

PREDICTORS OF OUTCOME IN PATIENTS WITH DYSPNOEA OF PULMONARY ORIGIN ADMITTED TO A RESPIRATORY INTENSIVE CARE UNIT: A PROSPECTIVE OBSERVATIONAL STUDY

Davinder Kumar Kundra¹, Vikas Jaiswal², Sachin Baliyan³

Received : 26/08/2026
Received in revised form : 20/09/2026
Accepted : 03/10/2026

Keywords:

Dyspnoea; respiratory intensive care unit; pulmonary disease; mortality; arterial blood gas; SOFA score.

Corresponding Author:

Dr. Davinder kumar kundra,
Email: drkundra@gmail.com

DOI: 10.47009/jamp.2026.8.5.82

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (5); 527-532



¹Senior Consultant, Unit Head Dept of Pulmonology (Respiratory and Sleep Medicine) Manipal Hospital, Dwarka, Delhi, India New Delhi, India

²Sr.Consultant Pulmonologist Respiratory Medicine Rajinder Integrated Multispeciality Hospital & Research Centre Varanasi, Uttar Pradesh, India

³Senior Consultant Pulmonary Medicine Oncoplus Hospital, Delhi, India

ABSTRACT

Background: Adults admitted to a respiratory intensive care unit (RICU) with pulmonary-origin dyspnoea have diverse diagnoses and outcomes. Identifying adverse findings at admission may support early risk assessment. This study examined clinical and physiological factors associated with RICU mortality.

Materials and Methods: In this prospective observational study conducted at a tertiary care centre in North India from January to December 2024, 114 adults admitted with pulmonary-origin dyspnoea were followed until RICU discharge or death. Admission characteristics were compared between survivors and non-survivors. Age, arterial pH ≤ 7.25 and Sequential Organ Failure Assessment (SOFA) score were examined in a multivariable logistic regression model.

Result: Twenty-eight patients died in the RICU (24.6%). COPD exacerbation (36.8%) and pneumonia (25.4%) were the most frequent primary diagnoses. Compared with survivors, non-survivors were older (68.0 ± 11.4 versus 56.8 ± 13.0 years), had a lower $\text{PaO}_2/\text{FiO}_2$ ratio (136.9 ± 39.6 versus 188.4 ± 60.4 mmHg), and had a higher SOFA score (6.0 ± 1.5 versus 4.5 ± 1.8); all $p < 0.001$. Admission pH ≤ 7.25 occurred in 39.3% of non-survivors versus 10.5% of survivors ($p = 0.001$). Mortality was associated with age per decade (adjusted odds ratio [aOR] 2.48; 95% confidence interval [CI] 1.48–4.17), pH ≤ 7.25 (aOR 5.89; 95% CI 1.63–21.25) and SOFA score per point (aOR 1.58; 95% CI 1.14–2.18). **Conclusion:** Older age, marked acidemia and greater organ dysfunction were associated with RICU mortality. These admission measures may help identify patients who need closer monitoring.

INTRODUCTION

Dyspnoea of pulmonary origin is a frequent reason for admission to a respiratory intensive care unit (RICU). It may arise from an exacerbation of chronic obstructive pulmonary disease (COPD), pneumonia, acute respiratory distress syndrome (ARDS), or another acute lung disorder. Although these conditions differ in cause and treatment, patients may present with similar respiratory distress while facing substantially different risks. In a multicentre study, 458 of 1,449 critically ill patients (32%) had acute respiratory failure at ICU admission; their ICU mortality was 34%, compared with 16% among patients without respiratory failure.^[1] The LUNG SAFE study likewise documented a substantial mortality burden among patients with ARDS across 50 countries.^[2]

The intensity of dyspnoea may add prognostic information to physiological measurements. Among patients receiving non-invasive ventilation (NIV), persistent dyspnoea was associated with NIV failure and mortality.^[3] A subsequent study of acute hypoxaemic respiratory failure linked greater dyspnoea at presentation with a higher risk of intubation.^[4] Other indicators may vary by diagnosis: an Indian COPD cohort identified admission APACHE II score and serum albumin as predictors of hospital mortality,^[5] while an Indian ARDS cohort found associations with non-pulmonary organ failure and illness severity.^[6] A multicentre ventilatory-support cohort also identified age, comorbidity, organ dysfunction and NIV failure as prognostic factors.^[7]

