CNRM
COLLEGE NATIONAL DE REANIMATION MEDICALE

كلية بتونس
FACULTE DE MEDECINE DE TUNIS

Exacerbations sévères de BPCO: Diagnostic et traitement

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INTRODUCTION

- BPCO ➔Trois premières causes de décès

- Problème de santé public ➔ Traitement

➔Prévention

Global Initiative for Chronic Obstructive Lung Disease

2023 REPORT

Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease

DEFINITIONS

Bronchite chronique

- Abandonnée par le GOLD

Insuffisance respiratoire chronique

- Incapacité permanente de l'appareil respiratoire à assurer une *hématose*
- La définition est *gazométrique*: $PaO_2 < 70$ mmHg sur 2 gaz du sang artériels en état stable à ≥ 3 mois d'intervalle
- Insuffisance respiratoire grave si $PaO_2 \leq 55$ mmHg ou < 60 si *polyglobulie* ou *insuffisance cardiaque droite* associée.

Bronchopneumopathie chronique obstructive (BPCO)

- Maladie respiratoire hétérogène
- Symptômes respiratoires chroniques
- Obstruction permanente et progressive des voies aériennes et/ou alvéolaires

Exacerbation de BPCO

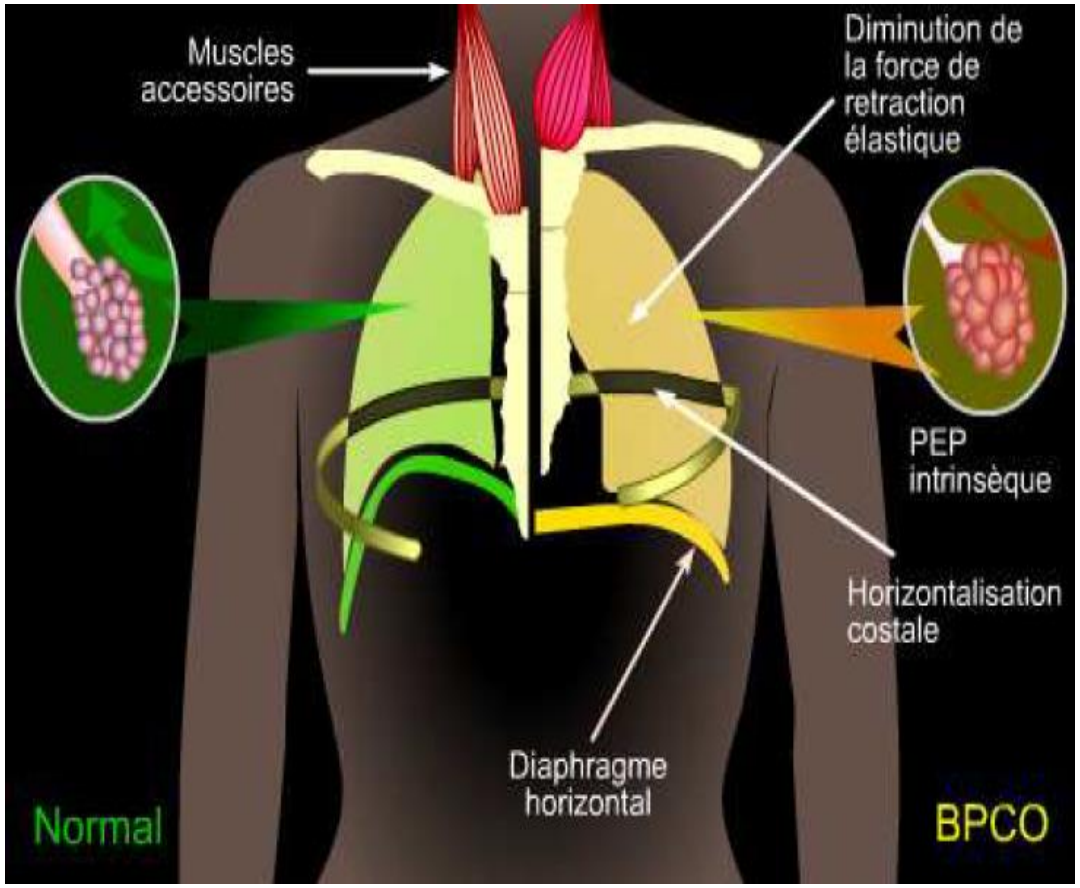
- Le diagnostic est clinique
- Aggravation de dyspnée et/ou de la toux et des expectorations
- En moins de 14 jours

Exacerbation de BPCO

- Définitions après résolution de l'exacerbation (*GOLD 2023*)
- Exacerbations fréquentes: ≥ 2 exacerbations/an
  (1er facteur prédictif de survenue d'EBPCO)

PHYSIOPATHOLOGIE

ANOMALIES ANATOMIQUE

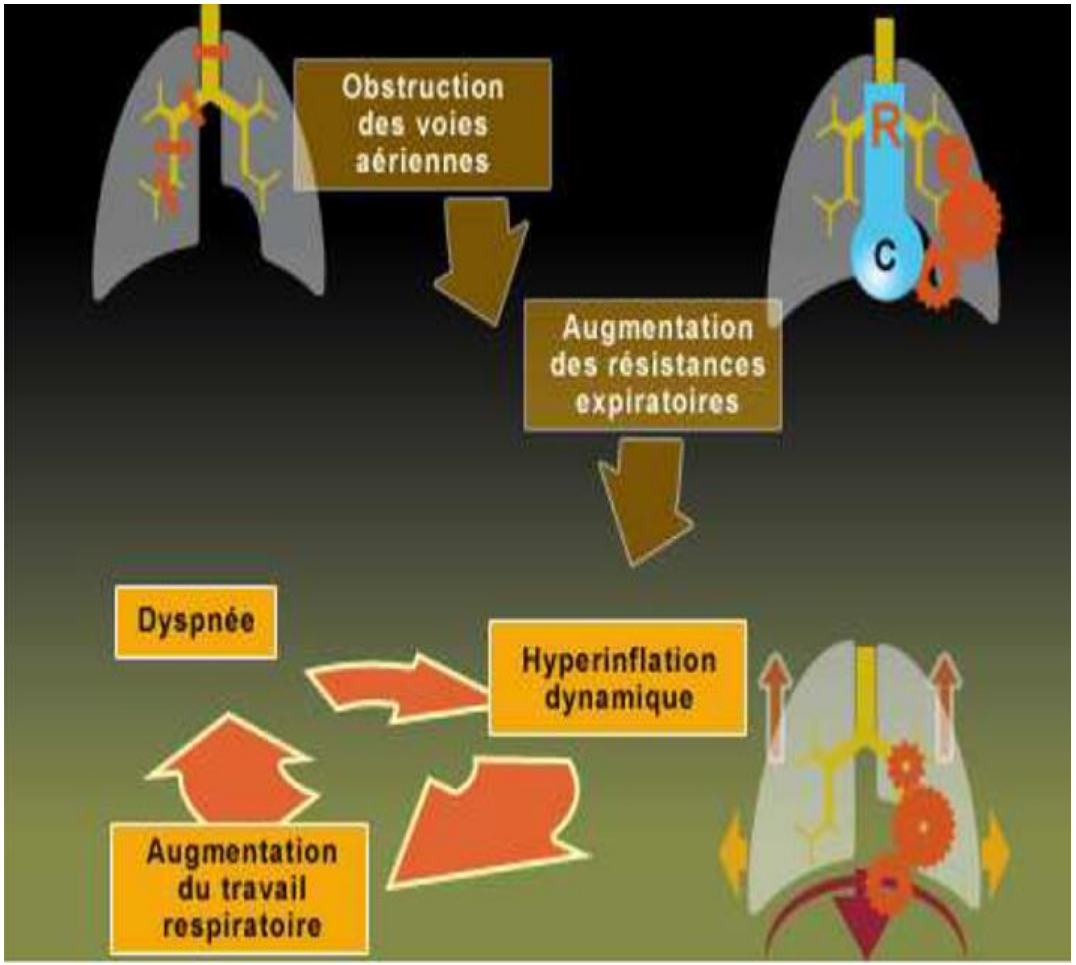


- **Atteinte bronchique:**
  - **Hypersécrétion**
  - **œdème**
  - **Bronchoconstriction**
- **Atteinte parenchymateuse:**
  - **Perte des forces de rétraction élastiques du poumon**

