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Should All Patients With Pulmonary Hypertension Undergoing Non-Cardiac Surgery Be Managed by Cardiothoracic Fellowship-Trained Anesthesiologists?

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Abstract

Objectives: To identify differences in practice patterns and outcomes related to the induction of general anesthesia for patients with pulmonary hypertension (PH) performed by anesthesiologists who have completed a cardiothoracic fellowship (CTA group) vs those who have not (non-CTA group).

Design: Retrospective study with propensity score matching.

Setting: Operating room.

Participants: All adult patients with PH undergoing general anesthesia requiring intubation at a single academic center over 5 years.

Interventions: Patient baseline characteristics, peri-induction management variables, post-induction mean arterial pressure (MAP), and other outcomes were compared between CTA and non-CTA groups.

Methods and main results: Following propensity scoring matching, 402 patients were included in the final model, 100 in the CTA group and 302 in the non-CTA group. Also following matching, only cases of mild to moderate PH without right ventricular dysfunction remained in the analysis. Matched groups were overall statistically similar with respect to baseline characteristics; however, there was a greater incidence of higher ASA class ($P = .025$) and cardiology and thoracic procedures ($P < .001$) being managed by the CTA group. No statistical differences were identified in practice patterns or outcomes related to the induction of anesthesia between groups, except for longer hospital length of stay in the CTA group ($P = .008$).

Conclusions: These results provide early evidence to suggest the induction of general anesthesia of patients with non-severe PH disease can be comparably managed by either anesthesiologists with or without a cardiothoracic fellowship. However, these findings should be confirmed in a prospective study.

Keywords

pulmonary hypertension, post-induction hypotension, cardiothoracic anesthesiology fellowship, non-cardiac surgery, practice patterns

Introduction

Pulmonary hypertension (PH) affects 1% of the general population and threatens right ventricular (RV) function.[1] A database analysis of over 17 million patients hospitalized after surgery demonstrated patients with PH were 43% more likely to suffer major adverse cardiac events after adjusting for other variables, compared to those without the diagnosis.[2] In a separate study, 62 patients with severe PH were case-matched with

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controls and more likely to suffer post-operative heart failure, delayed extubation, and increased mortality.[3] There are a variety of potential mechanisms by which patients with PH are more susceptible to poorer outcomes following perioperative stressors, though the hemodynamic effects of anesthetic agents during the induction phase in this vulnerable population are largely unknown.[4] During anesthetic induction, vasodilation and venodilation lead to decreased coronary blood flow, and a transition from negative to positive pressure ventilation can further exacerbate RV afterload. PH predisposes patients to RV failure and inadequate perfusion in the setting of additional peri-surgical risk factors that further compromise RV function.

Surgeons often recognize that patients with complex cardiac comorbidities are at increased perioperative risk and make special requests for anesthesiologists who have completed a cardiothoracic anesthesiology fellowship to manage these cases.[5] The rationale for this practice stems from the assumption that this group has more experience in managing patients with complex cardiac pathophysiology and therefore might help mitigate their patients' risk. How this subspecialty training and experience influences anesthesiologists' conduct of anesthetic induction in complex cardiac patients undergoing non-cardiac surgery has yet to be described. Furthermore, despite the lack of evidence suggesting improvement in outcomes, academic institutions are seeing an increasing number of requests for cardiothoracic anesthesiologists for non-cardiac cases, and this can be logistically challenging.[6] Therefore, in this study, we aimed to compare practice patterns and outcomes of anesthetic induction between anesthesiologists who have completed a CTA fellowship vs those who have not, for a population of patients with a diagnosis of PH.

The transition from wakefulness to general anesthesia can have dramatic hemodynamic consequences that are determined solely by decisions made by the anesthesiologist and the patient's physiological responses to those decisions. Therefore, there should be few confounding variables related to the procedure that also impact the occurrence of post-induction hypotension. Although focusing on a single phase of management does not allow for a comprehensive appraisal of the value of a subset of anesthesiologists, the degree of post-induction hypotension was selected to serve a surrogate for how care is taken to anticipate and prevent sudden drops in blood pressure. We hypothesized we would identify differences in peri-induction practice patterns between cardiothoracic and non-cardiothoracic anesthesiologists and that those differences would be associated with less hemodynamic instability following anesthetic induction in the cardiothoracic group.

