Section: CTVS

Original Research Article

IMPACT OF CEREBRAL PERFUSION PRESSURE DURING CARDIOPULMONARY BYPASS ON EARLY POSTOPERATIVE NEUROCOGNITIVE DYSFUNCTION

G. N. Prabhu[1], R. Swaminathan Veerasamy[2], Ms. Caroline Rachel Kumara Das[3], M. Dharmesh Raj[4]

[1]*Associate Professor, Department of Cardiothoracic and Vascular Surgery, SRM Medical College Hospital and Research Centre, Kattankulathur, Chengalpattu District, Tamil Nadu, India.*
[2]*Medical Superintendent and Senior Consultant, Department of Cardio Thoracic and Vascular Surgery, Institute of Cardiac Sciences, SRM Global Hospitals, SRM Nagar, Kattankulathur, Chengalpattu District, Tamil Nadu, India.*
[3]*Research Scholar (Ph.D), Department of Cardio Thoracic and Vascular Surgery, SRM Medical College Hospital and Research Centre, Kattankulathur, Chengalpattu District, Tamil Nadu, India.*
[4]*Junior Resident, Department of Cardio Thoracic and Vascular Surgery, SRM Medical College Hospital and Research Centre, Kattankulathur, Chengalpattu District, Tamil Nadu, India.*

Received *: 25/07/2026*
Received in revised form *: 19/08/2026*
Accepted *: 01/09/2026*

Corresponding Author:
Dr. G. N. Prabhu,
Associate Professor, Department of Cardiothoracic and Vascular Surgery, SRM Medical College Hospital and Research Centre, Kattankulathur, Chengalpattu District, Tamil Nadu, India.
Email: prabhu050563@yahoo.com

DOI: 10.70034/ijmedph.2026.3.675

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

Int J Med Pub Health
2026; 16 (3); 4317-4321

ABSTRACT

Background: Early postoperative neurocognitive dysfunction remains an important complication after cardiac surgery with cardiopulmonary bypass (CPB). Cerebral perfusion pressure (CPP) depends on mean arterial pressure (MAP) and intracranial pressure (ICP). Because ICP was not routinely monitored, this study examined cerebral perfusion indirectly using MAP during CPB.

Materials and Methods: This retrospective observational study included 90 adults aged 35-75 years who underwent CPB-supported cardiac surgery between January 2023 and December 2025. Mean CPB MAP was the principal pressure variable. MMSE was assessed preoperatively and on postoperative day 5. Early postoperative neurocognitive dysfunction was defined as a decrease of at least 2 points.

Results: Early neurocognitive dysfunction occurred in 28 patients (31.1%). Mean CPB MAP was lower in patients with dysfunction (61.61 ± 3.80 vs. 66.90 ± 5.59 mmHg; $P < 0.001$), and time with MAP <60 mmHg was longer (7.46 ± 7.85 vs. 3.03 ± 6.37 min; $P = 0.006$). Mean MAP correlated with MMSE change ($r = 0.346$, $P = 0.001$). After adjustment for age and CPB duration, higher mean MAP remained associated with lower odds of dysfunction (adjusted OR 0.787 per mmHg; 95% CI 0.687-0.901; $P = 0.001$).

Conclusion: Lower mean MAP and longer exposure to MAP <60 mmHg during CPB were associated with early postoperative neurocognitive dysfunction. Prospective studies are needed to evaluate individualized perfusion strategies and optimal pressure targets.

Keywords: cardiopulmonary bypass; mean arterial pressure; cerebral perfusion pressure; neurocognitive dysfunction; MMSE; cardiac surgery.

INTRODUCTION

Cardiac surgery with cardiopulmonary bypass (CPB) is associated with postoperative neurological and cognitive complications that may delay recovery. Current consensus uses perioperative neurocognitive disorders as an overarching term; cognitive decline within 30 days is classified as delayed neurocognitive recovery when formal diagnostic criteria are met.[1] Because this study used MMSE screening rather than a multidomain neuropsychological battery, the outcome is described as study-defined early postoperative neurocognitive dysfunction.