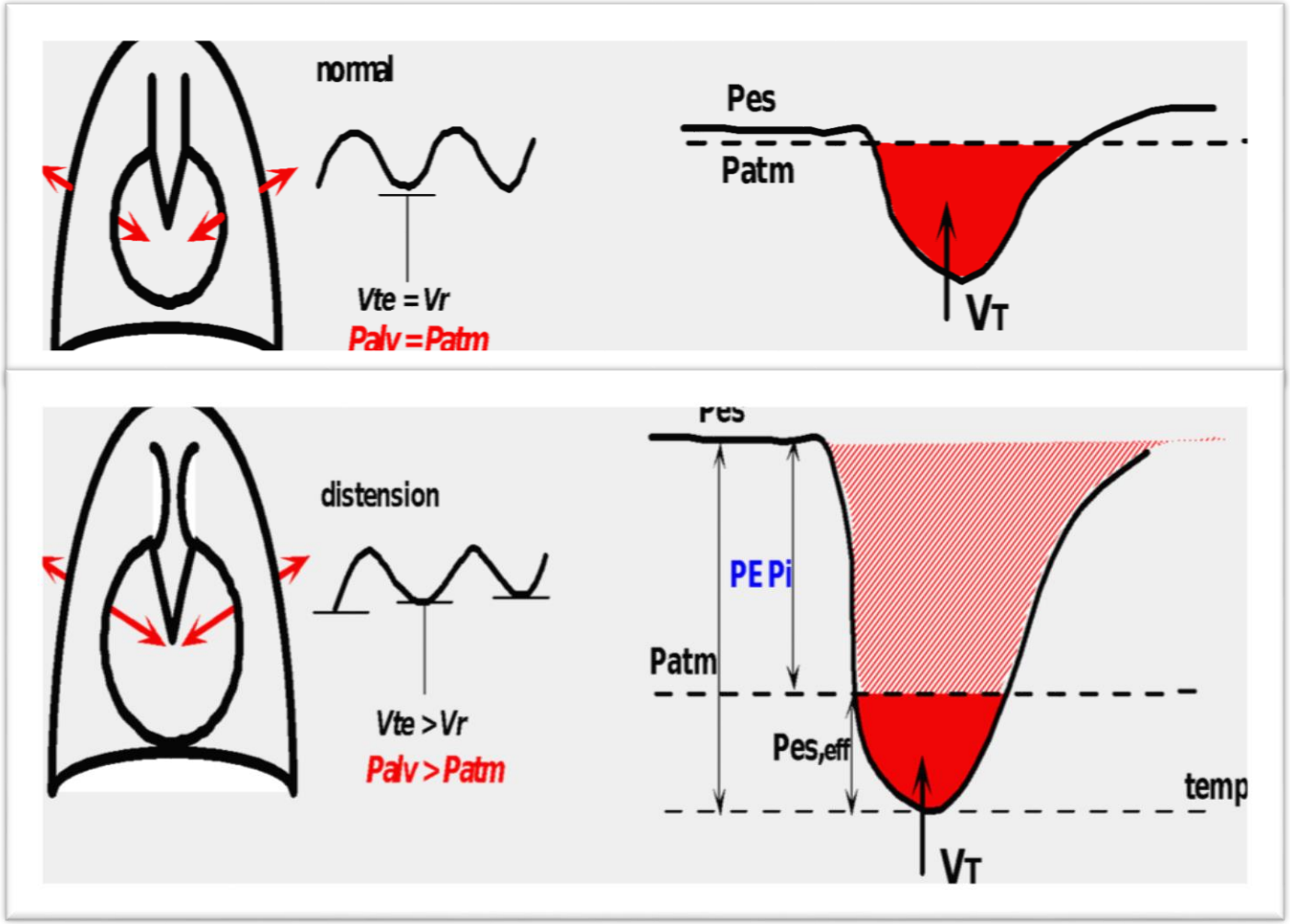
OBSTRUCTION

⇒**LIMITATION DÉBIT EXPIRATOIRE**

CONSÉQUENCES SUR LA MÉCANIQUE RESPIRATOIRE



D'UNE ANOMALIE EXPIRATOIRE À UNE CHARGE INSPIRATOIRE



CONSÉQUENCES GAZOMÉTRIQUES

- **Hypoxémie par inégalité VA/Q**

- **Hypercapnie par ↑ espace mort ,**
  hypoventilation alvéolaire secondaire à la fatigue muscles respiratoires

Exacerbations

DIAGNOSTIC

BPCO

CRITÈRES ANAMNESTIQUES

- **Facteurs de risque**
- Tabagisme
- Pollution atmosphérique
- Chauffage au bois ou au charbon
- Exposition professionnelle

- **histoire familiale de BPCO**

BPCO

CRITÈRES ANAMNESTIQUES

Dyspnée

Progressive

Aggravée à l'effort

Persistante

Toux chronique

intermittente

productive ou non

sifflement expiratoire

Expectoration chronique

Infections récurrentes des voies aériennes inférieures

BPCO

Examen clinique

➔Leurs absence n’exclus pas le diagnostique

- Déformation du thorax
- Hippocratisme digital
- Cyanose
- Signe de lutte
- Une respiration à « lèvres pincées »…

BPCO

Spiromètre

- *Trouble ventilatoire obstructif* (VEMS/CVF < 0,7) non réversible
- Non spécifique
- Contexte

Role of Spirometry in COPD

Table 2.5

- **Diagnosis**
- **Assessment of severity of airflow obstruction (for prognosis)**
- **Follow-up assessment**
  - Therapeutic decisions
    - Pharmacological in selected circumstances (e.g., discrepancy between spirometry and level of symptoms)
    - Consider alternative diagnoses when symptoms are disproportionate to degree of airflow obstruction
    - Non-pharmacological (e.g., interventional procedures)
  - Identification of rapid decline

