



Correlation of Carbohydrate and Fat Intake with PaCO₂ Levels in Critically Ill Patients Receiving Enteral Feeding

Ayling Lie Pannadewi^{1*} and Anggun Rindang Cempaka¹ 

¹Department of Nutrition, Faculty of Health Sciences, Universitas Brawijaya, Malang, Indonesia
aylinglie@student.ub.ac.id

Abstract. Critically ill patients admitted to the Intensive Care Unit (ICU) often experience metabolic changes that affect acid-base balance and blood gas levels, including the partial pressure of carbon dioxide (PaCO₂). Carbohydrates and fats are the main sources of nonprotein calories that contribute to CO₂ production through metabolic processes. This study aimed to explore the correlation between carbohydrate and fat intake and PaCO₂ levels in critically ill patients receiving enteral feeding at Dr. Saiful Anwar General Hospital, East Java Province, Indonesia. This study employed an observational, cross-sectional design (n = 20). Data on carbohydrate and fat intake were collected from multiple 24-hour enteral feeding records, while PaCO₂ levels were measured via arterial blood gas analysis. The correlation was assessed with Pearson correlation test, and mean differences were examined using One-Way ANOVA. Results indicated that the average carbohydrate intake was 157.32±46.61 g, with an adequacy of 77.92±26.23%, and fat intake was 40.11±12.73 g, with an adequacy of 82.66±30.10%. No significant association was found between PaCO₂ levels and either carbohydrate or fat intake and adequacy (p > 0.05). This study concludes that carbohydrate and fat intake are not significantly associated with PaCO₂ levels in critically ill patients receiving enteral feeding. Further study is recommended to investigate macronutrient intake in critically ill patients with stricter control of respiratory variables.

Keywords: Carbohydrate Intake, Fat Intake, PaCO₂, Critically Ill Patients, Enteral Feeding

1 Introduction

Critically ill patients are individuals experiencing life-threatening conditions characterized by dysfunction of vital organs and a high risk of rapid clinical deterioration [1]. In Indonesia, the prevalence of critically ill patients has increased substantially, from 33,148 cases in 2019 to 52,719 in 2021, with an average intensive care unit (ICU) occupancy rate of 64.83% [2]. This growing burden is accompanied by high mortality rates in intensive care units. A five-year retrospective cohort study by Melaku et al. (2024) reported an overall ICU mortality rate of 35.9%, with nearly half of all deaths

attributed to septic shock, congestive heart failure, severe community-acquired pneumonia, and stroke [3]. Similarly, Munawaroh et al. (2024) reported an ICU mortality rate of 75.9% at Aisyiyah Islamic Hospital, Malang, in 2023 [4]. A considerable proportion of these patients develop respiratory failure and require mechanical ventilation, highlighting the importance of appropriate management strategies to preserve organ function and improve clinical outcomes [5].

Critically ill patients commonly experience metabolic stress characterized by hypermetabolism and hypercatabolism, leading to increased energy requirements [6]. Adequate nutritional support, delivered either enterally or parenterally, is essential to promote anabolic responses and improve recovery [7]. However, determining the optimal macronutrient composition remains challenging, as the metabolism of carbohydrates, fats, and proteins produces carbon dioxide (CO₂) as a byproduct, albeit in different amounts. Carbohydrates have the highest respiratory quotient (RQ $\approx$ 1.0), compared with protein ($\approx$ 0.8) and fat ($\approx$ 0.7), indicating that higher carbohydrate intake may increase CO₂ production and, consequently, respiratory workload [8], [9]. This issue is particularly relevant in patients with impaired pulmonary function.

The impact of metabolic processes on respiratory function can be assessed through arterial partial pressure of carbon dioxide (PaCO₂), which is considered the gold standard indicator of alveolar ventilation efficiency [10]. Under normal physiological conditions, PaCO₂ is maintained within the range of 35–45 mmHg. However, excessive CO₂ production that is not adequately compensated by ventilation may lead to hypercapnia, potentially worsening respiratory failure and prolonging the duration of mechanical ventilation [11], [12].

