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Predictive Value of the Braden Scale for Mechanical Ventilation Weaning Failure in Severe Pneumonia: A Retrospective Cohort Study with Incremental Value Analysis

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Abstract

Background: Identifying patients at high risk for mechanical ventilation weaning failure remains a persistent challenge in critical care. The Braden Scale, a bedside functional assessment tool already routinely administered in ICUs, may capture systemic functional status relevant to weaning capacity, yet its predictive value for weaning outcomes and its incremental contribution beyond disease severity scores remain unknown. This study evaluated the association between Braden score and weaning failure in severe pneumonia patients and systematically assessed its incremental predictive value beyond APACHE II.

Methods: A retrospective cohort study was conducted on 184 mechanically ventilated patients with severe pneumonia admitted to the ICU of a tertiary hospital from January 2021 to December 2025. Patients were divided into weaning success group (n = 85) and weaning failure group (n = 99). Multivariate logistic regression was used to analyze the association between Braden score and weaning failure. Four models were constructed upon a base model (age, sex, tracheostomy, vasopressors): Model A (base+Braden), Model B (fully adjusted), Model E (base+APACHE II), and Model F (base+APACHE II+Braden). Incremental value was assessed using ROC curves, decision curve analysis (DCA), net reclassification improvement (NRI), and integrated discrimination improvement (IDI). Bootstrap 1,000 iterations were used for internal validation. Subgroup analysis was performed with Benjamini-Hochberg FDR correction.

Results: Braden score was independently associated with weaning failure (OR = 0.77, 95% CI 0.65–0.92, P = 0.004), with a significant dose-response relationship across risk strata (P-trend = 0.041). Braden provided significant incremental value beyond the base model (ΔAUC = 0.029, NRI = 0.389, IDI = 0.044; all P < 0.05). However, its incremental value beyond APACHE II was limited (ΔAUC = 0.009, P = 0.224), indicating substantial information overlap. Bootstrap validation confirmed model stability (optimism-corrected AUC = 0.754). Subgroup analysis with FDR correction identified tracheostomy as a significant effect modifier (OR = 0.60, FDR P = 0.034), suggesting Braden may be particularly informative in tracheostomized patients.

Conclusions: The Braden Scale is independently associated with weaning failure and offers a practical bedside tool for functional status assessment. Its incremental value beyond APACHE II is limited, supporting its use as a complement rather than a replacement for disease severity scores. The tracheostomy subgroup finding warrants further investigation.

Trial registration: Clinical trial number: not applicable. This was a retrospective observational cohort study, not a clinical trial.

Background

Severe pneumonia is one of the most common critical illnesses in the ICU. Worldwide, millions of people require ICU admission for severe pneumonia each year [1]. Severe hypoxemia, acute respiratory distress

syndrome (ARDS), and respiratory failure are the main indications for invasive mechanical ventilation in severe pneumonia patients [1, 12]. Although mechanical ventilation is life-saving, prolonged ventilation leads to complications including respiratory muscle dysfunction, diaphragmatic atrophy, ICU-acquired weakness (ICU-AW), and ventilator-associated pneumonia (VAP) [2, 13]. Therefore, timely liberation from mechanical ventilation when conditions permit is critical for improving patient outcomes. However, weaning is not always straightforward. The WIND study by Beduneau et al. [3] included 2,760 mechanically ventilated patients and found that approximately 31.6% experienced at least one weaning failure, with higher ICU mortality (17.6%) and hospital mortality (26.5%) in weaning failure patients. Weaning failure prolongs mechanical ventilation duration and ICU stay, increases infection risk and healthcare costs, and may cause respiratory muscle fatigue, psychological trauma, and long-term functional impairment [3, 14]. Accurate identification of high-risk patients for weaning failure is therefore essential for optimizing weaning strategies.

Current tools for assessing weaning readiness each carry practical limitations. Spontaneous breathing trials (SBT) remain the standard screening method, but their criteria rely heavily on clinical observation with inherent subjectivity; the rapid shallow breathing index (RSBI) is simple but influenced by multiple factors with variable predictive performance [15]. APACHE II [19] and SOFA scores correlate with weaning outcomes but are computationally complex, require multiple laboratory parameters, and were primarily designed for disease severity assessment rather than weaning evaluation [4]. A gap therefore exists at the bedside: clinicians lack a simple, rapidly repeatable tool that comprehensively captures functional status relevant to weaning readiness.

The Braden Scale, developed by Bergstrom et al. [6] in 1987, may bridge this gap. Originally designed as a pressure ulcer risk assessment tool, it comprises six dimensions — sensory perception, moisture, activity, mobility, nutrition, and friction/shear — with total scores ranging from 6 to 23 (lower scores indicate higher risk). It is already routinely administered in ICUs worldwide for pressure ulcer screening [7–8], requiring no additional equipment, laboratory tests, or specialized training beyond standard protocols. What makes the Braden Scale potentially relevant to weaning is that several of its dimensions tap into physiological domains known to influence weaning success. Activity and mobility, for instance, reflect the physical functioning whose decline underlies ICU-AW and diaphragmatic dysfunction — both major barriers to liberation from the ventilator [2]. Nutritional status, captured in the nutrition dimension, has long been linked to weaning outcomes through the well-established effects of hypoalbuminemia and malnutrition on respiratory muscle strength [4–5]. Consciousness, reflected in the sensory perception dimension, is itself a prerequisite for spontaneous breathing trial success [3]. In this way, a single bedside score may inadvertently integrate functional information that no current weaning-specific tool directly captures. Li et al. [9] found that Braden score was associated with prognosis in elderly severe pneumonia patients, but its predictive value for mechanical ventilation weaning outcomes has not been adequately studied.

Whether the Braden Scale provides predictive information independent of disease severity scores — that is, its incremental predictive value beyond APACHE II — is a clinically important but unresolved question.

To date, no study has systematically evaluated this incremental value using metrics such as net reclassification improvement (NRI), integrated discrimination improvement (IDI), and decision curve analysis (DCA). Prior studies have examined the association between Braden score and prognosis in specific populations [9], but have not quantified the additional predictive contribution relative to established severity scores, nor explored potential effect modification across clinical subgroups. Answering this question has direct clinical implications: if the Braden Scale offers incremental value, it could be integrated into weaning decision-making to enhance multidisciplinary assessment; if incremental value is limited, clarifying whether it serves as an independent predictor or an indirect reflection of disease severity would still inform its appropriate clinical use.

This study retrospectively analyzed clinical data from 184 mechanically ventilated patients with severe pneumonia to investigate the association between Braden score and weaning outcomes and to systematically evaluate its incremental predictive value beyond APACHE II using a comprehensive analytical framework — multivariable logistic regression, ROC/AUC with DeLong testing, DCA, NRI, IDI, bootstrap internal validation, and FDR-corrected subgroup analysis — aiming to provide robust evidence for clinical assessment and weaning decisions.