Exacerbation BPCO

1 .Données clinique

2 . Données para-cliniques

3 . Diagnostics différentiels

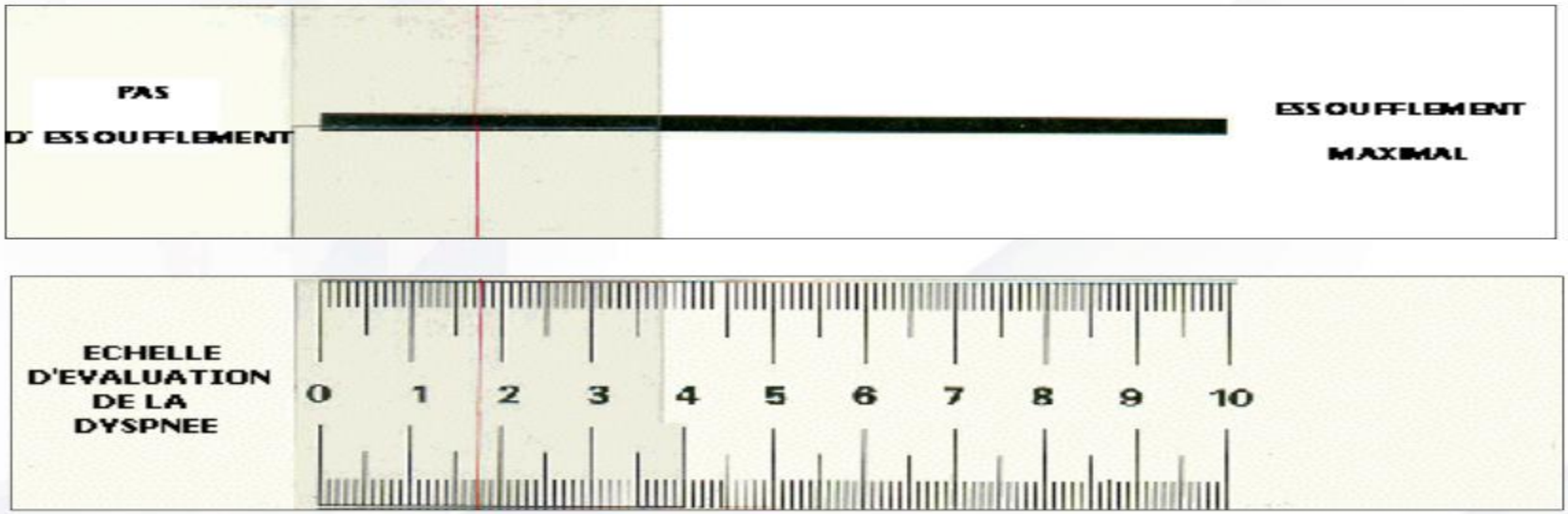
4 . Classification selon la sévérité

5 . Le facteur déclenchant

Exacerbation BPCO

Données clinique

L'Echelle Visuelle Analogique appliquée à la dyspnée



Gift. validation of a vertical vas as a measure of clinical dyspnea. Rehab nurs 1989; 14:313-25

Exacerbation BPCO

Données clinique

Examen respiratoire
- Fréquence respiratoire
- Présence de signe de lutte
- SpO2
- Aspect et volume des secrétions
- Cyanose

Examen cardiovasculaire
-Fréquence cardiaque
-PA

Examen neurologique
-GCS
-flapping tremor

Exacerbation BPCO

Données para-cliniques

Examens systématique	
Gaz du sang artériels	**Evaluation de la sévérité de l'exacerbation**
NFS	Facteur déclenchant
CRP	**Evaluation de la sévérité**
Radiographie de thorax	Facteur déclenchant
ECG	Facteur déclenchant

Exacerbation BPCO

Diagnostics différentiels
Dyspnée

Pneumopathie aiguë communautaire

Insuffisance cardiaque

Embolie pulmonaire

Exacerbation BPCO

Classification selon la sévérité

Exacerbation légère

EVA dyspnée <5
FR<24 c/min
FC< 95 bpm
SpO2 ≥92% ou ↘ de moins de 3%
CRP< 10 mg/l

Exacerbation BPCO

Classification selon la sévérité

Exacerbation Modérée (au moins 3)

- EVA dyspnée >5
- FR ≥ 24 c/min
- FC ≥ 95 bpm
- SpO2 <92% ou ↘ de plus de 3%
- CRP ≥ 10 mg/l
- PaO2<60mmHg et/ou PCo2 >45 mmHg sans acidose

Exacerbation BPCO

Classification selon la sévérité

Exacerbation Sévère

- EVA dyspnée >5
- FR ≥ 24 c/min
- FC ≥ 95 bpm
- SpO2 <92% ou ↘ de plus de 3%
- CRP ≥ 10 mg/l
- PaO2<60mmHg ou PCo2 >45 mmHg
- Acidose respiratoire

Exacerbation BPCO

Classification selon la sévérité

Après résolution de l'exacerbation

➔**Exacerbation légère**

Résolutive en majorant les bronchodilatateurs de courte durée d'action

➔**Exacerbation modérée**

Nécessitant bronchodilatateurs de courte durée
Corticoïdes oraux

± Antibiotiques

➔**Exacerbation sévère**

Passage aux urgences ou hospitalisation

IRA

Exacerbation BPCO

Le facteur déclenchant

Anamnèse

➔Iatrogénie

-Fort débit d'O2

-Prise de psychotropes : neuroleptiques, antidépresseurs

-Prise de diurétiques

-Prise d'antitussifs

Exacerbation BPCO

Le facteur déclenchant

Anamnèse

➔ **Pollution:**

-Ozone

-Dioxyde de soufre,

-Dioxyde d’azote

Exacerbation BPCO

Le facteur déclenchant

Anamnèse

➔Observance au traitement

Exacerbation BPCO

Le facteur déclenchant

Examens à réaliser au cas par cas selon les hypothèses diagnostiques	
Suspicion d'infection	- HC - ECBC
Suspicion d'insuffisance cardiaque gauche	- NT-proBNP - Echocardiographie
Suspicion d'embolie pulmonaire	- D-dimères - Doppler des membres inférieures - Angioscanner thoracique - Echocardiographie

TRAITEMENT

Oxygénothérapie

- Elément essentiel
- Objectif SpO2 entre 88-92%
- Surveillance GDS

Oxygénothérapie haut débit

- FiO2 précise
- Effet peep → recrutement alvéolaire fermée
- Un wash-out de l'espace mort → amélioration de la capnie
- Une humidification et un réchauffement de l'air (31-37°C) → fluidification des sécrétions

Oxygénothérapie haut débit

Diminution de la FR et du travail respiratoire

Amélioration des échanges gazeux

Oxygénothérapie haut débit

Place au cours de l'exacerbation BPCO

Pulmonol. 2019;25(6):348–354

PULMONOLOGY

www.journalpulmonology.org

REVIEW

High flow through nasal cannula in exacerbated COPD patients: a systematic review

L. Pisani[a,*], M. Astuto[b], I. Prediletto[a], F. Longhini[c]

[a] Respiratory and Critical Care Unit, S. Orsola-Malpighi Hospital, Alma Mater University, Bologna, Italy
[b] Department of Clinical and Experimental Medicine, University of Catania, Catania, Italy
[c] Intensive Care Unit, University Hospital Mater Domini, Department of Medical and Surgical Sciences, Magna Graecia University, Catanzaro, Italy

Study design

Of the 5 studies included in the systematic review, only one was multicentered,[9] while the remaining four were single centered.[14–17] Two studies were randomized controlled trials,[14,17] while the others were cross-over randomized trials.[9,15,16] The median [interquartile range] of the mean duration of the studies was 19 [14–22] months. All studies were performed in a university hospital, two (40%) in Italy,[9,15] one (20%) in China,[14] one (20%) in New Zealand[16] and one (20%) in South Korea.[17]

Conclusions

Although the available clinical data for application of HFNC in COPD exacerbation are increasing over time, there are still some unanswered questions regarding practical aspects of its use.

Randomized controlled trials are needed to clarify the real efficacy of HFNC and the best target population during COPD exacerbation. In addition, data should confirm whether therapy with HFNC could play a role in addition to or as an alternative to COT and NIV in the various settings described above.

Oxygénothérapie haut débit

Place au cours de l'exacerbation BPCO

Systematic Review Article

High Flow Through Nasal Cannula in Stable and Exacerbated Chronic Obstructive Pulmonary Disease Patients

Author(s): Andrea Bruni, Eugenio Garofalo, Gianmaria Cammarota, ">Paolo Murabito, ">Marinella Astuto, Paolo Navalesi, ">Francesco Luzza, ">Ludovico Abenavoli and Federico Longhini*

Results: Twenty-six studies have been included. HFNC: 1) provides heated and humidified airoxygen admixture; 2) washes out the anatomical dead space of the upper airway; 3) generates a small positive end-expiratory pressure; 4) guarantees a more stable inspired oxygen fraction, as compared to conventional oxygen therapy (COT); and 5) is more comfortable as compared to both COT and non-invasive ventilation (NIV).

Conclusion: Though evidence of superiority still lacks and further studies are necessary, HFNC might play a role in the treatment of both stable and exacerbated COPD patients.

