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Contents

Comparison of intraoperative electroencephalographic measures of anesthetic sensitivity for predicting postoperative delirium in elderly surgical patients

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232

AUTHORS

Morisson, Louis;[1,2] Laferrière-Langlois, Pascal;[1,2] Ouahdi, Youcef;[3] Denault, André;[2] Richebé, Philippe;[4] Duclos, Catherine[2,5,6]

[1]Department of Anesthesiology and Pain Medicine, Hôpital Maisonneuve-Rosemont, CIUSSS de l'Est de l'Ile de Montréal, Montréal, Québec, Canada; [2]Department of Anesthesiology and Pain Medicine, Université de Montréal, Montréal, Québec, Canada; [3]Faculty of Medicine, Université de Montréal, Montréal, Québec, Canada; [4]Department of Anesthesia and Intensive Care, Polyclinique Bordeaux Nord Aquitaine (PBNA), Bordeaux, France; [5]Department of Neuroscience, Université de Montréal, Montréal, Québec, Canada; [6]Center for Advanced Research in Sleep Medicine, CIUSSS du Nord-de-Île-de-Montréal (CIUSSS-NIM), Montréal, Québec, Canada

INTRODUCTION

Heightened sensitivity to anesthetics has been proposed as a marker of brain vulnerability associated with postoperative delirium (POD). Several intraoperative electroencephalographic (EEG) definitions have emerged: EEG suppression at lower volatile anesthetic concentrations using mixed-effects modeling[1], the Duke Anesthesia Resistance Scale (DARS) based on processed EEG[2] and intraoperative anesthetic dose-adjusted EEG alpha power[4]. However, these methods have not been directly compared. Furthermore, we previously showed that post-induction burst-suppression (BS) following standardized propofol induction identifies vulnerable patients with distinct preoperative EEG signatures. The relationship between this phenotype and other intraoperative sensitivity measures remains unknown. We aimed to compare four intraoperative anesthetic sensitivity definitions and evaluate their associations with POD in elderly surgical patients.

METHODS

This secondary analysis included 240 patients aged 70 years or older from an ongoing randomized trial (POEGEA study) undergoing major non-cardiac surgery. We calculated four anesthetic sensitivity measures: post-induction BS following standardized propofol induction (1-1.5 mg/kg); Fritz sensitivity defined as random intercept above population median from a mixed-effects logistic model predicting EEG suppression at each end-tidal volatile anesthetic concentration; low DARS defined as mean bispectral index divided by (2.5 minus age-adjusted minimum alveolar concentration) below 28.755; and low dose-adjusted

frontal alpha power. The optimal cutoff for dose-adjusted alpha power was determined using Youden's index with bootstrap resampling for confidence intervals. POD was assessed using the Confusion Assessment Method on postoperative days 1, 2, and 7. We compared delirium rates across sensitivity definitions using Fisher exact tests, calculated odds ratios with 95% confidence intervals, and evaluated diagnostic performance (sensitivity, specificity). Agreement between measures was assessed using Cohen's kappa coefficient.

RESULTS

POD occurred in 14 patients (5.8%). The prevalence of heightened sensitivity varied by definition: post-induction BS 19%, high Fritz sensitivity 50%, low DARS 31%, and low dose-adjusted alpha (optimal cutoff 3.5 dB) 28%. Fritz sensitivity showed the strongest association with POD (OR 14.5, 95% CI 2.8-266, p=0.01; POD rate 11.2% vs 0.9%), followed by intraoperative low dose-adjusted alpha power (OR 7.3, 95% CI 2.4-27.6, p=0.001) and post-induction BS (OR 6.5, 95% CI 2.2-20.9, p=0.001). Low DARS was not significantly associated with POD (OR 2.0, 95% CI 0.6-6.3 p=0.22). Fritz sensitivity captured 93% of delirium cases compared to 71% for low alpha, 57% for BS, and 43% for DARS. Agreement was moderate between Fritz sensitivity and dose-adjusted alpha power (kappa=0.42), but poor between DARS and other measures (kappa<0.20). Notably, 92% of patients with post-induction BS also demonstrated high Fritz sensitivity.

DISCUSSION

Intraoperative anesthetic sensitivity measures identify overlapping but not identical patient populations at risk for POD. Fritz sensitivity and dose-adjusted alpha power showed the strongest associations with delirium, while DARS did not predict POD in this non-cardiac surgical cohort. The high concordance between post-induction BS and Fritz sensitivity validates that both methods capture similar vulnerable phenotypes. These findings suggest that EEG-derived measures of anesthetic sensitivity may be more clinically relevant than processed EEG indices. Prospective validation is warranted.

REFERENCES

1. Fritz BA, et al. Br J Anaesth. 2018;121:241-248.
2. Cooter Wright M, et al. Anesth Analg. 2022;134:149-158.
3. Reese M, et al. Br J Anaesth. 2025;135:109-120.