Postoperative cognitive decline is multifactorial and may involve cerebral microembolization, inflammation, haemodilution, anaemia, altered carbon dioxide tension, temperature changes, reduced oxygen delivery, and disturbed cerebral

blood flow. CPB-related exposures have been associated with postoperative cognitive change.[2]

Cerebral blood flow during CPB depends partly on MAP, pump flow, oxygen content, vascular resistance, carbon dioxide tension, and temperature. Autoregulatory limits vary between patients and may shift with age and vascular disease, so a single MAP target may not provide equivalent cerebral perfusion for all patients.[3]

Previous pressure-target studies have reported mixed findings. Gold et al. found fewer combined cardiac and neurological complications with higher CPB MAP targets, although cognitive outcomes were not significantly different.[4] Siepe et al. reported less early cognitive dysfunction and delirium at higher perfusion pressures.[5] Conversely, pressure above the upper autoregulatory limit has been associated with delirium.[6] Autoregulation-guided MAP management has therefore been investigated as an individualized strategy.[7]

The primary objective was to determine whether mean CPB MAP was associated with study-defined early postoperative neurocognitive dysfunction. Secondary objectives were to examine cumulative exposure to MAP below 60 mmHg and associations between pressure variables and postoperative MMSE change.

MATERIALS AND METHODS

Study design and setting

This retrospective observational study included adults undergoing CPB-supported cardiac surgery at SRM Medical College Hospital & Research Centre, Kattankulathur, Chennai, between January 2023 and December 2025. Reporting followed STROBE recommendations.[8]

Study population

Ninety records were included. Eligible patients were 35-75 years old, underwent cardiac surgery requiring CPB, and had complete perioperative, perfusion, and preoperative and postoperative MMSE records. Patients with dementia, severe pre-existing neurological impairment, incomplete CPB data, absent cognitive assessments, or surgery without CPB were excluded.

Clinical and perfusion variables

Collected variables included age, sex, BMI, hypertension, diabetes, previous stroke/TIA, LVEF, preoperative haemoglobin, baseline MMSE, procedure type, CPB duration, cross-clamp duration, nadir haematocrit, and MAP measurements. CPB management followed contemporary adult cardiac perfusion guidance.[9]

Cerebral perfusion pressure is physiologically defined as CPP = MAP - ICP. Contemporaneous ICP was not routinely available; therefore, direct patient-specific CPP could not be calculated. Mean CPB MAP was prespecified as the principal pressure variable representing the arterial-pressure component of cerebral perfusion. Minimum and maximum MAP and cumulative time with MAP below 60 mmHg were also recorded.

Cognitive outcome assessment

Cognitive function was assessed using the Mini-Mental State Examination (MMSE),[10] preoperatively and on postoperative day 5. The primary cognitive outcome was MMSE change. Early postoperative neurocognitive dysfunction was operationally defined as a decline of at least 2 points. This study-defined threshold was not considered equivalent to a formal perioperative neurocognitive disorder diagnosis. Delirium and clinical stroke were recorded separately.[11]

Statistical analysis

Continuous variables are reported as mean ± standard deviation and categorical variables as n (%). Complete-case analyses compared NCD groups using independent-samples t tests and chi-square or Fisher exact tests, as appropriate. Preoperative and postoperative MMSE scores were compared with a paired t test. Pearson correlation assessed associations between pressure variables and MMSE change. The principal MAP difference is reported with a 95% confidence interval.

Multivariable logistic regression evaluated adjusted associations with early NCD. With 28 outcome events, predictors were restricted to reduce overfitting.[12] The primary model included mean CPB MAP, age, and CPB duration; the secondary model replaced mean MAP with time below 60 mmHg. Adjusted ORs are reported with 95% CIs. Coefficients were also rescaled to 5-mmHg and 10-minute increments for clinical interpretation. Tests were two-sided with $P < 0.05$. Residual confounding remains possible.

Ethical considerations

The study was conducted in accordance with the Declaration of Helsinki and institutional ethical requirements. Institutional Ethics Committee approval was obtained before study commencement. Written consent was waived where applicable for this retrospective record-based study, and data were anonymized.

