**A Critical Examination of Therapeutic Procedures Used to**

**Provide Emergency Support for Dogs Presenting with Dyspnea:**

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This presentation provides practical information to the emergency and general practice clinician on which procedures to perform to gain the best possible resolution for dogs presented with dyspnea based on their clinical presentation.

Recommendations made here were based on clinical outcome data that critically examined the effectiveness of these procedures.  Criteria used to evaluate the effectiveness of the procedures examined included presenting signs (upper verses lower or mixed respiratory patterns), resolution of clinical signs such as decrease in work of breathing, level of oxygenation achieved, staff time and special devices or equipment required, ease of implementation, complications noted, and follow-up as prospectively studied in several busy emergency and critical care/ referral facilities. NOTE: The true complete definition of dyspnea is that of a *symptom* of a patient *saying* that they are having difficulty in breathing, but the expanded definition includes the *clinical signs* noted of difficult breathing as judged by the owner and / or clinical professionals.

**ASSESSMENT:**

**Clinical assessment - By far and away the most important evaluation of the patient that is having difficulty breathing (\*dyspnea) is a good observational assessment of signalment, history, and physical exam findings. *Often the first few minutes of assessment and care determine the outcome if the patient*” .breathing his or her last”” Because oxygen is 20 times less diffusible than carbon dioxide the immediate need for most patients to provide supplemental oxygen and often sedation. SUPPLEMENTAL OXYGEN IS PROVIDED WITH OXYGEN TUBING ATTACHED TO A 14 G IV CATHETER AND THE OXYGEN STEAM BLOWN TO THE PATIENTS FACE LIKE A WATER STEAM AIMED AT A FIRE. A CLEAR PASTIC BAG CAN ALSO BE PLACED OVER THE ANIMAL LIKE A PORTABLE OXYGEN TENT FILLED WITH O2 AND THEN ASSESSMENT CONTINUED:**

A, THE PATTERN OF BREATHING IS ASSESSED and BREATH SOUNDS ARE CHARACTERIZED.

BLOW BY with CATHETER NOZZLE – to the face as the breathing pattern is noted:

**Characterization of the breathing pattern:**

Short Rapid Difficult Breaths = Restrictive Patterns = Lung can not expand – commonly associated with pneumothorax, pleural fluid buildup, diaphragmatic hernia, chylothorax, hemothorax – severe abdominal distension

Long slow breaths witb added effort to inhale (literally looking like the patient is sucking on a small straw) = Upper Airway Resistance Pattern. Seen with upper airway obstruction such as that seen with laryngeal collapse or tracheal stenosis or masses.

Longer slower breaths with additional force seen for exhalation = noted with lung parenchyma and small airways problems such as asthma or pneumonia

**Characterization of the breathing generated sound from a distance:**

Sounds normally are quiet and not obvious other than hard panting. If these added sounds (called adventitial sounds) they are characterized:

 Low pitched guttural sounds heard on inhalation = pharyngeal obstruction

High pitched sibilant sounds heard on inhalation = laryngeal obstruction

**Characterization of breath sounds on stethoscope auscultation:**  Then listen to the breath sounds bilaterally – pneumothorax does occur unilaterally initially. Breath sounds are generally quiet with a slightly more soft F sound on exhalation - Sounds other than these are adventitial sounds and are characterized:

Increased sounds on inhalation = generally associated with larger airway resistance as what might be seen with a tracheal injury and some stenosis

Increased sounds on exhalation = generally associated with increases in resistance in the small airways such as that associated with an increase in interstitial edema (as observed in early fluid overload) or inflammation (as is seen in very early pneumonia). These are called bronchovesicular sounds.

Musical adventitial exhalational sounds = those that are musical like wheezes indicate some bronchial constriction as occurs with asthma.

Water like adventitial water like sounds = those associated with fluid or exudate within the bronchi which can be associated with pneumonia An older term was rales but these often are wet sounds and are heard with exudate within the airways. Rales are also seen with more advanced pulmonary edema, where fluid is accumulating into the small bronchial tubes. Rattles are more obvious and louder than rales and indicate an even more fluid accumulation within the small

**Characterization of sounds heard and energy waves felt on percussion;**

Percussion is difficult to do and interpret but can be useful in cases where air is accumulating rapidly as apposed to fluid that generally takes more time to do so. Immediate percussion done with one of the operators hands on the chest wall and the middle two fingers of the opposite hand used to thump the back of the opposite hand that is remaining on the chest wall. The operator does this procedure on both sides and compares the sound and the palpable characteristics of this thump. The sounds heard are then characterized:

Sounds same on both sides and are neither low base like or higher thud like = normal

Hollow sound like a base drum = air accumulation in the pleural space on that side

Dull thud like sound = fluid or solid mass or structure on that side

**Auscultation of upper airway sounds generated with panting**:

Similar to the physical test called “egophany” where the human patient is told to say the sound E and the stethoscope is used to hear the referred upper airway laryngeal sound E (termed Egophany) through the patent’s bronchi, the animal’s upper airway panting sounds are transferred through the bronchi and is used to detect blocked main sections of the bronchi. This diagnostic test I have termed “pantophany” is not difficult to do and does help in the detection of lung collapse and major bronchi blockage.

**The NEW STETHOSCOPE = ULTRASOUND:**

This is becoming the new stethoscope in practice. All animals with dyspnea on arrival, after supplemental oxygen is begun, are soaked with alcohol, applied to the lateral thoracic wall on both sides and the ultrasound probe is then applied and used to assess for pneumothorax, free pleural fluid, pulmonary parenchyma, pericardium, myocardium contractility and chamber size, and diaphragm**:**

**Probe placed on the thoracic wall dorsally and thoracic structures visualized:**

Examined for a glide sign – if not seen = pneumothorax,

Examine for lung rockets **-** If seen indicate increased pulmonary density as may be seen with pulmonary edema, pulmonary contusions, and pneumonia. Three or more lung rockets seen per field = indicate more increased water content than normal lungs

Examine for free fluid in the pleural space - seen as fluid between the parietal pleura and the visceral pleura = see with blood, chyle, or other types of free fluid; the US probe can be then used to aspitate some of this fluid if there enough that can be withdrawn safely. Occasionally pulmonary masses can also be observed.

