

Case Examples of Mechanical Ventilation

Jon Palmer, VMD, DACVIM
New Bolton Center

Three cases



Case 1

- 50-day-old Morgan colt
- June 13
 - Normal in the morning
 - Evening found down in the field
 - Weak
- Rx
 - Intravenous fluids
 - Antibiotics
 - Tube fed milk
- June 14 6:00 a.m.
 - Respiratory distress
 - Cyanotic

Ventilation Case 1

- Admission PE
 - Weak, no eyelid tone
 - No tongue tone, weak tail tone
 - Shallow, rapid respiratory pattern
 - Mark nostril flare
- Therapy
 - Botulism antitoxin
 - Intravenous fluids
 - Intravenous ceftiofur sodium
 - Indwelling nasogastric tube
 - Ventilation



Case 1

	Adm	40 min	2 hr
pH	7.325	7.265	7.289
Pco ₂	56	68	70
Po ₂	40	229	243
SAT	64.5	99.7	99.7
HCO ₃	29.6	31.1	33.5
BE	+2.7	+2.6	+5.5
RA		10 lpm	10 lpm



Ventilator Set Up

- Goals
 - Decrease work of breathing
 - Maintain FRC
- Mode: Pressure Support with CPAP
 - PS initially set at 9
 - Normal lungs
 - CPAP initially set at 4
 - Normal lungs
- Parameters set by foal
 - Tidal Volume = 5.6 – 6.2 ml/kg (7 ml/kg)
 - RR 32
 - PIP = 18-20 mmH₂O

Case 1

	7 hr	HD 2	HD 2	HD 7		
pH	7.393	7.385	7.396	7.414		
Pco ₂	53	51	51	48	mode	PS
Po ₂	97	74	127	114	TV	550-680
SAT	96.7	92.8	98.4	98.4	RR	32 – 26
Cont		17	17.4	17.1	PIP	20 – 18
HCO ₃	32	31	31	31	P _{Plat}	24 – 18
BE	+6.5	+5.2	+6.0	+6.0		
FIO ₂	0.3	0.3	0.4	0.3		
PEEP	4	4	5	5		
ETCO ₂	53	48	49	48		
PS	9	11	11	11		



Problems

- $\text{ETCO}_2 = 0$
- Long inspiration
- Flow meter shows a dramatic \downarrow TV
- Common problem
 - Have foal sitter monitor cuff
 - Often slow leak
 - Bad valve – use hemostat or clamp
 - Leaking cuff – replace endotracheal tube



Case 1

	HD 9	HD 9
pH	7.304	7.443
Pco ₂	76	52
Po ₂	115	111
SAT	97.5	98
Cont	16.0	16.3
HCO ₃	38	36
BE	+9.6	+10.8
FIO ₂	0.35	0.35
ETCO ₂	72	52
Mode	PS	PS
PS	20	12





Weaning

- 1st weaning challenge HD 6
 - Off the ventilator
 - Good breathing efforts
 - ETCO₂ increased
 - Foal became cyanotic (on INO₂)
 - Aerophagia - increased abdominal size
 - 10 minute trial
- 2nd weaning trial day 8
 - After 22 minutes Paco₂ 48 → 60



3rd Weaning Attempt

	HD 10	3 pm	6 pm	3 am	6 am	HD 14
pH	7.443	7.365	7.394	7.338	7.031	7.420
Pco ₂	52	55	50	64	129	49
Po ₂	111	72	184	79	49	120
SAT	98	91	99	92	60	98
HCO ₃	36	32	31	34	34	32
BE	+10.8	+5.4	+5.5	+6.7	-1.3	+7.0
FIO ₂	0.35	8 lpm	10 lpm	8 lpm	0.5	4 lpm
ETCO ₂	52				84	
Mode	PS	off	off	off	PS	off

Outcome

- Successful weaning HD 14
- Standing day 15
- Dysphagia
 - HD 22 – able to swallow water
 - HD 23 – able to swallow solids
- Hospital Discharge HD 30



Case 2



Case 2

Clinical Problems

- Septic Shock
- Bacteremia/Sepsis
 - *Pantoea agglomerans*
- Neonatal Encephalopathy
 - Somnolent, Facial nerve paresis
 - Seizure-like activity
- Neonatal Enteropathy
 - Fetal diarrhea, dysmotility
- Neonatal Nephropathy
- Other problems
 - Urachitis, hepatomegaly
 - Linear dermal necrosis, patent urachus
 - Angular limb deformity



Case 2

	Adm	1 hr
pH	7.339	7.349
Pco ₂	60	58
Po ₂	44	144
SAT	77	100
Cont	13	15
HCO ₃	32	32
BE	+5.6	+5.9
INO ₂	RA	10 lpm



Chester

Neonatal Encephalopathy

- *4 hours*
 - *Respiratory effort decreased*
 - *Apneustic breathing (breath holding)*



Case 2

	Adm	1 hr	4 hr	5 hr
pH	7.339	7.349	7.284	7.354
Pco ₂	60	58	82	62
Po ₂	44	144	80	250
SAT	77%	100	97	100
Cont	13.3	14.9	15.4	16.4
HCO ₃	32	32	34	35
BE	+5.6	+5.9	+10.5	+8.2
INO ₂	RA	10 lpm	10 lpm	15 lpm



Caffeine for Central Hypoventilation

- Naturally occurring methylxanthine
 - Theophylline, aminophylline
- Mechanism of action
 - Mild, direct general CNS stimulant
 - Increases respiratory center output
 - Increases chemoreceptor sensitivity to CO₂
 - Cardiac stimulate - increases cardiac output
 - Increases renal blood flow
 - Mild diuretic

