

Neurological Assessment

Following stabilization of airway, respiratory function, and cardiovascular function, a complete neurological assessment of the patient should be carried out, and a coma score evaluation completed. Particular attention should be given to the patient's level of consciousness, ocular responses, and ability to effectively guard its airway and prevent aspiration of gastric, esophageal, and oral secretions. In addition, a complete evaluation of spinal reflexes, presence of superficial, and deep pain, anal tone, and bladder function should be carried out and repeated if results are inconclusive at presentation. Subsequent neurological assessment should be scheduled every 6-12 hours.

1. **Patients that present with seizures and status epilepticus** – patients that are in status epilepticus present a unique challenge to the emergency clinician. A protocol for management of seizures is included (Appendix A).
2. **Patients that present with Stupor and Coma –** are managed in accordance with the protocol in Appendix B.

Supportive Care of the Critically Ill or Traumatized Patient

During and following stabilization of the critically ill patient, the patient must be adequately supported to ensure recovery. In many instances of illness and severe trauma, pro- and anti-inflammatory cytokines, vasoactive mediators of inflammation, and infectious organisms will impact on patient recovery several days following the initial trauma. These mediators of systemic

inflammation and sepsis cause maldistribution of blood flow, arteriovenous shunting of blood flow through organs such as the gastrointestinal tract; increased capillary permeability, and third space loss of intravascular fluid. The net result is decreased tissue blood flow and tissue oxygen delivery to the tissues, resulting in organ dysfunction and eventually organ failure. Every effort should be made to ensure that tissue oxygen delivery remains adequate through maintaining adequate blood pressure, central venous pressure, heart rate and rhythm, urine output, control of infection, and maintenance of colloid oncotic pressure and hemoglobin concentrations.

General nursing care, including pain management, prevention of aspiration pneumonia, decubital ulcer prevention, and management of intestinal ileus and gastroparesis is critical in the management of these patients.

Early provision of nutritional support in critically ill patients has been shown to reduce mortality and morbidity, infection rates, and hospital stays in numerous human studies. There are currently a large number of human and veterinary products available for enteral nutrition in critically ill patients.