VNI

1 ère modalité ventilatoire

INDICATIONS

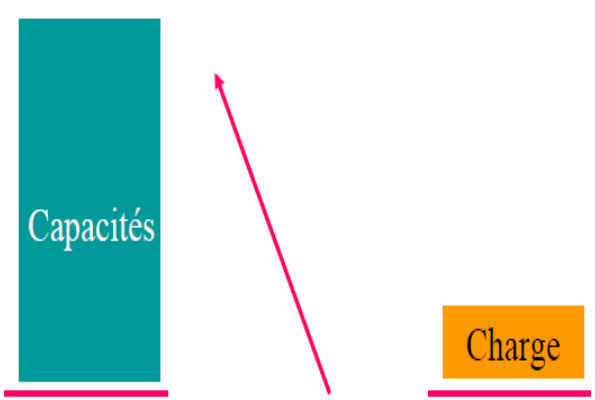
Indications for Noninvasive Mechanical Ventilation (NIV) — Table 5.7

At least one of the following:

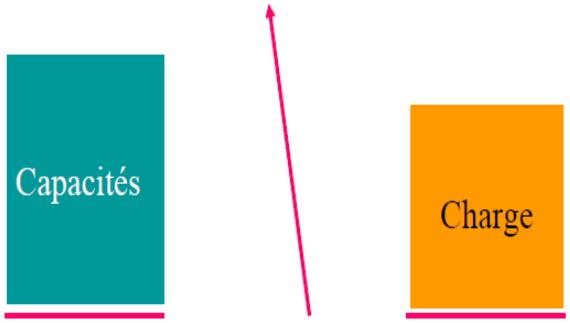
- Respiratory acidosis ($PaCO_2 \geq 6.0$ kPa or 45 mmHg and arterial pH ≤ 7.35)
- Severe dyspnea with clinical signs suggestive of respiratory muscle fatigue, increased work of breathing, or both, such as use of respiratory accessory muscles, paradoxical motion of the abdomen, or retraction of the intercostal spaces
- Persistent hypoxemia despite supplemental oxygen therapy

VNI

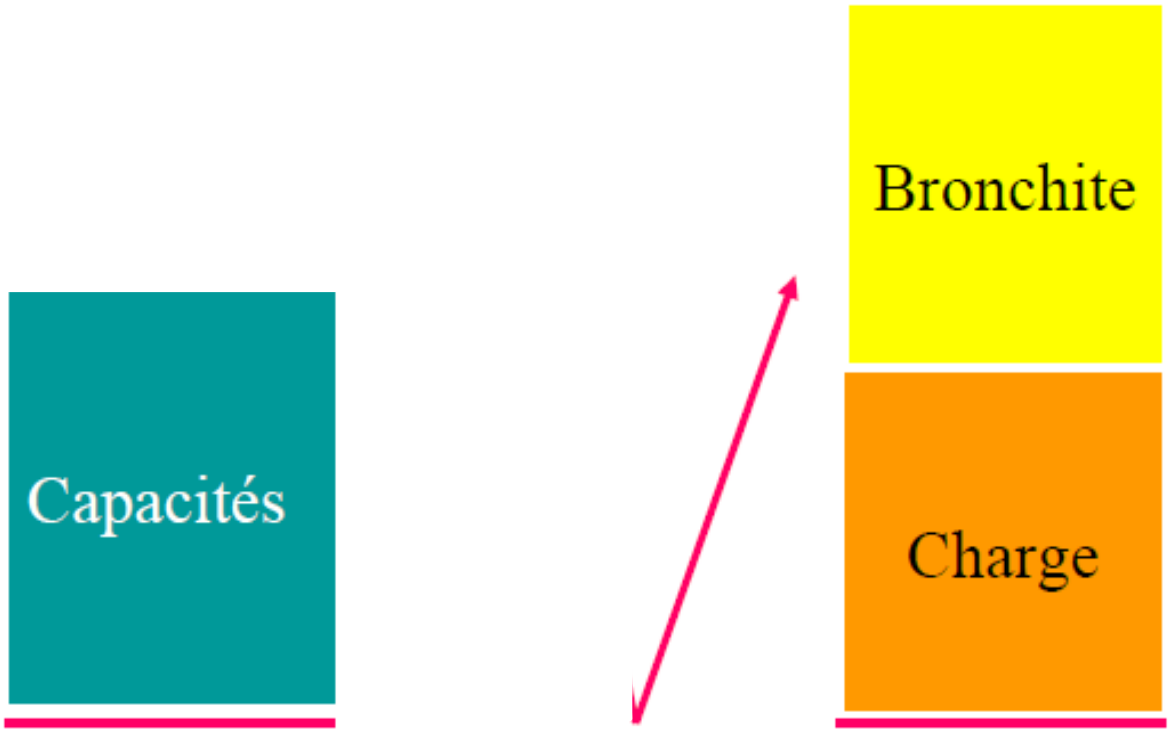
Sujet sain au repos



Sujet BPCO au repos

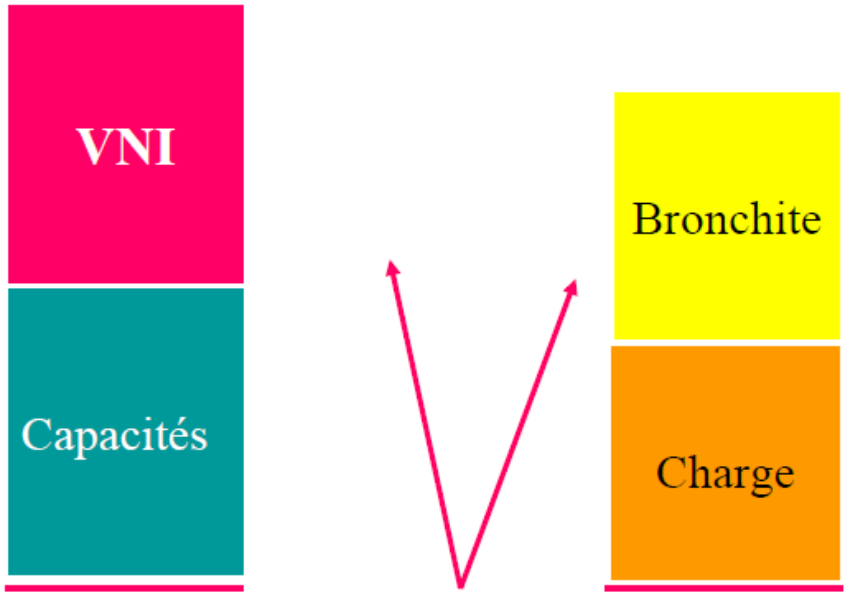


BPCO en Exacerbation



VNI

Sujet BPCO décompensé



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Effets

Réduction du travail inspiratoire

PEP extrinsèque

↓ gradient de pression pour initialiser l'inspiration en contrebalançant la PEP intrinsèque

VNI

<u>Pression Inspiratoire Positive: Aide Inspiratoire</u> (AI)

- **Améliore les échanges gazeux:**
  - ✓ **Augmentation de la ventilation alvéolaire (+++)**
  - ✓ **Amélioration des rapports VA/Q (+)**
- **↑ le Vt , ↓ la FR**

VNI

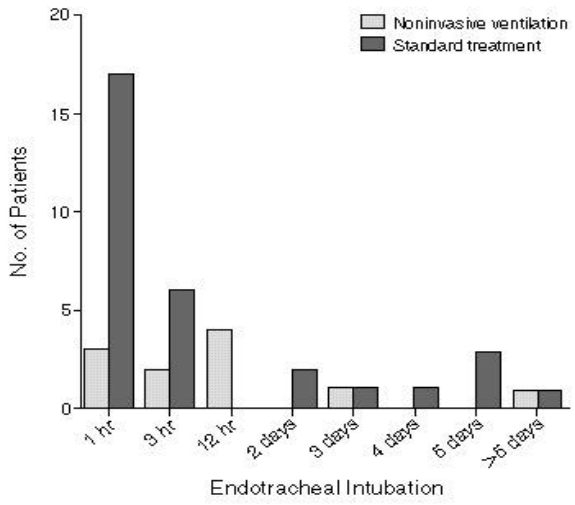
BENEFICES

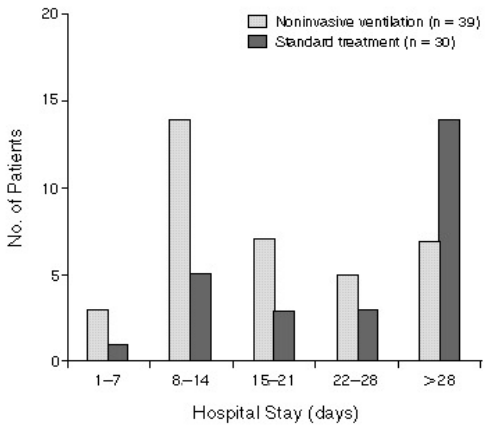
ORIGINAL ARTICLE

Noninvasive Ventilation for Acute Exacerbations of Chronic Obstructive Pulmonary Disease

