Quoi de neuf en 2003?

Morceaux choisis

- Non exhaustif, mais uniquement 2003
- Utile pour la pratique courante
- Articles de revues, articles originaux, pas expérimentaux
- Les domaines suivants:
  - Syndrome coronarien aigu
  - Ventilation: VNI, Ventilation mécanique
  - Prévention de l'insuffisance rénale
  - Prophylaxie des ulcères de stress
  - Investigation hémodynamique: invasive, non-invasive, monitoring du remplissage
  - Infections: VAP, choc septique (corticoïdes, Protéine C)
  - Endocrino: insuffisance surrénalienne, insuline
  - Nutrition
  - Sédation
  - Embolie pulmonaire
  - RCP…….

Prise en charge de l'infarctus aigu du myocarde

- PRAGUE II (*Eur Heart J*): Long distance transport for primary angioplasty vs immediate thrombolysis in acute myocardial infarction
- CAPTIM (*Circulation*): Impact of time to treatment on mortality after prehospital fibrinolysis or primary angioplasty
- DANAMI II (*N Eng J Med*): A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction
- ASSENT 3 plus (*Circulation*)
- ….
- Méta-analyse *Lancet 2003*: PTCA vs Thrombolyse
- Meta-analyse *Circulation 2003*: Transfert pour PTCA vs Thrombolyse immédiate

Articles

Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials

Ellen C Keeley, Judith A Boura, Cindy L Grines

Summary

Background Many trials have been done to compare primary percutaneous transluminal coronary angioplasty (PTCA) with thrombolytic therapy for acute ST-segment elevation myocardial infarction (AMI). Our aim was to look at the combined results of these trials and to ascertain which reperfusion therapy is most effective.

Methods We did a search of published work and identified 23 trials, which together randomly assigned 7739 thrombolytic-eligible patients with ST-segment elevation AMI to primary PTCA (n=3872) or thrombolytic therapy (n=3867). Streptokinase was used in eight trials (n=1837), and fibrin-specific agents in 15 (n=5902). Most patients who received thrombolytic therapy (76%, n=2939) received a fibrin-specific agent. Stents were used in 12 trials, and platelet glycoprotein IIb/IIIa inhibitors were used in eight. We identified short-term and long-term clinical outcomes of death, non-fatal reinfarction, and stroke, and did subgroup analyses to assess the effect of type of thrombolytic agent used and the strategy of emergent hospital transfer for primary PTCA. All analyses were done with and without inclusion of the SHOCK trial data.

Findings Primary PTCA was better than thrombolytic therapy at reducing overall short-term death (7% [n=270] vs 9% [360]; p=0·0002), death excluding the SHOCK trial data (5% [199] vs 7% [276]; p=0·0003), non-fatal reinfarction (3% [80] vs 7% [222]; p<0·0001), stroke (1% [30] vs 2% [64]; p=0·0004), and the combined endpoint of death, non-fatal reinfarction, and stroke (8% [253] vs 14% [442]; p<0·0001). The results seen with primary PTCA remained better than those seen with thrombolytic therapy during long-term follow-up, and were independent of both the type of thrombolytic agent used, and whether or not the patient was transferred for primary PTCA.

Interpretation Primary PTCA is more effective than thrombolytic therapy for the treatment of ST-segment elevation AMI.

Lancet 2003; **361**: 13–20

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Introduction

In the mid-1970s, acute myocardial infarction (AMI) was identified, in nearly all cases, as being the result of a ruptured atherosclerotic plaque, causing thrombosis and occlusion of the coronary artery.[1] Subsequently, the reperfusion era was ushered in with the realisation that an occlusive thrombus in a coronary artery could be managed by use of a combination of a guidewire to mechanically initiate coronary blood flow and the intracoronary infusion of streptokinase.[2] The recognition that the prompt restoration of flow salvages myocardium, reduces infarct size, and prolongs life has been the driving force behind a large number of clinical trials, assessing thrombolytic therapy for AMI. The results of these trials, done in the early 1980s and involving tens of thousands of patients, unequivocally showed that thrombolytic therapy resulted in preserved left-ventricular function and decreased mortality in patients with AMI.[3]

Primary percutaneous transluminal coronary angioplasty (PTCA), defined as balloon angioplasty undertaken as the primary reperfusion strategy for AMI without previous or concomitant thrombolytic therapy, was initially compared with intracoronary thrombolytic therapy.[4] Over the next decade, ten trials, comparing primary PTCA with intravenous thrombolytic therapy for ST-segment elevation AMI were undertaken. In 1993[5] and in 1997,[6] systematic reviews of this topic were published, with the later analysis of 2606 patients, showing improved short-term clinical outcomes, including death, with primary PTCA compared with thrombolytic therapy. Since this quantitative review, however, 13 new trials, comparing these two reperfusion modalities, have been done, more than doubling the number of randomised trials, and tripling the number of patients studied. Moreover, long-term clinical outcomes are now available for many of these trials. Our aim was, therefore, to provide an updated quantitative analysis of the results of the randomised trials of primary PTCA versus thrombolytic therapy.

Methods

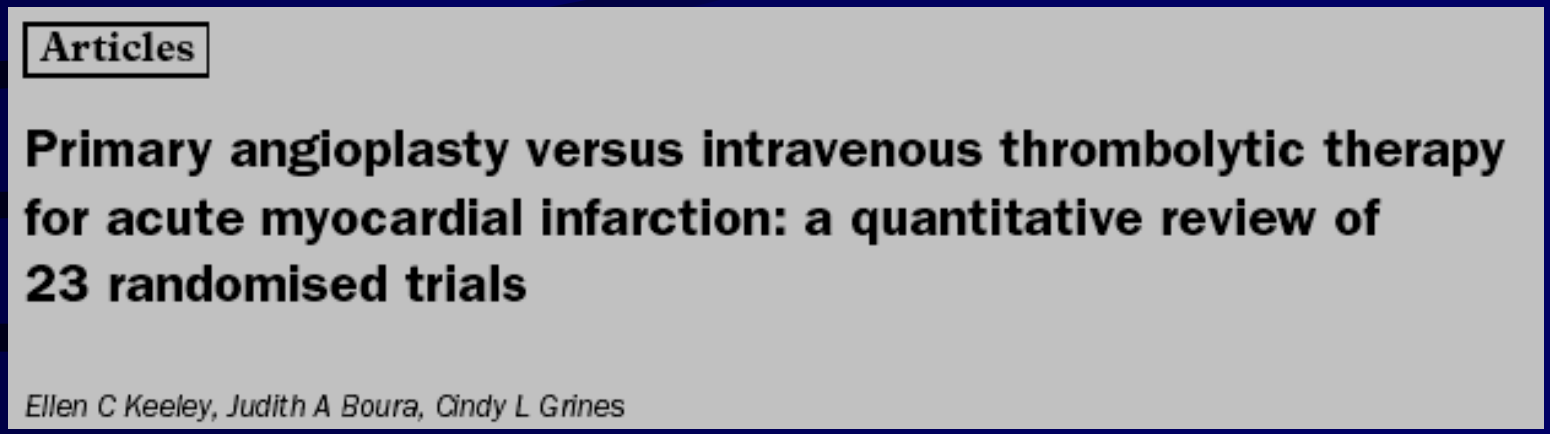
Protocol

We identified all randomised trials done to date, published and unpublished, comparing primary PTCA with thrombolytic therapy for the treatment of acute ST-segment AMI, by searching the Medline database. We also searched scientific sessions abstracts in the *New England Journal of Medicine*, *Journal of the American College of Cardiology*, *Circulation*, *European Heart Journal*, *Heart*, and *Clinical Cardiology*, and questioned the principal investigators of most trials to ensure validity of the data, obtain additional unpublished data, and to verify that the study was randomised in design.

Our primary aim was to ascertain which reperfusion therapy, intravenous thrombolytic therapy or primary PTCA, is most effective for treatment of patients with

- 70's: infarctus résulte de la rupture d'une plaque d'athérosclérose avec thrombose et occlusion coronaire (*Br Heart J 1976*)
- Reperfusion par perfusion intra coronaire de SK ➔ diminue la taille de l'infarctus et prolonge la vie (*Clin Cardiol 1979*)
- 80's: grands essais sur la fibrinolyse incluant des dizaines de milliers de patients: la fibrinolyse diminue la mortalité et préserve la fonction ventriculaire gauche
- Primary percutaneous transluminal coronary angioplasty (PTCA) fut d'abord comparée à la fibrinolyse intra coronaire (N Eng J Med 1986)
- 90's: 10 essais PTCA vs fibrinolyse : amélioration clinique et décès à court terme (2606 patients, Circulation 1995, JAMA 1997).

- Depuis lors, 13 nouvelles études ont été publiées sur ce sujet
- Intérêt cette nouvelle méta-analyse:
  - 2x plus d'études, 3x plus de patients que la précédente
  - données disponibles sur effets cliniques à long terme
  - 9/13 études utilisent des agents fibrin-specific
  - amélioration de la technique PTCA (12/13 utilisent des stents, 8/13 des inhibiteurs G IIb/IIIa)


Articles

Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials

Ellen C Keeley, Judith A Boura, Cindy L Grines

- Comparaison PTCA primaire vs fibrinolysis pour infarctus du myocarde avec sus decalage du segment ST

- Mortalité, réinfarcissement, récidive d'ischémie, stroke, stroke hémorragique, hémorragie
- Court terme (4-6 sem), long terme (6-18 mois)
- 23 études, 7739 patients

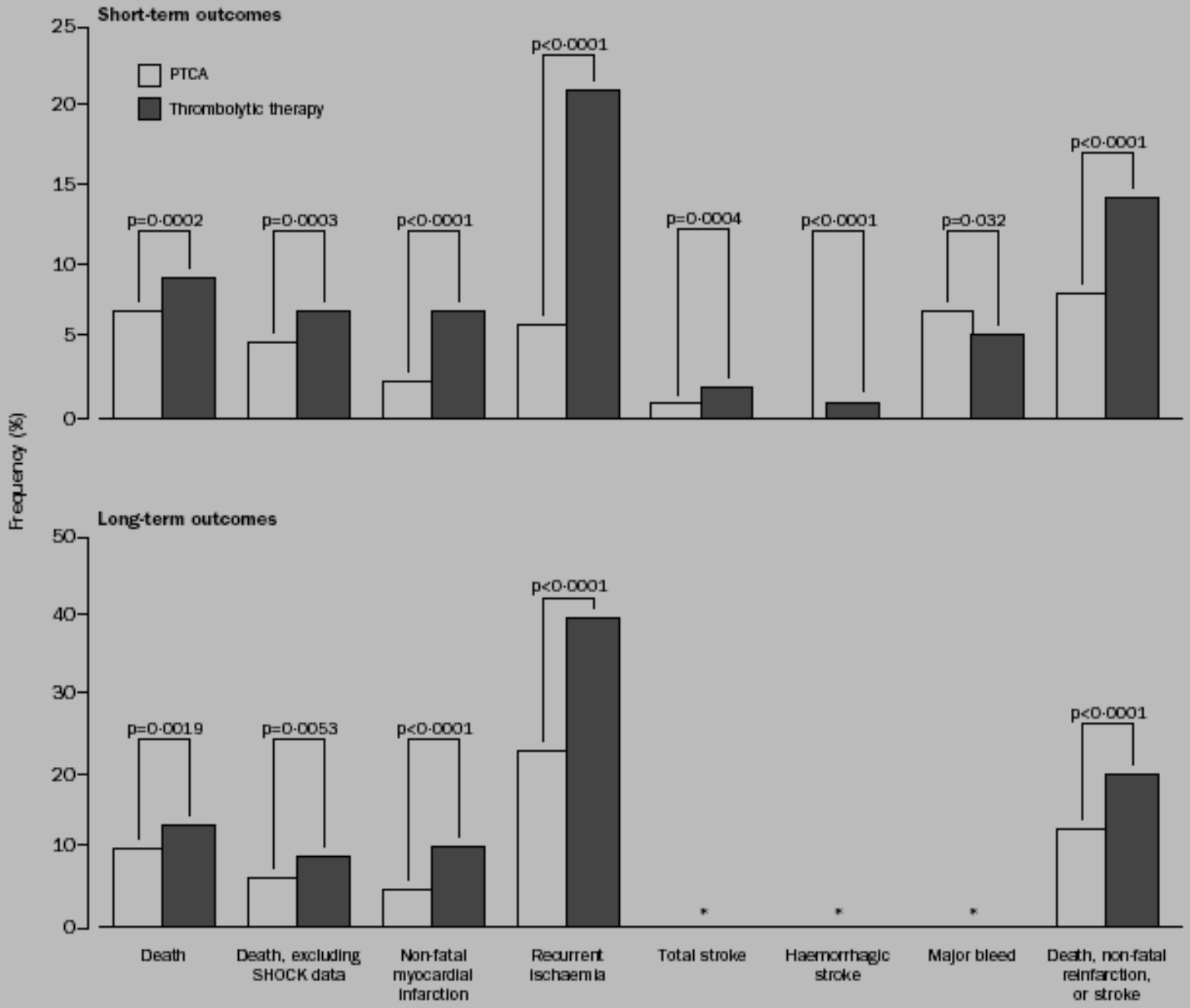


Figure 2: **Short-term[7–21] and long-term[7–9,11,12,14,17,18,20,22–24,26,27,29,30,33–38] clinical outcomes in individuals treated with primary PTCA or thrombolytic therapy**

