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Original Article

Mean arterial pressure in critically ill adults receiving vasopressors: A multicentre, observational study

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ABSTRACT

Objective: Mean arterial pressure (MAP) management is a key aspect of treatment in critically ill patients receiving vasopressor therapy. Guidelines in different clinical subgroups have proposed various target MAP values. This study aimed to describe delivered MAP values and corresponding vasopressor doses in such patients.

Design: Multicenter, retrospective cohort study of adult intensive care unit (ICU) admissions.

Setting: 12 ICUs in Queensland, Australia, from January 1, 2015, to December 31, 2021.

Participants: Patients receiving vasopressors for at least six continuous hours in the ICU. We studied the delivered MAP values using hourly data based on averaging all validated values obtained from the ICU monitors and average hourly doses of vasopressors.

Main Outcome Measure: The primary outcome was the mean MAP during the entire cohort's first 72 hours of ICU admission, whilst vasopressors were administered.

Results: In 26,519 patients who received vasopressors for at least six continuous hours, the median age was 62 years, and 9,373 (35%) were admitted after elective surgery. The median time from ICU admission to vasopressor commencement was 2 hours, and the median duration of vasopressor therapy was 27 hours. At 72 hours, 6,627 (25.0%) patients remained on vasopressors. The mean hourly MAP was 72 mmHg in the first six hours, then steadily increased to ≈75 mmHg at 72 hours. In the first 72 hours, 11,032 (41.6%) patients had a mean MAP of 70-74 mmHg, and 5,914 (22.3%) had a mean MAP of 75-79

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mmHg. For every clinical subgroup, a MAP of 70-74 mmHg was the most common mean MAP, and the proportion of patients with a mean MAP of 60-65mmHg was less than 5%.

Conclusions: In a large, multicenter study of heterogeneous critically ill patients on vasopressors, the mean hourly MAP was > 70 mmHg. This mean hourly MAP was observed consistently over diverse clinical subgroups and is higher than recommended by guidelines.

1. Introduction

A low mean arterial blood pressure (MAP) is a common reason for admission to the intensive care unit (ICU).[1,2] Patients with such hypotension have an increased risk of morbidity and mortality.[2] Prompt treatment of hypotension is a cornerstone of ICU management, and vasopressors are commonly used for its treatment.[3] These medications can counteract vasodilation and restore MAP to target levels. However, their effects are diverse, and their use has also been linked to potential harm.[4]

Balancing the risk of hypotension with the risk of vasopressors-induced harm is a key challenge in the ICU. This is done by titrating the dose of vasopressors to the chosen target MAP. Guidelines, such as the Surviving Sepsis Guidelines, recommend a MAP target of 65 mmHg for critically ill patients receiving vasopressor therapy based on low-certainty evidence.[5] A recent systematic review of 11 randomised clinical trials (RCTs) found a lower MAP, ranging from MAP 50—70 mmHg, compared to higher targets, reduced mortality, suggesting that the MAP target may impact patient outcomes.[6,7] Moreover, a recent international survey of intensive care doctors reported 65 mmHg as the most common minimum MAP target prescribed in clinical practice. However, the survey also highlighted that the optimal threshold for most clinical scenarios remains uncertain.[8]

Considering the significant global impact of hypotension requiring vasopressor support and the need to balance the harms and benefits of hypotension and vasopressor therapy, ensuring optimal MAP management seems critical. However, the conduct of future randomised controlled trials to define such optimal MAP must be based on a clear understanding of current usual care. Unfortunately, there is no detailed knowledge of the MAP values delivered in current practice in Australia and New Zealand. We aimed to test the hypothesis that the average MAP would be close to the recommended 65 mmHg in patients receiving vasopressor therapy.

2. Methods

2.1. Study design

Multicenter, retrospective cohort study of routinely collected electronic medical record (EMR)-based clinical data.

2.2. Setting

The study was conducted in 12 closed-model, mixed (medical and surgical) ICUs in Queensland, Australia, including five tertiary, three outer metropolitan, and four regional ICUs. Patient data were collected for the entire ICU admission, and all patients were followed up for one year from their ICU admission.

2.3. Participants

All adult patients, age greater than or equal to 18, admitted between January 1, 2015, and December 31, 2021, were assessed for inclusion. Patients admitted with palliative intent, pregnant patients, or patients admitted with acute aortic syndrome, traumatic brain, or spinal injury were excluded. Patients readmitted or transferred from another hospital were excluded from the analysis.

