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本期封面：*Rainbow Bridge, Utah* 汪 红 摄影作品（*Instagram @howang248*）

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。

We appreciate your attention to this issue of CASA's publication. As this journal does not employ a peer review process, academic articles submitted and published in this journal retain the potential for future publication in peer-reviewed journals. Additionally, officially published articles may be reprinted in this journal. The editorial board strongly encourages our colleagues to actively submit articles and contribute to the advancement of anesthesiology.

本期导读

本期的会员访谈，我们有幸邀请到了胡辉医生和我们分享他的执业经历和丰富多彩的个人生活。

一年一度的Match季在三月份结束，我们编辑部诚挚的祝贺每一位成功进入麻醉医学培训医生。为了帮助还在准备申请的医学生，CASA在四月份组织了2025年美国麻醉住院医及亚专科Match分享会，黄少鹏医生为本刊撰写了会议简报，分享了会议的精彩内容。

随着GLP-1 受体激动剂(GLP-1 RAs)的越来越广泛的应用，为我们的日常麻醉管理提出了新的挑战。本刊编辑张扬根据前会长彭勇刚和会员李娟以及黄少鹏最近发表的一篇综述撰写了文献速递。

CASA候任会长王景平和其团队为我们介绍围术期使用esketamine (艾氯胺酮)治疗肿瘤痛期间药物对心血管系统的影响。

前CASA Bulletin主编苗宁团队为我们介绍了两例肾上腺皮质肿瘤(Adrenalcortical Carcinoma, ACC)术中出现紧急情况时的麻醉管理。

CASA会员李娟为本刊提供了一例非常成功的Mitral-Clip，患者在术后心输出量显著增加，同时降主动脉超声自发显影（spontaneous echo contrast）消失。

本期的最后依然是我们敬爱的CASA创始人之一王海明医生的回忆录。介绍了他的两位优秀女儿的成长以及他对乒乓球运动的热爱。

本期封面依然是来自于我们CASA前会长汪红的摄影作品，摄于犹他州的彩虹桥 (Rainbow Bridge)。它是世界上最大的自然形成的石桥之一，高250英尺，跨径275英尺。汪红的先生王乐一特意为此赋诗一首。

寻访美国犹他州彩虹桥自然保护区

王乐一

丛山峻岭穿长河，奇谷深藏彩虹桥。

陡壁水旋石幽险，曲途山峭景深娆。

左寻右顾路不见，天斧地工岩可削。

惊见一弧天际过，喜连情侣不愁遥。

CASA 会员访谈 （胡 辉 医生）

1. 您在哪年参加的 CASA? 您认为协会的价值和文化 是否符合你的预期，对今后进一步发展和改进有何 建议?

我是2023年在麻醉年会上加入CASA. 我是非常认同协会的价值和文化，希望协会今后能够吸引更多的新会员扩大组织。

2. 您认为几十年来麻醉最大的改变/进步是什么?您对未来的预期/发展

麻醉一直致力于安全性的发展，在这方面也取得了很大的进步，是其它学科的榜样。我觉得未来的AI技术引用会进一步加强麻醉的安全性。

3. 如果没有做麻醉，或者重新选择，这辈子您会做什么?

工程师

4. 工作之外有什么爱好吗?

爬山，跳舞, making YouTube teaching videos for nerve blocks using landmarks.

https://www.youtube.com/watch?v=KOaWrciqnpk&t=55s

https://www.youtube.com/watch?v=tN8stPO3GbE

https://www.youtube.com/watch?v=2hPUxHQc9m4&t=2s

https://www.youtube.com/watch?v=Z-Hq_n6urKg

https://www.youtube.com/watch?v=kipSqJyVtLo

5. 遇到过最有意思的病例

It was my first day on call as a brand new attending. Just as I arrived home, I received a call from an ENT surgeon. He asked me to come back to the hospital to intubate a patient who had undergone a radical neck dissection earlier that day. The patient's tracheostomy tube had fallen out, and the surgeon was hesitant to replace it, concerned about potentially damaging the fresh surgical site.

I asked the surgeon to be present during the intubation in case it was a difficult intubation. When I arrived at the hospital, the on-call CRNA had already brought the patient into the OR and was waiting for me.

I instructed the CRNA to begin titrating a remifentanil drip to maintain spontaneous breathing while I quickly reviewed the anesthesia record. Once the patient was adequately sedated, I prepared for intubation using a MAC 3 blade as the other anesthesiologist used earlier, applying lidocaine jelly on it for added comfort.

As I gently inserted the blade, all I could see was pink tissue—no clear landmarks. I stayed calm and asked the CRNA for a

bougie, even as I felt my heart pounding. I asked the CRNA to cover the stoma of the trachea, then I saw air bubbles and advanced the bougie toward where the air bubbles were without resistance. I then threaded a small endotracheal tube over the bougie, I could hear the air passing through the endotracheal tube, and asked the CRNA to slowly withdraw the bougie. Tube placement was confirmed.

After the successful intubation, the surgeon gave me a reassuring pat on my shoulder. It was an enormous relief—and a moment I'll never forget.

6. 回首往事，您做的最得意的事，最想让人记住的是什么？ 给年轻人的寄语?

多年来我一直尝试发明创造和开公司，目前有两个产品在中国市场销售，开了四家公司。我最得意的是开了med-gigs Inc, 它是一款专注于医疗排班的云端平台，提供给医疗人员和医疗机构之间便捷高效的纽带，感觉它前途广阔，也希望它能够走得很远。我建议年轻的同行跳出传统的思维框框，以年轻人的新眼光看问题，可以独辟蹊径。

2025 美国麻醉住院医及亚专科MATCH分享会简报 (黄少鹏撰稿)

2025 U.S. Anesthesiology Residency and Fellowship Match Experience Sharing Session

April 5, 2025 9:00 PM EST - 12:40 PM EST

Host:

Dr. Shaopeng Huang, MD

PGY-5, Cardiothoracic Anesthesia Fellow

Montefiore Medical Center

Guest Hosts:

•Dr. Yang Zhang, MD

Assistant Professor of Anesthesiology, Stanford Medical Center

•Dr. Qing Zhao Ruan, MD

Assistant Professor of Anesthesiology and Pain Specialist,

Brown University / Providence VA Medical Center

This virtual session brought together successfully matched applicants in 2025 and expert faculties to share first hand insights into the US anesthesiology residency and fellowship application processes. The event was designed to support and guide Chinese medical graduates (CMGs) in understanding match pathways, optimizing their applications, and exploring less conventional yet effective routes to practicing anesthesia in the U.S.

Featured Speakers

Residency Match Applicants:

•Dr. Chih-Xuan (Julia) Chen – Matched into Categorical Anesthesiology Residency, Cook County Health

•Dr. Yifan Bu – Matched into Categorical Anesthesiology Residency, Beth Israel Deaconess Medical Center (BIDMC)

•Dr. Lei Yang – Matched into Advanced Anesthesiology Residency (PGY-2), University of Washington

Fellowship Match Applicants:

•Dr. Qi Yu – Matched into Pediatric Anesthesiology Fellowship, Children's Hospital at Montefiore

•Dr. Xinhao Liu – Matched into Obstetric Anesthesiology Fellowship, Montefiore Medical Center

•Dr. Zhihao Wang – Matched into Regional Anesthesiology Fellowship, Westchester Medical Center

Highlights of the Session

Dr. Julia Chen shared her unique journey as a fresh graduate with no prior anesthesia experience. Her success in matching directly into a U.S. categorical anesthesiology residency offers an encouraging example for other recent CMG graduates.

Dr. Yifan Bu provided an in-depth overview of multiple pathways available to IMGs pursuing anesthesiology in the U.S., including both traditional (residency-to-attending) and non-traditional routes (fellowship-first, multiple fellowships, or direct practice for experienced anesthesiologists from abroad).

Dr. Lei Yang reflected on her personal journey to anesthesiology, offering valuable insight into the strategic considerations that guided her application process.

Drs. Qi Yu, Xinhao Liu, and Zhihao Wang gave practical and detailed presentations on how they successfully matched into U.S. anesthesia fellowships without prior U.S. residency. Their step-by-step breakdowns and explanations of the SF Match system were particularly helpful for attendees unfamiliar with this process.

Q&A Discussion

In the concluding segment, Drs. Huang, Ruan, and Zhang addressed questions submitted by attendees. They offered tailored advice on increasing competitiveness for the match, improving application materials, and identifying suitable programs. Their expertise provided valuable guidance to CMGs navigating these complex pathways.

文献速递 (张 扬 撰稿)

GLP-1 受体激动剂与择期手术围术期误吸风险和管理的文献综述

Li, J., Mohamed, B., Huang, S., & Peng, Y. G. (2025). Aspiration risk and strategic approach for patients receiving GLP-1 receptor agonists undergoing elective surgery. Current Medical Research and Opinion, 41(4), 699–712. https://doi.org/10.1080/03007995.2025.2494646

（原文作者为CASA会员李娟，黄少鹏和彭勇刚医生）

GLP-1 受体激动剂（GLP-1 RAs）的作用机制与药理特点

GLP-1 受体激动剂通过激活胰高血糖素样肽-1 受体，可有效促进体重减轻、改善糖尿病患者的血糖控制，并具有心脏和肾脏保护作用。然而，由于其减缓胃排空的作用，多个病例报告及回顾性研究提出其可能增加围术期误吸的风险。根据药代动力学特征，GLP-1 RAs 可分为短效和长效两类。例如，短效制剂 Exenatide（商品名：Byetta、Bydureon）半衰期约为3小时；而长效制剂 Semaglutide（商品名：Wegovy、Ozempic）半衰期可达约7天。

研究方法

本综述纳入了2000至2024年间所有关于GLP-1 RAs与围术期误吸风险、呼吸系统并发症风险的研究，包括随机对照试验、观察性病例对照研究、前瞻性与回顾性队列研究、系统评价、综述文章、病例报告及病例系列。

研究发现及其差异性

多数研究证实GLP-1 RAs可显著减缓胃排空，但其是否显著增加误吸风险仍存在争议。一些研究显示其与误吸风险正相关，而正另一些研究则未发现统计学上的显著关联。

研究结果不一致的可能原因

- 研究设计与人群差异：前瞻性与回顾性设计的差异，受试者合并疾病（如糖尿病、肥胖、高龄）对胃排空的影响，以及手术类型和样本量的不同；
- GLP-1 RAs 类型及剂量的差异
- 麻醉方式的不同：无气管插管的麻醉方式更易发生误吸；
- 评估胃排空的检测方法不同：如胃部超声、胃镜等，其敏感度与特异度存在差异。

研究中的几个有趣发现与观点

- 禁食时间与进食方式的调整是否影响误吸风险?

部分研究指出，在进行上消化道内镜检查（EGD）前，如果患者遵循术前一天仅进食清流质饮食的方案，则可以在继续使用GLP-1 RAs的情况下安全接受操作。

- 药效变化与胃排空延迟的影响

研究显示，GLP-1 RAs 所致的胃排空延迟在长期服药后可能减轻。胃肠道副作用通常在用药第12周最为明显，至第20周后逐渐缓解。若患者长期使用，且近期未调整药量，术前无消化道症状（如腹胀、恶心），则推测其胃排空功能可能恢复，误吸风险随之降低。

ASA 指南的更新与调整

美国麻醉医师协会（ASA）旧版指南建议：对于择期手术，日用型GLP-1 RAs应提前一天停药，周用型应提前至少一周停药。但新版指南指出，仅依据药物半衰期调整用药时间不足以有效降低其血药浓度或恢复胃排空功能。以Semaglutide为例，其半衰期约为1周，药物清除时间可达5至7周。

Original Article （原创文章）

A Review of the Effects of Perioperative Esketamine on the Cardiovascular System in Cancer Pain Management

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#Lili Zhu and Fei Liang contributed equally to this work.

1. INTRODUCTION

Cancer patients in the perioperative period (preoperative, intraoperative, and postoperative) often face significant challenges in pain management. Chemotherapy not only exacerbates pain perception but also induces side effects such as immune suppression and tissue damage, further intensifying perioperative pain [1][2]. Effective pain management plays a crucial role in enhancing patient comfort and quality of life, facilitating postoperative recovery, shortening hospital stays, and reducing the risk of complications [3][4]. Therefore, selecting an optimal analgesic regimen is essential for perioperative care in cancer patients receiving chemotherapy [5][6].

Esketamine, the S-enantiomer of ketamine, has gained attention for its potent analgesic and sedative properties. It exerts its analgesic effects primarily through N-methyl-D-aspartate (NMDA) receptor antagonism in the central nervous system [7] [8]. At low doses, esketamine has demonstrated relatively stable cardiovascular effects, making it a potential alternative to traditional opioid analgesics [9]. However, cancer patients undergoing chemotherapy often have an increased cardiovascular burden due to prolonged exposure to multiple medications, including cardiotoxic chemotherapeutic agents. While esketamine may improve perioperative pain control, its cardiovascular effects in this specific patient population remain under-explored. Most existing studies focus on general surgical patients or other disease groups, with limited research addressing cancer patients receiving chemotherapy.

