KLINICKÁ PRAVIDLA, KTERÁ BYCHOM MĚLI VŽDY RESPEKTOVAT …

VENTILACE A OXYGENOTERAPIE

Pavel Dostál

Klinika anesteziologie, resuscitace a intenzivní medicíny
Universita Karlova v Praze, Lékařská fakulta v Hradci Králové
Fakultní nemocnice Hradec Králové

Obsah

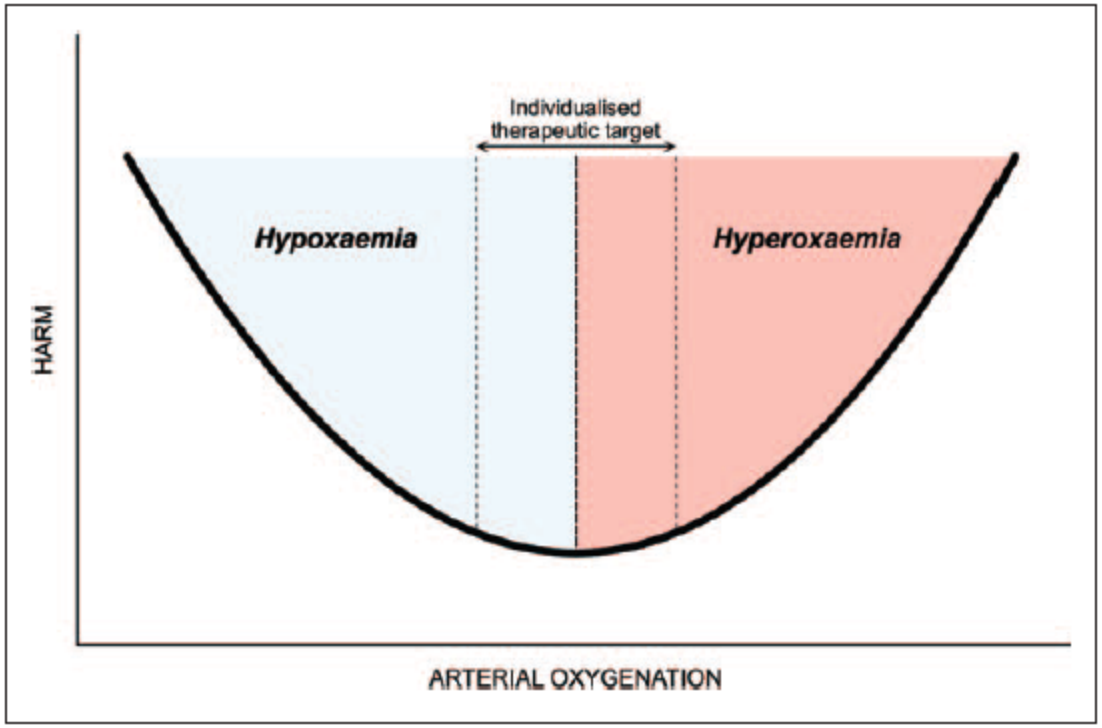
- Oxygenační cíle
- Ventilační cíle
- Nastavení ventilace

1. Oxygenační cíle

Oxygen Therapy in Critical Illness: Precise Control of Arterial Oxygenation and Permissive Hypoxemia*

(*Crit Care Med* 2013; 41:423–432)

Daniel Stuart Martin, BSc, MBChB, PhD, FRCA, FFICM[1,2];
Michael Patrick William Grocott, MBBS, MD, FRCA, FRCP, FFICM[1,3,4]



Permisivní hypoxémie

- Původně koncept plicní protekce při umělé plicní ventilaci (snížení rizika spojeného s vysokým PEEP a FiO_2)
- Akceptace hodnot SpO_2 < 90%
- Zdůrazňován více význam DO_2 a tkáňové oxygenace, než PaO_2
    - Pierson D. The future of respiratory care. Respir Care 2001; 47(7): 705-718.
    - Abdelsalam M. Permissive hypoxemia: Is it time to change our approach? Chest 2006; 129(1) :210-211.

Výšková hypoxie/hypoxemie

- **Klíčovým kompenzačním mechanismem je udržení obsahu kyslíku v arteriální krvi**
  - Hemokoncentrace
  - Alkalóza (posun disociační křivky)
  - Adekvátní srdeční výdej

- U nemocných v IP jsou již tyto mechanismy vyčerpány (Hb) nebo nejsou možné

Obsah kyslíku v arteriální krvi

- CaO_2 (ml/100ml) = 1,34 x Hb (g/dl) x SaO_2 + (0,003xPaO_2 (mmHg))
- Norma (Hb 15 g/dl) 20 ml O_2/100 ml krve
- Anémie (Hb 10 g/dl) 13,5 ml O_2/100 ml krve
- Hb 15 gd/l, SpO_2 90% 18 ml O_2/100 ml krve
- Anémie a SpO_2 90% 12,15 ml O_2/100 ml krve
- Hb 7 g/dl a SpO_2 95% 8,9 ml O_2/100 ml krve

- Hb 15 g/dl, SpO_2 75% 15 ml O_2/100 ml krve

Rizika permisivní hypoxémie

- Kongnitivní deficit
  - Zvýšená vnímavost po proběhlém inzultu
- Plicní hypertenze (oběhové selhání)
- Zvýšení renální vaskulární rezistence
  - Zvýšená vnímavost ledvin vůči dalším inzultům
- Iniciace zánětlivé odpovědi

Hypoxémie a stav vědomí

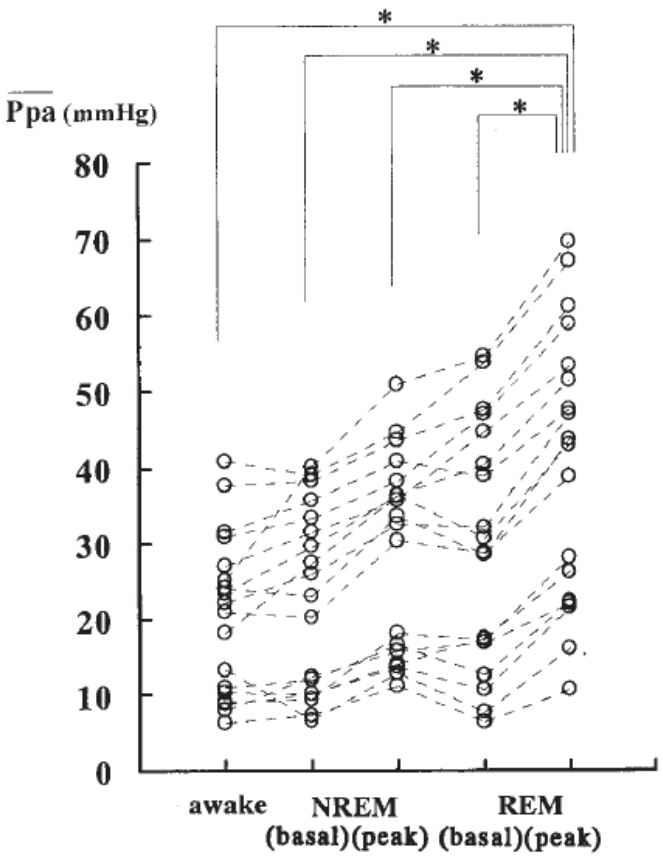
- Bezvědomí při poklesu PaO_2 pod 30-**20**-16 mmHg, důležitá rychlost vzniku
- Zhoršení krátkodobé paměti, poruchy vyjadřování, zhoršení jemné motoriky (nad 5000m)
  - Hornbein TF, Townes BD, Schoene RB, Sutton JR, Houston CS. The cost to the central nervous system of climbing to extremely high altitude. N Engl J Med. 1989;321:1714–9.
  - Regard M, Oelz O, Brugger P, Biol D, Landis T. Persistent cognitive impairment in climbers after repeated exposure to extreme altitude. Neurology. 1989;39:210–3.
- U pacientů s CHOPN PaO_2 35-50 mmHg kognitivní deficit
  - Andreou G, Vlachos F, Makanikas K. Effects of Chronic Obstructive Pulmonary Disease and Obstructive Sleep Apnea on Cognitive Functions: Evidence for a Common Nature. Sleep Disord. 2014; 2014: 768210.

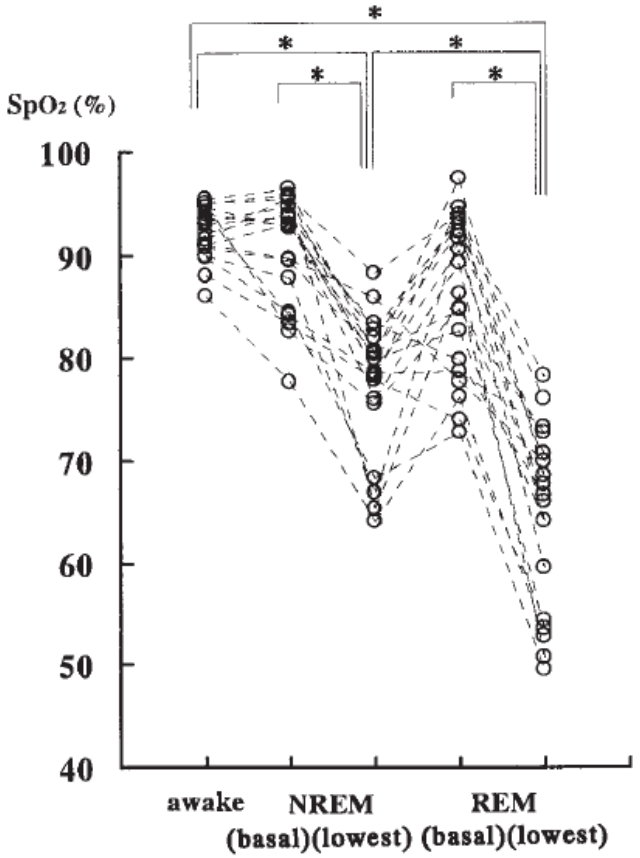
Hypoxií indukovaná plicní hypertenze

- Hypoxická plicní vasokontrikce při $PAO_2 < 75$ mmHg (2100 m nadmořské výšky)
- PaO_2 pod 60 mmHg spojena s aktivací hypoxické plicní vasokonstrikce
- Individuální variabilita, rozvoj plicní hypertenze v průběhu 6-8 h
- Augmentace hyperkapnii, především u nemocných s již přítomnou plicní hypertenzí