Although the concept of respiratory quotient provides a theoretical basis for the relationship between diet and CO₂ production, clinical evidence regarding the effect of macronutrient intake on PaCO₂ remains inconsistent. For example, Nourmohammadi et al. (2022) demonstrated that a low-carbohydrate, high-fat diet significantly reduced PaCO₂ levels, whereas other studies have reported no significant effect of macronutrient ratios in patients with hypercapnic respiratory failure [13], [14]. These discrepancies, along with the limited number of studies evaluating specific enteral formulas in local populations, highlight the need for further investigation. Therefore, this study aims to analyze the correlation between carbohydrate and fat intake and PaCO₂ levels in critically ill patients diagnosed with acute respiratory failure (ARF) and pneumonia who receive enteral feeding.

2 Methods

This study employed an analytical observational design with a cross-sectional approach and was conducted in the Intensive Care Unit (ICU) of Dr. Saiful Anwar Hospital, East Java Province, Indonesia, from July to September 2025. A total sampling technique was employed, whereby all eligible critically ill patients admitted to the ICU over the three-month study period who met the inclusion and exclusion criteria were included. As in previous single-center observational studies involving mechanically ventilated critically ill patients [15], eligibility was restricted to patients with specific clinical

characteristics and complete clinical data. The final sample consisted of 20 participants, representing the entire accessible study population during the study period. Ethical approval was obtained from the Health Research Ethics Committee of Dr. Saiful Anwar Hospital (Approval No. 400/171/K.3/120.7/2025).

Inclusion criteria comprised adult critically ill patients (≥ 18 years) receiving mechanical ventilation, diagnosed with acute respiratory failure (ARF) or pneumonia, receiving enteral nutrition (non-protein calorie formula), and having at least three recorded PaCO_2 measurements during ICU stay. Patients with recent shock (within 24 hours), defined by hemodynamic instability or vasopressor use, or those with incomplete data were excluded.

Baseline anthropometric data, including body weight and height, were obtained from the initial measurements documented in the patients' medical records. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2), and nutritional status was classified according to the World Health Organization (WHO) Asia-Pacific BMI classification [16].

Dietary intake data, including carbohydrate and fat intake, were obtained from two 24-hour enteral and parenteral feeding records documented in the hospital nutrition monitoring forms, each representing the patients' intake during the 24 hours preceding arterial blood gas (ABG) assessment. The mean values of the two dietary records were used for analysis. Arterial PaCO_2 levels were obtained from the second and third ABG measurements, and the mean of these two measurements was used to represent each patient's PaCO_2 level. Likewise, the mean values of the two dietary records were used for analysis.

Nutritional data were entered and processed using Microsoft Excel, and the statistical analyses were performed using IBM SPSS Statistics version 29.0. Carbohydrate and fat intake were analyzed as absolute intake (g/day) and adequacy percentage. Energy requirements were estimated at 25 kcal/kg body weight/day according to the ESPEN guideline for critically ill patients [17]. Target carbohydrate and fat requirements were determined as 55% and 30% of the estimated total energy requirement, respectively, based on standard macronutrient distribution recommendations [6]. Adequacy percentage was calculated as $(\text{actual intake} \div \text{estimated requirement}) \times 100\%$.

Descriptive statistics were used to summarize patient characteristics and nutrient intake distribution. The correlation between carbohydrate and fat intake and PaCO_2 levels was analyzed using the Pearson correlation test after confirming normal data distribution. In addition, differences in metabolic and nutritional variables across PaCO_2 categories were assessed using one-way ANOVA for normally distributed data and the Kruskal–Wallis test for non-normally distributed data, followed by Tukey HSD or appropriate non-parametric post hoc tests when applicable. Statistical significance was set at $p < 0.05$. All statistical analysis was performed by SPSS (Statistical Package for Social Sciences) version 29.0 (IBM Statistics, United States).

3 Results

3.1 Characteristics of Study Subjects

The primary characteristics of the study participants are shown in Table 1. This section summarizes the demographic and clinical profiles of the subjects, including age, sex, primary diagnosis, comorbidities, and nutritional status, to provide an overview of the study population.