Caffeine for Central Hypoventilation

- Pharmacokinetics
 - Well absorbed from the GI tract
 - Good levels in foals given per rectum
 - Metabolized in the liver, excreted in urine
 - Plasma half-life is long in neonates (40 - 230 hr)
- Dosage - caffeine base PO or PR
 - Loading 10 mg/kg - may repeat 2-3X
 - (Maintenance 2.5 mg/kg SID)
 - 2 mg caffeine citrate = 1 mg caffeine base



Caffeine for Central Hypoventilation

- Response monitored through ABG
- High therapeutic index
 - Effective blood levels 5-20 $\mu\text{g/ml}$
 - Toxic levels $> 40 - 50 \mu\text{g/ml}$ in humans
 - Safer than aminophylline
- Adverse effects
 - Hyperactive - more difficult to manage
 - Tachycardia - have not seen

Case 2

Neonatal Encephalopathy

- *10 hours*
 - *Apneic respiratory pattern*
 - *40 second apneic period*
 - *Cluster breathing in-between*



Case 2

	Adm	1 hr	4 hr	5 hr	10 hr	12 hr
pH	7.339	7.349	7.284	7.354	7.276	7.451
Pco ₂	60	58	82	62	85	45
Po ₂	44	144	80	250	105	141
SAT	77%	100	97	100	100	100
Cont	13.3	14.9	15.4	16.4	16.1	15.5
HCO ₃	32	32	34	35	40	32
BE	+5.6	+5.9	+10.5	+8.2	+11	+7.4
INO ₂	RA	10 lpm	10 lpm	15 lpm	10 lpm	10 lpm

Chester

Neonatal Encephalopathy

- *12 hours*
 - *Periods of somnolence and nonresponsiveness*
 - *Apneic respiratory pattern with cluster breathing*
 - *Facial nerve paresis*
 - *Right ear lower and slower to respond*
 - *Ears are not synchronized*
- *21 hours*
 - *Seizure-like activity*
 - *Opisthotonus*
 - *Tonic/Clonic marching activity*
 - *Treated with intravenous phenobarbital*

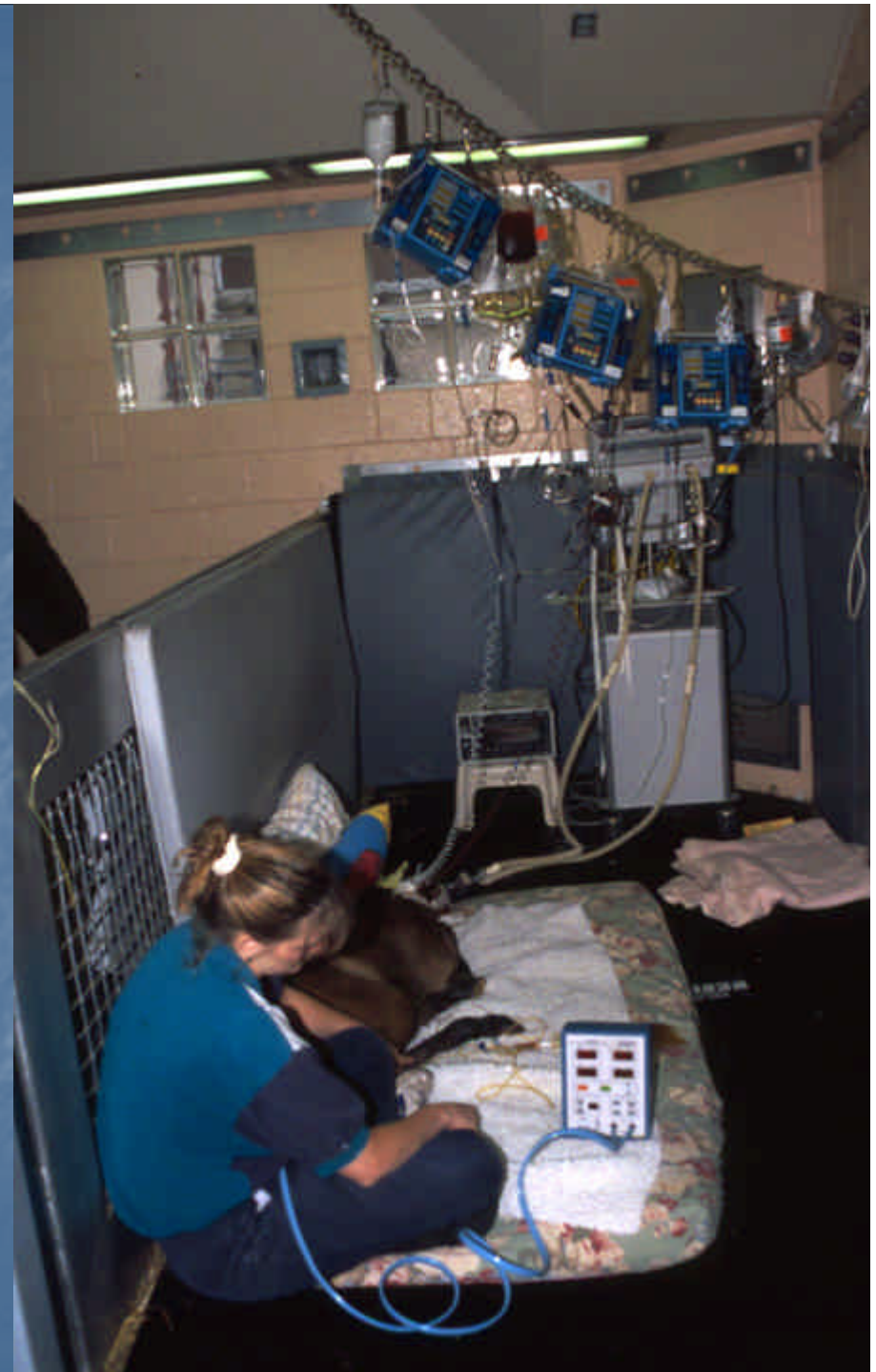
Case 2

	27 hr	29 hr
pH	7.313	7.269
Pco ₂	75	85
Po ₂	118	119
SAT	100	100
Cont	14.5	14.7
HCO ₃	38	39
BE	+10.4	+10.3
INO ₂	10 lpm	10 lpm