Manifestation of Pulmonary Hypertension During REM Sleep in Obstructive Sleep Apnea Syndrome

MAFUMI NIIJIMA, HIROSHI KIMURA, HIDENORI EDO, TOSHIHIDE SHINOZAKI, JIAN KANG, SHIGERU MASUYAMA, KOICHIRO TATSUMI, and TAKAYUKI KURIYAMA

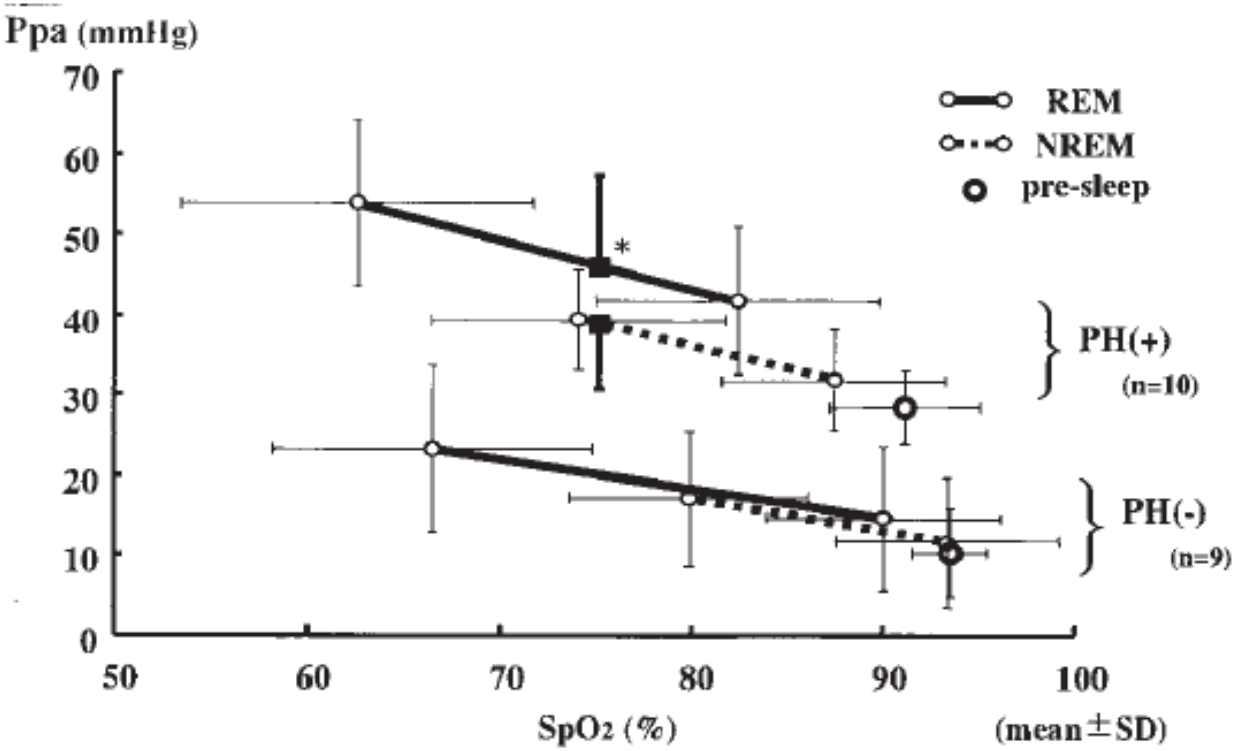




AM J RESPIR CRIT CARE MED 1999;159:1766–1772.

Manifestation of Pulmonary Hypertension During REM Sleep in Obstructive Sleep Apnea Syndrome

MAFUMI NIIJIMA, HIROSHI KIMURA, HIDENORI EDO, TOSHIHIDE SHINOZAKI, JIAN KANG, SHIGERU MASUYAMA, KOICHIRO TATSUMI, and TAKAYUKI KURIYAMA

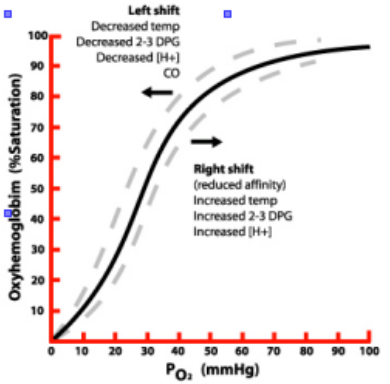


AM J RESPIR CRIT CARE MED 1999;159:1766–1772.

Individualizace permisivní hypoxémie?

- Laktát?
- Transaminázy, markery poškození ledvin ?
- Sledování PAP, CO, echo?
- Dynamika cytokinů?
- Stav vědomí?

Co lze (snad) akceptovat?



- Neexistují randomizované studie, extrapolace
- PaO_2 nad 65 až 70 mmHg nelze doporučit u spont. ventil. nemocných s chronickou hyperkapnii
- PaO_2 pod 30 mmHg vede k poruše vědomí
- PaO_2 pod 50 mmHg je spojeno s rizikem kognitivního deficitu
- PaO_2 50-60 mmHg je (snad) akceptovatelné u nemocných bez dekompenzovaného cor pulmonale

Neuropsychological Sequelae and Impaired Health Status in Survivors of Severe Acute Respiratory Distress Syndrome

AM J RESPIR CRIT CARE MED 1999;160:50–56.

RAMONA O. HOPKINS, LINDELL K. WEAVER, DONNA POPE, JAMES F. ORME, Jr., ERIN D. BIGLER, and VALERIE LARSON-LOHR

PEARSON CORRELATIONS OF NEUROPSYCHOLOGICAL DATA WITH CONTINUOUS PULSE OXIMETRY DATA

Neuropsychological Test	< 90%	< 85%	< 80%
WMS-R Attention/Concentration	$r^2 = 0.393^*$ $p = 0.004$	$r^2 = 0.332^\dagger$ $p = 0.016$	$r^2 = 0.328^\dagger$ $p = 0.018$
Full-Scale Intelligence Quotient	$r^2 = 0.462^*$ $p = 0.000$	$r^2 = 0.362^*$ $p = 0.008$	$r^2 = 0.340^\dagger$ $p = 0.013$
Verbal Intelligence Quotient	$r^2 = 0.376^*$ $p = 0.005$	$r^2 = 0.320^\dagger$ $p = 0.018$	$r^2 = 0.310^\dagger$ $p = 0.022$
Performance Intelligence Quotient	$r^2 = 0.466^*$ $p = 0.000$	$r^2 = 0.348^\dagger$ $p = 0.011$	$r^2 = 0.316^\dagger$ $p = 0.021$
Digit Span	$r^2 = 0.324^\dagger$ $p = 0.017$	$r^2 = 0.288^\dagger$ $p = 0.034$	$r^2 = 0.305^\dagger$ $p = 0.025$
Block Design	$r^2 = 0.360^*$ $p = 0.008$	$r^2 = 0.237$ $p = 0.087$	$r^2 = 0.185$ $p = 0.184$
Digit Symbol	$r^2 = 0.413^*$ $p = 0.002$	$r^2 = 0.341^\dagger$ $p = 0.013$	$r^2 = 0.206$ $p = 0.151$
Rey-Osterrieth Complex Figure Delay Recall	$r^2 = 0.311^*$ $p = 0.023$	$r^2 = 0.224$ $p = 0.104$	$r^2 = 0.173$ $p = 0.216$
Trails B T-score	$r^2 = 0.439^*$ $p = 0.001$	$r^2 = 0.325^\dagger$ $p = 0.018$	$r^2 = 0.344^\dagger$ $p = 0.012$

* Significant at ≤ 0.01.
† Significant at ≤ 0.05.

Průměrný čas s SpO_2
pod 90% 120 ± 144 h
pod 85% 13 ± 28h
Pod 80% 1 ± 3h

The Adult Respiratory Distress Syndrome Cognitive Outcomes Study

Long-Term Neuropsychological Function in Survivors of Acute Lung Injury

Mark E. Mikkelsen[1,2*], Jason D. Christie[1,2*], Paul N. Lanken[1], Rosette C. Biester[3,4], B. Taylor Thompson[5], Scarlett L. Bellamy[2], A. Russell Localio[2], Ejigayehu Demissie[1,2], Ramona O. Hopkins[6,7], and Derek C. Angus[8]