Table 1. Characteristics of study subjects.

Characteristics	n	%
Sex		
Female	15	75
Male	5	25
Age		
19 - 44 years (Adults)	5	25
45 – 59 years (Middle-aged)	9	45
≥ 60 years (Elderly)	6	30
Current Medical Diagnosis		
ARF	1	5
Pneumonia	1	5
ARF + Pneumonia	18	90
Comorbidities		
Adenocarcinoma	1	5
Chronic asthma	1	5
Pulmonary contusion	2	10
COPD	1	5
Encephalopathy	1	5
Guillain-Barre Syndrome	1	5
Moderate ARDS	1	5
Moderate hypoxemia	1	5
Cerebral space-occupying lesion	1	5
Stroke	8	40
Head injury	2	10
Nutritional Status		
Normal	7	35
Overweight	5	25
Obesity class I	6	30
Obesity class II	2	10

ARF: acute respiratory failure, COPD: chronic obstructive pulmonary disease, ARDS: acute respiratory distress syndrome.

Among the 20 critically ill patients included, the majority were female (75%), with a mean age of 52.90 ± 15.14 years. Most participants were middle-aged (45%) or elderly (30%). Acute respiratory failure (ARF) combined with pneumonia was the predominant diagnosis (90%). Stroke was the most common comorbidity (40%), followed by head

injury (10%) and pulmonary contusion (10%). The mean body mass index (BMI) was $24.45 \pm 3.50 \text{ kg/m}^2$, with 35% of patients classified as having normal nutritional status, 25% as overweight, 30% as obesity class I, and 10% as obesity class II.

3.2 Carbohydrate and Fat Intake

Table 2 summarizes the estimated requirements, actual intake, and adequacy of carbohydrate and fat among the study participants. Values are presented as mean $\pm$ SD to reflect inter-individual variation.

Table 2. Carbohydrate and fat intake.

Intake	Mean Requirement (g)	Total Intake (g)	Adequacy (%)
Carb	203.50 $\pm$ 20.19	157.32 $\pm$ 46.61	77.92 $\pm$ 26.23
Fat	49.33 $\pm$ 4.90	40.11 $\pm$ 12.73	82.66 $\pm$ 30.10

Values are based on mean $\pm$ SD. Carb: carbohydrate.

Mean carbohydrate intake (157.32 $\pm$ 46.61 g) was lower than the estimated requirement (203.50 $\pm$ 20.19 g), with an adequacy level of 77.92 $\pm$ 26.23%. Similarly, mean fat intake (40.11 $\pm$ 12.73 g) was below the requirement (49.33 $\pm$ 4.90 g), with an adequacy of 82.66 $\pm$ 30.10%.

3.3 PaCO₂ Levels

PaCO₂ levels were monitored as the primary outcome variable. The mean PaCO₂ values obtained from the second and third arterial blood gas (ABG) measurements are presented in Table 3.

Table 3. Average PaCO₂ levels.

Variable	Measurement 1	Measurement 2
Mean PaCO ₂ (mmHg)	39.90 $\pm$ 10.78	38.83 $\pm$ 8.28

Values are based on mean $\pm$ SD.

As shown in Table 3, the mean PaCO₂ level decreased slightly from 39.90 $\pm$ 10.78 mmHg at the second measurement to 38.83 $\pm$ 8.28 mmHg at the third measurement.

3.4 Correlation of Carbohydrate, Fat, and PaCO₂

The relationship between nutritional status and respiratory outcomes was further examined using correlation analysis. This analysis evaluated the correlation between carbohydrate and fat intake and PaCO₂. The results, including the correlation coefficients (r) and p-values (p), are summarized in Table 4.