Understanding the cardiovascular safety and efficacy of esketamine in perioperative pain management for chemotherapy patients is of great clinical importance. This review aims to summarize current literature on the perioperative use of esketamine in cancer patients undergoing chemotherapy, with a particular focus on its cardiovascular effects. By systematically analyzing relevant studies, this review seeks to provide insights into the analgesic benefits and cardiovascular implications of esketamine, contributing to optimized pain management strategies and guiding future research.

2.Pharmacological Effects of Esketamine on the Cardiovascular System

Esketamine, the S-enantiomer of ketamine, is widely used in anesthesia for both induction and maintenance. It serves as an adjunct anesthetic, reducing the need for additional sedatives and analgesics, enhancing pain relief and sedation, and minimizing anesthesia-related adverse effects. The recommended induction dose for general anesthesia is 0.5 mg/kg, with a maintenance dose of 0.5 mg/kg/h, though its optimal adjunctive dosing remains under investigation [[10][11]]. Due to its rapid onset and short recovery time, esketamine is frequently used in procedures such as painless gastrointestinal endoscopy.

Esketamine primarily exerts its effects by blocking N-methyl-D-aspartate (NMDA) receptors, leading to analgesia and sedation.

However, it also influences multiple other pathways, affecting the cardiovascular system through complex mechanisms (Figure 1).

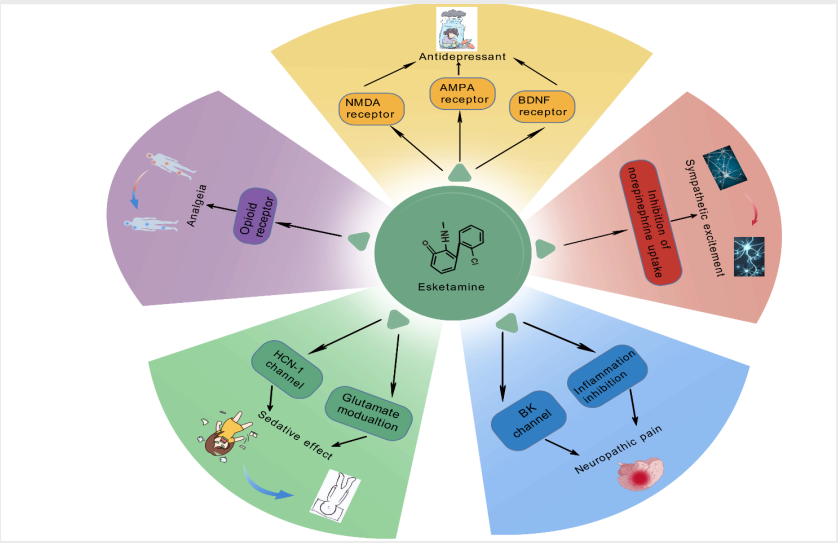

Figure 1. The Multiple Mechanisms of Esketamine in Clinical Pain Management：Esketamine alleviates pain through multiple mechanisms: it directly reduces pain by activating opioid receptors; inhibits pain signal transmission by blocking NMDA receptors and activating AMPA and BDNF receptors; provides sedative effects and regulates neural activity by modulating HCN channels and the glutamate pathway to alleviate pain; addresses neuropathic pain by suppressing inflammatory responses; and mitigates pain caused by stress or anxiety by inhibiting sympathetic nervous system excitability. NMDA: N-Methyl-D-aspartate;AMPA: α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid;BDNF: Brain-Derived Neurotrophic Factor;HCN: Hyperpolarization-activated cyclic nucleotide-gated channel;BK channel: Big Potassium Channel.

Antidepressant Effects – As a novel agent, esketamine significantly alleviates depressive symptoms. Its antidepressant action is primarily mediated through NMDA and AMPA receptor activation, along with increased expression of brain-derived

neurotrophic factor (BDNF), which promotes neuroplasticity and synaptic function [[12-15]].

Opioid Receptor Interaction and Analgesic Effects – Esketamine may enhance analgesia by directly acting on opioid receptors or interacting with them to amplify pain relief, effectively reducing pain perception [[16-19]].

Sympathetic Nervous System Modulation – By inhibiting norepinephrine reuptake, esketamine increases norepinephrine levels in the synaptic cleft, leading to heightened sympathetic nervous system activity and balancing neural function [[7][20][21].

Sedative and Anxiolytic Effects – Esketamine produces sedation by modulating the HCN-1 channel and glutamate signaling pathways, helping patients alleviate anxiety and tension [[22-25]].

Neuropathic Pain Relief – In the treatment of neuropathic pain, esketamine effectively alleviates chronic pain caused by nerve injury by inhibiting the Big Potassium Channel (BK channel) and reducing inflammation [[26][27]].

Through these mechanisms, esketamine not only provides analgesia and sedation but also exerts notable cardiovascular effects. While its ability to transiently elevate blood pressure and heart rate may be beneficial in certain clinical scenarios, caution is necessary in patients with cardiovascular disease. Future studies should further investigate its hemodynamic effects and optimize its perioperative use in high-risk populations.

3. Effects of Esketamine on the Cardiovascular System

Research has shown that esketamine (the S-enantiomer of ketamine) exerts its pharmacological effects primarily by blocking N-methyl-D-aspartate (NMDA) receptors [7]. While NMDA receptors play a crucial role in the central nervous system (CNS)—mediating sedation, anesthesia, and antidepressant effects—they are also expressed in the heart, where they participate in regulating cardiac function. Consequently, esketamine's NMDA receptor blockade may influence cardiac electrophysiology and function. Specifically, blocking NMDA receptors could alter intracellular calcium ion flux in cardiomyocytes, potentially affecting myocardial contraction and conduction. The following sections summarize the major pharmacological effects of esketamine on the cardiovascular system based on current research (Figure 2).

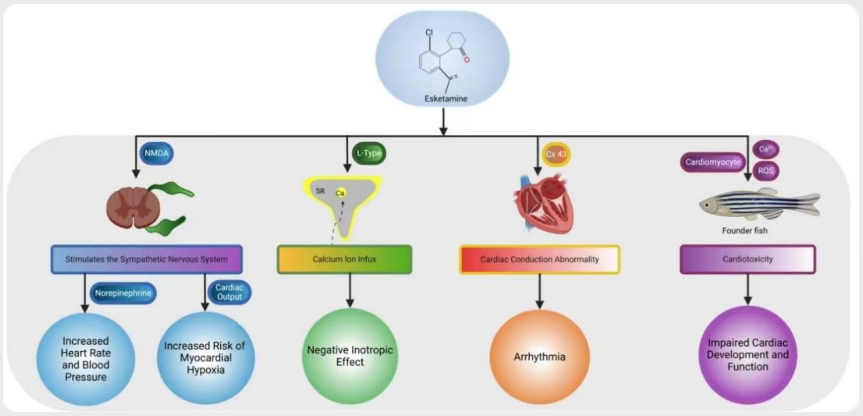

Figure 2. Mechanisms of Esketamine's Effects on the Cardiovascular System.Esketamine activates the sympathetic nervous system, increasing norepinephrine release, which leads to elevated heart rate and blood pressure, subsequently increasing myocardial oxygen consumption. It also interferes with L-type calcium channels, reducing calcium ion influx, which in turn decreases myocardial contractility and conduction ability. Furthermore, esketamine affects cardiomyocyte action potentials and Connexin43 (Cx43) expression, potentially inducing arrhythmias. Additionally, by modulating calcium ion channels, reactive oxygen species (ROS), and gene expression, esketamine may impact cardiac development and function, potentially resulting in cardiotoxicity. NMDA:N-Methyl-D-Aspartate Receptor; L-Type :L-Type Calcium Channel; SR: Sarcoplasmic Reticulum;Cx34:Connexin43;Ca2+: Calcium ion; ROS: Reactive Oxygen Species.

3.1 Induction of Tachycardia, Increased Blood Pressure, and Myocardial Oxygen Consumption

Esketamine may activate the sympathetic nervous system (SNS), leading to increased heart rate (HR) and elevated blood pressure (BP). Suleiman A et al. reported that esketamine enhances respiratory drive, promotes arousal, and activates the SNS, thereby affecting cardiac function. The heightened sympathetic activity boosts myocardial contractility and cardiac output, maintaining circulatory stability. These effects are attributed to NMDA receptor antagonism and enhanced AMPA receptor

activation, which intensify sympathetic outflow. At moderate to high doses (incremental infusion over 3 hours, reaching 1 mg/kg), esketamine stimulates SNS activity and adrenaline release, leading to increased HR and systolic BP. In patients with cardiovascular diseases, this may elevate the risk of adverse events [28].Qi [29] reported that 0.25 mg/kg intravenous (IV) esketamine helps stabilize hemodynamics during anesthesia and shows significant potential in treating refractory depression. Its sympathomimetic effects increase cardiac output and peripheral vascular resistance, counteracting the vasodilatory effects of spinal anesthesia.

Additionally, esketamine-induced SNS activation increases myocardial oxygen demand and consumption (MVO_2). This effect partially counteracts the circulatory depression caused by propofol and remifentanil, primarily by blocking NMDA receptors and activating the SNS, thereby boosting cardiac output and mean arterial pressure (MAP). Studies indicate that at a 0.3 mg/kg dose, esketamine significantly increases cardiac output and MAP, ensuring circulatory stability during anesthesia induction.However, increased myocardial oxygen demand due to elevated HR and enhanced myocardial contractility may worsen myocardial ischemia, particularly in patients with hypertension or coronary artery disease (CAD). Thus, caution is advised when using esketamine in patients with impaired cardiac function [30]. Doherty[31] assessed intranasal esketamine for treating refractory depression and its cardiovascular safety. The fixed doses (56 mg or 84 mg, administered 1–2 times per week) transiently increased systolic and diastolic BP, peaking 40 minutes post-administration and returning to baseline within 1–2 hours. The concurrent HR increase was synchronized with BP changes, likely due to sympathetic activation and norepinephrine release, leading to vasoconstriction and tachycardia. Therefore, caution is necessary in patients with cardiovascular conditions (e.g., hypertension, CAD) due to the risk of increased myocardial oxygen consumption and BP elevation.

3.2 Effects on Cardiomyocyte Calcium Regulation

Esketamine blocks calcium ion (Ca^{2+}) channels, which relaxes airway smooth muscles but also exerts negative inotropic effects on the heart. This suggests that esketamine may alter cardiomyocyte Ca^{2+} influx, providing insights into its use in anesthesia for specific patient populations.The Ca^{2+} channel blockade allows esketamine to be used in patients with airway hyperreactivity during anesthesia induction and maintenance. However, it may also introduce potentially adverse cardiac

effects [32]. The mechanisms involved include: L-type Ca^{2+} channel blockade, reducing Ca^{2+} influx, decreasing sarcoplasmic reticulum Ca^{2+} release, and impairing excitation-contraction coupling in cardiomyocytes. NMDA receptor antagonism, inhibiting NMDA receptor activity in the heart, thereby reducing intracellular Ca^{2+} levels. SNS inhibition, reducing norepinephrine release, which may weaken positive inotropic effects.Further studies are needed to explore these potential mechanisms.

3.3 Induction of Cardiac Arrhythmias

Esketamine may alter cardiac action potential morphology at certain doses, affecting resting membrane potential and action potential duration, which may induce arrhythmias. The dose-dependent effects of esketamine on cardiac conduction have been documented. Studies indicate that as esketamine dose increases, cardiac conduction time lengthens, correlating with heterogeneous Connexin43 (Cx43) expression. This results in spatially uneven conduction, increasing arrhythmia susceptibility.Animal studies reveal Cx43 expression changes, leading to higher arrhythmia incidence. High-dose esketamine has been shown to significantly increase arrhythmia occurrence, potentially inducing abnormal conduction at high heart rates, resulting in ventricular fibrillation, atrial fibrillation, or other arrhythmias [33].

3.4 Cardiotoxicity

Huang[34] demonstrated that esketamine may disrupt Ca^{2+} channels, cardiomyocyte function, and gene expression, leading to abnormal zebrafish heart development and function. This study provides critical experimental evidence regarding the potential cardiac side effects of esketamine, particularly its perioperative and long-term cardiovascular risks.

Although the exact mechanism by which esketamine induces changes in consciousness remains unclear, its acute effects are strictly dose-dependent as a “dissociative analgesic/anesthetic.” Low and sub-anesthetic doses are primarily associated with analgesia, antidepressant effects, mild psychedelic experiences, and a sense of "disconnection" in consciousness. As the dose increases, it can lead to enhanced sedation and even loss of consciousness.