**Probe placed at midthoracic area (chest tube site) and structures visualized:**

The same interpretations are made.

**Probe placed at the ventral lung – pleural space area and structures visualized:**

The same interpretations are made.

**Probe placed over the heart, pericardium and diaphragm structures visualized;**

Observe for pericardial fluid, or other – if seen is pericardial hemorrhage or fluid; Pericardial diaphragmatic hernia can be seen nicely with this technique. One case of dyspnea lead to acute cardiac arrest that occurred in our hospital lobby; jugular veins were distended – probe identified this possible reason for acute decompensation and immediate thoracotomy was completed and opening of the pericardium and CPR lead to a complete recovery with good neurological function

Observe cardiac contractility – provides a index of cardiac output and possible cause of dyspnea and very useful in tracking response to fluid resuscitation, diuretic response, etc. Occasionally other reasons for the dyspnea will be detected, e.g., heart base mass.

Observe the diaphragm – provides a means of noting significant diaphragmatic hernia as often liver parenchyma is observed in these cases and this leads to the operator having a diagnosis or preliminary diagnosis within minutes of admission and without any added manipulation of the dyspnic patient. This can then lead to immediate intubation with a rapid induction and moving on to definitive repair as these patients can not be “stabilized” without rapid intervention.

**Probe placed on the abdominal side of the diaphragm**: - The cranial portion of the abdomen is visualized as an AFAST (a focused abdominal sound for trauma, tumors, etc). Some animals with abdominal conditions such as extreme sepsis will present with dyspnea and free fluid or an abdominal mass can be found. This would lead to a clinical workup for such.

**THERAPY REQUIRED:**

**This may range from oxygen supplementation (SEE BELOW) to nearly immediate resuscitative thoracotomy such as might occur with sudden cardiac arrest. This author has not seen very many patients in which closed chest CPR in the face of dyspnea has been successful. Yet with open chest CPR he has observed noted “saves” when a resuscitative thoracotomy and subsequent CPR with recognition and resolution of the reason for the arrest was accomplished.**

Examples of Dyspnic Animals that required a Resuscitative Thoracotomy- the reason noted for the decompensation and resolution leading to a successful outcome:

Pericardial tamponade from a pericardial- diaphragmatic hernia

Severe tension pneumothorac from a ruptured pulmonary bulla

Severe tension pneumothorax from a dog bite associated lung/bronchus injury

Severe tension pneumomediastinum caused by a peritracheal wound

Severe tension pneumomedistainum following the creation of a tracheotomy that was required to be able to perform a partial arytenoidectiomy

**Supplemental Oxygen** - The first principle of resuscitative treatment of the dyspnic patient is to provide DtO2 to meet needs of all tissues of the Brain\*, Heart, GI, etc. Therefore this commonly dictates the need for *supplemental oxygen*..\* DtO2 is determined by FiO2 ~ pAO2~ paO2, [Hb], Q (t capillary), interstitial space. Since it is common to have VQ Mismatching in such conditions as shock, trauma, sepsis it is highly commended that supplemental O2 be provided in all critical patients especially if they exhibit dyspnea. The goal with supplemental oxygen therapy is to provide it as required to gain just enough increased FiO2 to allow for better tissue oxygenation to meet the metabolic oxygen demands for the cardiovascular, neurologic and gastrointestinal systems. The simplest and most clinically effective way to determine this initially is to observe the changes seen in both *breathing rate and effort*…. IF BREATING RATE AND EFFORT ARE BECOMING MORE NORMALIZED THESE OBSERVED SIGNS INDICATE that the supplemental oxygen is making a positive impact and should be continued as DO2 is at least getting close to satisfying the CNS VO2 (oxygen demand) and this includes the aortic arch.

Assessment may be also be enhanced through the attempted use of **PULSE OXIMETRY as this provides a digital means of determining the need and results of treatment.** Any SpO2 % above 94% is acceptable. HIGH FIDELETY SpO2 monitors like the Mosimo unit or those that provide nasal membrane oxygen placed saturation transducers may provide another means of oximetry. The common systems use transmissive oximetry methods that involve the detection of the amount of oxygen saturation in the red blood cells using a photodetector that is placed on one side of the tissue being interrogated and the other side where the photon beams are being generated. The device passes two wavelengths of light through the body part to the photodector. The changes in absorbance at each of the near infrared wavelengths is measured. These differences in absorbance are due to pulsing arterial blood in the small arterioles. Reflectance pulse oximetry may also be used as an alternative. It does not require the light beams to be transmitted through the skin or membranes and is therefore more effective when interrogating the chest, feet, and forehead. This technology has recently been introduced into smartphone technology such as the Samsung Galaxy S5. For pulse oximetry to been effective and reliable the pulse wave MUST be able to be seen and assessed (Photoplethysmography) so that the accuracy can be determined. There must be a good wave form for clinical interpretation.

Unfortunately in many cases pulse oximetry is NOT able to be used initially due to the difficulty in applying the sensor anywhere. As many experienced clinicians have said “ These patients should not be messed with” and attempts to place a pulse oximeter, at least at the onset of therapy is contrainidicated. This also includes the procurement of blood samples for laboratory analysis and the procurement of radiographs.