Early assessment must therefore consider the underlying pulmonary diagnosis alongside respiratory rate, consciousness, arterial blood gases, oxygenation, infection and organ dysfunction. This is

clinically relevant because deterioration during NIV and the need for invasive ventilation are associated with poorer outcomes, while oxygen-support strategies differ across forms of respiratory failure.^[8,9] So, this prospective observational study aims to describe the clinical and diagnostic profile of adults admitted to an RICU with dyspnoea attributed to pulmonary disease and to determine which characteristics recorded at admission or during early ICU assessment are associated with ICU mortality. Secondary objectives are to examine factors associated with the need for invasive mechanical ventilation and length of ICU stay.

MATERIALS AND METHODS

Study design and setting: This prospective observational study was conducted by the Department of Pulmonology in collaboration with the Emergency Department at a tertiary care centre in North India over 12 months, from January to December 2024. Consecutive eligible adults admitted to the respiratory intensive care unit (RICU) were enrolled and followed until RICU discharge or death. Investigations and treatment were directed by the treating team; study participation did not change clinical care.

Participants and eligibility: Patients aged 18 years or older were eligible when dyspnoea attributed primarily to pulmonary disease was the reason for RICU admission. Pulmonary origin was determined from the history, clinical examination, prior records, chest imaging and routinely obtained investigations. Eligible conditions included exacerbations of chronic obstructive pulmonary disease (COPD), asthma or bronchiectasis; pneumonia; acute deterioration of interstitial lung disease; pulmonary tuberculosis; and other documented pulmonary disorders. Patients whose dyspnoea was predominantly due to cardiac failure, acute coronary syndrome, severe anaemia, metabolic disturbance or another non-pulmonary cause were excluded. Where pulmonary and non-pulmonary conditions coexisted, the treating pulmonologist assigned the principal cause of the presenting dyspnoea; cases without a clearly predominant pulmonary cause were excluded. Only the first eligible RICU admission for each patient was analysed. So, a total of 114 patients were included in the study.

Clinical assessment and data collection: Data were collected prospectively using a structured case record form. Baseline variables included age, sex, smoking history, pre-existing lung disease, hypertension, diabetes mellitus, presenting symptoms and the principal pulmonary diagnosis. Findings from the initial RICU assessment included respiratory rate, heart rate, blood pressure, peripheral oxygen saturation, mental status, presence of shock or sepsis, and Sequential Organ Failure Assessment (SOFA) score. Haemoglobin, leucocyte count, serum creatinine, chest imaging and microbiological

findings were recorded when obtained during routine care.

The first arterial blood gas obtained during the initial RICU assessment was used to record pH, arterial oxygen tension (PaO_2), arterial carbon dioxide tension (PaCO_2) and bicarbonate. The $\text{PaO}_2/\text{FiO}_2$ ratio was calculated using the fraction of inspired oxygen (FiO_2) documented at sampling. Admission $\text{pH} \leq 7.25$ and $\text{PaO}_2/\text{FiO}_2 < 150$ mmHg were examined as clinically relevant severity categories. Where applicable, acute respiratory distress syndrome (ARDS) was identified using the Berlin definition, including acute onset, bilateral pulmonary opacities, respiratory failure not fully explained by cardiac failure or fluid overload, and impaired oxygenation assessed under the specified positive airway pressure conditions.

Initial respiratory support was classified as conventional oxygen, high-flow nasal oxygen, non-invasive ventilation (NIV) or invasive mechanical ventilation (IMV). Subsequent intubation, acute kidney injury and other complications during RICU care were recorded separately from admission findings. NIV failure was defined as subsequent intubation among patients initially managed with NIV.

Outcomes: The primary outcome was all-cause RICU mortality, defined as death before discharge from the unit. Patients discharged alive to a ward or another unit were classified as RICU survivors. Secondary outcomes were IMV initiated after admission and length of RICU stay, measured from admission to discharge or death. Analysis of subsequent intubation was restricted to patients who were not intubated initially. Respiratory support and complications developing during admission were assessed as features of the clinical course, not as predictors available at admission.