Methods

Study Design and Population

A retrospective cohort study was conducted collecting clinical data from mechanically ventilated severe pneumonia patients admitted to the ICU of a tertiary grade-A hospital from January 2021 to December 2025. Clinical trial number: not applicable. This was a retrospective observational cohort study, not a clinical trial. This study was approved by the hospital's ethics committee (Approval No. : 2026-ky-053), and informed consent was waived due to the retrospective nature. Severe pneumonia was diagnosed according to the 2016 IDSA/ATS criteria [12]: meeting major criteria (requiring invasive mechanical ventilation or septic shock requiring vasopressors) or at least 3 minor criteria (respiratory rate ≥ 30/min, PaO_2/FiO_2 ≤250, multilobar infiltrates, confusion/disorientation, BUN ≥ 7.14 mmol/L, infection-induced leukopenia, thrombocytopenia, hypothermia, hypotension requiring aggressive fluid resuscitation).

Inclusion criteria: (1) meeting severe pneumonia diagnostic criteria; (2) age ≥ 18 years; (3) receiving invasive mechanical ventilation ≥ 48 h; (4) complete Braden score data; (5) complete clinical records.

Exclusion criteria: (1) neuromuscular diseases (e.g., myasthenia gravis, Guillain-Barre syndrome, ALS) directly affecting weaning assessment; (2) pre-existing severe motor dysfunction (e.g., stroke sequelae, spinal cord injury); (3) terminal malignancy; (4) voluntary discharge or treatment withdrawal; (5) missing key data.

Weaning Protocol and Grouping

Weaning Protocol and Grouping

Weaning followed relevant guidelines from the Chinese Society of Critical Care Medicine [1]. When patients met prerequisite conditions (primary disease improved/controlled; $PaO_2/FiO_2 \geq 150$ mmHg with PEEP ≤ 8 cmH_2O; hemodynamic stability; conscious or arousable; no severe acidosis pH ≥ 7.25), weaning screening was initiated, followed by SBT using T-piece or pressure support ventilation (PS 5–7 cmH_2O) for 30–120 min.

Patients were grouped by weaning outcome: weaning success group (*n* = 85), first SBT passed with successful extubation, no reintubation or non-invasive ventilation within 48 h; weaning failure group (*n* = 99), including: (1) SBT failure; (2) reintubation within 48 h; (3) requiring non-invasive ventilation within 48 h and unable to wean [3, 14].

Data Collection

(1) Demographics: age, sex, BMI, smoking history, comorbidities (hypertension, diabetes, COPD). (2) Disease severity: APACHE II (worst value within 24 h of admission), SOFA. (3) Laboratory: albumin, CRP, PCT, WBC, NLR, platelets. (4) Functional assessment: Braden Scale, assessed by trained ICU staff within 24 h of admission and daily thereafter; inter-rater reliability ensured by periodic quality control (Kappa ≥ 0.80). (5) Treatment: mechanical ventilation duration, tracheostomy, vasopressor use, ICU stay. All data were extracted independently by two researchers and cross-verified.

Statistical Analysis

Python 3.11 (statsmodels 0.14, scipy 1.11, scikit-learn 1.3) was used. Normally distributed continuous variables were expressed as mean ± SD and compared using independent *t*-tests; skewed variables as median (Q1, Q3) with Mann–Whitney *U* tests. Categorical variables as *n* (%) with chi-square or Fisher's exact tests. Two-sided $P < 0.05$ was considered significant.

Multivariable logistic regression was used to construct four models upon a base model (age, sex, tracheostomy, vasopressors): Model A (base + Braden, primary model); Model B (Model A + APACHE II, SOFA, albumin, CRP, ventilation duration, fully adjusted); Model E (base + APACHE II, without Braden); and Model F (base + APACHE II + Braden). Events per variable (EPV) = 99/5 = 19.8, meeting the EPV ≥ 10 requirement [16].

ROC/AUC assessed discrimination; the DeLong test compared AUCs [20]. DCA evaluated net benefit [17]. NRI and IDI assessed incremental value for two comparisons: (1) Model A vs. base; (2) Model F vs. Model E. Bootstrap with 1,000 iterations assessed model stability (optimism-corrected AUC, OR direction stability) [22]. Subgroup analysis was stratified by age (< 70/≥70), sex, APACHE II (< 20/≥20), tracheostomy, and vasopressor use, using Model A covariates with automatic removal of non-varying variables and Benjamini–Hochberg FDR correction. The Hosmer–Lemeshow test assessed calibration

[21]. VIF assessed multicollinearity (VIF < 5 = no collinearity) [18]. Sensitivity analysis: Braden stratified (≤ 12/13–14/≥15), Cochran–Armitage and logistic trend tests.

Results

Baseline Characteristics

A total of 184 patients were included: weaning success 85 (46.2%), weaning failure 99 (53.8%). No missing data were present. Baseline characteristics are shown in Table 1. No significant differences between groups were observed in age, sex, BMI, smoking, or comorbidities ($P > 0.05$). The failure group had significantly higher APACHE II [26.10 ± 7.04 vs. 18.75 ± 6.15, $t = -7.56$, $P < 0.001$], SOFA [8.0 (6.0, 10.0) vs. 6.0 (4.0, 8.0), $Z = -4.012$, $P < 0.001$], tracheostomy rate [43.4% vs. 21.2%, $\chi^2 = 9.24$, $P = 0.002$], vasopressor use [68.7% vs. 32.9%, $\chi^2 = 22.01$, $P < 0.001$], and longer ventilation duration [9.0 (4.0, 17.0) vs. 7.0 (4.0, 10.0) d, $Z = -2.01$, $P = 0.045$]. Braden score was significantly lower in the failure group [12.0 (10.0, 13.0) vs. 12.0 (11.0, 14.0), $Z = -3.196$, $P = 0.001$].

Table 1
Baseline characteristics of the study population.

