Successful management of the post-operative, emergency and critically ill patient is dependent on several factors; such as the training and skills of personnel and the readiness of facility. The implementation of a team approach through the use of established policies, protocols and procedures allows for coordinated resuscitative efforts. The survival of the patient is also dependent on the collaborative efforts of the veterinary surgeon and veterinary nurse. The success of the team is not only dependent on the knowledge base and skill level of each member, but also on the consistency and repetition of their practice as a team. Thus, the nurse or technician should be skilled in patient monitoring, based on clinical and physiological parameters and knowledgeable at utilizing different types of monitoring.

**Golden Rule of Recovery**
Successful recovery from surgery depends largely on the quality of care delivered during this phase. The time required for recovery will vary with the type of surgical procedure performed, pre-operative status of patient, as well as anaesthetic agents used. During this period protective reflexes and homeostatic responses which control respiration and circulation return gradually. The veterinary nurse (VN) must continue to provide the same quality care as during the operative period, until the patient is conscious and their vital signs are stable. Above all, the patient should never be left unattended.

**Three Pillars of Critical Care**
Interventions, monitoring and assessments in the post-op patient are geared towards supporting and/or enhancing 3 main processes:

A: Improving and facilitating perfusion/rhealogy
B. Enhancing and facilitating O2 delivery and consumption.
C. Improving and facilitating adequate ventilation (CO2)

In critical care we are mostly concern with the adequacy of intravascular volume/fluids and their flow throughout the circulatory system. A surgical and post-operative patient is likely to experience hypovolemic shock due to intra or post-operative blood loss (hemorrhage).

**CEPSAF**
The Confidential Enquiry into Perioperative Small Animal Fatalities (CEPSAF; Brodbelt and et al, 2006) examined perioperative small animal fatalities. The results revealed that the postoperative period was a particularly common time for dogs, cats and rabbits to die. Over 60% of cats and rabbits, and nearly 50% of dogs died during this time period and of these postoperative deaths approximately half of the patients died within 3 hours of termination of the procedure. Cardiovascular and respiratory causes of death were the most common causes of death in dogs and cats with a substantial majority being of unknown cause. The results of the study suggest that
Improved monitoring of recovering patients could reduce the number of fatalities in this group of patients. This lecture will look at the most important aspects of post-operative care which are based around frequent patient monitoring and anticipation of patient needs, this includes frequent and accurate patient assessment, which will allow identification of arising problems and help guide therapy and also the use of monitoring equipment.

**Areas of assessment**
The veterinary nurse (VN) must continue to provide the same quality care as during the operative period, until the patient is conscious and his vital signs are stable. The VN will need to observe and assess the following:

- LOC
- Airway and Breathing
- Circulation and Perfusion
- Temperature control
- Renal Function
- Gastrointestinal Function
- Fluid and Electrolyte Balance
- Pain Management

**Neurological Status**
LEVEL OF CONSCIOUSNESS: When a patient recovers from anesthesia, reflexes return in the reverse order in which they disappeared: unconsciousness, response to stimuli, drowsiness, disorientation, and orientation. The most common cause for persistent somnolence is residual effects of anaesthetics, sedatives, and pre-operative medications. VNs should learn about the various anaesthetic agents most commonly used in the work-place and their recovery characteristics.

Hopefully, patients recovering from anesthesia have an IV catheter. It is important to remember that during a crisis such as a CPA, CPR drugs (Naloxone, Atropine, Vasopresin, Epinephrine and Lidocaine) can be delivered down the endotracheal tube. The drug dosage needs to be doubled when using the ET tube as the administration route.

The neurological status of a critical patient should be assessed in conjunction with the other body systems. A patient’s mental status and gait should be noted, and any variation from ambulatory, bright, alert and responsive, will require further investigation. Patients may have many variations in mental status from dullness and depression, to hyperexcitability and seizures. Central nervous system depression may be a result of primary CNS disease, or as a complication from metabolic disorders. Hypoglycaemic or patients with renal or hepatic compromise may display depressed mentation.

**Loss of Consciousness**
In most cases, some level of responsiveness should be achieved within 90 minutes following completion of the anaesthesia. The patient that does not regain
consciousness following general anaesthesia must be carefully evaluated. Preoperative factors such as drugs or toxins should be sought.

**Causes of Reduced Responsiveness**

Once pharmacological etiologies are ruled out, metabolic and structural etiologies are considered. Profound hypothermia can produce unconsciousness, as can profound abnormalities of serum glucose such as hyperglycemia or hypoglycemia. Blood glucose, electrolytes and blood gases should be evaluated. If the diagnosis remains unclear, neurologic abnormality should be sought. Increased intracranial pressure or intracranial hemorrhage can occur following head trauma or neurosurgery. Intraoperative hypoxia from hypoxaemia or poor cerebral perfusion can produce a diffuse encephalopathy.

**Airway and Breathing**

AIRWAY and BREATHING: Certain anaesthetic agents may continue to cause respiratory depression. The VN should observe for signs of shallow, slow breathing. The nurse should assess respiratory rate, rhythm, depth of ventilation, symmetry of chest wall movement, breath sounds and color of mucous membranes.