Additionally, low-dose esketamine typically causes mild and transient adverse effects. Table 1 provides dosage guidelines for different clinical applications of esketamine [35-47], detailing its cardiovascular effects and recommended management strategies to help balance efficacy and safety in clinical practice.

Table 1. Esketamine Dosage Guidelines, Cardiovascular Effects, and Management in Adults

Indication	Dose	Specifics	Cardiovascular Effects	Recommended Management	References
Analgesia/sedation in acute pain					
IV, low-dose	0.125–0.3 mg/kg	Provide CALM, friendly, and supportive atmosphere in case of adverse psychic events. Combine with GABA-agonist if clinically possible.	May cause mild increases in BP and HR.	Monitor BP/HR; avoid in uncontrolled hypertension.	[35][36][37]
IV, subanesthetic-dose	< 0.5 mg/kg		Potential moderate increases in BP and HR.	Combine with β-blockers if cardiovascular stress is significant.	[35][38]
IM	0.25–0.5 mg/kg		Similar to IV; slower onset of changes in BP and HR.	Use with caution in patients with cardiovascular risks.	[35][39][40]
IN	1.5–2.5 mg/kg		May cause transient elevations in BP.	Encourage nasal saline rinses post-administration.	[31][35][41]

Analgesic adjunct to general anesthesia					
Intraoperative repeated or continuous administration	0.12–0.6 $mg \cdot kg^{-1} \cdot h^{-1}$	Dose reduction of other drugs (e.g., inhalational anesthetics) often possible.	May stabilize BP due to reduced need for other depressant anesthetics.	Gradual titration to minimize abrupt BP changes.	[31][35][42]
Postoperative					
PCA	0.25–0.75 mg per bolus		Mild elevation in BP may occur.	Adequate hydration and monitoring required.	[35][43][44]
Continuous infusion	0.075–0.125 $mg \cdot kg^{-1} \cdot h^{-1}$		Stabilizing or minor increases in BP.	Close monitoring in high-risk patients.	[35][45]
Anesthesia					
Induction					
IV	0.5–2.5 mg/kg		Dose-dependent elevation in BP and HR.	Combine with GABA-agonist (e.g., propofol) for high doses.	[35][40][46]
IM	4.0–8.0 mg/kg		Similar cardiovascular effects as IV but slower onset.	Use only in controlled environments with appropriate monitoring.	[35][45][47]
Maintenance	0.5–2.5 $mg \cdot kg^{-1} \cdot h^{-1}$		Potential for increased BP and HR with prolonged infusion.	Adjust infusion rate based on real-time BP/HR monitoring.	[35][45]

4. The Impact of Cancer Chemotherapy Drugs on the Cardiovascular System

While cancer chemotherapy drugs improve tumor control and patient survival rates, they can also have significant adverse effects on the cardiovascular system. These effects mainly involve damage to myocardial function, disruption of cardiovascular regulation mechanisms, and chemotherapy-related complications, collectively known as "cardiotoxicity." Cardiotoxicity presents in various forms, including direct myocardial damage, arrhythmias, hypertension, heart failure, and an increased risk of cardiovascular events. These potential cardiovascular impacts, especially during the perioperative period, require more stringent patient management. Table 2 summarizes the specific effects of common chemotherapy drugs on the cardiovascular system, categorizing the cardiovascular complications caused by different drugs and their mechanisms

- Endothelial Dysfunction: Some chemotherapy drugs impair endothelial function by reducing nitric oxide (NO) production or increasing oxidative stress levels, leading to elevated blood pressure or arteriosclerosis [50]. Anti-angiogenic drugs (e.g., bevacizumab) exert their effects by inhibiting vascular endothelial growth factor (VEGF), while also increasing the risk of thrombosis [51].
- Cardiac Metabolic Abnormalities: 5-fluorouracil (5-FU) may interfere with myocardial energy metabolism, leading to angina or even myocardial ischemia [52].
- Calcium Ion Imbalance: Certain drugs affect calcium channels in myocardial cells, causing calcium overload and leading to arrhythmias or abnormal myocardial contraction [53]. For example, paclitaxel may cause QT interval prolongation [54].

4.2 Cardiovascular Complications Caused by Chemotherapy Drugs

- Tachycardia: Some chemotherapy drugs (e.g., procarbazine, cladribine, alemtuzumab, trastuzumab, and muromonab-CD3) may induce tachycardia by enhancing sympathetic nerve excitability or through direct toxic effects [55][56]. These drugs require special attention during the perioperative period to prevent hemodynamic abnormalities.
- Bradycardia: Docetaxel and lenalidomide may induce bradycardia through excessive stimulation of the vagus nerve or direct inhibition of myocardial cells, especially in the perioperative period, which may lead to blood pressure instability [57][58].
- Hypertension: Anti-VEGF drugs (e.g., bevacizumab, sorafenib, sunitinib, nilotinib) induce hypertension by

increasing peripheral resistance [59][60][61]. Chronic hypertension further increases the risk of cardiovascular events [62].

- Myocardial Ischemia and Infarction: Chemotherapy drugs (e.g., 5-FU and capecitabine) can cause myocardial ischemia or even myocardial infarction through vasospasm or direct toxicity [52][63]. Early intervention is required for these complications to prevent further damage to heart function.
- HeartFailure: Anthracyclines (e.g., doxorubicin) are commonly associated with heart failure after prolonged use, with cumulative doses significantly correlating with the risk of heart failure [64]. These drugs should be used under close monitoring of heart function.
- Arrhythmias: Electrolyte imbalances (e.g., hypomagnesemia) caused by cisplatin are one of the major causes of chemotherapy-related arrhythmias [65].
- Additionally, drugs like fludarabine may also induce arrhythmias [66].
- Thromboembolic Events: Chemotherapy drugs increase the risk of thrombosis by activating the coagulation system [67]. Perioperative patients require prophylactic anticoagulation therapy to reduce this risk.
- Inflammatory Factors and Immune Response: Chemotherapy-induced systemic inflammatory responses can exacerbate the cardiovascular burden during the perioperative period [68]. For instance, drugs like muromonab-CD3 and daclizumab may cause immune-mediated cardiovascular reactions.

Table 2 summarizes some common chemotherapy drugs and their associated cardiovascular adverse reactions, including arrhythmias, hypertension, myocardial damage, and others.

Table 2 Representative chemotherapy agents and Cardiovascular concerns

Organ System	Common Perioperative Concerns	Associated Chemotherapy Drugs	References
Cardiovascular	Tachycardia	Procarbazine, Cladribine, Alemtuzumab, Trastuzumab, Muromonab-CD3	[55][56][70]
	Cardiac arrhythmia	Pentostatin, Fludarabine, Palivizumab, Interferon alfa-2b, Erlotinib	[66][70]
	Bradycardia	Docetaxel, Lenalidomide	[57][58][70]
	Hypotension	Pentostatin, Vincristine, Alemtuzumab, Daclizumab, Muromonab-CD3, Denileukin diftitox	[68][70]
	Hypertension	Pentostatin, Vinblastine, Vincristine, Alemtuzumab, Bevacizumab, Trastuzumab, Daclizumab,	[59][60][62][70]
	Cardiomyopathy	Doxorubicin, Trastuzumab, Sunitinib, Dasatinib, Lapatinib	[49][65][70]
	Myocardial ischemia/infarction	5-FU, Capecitabine	[52][64]
	Heart failure	Doxorubicin, Trastuzumab	[65]
	Cardiac arrhythmia	Cisplatin (due to electrolyte imbalance such as hypomagnesemia)	[66]

5. The Potential Cardiovascular Effects of Esketamine When Used in Combination with Chemotherapy Drugs

5.1 The Role of Esketamine in Cancer Pain Patients Undergoing Chemotherapy

- Relief of Cancer Pain
  Mechanism of Action: Esketamine is a selective NMDA receptor antagonist that can effectively relieve cancer pain by reducing central sensitization and decreasing the transmission of pain signals [70]. Research has shown [10] [71] that esketamine can be used for pain management, particularly when opioid drugs have limited efficacy or increased tolerance. Under these circumstances, esketamine serves as an effective adjunct in cancer pain management, especially for opioid-resistant pain.

- Improvement of Chemotherapy-Induced Mood and Cognitive Issues
  Mechanism of Action: Chemotherapy-related depression and anxiety can significantly affect a patient's quality of life. Esketamine regulates glutamate signaling and has rapid antidepressant and anxiolytic effects. Studies by Zarate CA et al. have demonstrated that esketamine's antidepressant effects are rapid and may be effective for mood disturbances induced by chemotherapy [72].

5.2 Cardiovascular Effects of Esketamine When Used in Combination with Chemotherapy Drugs

Esketamine, when used in combination with chemotherapy drugs, has shown potential clinical benefits in cancer pain management. However, its synergistic or interactive effects on the cardiovascular system require careful evaluation. The mechanisms of cardiovascular damage caused by chemotherapy drugs are complex, typically manifesting as direct myocardial toxicity, vascular dysfunction, and metabolic disturbances. Esketamine itself has also been reported to have certain effects on the cardiovascular system. The following analysis examines the potential cardiovascular effects based on the combined mechanisms of both.

- Cardiovascular Excitatory Effects
  When used in combination with anthracyclines (e.g., doxorubicin), esketamine may produce a synergistic effect on the cardiovascular system, necessitating particular attention to its potential risks. Anthracyclines induce myocardial toxicity through oxidative stress, iron-dependent toxicity, and calcium metabolism disorders [73], while esketamine activates the sympathetic nervous system, leading to an increased heart rate and elevated blood pressure. Its excitatory effects on the central nervous system further promote catecholamine release, increasing myocardial workload [7][31][74]. Therefore, the combination of these two drugs may have an additive effect on heart rate and blood pressure, which could increase the risk of cardiovascular adverse events, especially in patients with pre-existing cardiovascular conditions. High doses of esketamine should be avoided, and the use of β-blockers or calcium channel blockers should be considered for cardioprotection. For patients with underlying hypertension, an optimized antihypertensive regimen should be implemented prior to treatment, with close monitoring of blood pressure changes.

- Myocardial Ischemia and Increased Oxygen Consumption
  Esketamine's cardiovascular effects, including enhanced myocardial contractility and increased heart rate, may elevate myocardial oxygen demand in the short term, particularly in patients with impaired heart function or restricted coronary artery blood flow, thereby increasing the risk of myocardial hypoxia. Chemotherapy drugs such as 5-fluorouracil (5-FU) can also induce myocardial ischemia or infarction by causing coronary artery spasm or endothelial dysfunction [52]. Therefore, when esketamine is combined with 5-FU or similar drugs, special attention must be paid to the risk of myocardial ischemia. In patients with chemotherapy-induced myocardial ischemia, high doses or rapid infusion of esketamine should be avoided. If necessary, vasospasm medications (e.g.,

nitrates) or anti-ischemic treatments should be used to protect myocardial oxygen supply.

- Synergistic Effects on Arrhythmias
  Esketamine may alter the dynamic balance of calcium ions within myocardial cells or modify the action potential duration, decreasing myocardial contractility and increasing the risk of arrhythmias [32][33][34]. This property complements the effects of positive inotropic drugs (e.g., dopamine, milrinone) or calcium sensitizers (e.g., levosimendan). Positive inotropic drugs increase myocardial contractility by enhancing intracellular cAMP levels or calcium influx, while calcium sensitizers improve heart function by enhancing troponin's sensitivity to calcium, without increasing the risk of calcium overload. However, when combined with chemotherapy drugs such as anthracyclines or cisplatin, which often cause electrolyte imbalances (e.g., hypomagnesemia, hypokalemia), the risk of ectopic excitation in myocardial cells and arrhythmias may be exacerbated [53]. Therefore, the combination of these drugs may increase the probability of ventricular tachycardia, atrial fibrillation, or QT interval prolongation. During combination therapy, electrolyte levels should be closely monitored, and calcium, potassium, and magnesium supplementation should be provided as needed. For patients with known QT prolongation or other arrhythmia risks, dynamic electrocardiographic monitoring is recommended.

- Cumulative Effects on Vascular Dysfunction
  Chemotherapy drugs (e.g., anti-VEGF agents) can induce endothelial dysfunction, leading to hypertension and an increased risk of thrombosis [51], while esketamine may exacerbate this effect by activating the sympathetic nervous system [28]. When used together, the combination may lead to further increases in peripheral vascular resistance, aggravating the vascular stress response. In patients receiving anti-VEGF drugs, careful evaluation of esketamine dosage is necessary, and treatment plans should be adjusted according to the patient's vascular function status. If necessary, vasodilators should be used to alleviate the increase in vascular resistance.