Variable	Weaning success (n = 85)	Weaning failure (n = 99)	Statistic	*P* value
Age (years), median (Q1, Q3)	80.0 (66.0, 85.0)	81.0 (73.0, 86.0)	$U = 3802$	0.260
Male sex, n (%)	53 (62.4)	62 (62.6)	$\chi^2 = 0.00$	1.000
BMI (kg/m^2), mean ± SD	22.56 ± 3.19	21.76 ± 3.35	$t = 1.66$	0.098
Smoking history, n (%)	38 (44.7)	52 (52.5)	$\chi^2 = 0.83$	0.363
Hypertension, n (%)	47 (55.3)	63 (63.6)	$\chi^2 = 1.00$	0.317
Diabetes, n (%)	27 (31.8)	28 (28.3)	$\chi^2 = 0.12$	0.724
COPD, n (%)	23 (27.1)	28 (28.3)	$\chi^2 = 0.00$	0.984
APACHE II, mean ± SD	18.75 ± 6.15	26.10 ± 7.04	$t = -7.56$	< 0.001
SOFA, median (Q1, Q3)	6.0 (4.0, 8.0)	8.0 (6.0, 10.0)	$Z = -4.012$	< 0.001
Albumin (g/L), median (Q1, Q3)	31.1 (29.2, 34.5)	30.4 (26.9, 33.7)	$U = 4734$	0.145
CRP (mg/L), median (Q1, Q3)	77.1 (39.6, 175.8)	104.8 (47.4, 182.1)	$U = 3820$	0.283
PCT (ng/mL), median (Q1, Q3)	2.2 (0.3, 6.1)	1.2 (0.4, 5.8)	$U = 4420$	0.556
WBC (×10^9/L), median (Q1, Q3)	9.7 (6.2, 13.6)	10.7 (6.9, 17.9)	$U = 3618$	0.102
NLR, median (Q1, Q3)	11.3 (6.7, 21.2)	12.0 (6.7, 24.1)	$U = 4080$	0.725
Platelets (×10^9/L), median (Q1, Q3)	188.0 (133.0, 288.0)	194.0 (135.0, 286.0)	$U = 4106$	0.780
Tracheostomy, n (%)	18 (21.2)	43 (43.4)	$\chi^2 = 9.24$	0.002
Vasopressor use, n (%)	28 (32.9)	68 (68.7)	$\chi^2 = 22.01$	< 0.001
Ventilation duration (d), median (Q1, Q3)	7.0 (4.0, 10.0)	9.0 (4.0, 17.0)	$U = 3486$	0.045
ICU stay (d), median (Q1, Q3)	14.0 (8.0, 23.0)	12.0 (7.0, 23.0)	$U = 4427$	0.543
Braden score, median (Q1, Q3)	12.0 (11.0, 14.0)	12.0 (10.0, 13.0)	$U = 5345$	0.001

SD, standard deviation; BMI, body mass index; COPD, chronic obstructive pulmonary disease; APACHE II, Acute Physiology and Chronic Health Evaluation II; SOFA, Sequential Organ Failure Assessment; CRP, C-reactive protein; PCT, procalcitonin; WBC, white blood cell; NLR, neutrophil-to-lymphocyte ratio.

Continuous variables: mean ± SD analyzed by independent *t*-test; median (Q1, Q3) analyzed by Mann-Whitney *U* test. Categorical variables analyzed by χ^2 test.

Braden Score Stratification and Weaning Failure Rate

Patients were stratified by Braden score: ≤12 (high risk, *n* = 114), 13–14 (moderate risk, *n* = 52), ≥ 15 (low risk, *n* = 18). Weaning failure rates were 58.8% (67/114), 50.0% (26/52), and 33.3% (6/18), showing a decreasing trend. Cochran–Armitage trend test *P* = 0.030, logistic trend test *P* = 0.041 (Table 2, Fig. 1). The absolute risk difference between the ≤ 12 and ≥ 15 strata was 25.5%, with a relative risk ratio of 1.76.

Table 2
Weaning failure rate stratified by Braden score.

Braden stratum	*n*	Failure count	Failure rate (%)	Braden median
≤ 12 High risk	114	67	58.8	11.0
13–14 Moderate risk	52	26	50.0	13.0
≥ 15 Low risk	18	6	33.3	15.5

Cochran-Armitage trend test *P* = 0.030; logistic trend test *P* = 0.041. Absolute risk difference between ≤ 12 and ≥ 15 strata = 25.5%; relative risk ratio = 1.76.

Multivariable Logistic Regression

In Model A (base + Braden), Braden score was an independent protective factor for weaning failure: each 1-point increase reduced the failure risk by 23% (*OR* = 0.77, 95% *CI* 0.65–0.92, *P* = 0.004). Tracheostomy (*OR* = 3.35, *P* = 0.001) and vasopressor use (*OR* = 4.16, *P* < 0.001) were independent risk factors. In Model E (base + APACHE II), APACHE II was a strong predictor (*OR* = 1.15, 95% *CI* 1.08–1.22, *P* < 0.001, AUC = 0.821). In Model F (base + APACHE II + Braden), the Braden score effect attenuated (*OR* = 0.84, 95% *CI* 0.69–1.02, *P* = 0.073), while APACHE II remained significant (*OR* = 1.14, *P* < 0.001, AUC = 0.829) (Table 3).

Table 3
Multivariable logistic regression results for weaning failure.

Model	Variable	β	*OR*	95% *CI*	*P* value
Model A (AUC = 0.774)	Braden	−0.261	0.77	0.65–0.92	0.004
	Age	0.015	1.02	0.99–1.04	0.300
	Male	0.109	1.12	0.56–2.22	0.757
	Tracheostomy	1.210	3.35	1.61–6.99	0.001
	Vasopressors	1.426	4.16	2.15–8.07	< 0.001
Model E (AUC = 0.821)	Age	0.007	1.01	0.98–1.04	0.677
	Male	−0.059	0.94	0.46–1.94	0.873
	Tracheostomy	0.999	2.72	1.29–5.72	0.009
	Vasopressors	0.753	2.12	1.01–4.45	0.046
	APACHE II	0.139	1.15	1.08–1.22	< 0.001
Model F (AUC = 0.829)	Braden	−0.175	0.84	0.69–1.02	0.073
	Age	0.003	1.00	0.97–1.03	0.843
	Male	0.008	1.01	0.49–2.10	0.983
	Tracheostomy	1.071	2.92	1.36–6.25	0.006
	Vasopressors	0.745	2.11	1.00-4.45	0.051
	APACHE II	0.128	1.14	1.07–1.21	< 0.001
Model B (AUC = 0.832)	Braden	−0.175	0.84	0.69–1.02	0.076
	Tracheostomy	1.098	3.00	1.28–7.02	0.011
	APACHE II	0.106	1.11	1.01–1.22	0.023
	SOFA	0.069	1.07	0.84–1.36	0.570
	Albumin	−0.023	0.98	0.91–1.05	0.503
	CRP	−0.000	1.00	1.00–1.00	0.862
	Vent duration	−0.001	1.00	0.97–1.03	0.947

Base model AUC = 0.745; Braden alone AUC = 0.635; APACHE II alone AUC = 0.791. Model A = base+Braden; Model E = base+APACHE II; Model F = base+APACHE II+Braden; Model B = fully adjusted (Model A + APACHE II, SOFA, albumin, CRP, ventilation duration). β, regression coefficient; *OR*, odds ratio; *CI*, confidence interval; Vent duration, mechanical ventilation duration.