Statistical analysis: Data were entered into IBM SPSS Statistics version 20.0 and checked for completeness and implausible values. Categorical variables were expressed as frequencies and percentages; continuous variables as mean $\pm$ standard deviation or median with interquartile range, according to distribution. Survivors and non-survivors were compared using the independent-samples t test, with unequal variance estimates where appropriate, or the Mann-Whitney U test. Categorical comparisons used Fisher's exact test; a Monte Carlo exact approach was used for larger tables with sparse cells. A limited multivariable logistic regression model examined associations of admission age (per 10 years), $\text{pH} \leq 7.25$ and SOFA score (per point) with RICU mortality. Adjusted odds ratios were reported with 95% confidence intervals. Tests were two-sided, with $p < 0.05$ considered statistically significant.

Ethical considerations: The study was approved by the Institutional Ethics Committee of the study centre. Written informed consent was obtained from participants or, when they lacked capacity, from an authorised representative in accordance with the

approved protocol. Records were coded and analysed without direct patient identifiers.

RESULTS

Of 114 patients, 86 survived and 28 died during RICU admission, giving a mortality rate of 24.6%.

Non-survivors were older than survivors (68.0 ± 11.4 versus 56.8 ± 13.0 years; $p < 0.001$). Men comprised 70.2% of the cohort. Smoking history, hypertension, diabetes mellitus and pre-existing chronic lung disease did not differ significantly between the groups [Table 1].

Table 1: Demographic characteristics and medical history according to RICU survival status.

Characteristic	All patients (N=114)	Survivors (n=86)	Non-survivors (n=28)	p value
	Frequency (%) / Mean $\pm$ SD			
Age, years	59.5 ± 13.5	56.8 ± 13.0	68.0 ± 11.4	<0.001
Male Gender	80 (70.2)	61 (70.9)	19 (67.9)	0.814
Smoking history	53 (46.5)	39 (45.3)	14 (50.0)	0.828
Hypertension	35 (30.7)	23 (26.7)	12 (42.9)	0.156
Diabetes mellitus	22 (19.3)	16 (18.6)	6 (21.4)	0.785
Pre-existing chronic lung disease	73 (64.0)	57 (66.3)	16 (57.1)	0.497

RICU, respiratory intensive care unit. Medical-history categories may overlap.

COPD exacerbation was the most frequent diagnosis (42/114, 36.8%), followed by pneumonia (29/114, 25.4%). Pneumonia accounted for 9 of 28 deaths (32.1%). Six of 12 patients with fibrotic ILD exacerbation died, corresponding to a diagnosis-

specific mortality of 50.0%. All nine patients admitted with acute asthma survived. The overall distribution of diagnoses did not differ significantly between survivors and non-survivors ($p = 0.179$) [Table 2].

Table 2: Primary pulmonary diagnoses and RICU outcome.

Primary diagnosis	All patients (N=114)	Survivors (n=86)	Non-survivors (n=28)	Mortality within diagnosis, %
	Frequency (%)			
COPD exacerbation	42 (36.8)	34 (39.5)	8 (28.6)	19.0
Pneumonia	29 (25.4)	20 (23.3)	9 (32.1)	31.0
Fibrotic ILD exacerbation	12 (10.5)	6 (7.0)	6 (21.4)	50.0
Bronchiectasis exacerbation	10 (8.8)	8 (9.3)	2 (7.1)	20.0
Acute asthma	9 (7.9)	9 (10.5)	0 (0.0)	0.0
Pulmonary tuberculosis	7 (6.1)	5 (5.8)	2 (7.1)	28.6
Other pulmonary diagnoses	5 (4.4)	4 (4.7)	1 (3.6)	20.0
Total	114 (100)	86 (100)	28 (100)	24.6

COPD, chronic obstructive pulmonary disease; ILD, interstitial lung disease.

Non-survivors had a higher admission respiratory rate than survivors (34.5 ± 5.4 versus 29.5 ± 5.1 breaths/min; $p < 0.001$) and a higher SOFA score (6.0 ± 1.5 versus 4.5 ± 1.8 ; $p < 0.001$). Altered

consciousness (28.6% versus 4.7%; $p = 0.001$), shock (25.0% versus 4.7%; $p = 0.004$) and sepsis (42.9% versus 5.8%; $p < 0.001$) were more frequent among non-survivors. Heart rate did not differ significantly ($p = 0.108$) [Table 3].