Research has proven that supplemental oxygen might be a means of care that provides a buffer between catastrophic decompensation and getting by while the history and exam (as best as can be done safety) are completed

**Research papers supporting supplemental oxygen: Head injury swine model supplemental O2 reduced the edema and improved outcome…. “ The MOST important treatment for all head injured**” Geoff Manley MD 2003; Chief Neuro-Trauma, Univ. California San Francisco, San Francisco General Hospital: 0% mortality w/ 100% O2 verses 71% mortality w/ 20% O2 in hemorrhagic shock pigs partially fluid resuscitated w/ hetastarch. .Meier J, et al: Hyperoxic ventilation reduces 6 hour mortality after partial fluid resuscitation from hemorrhagic shock. Shock 2004 Sept 22(3):240-247

**NOTE ABOUT SUPPLEMENTAL OXYGEN: Please refer to the manuscript entitled:**

**A Critical Look at Supplemental Oxygen Delivery Methods - What is the evidence. And a look at a new method: Using a High Flow Humidified Oxygen Cannula**- This presentation reviews all the current means of providing supplemental oxygen (blow by, cages of various types, open mask, sealed mask with BVM and reservoir, hood, collar, boat, nasal catheter, nasal cannula, bilateral nasal catheter, nasopharyngeal, bilateral nasopharyngeal, nasotracheal) and *relatively new method involving a nasal cannula fitted to deliver higher flow humidified oxygen.* These methods are compared regarding ease of use, patient acceptance, equipment/supplies needed and FiO2 achievable over given amounts of time, and oxygen flow rate required.(further information is provided that addresses these modalities is written in the proceedings entitled: A Critical Look at Supplemental Oxygen Delivery Methods

**Sedation as a critical therapy**:

This is one of the most important therapies that should be completed for the patient having difficult breathing. If anyone has ever had a respiratory crisis it is commonly remembered as one of the most frightening times in his or her entire life. Although we can not truly know what our animal patients are sensing or thinking I have noted in their eyes a look of panic and fright. This appeared to be greatly heightened as their breathing effort increased. This often becomes more exaggerated as they arrive at the very place (veterinary hospital) were they might remember other visits were they were subjected to examinations, vaccinations, blood draws, etc., that were associated with a sense of fright. **THEREFORE, as many other authors have recommended – these animals need substantial and very early treatment for their physiologically induced PANIC.**

**Protocols that have been critically examined and found to be effective in decreasing patient panic.** I have not found any clinically reproducible studies that will fit every patient however. Each patient will need a protocol that is tailored to their needed and the response observed to assure as much clinically safety and effectiveness as possible:

Butorphenol 0.2 mg/kg IM with either Acepromazine 0.01 mg.kg IM or Midazolam 0.2 mg/kg IM – acupuncture point LI-11, GV -20 and the calming point located behind the mastoid process – each activated with either pressure, red photonic light, low level laser or needed. I prefer the use of the photonic “torch” as it very quickly activates the AC point and attendant light based meridian. NOTE: This protocol can then be supplemented with added doses once an IV catheter is placed.

Hydromorphone 0.1 mg/kg with either Acepromazine 0.01 mg/kg or Midazolam 0.2 mg/kg IM along with acupuncture points as well – Of course these protocols can be used without the ACPs but the effect is enhanced by their use and in some cases the can be used along. IV doses of these combinations will also work well in most cases as long as they are not given rapidly and it is recommended that only half doses are given initially.

Ketamine 2-4 mg/kg, butorphenol 0.1-0.2 mg/kg, and acepromazine 0.01-0.04 mg/kg mixture in the same syringe given IM in the epaxial muscles (if IV access is not already available).

**Anesthesia Induction, Intubated and Positive Pressure Breathing Instituted:**

Anesthesia Induction with IV doses of these drugs with a small dose of propofol may also be needed for the very critical difficult breathing patient on occasion. This may be indicated when the dog or cat is literally “breathing their last” Blow by oxygen is given until the IV hydromorphone and Midazolam are on board and then a bag valve mask is added and assist ventilation is given. Then a small amount of propofol is provided (generally 1-4 mg/kg starting with the lower end of the dose. The trachea is intubated with the assistance of a laryngoscope (if available – which is especially helpful in cats and those dogs with airway compromises such as those that are brachycephalic. Positive pressure ventilation will then be instituted while other diagnostics and therapy such as chest taps , chest tube placement, or thoracic surgery such as what might be needed for a small dog that was attached by a large dog and sustaining injury to both the right and left thoracic wall and lung tissue, those with severe pulmonary edema from congestive heart failure, or centroneurogenic pulmonary edema from a choking episode.

**Protocol of use of a Bag Valve Mask (BVM) for assist ventilation with the addition of a PEEP Valve:**

When a pet arrives that is obviously having labored and difficult breathing an immediate course in treatment is to provide supplemental oxygen at high concentrations. This is may be best completed by use of a tight fitting face mask that is attached to a valve system and reservoir. Of course most of these patients will in a state of panic and will need the medications as discussed previously. Ideally a “non-rebreather” oxygen mask would be initially placed that would provide nearly 100% oxygen. In veterinary medicine, we lack this type of non-rebreather mask so the best option is to use a bag-valve-mask assembly or “AMBU bag” \* attached to a cone-shaped veterinary facemask. The AMBU has an exhalation valve that directs the exhaled air out into the atmosphere, and an inhalation valve that allows the inhalation of oxygen from the reservoir which is part of the AMBU. To the end of the exhalation port a positive end expiratory pressure (PEEP) valve\*\* can be attached. Using this added valve increases the amount of air left in the lung at the end of exhalation (increasing functional residual capacity). As the top of the valve is rotated it tightens a spring which increases pressure on a valve that controls the resistance to air flow out of the lungs. The reservoir is a clear plastic flexible bag which is filled with oxygen during the patient’s exhalation time and empties during bag compression as seen in this photo of a firefighter-paramedic providing bag-valve mask ventilations in the emergency training scenario. The amount of oxygen in the reservoir needed to satisfy the patient’s inhalation lung volume, which is frequently increased from the patient’s stressed state, must be able to be easily supplied in the breath effort in the ¼ to 2 seconds inspiratory time. This requires a flow rate of oxygen at the patient’s airway during inhalation calculated to be between 12and 100 Liters per minute (ave. 66 L/min)\*\*\*. This calculation is based on the tidal volume, minute volume and inhalation time of each breath. As can be seen from examination of the data presented, it is impossible to provide this amount of flow rate of inhaled oxygen without the use of a reservoir that can be easily emptied that contains 100% oxygen. Any other systems involving a face mask, where there is no reservoir or valves to control the direction of inhaled and exhaled air is not recommended to be used in emergency settings where the work of breathing must not be increased and high concentrations of oxygen are required*.* During the inhalation phase of breathing if there is no reservoir attached to this same type of bag-valve-system the highest % of oxygen that can be achieved during inhalation is 40-50% while with the reservoir oxygen concentrations can reach 100%. Of course most animals having difficulty with breathing they resist the use of the tight fitting mask. In these cases other simple ways of providing supplemental oxygen should be tried first. This includes the methods discussed in more detain in the paper on oxygen supplementation that is also in this proceedings.