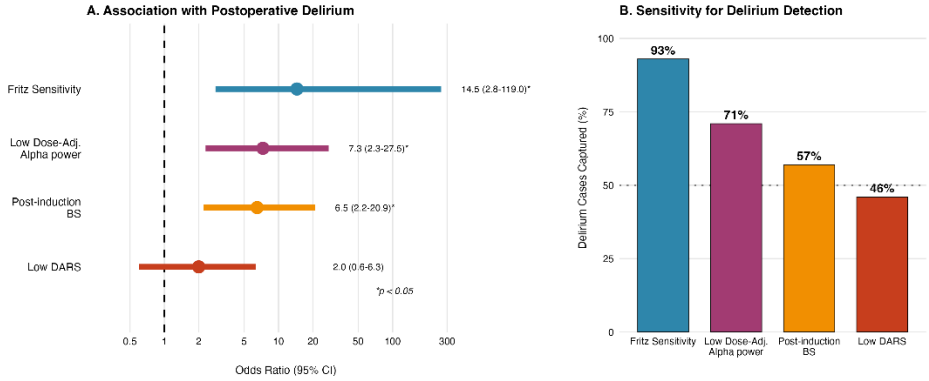
A. Association with Postoperative Delirium
Fritz Sensitivity
Low Dose-Adj. Alpha power
Post-Induction BS
Low DARS
14.5 (2.8-119.0)*
7.3 (2.3-27.5)*
6.5 (2.2-20.9)*
2.0 (0.6-6.3)
*p < 0.05
0.5
1
2
5
10
20
50
100
300
Odds Ratio (95% CI)
B. Sensitivity for Delirium Detection
Delirium Cases Captured (%)
100
75
50
25
0
93%
71%
57%
46%
Fritz Sensitivity
Low Dose-Adj. Alpha power
Post-induction BS
Low DARS

Figure 1

Harms of common interventional procedures for chronic noncancer spine pain: a systematic review and meta-analysis of non-randomized studies

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155

AUTHORS

Asif, Mohammad Saad;[1] Malam, Faheem;[2,3] Khalid, Muhammad F.;[5] Leafloor, Cameron W.;[5] Hong, Patrick J.;[6] Levit, Tal;[7] Zeraatkar, Dena;[2,8] Wang, Li;[2,8] Couban, Rachel J.;[8] Agarwal, Arnav;[2] Agoritsas, Thomas;[2,9] Busse, Jason W.;[2,8,10]

[1]Department of Anesthesiology and Pain Medicine, University of Ottawa, Ottawa, Canada; [2]Department of Health Research Methods, Evidence, and Impact, McMaster University, Hamilton, Canada; [3]Department of Family Medicine, University of Ottawa, Ottawa, Canada; [4]Department of Anesthesiology and Pain Medicine, University of Ottawa, Ottawa, Canada; [5]Department of Emergency Medicine, University of Ottawa, Ottawa, Canada; [6]Department of Anesthesiology and Pain Medicine, University of Toronto, Toronto, Canada; [7]Division of Plastic Surgery, McMaster University, Hamilton, Canada; [8]Department of Anesthesia, McMaster University, Hamilton, Canada; [9]Department of Medicine, University Hospitals of Geneva, Geneva, Switzerland; [10]Michael G. DeGroote National Pain Centre, McMaster University, Hamilton, Canada

INTRODUCTION

Chronic, non-cancer spine pain is a leading cause of morbidity, years lived with disability, and productivity loss globally[1]. Clinicians frequently offer patients living with chronic spine pain interventional procedures, such as joint or epidural injections with corticosteroids or anesthetics, medial branch blocks or radiofrequency ablation. These procedures may be associated with rare but serious harms, such as deep infection or paralysis[2]. Randomized trials in this area typically enroll small numbers of patients followed for short time frames, limiting their ability to inform infrequent harms.

We conducted a systematic review and meta-analysis of non-randomized studies to summarize the evidence on adverse events of interventional procedures for chronic non-cancer spine pain. Our findings, as well as a second systematic review[3], informed a parallel *BMJ Rapid Recommendation* addressing interventional procedures for chronic spine pain[4]. This evidence synthesis is part of the *BMJ Rapid Recommendations* project, a collaborative effort from the *MAGIC Evidence Ecosystem Foundation* and the *BMJ*[5].

METHODS

We performed a systematic review and meta-analysis. Data sources included MEDLINE, EMBASE and CINAHL. A parallel guideline panel provided input on the scope, design, and interpretation of this systematic review, including selection of adverse events for consideration. Using standardized pilot-tested forms, three pairs of trained reviewers screened titles and abstracts of identified citations independently and in duplicate. We included all non-randomized studies in which: 1) at least 80% of participants were adult patients (>18 years old), presenting with chronic (>12 weeks or explicitly defined by study authors as 'chronic') axial and/or radicular, noncancer spine pain, (2) received an interventional procedure considered by our guideline panel and, (3) reported harms as defined by the authors. Systematic literature screening, data abstraction and risk of bias appraisal was conducted independently and in duplicate by pairs of reviewers.

We used DerSimonian-Laird random-effects models for all meta-analyses. We conducted analyses for comparative and non-comparative studies separately. Where possible, we reported risk differences with associated 95% confidence intervals (95% CIs) for comparative studies. Subroup analyses were conducted only if there were two or more studies in each subgroup. To evaluate certainty of evidence, we used the GRADE approach.

RESULTS

We included 60 longitudinal studies (56 non-comparative, 4 comparative), that enrolled 4,966 patients with chronic spine-related pain. Low certainty evidence suggests that joint targeted steroid injection, and epidural steroid injection for chronic spine pain may result in an increased prevalence of temporary altered level of consciousness (prevalence: 2.1%; 95%CI 0.7 to 4.1), joint radiofrequency nerve ablation, joint targeted steroid injection, and epidural injection of local anesthetic and steroids may increase the risk of deep infection (prevalence: 0.4%; 95%CI 0 to 1.5), and epidural steroid injection, joint radiofrequency nerve ablation, and joint targeted injection of local anesthetic and steroids may increase the risk of dural puncture (prevalence: 1.6%; 95%CI 0.2 to 3.7). Several common interventional procedures may increase prevalence of metabolic complications, and prolonged sensory deficits, pain or stiffness (prevalence ranged from 8.2% to 16.3%), but the supporting evidence was only very low certainty.