Ventilate

- Goals
 - Increase alveolar ventilation
 - Maintain FRC
- Mode: SIMV/PS with PEEP/CPAP
 - TV = 460 ml (8.5 ml/kg)
 - PIP = 18 cmH₂O
 - PS initially set at 9 cmH₂O
 - Normal lungs
 - PEEP/CPAP = 4 cmH₂O
 - Normal lungs
 - Peak flow = 60 lpm
 - RR = 24
 - Foal's rate 33
 - FIO₂ = 0.4



Case 2

	27 hr	29 hr	31 hr	36 hr		
pH	7.313	7.269	7.353	7.428	mode	SIMV
Pco ₂	75	85	67	50	TV	460 ml
Po ₂	118	119	96	164	PF	60
SAT	100	100	99	100	RR	36
Cont	14.5	14.7	13.9	14.4	PEEP	4
HCO ₃	38	39	38	33	PS	9
BE	+10.4	+10.3	+10.9	+8.3	P _{peak}	24
FIO ₂	10 lpm	10 lpm	0.4	0.5	P _{plat}	18
ETCO ₂			54	46		

Weaning

- Began asking when? within 12 hours
- After 21 hours – PS trial

Case 2

	48 hr	52 hr	57 HR	60 hr	48 hr	52 hr
pH	7.447	7.473	7.392	mode	SIMV	PS/CPAP
Pco ₂	45	40	54	TV	460 ml	520-75
Po ₂	242	91	252	PE	60	
SAT	100	100	100	RR	36	22
Cont	14.5	14.1	14.3	PEEP	4	4
HCO ₃	31	29	33	BS	9	6
BE	+7.2	+6.0	+7.7	P _{peak}	21	
FIO ₂	0.5	0.35	10 lpm	B _{plat}	16	
ETCO ₂	42	38				



Case 3 Septic Shock



Case 3

- Admission - 8 hr old
- Septic shock - *Streptococcus* bacteremia
 - Minimally responsive
 - Hypothermic (98.2 F)
 - Hypotonia
 - Pupils were pinpoint, iris edema
 - Inappropriately low heart rate
 - Cold legs, and poor peripheral perfusion
- Admission lab work
 - Leukopenic (WBC = 528 cells/ul)
 - Cortisol of 31 ug/dl
 - Hypoglycemia – required 20 mg/kg/min to get > LO

Case 3 Therapy

- Intranasal oxygen
- Shock doses of fluids
- Plasma
- Antimicrobials
- Ventilation
- Dobutamine
- Norepinephrine



Benefits of Mechanical Ventilation

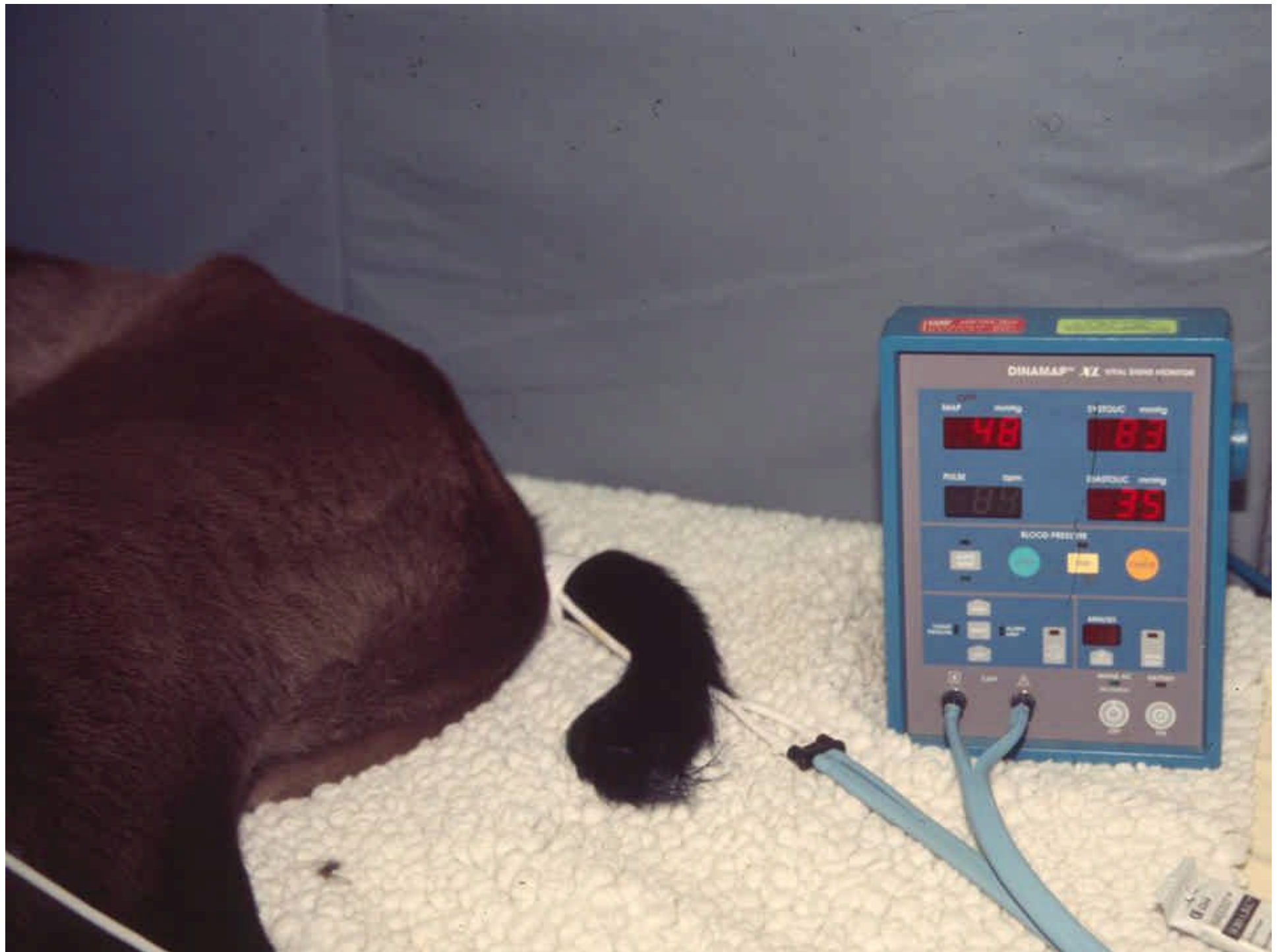
- Traditional
 - Improve gas exchange
 - Improve V/Q matching
 - Decrease shunt fraction
- Benefit of decreasing work of breathing
 - Normal quiet breathing
 - Inhalation active process
 - Requires energy
 - 3% - 5% O₂ consumed
 - Exhalation is a passive
 - Requires no energy, O₂

