Positive pressure ventilation in cardiogenic shock: friend or foe?

ACCA Masterclass 2017

Josep Masip MD, PhD, FESC

Disclosures: Novartis advisor, ThermoFisher consultant, Philips and Orion speaker fees, Menarini travel-congress support

WE ARE THE ESC

Acute Cardiovascular Care Association ACCA
A Registered Branch of the ESC

EUROPEAN SOCIETY OF CARDIOLOGY®

Respiratory disorders in cardiogenic shock

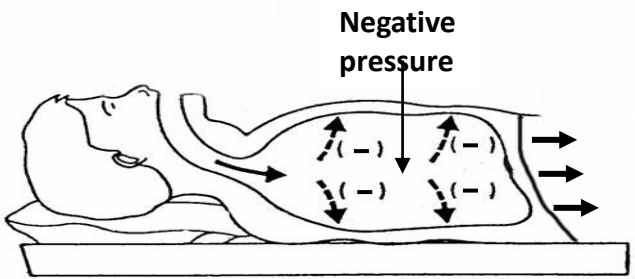
- Increase in dead-space (fall in pulmonary perfusion)
- Shunt effect (pulmonary edema - hypoxemia)
- Ventilation-Perfusion inequality (respiratory failure)
- Tissue hypoperfusion (Altered mental status)
  (Lactacidemia - metabolic acidosis – ↑ A-V difference- ↓ SVO2
- Respiratory muscle dysfunction (Hypoventilation – Hypercapnia)
- Pulmonary inflammation (Cytokines release – SIRS)
- Tachypnea – Increasing work of breathing

MAIN GOALS OF MECHANICAL VENTILATION IN SHOCK

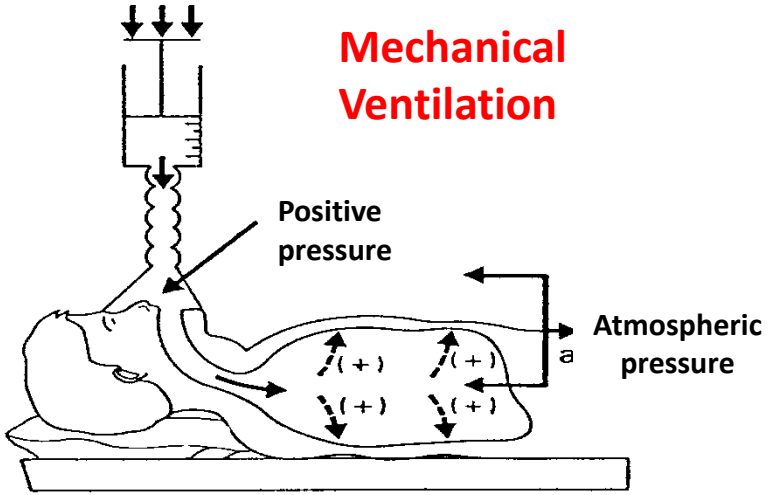
- Establish an adequate airway (CNS)
- Reduce VO2 (work of breathing)
- Improve oxygenation
- Reverse respiratory acidosis (hypercapnia)
- Decrease sympathetic tone
- Improve tissue perfusion and metabolic acidosis

Effects of MV in the thorax

Spontaneous breathing



Mechanical Ventilation

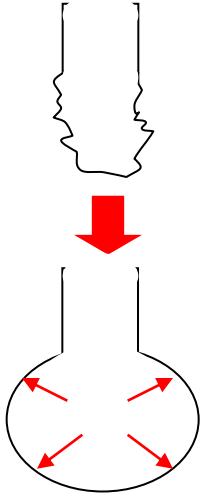


BENEFICIAL EFFECTS OF POSITIVE INTRATHORACIC PRESSURE

RESPIRATORY

- Recruitment of collapsed alveolar units
- Increase of FRC
- Maintenance of continuously opened alveoli
- Gas exchange during the whole respiratory cycle
- Intra-alveolar pressure against edema

Decrease work of breathing
Improvement in oxygenation



Alveolus

HEMODYNAMIC

- Decrease in pulmonary shunt

OTHER HEMODYNAMIC CHANGES WITH POSITIVE INTRATHORACIC PRESSURE

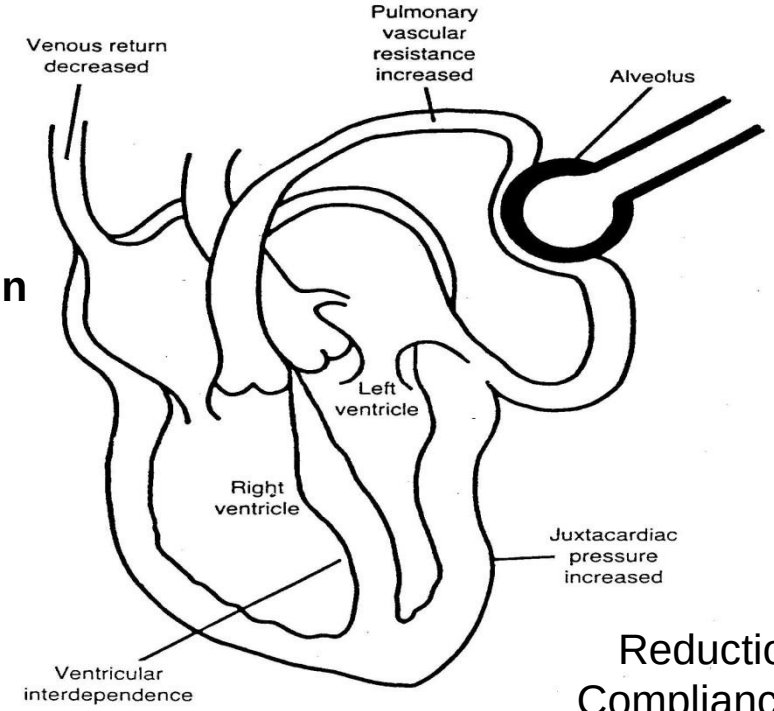
Decrease
Preload RV - LV

↓

Systemic hypotension
Reduction CO
Fluid retention

↑

Increase
RV Afterload



In AHF it may
Increase
cardiac output

Reduction
Compliance LV

Martin J Tobin. NEJM 2001

WE ARE THE ESC

Let's have a look at the real world

Acute Cardiovascular Care Association ACCA
A Registered Branch of the ESC

EUROPEAN SOCIETY OF CARDIOLOGY®

Table 1. Pathophysiology of Cardiogenic Shock.

- Acute myocardial infarction
  - Loss of critical left ventricular myocardium
  - Right ventricular pump failure
- Mechanical complications
  - Acute mitral regurgitation due to papillary-muscle dysfunction or rupture
  - Ventricular septal rupture
  - Free-wall rupture
  - Left ventricular aneurysm
- Miscellaneous conditions
  - End-stage cardiomyopathy
  - Myocardial contusion
  - Myocarditis
  - Left ventricular outflow tract obstruction
    - Aortic stenosis
    - Hypertrophic obstructive cardiomyopathy
  - Left ventricular inflow tract obstruction
    - Mitral stenosis
    - Left atrial myxoma
  - Sequela of cardiopulmonary bypass