DISCUSSION

In this systematic review of observational studies, we found low certainty evidence that suggests interventional procedures for chronic spine pain may increase the prevalence of several harms. Specifically, temporary altered level of consciousness following joint targeted steroid injection, and epidural steroid injection, deep infection following joint radiofrequency nerve ablation, joint targeted steroid injection, and epidural injection of local anaesthetic and steroids, and dural puncture following epidural steroid injection, joint radiofrequency nerve ablation, and joint targeted injection of local anaesthetic and steroids. Other harms are uncertain due to very low certainty evidence.

REFERENCES

1. Knezevic NN, Candido KD, Vlaeyen JWS, et al. Low back pain. *Lancet* 2021;398(10294):78-92. doi: 10.1016/s0140-6736(21)00733-9 [published Online First: 20210608]
2. Glaser SE, Falco F. Paraplegia following a thoracolumbar transforaminal epidural steroid injection. *Pain Physician* 2005;8(3):309-14.
3. Wang X, Martin G, Sadeghirad B, et al. Common interventional procedures for chronic non-cancer spine pain: a systematic review and network meta-analysis of randomised trials. BMJ 2025;388:e079971. doi: 10.1136/bmj-2024-079971 [published Online First: 20250219]
4. Busse JW, Genevay S, Agarwal A, Standaert CJ, Carneiro K, Friedrich J, et al. *Commonly used interventional procedures for non-cancer chronic spine pain: a clinical practice guideline.* BMJ 2025;388:e079970. doi: 10.1136/bmj-2024-079970 [published Online First: 20250219]
5. Siemieniuk RA, Agoritsas T, Macdonald H, et al. Introduction to BMJ Rapid Recommendations. *Bmj* 2016;354:i5191. doi: 10.1136/bmj.i5191 [published Online First: 20160928]

Table 1: GRADE evidence profile for adverse events associated with interventional procedures for chronic spinal pain in single-arm observational studies

Adverse Event	Types of Interventional Procedures Administered	No. of studies (range of follow-up)	No. of Patients	Prevalence (95% CI)	Reasons for Downgrading	Certainty of Evidence
Deep Infection	Joint radiofrequency ablation or denervation (3 studies) Fluoroscopy-guided epidural injections with both corticosteroid and local anesthetic (1 study) CT-guided joint injection with corticosteroid (1 study)	5 (12 to 90 weeks)	539	0.4% (0 – 1.5)	Risk of bias Imprecision	Low
Dural Puncture	Epidural corticosteroid injections (5 studies) Radiofrequency ablation (2 studies) Nerve blocks (1 study)	8 (12 to 52 weeks)	1097	1.6% (0.2 – 3.7)	Risk of bias Imprecision	Low
Temporary Altered Level of Consciousness	Epidural steroid injections (9 studies) Joint steroid injections (1 study)	10 (12 to 52 weeks)	1021	2.1% (0.7 – 4.1)	Risk of bias	Low
Prolonged (>48 hours) Sensory Deficits	Joint radiofrequency ablation dorsal root ganglion (2 studies) Radiofrequency ablation (2 studies) Radiofrequency neurotomy and corticosteroid injection (1 study	5 (12 to 104 weeks)	363	8.2% (1.8 – 17.9)	Risk of bias Imprecision	Very low
Prolonged (>48) Pain or Stiffness	Radiofrequency ablation procedures (16 studies)	16 (8 to 105 weeks)	1132	16.3% (7.7 – 27.1)	Risk of bias Imprecision	Very low

Incidence of postoperative intracranial hemorrhage in awake craniotomy patients receiving NSAIDS: a retrospective single-institution review

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52

AUTHORS

Elmestekawy, Mohamed;[1] Serhan, Mohamed;[1] Polis, Tom;[2] Sinclair, John;[3] Budiansky, Adele[2]

[1]Faculty of Medicine, University of Ottawa, Ottawa, Canada; [2]Department of Anesthesiology and Pain Medicine, University of Ottawa, Ottawa, Canada; [3]Division of Neurosurgery, Department of Surgery, University of Ottawa, Ottawa, Canada

INTRODUCTION

Awake craniotomy facilitates maximal resection of tumours involving eloquent cortical/subcortical white matter, requiring anesthetic strategies that balance adequate perioperative analgesia with intraoperative patient alertness and cooperation. Although non-steroidal anti-inflammatory (NSAID) medications can provide opioid-sparing analgesia, their use in patients undergoing craniotomy is often limited by theoretical concerns of postoperative intracranial hemorrhage (PICH). Recent meta-analyses report reoperation rates for PICH ranging from 1.3-2.1%[1-3]; however, this outcome has not been examined specifically in awake craniotomy cohorts. Our institution performs ~120 awake craniotomies annually in patients considered high risk for awake surgery, including older adults, and those with significant medical comorbidities. The distinct physiological and pharmacologic features of this approach introduce uncertainty regarding NSAID safety in this population. This study aimed to quantify PICH incidence in adult patients undergoing awake craniotomy at our institution who received perioperative NSAIDs and compare this incidence with rates reported in the literature.

METHODS

This study was completed under a Quality Improvement Waiver from or institutions's Health Science Network Research Ethics Board, received on December 20, 2024, for a quality assurance retrospective review and database of patients undergoing awake craniotomy for brain tumour resection at our institution. Patients ≥17 years treated between June 2019-July 2023 were included, and those receiving perioperative NSAIDs were identified for analysis.

All patients were managed under a standardized Monitored Anesthesia Care protocol including regional and multimodal analgesia. Perioperative NSAID use was defined as preoperative oral celecoxib or postoperative celecoxib and/or intravenous ketorolac

administered day of surgery at the anesthesiologists' discretion. The primary outcome was PICH requiring surgical evacuation during the postoperative admission with a secondary outcome of symptomatic PICH causing neurological deficit; identified through review of postoperative discharge summaries, with CT imaging obtained following clinical neurological assessment. Continuous and categorical variables were summarized as means (SD) and frequencies (%). PICH incidence was calculated as a proportion with a 95% confidence interval. Institutional performance was benchmarked against a weighted average reoperation rate of 1.3-2.1% derived from large contemporary international cohorts (N > 10,800)[1-3]. Comparison to the upper benchmark threshold of 2.1% was performed using a one-sample binomial test ($p<.05$).