Incremental Predictive Value

Table 4 and Figs. 2–3 summarize the incremental predictive value. Braden vs. base model: ΔAUC = 0.029 (*P* = 0.042), continuous NRI = 0.389 (*P* = 0.032), IDI = 0.044 (*P* = 0.004) — all significant, indicating that Braden provides additional discrimination information. Braden vs. APACHE II model: ΔAUC = 0.009 (*P* = 0.224), NRI = 0.157 (*P* = 0.218) — not significant; IDI = 0.014 (*P* = 0.032) — significant but minimal. The ΔAUC of 0.009 is far below the clinically meaningful threshold (0.025–0.050) [17]. APACHE II alone (AUC = 0.791) exceeded Model A (0.774). DCA showed that APACHE II-containing models (E/F) had superior net benefit over Model A in the 30–50% threshold probability range.

Table 4
Incremental predictive value of Braden score.

Comparison	New model AUC	Old model AUC	ΔAUC	ΔAUC *P*	NRI	NRI *P*	IDI	IDI *P*
Model A vs Base model	0.774	0.745	+ 0.029	0.042	+ 0.389	0.032	+ 0.044	0.004
Model F vs Model E	0.829	0.821	+ 0.009	0.224	+ 0.157	0.218	+ 0.014	0.032

NRI, net reclassification improvement; IDI, integrated discrimination improvement. Model A = base+Braden; Model E = base+APACHE II; Model F = base+APACHE II+Braden. The clinically meaningful ΔAUC threshold is 0.025–0.050 [17].

Bootstrap Internal Validation

Bootstrap internal validation results are presented in Table 5. Optimism was only 0.020 (~ 2.6% relative attenuation), indicating acceptable model stability. The Braden *OR* median was 0.76 (95% *CI* 0.63–0.91), with 99.9% of iterations showing *OR* < 1, demonstrating excellent effect direction stability.

Table 5
Bootstrap internal validation of Model A (1,000 iterations).

Metric	Estimate	95% *CI* lower	95% *CI* upper	*OR* < 1 proportion
Original AUC	0.774	—	—	—
Bootstrap apparent AUC	0.785	0.708	0.849	—
Bootstrap test AUC	0.765	0.741	0.777	—
Optimism	0.020	—	—	—
Optimism-corrected AUC	0.754	—	—	—
Braden *OR* (Bootstrap)	0.76	0.63	0.91	99.9%

AUC, area under the receiver operating characteristic curve; *OR*, odds ratio. Optimism = apparent AUC − test AUC; optimism-corrected AUC = original AUC − optimism. Relative attenuation = 0.020/0.774 ≈ 2.6%.

Calibration and Multicollinearity

For Model A, the Hosmer–Lemeshow $\chi^2 = 16.60$, $P = 0.035$ (calibration suboptimal). For Model F, $\chi^2 = 11.24$, $P = 0.188$ (good calibration). VIF analysis: Braden VIF = 1.18 (no collinearity); APACHE II VIF = 3.60, SOFA VIF = 3.50 (moderate correlation but below the threshold) (Table 6).

Table 6
Variance inflation factor (VIF) analysis for multicollinearity.

Variable	VIF	Assessment
Braden score	1.18	No collinearity
APACHE II	3.60	No collinearity
SOFA	3.50	No collinearity
Albumin	1.12	No collinearity
Age	1.13	No collinearity
Sex	1.03	No collinearity
Tracheostomy	1.04	No collinearity
Vasopressors	1.37	No collinearity

VIF, variance inflation factor. VIF < 5 indicates no multicollinearity [18].

Subgroup Analysis

Figure 5 and Table 7 present the subgroup analysis. Of 11 predefined subgroups, 4 remained significant after FDR correction (FDR $P < 0.05$): overall (*OR* = 0.77, FDR $P = 0.034$), age ≥ 70 (*OR* = 0.77, FDR $P = 0.047$), APACHE II ≥ 20 (*OR* = 0.77, FDR $P = 0.047$), and tracheostomy (*OR* = 0.60, FDR $P = 0.034$). Five subgroups were nominally significant but did not survive FDR correction ($0.05 <$ FDR $P < 0.10$).

Table 7
Subgroup analysis of the association between Braden score and weaning failure (Model A covariates).

Subgroup	*n*	Failures	*OR*	95% *CI*	*P* value	FDR *P*	Removed variables
Overall	184	99	0.77	0.65–0.92	0.004	0.034	None
Age < 70	41	16	0.68	0.46-1.00	0.048	0.066	None
Age ≥ 70	143	83	0.77	0.63–0.95	0.015	0.047	None
Male	115	62	0.81	0.66–0.99	0.043	0.066	Sex
Female	69	37	0.67	0.47–0.96	0.029	0.054	Sex
APACHE II < 20	62	14	0.81	0.52–1.26	0.357	0.357	None
APACHE II ≥ 20	122	85	0.77	0.62–0.96	0.017	0.047	None
No tracheostomy	123	56	0.87	0.70–1.07	0.186	0.205	Tracheostomy
Tracheostomy	61	43	0.60	0.42–0.87	0.006	0.034	Tracheostomy
No vasopressors	88	31	0.80	0.62–1.02	0.068	0.083	Vasopressors
Vasopressors	96	68	0.74	0.57–0.96	0.024	0.053	Vasopressors

FDR, false discovery rate (Benjamini-Hochberg correction). Bold FDR *P* values indicate significance after correction (FDR $P < 0.05$). Non-varying variables were automatically removed within each subgroup. Age < 70 subgroup: $n = 41$, failures = 16, EPV ≈ 4.0, limiting estimate stability [16].

Discussion

This study found that the Braden score is an independent protective factor for weaning failure in mechanically ventilated patients with severe pneumonia, with each 1-point increase reducing failure risk by 23% (*OR* = 0.77, 95% *CI* 0.65–0.92, $P = 0.004$). The biological basis for this association lies in what the Braden Scale actually measures. Although designed for pressure ulcer risk, its dimensions converge on several physiological domains directly relevant to weaning capacity. Activity and mobility reflect the patient's overall physical functioning, and their decline mirrors the ICU-acquired weakness and diaphragmatic dysfunction that frequently complicate prolonged mechanical ventilation [2, 13, 26]. The nutrition dimension, meanwhile, captures a domain where hypoalbuminemia and malnutrition have long been recognized as independent risk factors for weaning failure [4, 5, 24]. Sensory perception, which reflects consciousness and neurological integrity, is itself a determinant of spontaneous breathing trial success [3]. That a single bedside score integrating these domains predicts weaning outcomes is

therefore less surprising than it might initially appear — the Braden Scale inadvertently captures a composite picture of the patient's readiness to breathe without mechanical support.