2.4. Data sources

Data were collected from all centres using eCritical MetaVisionTM (iMDsoft, Boston, MA, USA) clinical information systems and the Australian and New Zealand Intensive Care Society (ANZICS) CORE Adult Patient Database (APD).[9] The data included mean hourly doses for all vasoactive medications and hourly haemodynamic measurements, including MAP, as well as patient demographics, diagnoses, severity of illness, and outcomes. The hourly data were derived by averaging all validated MAP values over 1 h. MAP and vasoactive dose values considered implausible based on prespecified parameters were marked as not administered (Supplementary Methods, Table S1).

Primary and secondary diagnoses from the International Classification of Diseases 10 Australia Modification (ICD-10-AM) codes and mortality data were collected from the Queensland Hospital Admitted Patient Data Collection (QHAPDC) and Queensland Births, Deaths, and Marriage Registry, respectively.[10]

2.5. Data variables

The mean hourly vasopressor dose, expressed as a weight-adjusted dose per hour, was collected for the entire ICU admission. Vasopressors were adrenaline, noradrenaline, vasopressin, dopamine, metaraminol, and phenylephrine. Other vasoactive medications, milrinone, dobutamine, and levosimendan were considered inotropes and not classified as vasopressors. Patients were identified as having received a vasopressor if they were administered any of the included drugs for at least six continuous hours during the first 72 h of their admission.

Admission diagnoses were categorised by system to optimise data accuracy and interpretability (Supplementary Methods, Table S2).[11] The Charlson-defined comorbidities and index were calculated from the ICD-10 codes (Supplementary Methods, Table S3).[12,13]

The prespecified subgroups were sepsis vs no sepsis, chronic hypertension vs. no chronic hypertension, elective non-cardiac surgery vs not elective noncardiac surgery, elective cardiac surgery vs not elective cardiac surgery, cardiogenic shock vs not cardiogenic shock, age $\geq$65 years vs <65 years, acute kidney injury vs no acute kidney injury, acute myocardial infarction vs no acute myocardial infarction, atrial fibrillation vs no atrial fibrillation, unplanned admission vs. planned admission, and mechanical ventilation vs. not mechanically ventilated. These subgroups were defined as detailed in the Supplementary Methods Table S4.

Mean hourly MAP was used for analysis, representing the mean value of all validated measurements within each hour. MAP measurements were taken from both invasive and noninvasive measurements, and when both were available for an hour, the invasive measurement was used for in the analysis. Only MAP values where vasopressors were administered were included in the MAP

analysis. For additional analysis, the MAP (in mmHg) was categorised into seven groups: <55, 55–59, 60–64, 65–60, 70–74, 75–79, ≥80.

2.6. Outcomes

The primary outcome was the mean MAP during the entire cohort's first 72 h of ICU admission, whilst vasopressors were administered. The secondary outcomes were the mean MAP for the first 72 h of vasopressor therapy for prespecified subgroups. Exploratory outcomes included the proportion of patients in prespecified MAP bands for each hour of vasopressor therapy during the first 72 h and characteristics of vasopressor therapy, including time of commencement, duration of treatment, and individual drug doses.

2.7. Statistical analysis

Descriptive statistics were expressed as frequencies and proportions for categorical variables and medians with interquartile ranges (IQR) for continuous variables. Pearson's Chi-square test was used to compare categorical variables for each subgroup pair. The Wilcoxon rank sum test was used to compare continuous variables for each subgroup pair. Statistical analyses were performed using R version 4.4.0 (R Foundation for Statistical Computing, Vienna, Austria)[9] with the packages 'dplyr',[14] 'ggplot2',[15] and 'gtsummary'.[16]

2.8. Statement of ethics

In Queensland, the Metro South Hospital and Health Service Human Research Ethics Committee (HREC/2022/QMS/82024) approved the study, with an individual waiver of consent granted.

3. Results

3.1. Patient characteristics and incidence of vasopressor therapy

During the study period, there were 89,117 admissions to the participating ICUs, and 71,406 (80.1%) patient admissions remained after exclusion for baseline characteristics (Supplementary Figure S1). Of these, 39,057 (55.0%) did not receive vasopressors and 5830 (7.5 %) patients received vasopressors for fewer than 6 h. In total, 26,519 (29.8%) patients admitted received at least six continuous hours of vasopressor therapy within 72 h of their ICU admission. For patients described in this analysis, there were 3,263,720 hourly MAP measurements during vasopressor therapy, of which 2,960,194 (90.7%) were taken from invasive arterial lines.

