



doi:10.7659/j.issn.1005-6947.2023.12.001  
http://dx.doi.org/10.7659/j.issn.1005-6947.2023.12.001  
China Journal of General Surgery, 2023, 32(12):1819-1828.

· 述评 ·

## 主动脉弓腔内修复术后卒中的防治研究进展

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### 摘要

传统开放性主动脉弓修复手术存在较高的围术期并发症和死亡风险。主动脉弓腔内修复术(EAAR)为开放手术高风险患者提供了微创治疗选择。然而,与传统开放手术相比,EAAR仍具有较高的卒中风险。分支技术是腔内治疗主动脉弓部疾病最具应用前景的技术之一,尽管其早期卒中风险略高于传统开放手术,但对于高风险患者来说,这样的风险是可以接受的。导致术后卒中的主要原因包括固体栓塞、气体栓塞和脑灌注不足。术前评估、围术期监测、药物预防和优化术中操作是预防EAAR术后卒中发生的关键策略。对于已经发生卒中的患者,及时诊断和评估、药物治疗和必要的手术干预是治疗的基石,而多学科有效协作对于改善患者的病情和预后亦尤为重要。目前,EAAR术后卒中的防治仍有很大的研究空间,因此,笔者就EAAR术后早期卒中的发生率、发生机制、危险因素以及预防和治疗策略方面进行论述,以期为临床工作提供思路。

### 关键词

主动脉, 胸; 血管内操作; 卒中; 围手术期

中图分类号: R654.3

## Research progress in the prevention and treatment of stroke after endovascular aortic arch repair

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### Abstract

Conventional open repair of the aortic arch is associated with a higher risk of perioperative complications and mortality. Endovascular aortic arch repair (EAAR) offers a minimally invasive treatment option for patients deemed high risk for open surgery. However, compared to conventional open surgery, EAAR still carries an elevated risk of stroke. Branch TEVAR represent one of the most promising techniques for the endovascular treatment of aortic arch diseases. Although the early stroke risk is slightly higher than that of conventional open surgery, such risks are considered acceptable for high-risk patients. The primary causes of postoperative stroke include solid embolism, gas embolism,

基金项目: 国家重点研发计划基金资助项目(2020YFC1107701)。

收稿日期: 2023-10-09; 修订日期: 2023-12-07。

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and cerebral hypoperfusion. Key strategies for preventing stroke following EAAR include preoperative assessment, perioperative monitoring, pharmacological prevention, and optimization of intraoperative procedures. For patients who have already developed a stroke, prompt diagnosis and assessment, pharmacological treatment, and appropriate surgical interventions form the cornerstone of treatment, and effective multidisciplinary collaboration is particularly crucial for improving the patient's condition and prognosis. Currently, there is still considerable research space in the prevention and treatment of stroke associated with EAAR. Therefore, the authors address the incidence, mechanisms, risk factors, as well as prevention and treatment strategies for early postoperative stroke following EAAR, to provide insights for clinical work.

**Key words** Aorta, Thoracic; Endovascular Procedures; Stroke; Perioperative Period

**CLC number:** R654.3

传统开放性主动脉弓修复手术是心血管领域最具挑战的手术之一。随着深低温停循环和选择性顺行脑灌注技术的发展,主动脉弓置换术相关的病死率和脑卒中风险大大改善<sup>[1]</sup>,并始终保持着其作为所有新技术必须对照的金标准地位<sup>[2]</sup>。然而,由于并非所有患者都能够耐受体外循环和深低温停循环操作,杂交主动脉弓修复手术以及主动脉弓腔内修复术(endovascular aortic arch repair, EAAR)应运而生<sup>[2-4]</sup>。其中以冰冻象鼻术为代表的杂交手术因通常仍需体外循环和低温停搏技术的支持,并不能算是真正意义上的微创,且目前也没有明确的证据支持该类杂交技术相较于传统开放手术在早期病死率和围术期并发症方面的显著优越性<sup>[5-6]</sup>;而以去分支技术创造足够近端锚定区进行胸主动脉腔内修复术(thoracic endovascular aortic repair, TEVAR)的I型和IV型杂交手术避免了体外循环,尤其是IVb型同时避免了开胸操作,实现了真正意义上的微创治疗,也为EAAR奠定了基础<sup>[7]</sup>。因此,严格来说,杂交手术可以被看作是传统主动脉弓开放手术向腔内手术发展过程中的一种过渡。