Methods

This study was approved by the Institutional Review Board (IRB# 21-0412) at the University of North Carolina at Chapel Hill on June 7, 2021. For this retrospective observational study, all patients having undergone surgery at a single academic institution from October 2015 to December 2020 with an International Classification of Disease diagnostic code in their electronic health record for pulmonary hypertension (ICD-10 I27) were identified using BusinessObjects™ (SAP®, Paris, France). Patients were included if they were >18 years old, underwent non-cardiac surgery, and were undergoing general anesthesia with intubation. Exclusion criteria included mechanical circulatory support, anesthesia induction prior to the surgical encounter, or inadequate documentation of blood pressure or induction medications. Patient data were collected by either BusinessObjects™ or manual data extraction directly from the electronic health records. Sensitive patient information was stored using a RedCap® database system (RedCap Consortium, Vanderbilt University, Nashville, TN). For internal quality control, numerous audits were performed by members of the data collection team who did not originally collect the data to minimize the effect of bias and human error.

Baseline patient demographic and comorbidity data were collected, as well as potential predictors of post-induction hemodynamics derived from preoperative cardiac catheterization and echocardiography reports. Perioperative information was collected regarding anesthetic management of induction, including whether the anesthesiologist had completed a fellowship in cardiothoracic anesthesiology, the placement of a pre-induction arterial line, type and dose of anesthetic agents used, the pre-emptive use of vasopressors or inotropes (i.e., documented before or with the initial anesthetic agent administration), and the preservation of spontaneous ventilation during induction. Baseline mean arterial pressure (MAP) was determined using the mean intraoperative blood pressure recordings preceding induction, and post-induction MAP nadir was assessed within 10 min after induction.

The groups compared in this study were patients managed by anesthesiologists who had received subspecialty training in cardiothoracic anesthesiology (CTA group) vs those who had not (non-CTA group). The primary outcome compared between groups was the occurrence of post-induction MAP <55 mmHg. Secondary outcomes included the absolute and fractional decrease in MAP relative to baseline, the reactive use of vasopressor or inotropes (i.e., following but within 10 minutes of induction), the occurrence of any major adverse post-induction hemodynamic event, post-operative hospital length of stay, and 30-day mortality.

Statistical Analysis

Following exclusions, patients remaining in the sample population underwent propensity score matching. Variables included in the matching process included all of those listed in Table 1. With CTA and non-CTA groups defined as the response variable, inverse probability weights were estimated

Table 1. Baseline Demographic and Clinical Characteristics of the Matched Sample Population.

	Non-CTA Trained (n = 302)	CTA Trained (n = 100)	*P* Value
Demographics			
Age (years)	64 ± 14	64 ± 14	.665[b]
Female gender	198 (66%)	58 (58%)	.188[c]
Race			.324[c]
White	166 (55%)	59 (59%)	
Black	131 (43%)	38 (38%)	
Hispanic	1 (<1%)	2 (2%)	
Asian	2 (1%)	0 (0%)	
Other	2 (1%)	1 (1%)	
Body mass index (kg/m^2)	31 ± 8	31 ± 8	.563[b]
American Society of Anesthesiology class			.025[c]
II	13 (4%)	0 (0%)	
III	183 (61%)	55 (55%)	
IV	106 (35%)	45 (45%)	
Emergent surgery	14 (5%)	1 (1%)	.130[c]
Code status for surgery			.154[c]
Full code	301 (>99%)	98 (98%)	
"Do not resuscitate"	1 (<1%)	2 (2%)	
Past medical history			
Hypertension	238 (79%)	87 (87%)	.079[c]
Diabetes	111 (37%)	36 (36%)	1.000[c]
Chronic obstructive pulmonary disease	89 (30%)	28 (28%)	.801[c]
Chronic kidney disease	12 (37%)	44 (44%)	.237[c]
Coronary artery disease	97 (232%)	30 (30%)	.712[c]
Carotid disease	21 (7%)	6 (6%)	1.000[c]
Pulmonary embolism	22 (7%)	5 (5%)	.499[c]
Left heart failure	159 (53%)	50 (50%)	.729[c]
Right heart failure	16 (5%)	10 (10%)	.104[c]
Preoperative medications			
Angiotensin pathway disruptor	29 (10%)	7 (7%)	.546[c]
Beta blocker	38 (13%)	14 (14%)	.732[c]
Cardiac studies[a]			
Left ventricular ejection fraction (%)	56 ± 9	56 ± 9	.781[b]
Left ventricular diastolic function			.740[c]
Normal	94 (231%)	32 (32%)	
Grade I dysfunction	99 (33%)	38 (38%)	
Grade II dysfunction	20 (7%)	4 (4%)	
Grade III + dysfunction	1 (<1%)	0 (0%)	
Right ventricular function			.435[c]
Normal	257 (85%)	81 (81%)	
Mild dysfunction	36 (12%)	13 (13%)	
Moderate dysfunction	6 (2%)	4 (4%)	
Severe dysfunction	3 (1%)	2 (2%)	
Valvular dysfunction (at least moderate severity)			
Mitral regurgitation	26 (9%)	5 (5%)	.286[c]
Mitral stenosis	2 (1%)	1 (1%)	1.000[c]
Aortic insufficiency	9 (3%)	2 (2%)	1.000[c]
Aortic stenosis	7 (2%)	7 (7%)	.052[c]
Tricuspid regurgitation	38 (13%)	14 (14%)	.732[c]
Tricuspid stenosis	0 (0%)	0 (0%)	1.000[c]
Pulmonic insufficiency	2 (1%)	0 (0%)	1.000[c]
Pulmonic stenosis	0 (0%)	0 (0%)	1.000[c]