Laurent Brochard, M.D., Jordi Mancebo, M.D., Marc Wysocki, M.D., Frédéric Lofaso, M.D., Giorgio Conti, M.D., Alain Rauss, M.D., Gérald Simonneau, M.D., Salvador Benito, M.D., Alessandro Gasparetto, M.D., François Lemaire, M.D., Daniel Isabey, Ph.D., and Alain Harf, M.D.

September 28, 1995
N Engl J Med 1995; 333:817-822





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BENEFICES

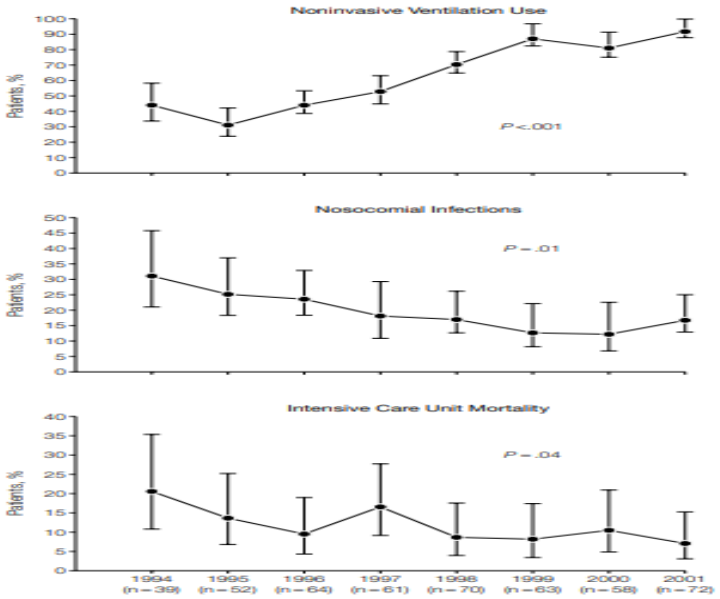
Secular Trends in Nosocomial Infections and Mortality Associated With Noninvasive Ventilation in Patients With Exacerbation of COPD and Pulmonary Edema

Emmanuelle Girou, PharmD
Christian Brun-Buisson, MD
Solenne Taillé, Biomed Eng Master
François Lemaire, MD
Laurent Brochard, MD

Context Randomized controlled trials have shown that the use of noninvasive ventilation (NIV) reduces the need for endotracheal intubation and invasive mechanical ventilation and reduces complication rates and mortality in selected groups of patients. But whether these benefits translate to a clinical setting is unclear.

Objective To evaluate longitudinally the routine implementation of NIV and its effect on patients admitted to the intensive care unit (ICU) with acute exacerbation of chronic obstructive pulmonary disease (COPD) or severe cardiogenic pulmonary

Figure 2. Long-term Noninvasive Ventilation–Use Trends Over 8 Years



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Cochrane Library

Cochrane Database of Systematic Reviews

Non-invasive ventilation for the management of acute hypercapnic respiratory failure due to exacerbation of chronic obstructive pulmonary disease (Review)

Osadnik CR, Tee VS, Carson-Chahhoud KV, Picot J, Wedzicha JA, Smith BJ

Figure 1. Study flow diagram for 2004-2017 literature searches.

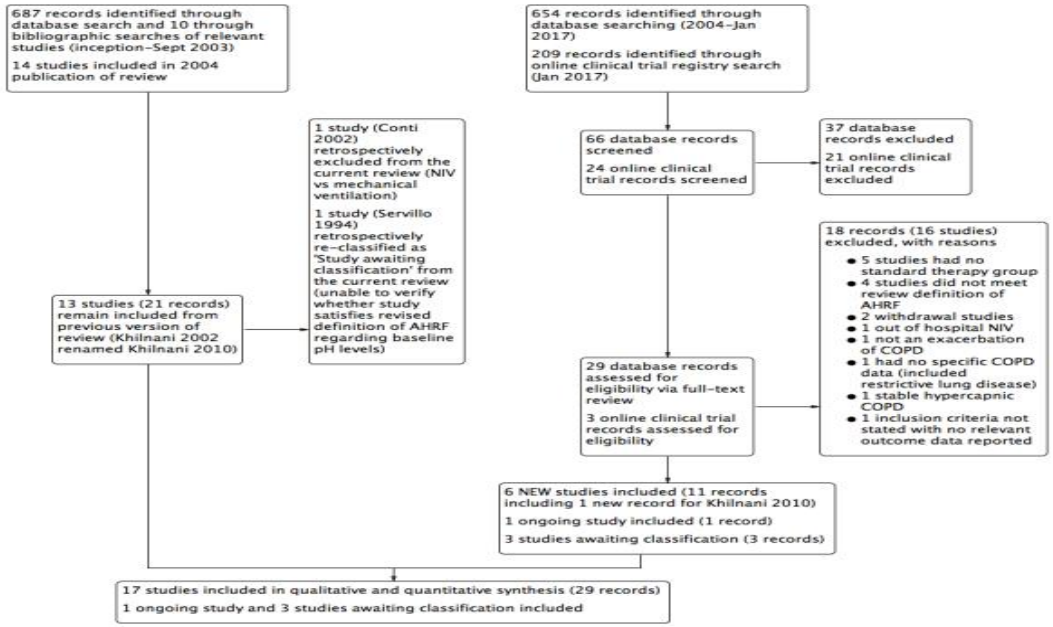

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Figure 6. NIV vs usual care (overall) - Need for endotracheal intubation

Study or Subgroup	NIV Events	NIV Total	Usual care Events	Usual care Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Avdeev 1998	3	29	8	29	4.2%	0.38 [0.11, 1.27]
Barbe 1996	0	14	0	10		Not estimable
Bott 1993	0	30	2	30	1.3%	0.20 [0.01, 4.00]
Brochard 1995	11	43	31	42	16.7%	0.35 [0.20, 0.60]
Carrera 2009	5	37	13	38	6.8%	0.40 [0.16, 1.00]
Celikel 1998	1	15	2	15	1.1%	0.50 [0.05, 4.94]
Collaborative 2005	6	100	17	91	9.5%	0.32 [0.13, 0.78]
del Castillo 2003	1	20	3	21	1.6%	0.35 [0.04, 3.09]
Dikensoy 2002	2	17	7	17	3.7%	0.29 [0.07, 1.18]
Khilnani 2010	3	20	12	20	6.4%	0.25 [0.08, 0.75]
Kramer 1995	1	11	8	12	4.1%	0.14 [0.02, 0.92]
Liu 2005	2	18	8	18	4.2%	0.25 [0.06, 1.02]
Matuska 2006	3	30	10	30	5.3%	0.30 [0.09, 0.98]
Plant 2001	18	118	32	118	17.0%	0.56 [0.34, 0.94]
Samaria 2009	4	20	11	20	5.8%	0.36 [0.14, 0.95]
Thys 2002	0	7	5	5	3.3%	0.07 [0.00, 1.01]
Zhou 2001	7	30	17	30	9.0%	0.41 [0.20, 0.85]
Total (95% CI)		**559**		**546**	**100.0%**	**0.36 [0.28, 0.46]**
Total events	67		186			