6. Conclusion

Perioperative pain management in cancer patients undergoing chemotherapy presents multiple challenges, including intraoperative and postoperative pain control, drug interactions, and the potential risk of cardiovascular complications. In this context, we explored the application of esketamine in perioperative pain management for cancer patients and its potential impact on the cardiovascular system. Esketamine has demonstrated significant efficacy in alleviating pain and mitigating depressive symptoms, particularly in cases where traditional opioid analgesics are insufficient for pain control. This study highlights the potential value of esketamine in cancer patients, especially in those receiving chemotherapy, where careful attention must be paid to its effects on the heart. Chemotherapy drugs may impair the heart's compensatory capacity, making patients more susceptible to cardiovascular adverse effects associated with medication use. Esketamine may further exacerbate cardiac burden by altering cardiac electrophysiology or hemodynamics. Therefore, when using esketamine in combination with chemotherapy drugs, a comprehensive assessment of the patient's cardiac health is essential, particularly for those who have previously received cardiotoxic chemotherapy agents. Clinical application should incorporate individualized dose adjustments to minimize potential cardiac risks. Additionally, close monitoring of cardiovascular indicators, including electrocardiograms, myocardial enzyme profiles, and hemodynamic changes, is recommended to detect early signs of cardiotoxicity and to explore an optimal dosage range that balances pain relief and mood improvement while minimizing cardiovascular risks. Furthermore, future research should further investigate the interaction mechanisms between esketamine and different chemotherapy drugs to provide more precise clinical guidance. In conclusion, the use of esketamine in cancer patients requires a comprehensive consideration of its synergistic effects with chemotherapy drugs, particularly its potential impact on the heart. Optimizing dosage, enhancing monitoring, and

incorporating individualized assessments will help improve medication safety, ensure cardiovascular health, and contribute to the advancement and refinement of perioperative pain management strategies.

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Case Report (病例报告)

Anesthesia emergency during adrenocortical carcinoma resection

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Introduction

An adrenocortical carcinoma (ACC) is an extremely rare and aggressive tumor of the adrenal cortex, with an annual estimated incidence of 0.7 to 2.0 cases per million population in the United States [1, 2]. The etiology of ACC is largely unknown, and may be related to a sporadic occurrence, genetic predisposition, or can be encountered in the setting of hereditary tumor syndromes [3-5]. ACC usually appears in the fourth to fifth decade of life, and with a median age at diagnosis of 46 years [3, 4, 6]

ACC may be categorized as either functional and non-functional based on the overproduction of adrenocortical hormones, such as glucocorticoids, mineralocorticoids, androgens, and estrogens, with the rare possibility of various peptides being involved. In cases of non-functional ACC, the presentation may include an abdominal mass, discomfort or pain, or symptoms indicative of compression resulting from either the primary tumor or distant metastases. [1, 5].

The preferred treatment modality for localized primary or recurrent tumors is radical surgical resection, which offers the most favorable opportunity for extended recurrence-free survival. However, it is important to note that patients presenting with recurrent or metastatic disease often cannot achieve cure through surgical intervention alone. A considerable number of patients who show no objective or biochemical indicators of residual tumor following surgery may ultimately experience relapse.

The efficacy of chemotherapy is somewhat limited, but it is noteworthy that platinum-based therapies exhibit a response rate between 25% and 30%. Mitotane, a derivative of dichlorodiphenyltrichloroethane, has been employed in the management of advanced adrenocortical carcinoma since the 1960s, although its application as an adjunct postoperative therapy remains a subject of debate [7, 8]. More recent findings suggest that mitotane treatment following macroscopically complete excision of adrenocortical carcinoma is associated with improved outcomes [8].

In cases of advanced disease where radical resection is not feasible, the incorporation of cytotoxic agents alongside mitotane may be beneficial. The most promising treatment regimens, etoposide, doxorubicin, cisplatin in combination with mitotane (EDP-M), and streptozotocin plus mitotane (S-M), were evaluated in an international phase III trial. The study showed that the EDP-M regimen demonstrated superiority over the S-M regimen. Other targeted therapies have been explored, including IGF-1 inhibitors, sunitinib, sorafenib, and axitinib; however, till now, no targeted therapy has been identified as providing substantial therapeutic advantage [1, 9].

Given rarity of ACC, there is limited experience pertaining to the anesthetic management during surgery posing a significant challenge for practitioners. Anesthesia providers must possess comprehensive knowledge on both the administration of anesthesia for ACC surgeries but also the tumor's characteristics, stages, progression, surgical complexities, and perioperative complications.

In this report, we discuss an intraoperative anesthesia emergency involving two cases: 1) a patient undergoing en bloc resection of a large metastatic left ACC, during which acute bronchospasm and vasoplegic syndrome occurred, and 2) a patient undergoing en bloc left upper quadrantectomy for recurrence of ACC, during which vasoplegic syndrome occurred. In both cases, the early identification and treatment was facilitated by continuous cardiac output monitoring helped guide the differential diagnosis as well as appropriate treatment. A single case report of concurrence of vasoplegia and ACC has been previously reported, however, its frequency may be higher and frequently undiagnosed warranting further discussion especially since successful management necessitates rapid diagnosis and treatment.

Case presentation 1

A 43-year-old male patient was admitted to the NIH Clinical Center for surgery following a recent diagnosis of metastatic ACC. He underwent surgical resection of the large primary left adrenocortical mass along with adjacent organs in an en bloc fashion. Recent imaging studies revealed a 7.3 cm x 5.6 cm subcarinal mediastinal mass and multiple pulmonary metastases. Additionally, there was narrowing of the right middle and lower lobe bronchsecondary to significant right hilar adenopathy. A 14.2 cm by 12.9 cm mass in the left adrenal gland was identified which was inseparable from the left kidney. Additional studies demonstrated metastatic disease to the liver, pancreas, left upper quadrant of the abdomen, and the L1 vertebral body (Fig. 1). A bronchial biopsy of the subcarinal lymph node yielded pathology results consistent with metastatic adrenocortical carcinoma, confirmed by immunostaining that tested positive for synaptophysin, inhibin, and Melan-A. The patient was known to have hypersecretory function of his tumors with associated hypercortisolism and hyperaldosteronism resulting in severe hypokalemia and muscle weakness that had required repeat hospitalizations.

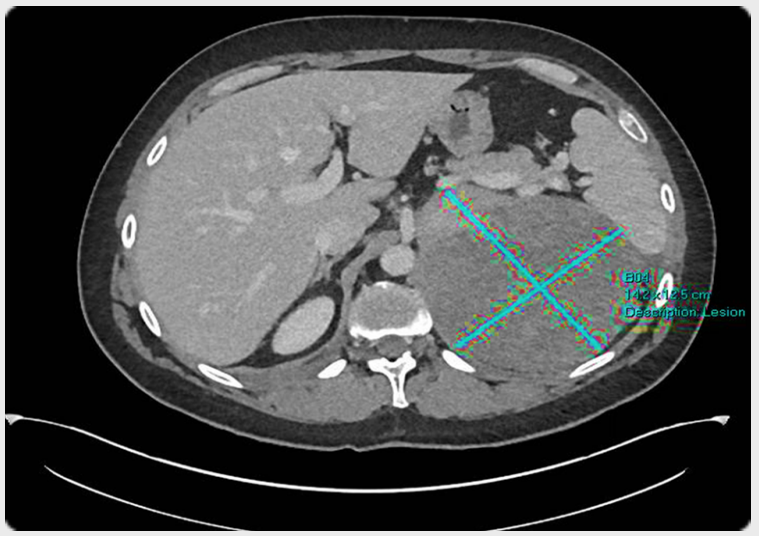

Figure 1A. Large ACC mass in the left adrenal fossa (14.2X12.9cm)

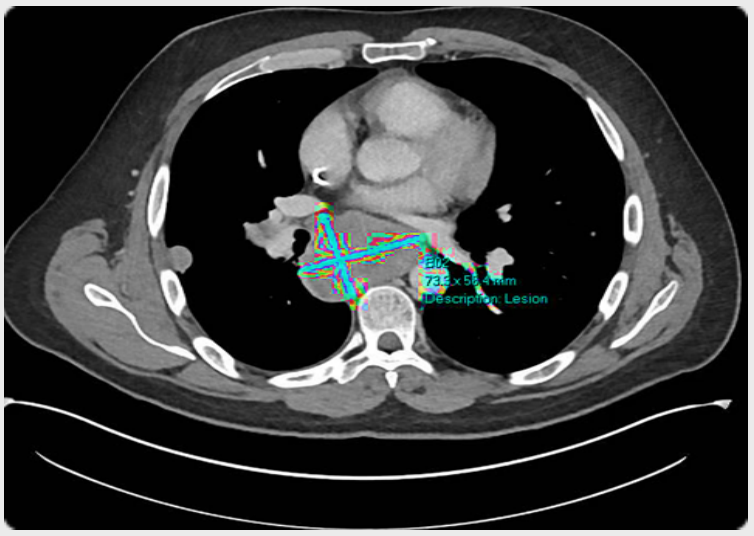

Figure 1B. Mediastinal subcarinal mass (7.3 cm x 5.6cm)

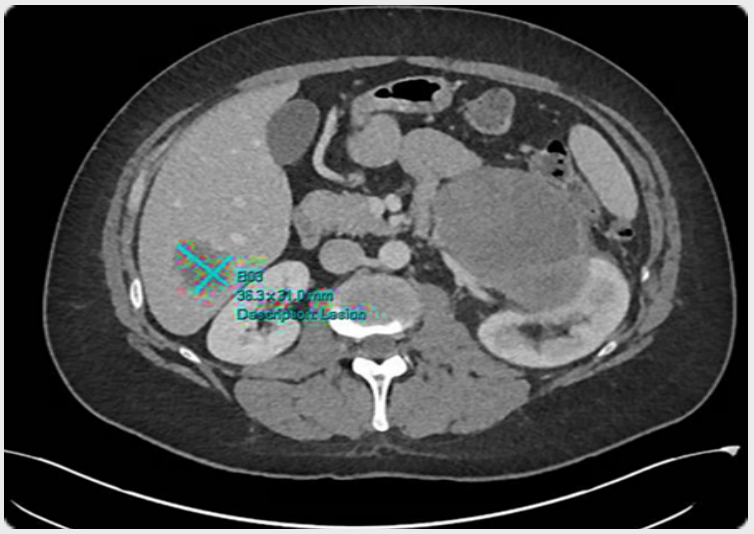

Figure 1C. Hepatic metastasis mass (3.6x 3.1cm)

The patient reported no respiratory or cardiovascular diseases, or recent infections. On examination, the patient exhibited clinical manifestations of truncal obesity, hypertension, severe hypokalemia, and muscle weakness, all presumed to be secondary to the hypersecretory components of his ACC. His urine and plasma cortisol and aldosterone levels were elevated; consistent with functional ACC. The remainder of his laboratory evaluations, including complete blood count (CBC), electrolyte levels, liver function tests, renal panel, electrocardiogram (EKG), and echocardiogram, were within normal ranges. Catecholamines and metanephrines were within normal limits.

The patient's hypertension was effectively managed with lisinopril, eplerenone, terazosin and carvedilol, while hypokalemia was managed with potassium supplementation and potassium sparing diuretics. The patient completed two cycles of systemic chemotherapy (etoposide, doxorubicin and cisplatin) and was currently undergoing treatment with mitotane, metyrapone, and osilodrostat to manage his hypercortisolemia in preparation for surgery.

The surgical team plan included an exploratory laparotomy with hepatic resection/ablation of liver component, cholecystectomy, and en bloc resection of mass and adjacent kidney, adrenal gland, distal pancreas, spleen, and left diaphragm. The expectation was that this resection would reduce his tumor burden to potentially improve efficacy of further therapies and obtain control of his primary ACC. The thoracic component of his disease was to be addressed with a future surgical procedure. General anesthesia was induced, and the patient intubated and placed on volume-controlled ventilation. Invasive monitoring included an arterial line, an internal jugular vein (Cordis 9F catheter), and advanced hemodynamic monitoring (Edwards Lifesciences LLC, HemoSphere Advanced Monitor) to facilitate continuous intraoperative assessment of various derived hemodynamic parameters.

A rapid transfusion system (Belmont Medical Technology) was connected to the Cordis catheter to facilitate intraoperative blood transfusions, medication administration, and central venous pressure measurements. Management of intravenous fluids and blood products was conducted based on estimated blood loss, urinary output, arterial blood gas results, and thromboelastography findings (TEG 6s, Haemonetics), in conjunction with the department's realtime hemodynamic monitoring protocols (blood pressure, heart rate, cardiac pre/afterload and contractility parameters, oxygenation and ventilation) guiding goal-directed fluid therapy IV fluids and blood product replacement.

The surgical team had mobilized the liver and resected a large lesion in segment 6 and several liver metastases around segment 6. Two and a half hours into the case when the surgical team was ablating the smaller liver lesions, the patient exhibited a sudden rise in peak airway pressures and significant hypotension. The surgical team immediately released the Pringle maneuver and returned the liver to its anatomical position.