Califf R. NEJM 1994

Table 1. Causes of cardiogenic shock

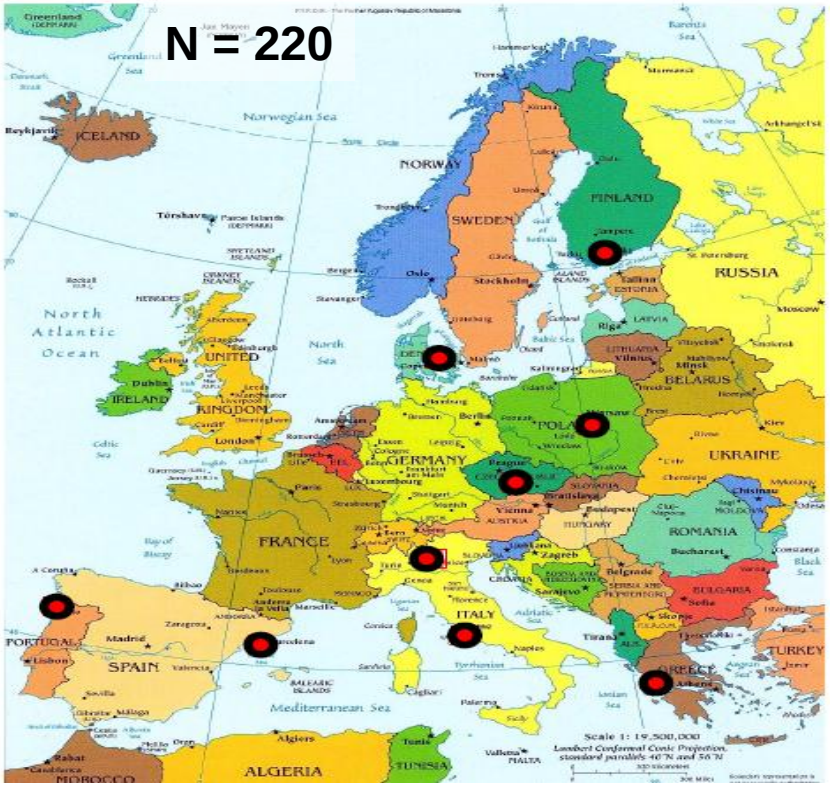
- Acute myocardial infarction
  - Pump failure
    - Large infarction
    - Smaller infarction with preexisting left ventricular dysfunction
    - Infarction extension
    - Severe recurrent ischemia
    - Infarction expansion
  - Mechanical complications
    - Acute mitral regurgitation caused by papillary muscle rupture
    - Ventricular septal defect
    - Free-wall rupture
    - Pericardial tamponade
  - Right ventricular infarction
- Other conditions
  - End-stage cardiomyopathy
  - Myocarditis
  - Myocardial contusion
  - Prolonged cardiopulmonary bypass
  - Septic shock with severe myocardial depression
  - Left ventricular outflow tract obstruction
    - Aortic stenosis
    - Hypertropic obstructive cardiomyopathy
  - Obstruction to left ventricular filling
    - Mitral stenosis
    - Left atrial myxoma
  - Acute mitral regurgitation (chordal rupture)
  - Acute aortic insufficiency
  - Acute massive pulmonary embolism
  - Acute stress cardiomyopathy
  - Pheochromocytoma

Topalia S, et al Crit Care Med 2008

CARDSHOCK STUDY

CardShock Study

GREAT
global research on acute conditions team

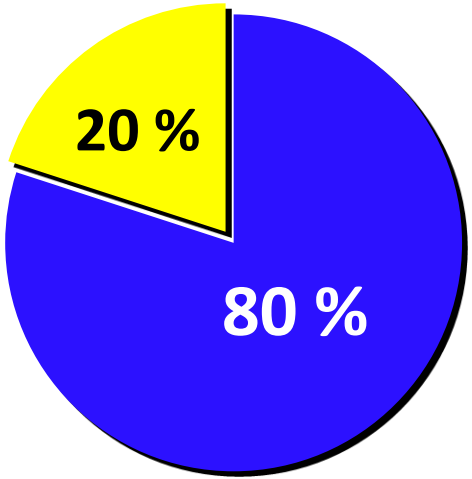


www.escardio.org/ACCA

Acute Cardiovascular Care Association ACCA
A Registered Branch of the ESC

EUROPEAN SOCIETY OF CARDIOLOGY®

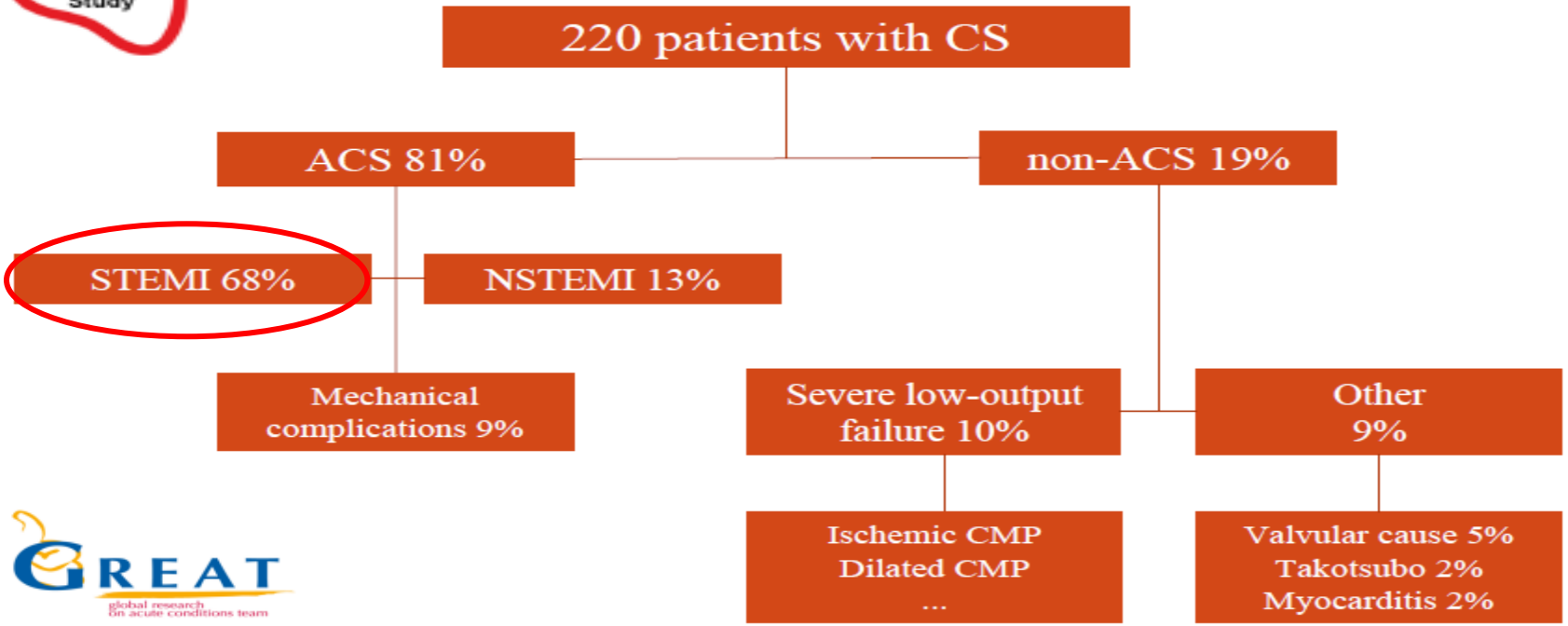
Causes of Cardiogenic shock



N=220

- Acute Coronary Syndrome
- Other causes

Harjola V-P. Eur J Heart Fail 2015



Harjola V-P. Eur J Heart Fail 2015

CAUSES OF MECHANICAL VENTILATION IN ACS

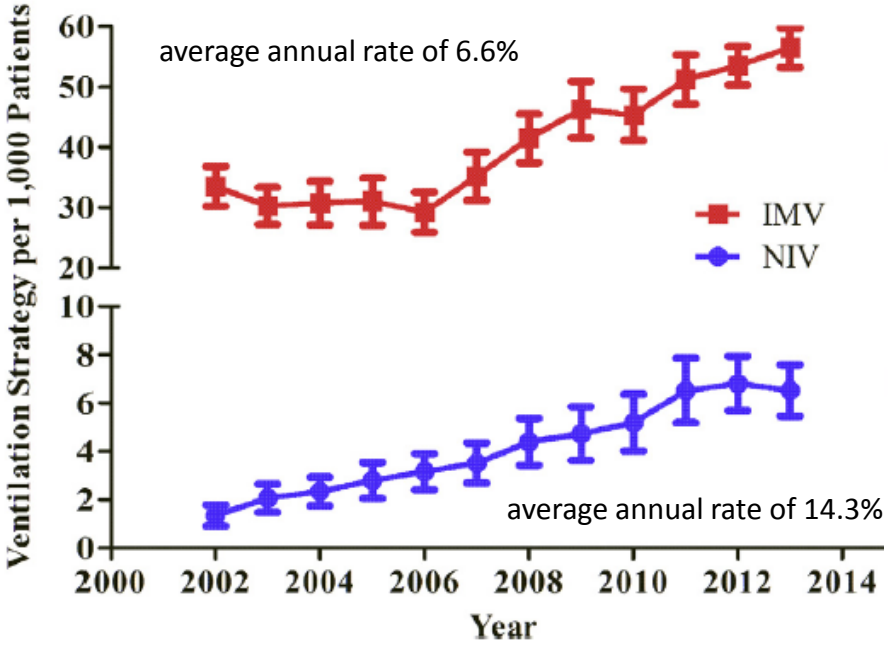
7.6% 1231 STEMI | 10 (9.5%) | 64 (60.4%) | 32 (30.1%) — *Lazzeri Ch. Cardiol J 2013*