RESULTS

Baseline characteristics

Among 90 patients, mean age was 55.09 ± 12.27 years and 63 (70.0%) were male. Hypertension was present in 43 (47.8%), diabetes in 23 (25.6%), and previous CVA/TIA in 5 (5.6%). Early NCD occurred in 28 patients (31.1%).

Patients with early NCD were older (60.11 ± 11.61 vs. 52.82 ± 11.97 years; $P = 0.008$) and more often hypertensive (64.3% vs. 40.3%; $P = 0.035$). Other baseline characteristics, including preoperative MMSE, did not differ significantly. [Table 1]

Table 1: Baseline demographic and clinical characteristics

Variable	Total (n=90)	No NCD (n=62)	Early NCD (n=28)	P value
Age, years	55.09 ± 12.27	52.82 ± 11.97	60.11 ± 11.61	0.008
Male sex, n (%)	63 (70.0)	43 (69.4)	20 (71.4)	1.000
BMI, kg/m²	25.63 ± 3.72	25.27 ± 3.57	26.41 ± 3.97	0.179
Hypertension, n (%)	43 (47.8)	25 (40.3)	18 (64.3)	0.035
Diabetes mellitus, n (%)	23 (25.6)	13 (21.0)	10 (35.7)	0.138
Previous CVA/TIA, n (%)	5 (5.6)	2 (3.2)	3 (10.7)	0.172
LVEF, %	50.52 ± 8.26	49.65 ± 7.86	52.46 ± 8.93	0.135
Preoperative haemoglobin, g/dL	13.15 ± 1.27	13.13 ± 1.29	13.21 ± 1.24	0.793
Preoperative MMSE	28.09 ± 1.31	28.19 ± 1.34	27.86 ± 1.24	0.263

BMI, body mass index; CVA, cerebrovascular accident; TIA, transient ischaemic attack; LVEF, left ventricular ejection fraction; MMSE, Mini-Mental State Examination.

Operative and perfusion characteristics

CABG was performed in 54 patients, valve surgery in 18, and combined CABG plus valve surgery in 18. Procedure distribution differed between groups (P = 0.009), with combined surgery more frequent in early NCD. CPB and cross-clamp durations did not differ significantly.

Mean CPB MAP was lower in early NCD (61.61 ± 3.80 vs. 66.90 ± 5.59 mmHg; P < 0.001; mean difference -5.29 mmHg, 95% CI -7.30 to -3.28). Minimum and maximum MAP were also lower, while time below 60 mmHg was longer (7.46 ± 7.85 vs. 3.03 ± 6.37 min; P = 0.006). Nadir haematocrit did not differ significantly. [**Table 2]**

Table 2: Operative and cardiopulmonary bypass characteristics

Variable	Total	No NCD	Early NCD	P value
Procedure type, n (%)				**0.009**
CABG	54 (60.0)	41 (66.1)	13 (46.4)	
Valve surgery	18 (20.0)	14 (22.6)	4 (14.3)	
CABG + valve	18 (20.0)	7 (11.3)	11 (39.3)	
CPB duration, min	108.83 ± 26.72	106.77 ± 21.15	113.39 ± 36.22	0.279
Cross-clamp duration, min	79.08 ± 23.47	77.02 ± 19.04	83.64 ± 31.07	0.217
Mean CPB MAP, mmHg	65.25 ± 5.65	66.90 ± 5.59	61.61 ± 3.80	<0.001
Minimum MAP, mmHg	52.92 ± 6.18	54.67 ± 6.05	49.04 ± 4.55	<0.001
Maximum MAP, mmHg	78.17 ± 6.32	79.89 ± 6.15	74.35 ± 4.91	<0.001
Time MAP <60 mmHg, min	4.41 ± 7.12	3.03 ± 6.37	7.46 ± 7.85	0.006
Nadir haematocrit, %	24.51 ± 2.26	24.67 ± 2.36	24.15 ± 2.04	0.323

CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; MAP, mean arterial pressure.