Most patients are admitted to the recovery area with an oral airway or endotracheal (ET) tube in place to maintain a patent airway until effective and comfortable breathing resumes at a normal rate. As respiratory function returns, the VN will observe for a swallowing or gag reflex in the patient, thus denoting the patient’s ability to guard its airway. The nurse can then proceed to extubate the patient.

To assess airway patency the VN should place his/her hand over the patient nose (if extubated) or near the open end of the endotracheal tube. In the unconscious patient the airway should be visualized utilizing a laryngoscope. When the patient is in lateral recumbence, the neck should be maintained extended to prevent occlusion of the airway at the pharynx.

The nurse should suction the artificial airway and the oral cavity for mucus secretions. Care must be taken to avoid continually eliciting the gag reflex, which might cause vomiting. Before an artificial airway is removed, the VN should suction the back of the oral cavity so that mucus plugs or secretions are not retained. The VN should also feel the amount of air exchange that occurs with each breath the patient takes. Check bilaterally for rise and fall of the chest wall. Do not assume that because the chest is moving, the patient is breathing effectively. Bilateral chest expansion is a backup observation that reveals depth and equality of muscle exertion. Auscultate the lungs. The VN should hear breath sounds bilaterally. If breath sounds are absent on one side, the patient’s ET tube may have become displaced and lodged in either main bronchus.
Airway Obstruction

The major respiratory complications encountered in the postoperative recovery period are airway obstructions, hypoxaemia, hypercapnea and aspiration. The most common cause of postoperative airway obstruction is pharyngeal obstruction from a sagging tongue or mucus plug in the unconscious patient. Laryngeal obstruction can also occur secondary to laryngeal spasm, oedema or due to direct injury to the airway.

In the conscious patient, if the air passage is obstructed, the patient may show signs of distress with unsuccessful attempts to ventilate. Upper airway obstructions are generally accompanied by loud breathing sounds. The conscious patient that is “oxygen starving” will require sedation in order to establish an airway.

If the patient is unconscious and has no ET tube, open the airway, pull the tongue out, and extend the neck. Look, listen, and feel for breathing, then suction the oropharynx and reassess breathing. Postoperative mucus secretions are another common cause of obstruction. Never leave your patient alone. Have someone else get help, or call for help as loud as you can. If attempts to dislodge the obstruction are unsuccessful, the patient will require basic life-support measures until help arrives. The time to know how much and what is required of one as a nurse is before a disaster occurs. The VN should ask that the veterinarian review with them the guidelines and procedures to follow in such situations.

Depending on the severity of the problem, re-intubation may be necessary. If an oral airway is in place, check its position and suction the patient’s oropharynx; then check respirations. When caring for a patient with an ET tube, suction the ET tube and attempt to ventilate with an Ambu bag.

Shallow breathing should warrant the nurse to assess for signs of cyanosis. Hypoventilation will cause mucous membranes to exhibit blue (cyanotic) color. The nurse should provide the patient with 100% oxygen via high flow. Hypoventilation is defined as reduced alveolar ventilation resulting in an increase in arterial carbon dioxide tension.

During the postoperative period, hypoventilation occurs as a result of poor respiratory drive, poor respiratory muscle function, a high production rate of carbon dioxide, or as the direct result of acute or chronic lung disease. Respiratory depression or poor drive is mainly due to anaesthetic agents such as narcotics. Narcotic-induced respiratory depression can be reversed by use of a narcotic antagonist. An inadequate central respiratory drive can also be seen after certain neurosurgical procedures such as cervical hemilaminectomies.

Poor respiratory muscle function occurs following surgery, sometimes due to the site of the incision affecting the ability of the patient to take large breaths. Failure to reverse neuromuscular blockades can also contribute to inadequate respiratory muscle function. Obesity, gastric dilatation, tight dressings or thoracic casts also inhibit respiratory muscle function and can predispose to carbon dioxide retention.
A high level of carbon dioxide production from sepsis or shivering can result in carbon dioxide retention, especially if the patient cannot increase minute ventilation.

Aspiration involves the inhalation of gastric contents, food, liquids, or other foreign material into the tracheobronchial tree. Postoperatively, this usually results from regurgitation of stomach contents in a heavily sedated patient or a patient with an absent gag reflex. Aspiration of large quantities of stomach contents results in chemical irritation from the extreme acidity in the content. Post anesthesia aspiration may not be noticeable because of the sedated patient’s inability to cough or protect its airway.

Regurgitation, unlike vomiting, is a passive process indicated by gurgling in the throat and signs of airway obstruction. If the VN should notice the patient is regurgitating, the following measures should be taken:

Turn the patient’s head to one side and suction the mouth and oropharynx.
Take necessary steps to clear the airway. If the patient regurgitates a large amount, the VN may have to tilt the patient’s head down to allow the fluid to exit (via gravity) and then proceed to suction the mouth and oropharynx with the blunt end of the suction tubing or a large Yankauer suction tip.
Place the head in a dependent position, if possible.
Notify the veterinarian immediately.
Medical treatment involves chest radiographs, possible intubation, arterial blood gases, and placement of a nasogastric (NG) tube for gastric decompression. Massive aspiration usually does not occur in patients who have received nothing by mouth (NPO status) several hours before surgery. It occurs more often in patients who have undergone emergency surgery and in those who experience some type of gastrointestinal pathology.