A 'shark-fin' capnography pattern was observed, and inspiratory/expiratory wheezes were auscultated; On skin assessment, there was no evidence of flushing, urticaria or angioedema. Tidal volume (TV) and minute ventilation (MV) decreased from 650 mL to 67 mL and from 7.6 L/min to 0.9 L/min, respectively, airway peak inspiratory pressure (PIP) and mean airway pressure (Pmean) increased, and arterial blood pressure and systemic vascular resistance (SVR) dropped from 1000 to 380 dynes/sec/cm2 despite a normal or increased cardiac index (CI maintained 3-3.7 L/min/m2). The fraction of inspired oxygen (FiO2) was increased from 0.6 to 1.0. The anesthesia provider called for additional help as mean arterial pressure (MAP) continued to decline to 27 mmHg (Table 1).As a precautionary measure, the surgeon implemented a right side 28Fr surgical chest tube for possible tension pneumothorax, The surgeon noted that there was no audible rush of air and there were significant adhesions appreciated in right pleural cavity secondary to patient's metastatic disease that would have prevented lung from collapsing.

Table 1 Intraoperative vital signs, ventilating and hemodynamic parameters changes.

	09:15	10:15	11:15	12:15	13:15	14:15	15:15
MAP (mmHg)	80	78	27	70	78	82	81
HR (bpm)	78	82	90	100	98	95	90
POX (%)	98	98	98	98	98	98	98
EtCO2 (mmHg)	33	34	32	33	31	30	30
CVP (mmHg)	10	22	38	24	23	15	23
Temp (C)	36.4	36.4	36.7	37.1	37.1	36.9	36.8
Tv (ml)	695	643	67	585	647	644	647
RR (bpm)	12	12	12	14	14	14	14
Pmean (cm H2O)	9	8	27	12	13	10	12
MV (L/min)	7.7	8.1	0.9	9.4	10	9.4	8.9
PIP (cm H2O)	27	23	47	26	29	21	25
PLAT (cm H2O)	23	18	?	17	20	19	23
SVV (%)	8	12	18	10	9	8	9
CI (L/min/m^2)	3.9	3.5	4. 5	4.8	3.7	3.8	3.7
SVR (dynes/sec/cm^2)	722	630	289	471	520	631	710

Given the significant increase in airway peak pressure and hypotension unresponsive to fluid administration, blood transfusion, phenylephrine, norepinephrine infusion, and vasopressin bolus, the possibility of acute bronchospasm and vasoplegic syndrome were considered. The surgery was halted while anesthesia providers administered a series of medications including 100 mcg of epinephrine, 20 mg of famotidine, 25 mg of diphenhydramine, and 25 mg of methylene blue, and albuterol via an inhaler attached to the endotracheal tube. A flexible bronchoscopy placed through the endotracheal tube revealed no displacement, kinking, mucus plugging, or significant airway edema. Within five minutes of these interventions, the patient's mean arterial pressure (MAP) returned and stabilized at 70-80 mmHg, bilateral airway sounds were equalized, and wheezing resolved. Tidal volume, airway pressures, and advanced hemodynamic parameters similarly returned to baseline levels.

The treatment was discussed with the surgical team and the surgical team proceeded with a partial hepatectomy, cholecystectomy, resection of a mass in the left upper quadrant, splenectomy, distal pancreatectomy, and left nephrectomy/ adrenalectomy (see Fig. 2).

Figure 2. En block resection of ACC tumors and confirmed primary and met ACC by pathology report postoperatively

Throughout the surgery, the patient's condition was continuously assessed utilizing advanced hemodynamic monitoring techniques. Pressor administration included norepinephrine at doses ranging from 4 to 10 mcg/min and phenylephrine at 20 to 40 mcg/min to maintain systemic vascular resistance (SVR) and mean arterial pressure (MAP). Intravenous fluid infusion and blood product replacement was guided by hemodynamic monitoring, ABG, and TEG, and directed. The estimated blood loss (EBL) was 5000 ml, and urine output (UOP) 800 ml. Fluid replacement consisted of intravenous crystalloid 8500 ml, 25% albumin 700 ml, packed red blood cells (PRBC) 15 units, fresh frozen plasma (FFP) 10 units, platelets 20 units and cryoprecipitate 20 units.

Postoperatively, the patient remained intubated and ventilated and was transferred directly to the Intensive Care Unit (ICU) with a norepinephrine infusion. Initial post-operative lab results upon arrival, including ABG, TEG, complete blood count (CBC), and electrolyte levels were within normal parameters. On post-operative day (POD) #1, the patient was extubated and weaned off of all pressors. Epidural patient-controlled analgesia (PCA) was used for effective post-operative pain management. The patient experienced acute kidney injury (AKI) that rapidly improved and on POD #4, the patient was successfully transferred from the ICU to the post-surgical care unit without any complications.

Case presentation 2

A 58-year-old female patient with recurrent functional adrenocortical carcinoma (ACC) despite multiple surgical resections, status post mitotane and radiation therapy was evaluated for an en-bloc resection. Five years prior, she was first diagnosed with a left ACC which resulted in a left adrenalectomy of a 7.1cm low grade ACC. Two years later, she had a local recurrence requiring a left partial nephrectomy of a 1.5cm high grade local ACC recurrence. After the surgery, the patient was started on adjuvant mitotane and required hydrocortisone for adrenal insufficiency. The patient's disease progressed with additional local recurrence at the left adrenal vein stump (Fig.3), which required resection and radiation therapy to the left adrenal bed (5000cGy over 25 fractions). Further disease recurrence necessitated the patient to seek a second opinion for an en-bloc resection.

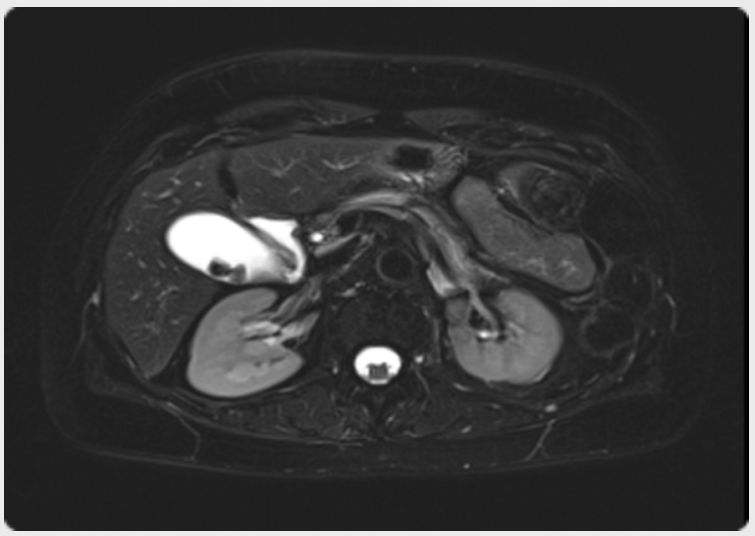

Figure 3. Left adrenal vein stump recurrence

The patient's history was significant for a 4.5 pack year smoking history but quit 20 years prior, hypertension, GERD, post-operative nausea and vomiting, post-menopausal, melanoma, adrenal insufficiency (on steroid therapy) with a history of adrenal crisis two years previously. The patient history was significant for hypertension that was managed with amlodipine, hydrochlorothazide-triamteren, and atenolol. and adrenal insufficiency with hydrocortisone. Other medications included estradiol to treat post-menopausal symptoms. The patient completed two cycles of systemic chemotherapy (etoposide, doxorubicin and cisplatin) and was currently undergoing treatment with mitotane, metyrapone, and osilodrostat to manage her hypercortisolemia in preparation for surgery.

Laboratory evaluations (complete blood count, electrolyte levels, liver function tests, renal panel, electrocardiogram, and echocardiogram, were within normal ranges. , while the

General anesthesia was induced, intubated and placed on volume-controlled ventilation. An arterial line, internal jugular vein (Cordis 9F catheter), and advanced hemodynamic continuous cardiac output monitoring (CCOM) (Edwards Lifesciences LLC, HemoSphere Advanced Monitor) was established. A rapid transfusion system (Belmont Medical Technology) was connected to the Cordis catheter to support intraoperative blood transfusions, medication administration, and central venous pressure measurements. Management of intravenous fluids and blood products was conducted based on estimated blood loss, urinary output, arterial blood gas results, and thromboelastography (TEG) findings (TEG 6s, Haemonetics), in conjunction with standard and dynamic hemodynamic monitoring protocols. Throughout the case, the patient blood pressure, heart rate, cardiac pre/afterload and contractility parameters, oxygenation and ventilation were continuously monitored. IVF and blood transfusion were administered based on goal-directed fluid therapy based on ABG and TEG results and real-time hemodynamic monitoring.

The surgical team performed a left upper quadrantectomy to include removal of the left kidney, distal pancreas, splenic flexure of colon, spleen, adjacent section of the left diaphragm, gastric fundus wedge resection and gallbladder (Fig.4). Four hours after skin incision, the patient exhibited significant hypotension unresponsive to phenylephrine, norepinephrine, and vasopressin. CCOM parameters demonstrated significant decrease in Systemic Vascular Resistance (SVR) (381) while maintaining a normal cardiac index (3.4) with the MAPs trending into the 20s (Table 2). A diagnosis of vasoplegia was presumed and 50 mg of Methylene blue was administered as a bolus over the course of one minute, with an immediate recovery of MAPs into the 110s. After stabilization of the patient's hemodynamic state, the procedure was resumed.

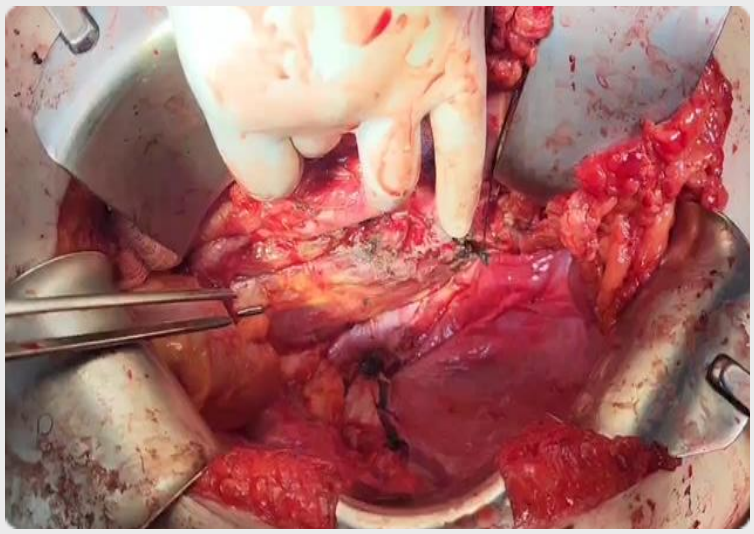

Figure 4. Surgical field of a left upper quadrantectomy to include removal of the left kidney, distal pancreas, splenic flexure of colon, spleen, part of the left diaphragm, gastric fundus wedge resection and gallbladder.

Table 2, Intraoperative vital signs and hemodynamic parameters changes.

	11:15	12:15	13:15	13:50	14:15	15:15
MAP (mmHg)	75	82	82	36	89	77
HR (bpm)	83	89	90	94	95	91
SVV (%)	13	13	15	15	12	8
CI (L/min/m^2)	3.1	3.0	3.6	3.4	3.2	3.7
SVR (dynes/sec/cm^2)	936	1086	765	381	1125	823

The patient's condition was continuously monitored utilizing advanced hemodynamic monitoring techniques. Intravenous infusion of norepinephrine (2 to 4 mcg/min) and phenylephrine (5 to 40 mcg/min) was initiated to maintain systemic vascular resistance (SVR) and mean arterial pressure (MAP). Blood products and fluid administration was guided by ABG and TEG analysis. The estimated blood loss (EBL) was 3500 ml, and urine output (UOP) was 3700 ml. Fluid replacement consisted of intravenous crystalloid 6800 ml, 25% albumin 400 ml, packed red blood cells (PRBC) 5 units, fresh frozen plasma (FFP) 2 units, platelets 13 units and cryoprecipitate 20 units.

The patient was extubated in the operating room and transferred to the ICU spontaneously ventilating on face mask requiring no vasoactive support. Initial post-operative lab results, including ABG, TEG, complete blood count (CBC), and electrolyte levels were within normal parameters. Epidural patient-controlled analgesia (PCA) was used for effective pain management and the patient was discharged from the ICU on POD #2 to the medical-surgical unit without event.

Discussion

ACC is an exceedingly rare and highly lethal tumor originating from the adrenal cortex, categorized into either a functional or non-functional clinical presentation [1-5]. The overproduction of hormones by a functional ACC is primarily responsible for conditions such as Cushing's syndrome and Conn's syndrome [1, 5, 9]. In both case presentations, despite elevated cortisol and aldosterone levels resulting in hypertension and fluid retention, symptoms were effectively managed, and their conditions remained stable prior to surgery.