As shown in Supplementary Table S5, patients included in this cohort had a median age of 62 years (IQR 48–71), a median body mass index of 27.8 (IQR 24.4–31.9) and a median Charlson comorbidity index of 3 (IQR 1–5). The most common medical comorbidity was diabetes, which occurred in 6467 (24%) of patients, followed by congestive heart failure (3419; 13%), ischaemic heart disease (3407: 13 %), and chronic kidney disease (2844; 11%). 7569 (29%) patients had more than one chronic comorbidity. The most common diagnostic group was cardiovascular (9373; 35%). Overall, 35% (9328) were patients admitted after elective surgery, and 8.9% (2316) were admitted after a rapid response team review. The median Acute Physiology and Chronic Health Evaluation (APACHE) III score was 56 (IQR 42–75), and on the day of admission, most patients required ventilation (19,183; 72%); the median maximum sequential organ failure assessment (SOFA) score was 6 (IQR 5–9). Most patients were admitted to tertiary ICUs (20,107; 76%) instead of a regional (4079; 15%) or outer metropolitan (2333; 8.8%) ICU. The operating theatre (13,421; 52%) was the most common source of ICU admission.

3.2. Vasopressor therapy characteristics

The median time from ICU admission to vasopressor commencement was 2 h (IQR 1–4), including patients admitted with vasopressor infusion, and the median duration of vasopressor therapy was 27 h (IQR 15–55). After 72 h, 6627 (25.0 %) patients received at least 1 h of vasopressors during the remainder of their ICU admission (Supplementary Figure S2).

The most common vasopressor administered was noradrenaline (21,440; 81%), with metaraminol (5502; 21%), dopamine (3948; 15%), vasopressin (4677; 18%), and adrenaline (3488; 13%) being the next most common (Table 1). 7991 (30%) patients received more than one concurrent vasopressor during their ICU admission. The median doses for vasopressors were as follows: noradrenaline 0.06 μg/kg/min, metaraminol 0.26 μg/kg/min, dopamine 3.45 μg/kg/min, vasopressin 0.034 units/min, and adrenaline 0.07 μg/kg/min.

3.3. Mean arterial pressure for entire cohort

The mean hourly MAP whilst vasopressors were administered for the entire cohort is shown in Fig. 1. The mean hourly MAP was 72 mmHg in the first 6 h of the ICU admission, then steadily increased to a MAP of just over 75 mmHg at 72 h. Over the 7-year study period, the mean hourly MAP during the first 72 h of ICU admission ranged between 73.2 mmHg and 73.5 mmHg (Supplementary Figure S3).

The mean hourly MAP, categorised into seven distinct MAP groups, demonstrated no dominant MAP group. For each hour of the 1st 72 h, 17.6%–25.4% of patients had a MAP of 65–69 mmHg, which was comparable to 18.1%–26.1% with a MAP of 70–74 mmHg and 15.1%–20.3% with a MAP of 75–79 mmHg (Fig. 2).

When the mean MAP over the first 72 h of an ICU admission was examined, the largest group of patients (11,032; 41.6%) had a mean MAP of 70–74 mmHg, followed by 75–79 mmHg (5914; 22.3%) and 65–69 mmHg (5383; 20.3%) (Fig. 3).

3.4. Mean arterial pressure in subgroups

The mean MAP over the first 72 h of admission was examined in the prespecified subgroups (Fig. 4). When compared to the remainder of the cohort, the mean MAP over the first 72 h of ICU admission for each subgroup is presented in Table 2. In all subgroup pairs, except for chronic hypertension vs not and acute myocardial infarction vs not, there was evidence that mean MAP varied significantly by subgroup. However, the magnitude of the differences was small; for example, patients with sepsis had a mean MAP of 73.6 mmHg (5.4) compared to patients without sepsis, 73.3 mmHg (6.3) ($p < 0.001$ for interaction).

The number and percentage of patients in each MAP group by prespecified subgroup are reported in Supplementary Table S6. For patients diagnosed with sepsis, most patients (4016; 45%) had a mean MAP of 70–74 mmHg, followed by 75–79 mmHg (2201; 25%) and 65–69 mmHg (1493; 17%) over the first 72 h of admission. When their MAP was analysed hourly, the MAP was greater than 70 mmHg in 67.7% of measurements. Patients aged 65 or greater (11,410) had a similar distribution of mean MAP, where most (4748;

Table 1
Characteristics of vasopressor administration.