(continued)

Table 1. (continued)

	Non-CTA Trained (n = 302)	CTA Trained (n = 100)	*P* Value
Pulmonary artery systolic pressure (mmHg)	51 ± 18	50 ± 21	.449[b]
Right atrial pressure (mmHg)	8 ± 5	7 ± 5	.428[b]
Classification of PH			.617[c]
Group 1, pulmonary arterial hypertension	14 (5%)	4 (4%)	
Group 2, congestive left heart disease	95 (32%)	38 (38%)	
Group 3, chronic hypoxic lung disease	25 (8%)	8 (8%)	
Group 4, chronic thromboembolic	6 (2%)	0 (0%)	
Group 5, unclear/multifactorial	93 (31%)	25 (25%)	
Unknown	69 (23%)	25 (25%)	
Intraoperative details			
Type of surgery			<.001[c]
Thoracic	2 (1%)	10 (10%)	
Vascular	6 (2%)	5 (5%)	
Ear–nose–throat/oromaxillofacial	28 (9%)	11 (11%)	
Ophthalmology	4 (1%)	0 (0%)	
Gastroenterology	60 (20%)	14 (14%)	
General surgery	43 (14%)	6 (6%)	
Surgical oncology	10 (3%)	3 (3%)	
Obstetrics/gynecology	15 (5%)	0 (0%)	
Urology	26 (9%)	6 (6%)	
Orthopedic	50 (17%)	4 (4%)	
Neurosurgery	8 (3%)	1 (1%)	
Trauma	17 (6%)	7 (7%)	
Plastic	11 (4%)	2 (2%)	
Transplant	5 (2%)	4 (4%)	
Cardiology	6 (2%)	20 (20%)	
Pulmonology	11 (4%)	7 (7%)	
Pre-induction mean arterial pressure (mmHg)	99±19	97±18	.668[b]

Variables expressed as mean (±SD) or as total (%).
[a]Echocardiographic data were only available for 781 (94.2%) of patients. Pulmonary artery systolic pressure and right atrial pressure assessments were available in 678 (81.7%) of patients.
[b]Kruskal–Wallis rank sum test.
[c]Fisher's exact test.

via a logistic model and baseline demographic and clinical characteristics were adjusted. Next, 1:1 matching with replacement was performed based on the fitted propensity score. In particular, propensity scores were calculated for each patient. They were further used as sampling probabilities to sample with replacement from the two groups to get matched samples. Variables were then compared between CTA and non-CTA groups to identify statistical differences in baseline characteristics, practice patterns, and outcomes using either Fisher's exact test or Kruskal–Wallis rank sum tests, as appropriate. Results were considered statistically significant when $P < .05$. The statistical software used for this analysis was R Version 4.0 (R Foundation for Statistical Computing, Vienna, Austria)