O2 supplementation
Oxygen supplementation increases the O2 content of blood, increases the partial pressure of oxygen (PO2) in the capillary blood, and improves tissue delivery of O2. In addition to improving tissue oxygenation, the administration of O2 may improve the function of O2-dependent cellular systems, such as cytochrome P450 system, which is important to drug and toxin metabolism; nitric oxidase synthase, which regulates vasodilatation; and host defense systems. Improved tissue oxygenation is also beneficial for wound healing.

FiO2 concerns
A patient breathing room air, with PO2 values of less than 80 mm Hg is indicative of the potential for hypoxemia. If the PO2 decreases to < 60 mm Hg, supplemental oxygen is indicated. A number of devices are available for the delivery O2. The method chosen should be based on the desired FiO2, equipment availability, and anticipated treatment duration as well as the patient’s clinical condition, size and temperament.
The appropriate O2 flow is based on patient size, respiratory rate/pattern, and degree of open-mouth breathing. In general, a higher FiO2 is achieved in patients with tachypnea and low tidal volumes than in those with patients with a normal respiratory rate at the same flow rate. Each litre per minute increase in O2 flow raises the FiO2 by 3% to 4%.

Assess patient /tube
Animals requiring oxygen therapy or under anesthesia should have their SaO2 monitored as well as monitoring for physical signs of hypoxia (e.g. decreased LOC, tachycardia, arrhythmias, restlessness, WOB (Work Of Breathing) altered blood pressure, increased respiratory rate and changes in mucous membrane colour.

Assess efficacy
Pulse oximetry is a quick and reliable non-invasive method of measuring arterial oxygen saturation (SaO2). Oxygen saturation is the percentage of hemoglobin sites that are chemically combined with oxygen. Oxygen saturation and pulse rate are determined by passing two wavelengths of light, one red and one infrared, through body tissue to a photo detector. The signal strength resulting from each light source determines the SaO2. Pulse oximetry can be affected by the color and thickness of body tissues, the probe placement, the intensity of the light source, and the absorption of the arterial and venous blood in the body tissue.

There are several types of probes that can be placed. The probes that are clamps can be placed on the tongue or on a shaved, non-pigmented skin surface. The rectal probe is placed against the rectal mucosa, which has been cleared of faeces. The oximeter is turned on and the SaO2 and pulse rate is digitally reported. Any sudden decrease in the oxygen saturation with proper probe placement requires immediate notification of the veterinarian and rapid assessment of the animal's cardiopulmonary function. The oxygen concentration may need to be increased or the method of ventilation improved.

Limitations
Limitations of pulse oximetry include its inability to differentiate carboxyhaemoglobin (seen with carbon monoxide poisoning) from haemoglobin. The pulse oximeter cannot distinguish a declining PaO2 that is above 100mmHg (e.g. a fall from 330 mmHg to100 mmHg will still report a SaO2 of 100%) or the presence of methaemoglobin. Results can be erroneous in animals with poor peripheral perfusion, heavily pigmented skin, hypothermia, icterus and anemia.

Hypoxaemia
The use of pulse oximetry for monitoring oxygen saturation and pulse rate can provide early warning of pulmonary or cardiovascular deterioration before it is clinically apparent. Normal SaO2 is 98%. Values below 90% are correlated with PaO2 <60 mmHg and cyanosis is eminent. It is of most value when the arterial oxygen saturation is between 90% and 95%. The accuracy of pulse oximetry becomes unreliable at values below 85%.
Assess Patient/Tube
Desaturation following re-intubation warrants the repositioning of the ET tube. The patient in the slide was presented with respiratory distress and loud upper airway sounds. The patient was stabilized (SaO2: 98%) and x-rays were taken revealing a collapsing trachea. During transport the ET was dislodged requiring re-intubation. The patient became severely distressed, on auscultation lung sounds were audible on the right side of thorax only and SaO2 was 86%. The ET was retracted a few centimeters and was secured in place. Patient’s SaO2 reading was 99%. A new x-ray showed that even though the ET was retracted; the tip of the ET was in a position close by the heart.

This is the perfect example of when certain intervention such as properly securing the airway, proper size of equipment (ET length), and verification of placement (bilateral auscultation) can determine the successfulness of case management.

Breathing patterns
Respiratory patterns guide the veterinary team to localize the anatomical site of disease for life-saving intervention. Loud breathing or stridor (heard without the aid of a stethoscope) indicates large airway disease (nasal passages, larynx/pharynx, or trachea). Inspiratory stridor directs investigation of the extrathoracic airways, especially the larynx. Expiratory stridor is usually due to intrathoracic tracheal changes. Rapid, shallow breathing is suggestive of pleural space disease (e.g. air or fluid). Laboured breathing on both inspiration and expiration is most typical of lung parenchymal disease. Distress on expiration, with a short inspiration directs attention to the small airways.

Auscultation can help distinguish pleural disease from lung disease. Moist lung sounds suggest fluid in the lung tissues. Dry, coarse sounds on inspiration and expiration suggest fibrosis of the lung. Absence of lung sounds indicates that air or fluid in the pleural space is dampening airway noises.

