INSUFFISANCE RESPIRATOIRE AIGUE EN ONCOHEMATOLOGIE

Dr Ayed Samia

Service de Réanimation Médicale Ariana

Collège de Réanimation Médicale

le 07/10/2017

PLACE DU PROBLEME…

- Nombre de patients immunodéprimés en constante croissance:
  - Augmentation du nombre de cancers et d'hémopathies
  - Augmentation du nombre de greffes de moelle ou d'organes solides,
  - Plus large utilisation de traitements immunodépresseurs dans des indications non oncologiques: maladies de système et maladies inflammatoires chroniques.

- IRA: première cause d'admission en soins intensifs
  - **5% en oncologie Vs 20% en cas d'hémopathies**
  - **30% Allogreffe de moelle osseuse**

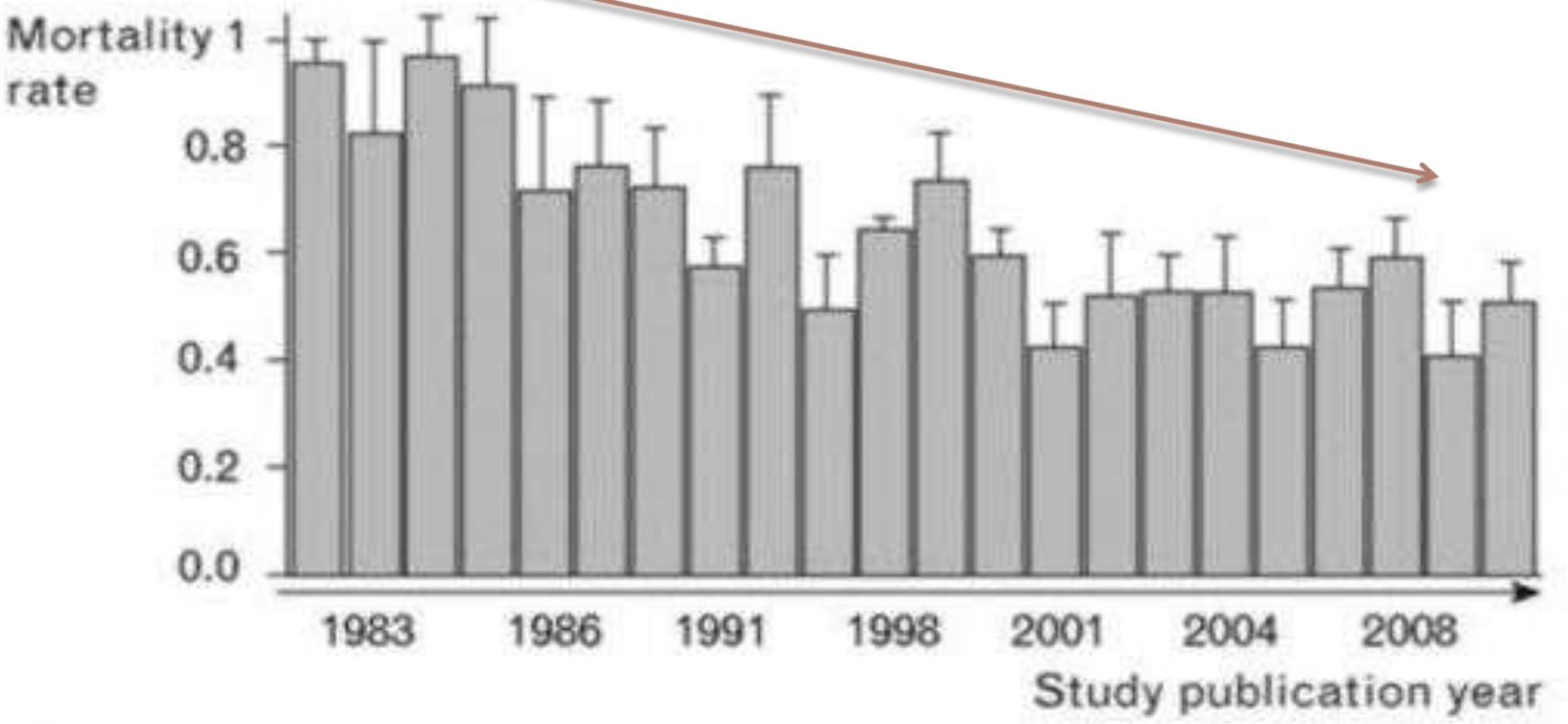
- **Incidence croissante:**
  - Constante augmentation des pathologies malignes
  - Allongement de l'espérance de vie des POH grâce à l'amélioration des traitements de support et à l'administration de traitements curatifs de plus en plus intensifs

    (mais de plus en plus immunosuppresseurs ou toxiques)

- Mortalité élevée: 50%
  - 75% si ARDS
  - 90% si ventilation invasive

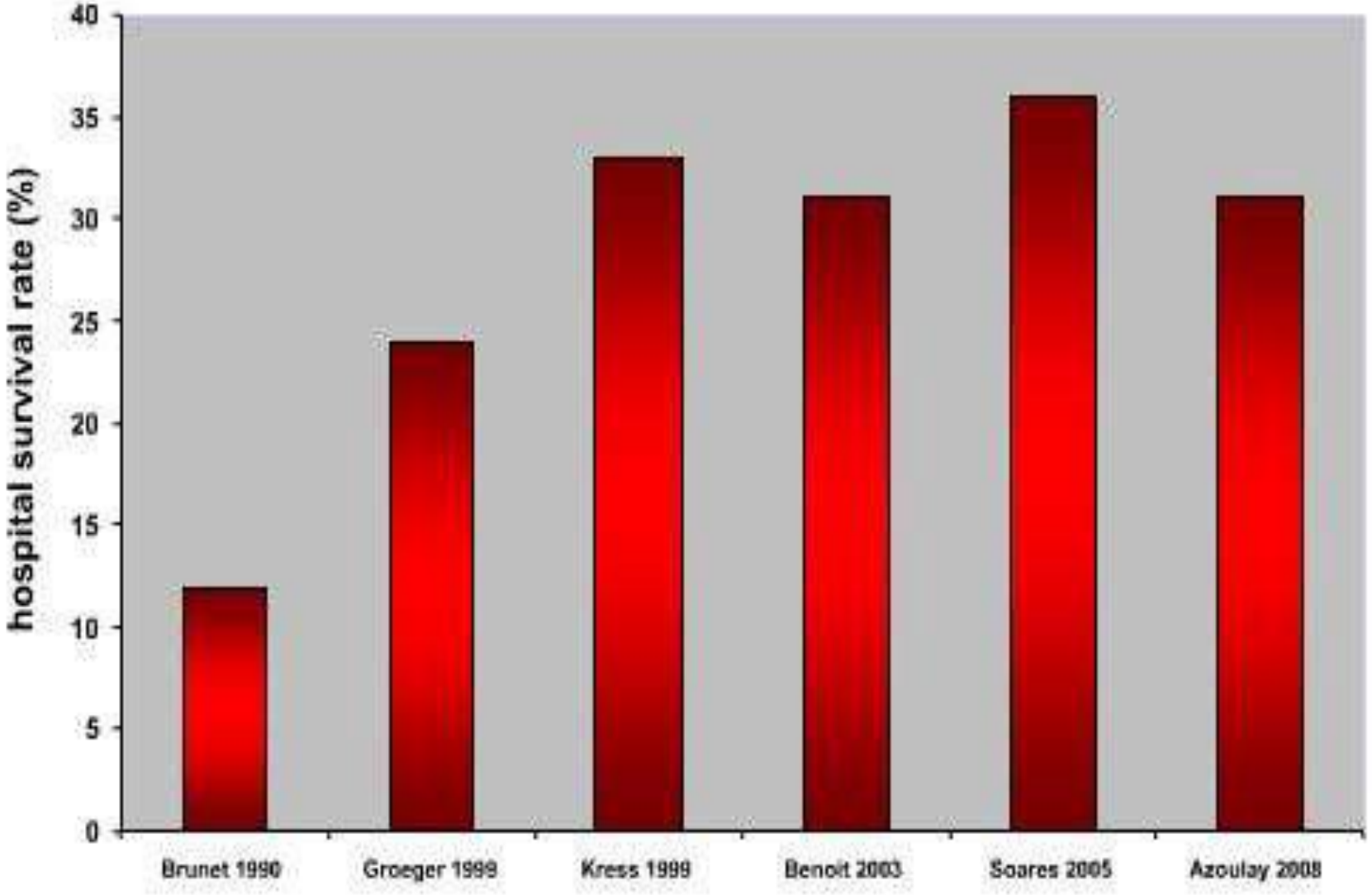
- Facteurs de pronostic:
  - État général altéré (performance status)
  - Retard d'admission en réanimation
  - Étiologie de l'IRA
  - Greffe allogénique de moelle
  - Défaillance multiple d'organes
  - Ventilation mécanique invasive