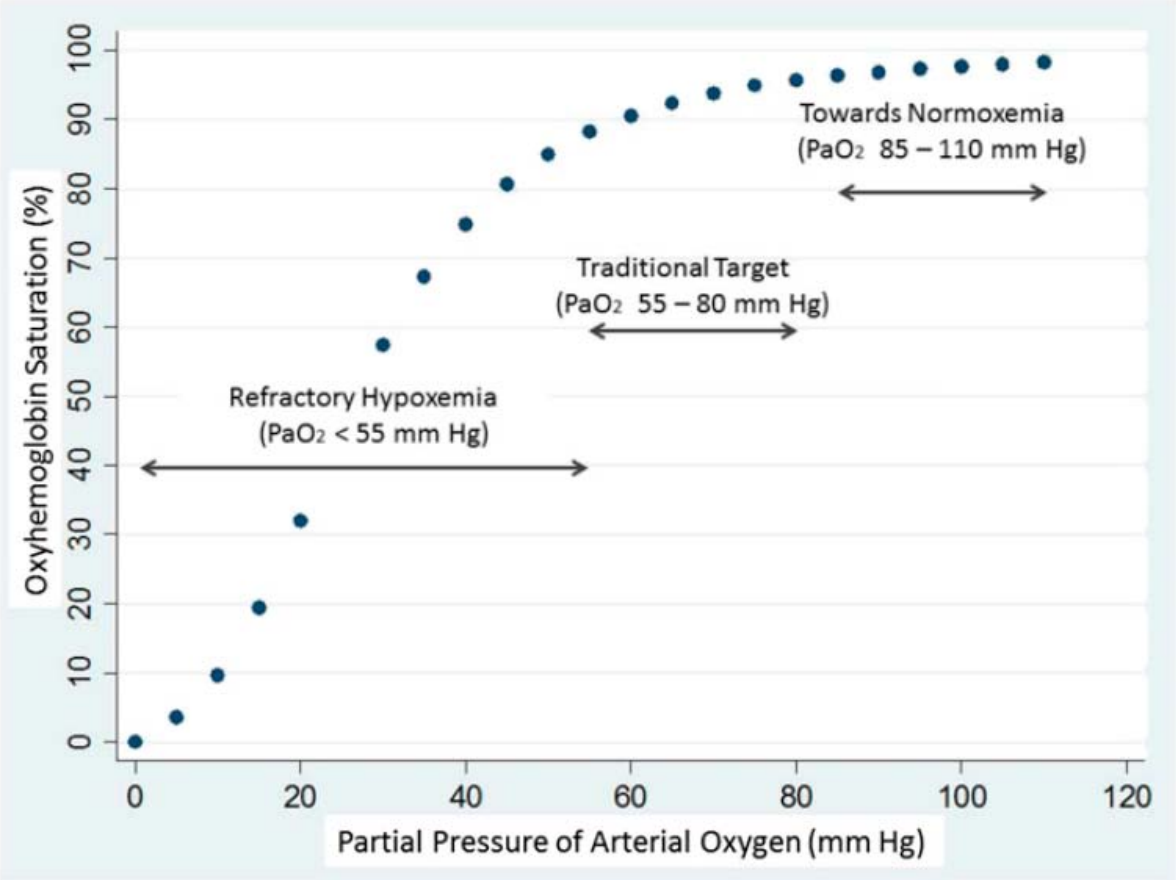
	Long-Term Cognitive Impairment				
	Not Impaired ($n = 34$)	Impaired ($n = 41$)	*P* Value	Unadjusted Odds Ratio (95% CI)	Adjusted Odds Ratio (95% CI)*
Candidate risk factors					
Duration of mechanical ventilation	8 (4–11)	6 (4–9)	0.43		
Conservative fluid-management strategy, %	32	66	0.004	4.03 (1.53–10.59)	3.35 (1.16–9.70)–5.46 (1.92–15.53)
Hypotension (hemodynamic data on-study)†					
Systolic blood pressure (mm Hg)	108 (102–113)	104 (96–112)	0.32		
Cardiac index (L/min/m^2)‡	4.5 (3.8–5.3)	4.5 (3.7–5)	0.49		
Shock, %†	32	29	0.77		
Vasopressor use	26	24	0.84		
Hypoxemia (respiratory variables on-study)					
Pa_{O_2}	86 (70–98)	71 (67–80)	0.02	1.56 (1.09–2.24)§	1.51 (1.01–2.26)–1.68 (1.14–2.49)§
Pa_{O_2}:$F_{I_{O_2}}$	152 (132–192)	157 (133–190)	0.63		
Oxygenation index	7.38 (4.55–10.42)	7.67 (5.97–10.02)	0.57		
Oxygen saturation (%)	95.1 (93.3–96.8)	94.2 (92.6–95.8)	0.10		

67-80 vs 70-98 mmHg

Am J Respir Crit Care Med Vol 185, Iss. 12, pp 1307–1315, Jun 15, 2012

Can We Optimize Long-Term Outcomes in Acute Respiratory Distress Syndrome by Targeting Normoxemia?

Mark E. Mikkelsen[1–3], Brian Anderson[1,2], Jason D. Christie[1,2], Ramona O. Hopkins[4,5], and Paul N. Lanken[1]



Ann Am Thorac Soc Vol 11, No 4, pp 613–618, May 2014

Can We Optimize Long-Term Outcomes in Acute Respiratory Distress Syndrome by Targeting Normoxemia?

Mark E. Mikkelsen[1–3], Brian Anderson[1,2], Jason D. Christie[1,2], Ramona O. Hopkins[4,5], and Paul N. Lanken[1]

	Traditional Target	Toward Normoxemia
Oxygenation target	55–80 mm Hg	85–110 mm Hg
Potential advantages	• Mitigate risk of hyperoxia-induced lung injury • Reduce duration of mechanical ventilation	• Mitigate risk of long-term cognitive impairment
Potential disadvantages	• Increase risk of long-term cognitive impairment	• Increase risk of hyperoxia-induced lung injury • Increase duration of mechanical ventilation

Ann Am Thorac Soc Vol 11, No 4, pp 613–618, May 2014

BTS guideline for emergency oxygen use in adult patients

B R O'Driscoll,[1] L S Howard,[2] A G Davison[3] on behalf of the British Thoracic Society

[1] Department of Respiratory Medicine, Salford Royal University Hospital, Salford, UK; [2] Hammersmith Hospital, Imperial College Healthcare NHS Trust, London, UK; [3] Southend University Hospital, Westcliff on Sea, Essex, UK

Correspondence to:
Dr B R O'Driscoll, Department of Respiratory Medicine, Salford Royal University Hospital, Stott Lane, Salford M6 8HD, UK;
ronan.o'driscoll@srft.nhs.uk

Received 11 June 2008
Accepted 11 June 2008

EXECUTIVE SUMMARY OF THE GUIDELINE

Philosophy of the guideline

- Oxygen is a treatment for hypoxaemia, not breathlessness. (Oxygen has not been shown to have any effect on the sensation of breathlessness in non-hypoxaemic patients.)
- The essence of this guideline can be summarised simply as a requirement for oxygen to be prescribed according to a target saturation range and for those who administer oxygen therapy to monitor the patient and keep within the target saturation range.
- The guideline suggests aiming to achieve normal or near-normal oxygen saturation for all acutely ill patients apart from those at risk

Monitoring and maintenance of target saturation

- Oxygen saturation and delivery system should be recorded on the patient's monitoring chart alongside the oximetry result.
- Oxygen delivery devices and flow rates should be adjusted to keep the oxygen saturation in the target range.
- Oxygen should be signed for on the drug chart on each drug round.

Weaning and discontinuation of oxygen therapy

- Oxygen should be reduced in stable patients with satisfactory oxygen saturation.
- Oxygen should be crossed off the drug chart

Oxygen should be prescribed to achieve a target saturation of 94–98% for most acutely ill patients or 88–92% for those at risk of hypercapnic respiratory failure (tables 1–3).

2. Ventilační cíle

Akutní hyperkapnie

- Náhodná
- Permisivní - 90.léta
  - Hickling, 1990, 1994
- Terapeutická – cca od konce 90.let
  - Laffey 1999

Patofyziologické změny

- Zvýšení CBF, zvýšení ICP
- Zvýšení dechové aktivity, nárůst dechové práce
- Zvýšení srdečního výdeje
- **Vazokonstrikce v plicním řečišti, zvýšení afterloadu PK**
- **Periferní vazodilatace, tachykardie, hypotenze**
- **Hypoxémie**
- Respirační acidóza, posun disociační křivky Hb
- Reabsorbce bikarbonátu v ledvinách a metabolická alkalóza

Resuscitation from Severe Acute Hypercapnia*

Determinants of Tolerance and Survival

Ralph T. Potkin, M.D., F.C.C.P.; and Erik R. Swenson, M.D.

A 46-year-old man underwent cosmetic facial surgery under general anesthesia. He was ventilated by mask with an oxygen-enriched gas mixture for 4 to 6 h and monitored by pulse oximetry. Despite adequate arterial saturation (SaO_2 >90 percent) throughout the procedure, he remained in a deep coma after termination of anesthesia. Initial arterial blood gas analysis revealed a pH of 6.60 and a $PaCO_2$ of 375 mm Hg. The patient was intubated and placed on mechanical ventilation. As his respiratory acidosis resolved, he regained consciousness quickly and recovered without any neurologic deficits. This case of record extreme hypercapnia and review of the literature demonstrates that survival is possible in acute severe respiratory acidosis as long as tissue anoxia and ischemia are prevented. We discuss the tissue effects of acute hypercapnia and newer aspects of the nature of intracellular pH regulation in critical tissues that afford considerable tolerance to acidosis. The dependence of these mechanisms upon active ion transport underscores the importance of adequate tissue oxygenation and perfusion. ***(Chest 1992; 102:1742-45)***

Table 1—*Arterial Blood Gas Data during Successful Resuscitation from Severe Hypercapnia*

Time	pH	$PaCO_2$, mm Hg*	PaO_2, mm Hg*	HCO_3^-, mEq/L	Base excess, mEq/L
Admission	6.60	375	40	34	−16
5 min of mask ventilation	6.91	151	244	29	−9
25 min of mechanical ventilation	7.08	68	56	25	−7
90 min of mechanical ventilation	7.19	58	65	21	−5