The dose-response relationship observed in stratified analysis adds weight to this association. The weaning failure rate decreased progressively from 58.8% in the ≤ 12 (high-risk) stratum to 33.3% in the ≥ 15 (low-risk) stratum (Cochran-Armitage trend $P = 0.030$, logistic trend $P = 0.041$), with an absolute risk difference of 25.5% and a relative risk ratio of 1.76 between the extreme strata. Subgroup analysis revealed that 4 of 11 predefined subgroups remained significant after Benjamini-Hochberg FDR correction (FDR $P < 0.05$): the overall population, age ≥ 70, APACHE II ≥ 20, and tracheostomy subgroups. The tracheostomy subgroup demonstrated the strongest protective effect ($OR = 0.60$, FDR $P = 0.034$), suggesting that Braden score may be particularly informative among patients with tracheostomy, who often have more prolonged illness courses and greater functional impairment [25]. The age < 70 subgroup included only 41 patients with 16 events, yielding an EPV of approximately 4.0, which substantially limits the stability of estimates in this subgroup [16]; the nominally significant result ($OR = 0.68$, $P = 0.048$) did not survive FDR correction and should be interpreted with caution.

Whether the Braden Scale adds predictive information beyond what disease severity scores already capture is a question with direct clinical implications but has not been previously addressed with rigorous methodology. This study is, to our knowledge, the first to answer this question using NRI, IDI, and DCA in a mechanically ventilated population, and the findings are clear: the incremental predictive value of Braden score beyond APACHE II is limited. Although Braden score was a significant independent predictor in Model A (base+Braden, $OR = 0.77$, $P = 0.004$), its effect attenuated substantially after adjustment for APACHE II (Model F: $OR = 0.84$, $P = 0.073$), and the ΔAUC of Model F versus Model E was only 0.009 ($P = 0.224$), far below the clinically meaningful threshold of 0.025–0.050 [17]. APACHE II alone achieved an AUC of 0.791, exceeding Model A (0.774), and APACHE II-containing models (E and F) demonstrated superior net benefit over Model A in decision curve analysis within the 30–50% threshold probability range. These findings indicate substantial information overlap between Braden score and disease severity. Multicollinearity was excluded (Braden VIF = 1.18), confirming that the attenuation reflects shared predictive information rather than statistical collinearity. This is consistent with the findings of Pan et al. [4], who identified disease severity and nutritional status as dominant predictors of weaning difficulty, and with recent systematic reviews highlighting the importance of bedside functional indicators in predicting weaning outcomes [23]. The Braden Scale captures functional consequences of severe illness that are also reflected, albeit through different parameters, in APACHE II — explaining the limited additional discrimination. While this finding may appear negative, it is clinically informative: it definitively establishes that the Braden Scale cannot replace disease severity scores, and it redirects attention toward identifying the specific clinical contexts where Braden may still add value.

What, then, is the clinical role of the Braden Scale given its limited incremental value beyond APACHE II? The answer lies in practicality. APACHE II requires multiple laboratory parameters and complex computation, whereas the Braden Scale can be completed at the bedside within minutes by clinical staff without additional equipment, making it suitable for repeated daily assessment. The clear risk

stratification observed in this study — a 25.5% absolute difference in failure rate between the highest and lowest Braden strata — provides actionable thresholds for bedside decision-making. We therefore position the Braden Scale not as a competitor to disease severity scores but as a practical complement: its primary value lies in dynamic monitoring, where serial assessments can detect functional deterioration early and prompt timely clinical interventions [4]. The prognostic utility of the Braden Scale has been increasingly recognized beyond pressure ulcer prediction, including its association with mortality in ICU patients with heart failure [28], supporting its broader role as a comprehensive functional status indicator. The Braden dimensions also directly inform targeted interventions: the activity and mobility dimensions align with evidence that early physical and occupational therapy improves functional outcomes in mechanically ventilated patients [10], while the nutrition dimension connects to established nutritional interventions for weaning optimization. Integrating Braden-guided assessment with early mobilization, nutritional support, and sedation management [27] may optimize weaning outcomes through a multidimensional approach.

Several limitations should be acknowledged. This was a single-center retrospective study with a sample size of 184 patients, and no external validation was performed, limiting generalizability. The calibration of Model A was suboptimal (Hosmer-Lemeshow $P = 0.035$), suggesting potential overfitting or miscalibration that warrants further validation. Although bootstrap internal validation showed acceptable stability (optimism = 0.020, optimism-corrected AUC = 0.754), this represents only internal validation. The ≥ 15 Braden stratum included only 18 patients, limiting the precision of estimates in the low-risk category, and the age < 70 subgroup had an EPV of approximately 4.0, well below the recommended threshold [16], compromising estimate stability. This study analyzed the total Braden score without dimension-level analysis, precluding identification of which specific dimensions drive the association. Diaphragmatic ultrasound and RSBI were not included as comparators [15], and only baseline Braden scores were analyzed, without trend analysis of serial measurements. Future studies should adopt multicenter prospective designs, perform external validation, conduct dimension-level analyses, and incorporate serial Braden trajectories.

Conclusions

The Braden Scale is independently associated with mechanical ventilation weaning failure in severe pneumonia ($OR = 0.77$, 95% CI 0.65–0.92, $P = 0.004$), with a dose-response relationship across risk strata. Its incremental predictive value beyond APACHE II is limited, confirming that the two measures capture overlapping rather than complementary prognostic information. The Braden Scale is best positioned as a practical bedside tool that complements disease severity scores through rapid, repeatable functional assessment, with particular promise in tracheostomized patients where it demonstrated its strongest effect. Multicenter prospective studies with external validation are needed to confirm these findings and explore dimension-level contributions.

Abbreviations

APACHE II: Acute Physiology and Chronic Health Evaluation II; ARDS: Acute Respiratory Distress Syndrome; ATS: American Thoracic Society; AUC: Area Under the Curve; BMI: Body Mass Index; CI: Confidence Interval; COPD: Chronic Obstructive Pulmonary Disease; CRP: C-Reactive Protein; DCA: Decision Curve Analysis; EPV: Events Per Variable; FDR: False Discovery Rate; HL: Hosmer-Lemeshow; ICU: Intensive Care Unit; ICU-AW: ICU-Acquired Weakness; IDI: Integrated Discrimination Improvement; IDSA: Infectious Diseases Society of America; IQR: Interquartile Range; NLR: Neutrophil-to-Lymphocyte Ratio; NRI: Net Reclassification Improvement; OR: Odds Ratio; PCT: Procalcitonin; ROC: Receiver Operating Characteristic; RSBI: Rapid Shallow Breathing Index; SBT: Spontaneous Breathing Trial; SD: Standard Deviation; SOFA: Sequential Organ Failure Assessment; STROBE: Strengthening the Reporting of Observational Studies in Epidemiology; VAP: Ventilator-Associated Pneumonia; VIF: Variance Inflation Factor; WBC: White Blood Cell; WIND: Weaning according to a New Definition

Declarations

This study was reported following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies. A completed STROBE checklist is available as Supplementary Material.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of The Second People's Hospital of Hefei (Approval No.: 2026-ky-053). Written informed consent was waived by the Ethics Committee due to the retrospective nature of the study and the use of de-identified data from existing medical records.

Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any form (including individual details, images, or videos).