Benefits of Mechanical Ventilation

- Pulmonary failure 2ndary to septic shock
 - Respiratory distress
 - Work of breathing
 - O₂ required up to 50% of available O₂
 - Diverts perfusion resources
 - Accessory muscles recruited
- Relieving work of breathing
 - Redistribution of O₂
 - Redistribution of perfusion
 - Sparing energy resources
- Ventilation foals with septic shock
 - Improve perfusion, increase BP
 - Improved glucose balance







Case 3



Ventilate

- Goals
 - Decrease the work of breathing
 - Correct pulmonary hypertension
 - Maintain FRC
- Initial settings
 - Mode: PS with CPAP
 - PS initially set at 18 cmH₂O
 - Based on ease of breathing and resulting TV
 - PEEP/CPAP = 8 cmH₂O
 - FIO₂ = 1.0
- Set by foal
 - TV = 180 ml (7 ml/kg)
 - PIP = 32 cmH₂O
 - RR = 48



Case 3

	Adm	1 hr	1.5 hr	Mode	PS
pH	7.220	7.072	7.073	TV	180
Pco ₂	65	70	62	RR	48
Po ₂	22	20	248	PEEP	8
SAT	27.7	21.2	99.6	PS	18
Cont	5.1	3.4	14.9	P _{peak}	32
HCO ₃	27	20	18		
BE	-1.7	-9.6	-11.3	NO	20 ppm
FIO ₂	0.21	1.0	1.0		

Case 3

	3.75 hr	4 hr	5 hr	6 hr	Mode	PS
pH	7.000	7.062	7.089	7.061	TV	220
Pco ₂	96	92	84	98	RR	45
Po ₂	73	25	146	96	PEEP	10
SAT	86.3	29.4	97.8	92.3	PS	24
Cont	11.4	4.1	14.1	13.2	P _{peak}	35
HCO ₃	24	26	26	28		
BE	-7.5	- 4.6	- 4.6	- 3.1	NO	20 ppm
FIO ₂	1.0	1.0	1.0	0.50		

Case 3

ADV

	1 hr	1.5 hr	3.75 hr	4 hr	5 hr	6 hr
Pco ₂	70	62	96	92	84	98
ETCO ₂	34	34-54	46-64	33	43-56	25-33
ADV	51%	13-45%	33-52%	64%	33-49%	66-75%

Case 3

- Multifocal necrotizing interstitial pneumonia



Pulmonary Hypertension

- Sequela to many cases of ALI
- Increased pulmonary vascular resistance
 - Inflammatory mediators
 - Severe hypoxemia

Pulmonary Hypertension

- Neonate
 - Right to left shunting
 - Foramen ovale
 - Ductus arteriosus
 - Reversion to fetal circulation
 - Adaptive advantage
 - Achieve adequate systemic cardiac output
 - Neonate's unique ability
 - Exist in a hypoxemic state
 - Regain CO by shunting
 - Survive pulmonary hypertension without systemic ischemia

Pulmonary Hypertension

- 1.0 F_{IO_2} trial
 - $P_{aO_2} < 100$ torr after 15-20 min
 - Shunt fraction $> 30\%$
 - Cause of the hypoxemia extrapulmonary
 - Large cardiac shunt
 - PPHN

Pulmonary Hypertension

- Pulmonary hypertension
 - Failure to make the birth transition – PPHN
 - Imbalance of vasodilators and vasoconstrictors
 - Nitric oxide and endothelin
 - Regression to fetal circulation – PPHN
 - Perinatal hypoxemia
 - Cytokine showers
 - Secondary
 - Pulmonary disease
 - Septic shock
 - ALI