Heterogeneity: $Chi^2 = 6.68$, df = 15 (P = 0.97); $I^2 = 0\%$
Test for overall effect: Z = 8.22 (P < 0.00001)

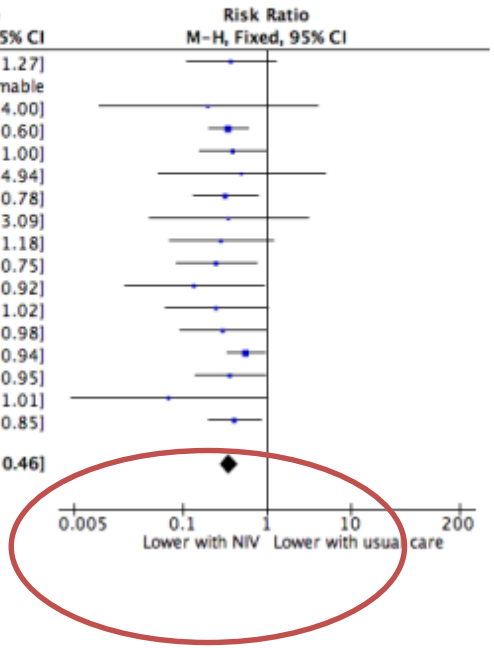

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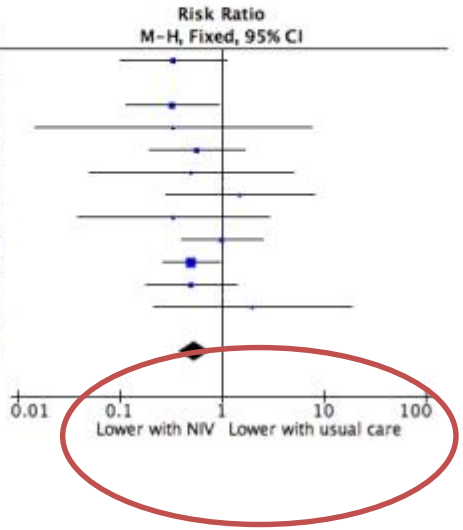
Figure 3. NIV vs usual care (overall) - Mortality

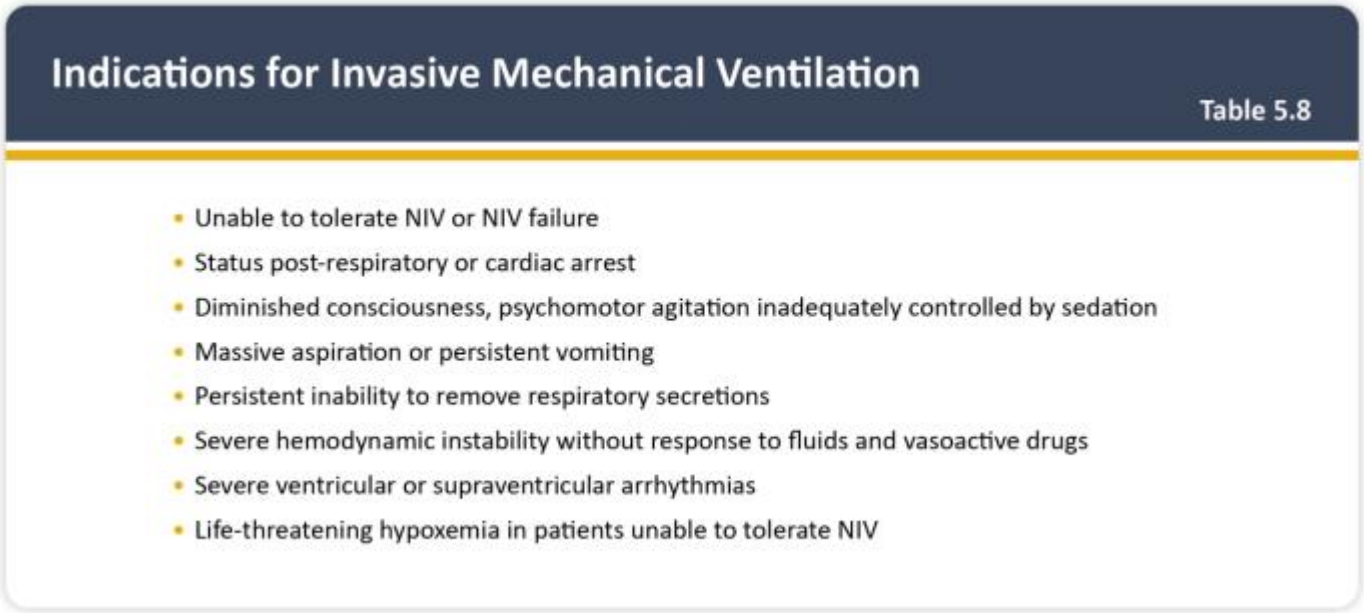
Study or Subgroup	NIV Events	NIV Total	Usual care Events	Usual care Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Avdeev 1998	3	29	9	29	11.5%	0.33 [0.10, 1.11]
Barbe 1996	0	14	0	10		Not estimable
Brochard 1995	4	43	12	42	15.6%	0.33 [0.11, 0.93]
Celikel 1998	0	15	1	15	1.9%	0.33 [0.01, 7.58]
Collaborative 2005	5	100	8	91	10.7%	0.57 [0.19, 1.68]
Dikensoy 2002	1	17	2	17	2.6%	0.50 [0.05, 5.01]
Khilnani 2010	3	20	2	20	2.6%	1.50 [0.28, 8.04]
Liu 2005	1	18	3	18	3.8%	0.33 [0.04, 2.91]
Matuska 2006	7	30	7	30	9.0%	1.00 [0.40, 2.50]
Plant 2001	12	118	24	118	30.8%	0.50 [0.26, 0.95]
Samaria 2009	4	20	8	20	10.3%	0.50 [0.18, 1.40]
Thys 2002	2	10	1	10	1.3%	2.00 [0.21, 18.69]
Total (95% CI)		**434**		**420**	**100.0%**	**0.54 [0.38, 0.76]**
Total events	42		77			

Heterogeneity: $Chi^2 = 6.36$, df = 10 (P = 0.78); $I^2 = 0\%$
Test for overall effect: Z = 3.49 (P = 0.0005)



Ventilation mécanique invasive

INDICATIONS



Ventilation mécanique invasive

INTUBATION

- **Pré-oxygénation à fort débit d'O2**
- **Séquence rapide:**

  - Hypnotique: Kétamine

  - Curare: célocurine

- **Sonde d'intubation de bon calibre :** diminuer les résistances, drainage des secrétions

Ventilation mécanique invasive

Hyperinflation dynamique

Privilégier la vidange pulmonaire → EXPIRATION

- **Mode VAC**
- **VT:** 6 à 7 ml/kg
- **FR:** 12 à 14 cycles /min
- **FiO2 :** objectif SpO2 90-92%
- **I/E:** 1/3- 1/4
- **Débit inspiratoire:** 60 l/min
- **ZEEP**
- **Humidificateur chauffant**

Traitement pharmacologique

OBJECTIFS

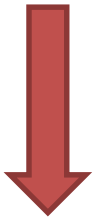
- **Diminuer l'obstruction**
- **Traiter l'inflammation**
- **Traiter le facteur déclenchant**

Traitement pharmacologique

Bronchodilatateur

Bronchodilatateurs de courte durée d'action

Diminuent la charge en diminuant les résistances bronchiques



Amélioration du débit expiratoire et de la surdistension

Traitement pharmacologique

Bronchodilatateur

Bronchodilatateurs de courte durée d'action

- Nébulisation sous air
- Béta 2 agonistes en première intension (salbutamol ou terbutaline 5 mg)

Traitement pharmacologique

Bronchodilatateur

Association d'un anticholinergique (ipratromium 0,5 mg)