Comparaison fibrinolyse sur place et transfert pour PTCA (5 études)

- Délai moyen de transfert 39 min
- 0.5% décès pdt le transfert, 0.7 à 1.4% risque d'arythmie ventriculaire, 2% de risque de BAV 2e ou 3e degré

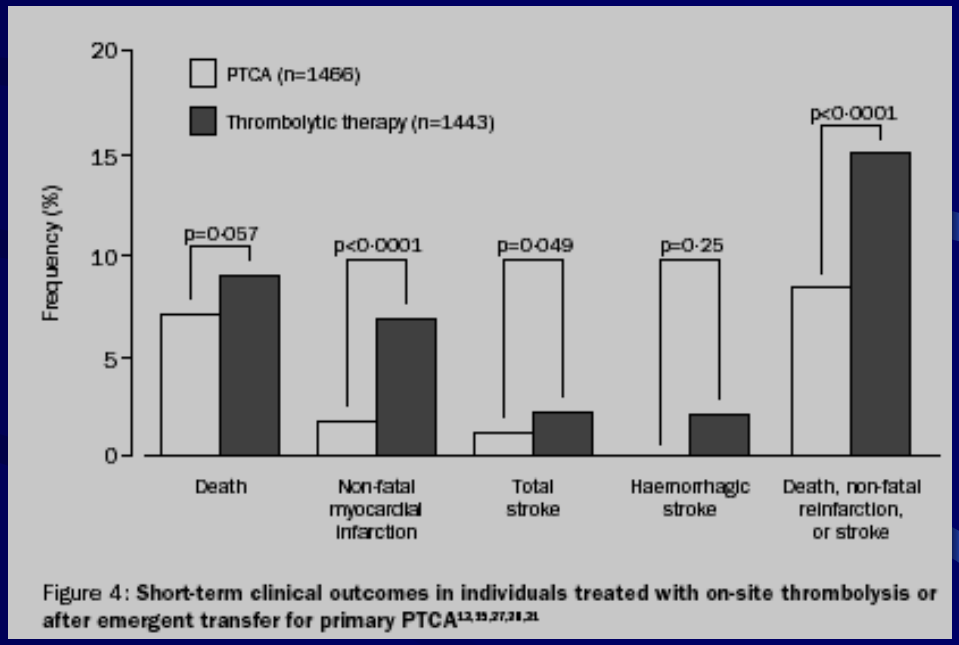


Figure 4: **Short-term clinical outcomes in individuals treated with on-site thrombolysis or after emergent transfer for primary PTCA**[12,15,27,28,31]

Conclusions

- PTCA meilleure que fibrinolyse : réduit les événements cardiaques majeurs y compris le décès et la récidive. Ces effets sont conservés à long terme.
- Ces effets sont indépendants du type de thrombolytique utilisé
- Ces effets sont conservés en cas de transfert pour PTCA (! Timing)

- PTCA technique de reperfusion de choix
- Les études sont réalisées dans des centres disposant des deux techniques (et souvent de grande taille): biais?
- Dans la réalité la majorité des patients se présentent dans des centres qui ne disposent pas des deux techniques: que faire? Thrombolyser ou transférer pour PTCA?

Transfer for Primary Angioplasty Versus Immediate Thrombolysis in Acute Myocardial Infarction

A Meta-Analysis

M. Dalby, MD; A. Bouzamondo, MD; P. Lechat, MD; G. Montalescot, MD, PhD

Summary of Characteristics in Trials Comparing Immediate Thrombolysis to Transfer for Primary Angioplasty

Study	Inclusion Criteria	No. PCI	Time to PCI (From Randomization)	No. Lytic	Time to Lytic (From Randomization)	Lytic Agent	Trial Weight, %	Primary End Point
MAASTRICHT*	Pain >30 min and <6 h ST elevation	75	85	75	10	tPA	6	Test safety and feasibility of acute transfer for PCI
PRAGUE-1†	Pain <6 h ST elevation or new LBBB	101	80	99	10	SK	7	Death, re-MI, or stroke (30 days)
AIR-PAMI‡	Pain <12 h with ST elevation/new LBBB, plus 1 or more high-risk criteria‡	71	122	66	19	By center. If tPA, bolus plus 72 h heparin infusion	4	Death, re-MI, or disabling stroke (30 days)
CAPTIM§	Pain >30 min and <6 h ST elevation/new LBBB	421	82	419	23	tPA	15	Death, re-MI or stroke (30 days)
DANAMI-2‖	Pain <12 h with ST elevation	790	NA	782	NA	tPA	43	Death, re-MI, or disabling stroke (30 days)
PRAGUE-2¶	AMI <12 h	429	97	421	17	SK	25	Mortality (30 days)

LBBB indicates left bundle-branch block; MI, myocardial infarction.

*Prospective, randomized comparison between thrombolysis, rescue percutaneous transluminal coronary angioplasty (PTCA) and primary PTCA in patients with extensive myocardial infarction admitted to a hospital without PTCA facilities: a safety and feasibility study.[6]

†Multicenter randomized trial comparing transport to primary angioplasty versus immediate thrombolysis versus combined strategy for patients with acute myocardial infarction presenting to a community hospital without a catheterization laboratory. The PRAGUE Study.[3]

‡The Air Primary Angioplasty in Myocardial Infarction study.[7] In Air-PAMI, high-risk criteria were age >70, HR >100/min, SBP <100 mm Hg, Killip class II/III, ECG LBBB, or anterior MI.

§Comparison of primary angioplasty and prehospital thrombolysis in the acute phase of myocardial infarction.[8]

‖The Danish multicenter randomized study on thrombolytic therapy vs acute coronary angioplasty in acute myocardial infarction. (DANAMI-2).[4]

¶PRAGUE-2. Long-distance transport for primary angioplasty vs immediate thrombolysis in acute myocardial infarction.[5]

Effets sur la récidive et le stroke

- Récidive infarctus: RR 0.32 (p < 0.001): NNT 33

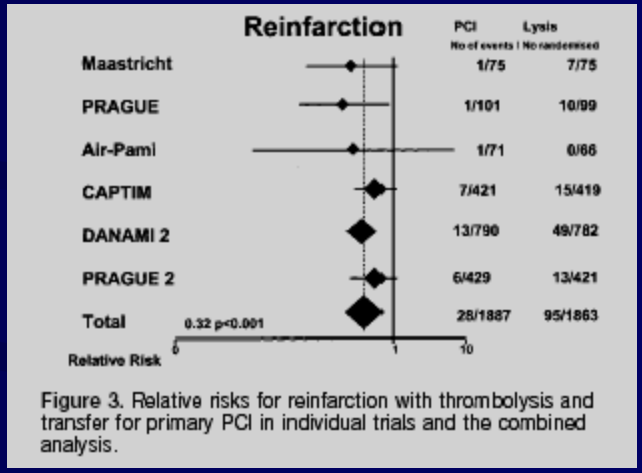


Figure 3. Relative risks for reinfarction with thrombolysis and transfer for primary PCI in individual trials and the combined analysis.

- Stroke: 0.44 (p = 0.015); NNT 86

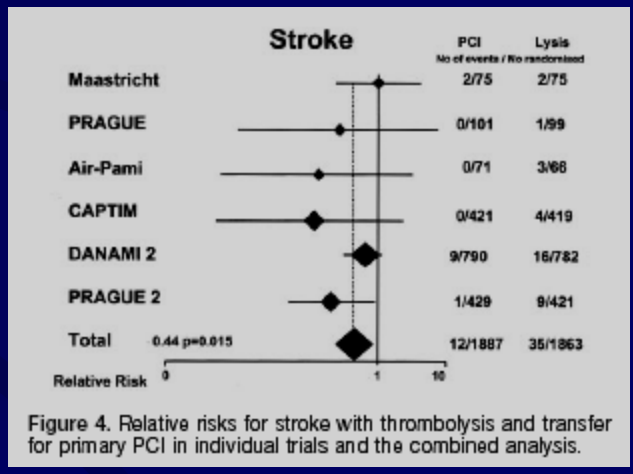


Figure 4. Relative risks for stroke with thrombolysis and transfer for primary PCI in individual trials and the combined analysis.

Effet isolé sur le décès

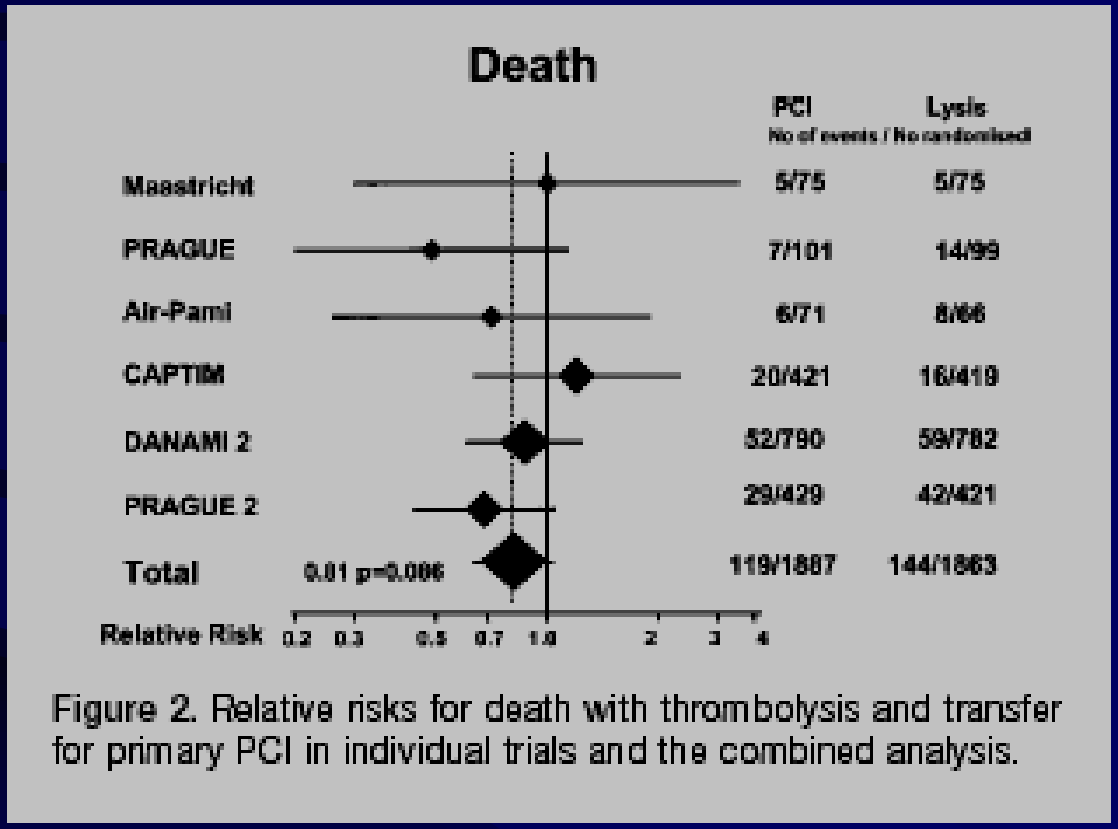


Figure 2. Relative risks for death with thrombolysis and transfer for primary PCI in individual trials and the combined analysis.

