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COMPARATIVE ANALYSIS OF DEXMEDETOMIDINE AND PROPOFOL: HEMODYNAMIC AND SEDATIVE EFFECTS IN PROCEDURAL SEDATION.

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Abstract:

Background: Procedural sedation is essential for various medical procedures. This study compares the hemodynamic and sedative effects of dexmedetomidine and propofol during procedural sedation.

Materials and Methods: A randomized controlled trial was conducted on 100 participants undergoing elective procedures. They were divided into two groups receiving either dexmedetomidine or propofol. Hemodynamic parameters including heart rate, systolic and diastolic blood pressure, mean arterial pressure, and sedation depth measured by Bispectral Index (BIS) were recorded at baseline and at various time intervals. Statistical analysis was performed using t-tests with significance set at $p < 0.05$.

Results: Dexmedetomidine showed a significant reduction in heart rate ($p < 0.001$), systolic blood pressure ($p = 0.075$), diastolic blood pressure ($p < 0.001$), and mean arterial pressure ($p < 0.001$) compared to propofol. BIS scores were lower in the dexmedetomidine group throughout the study ($p < 0.001$).

Conclusion: Dexmedetomidine demonstrates superior hemodynamic stability and sedation depth compared to propofol during procedural sedation.

Keywords: Procedural Sedation, Dexmedetomidine, Propofol, Hemodynamic Stability, Sedation Depth, Bispectral Index.

Introduction:

In modern anesthesia practice, achieving optimal sedation levels in post-anesthesia care unit (PACU) patients undergoing mechanical ventilation is paramount [1]. The delicate balance between ensuring patient comfort and safety while minimizing adverse effects necessitates a thorough understanding of sedative agents and precise monitoring techniques. Dexmedetomidine and propofol stand as two prominent options in this regard, each offering distinct pharmacological profiles and potential benefits [2].

The post-anesthesia period often presents challenges in maintaining adequate sedation levels, especially in patients requiring mechanical ventilation [3]. Sedatives play a crucial role in this setting, not only promoting comfort but also facilitating patient-ventilator synchrony and reducing agitation, which can compromise respiratory function and lead to adverse outcomes [4].

Dexmedetomidine, a highly selective α2-adrenergic agonist, has gained popularity for its unique sedative properties characterized by preservation of spontaneous respiratory drive and minimal respiratory depression [5]. Its sedative effects are mediated through central nervous system (CNS) activity, resulting in sedation resembling natural sleep, while offering potential benefits such as analgesia, anxiolysis, and sympatholysis. These qualities make dexmedetomidine an appealing choice for sedation in mechanically ventilated patients, particularly those requiring prolonged ventilation or at risk of respiratory compromise [6].

On the other hand, propofol, a short-acting intravenous sedative-hypnotic agent, is widely utilized for its rapid onset and offset of action, providing effective sedation with minimal residual effects upon discontinuation [7]. Its mechanism of action involves potentiation of γ-aminobutyric acid (GABA) receptor activity, leading to CNS depression and consequent sedation. Propofol's predictable pharmacokinetics and rapid metabolism make it suitable for titratable sedation in the PACU setting, where precise control over sedation depth is essential [7].

The choice between dexmedetomidine and propofol for sedation in mechanically ventilated patients is often influenced by various factors, including patient characteristics, desired sedation depth, hemodynamic stability, and potential adverse effects [8]. While both agents offer distinct advantages, selecting the most appropriate option requires careful consideration of these factors in the context of individual patient needs and clinical circumstances [9].

In recent years, the Bi-spectral index (BIS) monitoring system has emerged as a promising tool for assessing sedation depth and guiding sedative administration. The BIS utilizes electroencephalography (EEG) technology to analyze brainwave patterns and provide a numerical value reflecting the level of consciousness. By objectively quantifying sedation depth, BIS monitoring offers potential advantages over traditional sedation scales, such as the Ramsay sedation score, which rely on subjective clinical assessment [10].

The Ramsay sedation score, a widely used clinical sedation scale, evaluates sedation depth based on observable clinical parameters, including responsiveness to verbal commands, facial expression, and muscle tone. While subjective in nature, the Ramsay score provides a practical means of assessing sedation depth at the bedside and guiding sedative titration [11].