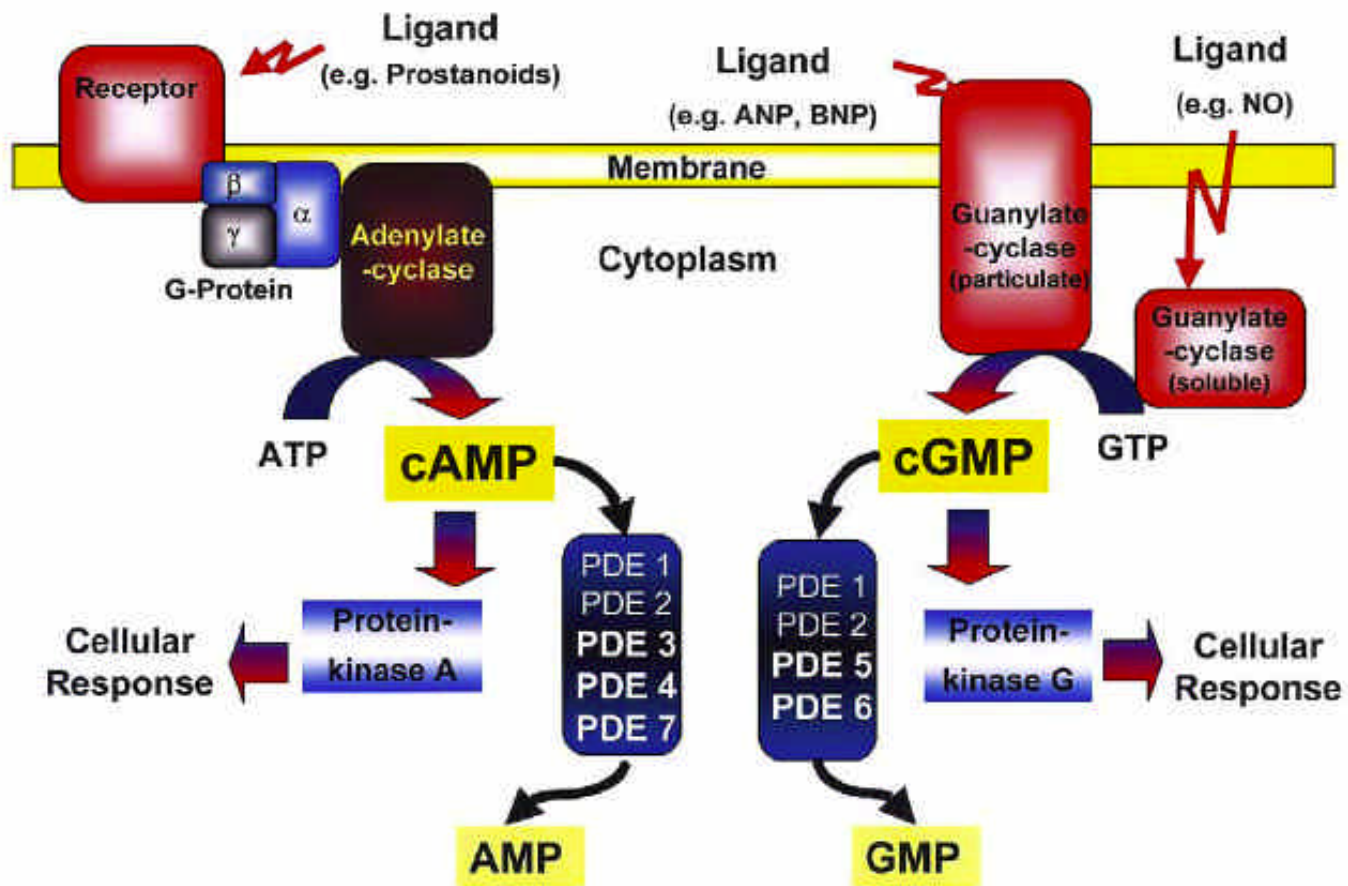
Pulmonary Hypertension Therapy

- Traditional therapy
 - Maximize exposure to O₂
 - Ventilation with 100% oxygen
 - Alkalinize arterial pH
 - Mild hyperventilation
 - Treatment with bases
 - Maintain systemic blood pressure
 - Counterbalance the pulmonary pressure
 - ALI will counteract these approaches
- Inhaled NO therapy
 - 5 to 20 ppm
 - Immediate effect
 - Significant pulmonary toxicity possible
 - Free radicals



Pulmonary Hypertension Therapy

- NO - Mechanism of action
 - Vasodilation
 - Increasing cGMP levels
 - Relaxation of the pulmonary vasculature
- Type V phosphodiesterase inhibitors
 - Selectively prevent cGMP destruction
 - Endogenous nitric oxide
 - Pulses of exogenous nitric oxide
 - Currently available
 - Sildenafil
 - Vardenafil
 - Tadalafil



From: Ghofrani HA, et. al. J Am Coll Cardio 44(7):1488-96, 2004



CASE EXAMPLES OF MECHANICAL VENTILATION

Jonathan Palmer, VMD, DACVIM

Director of Neonatal/Perinatal Programs, Chief Neonatal Intensive Care Service
New Bolton Center, University of Pennsylvania, Kennett Square, Pennsylvania