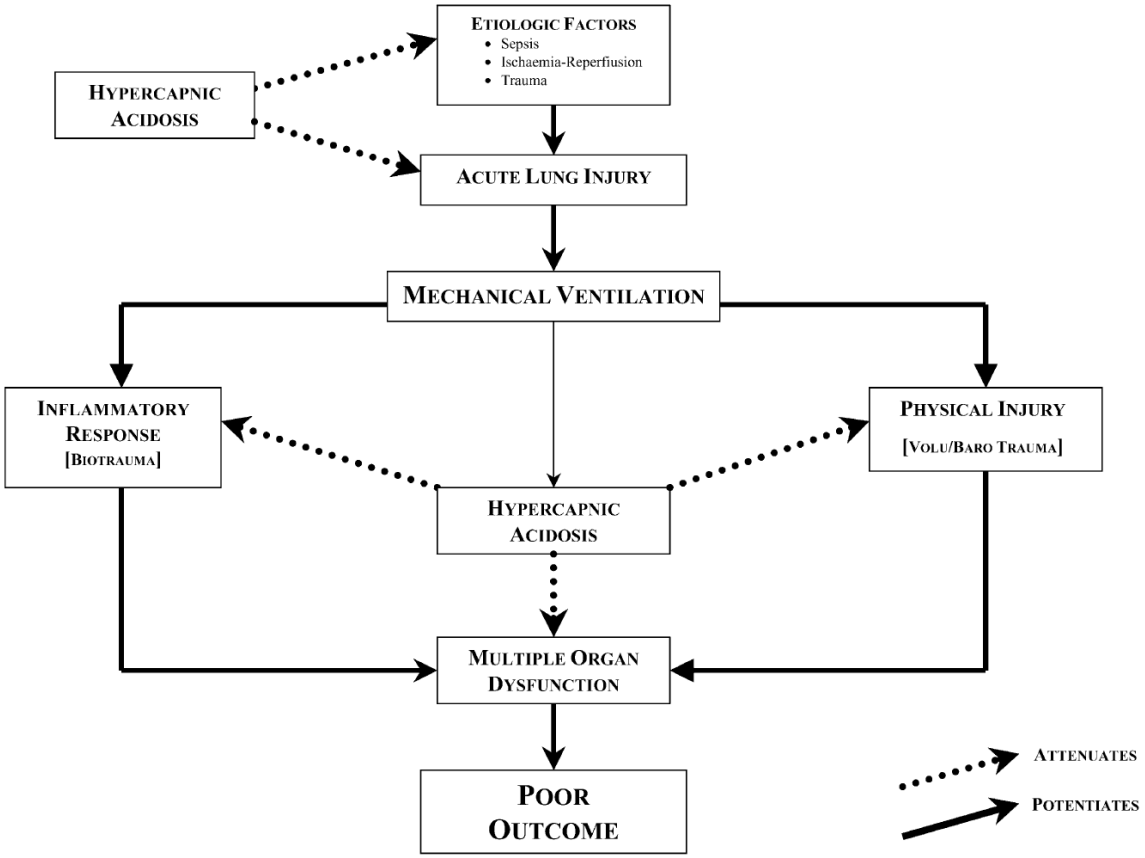
*Conversion of traditional units to SI: 1 mm Hg = 0.133 kPa.

Intensive Care Med (2004) 30:347–356
DOI 10.1007/s00134-003-2051-1

REVIEW

John G. Laffey
Donall O'Croinin
Paul McLoughlin
Brian P. Kavanagh

Permissive hypercapnia — role in protective lung ventilatory strategies



Carbon dioxide in critically ill – too little of a good thing?

Laffey J.G., Kavanagh B.P., Lancet 1999

- „keep $PaCO_2$ high, if necessary make it high, and above all prevent it from being low“

Pozitivní plicní účinky HCA

- Antinflamatorní účinek v modelech ALI
- Snížení kapilární permeability
- Zlepšení tkáňové oxygenace
- Snížení poškození plic v krátkodobém modelu pneumonie

Sustained hypercapnic acidosis during pulmonary infection increases bacterial load and worsens lung injury*

Donall F. O'Croinin, MD, FCARCSI; Alistair D. Nichol, MB, FCARCSI; Natalie Hopkins, PhD; John Boylan, MB, FRCPC; Sorca O'Brien, MSc; Clare O'Connor, PhD; John G. Laffey, MD, FFARCSI; Paul McLoughlin, MB, PhD

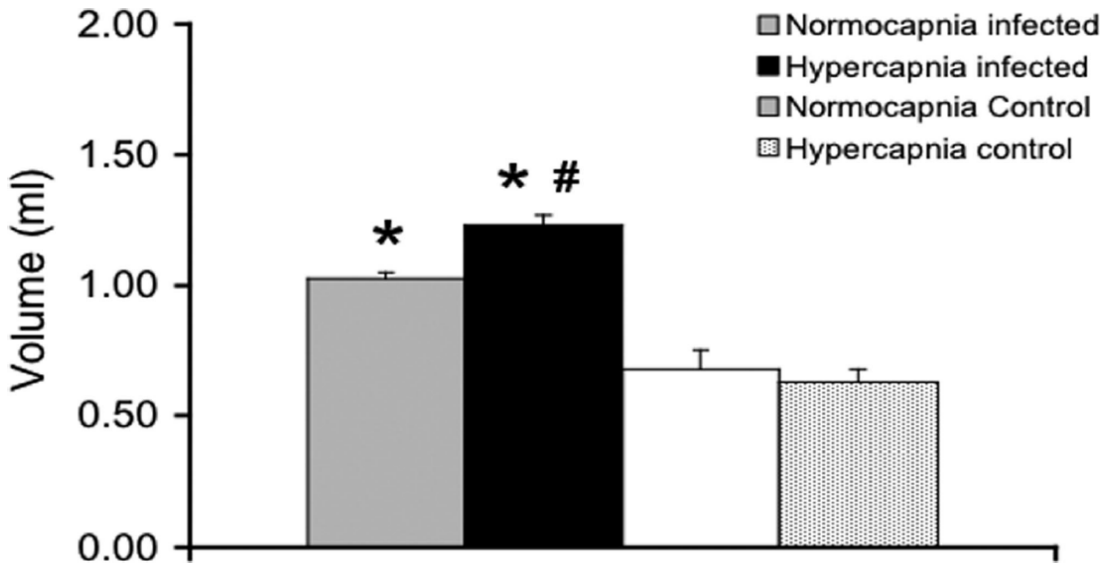


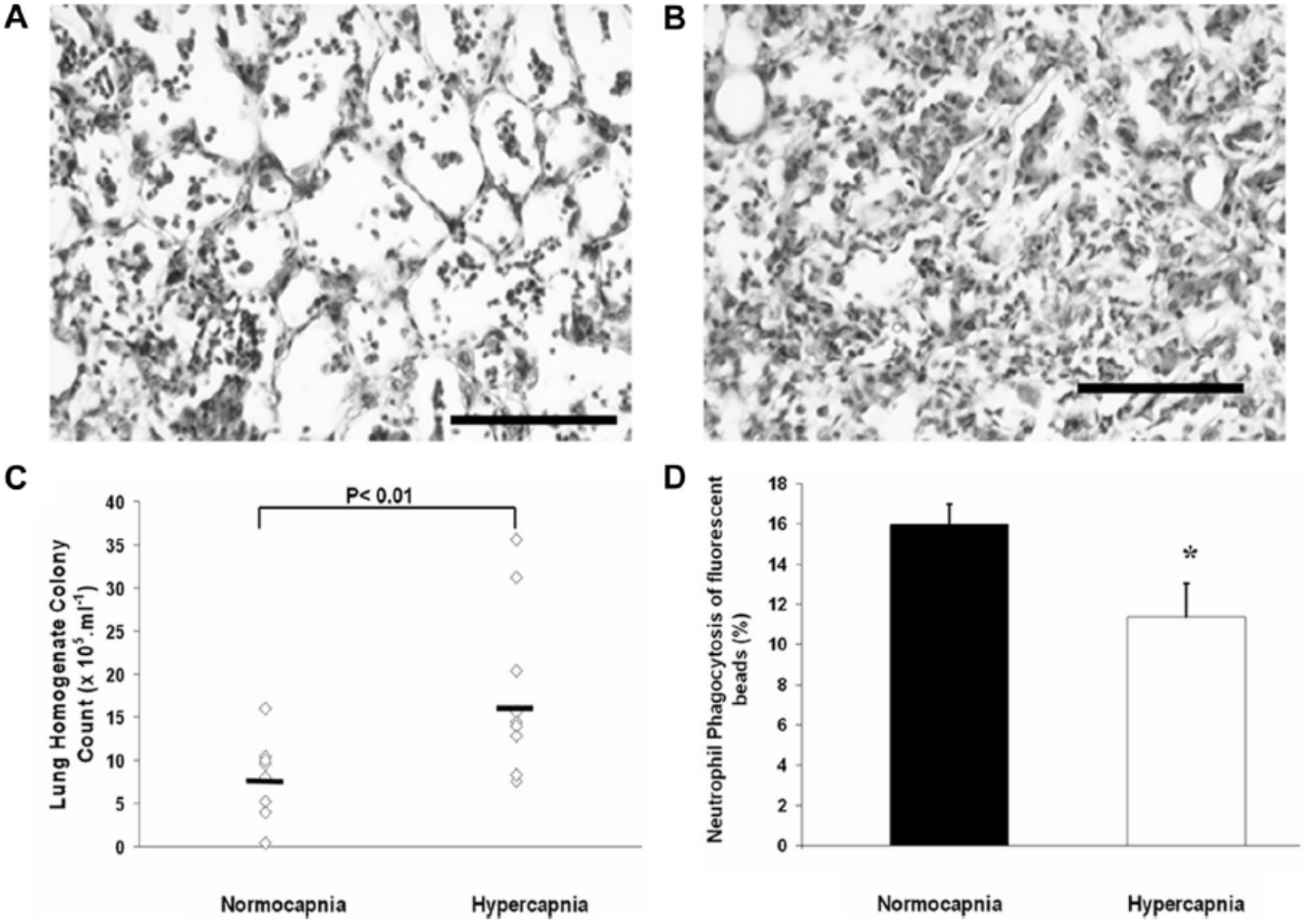
Figure 2. Graph representing mean (SEM) lung intra-acinar tissue volume in *Escherichia coli*-infected groups exposed to normocapnia (inspired CO_2 0%) or hypercapnia (inspired CO_2 5%) and in noninfected lungs exposed to normocapnia or hypercapnia. #Significant difference from normocapnia-infected group ($p < .05$). *Significant difference from normocapnia control (uninfected) group ($p < .05$).