The first patient experienced acute bronchospasm during tumor manipulation and resection of the large, functional, metastatic left adrenal ACC. The intraoperative wheezing, decreased tidal volume, and increased peak and plateau airway pressures are consistent with the onset of bronchospasm. Bronchospasm can be triggered by numerous factors, including hypersensitivity reactions (such as those associated with anesthesia and surgical stimuli), medications, or pre-existing medical conditions, potentially via the parasympathetic nervous system or immunologic-inflammatory pathways [10-15]. While the onset of intraoperative bronchospasm is known albeit infrequent, to our knowledge, there has been no prior report establishing an association between the surgical resection of ACC and acute bronchospasm [12, 15]. Our observation suggests that the bronchospasm may have been associated with this patient's primary functional ACC. A thorough differential diagnosis was undertaken to exclude potential causes related to the patient, surgery, and anesthesia, including a history of asthma, infection, anaphylaxis, surgical stimulation, and equipment-related issues. It is plausible that a sudden release of vasoactive substances and inflammatory mediators from the surgical manipulation of the ACC contributed to the acute bronchospasm. Immediate interventions included increasing the oxygen concentration to 100%, manual bag ventilation to evaluate pulmonary compliance, and deepening anesthesia to mitigate the bronchospasm. A low-dose intravenous bolus of epinephrine,

antihistamines, and β2-adrenergic agonists (albuterol) administered via the endotracheal tube resulted in successful resolution of the bronchospasm within minutes, with airway pressures and tidal volume returning to baseline prior to the conclusion of the surgery.

Vasoplegic syndrome (VPS) is a rare, presenting as a rapidly deteriorating state condition associated with persistent hypotension and vasodilation with normal or elevated cardiac output, and involving inflammatory responses and dysregulation of vasoactive substances [16-18]. VPS is more common and occurs more frequently in cardiac surgeries, as well as in cases of anaphylaxis, sepsis, certain medications, as a result of pre-existing patient comorbidities, but is exceptionally uncommon during the resection of primary and metastatic ACC [19]. In both cases with functional ACC, surgical manipulation and resection of the liver metastatic tumor led to a sudden drop in blood pressure, low systemic vascular resistance and elevated cardiac output, with no evidence of hemorrhagic, septic, cardiogenic, or anaphylactic shock. Despite adequate intravenous fluid administration and prompt boluses of phenylephrine, norepinephrine, epinephrine, and vasopressin, normal mean arterial pressure could not be achieved. Ultimately, only the administration of methylene blue intravenously resulted in the rapid restoration of mean arterial pressure, allowing the patients to tolerate the surgical procedure. Postoperatively, both patients continued to receive intravenous fluid resuscitation due to the capillary leak syndrome but recovered without further complications.

It is acknowledged that while catecholamines (such as epinephrine, norepinephrine, phenylephrine, and dopamine) are typically considered first-line treatment for vasoplegic syndrome, the early introduction of non-catecholamine agents (including vasopressin, methylene blue, and vitamins C and B12, as well as steroids) may be lifesaving when used in conjunction with catecholamines and intravenous fluid resuscitation to maintain mean arterial pressure and ensure adequate organ perfusion [16-18, 20, 21]. The administration of methylene blue (MB), a water-soluble dye and nitric oxide (NO) free radical scavenger, has been reported to effective in treating vasodilatory shock [22]. A systematic review and meta-analysis by Cong-Cong Zhao et al [22] has shown that the administration of the combination of MB and vasopressors improved survival and lowered lactate levels by improving blood pressure while lowering the need for vasopressors. Our experience supports the use of MB administration intraoperatively in the patients with functional ACCs undergoing surgical resection that are hypotensive and unresponsive to first line agents.

Though the specific etiology and triggering factors of these patient's episodes remain uncertain, three potential reasons for the occurrence of vasoplegic syndrome are proposed: 1) the preoperative administration of alpha and beta-blocker antihypertensive medications may heighten the risk of developing vasoplegic syndrome [16, 23]; 2), the use of preoperative cortisol inhibitors may diminish cortisol levels and alter vascular tone and responsiveness to vasoconstrictors [20, 21]; and 3), ACC can produce a diverse array of vasoactive substances, including local inflammatory growth factors and cytokines, which may play a significant role in promoting vasodilation and triggering vasoplegic syndrome [11, 14]. These factors may contribute to a decreased affinity of catecholamine receptors to vasopressors and/or increased nitric oxide production, resulting in vascular hypo-responsiveness [16, 24].

Conclusion

ACC is a rare and devastating malignancy that portends a poor prognosis. Surgery remains the most effective curative/debulking treatment for ACC. It is paramount that, during surgery, anesthesia providers are cognizant that ACC clinical manifestations tend to be highly variable. Most importantly, while rare, intraoperative anesthesia and surgical stimulation might cause acute bronchospasm and vasoplegic syndrome from various possible risk factors, including vasoactive substances secreted from ACC. Anesthesia providers should differentiate various triggering factors, and promptly use catecholamine and non-catecholamine vasopressors to maintain patient stability. MB and other drugs for the treatment of VPS should be considered if the patient continues to experience profound, refractory hypotension.

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Case Report (病例报告)

Managing Spontaneous Echo Contrast in the Descending Aorta of a Patient Undergoing Mitral Valve Transcatheter Edge to Edge Repair

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The presence of thick spontaneous echo contrast and overt thrombosis in the intracardiac chambers and aorta is consistently linked to poor outcomes. In such cases, administering epinephrine and heparin can help restore microcirculatory flow, protect organ function, and improve overall prognosis. 1 Additionally, transcatheter edge-to-edge repair (TEER) increases forward stroke volume and cardiac output, reducing the likelihood of future spontaneous echo contrast in the descending aorta (SECDA). This report presents the first documented case of an incidental finding of spontaneous echo contrast during a TEER procedure, which was successfully managed with heparinization, allowing the procedure to proceed without complications.

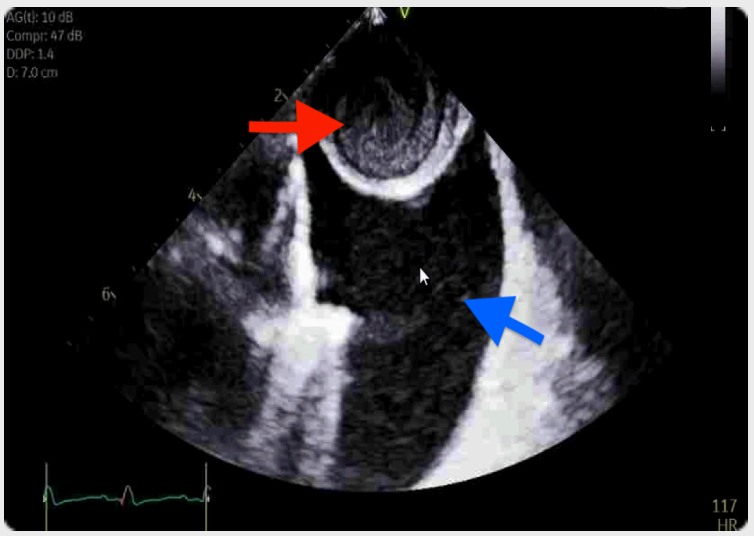

Figure 1. Spontaneous echo contrast in the descending aorta (SECDA)

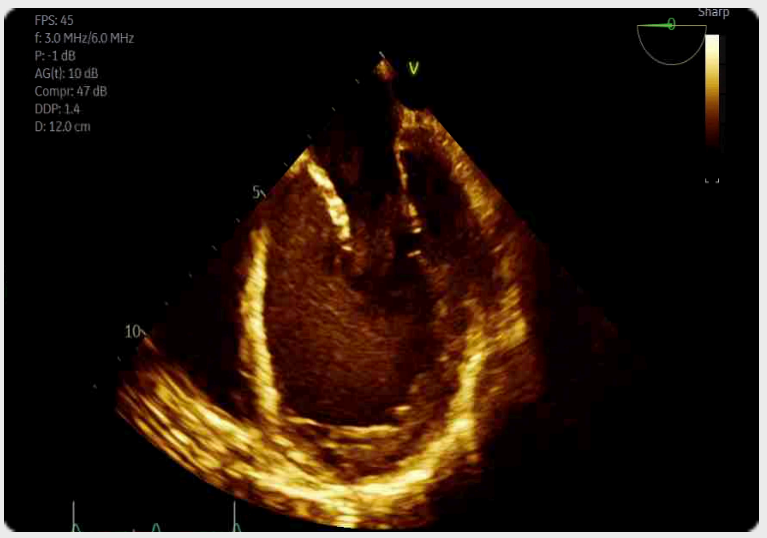

Figure 2 Severely dilated LV cavity

Case Presentation

This is a 74-year-old male with a medical history significant for ischemic cardiomyopathy with left ventricular heart failure with reduced ejection fraction with severe mitral regurgitation. He was scheduled for a transcatheter edge-to-edge repair (TEER) on September 19, 2024, under planned general anesthesia. Post-induction transesophageal echocardiography revealed SECDA (Figure 1), and severely dilated LV cavity with left ventricular ejection fraction (LVEF) about 10% (Figure 2). Functional severe mitral regurgitation (MR) was also identified by 2D and 3D view (Figure 3). Upon identifying severe LV global hypokinesis with thrombosis and DASEC, doppler ultrasound measured a cardiac output (CO) of only 1.3 L/min (Figure 4). To assess if the patient retained some viable cardiac reserve, an epinephrine infusion was initiated at 0.03 mcg/kg/min.

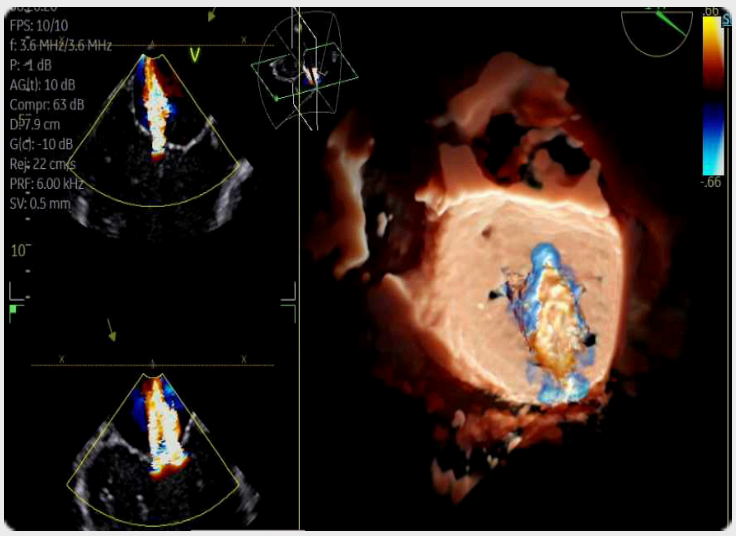

Figure 3. Functional severe MR identified by 2D and 3D views.

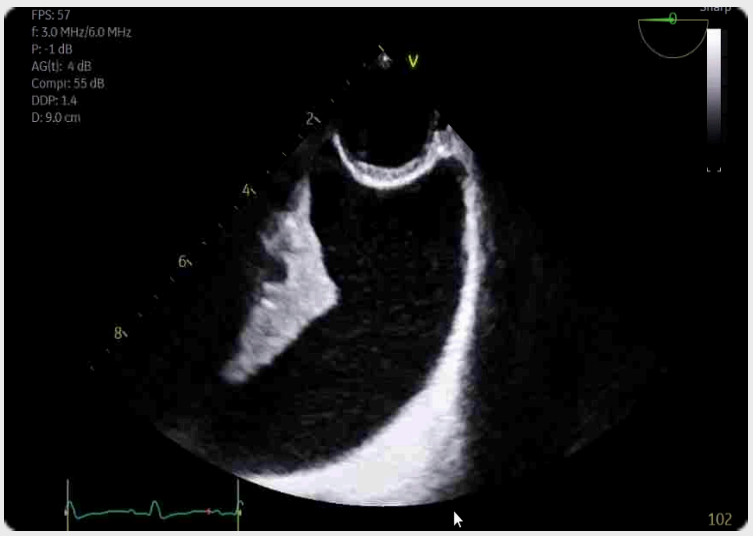

Figure 5. Decreased SECDA post Mitral-Clip.