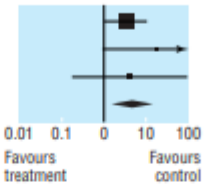
Although there is no high-quality evidence from RCTs, it is recommended that short-acting inhaled beta$_2$-agonists, with or without short-acting anticholinergics, are the initial bronchodilators for acute treatment of a COPD exacerbation.[56,57] A systematic review of the route of delivery of short-acting bronchodilators found no significant

Traitement pharmacologique

Panel A
Comparison: Adverse effects
Outcome: Effect of methylxanthines on nausea/vomiting

Study	Treatment n/N	Control n/N	Odds ratio (95% CI fixed)	Weight %	Odds ratio (95% CI fixed)
Ram 2000	16/27	7/23		76.6	3.32 (1.03 to 10.75)
Rice 1987	6/15	0/13		8.1	18.47 (0.93 to 368.78)
Wrenn 1991	2/23	0/16		13.4	3.84 (0.17 to 85.47)
Total (95% CI)	24/65	7/52		100.0	4.62 (1.70 to 12.56)

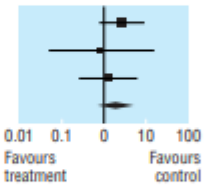
Test for heterogeneity: χ^2=1.14, df=2, P=0.57
Test for overall effect: z=3.00, P=0.003



Panel B
Comparison: Adverse effects
Outcome: Effect of methylxanthines on tremor

Study	Treatment n/N	Control n/N	Odds ratio (95% CI fixed)	Weight %	Odds ratio (95% CI fixed)
Ram 2000	21/27	23/23		45.3	2.69 (0.79 to 9.17)
Rice 1987	1/15	1/13		14.5	0.88 (0.05 to 15.23)
Wrenn 1991	5/23	3/16		40.2	1.20 (0.24 to 5.96)
Total (95% CI)	27/65	17/52		100.0	1.83 (0.73 to 4.56)

Test for heterogeneity: χ^2=0.91, df=2, P=0.63
Test for overall effect: z=1.29, P=0.2



Panel C
Comparison: Adverse effects
Outcome: Effect of methylxanthines on palpitations/arrhythmias

Study	Treatment n/N	Control n/N	Odds ratio (95% CI fixed)	Weight %	Odds ratio (95% CI fixed)
Ram 2000	8/27	1/23		40.2	9.26 (1.08 to 80.94)
Wrenn 1991	1/23	1/16		59.8	0.68 (0.04 to 11.77)
Total (95% CI)	9/50	2/38		100.0	4.14 (0.87 to 19.61)

Test for heterogeneity: χ^2=2.07, df=1, P=0.15
Test for overall effect: z=1.79, P=0.07

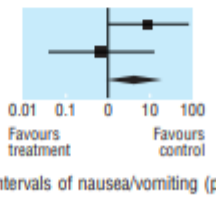


Fig 4 Odds ratio and 95% confidence intervals of nausea/vomiting (panel A), tremor (panel B), and palpitations/arrhythmias (panel C) for methylxanthines compared with placebo use

Comparison: Effect of methylxanthines on symptom scores
Outcome: Change in symptoms core over 3 days

Study	Treatment n	Treatment Mean (sd)	Control n	Control Mean (sd)	Standardised mean difference (95% CI fixed)	Weight %	Standardised mean difference (95% CI fixed)
Ram 2000	27	7.69 (20.04)	23	-2.89 (20.54)		51.0	0.52 (-0.04 to 1.09)
Rice 1987	15	2.70 (0.88)	13	5.70 (0.66)		49.0	-3.31 (-4.50 to -2.12)
Total (95% CI)	42		36			100.0	-1.36 (-5.11 to 2.40)

Test for heterogeneity: χ^2=32.31, df=1, P=0.001
Test for overall effect: z=0.71, P=0.5

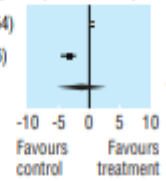


Fig 3 Standardised mean difference in symptom score (0-100) at three days and 95% confidence intervals between methylxanthines compared with placebo use

lnalysis of randomised trials

nargo Jr

Traitement pharmacologique

Bronchodilatateur

METHYLXANTHINE

exacerbation or to start these medications as soon as possible before hospital discharge. Intravenous methylxanthines (theophylline or aminophylline) are not recommended to use in these patients due to significant side effects.[60,61] If a

Traitement pharmacologique

Characteristics	Oral corticosteroids (n=28)	Placebo (n=22)

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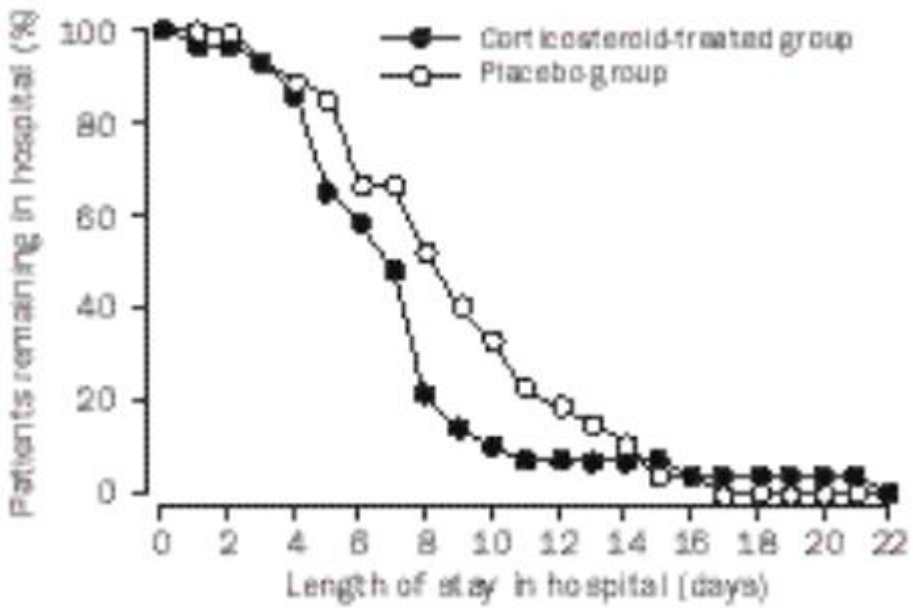
a prospecti…

L Davies, R M Angus, …



SGRQ=St George's respiratory questionnaire. Values are means (SE) or numbers (%).

Table 2: **Demography of patients on admission**

Traitement pharmacologique

JAMA Network

JAMA Internal Medicine

GLUCOCORTICOIDES

Nov 28, 2011

Efficacy of Corticosteroid Therapy in Patients With an Acute Exacerbation of Chronic Obstructive Pulmonary Disease Receiving Ventilatory Support

Conclusion Systemic corticosteroid therapy in patients with COPD exacerbations requiring mechanical ventilation is associated with a significant increase in the success of noninvasive mechanical ventilation and a reduction in the duration of mechanical ventilation

Traitement pharmacologique

GLUCOCORTICOIDES

- Diminue la durée de récupération
- Améliore la fonction pulmonaire
- Améliore l'oxygénation
- Diminue la durée d'hospitalisation

Traitement pharmacologique

GLUCOCORTICOIDES

- 40 mg de prednisone par jour pendant 5 jours
- Voie orale équivalente à la voie intraveineuse

CHEST

Volume 132, Issue 6, December 2007, Pages 1741-1747

Original Research
COPD

Oral or IV Prednisolone in the Treatment of COPD Exacerbations

Ynze P. de Jong MD [a], Steven M. Uil MSc [a], Hans P. Grotjohan MD, PhD [a], Dirkje S. Postma MD, PhD [b], Huib A.M. Kerstjens MD, PhD [b],

Conclusion

Therapy with oral prednisolone is not inferior to IV treatment in the first 90 days after starting therapy. We suggest that the oral route is preferable in the treatment of COPD exacerbations.