Pearson correlation analysis showed no significant correlation between carbohydrate intake and PaCO₂ (r = 0.065, p = 0.786) or between carbohydrate adequacy and PaCO₂ (r = 0.069, p = 0.771). Likewise, neither fat intake (r = 0.103, p = 0.666) nor fat adequacy (r = 0.111, p = 0.641) was significantly correlated with PaCO₂ levels. To further

evaluate differences according to PaCO₂ categories, trend and group comparison analyses were performed for energy intake, macronutrient intake, and metabolic parameters. The results are summarized in Table 5.

Table 4. Correlation of carbohydrate and fat intake with PaCO₂

Variable	Mean±SD	r	p
Carb intake (g)	157.32±46.61	0.065	0.786
Carb adequacy (%)	77.92±26.23	0.069	0.771
Fat intake (g)	40.11±12.73	0.103	0.666
Fat adequacy (%)	82.66±30.10	0.111	0.641

Values are based on mean±SD. Carb: carbohydrate

p-values < 0.05 are considered significant.

Table 5. Intake energy, carbohydrate, fat, and metabolic parameters across PaCO₂ categories

Variable	PaCO ₂ Categories			p trend	p-value
	Low (n=6)	Normal (n=9)	High (n=5)		
Intake					
Energy intake (kcal)	1094.60±309.50	911.03±347.29	1107.20±287.16	0.949	0.444
Energy adequacy (%)	76.34±24.68	59.59±22.04	78.24±28.71	0.899	0.302
Carbohydrate intake (g)	168.76±43.15	143.57±53.70	168.33±38.10	0.988	0.516
Carbohydrate adequacy (%)	85.63±26.02	68.15±24.73	86.27±28.43	0.968	0.339
Fat intake (g)	42.73±13.41	35.39±13.07	45.46±10.35	0.725	0.321
Fat adequacy (%)	89.70±32.74	70.49±25.54	96.11±31.84	0.723	0.257
Metabolic Profile					
Systolic BP (mmHg)	122.67±16.02	131.89±16.78	124.80±10.71	0.821	0.490
Diastolic BP (mmHg)	76.33±7.39	78.11±6.23	75.60±6.19	0.856	0.766
Hb (g/d)	10.77±2.50	11.18±2.48	10.76±2.30	0.996	0.930
PaO ₂ (mmHg)	122.75 (29.93)	108.55 (34.00)	86.00 (153.53)	0.707	0.419
HCO ₃ ⁻ (mEq/L)	23.68±2.61 ^a	26.64±4.17 ^a	34.29±3.60 ^b	<0.001	<0.001

Values are based on mean±SD or median (IQR). BP: blood pressure, Hb: haemoglobin, PaO₂: partial pressure of arterial oxygen.

Low category: <35 mmHg, normal category: 35-45 mmHg, high category: >45 mmHg.

P-trend derived from polynomial contrast. P-value for group difference derived from one-way ANOVA or Kruskal–Wallis. Different superscript letters indicate statistically significant differences between groups based on Tukey's HSD post hoc test ($p < 0.05$); values sharing the same superscript letter are not significantly different. P-values < 0.05 were considered statistically significant.

No significant differences were observed in energy intake, energy adequacy, carbohydrate intake, carbohydrate adequacy, fat intake, or fat adequacy across the low, normal, and high PaCO₂ groups (all $p > 0.05$). Likewise, no significant differences were found in systolic blood pressure, diastolic blood pressure, haemoglobin, or PaO₂ among the three groups.

In contrast, HCO₃⁻ levels differed significantly across PaCO₂ categories ($p < 0.001$). Post hoc analysis demonstrated that the high PaCO₂ group had significantly higher HCO₃⁻ levels than both the low ($p < 0.001$) and normal PaCO₂ groups ($p = 0.004$), whereas no significant difference was observed between the low and normal groups ($p = 0.297$).