MORTALITE EN REANIMATION DES PATIENTS D'ONCOHEMATOLOGIE

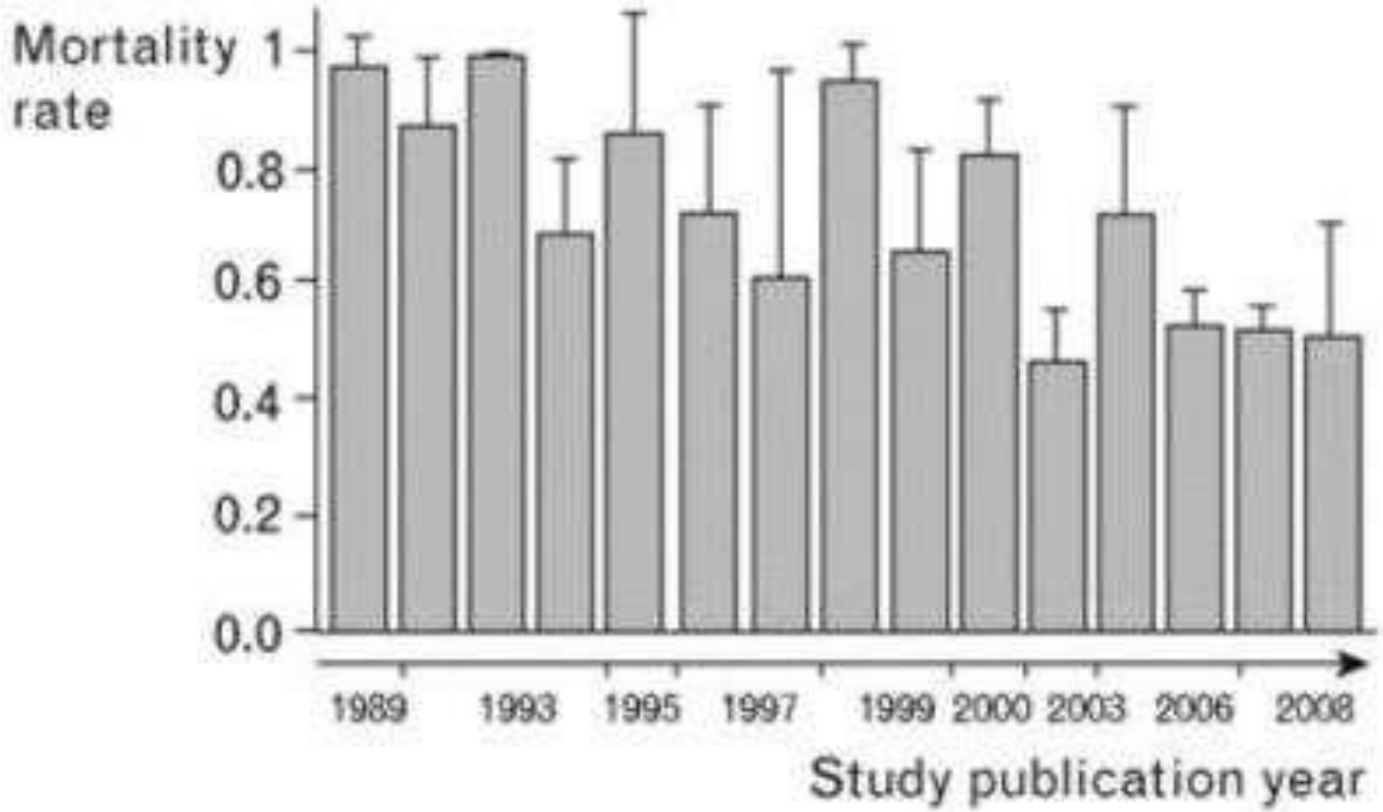


Darmon, Curr Op Oncol 2009

SURVIE EN REANIMATION DES PATIENTS D'ONCOHEMATOLOGIE

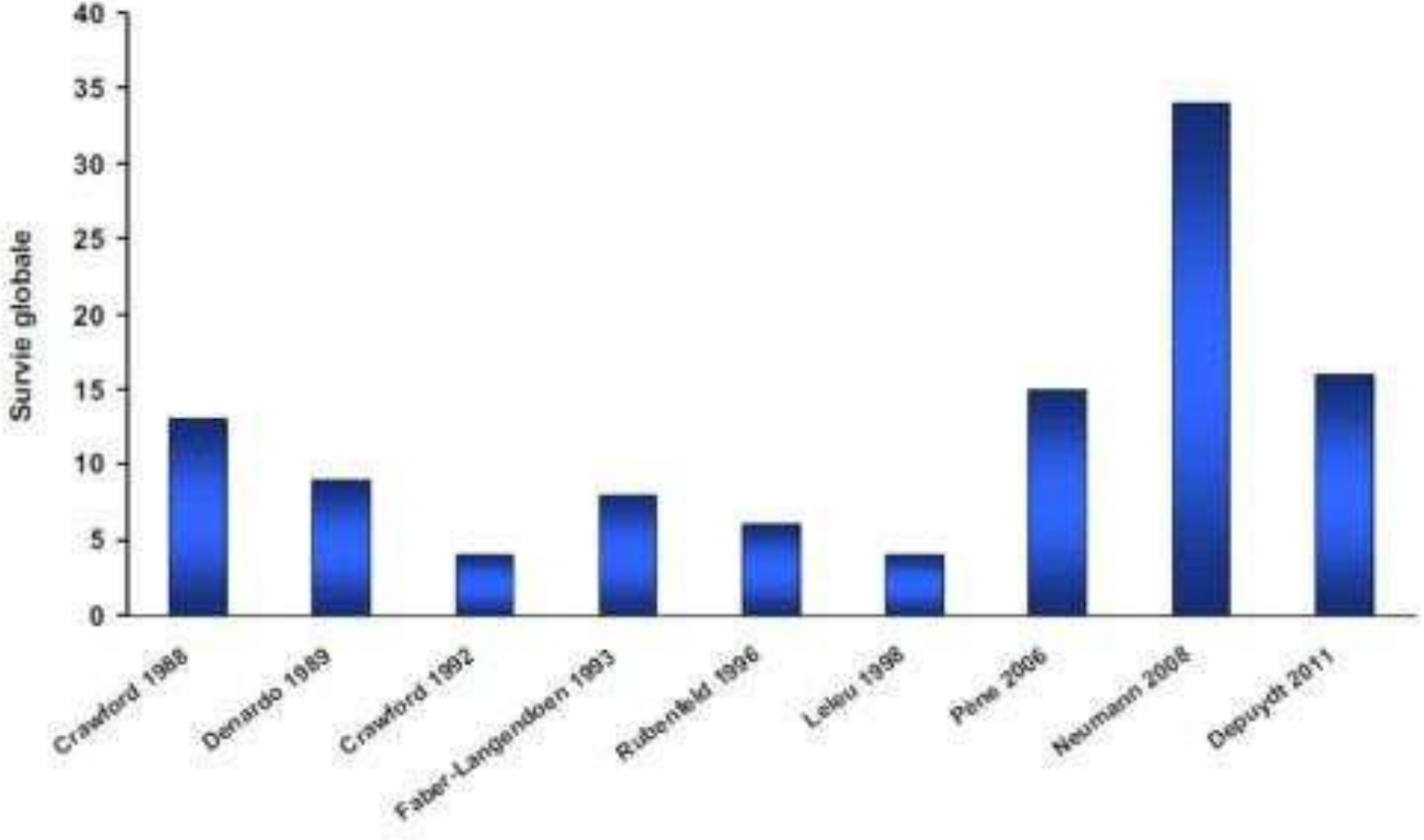


MORTALITE DES ALLOGREFFES DE MOELLE EN REANIMATION



Darmon, Curr Op Oncol 2009

SURVIE DES ALLOGREFFES DE MOELLE MIS SOUS VM



- **Pronostic en réanimation amélioré à plusieurs niveaux:**

  - Admission précoce en réanimation
  - Introduction de la VNI (alternative à la ventilation invasive)
  - Stratégie non invasive pour le diagnostic étiologique (mini-diagnostic stratégie…).
  - Prise en charge agressive des états de choc septiques
  - Arsenal thérapeutique antitumoral plus puissant et agressif

- **Fin de l'adage de non admission en réanimation des patients d'oncohématologie en détresse vitale**

The ICU Trial: A new admission policy for cancer patients requiring mechanical ventilation*

Lucien Lecuyer, MD; Sylvie Chevret, MD, PhD; Guillaume Thiery, MD; Michael Darmon, MD; Benoît Schlemmer, MD; Élie Azoulay, MD, PhD

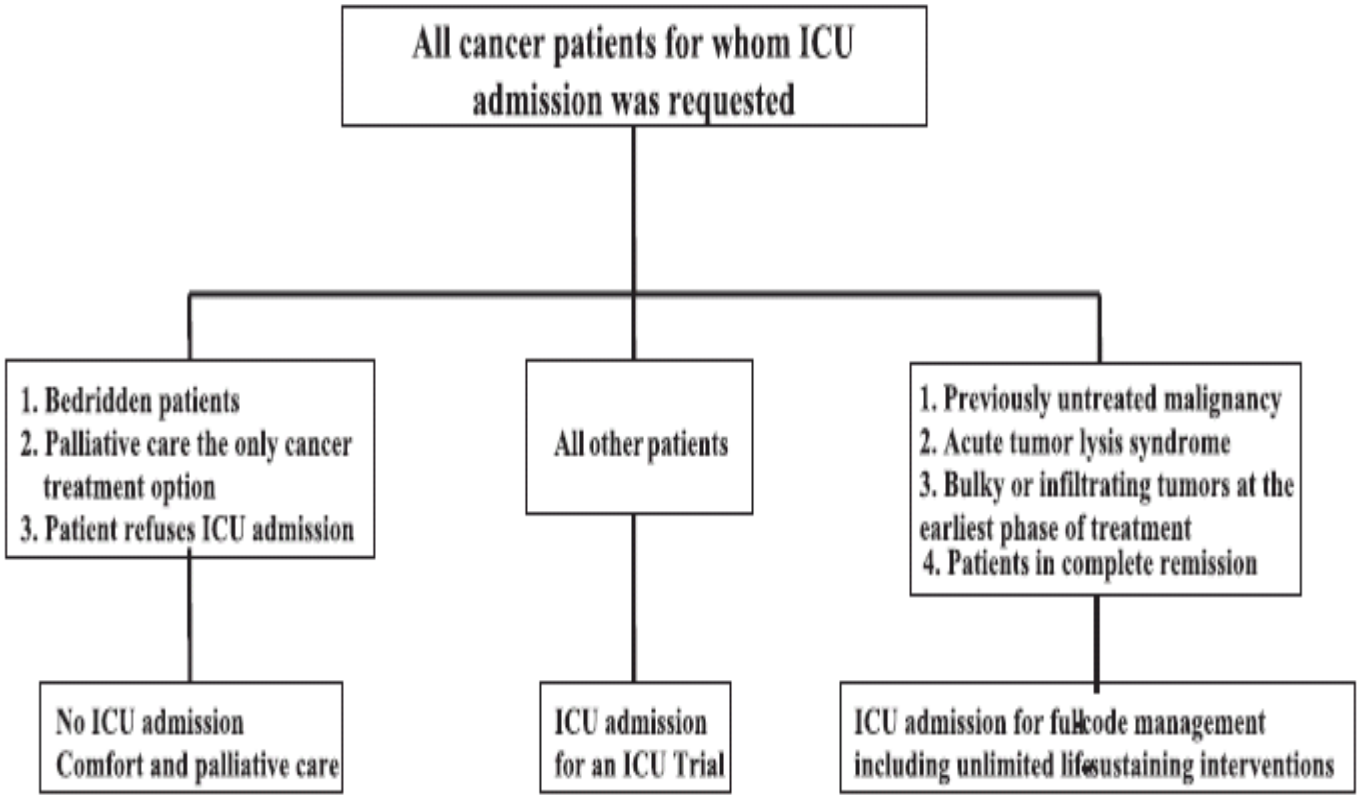


Figure 1. Intensive care unit (*ICU*) admission policy initiated in 2001 at the Saint-Louis Hospital.

DIAGNOSTIC ETIOLOGIQUE D'UNE IRA DES PATIENTS D'ONCOHEMATOLOGIE

SIGNES CLINIQUES

- Tachypnée
- Mise en jeu des muscles respiratoires accessoires voire épuisement
- SaO2 < 90% à l'AA
- Besoins d'O2 à fort débit ou support ventilatoire

E.AZOULAY. REV MAL RESPIR 2008;25: 433-49

Intensive Care Med (2006) 32:808–822
DOI 10.1007/s00134-006-0129-2

REVIEW

Élie Azoulay
Benoît Schlemmer

Diagnostic strategy in cancer patients with acute respiratory failure

The DIRECT approach: a guide to select initial antimicrobial treatments and appropriate investigations

Delay since malignancy onset or BMT
Patterns of Immune deficiency
Radiographic appearance
Clinical Experience and knowledge of the literature
Clinical picture
Findings by the high resolution computed Tomodensitometry (HRCT)

Tableau III.

Diagnostic étiologique de l'insuffisance respiratoire aiguë d'après les données cliniques et radiographiques [51].

Tableau radioclinique	1	2	3	4	5
Vitesse de progression	Lente	Rapide	Rapide	Modérée à rapide	Modérée à rapide
Fièvre	NON	OUI	OUI	OUI	OUI
Radiographie	Infiltrat diffus	Infiltrat diffus	Alvéolaire focal ou diffus	Nodules ± excavation	Alvéolaire focal
Diagnostics suspectés	Insuffisance cardiaque congestive Toxicité médicamenteuse Infiltration spécifique	Infection opportuniste (*P. jirovecii*, CMV, Tuberculose) Toxicité médicamenteuse Infiltration spécifique	Bactéries Sepsis SDRA	Champignons Legionella Tuberculose Maladie thromboembolique	Mycobactéries Nocardia Rhodococcus BOOP Tumeur
Examens de première ligne	Échocardiographie FB-LBA Biopsie pulmonaire	FB-LBA	Hémocultures ECBC Prélèvement distal protégé Traitement immédiat	Scanner Biopsie sous scanner FB+ BTB Biopsie pulmonaire chirurgicale	FB Biopsie

FB : fibroscopie bronchique; FB-LBA : fibroscopie bronchique avec lavage broncho-alvéolaire; BTB : biopsie trans-bronchique ; BOOP : bronchiolite oblitérante avec pneumopathie organisée ; Vitesse de progression : rapide signifie < 2 jours, modérée à rapide signifie 2 à 7 jours, lente signifie > 7 jours.

E.AZOULAY. REV MAL RESPIR 2008;25: 433-49

Tableau I.
Causes d'insuffisance respiratoire aiguë chez les patients souffrant de pathologies malignes.

Causes infectieuses	Causes non infectieuses
Infections bactériennes	Œdème pulmonaire cardiogénique
• Bactéries pyogènes	Syndrome de fuite capillaire
– Streptococcus pneumoniae	Infiltration pulmonaire spécifique
– Staphylococcus aureus	Toxicité médicamenteuse
– Haemophilus influenzae	Hémorragie alvéolaire
– Pseudomonas aeruginosa et Enterobactéries	Syndrome respiratoire aigu post-transfusionnel
• Bactéries intracellulaires	Pneumopathie radique
– Legionella pneumophilia	Protéinose alvéolaire
– Chlamydia et Mycoplasma pneumoniae	Dommage alvéolaire diffus
• Autres bactéries	Bronchiolite
– Actinomyces israeli,	Bronchiolite oblitérante avec pneumonie organisée
– Nocardia spp.	Second cancer
Pneumocystis jirovecii	
Cryptococcus neoformans	
Toxoplasmose	
Anguillulose	
Infections fongiques invasives	
• Champignons filamenteux	
– Aspergillus	
– Champignons émergents :	
– Trichosporose, Fusariose, Zygomycètes	
• Levures	
– Atteinte pulmonaire des candidémies	
• Infections fongiques endémiques	
– Histoplasmose, coccidioidomycose,	
– Blastomycose	
Infections virales (primo-infections ou réactivations)	
• Virus respiratoires saisonniers	
– Influenzae, parainfluenzae, rhinovirus	
– Virus respiratoire syncytial	
• Herpes virus	
– CMV, HSH, VZV, HHV6	
• Autres virus : adénovirus	
Infections mycobactériennes	
• Tuberculose et mycobactéries atypiques	

E.AZOULAY. REV MAL RESPIR 2008;25: 433-49

1ére ETAPE

- Éliminer OAP hémodynamique
  - Examens complémentaires inutile 10% des cas
  - Meilleur pronostic

- ATCDS/ SIGNES CLINIQUE/ ETT/ NT PROBNP

E.AZOULAY. REV MAL RESPIR 2008;25: 433-49

ORIGINE INFECTIEUSE SELON TYPE D'IMMUNOSUPPRESSION

Table 2 Nature of immune deficiencies and infections according to the diagnosis

Diagnosis	Deficiencies	Main infections
Acute myeloid leukemia	Phagocytosis	Bacteria
	Cell-mediated immunity	Yeasts
Acute lymphocytic leukemia	Phagocytosis	Bacteria
	Cell-mediated immunity	Yeasts, herpes viruses, *P. jirovecii*
Lymphomas	Cell-mediated immunity	*P. jirovecii* , yeasts, bacteria, encapsulated bacteria
Myelomas	Immunoglobulins	Encapsulated bacteria
Chronic lymphocytic leukemia	Phagocytosis	Encapsulated bacteria
	Cell-mediated immunity	Intracellular organisms
Chronic myeloid leukemia	Phagocytosis	Bacteria
Solid cancer	Compression, obstruction, ulceration	Bacteria
Bone marrow transplantation	Phagocytosis	Bacteria
	Cell-mediated immunity	Encapsulated bacteria
	Immunoglobulins	Yeasts, *P. jirovecii*
Associated condition	Asplenia in general associated with defect in immunoglobulins, altered phagocytosis and cell-mediated immunity	Encapsulated bacteria

Table 7 Noninvasive diagnostic investigations for cancer patients with acute respiratory failure

Radiography
Chest radiography
Thin-section high-resolution computed tomography
Echocardiography or pleural ultrasonography
Sputum
Bacteria
Tubercle bacillus
Fungi (aspergillus)
Tests for *Pneumocystis jirovecii* (MGG staining and immunofluorescence)
PCR for *Pneumocystis jirovecii*
Blood cultures
Serum tests
Serology: Chlamydia, Mycoplasma, Legionella
Herpes consensus PCR test
Circulating aspergillus antigen
Circulating cytomegalovirus antigen
Nasopharyngeal aspiration
Tests for viruses (PCR and immunofluorescence)
Urine tests
Cytology, bacteriology
Legionella antigen
Biological markers
Brain natriuretic peptide (BNP) or ProBNP
C reactive protein
Fibrin
Procalcitonin

Place du LBA

- Si outils non invasifs de diagnostic (antigénémie aspergillaire, antigénuries…) non contributifs.
  - Absence de diagnostic: surmortalité (OR 8,65)

- FB-LBA: diagnostic dans 55%des cas
  - seul examen à apporter un diagnostic dans 34 % des cas.
  - Arrêt de traitement dans un tiers des cas

- Meilleur rendement si précoce et avant toute antibiothérapie

E.Azoulay. CCM2008

Table 5 Diagnostic yield of bronchoalveolar lavage in hematology patients (*HM* hematological malignancy)

Reference	*n*	Diagnosis	Diagnostic impact	Therapeutic impact
Stover et al. [96]	97	HM	66	–
Martin et al. [142]	100	HM	30	–
Xaubet et al. [143]	96	HM	49	31
Campbell et al. [144]	22	HM	55	–
Pisani et al. [145]	150	HM	39	–
Maschmeyer et al. [146]	46	Neutropenia	30	–
Cordonnier et al. [100]	56	Neutropenia	53	24
Cazzadori et al. [147]	142	HM	36	–
Von Eiff et al. [40]	90	HM	66	65
White et al. [3]	68	HM	31	24
Ewig et al. [28]	49	HM	31	16
Gruson et al. [18]	41	Neutropenia	63	28
Hilbert et al. [22]	24/46	HM	62	71
Murray et al. [2]	27	HM	33	28
Azoulay et al. [4]	203	HM	49.5	45.1
Pagano et al. [148]	127	HM	53	14
Jain et al. [82]	104	HM	56	–
Hohenadel et al. [81]	95	HM	30	–
Total	1537		46.2	34.6