RESULTS

A total of 429 charts of patients who underwent awake craniotomy were reviewed. Out of these, 340 adult patients received perioperative NSAIDs (mean age 54.8 (15.9) years, range 17-87; 44% female; mean BMI 26.7 (5.5) kg/m^2, range 15.1-47.5). The majority were classified as ASA physical status III (69%) or IV (26%), with high rates of cardiac (30%) and respiratory (21%) disease. Data regarding baseline kidney function was not available. Preoperative celecoxib was the predominant NSAID administered (79%). Symptomatic PICH causing neurological deficit occurred in 11 patients (3.2%). PICH requiring surgical evacuation occurred in 6 patients (all of whom underwent a primary rather than repeat awake craniotomy), yielding an overall incidence of 1.8% (95% CI: 0.7% to 3.8%). This incidence was not statistically significantly different from the upper benchmark of 2.1% reported in general craniotomy cohorts ($p=0.85$).

DISCUSSION

The 1.8% reoperation rate observed in this cohort, among the largest reported for NSAID-exposed craniotomy patients, is consistent with established craniotomy benchmarks[4-5]. These results suggest that the use of NSAIDs as part of a multimodal analgesic strategy in clinically complex patients undergoing awake craniotomy is safe. However, there are multiple limitations to this study, including its retrospective nature and confounding by indication, as discretionary NSAID use may have favoured patients with lower baseline bleeding risk. Accordingly, while these findings are reassuring, future studies using matched cohorts are necessary to more definitively establish safety and to further refine hemorrhage risk stratification.

REFERENCES

1. Wang C, Niu X, Ren Y, Lan Z, Zhang Y. Risk Factors for Postoperative Intracranial Hemorrhage After Resection of Intracranial Tumor in 2259 Consecutive Patients. World Neurosurg. 2019 Sep;129:e663-e668. doi: 10.1016/j.wneu.2019.05.239. Epub 2019 Jun 6. Erratum in: World Neurosurg. 2020 Feb;134:684. doi: 10.1016/j.wneu.2019.11.082. PMID: 31176060.

2. Kageji T, Nagahiro S, Mizobuchi Y, Nakajima K. Postoperative Hematoma Requiring Recraniotomy in 1149 Consecutive Patients With Intracranial Tumors. Oper Neurosurg. 2017 Jun 1;13(3):392-397. doi: 10.1093/ons/opw045. PMID: 28521349.
3. Senders JT, Goldhaber NH, Cote DJ, Muskens IS, Dawood HY, De Vos FYFL, Gormley WB, Smith TR, Broekman MLD. Venous thromboembolism and intracranial hemorrhage after craniotomy for primary malignant brain tumors: a National Surgical Quality Improvement Program analysis. J Neurooncol. 2018 Jan;136(1):135-145. doi: 10.1007/s11060-017-2631-5. Epub 2017 Oct 16. PMID: 29039075; PMCID: PMC5754452.
4. Perez JL, Naylor RM, Bauman MMJ, Harrison DJ, Knowles K, Burns TC, Lanzino G, Link MJ, Peris Celda M, Meyer FB, Van Gompel J, Parney IF. Safety of the immediate use of nonsteroidal anti-inflammatory drugs after adult craniotomy for tumor. J Neurosurg. 2024 Feb 2;141(1):117-122. doi: 10.3171/2023.11.JNS231600. PMID: 38306640.
5. Sriram S, Miller P, Reilly T, Ebrahim G, Ali M, Chowdhury MAB, Sorrentino Z, Chen S, Ghiaseddin A, Koch M, Rahman M. Safety and Efficacy of Ketorolac After Craniotomy for Tumor Resection. World Neurosurg. 2025 Feb;194:123339. doi: 10.1016/j.wneu.2024.10.068. Epub 2024 Nov 12. PMID: 39447744.

Non-invasive assessment of cardiac oxygenation using photoacoustic imaging

Submission ID

185

AUTHORS

Noble, Ronan;[1,2] Vu, Jennie;[2,3,4] Khodabocus, Ibrahim;[2,3,5] Derzi, Simone;[6] Henry, Matthew;[5,7] Bourque, Stephane[2,3,4,5,6,7]

[1]Department of Anesthesiology, Perioperative and Pain Medicine, Cumming school of medicine, University of Calgary; [2]Women and Children's Health Research Institute, University of Alberta, Edmonton, Canada; [3]Cardiovascular Research Institute, University of Alberta, Edmonton, Canada; [4]Department of Physiology, University of Alberta, Edmonton, Canada; [5]Department of Pediatrics, University of Alberta, Edmonton, Alberta, Canada; [6]Department of Anesthesiology and Pain Medicine, University of Alberta, Edmonton, Alberta, Canada; [7]Division of Pediatric Cardiology, University of Alberta, Edmonton, Canada

INTRODUCTION

Perioperative incidents such as hypoxic cardiac injury, present significant diagnostic challenges due to their often subtle or nonspecific clinical presentation [1]. However, reductions in myocardial oxygenation and perfusion precedes biochemical changes, as well as changes in ECG and wall motion abnormalities [2,3]. Photoacoustic imaging (PAI) is a modality that uses laser irradiation of a tissue to generate ultrasonic waves, which can then be detected by ultrasound. PAI enables spatially resolved quantitative mapping of various molecules, such as oxygenated and deoxygenated hemoglobin (based on their differential absorption spectra of 850 and 750 nm respectively) [4]. The objective of this study was to evaluate the application of PAI in monitoring oxygenation of the myocardium and great vessels and benchmark its performance against conventional ECG and echocardiography as a potential early warning sign for myocardial hypoxemia. We then sought to test the myocardial response to select vasoactive and inotropic agents.