Clinical indications for mechanical ventilation in the neonate include persistent pulmonary hypertension, acute respiratory failure, neonatal encephalopathy associated weakness or central respiratory center failure, weakness associated with prematurity or IUGR, central or sepsis induced hypotension, septic shock and neuromuscular disorders such as botulism. Acute respiratory failure includes acute respiratory distress syndrome, organ dysfunction secondary to sepsis, infectious pneumonia, non-infectious pneumonia and trauma secondary to fractures ribs. The practical clinical approach to ventilating individual patients depends on the underlying reason for ventilation and the goal of therapy. Foals with botulism or central weakness secondary to neonatal encephalopathy or sepsis respond well to pressure support ventilation. Some patients with neonatal encephalopathy, sepsis or drug therapy causing central depression may respond well to SMIV whereas others to the combination of central respiratory stimulants (e.g. caffeine) and pressure support ventilation. Foals with septic shock, before the development of respiratory failure, often benefit from pressure support therapy. Foals with upper airway obstruction and secondary fatigue often do well on pressure support ventilation, if stenting the airway with the placement on an endotracheal tube is not sufficient. Examples of approaches to some of these situations will be given during the talk. In all of these situations, the approach to placing the patient on the ventilator and monitoring their response is similar as presented below.

Preparing to place a foal on a ventilator

The time between the decision to use mechanical ventilation as a therapeutic intervention and initiation of ventilation can be minimized by following a routine for ventilator setup. In clinics where mechanical ventilation of patients is a rare event, having a ventilator setup checklist with pictures, dry runs with the staff practicing setting up the equipment and sessions covering equipment troubleshooting procedures can minimize confusion and uncertainty during the heat of battle. Typical equipment needed include: the ventilator, access to oxygen (with minimal interruption of the patient's oxygen insufflation), access to medical grade compressed air or a compressed air generator, interface lines, gas blender (often built into the ventilator), capnograph with lines and adaptor, humidifying device (often an HME filter), ventilator circuit, endotracheal tube, sterile gloves, sterile lubrication, means of securing the endotracheal tube, stethoscope, self inflating bag (in case of an emergency) and most importantly, adequate trained help to restrain the foal, intubate the foal, secure the endotracheal tube and begin adjusting the ventilator settings as soon as the foal is intubated.

As part of setting up the ventilator, the circuit and other attachments should be inspected to be certain that everything is in proper working order and the circuit should be checked for leaks. This can be done by attaching an artificial lung to the circuit, occluding the exhalation port and charging the circuit with a manual breath or checking the ventilator's ability to maintain PEEP. It is convenient to select the initial ventilator setting after checking for leaks. It is also important to check the integrity of the endotracheal tube's cuff before intubation. Leaking endotracheal cuffs are a very common problem during ventilation. Sterile endotracheal tubes should be used to minimize introduction of nosocomial bacteria during intubation. While checking the cuff, care should be

taken to maintain sterility of the tube beyond the cuff inflation port. During set up, choice of heating and humidifying the delivered gas should be made and set up.

Monitoring during ventilation

The object of monitoring during ventilation is to allow dynamic adjustment of mechanical ventilation parameters, to understand and correct underlying pathophysiology, to prevent damage from the act of mechanical ventilation and decide if it is time to discontinue mechanical ventilation. Monitoring can take many forms and vary greatly in sophistication. The following are a selection of the most useful parameters which can be monitored by the minimum equipment which should be available in support of mechanical ventilation.

Arterial blood gas (ABG) measurement: Although noninvasive monitoring such as pulse oximetry and capnography can be useful, they are not a substitute for an ABG in a critical neonate. If these noninvasive techniques are being used when an ABG is drawn, comparison of the results recorded as the syringe is filling and understanding the underlying pathophysiology causing discrepancies between the 2 monitoring techniques can be very useful in understanding the clinical condition of the patient. Whenever an ABG is drawn, not only should ventilator settings be recorded to help with dynamic adjustments, the capnograph reading as the syringe is filling should also be noted. ABG should be drawn before beginning ventilation, within 30 minutes of initiation of ventilation and again after 2 to 3 hours of ventilation. Timing of further ABG samples should be taken as dictated by the initial samples and the clinical condition of the patient. Stable patients without primary pulmonary disease or cardiovascular instability (e.g. a botulism case) may only require 1 ABG a day. Others may require samples every few hours. The goal is to keep the $P_{aO_2} > 80$ torr and < 120 torr with $SAT > 92\%$ and to keep the $pH > 7.340$ but < 7.420 .

Capnography: Capnography can be a very valuable monitoring technique and should be used continuously during ventilation. It can be useful for simple, but vital matters such as detecting loss of endotracheal cuff integrity, an all too common problem during ventilation. In this situation, the end tidal CO_2 ($ETCO_2$) will drop to zero as if the foal is apneic but the cycling of the ventilator will suggest that apnea is not the problem. This scenario is often caused by a deflated endotracheal cuff with leakage of exhaled gases around the endotracheal tube. Capnography can also help diagnose and monitor complex pathophysiologic conditions.

In patients with normal hemodynamics and pulmonary function $ETCO_2$ will be 2 to 5 torr less than P_{aCO_2} , having a strong enough correlation to safely be relied upon as a surrogate for P_{aCO_2} . But in patients with severe lung disease or hemodynamic instability, the P_{ETCO_2} is not a good predictor of P_{aCO_2} because the difference between the two measurements varies with changing V/Q relationships in the lungs. In these cases the emphasis should be on more ABG measurements until the V/Q mismatch improves (improved hemodynamics and pulmonary function) and a more consistent relationship between $ETCO_2$ and P_{aCO_2} is established. Establishment of consistent relationship implies an improvement in the V/Q status of the patient.

$ETCO_2$ is a function of P_{aCO_2} , cardiac output, alveolar dead space ventilation (pulmonary perfusion), airway time constants, CO_2 production (metabolic rate) and bicarbonate therapy. Capnography can be used to determine the adequacy of alveolar ventilation, the patency and placement of endotracheal tube, the relationship of alveolar ventilation and pulmonary perfusion, cardiac output and pulmonary blood flow (especially useful during CPR) and proper functioning of the ventilator (especially if rebreathing of CO_2 is occurring).