!!Sans resultats CAPTIM: RR 0.76 p = 0.035

Effets combinés mort/réinfarctus/stroke

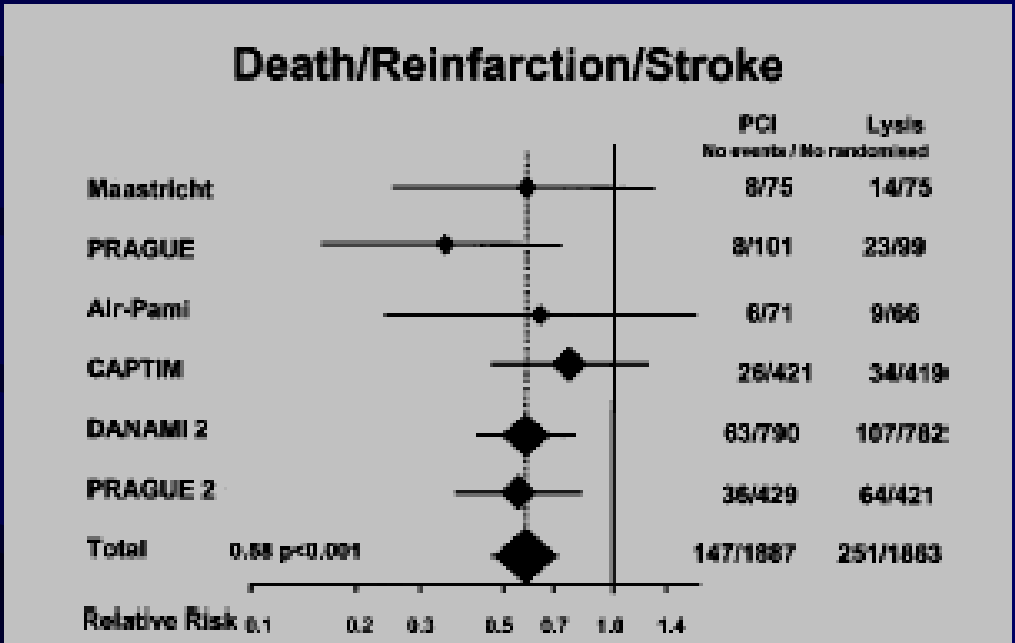
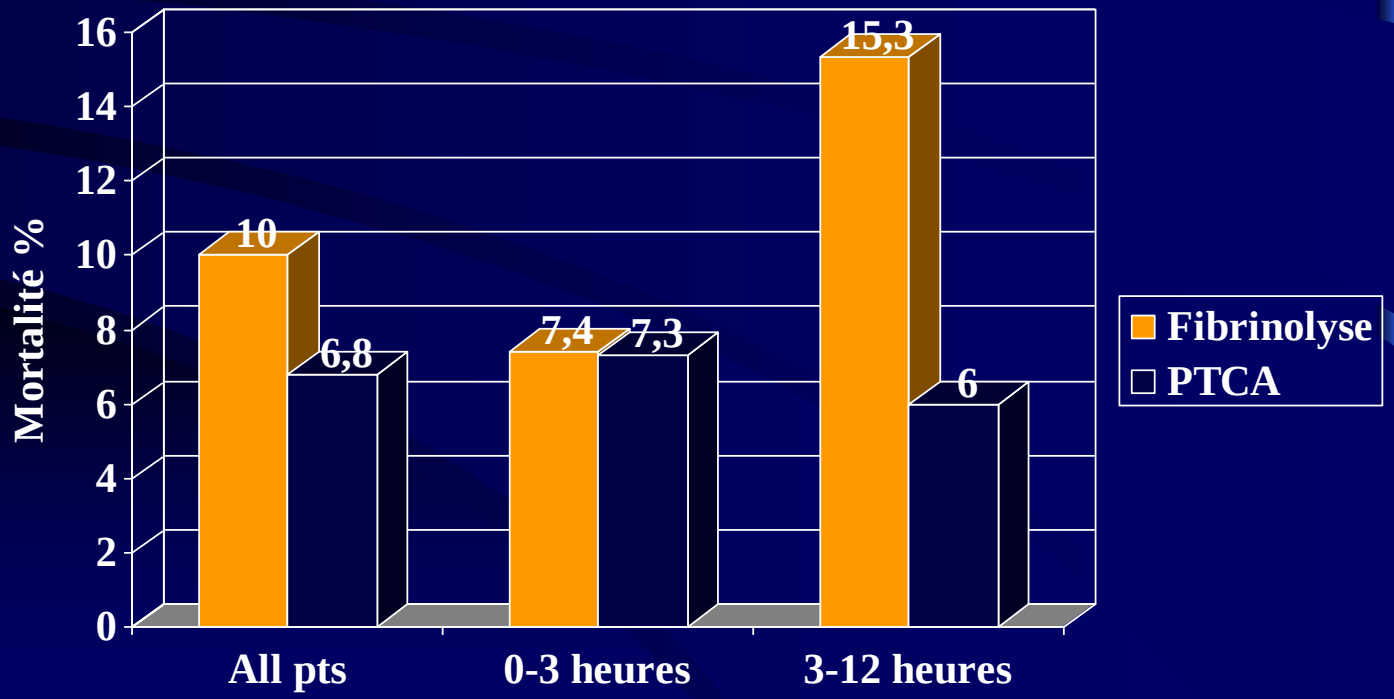


Figure 1. Relative risks for the composite of death/reinfarction/stroke with thrombolysis and transfer for primary PCI in individual trials and the combined analysis.

30 days NNT: 19 patients

Conclusions

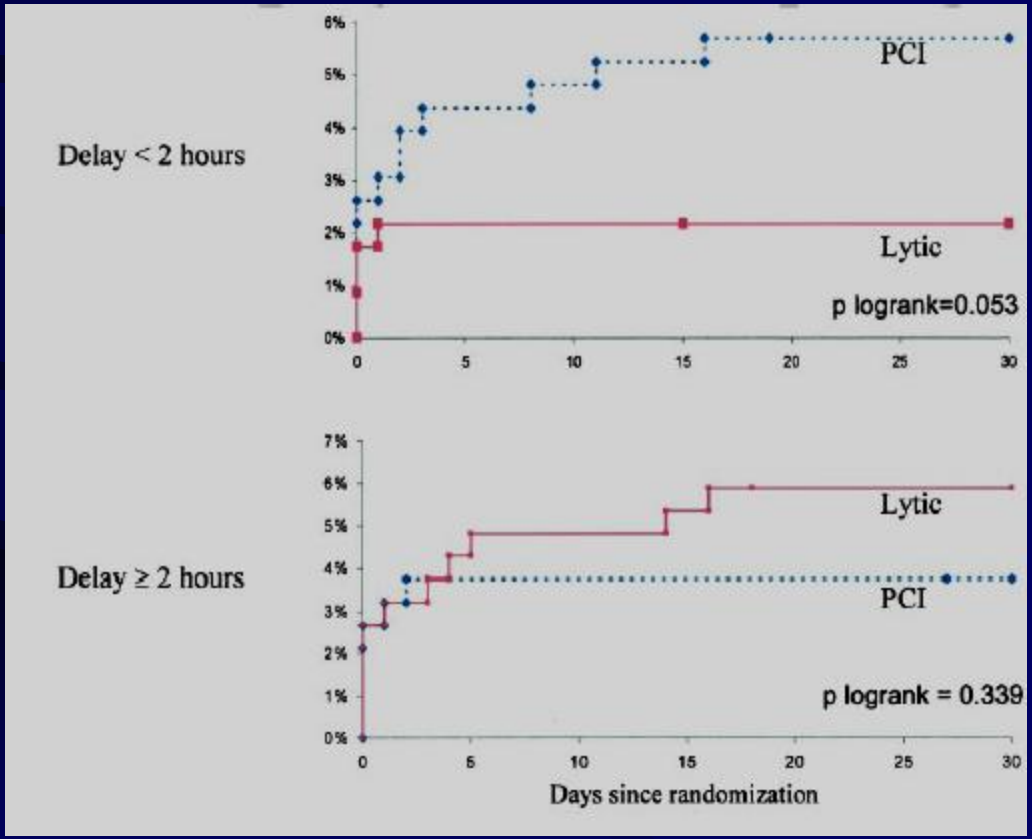
- Bien que le transfert accroisse clairement le délai de reperfusion, le bénéfice reste présent:
  - Additionnal time to treat 70 à 103 minutes
- Différence entre fibrinolyse et PTCA dépend aussi du délai de début des symptômes:
  - PRAGUE-2:



Impact of Time to Treatment on Mortality After Prehospital Fibrinolysis or Primary Angioplasty

Data From the CAPTIM Randomized Clinical Trial

Philippe Gabriel Steg, MD; Eric Bonnefoy, MD; Sylvie Chabaud, MSc; Frédéric Lapostolle, MD; Pierre-Yves Dubien, MD; Pascal Cristofini, MD; Alain Leizorovicz, MD; Paul Touboul, MD; for the Comparison of Angioplasty and Prehospital Thrombolysis In acute Myocardial infarction (CAPTIM) Investigators*



(*Circulation*. 2003;108:2851-2856.)

EDITORIALS

Primary Angioplasty for Acute Myocardial Infarction — Is It Worth the Wait?

Alice K. Jacobs, M.D.

Nearly two decades after clinical trials established that fibrinolytic therapy for acute myocardial infarction preserves left ventricular function and reduces mortality, there is evidence that mechanical reperfusion therapy is superior in reducing the rates of death, reinfarction, intracranial bleeding, reocclusion of the infarct-related artery, and recurrent ischemia. Initially introduced as an alternative to fibrinolytic therapy (to circumvent contraindications to its use and the risk of intracranial bleeding), primary percutaneous coronary intervention is now increasingly recognized as the reperfusion therapy of choice. The ability to restore robust coronary flow promptly in more than 90 percent of patients and the nearly linear relation between patency of the infarct-related artery at 90 minutes after the initiation of reperfusion therapy and in-hospital mortality rates lend credibility to the momentum behind primary percutaneous coronary intervention for patients with myocardial infarction associated with ST-segment elevation. In fact, a quantitative review[1] of 23 randomized trials in which primary percutaneous coronary intervention was compared with fibrinolytic therapy revealed that the former was superior in reducing the short-term rates of death (7 percent, vs. 9 percent with fibrinolytic therapy; P<0.001), nonfatal reinfarction (3 percent vs. 7 percent; P<0.0001), stroke (1 percent vs. 2 percent; P=0.0004), and the combined end point of death, nonfatal reinfarction, and stroke (8 percent vs. 14 percent; P<0.001).

Nevertheless, fibrinolytic therapy remains the mainstay of reperfusion treatment around the globe because it is more widely available than coronary angioplasty. Even in the United States, the majority of hospitals do not have angioplasty capabilities, and in many that do, nearly 50 percent of the patients with myocardial infarction associated with ST-segment elevation are treated with fibrinolytic agents. The widespread unavailability of primary percutaneous coronary intervention appears to negate the superiority of this strategy as compared with fibrinolysis. It also raises the obvious question of whether primary percutaneous coronary intervention performed after a patient is transferred to a facility where it is available will still be superior to fibrinolytic therapy administered at the referral hospital. Given the inherent delay before transfer and the risks associated with transportation during acute myocardial infarction, the answer is not intuitive.