METHODS

Male CD-1 Elite mice were positioned supine and anesthetized with isoflurane (4% induction, 2% maintenance) carried in 1 L/min medical-grade gas (composition described below), as previously described [5]. Four imaging protocols were conducted in three separate cohorts of mice. Photoacoustic and simultaneous B-mode ultrasound images of the myocardium and right ventricular outflow tract (RVOT), as well as the pulmonary artery and aorta were acquired at steady states with a fractional inspired oxygen fraction (FiO_2) of 21% (medical grade air) and then 100% (medical grade oxygen). In the same cohort of mice, the FiO_2 was then reduced to 10% for 60 seconds by introducing medical grade nitrogen

using a gas mixer, and then restored to 100%. A second cohort of mice were anesthetized and instrumented with a 26G IV. Photoacoustic and simultaneous B-mode ultrasound images of the myocardium and right ventricular outflow tract (RVOT) were again recorded. Mice received IV phenylephrine and isoproterenol at a 2:1 ratio doubling every 8 minutes. Finally, in a third cohort of mice the individual effects of various inotropes and vassopressors were assessed. Each mouse was given increasing doses of phenylephrine, isoproterenol, or norepinephrine. Each protocol was stopped when myocardial oxygen saturation decreased by ~50%.

RESULTS

PAI reliably distinguished changes in oxygenation in the myocardium, RVOT chamber, aorta, and pulmonary artery when the FiO_2 was changed from 100% to 21% ($P<0.0001$) and from 21% to 10% ($P<0.0001$). With escalating doses of phenylephrine and isoproterenol a progressive myocardial desaturation as well as decreasing RVOT oxygenation occurred (-50%, $P<0.001$). Importantly, we found when the myocardium is stressed its oxygen saturation drops below that of the RVOT oxygen saturation indicating the unique sensitivity of the heart ($P<0.01$). Cardiac contractility, measured from B-mode ultrasound, did not significantly reduce until myocardial oxygenation decreased by 17% ($P<0.05$). Further, PAI detected a 10% reduction in myocardial oxygenation prior to the onset of ECG abnormalities ($P<0.05$). Finally, we tested the response to individual agents. Isoproterenol ($P<0.01$) and phenylephrine ($P<0.001$) alone each caused a more rapid desaturation than when the agents were given together. Mice demonstrated a greater tolerance to norepinephrine than phenylephrine or a combination of isoproterenol and phenylephrine.

DISCUSSION

While PAI is presently studied in humans for other purposes, the ability to measure oxygenation within the aorta and pulmonary artery has the potential to offer a more central measure of oxygenation than conventional pulse oximetry, as well as serve as a non-invasive measure of oxygen extraction. This work also demonstrates the potential for PAI to be integrated into the existing monitoring armamentarium, as it may offer an earlier warning of cardiac dysfunction and injury compared to existing monitoring devices. Finally, this work compared various pharmacological agents and showed how at large doses norepinephrine may be preferred over phenylephrine.

REFERENCES

1. Wittmann M, Dinc T, Kunsorg A, Marcucci M, Ruetzler K. Preventing, identifying and managing myocardial injury after non cardiac surgery - a narrative review. Curr Opin Anaesthesiol. 2025 Feb 1;38(1):17-24. doi: 10.1097/ACO.0000000000001454.
2. Landesberg G, Mosseri M, Wolf Y, Vesselov Y, Weissman C. Perioperative myocardial ischemia and infarction: identification by continuous 12-lead electrocardiogram with

online ST-segment monitoring. Anesthesiology. 2002 Feb;96(2):264-70. doi: 10.1097/00000542-200202000-00007.
3. Arnold JR, Karamitsos TD, Bhamra-Ariza P, Francis JM, Searle N, Robson MD, Howells RK, Choudhury RP, Rimoldi OE, Camici PG, Banning AP, Neubauer S, Jerosch-Herold M, Selvanayagam JB. Myocardial oxygenation in coronary artery disease: insights from blood oxygen level-dependent magnetic resonance imaging at 3 tesla. J Am Coll Cardiol. 2012 May 29;59(22):1954-64. doi: 10.1016/j.jacc.2012.01.055.
4. Attia ABE, Balasundaram G, Moothanchery M, Dinish US, Bi R, Ntziachristos V, Olivo M. A review of clinical photoacoustic imaging: Current and future trends. Photoacoustics. 2019 Nov 7;16:100144. doi: 10.1016/j.pacs.2019.100144.
5. Noble RMN, Kirschenman R, Wiedemeyer A, Patel V, Rachid JJ, Zemp RJ, Davidge ST, Bourque SL. Use of Photoacoustic Imaging to Study the Effects of Anemia on Placental Oxygen Saturation in Normoxic and Hypoxic Conditions. Reprod Sci. 2024 Apr;31(4):966-974. doi: 10.1007/s43032-023-01395-6.