The P_{aCO_2} is determined by the P_{CO_2} of all perfused alveoli (whether or not they are ventilated) and the $ETCO_2$ represents the P_{CO_2} of all ventilated alveoli (whether or not they are

perfused). So primary V/Q abnormalities will affect the gradient between P_{aCO_2} and $ETCO_2$ reflects alveolar dead space ventilation ($V/Q = \infty$). Alveolar dead space is the volume of alveoli that are ventilated but not perfused. It represents a failure of pulmonary perfusion and is the other end of the spectrum from shunting or venous admixture in the continuum of V/Q abnormalities. Since the gas leaving alveoli that are ventilated but not perfused will contain no CO_2 , it will have a diluting effect on the CO_2 leaving other areas of the lung, lowering the $ETCO_2$. The gradient between P_{aCO_2} and $ETCO_2$ will reflect the percent of alveolar dead space ventilation and can easily be calculated with the following formula: % alveolar dead space = $(P_{aCO_2} - ETCO_2/P_{aCO_2}) \times 100$. The 2 most common reasons for increased alveolar dead space ventilation are decreased perfusion secondary to decreased cardiac output and decreased perfusion secondary increased pulmonary vascular resistance as occurs during ventilation with increased alveolar pressure (PEEP level/peak airway pressure/average airway pressure) causing alveolar capillary compression.

Although usually the P_{aCO_2} - $ETCO_2$ gradient reflects alveolar dead space ventilation, since there are other factors affecting $ETCO_2$ levels, this is not always true. The $ETCO_2$ is a measure of the CO_2 in the last alveoli to empty. If the lungs have uniform conditions throughout most areas, this will reflect the average alveoli. But often there are areas of the lungs with different time constants (reflecting how quickly gas enters and leaves these alveoli). When there are areas of the lungs with long time constants, these alveoli will receive less ventilation so the P_{CO_2} in these alveoli will be higher and gas from these alveoli will be the last to leave the lungs and will heavily influence the P_{CO_2} in the end tidal gas, thus resulting in a $ETCO_2$ which is higher than the average alveoli. In fact, in some cases the $ETCO_2$ may be higher than the P_{aCO_2} . Thus the P_{aCO_2} - $ETCO_2$ gradient will underestimate the alveolar dead space ventilation. Inspection of the capnogram will readily reveal this situation. This is one of several reasons that in addition to $ETCO_2$ monitoring it is important to use capnography, which is the continuous measurement of exhaled CO_2 .

Capnography consists of continuous real time recording of CO_2 as measured from a sample taken at the ventilator end of the endotracheal tube either relative to time or to volume of exhaled gases. A volume capnogram is a graph of exhaled CO_2 relative to the volume of gas exhaled. There has been renewed interest in this mode of capnography with the advent of noninvasive cardiac output measurements using CO_2 excretion. Whether or not this technique, which appears valuable in patients with normal physiology, will live up to its promise when applied to critically ill patients remains to be proven. The clinically more common time capnogram graphs CO_2 relative to time throughout the respiratory cycle. In a time capnogram of a normal individual at the initiation of exhalation the CO_2 will be zero as the first gas to leave the airway will be from anatomic dead space (phase I). As anatomical dead space gas begins to mix with alveolar gas there is a sudden upstroke of the curve (phase II) which is almost at a right angle to the baseline. The up stroke rapidly reaches a plateau (phase III). The end of the plateau marks the end of exhalation and that point is the $ETCO_2$. The initial part of inhalation is marked by the down stroke which rapidly returns to baseline followed by a period with the CO_2 of zero (phase O).

Careful observation of the curve for variations from normal can help characterize abnormalities. An elevated base line and upstroke (phase II) indicate CO_2 rebreathing if it develops gradually. A sudden increase suggests contamination of sample cell with water, mucus or dirt. A prolonged or sloped upstroke which does not meet a clear plateau suggests an obstruction to expiratory gas flow (e.g. bronchospasm, obstructive pulmonary disease, kinked endotracheal tube) or leaks in the breathing circuit. Even in normal patients, the plateau, which is alveolar gas, usually has a slight positive slope, indicating a slight rise in CO_2 during expiration. There are 2 reasons for

this increase. First because CO_2 is being continuously excreted into the alveoli which are becoming progressively smaller as expiration continues, the last gas emptied from the alveoli has a higher concentration of CO_2 . Second, even in normal lungs there are a wide range of V/Q ratios in different areas of the lungs. Some alveoli have a higher V/Q ratio because they are more readily ventilated (have shorter time constants) so have a relatively lower Pco_2 . Others have a lower V/Q ratio because of under ventilation (have longer time constants) resulting in a relatively higher Pco_2 . The delayed emptying of these alveoli with low V/Q (high Pco_2) contributes to the rising slope of the plateau. Factors such as changes in cardiac output, CO_2 production, airway resistance and functional residual capacity (FRC) may further affect the V/Q status of the various areas in the lung, and thus influence the height or the slope of the plateau. The presence of a steep slope of this plateau indicates abnormalities in V/Q mismatch of the lung. The V/Q difference can be great enough that the ETCO_2 may be higher than the Paco_2 , which reflects the average perfused alveolar CO_2 . When there is a significant slope to the plateau phase, the Paco_2 - ETCO_2 gradient will underestimate the amount of alveolar dead space ventilation. .