Results

Of the initial 2726 patients identified by searching by ICD code for PH, patients were included if they were >18 years old, underwent non-cardiac surgery, and were undergoing general anesthesia with intubation, yielding 852 patients. Following the exclusion of patients sustained on mechanical circulatory support (n = 9), having had anesthesia induction prior to the surgical encounter (n = 6), or with inadequate documentation of blood pressure (n = 4) or induction medications (n = 4), 829 patients remained in the sample (589 in the non-CTA group and 240 in the CTA group). Of these initial unmatched patients (see Supplemental Tables 1-3), those in the CTA group were more likely to have higher American Society of Anesthesiology physical status ($P < .001$), used a β-blocker ($P = .038$), have lower mean left ventricular ejection fraction ($P = .011$), have a higher incidence of moderate to severe aortic stenosis ($P = .011$), underwent non-emergent surgery ($P = .030$), and underwent a different distribution of surgery types ($P < .001$).

Following propensity score matching, 402 patients remained in the final model (302 in the non-CTA group and 100

in the CTA group). Matching was performed using 34 baseline demographic and clinical variables, all listed in Table 1. These groups were different with respect to surgery type performed as a univariable ($P < .001$) but were overall statistically similar following matching with respect to these baseline characteristics. Matched samples were statistically similar with respect to all variables not included in the matching algorithm. Stated another way, following propensity score matching, there were no differences observed between CTA and non-CTA groups with respect to peri-induction practice patterns (Table 2) and primary and secondary outcome measures (Table 3).

Discussion

Surgeons frequently make requests for cardiothoracic anesthesiologists to manage patients with cardiovascular risk factors.[5] This action may be fueled by both a clinical and medicolegal attempt to mitigate risk, especially in situations where a patient's medical optimization for surgery is questioned. Whatever the incentive, a mindset shared by many that are drawn to pursue a career in cardiothoracic anesthesiology is one of intellectual satisfaction when managing more complex patients, even in the context of non-cardiac surgery. In addition to advanced certification in echocardiography, the regular performance of cardiac anesthesia likely contributes to a heightened understanding of more severe forms of cardiac disease states and hemodynamic consequences, better familiarity with the use of vasopressors, inotropes, massive blood product resuscitation, advanced mechanical support devices and complicated critical care transports, and closer professional relationships with perfusionists, cardiologists, and cardiac and thoracic surgeons that might need to be called upon to assist in dire circumstances. But the question remains, does a cardiac anesthesiologist need to be involved in patients with certain types of cardiac

Table 2. Practice Patterns of Anesthesiologists Without and With Fellowship Training in Cardiothoracic Anesthesiology (CTA); Results Shown for the Matched Population.

	Non-CTA Trained (n = 302)	CTA Trained (n = 100)	*P* Value
Pre-induction arterial line	29 (10%)	19 (19%)	.019[a]
Type of airway			.074[a]
Endotracheal tube	251 (83%)	91 (91%)	
Laryngeal mask airway	51 (17%)	9 (9%)	
Preservation of spontaneous ventilation	22 (7%)	9 (9%)	.665[a]
Pre-emptive inotrope or vasopressor use	25 (8%)	24 (24%)	<.001[a]
Primary induction agent			.806[a]
Propofol	287 (95%)	96 (96%)	
Etomidate	10 (3%)	2 (2%)	
Ketamine	1 (<1%)	0 (0%)	
Opioid/benzodiazepine	1 (<1%)	0 (1%)	
Combination	3 (1%)	2 (2%)	
Propofol dose (mg)	126 (49)	116 (54)	.064[b]

Variables expressed as mean (±SD) or as total (%).
[a]Fisher's exact test.
[b]Kruskal–Wallis rank sum test.

Table 3. Comparison of Post-Induction Hemodynamics and Other Outcomes; Results Shown for the Matched Population.