Hypercapnia and Acidosis in Sepsis

A Double-edged Sword?

Gerard Curley, M.B., F.C.A.R.C.S.I.,* Maya Contreras, M.B., F.C.A.R.C.S.I.,*
Alistair D. Nichol, M.B., F.C.A.R.C.S.I., Ph.D.,† Brendan D. Higgins, B.Sc., Ph.D.,‡
John G. Laffey, M.D., M.A., B.Sc., F.C.A.R.C.S.I.§

48 h



Negativní plicní účinky

- Zhoršuje průběh pneumonie (E.coli)
  - O'Croinin DF, Nichol AD, Hopkins N, Boylan J, O'Brien S, O'Connor C, Laff ey JG, McLoughlin P: Sustained hypercapnic acidosis during pulmonary infection increases bacterial load and worsens lung injury. *Crit Care Med 2008*, 36:2128-2135.
- Zhoršuje clearence alveolární tekutiny
  - Briva A, Vadasz I, Lecuona E, Welch LC, Chen J, Dada LA, Trejo HE, Dumasius V,Azzam ZS, Myrianthefs PM, Batlle D, Gruenbaum Y, Sznajder JI: High CO2 levels impair alveolar epithelial function independently of pH. *PLoS One* 2007, 2:e1238.
- Zpomaluje hojení plicního poškození
  - O'Toole D, Hassett P, Contreras M, Higgins BD, McKeown ST, McAuley DF, O'Brien T, Laff ey JG: Hypercapnic acidosis attenuates pulmonary epithelial wound repair by an NF-κB dependent mechanism. *Thorax 2009*, 64:976-982.

J Appl Physiol 94: 1543–1551, 2003.
First published December 13, 2002; 10.1152/japplphysiol.00890.2002.

Human pulmonary vascular response to 4 h of hypercapnia and hypocapnia measured using Doppler echocardiography

George M. Balanos, Nicholas P. Talbot, Keith L. Dorrington, and Peter A. Robbins

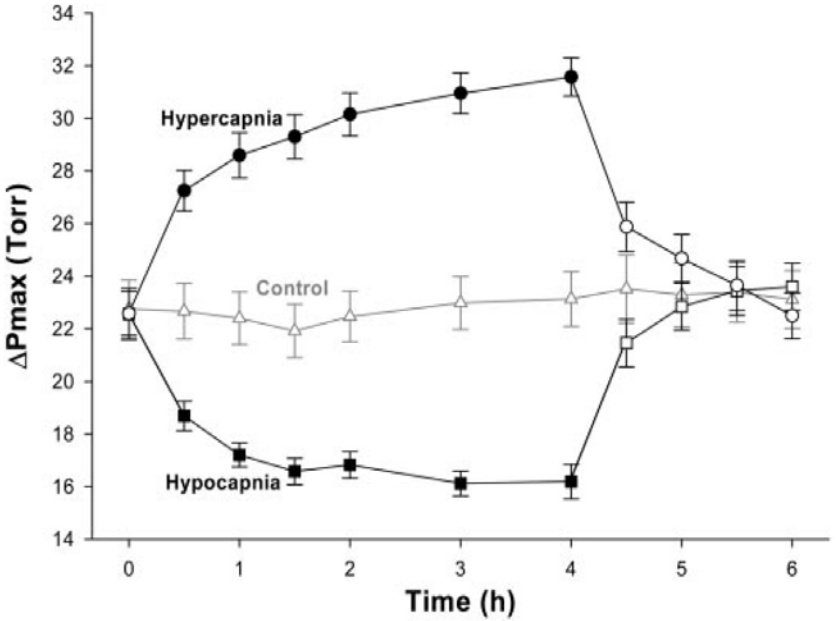


Fig. 2. Tricuspid valve maximum pressure gradient (ΔP_{max}) for the hypercapnia (circles), hypocapnia (squares), and control protocols (triangles). Closed symbols, measurements made under hypercapnic (hypercapnia protocol) or hypocapnic (hypocapnia protocol) conditions; open symbols, measurements made under eucapnic conditions. Data are means ± SE for $n = 12$ subjects. ΔP_{max} was significantly affected by both hypercapnia and hypocapnia compared with control ($P < 0.001$, protocol by time, repeated-measures ANOVA in both cases).

Effects of hypercapnia and hypocapnia on ventilatory variability and the chaotic dynamics of ventilatory flow in humans

Marie-Noëlle Fiamma,[1] Christian Straus,[1,2] Sylvain Thibault,[3] Marc Wysocki,[1,4] Pierre Baconnier,[3] and Thomas Similowski[1,5]

Table 2. *Ventilatory variables during hypo-, normo-, and hypercapnia*

	Hypocapnia	Normocapnia	Hypercapnia	ANOVA
Instantaneous ventilation, l/min	8.940±2.040†	7.080±2.040‡	46.440±9.06†‡	$P<0.0001$
Tidal volume, liter	0.562±0.272†	0.617±0.171‡	2.091±0.483†‡	$P<0.0001$
Mean inspiratory flow, l/s	0.327±0.089†	0.260±0.047‡	1.590±0.349†‡	$P<0.0001$
Total ventilatory cycle time, s	3.878±1.596*	5.757±2.494*‡	2.725±0.296‡	$P=0.0023$
Inspiratory time, s	1.742±0.655*	2.466±0.743*‡	1.334±0.170‡	$P=0.0017$
Expiratory time, s	2.132±0.958*	3.291±1.878*‡	1.392±0.183‡	$P=0.0055$
Duty cycle	0.463±0.046	0.458±0.083	0.490±0.034	$P=0.2992$

Values are means ± SD. Significant differences ($P < 0.005$) between 2 conditions are as indicated: *difference between hypocapnia and normocapnia; †difference between hypocapnia and hypercapnia; ‡difference between normocapnia and hypercapnia.

Use of Sedatives and Neuromuscular Blockers in a Cohort of Patients Receiving Mechanical Ventilation*

Alejandro Arroliga, MD, FCCP; Fernando Frutos-Vivar, MD; Jesse Hall, MD; Andres Esteban, MD; Carlos Apezteguía, MD; Luis Soto, MD; Antonio Anzueto, MD; for the International Mechanical Ventilation Study Group†

Permisivní hyperkapnie

- OR 8,45 pro hlubokou sedaci
- OR 11,22 pro použití NMBA

Limity permisivní hyperkapnie

- Kontraindikace
  - Riziko nitrolební hypertenze
  - Srdeční selhání, závažné arytmie
  - (Hypovolémie)
  - (Betablokátory - negativně inotropní efekt hyperkapnie)
    - www.uptodate.com
- Limitní hodnoty $PaCO_2$
  - Porucha vědomí - nad 90-120 mmHg, rychlost vzniku a adaptace
    - Nunn JF. Ventilatory failure. In: Nunn's Applied Respiratory Physiology. 4th ed. Butterworth-Heinemann; 1993:418-430

Konvenční indikace k ventilační podpoře při AE CHOPN

- pH < 7,35 – NIV
- pH < 7,20 – zvážit invazivní ventilaci, vyšší riziko selhání NIV
- *Kontraindikace nebo selhání NIV – invazivní ventilace*

Ventilační cíle u invazivní ventilace (CHOPN)

- pH > 7.2
- Krátkodobě lze připustit pH > 7.0
- Cílové hodnota CO_2 ?

Anesthesiology 2009; 111:699–700

Ventilator-induced Lung Injury

Less Ventilation, Less Injury

When the $Paco_2$ becomes uncomfortably high (60 mmHg? 80 mmHg?), then carbon dioxide needs to be eliminated in different ways.

$ECCO_2R$ v non-ARDS indikaci (CHOPN)

- Při selhání NIV nebo „v riziku selhání NIV“
  - Snížení potřebné minutové ventilace
  - „Odtížení“ dýchacích svalů
- Předpokládá, že invazivní ventilace plic je rizikovější než $ECCO_2R$
- Facilitace ukončení invazivní ventilace
- „Bridge to transplant“

Extracorporeal Co_2 Removal in Hypercapnic Patients At Risk of Noninvasive Ventilation Failure: A Matched Cohort Study With Historical Control*

Lorenzo Del Sorbo, MD[1]; Lara Pisani, MD[2]; Claudia Filippini, PhD[1]; Vito Fanelli, MD[1]; Luca Fasano, MD[2]; Pierpaolo Terragni, MD[1]; Andrea Dell'Amore, MD[3]; Rosario Urbino, MD[1]; Luciana Mascia, MD, PhD[1]; Andrea Evangelista, MD[4]; Camillo Antro, MD[5]; Raffaele D'Amato, MD[1]; Maria José Sucre, MD[1]; Umberto Simonetti, MD[1]; Pietro Persico, MD[1]; Stefano Nava, MD[2]; V. Marco Ranieri, MD[1]

Mechanical events
Patient 2 clots in the circuit
Patient 6 clots in the circuit
Patient 9 clots in the circuit
Patient 14 clots in the circuit
Patient 16 membrane lung failure
Patient 18 pump malfunction
Patient 19 clots in the circuit
Patient 22 clots in the circuit
Patient 25 pump malfunction
Patient-related events
Patient 1 significant bleeding (hematuria)
Patient 4 significant bleeding (retroperitoneal hematoma)
Patient 13 vein perforation at cannula insertion
Patient 23 significant bleeding (groin)

Komplikace u 50% pacientů

(*Crit Care Med* 2015; 43:120–127)

Permisivní hyperkapnie - závěry

- Limitní hodnoty CO_2 nejsou jasné
- Zásadní pro rozhodování hodnota pH
- Snaha o intenzivnější korekci u nemocných s dekompenzovaným cor pulmonale
- Rozvoj alternativních metod ventilační podpory t.č. s nejasným benefitem, nelze zatím považovat za standard péče

3. Protektivní ventilace?

Možnosti prevence komplikací

- NIV
- Sedace
- Nutriční podpora
- Prevence VAP
- ..
- ..