Availability of data and materials

The datasets generated and analyzed during the current study are not publicly available due to patient privacy regulations but are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

ML and YW contributed equally to this work. ML and YW designed the study, performed the statistical analysis, and drafted the manuscript. XF, JZ, and ZH collected the data and performed quality control. LY conceived the study, supervised the project, and revised the manuscript. All authors have read and approved the final manuscript.

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Figures

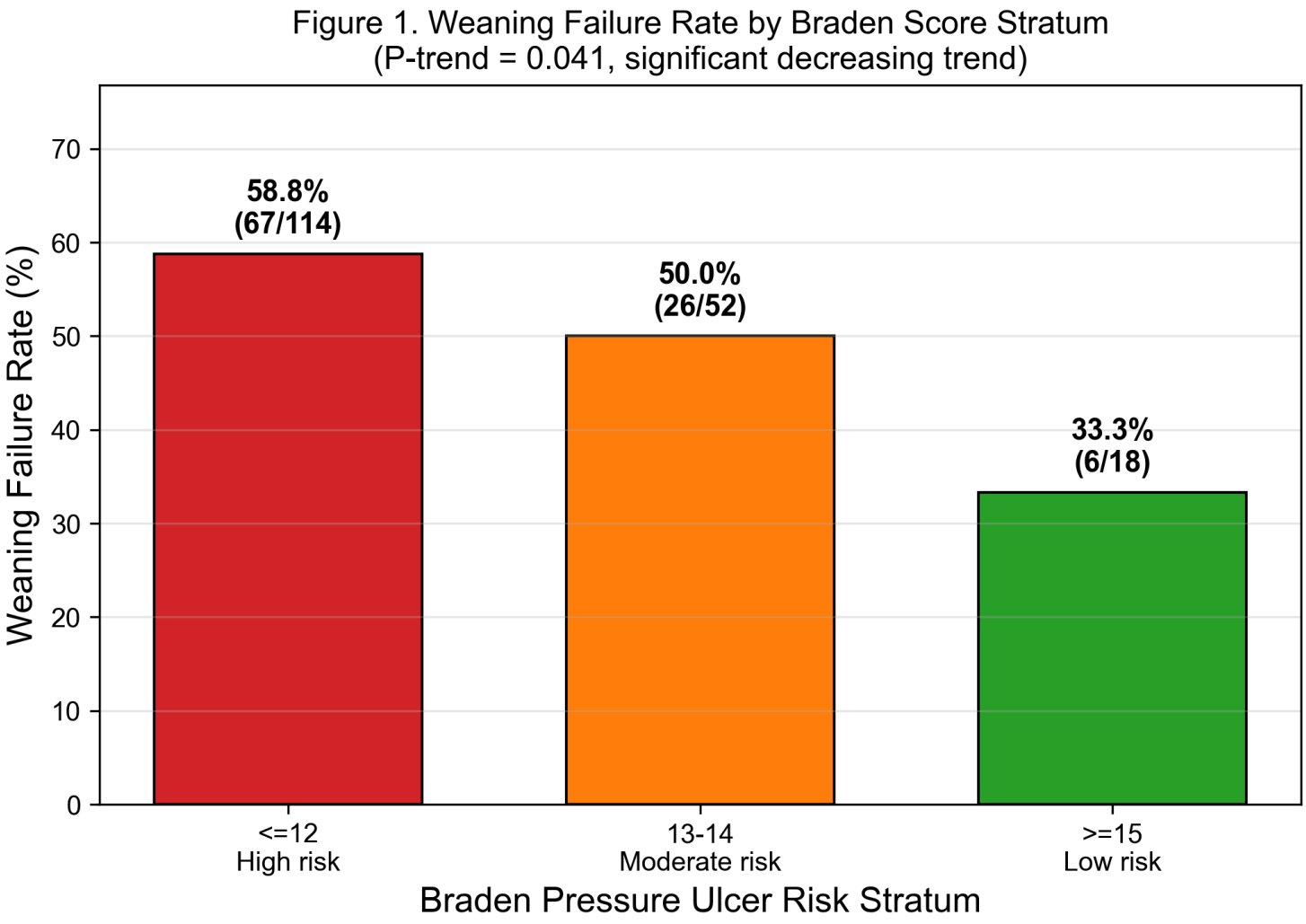


Figure 1

Weaning failure rate stratified by Braden score. Failure rates decreased progressively from 58.8% (≤12) to 50.0% (13-14) to 33.3% (≥15), with a significant decreasing trend (Cochran-Armitage *P*=0.030; logistic trend *P*=0.041).