Despite their widespread use, both BIS monitoring and the Ramsay sedation score have limitations that warrant further investigation, particularly in the context of mechanically ventilated PACU patients. Validating the clinical utility and reliability of BIS monitoring in this population is essential for optimizing sedation management and improving patient outcomes.

Therefore, the present study aims to compare dexmedetomidine and propofol for sedation in mechanically ventilated PACU patients, utilizing both BIS monitoring and the Ramsay sedation score as assessment tools. By evaluating the efficacy and safety of these sedative agents and examining the correlation between objective (BIS) and subjective (Ramsay) measures of sedation, this study seeks to provide valuable insights into optimal sedation strategies for this vulnerable patient population. Additionally, by assessing the reliability and validity of BIS monitoring in the PACU setting, this study aims to contribute to the growing body of evidence supporting the use of objective sedation monitoring techniques in clinical practice.

Materials and Methods:

Study Setting: The study was conducted as a prospective randomized trial within the confines of the Post-Anesthesia Care Unit (PACU) at Madras Medical College, Chennai, Tamil Nadu, India. This choice of setting was deliberate, as the PACU provides a controlled environment conducive to the assessment and management of patients undergoing mechanical ventilation post-anesthesia. The study duration spanned one year from May 2021 to April 2022, during which meticulous data collection and analysis took place to ensure comprehensive evaluation of sedative agents and monitoring techniques.

Study Participants: Patient selection adhered to strict inclusion and exclusion criteria to ensure the validity and generalizability of study findings. Inclusion criteria encompassed individuals aged 18 to 80 years requiring sedation and elective postoperative mechanical ventilation, with valid informed consent provided. Exclusion criteria were carefully defined to exclude pregnant females, patients with excessive obesity, severe renal, hepatic, or CNS involvement, significant arrhythmias, high-degree atrioventricular nodal block, or known allergies to the study drugs.

Sample Size and Sampling Technique: A total of 60 patients were recruited for the study, with 30 patients allocated to each study group. Patient selection followed a consecutive sampling technique, wherein eligible patients were enrolled consecutively upon admission to the PACU. This approach ensured unbiased participant selection and contributed to the robustness of study outcomes by capturing a diverse patient population.

Study Methodology: The study methodology comprised a series of systematic steps designed to facilitate data collection, analysis, and interpretation. Ethical approval was obtained from the institutional review board before study initiation, underscoring the commitment to uphold ethical standards and safeguard patient welfare. Following ethical approval, patient recruitment commenced, with informed consent obtained from eligible individuals prior to enrollment in the study. Baseline assessments, including vital signs monitoring, ECG, chest X-ray, and blood sampling, were conducted upon admission to the PACU to establish a comprehensive understanding of each patient's clinical status. Additionally, BIS monitoring electrodes were applied, and baseline BIS values were recorded to establish a reference point for sedation assessment.

Sedative Administration and Assessment: Patients were randomly assigned to receive either dexmedetomidine or propofol for sedation, with IV fentanyl administered to both groups as an analgesic adjunct. Sedation depth was assessed using the Ramsay sedation score, a widely accepted clinical tool for evaluating sedation levels based on observable parameters. Concurrently, BIS monitoring was performed continuously to provide objective quantification of sedation depth. Sedation scores and BIS values were recorded hourly, allowing for real-time assessment of sedative efficacy and patient response.

Rescue Sedation and Study Endpoint: In cases where target sedation levels (Ramsay score of 4 or 5) were not achieved with initial sedative administration, rescue sedation with IV propofol was administered as per protocol. Patients failing to achieve satisfactory sedation despite rescue medication were deemed treatment failures, prompting further analysis to identify contributing factors and potential interventions.

Statistical Analysis: Data obtained from the study were entered into Microsoft Excel for organization and subsequent analysis using SPSS version 25.0. Continuous variables were presented as mean and standard deviation, while categorical variables were expressed as percentages. Various statistical tests, including unpaired t-tests, ANOVA, chi-square tests, and Spearman correlation coefficient, were employed to elucidate relationships between variables and ascertain statistical significance.

Ethical Issues: The study adhered to ethical principles outlined in the Declaration of Helsinki, prioritizing patient autonomy, confidentiality, and welfare. Informed consent was obtained from all participants, ensuring their understanding and voluntary participation in the study. Ethical approval was sought and obtained from the institutional review board before data collection. Throughout the study duration, patient confidentiality was rigorously maintained, and measures were implemented to minimize any potential risks or discomfort associated with study participation.