- Protektivní ventilace

Protektivní ventilace

- Redukovaný dechový objem a limitovaný endinspirační tlak
- PEEP
- Doplňující postupy
  - PEEP + RM ?
  - *Pronační poloha*
  - *Svalová relaxace*
  - *Indikace ECLA*

Lung-Protective Ventilation With Low Tidal Volumes and the Occurrence of Pulmonary Complications in Patients Without Acute Respiratory Distress Syndrome: A Systematic Review and Individual Patient Data Analysis*

(*Crit Care Med* 2015; 43:2155–2163)

TABLE 1. Baseline Characteristics of Included Patients According to Tidal Volume Received[a]

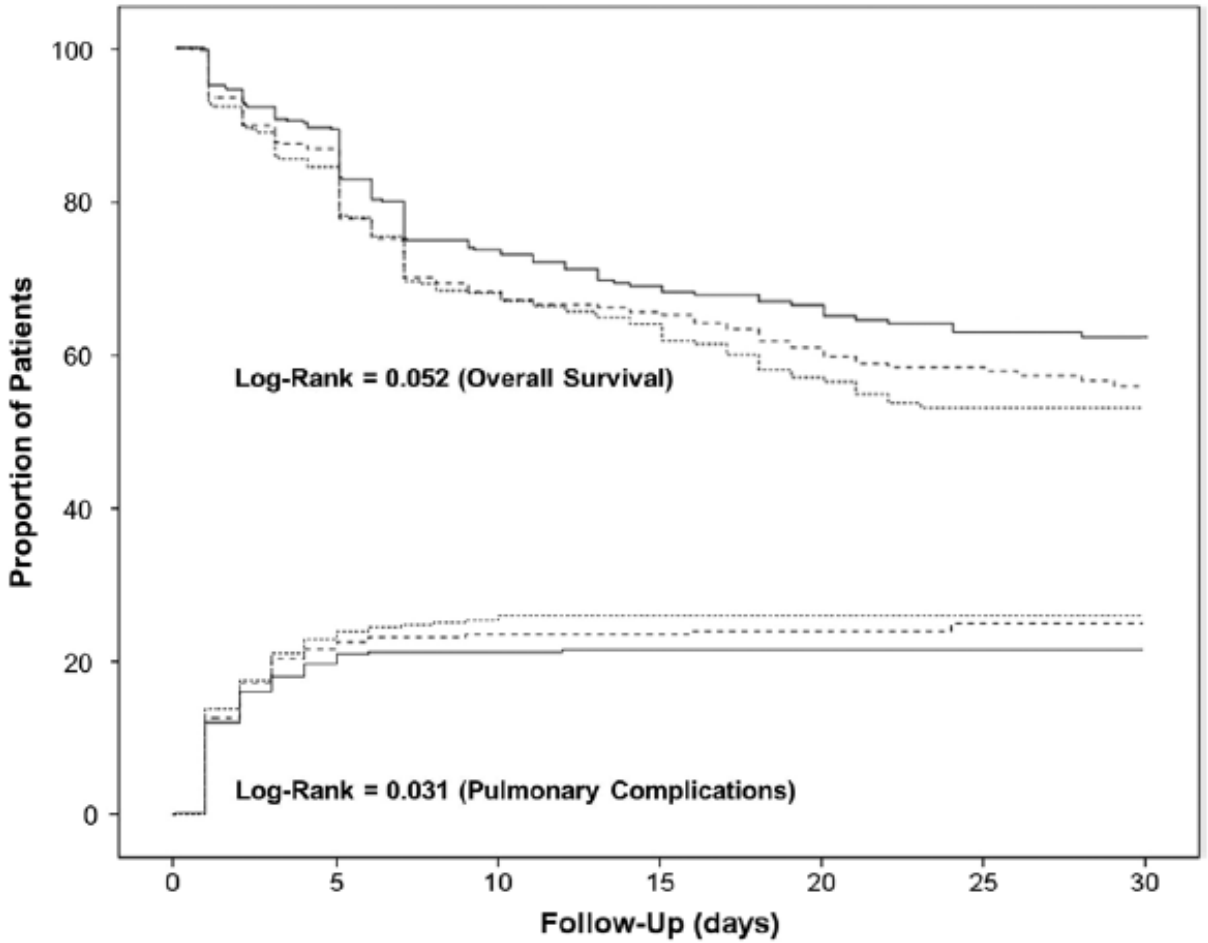
Variables	Less Than or Equal to 7 mL/kg PBW ($n = 720$)	Greater Than 7 and Less Than 10 mL/kg PBW ($n = 754$)	Greater Than or Equal to 10 mL/kg PBW ($n = 710$)
Age, yr	62.1 ± 16.6	63.5 ± 15.7	64.2 ± 16.0
Gender, female	232 (32.2)	296 (39.2)	253 (35.6)
PBW, kg	69.7 ± 9.7	64.8 ± 9.9	60.8 ± 11.8[b]
Design of the study, randomized controlled trial	106 (14.7)	35 (4.6)	84 (11.8)[b]
Acute Physiology and Chronic Health Evaluation II	21.67 ± 8.6	21.6 ± 8.2	21.3 ± 8.5
Pao_2/Fio_2	272.9 ± 142.9	274.6 ± 124.7	278.6 ± 130.3

Lung-Protective Ventilation With Low Tidal Volumes and the Occurrence of Pulmonary Complications in Patients Without Acute Respiratory Distress Syndrome: A Systematic Review and Individual Patient Data Analysis*

(*Crit Care Med* 2015; 43:2155–2163)

TABLE 2. Primary and Secondary Outcomes According to Tidal Volume Received[a]

Variables	Less Than or Equal to 7 mL/kg PBW	Greater Than 7 and Less Than 10 mL/kg PBW	Greater Than or Equal to 10 mL/kg PBW	Adjusted OR (Low vs High) (95% CI)[b]	*p*	Adjusted OR (Intermediary vs High) (95% CI)[b]	*p*
Pulmonary complications	166 (23)	211 (28)	220 (31)	0.72 (0.52–0.98)	0.042	0.93 (0.69–1.24)	0.635
Acute respiratory distress syndrome	86 (12)	121 (16)	163 (23)	0.48 (0.32–0.71)	< 0.01	0.73 (0.52–1.03)	0.074
Pneumonia	122 (17)	158 (21)	106 (15)	1.47 (0.89–2.21)	0.093	1.27 (0.86–1.86)	0.223
In-hospital mortality	245 (34)	279 (37)	270 (38)	0.82 (0.65–1.02)	0.081	0.90 (0.73–1.10)	0.319

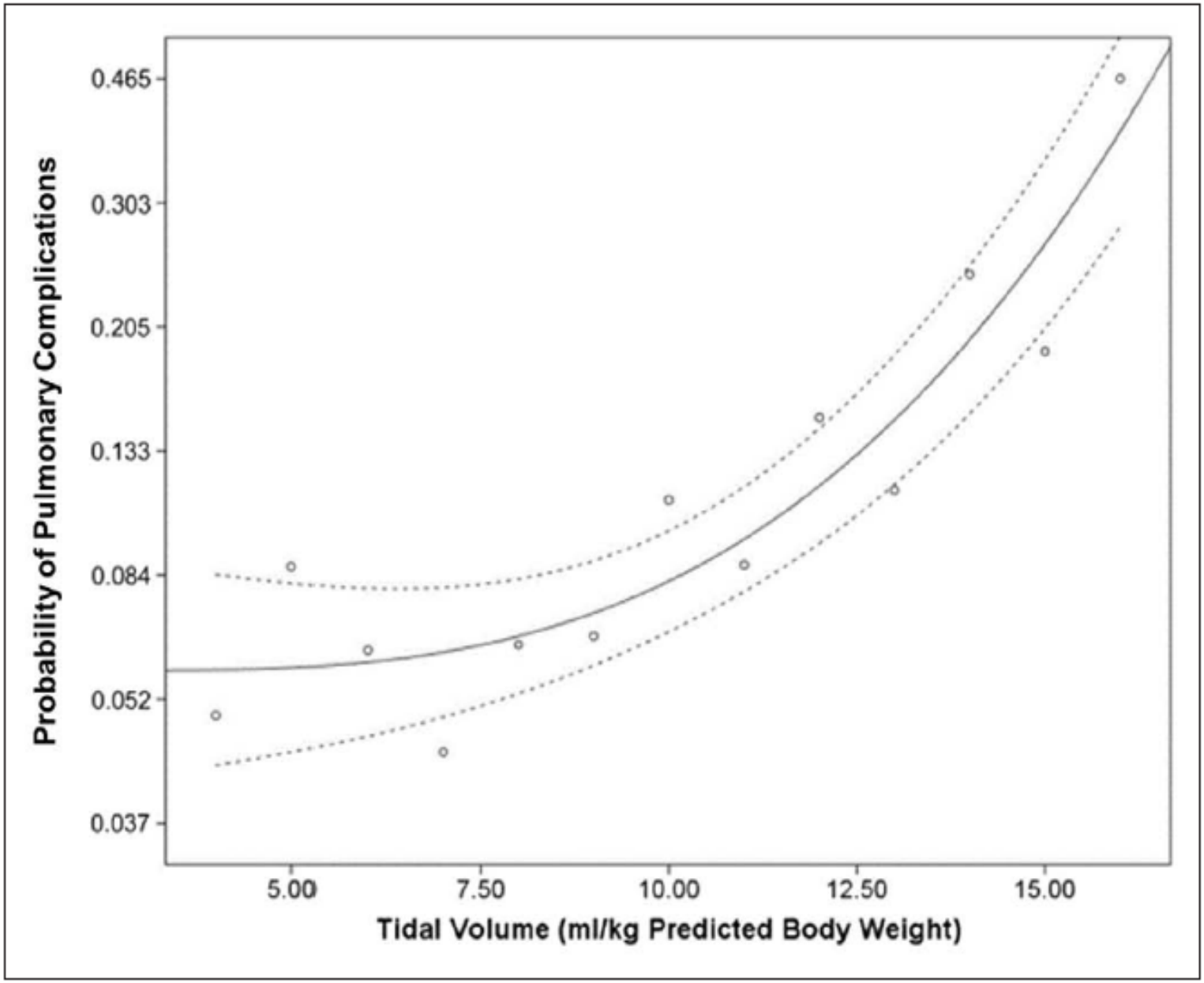


Number at Risk for Overall Survival

≤ 7 ml/kg PBW	512	385	239	179	142	93	35
7 to 10 ml/kg PBW	598	436	243	189	145	101	45
≥ 10 ml/kg PBW	498	353	199	149	110	72	29

