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OUR GREATEST GLORY IS NOT IN NEVER FAILING, BUT IN RISING EVERY TIME WE FAIL— *CONFUCIUS*

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尊敬的读者：

感谢您正在阅读本期 CASA 协会的刊物。鉴于本刊并未设定同行评审（peer review）机制，于本刊所投及发表的学术文章可仍于今后发于 Peer review 刊物。已正式发表的文章亦可于本刊物转载。本编辑部鼓励专业同行积极投稿，为我们麻醉事业的发展努力。

主编之言

又是一年的 match 季节过去了， 莘莘学子在经过 4 年的努力奋斗后收获满满。在此祝贺 Yu-Hsin Ting，Yuli Lee，Yinqian Kang 及未有及联系到的同学，即将进入麻醉专业的学习和培训，也感谢为帮助年轻学子做出贡献的老师们。

在美国医学专业如此竞争的环境下，麻醉学科以其独特的专业需求，在市场上占有重要的席位。不仅如此，随着科学技术的进展，对麻醉专业的研究也有了更深的进展。本期发表的原创和转载 CASA 会员已发表的文章，对麻醉的临床管理均具有借鉴意义。

ASK THE EXPERT 是 CASA 组织的新的不定期讨论会，以 Zoom 做为交流方式，探讨在麻醉中的管理及新进展。讨论会邀请中美在业界中对某专题有更深入研究的专家， 围绕大家感兴趣的话题进行讨论。请大家关注微信通知，积极参与，以不同的视角扩展对某临床问题的理解。本刊也会于之后发表会议讨论的总结。

盛暑将至，编辑部成员祝大家有一个开心的夏天，也鼓励各位读者将生活的经历，学术的成果投予本刊以期与大家共享。

麻醉热点讨论

How To Deal with A Patient Who Has Taken A GLP-1 Receptor Agonist Undergoing Upper Endoscopy Procedure: Cancel or Proceed?

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Disclosures: The presenters have no financial relationships with commercial interests.

Objectives:

After the case discussion, we can learn from the following aspects:

1) Describe the pharmacodynamic mechanism of a glucagon-like peptide-1 receptor agonist (GLP-1 RA), its side effects, and its impact on gastric emptying.

2) Determine the potential aspiration risk for patients who have taken a GLP-1 RA.

3) Discuss the systematic approach including point-of-care ultrasound (PoCUS) in preoperative stratification for patients who have taken a GLP-1 RA.

Introduction:

A 55-year-old man is scheduled for esophagogastroduodenoscopy (EGD). His past medical history is significant for hypertension, hyperlipidemia, s/p coronary artery stent for coronary artery disease, chronic obstructive pulmonary disease (COPD), gastroesophageal reflux disease (GERD), diabetes mellitus, and chronic kidney disorder (CKD). He has no known drug allergies. He takes lisinopril, metoprolol, clopidogrel, asprin, albuterol PRN, insulin, metformin, and dulaglutide injection qWeek (last dose 3 days ago) at home. His weight is 80 kg, and his body mass index is 31.

Physical Examination: The patient displayed no acute distress and was pleasant during the preoperative evaluation.

Vital signs*:* BP 140/75 mmHg, HR 80, RR 14, SpO2 99%, T 36.6 F.

Key Questions:

You evaluate the patient for surgical procedure in the preoperative holding area.

1. Are there any clinical concerns for this patient undergoing EGD?

2. How does a GLP-1 RA slow gastric emptying, what is its half-life, and what effects does it have?

3. What is your anesthesia plan for the patient?

4. What else do you plan to do before you decide to provide anesthesia for the patient?

Based on a new American Society of Anesthesiologists (ASA) NPO guideline published in October 2023, the anesthesia team communicated with the gastroenterology team and expressed concerns that the patient might still have been considered a full stomach since he did not stop taking a GLP-1 RA until 3 days ago, which put him at higher risk of pulmonary aspiration during EGD. The anesthesia and gastroenterology teams both agreed that the elective EGD procedure should be postponed. When the anesthesiologist informed the patient about the aspiration risk and potential complications, the patient changed his "story"; he stated that his last dose of dulaglutide was 10 days ago and insisted on proceeding with the case. Since there was some difficulty in convincing the patient to cancel the EGD procedure, the anesthesiologist decided to perform a gastric PoCUS exam to evaluate the patient's stomach status.

5. Can you describe how to perform a gastric PoCUS exam?

6. What does solid food and clear liquid look like on ultrasound?

7. What criteria are used to determine on ultrasound that the patient has a full stomach, which increase his risk for aspiration?

The anesthesia team performed a gastric PoCUS exam, as shown in ***Figure 1****.*

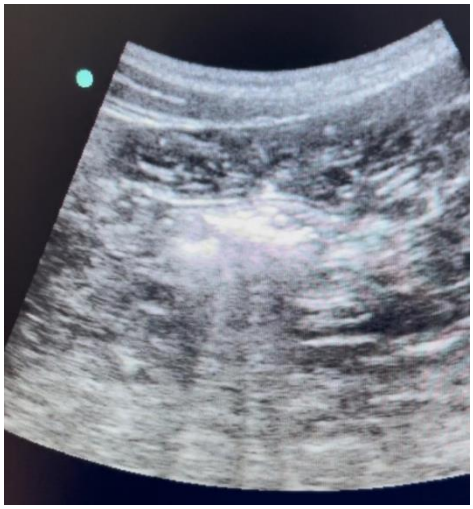

Figure 1. Gastric Ultrasound Picture with the Patient in the Right Lateral Decubitus Position

8. For elective procedures like EGD, what would you do if presented with the gastric ultrasound shown above?
9. What are the risk factors for aspiration?

The anesthesia team explained to the patient that solid food was found in his stomach, that he had a very high risk of aspiration with sedation for EGD, and that there was no guarantee of preventing aspiration with an endotracheal tube and gastrointestinal (GI) suction. The patient later admitted that he took the dulaglutide 3 days ago rather than 10 days. He "just wanted the EGD to get done" and agreed to cancel the case after his full stomach was confirmed with ultrasound.

10. According to new ASA recommendations, what is the safe time window to perform EGD for patients who have taken a GLP-1 RA and do not have an adequate elapsing date?
11. What is the new research about aspiration risk for patients on a GLP-1 RA to have sedation/monitored anesthesia care (MAC) cases? General anesthesia cases?

The use of GLP-1 RAs is becoming more popular in the treatment of type 2 diabetes and obesity (**Table 1**). These patients are increasingly likely to be taking GLP-1 RAs during perioperative periods. Multiple organ systems contain GLP-1 receptors, including the brain, heart, gastrointestinal tract, and pancreas (**Figure 2**). Stimulating this receptor leads to losing weight, better glycemic management in diabetic patients, and improved cardiac and renal functions.[1]

Some side effects related to GLP-1 RAs include nausea, vomiting, and diarrhea. These symptoms may subside if patients continue to take the GLP-1 RA. There have also been reports of gallbladder and biliary disease, including cholecystitis. In case reports, angioedema and anaphylaxis are also described.[1]

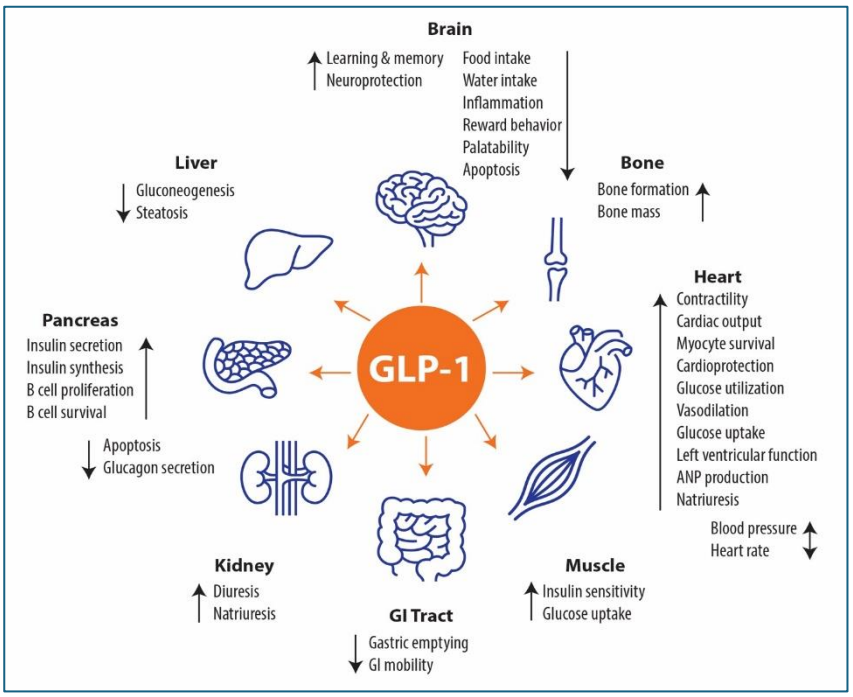

Figure 2. An Overview of GLP-1's Metabolic Effects Metabolism is affected in both direct and indirect ways by GLP-1.

Daily Dosing	**Half-Life**	**Special Considerations**
Exanetide (Byetta®, Bydureon®)	3 h	Associated with immune-mediated thrombocytopenia
Lixisenatide (Adlyxin®)	3 h	No longer available in the United States
Liraglutide (Saxenda®, Victoza®)	12.5 h	Approved for weight loss
Weekly Dosing		
Semaglutide (Wegovy®, Ozempic®)	7 d	Approved for weight loss
Dulaglutide (Trulicity®)	4.5 d	Approved for DM-2 and weight loss
Tirzepatide (Mounjaro®)	5 d	Approved for weight loss

Table 1. Common GLP-1 Agonists
Abbreviation: DM-2, type 2 diabetes mellitus.

GLP-1 RAs may pose anesthetic risks. GLP-RAs can slow gastric emptying and colon transit time. that It is still possible for patients on a GLP-1 RA to aspirate liquid or solid gastric contents under anesthesia, even when they have fasted for a conventional time per the ASA practice guidelines.[5]

Pulmonary aspiration under anesthesia is a rare event. However, the consequences can be devastating. In addition, the ASA reports that aspiration is one of the top 3 adverse events associated with airway management. Passive or active regurgitation of gastric contents is a leading cause of aspiration. Therefore, it is very important to recognize patients with an elevated risk of increased gastric volume.[1]

Patients undergoing anesthesia with unprotected airways are at risk of pulmonary aspiration, especially those with higher risk factors (**Table 2**). There are case reports as well as retrospective studies showing that patients on GLP-1 RAs undergoing endoscopy have an increased volume of gastric contents.[1] Wu et al found that even with the recommended 8-hour fasting by ASA guidelines, GLP-1 RA patients have a higher residual gastric content and a potentially higher risk of pulmonary aspiration.[6]

Compared with control groups, GLP-1 RA patients had an odds ratio of 5.8 (95% CI, 1.7 to 19.3) for residual gastric contents. In addition, one patient on a GLP-1 RA was started on MAC but had to be converted to general anesthesia due to large residual gastric contents discovered during the endoscopy, and another case witnessed pulmonary aspiration.[6]

In March 2024, Yeo et al. published a retrospective cohort study of 70 047 074 adults and 963 184 patients with 916 249 (95.1%) nonusers and 46 935 (4.9%) users of GLP-1 RAs.[5] There was a higher incidence of aspiration pneumonia among GLP-1RA users (0.83% vs 0.63%). Aspiration pneumonia was significantly more common in

users (hazard ratio [HR], 1.33; 95% CI, 1.02-1.74; $P = 0.036$) than in nonusers. It is important to pay particular attention to upper endoscopies without protected airways, propofol sedation, and combined upper and lower endoscopies.[5]

Dixit et al. published a retrospective cohort study in April 2024 which found that the use of GLP-1 RAs before surgery did not result in respiratory complications following surgery. However, only 2.8% of the procedures involved upper endoscopies, while the rest were emergent cases requiring general anesthesia.

Esophageal-related problems	Achalasia
	Previous esophagectomy (eg, Ivor Lewis)
	Tracheoesophageal fistula
Intra-abdominal Obstruction	Gastric outlet
	Small bowel
	Colonic
Gastroparesis	Longstanding diabetes
	Neuromuscular disorders
	Medication (eg, GLP-1 RA)
High risk for ileus/bowel dismobility	Acute pancreatitis
	Recent intra-abdominal surgery
	Inpatient receiving opioids/prolong bedrest
Emergency case	
Case with prolonged duration or complexity	
Pregnancy	
Active GI Bleed	

Table 2. Risk Factors for Aspiration[1]
Abbreviation: GI, gastrointestinal; GLP-1 RA, glucagon-like peptide-1 receptor agonist.

Right lat CSA	Age(y)						
	20	30	40	50	60	70	80
2	**31**	**18**	**5**	**0**	**0**	**0**	**0**
3	**45**	**32**	**20**	**7**	**0**	**0**	**0**
4	**60**	**47**	**34**	**21**	**9**	**0**	**0**
5	**74**	**62**	**49**	**36**	**23**	**10**	**0**
6	**89**	**76**	**63**	**51**	**38**	**25**	**12**
7	103	**91**	**78**	**65**	**52**	**40**	**27**
8	118	105	**93**	**80**	**67**	**54**	**41**
9	133	120	107	**94**	**82**	**69**	**56**
10	147	135	122	109	**96**	**83**	**71**
11	162	149	136	123	111	**98**	**85**
12	177	164	151	138	125	113	100
13	191	178	165	153	140	127	114
14	206	193	180	167	155	142	129
15	220	207	194	182	169	156	143
16	235	222	209	200	184	171	158
17	249	236	224	211	198	185	173
18	164	251	239	226	213	200	187
19	278	266	253	240	227	214	202
20	293	281	268	255	242	229	217
21	307	295	282	269	256	244	231
22	323	310	297	284	271	259	246
23	337	324	311	298	285	273	260
24	352	339	326	313	301	288	275
25	366	353	340	327	315	302	289
26	381	368	355	343	330	317	304
27	395	382	369	357	344	331	318
28	410	397	385	372	359	346	333
29	424	411	398	386	373	360	347
30	439	427	414	401	388	375	363

Table 3. Gastric Volume and the Cross-Sectional Area of the Antrum (CSA) are Linearly Related[8]

GRADE	ANTRAL PRESENTATION	VOLUME IMPLICATIONS	ASPIRATION RISK
0	Empty in both supine and RLD position	Minimal	Low risk
1	Empty in supine, clear fluid visible in the RLD	⩽ 1.5 mL/kg, compatible with baseline gastric secretions	Low risk
2	Clear fluid visible in both positions	> 1.5 mL/kg, likely in excess of baseline gastric secretions	High risk

Table 4. Antral Grading System[8]. Abbreviation: RLD, right lateral decubitus (position).

The ASA has released updated, consensus-based guidance on preoperative management of GLP-1 RAs. For elective procedures, 1 day prior to the procedure, daily GLP-1 RAs should be withheld, and weekly dosed GLP-1 RAs should be withheld for at least 1 week. Furthermore, patients should be asked about their GI symptoms on the day of their procedure, including nausea, vomiting, abdominal pain, and abdominal distention. It is strongly recommended that elective procedures be postponed among symptomatic patients. Perioperatively, if a patient does not have the aforementioned GI symptoms and GLP-Ras are held in accordance with recommendations, the procedure can be performed. Patient without GI symptoms but not holding the medication as advised should proceed with the procedure with "full stomach" precautions. In addition, there is no evidence that patients taking GLP-1 RAs should fast for a specific amount of time. Using ultrasound to determine gastric volume is also recommended (**Figure 3**). The algorithm for performing a gastric ultrasound is shown in Figure 4.[1] Gastric ultrasound can help with decision-making (**Table 3 and Table 4**).

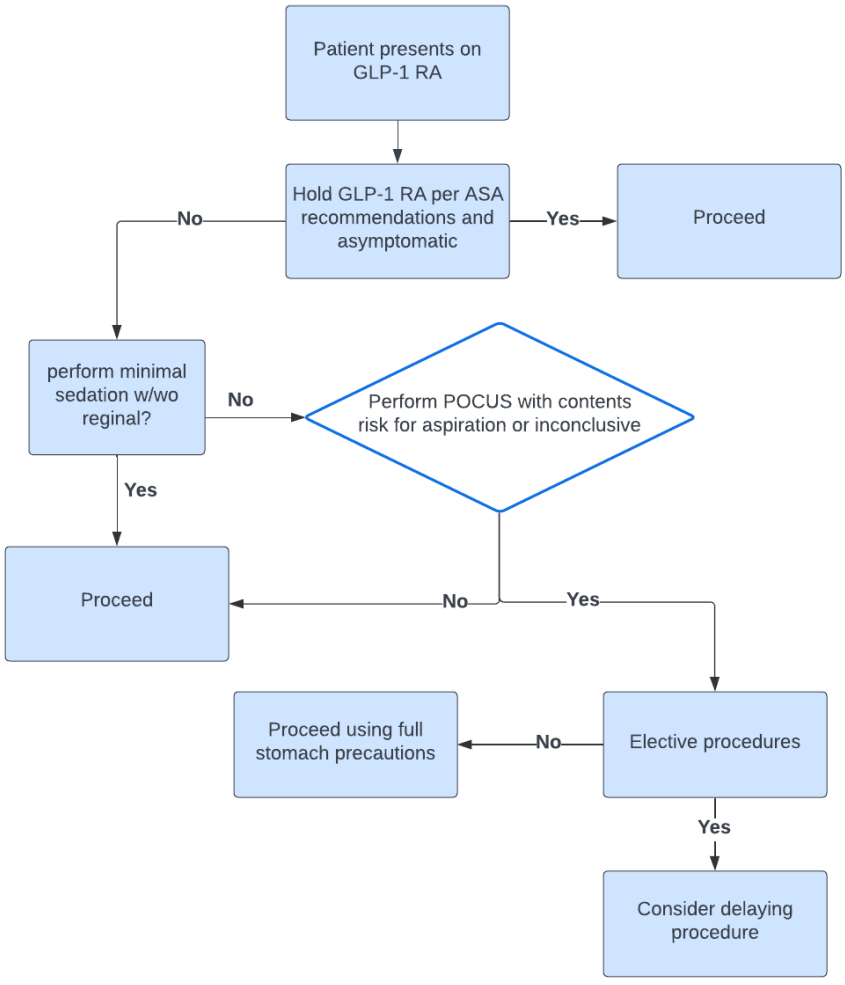

Figure 3. Algorithm for Patients Presenting for Elective Surgery While Taking a GLP-1 RA

In patients who have taken GLP-1 RAs, gastric ultrasound is recommended to evaluate the gastric contents before anesthesia.[1] According to Sherwin et al's prospective observational study, residual gastric solids were detected with gastric ultrasound. Aspiration risk during anesthesia care may be impacted by GLP-1 RAs following an overnight fast and 2 hours after clear liquids, which may have implications for gastric emptying and residual gastric contents.[9]

A rapid sequence induction of general anesthesia and decompression of the gastric contents before emergence could be considered in the event of unknown gastric contents. Solid foods are not easily decompressed by gastric suction, so there is a risk of aspiration during emergence if there is residual solid content in the stomach.[1]

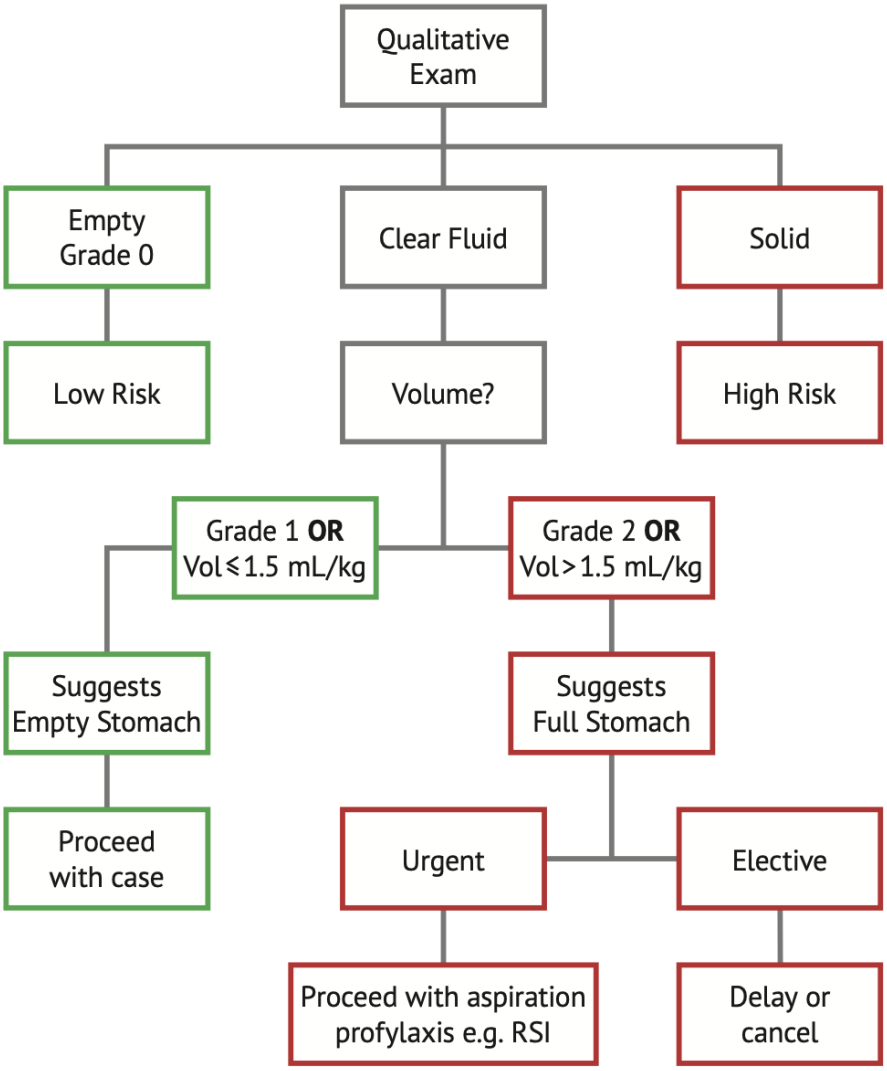

Figure 4. The Algorithm for Performing PoCUS Gastric Ultrasound

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转载文章

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One Size Does Not Fit All: Perioperative Management of Patients with Heart Failure with Preserved Ejection Fraction

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Running title: Perioperative management of HFpEF

Abstract

Heart failure with preserved ejection fraction (HFpEF) is recognized as an important risk factor for perioperative complications. However, anesthesia management of HFpEF patients remains a considerable challenge without clear guidance. HFpEF is heterogeneous in its pathophysiological processes, diverse clinical presentations, adverse remodeling of cardiovascular and other organs, and clinical outcomes. It is difficult to manage the disease with one fixed approach because of this. This review phenotypes HFpEF patients by combining their clinical features and anesthesia care issues. Five phenotypes of HFpEF patients are identified: A, O, P, C, and Y. The clinical features, anesthesia implications, and anesthesia management for each phenotype are highlighted and discussed. Such an approach to HFpEF patients in the operating room could deliver safe, high-quality perioperative care.