与传统开放相比,EAAR手术的围术期并发症发生率和病死率较低<sup>[8]</sup>。然而,术后脑卒中一直是限制该技术推广应用的一个重要并发症。研究<sup>[9]</sup>表明,EAAR术后卒中死亡风险是其他主要并发症死亡风险的10倍,这一问题在早期用于EAAR的外分支移植系统中很明显,这些装置显示出了令人失望的高卒中发生率和病死率<sup>[10]</sup>。随着器材设计及输送系统的改进,尤其是内分支模块化系统的研发,使得EAAR术后卒中风险得到了极大降

低<sup>[11]</sup>。本文介绍近年来关于EAAR术后早期卒中的防治研究进展,包括卒中的发生率、发生机制、预防措施和治疗策略等方面,以期为临床提供指导和参考。

## 1 EAAR及其术后早期脑卒中发生率

EAAR结合去分支、烟囱、开窗以及分支支架等技术能够为解剖学适合且不适合接受传统开放手术的主动脉弓部疾病患者提供微创治疗,特别是那些高开放手术风险或既往行开胸手术的患者<sup>[12-13]</sup>。传统的弓上去分支技术有一定的神经损伤(膈神经麻痹、喉返神经损伤)和淋巴损伤风险<sup>[14]</sup>;平行支架技术存在较高的“Gutter”相关Ia型内漏风险<sup>[15-17]</sup>;定制开窗往往需要较长的等待时间,对于紧急情况无法适用,且存在等待期发生严重不良事件的风险<sup>[18-19]</sup>;而原位开窗或预开窗技术则存在开窗撕裂扩大导致III型内漏、支架结构性移位导致分支闭塞、织物栓塞等风险<sup>[20-22]</sup>,同时原位开窗的快慢对于大脑短暂性缺血的耐受问题也是另一个挑战。因此,分支EAAR(尤其是内分支支架系统)被认为是目前腔内治疗主动脉弓部疾病的理想手段<sup>[23]</sup>。

目前大多数关于EAAR的研究都是病例系列研究,且仅有少数为大样本研究,加上各个研究纳入的患者基线特征相差较大,腔内干预及近端锚定区各不相同,其术后早期卒中发生率也从0%到42.9%不等<sup>[10, 24]</sup>。表1列举了最新的关于开窗、烟囱、分支等技术腔内治疗主动脉弓部疾病术后早期卒中和死亡的发生风险<sup>[11, 13, 25-27]</sup>。鉴于分支

EAAR在治疗主动脉弓部疾病中的应用前景,这里主要介绍分支EAAR相关的早期卒中发生率。2023年Spath等<sup>[13]</sup>发表的系统综述显示,锚定区位于0~2区的分支EAAR早期卒中发生率6.8%(95% CI=4%~11%),早期病死率7.2%(95% CI=4%~13%);Basha等<sup>[26]</sup>报道,锚定区位于0区的分支EAAR的术后早期卒中发生率14%(95% CI=8%~24%),早期病死率16%(95% CI=8%~26%);笔者团队<sup>[11]</sup>近期发

表的系统综述显示,内分支系统用于锚定区位于0~1区的EAAR术后早期卒中发生率10.6%(95% CI=7.0%~14.2%),早期病死率4.9%(95% CI=2.0%~7.8%)。结合表1可见,目前EAAR的早期卒中风险高于传统弓部开放手术的金标准<sup>[5-6]</sup>,但考虑到其主要应用于高开放手术风险的患者人群<sup>[2]</sup>,这样的早期卒中和死亡风险是可以接受的。