**However, in summary research that we performed and published in 2004 (Engelhardt, Crowe) revealed that although oxygen cages can be used for the treatment of these critical patients, they unfortunately force isolation of the pet away from the care givers and they require at least 20-25 minutes of oxygen delivery to see oxygen concentrations approach 40% delivered to the patient.** This is of concern because of the time lag that is seen with the use of oxygen cages. Other alternatives that are available that provide higher concentrations of oxygen in a much shorter period of time include blow-by and jet blow-by (reaching 40% oxygen within 2 minutes), placement of the patient in a smaller container (compared to the standard oxygen cage) (reaching 60% oxygen within 5 minutes), use of a Crowe collar; which is an Elizabethan collar that is one size larger that would otherwise be used and its most rostral ventral half covered with clear plastic wrap to provide a “boat effect” for the holding of the delivered oxygen around the patient’s head (providing 70% oxygen within 2-3 minutes and 80% a a flow rate of 5 L/min.), nasal cannula (providing 40% oxygen with in 1 minute with an oxygen flow rate of 4 – 5 L/min.) and nasal catheters that are flexible feeding tubes that can be placed unilaterally or bilaterally following slight sedation (providing 40-70% oxygen within 2-3 minutes of the beginning of oxygen delivery at 50-100 ml per kg), and even simply placing the patient in firm or flexible plastic bubble that is connected to an oxygen supply line (providing 50-70% oxygen within1-3 minutes with oxygen delivery at 5-10 L/min.)

If after the oxygen has been administered for from a few to 30 minutes with any of these non-mask systems the patient continues to have serious respiratory effort (and a quick look ultrasound has ruled out significant pleural space disease) the patient may benefit significantly from the application of mild sedation and placement of a tight fitting mask on the patient’s head and using an AMBU resuscitation bag, and adding breath assistance with the squeezing of the AMBU bag with each breath. The positive pressure breathing is begun, timing as best as possible, the squeezing of the bag with the onset of each spontaneous breath the patient takes. Within a few minutes the patient’ respiratory rate and effort should be decreasing and becoming less labored. This simple but very effective technique called “assisted ventilation” is very effective in decreasing the effort the patient takes to continue to breath. It has been my experience with many patients that often as they get used to what is trying to be accomplished and settle into a rhythm, and literally fall asleep within a few minutes because they were so exhausted from trying to breath. Emergency patients that arrive in significant shock and pulmonary injury should be managed immediately with this bidirectional positive airway pressure (Bi-PAP) ventilation technique if they are still conscious.

Continuing the assisted ventilation for at least 30 minutes is recommended in most of these cases if they are still conscious and positive clinical effects are observed. Once the shock is being addressed the addition of a PEEP valve on the exhalation side of the AMBU bag can be instituted to treat pulmonary edema, contusions, etc., This valve has a spring that gets tighter and increases the amount of pressure needed to open up the exhalation valve. With the valve pressure set at 5 cmH20 the airway pressure at the end of exhalation is elevated, i.e., positive end exhalation pressure (PEEP). When positive pressure ventilation is added to the AMBU bag- valve-mask that is applied tightly BiPAP or Bi-directional positive airway pressure ventilation is being performed. If breathing is not assisted with the addition of positive pressure breathing this is called CPAP (continuous positive airway pressure). Both types of breathing will increase the patient’s functional residual capacity and is easily accepted by most patients. If not accepted then a small dose of butorphenol and very small dose of acepromazine generally works well to allow the animal to relax and accept the mask, which must be tight fitting to allow the positive pressure breathing to be successful.

In most cases the addition positive pressure to assist inhalation with each breath with the PEEP valve set at 5-10 cmH2O (Bi-PAP) is begun initially with patients that are not responding to supplement oxygen alone. The Bi-PAP is done for 20-30 minutes and then the patient is re-evaluated. Re-evaluation may include watching the patient’s breathing pattern and effort, auscultation of breath sounds, thoracic radiographs, thoracic ultrasound, and arterial blood gas analysis. Hopefully there are changes that are positive such as the dog who had been hit by a car that received a repeated thoracic radiographs following Bi-PAP that showed a significant improvement.

In patients that do not respond well invasive positive pressure ventilation with intubation of the trachea and placing the patient on continuous positive pressure ventilation and PEEP. In those patients that make improvement with the non-invasive ventilation it has been my experience that approximately 50% will only need a few more Bi-PAP treatment sessions and then provided with continued supplemental oxygen therapy with methods that are very effective, even if a small amount of sedation is required to keep the patient comfortable and accept the therapy such as bilateral nasal catheter placement or Crowe collar. The use of this technique has been used by the author in an estimated 400 patients over a period of 10 years, beginning in 1997. It is recommended to consider this life –saving technique as it has been very successful as a rescue procedure. I continue to use similar techniques in my human patients working as an EMT Intermediate and first responder on the rescue unit of the county. I estimate that without the use of this technique mortality would have been 100%. But through its use over 80% were able to become salvaged.

I also recommend the use of non-invasive ventilation with a bag-valve - mask for a few breaths at least BEFORE intubation in cases of the pulseless non-breathing and unconscious patient. It has been my experience that if the patient is not oxygenated well and is acidotic that any movement can induce ventricular asystole or fibrillation caused by vasovagal and sympathetic stimulation. Bag-valve mask ventilation only requires the head to be extended and the tongue pulled forward and the jaw closed and the application of the tight fitting mask, as opposed to all the manipulation necessary to perform endotracheal intubation.