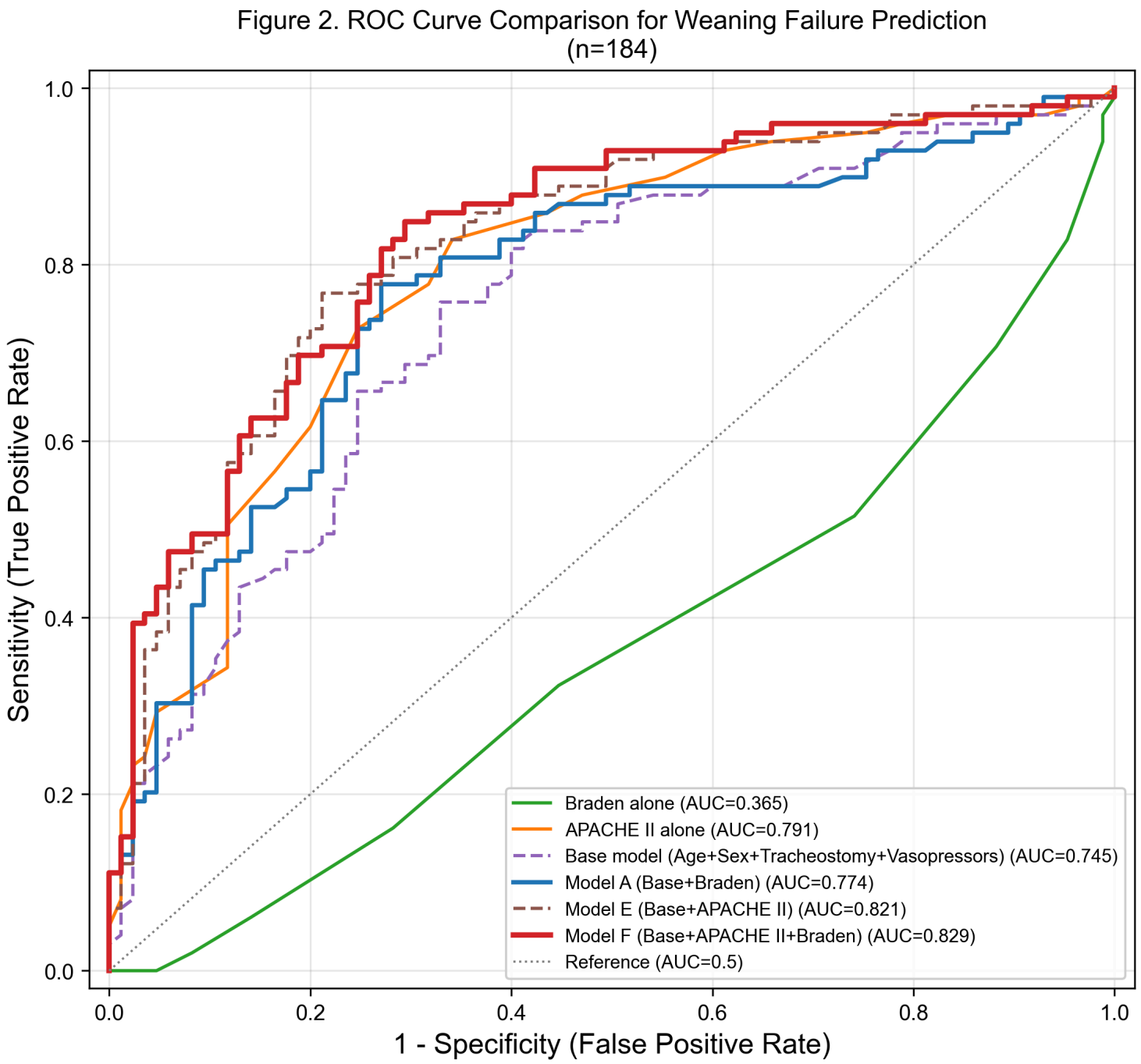


Figure 2

Receiver operating characteristic (ROC) curves for predictive models. Model A (base+Braden, AUC=0.774) significantly improved discrimination over the base model (AUC=0.745; ΔAUC=0.029, *P*=0.042). Model F (base+APACHE II+Braden, AUC=0.829) showed limited incremental value over Model E (base+APACHE II, AUC=0.821; ΔAUC=0.009, *P*=0.224). APACHE II alone (AUC=0.791) exceeded Model A.

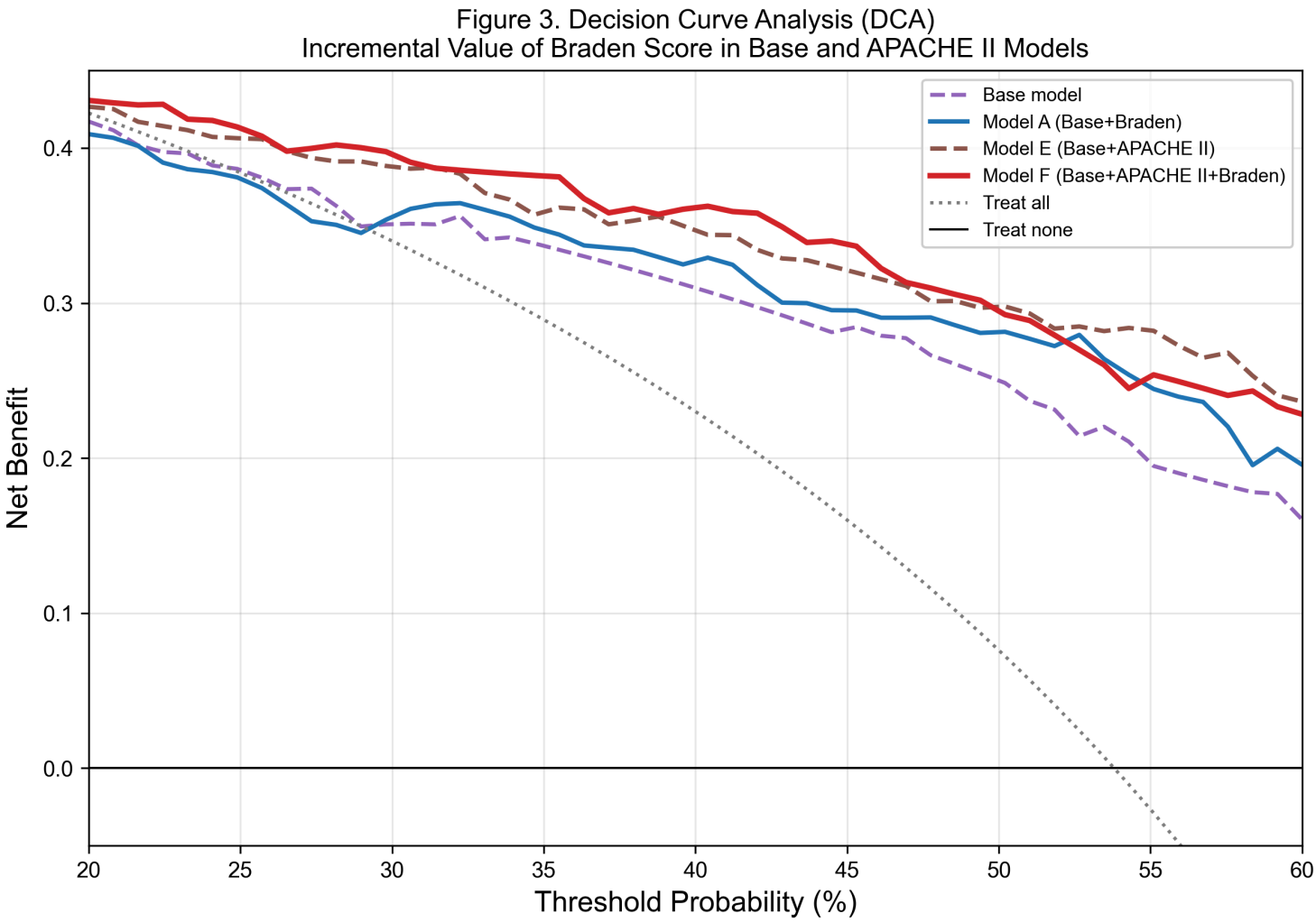


Figure 3

Decision curve analysis (DCA). APACHE II-containing models (Model E and Model F) demonstrated superior net benefit over Model A within the 30-50% threshold probability range, indicating that adding APACHE II yields greater clinical utility than adding Braden alone.