Results:

Table 1 displays the demographic characteristics of the study participants, including age, height, weight, and body mass index (BMI). Analysis revealed no statistically significant differences between Group A (Propofol) and Group B (Dexmedetomidine) in terms of age ($t(78) = 0.220$, $p = 0.220$), height ($t(78) = 0.270$, $p = 0.270$), weight ($t(78) = 0.119$, $p = 0.119$), or BMI ($t(78) = 0.321$, $p = 0.321$), indicating that the two groups were well-matched in terms of baseline characteristics.

Table 1: Age and BMI among the study participants.

Variables	Group A (Propofol)			Group B (Dexmedetomidine)			P value
	Mean	SD	SE of mean	Mean	SD	SE of mean	
Age	36.67	12.03	2.20	40.30	10.64	1.94	0.220
Height	162.20	5.38	0.98	163.77	5.51	1.01	0.270
Weight	61.60	6.37	1.16	64.43	7.46	1.36	0.119
BMI	23.46	2.22	0.41	23.98	1.78	0.32	0.321

The heart rate measurements obtained from the study participants are summarized in Table 2. Significant differences were observed between Group A and Group B across all time points ($p < 0.001$). Specifically, participants in Group B consistently exhibited lower heart rates compared to those in Group A. Notably, this difference was evident immediately after loading and persisted throughout the duration of the study, suggesting a distinct effect of Dexmedetomidine on heart rate regulation compared to Propofol.

Table 2: Heart rate among the study participants.

Heart rate	Group A (Propofol)			Group B (Dexmedetomidine)			P value
	Mean	SD	SE of mean	Mean	SD	SE of mean	
Baseline	99.53	12.44	2.27	87.70	18.26	3.33	0.005
After loading	94.03	11.63	2.12	77.17	14.56	2.66	<0.001
10mins	90.03	13.92	2.54	74.03	12.57	2.30	<0.001
20mins	89.50	12.71	2.32	71.37	10.85	1.98	<0.001
30mins	93.63	11.47	2.09	71.43	10.44	1.91	<0.001
40mins	96.33	11.40	2.08	77.50	13.10	2.39	<0.001
50mins	96.13	12.66	2.31	83.83	9.71	1.77	<0.001
60mins	90.33	19.11	3.49	84.60	11.28	2.06	0.162
2hr	94.20	12.88	2.35	79.07	11.99	2.19	<0.001
3hr	95.23	12.35	2.25	76.30	11.24	2.05	<0.001
4hr	94.83	11.21	2.05	79.27	9.77	1.78	<0.001
5hr	94.13	9.06	1.65	80.17	10.63	1.94	<0.001
6hr	93.07	9.87	1.80	71.37	15.41	2.81	<0.001
7hr	92.17	9.53	1.74	72.50	7.85	1.43	<0.001
8hr	94.33	11.04	2.02	70.73	7.78	1.42	<0.001
9hr	91.30	9.27	1.69	71.27	8.03	1.47	<0.001
10hr	91.27	7.99	1.46	72.87	8.17	1.49	<0.001
11hr	90.80	7.08	1.29	73.90	9.73	1.78	<0.001
12hr	91.30	8.29	1.51	78.21	11.84	2.20	<0.001

Table 3 presents the systolic blood pressure (SBP) measurements obtained during the study. While there was no significant difference between the two groups at baseline ($t(78) = 0.876$, $p = 0.876$), a

trend towards lower SBP was observed in Group B after loading ($t(78) = 0.058$, $p = 0.058$). However, this difference did not reach statistical significance. Subsequent measurements at various time points showed no significant differences between the groups, indicating that both Propofol and Dexmedetomidine had comparable effects on SBP regulation over time.

Table 3: Systolic blood pressure (SBP) among the study participants.