Variable	Hour of ICU Admission			
	Overall N = 26,519[a]	0-24 N = 26,519[a]	25-48 N = 16,774[a]	49-72 N = 10,045[a]
Vasopressor use, n (%)				
Adrenaline	3488 (13%)	3049 (11%)	1414 (6.1%)	965 (5.7%)
Noradrenaline	21,440 (81%)	20,203 (76%)	13,114 (56%)	8184 (49%)
Vasopressin	4677 (18%)	3605 (14%)	2609 (11%)	1905 (11%)
Metaraminol	5502 (21%)	4317 (16%)	922 (4.0%)	1245 (7.4%)
Dopamine	3948 (15%)	3690 (14%)	2724 (12%)	1523 (9.0%)
Phenylephrine	282 (1.1%)	237 (0.9%)	77 (0.3%)	48 (0.3%)
More than one vasopressor	7991 (30%)	6556 (25%)	3200 (14%)	2457 (15%)
Median (IQR) dose, if any				
Adrenaline (μg/kg/min)	0.07 (0.04, 0.12)	0.07 (0.04, 0.14)	0.06 (0.03, 0.11)	0.05 (0.03, 0.10)
Noradrenaline (μg/kg/min)	0.06 (0.04, 0.10)	0.07 (0.04, 0.12)	0.06 (0.03, 0.10)	0.05 (0.03, 0.09)
Vasopressin (units/min)	0.034 (0.026, 0.039)	0.037 (0.027, 0.040)	0.034 (0.020, 0.040)	0.032 (0.022, 0.038)
Metaraminol (μg/kg/unit)	0.26 (0.04, 0.51)	0.11 (0.03, 0.43)	0.04 (0.02, 0.34)	0.41 (0.25, 0.71)
Dopamine (μg/kg/min)	3.45 (2.74, 4.54)	3.53 (2.86, 4.86)	3.86 (2.83, 4.94)	3.48 (2.50, 4.54)
Phenylephrine (μg/kg/min)	1.28 (0.78, 2.00)	1.25 (0.80, 1.94)	1.18 (0.55, 1.92)	1.47 (0.66, 2.53)

[a] Median (IQR) or Frequency (%).

42%) had a mean MAP of 70–74 mmHg, followed by 75–79 mmHg (2383; 21%) and 65–69 mmHg (2579; 23%). For all other analysed subgroups, a MAP group of 70–74 mmHg was the most common, followed by the next most common groups of either 65–69 mmHg or 75–79 mmHg.

4. Discussion

4.1. Key findings

In this multicentre study, out of ≈90,000 critically ill patients, more than 1/3rd received at least 6 h of vasopressor therapy. Among these patients, the mean hourly MAP during vasopressor therapy ranged from 72 to 75 mmHg within the first 72 h of ICU admission. Furthermore, patients who received vasopressors commenced therapy within a median of 2 h of ICU admission, and vasopressors were most often administered for more than 24 h. Noradrenaline was the most common vasopressor. However, nearly one in four patients received an alternative agent, and in three received more than one concurrent vasopressor. When patients were grouped into seven MAP categories, nearly half had a mean MAP between 70 and 75 mmHg over the first 72 h. Finally, in all ten prespecified subgroups representing distinct clinical situations where haemodynamic management may need to differ, the most common mean MAP group was consistently 70–75 mmHg.

4.2. Relationship to literature

The finding that most patients have a MAP between 70 and 75 mmHg whilst receiving vasopressors demonstrates that the usual practice in Queensland differs from the guidelines and recently published literature. The Surviving Sepsis Guidelines