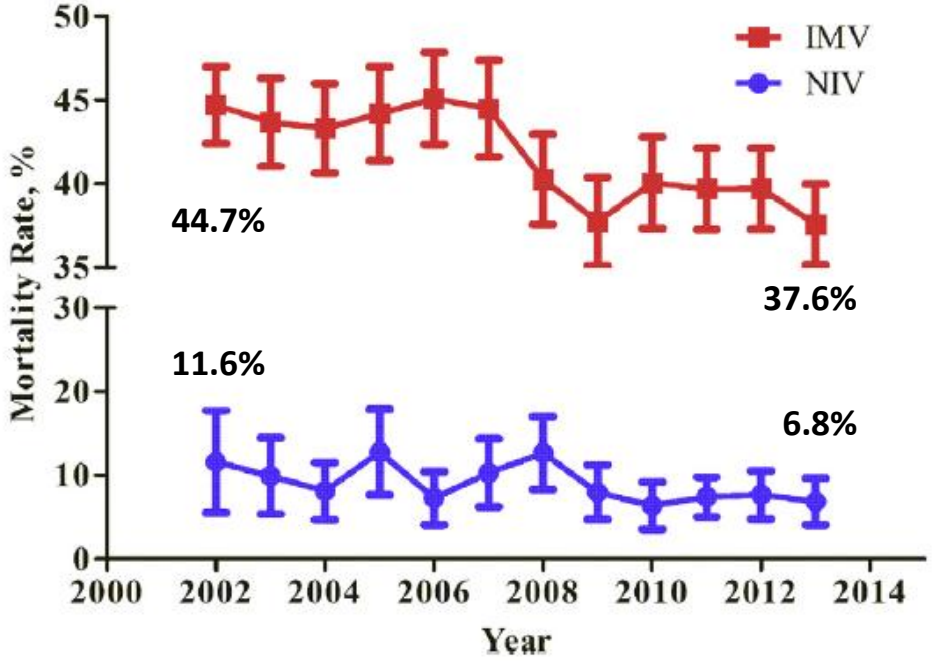
5.8% 1821 ACS

	AHF n=27 (26%)	Shock n=14 (13%)	Cardiac Arrest n=65 (61%)	p
Age	68	69	58	.000
Diabetes	59	43	22	.002
HTA	82	86	46	.001
Smoker	26	21	83	.001
In hospital ETI (%)	63	54	16	.001
NIV	9	-	3	.001
Swan Ganz (%)	37	36	14	.024
IABP (%)	56	50	15	.001
Renal RT (%)	15	15	2	.030
Major bleeding (%)	11	29	3	.006
Transfusions (%)	26	36	11	.032
In H mortality (%)	22	43	33	0.4
Mortality (%)	41	43	33	0.4
Non-card. Mortality	36	17	52	0.2

Ariza A. Eur Heart J Acute Card Care 2013

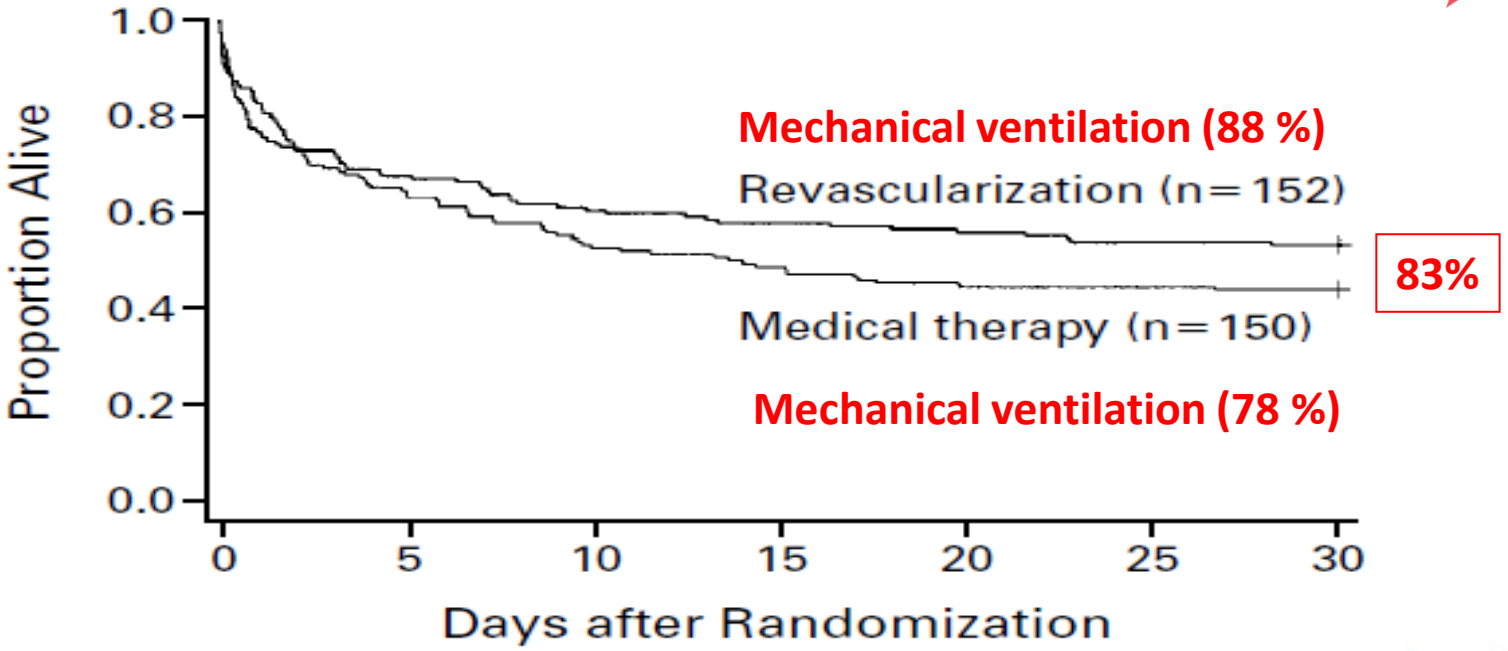
USA National Inpatient Sample (NIS) from 2002 to 2013: 1.867.114 STEMI
72.220 IMV (3.9%) and 7.030 NIV (0.4%)





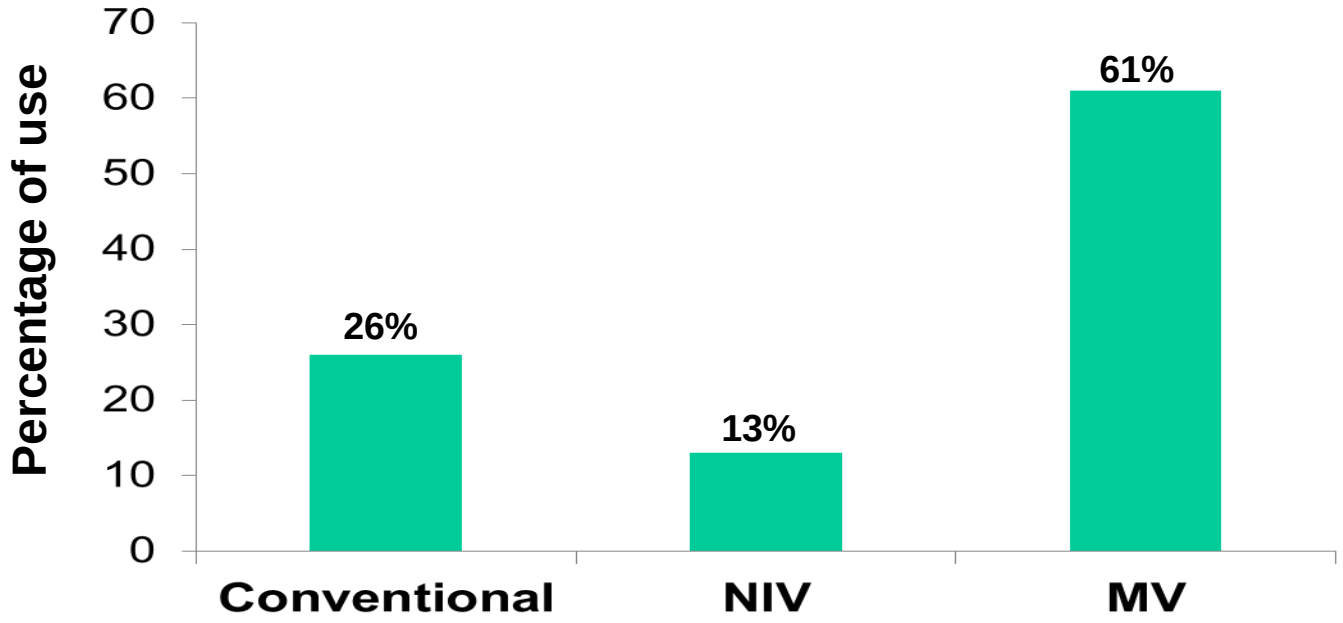
Metkus T. Am J Cardiol 2013

SHOCK Trial



Hochman J et al. NEJM 1999

Oxygen Therapy in Card-Shock Study



Hongisto M. International J Cardiol 2017

CHARACTERISTICS OF THE PATIENTS WITH CS ACCORDING TO THE TYPE OF OXYGEN THERAPY

Blood analysis

	MV (n = 137)	NIV (n = 26)	Oxygen (n = 56)	p
Hemoglobin (g/L)	130	125	124	0.3
Arterial lactate (mmol/L)	3.7	1.7	2.3	0.001
Hs-TroponinT (ng/L)	1597	3631	2427	0.06
NT-proBNP (pg/mL)	2367	7375	1860	0.04
Creatinine (mmol/L)	110	100	107	0.1
eGFR (mL/min/1.73 m2)	64	67	59	0.6
CRP (g/L)	15	37	15	0.2