Introduction

Heart failure with preserved ejection fraction (HFpEF) is increasingly recognized as a crucial risk factor for perioperative mortality and morbidity in cardiac and non-cardiac surgeries [1-3]. HFpEF is becoming the dominant form of heart failure (HF),[4] yet there is no clear guidance to manage these patients during the perioperative period. HFpEF is associated with comorbidities such as hypertension, obesity, diabetes, pulmonary hypertension, cardiac arrhythmias, systematic inflammation, increased stiffness in tissues, and impaired oxygen utilization [3,4]. Anesthesiologists face great challenges when delivering anesthesia care to HFpEF patients [3].

The pathophysiological processes leading to HFpEF are even more complex and complicated, involving numerous cellular and molecular signaling pathways [5]. Thus, the multifaceted clinical presentation, poorly understood underlying mechanisms, and lack of effective treatment render it challenging to mitigate HFpEF perioperative risks. This review covers the phenotyping of HFpEF patients and the implications for anesthesia management.

HFpEF diagnosis and treatment

HFpEF can be readily suspected with signs and symptoms of HF (**Table 1**) and a normal EF (i.e. ≥50%). However, these classic clinical signs and symptoms of HF are non-specific, non-obvious, and sometimes can only be elicited by stress testing. Therefore, HFpEF diagnosis usually requires objective evidence of abnormalities in cardiac structure and function related to HF (c.f. imaging studies, i.e., echocardiography), plasma BNP (>35 pg/ml), and NT-proBNP (>125 pg/ml) elevation.[6] Recently, a score-based approach, which incorporated the six strongest independent predictors of HFpEF (hypertension, obesity, atrial fibrillation (a-fib), pulmonary hypertension, elderly, filling pressure—H_2FpEF) was proposed (**Table 2**) [7].

Table 1
Symptoms and signs of heart failure (HF).

Symptoms	Signs
Shortness of breath (dyspnea)	Jugular vein distension
Orthopnea	Low extremity edema
Persistent coughing or wheezing	Pulmonary edema
Tiredness, fatigue	Hepatojugular reflex
Lack of appetite	Cardiomegaly
Confusion, impaired thinking	S3 gallop
Tachycardia	Pleural effusion
Weight loss	Decreased vital capacity

Table 2
Criterion used to diagnose HFpEF: H_2FpEF scoring system. [7].

Label	Clinical Variable	Parameters	Score
H_2	**H**eavy	BMI >30 kg/m^2	**2**
	Hypertension	2 or more antihypertensive medicine	**1**
F	Atrial **F**ibrillation	Proximal or persistent	**3**
P	**P**ulmonary hypertension	PASP >35 mmHg (echocardiography)	**1**
E	**E**lder	Age > 60 years old	**1**
F	**F**illing pressure	E/e' > 9 (Doppler echocardiography)	**1**

BMI body mass index, **PASP** pulmonary arterial systolic pressure, **E/*e'*** ratio between early mitral inflow velocity and mitral annular early diastolic velocity.

This scoring system evaluates a symptomatic patient suspected of HFpEF for these six predictors (maximum score is 9). A score of 0-1 suggests low probability, 2-5 intermediate probability, and > 6 high probability. Patients with scores of 2-5 require additional exercise/stress testing for diagnosis.

The European Society of Cardiology developed a similar scoring system [8]. This system emphasizes cardiac structural (chamber size/volume) (Domain 1) and functional (diastolic) (Domain 2) abnormalities, levels of plasma BNP and NT-proBNP in patients with sinus rhythm (Domain 3), and a-fib (Domain 4). Each domain has a score of 2, for a maximum score of 8. A score of 0-1 indicates no disease, 2-4 intermediate probability, and >5 high probability.

Both systems emphasize the importance of additional stress exercise testing in intermediate-risk patients. The H_2FPEF system seems more practical and easier to use by the general practitioner, as it does not require echocardiography expertise. However, different scores assignment to each factor would increase probability intervals for diagnosis. In addition, echocardiographic diagnosis of PASP and E/e' ratio is not always reliable. Therefore, the H2EPEF scoring system can be improved in order to make the diagnosis of HFpEF easier and more reliable. Thus, a firm diagnosis of HFpEF requires the presence of HF signs and symptoms *and* expert echocardiography examination and, sometimes, invasive testing, especially in patients with intermediate scores.

In general, HFpEF patients are managed and treated according to their HF stages as described in the guidelines recently published [9]. Majority of the treatments involves pharmacological therapies. At early stages, management includes risk factor reduction, patient and family education, and treatment of underlying diseases such as hypertension, diabetes mellitus, coronary artery disease, and dyslipidemia. Angiotensin receptor-neprilysin inhibitor (ARNi), angiotensin converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB), and sodium-glucose cotransporter-2 inhibitor (SGLT2i) are recommended drugs. Patients with more advanced stages are treated with diuretics as first line therapy, supplemented by SGLT2i and possibly other drugs mentioned above. However, unlike heart failure management for reduced ejection fraction (HFrEF), there is no established guide to drug therapy for HFpEF patients due to lack of clinical benefits. Nevertheless, SGLT2i has recently been shown to reduce HF hospitalization or cardiovascular death but not all-cause mortality [10].

Treatment approaches adopted from HFrEF have been largely unsuccessful in HFpEF due to substantial heterogeneity in its clinical presentation and etiologies [4]. Researchers have thus proposed phenotype-specific management strategies [11]. Treatment plans are derived from either a matrix of clinical presentation phenotypes (i.e., presentations showing organ-specific involvement /characteristics) or predisposing phenotypes (i.e., etiological factors). The success of phenotype-based management of HFpEF has yet to be seen due to a lack of consensus on criteria for profiling these patients. Nevertheless, this phenotype framework provides an impetus to design HFpEF patient perioperative care out of the traditional box.

Clinically subgrouping HFpEF patients and outcomes

As discussed above, HFpEF diagnosis combines clinical signs and symptoms with documentation of diastolic dysfunction of the heart (structural or functional) [7,8]. Yet, it has been difficult for clinicians to readily determine the degree to which pathological processes are present in each HFpEF patient because of varied pathological processes (beyond coronary artery disease alone) with varied cardiac structure and function alterations. Therefore, attempts have been made to phenotype HFpEF patients according to their clinical profiles/features, examination and laboratory results, disease progress, and therapy response [12-14]. These studies showed group differences in outcomes.

Kao et al. identified six sub-groups of HFpEF patients from the Irbesartan in Heart Failure with Preserved Ejection Fraction Study (I-PRESERVE) trial database (4,113 HFpEF patients) [12]. Identification was based on 11 clinical features (age, gender, BMI, a-fib, coronary artery disease (CAD), diabetic mellitus (DM), hyperlipidemia, valvular disease, alcohol use, estimated glomerular filtration rate (eGFR), and hematocrit). Two subgroups of HFpEF patients had the worst primary (all causes of mortality or hospitalization due to cardiovascular reasons) and secondary (HF hospitalization or death from HF or sudden death)

outcomes, with a mean follow-up of 49.5 months. One group was older (>70 yr) and morbid obese (BMI > 30). These patients also had, CAD, hyperlipidemia, diabetes mellitus, renal failure, and hematocrit <40 %. The other group was older (>80 yr), with atrial fibrillation, renal failure, hematocrit <40 %, and higher NT-proBNP (950 lg/ml).

Based on the clinical features, biomarkers, and echocardiography, Cohen et al. identified three HFpEF phenogroups in 3,445 patients enrolled in a multicenter internal trial: Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist Trial (TOPCAT) [14]. These three phenogroups included: Phenogroup 1 (mean age 61 years, smoking 24%, diabetes 9%), Phenogroup 2 (mean age 77 years, women 56%, a-fib 49%, eGFR 58 ml/min/1.73 m^2), and Phenogroup 3 (mean age 66 years, obesity 98%, diabetes mellitus 88%, chronic renal insufficiency 57%, depression 36%, black patients 21%, NYHA Class III or IV 54%). Phenogroup 1 had the lowest NT-proBNP levels, least LV hypertrophy with the largest left ventricular (LV) cavities, lowest left atrial (LA) volumes and mitral E/e′ ratios, and the lowest arterial stiffness; Phenogroup 2 had the highest levels of biomarkers for innate immune response, the smallest LV cavities with the lowest LV mass, largest LA, lowest mitral annular a′ and s′ velocities, and highest arterial stiffness; Phenogroup 3 had the highest levels of tumor necrosis factor-mediated inflammatory markers, the highest LV wall thickness and LV mass, highest E/*e*′ ratios, and compliant arteries. Importantly, Phenogroups 2 and 3 had a significantly higher risk for primary endpoint (composite of cardiovascular death, HF hospitalization, or aborted cardiac arrest), all-cause mortality, and HF hospitalizations than Phenogroup 1. Interestingly, spironolactone therapy significantly increased the probability of survival for patients in Phenogroup 3.

Clearly, phenotyping HFpEF patients provides insight into factors underlying their differences in disease presentation, progression, and prognosis. More importantly, phenotyping helps to identify subgroups of HFpEF patients who may respond to specific therapies.

Table 3
Common medicines taken by HFpEF patients and perioperative management.

Drugs	Action	Recommendation	Others
β-blockers	↓myocardial ischemia ↓arrhythmias, ↓BP	Continue throughout peri-operative period	Avoid acute withdraw
α2-agonists	↓BP	Continue throughout peri-operative period	For clonidine: oral can be replaced by transdermal 3 days before surgery
Ca^{2+} channel blockers	↓BP, ↓arrhythmias, ↓myocardial ischemia ↑relaxation/dia. function	Continue throughout peri-operative period	Oral can be replaced by IV
ACEi/ARBs /ANRis	↓BP, ↑survival(?) Renal benefit	Well-controlled BP: hold 24 h. before surgery (long-acting drugs) Poorly-controlled BP: Continue	Restart <48 h. post-operatively. If continuing preoperatively, be aware of intraoperative ↓BP and postoperative ↑BP
Diuretics	↓BP, ↓blood volume	BP and well-controlled blood volume: hold in the morning of surgery Poorly controlled volume: continue	Watch hypokalemia Give IV diuretic if holding causes volume overload during perioperative period
Digoxin	↓hospitalizations, ↓a-fib	Continue	Usually no need to check levels
Statins	↓LDL ↓CV risks overall ↓inflammation, ↓A-fib Stabilizing plaques	Continue	Start as early as possible in untreated patients during perioperative period
SGLT2i	↓LDL, ↓HF mortality(?) metabolic benefits	Discontinue for 3 days (4 days for ertugliflozin)	Monitoring blood glucose during discontinuation Restart when oral intake possible or risk of ketoacidosis reduced
GLP-1 agonist	↑insulin sensitivity ↓blood glucose, ↓body weight ↓cardiovascular risks ↑relaxation/diastolic function	If taken daily, hold on day of surgery If taken weekly, hold a week before surgery	If not held, and no GI symptoms, proceed with full stomach precaution If GI symptoms present, delay elective surgery

BP blood pressure, **ACEi** angiotensin converting enzyme inhibitor, **ARBs** angiotensin receptor blockers, **a-fib** atrial fibrillation, **CV** cardiovascular, **LDL** low density lipoprotein, **SGLT2i** sodium-glucose cotransporter 2 inhibitor, **GLP-1** glucagon-like peptide-1, **GI** gastrointestinal.

Perioperative management

Perioperative risks for HFpEF patients

Despite normal EF (i.e., normal cardiac output), perioperative risks are no different from those of HFrEF, revealed by retrospective studies with large sample sizes from non-cardiac [2,15] and cardiac surgical patients [1]. For example, a retrospective analysis of ~300,000 HF patients in 19 moderate-to-high non-cardiac surgeries showed that 40.8% and 54.5% of HFpEF patients developed cardiopulmonary and non-cardiopulmonary complications after surgery, respectively, with a ~5% in-hospital mortality [2]. In acute hip fracture surgeries, HFpEF patients had an adjusted odds ratio (aOR) of 1.69 for major adverse cardiac and cerebral

events (MACCE), 1.71 for respiratory failure, and 1.52 for renal failure [15].

In HFpEF patients, causes of death are primarily cardiovascular (~60-70%). Sudden cardiac death and HF account for ~45-55%% and vascular 5-15%. Non-cardiac death is related to renal failure, respiratory failure, infection/sepsis, and malignancy (20-30% of all deaths) [16]. The high prevalence of cardiovascular-related deaths in HFpEF patients renders these patients more susceptible to cardiovascular complications during the perioperative period.

Perioperative medications for HFpEF patients

Because of the complex and heterogenous pathophysiological mechanisms involved in HFpEF, these patients are commonly on multiple "housekeeping" medicines for symptoms relieve and control of baseline comorbidities [17]. **Table 3** shows the medications that HFpEF patients likely take when they come for surgeries/procedures and their perioperative management. Whether patients should continue or hold these medicines is still under investigation. But the general consensus is that most of these medicines can be continued, with a number of caveats. For ACEi/ARBi/ANRi, continuation before surgery is not associated with adverse outcomes [18-20], but associated with intraoperative hypotension. Therefore, caution should be exercised to monitor BP closely and treat hypotension promptly. For SGLT2i, which has been shown to decrease hospitalizations and mortality in HFpEF patients [10], holding (for at least 3-4 days) before surgery is recommended by the FDA [21,22], due to the development of euglycemic ketoacidosis (EGKA) [23]. The mechanism of SGLT2i-induced EGKA is depicted in **Figure 1**. Risk factors for development of EGKA for patients taking SGLT2i include acute illness (infection, myocardial infarction, pulmonary embolism), malignancy, dietary changes, cocaine abuse, pregnancy, and preoperative/postoperative fasting [24]. Common features of EGKA are metabolic acidosis with an anion gap, ketonemia and ketonuria, and blood glucose <200-300 mg/dl. Glucagon-like peptide-1 (GLP-1) receptor agonists have been shown to reduce mortality and HF hospitalizations in diabetic patients [25]. However, there is no guidance regarding their use nor known benefits in HFpEF patients. Nevertheless, some HFpEF patients may have started on GLP-1 agonists given the positive effects on mitigating diabetes and cardiovascular risks in these patients [26]. One risk of taking GLP-1 agonists is accumulation of gastric content as a result of delayed gastric emptying [27]. In this regard, ASA has issued a guideline on holding and management of patients who are on the drug [28]. Depending on the dosing frequency, GLP-1 inhibitor can be held on the day (daily dosing) or the week (weekly dosing) before surgery. If the drug is not stopped and the patient has no gastrointestinal (GI) symptoms, anesthesia/surgery should proceed with "full stomach" precautions. If patient has GI symptoms, one should consider delaying elective procedures

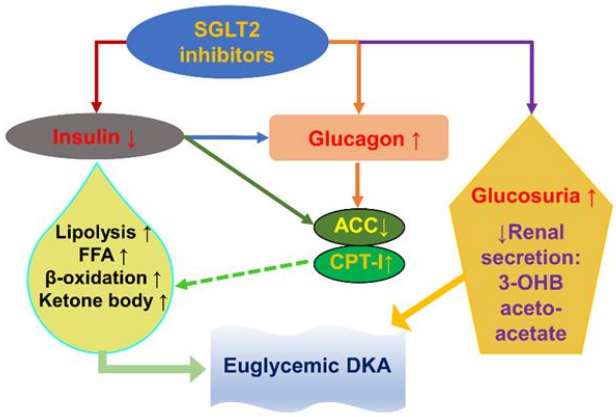

Figure 1. Mechanism of SGLT2i-induced euglycemic ketoacidosis. SGLT2i causes glucosuria, decreases insulin secretion, and promotes glycogen secretion. As a result, lipolysis increases and levels of FFA increase. Augmentation of FFA β-oxidation and accumulation ketone bodies occur. **SGLT2i** sodium-glucose co-transporter 2 inhibitor, **FFA** free fatty acid, **ACC** acetyl-CoA carboxylase, **CPT-I** carnitine palmitoyltransferase–I, **3-OHB** hydroxybutyrate, **DKA** diabetic ketoacidosis.

regardless of whether the drug is held or not. In emergency, the potential risks of regurgitation and pulmonary aspiration should be discussed with the surgeon, the patient, and family.

Phenotyping HFpEF Patients Coming to the Operating Room

Given the multifactorial and diverse nature of clinical presentations, response to therapies, and prognosis, HFpEF patients will behave heterogeneously when they come for surgeries and procedures and present challenges to perioperative management. Thus, it seems necessary for anesthesiologists to address the diversity of HFpEF to deliver better perioperative care to these patients.

Figure 2 illustrates the proposed phenotypes for HFpEF patients pertinent to perioperative anesthesia management based on considerations of HFpEF clinical features and implications to anesthesia care. There are five phenotypes of HFpEF patients: (1) elderly patients with HFpEF, or *phenotype **A***; (2) obese patients with HFpEF, or phenotype **O**; (3) HFpEF patients with pulmonary hypertension, or *phenotype **P***; (4) HFpEF with cardiac ischemia and dysfunction (cardiomyopathy), or *phenotype **C***; (5) younger patients with HFpEF, or *phenotype **Y***. The following will discuss the clinical features of each phenotype and their anesthesia implications. More importantly, we will highlight the issues in anesthesia management associated with each phenotype.