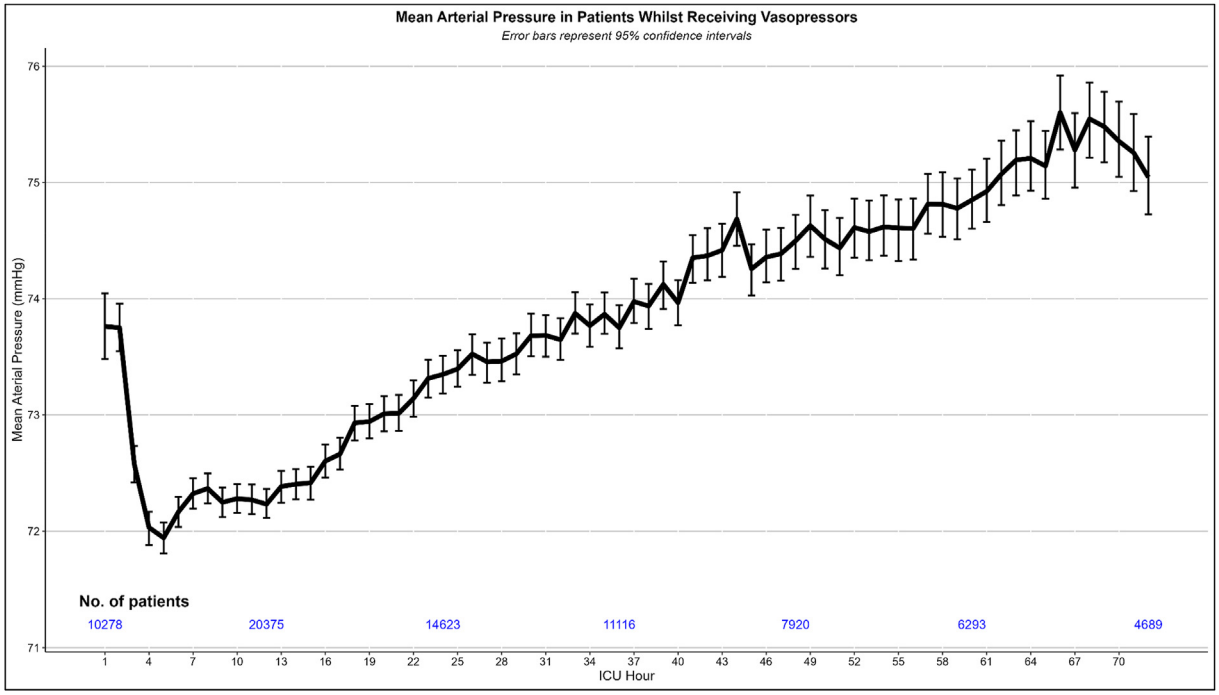

Fig. 1. Mean arterial pressure in patients receiving vasopressors.

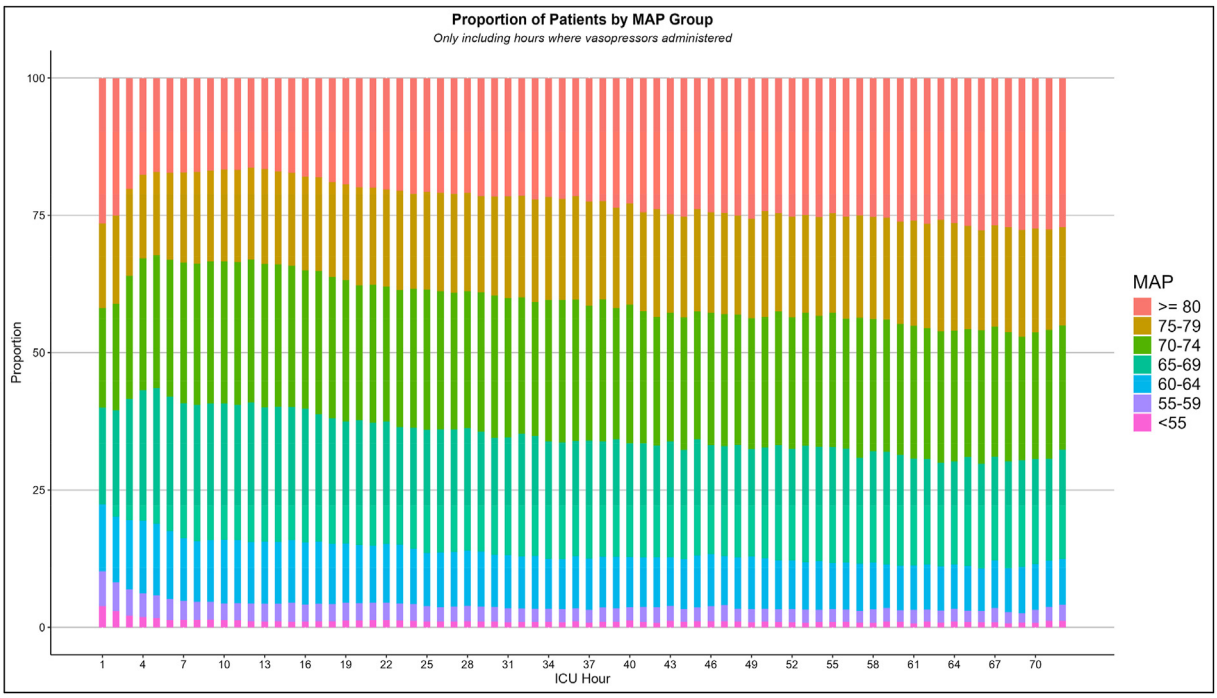

Fig. 2. Proportion of patients by MAP. Mean arterial pressure (MAP) values were obtained from the hourly average of MAP values and were categorised into one decimal place (example 74.3).