Table 6 Diagnostic yield of bronchoalveolar lavage in bone marrow transplant recipients (*auto* autologous bone marrow transplantation, *allo* allogeneic bone marrow transplantation)

Author	*n*	Type of patients	Diagnostic impact	Therapeutic impact	Complications
Springmeyer et al. [20]	22	Auto-allo	58	–	13
Cordonnier et al. [17]	52	Allo	50	–	0
Cordonnier et al. [9]	69	Allo	66	–	–
Milburn et al. [19]	40	Allo	80	76	0
Springmeyer [78]	15	Auto-allo	89	–	40
Heurlin et al. [149]	18	Auto-allo	61	–	–
Weiss et al. [80]	47	Auto-allo	47	–	12
Campbell et al. [79]	27	–	74	63	11
AbuFarsakh et al. [150]	77	Auto-allo	42	–	–
White et al. [93]	68	Auto-allo	31	24	15 (7% MV)
Dunagan et al. [1][a]	71	Auto-allo	38	42	27 (4% MV)
Glazer et al. [151]	79	Auto-allo	67	62	–
Gruson et al. [39]	38	Auto-allo	42	–	–
Gruson et al. [18]	52	Auto-allo	38	28	17
Huaringa et al. [127]	89	Auto-allo	42	–	–
Total	764	Auto-allo	55	49	0 to 40%

[a] 32% mechanical ventilation

- **Hantise:**
  - Aggravation clinique et gazométrique après fibroscopie (10 à 40% dans la littérature)
- Étude au laboratoire d'endoscopie bronchique:
  - Baisse de la PaO2 d'environ 10 mmHg après FB
  - Baisse jusqu'à 23 mmHg après un LBA sans complications
  - Persistance de la dégradation de l'hématose pendant 15 à 24 heures
- **FB/LBA:** classiquement non recommandée en cas d'hypoxémie profonde (PO2/FiO2<75 mmHg en ventilation spontanée)

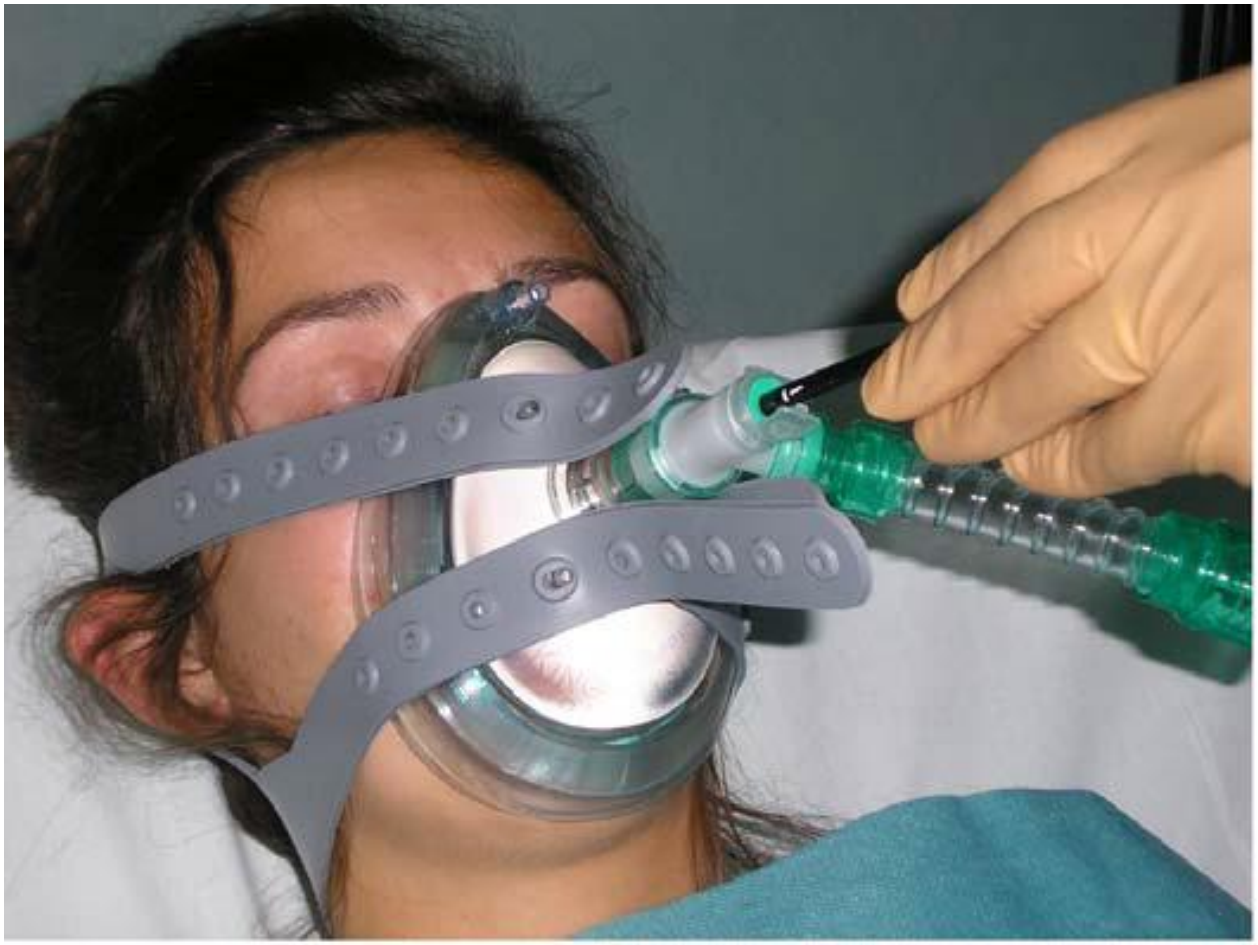


Figure 1: Réalisation pratique d'une fibroscopie chez un sujet hypoxémique non intubé en réanimation sous VNI

Diagnostic Strategy for Hematology and Oncology Patients with Acute Respiratory Failure

Randomized Controlled Trial

Am J Respir Crit Care Med Vol 182. pp 1038-1046, 2010

Élie Azoulay[1], Djamel Mokart[2], Jérôme Lambert[3], Virginie Lemiale[4], Antoine Rabbat[5], Achille Kouatchet[6], François Vincent[7], Didier Gruson[8], Fabrice Bruneel[9], Géraldine Epinette-Branche[1], Ariane Lafabrie[1], Rebecca Hamidfar-Roy[10], Christophe Cracco[11], Benoît Renard[12], Jean-Marie Tonnelier[13], François Blot[14], Sylvie Chevret[3], and Benoît Schlemmer[1]

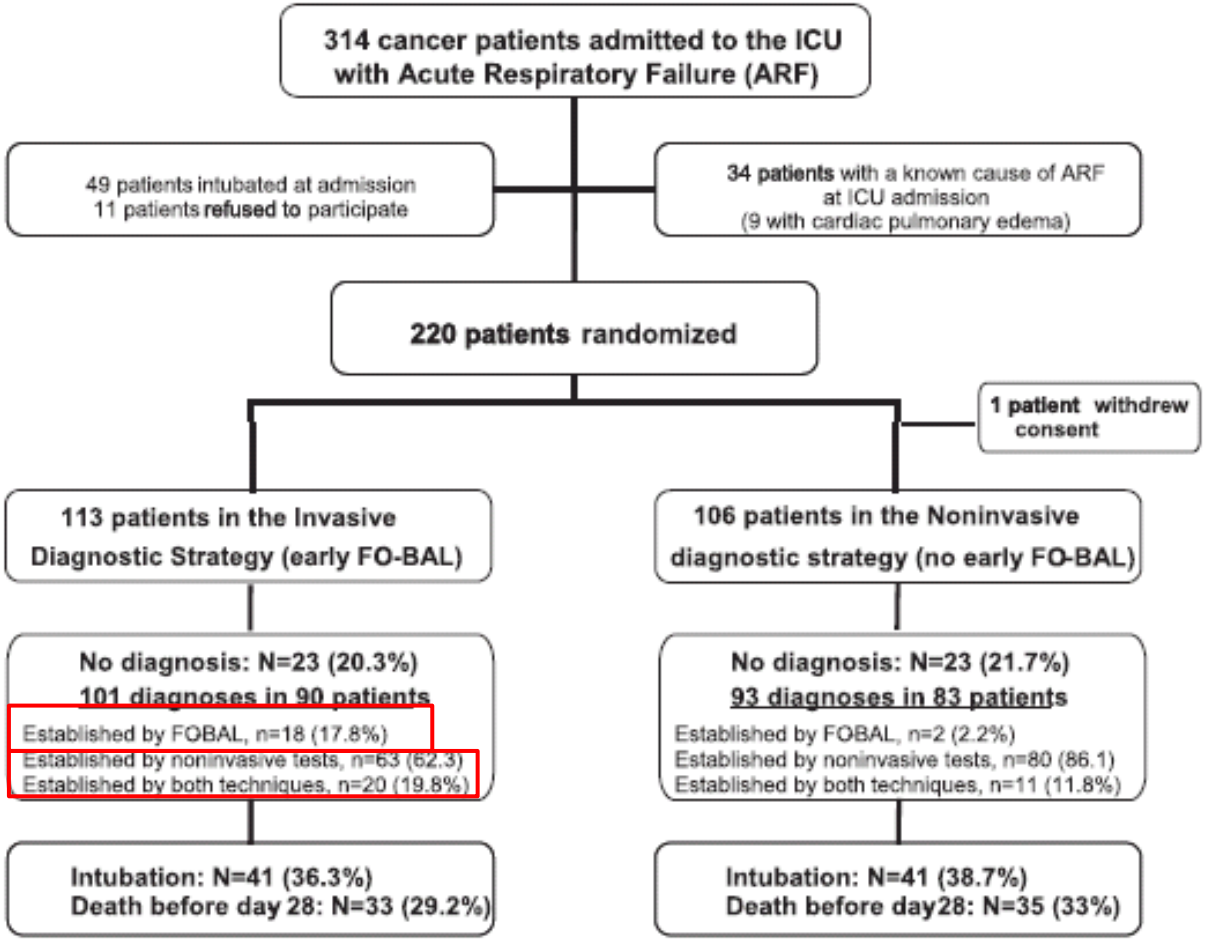


TABLE 2. CAUSES OF ACUTE RESPIRATORY FAILURE IN THE TWO DIAGNOSTIC-STRATEGY GROUPS

Cause	Routine Day 1 FO-BAL, N = 113 (%)	N (%) Diagnosed by BAL	± Day 3 FO-BAL, N = 106 (%)	N (%) diagnosed by BAL
Bacterial pneumonia	47 (41.6)		39 (36.8)	
Microbiologically documented	32 (68)	17 (36.2)	27 (70)	4 (10.2)
Clinically documented	15 (32)	—	12 (30)	—
Viral pneumonia	7 (6.2)	6 (86)	19 (17.9)	10 (55.5)
Invasive yeast and mold infections	14 (12.4)	7 (50)	9 (8.5)	3 (33.3)
Including invasive pulmonary aspergillosis only	10 (8.8)	6 (60)	8 (7.5)	3 (37)
Pneumocystis pneumonia*	9 (8)	9 (100)	10 (9.4)	1 (10)
Pulmonary infiltration by the malignancy	10 (8.8)	3 (30)	6 (5.7)	0
Cardiogenic pulmonary edema	7 (6.2)	0	3 (2.8)	0
Respiratory failure during neutropenia recovery	2 (1.8)	0	1 (0.9)	0
Pulmonary toxoplasmosis	1 (0.9)	1 (100)	1 (0.9)	0
Pulmonary tuberculosis	0		1 (0.9)	0
Cryptogenic organizing pneumonia	1 (0.9)	0	1 (0.9)	0
Idiopathic alveolar hemorrhage	1 (0.9)	1 (100)	0	
Miscellaneous†	2 (1.8)	0	3 (2.8)	0
Total number of identified causes	101		93	
Patients with two identified causes	7 (6.2)		8 (7.5)	
Patients with three identified causes	2 (1.8)		1 (0.9)	
No identified cause	23 (20.3)		23 (21.7)	

Definition of abbreviation: FO-BAL = fiberoptic bronchoscopy with bronchoalveolar lavage. Definition of cardiac dysfunction is detailed in the online supplement.

* Although polymerase chain reaction testing was positive in all cases of *Pneumocystis* pneumonia, retrieval of the pathogen in induced sputum or BAL fluid was required for the diagnosis.

† The five diagnoses were drug-related pulmonary toxicity (n = 2); alveolar proteinosis (n = 1); nontumoral eosinophilic pneumonia (n = 1); and capillary leak syndrome (n = 1).

Am J Respir Crit Care Med Vol 182. pp 1038-1046, 2010

TABLE 3. OUTCOMES AND ICU MANAGEMENT IN THE TWO DIAGNOSTIC-STRATEGY GROUPS

	Routine Day 1 FO-BAL (*n* = 113)	Noninvasive Group (*n* = 106)	
Primary end point			*P* Value
Need for endotracheal mechanical ventilation, pts	40	41	0.68
Estimated rate (95% CI)	35.4% (26.6–45)	38.7% (29.4–48.7)	OR = 0.87 (0.50–1.50) Adjusted OR* = 1.11 (0.58–2.11); *P* = 0.76
Major secondary end point			
Number of patients with no identified diagnosis	23	23	Between-group risk difference (90% CI), %
Estimated rate (95% CI)	20.4% (13.4–29)	21.7% (14.3–30.8)	−1.3 (−10.4 to 7.7)
Other secondary end points			*P* value
Day 28 deaths	33	35	
Estimated rate (95% CI)	29.2% (21–38.5)	33% (24.2–42.8)	0.56
Median number of antibiotic-free days (Q1–Q3)	2 (0–2)	2 (0–3)	0.78
ICU-acquired infections	9	13	
Estimated rate (95% CI)	8% (3.7–14.6)	12.2% (6.7–20.1)	0.37
Acquisition of multiresistant bacteria	6	10	
Estimated rate (95% CI)	25.3% (2–11.2)	9.4% (4.6–16.7)	0.30

Conclusions

In hematology and oncology patients with early hypoxemic ARF, FO-BAL is safe when performed in the ICU. Non-invasive diagnostic tests provide the diagnosis in most of these patients. Because 18% of patient diagnoses are made only by FOB-BAL, this procedure should be added to noninvasive tests if feasible early after ICU admission.