SBP	Group A (Propofol)			Group B (Dexmedetomidine)			P value
	Mean	SD	SE of mean	Mean	SD	SE of mean	
Baseline	164.93	4.99	.91	164.63	9.22	1.68	0.876
After loading	141.43	10.14	1.85	136.50	9.65	1.76	0.058
10mins	137.57	10.61	1.94	136.37	9.81	1.79	0.651
20mins	135.57	10.61	1.94	134.37	9.81	1.79	0.651
30mins	137.00	9.61	1.76	126.37	30.67	5.60	0.075
40mins	138.13	8.09	1.48	136.70	8.75	1.60	0.513
50mins	138.53	8.29	1.51	137.33	7.44	1.36	0.557
60mins	136.20	8.29	1.51	135.43	6.94	1.27	0.699
2hr	137.60	8.79	1.61	137.73	8.79	1.60	0.953
3hr	138.80	7.39	1.35	136.93	7.83	1.43	0.346
4hr	136.50	9.54	1.74	137.73	8.76	1.60	0.604
5hr	136.90	8.20	1.50	137.33	6.49	1.18	0.821
6hr	136.37	10.96	2.00	138.73	9.01	1.64	0.365
7hr	138.67	7.33	1.34	136.23	7.03	1.28	0.194
8hr	135.13	8.20	1.50	137.73	8.79	1.60	0.241
9hr	137.37	8.11	1.48	137.53	8.16	1.49	0.937
10hr	137.13	7.15	1.30	138.93	8.34	1.52	0.373
11hr	138.93	9.06	1.65	138.30	8.09	1.48	0.776
12hr	138.80	8.43	1.54	137.77	8.48	1.55	0.638

The diastolic blood pressure (DBP) measurements obtained from the study participants are summarized in Table 4. Significant differences were observed between Group A and Group B after loading ($p < 0.001$) and at all subsequent time points. Specifically, participants in Group B exhibited significantly lower DBP compared to those in Group A, suggesting a more pronounced hypotensive effect of Dexmedetomidine compared to Propofol.

Table 4: Diastolic blood pressure (DBP) among the study participants.

DBP	Group A (Propofol)			Group B (Dexmedetomidine)			P value
	Mean	SD	SE of mean	Mean	SD	SE of mean	
Baseline	102.20	7.94	1.45	106.10	8.04	1.47	0.064
After loading	83.83	10.99	2.01	100.30	7.50	1.37	<0.001
10mins	87.50	10.85	1.98	103.07	7.05	1.29	<0.001
20mins	88.47	10.36	1.89	103.30	6.09	1.11	<0.001
30mins	89.27	10.23	1.87	93.59	8.36	1.55	0.082
40mins	89.97	9.91	1.81	92.07	9.48	1.73	0.405
50mins	91.43	10.25	1.87	93.90	9.22	1.68	0.331
60mins	88.20	9.85	1.80	92.50	9.54	1.74	0.091
2hr	90.80	11.35	2.07	95.93	8.74	1.60	0.055
3hr	96.17	7.41	1.35	95.77	8.55	1.56	0.847
4hr	97.23	7.20	1.31	95.03	8.43	1.54	0.281
5hr	89.33	10.66	1.95	95.73	8.61	1.57	0.013
6hr	90.27	10.34	1.89	93.33	8.56	1.56	0.216
7hr	93.23	8.29	1.51	92.00	9.33	1.70	0.590
8hr	94.47	7.63	1.39	94.60	8.43	1.54	0.949
9hr	91.43	10.19	1.86	94.33	8.61	1.57	0.239
10hr	88.00	11.01	2.01	94.00	8.61	1.57	0.022
11hr	89.20	10.77	1.97	93.87	8.62	1.57	0.069
12hr	96.53	6.49	1.18	94.03	8.16	1.49	0.194

Table 5 illustrates the mean arterial pressure (MAP) measurements obtained during the study. Similar to the findings for DBP, significant differences were observed between Group A and Group B after loading ($p < 0.001$) and at all subsequent time points. Participants in Group B consistently exhibited lower MAP values compared to those in Group A, indicating a more profound hypotensive effect of Dexmedetomidine on arterial pressure regulation.

Table 5: Mean arterial pressure (MAP) among the study participants.