Hongisto M. International J Cardiol 2017

CHARACTERISTICS OF THE PATIENTS WITH CS ACCORDING TO THE TYPE OF OXYGEN THERAPY

Baseline arterial blood gases	MV (n = 137)	NIV (n = 26)	Oxygen (n = 56)	p
pH	7.27	7.39	7.38	<0.001
PaO2 (mm Hg)	96.7	84	105.1	0.2
PaCO2 (mm Hg)	41.2	33.8	36.8	0.01
HCO3 mmol/L	19.6	22	21.9	0.001
FiO2 (%)	76	60	32	0.001
PaO2/FiO2 (mm Hg)	141	167	311	0.3
200–300 n (%)	35	7	7	0.9
100–200 n (%)	54	14	7	0.2
<100 n (%)	40	4	0	0.1

Hongisto M. International J Cardiol 2017

CHARACTERISTICS OF THE PATIENTS WITH CS ACCORDING TO THE TYPE OF OXYGEN THERAPY

Devices and outcomes

	MV (n = 137)	NIV (n = 26)	Oxygen (n = 56)	p
Coronary angiogram	114 (83)	23 (89)	45 (80)	0.8
PCI	90 (66)	19 (73)	40 (71)	0.5
CABG	5 (4)	3 (12)	1 (2)	0.1
IABP	85 (62)	16 (62)	21 (38)	1.0
In-hospital mortality	62 (45)	5 (19)	13 (23)	0.01
90-day mortality	67 (49)	7 (27)	15 (27)	0.03
ICU/CCU (days)	6	4	3	0.2
In-hospital (days)	17	12	8	0.2

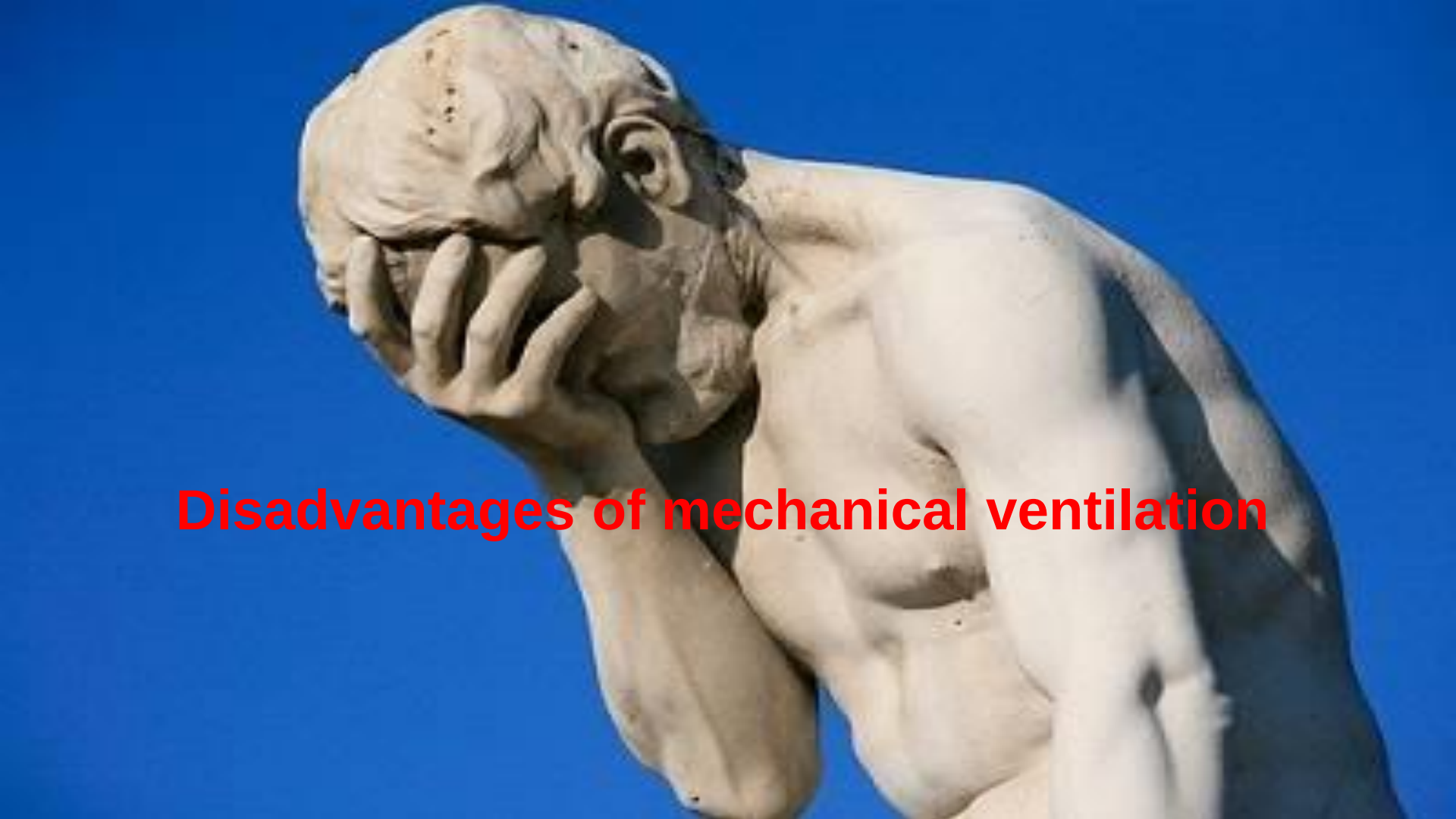
Hongisto M. International J Cardiol 2017

CHARACTERISTICS OF THE PATIENTS WITH CS ACCORDING TO THE TYPE OF OXYGEN THERAPY

Clinical Findings

	MV (n = 137)	NIV (n = 26)	Oxygen (n = 56)	p
Systolic BP (mmHg)	78	83	75	0.03
Heart rate (b/m)	91	87	89	0.2
LVEF (%)	32	33	36	0.7
Confusion n (%)	113	8	26	0.001

Hongisto M. International J Cardiol 2017


Disadvantages of mechanical ventilation

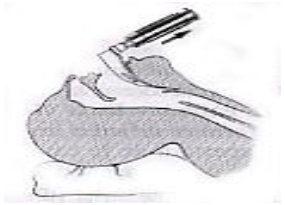
Disadvantages of mechanical ventilation

- Artificial airway (intubation-tracheostomy)
- Need for Sedation
- Initial hypotension
- Atrophy (ciliar)
- Ventilator lung injury
- Diaphragmatic dysfunction
- Ventilator associated pneumonia
- Increased RV afterload → Acute Cor Pulmonale

Inconvenients of Tracheal Intubation

At the time of Intubation

- Gastric aspiration
- Barotrauma
- Hypotension and arrhythmias
- Sedation
- Local trauma (dental, pharynge, larynge or trachea)