Number at Risk for Incidence of Pulmonary Complications

≤ 7 ml/kg PBW	508	325	235	206	188	175	86
7 to 10 ml/kg PBW	478	283	190	173	160	152	77
≥ 10 ml/kg PBW	610	338	197	165	145	132	65


Probability of Pulmonary Complications
0.465
0.303
0.205
0.133
0.084
0.052
0.037
5.00
7.50
10.00
12.50
15.00
Tidal Volume (ml/kg Predicted Body Weight)

High versus low positive end-expiratory pressure during general anaesthesia for open abdominal surgery (PROVHILO trial): a multicentre randomised controlled trial

Lancet 2014; 384: 495–503

The PROVE Network Investigators for the Clinical Trial Network of the European Society of Anaesthesiology*

Methods In this randomised controlled trial at 30 centres in Europe and North and South America, we recruited 900 patients at risk for postoperative pulmonary complications who were planned for open abdominal surgery under general anaesthesia and ventilation at tidal volumes of 8 mL/kg. We randomly allocated patients to either a high level of positive end-expiratory pressure (12 cm H_2O) with recruitment manoeuvres (higher PEEP group) or a low level of pressure (≤2 cm H_2O) without recruitment manoeuvres (lower PEEP group). We used a centralised computer-generated randomisation system. Patients and outcome assessors were masked to the intervention. Primary endpoint was a composite of postoperative pulmonary complications by postoperative day 5. Analysis was by intention-to-treat. The study is registered at Controlled-Trials.com, number ISRCTN70332574.

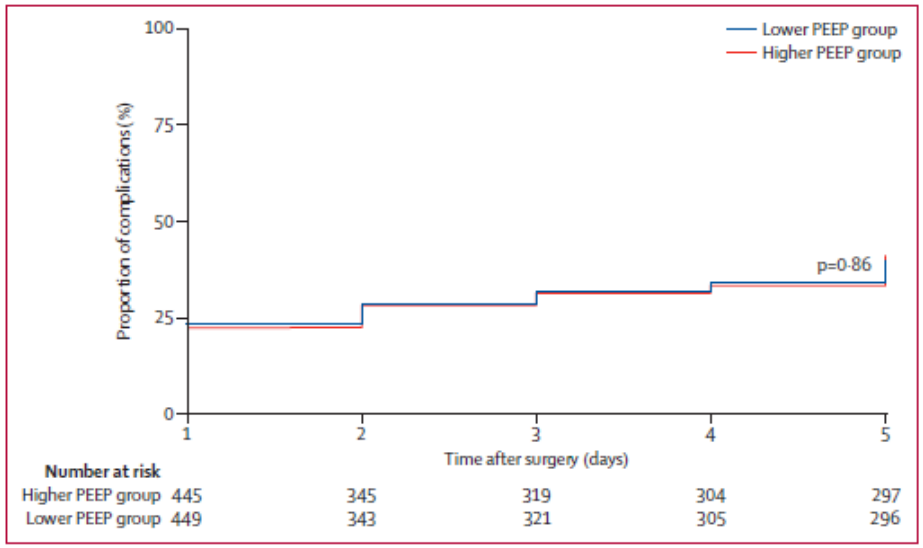


Figure 2: Kaplan-Meier curve showing the probability of postoperative pulmonary complications by postoperative day 5

High versus low positive end-expiratory pressure during general anaesthesia for open abdominal surgery (PROVHILO trial): a multicentre randomised controlled trial

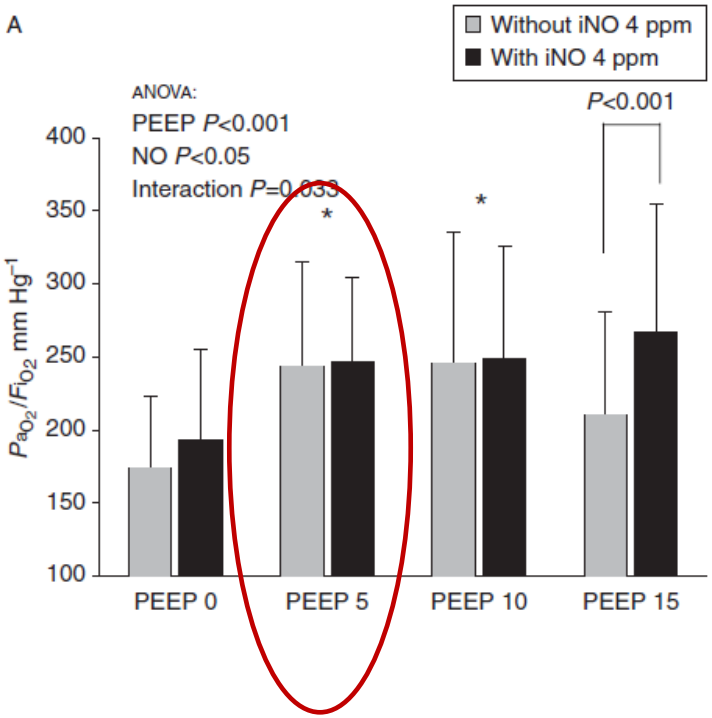
Lancet 2014; 384: 495–503

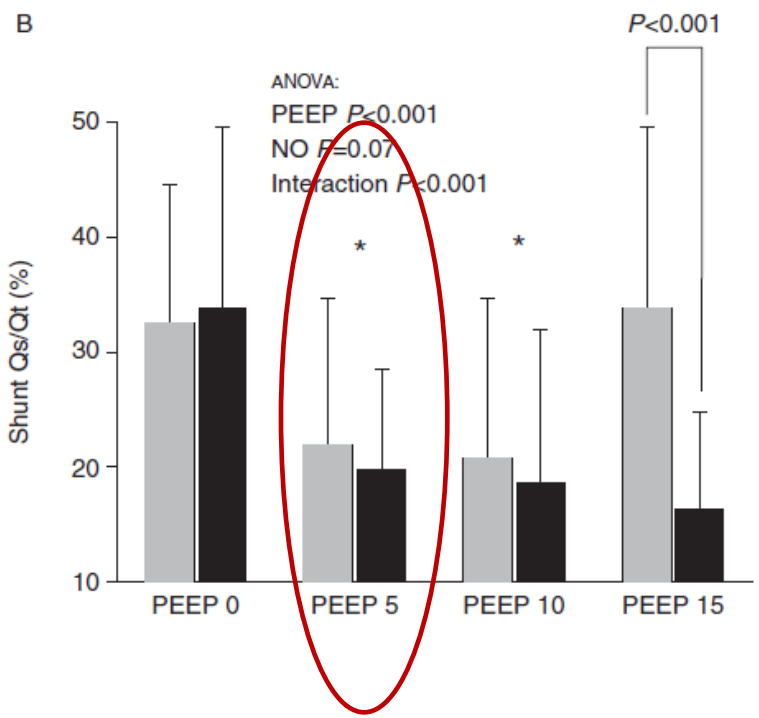
The PROVE Network Investigators for the Clinical Trial Network of the European Society of Anaesthesiology*

Intraoperative complications				
Rescue strategy for desaturation	11/442 (2%)	34/445 (8%)	0·34 (0·18–0·67)	0·0008
Hypotension††	205/441 (46%)	162/449 (36%)	1·29 (1·10–1·51)	0·0016
Vasoactive drugs needed	274/444 (62%)	228/445 (51%)	1·20 (1·07–1·35)	0·0016
New arrhythmias needing intervention	12/442 (3%)	5/445 (1%)	2·38 (0·84–6·70)	0·09
Follow-up				
Impaired wound healing‡‡	71/444 (16%)	58/446 (13%)	1·23 (0·89–1·70)	0·21
Need for new or continued mechanical ventilation	18/437 (4%)	24/443 (5%)	0·77 (0·42–1·40)	0·74
Admission to intensive-care unit	106/442 (24%)	104/452 (23%)	1·03 (0·81–1·32)	0·79
Length of hospital stay (days)	10 (7–14)	10 (7–14)	..	0·24
Hospital-free days, at day 90	79 (71–83)	79 (70–82)	..	0·33
Mortality by day 5	2/443 (<1%)	1/448 (<1%)	2·02 (0·18–22)	0·56
In-hospital mortality	7/438 (2%)	7/442 (2%)	1·01 (0·36–2·85)	0·99

Effects of PEEP on oxygenation and respiratory mechanics during one-lung ventilation

P. Michelet[1]*, A. Roch[1], D. Brousse[2], X.-B. D'Journo[3], F. Bregeon[5], D. Lambert[1], G. Perrin[1], L. Papazian[4], P. Thomas[3], J.-P. Carpentier[2] and J.-P. Auffray[1]





Br J Anaesth 2005; **95**: 267–73

Lung anatomy, energy load, and ventilator-induced lung injury

Protti *et al. Intensive Care Medicine Experimental* (2015) 3:34

Conclusions

We found that the threshold of VILI in the healthy lung is the region defining the inspiratory lung capacity, i.e., the anatomical limits of lung expansion. When the inflated volume is below the threshold, VILI does not occur, and if it is within the limits, it appears as a main function of its dynamic component. If it exceeds the total lung capacity, stress at rupture occurs. A unifying explanation is that the trigger for VILI is an excessive energy/power load, which encompasses pressures, volume and—though not tested in the present study—respiratory rate and flow. PEEP, not associated with energy input, appears to prevent VILI if the tidal volume is lower. Otherwise, PEEP may be harmful as it just boosts inflation closer to the total lung capacity.