Am J Respir Crit Care Med Vol 182. pp 1038-1046, 2010

ETIOLOGIES EMERGENTES:

- Pneumopathie de lyse tumorale:
  - Atteinte alvéolaire diffuse responsable d'une IRA survenant avec l'initiation d'une chimiothérapie pour une leucémie aiguë (10 heures à 20 jours après le début)
  - peut se compliquer d'hémorragie alvéolaire diffuse.
  - Traitement: celui de tout SDRA.

- Leucostase pulmonaire:
  - SDRA associé à une atteinte neurologique
  - Hyperleucostase > 100.000/mm3
  - Traitement: leukaphérèses, support ventilatoire et chimiothérapie ou corticothérapie

PRISE EN CHARGE SYMPTOMATIQUE

VM 75.1%

VNI 51.4%

Authors	Year	Journal	Number of patients	Hospital Mortality
Kress	1999	AJRCCM	153 MV	67%/
Hilbert	2000	CCM	/	/
			64 NIMV	68%
Hilbert	2001	NEJM	14 MV	93 %
			8 NIMV	53 %
Azoulay	1999	ICM	46 MV	78 %
			9 NIMV	22 %
Azoulay	2001	CCM	189 MV	70.8 %
			48 NIMV	48.7 %
Larché	2003	ICM	68 MV	79.4 %
			12 NIMV	75 %
Khassawneh	2002	Chest	78 MV	75%
Darmon	2002	ICM	/	/
			49 MV	71.4 %
			42 NIMV	45 %
Kroschinsky	2002	ICM	54 MV	74 %
Benoit	2003	CCM	/	/
			87 MV	67.8 %
Massion	2002	CCM	/	/
			48 MV	75 %
Azoulay	2004	Medicine	/	/
			114 MV	75 %
			79 NIMV	48 %

Intensive Care Med (2000) 26: 1817–1823

ORIGINAL

E. Azoulay
D. Moreau
C. Alberti
G. Leleu
C. Adrie
M. Barboteu
P. Cottu
V. Levy
J.-R. Le Gall
B. Schlemmer

Predictors of short-term mortality in critically ill patients with solid malignancies

Table 4 Multivariate analysis: independent predictors of 30-day mortality; goodness of fit (Hossmer-Lemeshow) $p > 0.05$ (χ^2)

Parameters	Odds ratio	95% CI
Mechanical ventilation	3.55	1.26–6.7
LOD	1.26	1.09–1.44
Surgical treatment of the cancer	0.20	0.07–0.58

Intensive Care Med (1999) 25: 1395–1401

ORIGINAL

E. Azoulay
C. Recher
C. Alberti
L. Soufir
G. Leleu
J. R. Le Gall
J. P. Fermand
B. Schlemmer

Changing use of intensive care for hematological patients:

the example of multiple myeloma

Table 3 Comparison between patients admitted to our ICU before versus after 1996

	1992–1995 41 patients (55 %)	1996–1998 34 patients (45 %)	P value
Knaus scale C or D	26 (66.5 %)	13 (38.2 %)	0.02
Chronic renal failure	3 (7.5 %)	14 (41 %)	0.0009
Stage III disease	34 (83 %)	21 (62 %)	0.03
Bone marrow transplantation	14 (34 %)	14 (41 %)	0.25
Remission	16 (40 %)	11 (32 %)	0.76
Quantification of the monoclonal immunoglobulin (G/L)	42.5 ± 19	19.5 ± 16	0.008
Quantification of the urinary Bence Jones protein excretion	0.8 ± 0.4	2.28 ± 0.8	0.11
Mean SAPS II	54 ± 18	64 ± 22	0.055
ICU admission for acute respiratory failure	21 (51.2 %)	18 (53 %)	1
Mechanical ventilation	28 (68 %)	18 (53 %)	0.17
Noninvasive mechanical ventilation	2 (5 %)	7 (20.6 %)	0.03
ICU admission for shock	19 (46.3 %)	12 (35 %)	0.35
Need for vasopressors	31 (75.6 %)	19 (56 %)	0.07
Median duration of vasopressor use (days)	5 (0–11)	3 (0–7.5)	0.16
Dialysis	9 (22 %)	15 (44 %)	0.04
30-day outcome: number of deaths	31 (75.6 %)	12 (35 %)	0.0008

Table 2 Multivariate analysis

A: Results of the stepwise logistic regression model			
Parameters	Odds ratio	95 % CI	P value
Female gender	5.12	[1.20–29.1]	0.04
Need for vasopressor or inotropic agents	5.7	[1.2–30]	0.03
Need for mechanical ventilation	16.7	[3.9–96.7]	0.0005
ICU admission after 1995	0.09	[0.02–0.40]	0.002
Remission	0.16	[0.03–0.80]	0.03
Goodness of fit (Hosmer-Lemeshow) chi-square P-value > 0.05			

British Journal of Anaesthesia **108** (3): 452–9 (2012)
Advance Access publication 31 January 2012 · doi:10.1093/bja/aer449

BJA

CRITICAL CARE

Outcomes and prognostic factors in patients with haematological malignancy admitted to a specialist cancer intensive care unit: a 5 yr study

G. T. Bird[1], P. Farquhar-Smith[1], T. Wigmore[1], M. Potter[2] and P. C. Gruber[1]*

Table 4 Variables predictive of in-hospital mortality on multivariate analysis

Variable	Odds ratio	95% confidence interval
Invasive mechanical ventilation	3.03	1.33–6.90
Failure of ≥2 organ systems	5.62	2.30–13.70

Intensive Care Med (1998) 24: 1283–1288

ORIGINAL

G. Conti
P. Marino
A. Cogliati
D. Dell'Utri
A. Lappa
G. Rosa
A. Gasparetto

Noninvasive ventilation for the treatment of acute respiratory failure in patients with hematologic malignancies: a pilot study

Table 2 Changes in arterial blood gases, PaO_2/FIO_2, respiratory rate *RR*, mean arterial pressure *MAP* and heart rate *HR* during the study. Values are mean ± SD (*T0* baseline, *T1* after 1 h, *T2* after 3 h, *T3* after 12 h, *T4* after 24 h)

	T0	T1	T2	T3	T4
pH	7.5 ± 0.04	7.43 ± 0.03	7.44 ± 0.04	7.44 ± 0.03	7.40 ± 0.01
PaO_2 (mmHg)	43 ± 10	88 ± 37	91 ± 25	96 ± 26	90 ± 26*
$PaCO_2$ (mmHg)	32 ± 4	35 ± 8	35 ± 4	33 ± 3	38 ± 7
SaO_2 (%)	81 ± 9	95 ± 4	96 ± 3	97 ± 2	96 ± 4*
PaO_2/FIO_2 (breaths/min)	87 ± 22	175 ± 60	203 ± 91	210 ± 90	185 ± 84*
RR	42 ± 7	34 ± 10	34 ± 10	33 ± 8	33 ± 7
MAP (mmHg)	89 ± 11	78 ± 11	81 ± 11	85 ± 15	75 ± 12
HR (beats/min)	112 ± 17	102 ± 14	97 ± 10	99 ± 16	96 ± 22

* $p < 0.01$

NONINVASIVE VENTILATION IN IMMUNOSUPPRESSED PATIENTS WITH PULMONARY INFILTRATES, FEVER, AND ACUTE RESPIRATORY FAILURE

GILLES HILBERT, M.D., DIDIER GRUSON, M.D., FRÉDERIC VARGAS, M.D., RUDDY VALENTINO, M.D., GEORGES GBIKPI-BENISSAN, M.D., MICHEL DUPON, M.D., JOSY REIFFERS, M.D., AND JEAN P. CARDINAUD, M.D.

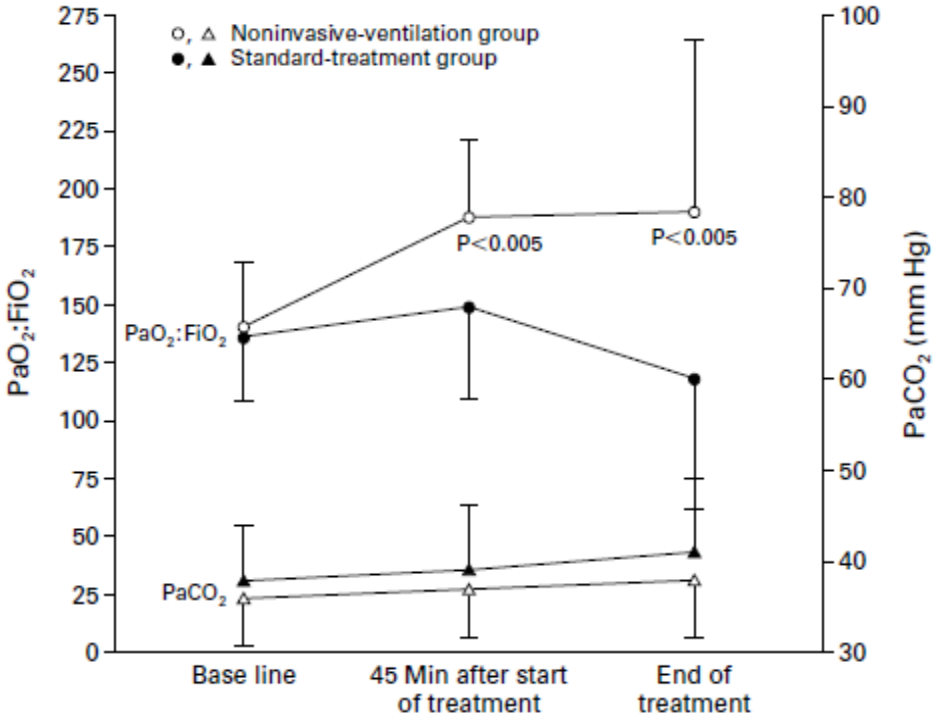


Figure 1. Mean (±SD) Changes in the Ratio of the Partial Pressure of Arterial Oxygen (PaO_2) to the Fraction of Inspired Oxygen (FiO_2) and in the Partial Pressure of Arterial Carbon Dioxide ($PaCO_2$) over Time in the 26 Patients in the Noninvasive-Ventilation Group and the 26 in the Standard-Treatment Group.

The "end of treatment" refers to the last arterial blood gas value obtained before the patient was either intubated or discharged from the intensive care unit. P values are for the comparisons with base-line values.

Improved survival in cancer patients requiring mechanical ventilatory support: Impact of noninvasive mechanical ventilatory support

Elie Azoulay, MD; Corinne Alberti, MD; Caroline Bornstain, MD; Ghislaine Leleu, MD; Delphine Moreau, MD; Christian Recher, MD; Sylvie Chevret, MD, PhD; Jean-Roger Le Gall, MD; Laurent Brochard, MD, PhD; Benoît Schlemmer, MD

Table 3. Multivariable analysis: Additive predictors of 30-day mortality

Variables	Odds ratio	95% CI	*p* Value
Noninvasive mechanical ventilation	0.343	0.16–0.73	<.0001
ICU admission between 1996 and 1998	0.24	0.12–0.50	<.0001
SAPS II score (per point)	1.04	1.02–1.06	<.0001

CI, confidence interval; ICU, intensive care unit; SAPS, simplified acute physiology score.
Goodness-of-fit (Hosmer-Lemeshow) chi-square *p* Value = .688.

(Crit Care Med 2001; 29:519–525)

MODALITES:

- **Ventilateur:** ventilateur de réanimation, ou appareil dédié à la VNI

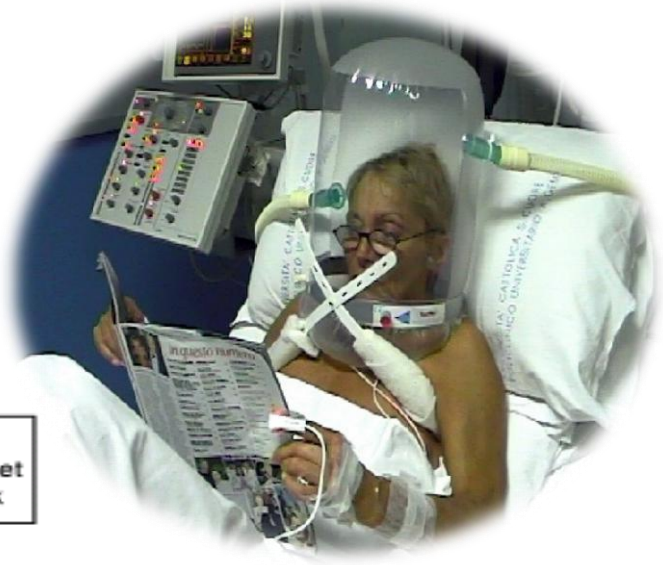
  (disposant d'un mélangeur d'oxygène pour délivrer des FiO2 élevées).
- **Modes ventilatoires :** mode en Pression (AI ou BIPAP)
- **Interfaces :** masque facial ou bucconasal recommandé en première intention

Intensive Care Med (2004) 30:147–150
DOI 10.1007/s00134-003-2056-9

BRIEF REPORT

Tiziana Principi
Simona Pantanetti
Francesca Catani
Daniele Elisei
Vincenzo Gabbanelli
Paolo Pelaia
Pietro Leoni

Noninvasive continuous positive airway pressure delivered by helmet in hematological malignancy patients with hypoxemic acute respiratory failure