Table 3: Clinical findings at RICU admission by survival status.

Admission finding	All patients (N=114)	Survivors (n=86)	Non-survivors (n=28)	p value
	Frequency (%) / Mean $\pm$ SD			
Respiratory rate, breaths/min	30.7 ± 5.6	29.5 ± 5.1	34.5 ± 5.4	<0.001
Heart rate, beats/min	109.2 ± 15.7	107.8 ± 15.4	113.5 ± 16.1	0.108
SOFA score	4.9 ± 1.8	4.5 ± 1.8	6.0 ± 1.5	<0.001
Altered consciousness	12 (10.5)	4 (4.7)	8 (28.6)	0.001
Shock at admission	11 (9.6)	4 (4.7)	7 (25.0)	0.004
Sepsis at admission	17 (14.9)	5 (5.8)	12 (42.9)	<0.001

SOFA, Sequential Organ Failure Assessment. Shock and sepsis were assessed at admission.

Initial arterial pH was lower among non-survivors than survivors (7.257 ± 0.067 versus 7.328 ± 0.061 ; $p < 0.001$). A $pH \leq 7.25$ occurred in 39.3% of non-survivors and 10.5% of survivors ($p = 0.001$). Non-survivors also had a lower mean PaO_2/FiO_2 ratio

(136.9 ± 39.6 versus 188.4 ± 60.4 mmHg; $p < 0.001$), and more frequently had a ratio < 150 mmHg (64.3% versus 25.6%; $p < 0.001$). $PaCO_2$ did not differ significantly ($p = 0.248$) [Table 4].

Table 4: Initial arterial blood gas and oxygenation findings by survival status.

Parameter	All patients (N=114)	Survivors (n=86)	Non-survivors (n=28)	p value
	Frequency (%) / Mean $\pm$ SD			
Arterial pH	7.311 $\pm$ 0.069	7.328 $\pm$ 0.061	7.257 $\pm$ 0.067	<0.001
Arterial pH $\leq$ 7.25	20 (17.5)	9 (10.5)	11 (39.3)	0.001
PaCO ₂ , mmHg	54.4 $\pm$ 14.3	53.5 $\pm$ 14.3	57.1 $\pm$ 14.0	0.248
PaO ₂ /FiO ₂ ratio, mmHg	175.8 $\pm$ 60.2	188.4 $\pm$ 60.4	136.9 $\pm$ 39.6	<0.001
PaO ₂ /FiO ₂ ratio <150 mmHg	40 (35.1)	22 (25.6)	18 (64.3)	<0.001

PaCO₂, arterial carbon dioxide tension; PaO₂/FiO₂, ratio of arterial oxygen tension to fraction of inspired oxygen.

Initial respiratory support differed between groups ($p < 0.001$). IMV was used at admission in 13/28 non-survivors (46.4%) and 4/86 survivors (4.7%). Among patients not initially intubated, subsequent IMV was required in 6/15 non-survivors (40.0%) and 12/82

survivors (14.6%; $p = 0.031$). Twelve of 57 patients initially receiving NIV subsequently required intubation (21.1%). IMV at any time was recorded in 67.9% of non-survivors and 18.6% of survivors ($p < 0.001$). Acute kidney injury was more frequent among non-survivors (21.4% versus 3.5%; $p = 0.007$), while their median RICU stay was shorter (6 versus 7 days; $p = 0.006$) (Table 5).

Table 5: Respiratory support, complications and RICU stay by survival status.