	Non-CTA Trained (n = 302)	CTA Trained (n = 100)	*P* Value
Post-induction mean arterial pressure <55 mmHg	83 (28%)	27 (22%)	1.000[a]
Post-induction decrease in mean arterial pressure (mmHg)	31 ± 18	32 ± 17	.736[b]
Post-induction % decrease in mean arterial pressure	31 ± 16	32 ± 15	.890[b]
Reactive inotrope or vasopressor use	129 (43%)	41 (41%)	.816[a]
Major post-induction hemodynamic events	2 (1%)	1 (1%)	1.000[a]
Post-operative hospital length of stay	4 ± 9	7 ± 18	.008[b]
30-day mortality	3 (1%)	1 (1%)	1.000[a]

Variables expressed as mean (±SD) or as total (%).
[a]Fisher's exact test.
[b]Kruskal–Wallis rank sum test.

disease undergoing non-cardiac surgery and how should this decision be made?

The goal of this study was to investigate this very complex question by focusing on a specific pathology and the hemodynamic consequences of anesthetic induction. At our institution, patients with PH undergoing non-cardiac surgery are identified as a high-risk population, and a request is often made for a cardiac anesthesiologist to be involved in their care. In this study, the primary aim was to identify differences in post-induction blood pressure for patients with PH managed by anesthesiologists having completed a cardiothoracic fellowship (CTA group), compared to those who have not (non-CTA group). After propensity matching of patient characteristics, no statistical difference between the groups was noted in induction technique or with the primary outcome. It is important to note however that after propensity matching our study population was limited to those with mild to moderate pulmonary hypertension and most without any RV dysfunction. This suggests that for this subpopulation of patients with milder disease, the hemodynamic consequences of anesthetic induction are similar among anesthesiologists, regardless of cardiothoracic anesthesiology fellowship training. Assuming post-induction drop in blood pressure may be used as a surrogate to represent general hemodynamic management decisions, it might be reasonable to interpret our findings as evidence that patients with mild to moderate PH without RV dysfunction might be safely managed by anesthesiologists not having completed a cardiothoracic fellowship. It is important to note, however, that although 30-day mortality was similar between groups, hospital length of stay was notably longer in the CTA group. This might have been related to more patients managed by that group to be of higher ASA class and to have undergone a greater number of cardiology and thoracic procedures, despite overall propensity score matching. It was for these reasons that post-operative outcomes are reported but considered as secondary outcomes and should be interpreted with caution. In addition, it is not possible to know whether CTAs were requested due to a history of PH and the level of procedural risk or for other patient comorbidities.

This study not only reached an unexpected conclusion about similar post-induction hypotension between groups but brings to light an interesting observation. Life expectancy and patient acuity continue to increase for those undergoing non-cardiac surgery.[7] Anesthesia training has subsequently evolved to involve more advanced hemodynamic monitoring, point of care ultrasound training, advanced regional techniques, and enhanced recovery modalities.[8] It can be argued that anesthesia training has also normalized high-risk patient populations, and thus, the average anesthesiologist, regardless of fellowship training, may practice similarly when such high-risk patients are encountered. This study specifically looked at pre-emptive vasopressor use as well as pre-emptive arterial line placement, and both the CTA group and the non-CTA group made similar choices in practice patterns and had similar incidences of post-induction hypotension. There are many factors that may explain these similarities—most notably is that with the advances in anesthesia residency training graduates are more comfortable with the care of complex patients. Another reason for these similarities is the frequent sharing of practice patterns between cardiac and non-cardiac trained anesthesiologist either through shared academics or informal preoperative consultation. Based on the above results, the authors would recommend future investigation into other high-risk cardiac patients such as low ejection fraction and aortic stenosis and see if there is a similar result when undergoing non-cardiac surgery.

For patients with left ventricular assist devices undergoing non-cardiac surgery managed by either cardiothoracic or non-cardiothoracic anesthesiologists, Brown et al[9] reported no difference in the occurrence of MAPs <55 mmHg. However, as part of this study, standards for managing these patients were established, and education was provided that was guided by the group of cardiothoracic anesthesiologists at the institution performing the study to guide the practices of the non-cardiothoracic anesthesiologists. No such quality improvement initiative was in place at the current study's institution for patients with PH, and nonetheless, the incidence of post-induction hypotension was still not statistically different. Therefore, in contrast to what Maxwell et al had optimistically proposed are anesthesiologists simply, "…interchangeable cogs in a health services machine…"?[10] This editorial by Maxwell et al was first written to describe a study that had attempted to show how the anesthesiologist involved in a case might have had an effect on patient outcomes and however was later retracted on methodological grounds.[11] Unfortunately, very little research has been done on this topic otherwise. The results of the current study are striking, though should be carefully interpreted given the associated limitations. As mentioned above, further data are needed to expand these findings to include those with severe PH and RV dysfunction. In the meantime, this analysis may help to prevent requiring a cardiac anesthesiologist for all patients with PH by default. At a minimum, we hope it prompts further research into this very complex question.