 I have observed that noninvasive ventilation as described, provides a very rapid way to provide oxygen and lower CO2 in the blood stream and tissues, thus helps prevent catastrophic complete arrest in patients that were near arrest. It is recommended to perform non-invasive ventilation with the AMBU bag and reservoir connected to an oxygen supply and being delivered at from 5-15 L/minute immediately on all unconscious patients that are unresponsive and not already intubated. The technique has been one of the best life-saving procedures I have ever done.

**Protocol for care in the dyspnic patient that is NOT responding to Supplemental Oxygen and Assist Ventilation with the Bag-Valve- Mask and PEEP Valve .**

Response to supplemental oxygen therapy can usually be gauged by monitoring respiratory rate and effort, presence of cyanosis and pulse oximetry readings. Pulse oximetry readings may be unreliable in the animal with pigmented skin, where this is inappropriate thickness of skin between the oximetry clamp, and in the patient with poor perfusion.

If the patient does not respond to supplemental oxygen and assist ventilation with bag-valve-mask and pneumothorax is ruled out as well as diaphragmatic hernia or in the pleural space via auscultation, tap, ultrasound or rapid thoracic radiograph while assist-ventilation is being performed then rapid sequence induction, intubation and ventilation should be strongly considered. In many cases assisted ventilation with the use of sedation followed by placement of a tight fitting mask and positive pressure support ventilation, timed with each patient breath, is at least initially done. Artificial ventilation is indicated in the patient with ventilatory failure (inability to exhale carbon dioxide as well as inability to oxygenate) until the cause of the ventilatory failure can be resolved. It is important to recognize these patients early. Patients who have been working hard to breathe for an extended period of time may die from ventilatory failure secondary to exhaustion. The goal in these patients should be rapid induction, intubation and ventilation.

Before this is accomplished simply sedating with an IM injection in the epaxial muscles (if IV access is not already available) of a mixture of ketamine 2-4 mg/kg, butorphenol 0.1-0.2 mg/kg, and acepromazine 0.01-0.04 mg/kg as outlined prior can also be used to provide enough calming to allow the placement of a bag-valve-mask attached to reservoir and oxygen supply delivering 5-15L/minutes. The mask is applied firmly and with the patient’s head and neck extended (and the tongue pulled out and the mouth closed over the extended tongue (if the patient allows) the bag is squeezed with every breath the patient takes, thus assisting him with positive pressure breathing. (see also the section below discussion CPAP). Timing each squeeze of the bag with the beginning of each beginning breath the patient takes gives tremendous relief to the patient’s respiratory difficulty. This also helps oxygenate the patient before induction is proceeded rapidly with an IV administration of a mixture of ketamine (5 mg/kg) and diazepam (0.25 mg/kg), given to effect to allow tracheal intubation. Other drugs can be used such as 2.5% thiopental (5-10 mg/kg) and Propofol (3-8 mg/kg), but in my hands produce more significant cardiovascular depression and should are not my drugs of choice. Neuromuscular blockers (succinylcholine 0.01 mg/kg, or atracurium 0.25 mg/kg) can also be used for rapid induction after small amounts ketamine, diazepam, or an opioid are given. (Atricurium is preferred over succinylcholine as it does not cause muscular contractions as later does). In the compromised patients doses should always be titrated, as much less drug will generally be required than needed to induce the healthy patient. Once intubated (preferably with a laryngoscope, as this provides for less laryngeal manipulation and possible vaso-vagal response), positive pressure ventilation is continued.

After the patient is intubated the choices of ventilator include 1. By-hand bagging either with a bag-valve with reservoir (AMBU), or anesthetic machine; 2. Anesthetic ventilator; 3. ICU ventilator. At the same time the hunt for the cause should continue and specific therapy performed when the cause is determined.

One patient illustrating the use of an anesthetic ventilator for ICU ventilation in a newborn. This case seems to break all traditional dogma of the following: 1.you cannot ventilate with 100 % oxygen for more that 12 -18 hours as you will be irreversible oxygen toxicity and lung injury. 2 you can not give 100% oxygen to a newborn without causing significant eye, neuro and lung issues: The dog was a newborn German Short Haired Pointer – he and the rest of his litter mates had been delivered by C-section and a number of the newborn were not thriving. He was given mouth to those ventilation and then moved onto a small Ambu bag and mouth to mask ventilation. You still did not ventilate despite several doses of naloxone. A eight French feeding tube was used to intubate the trachea and it was attached to a 3 mm ET tube connector using tape to secure the connection. Again a small AMBU bag-valve resuscitator was used to provide ventilations but the little one still did not begin to ventilate spontaneously. Therefore Hollowell anesthetic ventilator was used to provide a means of positive pressure breathing using a pediatric circuit attached to the 3 mm ET tube connector. The lowest settings on the ventilator was used but this was still large an amount of oxygen to be given with each ventilator given breath. So this was remedied by making a hole on the inhalation side of the tubing going to the Y connector. The hole was continued to be made until the end of the Y connector and an attached thumb of a surgical glove provided just enough airflow that the surgical thumb tip would expand slightly as the ventilator circuit kicked on and gave a breath. The Y connector was then attached to the 3 mm ET connector and the chest rise of the newborn was observed as the ventilator kicked on. The patient was then left on the ventilator throughout the night and on into the next day, and he was able to be weaned off the ventilator and then supplemented with oxygen using a 3 Fr. nasal oxygen catheter. The dog recovered from the ordeal and became a national champion (as per conversations had with the veterinarians that had provided most of the care for the newborn until discharge. The owner was also a veterinary technician that worked at the hospital.

Use of an intensive care ventilator is very important and a critical piece of equipment necessary for intensive care of many of the most severe pulmonary cases. Many can you purchase used. It is recommended that when making a purchase that an air compressor be involved as then the amount of oxygen delivered can be tailored to just what is necessary. Some of the newer models do not require oxygen and air mixes outside the ventilator. It is beyond the scope of this paper to go into all the modalities of the use of ventilator support in sick patients, but suffice it to say that positive pressure support with simultaneous intermittent ventilation time with the patients ventilations are ideal. Units to provide humidification her also recommended.