Variable	All patients (N=114)	Survivors (n=86)	Non-survivors (n=28)	p value
	Frequency (%) / Mean $\pm$ SD / median (IQR)			
Initial respiratory support				
Conventional oxygen	23/114 (20.2)	21/86 (24.4)	2/28 (7.1)	<0.001 †
High-flow nasal oxygen	17/114 (14.9)	16/86 (18.6)	1/28 (3.6)	—
Non-invasive ventilation	57/114 (50.0)	45/86 (52.3)	12/28 (42.9)	—
Invasive mechanical ventilation	17/114 (14.9)	4/86 (4.7)	13/28 (46.4)	—
Subsequent invasive ventilation among those not initially intubated	18/97 (18.6)	12/82 (14.6)	6/15 (40.0)	0.031
NIV failure requiring intubation, among those initially receiving NIV	12/57 (21.1)	6/45 (13.3)	6/12 (50.0)	0.012
Invasive ventilation at any time	35/114 (30.7)	16/86 (18.6)	19/28 (67.9)	<0.001
Acute kidney injury during RICU stay	9/114 (7.9)	3/86 (3.5)	6/28 (21.4)	0.007
RICU stay, days	7 (5–9)	7 (5–10)	6 (4–8)	0.006

IQR, interquartile range; HFNO, high-flow nasal oxygen; NIV, non-invasive ventilation; IMV, invasive mechanical ventilation. Subsequent IMV was assessed among 97 patients not initially intubated; NIV failure was assessed among 57 patients initially receiving NIV.

Older age was associated with higher odds of RICU mortality (aOR per 10-year increase, 2.48; 95% CI, 1.48–4.17; $p < 0.001$). Admission pH $\leq$ 7.25 was associated with mortality (aOR 5.89; 95% CI, 1.63–21.25; $p = 0.007$), as was each one-point increase in SOFA score (aOR 1.58; 95% CI, 1.14–2.18; $p = 0.005$) [Table 6].

Table 6: Multivariable associations of admission factors with RICU mortality.

Admission variable	Adjusted odds ratio	95% confidence interval	p value
Age, per 10-year increase	2.48	1.48–4.17	<0.001
Initial arterial pH $\leq$ 7.25	5.89	1.63–21.25	0.007
SOFA score, per 1-point increase	1.58	1.14–2.18	0.005

The outcome was death before RICU discharge (28 events). Adjusted odds ratios (aORs) include age, admission pH category and SOFA score in the same model; 95% CIs are confidence intervals. SOFA, Sequential Organ Failure Assessment.

DISCUSSION

In this 114-patient cohort, RICU mortality was 24.6%. COPD exacerbation was the leading admission diagnosis (36.8%), followed by pneumonia (25.4%). Sen et al., reported higher mortality (39.5%) in a 76-patient Eastern Indian RICU cohort with pulmonary-origin dyspnoea.^[10] Differences in admission thresholds, diagnostic mix

and severity could explain the contrast, but the cohorts cannot be compared directly. Obstructive disease was also prominent in the multicentre YETI study by Dupuis et al., of acute hypercapnic respiratory failure.^[11] In a North Indian RICU, Agarwal et al., found that NIV failed less often in COPD-related respiratory failure than in respiratory failure from other causes (3/24 versus 15/39; $p = 0.03$).^[12] Thus, the frequency of COPD admissions alone would not be expected to determine overall mortality.

Diagnosis-specific mortality was highest in fibrotic interstitial lung disease (ILD) exacerbation (6/12, 50.0%), although the overall association between diagnosis and outcome was not significant ($p = 0.179$).

In a North Indian series of 145 patients with acute respiratory failure related to diffuse parenchymal lung disease, Sehgal et al., reported 45.5% mortality and identified poor oxygenation and worsening organ dysfunction as adverse factors.^[13] Limited recruitable lung tissue, severe gas-exchange impairment and, in some patients, less reversible disease may contribute to this pattern.

Non-survivors were older and had higher admission SOFA scores; both variables remained associated with mortality in the three-variable model (adjusted odds ratios [aORs] 2.48 per decade and 1.58 per SOFA point). Altered consciousness, shock and sepsis were also more common among those who died. Raith et al., demonstrated the prognostic value of SOFA in a large ICU population with suspected infection.^[14] while Ferreira et al., showed that organ-failure assessment over time adds prognostic information.^[15] SOFA captures dysfunction beyond the lungs, helping explain why respiratory diagnosis alone was insufficient to distinguish outcomes. The association with age may reflect reduced physiological reserve and vulnerability to concurrent organ failure, although those mechanisms were not measured directly in this cohort.