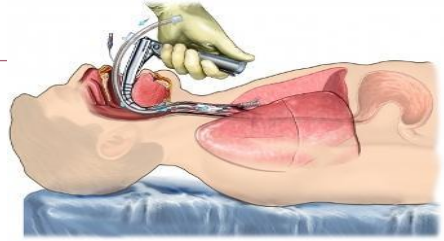


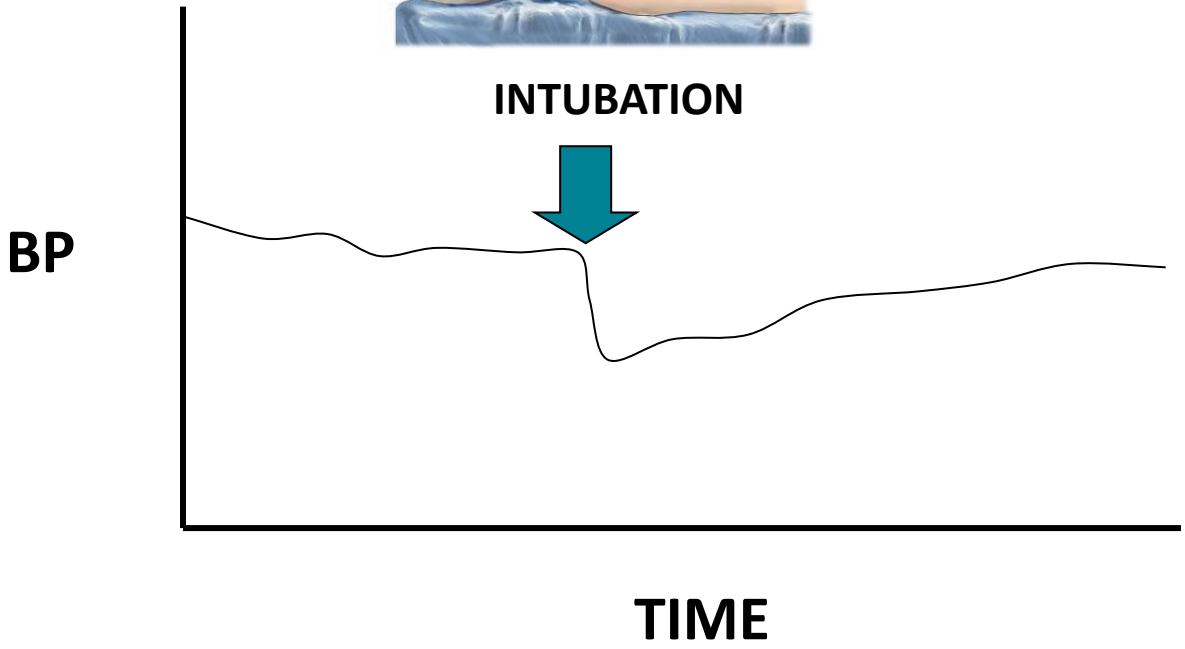
Related to Extubation

- Dysphagia, odinophagia or dysphonia
- Hemoptisis
- Obstruction (chordae dysfunction/edema)
- Tracheal stenosis

Tracheostomy

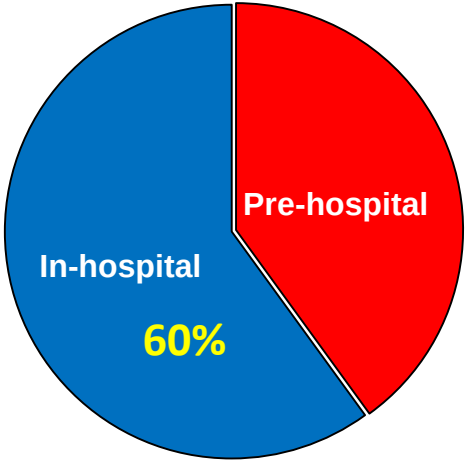
- Hemorrhage
- Infection or obstruction
- False lumen
- Mediastinitis
- Lesions in trachea, esophagus and blood vessels




INTUBATION
BP
TIME

ENDOTRACHEAL INTUBATION IN ACS

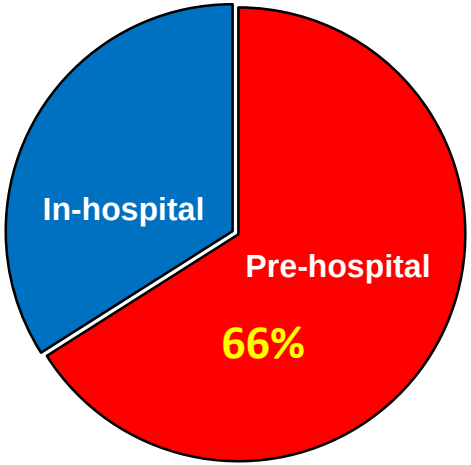
2001-2002: 458 patients
Germany (BEAT registry)



Mortality 48%

Kouraki K. Clin Res Cardiol 2011

2009-2012: 106 patients
Barcelona
Primary PCI 74%



Mortality 29%

Ariza et al. EHJ Acute Cardiovasc Care 2013

VENTILATOR - ASSOCIATED EVENTS

CDC surveillance paradigm (2013)

Incidence rates range:
10–15 events per 1.000 ventilator-days or
4 – 7 events per 100 episodes of MV

VAEs are approximately twice as likely to die, associated with more time on MV, longer ICU stays, and higher rates of antimicrobial use

Klompas. Am J Resp Crit Care 2015

Ventilator Associated Events (VAEs)

NEW PARADIGM

at least 2 days of stable or decreasing ventilator settings followed by at least 2 days of increased ventilator settings

PEEP : 3 cm H2O

(FIO2) of at least 20 points

Date	PEEP (min)	FiO2 (min)
Jan 1	10	100
Jan 2	5	50
Jan 3	5	40
Jan 4	5	40
Jan 5	8	60
Jan 6	8	50
Jan 7	8	40
Jan 8	5	40
Jan 9	5	40

Clinical Events Associated with Ventilator-associated Events

	Klompas *et al.* (15)* (*n* = 44)	Hayashi *et al.* (17) (*n* = 153)	Klein Klouwenberg *et al.* (12)* (*n* = 81)	Boyer *et al.* (20) (*n* = 67)	All Studies Combined* (*n* = 345)
Pneumonia and/or aspiration	10 (23%)	66 (43%)	28 (35%)	21 (31%)	125 (36%)
P					
A					
Acute respiratory distress syndrome	7 (16%)	10 (6.5%)	—	14 (21%)	31 (9.0%)
Mucous plugging	1 (2%)	—	—	—	1 (0.3%)
Abdominal distension/compartment syndrome	1 (2%)	2 (1.3%)	9 (11%)	—	12 (3.5%)
Pulmonary embolus	1 (2%)	3 (2.0%)	—	—	4 (1.2%)
Pneumothorax	—	—	2 (2.5%)	2 (3.0%)	4 (1.2%)
Radiation pneumonitis	1 (2%)	—	—	—	1 (0.3%)
Sepsis syndrome/extrapulmonary infection	1 (2%)	—	9 (11%)	3 (4.5%)	13 (3.8%)
Poor pulmonary toilet	1 (2%)	—	—		1 (0.3%)
Acute neurological event	—	—	10 (12%)		10 (2.9%)
Transfusion-associated lung injury	—	—	—	2 (3.0%)	2 (0.6%)
Other	—	—	—	9 (13%)	9 (2.6%)
No apparent pulmonary complication	18 (41%)	17 (11%)	10 (12%)	—	45 (13%)

PNEUMONIA, EXCESS FLUID, ATELECTASIS, and/or ARDS

*Some ventilator-associated events were attributed to multiple etiologies; hence the percentages exceed 100%.

Three major approaches to prevent VAEs:

(1) Avoid intubation: … Use of NIV

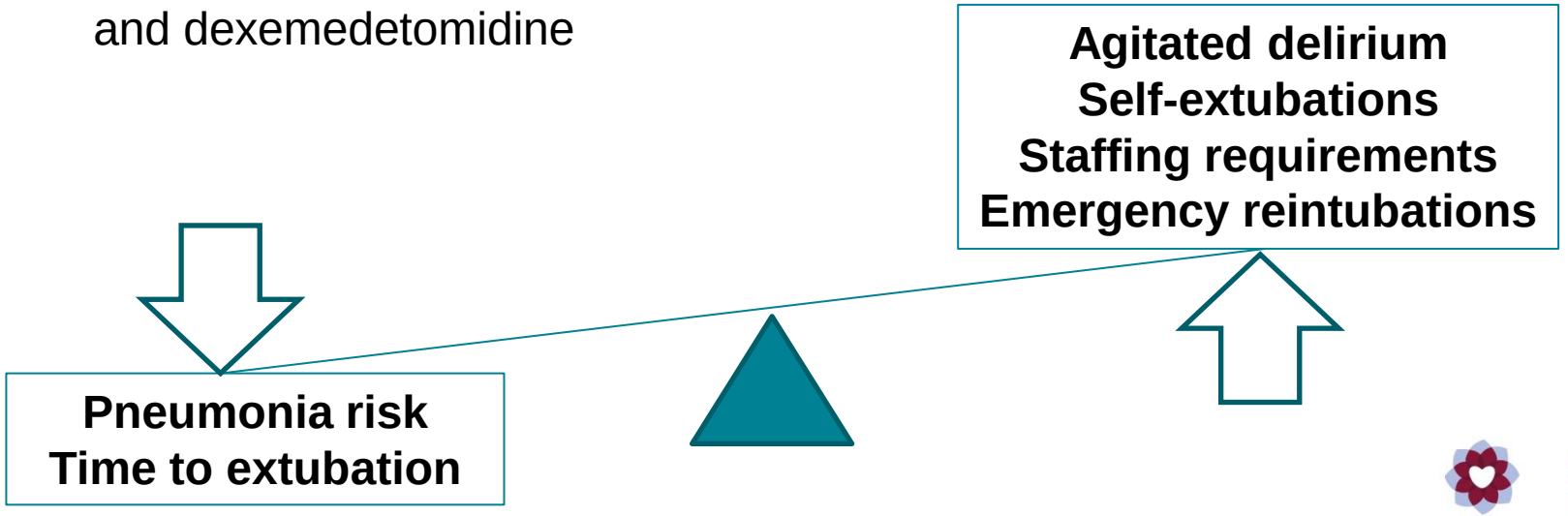
(2) Minimize duration of MV

(3) Target the specific conditions that most frequently trigger VAEs

1. Minimize Sedation

Sedation protocol (RASS Scales, Frequent controls)