Five randomized trials have attempted to address this question. The Danish Multicenter Randomized Study on Fibrinolytic Therapy versus Acute Coronary Angioplasty in Acute Myocardial Infarction (DANAMI-2), reported in this issue of the Journal,[2] is noteworthy for its randomized design, its practical approach to a critical question, and its careful consideration of the time between the onset of symptoms (in addition to arrival at the hospital) and reperfusion in its comparisons of strategies and treatment centers. Among patients at referral hospitals who were randomly assigned to be transferred to another center for primary angioplasty or to receive fibrinolytic therapy on site, the primary end point (a composite of death, reinfarction, or disabling stroke at 30 days) was reached in 8.5 percent of the patients in the former group, as compared with 14.2 percent of those in the fibrinolytic-therapy group (P=0.002), and the difference was driven by a reduction in the rate of reinfarction in the angio-

Impact sur la stratégie de reperfusion

- Si douleur depuis moins de 2-3h:
  - **Fibrinolyse** si Door to needle time -> 30 minutes
  - **PTCA** si: fibrinolyse CI ou échec, choc cardiogénique, transfert < 60 min
- Si douleurs > 3h:
  - Transfert pour **PTCA**
- Si difference entre *door-to-needle* et *door-to-balloon:*
  - > 60 min: pas de différence de mortalité
  - > 90 min: pas de différence de mortalité, de réinfarction, stroke entre les deux procédures
- PTCA sauve 20 vies et évite 60 événements pour 1000 patients traités

N ENGL J MED 349;8 AUGUST 21, 2003

Nevertheless, now is the time for evidence-based therapy to dictate optimal patient care. Now is the time to discard the practice of transporting patients with acute myocardial infarction to the nearest hospital and to transport them preferentially to centers of excellence for primary percutaneous coronary intervention (Fig. 1). This practice will foster the availability of highly experienced angioplasty teams that can perform primary percutaneous coronary intervention with minimal delay. The model for this type of care exists in the emergency system comprising regional centers of excellence for trauma victims. Moreover, now is the time for tertiary hospitals capable of performing primary angioplasty to offer it 24 hours a day, seven days a week.

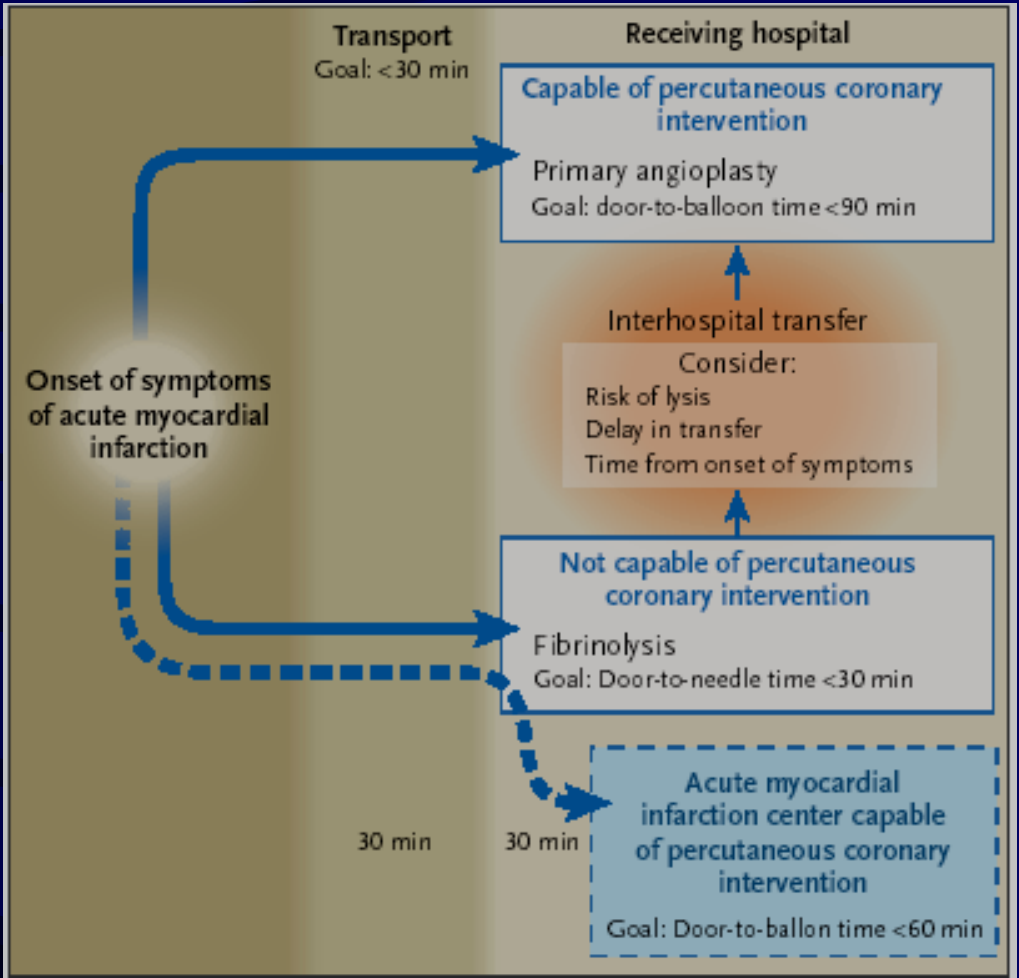


Figure 1. Suggested Triage of Patients with Myocardial Infarction and ST-Segment Elevation.

The solid arrows represent existing triage to the nearest receiving hospital, and the dashed arrow the ideal triage to a designated center of excellence for the treatment of acute myocardial infarction. The additional time necessary for transport would be negated by the availability of an experienced team performing primary percutaneous coronary intervention with a door-to-balloon time of less than 60 minutes. Adapted from Armstrong et al.[6]

VNI

- Après presque 20 ans d'utilisation, certaines questions persistent:
- Utilité ET sécurité dans l'OAP
- Utilité dans le sevrage respiratoire
- Utilité dans l'hypoxémie hypocapnique

Noninvasive Ventilation during Persistent Weaning Failure

A Randomized Controlled Trial

Miquel Ferrer, Antonio Esquinas, Francisco Arancibia, Torsten Thomas Bauer, Gumersindo Gonzalez, Andres Carrillo, Robert Rodriguez-Roisin, and Antoni Torres

Unitat de Vigilància Intensiva Respiratòria, Servei de Pneumologia, Institut Clínic de Pneumologia i Cirurgia Toràcica, Hospital Clínic, Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Universitat de Barcelona, Barcelona; and Unidad de Cuidados Intensivos, Hospital Morales Meseguer, Murcia, Spain

Am J Respir Crit Care Med Vol 168. pp 70–76, 2003

VNI et sevrage respiratoire

- Ventilation invasive est associée à un risque accru de pneumonie nosocomiale et à une augmentation de mortalité
- La ventilation mécanique prolongée, conséquance parfois d'échecs de sevrage, est un facteur de risque majeur de pneumonie nosocomiale et augmente la mortalité et la morbidité (Chastre et al.*AJRCCM 2002*)

- On recommande actuellement un essai quotidien de sevrage chez tous les patients stables, FiO2 < 50, Peep < ou = 5, réponse aux ordres simples et si bonne tolérance ➔ extubation (Ely et al.*N Eng J Med 1996*)
- VNI permet de réduire le nombre de recours à la ventilation mécanique dans certains sous groupe de patients (Brochard et al. *N Eng J Med 1995*)

MAIS

- Quel est l'effet de la VNI chez le patient avec un échec de sevrage répété?

Noninvasive Ventilation during Persistent Weaning Failure

A Randomized Controlled Trial

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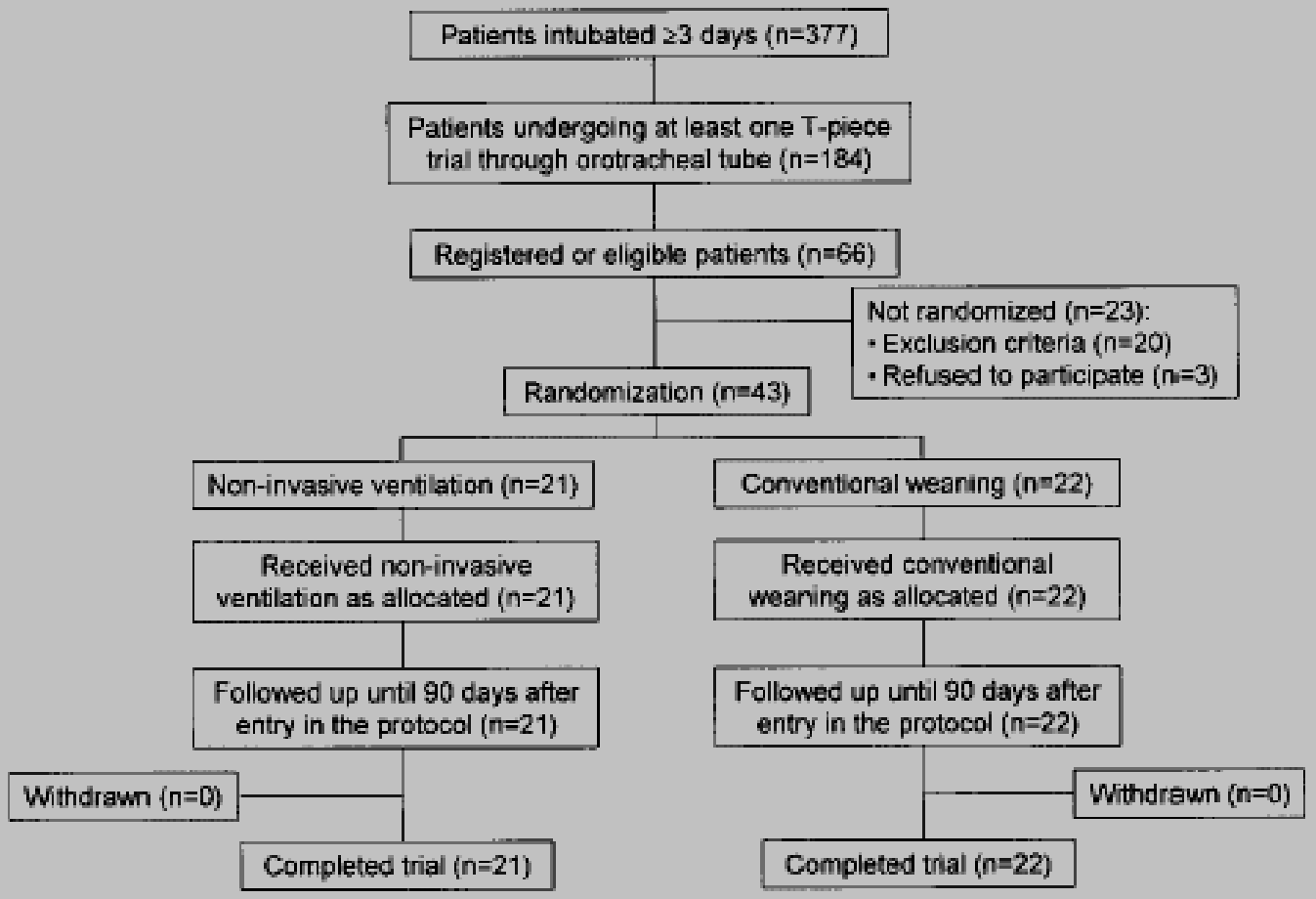
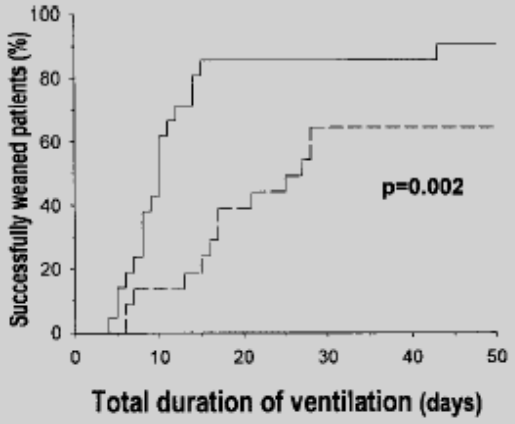