Phenotype A

Phenotype A patient characteristics

Those patients are older with age over 75-80 years old and above. In addition to significantly advanced age, elderly

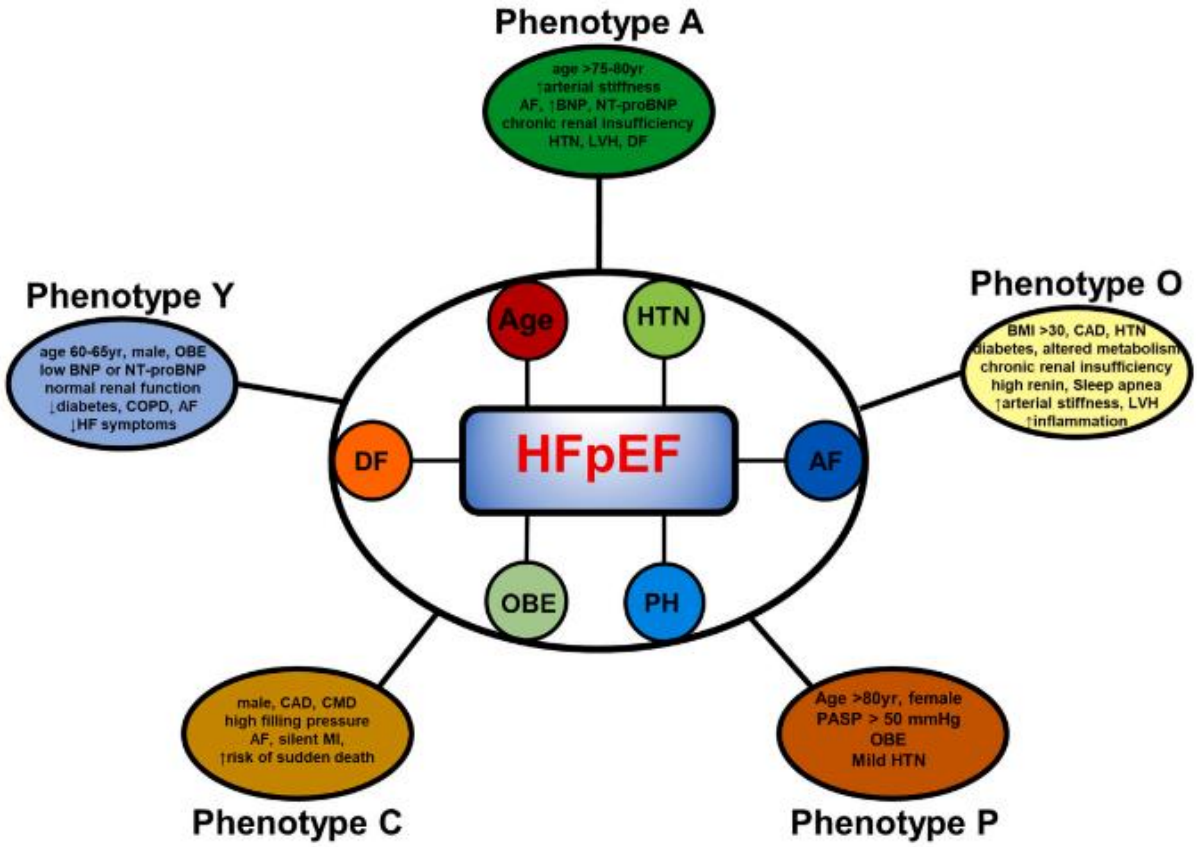

Figure 2. Schematic for phenotype classification. The core clinical features of HFpEF are presented within the circle. Five phenotypes with key clinical features are illustrated (see text for details). **HFpEF** heart failure with preserved ejection fraction, **HTN** hypertension, **AF** atrial fibrillation, **DF** diastolic dysfunction, **PH** pulmonary hypertension, **OBE** obesity, **LVH** left ventricular hypertrophy, **CAD** coronary arterial disease, **CMD** coronary microvascular disfunction, **MI** myocardial infarction, **PASP** pulmonary arterial systolic pressure.

patients have increased cardiovascular stiffness (both arteries and heart) with widened pulse pressure [29,30], which contributes significantly to the development of HFpEF. They also have concentric LV hypertrophy with diastolic dysfunction, severely enlarged LA accompanied by a-fib, significantly elevated plasma BNP and NT-proBNP levels, and a higher incidence of chronic kidney insufficiency [12,14]. Anemia is not uncommon in these patients.

Phenotype A anesthesia implications

Age >80 years is a risk factor for complications in moderate-to-high-risk surgeries. A meta-analysis showed that the 30-day postoperative mortality and length of hospital stay more than doubled in patients aged 80 and over after pancreaticoduodenectomy[31]. There was also a 50% increase in postoperative complication rate. However, low-risk laparoscopic procedures (including laparoscopic hepatectomy and gastrectomy) were as safe as in younger patients [32,33].

Frailty, which increases with ageing [34], is another risk factor for perioperative adverse outcomes [35]. Postoperative mortality is significantly increased in patients with frailty, especially the very frail, and can be as high as 43% at 180 days postop [35]. Thus, preoperative frailty assessment is paramount for early intervention and better outcomes. It should mention that frail patients often have cardiac dysfunction, especially diastolic dysfunction (both LV and RV).

Increased cardiovascular stiffness in elderly HFpEF patients has important implications [36]. Systolic pressure, especially in the mid-to-late phase, is elevated, accompanied by widened pulse pressure, due to increased arterial stiffness [36-38], leading to augmentation of systolic pressure and reduction of early diastolic pressure thus coronary perfusion time. Another pathophysiologic effect of increased pulse pressure is increased pulse energy, which can damage the microvasculature of flow-rich organs (such as the kidney and brain [36]).

Cardiac remodeling also occurs in elderly HFpEF patients, manifesting as hypertrophy, diastolic dysfunction, and increased LA size. These patients are prone to cardiac ischemia and arrhythmias, which can lead to heart failure. Renal insufficiency will lead to renal or other organ failures (heart, respiratory, coagulation).

Phenotype A anesthesia management

Preoperative comprehensive assessment of these patients should include systematic evaluation of comorbidities, functional status, cardiopulmonary function, neurocognitive function, sensory impairment, frailty, nutrition, and medication [39]. Such evaluation would aim to understand the functional status of vital organs, establish frailty status, and know baseline values of blood chemistry and coagulation. If identified, anemia should be treated before surgery. Intraoperative management includes mindful use of medicines (anxiolytics, narcotics, and other drugs) and invasive monitoring for moderate-to-high-risk procedures (arterial line, central venous pressure). These patients have increased arterial stiffness and, therefore, a smaller window for blood pressure management to optimize (diastolic) coronary perfusion and minimize afterload. Minimizing pulse pressure also protects the kidney and brain from injury.

The best strategy would be an aggressive goal-directed blood pressure management plan, which includes (1) goals for systolic and diastolic pressure based on baseline values; (2) using fluids, vasopressors and dilators to maintain pulse pressures at set levels; (3) appropriate depths of anesthesia (EEG or BIS monitoring preferred) and pain control.

Adverse cardiac remodeling renders these patients prone to cardiac ischemia and arrhythmias, decreasing cardiac output. Thus, tachycardia and hypoxia should be avoided at all costs. A-fib should be treated promptly, including immediate cardioversion if necessary. Postoperative management is no different than for other geriatric patients [39]. But monitoring for cardiac, pulmonary and renal function should be extended during the postoperative period. Frailty monitoring and management should be part of postoperative care.

Phenotype O

Phenotype O patient characteristics

These patients are obese (BMI >30) and can be morbidly obese (BMI >40) [40]. Obesity is usually central and more prevalent in American African women. Diabetes and altered metabolism (glucose, lipids, etc.) are often present due to liver dysfunction. Decreased renal function and high renin levels are common in these patients. Sleep apnea is a prominent feature often associated with PH or RV dysfunction and COPD. Inflammation is another feature, with increased arterial stiffness promoting cardiovascular dysfunction. As in other HFpEF patients, hypertension, CAD, and hypertrophy are present.

Significant cardiovascular remodeling occurs in this group.[40] Blood volume increases, leading to increased cardiac loading and remodeling (chamber dilation and hypertrophy). Increased filling pressure is another crucial feature, positively related to body mass and blood volume in obese patients with HFpEF. Heart volume (chamber volume, wall thickness, and pericardial fat) increases pericardial restraint and promotes interventricular independence. This altered interventricular interaction includes distortion (flattening) of the septum, higher right atrial pressure/pulmonary capillary wedge pressure (PCWP) ratio (i.e., equalization), and changed LV PCWP and pre-loading coupling (i.e., higher PCWP at similar transmural LV filling pressure), promoting LA-LV uncoupling.

Phenotype O anesthesia implications

Obstructive sleep apnea (OSA) significantly impacts the cardiovascular system [41], which is already remodeled in HFpEF. In fact, OSA is a risk factor for HFpEF due to its high prevalence with obesity. Airway obstruction can decrease intrathoracic pressure and increase LV transmural pressure, promoting increased afterload. Simultaneously, the drop in intrathoracic pressure increases venous return and RV filling, shifting the septum and reducing LV filling and stroke volume [42]. Increased heart volume and pericardial strain magnify the septum shift. These patients tend to have tachycardia and elevated pressure due to sympathetic stimulation by hypoxia. Thus, they are at risk for cardiac ischemia and dysfunction.

Chronic intermittent hypoxia in OSA can cause airway inflammation, triggering or exacerbating asthma, limiting airflow [43] and promoting lung injury [44]. OSA increases the risk of COPD exacerbations and death (i.e., so-called “overlap syndrome” [45]).

Phenotype O anesthesia management

OSA severity should be thoroughly evaluated to determine OSA-related risks per published guidelines [46]. Preoperative treatment of severe OSA is desirable but not mandatory. Difficult airway is always suspected in these patients. Endotracheal intubation is preferable to deep sedation without a secured airway, given the potential ventilation obstruction and subsequent hemodynamic consequences (increased afterload, septal shift towards left impairing LV filling and cardiac output, cardiac arrhythmias). Heart failure is always expected since additional factors contribute to increased LV filling pressure in these patients (i.e., volume dependence, pericardial restraint, ventricular interaction, RV afterload). Therefore, invasive monitoring for volume and cardiac function/hemodynamics should be considered. Increased volume status and filling pressure respond better to diuretics. Also, prompt inotropic circulatory support should begin without delay, especially in moderate-to-high-risk surgeries. Because of RV-PA uncoupling, using pulmonary vasodilators is highly recommended. In intubated patients, a lung protection strategy is desirable to prevent

further damage to lung tissue (including endothelium).

During the postoperative period, we should expect heart failure exacerbations based on clinical manifestations, not on plasma levels of NT-proBNP. Oxygen supplementation should continue to avoid hypoxia. CPAP should begin if indicated. Pain management, sedation, and reversal are optimized to prevent hypoventilation. Continuous monitoring is desirable during the postoperative period.

Phenotype P

Phenotype P patient characteristics

These patients are usually older (~80 years), and about 60% are female. They have significant pulmonary hypertension (i.e., PASP over 50 mmHg) [47]. Echocardiography shows characteristics of markedly increased E/*e'* ratio, LA volume, and PCWP. PASP is higher in these patients at the same level of PCWP compared to patients with hypertension, suggesting that these patients have combined pre- and post-capillary PH (i.e., cpc-PH [48]). These patients also have borderline or mild HTN. The prevalence of CAD (53%), a-fib (31%), diabetes (34%), COPD (~32%) and chronic renal insufficiency (CRI) (50%) are similar to other HFpEF patients.[47] This phenotype is often associated with obesity.

Phenotype P anesthesia implications

The degree of PASP predicts outcomes in HFpEF.[47] Therefore, effective PASP management should produce better perioperative outcomes in this phenotype group. Pulmonary arterial capacitance (PAC), the ratio of stroke volume (SV) over pulmonary pulse pressure (PP), is a robust independent predictor of mortality in PH patients [49]. The PAC-PVR relationship shifts downward and left when wedge pressure is increased [50], as in HFpEF patients during exercise [51]. Since pre-capillary components contribute to PH, vascular therapies targeting the pulmonary artery and volume control to reduce congestion may be considered. However, caution must be exercised when attempting to decrease pulmonary arterial resistance. This effort will promote RV output, further overloading the left heart with significant diastolic dysfunction and over-extended LA. Therefore, testing whether the patient will tolerate pulmonary vasodilation is crucial before implementing such therapy.

Pulmonary vascular remodeling not only affects hemodynamics but also affects the lung's efficiency for gas exchange [52]. Diffusion capacity of the lungs (measured as DL_{CO2}) and alveolar-capillary membrane conductance (D_M) decrease in HFpEF patients compared to controls. Recruiting vascular volume is limited during maximal exercise. These changes restrict gas transfer and promote exercise intolerance in these patients. Thus, during anesthesia, efforts to protect the lungs should be made to avoid further damage to the alveoli and maximize O_2 delivery.

The right heart undergoes compensation in hypertrophy and chamber size during PH in HFpEF patients [53]. RV diastolic dysfunction usually occurs before RV systolic deterioration (dysfunction) [54]. RV failure is a significant predictor of mortality in PH patients [55]. Therefore, management of RV failure is a priority should it occur, including inotropic support and correcting RV-PA uncoupling.

Phenotype P anesthesia management

The degree of PH should be recognized, and the contribution of pre- and post-capillary components to PH should be assessed. Patients with severe PH are expected to be treated as outpatients before coming to surgery. We should know the medicines used to treat and the patient's response to them. Treating severe PH during the immediate perioperative period is desirable, if possible. The degree of cardiac, especially RV remodeling, should also be evaluated in terms of hypertrophy, chamber size, and function (systolic and diastolic). Despite expected normal systolic function, the remodeled heart in these patients is at higher risk of cardiac ischemia and abnormal responses to drugs and volume.

Lung function should be assessed, given the decrease in gas diffusion capacity. Lung function tests are recommended, especially baseline function. Invasive arterial blood pressure monitoring is needed before induction. Monitoring RV function using TEE or PA pressure should be implemented for moderate to high-risk surgeries. Volume restriction and pulmonary vasodilators should be used if possible. Systolic RV failure must be treated aggressively using inotropic support. There are no specific therapies for RV diastolic dysfunction and failure at present [54], but close monitoring of these patients for signs is recommended, especially for elderly patients at high risk of frailty. The aim of ventilation is two-fold: (a) protecting the (already injured) lungs from additional mechanical ventilation injury and (b) maximizing O_2 delivery to compensate for decreased gas exchange capacity. Protective lung ventilation with optimized FiO_2 is desirable. Hypoventilation, hypoxia, and V/Q mismatch (in case of one lung ventilation) must be avoided. These patients should be extubated after surgery whenever possible. During the postoperative period, if patients need intubation, the duration should be as short as possible to prevent further lung injury. Also, they should be closely monitored for worsening PH and RV dysfunction. Postoperative admission to monitored or ICU beds for high-risk patients or high-risk surgeries may prevent complications and bad outcomes for these patients.

Phenotype C

Phenotype C patient characteristics

This patient group has significant CAD or coronary microvascular dysfunction (CMD) with normal EF [56]. Over 60% of HFpEF patients with CAD are male. CMD is a reflection of inflammation-associated endothelium injury seen in HFpEF [57]. Cardiac MRI often shows decreased

myocardial perfusion, increased fibrosis, and increased extracellular volume.[56] Of those with CAD, 50% had no prior clinical CAD history. Over 60% of patients have CMD regardless of CAD. HFpEF patients with CAD tend to have higher LV filling pressures, and those with CMD tend to have more atrial fibrillation. Most are NYHF Class III-IV.

Phenotype C anesthesia implications

CAD and CMD render patients prone to cardiac ischemia and dysfunction, especially under stress. Since HFpEF patients with CAD have a higher mortality risk than those without CAD [58], perioperative morbidity and mortality may be reduced with pre-surgical coronary angioplasty. HFpEF patients with CMD are equally at risk for perioperative cardiac complications due to decreased coronary perfusion. The microvascular dysfunction is endothelium-independent and caused by abnormal vascular remodeling (rather than vascular dysfunction), extrinsic compression, and microvascular rarefaction. Thus, coronary vasodilator therapy may be less effective than controlling volume and filling pressure to optimize coronary perfusion (relieving extrinsic compression of the microvasculature). CMRI shows evidence of myocardial infarction in some patients without clinical history. The (clinically) unrecognized MI is another risk factor for these patients' cardiac dysfunction (failure). HFpEF patients with CAD have a higher incidence of sudden (cardiac) death, with an odds ratio of 3.36 (univariate analysis) and 2.22 (multivariate analysis) [59].

Phenotype C anesthesia management

All HFpEF patients with CAD should also be suspected of having CMD, given the high prevalence (i.e., >60%) [56]. They should also be suspected of having an infarcted myocardium, even without clinical history. Thus, the presence and degree of myocardial ischemia should be carefully assessed (i.e., reviewing cardiac catheterization, coronary vascular activity testing, and other imaging studies). Although they have a maintained EF, the risk of cardiac functional deterioration must be evaluated, considering the structural remodeling of heart chambers, valvular functions, hemodynamics, and (when possible) myocardial stress/strain. Intraoperative management should focus on monitoring/identifying cardiac ischemia and functional deterioration and managing them promptly. Invasive monitors are preferred in intermediate- and high-risk patients or surgeries. Oxygen demand/supply balance is maintained by multimodal approaches (anesthesia types, volume/fluid management, vasoactive agents use, anesthetic agent use, and pain management). Cardiac functional status can be readily monitored by transesophageal echocardiography (TEE), with heart failure treated promptly. One should also be on high alert for the risk of sudden cardiac death and manage it effectively when it occurs. These patients should be monitored for cardiac ischemia and arrhythmias during the postoperative period, and if needed, they should be admitted for continuous monitoring for an extended period.

Phenotype Y

Phenotype Y patient characteristics

These patients are usually 60-65 or younger, primarily male, and obese. They have less severe heart failure symptoms (i.e., < II NYHA class) and low NT-proBNP levels [14]. The prevalence of other comorbidities is low in this group. For example, over 80% of these patients have normal renal function (i.e., eGFR >30 ml/min). Less than 20% have diabetes, COPD, and a-fib [60]. About 30% have ischemic heart disease, and 32% have diastolic pressure >80 mmHg. Cardiovascular remodeling is less severe: they have the least concentric LVH and wall thickness, largest LV volume, smallest LA volume, smallest E/*e′* ratio, lowest SVR, highest arterial compliance, and lowest arterial stiffness among the phenotypes.[14] However, these patients have similar filling pressures and E/*e′* as their older counterparts if the levels of NT-proBNP are similar [60]. In certain ethnic groups (Asian Malay and Indian men), young HFpEF patients have similar cardiac remodeling as older patients with similar symptom burdens (but a lower comorbidity burden) [60].

Phenotype Y anesthesia implications

This group represents the healthiest "garden variety" HFpEF patients. Because of this, one should consider the possibility that these patients may have been misdiagnosed with HFpEF. They are well compensated with few clinical symptoms and comorbidities. About a quarter of them are chronic smokers. In general, their clinical outcome is more favorable than other phenotypes. Likewise, their perioperative risks for cardiovascular and other complications are mainly determined by the risk of procedures/surgeries. However, we should not overlook the high prevalence of obesity in this group. Also, we should be wary of potential cardiac complications due to cardiac remodeling. Last but not least, certain ethnic groups, such as Asian, Malay, and Indian young men, may have disease burdens as substantial as older patients, and caution is warranted when caring for them during the perioperative period.

Phenotype Y anesthesia management

Because most of these patients are obese, the degree of obstructive apnea and airway should be evaluated carefully. We should also consider comorbidities in patients from certain ethnic groups (Asian Malays, Asian Indians, and African Americans) during preoperative assessment. COPD should be sought, given the higher smoking prevalence in these groups. In these patients, routine, standard monitoring is sufficient for low- and some intermediate-risk procedures/surgeries, with invasive monitoring reserved for high-risk surgeries. If COPD is present, peripheral nerve block or regional is preferred if indicated. Intraoperative management is no different from managing other non-HFpEF patients, such as being vigilant for hemodynamic disturbances and treating them promptly. The surgeries

dictate postoperative care regarding whether patients need continuous close monitoring and intensive management.

Clinical Perspective

Given the recognized risk of perioperative complications for patients with HFpEF, an effective management strategy is needed to mitigate risk. The heterogeneous nature of HFpEF prompted the idea of personalized therapy based on phenotyping HFpEF patients [4,11]. Phenotyping has successfully identified specific subgroups with distinct clinical features, outcomes, and responses to therapies [12,14].

Our attempt to phenotype HFpEF patients coming to the operating room represents a needed first step to optimize the perioperative care of these patients. Whether such an approach will improve outcomes in HFpEF patients remains to be seen. A parallel attempt is to analyze an existing database using similar phenotyping to identify subgroups of patients who underwent surgeries with associated outcomes. Ultimately, an individualized approach based on phenotyping is needed for HFpEF patients to deliver safe, high-quality care when they come for surgery.