Erica Aranha Suzumura
Mabel Figueiró
Karina Normilio-Silva
Lígia Laranjeira
Claudia Oliveira
Anna Maria Buehler
Diogo Bugano

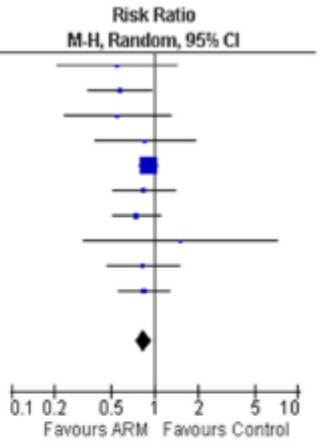
Effects of alveolar recruitment maneuvers on clinical outcomes in patients with acute respiratory distress syndrome: a systematic review and meta-analysis

Intensive Care Med (2014) 40:1227–1240
DOI 10.1007/s00134-014-3413-6

Effect on in-hospital mortality

Study or Subgroup	ARM Events	ARM Total	Control Events	Control Total	Weight	Risk Ratio M-H, Random, 95% CI	Year
Park 2003	4	11	4	6	1.6%	0.55 [0.21, 1.43]	2003
Lim 2003	10	20	7	8	5.7%	0.57 [0.34, 0.95]	2003
Long 2006	5	16	8	14	2.0%	0.55 [0.23, 1.29]	2006
Wang 2007	6	14	7	14	2.3%	0.86 [0.39, 1.91]	2007
Meade 2008	173	475	205	508	58.9%	0.90 [0.77, 1.06]	2008
Huh 2009	14	30	15	27	5.7%	0.84 [0.50, 1.40]	2009
Xi 2010	23	55	31	55	9.8%	0.74 [0.50, 1.09]	2010
Hodgson 2011	3	10	2	10	0.6%	1.50 [0.32, 7.14]	2011
Liu 2011	14	50	17	50	4.3%	0.82 [0.46, 1.48]	2011
Kacmarek 2014	29	99	35	101	9.0%	0.85 [0.56, 1.27]	2014
Total (95% CI)		**780**		**793**	**100.0%**	**0.84 [0.74, 0.95]**	
Total events	281		331				

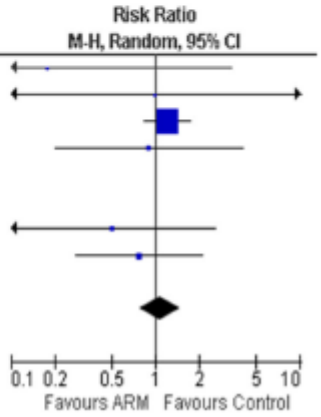
Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 5.71$, df = 9 (P = 0.77); $I^2 = 0\%$
Test for overall effect: Z = 2.86 (P = 0.004)



Effect on barotrauma

Study or Subgroup	ARM Events	ARM Total	Control Events	Control Total	Weight	Risk Ratio M-H, Random, 95% CI	Year
Long 2006	0	16	2	14	1.2%	0.18 [0.01, 3.39]	2006
Wang 2007	1	14	1	14	1.5%	1.00 [0.07, 14.45]	2007
Meade 2008	53	475	47	508	78.2%	1.21 [0.83, 1.75]	2008
Huh 2009	3	30	3	27	4.7%	0.90 [0.20, 4.09]	2009
Xi 2010	0	55	0	55		Not estimable	2010
Hodgson 2011	0	10	0	10		Not estimable	2011
Liu 2011	2	50	4	50	4.0%	0.50 [0.10, 2.61]	2011
Kacmarek 2014	6	99	8	101	10.4%	0.77 [0.28, 2.13]	2014
Total (95% CI)		**749**		**779**	**100.0%**	**1.07 [0.77, 1.48]**	
Total events	65		65				

Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 3.12$, df = 5 (P = 0.68); $I^2 = 0\%$
Test for overall effect: Z = 0.38 (P = 0.70)



Děkuji za pozornost.

Continuous endotracheal tube cuff pressure control system protects against ventilator-associated pneumonia

Lorente *et al. Critical Care* 2014, **18**:R77
http://ccforum.com/content/18/2/R77

Table 1 Characteristics of intermittent and continuous endotracheal-tube cuff-pressure control system patient groups *(Continued)*

VAP, patients, n (%)	33 (22.0)	15 (11.2)	0.02
Tracheobronchitis, patients, n (%)	10 (6.7)	5 (3.7)	0.30
VAP or tracheobronchitis, patients, n (%)	43 (28.7)	20 (14.9)	0.01
Time of MV free of VAP, days, mean ± SD	10.31 ± 10.56	12.75 ± 14.05	0.10
Duration of MV, days, mean ± SD	15.65 ± 20.78	15.21 ± 15.23	0.84
ICU mortality, patients, n (%)	55 (36.7)	51 (38.1)	0.90

- Intermitentní SS
- Kanyly s PVC manžetou

Continuous control of tracheal cuff pressure for VAP prevention: a collaborative meta-analysis of individual participant data

Saad Nseir[1,2*], Leonardo Lorente[3], Miquel Ferrer[4], Anahita Rouzé[1], Oswaldo Gonzalez[3], Gianluigi Li Bassi[4], Alain Duhamel[2,5] and Antoni Torres[4]

Nseir *et al. Ann. Intensive Care (2015) 5:43*
DOI 10.1186/s13613-015-0087-3

	Valencia et al. [21]	Nseir et al. [22]	Lorente et al. [23]
Number of included patients	137	122	284
Type of study	Randomized controlled	Randomized controlled	Quasi-randomized controlled
Primary objective	VAP	Microaspiration	VAP
Device	Electronic	Pneumatic	Electronic
Target P_{cuff} (cmH_2O)	25	25	25
Surgical patients	28	0	28
Chronic respiratory disorders	38	27	15
VAP preventive measures			
Oral care	CHX 0.12 % X3/days	CHX 0.10 % X3/days	CHX 0.12 % X3/days
Semirecumbent position	Yes	Yes	Yes
Subglottic secretion drainage	No	No	Yes
VAP incidence in control group	15	26	22
Reduction in VAP rate	NS	62	51

Continuous control of tracheal cuff pressure for VAP prevention: a collaborative meta-analysis of individual participant data

Saad Nseir[1,2*], Leonardo Lorente[3], Miquel Ferrer[4], Anahita Rouzé[1], Oswaldo Gonzalez[3], Gianluigi Li Bassi[4], Alain Duhamel[2,5] and Antoni Torres[4]

Nseir et al. Ann. Intensive Care (2015) 5:43
DOI 10.1186/s13613-015-0087-3

	Continuous Control of Pcuff		Routine Care		HR	
	Events	Total	Events	Total	(95%CI)	P Value
Valencia, 2007	15	68	20	69	0.79 (0.40-1.56)	0.50
Nseir , 2011	6	61	19	61	0.22 (0.09-0.57)	0.002
Lorente, 2014	15	134	33	150	0.46 (0.25-0.85)	0.013
Overall	36	263	72	280	0.47 (0.31-0.71)	0.0003
Heterogeneity	I^2=58% (P=0.085)					

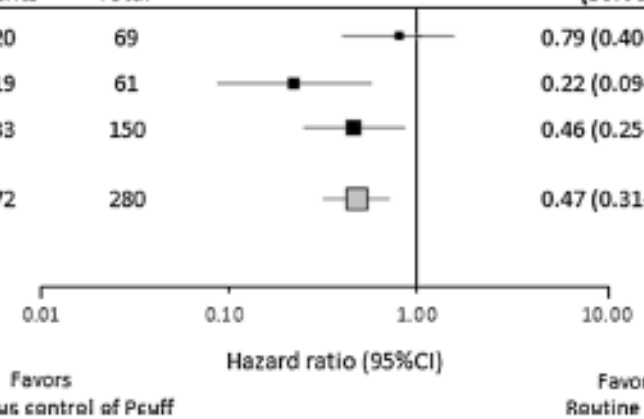


Fig. 2 VAP-free survival curves for patients assigned to continuous control of cuff pressure and routine care groups. *p* value, and hazard ratio were calculated using Cox proportional hazard model stratified on trial

Continuous control of tracheal cuff pressure for VAP prevention: a collaborative meta-analysis of individual participant data

Saad Nseir[1,2*], Leonardo Lorente[3], Miquel Ferrer[4], Anahita Rouzé[1], Oswaldo Gonzalez[3], Gianluigi Li Bassi[4], Alain Duhamel[2,5] and Antoni Torres[4]

Nseir et al. Ann. Intensive Care (2015) 5:43
DOI 10.1186/s13613-015-0087-3

	Continuous control of P_{cuff}		
	Yes ($n = 263$)	No ($n = 280$)	p value
MV duration (day)	8 (4, 16)	8 (4, 16)	0.681
MV free days	3 (0, 6)	2 (0, 5)	0.426
ICU length of stay (day)	11 (6, 24)	12 (7, 21)	0.440
Duration of antibiotic treatment	9 (6, 15)	10 (6, 15)	0.778
ICU mortality	86 (33)	91 (32)	>0.999

Data are number (%), or median (interquartile range)