Am J Respir Crit Care Med Vol 168. pp 70–76, 2003

TABLE 3. WEANING RESULTS, LENGTH OF STAY, OUTCOME VARIABLES, AND CAUSES OF DEATH FOR THE NONINVASIVE VENTILATION AND THE CONVENTIONAL-WEANING GROUPS

	NIV Group (*n* = 21)	Conventional-Weaning Group (*n* = 22)	p Value
Duration of invasive ventilation, d	9.5 ± 8.3	20.1 ± 13.1	0.003
Total period of ventilatory support*, d	11.4 ± 8.0	20.1 ± 13.1	0.012
ICU stay, d	14.1 ± 9.2	25.0 ± 12.5	0.002
Hospital stay, d	27.8 ± 14.6	40.8 ± 21.4	0.026
Reintubation, n (%)	3 (14)	6 (27)	0.457
Main causes of reintubation, n			
Severe persistent hypoxemia	1	3	
Severe dyspnea	–	2	
Inability to manage secretions	2	–	
Hemodynamic instability	–	1	
Tracheotomy, n (%)	1 (5)	13 (59)	<0.001
ICU survival, n (%)	19 (90)	13 (59)	0.045
Causes of death within 90 d after entry in the study			
Septic shock/MOF	1	9	
Refractory hypoxemia	1	2	
Cardiac arrest	2	1	
Pneumothorax	–	1	
Stroke	1	–	
Pulmonary embolism	1	–	



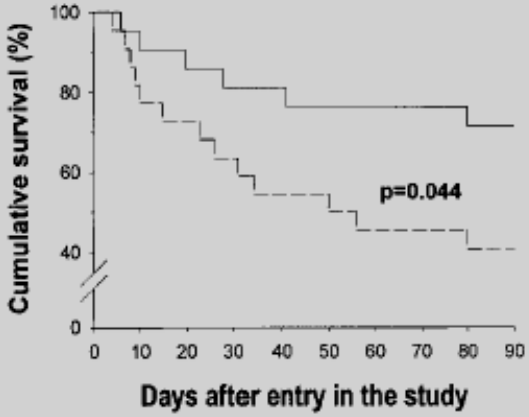


TABLE 4. SERIOUS COMPLICATIONS DIAGNOSED IN THE INTENSIVE CARE UNIT AFTER ENTRY INTO THE STUDY

	NIV Group ($n = 21$)	Conventional-Weaning Group ($n = 22$)	p Value
Total number of patients	5	16	0.004
Nosocomial pneumonia	5	13	0.042
Catheter-related sepsis	–	2	–
Sacrum-infected ulcer	–	1	–
Urinary tract infection	–	1	–
Chest wall abscess	–	1	–
Gastrointestinal bleeding	1	–	–
Pneumothorax	–	1	–
Septic shock	2	9	0.045

Conclusions

- VNI est efficace pour réduire la durée de ventilation chez les patients en échec de sevrage
- La VNI n'est pas la solution miracle mais en l'absence de contre indication et surtout chez les patients BPCO elle permet de diminuer:
  - la mortalité
  - le nombre d'infections nosocomiales
  - la longueur du séjour à l'USI
  - la durée de l'hospitalisation

Relative contraindications

Failure of prior attempts at noninvasive ventilation
Hemodynamic instability or life-threatening arrhythmias
High risk of aspiration
Impaired mental status
Inability to use nasal or face mask
Life-threatening refractory hypoxemia (PaO_2 <60 mm Hg with 1.0 FIO_2)*

Noninvasive Ventilation in Severe Hypoxemic Respiratory Failure

A Randomized Clinical Trial

Miquel Ferrer, Antonio Esquinas, Miguel Leon, Gumersindo Gonzalez, Antonio Alarcon, and Antoni Torres

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Am J Respir Crit Care Med Vol 168. pp 1438–1444, 2003

- Détresse respiratoire aiguë avec hypoxie évolue fréquemment vers l'intubation
- VNI peut être une alternative à l'intubation pour certains sous groupe de patients: BPCO, immunodéprimés, post pneumectomie,…
- cPAP inefficace dans ALI (Declaux et al. *JAMA 2000*)
- Intérêt de la VNI chez les patients hypoxiques non-hypercapniques? (Peter JV *CCM2002*)

Critères d'inclusion

- Prospectif , randomisé, multicentrique
- PO2 < 60 ou Satu < 90% sans hypercapnie
- En l'absence de nécessité d'intubation immédiate (altération conscience , instabilité hemodyn, trauma facial, fatigue)
- En l'absence d'encombrement bronchique majeur
- En l'absence de saignement digestif haut

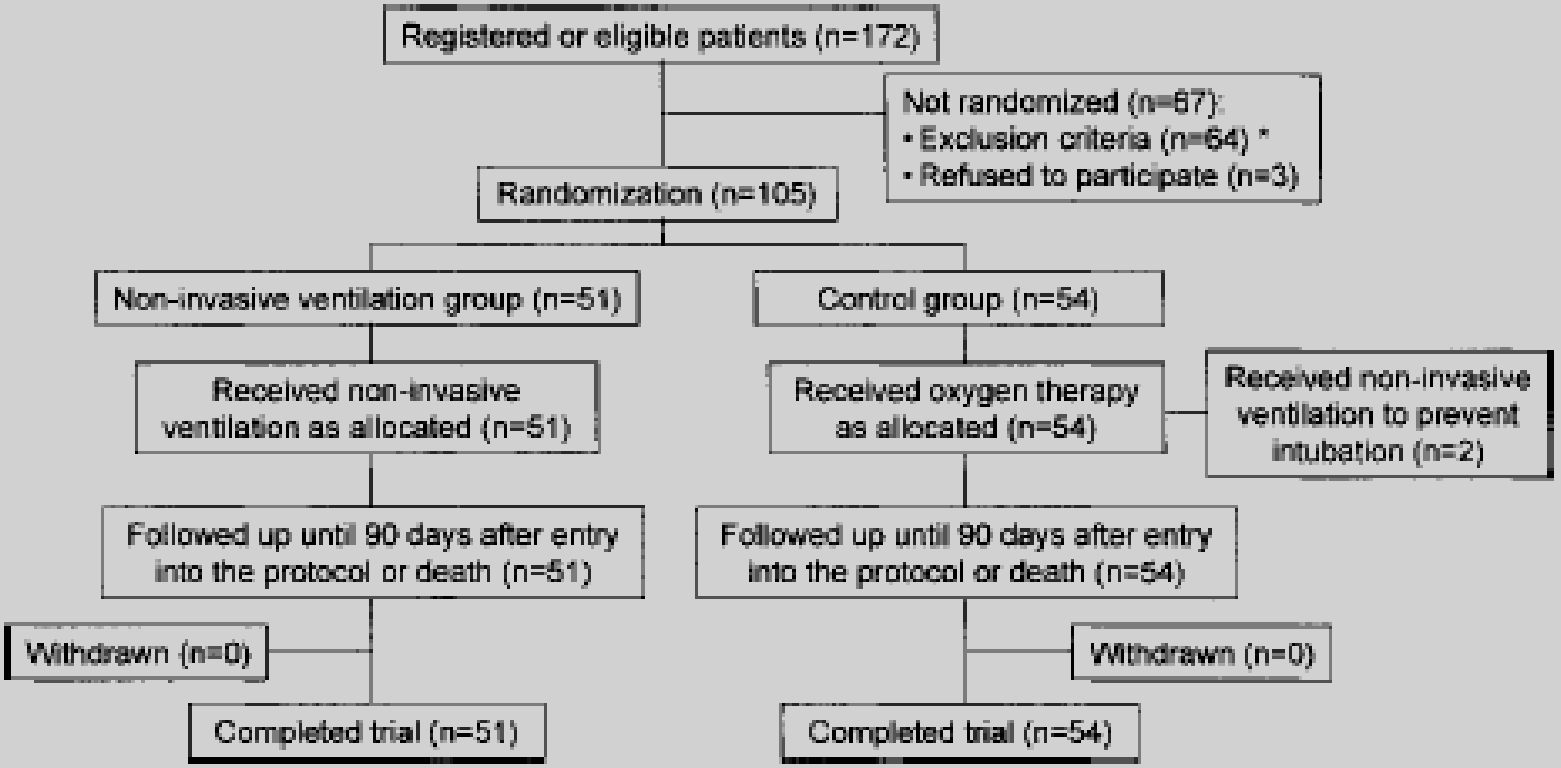


Figure 1. Trial profile. *The main exclusion criteria, as defined, were a lack of cooperation, including agitation and mild to moderate altered mental status (n = 45), the need for immediate intubation (n = 10), severely decreased consciousness (n = 5), and severe hemodynamic instability (n = 4).

TABLE 2. INTUBATION, LENGTH OF STAY, AND OUTCOME VARIABLES*

	Noninvasive Ventilation Group ($n = 51$)	Control Group ($n = 54$)	p Value
Intubation rate, n, %†	13 (25)	28 (52)	0.010
Pneumonia, n/tot	5/19	11/15	0.017
Cardiogenic pulmonary edema, n/tot	1/15	2/15	> 0.999
Thoracic trauma, n/tot	1/6	5/11	0.333
ARDS, n/tot	6/7	8/8	0.467
Other, n/tot	0/4	2/5	—
Indications for intubation and other relevant features at the time of intubation‡			
Signs of exhaustion	11	22	
Neurologic impairment	2	5	
Respiratory pauses and gasping	1	2	
Severe hemodynamic instability	2	5	
Respiratory or cardiac arrest	2	0	
Aspiration	1	1	
Inability to clear secretions	1	2	
Major agitation	2	3	
Refractory hypoxemia§	2	10	
Respiratory acidosis	1	3	
Metabolic acidosis	1	11	
Respiratory rate of more than 35 min^{-1}	5	13	
ICU stay, d	9.6 ± 12.6	11.3 ± 12.6	0.510
Among ICU survivors	8.0 ± 7.6	10.1 ± 10.7	0.339
Hospital stay, d	20.7 ± 16.6	26.8 ± 19.8	0.090
Among ICU survivors	21.1 ± 14.8	30.2 ± 21.3	0.043
Intensive care unit mortality, n (%)	9 (18)	21 (39)	0.028
Pneumonia, n/tot	3/19	8/15	0.030
Cardiogenic pulmonary edema, n/tot	1/15	2/15	> 0.999
Thoracic trauma, n/tot	0/6	3/11	0.515
ARDS, n/tot	5/7	7/8	0.569
Other, n/tot	0/4	1/5	—

TABLE 3. MULTIVARIATE ANALYSES OF RISK FACTORS FOR INTUBATION*

	Adjusted Odds Ratio	95% CI	p Value
Noninvasive ventilation†	0.20	0.07–0.58	0.003
Cardiogenic pulmonary edema†	0.14	0.04–0.56	0.005
ARDS	28.5	3.2–249.8	0.003