 The modality of high-frequency or high-frequency jet ventilation are also effective in some patients. Even in some of the older mechanical ventilators can be placed into a high-frequency mode ands may be of help in some patients who continue to be tachypneic. High frequency oscillatory ventilation allows movement of gases with the homeowner tree to be done without increasing airway pressure. The author has used these on occasion. Some people believe that they are very effective, especially in the neonates in human pulmonary critical care.

**Alternatives to RSI** - 1. Continued non-invasive ventilation with the AMBU BAG, anesthetic machine, or ventilator; 2. Direct awake tracheostomy tube placement generally under mild sedation and the use of lidocaine. 3. Following careful general anesthesia following initial sedation and tracheal intubation. This is the most common method used. The procedure I will describe I have as a video on VIN

**Temporary Tracheostomy -**  in my experience I have been very impressed how placement of a tracheostomy tube has allowed time to get to the bottom of the problem. The tracheotomy also decreases the work of breathing (short straw breathing verses long straw breathing). It has been able to allow for tracheal toileting and clearing of the large airways via suctioning. It also allows the bronchial tree to be directly medicated. If a commercial tracheostomy tube is available that is OK however I have also found through the performance of well over a 100 tracheotomies that an endotracheal tube fashioned in to a tracheostomy tube also works well. It is cut along its top and bottom longitudinally and the balloon inflation mechanism spared. The plastic adaptor is reinserted and the long ends of the two split sections are cut off partway. Holes are placed through the split sections that are left. Through these holes are placed sections of IV tubing used to tie the tracheotomy tube in place. Tracheotomy tubes should be changed every 6-8 hours or as needed (blocked or near blocked). Pets with T-tubes in place must always be watched as obstruction can occur quickly.

**Our experience has shown that the institution of temporary tracheotomy tubes has been life-saving in the following cases:**

**Pneumonia that is progressive where this significant exudate as the T-tube allows for direct suctioning of the exudate, humidification, direct irrigation with saline as this is a mucolytic and micronebulization of antibiotics such a Amikacin in the treatment of Gram negative associated pneumonia such as Klebsiella pneumonia**. This organism is encapsuilated and a facultative anaerobic organism that now a very important cause of nosocomial pneumonia that often is very resistant to many other antibiotics. Having the ability to treat the lung infection by micronebulization of the amikacin via the trach tube has been a rescue technique in my hands. There are of course some Klebsiella organisms that seem to be resistant to every commercial antibiotic known. In this case the use of meropenem may produce the best bacterial clearing. Caution is advised with each of these serious infections and the organisms can be spread to humans as well so very good isolation and hygiene protocols be used.

Here is one example: A four month old female German Shepherd with severe pneumonia was referred after having seen several veterinarians who are diagnosed the significant pneumonia and had place the dog on several types of antibiotics. Despite the administration of the antibiotics the patient was getting worse. Radiographs revealed both right and left lung fields were significantly involved with and air bronchogram pattern. Consolidation was also seen in some areas of both right and left lung fields. A tracheotomy was performed under mild amount of sedation and a local block Pre oxygenation was performed and then suction applied. A large amount suppurative exudate was able to be removed and following irrigation with saline more exudate was aspirated. I went over to the local hospital and receive some nebulization and humidification equipment from the respiratory therapist who work there and took that back to the veterinary hospital and started treating the dog was intermittent ventilation with micronebulizer and added a tracheotomy cup or bowel that was attached to a humidifier. The culture and Gram stain of the exudate was done and systemic antibiotics were chosen based on what the Gram stain showed. It appeared that a gram-negative rod was most likely the prime organism involved in the infection and intravenous Amikacin was started as well as it being used in the micronebulization treatments that were being done every 4 hours. Oral Baytril was also administered. Intravenous fluids were also provided to keep the patient hydrated. The dog went on to have a successful recovery from the pneumonia after remaining in the hospital for three days at which time the tracheotomy tube was removed and the patient was discharged. She was seeing on a daily basis to continue with the injectable Amikacin as well as receiving of postural drainage, coupage and oral antibiotics as described prior.

**Trauma to the lung that leads to severe pulmonary edema. The tracheotomy tube then allows for direct suctioning of blood and edema fluid . Here is an example:**

Puppy” a 2 mo old Jack Russell “squashed by a horse” “stepped on bad”: He arrived with difficult breathing, and crackles; Pale mm, CRT > 5, HR 200+, no pulses to speak of; breathing was very labored; Jet-blow-by O2, all peripheral vessels flat, IV established by CD on the rt. jugular, kept providing bag-valve-mask ventilation and oxygen while IV hypertonic saline and hetastarch was provided in 5 ml /kg increments, still remained unconscious, so intubated and provided IPPV and took radiographs that showed all ling fields white out; Performed a tracheotomy to allow tracheal toileting of all the blood and foam; Suctioned out red foamy fluid; Placed on an old Bird ventilator for 40 hrs using pentobarbital at 1 mg.kg.hour to keep him still. (now as there is no pentobarbital available we use hydromorphone or fentanyl as a CRI and acepromazine or midazolam **as a CRI as well)(Rarely need to paralyze the patient to allow the ventilator to do its work** and not have the patient buck it so hard). Were able to wean off and pull the ET trach and yet allowing the trach tube to be near so to still be able to replaced if it were necessary. Placed on a nasal O2 catheter + some CPAP (periodically for a day). Received Norm-R, Blood (fresh whole), HS and hetastarch. He went home day 4 post injury. The trach site healed well by second intension. A follow-up call at 6 month and a year later: Owners said doing well!