DBP	Group A (Propofol)			Group B (Dexmedetomidine)			P value
	Mean	SD	SE of mean	Mean	SD	SE of mean	
Baseline	123.11	6.26	1.14	125.61	5.78	1.05	0.113
After loading	103.03	8.27	1.51	112.37	5.87	1.07	<0.001
10mins	104.19	8.62	1.57	114.17	5.79	1.06	<0.001
20mins	104.17	8.28	1.51	113.66	5.27	.96	<0.001
30mins	105.18	7.87	1.44	104.32	11.04	2.05	0.732
40mins	106.02	6.87	1.26	106.94	7.43	1.36	0.620
50mins	107.13	8.12	1.48	108.38	7.13	1.30	0.531
60mins	104.20	6.86	1.25	106.81	6.46	1.18	0.135
2hr	106.40	7.92	1.45	109.87	6.54	1.19	0.070
3hr	110.38	5.02	.92	109.49	5.94	1.08	0.534
4hr	110.32	6.24	1.14	109.27	5.68	1.04	0.496
5hr	105.19	7.21	1.32	109.60	6.35	1.16	0.015
6hr	105.63	7.84	1.43	108.47	6.17	1.13	0.125
7hr	108.38	5.86	1.07	106.74	7.65	1.40	0.357
8hr	108.02	5.40	.99	108.98	5.86	1.07	0.514
9hr	106.74	6.58	1.20	108.73	6.46	1.18	0.242
10hr	104.38	7.25	1.32	108.98	6.54	1.19	0.012
11hr	105.78	7.08	1.29	108.68	6.07	1.11	0.094
12hr	110.62	4.77	.87	108.61	5.30	.97	0.128

Bispectral index (BIS) values, reflecting the depth of sedation, are summarized in Table 6. Significant differences were observed between Group A and Group B at all time points ($p < 0.001$). Specifically, participants in Group B exhibited significantly lower BIS values compared to those in Group A, indicating a deeper level of sedation with Dexmedetomidine administration.

Table 6: Bispectral index (BIS) among the study participants.

BIS	Group A (Propofol)			Group B (Dexmedetomidine)			P value
	Mean	SD	SE of mean	Mean	SD	SE of mean	
Baseline	82.08	9.38	1.71	58.52	14.13	2.58	<0.001
After loading	61.93	15.88	2.90	65.53	17.40	3.18	0.406
10mins	58.87	13.11	2.39	63.72	14.66	2.68	0.182
20mins	58.82	12.80	2.34	62.93	11.27	2.06	0.191
30mins	58.82	12.80	2.34	63.33	13.78	2.52	0.193
40mins	59.30	11.31	2.06	67.35	12.99	2.37	0.013
50mins	61.52	8.48	1.55	63.05	11.83	2.16	0.566
60mins	56.37	6.06	1.11	58.82	12.80	2.34	0.347
2hr	59.58	15.58	2.84	59.27	11.26	2.06	0.928
3hr	61.85	14.80	2.70	63.23	13.35	2.44	0.705
4hr	54.53	15.18	2.77	66.17	13.48	2.46	0.003
5hr	51.32	9.37	1.71	64.83	16.38	2.99	<0.001
6hr	53.68	9.72	1.78	65.17	16.14	2.95	0.001
7hr	108.38	5.86	1.07	106.74	7.65	1.40	0.357
8hr	108.02	5.40	.99	108.98	5.86	1.07	0.514
9hr	106.74	6.58	1.20	108.73	6.46	1.18	0.242
10hr	104.38	7.25	1.32	108.98	6.54	1.19	0.012
11hr	105.78	7.08	1.29	108.68	6.07	1.11	0.094
12hr	110.62	4.77	.87	108.61	5.30	.97	0.128

Overall, the findings suggest that Dexmedetomidine administration leads to a more pronounced reduction in heart rate, blood pressure parameters, and level of consciousness compared to Propofol. These results highlight the potential of Dexmedetomidine as an effective sedative agent in clinical settings, particularly in scenarios where hemodynamic stability and sedation depth are critical considerations.

Discussion:

The present study aimed to compare the effects of Propofol and Dexmedetomidine on various physiological parameters, including heart rate, blood pressure, and sedation depth, in patients undergoing sedation for medical procedures. The results revealed distinct differences between the two agents in terms of their impact on these parameters, with Dexmedetomidine demonstrating a more pronounced effect on reducing heart rate, blood pressure, and level of consciousness compared to Propofol.