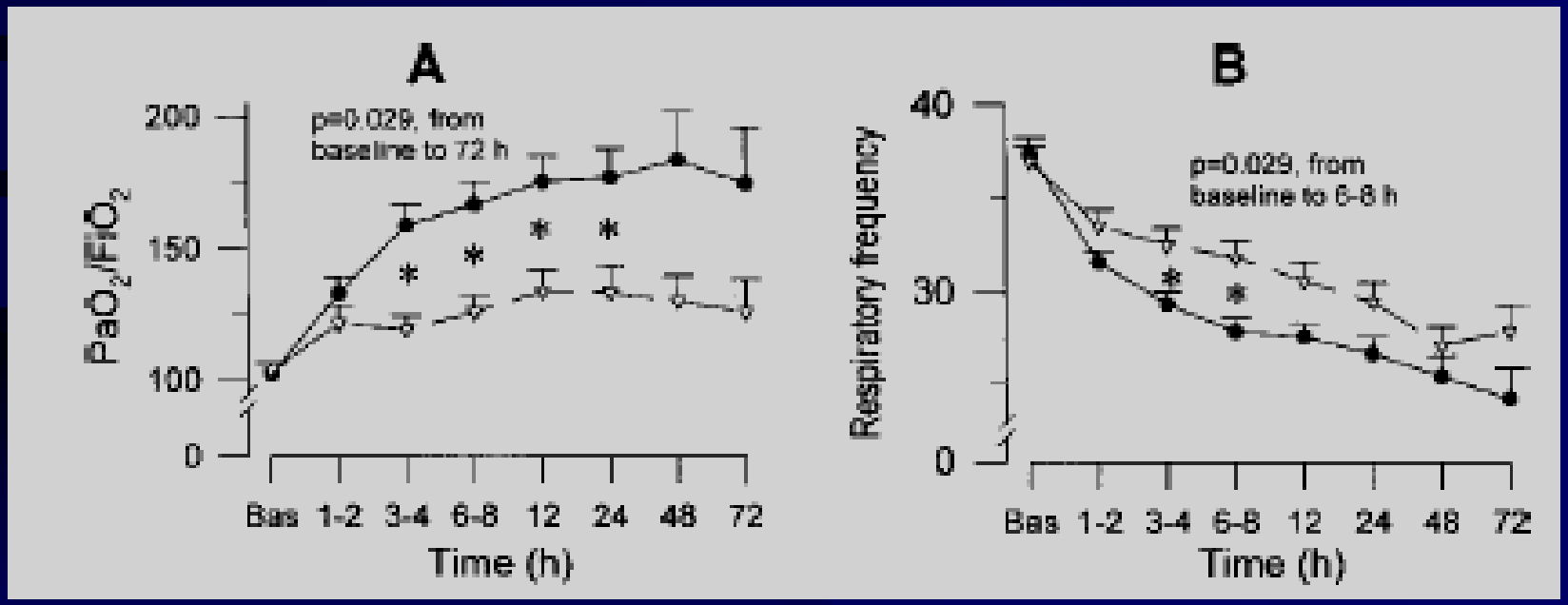


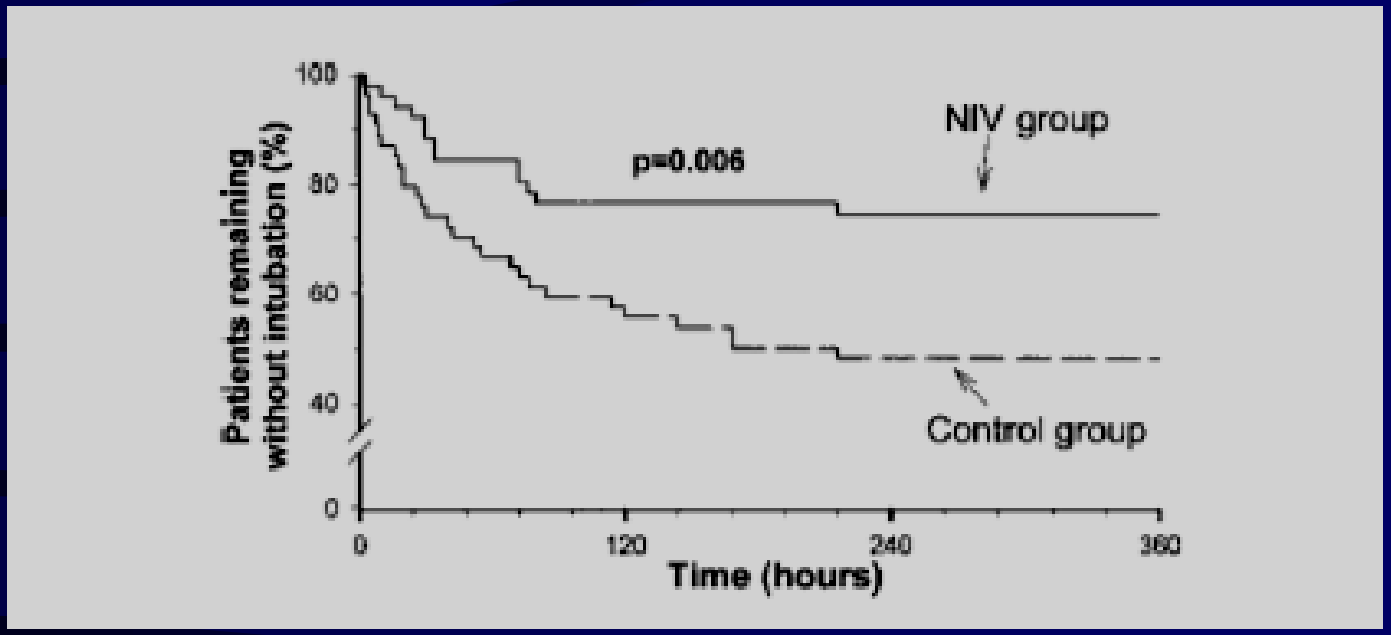
TABLE 5. MULTIVARIATE ANALYSES OF DECREASED 90-DAY SURVIVAL*

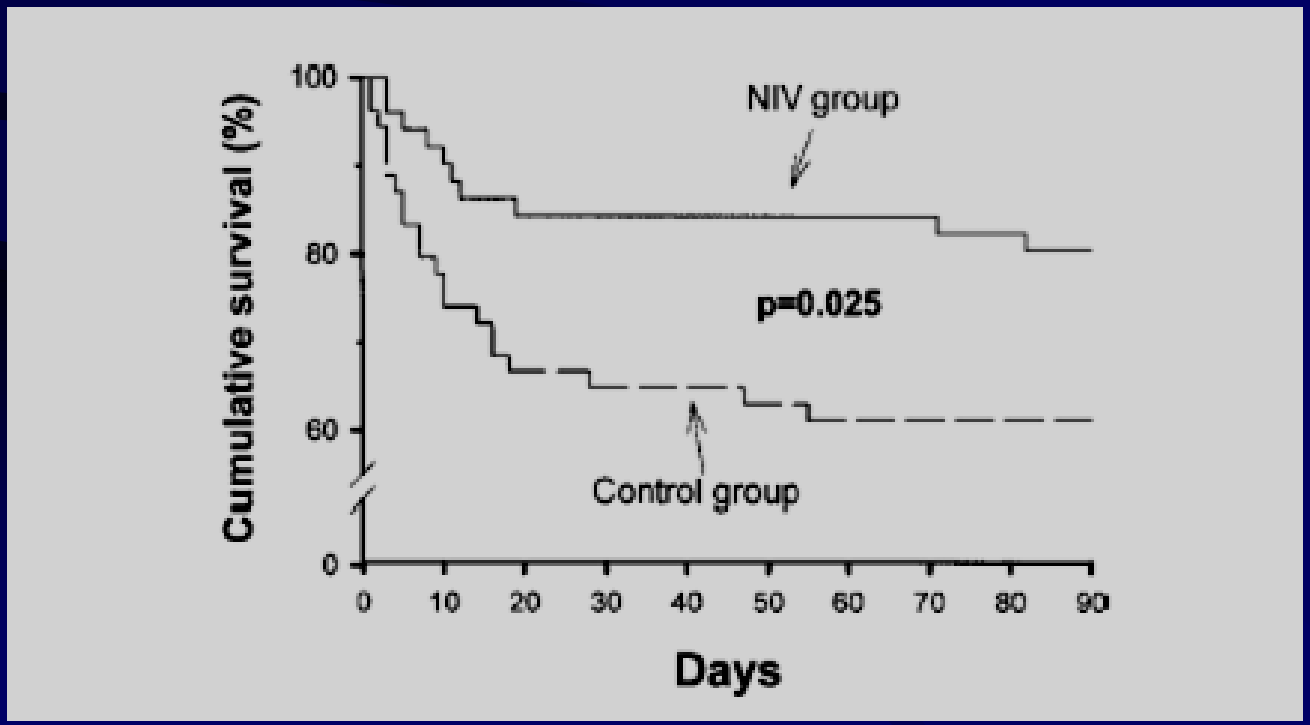
	Adjusted Odds Ratio	95% CI	p Value
All variables			
Intubation	38.3	9.1–161.4	< 0.001
Variables present at entry into the study only			
Noninvasive ventilation†	0.39	0.18–0.84	0.017
ARDS	5.1	2.4–11.0	< 0.001
SAPS-II of more than 37 on admission	2.4	1.1–5.0	0.021

Definition of abbreviations: ARDS = acute respiratory distress syndrome; CI = confidence interval; SAPS-II = simplified acute physiology score-II.

* Together with the randomized groups, the variables tested for association to 90-day survival are shown in the online data supplement.

† Adjusted odds ratio and 95% confidence intervals below one mean a beneficial effect on 90-day survival.


Patients remaining without intubation (%)
100
80
60
40
0
p=0.006
NIV group
Control group
0
120
240
360
Time (hours)


Cumulative survival (%)
100
80
60
0
NIV group
p=0.025
Control group
0
10
20
30
40
50
60
70
80
90
Days

Conclusions

- VNI efficace dans l'insuffisance respiratoire aiguë des sujets non hypercapniques (pas dans l'ARDS)
- Diminution
  - du nombre d'intubation
  - de l'incidence de choc septique
  - de tachypnée et hypoxie
  - des complications inhérentes à la ventilation invasive
  - conduit à une amélioration de la survie des patients en USI et à 90 jours
- En l'absence de contre-indications la VNI doit être utilisée en première ligne dans l'insuffisance respiratoire aiguë

Noninvasive Ventilation in Cardiogenic Pulmonary Edema

A Multicenter Randomized Trial

Stefano Nava, Giorgio Carbone, Nicola DiBattista, Andrea Bellone, Paola Baiardi, Roberto Cosentini, Mauro Marenco, Fabrizio Giostra, Guido Borasi, and Paolo Groff

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Am J Respir Crit Care Med Vol 168. pp 1432–1437, 2003

VNI dans l'OAP

- OAP, cause fréquente de dyspnée
- Dyspnée brutale, hypoxie, augmentation du travail respiratoire, acidose mixte, hypertension, tachycardie
- Trouble de la fonction diastolique est souvent une composante majeure de l'augmentation des PAP
- Ventilation souvent requise malgré traitement médical bien conduit

VNI dans l'OAP (2)

- VNI souvent employée dans cette indication, plus particulièrement la cPAP:
  - Augmentation de la CRF, recrutement alvéolaire, amélioration de l'oxygenation, diminution du travail respiratoire.
- Effets cardiovasculaires: diminution de la précharge et de la postcharge

VNI dans l'OAP (3)

- Etude physiologique montre que la Bipap plus efficace que la cPAP pour diminuer le travail respiratoire
- La Bipap plus efficace que la cPAP chez le BPCO décompensé
- Pas de recommandations claires pour l'utilisation de la Bipap dans l'OAP, ne serait efficace que si hypercapnie associée? Augmente le nombre d'infarctus? (Sharon et al. *JACC 2000*, Mehta et al. *CCM 1997*)
- Une seule étude randomisée (Masip et al.*Lancet 2000*): 40 patients, pas de différence de LOS ou mortalité mais moins d'intubation (5% vs 33%, $p < 0.05$)