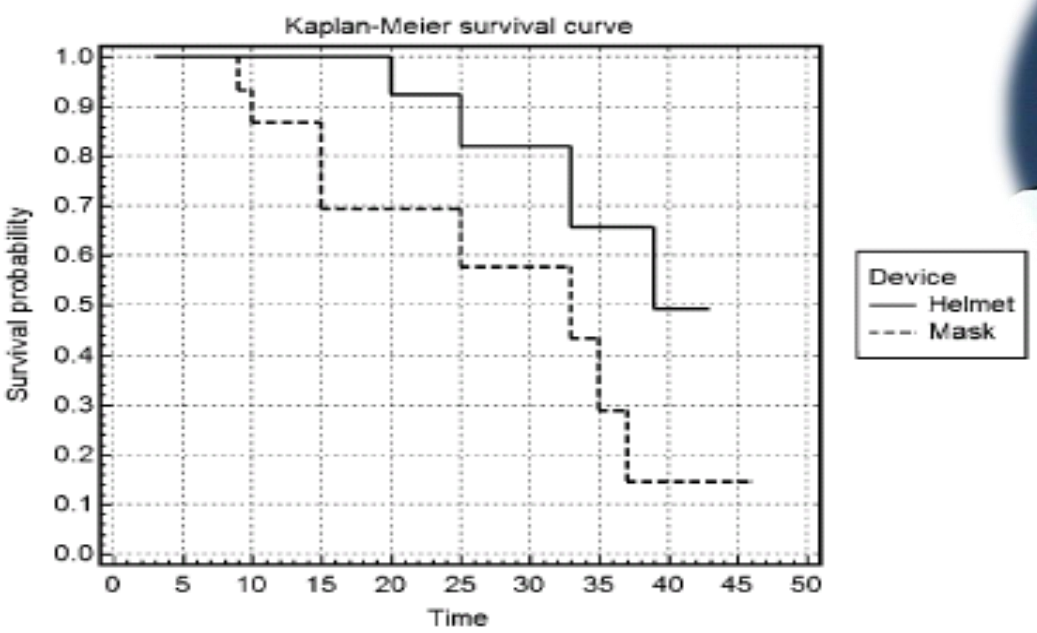


Fig. 1 Death rate assessed by Kaplan-Meier analysis. Four patients in the helmet group (23%) vs. eight (47%) in the face mask group died in the Hematology Department, representing an apparent risk of death reduction of 49%. Hospital mortality in the helmet group was significantly lower than that in the face mask group (Mantel-Cox log-rank test, $p<0.05$)

Intensive Care Med (2010) 36:1666–1674
DOI 10.1007/s00134-010-1934-1

ORIGINAL

Vincenzo Squadrone
Massimo Massaia
Benedetto Bruno
Filippo Marmont
Michele Falda
Carlotta Bagna
Stefania Bertone
Claudia Filippini
Arthur S. Slutsky
Umberto Vitolo
Mario Boccadoro
V. Marco Ranieri

Early CPAP prevents evolution of acute lung injury in patients with hematologic malignancy

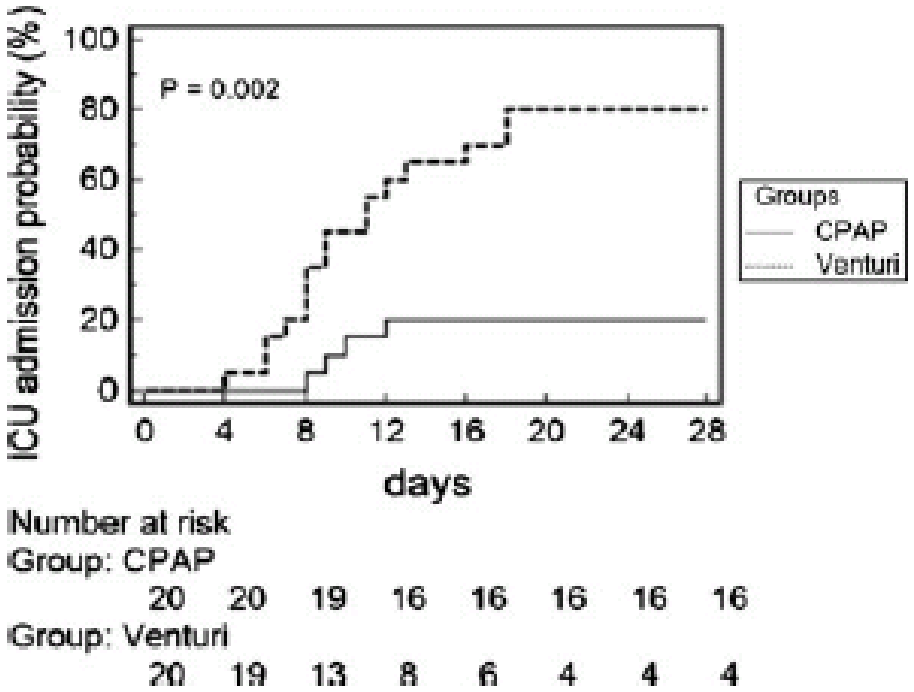
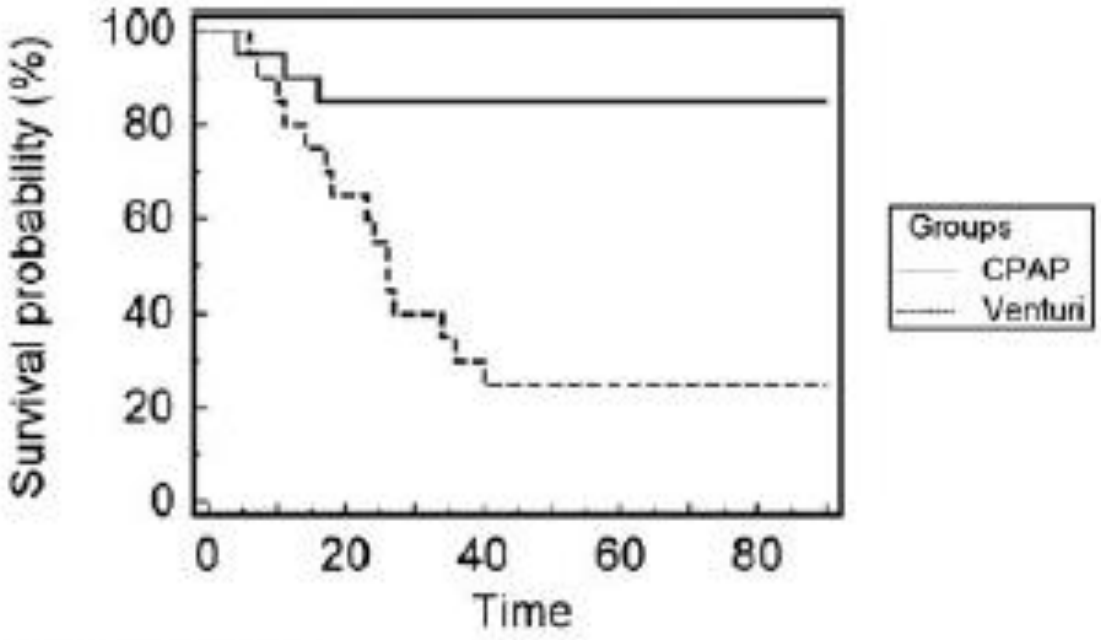


Fig. 2 Kaplan–Meier curves of development of acute lung injury requiring ventilatory management and ICU admission

Table 3 Study outcome variables

	Control ($n = 20$)	CPAP ($n = 20$)	Relative risk (95% CI)	P value
Intubation and invasive ventilation at ICU entry (no.)	8	2	0.5 (0.29–0.85)	0.03
Noninvasive ventilation at ICU entry (no.)	8	2	0.5 (0.29–0.85)	0.03
Failure on noninvasive ventilation requiring intubation (no.)	5	0	0.42 (0.29–0.63)	0.017

ICU intensive care unit, *CPAP* continuous positive airway pressure



Number at risk

Group: CPAP										
	20	19	17	17	17	17	17	17	17	17
Group: Venturi										
	20	17	13	8	5	5	5	5	5	5

Fig. 3 Kaplan–Meier curves of hospital mortality

High-Flow Nasal Cannula Oxygenation in Immunocompromised Patients With Acute Hypoxemic Respiratory Failure: A Groupe de Recherche Respiratoire en Réanimation Onco-Hématologique Study

Virginie Lemiale, MD[1]; Matthieu Resche-Rigon, MD[2]; Djamel Mokart, MD[3]; Frédéric Pène, MD, PhD[4]; Laurent Argaud, MD[5]; Julien Mayaux, MD[6]; Christophe Guitton, MD[7]; Antoine Rabbat, MD[8];

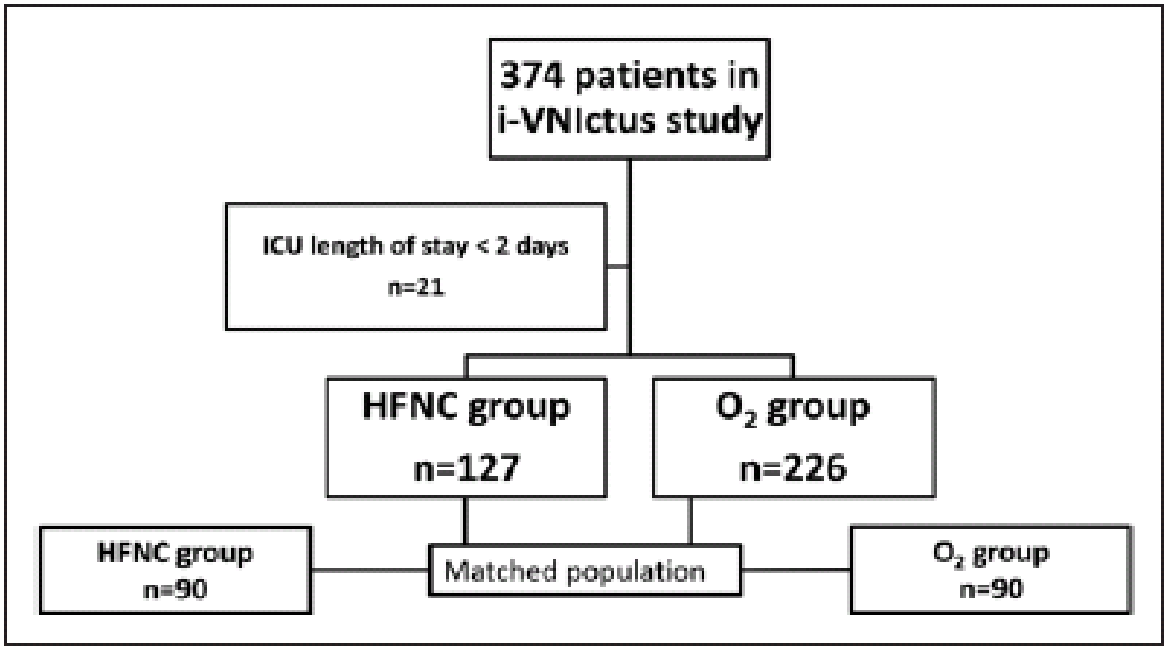


Figure 1. Patient diagram. HFNC = high-flow nasal cannula. O_2 = oxygen.

(*Crit Care Med* 2017; 45:e274–e280)

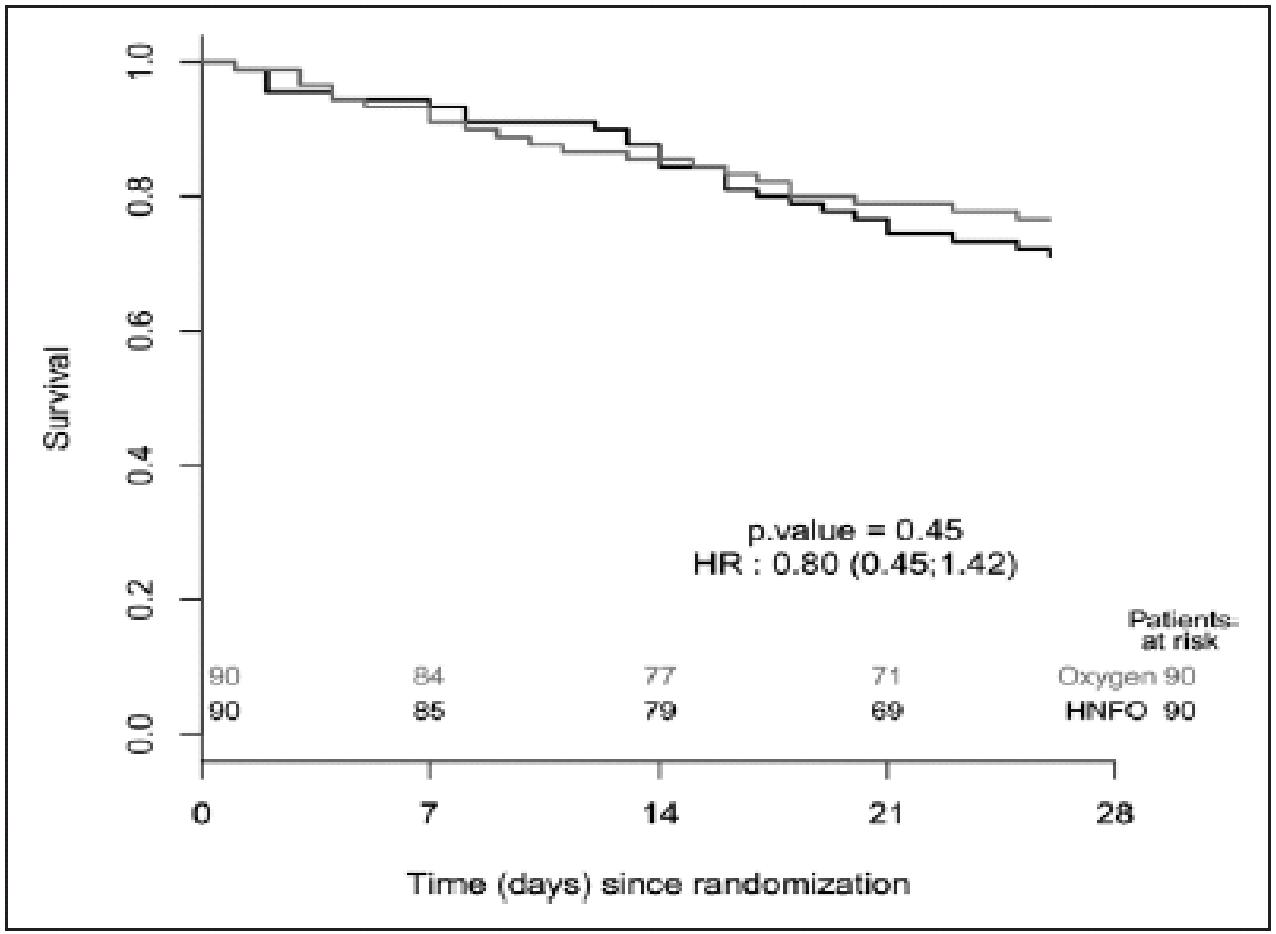


Figure 2. Probability of survival of the risk of day-28 mortality. Kaplan-Meier estimates of the probability of day-28 mortality in immunocompromised patients with acute respiratory failure receiving either high-flow nasal oxygen (HFNO) or oxygen. HFNO (*black line*), oxygen group (*gray line*).