As the work of breathing progresses, the animal will assume a posture to assist their efforts. Cats often sit crouched with their sternum elevated from the surface and dogs extend their neck, abduct their elbows and arch their back.

Respiratory rates below 8 or above 30 are considered abnormal. Low respiratory rates can be caused by trauma to the brain or spinal cord, diseases affecting respiratory drive (e.g. chronic obstructive pulmonary disease, low blood carbon dioxide level), and drugs (e.g. sedatives). Increased respiratory rates can be caused by fever, pain, anxiety, trauma to the brain or chest, metabolic alterations (e.g. alkalosis), pulmonary diseases (e.g. pneumonia or oedema of the lungs), and drugs (e.g. oxymorphone).

Any change in breathing pattern or effort warrants immediate notification of the veterinarian. The VN should administer oxygen until a complete assessment can be made. When respiratory distress is severe, endotracheal tubes and a laryngoscope
should be placed by the cage, or tracheotomy made ready as directed by the veterinarian.

**Hypoxaemia**

Hypoxemia is defined as low arterial oxygen pressure (PaO2) (usually < 60 mm Hg). This is characterized by nonspecific signs and symptoms ranging from agitation to lethargy, hypertension to hypotension, and tachycardia to bradycardia. Pulse oximetry may confirm low O2 saturation (90%); arterial blood gas analysis may confirm a PaO2 of less than 60 mmHg. Hypoxemia, if untreated, can result in organ ischemia.

**Hypoventilation**

Hypoventilation: Central respiratory depression is caused by most anaesthetic agents. This may lead to significant hypoventilation and hypercarbia (increase CO2). Impaired respiratory muscle function, particularly after upper abdominal surgery, may contribute to the problem of carbon dioxide elimination. Other contributing factors may include tight dressings, obesity and gastric dilatation. In addition, increased carbon dioxide production may occur as a result of shivering or sepsis. This leads to hypercarbia in patients unable to increase ventilation enough to compensate.

Hypercarbia resulting from post-operative hypoventilation may cause hypertension and tachycardia, increasing the risk of myocardial ischemia in susceptible patients. Hypoventilation, by itself or in combination with other factors previously described, can cause hypoxemia. Very high levels of CO2 may have sedative effects. Evaluation of suspected hypoventilation can be done measuring end tidal CO2 (etCO2) or an arterial blood gas.

The availability of nasal prongs that allow for the measurement of etCO2 make it now possible to assess CO2 not only in the post-op patient but also in those presented with altered LOC (stupor, coma, extremely lethargic) that are at risk of hypoventilating.

Careful titration of opioid antagonist, such as Naloxone, may be effective in improving ventilation without compromising pain relief. Planning ahead to provide adequate postoperative pain relief is essential for maintaining adequate postoperative ventilation, particularly in patients undergoing abdominal or thoracic procedures. Hypoventilation, in the post-op patient, that does not improve sufficiently by non-invasive means requires intubation and mechanical ventilation until the patient is able to maintain adequate ventilation.

**Capnometry**

Capnometry is a non-invasive mode of monitoring partial pressure of end-tidal CO2. It is defined as the measurement and numeric display of End-tidal CO2 (ETCO2). Capnography is the measurement and graphic display of expired carbon dioxide PCO2 versus time. Exhaled CO2 is a reflection of CO2 production (metabolism), transport (blood and circulation) and elimination (ventilation). Though a variety of
techniques can be used to measure CO2 (colorimetric, mass spectrometry, Raman analysis), the majority of capnographs rely on infrared absorption. Use of this technique can reliably and quantitatively provide vital information regarding the respiratory status of operative and critically ill patients.

**Normal capnogram**
1. Phase I: represents the beginning of exhalation, during which PCO2 remains almost zero while gas from the anatomic dead space leaves the upper airway.
2. Phase II: depicts the waveform rising sharply as exiting alveolar gas mixes with dead-space gas.
3. Phase III: the capnogram reaches a plateau representing gas from the alveolar space:
   a. The terminal and highest portion of plateau represents PetCO2.
   b. The slope of phase III is determined by ventilation/perfusion (V/Q) status of lungs.
   c. Patients that have increased dead space have a steeper phase III and may not reach a plateau.
4. Phase IV: the waveform sharply declines as inspiration begins.

**Abnormal ETCO2**
Normally, ETCO2 is 2-5 mmHg lower than PaCO2. Increased levels may be due to increased production, depression of respiratory centre, or hypoventilation. Abnormally low levels of ETCO2 most often are associated with hyperventilation or due to increased dead space. A sudden or abrupt decrease in ETCO2 can be due to ventilator disconnection, leakage in circuit, an obstructed ET, acute hypotension, hyperventilation or massive pulmonary embolism. Accidental placement of ET into the oesophagus or ET dislodgement will result in total absence of waveform. Elevation of baseline or phase I is indicative of CO2 rebreathing and suggests that either the absorbance of the CO2 is exhausted or malfunctioning valves. Prolongation or slanting of phase II occurs with obstruction of expiratory gas flow (kinked ET) or leak in breathing system. Increases in the slope of phase III can be due to events that impede or obstruct expiration.