- Prospectif, randomisé monocentrique, 130 patients

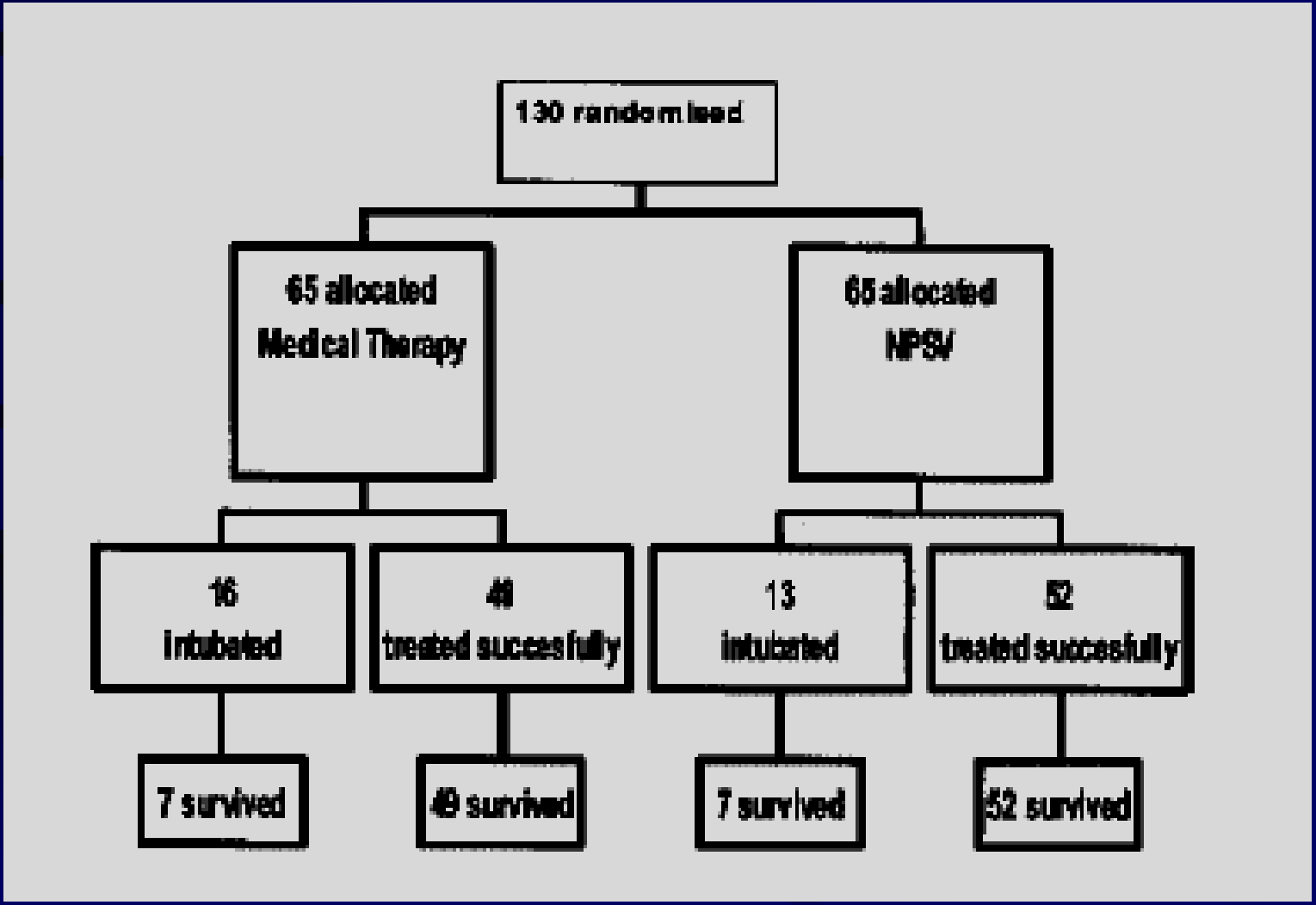

TABLE 2. INTUBATION RATE AND IN-HOSPITAL MORTALITY

	Standard Treatment	NPSV	p Value	OR
Intention to Treat				
Intubated	16/65 (25%)	13/65 (20%)	0.530	1.30
Died	9/65 (14%)	6/65 (8%)	0.410	1.58
Subgroup Analysis				
Pa_{CO_2} ≥ 45 mm Hg				
Intubated	9/31 (29%)	2/33 (6%)	0.015	6.34
Died	5/31 (16%)	1/33 (3%)	0.100	6.15
Pa_{CO_2} < 45 mm Hg				
Intubated	7/34 (21%)	11/32 (34%)	0.210	0.40
Died	4/34 (12%)	5/32 (15%)	0.650	0.72

Definition of abbreviations: NPSV = noninvasive pressure support ventilation; OR = odds ratio.

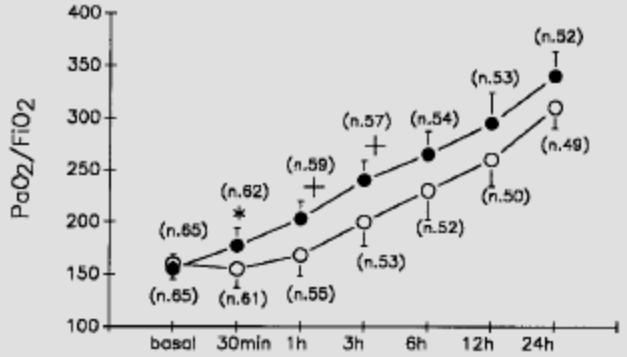

Figure 2. Oxygenation (Pa_{O_2}/Fi_{O_2} ratio) over time in the two randomized groups. *n* = number of patients not needing intubation or dead; *solid circles* = noninvasive pressure support ventilation (NPSV); *open circles* = standard treatment. Data represent means ± standard error. $^{+}p < 0.01$ NPSV versus standard treatment; $^{*}p < 0.05$ NPSV versus standard treatment. Repeated measures two-way analysis of variance.

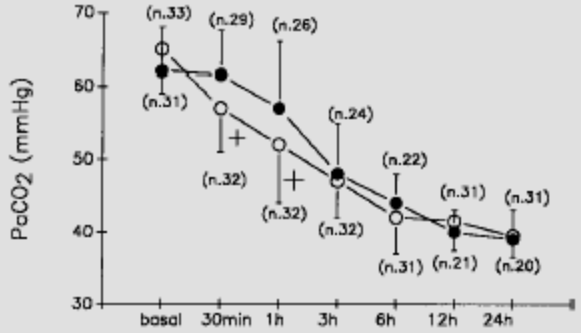

Figure 3. Measurements of arterial pressure of carbon dioxide (Pa_{CO_2}) over time in the two randomized groups of patients presenting at enrollment a $Pa_{CO_2} \geq 45$ mm Hg. *n* = number of patients not needing intubation or dead; *open circles* = NPSV; *solid circles* = standard treatment. Data represent means ± standard error. $^{+}p < 0.01$ NPSV versus standard treatment. Repeated measures two-way analysis of variance.

VNI dans l'OAP

- Amélioration plus rapide des échanges gazeux, diminution plus rapide de la fréquence respiratoire et de la dyspnée mais pas de différence en terme d'évolution clinique globale
- Réduction significative du nombre d'intubation dans le sous-groupe hypercapnique
- Nécessité de comparer cPAP et Bipap (une seule étude actuellement Mehta et al.*CCM 1997:* arrêt prématuré car infarctus dans le bras Bipap!)
- Recommandations actuelles: cPAP si hypoxique malgré traitement médical, Bipap si cPAP inefficace.(British Thoracic Society on the use of non invasive Thorax 2002)

- Encourage l’utilisation de la VNI dans l’insuffisance respiratoire aiguë même non hypercapnique et dans le processus de sevrage difficile (même pour les non BPCO)
- Seule son utilisation dans l’ARDS ne semble pas recommandée (risque d’hypoxie sévère lors de la déconnexion, souvent nécessité d’une ventilation mécanique prolongée)

PREVENTION DE L'INSUFFISANCE RENALE

- Néphropathie induite par agents de contraste:
  - 5% en cas d'insuffisance rénale légère
  - 50% en cas d'insuffisance rénale modérée et de diabète

- Mortalité des patients qui développent une néphropathie au produit de contraste 20% à l'hôpital et 35% à un an. Ceux qui requièrent une EER post PTCA ont une mortalité de 40 à 60%
- Augmente la morbidité, la mortalité, LOS, et peut être irréversible

➔ Intérêt de prévenir la toxicité

- Deux mécanismes:
  - Vasoconstriction et ischémie médullaire
  - Néphrotoxicité

  Les lésions ischémiques et toxiques peuvent être induites par la génération de radicaux libres

Acetylcysteine for Prevention of Acute Deterioration of Renal Function Following Elective Coronary Angiography and Intervention

A Randomized Controlled Trial

JAMA. 2003;289:553-558

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Context The antioxidant acetylcysteine prevents acute contrast nephrotoxicity in patients with impaired renal function who undergo computed tomography scanning. However, its role in coronary angiography is unclear.

Objective To determine whether oral acetylcysteine prevents acute deterioration in renal function in patients with moderate renal insufficiency who undergo elective coronary angiography.

Design and Setting Prospective, randomized, double-blind, placebo-controlled trial conducted from May 2000 to December 2001 at the Grantham Hospital at the University of Hong Kong.

Participants Two hundred Chinese patients aged mean (SD) 68 (6.5) years with stable moderate renal insufficiency (creatinine clearance <60 mL/min [1.00 mL/s]) who were undergoing elective coronary angiography with or without intervention.

Intervention Participants were randomly assigned to receive oral acetylcysteine (600 mg twice per day; n=102) or matching placebo tablets (n=98) on the day before and the day of angiography. All patients received low-osmolality contrast agent.

Main Outcome Measures Occurrence of more than a 25% increase in serum creatinine level within 48 hours after contrast administration; change in creatinine clearance and serum creatinine level.

Results Twelve control patients (12%) and 4 acetylcysteine patients (4%) developed a more than 25% increase in serum creatinine level within 48 hours after contrast administration (relative risk, 0.32; 95% confidence interval [CI], 0.10-0.96; $P=.03$). Serum creatinine was lower in the acetylcysteine group (1.22 mg/dL [107.8 µmol/L]; 95% CI, 1.11-1.33 mg/dL vs 1.38 mg/dL [122.9 µmol/L]; 95% CI, 1.27-1.49 mg/dL; $P=.006$) during the first 48 hours after angiography. Acetylcysteine treatment significantly increased creatinine clearance from 44.8 mL/min (0.75 mL/s) (95% CI, 42.7-47.6 mL/min) to 58.9 mL/min (0.98 mL/s) (95% CI, 55.6-62.3 mL/min) 2 days after the contrast administration ($P<.001$).The increase was not significant in the control group (from 42.1 to 44.1 mL/min [0.70 to 0.74 mL/s]; $P=.15$). The benefit of acetylcysteine was consistent among various patient subgroups and persistent for at least 7 days. There were no major treatment-related adverse events.

Conclusion Acetylcysteine protects patients with moderate chronic renal insufficiency from contrast-induced deterioration in renal function after coronary angiographic procedures, with minimal adverse effects and at a low cost.

Caractéristiques des patients

Characteristic	Study Group: Control (n = 98)	Study Group: Acetylcysteine (n = 102)	*P* Value
Age, median (IQR), y	69 (48-82)	69 (50-81)	.60
Men, No. (%)	62 (63)	61 (60)	.62
Body mass index, mean (SD)	23.7 (3.0)	23.7 (3.2)	.84
Causes of renal impairment, No. (%)			
Diabetic nephropathy	29 (30)	38 (37)	.34
Hypertensive nephropathy	36 (37)	33 (32)	
Obstructive nephropathy	23 (23)	16 (16)	
Other	2 (2)	1 (1)	
Unknown	8 (8)	14 (14)	
Serum urea, median (IQR), mg/dL*	18.8 (9.2-60.2)	17.4 (7.8-39.2)	.50
Serum creatinine, median (IQR), mg/dL*	1.26 (0.75-3.64)	1.24 (0.77-2.99)	.65
Serum creatinine >2.5 mg/dL, No. (%)	3 (3)	4 (4)	.74
Estimated creatinine clearance, mL/min†	44.8 (16.0-58.6)	46.4 (13.9-57.8)	.46
24-h creatinine clearance, mL/min	45 (12.7-59.8)	47 (14.0-59.4)	.11
Volume of contrast agent, median (IQR), mL	120 (70-380)	130 (75-320)	.29
Volume of contrast agent per body weight, median (IQR), mL/kg	2.1 (1.1-7.6)	2.2 (1.1-5.5)	.74

JAMA. 2003;289:553-558

Table 2. Clinical Outcomes in the Control and Acetylcysteine Groups

Outcome	Study Group: Control (n = 98)	Study Group: Acetylcysteine (n = 102)	RR (95% CI)	*P* Value
Acute contrast-induced reduction in renal function, No. (%)*	12 (12)	4 (4)	0.32 (0.10-0.96)	.03
Serum creatinine level increased >50% over baseline, No. (%)	2 (2)	0 (0)		.15
Oliguria†	3	1	0.32 (0.03-3.03)	.29
Length of hospitalization, mean (SD), d‡	3.9 (2.0)	3.4 (0.9)	0.52 (0.08-0.96)§	.02

Abbreviations: CI, confidence interval; RR, relative risk.
*Defined as >25% increase in serum creatinine level within 48 hours after exposure to contrast agent.
†Hourly urine output <0.5 mL × body weight in kg.
‡From admission to discharge.
§Values indicate difference between groups (95% CI).