This study was primarily limited by its retrospective design, and therefore, data quality may have been affected by documentation errors. In particular, it can be difficult to accurately document the timing of anesthesia induction and of peri-induction medication administration, given that anesthesia providers are often pre-occupied with the induction procedure during these events and not always available to simultaneously chart into the electronic health record. For instances when documentation was inadequate (<1% of total cases), patients were excluded from the analysis. Although hemodynamic data auto-populated directly into the record, instances of inadequate or missing MAP measurements were also excluded. Although this also included <1% of total cases, it is possible that extreme cases of hypotension were not captured as a result of oscillometric blood pressure cuff

failure due to the hypotension itself. It was not routine for all patients to have cardiac studies performed before surgery and so it was not possible to systematically grade the severity of pulmonary hypertension and include a detailed description of cardiac risk factors for the entire population. It is also possible that many patients with pulmonary hypertension during the study period might have been missed. This could have been due to either the diagnosis not having been known or to the non-inclusion of diagnostic codes in patients' medical records, despite evidence of the diagnosis embedded within stored echocardiography reports. Another factor not included in the assessment was the comparative incidence of cases between groups that were either cancelled or not performed under general anesthesia out of concern of unacceptably elevated perioperative risk. The impact of hubris or institutional pressures might have conceivably influenced one group to have proceeded with general anesthesia for cases the other might not have. In addition, given that a relatively low number of patients included in this study had significantly reduced right or left ventricular function, the reported low incidence of poor outcomes might not be generalizable to that population.

Another limitation of this study is the fact that it is a single center study at an academic institution. There are general trends or similarities in how people practice in a single institution. "Curbside" consultation with CTAs by non-CTAs might also have led to the observed similarity in practice patterns, and such informal discussions are not retrievable from the medical record. Furthermore, academic state institutions, such as this one, often recruit individuals who are interested in higher acuity patients and thus have more experience taking care of these patients for non-cardiac surgery. A more robust sample would involve multiple institutions. Sample size is also a factor. After exclusions and propensity matching, the final patient sample was 402 patients. A larger, multi-centered analysis would likely yield a more powerful result. Nonetheless, the above study provides some guidance for further research and potentially gets us closer to answering the question of whether higher risk patients undergoing non-cardiac surgery should be cared for by cardiac trained anesthesiologists or not. The implications of this study could potentially have a significant impact on staffing and logistical models for patient care and should be explored further.

Conclusion

Following propensity score matching of baseline variables, peri-induction practice patterns and the incidence of post-induction hypotension were not statistically different for patients with mild to moderate PH without RV dysfunction managed by anesthesiologists with or without fellowship training in cardiothoracic anesthesiology. Although a more comprehensive comparison of groups of anesthesiologists should be performed in the future, this study provides early evidence to suggest that patients with mild to moderate PH and normal RV function can be safely managed by either group. Hopefully, this information can help alleviate the scheduling burden in systems wherein cardiothoracic anesthesiologists are often called upon by default to manage all patients with a history of PH. Preoperative screening of these patients should include the determination of the severity of PH and RV dysfunction, with patients with milder disease triaged to be managed by non-cardiothoracic anesthesiologists. As a result, cardiothoracic anesthesiologists can therefore be more available to manage patients with more severe PH, RV dysfunction, or other cardiac pathologies. Although efforts were made to minimize the effect of confounding variables, future prospective studies are warranted to confirm these findings and further study to look at other high-risk populations should be done.

Declaration of Conflicting Interests

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Ethical statement

Ethical approval and consent

This study was approved by the Institutional Review Board (IRB# 21-0412) at the University of North Carolina at Chapel Hill on June 7, 2021. Consent not applicable.

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Supplemental Material

Supplemental material for this article is available online.

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