Decrease the use of benzodiacepines vs
No sedation, propofol, remifentanil
and dexemedetomidine



2. Daily Spontaneous Awakening Trials and Breathing Trials

Weaning protocol

30 min to 2 h
of
SBT or Pressure Support Ventilation

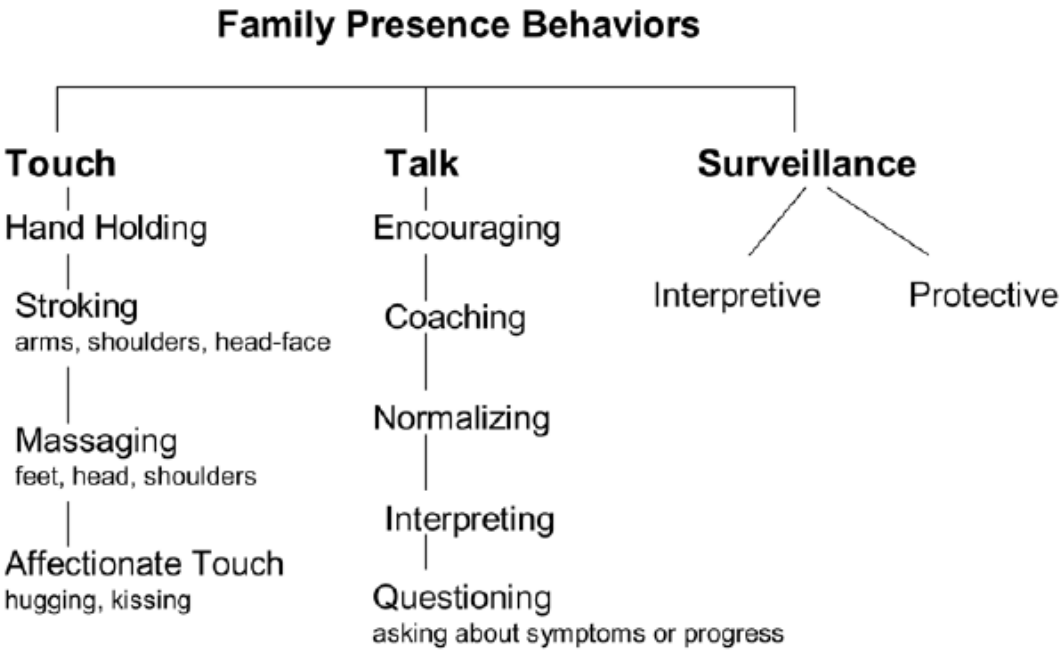
Reconnect Ventilation for 1 h before extubation

3. Programs of Early Exercise and Mobility

- **Physiotherapists**
- **Mobilization protocol**
- **Nurse training**
- **Family collaboration**

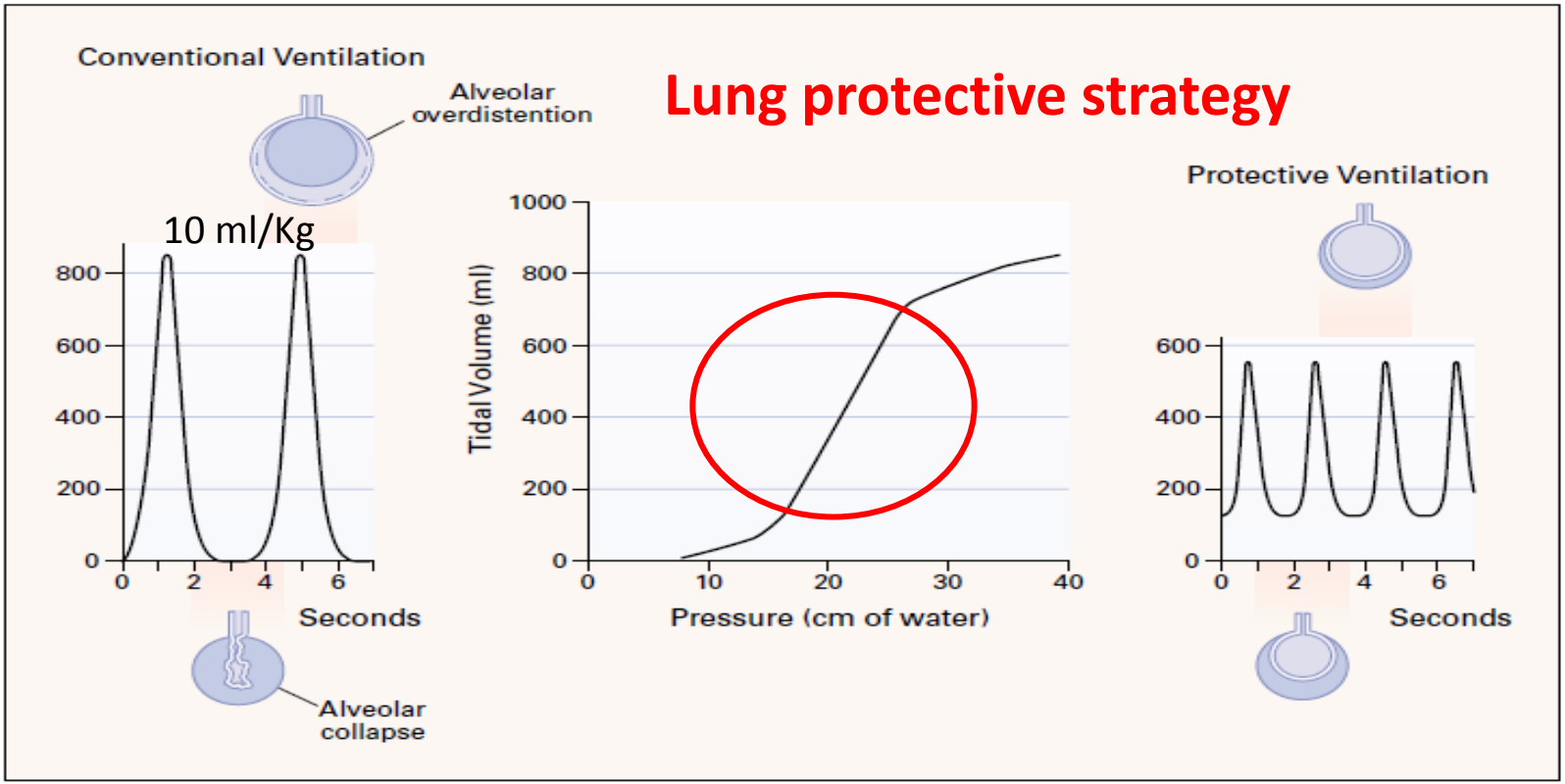
ABCDE package
Awakening and Breathing Coordination
Delirium monitoring and management
Early exercise and Mobility

Family collaboration



Happ MB. Heart Lung. 2007

4. Appropriate ventilation strategy



Tobin. M, N Engl J Med 2001

Appropriate ventilation strategy

- Low tidal volumes (6-7 ml/Kg)
- Higher frequencies
- Adjust PEEP
- Low plateau pressure (<27cmH2O) and driving pressure (<17cmH2O)
- Permissive, but controlled hypercapnia
- Meassures to prevent VAP
- Avoid F_IO_2 >0.6 Prone position