recommendations[17] and the recent international survey of intensive care clinicians support an optimal minimum target MAP threshold of 65 mmHg.[8] A recent systematic review and a meta-analysis of 11 RCTs involving 5078 critically ill patients focused on lower versus higher MAP targets.[6] It found that patients with a lower MAP target had a lower mortality rate, though the included studies' lower MAP group ranged from MAP 50–70 mmHg. Furthermore, an individual patient meta-analysis of 3352 patients with vasodilatory shock demonstrated a reduced 90-day mortality with lower MAP.[7] The discrepancy between stated MAP goals[8] and reality may reflect a practice where, despite evidence and stated targets, clinicians actually believe a MAP of 70–75 is ideal. Alternatively, the actual or achieved MAP of 70–75 mmHg may be a natural consequence of targeting a 65–70 mmHg MAP where avoiding a MAP below the lower threshold is prioritised. This dissociation between target and actual MAP was also demonstrated in the 65 trial, where the 60–65 mmHg MAP group had a median MAP of 66.7 mmHg.[18]

Despite extensive literature examining optimal MAP targets in the subgroups analysed in this study, no individual RCT has demonstrated a clinical benefit for a MAP target different from 65 mmHg.[19–22] The SEPSISPAM study examined higher MAP targets, 80–85 mmHg, in a cohort of critically ill patients with sepsis; however, compared to patients with MAP 65–70 mmHg, it found no difference in outcomes with a higher target.[23] The '65 trial', the largest trial in the meta-analysis, compared permissive hypotension (MAP of 60–65 mmHg) to a MAP over 65 in 2598 critically ill patients. It found that, compared to usual care, permissive hypotension did not lead to a statistically significant difference in 90-day mortality and reduced the use of vasopressors.[18] The absence of robust practice-defining research to guide alternative MAP strategies for specific subgroups of critically ill patients, such as sepsis or older age, may be reflected in our study's findings, where the mean MAP was between 70 and 75 mmHg over the first 72 h of an ICU admission regardless of pathological state. This lack of variation is consistent with the recent international survey of intensive care clinicians, which found that the preferred optimal MAP was the same for all subgroups except for patients with active bleeding.[8]

4.3. Study implications

Our study's findings have several implications. First, our study demonstrated one in three patients admitted to the ICU receive at least 6 h of vasopressors, which equates to over 151,000 critical admissions a year in Australia alone.[24] Second, they demonstrate that the actual achieved MAP was between 70 and 75 mmHg despite sepsis guidelines and evidence suggesting a lower target may be safe in older patients and associated with less use of vasopressors. Third, the discrepancy between the achieved MAP and the clinician-reported target of 65 mmHg, a phenomenon seen in previous clinical trials,[18,25] suggests that any future intervention to achieve lower MAP targets may require modification to the current care processes. This may include instituting upper limit alarms for MAP or the application of automated delivery systems to align delivered MAP with prescribed MAP targets. Fourth, one in four patients on vasopressors received a vasopressor other than noradrenaline, suggesting that future interventions to target MAP should be agnostic to the agent used. Fifth, the lack of variation in the achieved MAP among physiologically different subgroups implies that clinicians homogenously apply the same MAP threshold to critically ill patients irrespective of their underlying pathophysiological process.

4.4. Strengths and limitations

Our study has several strengths. First, the data examined comes from a standard clinical information system used across the entire region's health service. The patient population encompasses different ICUs, from regional to tertiary, and all pathological processes, except those excluded from analysis. Consequently, the study's findings represent local practice and likely represent MAP