**Nasal Capnography**
ETCO2 is most commonly measured in intubated patients, however if nasal cannula are placed they can be adapted (with the addition of a Y connector) to allow connection of the patients to a capnograph, or nasal prongs with measure ETCO2 as well as delivering O2 can be obtained. The capnogram using these ‘nasal’ techniques may differ from that in anaesthetised patients.

**Circulation**
CIRCULATION and PERFUSION: Any postoperative patient is at risk of cardiovascular complications resulting from actual or potential blood loss from surgical site, side effects of anaesthesia, electrolyte imbalances, and depression of normal circulatory regulating mechanisms. Monitoring of the heart rate and rhythm and blood pressure aids in the assessment of the patient’s cardiovascular status. The VN should compare preoperative vital signs with postoperative values.
Vitals
Evaluation of the patient through physical findings is the "gold standard" for determining patient status. These vital signs, to include level of consciousness, respiratory rate and effort, heart rate and rhythm, and perfusion parameters, provide clinical signposts that point to trends of change, warning the team of complications. Monitored parameters such as urine output, blood pressure, central venous pressures, and pulse-oximetry provide information regarding the basic physiologic functions of the animal. Obtaining and assessing physical and physiologic parameters is a serial process, establishing trends with the initial value acting as the baseline. The veterinary nurse must recognize what is normal for the animal and what the trend was from the previous shift. Any declining trend of change directs the veterinary nurse to inform the veterinarian and to intensify monitoring procedures.

Temperature
TEMPERATURE CONTROL: Hypothermia during anesthesia is the consequence of decreased basal metabolic rate and muscular activity and increased heat loss associated with depilation, the application and evaporation of antiseptic solutions, exposure to cold table surfaces, and open body cavities. The patient’s depressed level of body function results in a lowering of metabolism and fall in body temperature. When patients begin to awaken, they will feel cold and uncomfortable.

Heat loss in the surgery suite can be reduced by minimizing duration of preparation of surgical site, by protecting patient from the cold environment by placing towels or blankets over gurneys and metal surfaces, by minimizing surgical time, and by actively warming the patient or its environment. During surgery, open cavities may be flushed with warm sterile saline (< 105.0 F, [< 42 C]).

- The body maintains temperature homeostasis by balancing heat production with heat loss through a thermostatic feedback mechanism in the hypothalamus of the brain. During illness or central nervous system disorder, this mechanism may be altered. Chemical substances released in disease can reset the thermoregulatory centre, increasing the metabolic rate, producing and conserving heat and elevating body temperature. These chemicals may be pyrogens secreted by bacteria or cytokines associated with inflammation. Primary brain disease (e.g., cerebral oedema, neurosurgery, brain trauma, or tumours) can reset the thermostat to a higher level.
- Emergency patients should have their temperature monitored several times daily. Patients with infections, excessive panting, hyperactivity, and postoperative patients should have their temperatures checked more frequently. Temperatures are ideally monitored from the same site, usually rectally. Other sites of monitoring include the axillary and inguinal region. These areas generally are 1 degree lower than rectal temperature. Ear temperatures can be obtained using an ear probe. Serial temperatures taken from the same area are more important than single values.
Hypothermia
Hypothermia results in a reduced metabolic rate and enzyme functions. There is a decrease in oxygen consumption and a decrease in the ability of haemoglobin to release oxygen to tissues. Hypothermia affects the cardiovascular system by causing peripheral vasoconstriction, decreased heart rate and hypotension. Gastrointestinal motility is decreased, and ileus may occur.

Shivering, besides being a sign of hypothermia can be a side effect of certain anaesthetic agents. In paediatric patients and toy breeds, shivering can lead to hypoglycemia, because muscles activity will lead to rapid utilization of greater amounts of glucose. If the patient’s glucose drops to dangerous levels (< 60 mg/dL), seizures can occur. In rare instances malignant hyperthermia develops. A life-threatening complication of anaesthesia, this condition causes extremely high fevers >105 F (> 41 C), tachycardia, metabolic changes, and even convulsions.

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- Hypothermic patients can be treated by covering them with a warm blanket and placing warm water bottles around them. The patient can be placed under a heating lamp, but strict monitoring is essential to prevent burns. Circulating warm water blankets are preferred over heating blankets and lamps because there is less chance of accidental thermal burns. No surface heat should be provided without volume replacement, because vasodilatation from the heat may exacerbate their condition. Severe or prolonged hypothermia may require more aggressive approaches such as active core warming with IV warmed fluids, warm peritoneal lavage or intracolonic lavage with warm isotonic fluids. The recumbent patient should be turned every 2-4 hours to avoid thermal injury. Heating should be discontinued after the rectal temperature is low normal.

Addressing hypothermia
The VN should measure body temperature and provide warm blankets, warm water bottles or circulating heating pads to warm the patient and his environment. Warm water bottles should be changed when their measured temperature is below that of core temperature, because at this point they absorb heat from the patient. Increasing body warmth causes patient’s metabolism to rise and circulatory and respiratory functions to improve.