Aucun patient ne nécessita une EER

Conclusions

- Administration of acetylcysteine est sûre et peu coûteuse et previent la nephrotoxicité du produit de contraste grâce à ses propriétés antioxydantes et aussi vasodilatatrices rénales
- D'autres études sont nécessaires pour voir si acetylcysteine diminue la morbidité (EER) ou la mortalité

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

The Prevention of Radiocontrast-Agent–Induced Nephropathy by Hemofiltration

Giancarlo Marenzi, M.D., Ivana Marana, M.D., Gianfranco Lauri, M.D., Emilio Assanelli, M.D., Marco Grazi, M.D., Janess Campodonico, M.D., Daniela Trabattoni, M.D., Franco Fabbiocchi, M.D., Piero Montorsi, M.D., and Antonio L. Bartorelli, M.D.

N Engl J Med 2003;349:1333-40.

Inclusion

- Randomisé, monocentrique
- 114 patients creat > 2mg/dl et clairance < 50 ml/min.
- Hemofiltration 4 à 8 h avant et 18 à 24 h après (1L/h sans perte)
- OU hydratation saline simple 1 ml/kg/h ou 0.5 ml/kg/h si FE < 40%

Table 1. Base-Line Characteristics of the Study Patients.*

Characteristic	Hemofiltration Group (N=58)	Control Group (N=56)	P Value
Clinical characteristics			
Age — yr	69±10	69±11	0.75
Male sex — no. (%)	46 (79)	43 (77)	0.74
Diabetes — no. (%)	17 (29)	17 (30)	0.90
Hypertension — no. (%)	40 (69)	38 (68)	0.90
Prior myocardial infarction — no. (%)	18 (31)	16 (29)	0.77
Prior CABG — no. (%)	6 (10)	7 (12)	0.71
Prior PTCA — no. (%)	2 (3)	2 (4)	1.00
Left ventricular ejection fraction — %	50±13	49±12	0.67
Left ventricular ejection fraction <40% — no. (%)	14 (24)	14 (25)	1.00
Medications			
ACE inhibitors — no. (%)	7 (12)	8 (14)	0.72
Aspirin — no. (%)	25 (43)	29 (52)	0.35
Diuretics — no. (%)	32 (55)	32 (57)	0.83
Laboratory measures†			
Serum creatinine — mg/dl	3.0±1.0	3.1±1.0	0.84
Creatinine clearance — ml/min	26±9	26±8	0.63
Blood urea nitrogen — mg/dl	58±21	63±21	0.18

Table 2. Procedures Involving Radiocontrast Agent.*

Variable	Hemofiltration Group (N=58)	Control Group (N=56)	P Value
Coronary angiography — no. (%)	58 (100)	56 (100)	1.00
PTCA and stenting — no. (%)	51 (88)	48 (86)	0.94
Single-vessel	45 (78)	42 (75)	0.92
Multivessel	6 (10)	6 (11)	0.81
Associated procedures — no. (%)	18 (31)	15 (27)	0.77
Aortic angiography	10 (17)	8 (14)	0.86
Peripheral angioplasty	2 (3)	2 (4)	1.00
Renal angioplasty	6 (10)	5 (9)	0.80
Other	4 (7)	3 (5)	0.73
Volume of contrast agent used — ml	247±125	258±132	0.70

Table 3. Postprocedural Complications.*

Complication	Hemofiltration Group (N=58)	Control Group (N=56)	P Value
	no. (%)		
Myocardial infarction			
Q-wave	0	2 (4)	0.24
Non–Q-wave	1 (2)	1 (2)	1.00
Emergency CABG required	0	0	1.00
Pulmonary edema	0	6 (11)	0.02
Hypotension or shock	1 (2)	3 (5)	0.36
Blood transfusion required	1 (2)	3 (5)	0.36
Renal-replacement therapy required	2 (3)†	14 (25)	<0.001
All clinical events	5 (9)	29 (52)	<0.001

* CABG denotes coronary-artery bypass grafting. Renal-replacement therapy consisted of hemodialysis or hemofiltration.

† These two patients underwent prolonged prophylactic treatment with hemofiltration.

N Engl J Med 2003;349:1333-40.

- Risque relatif de décès à 1 an:
  - 1.16 (p 0.11) si créatinine < 4 mg/dl
  - 3.53 (p = 0.002) si créatinine > 4 mg/dl

EDITORIALS

Editorials represent the opinions of the authors and The Journal and not those of the American Medical Association.

Prevention of Contrast Nephropathy

Gary C. Curhan, MD, ScD

JAMA, February 5, 2003—Vol 289, No. 3 (Reprinted)

Invasive Cardiovascular Procedures — Optimizing Patient Safety

John W. Hirshfeld, Jr., M.D.

N ENGL J MED 349;14 WWW.NEJM.ORG OCTOBER 2, 2003

Recommandations

- Evaluation de la fonction rénale: clairance créatinine > 60 ml/min
- Perfusion 1ml/kg/h LP 12h avant et 6h après, utilisation d'agents de contraste non-ionique et à osmolalité basse, limitation de la dose de contraste < 100 ml
- Pour les patients avec fonction rénale réduite on peut suggérer d'administrer 600 mg acetylcysteine 2x/j
- Hémofiltration pour les patients à haut risque?

Prévention ulcères de stress

Intensive Care Med (2003) 29:1306–1313
DOI 10.1007/s00134-003-1863-3

ORIGINAL

Christophe Faisy
Emmanuel Guerot
Jean-Luc Diehl
Eléonore Iftimovici
Jean-Yves Fagon

Clinically significant gastrointestinal bleeding in critically ill patients with and without stress-ulcer prophylaxis

- Fréquence de saignement significatif varie de 0.6 à 6% selon les études et a diminué au cours des 20 dernières années
- Deux facteurs de risque indépendants de saignement digestif ont été identifiés (Cook et al. *N Eng J Med 1994*):
  - Insuffisance respiratoire aiguë nécessitant ventilation > 48 h
  - Coagulopathies
  - Chez les patients ventilés: insuf rénale, absence de nutrition entérale, absence de prophylaxie = risque de saignement (Cook et al. *CCM 1999*)
- Une trop large utilisation de la prévention des ulcères de stress pourrait gommer le bénéfice de la diminution de leur saignement:
  - Augmentation des coûts
  - Augmentation du risque de pneumonie nosocomiale

	Prophylaxis (*n*=736)	No prophylaxis (*n*=737)
Age (years)	58±21 (57–59)	58±20 (56–59)
SAPS II at ICU admission	31±19 (30–32)*	33±22 (31–35)
Sex (%)		
Male	49 (45–53)	51 (47–55)
Female	51 (47–55)	49 (45–53)
Medical ICU admission (%)	93 (91–95)	94 (92–96)
Acute respiratory failure	40 (36–43)	44 (40–48)
Cardiovascular failure	16 (13–19)	18 (15–21)
Drug overdose	21 (18–24)	18 (15–21)
Neurological failure	7 (5–9)	7 (5–9)
Metabolic disorders	7 (5–9)	6 (4–8)
Other	2 (1–3)	1 (0–2)
Surgical ICU admission (%)	7 (5–9)	6 (4–8)
Obstetric	0.8 (−1 to +3)	2.3 (1–3)
Trauma	1.3 (0–2)	2.7 (1–4)
Cervical	4.9 (3–6)*	1 (0–2)

Cause of bleeding	Prophylaxis (*n*=736)	No prophylaxis (*n*=737)
Overt gastrointestinal bleeding	1.9 (0.9–2.9)	1.6 (0.7–2.5)
Clinically significant gastrointestinal bleeding	1.4 (1.5–2.2)	1.1 (0.3–1.8)
Confirmed extradigestive bleeding	4.6 (3.1–6.1)*	9 (7–11)
Probable extradigestive blood loss	2.2 (1.2–3.2)	3 (1.8–4.2)

Reason for transfusion	Prophylaxis (*n*=736)	No prophylaxis (*n*=737)
Overall transfusion		
Number of patients	60	96
Total number of blood units transfused	292	461
Blood units transfused per patient	5±3 (4–6)	5±7 (3–6)
Clinically significant gastrointestinal bleeding		
Number of patients	10	8
Total number of blood units transfused	69	93
Blood units transfused per patient	7±3 (5–9)	12±17 (0–24)
Confirmed extradigestive bleeding		
Number of patients	34	66
Total number of blood units transfused	159	303
Blood units transfused per patient	5±3 (4–6)	5±5 (3–6)
Probable extradigestive blood loss		
Number of patients	16	22
Total number of blood units transfused	64	65
Blood units transfused per patient	4±3 (3–5)	2±1 (2–3)

Risk factors for CSGB	Prophylaxis (*n*=736)	No prophylaxis (*n*=737)
MV >48 h		
Number of patients	228*	284
Age (years)	65±17 (63–68)	65±18 (63–67)
SAPS II	43±19 (41–46)	46±20 (44–48)
CSGB (%)	4.4 (2–7)	2.8 (1–5)
Length of ICU stay (days)	13±12 (11–15)	14±18 (12–16)
ICU mortality (%)	32 (26–38)	32 (27–37)
Coagulopathy		
Number of patients	90*	115
Age (years)	62±20 (58–66)	64±18 (61–67)
SAPS II	42±24 (37–47)	47±27 (42–52)
CSGB (%)	5.5 (1–10)	3.5 (0–7)
Length of ICU stay (days)	9±7 (7–10)	11±19 (9–15)
ICU mortality (%)	31 (21–40)	32 (23–40)
Acute renal failure		
Number of patients	85*	116
Age (years)	70±17 (66–74)	69±17 (66–72)
SAPS II	53±25 (47–58)	57±18 (54–60)
CSGB (%)	3.5 (0–7)	3.4 (0–7)
Length of ICU stay (days)	8±6 (6–9)	11±15 (8–14)
ICU mortality (%)	43 (32–53)	56 (47–65)
MV >48 h + coagulopathy		
Number of patients	50*	77
Age (years)	61±18 (56–66)	66±16 (63–70)
SAPS II	52±26 (45–59)	55±28 (49–61)
CSGB (%)	10 (7–12)	4 (0–8)
Length of ICU stay (days)	9±8 (6–12)	15±22 (10–20)
ICU mortality (%)	46 (39–53)	45 (34–56)
MV >48 h +coagulopathy + acute renal failure		
Number of patients	20*	42
Age (years)	65±18 (57–73)	66±16 (61–71)
SAPS II	71±27 (60–83)	68±28 (60–77)
CSGB (%)	10 (–3 to +23)	7 (–1 to +15)
Length of ICU stay (days)	7±6 (5–10)	14±20 (8–20)
ICU mortality (%)	85 (69–100)	69 (55–83)

Conclusions

- Pas d'impact sur le nombre de saignement et l'évolution des patients
- Mortalité des patients qui ont un saignement digestif significatif est 6 à 7x plus élevée, mais cela semble lié à la gravité de la pathologie de départ
- L'absence de prophylaxie (même dans le groupe à risque défini par Cook et al.) ne modifie pas le nomre de saignements digestifs
- La nutrition entérale et le maintien d'une hémodynamique stable jouent un rôle majeur dans ce rôle