Figure 4. Calibration Curve Comparison

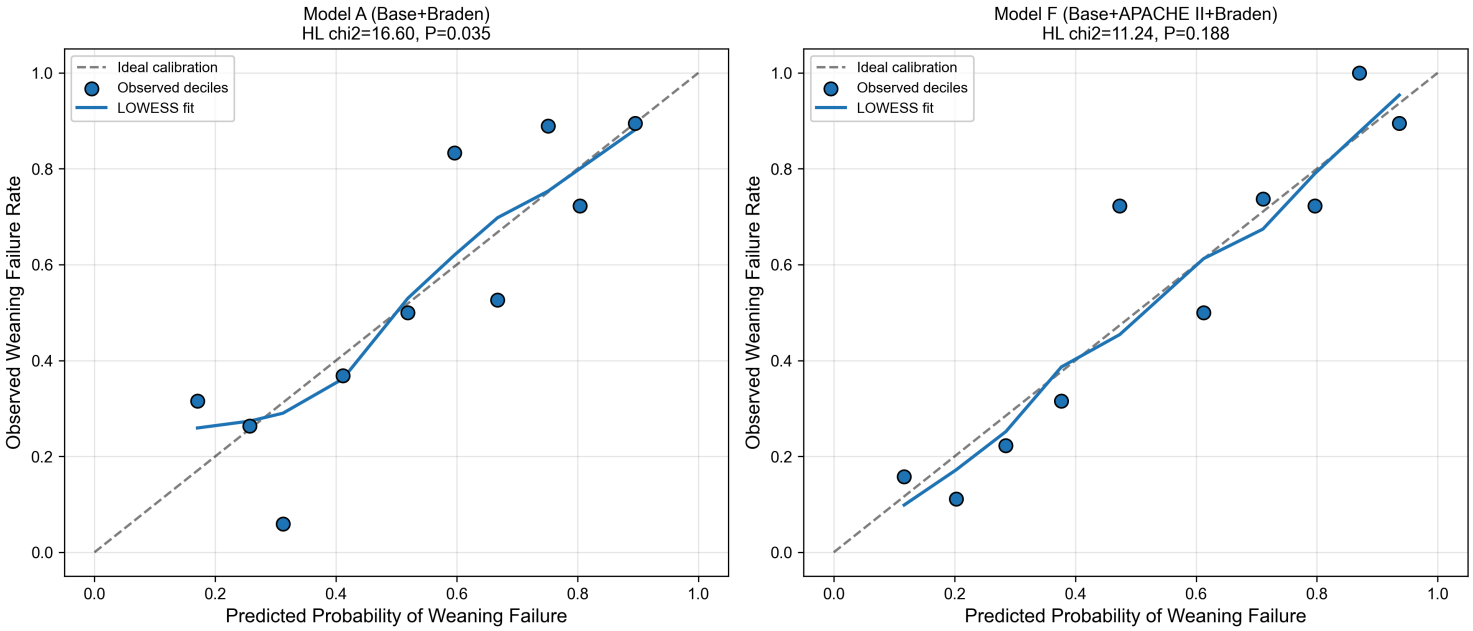


Figure 4

Calibration curve for Model A. The Hosmer-Lemeshow goodness-of-fit test indicated suboptimal calibration (χ^2=16.60, *P*=0.035), suggesting modest overfitting. Model F showed better calibration (χ^2=11.24, *P*=0.188).

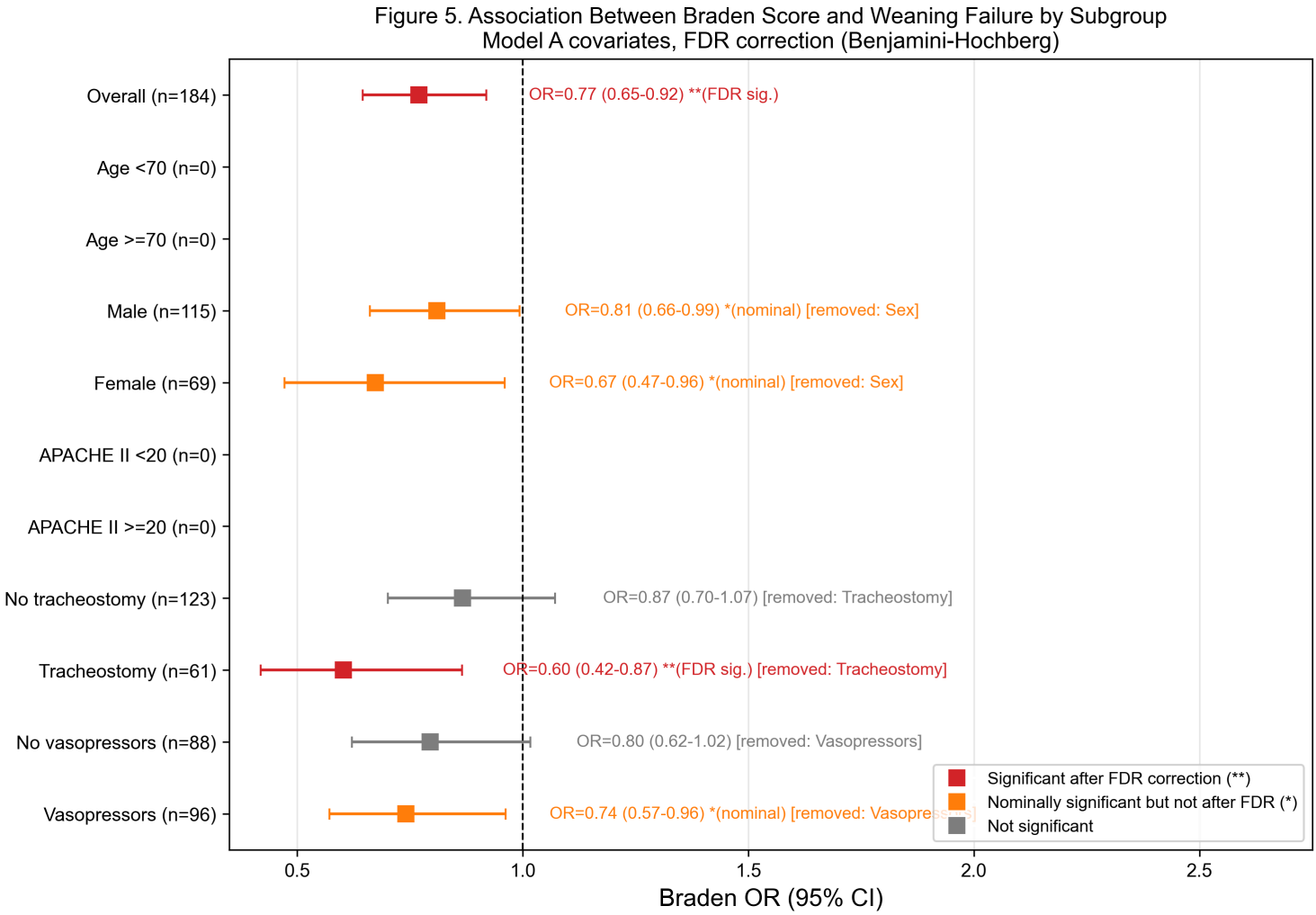


Figure 5

Forest plot of subgroup analysis for the association between Braden score and weaning failure. Four subgroups remained significant after Benjamini-Hochberg FDR correction (FDR *P*<0.05): overall, age ≥70, APACHE II ≥20, and tracheostomy. The tracheostomy subgroup showed the strongest protective effect (*OR*=0.60, FDR *P*=0.034).