Cognitive and postoperative outcomes

Mean MMSE decreased from 28.09 ± 1.31 preoperatively to 27.00 ± 1.86 on postoperative day 5 (mean change -1.09 ± 1.28; P < 0.001). Decline was greater in early NCD (-2.75 ± 0.84 vs. -0.34 ± 0.48; P < 0.001).

Early NCD was associated with longer mechanical ventilation, ICU stay, and hospital stay. Delirium occurred in 5 patients and was more frequent with early NCD (14.3% vs. 1.6%; P = 0.031). One patient developed clinical stroke **(Table 3).** These associations do not establish causal direction.

Table 3: Cognitive and postoperative outcomes

Variable	Total	No NCD	Early NCD	P value
Preoperative MMSE	28.09 ± 1.31	28.19 ± 1.34	27.86 ± 1.24	0.263
POD 5 MMSE	27.00 ± 1.86	27.85 ± 1.38	25.11 ± 1.31	<0.001
Change in MMSE	-1.09 ± 1.28	-0.34 ± 0.48	-2.75 ± 0.84	<0.001
Ventilation, h	10.78 ± 3.83	9.36 ± 3.19	13.91 ± 3.27	<0.001
ICU stay, days	2.75 ± 0.98	2.44 ± 0.85	3.45 ± 0.90	<0.001
Hospital stay, days	8.79 ± 1.73	8.39 ± 1.75	9.65 ± 1.36	0.001
Delirium, n (%)	5 (5.6)	1 (1.6)	4 (14.3)	0.031
Clinical stroke, n (%)	1 (1.1)	1 (1.6)	0	1.000

POD, postoperative day; ICU, intensive care unit; MMSE, Mini-Mental State Examination.

Association with MMSE change and multivariable analysis

Mean CPB MAP correlated positively with MMSE change (r = 0.346, P = 0.001), while time below 60 mmHg correlated negatively (r = -0.210, P = 0.047). The modest correlation is consistent with a multifactorial cognitive outcome.

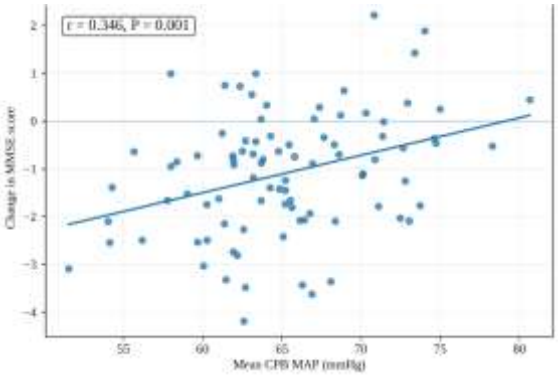

Figure 1: Association between mean CPB MAP and change in MMSE score (r = 0.346, P = 0.001)

After adjustment for age and CPB duration, higher mean CPB MAP was associated with lower odds of early NCD (adjusted OR 0.787 per mmHg; 95% CI 0.687-0.901; P = 0.001), equivalent to OR 0.30 per 5-mmHg increase. Age was significant, whereas CPB duration was not. In the secondary model, time below 60 mmHg was associated with higher odds of NCD (OR 1.078 per minute; 95% CI 1.008-1.153; P = 0.029), equivalent to OR 2.12 per 10 minutes **(Table 4).** These rescalings do not imply treatment effects.

Table 4: Multivariable logistic regression for early postoperative neurocognitive dysfunction

Variable	Adjusted OR	95% CI	P value
Primary model			
Age, per year	1.048	1.002-1.095	0.039
Mean CPB MAP, per mmHg	0.787	0.687-0.901	0.001
CPB duration, per min	1.005	0.986-1.024	0.590
Secondary model			
Age, per year	1.049	1.007-1.094	0.023
Time MAP <60 mmHg, per min	1.078	1.008-1.153	0.029
CPB duration, per min	1.007	0.989-1.025	0.448

OR, odds ratio; CI, confidence interval; CPB, cardiopulmonary bypass; MAP, mean arterial pressure.

DISCUSSION

Lower mean MAP and greater cumulative exposure to MAP below 60 mmHg during CPB were associated with early MMSE decline. Early NCD occurred in 31.1% of patients and was also associated with older age, hypertension, and combined CABG plus valve surgery. Mean MAP and low-pressure exposure remained associated with NCD after adjustment for age and CPB duration.