Wang et al. BMC Pulmonary Medicine (2016) 16:129
DOI 10.1186/s12890-016-0289-y

BMC Pulmonary Medicine

RESEARCH ARTICLE Open Access

Noninvasive versus invasive mechanical ventilation for immunocompromised patients with acute respiratory failure: a systematic review and meta-analysis

Tao Wang[1†], Lixi Zhang[2†], Kai Luo[3], Jianqiang He[1], Yong Ma[1], Zongru Li[4], Na Zhao[5], Qun Xu[3], Yi Li[1*] and Xuezhong Yu[1*]

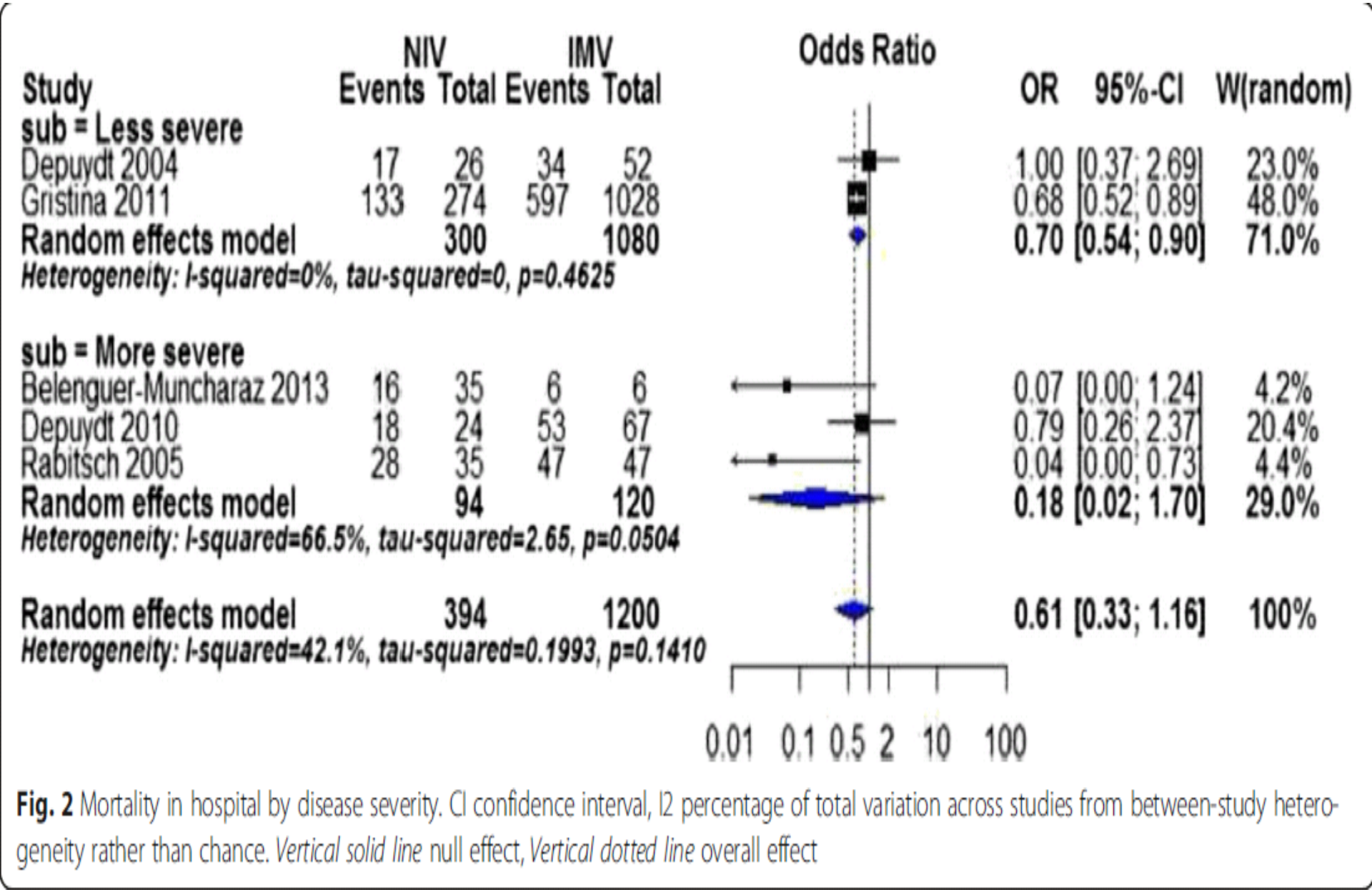


Fig. 2 Mortality in hospital by disease severity. CI confidence interval, I2 percentage of total variation across studies from between-study heterogeneity rather than chance. *Vertical solid line* null effect, *Vertical dotted line* overall effect