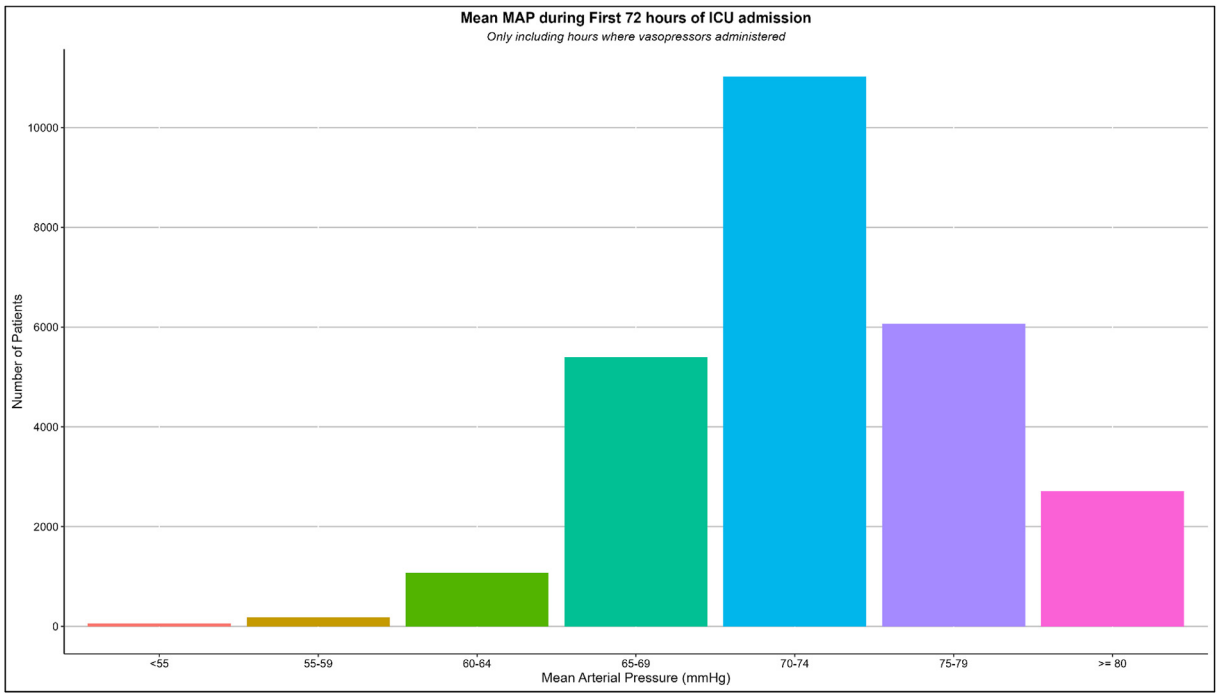

Fig. 3. Mean MAP during first 72 h of ICU admission. Mean arterial pressure (MAP) values were obtained from the hourly average of MAP values and were categorised to one decimal place (example 74.3).

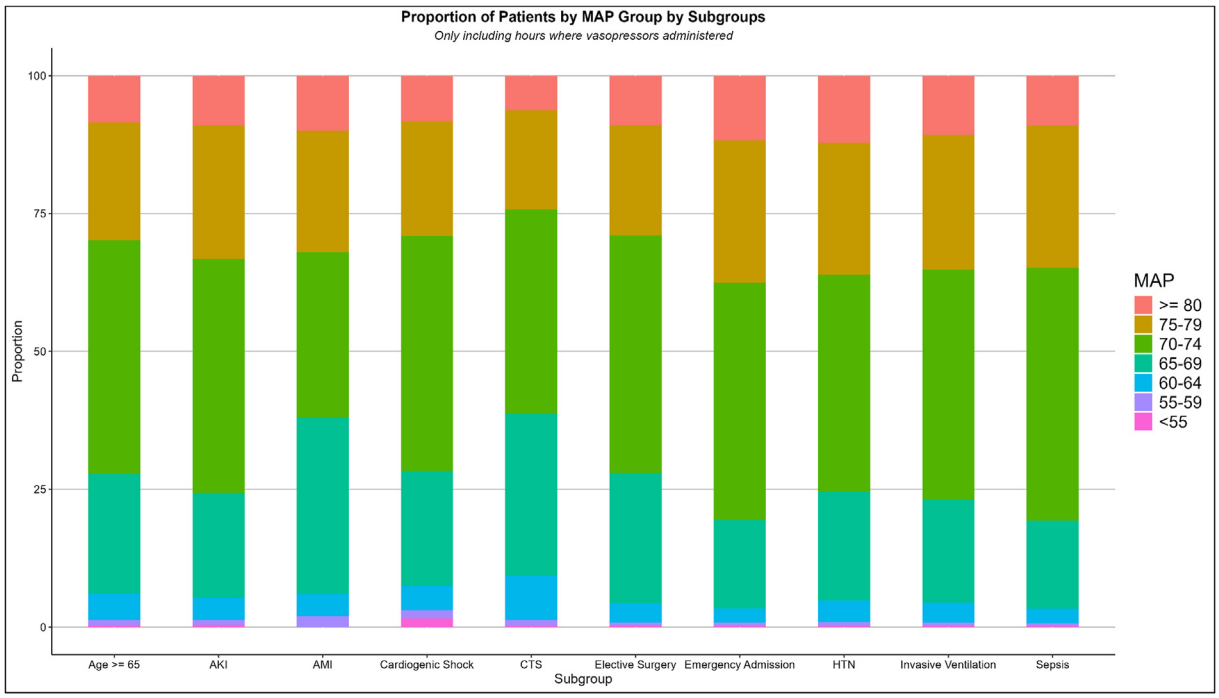

Fig. 4. Proportion of patients by MAP value bands according to clinical subgroups. Mean arterial pressure (MAP) values obtained from hourly average of MAP values and were categorised to one decimal place (example 74.3).
AKI = acute kidney injury; AMI = acute myocardial infarction; CTS = cardiothoracic surgery; HTN = chronic hypertension.