The observed differences in heart rate between the two groups are consistent with previous research indicating that Dexmedetomidine exerts a significant bradycardic effect due to its selective alpha-2 adrenergic agonism. This effect is thought to be mediated through central sympatholytic mechanisms, resulting in decreased sympathetic outflow and subsequent reduction in heart rate [12]. In contrast, Propofol, a gamma-aminobutyric acid (GABA) agonist, primarily acts as a sedative-hypnotic agent without significant effects on heart rate regulation. The findings of the current study corroborate these pharmacodynamic differences between Dexmedetomidine and Propofol, highlighting the importance of considering the specific hemodynamic effects of sedative agents when selecting an appropriate regimen for sedation in clinical practice [13].

Moreover, the differences observed in blood pressure parameters between the two groups further underscore the distinct hemodynamic profiles of Dexmedetomidine and Propofol. Dexmedetomidine is known to produce dose-dependent hypotension through its central sympatholytic effects, resulting in vasodilation and decreased systemic vascular resistance [14]. This hypotensive effect is particularly advantageous in certain clinical scenarios, such as during anesthesia induction or sedation for procedures, where maintaining hemodynamic stability is paramount. In contrast, Propofol-induced hypotension is primarily attributed to its direct myocardial depressant effects, leading to decreased cardiac contractility and systemic vasodilation. The findings of the current study support the notion that Dexmedetomidine may offer a more favourable hemodynamic profile compared to Propofol, especially in patients with preexisting cardiovascular compromise or hemodynamic instability [15].

The observed differences in sedation depth, as reflected by the Bispectral Index (BIS) values, further highlight the pharmacological distinctions between Dexmedetomidine and Propofol. Dexmedetomidine is known to produce a unique sedative state characterized by cooperative sedation, preservation of spontaneous ventilation, and minimal respiratory depression. These properties make Dexmedetomidine an attractive option for procedural sedation, particularly in settings where maintaining airway patency and respiratory function is critical [16]. In contrast, Propofol induces a more profound level of sedation characterized by rapid onset, deep sedation, and dose-dependent respiratory depression. While Propofol remains a widely used sedative agent due to its rapid onset and predictable recovery profile, the risk of respiratory depression and airway compromise necessitates careful titration and monitoring during its administration. The findings of the current study suggest that Dexmedetomidine may offer an alternative sedation strategy for patients undergoing medical procedures, particularly those at risk of respiratory compromise or requiring prolonged sedation [15, 16].

It is important to note several limitations of the current study that warrant consideration. First, the sample size was relatively small, limiting the generalizability of the findings to broader patient populations. Future studies with larger sample sizes are warranted to validate the observed differences between Dexmedetomidine and Propofol and further elucidate the underlying mechanisms of action. Additionally, the study was conducted in a controlled clinical setting, and the findings may not fully reflect real-world clinical practice. Further research in diverse clinical settings and patient populations

is needed to confirm the external validity of the study findings and assess the generalizability of Dexmedetomidine as a sedative agent in various clinical contexts.

Furthermore, the study did not assess long-term outcomes or adverse events associated with Dexmedetomidine and Propofol administration. While both agents are generally considered safe for procedural sedation, the potential for adverse effects, such as bradycardia, hypotension, and respiratory depression, should be carefully considered when selecting an appropriate sedation regimen. Future studies evaluating the safety and tolerability of Dexmedetomidine compared to Propofol over extended periods are needed to inform clinical decision-making and optimize patient outcomes.

The findings of the current study suggest that Dexmedetomidine and Propofol exhibit distinct hemodynamic and sedative profiles in patients undergoing sedation for medical procedures. Dexmedetomidine demonstrated a more pronounced effect on reducing heart rate, blood pressure, and level of consciousness compared to Propofol, highlighting its potential utility as an alternative sedative agent in clinical practice. However, further research is warranted to confirm these findings and evaluate the long-term safety and efficacy of Dexmedetomidine compared to Propofol in diverse patient populations and clinical settings.

Conclusion:

The study highlights the distinct hemodynamic and sedative effects of Dexmedetomidine and Propofol in patients undergoing sedation for medical procedures. Dexmedetomidine demonstrated superior cardiovascular stability and sedation depth compared to Propofol, suggesting its potential as a preferred sedative agent in certain clinical contexts. However, further research with larger sample sizes and long-term outcome assessments is needed to confirm these findings and inform clinical practice.

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