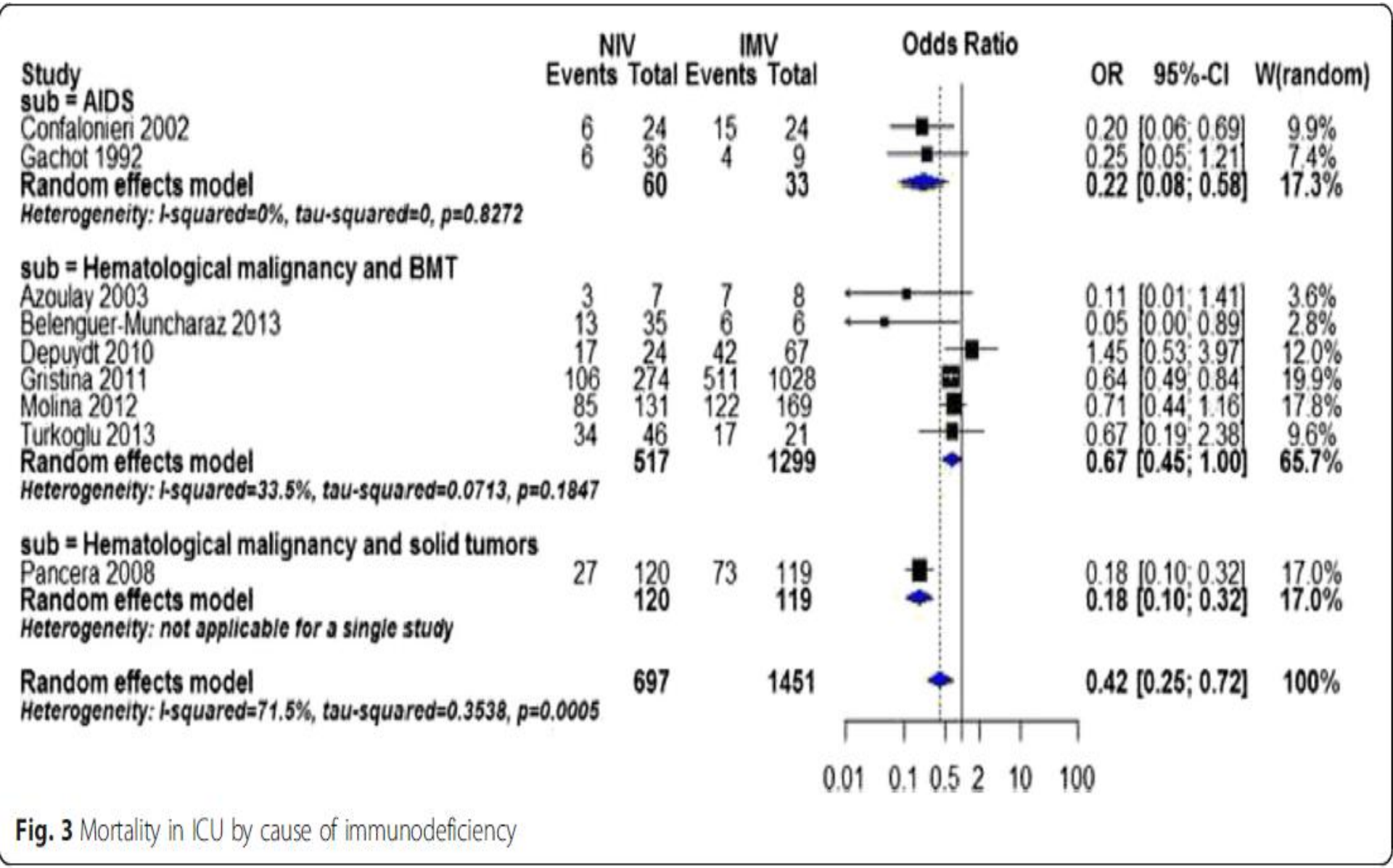


Fig. 3 Mortality in ICU by cause of immunodeficiency

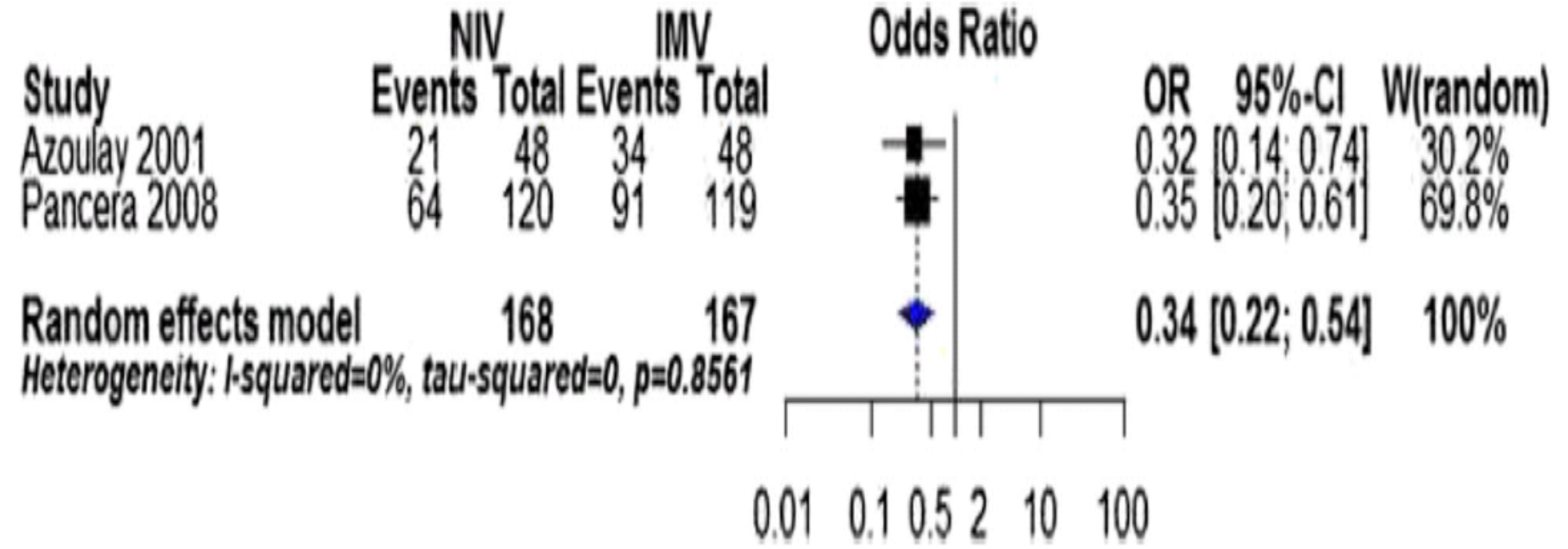


Fig. 4 30-day mortality

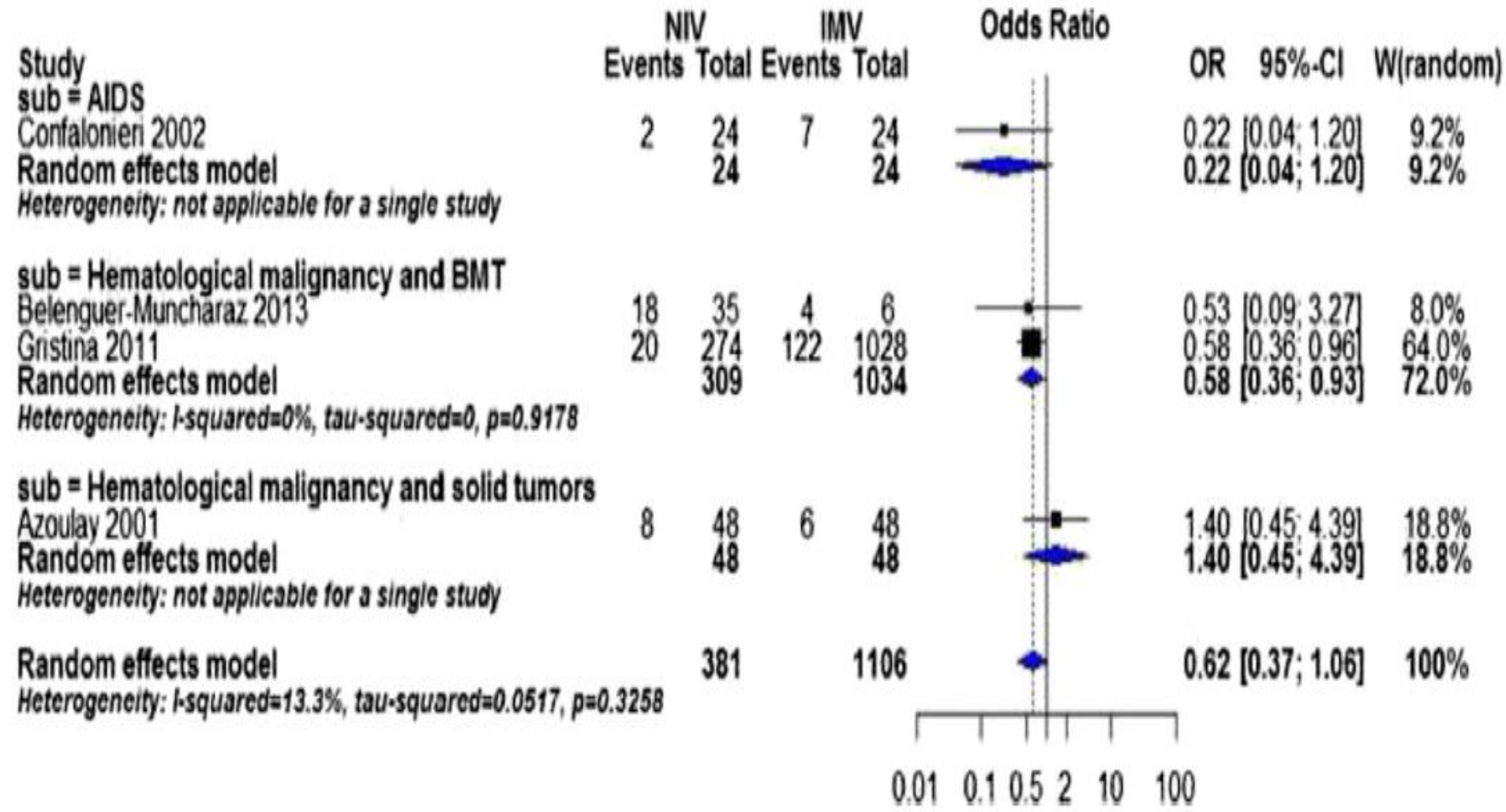


Fig. 5 Nosocomial infections by cause of immunodeficiency

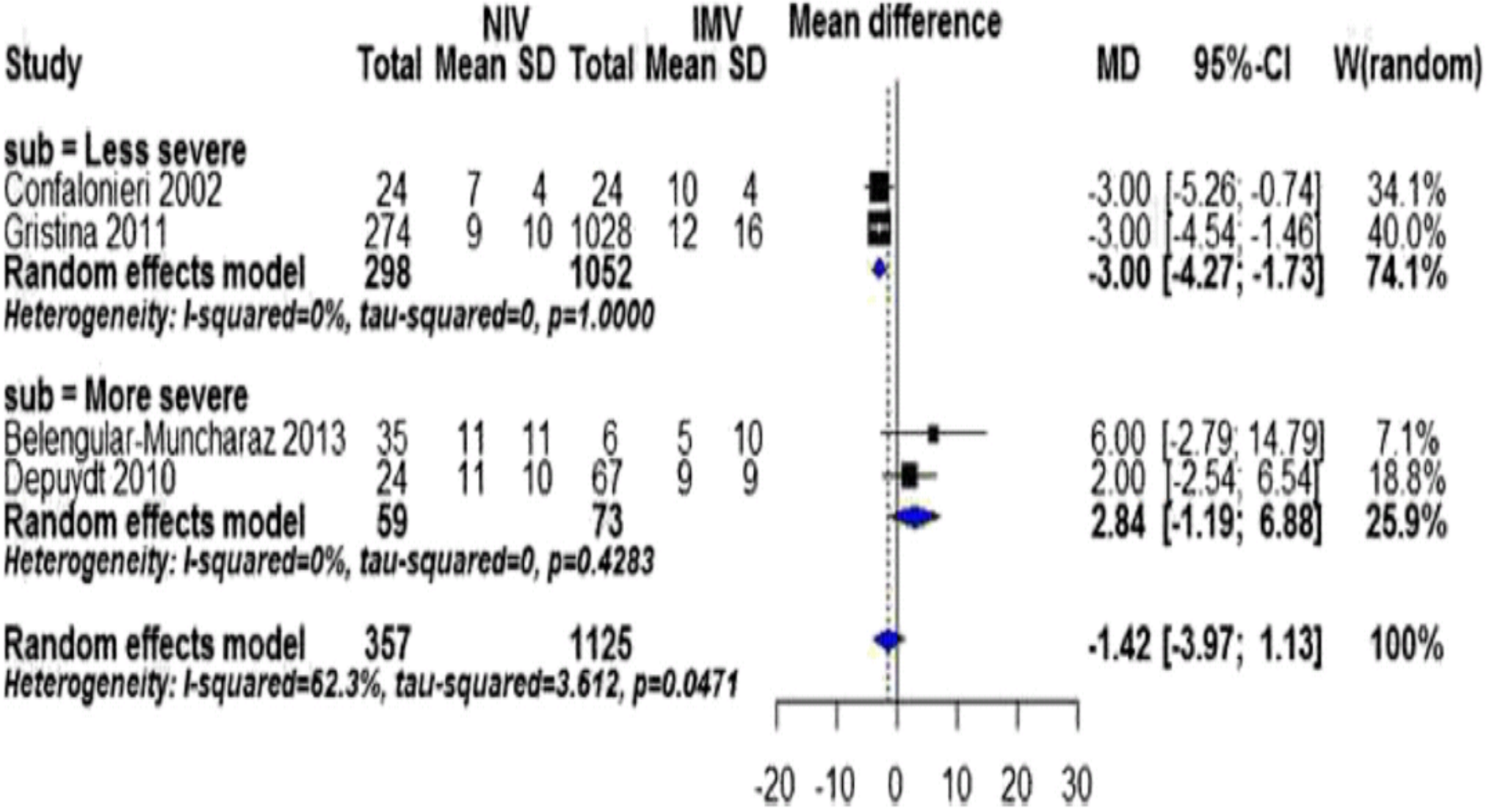


Fig. 6 Duration of ICU stay by disease severity. SD standard deviation

Intensive Care Med
DOI 10.1007/s00134-017-4947-1

SEVEN-DAY PROFILE PUBLICATION

Acute hypoxemic respiratory failure in immunocompromised patients: the Efraim multinational prospective cohort study

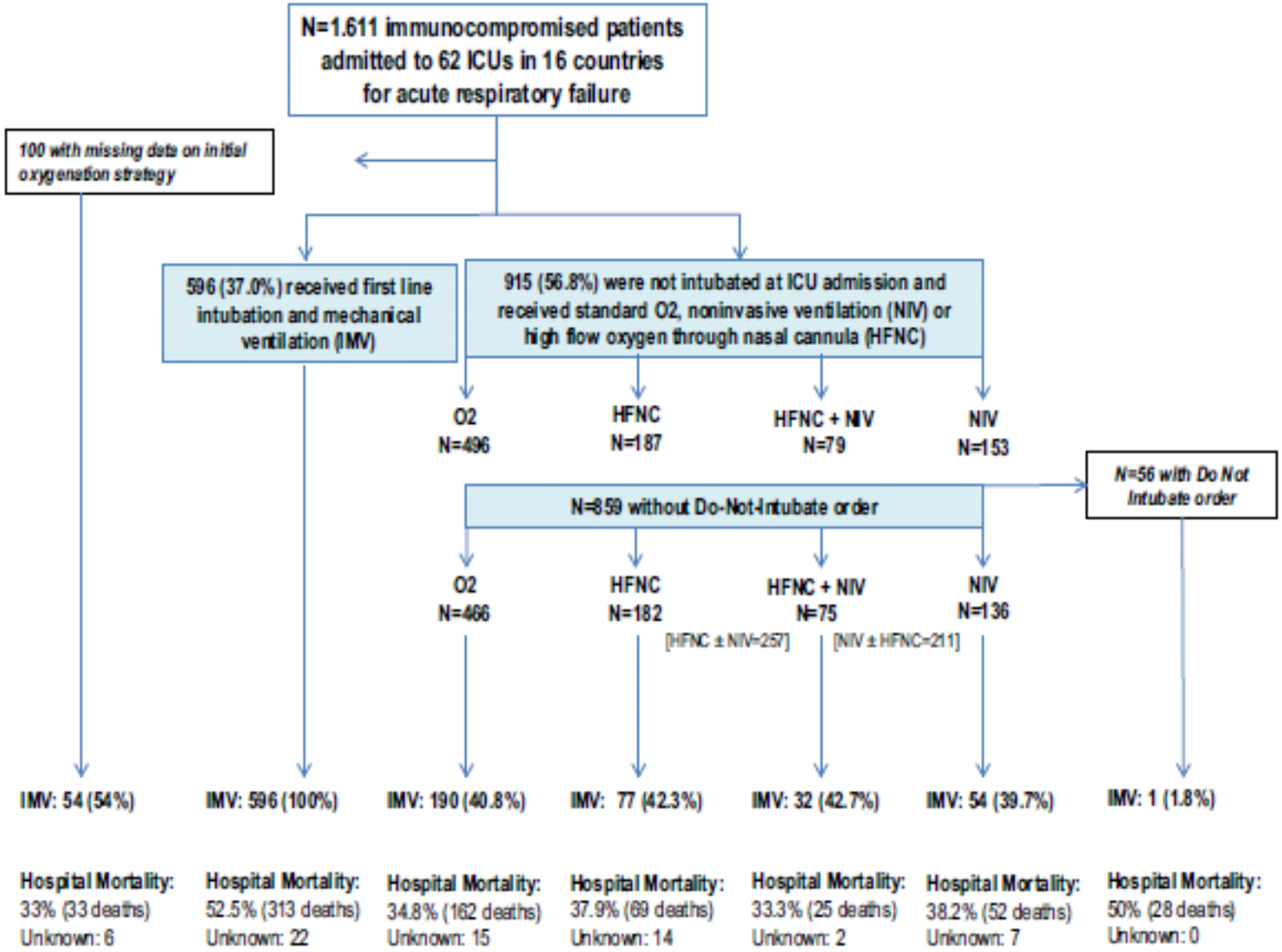
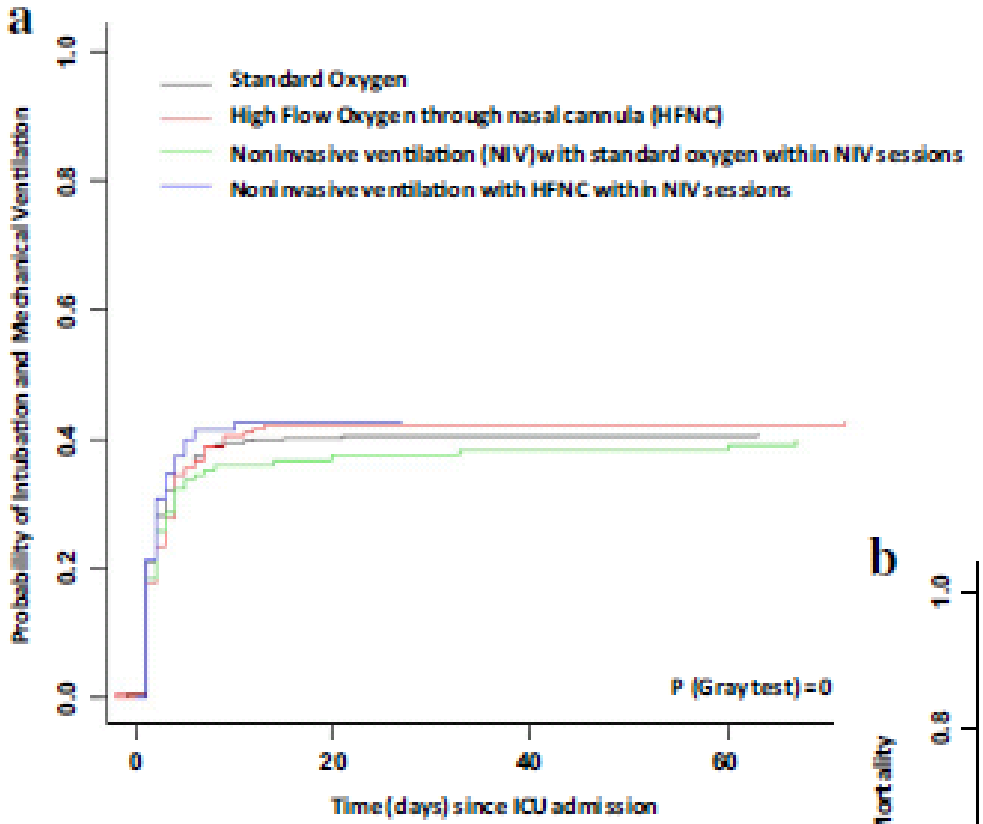
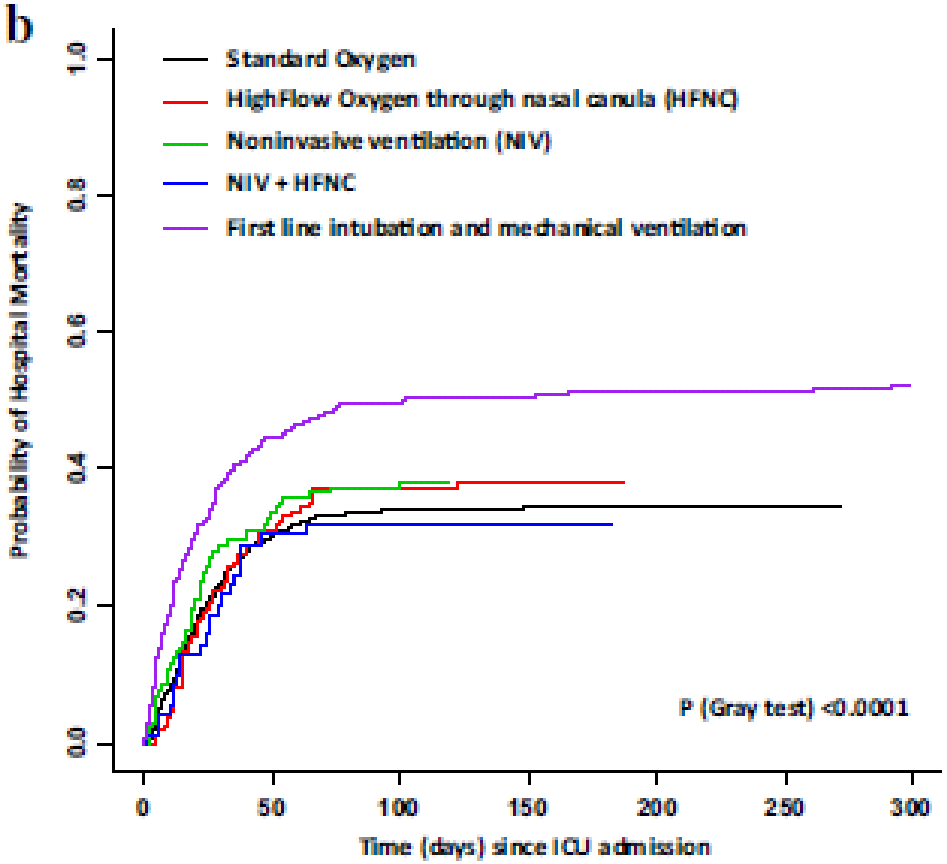
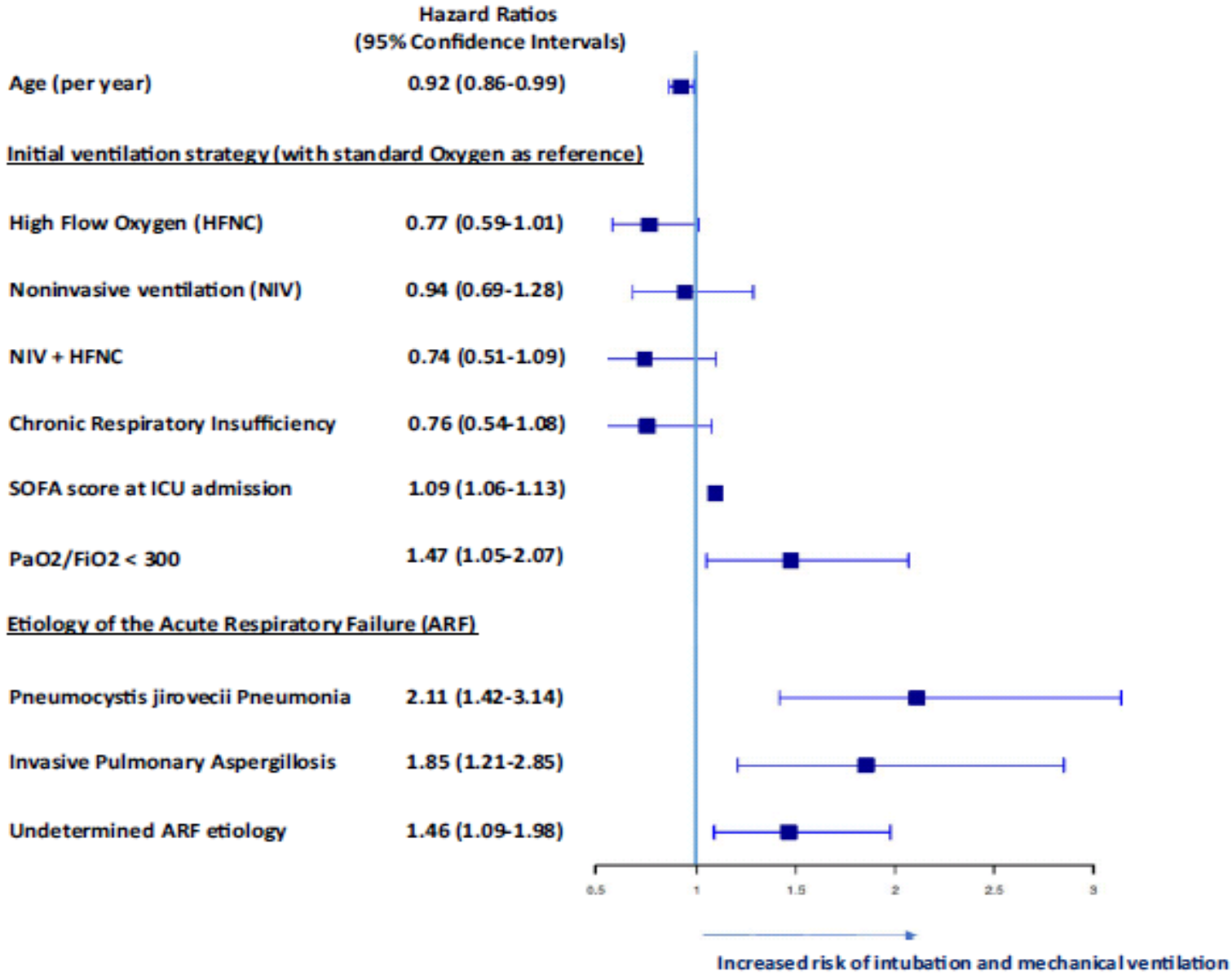