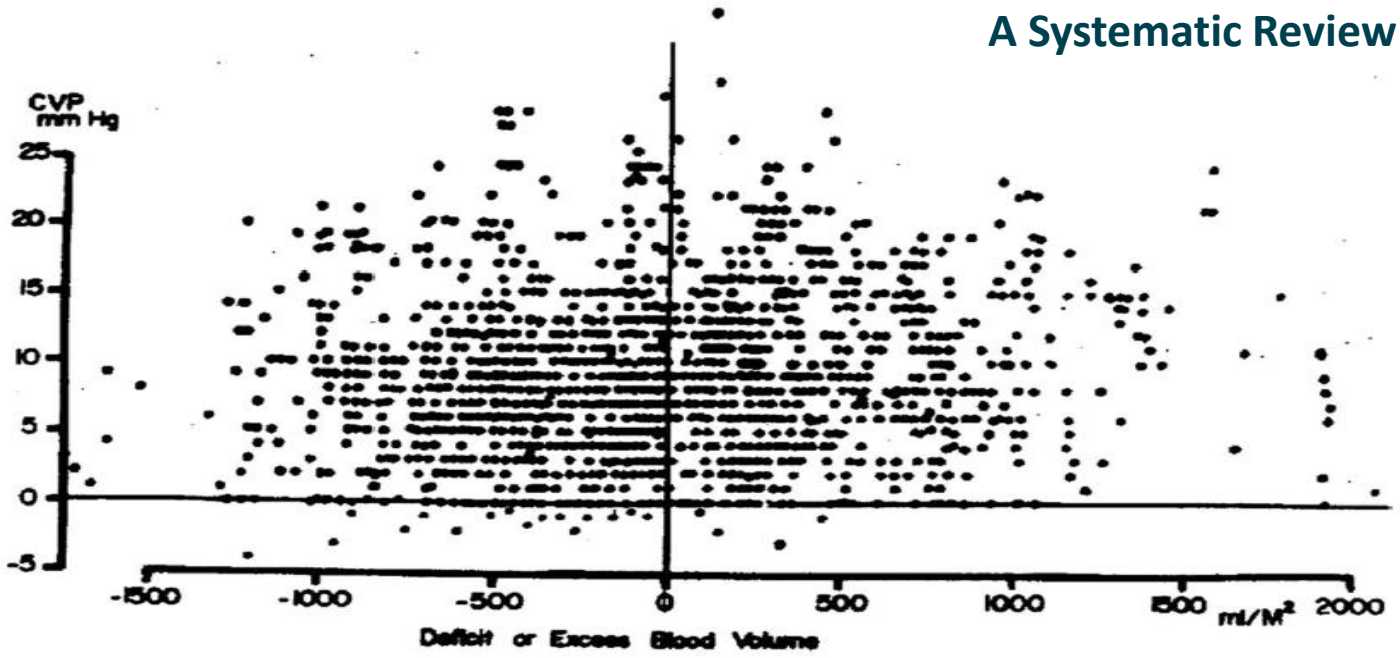
5. Conservative Fluid Management

20–40% of VAEs are attributable to fluid overload including congestive heart failure, pulmonary edema and new pleural effusions

Physical examination, CVP, PCWP, extravascular lung volume, mean arterial pressure, urinary output, cardiac index, IVC, E/E'

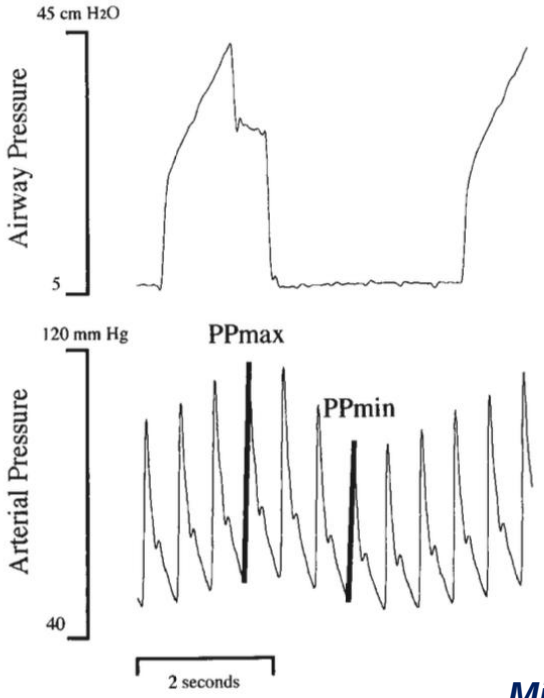
Central Venous Pressure and Fluid Responsiveness

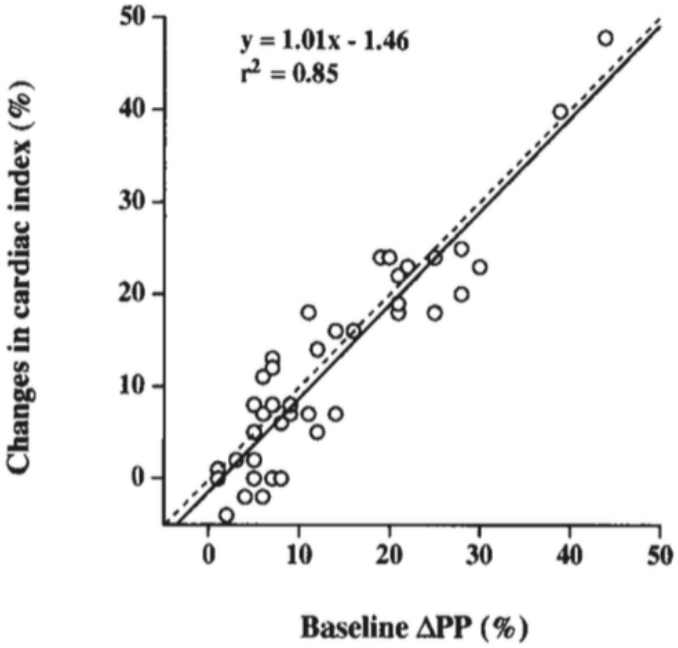
A Systematic Review



Marik PE et al. Chest 2008

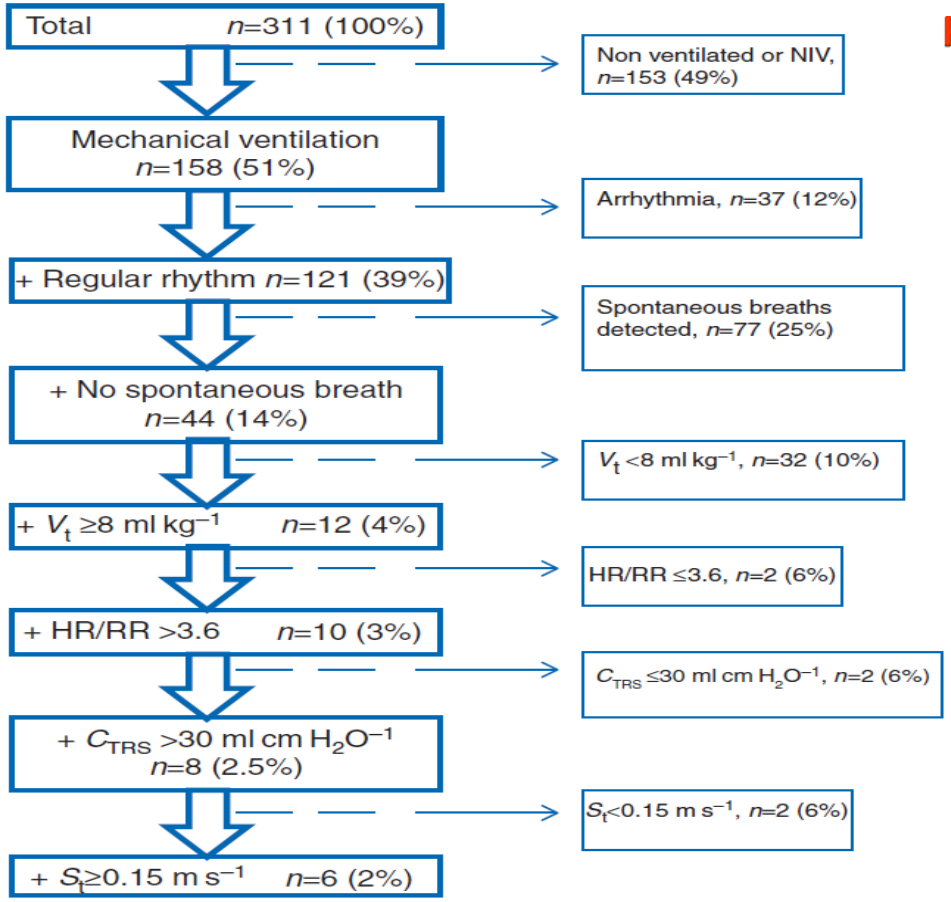
Pulse Pressure Variation with respiration





Michard F . Am J Respir Crit Care Med 2000

Pulse Pressure Variation (PPV)



Conditions:

- **Mechanical ventilation**
- **No arrhythmia**
- **No spontaneous breathing**
- **Constant Vt ≥7 ml/kg**
- **RR < 30**
- **No RVF**

Mahjoub Y et al Br J Anesthesia 2013

Passive Leg Raising

- **PLR and LVOT VTI (TTE) ↑ 12%**
- **PLR and SV (TTE) ↑ 12.5% predicts SV ↑ 15% after volume load (Sens. 77%; Spec. 100%)**

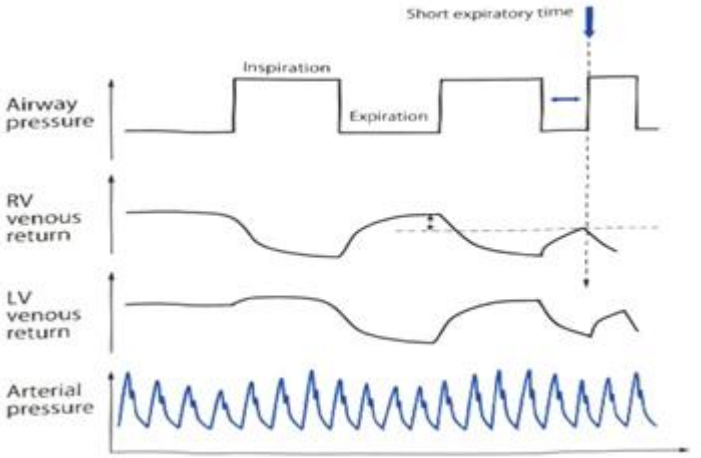
Monnet X. Intensive Care Med 2008

Lamia: Intensive Care Med 2007

Other methods :