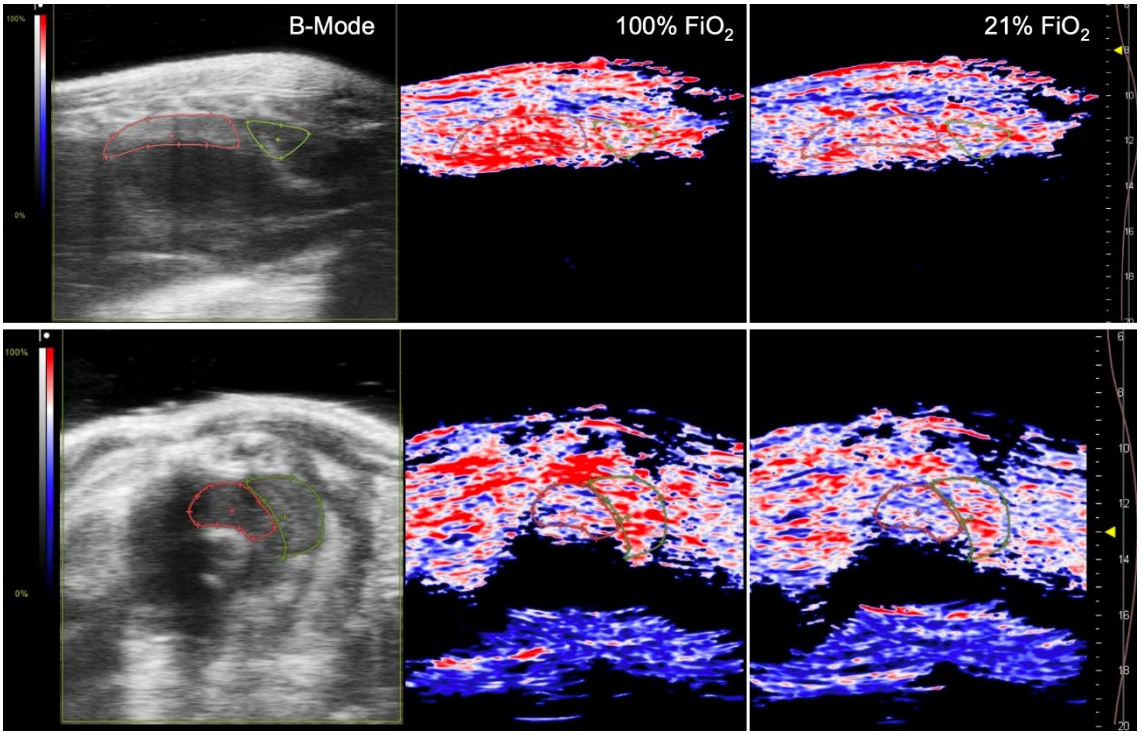

Figure 1

Predictors of delayed urinary catheter removal following enhanced recovery after cesarean delivery: a retrospective cohort study

Submission ID

47

AUTHORS

Barahi, Sunti;[1] Kuberan, Aswini;[1] Ye, Xiang;[2] George, Ronald B;[1] Siddiqui, Naveed;[1]

[1]Department of Anesthesia and Pain Management, Mount Sinai Hospital/ University of Toronto, Toronto, Canada; [2]Department of Biostatistics, Princess Margaret Cancer Center, UHN, Toronto, Canada

INTRODUCTION

Enhanced Recovery After Cesarean (ERAC) pathways endorse early postoperative urinary catheter removal to promote mobilization and reduce catheter-associated urinary tract infection (CAUTI).[1-3] The Society of Obstetric Anesthesia and Perinatology (SOAP) ERAC consensus specifically recommends removing the catheter within 6–12 hours postpartum in uncomplicated cesarean deliveries.[3] While the Enhanced Recovery Canada (ERC) pathway likewise encourages early removal, it notes ongoing uncertainty regarding the absolute optimal duration.[4] At our institution, early catheter removal is a key ERAC process measure linked to superior recovery outcomes and improved patient satisfaction. Nevertheless, recent local audit data reveal a significant implementation gap: approximately 30–40% of patients are discharged from the post-anesthesia care unit (PACU) with a catheter in situ. Prolonged catheterization may delay ambulation, increase CAUTI risk, and impair patient experience. Our study aims to identify clinical and process-of-care factors associated with delayed urinary catheter removal after cesarean delivery within our ERAC program.

METHODS

This retrospective cohort study was approved by the institutional research ethics board. All cesarean deliveries over a 12-month period at a high-volume tertiary obstetric centre with an established ERAC pathway were screened. Patients aged 18–49 years with BMI 18.5–50 $kg{\cdot}m^{-2}$ undergoing elective, urgent, or emergent cesarean under neuraxial or general anesthesia were included. Exclusion criteria included underlying renal or bladder pathology, pre-existing neurological deficits, bladder or bowel injury, postpartum hemorrhage, surgical duration >3 hours, or other clinical indications for prolonged catheterization. Data were abstracted from electronic records on demographics, cesarean urgency, anesthetic technique, neuraxial local anesthetic and opioid doses, intraoperative fluid volume, vasopressor and oxytocin use, surgical time, PACU motor block (Bromage 0–3) and sensory

levels. Catheter insertion time was defined as the start of anesthesia; removal time and any instances of recatheterization were obtained from nursing documentation. The primary outcome was delayed catheter removal, defined a priori as duration >360 minutes or the need for recatheterization. Univariate and multivariable logistic regression were used to identify independent predictors, with covariates selected based on clinical relevance and $p<0.10$ on univariable analysis.

RESULTS

Of 391 patients screened, 337 met inclusion criteria. Delayed catheter removal occurred in 151/337 patients (44.8%). Baseline age, BMI, gestational age, and cesarean urgency did not differ between groups. Delayed removal was associated with spinal (vs epidural) anesthesia, denser PACU motor block, ward (vs PACU) catheter removal, higher intraoperative fluid volumes, longer times to motor block assessment and complete motor recovery. In the final multivariable model, independent predictors of delay were PACU motor block, catheter removal setting, intraoperative fluid volume, time to motor block assessment, and time to Bromage 0. Compared with complete motor block, partial recovery (Bromage 1–2) was associated with lower odds of delay (OR 0.20 [95% CI 0.08–0.51] and 0.44 [0.20–0.95], respectively). Catheter removal in PACU markedly reduced delay (OR 0.13 [0.05–0.33]). Each 1200-mL increase in fluids tripled the odds of delay (OR 3.12 [1.53–6.35]); longer times to motor assessment and full recovery also increased risk.