Summary

HFpEF patients bring substantial risks to the operating room. Perioperative care for these patients is challenging due to the heterogeneous etiologies, clinical presentations, therapy responses, and clinical outcomes. Phenotyping (or sub-grouping) according to clinical features (and biomarkers) has identified different groups of HFpEF patients with distinct characteristics regarding disease presentation, clinical behaviors, and clinical outcomes. For HFpEF patients coming to surgical procedures, 5 (likely) phenotypes are identified with considerations to issues pertinent to anesthesia care: phenotypes A, O, P, C, and Y. Each phenotype has its own perioperative issues. We highlight the key issues relevant to anesthesia care and propose management principles for each phenotype throughout the perioperative period. Such an approach will address the challenges for HFpEF patients and deliver high-quality, safe perioperative care for these patients.

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Carbon Footprint of Total Intravenous and Inhalation Anesthesia in the Transcatheter Aortic Valve Replacement Procedure

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Introduction

Climate change is potentially the most significant long-term health threat of the twenty-first century, and it has emerged as a top priority for clinicians and healthcare leaders [1]. Inhaled anesthetics contribute significantly to healthcare-related greenhouse gas emissions [2]. The employment of low fresh gas flow during the administration of inhaled agents, the avoidance of desflurane and N2O, and, when possible, the use of total intravenous anesthesia (TIVA) have been touted as strategies to reduce the carbon footprint [3,4]. The data supporting these recommendations are based on both theoretical and small patient cohorts [5–7].

Since the first transcatheter aortic valve replacement (TAVR) device was approved in 2011, more than 276,000 patients in the United States have undergone a TAVR procedure [8]. Different approaches to anesthesia for TAVR include general anesthesia, moderate to deep sedation, and even minimalist sedation [9]. The growing enthusiasm for the use of sedation over general anesthesia has focused on the decreased use of pressor requirements and shorter lengths of stay [10]. To date, there has not been an environmental impact analysis of the different approaches.

We aimed to quantify and compare the respective carbon dioxide emissions (CO2e) for sedation with TIVA and inhaled agents during the TAVR procedure. We hypothesized that the overall carbon footprint of deep sedation with TIVA would be lower than that of general anesthesia with inhaled agents.

Materials and Methods

The study was reviewed by Tufts Medical Center Institutional Review Board (# STUDY00003471). After the approval, the anesthesia records of patients who underwent the TAVR procedure between January 1, 2018 and March 31, 2022 were reviewed. The related anesthesia records were downloaded from the Soarian Electronic Medical Record System and exported into Excel. The approach to anesthesia is determined by the anesthesiologist and his/her determination of suitability for sedation. Patients felt to be at higher risk of upper airway obstruction or who have alternative access sites such as trans-carotid, subclavian or transapical are provided general anesthesia. Patients receiving inhalation agents had airways managed with either an endotracheal tube or a laryngeal mask. It is

the general practice at our institution to run patients receiving inhaled agents at 0.5 +/- 0.1 minimum alveolar concentration (MAC). The depth of sedation with TIVA was titrated so that patients were not responsive to painful stimuli. Patients who required conversion to general anesthesia with an airway device and inhaled anesthetics after starting with deep sedation with TIVA were excluded from the analysis.

Each patient's age and gender, height and weight, ASA class, anesthesia type, anesthesia start and stop times, and airway management devices were collected. The type and amount of intravenous medication used during the anesthesia were obtained for each case. The time series of medical air, oxygen, N2O, and vaporizer flow rates were extracted from the anesthesia records.

The carbon footprint for a case was determined by calculating carbon dioxide equivalents (CO2e) incorporating all the pharmaceutical agents used including both intravenous and inhalational anesthetics as well as pressors, inotropes, neuromuscular blocking agents and antibiotics etc. The CO2e of intravenous medications during the anesthesia was converted with the CO2e data for anesthetic pharmaceuticals from Parvatker et al [11]. For the inhaled agents, the flow rate was captured at 15-minute intervals. The CO2e was calculated using the anesthetic gases calculator provided by the Association of Anesthetists based on the carrier gas flow rate, the vaporizer setting (%), and the global warming potential (GWP) of the inhalational anesthetics [12].

The information of airway management devices (oxygen mask, breathing circuit, laryngeal mask airway, endotracheal tube, stylet, when used disposable plastic laryngoscope blades, reusable metal laryngoscope blades and handles) were collected from the electronical anesthesia records. Many of these items have published carbon emissions equivalent data based on life-cycle assessments. These included: disposable plastic laryngeal mask airway [13], disposable plastic laryngoscope blades, reusable metal laryngoscope blades and handles [14]. For those items without available emission data, we measured the weight of the individual plastic items and converted them into carbon emissions based on the CO2e emission factors 3179 kg CO2e/ton with a 10% modifier to reflect the small non-plastic portion [15]. These items included: disposable oxygen mask, anesthetic breathing circuit including its associated face mask, endotracheal tube, and stylet. According to the manufacturer's data (Drager, GE, USA), the electricity consumed by the anesthesia machine while ventilating a patient was 1.8 Amperes at 110 volts AC current while the standby power consumption at 110 volts AC current was 1.6 Amperes [16]. The CO2e of anesthesia machine electricity was estimated by the local Energy Information Administration data [15]. The CO2e from carbon dioxide absorbent used during the general anesthesia with inhaled agents was also included (0.13kg CO2e /h from Zhong et al [17]). We did not include data for heating/ventilation/air conditioning or any surgical equipment because the comparison was in the same procedure in the same operating room. A heated underbody water blanket was used in each case of both groups to mitigate hypothermia. In addition, vasoconstricting agents (phenylephrine/ norepinephrine) are typically administered before induction to preemptively offset vasodilation and hypotension in the context of aortic stenosis. This practice, in conjunction with the warming blanket, likely offset the typical redistribution of heat seen with peripheral vasodilation on induction. There is a low likelihood for potential differences in heat redistribution in GA with inhaled agents and deep sedation with TIVA. The CO2e emissions were summarized as total data for each case.

Statistics

An *a priori* power analysis based on the results of McGain's study [5] demonstrated that a total sample size of 158 patients was needed for the statistical analysis with 80% power and 0.05% alpha. The SPSS Software (Version 27.0) was used for the statistical analysis. Chi-squared test or Wilcoxon rank-sum test was employed. All statistical analyses were performed

using a two-sided t test, with P<0.05 representing a statistically significant difference.

Results

We identified a total of 710 anesthesia records during the specified period. Of these cases, 18 (2.54%) anesthesia patients were converted from deep sedation to general anesthesia and were excluded from the analysis. 33 inhaled anesthesia records were excluded because more than one volatile sevoflurane, isoflurane, or desflurane was used. 55 records were excluded due to incomplete data (Appendix A). Finally, 285 general anesthesia with inhaled agents records and 319 deep sedation with TIVA records were analyzed (**Fig 1**).

There were no significant differences in the patient's demographic characteristics, such as age, gender, weight, height, and BMI. The percentage of ASA Class 3 in deep sedation with the TIVA group was higher than that in the general anesthesia with inhaled agents group (13.48% vs. 7.02%, P = 0.020, **Table 1**). The anesthesia duration in the general anesthesia with inhaled agents group was longer than that in the deep sedation with TIVA group (242 mins vs. 179 mins, P < 0.001, **Table 1**). The median flow rate during the maintenance phase of anesthesia with inhaled agents was 2.25 with a range of 1.3 to 3.2 L/min.

There was no significant difference between the kg CO2e per case generated by intravenous medications between the two groups (1.082 vs. 1.071, P = 0.103, **Table 2**). In the general anesthesia with inhaled anesthesia group, the average CO2e emission from the inhaled agents was 8.969 kg CO2e. The average kg CO2e of airway management devices during general anesthesia with inhaled agents was 2.6 per case. The carbon footprint of electricity from the anesthesia machine during general anesthesia was an average of 0.27 kg CO2e per case. In the deep sedation with TIVA group, the median of CO2e from the inhaled agents was 0.007 kg, from airway management devices was 0.25 kg, and from anesthesia machine electricity was 0.18 kg. In the analysis of total pharmaceuticals per minute including both inhaled and intravenous medications, patients receiving inhalation agents had more than seven-fold higher CO2e emission per minute than those receiving TIVA (0.040 vs. 0.006 kg CO2e/min, P < 0.001). The patients who received inhalation agents also had statistically higher total CO2e per case (16.188 vs. 1.518 kg CO2e, P < 0.001),

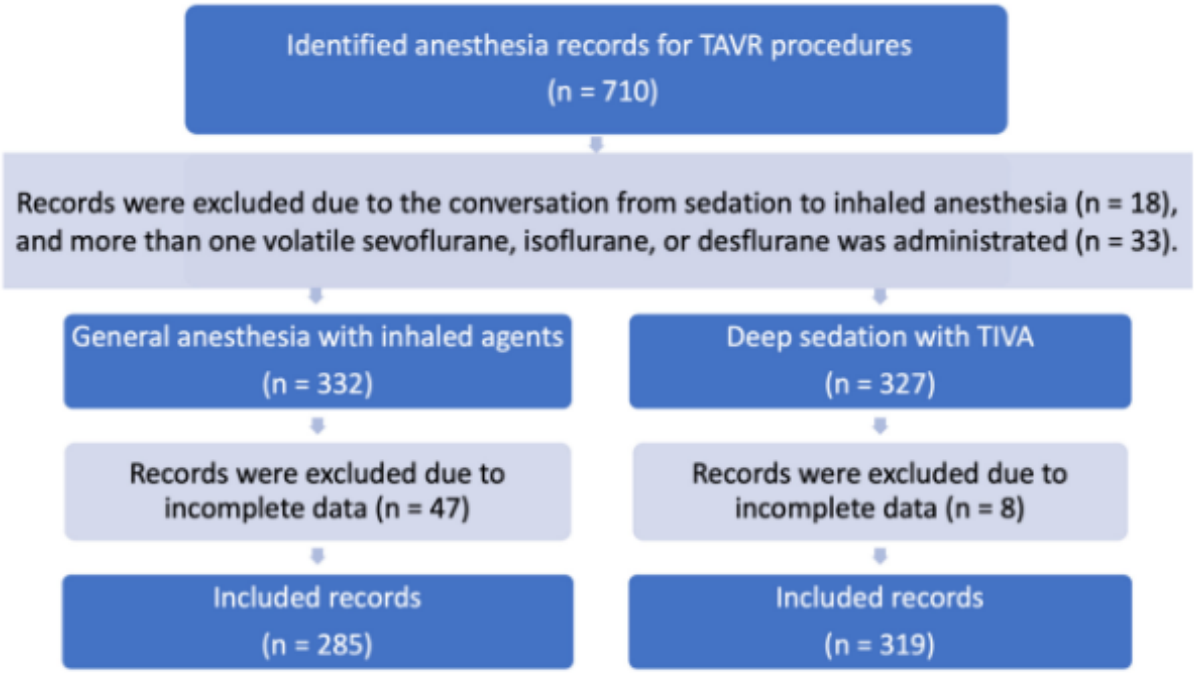

Fig 1. Study flow diagram. TAVR, transcatheter aortic valve replacement; TIVA, total intravenous anesthesia.

Table 1
Patient Demographics Characteristics

Items	General Anesthesia With Inhaled Agents (n = 285)		Deep Sedation With TIVA (n = 319)		p Value
	Median (IQR)	Range	Median (IQR)	Range	
Age (y)	78 (12)	38-94	80 (12)	57-95	0.235
Sex (male, %)	162	56.84%	180	56.43%	0.935
Weight (kg)	79.70 (25.00)	39.92-146.96	80.00 (22.90)	42.50-136.36	0.946
Height (in)	65.7 (6.7)	52.0-77.2	66.1 (6.7)	57.9-83.0	0.081
BMI	28.29 (8.84)	15.59-57.91	28.06 (6.75)	15.61-53.25	0.744
ASA class					0.020*
3	20	7.02%	43	13.48%	
4	262	91.93%	275	86.21%	
5	3	1.05%	1	0.31%	
Anesthesia duration (min)	242 (71)	114-490	179 (54)	101-395	< 0.001*

Abbreviations: ASA, American Society of Anesthesiologists; BMI, body mass index; TIVA, total intravenous anesthesia.
* Indicates that there is a significant statistical difference.

primarily due to the inhaled agents and secondarily due to airway management devices **(Table 2**).

Intravenous medications contributed to on average 1.071 kg (71.02%) for the deep sedation with TIVA and 1.082 kg CO2e (8.05%) for the general anesthesia with inhaled agents. For general anesthesia, inhaled agents were the principal contributor to the total CO2e (8.969 kg CO2e, 66.73%). Airway management devices contributed more greatly to the environmental impact in patients receiving inhaled anesthetics [2.6 kg CO2e (19.34%) vs. 0.25 kg CO2e (16.58%), P < 0.001]. The electricity of the anesthesia machine contributed 0.27 kg CO2e (2.01%) of the total CO2e in the general anesthesia with inhaled agents and 0.18 kg CO2e (11.94%) of the total CO2e in the deep sedation with TIVA (**Table 2, Fig 2**).

There were the masses and calculations of pharmaceuticals used during both general anesthesia with inhaled agents and deep sedation with TIVA, which were categorized by anesthetics, cardiovascular medications, antibiotics, and others. Patients in the sedation arm received a median dose of propofol 219.5 mg per case with a range from 7.09 to 1429 mg per case. Patients in the general anesthesia with inhaled agents received a median dose of propofol 261 mg per case with a range from 24 to 2028 mg per case (**Supplement Table 1**).

Desflurane has the highest global warming potential (GWP) at 2,540 within the inhaled anesthetics [18]. One patient received desflurane only with 95.71 kg CO2e. There were 21 patients receiving isoflurane only with a median of 9.30 kg CO2e and 155 patients receiving sevoflurane only with a median of 6.65 kg CO2e. Three patients received N2O only with an average of 62.12 kg CO2e. Compared with using the inhaled anesthetics alone, the CO2e emissions from using a combination of volatile anesthetics are more than 10-fold greater. There were 10 patients receiving both N2O and isoflurane, 95 patients receiving both N2O and sevoflurane. The average of CO2e were 71.84 kg and 27.58 kg respectively (**Table 3**).

In addition to the pharmacologic contribution to emissions, disposable items were included in our analysis. These airway management devices were calculated based on the material types and weights, and previous life-circle assessments. Both patients in general anesthesia with inhaled agents and deep sedation with TIVA groups were given an oxygen mask. Thirty-nine patients underwent general anesthesia with inhaled agents had a laryngeal mask placed, while 246 patients were intubated. The laryngeal mask airway has the highest carbon dioxide equivalent emissions due to the weight and material (**Table 4**).

The emissions from a given case that used inhalational agents was highly variable depending on the specific agent or combination of inhalations agents used (**Table 3**). A subgroup analysis of patients who only received sevoflurane was performed. There were no significant differences in the patient's demographic characteristics, such as age, gender, weight, height, and BMI, between the deep sedation with the TIVA group and the sevoflurane general anesthesia group.

The percentage of ASA Class 3 in the deep sedation with TIVA group was higher than that in the sevoflurane general anesthesia group (13.48% vs. 5.81%, P = 0.039, **Table 5**). There was no significant difference between the kg CO2e per case in intravenous medications between the two groups (1.083 vs 1.071, P = 0.050, **Table 5**). The total pharmaceuticals per min (kg CO2e per min) in sevoflurane anesthesia group was 4.5 times of that in deep sedation with TIVA group (**Table 5**).

Table 2
CO_2e Between General Anesthesia With Inhaled Agents and With Deep Sedation per Case

Items	General Anesthesia With Inhaled Agents (n = 285)		Deep Sedation With TIVA (n = 319)		p Value
	Median (IQR)	Range	Median (IQR)	Range	
Intravenous medications (kg CO_2e)	1.082 (0.073)	0.026-5.643	1.071 (0.115)	0.041-2.324	0.103
Inhaled agents (kg CO_2e)	8.969 (32.721)	0.227-455.161	0.007 (0.002)	0.004-0.016	< 0.001*
Total pharmaceuticals (kg CO_2e)	10.005 (32.787)	1.341-455.897	1.079 (0.115)	0.048-2.338	< 0.001*
Total pharmaceuticals per min (kg CO_2e/min)	0.040 (0.136)	0.006-1.546	0.0059 (0.0024)	0.0003-0.014	< 0.001*
Airway management devices (kg CO_2e)	2.6 (0.16)	2.6-13.49	0.25	0.25-0.25	< 0.001*
Anesthesia machine electricity (kg CO_2e)	0.27 (0.078)	0.13-0.55	0.18 (0.054)	0.10-0.39	< 0.001*
Carbon dioxide absorbent (kg CO_2e)	0.52 (0.152)	0.25-1.06	0	0	—
Total (kg CO_2e)	16.188 (33.705)	4.742-459.625	1.518 (0.278)	0.457-2.944	< 0.001*

Abbreviations: CO_2e, carbon dioxide emissions; TIVA, total intravenous anesthesia.
* Indicates that there is a significant statistical difference.

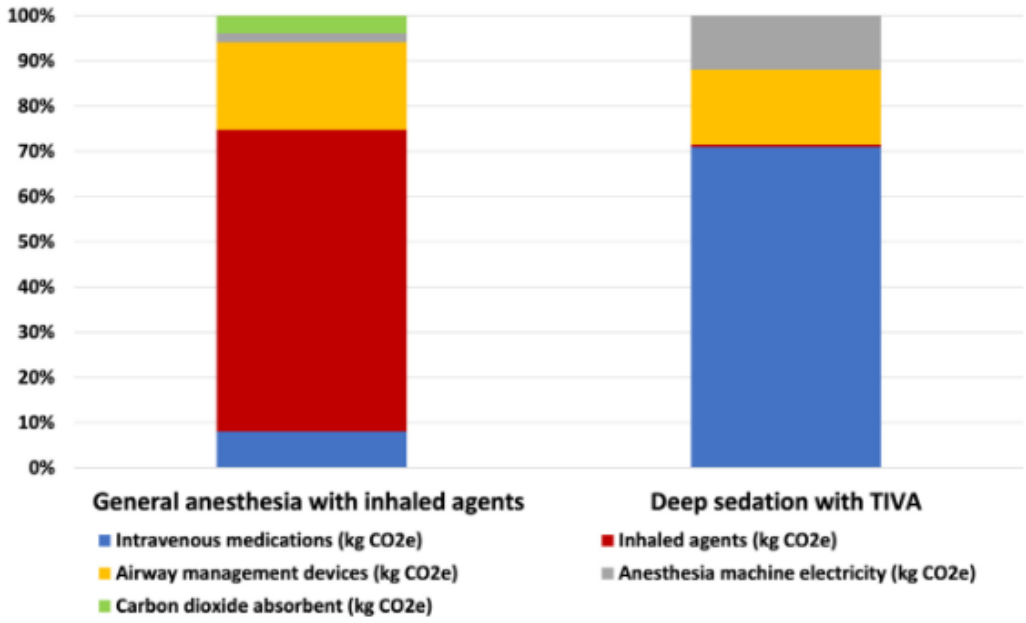

Fig 2. Categorizations of carbon dioxide emissions: general anesthesia with inhaled agents and deep sedation with total intravenous anesthesia. CO_2e, carbon dioxide emissions; TIVA, total intravenous anesthesia.

Discussion

Our results support our hypothesis that general anesthesia with inhalational agents has a higher carbon emission than deep sedation with TIVA during the TAVR procedure. This study is a real-world dataset to compare the carbon footprint of inhalational and intravenous anesthetics in clinical supply utilization rather than a modeling study. The findings reveal during the TAVR procedure that deep sedation with TIVA has a significantly lower carbon footprint when compared to general anesthesia with inhaled agents. The major components of CO2e emissions in general anesthesia with inhaled agents were inhaled agents themselves and airway management devices (endotracheal tubes and breathing circuits), while the main contributors to CO2e emissions in deep sedation with TIVA were

intravenous medications and airway management devices (oxygen masks).