Electric heating pads may easily overheat the contact area, causing burns and extensive skin sloughs. Heating blankets should be well insulated in towels to avoid direct contact with patient. Infrared lamps can be used during and after surgery. Optimum distance between the lamp and the patient is about 75 cm; a distance of
50 cm has been associated with excessive skin heating, whereas a distance of 100 cm was associated with ineffective warming.

A. Bair Hugger
B. Pillow case: we no longer buy the Bair Hugger blankets. We buy pillow cases and using a grommet, make a series of holes on one side of the pillow case. The color of the pillow denotes the area were the cases are to be returned after being laundered (Green=SX; Blue=ICU; Yellow=Isolation)
C. IV fluid line warmers
D. Warming Tent: 4 one gallon jugs are wrapped in towels. The side panels of the cage are covered with towels. We place one of the gallon jugs at each corner of the cage. Place a blanket over the top of the jugs. This creates a tented area where the patient is placed.

Hyperthermia
Hyperthermia creates increased tissue oxygen requirements. The body responds by increasing ventilation to release body heat. Should the PCO2 decrease too low, cerebral vasoconstriction and brain hypoxia can result. Cardiac work and oxygen demands increase. Peripheral vasodilatation occurs in an effort to release heat. Damage to vascular cells can lead to disseminated intravascular coagulation, sloughing of gastrointestinal mucosa, bacterial translocation, and significant intravascular volume deficits.

Blood pressure and Perfusion
Arterial blood pressure is a product of cardiac output (heart rate and stroke volume) and peripheral vascular resistance. Systolic pressure is the pressure exerted by the blood as a result of contraction of the left ventricle. Diastolic pressure is the pressure exerted by the blood within the vessel when the ventricle is at rest. The difference between the diastolic and the systolic pressure is called the pulse pressure. Mean arterial pressure (MAP) is the diastolic pressure plus one-third the pulse pressure. Anything that alters cardiac output or peripheral vascular resistance will alter the blood pressure.

Blood pressure can be measured by either direct or indirect methods. Direct (invasive) blood pressure measurement requires the insertion of a catheter into an artery (e.g. femoral or dorsal pedal artery) and connecting a transducer linked to a monitor. The direct arterial pressure is demonstrated in waveform on an oscilloscope with the high point being the systolic and the low point the diastolic pressures. Though direct measurement provides the most accurate pressure values, expensive and sophisticated monitoring equipment is required. A surgical approach is often necessary for arterial catheter placement in hypotensive or obese animals. Indirect (noninvasive) blood pressure measurements, though less accurate, are more easily obtained utilizing affordable equipment. Blood pressure cuffs can be placed around the distal portion of a leg or around the tail. The two most common methods used for indirect blood pressure instruments in veterinary medicine are oscillometric and Doppler. Systolic pressure should be above 100 mmHg. Systolic pressure below 80 mmHg is significant, and below 60 mmHg may be associated with
poor renal perfusion and oliguria. Cerebral circulation is compromised when systolic pressure falls below 50 mmHg, with brain ischemia occurring when systolic pressures are below 30-35 mmHg for 2 hours. Coronary perfusion is best maintained when systolic pressures are higher than 70 mmHg. Hypertension with systolic pressures above 200 mmHg can be associated with a hyperdynamic stage of shock, excessive endogenous production of renin, chronic renal failure, and excessive sympathetic stimulation.

The normal blood pressure in the dog and cat is 120 mmHg systolic and 80 mmHg diastolic. The mean arterial pressure is normally 80 - 90 mmHg. Increases in blood pressure can be caused by any condition that increases cardiac output, such as fever, exercise, and septic shock. Decreases in blood pressure can be caused by cardiac failure, hypovolemic shock, drugs (such as sedatives, opioids, and anaesthetics). Blood pressure should be evaluated together with the animal's perfusion parameters, urinary output, and disease state. As with any other monitoring parameter, repeated measurements are needed to detect a trend in change. Renal perfusion over the short term is considered adequate if BP is maintained above 60 mmHg.

Shivering, trembling, struggling, vasoconstriction, and inappropriate cuff size are common causes of erroneous measurements when utilizing the oscillometric method. There is also a decreased reliability when attached to animals less than 15 pounds. Erroneous results can occur with the Doppler method due to malpositioning of transducer, inappropriate cuff size, poor contact with coupling gel, and flexion of the limb. Any blood pressure outside the normal value must be reported to the veterinarian. Intervention by the veterinary team is performed after complete patient assessment including LOC, CVP, perfusion and UO. Hypotension may be treated by hemostasis, crystalloid or colloid infusion, and potentially positive inotropes or vasopressors. Hypertension can be treated by pain relief, diuretics or possibly vasodilators.

**CVP**

**CENTRAL VENOUS PRESSURE:** Pathophysiology: Central venous pressure (CVP) is a function of four independent forces: volume and flow of blood in the vena cava, distensability and contractility of the right chambers during filling, venomotor activity in the vena cava, and intrathoracic pressure. When right heart function and intrathoracic pressure are normal, CVP can be used as a reflection of intravascular volume. Changes in blood volume will result in pressure changes in the vena cava and are reflected by the CVP. Central venous pressure measurement requires placement of a central catheter into the cranial vena cava with the tip lying near the base of the heart (i.e. right atrium).