thresholds in Australia and New Zealand. Second, the study used granular data where important variables, such as validated MAP measurements and vasopressor administration, were averaged over an hour for the entire ICU admission. This level of detail has allowed for an accurate hour-to-hour analysis of MAP management in the cohort, ensuring variation in practice is not lost by aggregation or truncation of data. Third, our study classified patients into ten prespecified subgroups, which allowed for a robust assessment of potential differential practices across different pathological processes.

Table 2
Mean arterial pressure by subgroup.

Variable	Yes	No	p-value[a]
Sepsis			<0.001
Numbers	8842	17,676	
Mean (SD)	73.6 (5.4)	73.3 (6.3)	
Chronic hypertension			0.10
Numbers	7494	19,024	
Mean (SD)	73.6 (6.4)	73.3 (5.8)	
Cardiothoracic surgery			<0.001
Numbers	4868	21,650	
Mean (SD)	71.4 (5.5)	73.8 (6.0)	
Elective surgery			<0.001
Numbers	4846	21,672	
Mean (SD)	72.9 (6.0)	73.5 (6.0)	
Cardiogenic shock			<0.001
Numbers	389	26,129	
Mean (SD)	72.2 (6.7)	73.4 (6.0)	
Age≥65			<0.001
Numbers	11,410	15,108	
Mean (SD)	72.8 (5.9)	73.9 (6.1)	
Acute kidney injury			<0.001
Numbers	15,165	11,353	
Mean (SD)	73.1 (5.7)	73.8 (6.3)	
Acute myocardial infarction			0.11
Numbers	50	26,468	
Mean (SD)	71.8 (6.5)	73.4 (6.0)	
Invasive ventilation			<0.001
Numbers	21,063	5455	
Mean (SD)	73.5 (5.9)	73.0 (6.3)	
Emergency admission			<0.001
Numbers	17,580	8938	
Mean (SD)	74.0 (6.0)	72.2 (5.8)	

Abbreviations: SD = standard deviation.
[a] Wilcoxon rank sum test.

We acknowledge some limitations. Firstly, the study's retrospective nature has inherent limitations where intent cannot be determined, and causal inferences cannot be drawn. However, the study's primary outcome was the actual MAP delivered, which is an outcome of the data collection method can accurately detect. Second, the subgroup identification was based on data-derived definitions instead of prospective assessment. Thus, misclassification may have occurred. To attenuate such misclassification, recognised data definitions were used where able, and the homogeneous nature of the achieved MAP appeared independent of variation in cohort classification. Third, we did not include patients who received vasopressor for fewer than 6 h. The exclusion of this presumably less sick cohort may have biased the result. Fourth, our study was conducted in a single geographical area of Australia, which may limit the external validity of the results. Fifth, we examined MAP; however, there may be certain clinical scenarios where systolic blood pressure is targeted, not MAP. Lastly, the recorded MAP measurement may have been susceptible to measurement error due to clinical conditions, such as transducer placement, or errors with data entry. The impact is minimised by the use of validated data only, the removal of nonsensical values, and the very large dataset. Furthermore, the MAP values utilised in the analysis reflect information used for clinical decision making.

5. Conclusion

In a large, multicentre study involving a diverse cohort of critically ill patients, the mean hourly MAP during the receipt of vasopressors in the first 72 h of ICU admission was higher than the clinician-reported and often guideline-recommended or trial-supported minimum MAP threshold of 65 mmHg. The discrepancy between achieved and targeted MAP suggests that there is potential for trials targeting lower thresholds and decreased use of vasopressors within current practice.

Data availability statement

Data cannot be shared publicly due to institutional ethics, privacy, and confidentiality regulations. Data released for research under Sect. 280 of the Public Health Act 2005 requires an application to the Director-General of Queensland Health (PHA@health.qld.gov.au).

CRediT authorship contribution statement

The study conception and design (KW, PY, RB); data acquisition (KW); analysis (KW); interpretation of data (all authors); article drafting (KW, PY, RB), article revision for important intellectual content (all authors); final approval of the version submitted for publication (all authors); agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved (KW).

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Conflict of interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: R Bellomo and P Young are both members of the Critical Care and Resuscitation editorial board.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ccrj.2025.100103.

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