Fig. 1 Study flowchart


a
Probability of Intubation and Mechanical Ventilation
1.0
0.8
0.6
0.4
0.2
0.0
Standard Oxygen
High Flow Oxygen through nasal cannula (HFNC)
Noninvasive ventilation (NIV) with standard oxygen within NIV sessions
Noninvasive ventilation with HFNC within NIV sessions
P (Gray test) = 0
0
20
40
60
Time (days) since ICU admission


b
Probability of Hospital Mortality
1.0
0.8
0.6
0.4
0.2
0.0
Standard Oxygen
HighFlow Oxygen through nasal canula (HFNC)
Noninvasive ventilation (NIV)
NIV + HFNC
First line intubation and mechanical ventilation
P (Gray test) <0.0001
0
50
100
150
200
250
300
Time (days) since ICU admission



g. 3 Multivariate model of the cause-specific hazard of intubation. This analysis is restricted to the 915 patients not intubated on ICU admission. ots report variables independently associated with the need for intubation in the final model, with their 95% confidence intervals

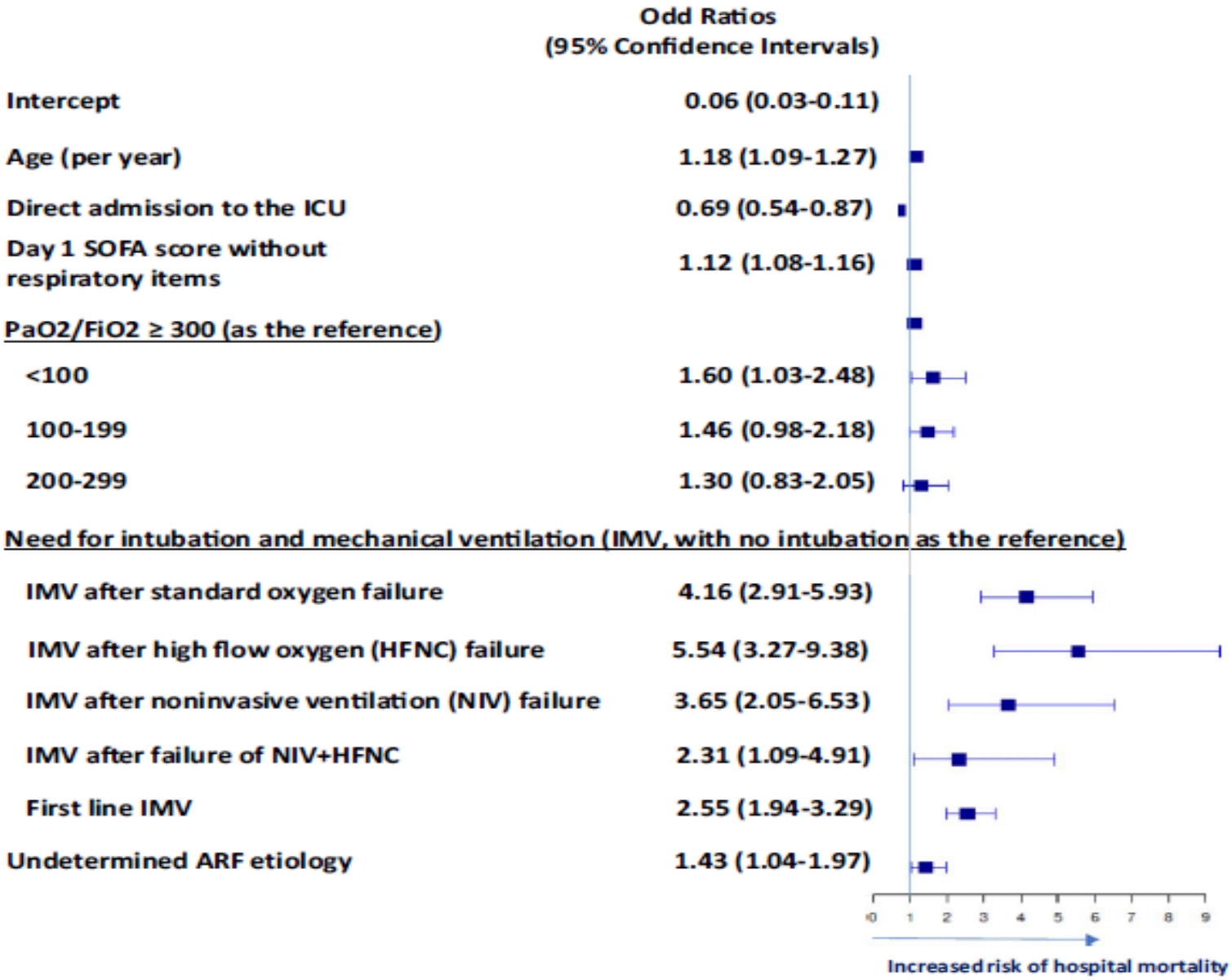


Fig. 4 Multivariate model of the prevalence of hospital death. This analysis is restricted to the 1545 patients with available status at hospital discharge. Plots report variables independently associated with hospital mortality in the final model, with their 95% confidence intervals

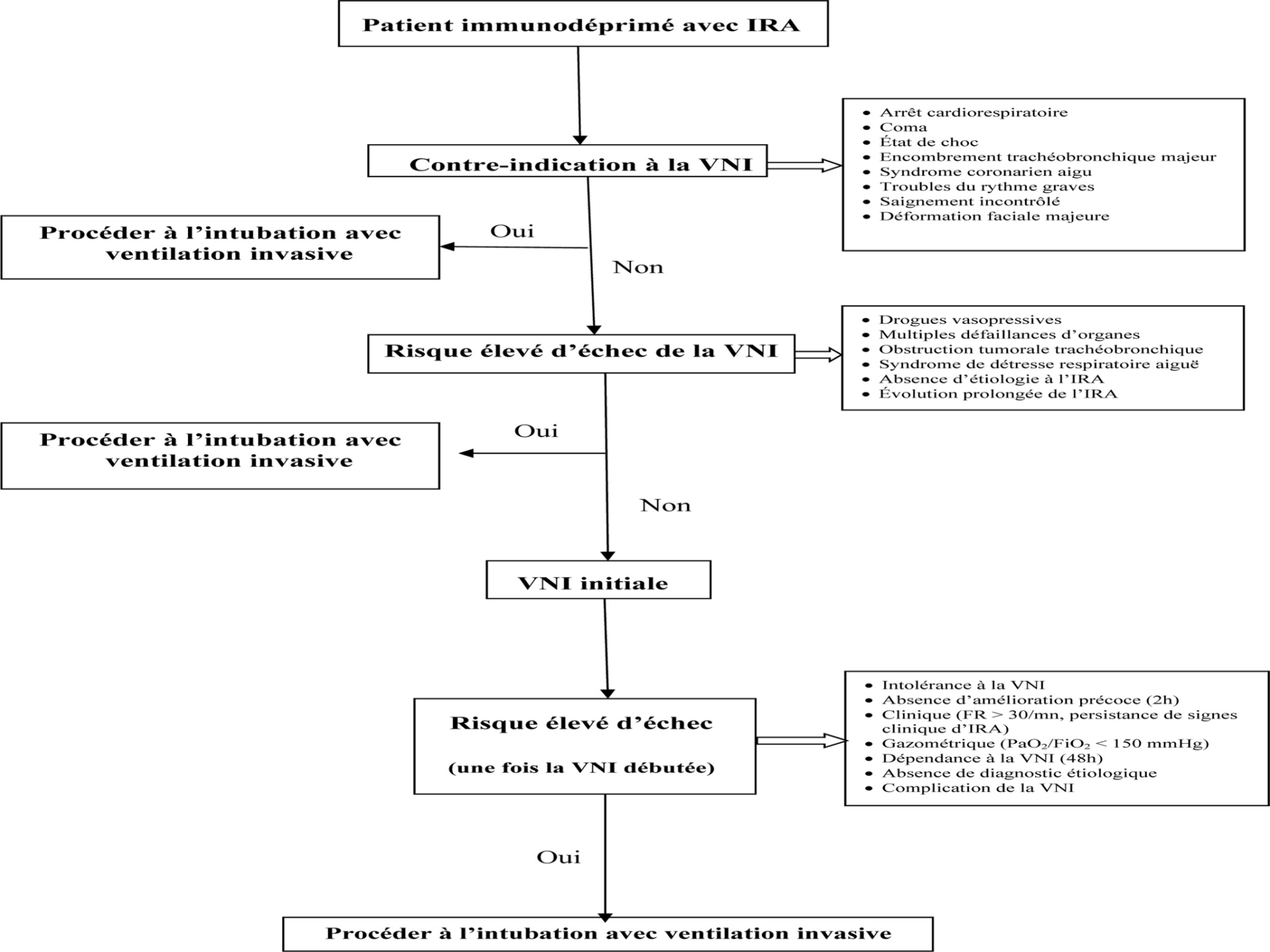
AUTRES INDICATIONS: LATA

- Modalité d'assistance ventilatoire maximale chez des patients immunodéprimés en cas de refus d'intubation ou chez des patients chez qui l'intubation et la VMI jugées déraisonnables (défaillances associées ou stade d'évolutivité de la maladie sous-jacente sans possibilité curative)

  Réanimation (2015) 24:586-598

- VNI palliative de la dyspnée chez des patients en LATA et en phase terminale, en particulier de maladies oncologiques

  Am J Hosp Palliat Care 24:417–21


Patient immunodéprimé avec IRA
Contre-indication à la VNI
Arrêt cardiorespiratoire
Coma
État de choc
Encombrement trachéobronchique majeur
Syndrome coronarien aigu
Troubles du rythme graves
Saignement incontrôlé
Déformation faciale majeure
Oui
Procéder à l'intubation avec ventilation invasive
Non
Risque élevé d'échec de la VNI
Drogues vasopressives
Multiples défaillances d'organes
Obstruction tumorale trachéobronchique
Syndrome de détresse respiratoire aiguë
Absence d'étiologie à l'IRA
Évolution prolongée de l'IRA
Oui
Procéder à l'intubation avec ventilation invasive
Non
VNI initiale
Risque élevé d'échec
(une fois la VNI débutée)
Intolérance à la VNI
Absence d'amélioration précoce (2h)
Clinique (FR > 30/mn, persistance de signes clinique d'IRA)
Gazométrique (PaO_2/FiO_2 < 150 mmHg)
Dépendance à la VNI (48h)
Absence de diagnostic étiologique
Complication de la VNI
Oui
Procéder à l'intubation avec ventilation invasive

IL EST TEMPS DE REVISER LE CONSENSUS…

Tableau 2 – Niveaux de reccommandation pour les indications de la VNI	
Intérêt certain Il faut faire (G1+)	Décompensation de BPCO OAP cardiogénique
Intérêt non établi de façon certaine Il faut probablement faire (G2+)	IRA hypoxémique de l'immunodéprimé Post-opératoire de chirurgie thoracique et abdominale Stratégie de sevrage de la ventilation invasive chez les BPCO Prévention d'une IRA post extubation Traumatisme thoracique fermé isolé Décompensation de maladies neuromusculaires chroniques et autres IRC restrictives Mucoviscidose décompensée *Forme apnéisante de la bronchiolite aiguë* *Laryngo-trachéomalacie*

SFAR-SRLF 2006

« TIME TO THINK DIFFERENTLY »

E.AZOULAY

ESICM 2017