表1 EAAR术后早期卒中及死亡风险最新系统综述

Table 1 Latest systematic review on early stroke and mortality risks after EAAR

系统综述	发表杂志	EAAR类型	近段锚定	纳入样本量(n)	早期卒中发生率	早期病死率
Nana, 等 <sup>[25]</sup> 2022	JEVT <sup>1)</sup>	开窗	0~1区	211	12.3%(范围:0~18.9%)	3.8%(范围:0~9.1%)
		分支	0区	348	13.2%(范围:0~27.9%)	6.0%(范围:0~28.5%)
		原位或预开窗	0~1区	635	3.5%(范围:0~13.5%)	2.3%(范围:0~4%)
		烟囱	0~2区	901	4.3%(范围:0~16.1%)	3.9%(范围:0~18.5%)
			0~1区	250	8.8%(范围:0~16.1%)	7.5%(范围:3.2%~18.5%)
Spath, 等 <sup>[13]</sup> 2023	EJVES <sup>2)</sup>	分支或开窗	0~2区	571	6.2%(95% CI=4%~9%)	6.7%(95% CI=5%~9%)
		开窗	0~2区	167	5.3%(95% CI=3%~10%)	6.4%(95% CI=4%~11%)
		分支	0~2区	280	6.8%(95% CI=4%~11%)	7.2%(95% CI=4%~13%)
		分支或开窗	0区	249	7.2%(95% CI=4%~12%)	7.8%(95% CI=5%~12%)
Basha, 等 <sup>[26]</sup> 2023	CJC <sup>3)</sup>	分支	0区	273	14.0%(95% CI=7.6%~24.3%)	15.6%(95% CI=8.7%~26.3%)
Kwan, 等 <sup>[27]</sup> 2023	JEVT <sup>1)</sup>	分支	0~2区	618	10.5%(95% CI=6.8%~14.3%)	5.5%(95% CI=2.6%~9.7%)
Gao, 等 <sup>[11]</sup> 2023	JEVT <sup>1)</sup>	内嵌分支	0~1区	289	10.6%(95% CI=7.0%~14.2%)	4.9%(95% CI=2.0%~7.8%)

注:1)Journal of Endovascular Therapy;2)European Journal of Vascular and Endovascular Surgery;3)Canadian Journal of Cardiology

Note: 1) Journal of Endovascular Therapy; 2) European Journal of Vascular and Endovascular Surgery; 3) Canadian Journal of Cardiology

## 2 EAAR术后脑卒中发生机制及危险因素

EAAR术后脑卒中的发生机制较复杂,通常为多因素共同作用。与腔内修复直接相关的因素主要有固体栓塞、气体栓塞、低灌注三种<sup>[28]</sup>。

### 2.1 固体栓塞

Grover等<sup>[29]</sup>采用术中经颅多普勒(transcranial doppler sonography, TCD)以及双滤网抗栓塞脑保护装置(cerebral embolic protection device, CEPD)观察锚定区位于2~3区的EAAR术中栓塞情况,研究发现100%的头臂干滤网及89%的左颈总动脉滤网内捕获到了固体碎片,病理检出的碎片各组分出现频率分别为急性血栓(95%)、动脉壁(63%)、异物(32%)、机化附壁血栓(21%)、瓣膜组织(16%)等;该研究还发现,TCD检测的固态栓子信号于导丝和猪尾导管操作期间最明显,其信号数量与磁共振弥散加权成像(DW-MRI)显示的新发病灶表面积增加相关( $P<0.01$ )。Schmidt等<sup>[30]</sup>通过双滤网系统收集经导管主动脉瓣置入(transcatheter

aortic valve implantation, TAVI)术中头臂干和左颈总动脉内的固体栓子,确定了五种主要碎片类型:血栓(91%),动脉壁组织(68%),瓣膜组织(53%),钙化(46%),异物(30%),其中急性血栓检出频率:头臂干80%,左颈总动脉76%。由此可见,腔内操作引起的急性微血栓、血管损伤以及主动脉粥样硬化碎片是EAAR术中固体栓塞的主要来源,既往研究也证实EAAR术中TCD检测到的微栓子信号总数与术后脑卒中、短暂性脑缺血发作以及死亡之间存在显著关联<sup>[31]</sup>。