Age is a recognized risk marker for postoperative cognitive dysfunction,[13] while diabetes has also been associated with increased risk.[14] The age association observed here is therefore biologically plausible and may reflect reduced cognitive reserve and cerebrovascular vulnerability, although the present study cannot establish mechanism.

The principal finding was a 5.29-mmHg lower mean CPB MAP in patients with early NCD, together with an adjusted MAP association and a modest correlation with MMSE change. This pattern suggests that arterial pressure may contribute to risk but explains only part of postoperative cognitive variability. Cerebral autoregulation provides a plausible framework because patient-specific pressure limits vary, and impaired autoregulation during CPB has been associated with cognitive deterioration.[15] The association with time below 60 mmHg further suggests that both pressure magnitude and duration may be relevant.

These findings are directionally consistent with prior pressure-target studies.[4,5] More recent work has emphasized individualized autoregulation-guided management: targeting MAP above a patient-specific lower autoregulatory limit reduced delirium in one nested trial,[16] while a larger randomized trial did not improve its primary neurological endpoint but reported favorable secondary delirium and memory findings.[7] Reviews therefore support further evaluation of individualized cerebral perfusion monitoring rather than a universal MAP target.[17]

Early NCD was also associated with longer ventilation, ICU stay, hospital stay, and delirium. These relationships may be bidirectional: postoperative illness can worsen cognitive performance, while neurological vulnerability may accompany more complicated recovery. Causal ordering cannot be determined from these data.

Strengths and limitations

Strengths include paired preoperative and postoperative cognitive assessment, routinely recorded CPB pressure metrics, analysis of both mean MAP and low-pressure exposure, and reporting of adjusted estimates with confidence intervals while limiting model size to the available outcome events.

Limitations include the retrospective single-centre design, complete-case selection, modest sample size, and residual confounding. Hypertension and procedure type differed between groups but were not included in the parsimonious models. MMSE is a global screening tool, the 2-point decline threshold was operational rather than diagnostic, and high baseline MMSE may reduce sensitivity to subtle domain-specific change. Follow-up ended on postoperative day 5. Direct CPP could not be calculated because contemporaneous ICP was unavailable; MAP therefore represents only the measured arterial-pressure component of cerebral perfusion. Autoregulation and cerebral oxygenation

data were also unavailable, so no individual optimal CPP or MAP target can be identified.

Clinical implications and future research

These findings support prospective studies using continuous haemodynamic data, multidomain neuropsychological testing, cerebral autoregulation and oxygenation monitoring, and longer follow-up. Larger samples should permit broader adjustment for comorbidity and surgical complexity. The rescaled MAP and low-pressure estimates are useful for hypothesis generation but should not be interpreted as expected treatment effects. Randomized or carefully designed prospective studies are needed before specific pressure targets are recommended.

CONCLUSION

Lower mean MAP and longer exposure to MAP below 60 mmHg during CPB were associated with study-defined early postoperative neurocognitive dysfunction. Older age was also associated with the outcome. Because direct CPP was unavailable and the study was retrospective with limited covariate adjustment, causality and optimal pressure targets cannot be established. Prospective studies incorporating autoregulation-guided perfusion assessment, cerebral oxygenation monitoring, and comprehensive cognitive testing are warranted.