4 Discussion

4.1 Characteristics of Study Subjects

The present study included predominantly middle-aged and elderly patients, with most participants diagnosed with ARF and pneumonia. This patient profile reflects the typical ICU population requiring mechanical ventilation. Advanced age has been associated with reduced respiratory reserve and impaired ventilatory compensation, which may contribute to altered gas exchange and respiratory outcomes [18]. Likewise, the high proportion of patients with ARF and pneumonia is consistent with the underlying pathophysiology of respiratory failure requiring ventilatory support [19]. In addition, stroke was the most common comorbidity, suggesting a substantial burden of neurological and respiratory compromise that may further affect ventilatory control. Furthermore, the variability in BMI observed in this study may affect respiratory mechanics and ventilatory function, as obesity has been associated with increased work of breathing and impaired gas exchange [20]. These clinical characteristics should be considered when interpreting PaCO₂ levels because they may influence respiratory function independently of nutritional intake.

4.2 Carbohydrate and Fat Intake

The present study showed that both carbohydrate and fat intake were below the estimated requirements, with mean adequacy levels of 77.9% and 82.7%, respectively. These findings differ from those of Javid et al. (2021), who reported a predominance of underfeeding among critically ill patients, possibly reflecting differences in nutritional assessment methods and feeding practices across healthcare settings [11].

Maintaining an appropriate carbohydrate intake is important in critically ill patients. Insufficient carbohydrate intake may promote muscle catabolism and delay ventilator

weaning, whereas excessive carbohydrate intake may increase carbon dioxide production and ventilatory burden [9], [21]. Therefore, carbohydrate intake should be carefully balanced to meet metabolic demands without imposing unnecessary respiratory burden. Similarly, appropriate fat intake is essential to support metabolic demands during critical illness. Although the mean fat adequacy observed in this study was within an acceptable range, the relatively large variability suggests that some patients experienced underfeeding whereas others may have received excessive fat intake. Reduced fat intake during the acute phase may be tolerated because of endogenous energy reserves [22], [23]; however, prolonged inadequacy or excessive energy provision may contribute to adverse metabolic consequences. Therefore, careful nutritional monitoring remains essential to optimize nutritional support while avoiding complications associated with overfeeding [24], [25].

4.3 PaCO₂ Levels

As shown in Table 3, a slight descriptive decline in mean PaCO₂ was observed between the two measurements. Although this trend may reflect physiological adaptation during clinical management, it should be interpreted with caution because no statistical comparison was performed between the two measurements.

Physiologically, PaCO₂ reflects the balance between carbon dioxide production and alveolar ventilation [26]. However, in critically ill patients, PaCO₂ is also influenced by multiple factors, including disease severity, respiratory mechanics, and ventilator settings. Furthermore, arterial blood gas measurements were obtained at variable intervals according to individual clinical conditions rather than at standardized time points. Therefore, the observed changes should not be attributed solely to enteral nutritional intake but interpreted within the broader clinical context.

4.4 Correlation of Carbohydrate, Fat, and PaCO₂

In the present study, neither carbohydrate nor fat intake, expressed as absolute intake or adequacy percentage, was significantly correlated with arterial carbon dioxide partial pressure (PaCO₂). These findings are consistent with those reported by Al-Dorzi et al., who similarly found that macronutrient composition was not significantly associated with PaCO₂ in patients with hypercapnic respiratory failure [14].

The absence of a significant association may be explained by the fact that energy intake in this study generally ranged from below the estimated requirement to near the prescribed target, with no evidence of hypercaloric feeding. Energy intake generally ranged from below the estimated requirement to near the prescribed target, with no evidence of hypercaloric feeding. Although carbohydrate metabolism theoretically generates more CO₂ because of its higher respiratory quotient (RQ = 1.0) compared with fat (RQ ≈ 0.7), this effect becomes clinically relevant primarily under conditions of overfeeding, when excess caloric substrates promote lipogenesis and additional CO₂ production [14], [27]. Therefore, variations in carbohydrate and fat intake alone, in the absence of excessive energy intake, may not be sufficient to produce clinically meaningful changes in PaCO₂.

From a physiological perspective, PaCO₂ reflects the balance between carbon dioxide production and alveolar ventilation [28]. In mechanically ventilated critically ill patients, ventilatory factors including tidal volume, minute ventilation, ventilation mode, respiratory mechanics, and the severity of the underlying disease are likely to exert a greater influence on PaCO₂ than modest variations in macronutrient intake [29], [30]. Furthermore, individualized ventilator management and heterogeneity in patients' clinical conditions may have obscured any subtle metabolic effects of nutrient intake on PaCO₂.