**Congestive heart failure that leads to severe and fulminant pulmonary edema as the tracheotomy tube allows for micronebulization of bronchodilators and suctioning of edema fluid and for ventilation of the edematous lung;**

**Trauma to the mouth, larynx or trachea that leads to edema and decreases in the airway diameters to the point of causing dyspnea**

**Brachycephalic syndrome that has the patient now in a respiratory crisis – Here is an example:** 4 month old English Bulldog that ate a large soup bone which got lodged in the esophagus and caused him to panic as he was unable to accommodate the bone in the proximal esophagus and the elongated soft palate. The airway was thick with ropey saliva and suctioning was to no avail. He was given butoprphenol and ketamine to allow him to be quiet and the examination showed a very severe laryngeal compromise with severe averted laryngeal saccules and a severe elongated soft palate; difficulty occurred in gaining an ET tube placed into the airway as swelling was also significant. A tracheotomy was completed and then the esophageal foreign body was able to be pushed into the stomach. It appeared to be mostly gristle. The dog then underwent elongated soft palate resection and removal of the significant everted laryngeal saccules and recovery was uneventful as the trach was able to provide an open airway and as the swelling went down, in two days, the trach tube was able to be removed and the site healed by second intension.

**Trauma to the head and neck - blunt or penetrating - Here is an example:**

Sara, the dog with a large hole in her head that she was breathing through: Its about 7 PM when we were all about to leave the hospital after a long day. You are tired! A man carrying in a Golden Retriever. Her name is Sara he says about 10 years old. She is

motionless, unconscious, and dripping blood from nose. This can be seen easily as the blood is dripping at a fairly brisk rate. As you do a further survey you see the man appears in emotional shock. He says his dog was just hit by a car a block away and says “Do all you can to save her. She’s my life!” He works as an accountant for the state he says. He is dressed in a suite and tie. His white shirt is covered in blood. On initial primary survey you note the following: A = bubbling – gurgling- large hole noted in the frontal sinus; B = breathing through this hole but not very effective; seems like little air is moving from the mouth and there is bubbling and gurgling sounds coming from the mouth; D = left eye appears possibly ruptured but difficult to tell because of all the blood covering her face; there is a poor but present gag reflex noted as an ET tube tries to be inserted. There’s so much blood that it is difficult to see her rima glottis; she is not responding otherwise and breathing efforts are poor; it is assumed that she has a traumatic brain injury (TBI); E = no other obvious injuries are noted 🡪 Immediate therapy is started by providing jet blow-by oxygen to her mouth and face and head as ts been noted that with brain injury with a compromised airway oxygen is needed within minutes to prevent hypoxia.

Research has implicated that a lack of oxygen to brain cells leads to excitotoxicity, and oxidative stress. (Perlman JM. Pathogenesis of hypoxic-ischemic brain injury Journal of Perinatology (2007) 27, S39–S46). With TBI that was also apparent with Sara there is primarily and secondarily associated massive release of excitatory amino acid neurotransmitters, particularly glutamate. This excess in extracellular glutamate availability affects neurons and astrocytes and results intra-stimulation of ionotropic and metabotropic glutamate receptors with consecutive Ca2+, Na+, and K+-fluxes.Although these events trigger catabolic processes including blood–brain barrier breakdown, the cellular attempt to compensate for ionic gradients increases Na+/K+-ATPase activity and in turn metabolic demand, creating a vicious circle of flow–metabolism uncoupling to the cell.

Oxidative stress relates to the generation of reactive oxygen species (oxygen free radicals and associated entities including superoxides, hydrogen peroxide, nitric oxide, and peroxinitrite) in response to TBI. The excessive production of reactive oxygen species due to excitotoxicity and exhaustion of the endogenous antioxidant system (e.g. superoxide dismutase, glutathione peroxidase, and catalase) induces peroxidation of cellular and vascular structures, protein oxidation, cleavage of DNA, and inhibition of the mitochondrial electron transport chain.Although these mechanisms are adequate to contribute to immediate cell death, inflammatory processes and early or late apoptotic programmes are also induced by oxidative stress.

Therefore most guidelines are to provide supplemental oxygen as rapidly as possible to patients suffering from TBI or other hypoxic to anoxic associated conditions so that there might be an interruption of the build up all the elements that cause further brain cell disruption. (Bateman NT, Leach RM. ABC of oxygen: acute oxygen therapy. *BMJ* 1998; 317: 798-801).

Sara received the following: She was first treated with supplemental oxygen by blow by jet to her face as mentioned earlier; A quick discussion was accomplished with the owner and written permission with a estimate was signed for further emergency care including surgery and postop supportive care as required; Because of the significant airway compromise with the blood in her airway and the TBI a resuscitative tracheostomy was performed; Then ventilation was started, first by use of an AMBU bag-valve and reservoir and then she was switched to an anesthetic machine with a mechanical ventilator (Hallowell); A mini-cutdown and the placement of a 14 g IV catheter was then done and 7,5% hypertonic saline and 6% hetastarch in saline was administered at 5 ml/kg; Oxyglobin, a Hemoglobin Based Oxygen Carrier (HBOC)(Biopure) at 5 ml/kg and fresh frozen plasma (FFP)was also administered at 5 ml/kg; Plasmalyte-A at a 15 ml/kg bolus and Hetastarch alone at a 2 ml/kg boluses every 15 minutes were given as needed to produce a Doppler blood flow that was audible to the point that a dichroitic (tish tish) sound with each heart contraction was heard and systolic blood pressure (BP) was adequate at 60 mmHg. NOTE: a slight amount of isoflurane was begun and ketamine 2 mg/kg and hydromorphone 0.2mg/kg

Acepromazine 0.02 mg/kg was given intravenously; This was followed by IV atricarium 0.25/kg, and together with the isoflurane, it was felt that adequate hypnosis, analgesia, and muscle relaxation were achieved. Preoperative IV cefazolin at 40 mg/kg and enrofloxicin at 10mg/kg were administered. The OR was already in a state of readiness as we always set it up right after each previous surgery.

The Operating Room – Readiness Protocol is the following:

All set up for a major surgery and anesthesia (machine and ventilator)

Emergency drugs: atropine/glyco, epi., P-lyte, HS

Suction with canister + suction tube set

Electro-surgery and “pencil” ValleyLab - Foremost

Monitor (ECG, ETCO2 , SPO2, Temp, BP)– Blue Tooth; a Doppler

Gowns, gloves, major pack, drapes, suture laid out

Warming blanket (Chill Buster), towels, plastic bag

IV pole, fluid warmer, “blood stop”, etc.