REFERENCES

1. Evered L, Silbert B, Knopman DS, Scott DA, DeKosky ST, Rasmussen LS, et al. Recommendations for the nomenclature of cognitive change associated with anaesthesia and surgery-2018. Br J Anaesth. 2018;121(5):1005-1012. doi:10.1016/j.bja.2017.11.087.
2. Greaves D, Psaltis PJ, Ross TJ, Davis D, Smith AE, Boord MS, Keage HA. Cognitive outcomes following coronary artery bypass grafting: a systematic review and meta-analysis of 91,829 patients. Int J Cardiol. 2019;289:43-49.
3. Caldas JR, Haunton VJ, Panerai RB, Hajjar LA, Robinson TG. Cerebral autoregulation in cardiopulmonary bypass surgery: a systematic review. Interact Cardiovasc Thorac Surg. 2018;26(3):494-503.
4. Gold JP, Charlson ME, Williams-Russo P, Szatrowski TP, Peterson JC, Pirraglia PA, et al. Improvement of outcomes after coronary artery bypass: a randomized trial comparing intraoperative high versus low mean arterial pressure. J Thorac Cardiovasc Surg. 1995;110(5):1302-1314. doi:10.1016/S0022-5223(95)70053-6.
5. Siepe M, Pfeiffer T, Gieringer A, Zemann S, Benk C, Schlensak C, Beyersdorf F. Increased systemic perfusion pressure during cardiopulmonary bypass is associated with less early postoperative cognitive dysfunction and delirium. Eur J Cardiothorac Surg. 2011;40(1):200-207. doi:10.1016/j.ejcts.2010.11.024.
6. Hori D, Nomura Y, Ono M, Joshi B, Mandal K, Cameron D, et al. Optimal blood pressure during cardiopulmonary bypass defined by cerebral autoregulation monitoring. J Thorac Cardiovasc Surg. 2017;154(5):1590-1598.
7. Hogue CW, Brown CH IV, Hori D, Ono M, Nomura Y, Balmert LC, et al. Personalized blood pressure management during cardiac surgery with cerebral autoregulation monitoring: a randomized trial. Semin Thorac Cardiovasc Surg. 2021;33(2):429-438. doi:10.1053/j.semtcvs.2020.09.032.
8. Ghaferi AA, Schwartz TA, Pawlik TM. STROBE reporting guidelines for observational studies. JAMA Surg. 2021;156(6):577-578.
9. Wahba A, Milojevic M, Boer C, De Somer FM, Gudbjartsson T, Van Den Goor J, et al. 2019 EACTS/EACTA/EBCP guidelines on cardiopulmonary bypass in adult cardiac surgery. Eur J Cardiothorac Surg. 2020;57(2):210-251.
10. Siqueira GS, Hagemann PD, Coelho DS, Santos FH, Bertolucci PHF. Can MoCA and MMSE be interchangeable cognitive screening tools? A systematic review. Gerontologist. 2019;59(6):e743-e763.
11. Kong H, Xu LM, Wang DX. Perioperative neurocognitive disorders: a narrative review focusing on diagnosis, prevention, and treatment. CNS Neurosci Ther. 2022;28(8):1147-1167.
12. van Smeden M, Moons KGM, de Groot JAH, Collins GS, Altman DG, Eijkemans MJC, Reitsma JB. Sample size for binary logistic prediction models: beyond events per variable criteria. Stat Methods Med Res. 2019;28(8):2455-2474.
13. Bowden T, Hurt CS, Sanders J, Aitken LM. Predictors of cognitive dysfunction after cardiac surgery: a systematic review. Eur J Cardiovasc Nurs. 2022;21(3):192-204.
14. Feinkohl I, Winterer G, Pischon T. Diabetes is associated with risk of postoperative cognitive dysfunction: a meta-analysis. Diabetes Metab Res Rev. 2017;33(5):e2884.
15. Kumpaitiene B, Svagzdiene M, Sirvinskas E, Adomaitiene V, Petkus V, Zakelis R, et al. Cerebrovascular autoregulation impairments during cardiac surgery with cardiopulmonary bypass are related to postoperative cognitive deterioration: prospective observational study. Minerva Anestesiol. 2019;85(6):594-603. doi:10.23736/S0375-9393.18.12358-3.
16. Brown CH IV, Neufeld KJ, Tian J, Probert J, LaFlam A, Max L, et al. Effect of targeting mean arterial pressure during cardiopulmonary bypass by monitoring cerebral autoregulation on postsurgical delirium among older patients: a nested randomized clinical trial. JAMA Surg. 2019;154(9):819-826.
17. Vranken NPA, Weerwind PW, Sutedja NA, Severdija EE, Barenbrug PJC, Maessen JG. Cerebral oximetry and autoregulation during cardiopulmonary bypass: a review. J Extra Corpor Technol. 2017;49(3):182-191.