Traitement pharmacologique

Results: Symptoms, pulmonary function and arterial blood gas analysis were significantly improved after treatment in both groups ($P < 0.05$), with no significant differences between them ($P > 0.05$), while incidence of adverse events in the budesonide group was lower ($P < 0.05$). No significant differences in CAT score, days of admission, blood gas analysis results and physiological and biochemical indexes were found between the two groups. Patients treated with methylprednisolone showed a higher degree of PaO_2 level improvement.

- [

Conclusion: Results show that inhalation of budesonide (2 mg 3 times/day) and systemic methylprednisolone (40 mg/day) had similar clinical outcome in AECOPD. In conclusion, inhaled budesonide is an alternative to systemic corticosteroids in AECOPD treatment.

Traitement pharmacologique

Des études suggèrent que les glucocorticoïdes sont plus efficaces chez les patients ayant une

American Journal of Respiratory and Critical Care Medi

Home > American Journal of Respiratory and Critical Care Medicine > List of Issues > Volume 186, Issue 1

Blood Eosinophils to Direct Corticosteroid Treatment of Exacerbations of Chronic Obstructive Pulmonary Disease

A Randomized Placebo-Controlled Trial

Mona Bafadhel [1], Susan McKenna [1], Sarah Terry [1], Vijay Mistry [1], Mitesh Pancholi [1], Per Venge [2], David A. Lomas [3], Michael R. Barer [1], Sebastian L. Johnston [4], Ian D. Pavord [1], and Christopher E. Brightling [1]

+ Author Affiliations

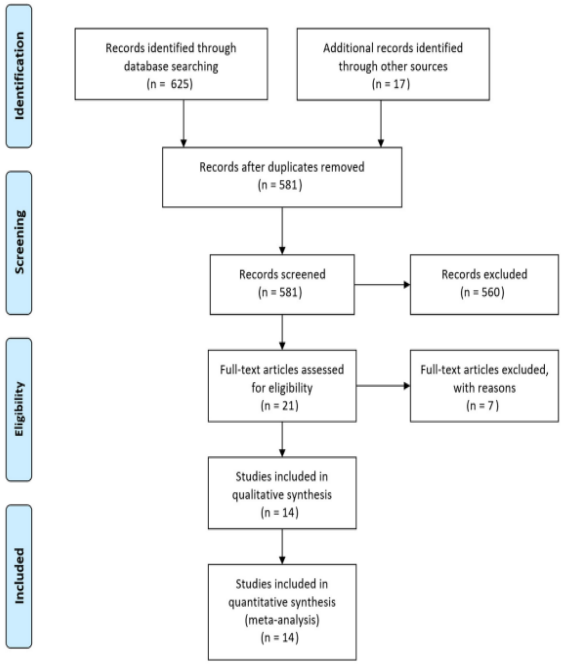
Traitement pharmacologique

Antibiothérapie

- ✓ Pas systématique
- ✓ Origine infectieuse
- ✓ Utilisation PCT controversée

Traitement pharmacologique

Antibiothérapie



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exclusively in the hospital setting. PCT was found to decrease overall antibiotic exposure in COPD exacerbations by 2.01 days ($p = 0.04$), while no apparent effects were found on clinical outcomes (length of hospital stay, $p = 0.88$; treatment failure $p = 0.51$; all-cause mortality $p = 0.28$). However, the majority of blood PCT

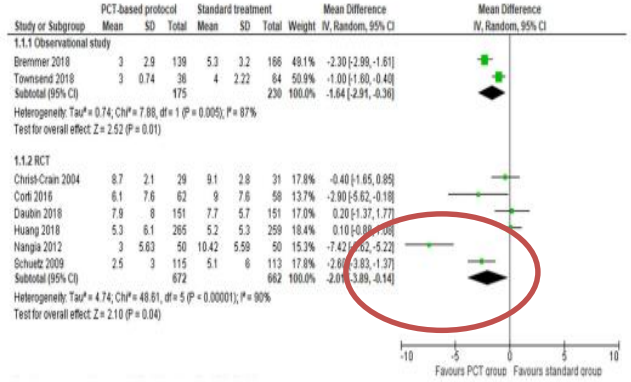

Fig. 3 Forest plot of the effects of PCT-based protocol versus standard treatment on length of antibiotic treatment

Traitement pharmacologique

SEVEN-DAY PROFILE PUBLICATION

Procalcitonin algorithm to guide initial antibiotic therapy in acute exacerbations of COPD admitted to the ICU: a randomized multicenter study

Cédric Daubin[1*], Xavier Valette[1], Fabrice Thiollière[2], Jean-Paul Mira[3], Pascal Hazera[4], Djillali Annane[5,6], Vincent Labbe[7], Bernard Floccard[8], François Fournel[9], Nicolas Terzi[10,11], Damien Du Cheyron[1] and Jean-Jacques Parienti[9,12] for the BPCTrea Study Group

Abstract

Purpose: To compare the efficacy of an antibiotic protocol guided by serum procalcitonin (PCT) with that of standard antibiotic therapy in severe acute exacerbations of COPD (AECOPDs) admitted to the intensive care unit (ICU).

Methods: We conducted a multicenter, randomized trial in France. Patients experiencing severe AECOPDs were assigned to groups whose antibiotic therapy was guided by (1) a 5-day PCT algorithm with predefined cutoff values for the initiation or stoppage of antibiotics (PCT group) or (2) standard guidelines (control group). The primary endpoint was 3-month mortality. The predefined noninferiority margin was 12%.

Results: A total of 302 patients were randomized into the PCT ($n = 151$) and control ($n = 151$) groups. Thirty patients (20%) in the PCT group and 21 patients (14%) in the control group died within 3 months of admission (adjusted difference, 6.6%; 90% CI −0.3 to 13.5%). Among patients without antibiotic therapy at baseline ($n = 119$), the use of PCT significantly increased 3-month mortality [19/61 (31%) vs. 7/58 (12%), $p = 0.015$]. The in-ICU and in-hospital antibiotic exposure durations, were similar between the PCT and control group (5.2 ± 6.5 days in the PCT group vs. 5.4 ± 4.4 days in the control group, $p = 0.85$ and 7.9 ± 8 days in the PCT group vs. 7.7 ± 5.7 days in the control group, $p = 0.75$, respectively).

Conclusion: The PCT group failed to demonstrate non-inferiority with respect to 3-month mortality and failed to reduce in-ICU and in-hospital antibiotic exposure in AECOPDs admitted to the ICU.

Keywords: Chronic obstructive pulmonary disease, Procalcitonin, Antibiotic stewardship, Respiratory tract infection, Community-acquired pneumonia, Viral infection

Mesures générales

- ✓ Réhydratation
- ✓ Correction des désordres hydro-électrolytiques éventuellement d'une hypokaliémie, hypophosphorémie, une hypomagesémie pouvant aggraver la dysfonction du diaphragme.
- ✓ Alimentation précoce
- ✓ Réduction de l'apport en hydrate de carbones pour diminuer la production endogène de CO_2.
- ✓ Arrêt du tabac

Traitement du facteur déclenchant

- ✓ Antibiothérapie
- ✓ TTT anticoagulant
- ✓ Drainage d’un pneumothorax
- ✓ Arrêt d’un traitement médicamenteux déclenchant

Traitements adjuvants

➔Kinésithérapie

➔ Prévention de la maladie thromboembolique

➔Évaluation de l'état nutritionnel

Prise en charge de l'exacerbation sévère de BPCO

-Evaluation des signes clinique de gravite
-GDS
-Radiographie du thorax
-Oxygénothérapie
-Bronchodilatateurs : beta 2 mimétique/ anticholinergique
-Corticothérapie
-Antibiothérapie si une infection bactérienne est suspectée
-VNI 1ere intension
-VM
-Monitorage du bilan hydrique
-Anticoagulation préventive
-Traitement du facteur déclenchant