To date there are four publications investigating the comparative environmental impact of different pharmacological approaches anesthesia [5–7,18]. A life cycle assessments involving a "cradle-to-grave" is a methodology to measure the environmental impact of a product during production until disposal [19]. Sherman et al. using a cradle-to-grave life cycle assessment in 2012 compared four inhaled anesthetics sevoflurane, isoflurane, desflurane, N2O and propofol. The analysis showed that desflurane had the greatest CO2e effect as 15 times that of isoflurane and 20 times that of sevoflurane. In contrast, propofol's CO2e was comparatively quite small [18]. Allen et al. in 2021 employing a theoretical model involving a life cycle assessment of propofol and remifentanil, concluded that a propofol TIVA anesthesia produced much less carbon emissions than a volatile-based anesthetic [7]. An additional study by Narayanan et al. investigated pediatric dosing using a mathematical simulation model to compare the two anesthesia approaches. Similarly, they demonstrated that TIVA was less environmentally detrimental [6]. Finally, McGain et al. prospectively compared the carbon footprints of general and spinal anesthesia with TIVA sedation with ten patients in each group. They found that inhaled agents had greater carbon emissions than that of spinal anesthesia with TIVA [5].

In our study, we analyzed 285 inhalational anesthetics against 319 TIVA with deep sedation. We considered the specific type and weight of each intravenous medication and the flow rate of every inhaled agent. We observed a substantial variation in carbon emissions within general anesthesia with inhaled agents group. This range (4.742-459.625) could be attributed to the choices of inhaled anesthetics and flow rates used. This finding is consistent with previously published findings on how variations in anesthesia practices, particularly the selection of gases with higher greenhouse warming potentials can negatively impact a cases overall emissions [20]. Therefore, we performed the subset analysis of patients receiving only sevoflurane which has the lowest emissions of the inhaled agents. In this analysis, we found that while the environmental impact of sevoflurane in TAVR procedures was less than other inhaled anesthetics, the environmental impact remained many fold higher than deep sedation with TIVA.

This project is subject to several limitations. As a single-center retrospective study, the specific results of this analysis may not be generalizable to other TAVR programs. As mentioned, there is a continuum of depth of anesthesia that is provided for TAVR patients [9]. Clearly, if moderate sedation or a minimalist approach to anesthesia had been used, the differences in the environmental impact would have likely been more pronounced. Given the real-world nature of this data, fresh gas flow rates for patients receiving inhaled agents varied in this retrospective study. Minimal flow or “closed-loop” anesthesia is not routinely employed in our institution, which has been advocated to reduce carbon emissions [21]. The generalizability of our results are dependent on fresh gas flow rates a clinician chooses to employ. The goal of the study was not just to compare the environmental impact of sedation versus general anesthesia in the TAVR

Table 3
CO_2 Equivalent Emissions of Different Inhaled Anesthetics per Case in Inhalation Anesthesia

Inhalation Anesthesia (n = 285)	IPCC GWP 100a (kg CO_2e)	N	Inhaled Anesthetics Median (kg CO_2e/case)	Inhaled Anesthetics Range (kg CO_2e/case)
Desflurane	2540	1	95.71	—
N_2O	310	3	62.12	42.16-100.20
Isoflurane	510	21	9.30	2.53-16.07
Sevoflurane	130	155	6.65	4.53-8.76
N_2O/Isoflurane	310/510	10	71.84	12.83-130.84
N_2O/Sevoflurane	310/130	95	27.58	10.80-44.37

Abbreviations: CO_2e, carbon dioxide emissions; GWP, global warming potential; IPCC, Intergovernmental Panel on Climate Change.

Table 4
CO_2 Equivalent Emissions of Different Airway Management Devices

Item	Kg CO_2e per Item	N
Oxygen mask	0.25	285 + 319 = 604
Breathing circuit	1.94	285
Laryngeal mask airway	11.3	39
Endotracheal tube and stylet	0.11	246
Reusable metal handle with low-level disinfection	0.08	246
Disposable plastic blade (McGrath/Glidescope)	0.38	88
Reusable metal blade (Mac/Miller) with sterilizing	0.22	158

Abbreviation: CO_2e, carbon dioxide emissions.

procedure per se but to study the environmental impact of the different agents. To achieve that end, the two groups ideally would have had identical depths of anesthesia. We do not routinely use the bispectral index to monitor the depth of sedation, so this kind of data was not available to us. Inhalation agents as mentioned in the methodology are typically run at 0.5 ± 0.1 MAC at our institution when providing general anesthesia together with paralysis to avoid involuntary movement. When deep sedation is used, we typically titrate the depth of anesthesia to avoid coughing or movement with painful stimulation when obtaining large-bore vascular access and holding pressure after removing sheaths. If absence of movement or coughing cannot be obtained while maintaining a patent airway despite employing maneuvers such as jaw thrusts, oral airways, or high flow nasal cannulas at greater than 50 liters/minute, patients are typically converted to general anesthesia with an inhalation agent. It is our clinical suspicion that the depth of anesthesia between the two groups was rather similar, although we cannot prove this in this retrospective dataset. Importantly, even if the depth of anesthesia used in the TIVA group were two-fold greater, this would not make up for the seven-fold difference in emissions observed between inhalational and intravenous techniques. For this reason, we feel that our global conclusions remain sound. Patients receiving inhalational agents in our study also had longer procedural durations which would obviously impact the total emissions for each case. For this reason, we included the analyses of total pharmaceuticals per min (kg CO2e/min) that does not include non-reusable items. After adjusting for differences in case duration in this way, we found that inhaled agents still had a many times higher emission rate than deep sedation with TIVA.

The proportion of missing data in anesthesia records was relatively higher in the general anesthesia with inhaled agents group than in the deep sedation with TIVA group (16% versus 3%). Because of the range of environmental inhaled agents with much higher greenhouse warming potentials than sevoflurane, this missing data could have a result of underappreciation of the global emissions produced by inhalational agents in our study. In attempt to address this, we studied the subgroup of sevoflurane only, we found that the emission per minute was 4.5 times higher than deep sedation with TIVA.

Finally, this study focuses on the comparative carbon footprints of deep sedation with TIVA versus general anesthesia with inhaled anesthesia during the TAVR procedure. We chose TAVR because of the internal consistence and a relatively high volume in our institution in order to properly power the study. We did not account for the wasted medication because we would like to assess the medications that were genuinely required for the anesthesia. We did not attempt to measure the overall environmental impact of the procedure itself. We ignored the contribution of the energy used for fluoroscopy or lighting, heating, ventilation, air-conditioning for the hybrid room and importantly, biohazard waste disposal and emissions associated with bioprosthetic aortic valve itself. In a study of coronary artery bypass grafting the environmental impact of anesthesia was comparably small to the overwhelming volume of biohazardous waste that required incineration [15]. We included carbon dioxide absorbent for general anesthesia with inhaled agents.

As climate change threatens our public health, it is imperative for the healthcare sector to consider the how we might “first do no harm.” We need to implement environmentally sustainable practices while providing

Table 5
CO_2 Equivalent Emissions Between Sevoflurane Inhalation Anesthesia and Intravenous Anesthesia per Case

Items	Sevoflurane Inhalation Anesthesia (n = 155)		Intravenous Anesthesia (n = 319)		p Value
	Median (IQR)	Range	Median (IQR)	Range	
Age (y)	78 (12)	38-94	80 (12)	57-95	0.087
Sex (male, %)	80	51.61%	180	56.42%	0.327
Weight (kg)	81.81 (26.83)	39.92-145.59	80.00 (22.90)	42.50-136.36	0.649
Height (in.)	65.7 (7.1)	52.0-77.2	66.1 (6.7)	57.9-83.0	0.133
BMI	28.42 (10.10)	15.59-57.91	28.06 (6.75)	15.61-53.25	0.695
ASA class					0.039*
3	9	5.81%	43	13.48%	
4	145	93.55%	275	86.21%	
5	1	0.64%	1	0.31%	
Anesthesia duration (min)	241 (70)	155-406	179 (54)	101-395	< 0.001*
Intravenous medications (kg CO_2e)	1.083 (0.0726)	0.026-5.643	1.071 (0.115)	0.041-2.324	0.050
Inhaled agents (kg CO_2e)	5.504 (4.470)	0.227-17.208	0.0072 (0.002)	0.004-0.016	< 0.001*
Total pharmaceuticals (kg CO_2e)	6.725 (4.431)	1.341-18.344	1.0785 (0.115)	0.048-2.338	< 0.001*
Total pharmaceuticals per min (kg CO_2e/min)	0.027 (0.0165)	0.006-0.056	0.0059 (0.0024)	0.0003-0.0138	< 0.001*
Airway management devices (kg CO_2e)	2.6	2.6-13.49	0.25	0.25-0.25	< 0.001*
Anesthesia machine electricity (kg CO_2e)	0.269 (0.0771)	0.173-0.454	0.1777 (0.054)	0.100-0.392	< 0.001*
Carbon dioxide absorbent (kg CO_2e)	0.522 (0.150)	0.336-0.880	0	0	—
Total (kg CO_2e)	10.955 (5.593)	4.742-29.096	1.518 (0.278)	0.457-2.944	< 0.001*

Abbreviations: ASA, American Society of Anesthesiologists; BMI, body mass index; CO_2e, carbon dioxide emissions.
* Indicates that there is a significant statistical difference.

the best possible care for individuals. Our data consistent with that of others suggests that we should use intravenous agents rather than inhaled agents when possible. Future research should prioritize the development and deployment of sustainable anesthetic solutions that provide patient safety and comfort while minimizing their environmental impact. This approach should encompass on not only the anesthetic medications but also the larger issue of minimizing the solid waste/disposing items in the perioperative space, which becomes the trend that more single-use items and parts are used.

Supplementary materials

Supplementary material associated with this article can befound in the online version at doi:10.1053/j.jvca.2024.02.027.

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临床病例报道

低体重新生儿先天性膈疝胸腔镜手术的麻醉管理

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先天性膈疝（congenital diaphragmatic hernia, CDH）是膈肌缺损导致不同程度的腹部内脏进入胸腔，发病率约为 1:4000[1]。CDH 可能是一个孤立的疾病，也可能并发其他畸形或症状，如 Beckwith-Wiedemann 综合征、CHARGE、各种三体染色体异常等[2, 3]。通常在新生儿期即出现呼吸窘迫，但约 10 %在新生儿期以后出现症状，肺泡膜增厚以及功能残气量降低引起通气交换受限，常伴随肺发育不良、肺动脉高压和气道反应性增高[1]，新生儿重症膈疝的病死率可达 70 %[4]。CDH 的根本治疗手段是手术修补膈肌，胸腔镜手术相对于传统开放手术的优势在于损伤小、视野好、术后疼痛更轻。然而，胸腔镜手术往往时间长，进一步压迫肺组织，更容易出现低氧血症、高碳酸血症及酸中毒，从而加重肺损伤[5, 6]，CDH 本身的病理生理特点也给麻醉带来极大困难[7-9]。现将我院新生儿外科成功收治的 1 例低体重先天性膈疝患儿麻醉管理经验总结如下。

一、 临床资料

一般资料：患儿，女，体重 2.2 kg，孕 37^{+2} 周，因生后 9 小时出现呼吸急促，伴颜面、四肢末端发绀入院。入院后患儿血氧饱和度降至 75%，在病房紧急行气管插管，血氧饱和度波动于 96 ~ 98 %。床旁超声示：颅内囊性结节，脾脏位于左侧胸腔-左侧膈疝，肝脏增大，动脉导管未闭，卵圆孔未闭，左右心室功能正常，未见明显肺动脉高压迹象。胸 CT 检查示脾脏位于左侧胸腔-左侧膈疝，右肺部分不张并散在实变影，左肺不张，见图 1。

术前诊断：1.先天性膈疝 2. 呼吸衰竭？3.肺发育不良 4.颅内囊性结节 5.消化道畸形？6.肝脏增大 7.低体重儿 8.动脉导管未闭 9.卵圆孔未闭 10.凝血功能异常。

拟行手术：胸腔镜探查、膈疝修补术。

麻醉管理：手术室温度预先调至 25 ℃左右，手术床铺好加温毯。患儿带气管插管辅助通气入手术室，脉搏 149 次/分，血压 65/36 mmHg，血氧饱和度 96 %，静脉给予阿托品 0.01 mg/kg。麻醉诱导：吸入七氟醚浓度为 6 %，氧流量为 6 L/min，氧浓度 80 %，七氟醚浓度逐渐降至 4%，氧流量调整为 4 L/min，氧浓度不变，患儿血氧饱和度 98%，行右侧桡动脉穿刺置管监测有创动脉压。麻醉维持：七氟醚吸入，根据情况给予芬太尼，新鲜气流量 2 L/min，氧浓度为 40 %，术中根据脉搏血氧饱和度情况进行适当调整，心率和血压值的波动尽量维持在基础值的 15 %范围内。通气采用压控模式，气道压力气胸前为 12 ~ 15 cmH_2O，呼吸频率 28 次/分，吸呼比 1:1.5，PEEP 5 cmH_2O，$P_{ET}CO_2$ 35 ~ 45 mmHg，CO_2 气胸流速为 2 L/min，压力≤ 6 mmHg，气胸后 $P_{ET}CO_2$ 升高，提高通气压力和呼吸频率，$P_{ET}CO_2$ 维持在 40 ~ 50 mmHg 之间。患儿术中血氧饱和

度维持在 95 %以上。镜下探查胸腔，左肺严重发育不良，可见胸腔内疝入脾、大部分小肠、回盲部及部分结肠，将腹腔脏器予以顺序复位，膈肌缺损约 4 cm×3 cm 大小，缝合修补膈肌缺损，放置胸腔闭式引流。手术顺利，术中各项生命体征平稳，历时 2 小时 15 min，术毕各项指标满意，患儿带管气管插管辅助通气安返 NICU，呼吸机呼吸频率 40 次/分，压力 12 cmH_2O，PEEP 2 cmH_2O，吸氧浓度 40 %，监测血氧饱和度 98 %，心率 145 次/分，血压 65/32 mmHg。患儿术后复查床旁胸部 X 片示左膈疝术后，脾和肠管还纳复位，见图 1。

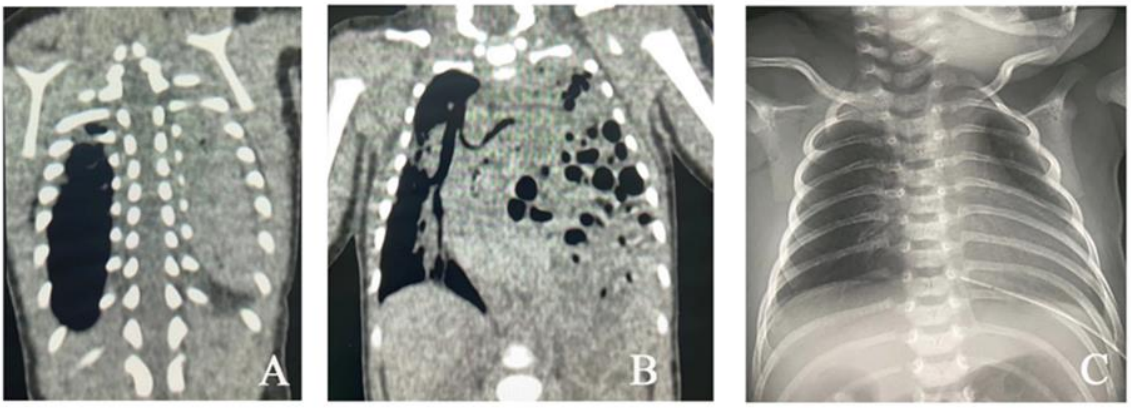

图 1. 患儿手术前后胸部影像学检查。术前：A 患儿 CT 冠状位重建纵隔窗显示脾疝入左侧胸腔；B 患儿 CT 冠状位重建纵隔窗显示肠管疝入左侧胸腔；术后：C 患儿床旁胸片显示左侧膈疝术后，脾和肠管还纳复位。

二、 讨论

先天性膈疝胸腔镜手术对新生儿呼吸循环影响均较大，围术期常见肺部并发症包括：低氧血症、高碳酸血症、缺氧性肺血管收缩受损、复张性肺水肿等，存在主要血管受损和大出血的风险[10]。本例患儿为低体重儿，整个脾、大部分小肠、回盲部和部分结肠疝入左侧胸腔，压迫左肺导致左肺不张，右肺部分不张并散在实变，且存在肺发育不良，在病房紧急行气管插管辅助通气，因此本例患儿麻醉风险很高，通过围术期恰当的处理麻醉手术经过顺利。现将本例患儿的麻醉管理体会总结如下：

1. 术前准备：

先天性膈疝患儿氧储备功能和对手术的耐受力极差，术中易出现呼吸循环功能紊乱及迷走神经反射致心跳骤停，连续监测为及时发现及抢救心跳骤停提供了可靠的依据[11]。麻醉前应充分给氧，应用阿托品可预防因刺激迷走神经所致的心动过缓，并且可明显减少唾液分泌。放置胃肠减压管，可以使胃肠内压力降低，减轻对肺脏的压迫，从而改善患儿的缺氧状况，同时减少反流误吸的发生。新生儿体温调节中枢发育不成熟，体温常随环境温度变化而变化，低体重儿更加明显，应注意保暖，可通过预先将手术室内温度调至 24 ~ 26 ℃，手术床上放置加温毯，输注

液加温等来预防低体温的发生。另外，应避免在下肢开通静脉通路，因为有可能由于下腔静脉受疝压迫，静脉回流受到限制而减少。

2. 麻醉诱导和维持：

本例患儿术前已在病房行气管插管呼吸机辅助通气，如果术前还没有建立机械通气，在预先吸入氧气后，可以选择清醒气管插管或快诱导气管插管。避免加压给氧，导致气体被挤入胃肠，进一步影响肺通气，使缺氧加重；还可能使原本萎陷的肺膨胀，使大血管和心脏受压加重，而发生心脏骤停。除了常规的监护设备外，可放置两个脉搏血氧仪（导管前和导管后）来监测分流程度。动脉导管前插管（右桡动脉）用于监测血压、血气、pH 值和其他血液参数。新生儿麻醉用药包括七氟醚、丙泊酚、肌松药或阿片类药物。因氧化亚氮能弥散入胸腔内的肠管，导致肠管扩张，使功能正常的肺组织进一步受压，应尽量避免应用。在动脉血氧合允许的情况下，在氧气内加入空气稀释氧气浓度，直到达到理想的氧气浓度。

胸腔镜修复新生儿先天性膈疝手术中麻醉气道管理非常重要[7, 8]。二氧化碳气腹可引起高碳酸血症和呼吸性酸中毒，Tytgat 等[12]研究发现当胸腔镜 CO_2 气胸压力为 5 mmHg 时，可引起导致血氧饱和度和 pH 值的可逆性降低和 $PaCO_2$ 的增加。可通调整通气压力和呼吸频率来改善，必要时间歇性放气来进行通气。术中机械通气期间应监测气道压力，保持气道压低于 25 ~ 30 cmH_2O，尽量减少气胸的危险。如果出现原因不明的肺顺应性下降、低氧血症或血压突然下降，表明可能发生了气胸。同时应必须避免低温，低温可引起肺血管阻力增加造成的右向左分流。低温也使耗氧量增加，这可能导致供氧不足及酸中毒，这又进一步增加肺血管收缩、加重血氧饱和度恶化。

手术过程中，麻醉医师和外科医师之间的及时、细致的沟通至关重要，尤其是在将疝入胸腔的脏器还纳回腹腔而引起继发性血流动力学改变时[1]。先天性膈疝新生儿还可能伴腹腔发育不良，过紧的腹部外科缝合增加腹内压，导致膈肌向头侧移位，功能残气量减少和下腔静脉受压，将脉搏血氧仪放于下肢，有助于及时发现腹腔间隔室综合征和循环系统影响。将疝入胸腔的腹腔内容物还纳入腹腔后，要小心膨肺，不要试图扩张发育不良的肺脏，因为肺脏扩张的可能性不太，而且气道正压过高，还会使健侧肺或发育相对正常的肺脏受损。

3. 术后管理：

新生儿先天性膈疝常有肺发育不良，脏器复位后萎陷的肺组织也不能立即膨胀，常需要呼吸机辅助通气呼吸治疗一段时间，因此术后管理也是一项重大挑战。新生儿常应用压力通气模式，以低压和最低吸入氧浓度进行通气支持。镇静和镇痛使患儿可以耐受插管和机械通气，亦有助于降低腹压。一些医疗中心使用硬膜外镇痛来减少阿片类药物引起的胸壁强直和呼吸抑制。

综上所述，低体重新生儿胸腔镜下膈疝修补术麻醉风险较高，全面的术前评估，个性化、细致的麻醉管理方案，持续保持警惕以尽早发现任何不良事件，以及麻醉医师和手术团队之间的良好沟通有助于手术平稳顺利的完成。