DISCUSSION

Despite an established ERAC pathway, nearly half of cesarean patients experienced delayed urinary catheter removal. Our findings highlight modifiable perioperative factors including intraoperative fluid administration, timing of motor block assessment, residual PACU motor block, and the location of catheter removal that strongly influence catheter duration. Process-of-care changes, such as formalizing PACU-based catheter removal protocols, standardizing Bromage-guided assessments, and promoting fluid stewardship may enhance adherence to ERAC, SOAP ERAC, and ERC recommendations,[1–4] reduce CAUTI risk and improve early mobilization. These data support targeting motor recovery and workflow factors, rather than patient demographics, when designing patient-safety interventions for post-cesarean urinary catheter management.

REFERENCES

1. Wilson RD, Caughey AB, Wood SL, et al. Guidelines for antenatal and intrapartum care in cesarean delivery (ERAS-related content). Am J Obstet Gynecol. 2018;219:523.e1–523.e15.
2. Caughey AB, Cahill AG, Guise JM, Rouse DJ. Safe prevention of the primary cesarean delivery. Am J Obstet Gynecol. 2014;210:179–193.

3. Bollag LA, Lim G, Sultan P, et al. Society for Obstetric Anesthesia and Perinatology consensus statement and recommendations for Enhanced Recovery After Cesarean Delivery (ERAC). Anesth Analg. 2021;132:1362–1377.
4. Enhanced Recovery Canada. Clinical Pathway for Cesarean Delivery. Version 1; 2022–2023. 5. Walker M, et al. Implementation of enhanced recovery after surgery for cesarean delivery: a quality improvement initiative. BMJ Open Qual. 2025;14:e003391

The association of air pollution on postoperative mortality after inpatient scheduled surgery: a systematic review and meta-analysis

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AUTHORS

Rathod, Dhruv;[1] Ding, Josephine;[2] Dourka, Jasmeen;[3] Kishibe, Teruko;[4] Evans, Greg;[5] Chung-Wai, Chow;[6] Brook, Jeffrey;[6] Baxter, Nancy;[7] Gershon, Andrea;[8] Sankar, Ashwin[4,9]

[1]Department of Medicine, Queen's University, Kingston, Canada; [2]Temerty Faculty of Medicine, University of Toronto, Toronto, Canada; [3]Schulich School of Medicine and Dentistry, Western University, London, Canada; [4]Li Ka Shing Knowledge Institute, Unity Health Toronto, Toronto, Canada; [5]Department of Chemical Engineering and Applied Chemistry, University of Toronto, Toronto, Canada; [6]Occupational and Environmental Health Division, Dalla Lana school of Public Health, University of Toronto, Toronto, Canada; [7]Department of General Surgery, Faculty of Medicine, University of Sydney, Sydney, Australia; [8]Division of Respirology, Sunnybrook Health Sciences Centre, Toronto, Canada; [9]Department of Anesthesiology and Pain Medicine, St. Michael's Hospital and the University of Toronto, Toronto, Canada

INTRODUCTION

In Canada, nearly 5% of patients die within 1-year after inpatient surgeries of which approximately 2.2 million are performed annually.[1] Few interventions improve these rates, suggesting that broader determinants of health, such as environmental factors, influence postoperative outcomes.[2] The World Health Organization attributes 23% of global deaths to environmental factors.[3] In the general population, air pollutant exposure is associated with inflammation, oxidative stress, and endothelial dysfunction – mechanisms that increase postoperative risk.[4] Although small observational studies have linked air pollution exposure to postoperative mortality, existing evidence remains limited to transplant populations and has not been comprehensively summarized.[5] Clarifying the impact of air pollution on outcomes after surgery could inform patient-level risk stratification and influence policy changes to mitigate population-level exposure. We conducted a systematic review and meta-analysis to quantify the association between air pollution exposure and postoperative mortality among patients undergoing scheduled inpatient surgery.

METHODS

This systematic review (PROSPERO CRD420251081067) is reported according to PRISMA guidelines. We searched MEDLINE, Embase, and Web of Science for English language studies published from January 2005 to March 2025. We included studies of adults (≥ 18 years) undergoing scheduled inpatient surgery and excluded emergency and outpatient

procedures. Eligible environmental exposures were ambient $PM_{2.5}$, PM_{10}, NO_2, and O_3 measured using fixed-site monitoring data, geospatial models, or individual monitors. Eligible study designs included randomized and observational studies. Two reviewers independently screened titles and abstracts, assessed full texts, and extracted data with disagreements resolved by a third reviewer. Risk of bias (ROB) was assessed using the Newcastle-Ottawa Scale. The primary outcome was postoperative mortality over any reported follow-up duration after surgery. Maximally adjusted hazard ratios were pooled with random-effects meta-analysis using restricted maximum likelihood estimation for between-study variance. Confidence intervals were calculated using Knapp-Hartung adjustments to account for small numbers of studies. Heterogeneity was quantified using the I^2 statistic. Pollutant exposure estimates were rescaled to a 10 $\mu g/m^3$ increase. Prespecified subgroup analyses examined type of surgery (transplant versus non-transplant), exposure measurement timepoint (preoperative versus postoperative air pollutant measurement), exposure duration (averaged pollutant exposure over less than a year or greater), and follow-up duration.