Normal CVP measurements are reported as -1 to 5 cmH2O. However, critical animals are resuscitated to supranormal values and the CVP is optimally maintained between 5-8 cm H2O. Values less than 5 are suggestive of insufficient intravascular volume. Values over 14 cm H2O are of concern for right heart failure or significant volume overload. Factors unrelated to right heart function and volume overload (such as
pleural, pericardial or mediastinal pressure, and increases in pulmonary hypertension) can also raise the CVP.

**Primary factors affecting CVP**
The CVP can be used to guide aggressive intravascular fluid resuscitation. When the CVP is low in a hypotensive animal, crystalloids and colloids are rapidly administered until the CVP is between 5-8 cm H2O. At that time, if hypotension persists, positive inotropes or pressor agents are administered

**CVP set up**
High CVP measurements warrant examination of the system for occlusion of the catheter. If the system is patent, then fluid overload or right heart failure are suspected. The fluid rate is lowered and the veterinarian will opt to administer diuretics or drugs specific for the cardiac condition. Any CVP measurements outside of the target values set by the veterinary team should be reported to the attending veterinarian.

Central venous pressure monitoring set-up: Central venous pressure measurement requires placement of a central catheter into the cranial vena cava with the tip lying near the base of the heart (i.e., right atrium). The catheter is attached to IV extension tubing, which is connected at right angles to a water manometer by a three-way stopcock. Across from the IV extension tubing on the stopcock is an IV line and fluids. The zero on the water manometer should be at the level of the right atrium. A horizontal line drawn between the thoracic inlet and the manometer establishes the zero reference level.

The stopcock is off to the manometer when the patient is receiving intravenous fluids. To measure CVP, the manometer is filled with fluid from the IV bag and then the stopcock is turned off to the bag leaving a column of fluid within the manometer. The stopcock is then opened towards the patient, allowing the fluid in the manometer to access the patient. The fluid level in the manometer is allowed to equilibrate with the pressure in the jugular vein. The fluid level may oscillate a few millimeters with each respiration or heart beat. Three values or readings are obtained to ensure consistent readings.

Normal CVP measurements are reported as -1 to 5 cmH2O. However, critical animals are resuscitated to supra-normal values and the CVP is optimally maintained between 5-8 cmH2O. Values less than 5 are suggestive of insufficient intravascular volume. Values over 14 cmH2O are of concern for right heart failure or significant volume overload. Factors unrelated to right heart function and volume overload (such as pleural, pericardial or mediastinal pressure, and increases in pulmonary hypertension) can also raise the CVP.

If readings do not fluctuate with respiration, the readings are inaccurate. Note which side the animal is positioned and, future reading should be made with the animal lying on the same side. Always use the same zero point reference (thoracic inlet), so that readings are comparable. Always obtain three or five consecutive readings at a
time. Each reading should be approximately close in measurement. Huge discrepancies in readings should alert the nurses to trouble shoot the CVP set-up for kinks, clogs or changes in catheter or patient position.

**Electrocardiogram**
An electrocardiogram (ECG) provides valuable information on rate and rhythm and is essential to assess arrhythmias. Arrhythmias are clinically important; they may cause or exacerbate low cardiac output states. It may be impossible to resolve congestive heart failure signs without controlling concurrent arrhythmias. Arrhythmias can contribute to myocardial ischaemia.

The ECG is the ‘gold standard’ for identification of an arrhythmia. It should be remembered that the ECG is not sensitive in detecting cardiac chamber enlargement (echocardiography and radiography are much more sensitive). An ECG provides no information about the ability of myocardium to contract, nor does it provide any information about the heart valves or endocardium. The ECG is the only way to diagnose the actual arrhythmia and hence provide appropriate treatment for the patient. Cardiac arrhythmias can contribute to morbidity and mortality in critically ill dogs and cats. Successful management of arrhythmias often involves investigation and correction of an underlying non-cardiac disorder, and management of contributing factors in cases with severe systemic illness. In animals with primary cardiac disease, arrhythmia management is usually accomplished with drug therapy or cardiac pacing.

**Renal function**
RENAL FUNCTION: The renal blood flow is a product of systemic arterial blood pressure and compliance of the renal vasculature. Therefore, renal blood flow and glomerular function may be depressed as a consequence of systemic hypotension, renal vasoconstriction, or a combination of the two. Irreparable kidney damage can occur in an animal that suffered from only mild renal compromise preoperatively.

Renal blood flow diminishes markedly if mean arterial blood pressure drops below 80 mm Hg. Even with adequate mean arterial pressure, renal perfusion can be inadequate if renal vasculature resistance is too high. Blood vessels of the kidneys are richly innervated by sympathetic nerve fibers that mediate vasoconstriction through alpha-adrenergic receptors. Stress, excitement, pain, and light anaesthetic planes all can result from sympathetic stimulation. Patients with pre-existing renal disease respond most favourably if given adequate sedation, analgesia, and stress-free handling.