笔者近期发表的一篇系统综述<sup>[11]</sup>显示,高龄、慢性阻塞性肺疾病(chronic obstructive pulmonary disease, COPD)、退行性主动脉病变(非主动脉夹层病变)与内分支EAAR术后30d内脑卒中发生率显著相关,提示术前主动脉粥样硬化斑块负荷对EAAR术后脑卒中发生具有重要影响。Hosaka等<sup>[32]</sup>通过CTA影像分析对EAAR患者术前主动脉粥样硬化负荷进行Shaginess评分,发现术后发生脑卒中

的患者人群主动脉 Shaginess 评分显著高于未发生脑卒中的患者人群 ( $P=0.04$ ); Maeda 等<sup>[33]</sup>同样发现主动脉 Shaggy 评分是 TEVAR 术后栓塞性脑卒中的独立预测因子 ( $P<0.001$ )。此外,弓上重建分支血管的动脉粥样硬化斑块负荷也会显著影响 EAAR 术后卒中风险。Kudo 等<sup>[34]</sup>在比较分支与烟囱技术在 0 区锚定的 EAAR 研究中发现,无名动脉粥样硬化分级  $\geq 2$  级是术后卒中的独立危险因素 ( $P<0.001$ )。

鉴于 EAAR 术中固体栓子的主要来源类型(急性微血栓、血管损伤、斑块碎片),腔内操作本身无疑是造成 EAAR 术中固体栓塞的最直接因素。多项研究<sup>[35-37]</sup>证实,EAAR 近端锚定区越近心,术后卒中风险越高,因为更近心端的锚定通常意味着需要增加主动脉弓上分支的重建,相应的上肢通路<sup>[38-39]</sup>以及主动脉弓内的导丝、导管等操作增多,手术复杂性明显增加,表现在手术时间上也越长<sup>[40]</sup>。最新的 Meta 分析<sup>[35]</sup>也证实,EAAR 术后卒中风险在 3 区及其远段最低,且随着锚定区向近心端移动而显著增加(2 区 vs. 3 区:  $RR=2.14$ ,  $P=0.000 2$ ; 1 区 vs. 2 区:  $RR=1.48$ ,  $P=0.000 2$ ; 0 区 vs. 1 区:  $RR=1.85$ ,  $P<0.000 01$ )。由此可见,锚定区前移引起的腔内操作复杂度升高对于 EAAR 术后卒中发生率具有重要影响。此外,器材设计、释放技巧以及主动脉弓的解剖(曲度<sup>[41]</sup>、牛弓<sup>[42]</sup>)同样会对腔内操作的复杂性以及术后卒中风险产生显著影响。例如,Inoue 外分支支架系统(PMTC, 日本)用于 EAAR 结果显示<sup>[10]</sup>,单分支系统的围术期卒中发生率 7.8%,双分支系统为 33%,三分支系统为 42%,分析其原因很可能是其一体式的外分支设计以及所采用的牵拉释放技术在多个外分支释放过程中的复杂性过高。而与之相反,采用模块化设计的双内嵌分支系统 Relay Branch (Terumo Aortic, 美国)和 Cook A-Branch (Cook, 美国)的术后 30 d 卒中发生率分别为 12.3%~25%<sup>[43-46]</sup>和 2.9%~11.1%<sup>[47-48]</sup>,分析其原因可能是模块化设计的各部分组件可以进行原位组装,相比一体式多分支支架的释放更简便,弓部侵扰更少。

## 2.2 气体栓塞

近年来人们逐渐认识到气体栓塞在 TEVAR 手术中的重要意义。Grover 等<sup>[29]</sup>通过术中 TCD 证实 TEVAR 全程所能监测到的微栓子信号 91% 为气态,且该气态微栓子信号于支架打开和释放过程最明显,研究<sup>[49]</sup>还证实气态微栓子信号数量的增加与 DW-MRI

病变数量的增加相关 ( $r=0.912$ ,  $P=0.01$ ),但对于病变表面积无明显影响,提示气态微栓子虽然可以在血液中弥散,但大量气态微栓子的累积效应仍会导致缺血性损伤,体外循环中的经验也证实气体过滤装置的使用具有改善术后认知功能的能力,提示气体栓塞对术后神经认知方面的不良影响。然而,TEVAR 术中产生的有限的气态微栓子<sup>[50]</sup>是否一定会引起远期神经系统功能障碍尚无确切证据。