Tidal Volume/Minute Volume: It is important to monitor actual tidal volume and minute volume rather than relying on the intended volumes, in order to monitor for leaks and ventilator malfunction. Also, in ventilator modes such as PSV, where the patient sets the tidal volume, which can change at any time, tidal volume monitoring is important. Most modern ventilators have built-in flow meters, but external flow meters are available for those that don't. One downfall of common turbine based flow meters (where gas moving past the sensor spins a turbine and the volume is measured by counting the revolutions) is that at high respiratory rates where the expiratory pause is too short to allow the turbine to stop spinning, tidal volume measurements can not be obtained.

Airway pressure: Many modern ventilators measure airway pressure, volumes and flows throughout the respiratory cycle and display pressure-volume (compliance) loops and flow-volume (resistance) loops. These graphs can be quite useful in adjusting and monitoring ventilator function. If these displays are not available, the most important airway pressures are peak inspiratory pressure (PIP), plateau pressure (P_{PL}) and the PEEP or baseline pressure. The PIP is the highest pressure usually at the end of inspiration. P_{PL} is the pressure after the end of inspiration when the gases have come to equilibrium and can usually be measured during the inspiratory pause, if the ventilator has this capability or by briefly occluding the expiratory port after full inspiration and recording the value once the pressure stabilizes (this technique will not work with PSV). The PEEP or baseline pressure can be measured between respiratory cycles, again when no air is moving.

PIP is the most frequently measured variable of ventilatory function during mechanical ventilation. It depends on lung compliance and airway resistance. Changes in the magnitude of PIP may reflect any of several potentially detrimental problems related to ventilation. In a practical sense, PIP should be considered an additional vital sign for patients on a ventilator. A sudden decrease in PIP suggests a major leak in the circuit but also can be caused by insufficient gas supply to the ventilator, inadvertent change in settings, unintended extubation, or failure or disconnection of the ventilator. Increases in PIP may indicate endotracheal tube occlusion by secretions or kinking, acute bronchospasm, pneumothorax, or conditions causing decreased lung compliance. High PIP may cause barotrauma and other acute lung injury. PIP is usually monitored by alarms which indicate high values or failure to reach a minimum PIP. The baseline pressure should be 0 or higher if there is a PEEP except during patient inspiratory efforts. Failure to maintain PEEP usually suggests a small circuit leak.

Compliance/Resistance: With measurement of tidal volume, PIP, P_{PL} and PEEP both effective compliance and airway resistance can be calculated. Effective compliance is the compliance of the lungs and the chest wall. Since chest wall compliance rarely changes acutely and since effective compliance does not require pleural pressure measurements, effective compliance is a readily attainable, clinically convenient parameter. Static effective compliance can be calculated by dividing the tidal volume by the difference between P_{PL} and PEEP. It will decrease if there is an abnormality of the chest wall (a flail chest), decrease in functional alveolar numbers as with pulmonary edema, pneumonia or atelectasis and for other similar reasons. It may also be used to determine the best PEEP and best tidal volume if serial measurements are obtained at trial settings and a response grid recorded.

Dynamic effective compliance can be calculated by dividing the tidal volume by the difference between PIP and PEEP. Dynamic compliance adds the effects of resistance to static compliance. Dynamic compliance will decrease with disorders of the airway, lung parenchyma, and chest wall. Dynamic compliance is less than static compliance when there is increased resistance such as with secretions in the airways or endotracheal tube, bronchospasm, or endotracheal tube kinking.

Changes in airway resistance are most easily detected by monitoring the difference between PIP and P_{PL}. As the difference increases, so does the airway resistance. PIP and P_{PL} pressures can not always be accurately measured with spontaneously initiated breaths, especially in PSV. In pressure support mode the airway pressure may be variably decreased by the magnitude of the patient's inspiratory effort. As the patient's effort increases and because the ventilator will cycle off based on flow rates and not volume or pressure, PIP may never reach a value close to that if the patient had the breath delivered by a volume or pressure machine breath. In fact, what is traditionally measure a PIP (at the end of inspiration) may actually be less than P_{PL}. Therefore, these measurements should only be made during a ventilator delivered, non-pressure support breath.

Endotracheal tube: Initially the endotracheal tube should be changed daily or more often in the face of increased airway resistance. Each time the tube is changed, the amount and quality of the discharge in the tube should be monitored. If the discharge is very viscous and difficult to clean from the tube, it suggests inadequate preconditioning of ventilator gases (usually too little humidity) which should be corrected. Increased quantity of clear discharge is also an indication of failure of preconditioning of gases. If the discharge becomes dark or sanguineous (in the absence of traumatic intubation) then infection should be suspected. In either case, periodic culturing of the lumen of the endotracheal tube is useful. Generally several microbes (2-6) can be recovered, even in the absence of significant disease. These isolates should not be viewed as invaders which need to be targeted with antimicrobial treatment, but rather as colonizers which represent the nosocomial population from which the next invader may originate, whether the route is via the respiratory tract, the GI tract or the intravenous access. If a secondary infection becomes evident, then these culture results gives weight to an educated guess of how to modify antimicrobial therapy before a definitive culture and sensitivity is available. Targeting these colonizers with antimicrobials before they invade is likely to select for more resistant nosocomial microbes. It should be remembered that cuffed endotracheal tubes provide a degree of protection from aspiration but this degree of protection is not complete. Studies in human medicine have found gastric contents in the bronchial secretions of approximately 25% of intubated patients.

Adapted with permission from: Palmer JE. Ventilatory Support of the Critically Ill Foal. Veterinary Clinics of North America: Equine Practice August 2005 21(2): in press.