All general anaesthetic drugs in animals with or without renal disease temporarily depress renal function as a result of decreased cardiac output. Epidural anaesthesia may produce minimal alterations in renal function. Epidural anaesthesia blocks sympathetic fibers in the thoracolumbar spine and prevents renal vasoconstriction. Renal effects of anaesthetics are dose related and are favourably influenced by adequate repletion of extracellular fluid with intravenous fluid therapy.
The main function of the kidneys is to excrete metabolic wastes and reabsorb vital electrolytes and water. The volume and contents of the urine produced is a result of the function of the population of nephrons, made up of the glomerulus and renal tubules. The volume of urine produced is dependent upon the glomerular filtration rate (GFR) and ability of the renal tubules to reabsorb sodium and water. The factors governing the GFR are the size of the glomerular capillary bed, the permeability of the capillaries, and the hydrostatic and oncotic pressure gradients across the capillary walls. The factors governing the function of the tubular cells include: oxygen utilization, glucose availability, and integrity of the cellular enzyme systems. Variations in these factors have predictable results. For example, should the mean arterial blood pressure falls below 60 mmHg, the hydrostatic pressure gradient declines across the glomerular capillary beds and glomerular filtration almost stops (oliguria). Severe prolonged hypoxia can cause the dysfunction or death of the glomerular and tubular cells, leading to inadequate urine production. In addition, interruption of the tubular cell ability to reabsorb sodium results in high urine sodium and an increased urine production.

**Procedure**

Accurate and frequent measurement of urine output (UO) requires bladder catheterization. A closed urinary collection system utilizing a sterile collection bag and IV line is attached with the bag maintained off the floor and below the level of the catheter. The bladder is immediately emptied and the time recorded as the 0 time (start of collection). The frequency of measuring UO is determined by the rate of onset and the severity of the disease. In general, UO is measured every 2 hours. Daily examination of urine sediment is performed to monitor for infection. Urinary catheters should be flushed with sterile saline and inspected for kinks and clots in the line at least every 8 hours or if there is a sudden decline in urine collected.

An indirect method of estimating UO is to place incontinence sheets in the patient's cage to collect the urine. The weight of a dry sheet is subtracted from the weight of a urine soaked sheet. Each 1g increase in weight equals 1ml of urine. An alternate method is to place the animal on a grate elevated off the cage floor. Urine is then collected and measured.

Fluid input and UO are recorded, including any fluids administered by enteral or parental routes. Quantities of fluid lost through vomiting and diarrhoea are estimated and recorded.

The technician must evaluate the UO in relation to the haematocrit, total solids, central venous pressure (CVP), blood pressure, heart rate, and body weight. Any decrease in UO in an adequately hydrated and perfused animal warrants immediate notification of the veterinary surgeon in charge of the case. The IV fluid rate is reduced and the urinary collection system examined for post-renal causes of urine outflow obstruction. If the origin of the condition is determined to be renal, the veterinarian may choose to administer either mannitol or frusemide and dopamine to stimulate urine production.
A polyuric animal will require a greater quantity of intravenous fluids for maintenance of normal hydration. Medullary washout often occurs and requires a slow tapering from IV fluids onto oral fluids to avoid significant dehydration. Potassium is commonly low in these animals and requires aggressive supplementation.

**Gastrointestinal Function**

GASTROINTESTINAL FUNCTION: Anaesthetics slow gastrointestinal (GI) motility and cause nausea. The VN should auscultate for abdominal sounds. One should normally hear faint or absent bowel sounds in all four quadrants during the immediate recovery period.

Inspection of the abdomen serves to rule out abdominal distention that may be caused by the accumulation of gas. Distention can also develop if internal bleeding occurs in the patient who has undergone abdominal surgery. The patient who is at high risk of vomiting should have a nasogastric (NG) tube placed. The VN should keep the tube patent using regular irrigations. Occlusion of NG tube results in the accumulation of gastric contents within the stomach. Because stomach emptying slows under anesthesia, the accumulated contents cannot escape, and nausea and vomiting occurs. Amounts suctioned should be recorded and the VN should inform the clinician if amounts being suctioned are not decreasing or increasing.

Specific therapy to enhance gastrointestinal motility is not administered following anaesthesia. If opioids have been selected in the anaesthetic protocol, use of opioid antagonist will reverse opioid-induced stasis.

The major causes for diarrhea in the postoperative/ICU patient include medications, enteral nutrition, and infection. Other causes include ischaemia to the bowel, possible sepsis and hypoalbuminaemia. Diarrhea can lead to further complications, such as volume depletion, and wound infection.

**Blood Gas Analysis**

**PaO2/FiO2 ratio**

PaO2:FiO2 ratio can be used to evaluate pulmonary function at any FiO2, which provides an advantage over the A-a gradient and the 120 rule. The value is acquired simply by dividing the PaO2 by the FiO2.

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\text{PaO2/FiO2} = \frac{\text{P}}{\text{F}} \text{ ratio}
\]

Where the FiO2 is expressed as a decimal. With normal pulmonary function, the P:F ratio should exceed 500mmHg. The P:F ratio can be used to approximate the severity of pulmonary dysfunction; values between 300 and 500mmHg are associated with mild dysfunction, between 300 and 200mmHg are considered to have moderate dysfunction, and less than 200mmHg are considered to have severe pulmonary dysfunction. The author finds this method useful as it is quick and can be used at any FiO2. The main disadvantage being the method disregards the effect of
ventilation (PaCO2), but this is only an issue when carrying out the calculation on room air.