随着临床对于 TEVAR 术中气体栓塞的重视,无症状性脑梗死(silent brain ischemia, SBI)的概念也逐渐被广泛认知。SBI 是指无明显局灶性神经系统异常、但被影像学证实的缺血性脑损伤,目前 SBI 已被确定为未来脑卒中、痴呆、抑郁以及认知障碍的独立预测因素<sup>[51-52]</sup>。近年来多项研究<sup>[53-55]</sup>证实接受 EAAR 的患者人群中  $>60\%$  存在 SBI;同时还发现该部分“沉默”脑缺血病变人群术后出现了神经认知能力的下降<sup>[55]</sup>,尤其以老年患者明显,提示老年患者对于缺血事件的耐受能力下降。

目前尚无直接证据证明 SBI 的栓子来源到底是以气体还是固体为主。Haussig 等<sup>[56]</sup>报道了在 TAVI 术中使用双滤网 CEPD 的随机对照研究显示,CEPD 组 97% 的 TAVI 病例中滤网捕获到了固态栓子,且新发 SBI 的数量和体积也较对照组显著减少,同时伴有早期神经系统结局改善<sup>[57]</sup>,提示固态栓子可能在 SBI 发生发展中发挥重要影响。鉴于 TCD 检测到的气态微栓子的高占比,不排除气体栓塞是 SBI 的另一重要来源。有学者<sup>[58]</sup>提出了使用  $CO_2$  冲洗来减少气体栓塞的方法,然而,STEP 研究<sup>[54]</sup>结果显示,即使经过充分的  $CO_2$  冲洗后,锚定区位于 0~3 区的 TEVAR 术后 SBI 的发生率仍达到 50%,多因素分析结果显示紧急手术是 SBI 的唯一独立预测因子。

## 2.3 脑灌注不足

EAAR 术后低灌注导致脑卒中最典型的例子就是左锁骨下动脉(LSA)的覆盖。尽管目前针对 LSA 覆盖后是否需要重建尚存少许争议<sup>[59-60]</sup>,但更多高级别证据支持 TEVAR 术中覆盖且不重建 LSA 会增加术后卒中风险<sup>[40, 61-63]</sup>。SVS 及 ESVS 发布的临床指南<sup>[64-65]</sup>也都提倡对接受选择性 TEVAR 患者术前行常规 LSA 血运重建,以防止 LSA 覆盖可能伴随的脑卒中、脊髓损伤、椎基底动脉供血不足及上肢缺血风险。

其他可能引起脑灌注不足的因素如颈动脉狭

窄<sup>[66]</sup>、Willis环不完整<sup>[67]</sup>、贫血<sup>[68]</sup>、术中低血压<sup>[69]</sup>、伴血流动力学不稳定的急诊手术<sup>[70]</sup>等也都可能会引起EAAR术后卒中风险升高。

### 3 EAAR术后卒中的预防策略

EAAR相关的研究报道大多为病例系列报道,样本量少,异质性大,因此目前暂没有预防EAAR术后卒中的高质量证据推荐,但笔者仍根据既往经验和文献报道来对此做出一些低证据级别的建议。

#### 3.1 术前评估

卒中的预防始于术前评估。术前全面评估患者的卒中风险因素和血管解剖结构,利用影像技术(CTA、MRA等)进行精细化分析,有助于制定个体化的手术方案。

首先是固体栓塞风险的评估,包括主动脉粥样硬化斑块负荷分级<sup>[32-33]</sup>、升主动脉及弓部有无明显的附壁/漂浮血栓、主动脉弓曲度<sup>[41]</sup>以及有无解剖变异<sup>[42]</sup>等。对于主动脉壁相关的固体栓塞风险较高的患者,如升主动脉漂浮血栓,开放手术仍然是最佳的治疗选择,因为在去分支或EAAR分支重建期间的广泛弓部操作很可能导致栓塞事件的发生。

其次就是低灌注风险的评估,包括颈动脉有无狭窄、Willis环是否完整、有无贫血、血流动力学是否稳定、LSA及椎动脉开放程度以及有无变异等。颅脑MRA可与TCD一起用于评估Willis环的完整性,有助于预测交叉血流不足和脑卒中风险。