This interpretation is further supported by the comparison of patients across PaCO₂ categories. Consistent with the correlation analysis, no significant differences were observed in energy intake, carbohydrate intake, fat intake, or their respective adequacy percentages among the low, normal, and high PaCO₂ groups. Likewise, systolic blood pressure, diastolic blood pressure, haemoglobin, and PaO₂ did not differ significantly across the three groups. These findings suggest that, under routine ICU nutritional management, variations in energy and macronutrient intake within the observed range may have limited influence on PaCO₂ levels, whereas respiratory status and ventilatory management are likely to play a more prominent role [26].

In contrast to the nutritional variables, serum bicarbonate (HCO₃⁻) levels increased progressively across the PaCO₂ categories, with significantly higher concentrations observed in the high PaCO₂ group than in the low and normal groups. This pattern is consistent with the physiological renal compensatory response to sustained hypercapnia, whereby bicarbonate retention helps maintain acid–base homeostasis despite elevated arterial carbon dioxide levels [31]. The close association between PaCO₂ and HCO₃⁻ observed in this study therefore reflects the expected physiological regulation of acid–base balance in critically ill patients rather than an effect of macronutrient intake.

4.5 Study Limitations

This study has several limitations. The relatively small sample size and single-center setting may have limited the statistical power to detect modest associations and reduced the generalizability of the findings. In addition, variations in disease severity, comorbidities, and ventilator settings among patients may have influenced PaCO₂ levels. Several potential respiratory confounding factors, including dead space ventilation, sedative use, fluid status, and disease progression, were also not fully controlled. Therefore, these findings should be interpreted with caution.

4.6 Clinical Implications

Despite these limitations, this study contributes to the limited evidence on the relationship between macronutrient intake and PaCO₂ levels in mechanically ventilated critically ill patients. Neither carbohydrate nor fat intake, expressed as absolute intake or adequacy percentage, was significantly associated with PaCO₂ levels. Clinically, these findings suggest that PaCO₂ should be interpreted in conjunction with patients' respiratory condition and ventilatory management rather than nutritional intake alone. Future

prospective multicenter studies with larger sample sizes and more comprehensive control of potential confounding factors are warranted to confirm these findings and further clarify the relationship between macronutrient intake and respiratory outcomes.

5 Conclusion

This study demonstrated that there was no significant correlation between carbohydrate and fat intake, including their adequacy levels, and PaCO₂ in critically ill patients receiving enteral nutrition. These findings indicate that macronutrient composition alone may not substantially influence PaCO₂ under controlled nutritional conditions. The lack of association may be explained by relatively adequate intake among patients, which reduces the likelihood of excess CO₂ production. Moreover, PaCO₂ levels are primarily determined by ventilatory factors, particularly in mechanically ventilated patients, where alveolar ventilation plays a key role in CO₂ elimination. The significant and progressive increase in HCO₃⁻ levels alongside higher PaCO₂ categories further demonstrates a coupled physiological compensatory response aimed at maintaining acid-base homeostasis in these patients. Therefore, nutritional management in critically ill patients should focus on achieving appropriate energy balance and individualized care rather than emphasizing macronutrient ratios alone. Further research with larger sample sizes and controlled interventions is needed to better elucidate the relationship between macronutrient composition and respiratory outcomes.

Acknowledgments. The authors would like to express their sincere gratitude to the physicians, nursing staff, and dietitians of the Intensive Care Unit at Dr. Saiful Anwar Hospital for their assistance during the data collection process. The authors also thank all patients who participated in this study and acknowledge the valuable guidance provided by the academic supervisors.

Disclosure of Interests. The authors have no competing interests to declare that are relevant to the content of this article.