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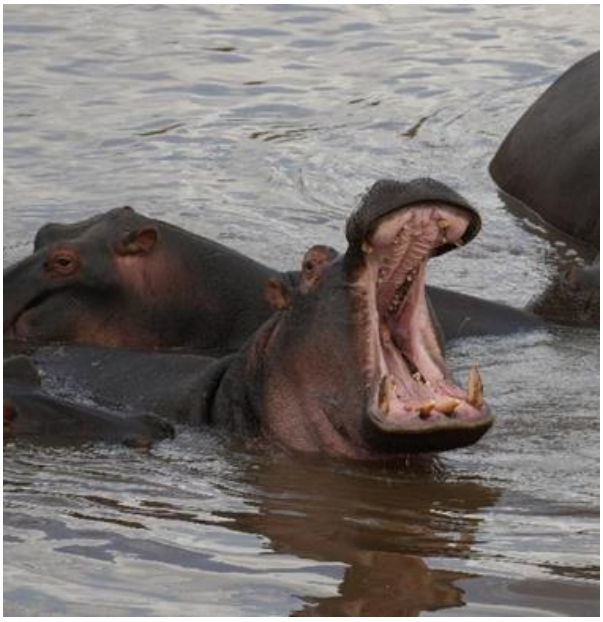


Henry Liu
(2023 CASA Photo Competition)

Hong Wang
(2023 CASA Photo Competition)

Ask the Expert

儿科麻醉-气道管理

CASA 的候任主席仲巍教授 主持并会议整理

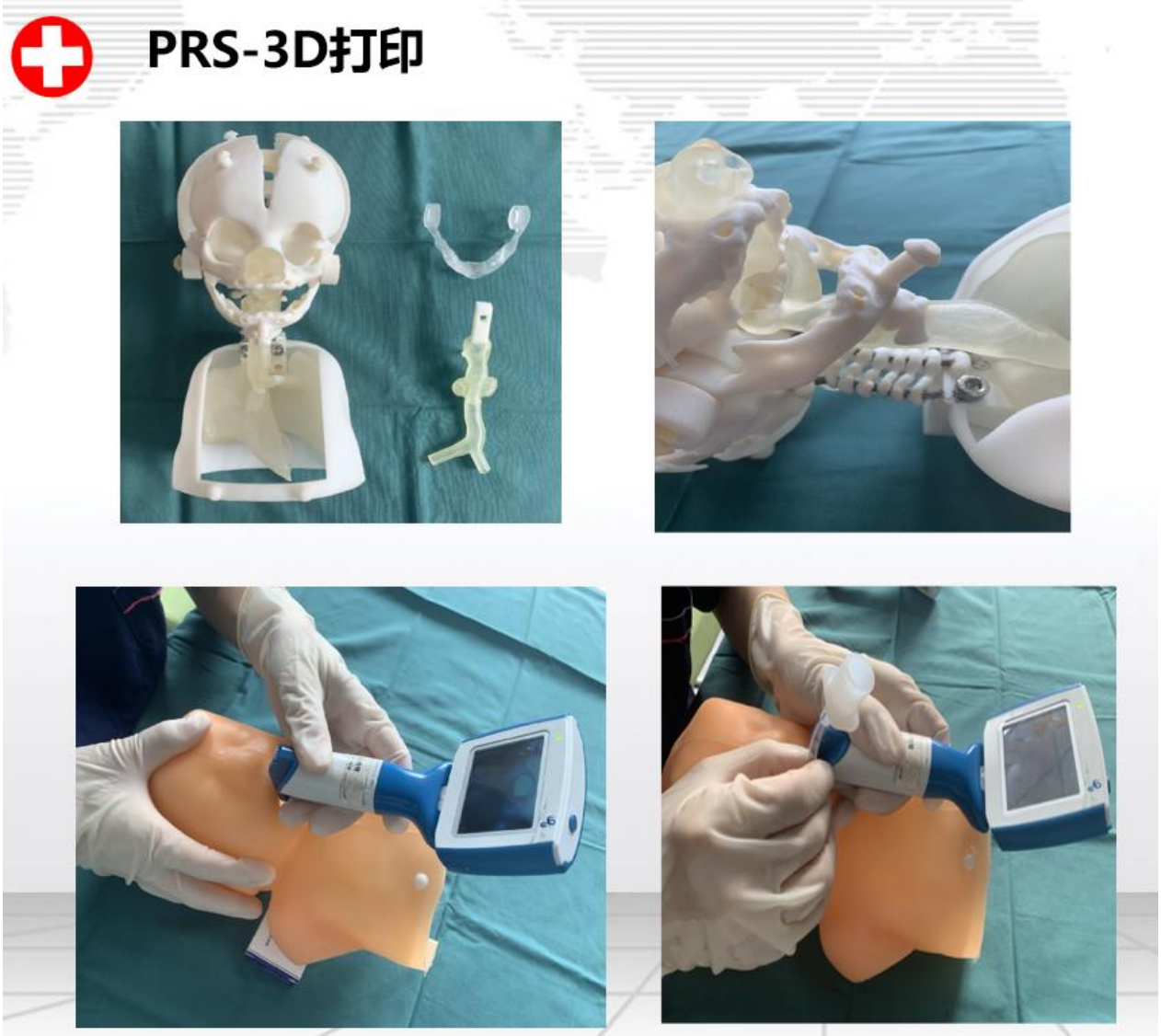
参与者
张莉主任, 南京儿童医院
徐颖主任, 重庆儿童医院
贾英萍主任，河南省儿童医院
梁小民教授, 印第安纳 Riley 儿童医院

今年 3 月 31 号适逢复活节， CASA 举办的<<询问专家系列>> 有幸请来南京儿童医院的张莉主任， 重庆儿童医院的徐颖主任， 河南省儿童医院贾英萍主任，印第安纳 Riley 儿童医院的梁小民教授来分享儿童困难气道管理的心得， 由 CASA 的候任主席仲巍教授主持。

首先南京儿童医院的张莉主任分享了他们对 Pierre Robin 儿童困难气道管理的经验。 Pierre Robin 综合征是一种以先天性颅面骨畸形行为特征的小下颌畸形-舌后缀-呼吸困难综合症， 发病率约 1/8，000-1/14，000。 出生后一个月内症状最严重，新生儿期即出现呼吸困难、喂养困难等急需处理的临床征象。他们麻醉管理的挑战在于气道管理，他们的口咽喉三轴线很难重合，暴露声门十分困难， 大大增加了插管的难度。然而由于插管时间的延长，反复试行插管而屡遭失败时往往加重原有的缺氧，可导致咽喉软组织创伤，较多血性分泌物而使视野模糊不清， 喉头出现创伤性水肿使声门暴露更加困难。水肿后气道阻力将成倍增加。

南京儿童医院曾收治四十名患儿，男婴 23 例，女婴 17 例，其中包括 9 例早产儿和 31 例足月儿。根据他们的临床表现、 体重和生长评估、呼吸困难评估、Cormack-Lehane 分级来综合评估。 综合评分在 6 到 9 分的患儿可以成功插管， 评分为 10 到 12 的患儿插管成功率仅为 47%，评分大于 13 分的患儿均无法成功插管。对于无法成功插管的患儿选择喉罩来管理困难气道。 为此他们针对性地制定了多项气道管理计划： 评分为 6 到 9 分的患儿可以由年轻麻醉医生尝试插管，麻醉诱导时可用肌松药更利于声门的暴露； 10 到 12 分的患儿由有经验的麻醉医生插管， 麻醉诱导可选择给肌松药控制呼吸或保留自主呼吸，等插管成功后再给肌松药； 评分大于 13 分的患儿由科里最有经验的麻醉医生来插管， 提前准备好纤支镜或可视软镜以及窒息氧合技术（ 经鼻湿化高流量氧气）， 尽量避免不能插管不能通气（CICV）的情况出现。

南京儿童医院张莉主任还与达拉斯儿童医院的仲巍教授、西安儿童医院的杨丽芳主任合作，分享了用薄层 CT 扫描通过三维打印重建 Pierre Robin 儿童困难气道， 用这种高保真的困难气道模型来训练年轻医生们的气道管理经验。



张莉总结到，Pierre Robin 患儿困难气道术前评估真的非常重要。 要根据评估结果提前制定相应的麻醉预案。临床气道管理的最高境界不是百战百胜而是防范于未然。敬畏气道才能安全处理气道。气道管理的核心是维持氧合! 患儿只会死于氧合失败而不会死于气管插管失败。

印第安纳 Riley 儿童医院的梁小民教授分享了他们对 Pierre Robin 患儿的气道管理经验。他们也会准备 CMAC, 可视喉镜, LMA。如果声门可见就直接插管, 如果不行的话用 LMA，将其当作一个可弯曲的 stylet, 用于引导插管。实在困难的话, 他们的耳鼻喉医生会来接手气道, 因为他们的纤维支气管镜技术更丰富, 而且需要建立外科气道的话也是他们来处理。他们好多年来只有一例外科建立气道； 他们用金属针环甲膜穿刺。 如果气道实在困难的话， 比如要四五个人才能建立起来， 他们的耳鼻喉医生会做气管切开术，并且根据下颌牵引器的牵引效果来重新评判气道困难的程度， 通过这个来决定何时来关闭气管切开。

重庆市儿童医院的徐颖主任分享了他们对 Pierre Robin 困难气道的处理。有时往往在急诊室的时候没有时间做很详细的气道评估，同时家长也在现场，所以他们先用 LMA 作为一个过渡，然后在手术室里再进行气管插管。 国内的纤维支气管镜直径较大， 有时候小的导管无法插上， 他们就用可视喉镜， 加上直

径较细的光棒， 建立气道。徐颖主任还分享了他们用高流量氧合保留自主呼吸完成了长达两小时以上喉裂手术。 而且他们有的耳鼻喉手术术前需要观察咽喉部位的动态状况，所以更需要保留自主呼吸才能反映病人的真正状况。

河南省的儿童医院的贾志平主任分享了他们的经验；他们有不少新生儿的咽部囊肿，声门下气道狭窄的手术， 他们用艾司氯胺酮和右美来保持自主呼吸，利多卡因雾化似乎效果不太理想。的确有时候国内小的纤维支气管镜，碰到 3.0 的气管导管无法使用， 有时碰到真正的困难气道，他们的经验是多学科联合会诊很重要，而且由经验最丰富的麻醉医生来负责建立气道，这样可以降低病人出现缺氧等并发症的机会。

仲巍教授分享了美国儿童麻醉协会里面的最早的困难气道兴趣小组衍生出来的儿童困难气道登记团队经验。他们从 2012 年形成之后在 2016 年《柳叶刀》学刊上发表了有里程碑意义的一篇文章，他们发现，从一千多例儿童困难气道的管理上总结经验，困难气道如果直接插管多于两次以上的话更容易出现并发症， 高达 20%，甚至出现心跳骤停需要心肺复苏来抢救。另外困难气道建立直接插管首成功的比例是 46%，纤维微支气管镜 28%， 可视喉镜 18%。因为达拉斯儿童医院就在这个儿童困难气道登记团队里面，所以近水楼台先得月，他们最新的研究结果还没发表或正在发表中我们就知道了初步结果：儿童困难气道登记团队最新的研究结果就是最近五年来的回顾， 与第一组一千多例的那组数据相比，现在是困难气道建立首次成功的比例提高了近 10%，而且反复插管由前五年的平均 2.7 次降到了现在的 2.2 次，跟南京儿童医院张莉主任分享的数据 2+1 相吻合。尽管两组数据的并发症发生率比较接近，最近五年是 19%，前五年是 20%，但是严重并发症表发生率后五年要好于前五年那组数据。仲巍教授还分享了他所在的达拉斯儿童医院最近一个 Pierre Robin 病例的紧急插管。

与会的各位主任、教授一致同意这种分享交流的方式特别好，希望今后能经常开展，选择一个儿科麻醉里面大家都感兴趣的话题来讨论，也可以用一个严重病例讨论的方式来进行。

Ask the Expert

产科麻醉-产程中血压及疼痛管理

记录总结：申建成, 李韵平，陆晓薇

CASA (Chinese American Society of Anesthesiology) 理事会联合 ICAA (International Chinese Academy of Anesthesiology) 和恒瑞医药于 2024 年 5 月 12 日成功举行了 ‘’Ask the experts: Obstetric Anesthesia" Zoom 线上研讨会。

CASA 主席李金蕾医生和 ICAA 主席夏云医生共同主持了研讨会， 由浙江医科大学陈新忠教授及哈佛医学院李韵平主任主讲。

陈新忠教授
浙江医科大学附属妇产科医院副院长
主任医师， 博士生导师
讲座题目：产科椎管内麻醉低血压的预防和治疗

李韵平
哈佛医学院麻醉副教授
产科麻醉冠名教授
哈佛碧芙以色列-迪肯尼斯医疗中心产科麻醉主任
讲座题目：产科麻醉进展：右美（Dexmedetomidine or Precedex) 的应用‘

夏云教授也向与会者介绍了北京首都医科大学附属妇产医院麻醉科主任，中国麻醉协会产科分会主委徐铭军教授。各位专家还就与会者提出的问题及产科麻醉的进展展开了讨论，进行了 SOAP 知识更新。陶为科和赵培山教授也参加了讨论。

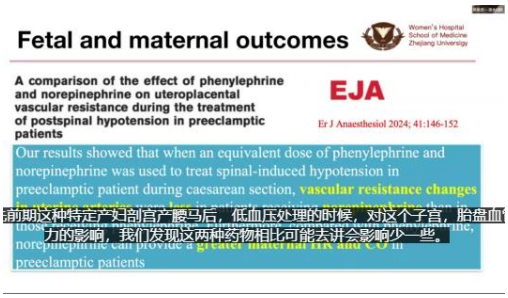
陈新忠教授
-产科椎管内麻醉低血压的预防和治疗：

1. 腰麻是剖宫产常见麻醉术式， 其造成的低血压发生率仍占高位。 低血压会给孕妇带来噁心，呕吐， 胎儿缺氧，酸中毒等。
2. 剖宫产腰麻造成的低血压处理：1）子宫左倾位；2）强调麻醉同时血液扩容(preload or co-load), 目前以 Co-load 更为提倡；3）血管活性药物应用；4）减少局麻药剂量及椎管内附加阿片类药物以减少低血压发生。
3. 治疗椎管内麻醉诱发的产妇低血压选择的血管活性药物（图 1），

I. 早期由于麻黄碱（ephedrine）的升压作用，被用作为剖宫产低血压的金标准。

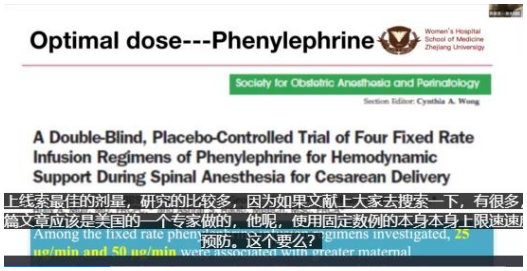
II. 之后，曾经的首选金标准的麻黄碱（ephedrine）过渡到了苯肾上腺素（phenylephrine ），在 2008 年的一项研究表明， 应用苯肾上腺素的产妇，婴儿脐带血 pH 值减低的机会更少， 而且呕吐较少，之后的一些研究也证实了这一点(图 2) 。 但苯肾上腺素可以造成产妇反射性心动过缓，从而可能引起心输出量减少， 基于这点，具有 a1 及 b1 作用的去甲肾上腺素(Norepinephrine)有了广泛的研究.

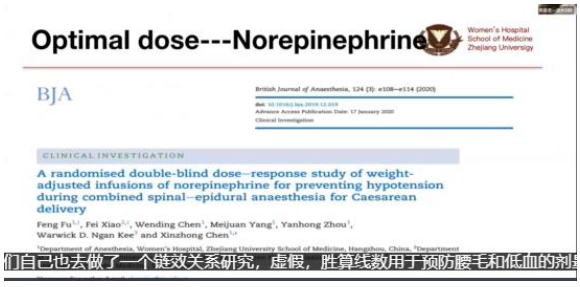
III. 有研究显示去甲肾上腺素(Norepinephrine)与苯肾上腺素对于控制腰麻水平作用相近， 但是去甲肾上腺素可更好的维持心率和心输出量，不易出现反射性心动过缓。 之后对 5 种血管活性药的研究也表明， 在对胎儿酸碱度的影响中，去甲肾上腺素的影响最低， 麻黄碱最高。(Slide3，4) 。更多的这方面的研究也在进行中。到目前为止去甲肾上腺素对产妇心率及对产妇腰麻后低血压的处理可能效果更好，但苯肾上腺素在临床中对于腰麻低血压的预防和治疗仍可作为一线药物使用。



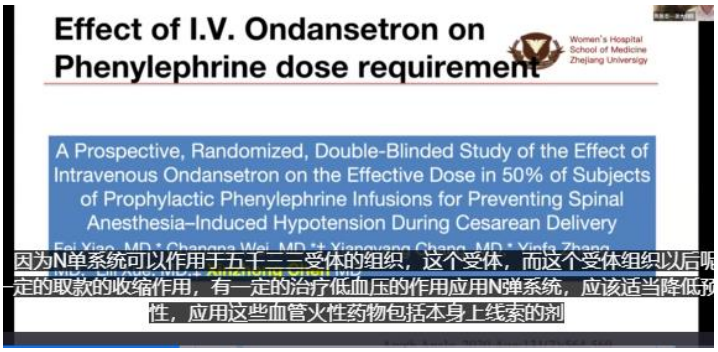
4. 血管活性药物的预防性应用因其早期干预低血压的发生故比补救应用更好（slides 5）

5. 在使用药物途径上，持续输注比单次给药更好， 特别是在预防性使用中。苯肾的预防持续输注可采用 25 到 50 微克/分钟，或 0.54 微克/公斤/分钟。用去甲肾上腺素预防低血压时，腰麻同时静脉给药的参考剂量是单次给药 6 微克，或连续输注 0.08 微克/公斤/分钟（slides 6，7，8）。





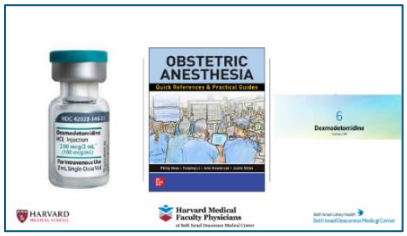
6. 预防应用血管活性升压药物时，腰麻中使用的局麻药的剂量可能要增加大约 20%。
7. 若病人应用了抗噁心的 Ondansetron，由于 5 羟色胺受体阻滞作用产生的对心脏血管的不同作用，预防应用血管活性药物剂量要减少大约 26%。



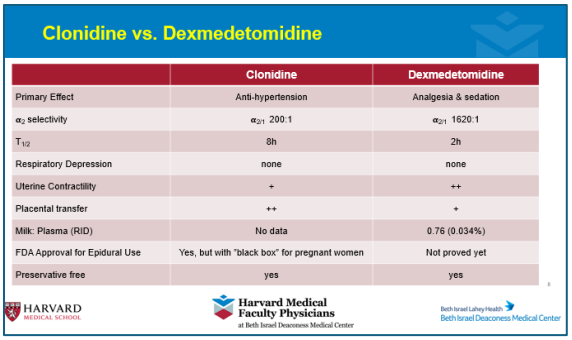
李韵平教授

-右美（Dexmedetomidine/Precedex）在产科分娩硬膜外麻醉中的使用

1. 硬膜外麻醉在产妇分娩的镇痛过程中起很大作用，但是其分娩镇痛的失败率约占 10-20%. 在李韵平教授最近出版的妇科麻醉一书中， 有专门的章节讨论右美在产科镇痛的相关知识。



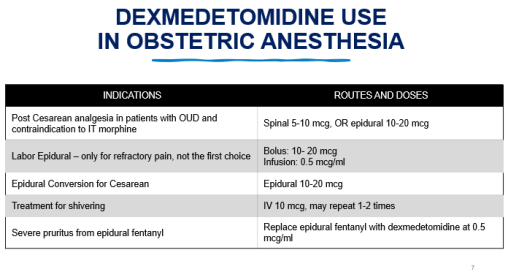
2. 右美是 Alpha2 激活剂，和 Clonidine 一样，作用于节前神经元，主要用于镇痛和镇静，比较于 clonidine, 其 Alpha2 受体的选择性比 Clonidine 大 8 倍， 血液半衰期两小时，没有呼吸抑制作用，通过胎盘的能力低于 Clonidine， 对胎儿影响较小。 右美在乳液中的分泌剂量很低， 故哺乳是安全的。