此外,通过机器学习构建个体化的卒中风险预测模型可能是有必要的<sup>[71]</sup>。EAAR患者的年龄、性别、基础疾病(高血压、糖尿病、COPD)、吸烟史、卒中史、冠心病史等基线特征都应当纳入考虑范围,并结合患者的固体栓塞风险、低灌注风险评估结果进行术后卒中概率的系统预测,包括手术时间、前后循环的卒中概率、死亡风险等,从而精准识别卒中高风险患者并为其量身定制高效的神经保护措施。

#### 3.2 围术期监测

围术期监测包括血流动力学监测和神经监测,可以及早发现卒中的预警信号。前者毋庸置疑,至于神经监测,目前ESVS指南<sup>[23]</sup>基于意见级别推荐的三种术中监测的手段主要有近红外光谱技术

(NIRS)、脑电图(EEG)以及运动诱发电位(MEP)/体感诱发电位(SSEP)。不过,与清醒患者监测相比,尚没有任何一种单一的监测方法能提供100%的敏感度和100%的特异度<sup>[72]</sup>,具体应用更可能取决于操作者的偏好。

基于血红蛋白(Hb)的NIRS可用于连续监测大脑皮层浅层区域的氧供需平衡(氧合Hb与脱氧Hb的浓度变化)。在EAAR中,脑组织氧合血红蛋白可能由于灌注不足、低血压、贫血等因素导致局部或全脑缺血而氧合Hb浓度下降,NIRS可以对此进行监测,必要时可针对局部脑组织氧合Hb降低进行程序化干预。NIRS的局限性则是术中双额区域脑血氧饱和度描图不能排除NIRS观察视野之外的局灶性脑缺血,但由于其显著有利的风险-获益比,无创连续NIRS监测在胸主动脉手术中的应用越来越多<sup>[73]</sup>。

EEG早期主要用于深低温停循环中脑电沉默监测,但目前随着中低温持续顺行灌注的广泛应用,EEG监测更多用于脑缺血、缺氧情况和麻醉水平不足的检测<sup>[73]</sup>,只是目前尚缺乏证据表明EEG监测可改善主动脉弓部开放手术、杂交手术或腔内修复术的主要结局。

MEP或SSEP监测可用于EAAR术中及术后麻醉恢复期间卒中发生的早期辅助诊断,从而指导治疗以及实现麻醉患者脑卒中的早期干预<sup>[74]</sup>。

#### 3.3 术中操作

从弓部腔内操作收集的固体栓塞物组成<sup>[29-30]</sup>可以看出,高质量的手术技巧和规范的操作对于预防卒中至关重要。操作者应当准确评估血管解剖结构,选择适当的器材和优化操作方案,从而最大程度避免血管损伤和斑块脱落。Perera等<sup>[75]</sup>通过介入机器人导航辅助TEVAR能显著减少术中TCD实时监测的固体微栓子信号数量,相比于手工操作,机器人辅助操作的控制性与稳定性可能减少了导丝导管与主动脉弓壁的接触,从而减少栓塞颗粒物的产生。

由于患者间基线特征和治疗方案的异质性,相应的预防措施也需要具有针对性。例如,对于行颈动脉切开穿刺的EAAR患者可以在支架释放过程中行颈动脉临时阻断以减少气态或固态碎片的人颅;对于弓上去分支联合TEVAR的杂交手术可以行远端脑保护装置来预防脑栓塞;采用CO<sub>2</sub>或大剂量盐水冲洗主动脉支架<sup>[76]</sup>等等。然而,以上推

荐的级别都很低，甚至部分预防措施本身就可能增加卒中风险的增加，如远端脑保护滤网的植入操作也会增加气态和固态微栓信号的显著增加<sup>[29]</sup>、术中滤网移位可能导致栓塞性卒中<sup>[77]</sup>等。

### 3.4 药物预防

药物治疗也是预防EAAR术后卒中发生的重要手段，如术前充分抗血小板治疗、术中充分抗凝能够有效降低血栓形成和栓塞风险。Grover等<sup>[29]</sup>发现术前未接受抗血小板治疗的急诊手术患者的术中滤网捕获的颗粒物的表面积和直径有增加的趋势，提示未充分抗栓治疗可能加重急性血栓为主的固体栓塞风险。