RESULTS

Our search identified 3861 records and included 13 studies; there were sufficient studies reporting associations between each of $PM_{2.5}$ and PM_{10} with mortality for meta-analysis. Each 10 $\mu g/m^3$ increase in $PM_{2.5}$ exposure was associated with higher risk of postoperative mortality (11 studies; HR 1.24, CI 1.06-1.46; I^2 = 97%). In subgroup analyses, associations between $PM_{2.5}$ and mortality differed by exposure measurement timepoint (postoperative HR 1.33, CI 1.02-1.72 versus preoperative HR 1.03, CI 0.76-1.41; subgroup differences $p < 0.001$) and exposure measurement duration (>1 year HR 1.35, CI 1.05-1.74 versus $\leq$1 year HR 1.08, CI 0.88-1.17; subgroup differences p = 0.03). Associations did not differ by transplant versus non-transplant surgery (p = 0.13) or follow-up duration (p = 0.73). PM_{10} exposure was not associated with postoperative mortality (6 studies; HR 1.15, CI 0.83-1.61; I^2 = 83%). Most studies were of low ROB in terms of outcome ascertainment.

DISCUSSION

Higher $PM_{2.5}$ exposure was associated with increased postoperative mortality after scheduled inpatient surgery. Heterogeneity was substantial due to diversity in included procedures, countries, and exposure assessments. Subgroup analyses suggested associations between $PM_{2.5}$ and mortality in subgroups with exposure measurement periods longer than one year, and included the postoperative period. These findings support considering exposure to airborne fine particulate matter over longer durations and during the postoperative period as a potential risk factor for death after surgery. Future research is needed to identify mechanisms underlying this association, and to identify pollutant exposure thresholds that support postoperative recovery.

REFERENCES

1. Sankar A, Thorpe K, McIsaac DI, Luo J, Wijeysundera DN, Gershon AS. Survival and health care costs after inpatient elective surgery: comparison of patients with and without chronic obstructive pulmonary disease. *CMAJ*. 2023 Jan 17;195(2):E62–E71. doi:10.1503/cmaj.220733.
2. POISE Study Group. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. Lancet. 2008 May 31;371(9627):1839-1847. doi:10.1016/S0140-6736(08)60601-7.
3. World Health Organization. Preventing disease through healthy environments: a global assessment of the burden of disease from environmental risks [Internet]. Geneva: World Health Organization; 2016 [cited 2025 Dec 24]. Available from: https://www.who.int/publications/i/item/9789241565196
4. Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation. 2010 Jun 1;121(21):2331-2378. doi:10.1161/CIR.0b013e3181dbece1.
5. Supphapipat K, Leurcharusmee P, Chattipakorn N, Chattipakorn SC. Impact of air pollution on postoperative outcomes following organ transplantation: evidence from clinical investigations. *Clin Transplant*. 2024;38(1):e15180. doi:10.1111/ctr.15180.

A

Study	Design	Sample Size (N)	Pollutants	Type of Surgery	Exposure Timepoint	Exposure Duration (>1 year or ≤1 year)	Follow-up Duration (years)[a]
Al-Kindi et al. (2019)	Retrospective Cohort	21,800	PM2.5	Heart Transplant	Postoperative	>1 year	Median 4.8
Ambuieya et al. (2024)	Retrospective Cohort	18,265	PM2.5	Lung Transplant	Postoperative	>1 year	Maximum 10
Chang et al. (2021)	Retrospective Cohort	112,098	PM2.5	Kidney Transplant	Postoperative	>1 year	Median 6
Chuang et al. (2024)	Retrospective Cohort	7426	PM2.5, PM10, O3, NO2	Orthopedic	Preoperative and Postoperative	>1 year	1 year
Dehom et al. (2021)	Retrospective Cohort	93,695	PM2.5, O3, NO2	Kidney Transplant	Postoperative	>1 year	Median 14.91
Deo et al. (2024)	Retrospective Cohort	26,403	PM2.5	Cardiac	Postoperative	>1 year	Median 4.9
Feng et al. (2021)	Retrospective Cohort	87,223	PM2.5	Kidney Transplant	Preoperative and Postoperative	≤1 year	Median 5.3
Kim et al. (2021)	Retrospective Cohort	1146	PM10	Kidney Transplant	Postoperative	≤1 year	1
Liu et al. (2023)	Prospective Cohort	3327	PM2.5, O3	Thoracic	Postoperative	≤1 year	Mean 2.14
Oh et al. (2024)	Retrospective Cohort	244,766	PM2.5, PM10, O3, NO2	Abdominal/Thoracic	Preoperative	≤1 year	1
Spencer-Hwang et al. (2011)	Retrospective Cohort	32,239	PM10, O3	Kidney Transplant	Postoperative	>1 year	Maximum 7
Urbanowicz et al. (2024)	Retrospective Cohort	283	PM2.5, PM10, NO2	Cardiac	Postoperative	>1 year	Median 5.3
Yoon et al. (2020)	Retrospective Cohort	398	PM2.5, PM10, O3, NO2	Neurosurgical	Preoperative	≤1 year	1

[a] Follow-up duration reported as presented in the original studies. Participants were followed until the outcome of interest, or follow-up was administratively censored at the study-specified maximum duration or at a fixed analytic window (e.g., 1-year mortality).

B

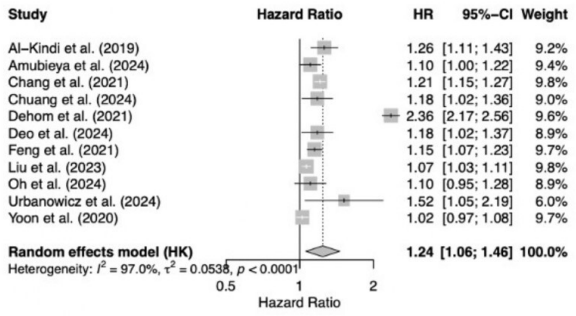

Figure 1. The association between air pollutant exposure with postoperative mortality following scheduled inpatient surgery. **(A)** Characteristics of included studies. **(B)** Primary analysis of association between PM2.5 exposure with postoperative mortality following scheduled inpatient surgery.