3. 右美的产科椎管内使用尚未得到 FDA 批准。目前的临床研究表明，右美的 off label 产科应用应该是安全和有效的。美国现有制剂不含防腐剂。

4. 右美不做为术后镇痛腰麻局麻药添加的首选，仅在鞘内吗啡有禁忌（例如病人有严重的瘙痒，或者吗啡成瘾的病人术后需要使用阿片类药物的 PCA）的情况下使用。腰麻时可添加 5 -10 微克，硬膜外添加 10-20 微克。

5. 右美不做为分娩镇痛的首选。临床的应用经验：
   a. 若发生了其他药物治疗失败的突破性疼痛，在硬膜外管追加局麻药及阿片类药两次后可使用。
   b. 分娩镇痛时，若硬膜外阿片类药引起了严重骚痒，可考虑停止使用阿片类药物，添加右美。可硬膜外给药 10-20 微克，或按 0.5 微克/毫升浓度随硬膜外局麻药持续泵入。



6. 中转剖宫产：对于分娩镇痛不理想的患者，可考虑硬膜外推注加量的局麻药同时加入 10-20 微克右美增加镇痛效果， 起效时间大约 10-15 分钟。

7. 右美可治疗术后寒战，可静脉给药 10 微克，一两分钟后可重复给药一次。

8. 需要强调的是，在稀释右美药物时， 需使用没有防腐剂的生理盐水。

讨论话题：

1. 右美的剂量是多少才可以帮助子宫收缩?
   a) -目前的研究限于体外实验。临床上尚未定论。但因临床使用剂量极小， 故不会引起胎心减慢或宫缩乏力。其镇静和助眠作用会帮助产妇恢复体力。（李韵平）

b) -右美对子宫平滑肌张力具有增加作用。产程初期，胎心会因宫腔压力增加而降低，故此情境下不建议使用右美。建议在宫缩不强及硬膜外镇痛不佳的情况下使用。（陈新忠）

2. 给与去氧肾上腺素（苯肾上腺素）会导致 CO 降低， BP 升高. 请问 CO 及 BP 与胎盘血流灌注的关系以及用神魔方法来检测胎盘血流灌注?
-子宫胎盘血流与 CO 无直接关联， 但与体循环压关系密切。 超声是目前最好的监测胎盘血流灌注的工具。去氧肾上腺素是外周血管阻力增加后血压上升，反射性 CO 抑制。（陈新忠）

3. 使用右美时产妇心率下降有什么好的治疗方法?
-临床使用右美的剂量极低，通常为 5-10 微克。很少导致产妇心率下降。（李韵平）

4. 无痛分娩可以做腰麻加 Nabuphine 吗?
-临床使用新药，第一要无防腐剂。第二要有大量的临床实验已证实其安全性。第三如果有纯的激动剂或拮抗剂， 我不会去选择混合效应的药物。因为混合效应药物含有激动剂和拮抗剂，会对之后的疼痛治疗带来困难。对于有阿片类药物成瘾的患者，会造成戒断症状（withdrawal syndrome）。（李韵平）

5. 右美和呼吸的关系是如何?
-右美对呼吸没有负面影响。可以用于肥胖及困难气道病人的插管用药（清醒插管）。（李韵平）

6. 和吗啡相比，右美在蛛网膜下腔注射时的持续时间?
-右美在蛛网膜下腔注射其镇痛作用持续 6 小时，通常不会超过吗啡的持续时间。我们的研究显示，给予 250 微克的吗啡，病人在 72 小时内不需要任何其他镇痛药，这是右美不能达到的。鞘内吗啡有封顶作用。当用量超过 300 微克时，镇痛效果不会增加，但其副作用会增加。（李韵平）

7. 腰麻和硬膜外联合给与，但麻醉效果不佳，如何处理?
-若麻醉平面不够，应先增加平面。 若是镇痛效果不佳，应增加药物浓度来增加阻断效果。（夏云）
-硬膜外平面应达到胸 5，且在明确导管在硬膜外腔时，推药速度可相对快些，压力可高些。（陈新忠）
-5-10%的产妇，会出现置管不佳，需要重新置管。药物浓度及阿片类药物使用要到位。（陶为科）

8. 血管加压药物对子宫收缩有无影响?
-通过对药物的药理特性，可以推导出有 Alpha1 受体的药物，可能会对子宫平滑肌的收缩有一定的作用。而具有 beta 受体的药物，会是子宫平滑肌张力下降。临床上会使用 beta 受体兴奋剂来解决子宫张力过高的状态。比如肾上腺素会使子宫张力下降， 而去甲肾上腺素致子宫张力升高。（陈新忠）
9. 请问对 DPE (Dual Puncture Epidural) 的看法?
-1）DPE 最好的作用是确定中线置管。 当有脑脊液流出，可证明是中线置管。而无脑脊液流出

时，DPE 有 25%失败率，源于单侧置管或 paravertebral 置管。2）DPE 的镇痛效果好坏或 onset time 决定于有多少药物渗透到蛛网膜下腔。刘志强教授的研究表明， PCEA（Patient-controlled epidural analgesia）不理想时，PIEB （ programmed intermittent epidural bolus）会有较好的结果，是因为在脉冲的效果下，会有极少数药物渗透到蛛网膜下腔。这种镇痛效果可以有帮助，但不很显著。（李韵平）

-DPE 对于脊柱侧弯，肥胖患者，剖宫产后的自产急诊手术，有很大帮助。探出脑脊液是成功率的关键。 No CSF, No go. （陶为科）

10. 置管后 12 小时后产生单侧镇痛效果不佳，如果考虑是因为胎位不正，为何要重新 CSE（combined spinal Epidural）置管?

-放置导管后 12 小时内镇痛均好，12 小时候后出现不佳，应考虑：

I. 1)导管设置位置正确。2) 药物耐药性（tachyphylaxis）, 因药物种类不同而出现时间不同。最早出现药物耐药性的药物是 Lidocaine. Fentanyl 也会产生同样的问题。3）胎位不正。3）重新置管（CSE）是考虑利用腰麻的作用使产妇得到短暂的疼痛缓解，打破恶性循环。4）而且可以考虑加入鞘内吗啡。（李韵平）

II. 骶尾部在第二产程镇痛比较困难，而多数产科医生对盆腔神经阻滞并无经验。 此时麻醉医生可考虑在检测到位的情况下大剂量推药，20-40ml.，或重新置管。（陶为科）

III. 因为头盆不正产妇所经历的疼痛会比头盆正位者要重，因此考虑加大药物剂量为先。（陈新忠）

IV. 考虑胎位不正不是主要原因，而是椎管镇痛有问题。补充：DPE 和硬脊膜孔的大小，硬膜外给药的浓度和速率都相关。（徐铭军）

11. 闭环血管升压系统目前的发展状况如何?

闭环血管升压系统要求精准度要高，而血压监测是一个连续及动态的过程。 二者的结合还需要有大量的数据来支撑。

12. 国外对于产妇镇痛开始的时间是怎样？需要考虑宫缩的因素吗?

1) ACO 指南是只要母亲有需求，而且医生可以安全地实行分娩镇痛，是可以考虑。我们医院是如果产妇有要求，这就是她分娩镇痛的开始。 少数产妇有宫缩，但未进入产程，子宫也未打开，只表现在 latent phase, 此时我不主张做分娩镇痛。在我们医院，可以采取 Morphine Sleep, 即给与静脉内阿片类药物：吗啡 10mg 静脉+10mg 肌肉注射。

2) 另一种情况是当宫口未开，机械性用水囊把子宫口撑开时，产妇不能耐受宫口扩开的疼痛时，可以展开分娩镇痛或是静脉给药。静脉给药只能是在 latent phase, 而不能在 active phase, 引起会影响胎儿呼吸。（李韵平）

补充讨论:

徐铭军： 腰硬联合的经验是 1）用脑脊液稀释药物。2）推注时反复推进和抽回，造成湍流现象，使局

麻药分布更广。3) 在 L2-3 穿刺，更易获得脑脊液，镇痛成功率更高。

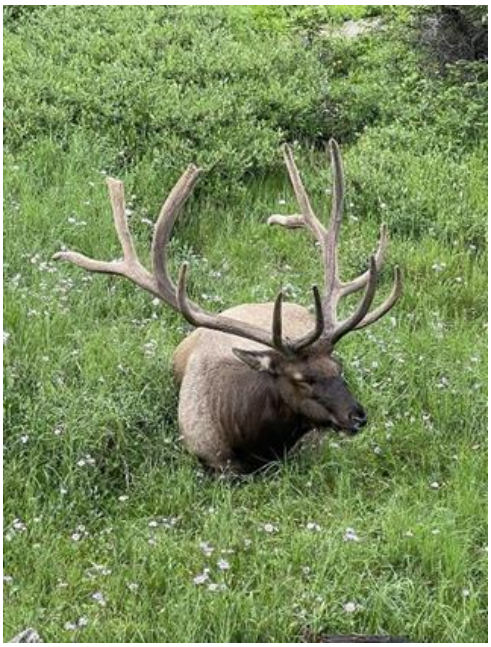
李韵平：右美在产妇硬膜外使用是 FDA 未批准的。需根据临床来做判断。对于 off-label 的药物使用，首先要做到对病人 no-harm., 参考回顾性研究。

赵培山：1）单侧硬膜外阻滞不佳，仍考虑为置管问题。2）DPE 的使用并不确定。若出现无脑脊液，但 loss of resistance positive 时，不易做决断，故我不建议我的学生常规去做 DPE。3）腰麻时有脑脊液回流，且给予常规剂量，但腰麻却工作不佳。建议应在等待 10-15 分钟后术前先测定平面。若无应达到的平面，我会再做二次腰麻，用第一次一半的剂量，保证腰麻平面。

李韵平：腰麻失败的原因：1）麻醉医生技术问题-腰麻针尖位于侧位，进出时脑脊液不确定。 2）病人解剖结构异常-Tarlov Cyst 致脑脊液容量增加，需要剂量增加。3）如只使用局麻药物而未加入阿片类药物（fentanyl or sufentanyl），镇痛的深度不及加入阿片类效果好。对于 Visceral Pain，只有用阿片类药物才可以控制。

赵培山：对于 L2-L3 进针，因部分病人的脊髓是终止于 L2，应加强和病人的互动，在病人不适时不要贸然给药。

徐铭军：1）腰麻成功决定于药物剂量及推送速度。L2-3 给药会帮助药物向上扩散。2）腰硬联合麻醉时，我会在硬膜外突破后，让针再向前移动一点点，方便置管，因为腰部硬膜外间隙可达到 5-6mm，危险降低。3）腰穿针头从针尖至侧孔约有 2mm 距离，故见到脑脊液时应向前在推动一些，以确保腰穿针的侧孔全部进入蛛网膜下腔，给药时才不会导致部分药物进入蛛网膜下腔，而部分药物进入硬膜外腔而致腰麻失败。



John Zhong
(2023 CASA Photo Competition)

Xiqing Cao
(2023 CASA Photo Competition)

ASA News

曲歌医生整理

April 17, 2024

Deeper Sedation with Propofol May Help Find Difficult-to-Detect Polyps During Colonoscopy

In patients undergoing colonoscopy to screen for colorectal cancer, deeper sedation using propofol may improve detection of "serrated" polyps — a type of precancerous lesion that can be difficult to detect, reports a study in the Online First edition of Anesthesiology (Quaye, et al. Association between Colonoscopy Sedation Type and Polyp Detection: A Registry-based Cohort Study. *Anesthesiology*. April 17, 2024.).

The study provides the first evidence that compared with moderate sedation or “conscious sedation”, monitored anesthesia care with propofol might increase detection of serrated polyps, which are harder to see during colonoscopy because they are often flatter and blend into the folds of the colon tissue.

The analysis included detailed information on more than 54,000 completed colonoscopies drawn from the New Hampshire Colonoscopy Registry between 2015 and 2020; The overall polyp detection rate was higher when colonoscopy was performed using propofol: 34%, compared to 24.5% with moderate sedation. The results were similar on analysis of another sample of about 19,000 colonoscopies: Propofol was associated with a clinically and statistically significant 13% higher likelihood of serrated polyp detection.

It is thought that propofol might improve detection of serrated polyps due to better patient comfort and relaxation and optimized smooth muscle relaxation in colon. Further studies are needed to clarify the possible advantages, and the potential to approach further optimizing the use of colonoscopy for early detection and prevention of colorectal cancer.

March 29, 2024

Certified Anesthesiologist Assistants (CAAs) Authorized to Practice in Washington State

SEATTLE – Washington state Gov. Jay Inslee signed into law Senate Bill 5184, which authorizes certified anesthesiologist assistant (CAA) licensure in the state. The law, which

is the first to introduce the role of CAAs to the Pacific Northwest, will be effective in June 2024.

Today, Washington joins over 20 states that allow both types of nonphysician anesthetists (nurse anesthetists and CAAs) to practice in the physician-led Anesthesia Care Team. The Washington State Society of Anesthesiologists (WSSA) and the American Society of Anesthesiologists (ASA) applaud this action, which will make CAA services available to Washington patients. This Law aims to improve health care workforce shortage in Washington State.

CASA News

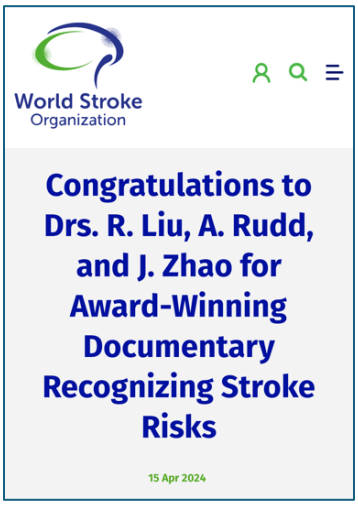
蒋天宇医生整理

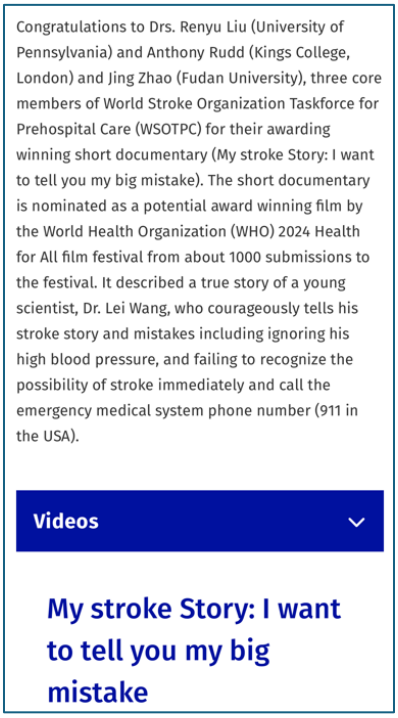
Congratulations!

Congratulations to Dr. Jiapeng Huang for becoming chair of the ASA Committee on Innovation for the 2025 governance year

Congratulations to #HMFP's Yunping Li, MD, MSc, Director of Obstetric Anesthesia at Beth lsrael Deaconess Medical Center and Associate Professor of Anaesthesia at Harvard Medical School, who was recently named the inaugural incumbent of the HMFP Nancy E. Oriol Chair in Obstetric Anaesthesia.

Congratulations to Dr. Renyu Liu

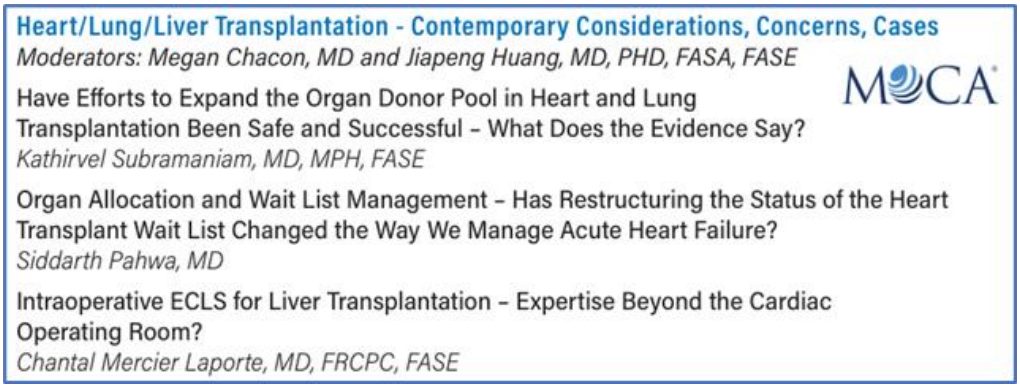




Meeting Information

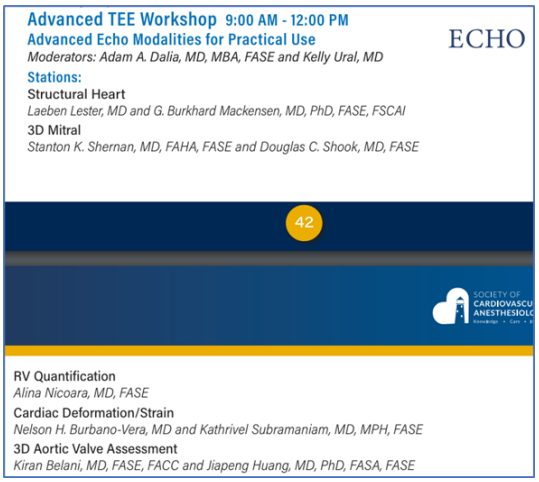
SCA 2024 at Toronto

Dr. Jiapeng Huang



SOAP International symposium was held on 05/01/2024


International Symposium: China



Articles and book publishing

刘仁玉：Non-scheduled short-acting opioid to taper off opioids?


CNS Neuroscience & Therapeutics / Volume 30, Issue 4 / e14705

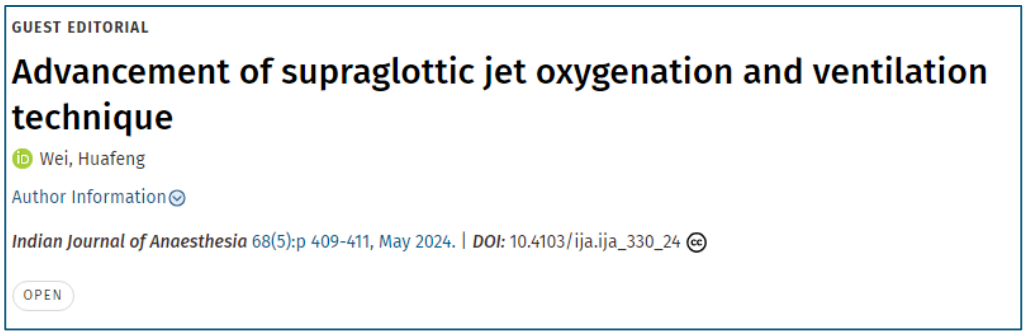
EDITORIAL COMMENTARY | Open Access

Non-scheduled short-acting opioid to taper off opioids?