## 4 EAAR术后卒中的治疗策略

EAAR患者术后出现脑卒中和认知功能障碍的主要原因是缺血，少数原因为脑出血<sup>[78]</sup>。脑部MRI是所有怀疑有神经血管事件或急性谵妄患者的首选和推荐检查<sup>[79]</sup>，若MRI无法实现，需行头颅CT以排除出血，也可以作为确认脑卒中的替代方法。

对于已经发生EAAR术后卒中的患者，积极采取合理的治疗策略可以降低其病情恶化和并发症的发生<sup>[9, 80]</sup>。首先，及时诊断和评估至关重要<sup>[81]</sup>。及时的脑血管造影、头颅MRI或头颅CT等影像学检查可以帮助确定卒中的类型和范围，有助于早期制定有效的治疗方案，尤其对于那些适合急诊腔内血栓切除术的患者<sup>[82]</sup>；其次，药物治疗是治疗EAAR术后卒中的基础。抗凝、溶栓和抗血小板等药物能够改善脑血流灌注，减少卒中面积和并发症发生<sup>[83]</sup>；此外，手术干预也是治疗EAAR术后卒中的重要手段。紧急血栓或异物清除可以恢复脑血流灌注，减轻神经损伤<sup>[82]</sup>。

总之，卒中干预过程中应注意多学科协作，需要神经外科、介入放射科、神经内科、重症医学科等多学科的紧密合作，以确保及时有效地治疗<sup>[57]</sup>。

## 5 小结与展望

EAAR术后卒中的防治研究进展涉及多个方面，包括手术方案、发生机制、预防策略和治疗策略等。目前的研究显示，EAAR虽然在围术期并发症发生率和病死率方面具有显著优势，但术后

脑卒中仍然是限制该技术推广应用的一个重要并发症。针对目前的研究现状，未来的工作可以从以下几个方面展开。

在术前评估方面，可以通过影像学技术进行精细化分析，以制定个体化的手术方案。此外，机器学习构建个体化的卒中风险预测模型用以精准识别高卒中风险患者并为其量身定制高效的神经保护措施可能是有必要的。

在围术期监测方面，应加强血流动力学监测和神经监测，以及早发现卒中的预警信号。术中操作方面，高质量的手术技巧和规范的操作能最大程度地避免血管损伤和斑块脱落。同时，药物治疗也是预防EAAR术后卒中发生的重要手段。

对于已经发生卒中的患者，积极采取合理的治疗策略可以防止其病情恶化和并发症的发生。及时的诊断和评估对于制定有效的治疗方案至关重要，包括脑血管造影、头颅MRI或头颅CT等影像学检查。药物治疗和手术干预也是治疗EAAR术后卒中的基础。

在未来的研究中，需要更多地关注如何进一步降低EAAR术后卒中的发生率，提高手术的安全性和成功率。同时，需要进一步探索更加精准的预防策略和治疗方案，以减少患者的术后并发症和提高生存质量。多学科协作也是未来研究和临床实践中需要重点关注的方向，不同学科之间的紧密合作将有助于更好地应对EAAR术后卒中的挑战。

利益冲突：所有作者均声明不存在利益冲突。

作者贡献声明：高江平、郭伟负责论文构思；高江平负责文献收集与初稿撰写；郭伟负责论文审阅与修订。

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( 本文编辑 宋涛 )

本文引用格式:高江平,郭伟. 主动脉弓腔内修复术后卒中的防治研究进展[J]. 中国普通外科杂志, 2023, 32(12):1819-1828. doi: [10.7659/j.issn.1005-6947.2023.12.001](https://doi.org/10.7659/j.issn.1005-6947.2023.12.001)

Cite this article as: Gao JP, Guo W. Research progress in the prevention and treatment of stroke after endovascular aortic arch repair[J]. *Chin J Gen Surg*, 2023, 32(12):1819-1828. doi: [10.7659/j.issn.1005-6947.2023.12.001](https://doi.org/10.7659/j.issn.1005-6947.2023.12.001)