Surgical headlight and loupes and juice boxes (for quick energy)

Sara was taken to surgery after the areas were clipped and prepped w/ Techni-Care a product containing chloroxylenol 3% (Care-Tech Labs 1- 800-325-9681). After draping

The nasal and frontal areas were explored and many free sections of bone from the frontal sinus required removal and other sections of bone just moved back into place.

The bleeding became severe at one point while removing and replacing the sections of bone and the frontal sinus and nasal cavity required packing and it was noted that there was a crack in the ethmoid region. The bleeding stopped with the pressure packing.

It was noted that there were internal contents of the left globe in the sinus cavity. All the contents and debris were removed and an enucleation of the rest of the eye was completed. A doubled sheet of commercially available porcine submucosa material from the small intestine was then used to cover the opening over the nasal passage and frontal cavity (A-Cell). Skin and subcutaneous tissue was able to then be advanced over the A-Cell cover and closed.

Post-operative care involved the following (briefly listed only): Ventilated for 4 hours with mild positive end expiratory pressure (PEEP) of 5 cmH2O; Respiratory and physical therapy; A continuous rate infusion (CRI) of Lidocaine, ketamine, morphine, and acepromazine; Enrofloxicin, metronidazole, and ampicillin IV; Partial parenteral feeding 2mg/kg/hr using FreAmine 3.5% with electrolytes; Trickle enteral feeding beginning 4 hr post-op, giving 2 ml per Kg slowly every 4 hours; Critical care continuous monitoring for 24 hr and HBOT was provided every 12 hours for 2 days; Discharged after several days with strict confinement. Remove the staples in the head, eye, and nasal regions in 2 weeks post injury and she was doing well. The tracheotomy had been left to granulate closed with only ostomy wound cleaning with saline needed.

Modalities critical in patients with respiratory difficulty include the following:

Thoracocentesis; both done with a needle or with a catheter

Placement of small bore with guide-wire thoracic drainage catheters

Placement of large bore chest tubes without the use of the trocar

NOTE: In the opinion of this author it is important and vital never to use a trocar point in pushing a chest tube through tissues and into the pleural space… ever. There have been major instances where the use of trocars has caused the death of both dogs, cats and humans. It is always best to gently penetrate the chest wall with a well controlled dissection with a curved hemostat and then allowing the lung just under the penetration area to deflate. That allows the chest tube to be placed into the pleural space without it causing any injury to the lung. Then with aspiration on the chest to the lung will re-inflate

The use of an underwater suction seal and drainage system is also critical in patients in which the lung continues to leak either blood or air following some type of penetration or injury. An indication for its use would be if there is either air or blood that continues to leak and have to be removed by periodic aspiration of the chest tube. Fortunately in most cases if continuous suction approximately 20 cm is placed on the plural space the leaking lung will generally re-expand and over the course of 24 to 48 hours the leak will stop. Having only a stopcock on the end of the chest tube and using a syringe to evacuate the air and blood intermittently may prevent the lung from expanding to the point of sealing and continue to stay sealed.

Two critical techniques that the will be addressed that has been found to be very successful are the use of either transtracheal oxygen or nasotracheal oxygen provided to the upper respiratory compromised patient with laryngeal paralysis. These techniques offer a way to support the dog in a relatively noninvasive way until the patient can have the tieback procedure done. The procedure also works on patients with tracheal collapse or laryngeal collapse as an effective stopgap measure that can be done and will lessen the stress these patients have while waiting until a more definitive procedure can be done. The principle is simple in that the insufflation of oxygen is provided distal to the area where the anatomic region of the blockage is. It is also critical when using his procedures to also keep the patient quiet and calm, as they cannot keep up with the oxygen demand in an excited or stress animal because of the limitations of the insufflation rate.

The last critical techniques that will be addressed are the following:

1. Emergent resuscitative approach to the cervical trachea to gain access to a major tracheal disruption – as may occur in a dog fight.
2. Emergent resuscitative thoracotomy – to provide decompression of a rapidly expanding and worsening tension pneumothorax.
3. Emergent resuscitative parasternotomy that may be necessary for access to the thoracic trachea should there be either a tracheal tear and leak from an iatrogenic cause or one caused by penetrating trauma, or a luminal blockage that can not be relieved and endoscopy is not available or can not be done due to the severity and speed of onset of the obstruction.

All of these techniques will be rarely needed but should the need arises not much time may be available to provide a watch and wait attitude as catastrophic hypoxia is occurring. A rapid limited thoracotomy may be the most common procedure needed to deflate the rapidly progressive tension pneumothorax. In other cases the dyspnic animal may go a full arrest and in these cases only an open chest CPR technique may be successful in resuscitation. In some cases when there is severe blood loss from a thoracic injury or tumor rupture it will be necessary to perform aorta cross-clamping (done by placing a feeding tube around the thoracic aorta as it becomes the descending aorta and sliding a hemostat down on it to tighten the loop created. Then the blood flow will be to only the brain and heart and this will help in the prevention of further hemorrhage. The same maneuver can be used to stop hemorrhage and air leaks when involving lung lobes. The stoppage of a major air leak caused by the injury at a major bronchial segment will be critical as it would stop most all of the inhaled air from moving into the pleural space and direct it to go to the lung, thus treating effectively the severe mechanical hypoxia caused by a bronchopleural fistula.

Summary

There are many therapeutic procedures that are useful in the care of the difficulty breathing (dyspnic) patient. Most are simple to perform. Studies are lacking that provide answers to which ones are the most effective. Some of the procedures like the use of an oxygen cage, have really never been critically examined in a large research project, and it’s always been assumed that the use if these is best thing for the dyspnic patient. This presentation provided an overview of many other procedures that may be required to definitively treat dyspnea. In some cases certain conditions, such as tension pneumothorax, must be recognized and specifically treated in an urgent manner to prevent catastrophic consequences. Further critical analysis of many of the procedures that are used such as the use oxygen cages is required. It is hoped that this review may act as a start for everyone’s “critical analysis” of the techniques we do and think they are the best to do for our difficult breathing patients..…the use of oxygen cages for a start. “Unless we investigate we will never know”