Admission acidemia was a further signal: pH ≤ 7.25 occurred in 39.3% of non-survivors versus 10.5% of survivors and remained associated with mortality after adjustment (aOR 5.89). Confalonieri et al., incorporated low pH, rapid breathing and impaired consciousness into a chart predicting NIV failure in COPD exacerbation.^[16] Duan et al., likewise used acidosis alongside consciousness, oxygenation and respiratory rate in an NIV-failure score.^[17] Acidemia may reflect inadequate ventilation, increased respiratory muscle load or systemic illness; the absence of a significant PaCO₂ difference here suggests that hypercapnia alone did not account for the pH–outcome association.

Non-survivors had lower PaO₂/FiO₂ ratios (136.9 versus 188.4 mmHg) and higher respiratory rates. A North Indian hypoxaemic respiratory-failure study by Agarwal et al., associated lower baseline PaO₂/FiO₂ with NIV failure.^[18] In severe pneumonia, Carrillo et al., found that oxygenation and organ-failure measures helped identify patients at risk of NIV failure.^[19] Carteaux et al., linked higher tidal volumes during NIV with failure in new-onset hypoxaemic respiratory failure.^[20] while Roca et al., combined respiratory rate and oxygenation to assess the risk of failure during high-flow oxygen therapy.^[21] Together, these studies support repeated assessment of respiratory effort and gas exchange rather than reliance on a single admission measurement.

IMV at any time was more frequent among non-survivors (67.9% versus 18.6%), and acute kidney injury occurred in 21.4% versus 3.5%. The multinational AKI-EPI study by Hostle et al., similarly associated acute kidney injury with worse ICU outcomes.^[22] IMV and kidney injury arose during the clinical course and may chiefly mark

deterioration; these associations do not show that ventilation caused death. The shorter median RICU stay among non-survivors (6 versus 7 days) may reflect earlier death. With only 28 deaths, the adjusted estimates particularly for pH remain imprecise and require confirmation in the actual cohort.

Limitations: This single-centre study included 114 patients and only 28 deaths, limiting precision and the number of variables that could be assessed in the regression model. Small diagnostic subgroups made disease-specific mortality estimates uncertain. Outcomes were recorded at RICU discharge, so subsequent hospital deaths and longer-term recovery were not captured.

CONCLUSION

Among adults admitted with dyspnoea of pulmonary origin, COPD exacerbation and pneumonia were the leading diagnoses, and RICU mortality was 24.6%. Non-survivors were older and presented with greater respiratory distress, worse oxygenation, more frequent acidemia and higher organ dysfunction scores. Age, arterial pH ≤ 7.25 and SOFA score retained associations with mortality after adjustment. The higher frequency of invasive ventilation and acute kidney injury among non-survivors describes the severity of their subsequent clinical course.

REFERENCES

1. Vincent JL, Akça S, De Mendonça A, et al. The epidemiology of acute respiratory failure in critically ill patients. *Chest*. 2002;121:1602–1609. doi:10.1378/chest.121.5.1602.
2. Bellani G, Laffey JG, Pham T, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. *JAMA*. 2016;315:788–800. doi:10.1001/jama.2016.0291.
3. Dangers L, Montlahuc C, Kouatchet A, et al. Dyspnoea in patients receiving noninvasive ventilation for acute respiratory failure: prevalence, risk factors and prognostic impact. *Eur Respir J*. 2018;52:1702637. doi:10.1183/13993003.02637-2017.
4. Demoule A, et al. Dyspnea is severe and associated with a higher intubation rate in de novo acute hypoxemic respiratory failure. *Crit Care*. 2024;28:174. doi:10.1186/s13054-024-04903-5.
5. Khilnani GC, Banga A, Sharma SK. Predictors of mortality of patients with acute respiratory failure secondary to chronic obstructive pulmonary disease admitted to an intensive care unit: a one year study. *BMC Pulm Med*. 2004;4:12. doi:10.1186/1471-2466-4-12.
6. Sharma SK, Gupta A, Biswas A, et al. Aetiology, outcomes & predictors of mortality in acute respiratory distress syndrome from a tertiary care centre in north India. *Indian J Med Res*. 2016;143:782–792. doi:10.4103/0971-5916.192063.
7. Azevedo LCP, Park M, Salluh JIF, et al. Clinical outcomes of patients requiring ventilatory support in Brazilian intensive care units: a multicenter, prospective, cohort study. *Crit Care*. 2013;17:R63. doi:10.1186/cc12594.
8. Bellani G, Laffey JG, Pham T, et al. Noninvasive ventilation of patients with acute respiratory distress syndrome: insights from the LUNG SAFE study. *Am J Respir Crit Care Med*. 2017;195:67–77. doi:10.1164/rccm.201606-1306OC.
9. Frat JP, Thille AW, Mercat A, et al. High-flow oxygen through nasal cannula in acute hypoxemic respiratory failure.