References

1. H. Damasnyah, P. Yunus, S. Monoarfa, and V. Taliki, "Pengaruh VAP Bundle Intervention Dalam Pencegahan VAP Pada Pasien Terpasang Ventilator Mekanik Di Ruangan ICU RSUD Prof. Dr. H. Aloei Saboe Kota Gorontalo," 2024.
2. R. Maryuni, R. Meilando, and S. Agustiani, "Pengaruh Abdominal Massage terhadap Penurunan Volume Residu Lambung Pasien Kritis di Intensive Care Unit," *Jurnal Penelitian Perawat Profesional*, vol. 5, pp. 961–972, 2023, doi: 10.37287/jppp.v5i3.1661.
3. E. Melaku, B. Urgie, F. Dessie, A. Seid, Z. Abebe, and A. Tefera, "Determinants of Mortality of Patients Admitted to the Intensive Care Unit at Debre Berhan Comprehensive Specialized Hospital: A Retrospective Cohort Study," *Patient Relat, Outcome Meas*, vol. 15, pp. 61–70, 2024, doi: 10.2147/PROM.S450502.
4. Munawaroh, D. Widodo, Marsaid, and K. Bahari, "Hubungan Spiritualitas dan Lama Perawatan dengan Kecemasan Keluarga Pasien Intensive Care Unit (ICU) RSI Aisyiyah Malang," 2024.

5. R. K. Kayambankadzanja *et al.*, "Towards definitions of critical illness and critical care using concept analysis," *BMJ Open*, vol. 12, p. e060972, 2022, doi: 10.1136/bmjopen-2022-060972.
6. L. K. Mahan, "Janice.L. Raymond," *Krause's Food & The Nutrition Care Process, 14th ed.*, Elsevier, 2017.
7. A. Hill *et al.*, "Combination of enteral and parenteral nutrition in the acute phase of critical illness: An updated systematic review and meta-analysis," *Journal of Parenteral and Enteral Nutrition*, vol. 46, pp. 395–410, 2022, doi: 10.1002/jpen.2125.
8. A. Goldenshluger *et al.*, "Effect of Dietary Strategies on Respiratory Quotient and Its Association with Clinical Parameters and Organ Fat Loss: A Randomized Controlled Trial," *Nutrients*, vol. 13, p. 2230, 2021, doi: 10.3390/nu13072230.
9. N. D. Kestriani, D. Budipratama, and E. Pradian, "Terapi Nutrisi pada Pasien di ICU," *Anesthesia & Critical Care*, vol. 33, pp. 226–234, 2015.
10. L. J. Hoffer and B. R. Bistran, "Nutrition in critical illness: a current conundrum," *F1000Res*, vol. 5, p. 2531, 2016, doi: 10.12688/f1000research.9278.1.
11. Z. Javid *et al.*, "Nutritional adequacy in critically ill patients: Result of PNSI study," *Clinical Nutrition*, vol. 40, pp. 511–517, 2021, doi: 10.1016/j.clnu.2020.05.047.
12. D. A. Godoy, M. Rovegno, C. Lazaridis, and R. Badenes, "The effects of arterial CO₂ on the injured brain: Two faces of the same coin," *J. Crit. Care*, vol. 61, pp. 207–215, 2021, doi: 10.1016/j.jcrc.2020.10.028.
13. M. Nourmohammadi, O. M. moghdam, M. N. Lahiji, and Z. V. Shariatpanahi, "High-fat low-carbohydrate enteral feeding enriched with olive oil and acute respiratory failure: A double-blind," *randomized, controlled trial, Clin. Nutr. ESPEN*, vol. 52, pp. 144–150, 2022, doi: 10.1016/j.clnesp.2022.10.017.
14. H. M. Al-Dorzi *et al.*, "Caloric intake and the fat-to-carbohydrate ratio in hypercapnic acute respiratory failure: Post-hoc analysis of the PermiT trial," *Clin. Nutr. ESPEN*, vol. 29, pp. 175–182, 2019, doi: 10.1016/j.clnesp.2018.10.012.
15. D. A. Purwaningrum, M. Jaelani, and S. Krisnamurni, "Asupan Karbohidrat," *Asupan Lemak Dan PaCO₂ Pada Pasien Kritis, Jurnal Riset Gizi*, vol. 2, pp. 7–12, 2014.
16. W. H. Organization, *The Asia-Pacific Perspective: Redefining Obesity and Its Treatment*. 2000.
17. P. Singer *et al.*, "Clinical Nutrition," vol. 38, pp. 48–79, 2019, doi: 10.1016/j.clnu.2018.08.037.
18. N. W. E. Satyawati and P. Andrika, "Hubungan Penyakit Komorbid dan Faktor Prognostik dengan Mortalitas pada Pasien COVID-19 Critical Ill Bulan Juni–Desember 2021 di Ruang Intensive Care Unit RSUP Prof. Dr. I.G.N.G Ngoerah Denpasar," *Indonesia Journal Chest*, vol. 10, pp. 1–6, 2023.
19. M. Umbrello, J. J. Marini, and P. Formenti, "Metabolic Support in Acute Respiratory Distress Syndrome: A Narrative Review," *J. Clin. Med.*, vol. 12, p. 3216, 2023, doi: 10.3390/jcm12093216.
20. A. Adimara, K. Prahasanti, and M. P. Airlangga, "Obesitas Mempengaruhi Tingkat Keparamahan Pasien COVID-19," *Jurnal Ilmiah Kedokteran Wijaya Kusuma*, vol. 10, pp. 222–242, 2021.
21. A. R. H. Van Zanten, "Full or hypocaloric nutritional support for the critically ill patient: is less really more?," *J Thorac Dis*, vol. 7, pp. 1086–1091, 2015.
P. C. Calder *et al.*, "Lipids in the intensive care unit: Recommendations from the ESPEN Expert Group," *Clinical Nutrition*, vol. 37, pp. 1–18, 2018, doi: 10.1016/j.clnu.2017.08.032.