Renyu Liu, Bonnie Milas, John Grothusen

First published: 07 April 2024
https://doi.org/10.1111/cns.14705

Huafeng Wei: Advancement of supraglottic jet oxygenation and ventilation technique


GUEST EDITORIAL

Advancement of supraglottic jet oxygenation and ventilation technique

Wei, Huafeng

Author Information

Indian Journal of Anaesthesia 68(5):p 409-411, May 2024. | *DOI:* 10.4103/ija.ija_330_24

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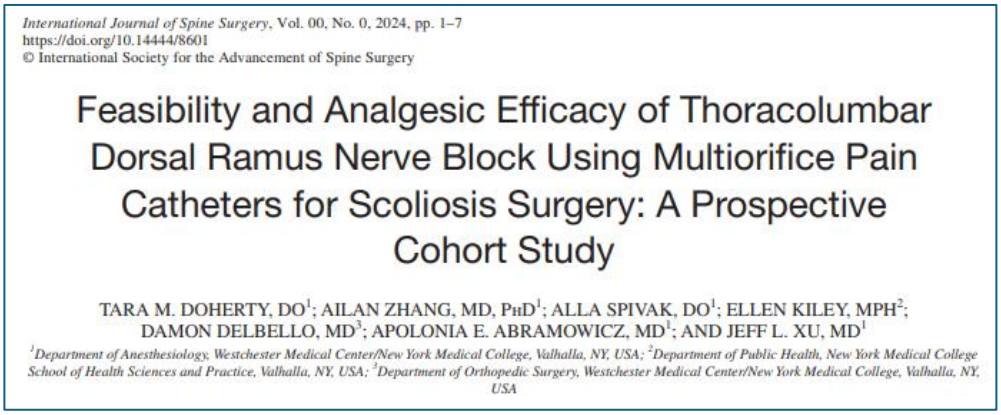
Jeff Xu：Feasibility and Analgesic Efficacy of Thoracolumbar Dorsal Ramus Nerve Block Using Multiorifice Pain Catheters for Scoliosis Surgery: A Prospective Cohort Study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Feasibility and Analgesic Efficacy of Thoracolumbar Dorsal Ramus Nerve Block Using Multiorifice Pain Catheters for Scoliosis Surgery: A Prospective Cohort Study

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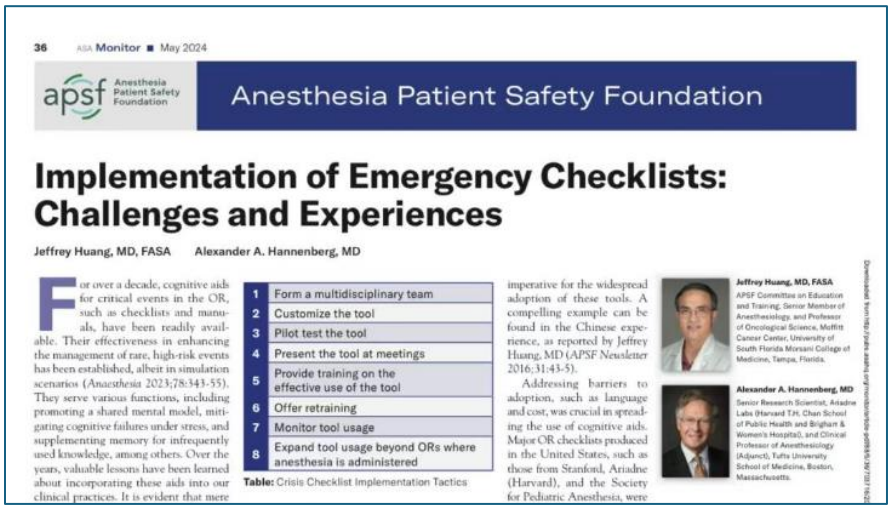

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Implementation of Emergency Checklists: Challenges and Experiences

Jeffrey Huang, MD, FASA Alexander A. Hannenberg, MD

For over a decade, cognitive aids for critical events in the OR, such as checklists and manuals, have been readily available. Their effectiveness in enhancing the management of rare, high-risk events has been established, albeit in simulation scenarios (*Anaesthesia* 2023;78:343-55). They serve various functions, including promoting a shared mental model, mitigating cognitive failures under stress, and supplementing memory for infrequently used knowledge, among others. Over the years, valuable lessons have been learned about incorporating these aids into our clinical practices. It is evident that mere

1	Form a multidisciplinary team
2	Customize the tool
3	Pilot test the tool
4	Present the tool at meetings
5	Provide training on the effective use of the tool
6	Offer retraining
7	Monitor tool usage
8	Expand tool usage beyond ORs where anesthesia is administered

Table: Crisis Checklist Implementation Tactics

imperative for the widespread adoption of these tools. A compelling example can be found in the Chinese experience, as reported by Jeffrey Huang, MD (APSF Newsletter 2016;31:43-5).

Addressing barriers to adoption, such as language and cost, was crucial in spreading the use of cognitive aids. Major OR checklists produced in the United States, such as those from Stanford, Ariadne (Harvard), and the Society for Pediatric Anesthesia, were

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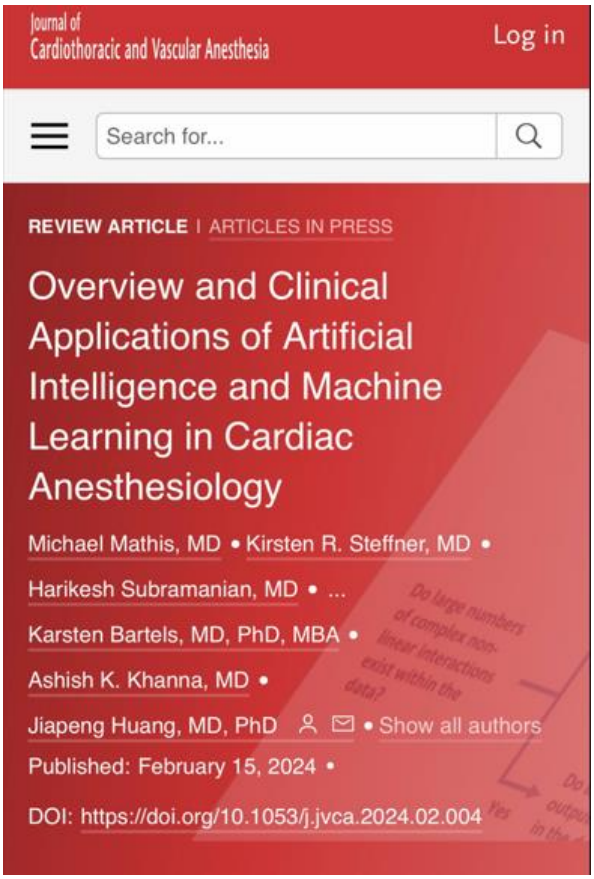
Jeffrey Huang: Implementation of Emergency Checklists: Challenges and Experiences

Jiapeng Huang Overview and Clinical Applications of Artificial Intelligence and Machine Learning in Cardiac Anesthesiology



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Overview and Clinical Applications of Artificial Intelligence and Machine Learning in Cardiac Anesthesiology

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Congratulations!

To 2024 medical graduate matched to anesthesia program

Yu-Hsin Ting Matched to Beth Israel Deaconess Medical Center + MetroWest Medical Center (TY)

Harvard T.H. Chan School of Public Health 2024
School of Medicine, National Yang Ming Chiao Tung University 2023

Growing up in Taiwan, I had a chance to move to Boston when I was in middle school and this experience has inspired me to pursue a career here in the U.S. I developed an interest in anesthesia during my clinical rotations and found myself enjoying the team dynamic and intellectually stimulating working environment.

Outside of medicine, music has been an intrinsic part of my life. I am also a big fan of all kinds of water sports, including swimming and diving. When I am not working, I enjoy cooking, running, hiking, and exploring the city. I greatly appreciate the warm and welcoming CASA community and am grateful for the support and encouragement from our incredible mentors. I am excited to share my inspiration and support with the CASA family and with future members who come after us.

Yuli Lee Matched to Case Western Reserve University- MetroHealth

Shanghai Jiao Tong University School of Medicine, Class of 2017

I grew up in Taiwan, surrounded by mountains and the tranquil yet ever-changing sea. I love to travel around exploring the world or take short trips to neighboring hiking trails. Additionally, as a big foodie, simply going out with friends and uncovering hidden culinary gems after a long day of work brightens my day. Not to mention sharing my never-ending list of must-try restaurants!

I am deeply grateful for the support and guidance I received from CASA. This warm community has helped me navigate the challenging yet rewarding path to matching into a residency. It is also my heartfelt aspiration to pay forward this kindness and assist future IMGs in their journey towards matching.

Yinqian Kang Matched to University of Iowa Hospitals and Clinics

Zhongshan School of Medicine, Sun Yat-Sen University, Class of 2021

Growing up in Guangzhou, I had the chance to explore my love for anesthesia during residency training in China for nearly two years after graduation. The experience deepened my passion for anesthesiology and the fulfilling lifestyle it offers.

Beyond the operating room, you'll find me hiking trails, hitting the gym, and experimenting in the kitchen – “a chef in the making”! This year, I feel incredibly fortunate to have successfully matched into the categorical track for anesthesia. Along the journey, I've been blessed with incredible support and encouragement from mentors of CASA. Joining the CASA family, I'm excited to pay it forward, fostering a culture of growth and camaraderie.

林熙， MD
Neurologist in Maryland

医生生活

加州有感

陶青 5/6/2024

岁月如梭，光阴似箭。十五年前的春假，与儿子 从夏威夷返回，在旧金山小住，顺便看看著名的斯坦佛大学及加州伯克利大学。那时儿子 12 岁，未上高中。参观伯克利校园时，我让他靠前一点，听得清楚些。他不耐烦地回答：“我永远不上伯克利，只上马里兰大学，只上马大！只上马大” 。当时在一群家长中间，我有苦难言，翻翻白眼作罢。

没想到从此与伯克利结缘。女儿夏天来加州大学旧金山分校读博士学位，儿子高中毕业后进了伯克利大学读本科。大概他早已忘记了当年“只上马里兰大学”的概念。女儿从加州大学旧金山分校博士毕业后，进入伯克利做博士后。两人很快以加州人自居。五年前，儿子与女友 在伯克利校园偶遇，相恋。如今女友也被伯克利录取，今年秋季入学，主攻公共政策研究硕士学位。女儿也几乎完成了博士后的研究。

曾几何时，两个孩子提到他们在加州的住所为“我的家”时，我心里愤愤不平，曾数次矫正他们，那不是家，只是你的宿舍，你的家在马里兰。如今的我，坦然接受新的“家”的慨念。大大方方的在女儿狭小“家“中作客，吃她做的炸酱面，味道甚佳。在儿子的“家”中做烧烤，坦然享用儿子的女朋友做的韩国菜肴。

最近，我买了一个佛罗里达州的小房子，焦虑中的我的正在 down size(退休后，大房子换小房子）。无奈中，卖家俱，卖钢琴，继而准备卖房，准备搬迁。劳心而费神，烦恼且痛心！这是怎么了？不曾忘记搬到这大房子的激动与振奋，不明不白为什么搬小房就这么令人沮丧，真难啊！终于明白了，没什么大不了的，不是谁的过错，只是此刻已是二十年之后，孩子们长大成人，有自己的工作与生活，儿子 和女友已经到了谈婚论嫁的时候，而我已退休，换言之，到了放飞自我的年龄，小小放纵任性吧。多年来喜欢佛州的白沙滩，棕榈树，是时候了。孩子们有各自的生活方式，我则与我的同龄朋友们一起分享属于自己的生活吧。

此次加州之行，当着儿子女友的面，重提当年儿子恼怒坚持只上马里兰大学的糗事，儿子反而惊呀不已！当时给了我巨大的头痛，人家反而早就忘光光啦！唉！这就是身为父母的独特感受，酸甜苦辣，样样不缺！看到孩子们准备的母亲节卡和蛋糕，感受尤其深刻，那小小的奶酪蛋糕，竟然如此香甜适口！因为这是三十余年的耕耘才得到的！

快乐的时光就这样在嘻笑中度过。 近距离观察孩子们的生活，愈发坚定我今后的信念，照顾好自己的身体，远距离与孩子们互动，尊重彼此的生活方式，需要时互相帮助。孩子们需要自己的生活与发展空间，而我也需要圆儿时的梦想，这就是我的未来，夕阳西下的无限憧憬与遐想。虽然近黄昏，但夕阳余晖仍无限的美好。

王海明回忆录 （续 5）

7. 毕业之后何去从 反复斟酌选未来

1993 年，克林顿总统夫人- 希拉里, 要医疗改革！ 一语惊医界！ 全美国，几乎各个科室，人们忐忑不安，纷纷冻结人员流动。麻醉界亦同样。许多麻醉住院医师毕业后，找不到工作。于是，骤然麻醉又成冷门。一些本已 matched 到麻醉的医师们惊慌中跳槽了。一位北医同学，已获哈佛 Brigham and Women’s Hospital 录取，他却改到内科去了。数年后，麻醉专业又火了，他后悔不已!

那年，我不急，去做 Fellow 深造。Fellow 中，我通过了 Board （专科执照 ）。这时，我杰出了！ 我既有专科执照，又有 Pain Management Fellowship！ 许多人找不到工作，而我得到三所医院聘请：1. NIH 疼痛临床研究中心。该院从美国各地筛选患者，提供机票，甚至免费诊治。主要研究疼痛发病机理，药物疗效等。要求我全职研究疼痛。我犹豫不定，因为我很喜欢各类麻醉和手术室工作，实不愿放弃麻醉。2. 田纳西州立大学医学院麻醉科主任约翰教授聘我为助理教授、疼痛诊疗中心主任、Tenure Track (有望成为终身教授 ）。我去面试感觉好；约翰主任又寄来二张往返免费机票，请我和丽再去。我被约翰教授感动！ 可惜田纳西州立牙科学院暂无教位给丽，我俩很失望 。3. 纽约上州 IBM 基地。一小城（Ｋ城 ）医院高薪聘请我一边麻醉，一边开辟疼痛诊疗服务。这正合我意。况且，此地交通便利，开车三个半小时可达多数常青藤大学！ 为了孩子的未来，我们选择了第三!

离开波士顿时依依不舍，波士顿是大学城，到处可见青年学生们，整个城市充满活力！ 我对丽说：将来我们会因为两个理由会重返波士顿：1. 孩子们可能来此读大学、医学院或牙科学院；2. 我们老了就在哈佛医学院附近买套公寓，诊治疾病方便。

如今回头看，对我们当初 （1994 年）的选择无怨无悔。虽然，我们放弃了大学教学和科研，可我们将自己锤炼成好医生。刚到此城，我的纽约行医执照（各州还有自己州的医学执照）还差 2 周未好。我们小家一起开车向东去了耶鲁；向北去了奥博尼（纽约州州府）；向西南去了普林斯顿；向南去了纽约市。参观了哥伦比亚大学、康奈尔大学医学院、世贸大楼双高塔。在Ｋ 城好区，很快买了一幢好房子，二英顷地，屋前有花园(翠竹林），房后有游泳池和标准的网球场。耶鲁大学赵浩生教授和夫人今泉智慧来访。他们喜欢我们屋前的翠绿竹林，说：“ 宁可食无肉，不可居无竹！ ”孩子们进了本地最好的学校。我努力帮丽找工作。几日后，医院对门牙医对我说：有一牙医突患脑瘤需手术，恳请张丽医师去顶栋梁。二年后，那牙医的听神经瘤治愈，回来工作了。丽便自己开业了。据说，600 病人可养一牙医和全家。数年前，丽牙科诊所的病历已过 5000（五千）。我总是骄傲地对朋友们说：Lily 是从纽约市到蒙特利尔（加拿大）之间最好的牙医之一！ 她对患者和蔼可亲，技术精益求精，设备总是最新最好，男女老少皆爱戴她！

美玲和美慧两个孩子均喜爱上学，均是年级好学生！ 后来，二人均毕业于藤校：哈佛、斯坦福、宾夕法尼亚大学，我们是多么幸运！

过去，曾有人赞我有先见之明：以最快速度进入美国临床工作 。而他（她）们先去读硕士和博士，耽误好几年 。我总说：非也！

我很羡慕那些读了 PhD 又进临床者。我至今仍喜欢阅读，没有读够书。

任何留学生或新移民倘若不为签证所限， 不为经济拮据所迫，而能随心所欲地选择自己有强烈兴趣的领域，从事深造和科学研究最好。

1987 年在曼哈顿，邱卫乔曾感慨地对我说：做科研是奢侈的享受。

我当初择工作时，的确反复论证过：1. 从教学科研单位可转去临床工作，可半临床、半科研、或只是集中精力做好医生（几无科研）。2. 先去临床工作（无科研），倘若不喜欢（痛失科研）还有机会返回科研教学中心，虽然不易，仍可行。那时，我巧遇 Dr. DeAmandi, 完成住院医后，他去了 Private Practice，不满意，换了一个组仍不满意。十年后回到麻省总院（ MGH ）一边工作（as Instructor）、一边读 MS （科研硕士、学习科研方法）。周末，则到周围私立医院加班 （ moonlighting) 多攒些钱，因为麻省总院的薪金太低 （麻省总医院从不缺人手，许多人带薪、甚至自费来学习科研，希望出成果，发表论文）。当然，他虽已婚，却无孩子，经济压力不大。在 MGH 努力几年后，他去一中西部大学医学院任麻醉科教授和主任。受他恳请，我曾介绍他去北京讲学。

Dr. Benjamin Covino 的故事可谓 传奇！ 他毕业于 Worcester Holy Cross College，去 Boston College 读一硕士，又到波士顿大学医学院（BU medical School) 获生理 PhD， 再去 University of Buffalo 获 MD！ 他研究局麻药和局麻药对心肌的作用。中年后，他去麻省总院麻醉科，入住院医师规培学习临床麻醉。他享特殊待遇：不值夜班。二年后一毕业，就到 Brigham and Women’s 医院（哈佛附属医院）麻醉科当主任和教授。他在 Boston 哈佛附属医院工作，却一直住在 Shrewsbury, Mass. (那是一个幽静、美丽的小城，陈立瑞老师和戴先生亦居于此，临近 UMass (麻省大学医学院），在波士顿城西三十余英里。Covino 教授 1991 年因心梗猝逝。我在麻醉会议上，曾有幸见过他几次。

不可否认，当初我确有先获得 financial security 的迫切愿望。2003 年后，我回中国各地讲学。在北京，曾因明教授请我去徐州医学院麻醉系交流，我抱歉地说：此次来京前，已将每日均排满了。下次来，我争取去，我可自付机票（我已冲破经济的束缚了）。我赞曾老师的得意弟子谢仲淙在麻省总医院麻醉科已渐杰出。曾教授殷切期盼谢仲淙教授能海归。我说：谢仲淙教授在哈佛大学工作； 多年来 一直力促中美麻醉和神经科学研究交流，我们是好朋友。曾因明教授的学生遍及中美各地。刘仁玉教授在宾夕法尼亚医学院也是非常杰出，对麻醉学研究和防治中风贡献突出。

我观察到：科研成功者并非自己埋头苦干即可；他（她）应幸运寻到 或巧遇杰出的导师；加入强壮的科研团队；幸运选择好课题；好的身心健康；不断地努力升华情操 ……， 天时、地利、运气均重要 …...。

有时我想：一个人成功与否确实是仁者见仁 ，智者见智。我虽未发刊许多论文，可我救治了许多患者；帮助了一些同仁；为中美麻醉交流努力贡献了；我有很杰出的朋友们……。

有一好友，王医师，博士毕业后到纽约市洛克菲勒大学 （此机构位于曼哈顿中城东侧，Sloan 肿瘤中心对门。虽称大学，并不招收本科生，只聘请很优秀的博士后或更高水平的科研人员，从事各医学尖端研究）。朝夕奋斗三年，仔细一看：距离诺贝尔奖遥远而无期，只好转做临床了。

数年前，我自己开车去纽约长岛北岸：冷泉港（ Cold Spring Harbor, National Lab) 国家级研究院参观。图书馆厅墙上挂着多张照片，均是诺奖获得者，我认出 了 James Watson （沃森 ）。他因在英国剑桥与他的一个朋友共同发明“DNA 的双螺旋结构 ” 获得诺贝尔医学生理奖。1986 年，我在 Worcester Foundation 生物医药研究所（此地一位美籍华人最早发明女性避孕药 ）聆听了沃森教授的精彩演讲。如今，他已是耄耋之年。可他的太太仍任图书馆长，热情、美雅。我告诉她，我早年来自北京，今特意慕名前来参观。她欢迎我多看看。那天，我还顺路参观了纽约州立大学石溪分校 （杨振宁教授以前工作的单位）。该大学虽不宏观壮丽，但有一栋 Charles Wang Center（ 王嘉廉，曾毕业于此校，因研发计算机软件而致富，那时计算机领域华裔中，名望大概仅次于王安博士）。

王安博士致富后，曾捐款翻建哈佛大学医学院麻省总医院急诊中心。大厅内悬挂着王安博士和夫人的巨幅画像。1993 - 1994，我曾在此急诊大楼诊治疼痛患者。

王安博士曾捐巨款给波士顿中国城，建成医疗中心大楼；还赠款于波士顿歌舞剧院以致更名为 “ Wang Center ” !

我阅读麻醉月刊杂志，每年至少参加一次纽约州或全美麻醉年会。不断更新知识，了解和掌握新药或新设备，努力与时俱进。

SAFELM™ VIDEO LARYNGEAL MASK SYSTEM

SafeLM makes insertion procedure of laryngeal mask become visualized, guiding operator to insert LMA with accuracy and efficiency. It helps to reduce stimulus to epiglottis, larynx, and other surrounding structures, and prevent foreign body entering airway.

Qing Wang
(2023 CASA Photo Competition)

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