- N Engl J Med. 2015;372:2185–2196. doi:10.1056/NEJMoa1503326.
10. Sen S, Pal A, Banerjee S, et al. Predictors of outcome in patients with dyspnoea of pulmonary origin admitted to a respiratory intensive care unit: a prospective observational study from Eastern India. *J Clin Diagn Res.* 2026;20(4):OC33–OC38. doi:10.7860/JCDR/2026/85110.22837.
 11. Dupuis C, Kamel T, Beyls C, et al. Acute hypercapnic respiratory failure in the ICU: a multicenter prospective observational study—the YETI study. *Ann Intensive Care.* 2026;16:100016. doi:10.1016/j.aicj.2025.100016.
 12. Agarwal R, Gupta R, Aggarwal AN, Gupta D. Noninvasive positive pressure ventilation in acute respiratory failure due to COPD vs other causes: effectiveness and predictors of failure in a respiratory ICU in North India. *Int J Chron Obstruct Pulmon Dis.* 2008;3:737–743. doi:10.2147/COPD.S3454.
 13. Sehgal IS, Agarwal R, Dhooira S, Prasad KT, Aggarwal AN, Behera D. Acute respiratory failure due to diffuse parenchymal lung diseases in a respiratory intensive care unit of North India. *Sarcoidosis Vasc Diffuse Lung Dis.* 2018;35:363–370. doi:10.36141/svdld.v35i4.7066.
 14. Raith EP, Udy AA, Bailey M, et al. Prognostic accuracy of the SOFA score, SIRS criteria, and qSOFA score for in-hospital mortality among adults with suspected infection admitted to the intensive care unit. *JAMA.* 2017;317:290–300. doi:10.1001/jama.2016.20328.
 15. Ferreira FL, Bota DP, Bross A, Mélot C, Vincent JL. Serial evaluation of the SOFA score to predict outcome in critically ill patients. *JAMA.* 2001;286:1754–1758. doi:10.1001/jama.286.14.1754.
 16. Confalonieri M, Garuti G, Cattaruzza MS, et al. A chart of failure risk for noninvasive ventilation in patients with COPD exacerbation. *Eur Respir J.* 2005;25:348–355. doi:10.1183/09031936.05.00085304.
 17. Duan J, Wang S, Liu P, et al. Early prediction of noninvasive ventilation failure in COPD patients: derivation, internal validation, and external validation of a simple risk score. *Ann Intensive Care.* 2019;9:108. doi:10.1186/s13613-019-0585-9.
 18. Agarwal R, Handa A, Aggarwal AN, Gupta D, Behera D. Outcomes of noninvasive ventilation in acute hypoxemic respiratory failure in a respiratory intensive care unit in North India. *Respir Care.* 2009;54:1679–1687.
 19. Carrillo A, Gonzalez-Díaz G, Ferrer M, et al. Non-invasive ventilation in community-acquired pneumonia and severe acute respiratory failure. *Intensive Care Med.* 2012;38:458–466. doi:10.1007/s00134-012-2475-6.
 20. Carreaux G, Millán-Guilarte T, De Prost N, et al. Failure of noninvasive ventilation for de novo acute hypoxemic respiratory failure: role of tidal volume. *Crit Care Med.* 2016;44:282–290. doi:10.1097/CCM.0000000000001379.
 21. Roca O, Caralt B, Messika J, et al. An index combining respiratory rate and oxygenation to predict outcome of nasal high-flow therapy. *Am J Respir Crit Care Med.* 2019;199:1368–1376. doi:10.1164/rccm.201803-0589OC.
 22. Hoste EAJ, Bagshaw SM, Bellomo R, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. *Intensive Care Med.* 2015;41:1411–1423. doi:10.1007/s00134-015-3934-7.