End-expiratory occlusion test

15s occlusion at end-expiration PP or PCA-CO ↑5%
Sens 87%, Spec 100% for response to 500 ml



Minifluid challenge

100 ml of colloid/1 min: LVOT VTI ↑ 10%
Sens 95%, Spec 78% for response to volume

Monnet X, Crit Care Med 2012 *Muller L, Anesthesiology 2011*

6. Conservative Blood Transfusion Thresholds

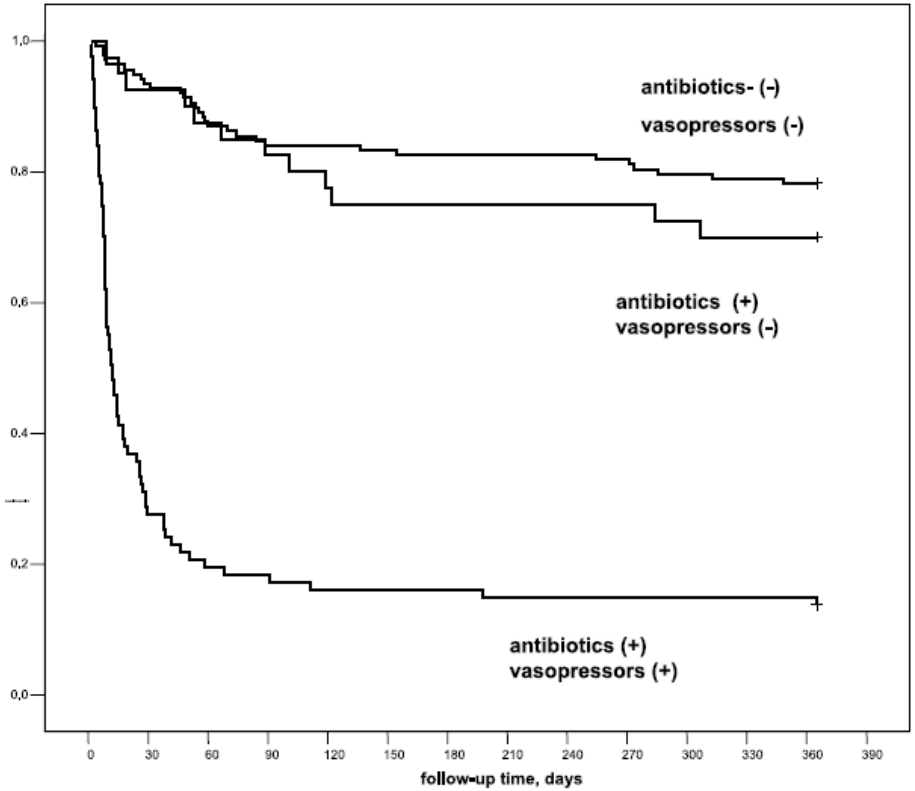
7. Ventilator Associated Pneumonia Prevention

Oral care with chlorhexidine **Subglottic secretion drainage**	**Unlikely**
Elevating the Head of the Bed **Hand washing** **Disposable gloves** **Sterile aspiration**	**Likely**

Campaigns: Pneumonia Zero, Bacteremia Zero, Resistance Zero

VAP: from 15/1000days **MV to** 5/1000 daysMV

Characteristics of Patients With Complicated ACS Requiring Prolonged Mechanical Ventilation



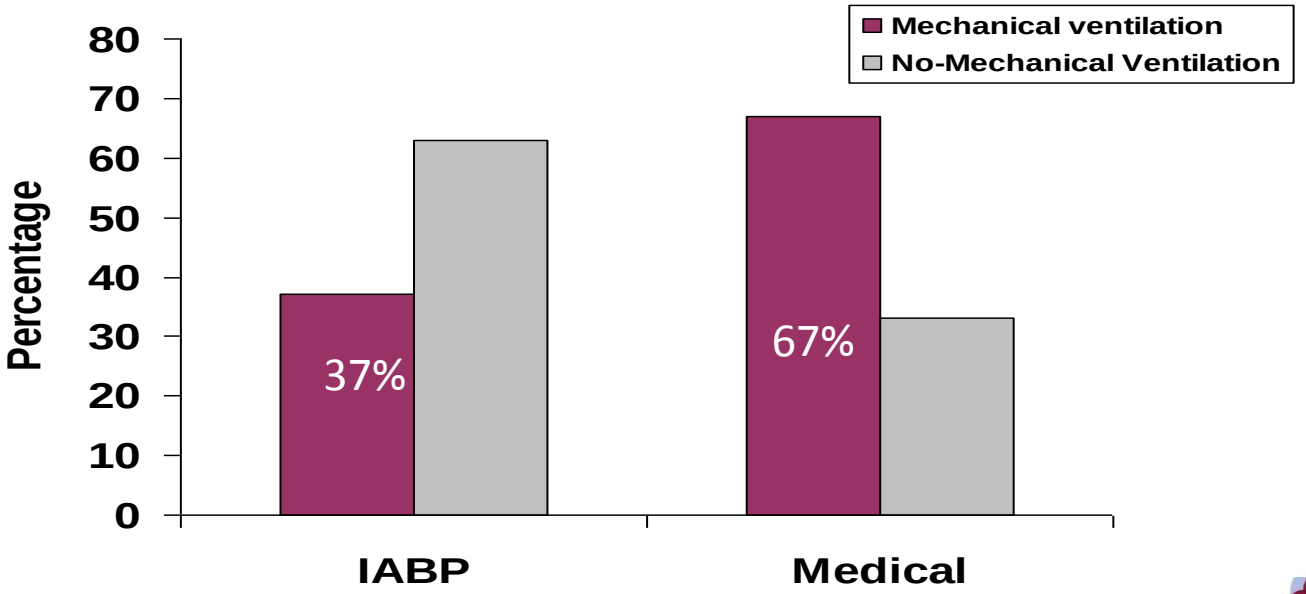
267 patients

Zahger D. Am J Cardiol 2005

COMPLEMENTARY EFFECTS OF MV AND IABP

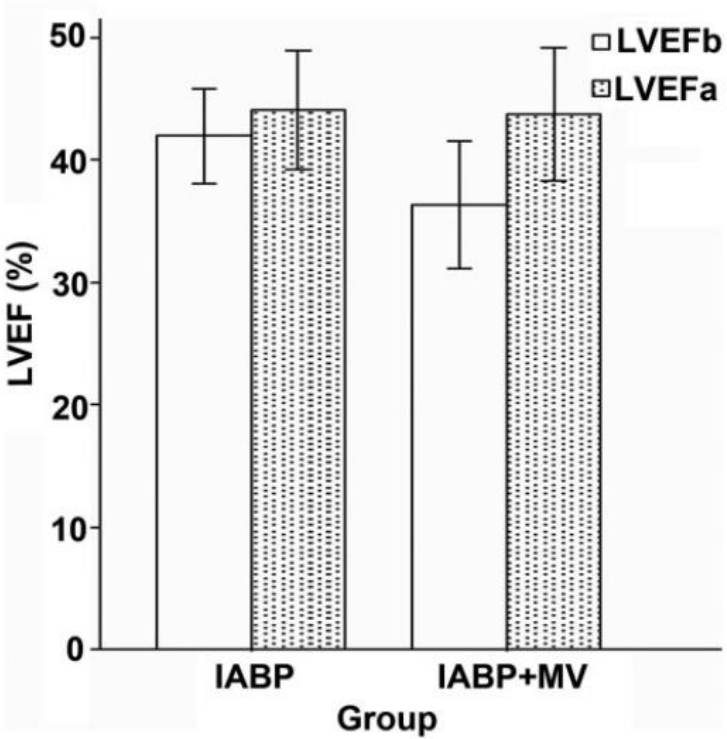
IABP- Shock Trial

45 patients AMI-PCI shock randomized to IABP



Prondzinsky R et al, Crit Care Med 2010

COMPLEMENTARY EFFECTS OF MV AND IABP



Liu H. J International Med Res 2016

CONCLUSIONS

MV is a good friend that has saved millions of lives

In patients with CS it should be used in cases with severe respiratory failure or altered mental status that can not be managed by other ways

The appropriate use of the technique targeted to shorten the duration of MV and avoiding ventilator events is essential to sustain this friendship


Thank you for your attention