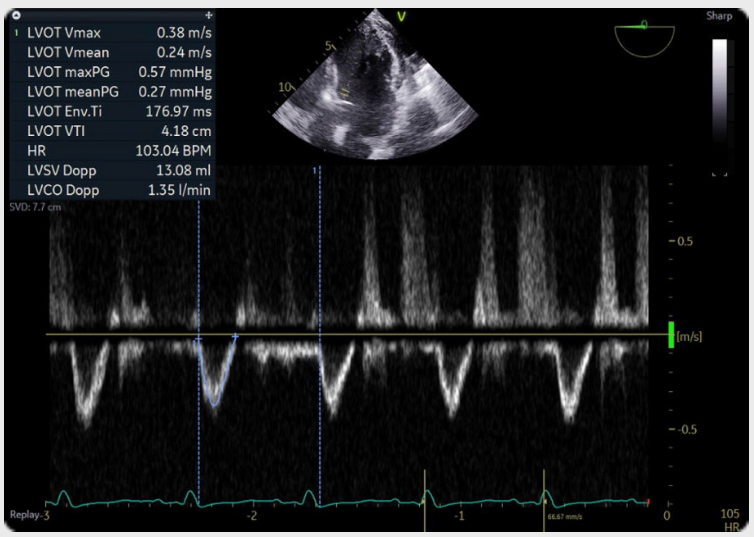

Figure 4. Cardiac output (CO) calculated by Doppler ultrasound.

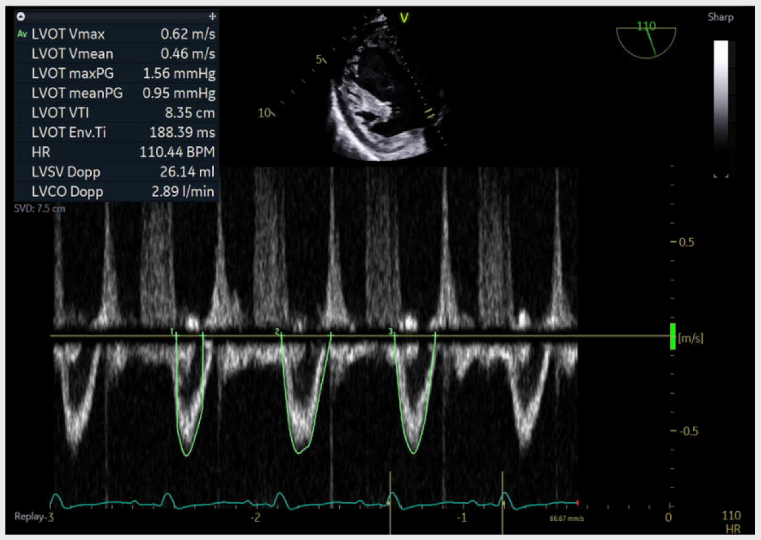

Figure 6. Improved CO post Mitral-Clip.

Simultaneously, the interventional cardiologist performed both right heart catheterization (RHC) and left heart catheterization (LHC) to evaluate ventricular filling pressures. Given the patient's demonstrated cardiac reserve and SECDA, it was concluded that proceeding with the TEER and heparinization would provide greater benefit than canceling the case. If the patient lacked myocardial reserve or the left ventricle could not tolerate an increased CO following TEER, the option of an intra-aortic balloon pump (IABP) was considered. The MitraClip was successfully placed and the SECDA was diminished (Figure 5). The patient was weaned off epinephrine with improving CO (Figure 6) and extubated without complications. And arterial blood gas analysis before and after the procedure indicated normal lactate levels. The patient was subsequently discharged without complications.

Discussion

Spontaneous echo contrast (SEC) appears as an echo-dense shadowing and must be distinguished from high-gain artifacts, typically evaluated using transesophageal echocardiography (TEE). SECDA has been associated with various clinical conditions, including altered hemostatic parameters,2,3 increased aortic diameter, reduced intravascular flow velocity and shear rate, and aortic plaques. 4 Additionally, it has been linked to left ventricular dysfunction and arrhythmias.5 These findings support the hypothesis that SECDA serves as a marker not only for embolic stroke related to atrial fibrillation but also for ischemic stroke influenced by multiple risk factors.6

Prior studies have suggested that patients with SECDA should undergo intensified medical management and lifestyle modifications to address cardiovascular risk factors.7 It is recommended to administer heparin to help treat SEC and prevent potentially catastrophic thrombotic events. A case report

describes DASEC in a patient who experienced cardiac arrest. During resuscitation, TEE revealed severely depressed biventricular function along with thick SEC in the ascending and descending thoracic aorta. In response, 10,000 units of unfractionated heparin were administered intravenously, and inotropic therapy was initiated. The SEC gradually resolved, biventricular function improved, and the patient made a full recovery, ultimately being discharged from the hospital a few days later.1 This could enable the early initiation of targeted therapy, leading to improved outcomes. As to this patient after initiation heparinization and proceed with TEER the patient had a favorable outcome and no more DASEC. The authors believe that the timely administration of heparin with the appearance of SEC in the left ventricle and ascending and descending aorta prevented further frank thrombus formation and a catastrophic outcome. Heparin administration in this situation may help in restoration of flow in the microcirculation and aid in organ protection and improved outcome.8-11 To the best of the authors' knowledge, this is the first report of continue to perform TEER with SECDA and LV thrombosis and the administration of heparin and its complete resolution and a favorable outcome. The gradual clearing of the SEC may be attributable to multiple factors in this case. Heparin administration may have improved the myocardial microcirculation by preventing thrombus formation and improving myocardial function. Increased stroke volume and forward flow with succeeded increasing shear rate cleared the DASEC.

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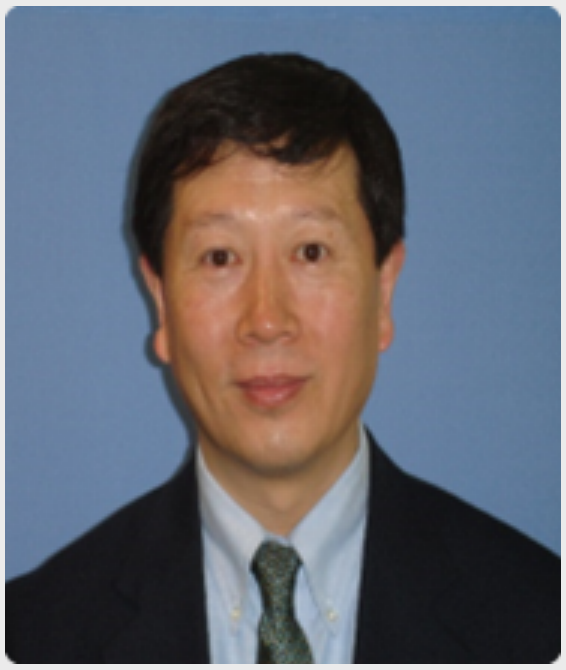
王海明医生回忆录 （续 9）

15. 子女不学父之过　父母当需好配合

1994，我们从波士顿迁居来K城前，波士顿 Baldwin 小学老师叮嘱我们：美玲的英文和算术远高于同级水平，应该跳一级。我们去找卡可伦校长，可他说：虽然这孩子数学和英文超众，但她的心理发育则未必也超前。总之不鼓励学生跳级。附近这所学校最好，只好依了老校长。可是，课堂上美玲的确感觉课程太简单。她继续学习钢琴，又学了小提琴，冬天参加了滑雪队。课余努力学习中文。



王海明 (10/1/1959 -08/23/2019) 1983年毕业于北京医学院，1985年赴美。1994年从Boston Elizabeth and MGH完成麻醉科培训。于2002年创立CASA 并出任首任会长，也曾任CASA Bulletin责任编辑。为CASA和中美麻醉医学的交流作出了杰出贡献。

美玲和美慧学习同样的中文课本。我从中国驻纽约总领事馆要来一套《 语文 》，共12本。美玲全学了。美慧学了部分。我和丽又去中国城书店，买来中国暨南大学编写的《 语文 》12大本。美玲和美慧均学一遍。学中文，寒暑假期：每日学一课。我先朗诵一遍（她俩看着书）；接着，我念一句，她俩跟着念一句。读罢，我问她俩是否有生字和词，是否有不懂的句子。我仔细解释，让她们给生字、词标上拼音（我已教会她们汉语拼音，她们已会查汉英字典了）。接着是看课文后的作业题，是否会做？没有问题了就可去抄写课文了。寒暑假每天学一课，开学期间，每周六学一篇课文。养成习惯了，届时我一喊：美玲、美慧学中文了。她们会放下手中一切，拿起中文作业和《语文》书跑到饭厅桌前，来上中文课。的确，美玲和美慧是比较听话的孩子。

下节课：我要请美玲或美慧读她们抄写的课文，我看着书，她们读着、看着抄写的课文。看一遍，不如读一遍，更不如写一遍 ！检查好抄写的课文，再查看课文后的各种作业是否完成得好。

复习好上一节课，马上学新的课文。日复一日，月复一月，年复一年。此生，我最自豪的成就之一，就是在家教 “ 中文私塾 ” 成功。两个孩子不仅会说汉语，而且认识许多汉字，因为她们抄写过许多课文，做过许多作业。在哈佛、宾大，她们仍学习中文，老师们多来自北大、北京师范大学。

美玲高中学习法语四年；美慧高中学西班牙语四年；每人通晓三种语言。二人均弹钢琴，拉小提琴，冬天滑雪。美玲曾反复为我弹奏 《留学生之歌》。

在波士顿，美玲五岁开始学弹钢琴，首位启蒙老师是乐为，她毕业于上海音乐学院，当时在波士顿新英格兰音乐学院学习钢琴。她父母原在上海电影制片厂乐团工作。一家三口住波士顿城西Waltham。乐为每周六上午教美玲一小时弹钢琴，收费20美元。

1996年，美慧从幼儿园要升小学了，卡可伦校长刚退休了。我去小学见新校长杰弗，简介了美慧的水平，请校长考虑是否允许她跳升一级。新校长开明，他说：只要能通过测验便可跳升。我说：好，请您们测试她。校长认真地组织了测试小组 ，测试美慧英语为二年级末，算术为三年级初，还有一项心理测试，美慧也通过了。我问美慧：心理学测验题是哪些方面？美慧说大多忘记了。我追问：仔细想想，说出一个考题也可。美慧沉思片刻说：有一题问：每次寄信前，为何要贴足邮票 ？ 我问：你是怎样回答的 ？ 美慧说：The mailman can get paid . 我说：正对！王美慧通过了各项考试从K-2 跳升至二年级。即使这样跳级，美慧仍觉得二年级课程太简单。

1991年冬，圣诞节前，我们一家四口聚在壁炉前开会。我对美玲说：妈妈和我计划送美玲你去菲利普读私立高中。美玲问：为何 ？ 我说：那高中是全美最好的，你去那里会受到比K城公立高中更好的教育，将来进常青藤大学的机会要高很多 ！ K城高中每

年入学600人，四年后只有300余人毕业。这只是美国平均公立高中水平。菲利普则是象牙塔式的顶尖高中，那学校好似大学，每班只有十一、二个学生。老师多是博士或硕士。我和妈妈愿意支付每年数万美元的高中学费。住在那样的学校，你将会认识许多来自世界各地的优秀学生，建立高质量的人脉，极有利你今后事业的发展。

美玲思索片刻说：我不想去。我急问：为何 ？ 她说：我们刚从波士顿转学来，刚与老师和同学们熟悉，感觉很好。您们希望我进常青藤大学，我将来一定能进！我说：你要知道纽约市、波士顿、华盛顿DC、休士顿、芝加哥、西雅图、旧金山、洛杉矶、圣地亚哥等许多好学校里有千千万万的好学生 ！ 请你再考虑考虑。美玲充满信心地说：我一定能入常青藤大学！我告诉她：希望她去读哈佛大学，哈佛医学院。然后，去斯坦福医学院做住院医师，毕业后就留在加州工作，那样妈妈和我就可去加州退休了。如果你做不到，希望美慧能做到。

进入高中后，美玲加入了好学生群，有优秀的学长帮助。美玲进发出极大的热情参与学生会活动。彰显出她杰出的领袖才能。我曾问美慧：姐姐到底有何超众的优点。美慧骄傲地告诉我：姐姐学习好；经常志愿帮助老师和同学，师生们皆爱戴她。她是：1）President of Student Government ；2）President of honor Society; 3) President of Science Olympia; 4) Captain of High School Girl Tennis Team; 5) Orchestral Leader and The First Violin; 6) Vice President of French Club; 7) She organizes Homecoming; 8) She was elected “ May Queen 被选为五月花葵” ；9) She speaks fluently Mandarin，and some French; 10) She is beautiful !

美慧对美玲的总结令我欣慰。为了扩建我院癌症大楼，美玲和同学们去商店和机关募捐，将一张五万美元支票交与我院CEO：汤姆迪。高二了，要参加 SAT考试。张丽注意到：美玲回家来很少复习资料备考，很担忧，“请爸爸管一管 ”。下班后，我去高中网球场看美玲和同学练球，与外来的球队比赛；或乘校车去外校赛网球。美玲两只眼和牙齿还白，全身几乎与非裔黑人一样了（日晒所致）。她若打球，队友们不断喝彩；其她队员打球时，美玲则是拉拉队长，忙得不亦乐乎。根本不看书!