22. F. Gavelli, J.-L. Teboul, and X. Monnet, "How can CO₂-derived indices guide resuscitation in critically ill patients?," *J Thorac Dis*, vol. 11, pp. S1528–S1537, 2019, doi: 10.21037/jtd.2019.07.10.
23. V.-D. Cehan, A.-R. Cehan, M. C. Pui, A. Lazar, and A. N. P. on O. in the I. C. U. (ICU): Challenges, "Dangers and Prevention Methods," *Life*, vol. 15, p. 828, 2025, doi: 10.3390/life15050828.
24. O. A. Tatucu-Babet and E. J. Ridley, "How much underfeeding can the critically ill adult patient tolerate?," *Journal of Intensive Medicine*, vol. 2, pp. 69–77, 2022, doi: 10.1016/j.jointm.2022.01.002.
25. D. Krishnaprasadh and S. Sharma, "Hypocarbica," in *StatPearls*, 2026.
26. S. A. McClave *et al.*, "Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient," *Journal of Parenteral and Enteral Nutrition*, vol. 40, pp. 159–211, 2016, doi: 10.1177/0148607115621863.
27. S. John, R. Ozanne, and K. M. HO, "Pathophysiological determinants of arterial carbon dioxide tension (PaCO₂) in spontaneously breathing and mechanically ventilated patients," *Journal of Emergency and Critical Care Medicine*, vol. 5, p. 30, 2021, doi: 10.21037/jeccm-21-7.
28. Z. Messina and H. Patrick, "Partial Pressure of Carbon Dioxide," in *StatPearls*, 2022.
29. K. Vaporidi, E. Akoumianaki, I. Telias, E. C. Goligher, L. Brochard, and D. Georgopoulos, "Respiratory Drive in Critically Ill Patients. Pathophysiology and Clinical Implications," *Am. J. Respir. Crit. Care Med*, vol. 201, pp. 20–32, 2020, doi: 10.1164/rccm.201903-0596SO.
30. J. E. Brinkman and S. Sharma, "Physiology," in *StatPearls*, 2023.

Open Access This chapter is licensed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits any noncommercial use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license and indicate if changes were made.

The images or other third party material in this chapter are included in the chapter's Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the chapter's Creative Commons license and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder.