晚饭后，我对美玲说：你必须暂停网球活动，你要抓紧时间，认真准备大学考试 - SAT。美玲说：打网球不会耽误考SAT。我说：非也。一下课，你就去网球场，直到天黑了、你也精疲力竭了才回家来。每日，你浪费了多少宝贵复习时间 ！ 你必须暂停网球 ！ 她说：不 ！ 我说：请你将手机和车钥匙放在电话旁的抽屉里。这均是妈妈和我给你提供的。高考分太低，常青藤大

学根本不会看你的申请 ！ 等考试过后，你可继续打网球！美玲果断地将手机和车钥匙放入抽屉里。次日凌晨，拉着美慧妹妹，和另外邻居二女同学坐校车上学去了。我一看对张丽笑道 ：嗬，有本事 ！ 看她能坚持多久？

当晚，我下班一进家门，美玲客气地对我说：爸爸，咱们需要谈判 ！ 没有手机和汽车严重影响我这学生会主席的活动！我说：手机和车钥匙就在抽屉里，未锁。你可拿去用，但是一定要暂停网球。她未语。次日早晨，开车载着美慧和邻居二女同学上学去了。

次日，我下夜班，在医院忙到中午才回家。近家门口，看到高中英语课威廉老师（高中女子网球队教练员）在我家门前踱步。我赶上前去，邀请他进屋坐。他犹豫地说：谢谢你，我只有几句话要对你讲。我说：可以。老师说：“ Margaret （美玲）不能停止网球训练和比赛！她是我们球队的灵魂人物！我求您了 ！” 等片刻，我说：老师过奖了。美玲球技一般，我已去球场看过了。之所以美玲妈妈和我坚持要她暂停网球，是因为大学考试在即，我们希望她能进最好的常青藤大学。我们是从中国北京来的，深知高考的重要。请您原谅，美玲高考过后可以继续网球训练。我们家将邀请您和所有队员来聚会。我们对Margaret 寄予厚望 ！ 听罢，威廉老师低头一语未发默默地走了 ！ 我觉得：我尽了父亲的责任 ！ 倘若疏忽，日后美玲入学不理想，我们会无比地遗憾和自责 ！

2004年12月14日傍晚5点，我在家忽听美玲大喊着奔我而来，“爸爸，我被哈佛大学录取了 ”！ 我问：你怎已知晓 ？ 美玲兴高采烈地说：我收到了email 。我说：会不会你的同学给你开玩笑呢 ！ 请你给我哈佛大学招生办的电话，明日我询问一下就可确定。次日上午，我电话哈佛大学招生办：请问我的女儿Margaret Meiling Wang 是否已被贵校录取 ？ 那位女士用优美动听的声音

说：很抱歉，我们不告知任何学生的信息，除非您女儿亲自来电话。我马上请美玲自己去问。几分钟后，美玲来电话：我的确已被哈佛大学录取。我说：尽快告诉妈妈和妹妹。张丽仍在诊所工作，闻知后，喜极而泣，那个自豪 ！当晚，喜讯传去中国，亲友们纷纷祝贺 ！ 美玲申请的是 “ Early Action ”。决定去哈佛大学，也就不必再申请其它大学了 ！

两年后，哈佛大学暂时取消 “ Early Action ”。我们鼓励美慧学习口腔医学。全美牙科最好的大学是宾夕法尼亚大学（ U Penn ）。美慧申请了 ，很快就被录取了（Early Decision:被录取后必须要去），也不需要申请其它大学了。

美玲和美慧的数学SAT均是满分，英语接近满分。进入大学后，二人均继续学习中文。一次，美玲在中国与几位青年谈话，一位美国小伙子以为美玲生长于中国，连赞美玲的英语很好。

小的时候，美慧学习中文态度不及美玲认真。抄写课文，有时竟然会漏掉一整行。一旦指出，立即补抄。美慧有她的长处：对美玲总是称“ 姐姐 ”，从未喊过美玲。在学校里，师生们常指Linda Wang 就是 “ Margaret ’s little sister ”。

美慧为了冲出姐姐的影子，做了许多尝试（除了与姐姐一样弹钢琴和拉小提琴之外）：田径长跑不杰出；尝试参加学校拉拉队，可我不允许她登高冒险；自学弹吉他；学了吹闪亮的长笛；学了 Voice （美声、唱歌剧）。一下午，我开车送她去学习小提琴，问她：声乐有进步吗 ？ 她说；有进步。接着为我唱了一段意大利歌剧。其实，美慧英文写作比姐姐强。

一日下午，我下夜班。美慧请我送她去学芭蕾舞课。我问：你是否已完成中文作业 ？ 她说：尚未。我说：我不送你去学芭蕾舞了。我要看着你写中文作业。她急得眼泪直流：芭蕾老师要责怪我了！我说：你不及时完成中文作业，我不高兴。她只好含泪抄写语文课文。自此，美慧再未忘记中文作业。

妈妈（张丽, Lily Zhang）是慈母，还时常给两个女儿手里塞（小量）现金。我们教会了孩子唱《世上只有妈妈好》。可当张丽管教孩子为难时，她会单独求助于我。我则去唱白脸（虎爸），有时会得罪孩子。比如：让美玲暂停网球；我曾让美慧因错，面壁思过半小时（面墙而立），姥姥悄悄告诉美慧：你爸爸忙其它事去了，你可坐一小会儿。姥姥要讨好美慧，也许姥姥心疼她 ，毕竟只有7岁。可美慧却诚实地说：再过一小会儿就到半小时了。后来，我岳母对我讲起此事，我反而佩服美慧!

美玲多年来，一直积极参与哈佛校友会活动：无论是在曼哈顿，还是加州斯坦福。每年，美玲均要春天赶回母校参加毕业典礼。由于表现好（也可能是运气好 ）她竟被选为哈佛大学校友会会长（2018-2019, President of Harvard Alumni Association，HAA ）。

16. 阅读令我如痴醉　打乒乓其乐无穷

我除了每月阅读麻醉专业期刊还经常与同仁交流，而且每年都去参加专业年会，力求麻醉知识和技能与时俱进。当两个孩子离家后，妈妈张丽若有所失。而我极欣喜。我又自由了。不用开车送孩子去学钢琴、小提琴、芭蕾舞和其它校内外活动。我开始打乒乓球（没有球友就游泳）。几年来，有两个较固定的球友伙伴。

林先生，祖籍福建农村，十余岁时经香港来美国。一头扎入曼哈顿唐人街学厨师。攒了一些钱后，来纽约上州开外卖餐馆。他负责厨房，年轻的妻子管前台。孩子们放学后，在餐馆做作业或帮工。服务态度好，出菜快，生意还好。又攒了钱，把餐馆房产买获。再攒些钱，自己开一洗衣店，将餐馆租给老乡。洗衣店获利较好，便租给另一老乡。他则去纽约市法拉盛白石桥旁买一民居，自己加门装修，出租给房客。如今，儿子是工程师。他成了我的球友。开始很长一段时间，他总不敌我。后来，去法拉盛同乡会找高手学习。前一阵子，我俩水平相当了。因为纽约市事情多，他迁去纽约市住了。我很思念他，他是位君子。

所幸，K 城有乒乓球俱乐部，每周四晚上均开馆打乒乓球。前几名高手多是白人。我寻得 Alex 作为新的球友。他的姥爷原是保加利亚驻意大利大使，在罗马乡下有别墅。后姥爷应召回国，赶上革命，因同情国王而被处死。妈妈毕业于罗马大学，嫁给一美国军官来了美国。他在纽约长大。读哈佛大学学建筑，可慢慢地移情绘画艺术。毕业后，为一哈佛大学艺术教授助教四年。又去芝加哥大学艺术学院读了硕士。回到曼哈顿下东区（低收入区）试着卖画为生，很快发现不现实，无名气，只好兼作管工，也修空

调。管工收入稳定，足可养家，两个儿子均是好孩子。妻子原是《华尔街日报》记者，她大学毕业后又读了两个写作学位。如今，他似乎已攒够了退休金。每周帮东助西，为朋友们修房，为教堂维修建筑。我俩球技水平相差无几，打球很开心。他长我几岁，可敬重我，我也同样珍惜与他的友谊。他是一位善者。经常打乒乓球，可以推迟脑痴呆。增强心肌储备。打球高峰时，我会汗流浃背，心率几乎加快一倍。运动之后，很有愉悦感。2007年11月，我组创“The Pingpang Association of North America Chinese Professionals，PANACP 北美华人专业人士乒乓球协会”，暂任群主。

我在中国学打乒乓球：每次回国，我均带二球拍和数球。中华大地，乒乓球台如满天繁星，争相闪烁。我曾在北京东单体育馆室内打乒乓球，那里高手如林。北京牛街，广安门外，西城区，朝阳区，海淀区等几处高档住宅区，我均以球会友。专找积分1500左右者打。偶遇高手，我先数银子，然后学招，年年进步。对方问我：何处贵干？吾答：悬壶济世！于是，就开打。国人普众多用飞鱼球，我总贡献“ 红双喜 ”；国人多用国产球拍，我用德、日蝴蝶。因我球技尚可，热情开朗又大方，球友多多，男女老幼皆吾友！大江南北，长城内外皆有球友。打球休息时，我则为友讲故事，古今中外，经典或演义，一个又一个，我手舞足蹈 ，两眼炯炯有神，口若悬河，引人入胜... 球友们既夸我博学，又夸我的球技，我呢，乐在其中！

讲到运动，一件事令我难忘。大学时， 临床课到北大医院。住在草岚子胡同，附近市区内 根本没有公共运动场地。一次， 我路过北京四中，进去参观，见该校有标准的足球场，大喜。立即找到该校体育老师，请求举办一次北医 - 四中足球友谊赛，我自荐为领队。回到宿舍楼，贴出告示：明日下午 4:00 PM将准时开赛。男生们高兴极了，纷纷摩拳擦掌，决定猛赢几个漂亮好球。是啊，大学高年级医学生对阵中学生，没有人担心会输 ！ 届时，参赛者众，均跃跃欲试 ！ 我这领队，看到我方豪情万丈，紧提醒众英豪：友谊第一，切勿伤小同学们 ！ 几分钟后，四中队进了一球，不久，四中队又进一球。 不对劲儿，我找来我方两位大侠咨询。明白人一指导，我立即看清场上攻守。四中队，人虽小，但训练有素，队员互相配合，成套的战略战术； 大学生们个个如猛虎，得到球后不传给其他队员，而是一路向对方球门冲 ！ 那四中队员看的真切，二人一起上前截堵，决不让大学生们靠近四中的球门。我方，得到球就一人带冲，均是独行侠。屡屡丢球，而当四中队反攻时，速度之快，个人球技之高竟令大学生们无奈。结局，比分 6 ： 0，中学生们轻松击败北医(北大医院)医学系和口腔医学联队。自此，我方体会 ： 何为兵败如山倒 ！ 战略战术及平时训练又多么重要！

WISONIC公司简介

华声医疗，成立于2013年，总部位于中国深圳；是一家拥有完全自主知识产权，集研发、制造、营销为一体的中国国家高新技术企业。 华声是全球POC专科应用领域一线品牌，以临床专业专科精准诊疗为基础，提供“专科专用”的产品和服务。 华声拥有“一核两翼”—— 其核心是云端医疗服务，两翼是生命信息支持、智能超声影像。 目前，华声与全球顶级医院共同建成培训基地，与多所院校建立技术前沿的联合实验室。华声致力于服务全球，聚焦全球中高端医疗用户；进口替代，走进国内400多家三级医院；出口升级，产品出口100多个国家和地区。 华声用智慧科技，呵护更多生命健康需求，让更多人分享专属生命关怀。

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指南针 (英文型号名： Navi S)： 秉承着“专科专用”的理念，经过大量的市场调研，了解了一线客户的痛点和需求，华声医疗于2016年推出了业内首款麻醉疼痛专用彩超——指南针。指南针以其19寸超大的全触屏设计、全面的穿刺解决方案，很好地克服了传统超声操作复杂、穿刺针显影不清等问题。通过近几年的市场推广、专家体验、学术合作等，在麻醉疼痛领域，目前在中国市场占有率稳居第一，在国际上也逐渐树立口碑。打破了进口品牌在相关领域的传统垄断局面。

北斗 （英文型号名：Labat SP）： 2019年，华声医疗推出了首款麻醉专科AI智能超声——北斗。作为一款高端彩超，北斗采用了HOLO BEAM全息平台，无需调节焦点，图像更清晰。同时，基于强大的硬件平台，北斗智能识别神经、血管及各类组织，配合专业的教学软件，使得超声下组织识别更为快速、简单，也极大地缩短了入门医生的学习曲线。此外，北斗的激光导航功能更是产学研合作的成果之一，创新地解决临床穿刺定位困难的问题。

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