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# 左心室卸负荷联合VA-ECMO治疗心源性休克：机制、方法与未来方向

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## 摘　要

**心源性休克是一种由心脏泵功能衰竭导致的高死亡率临床综合征，其治疗始终是临床重大挑战。静脉－动脉体外膜肺氧合(VA-ECMO)通过提供循环与呼吸支持，显著改善患者血流动力学及氧合状态，但其应用可能导致左心室后负荷增加，加剧心肌缺血与肺水肿。左心室卸负荷通过降低左心室前、后负荷，减少心肌氧耗，促进功能恢复，与VA-ECMO联合应用可协同提升疗效。本文系统综述了心源性休克的病理生理机制、VA-ECMO与左心室卸负荷的作用机制，并探讨了主动脉内球囊反搏、经皮左室辅助装置、房间隔分流及外科手术等卸负荷方法的临床效果与局限性。现有研究表明，联合治疗可改善患者生存率及预后，但适应证选择、装置优化及并发症管理仍是关键挑战。未来需通过个体化策略、新型装置研发及多中心随机对照研究，进一步明确最佳治疗方案，为临床实践提供循证依据。**

## 关键词

**心源性休克，静脉－动脉体外膜肺氧合，左心室卸负荷，主动脉内球囊反搏，经皮左室辅助装置，机械循环支持**

# Left Ventricular Unloading in Combination with VA-ECMO for the Treatment of Cardiogenic Shock: Mechanisms, Methods, and Future Directions

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## Abstract

**Cardiogenic shock is a clinical syndrome with high mortality caused by the failure of cardiac pump function, and its treatment has always a major clinical challenge. Venous-arterial extracorporeal membrane oxygenation (VA-ECMO) significantly improves patients' hemodynamics and oxygenation by circulatory and respiratory support, but its application may lead to increased afterload of the left ventricle, exacerbating myocardial ischemia and pulmonary edema, ventricular unloading, by reducing the pre- and afterload of the left ventricle, reducing myocardial oxygen consumption, and promoting functional recovery, can synerg improve the efficacy when used in combination with VA-ECMO. This article systematically reviewed the pathophysiological mechanisms of cardiogenic shock, the mechanisms of action of VAECMO and left ventricular unloading, and explored the clinical effects and limitations of unloading methods such as intra-aortic balloon counterpulsation, percutaneous left ventricular assist device, atrial septal shunt, and surgical treatment. Existing studies have shown that combined treatment can improve survival rate and prognosis, but the selection of indications, device optimization, and complication management remain key challenges. In the future, it is necessary to further clarify the best treatment plan through personalized strategies, the development of new devices, and multicenter randomized controlled trials, and to provide evidence-based basis for clinical practice.**

## Keywords

**Cardiogenic Shock, VA-ECMO, Left Ventricular Unloading, IABP, pLVAD, Mechanical Circulatory Support**



## 1. 引言

心源性休克(Cardiogenic Shock, CS)是指由于心脏泵血功能严重受损，导致全身器官和组织灌注不足的一种临床综合征[1]。其定义通常包括低血压(在血容量充足的情况下，收缩压 ≤ 90 mmHg 超过 30 分钟，或平均动脉压 ＜65 mmHg 超过 30 分钟)和至少一项脏器灌注不足的征象，如精神状态改变、肢端皮肤湿冷、少尿或无尿、代谢性酸中毒等。心源性休克的病因主要包括急性心肌梗死、心肌病、心肌炎、严重心律失常等，其中急性心肌梗死是最常见的原因[2]。全球范围内，心源性休克的发病率和死亡率均较高。据统计，心源性休克的院内病死率可达 40%左右，是急性心肌梗死患者中最严重的并发症之一[3]。在一项纳入 150 例急性心肌梗死合并心源性休克患者的研究中，院内病死率高达 60% [4]。此外，心源性休克的预后与多种因素相关，如发病时间、治疗时机、合并症等[2]。

静脉－动脉体外膜氧合(Veno-Arterial Extracorporeal Membrane Oxygenation, VA-ECMO)是一种体外生命支持技术，通过将静脉血引出体外，经过氧合器氧合后，再通过动脉回输到体内，以替代心脏的泵血功能，提供有效的循环支持[5]。VA-ECMO 的原理是利用体外机械泵支持外周器官的血液灌注，同时提供部分呼吸支持。VA-ECMO 的应用范围广泛，主要用于治疗严重的心脏和呼吸衰竭，如心源性休克、急性心肌梗死、心脏手术后心功能衰竭等[5]-[7]。VA-ECMO 的临床优势包括：1) 能够提供有效的循环支持，维持血压和器官灌注；2) 可以同时提供呼吸支持，改善氧合和二氧化碳清除；3) 操作相对简单，

可在床边进行，适用于急诊和重症患者[8]。此外，VA-ECMO 在心源性休克患者中的应用，能够显著改善患者的血流动力学状态，提高生存率[5]。

左心室卸负荷是指通过减少左心室的前负荷和后负荷，降低心室壁的应力，从而减轻心肌的氧耗需求，改善心功能[9]。左心室卸负荷的意义在于保护心肌，减少心肌损伤，改善心功能，提高患者的生存率[10]。左心室卸负荷的方法包括药物和机械方法。药物方法主要包括使用 $\beta$ 受体阻滞剂、硝酸酯类药物等，通过扩张血管、降低心率等机制减轻心脏负担。机械方法则包括使用左心室辅助装置(LVAD)、主动脉内球囊反搏(IABP)等[11]。

心源性休克作为一种极其危急且严重威胁患者生命健康的心血管急症，一直是临床治疗中的难题。其发病机制复杂，病情进展迅速，死亡率居高不下。心室辅助装置(VAD)的出现为心源性休克的治疗带来了新的希望，尤其是 VA-ECMO，在改善患者氧合和循环支持方面发挥了重要作用。然而，随着临床应用的不断深入，人们逐渐发现 VA-ECMO 治疗过程中存在一些问题，如左心室后负荷增加等，这可能影响患者的预后。因此，左心室卸负荷联合 VA-ECMO 治疗心源性休克的研究应运而生，旨在进一步提高治疗效果，降低死亡率。本综述旨在梳理现有证据，指导左心室卸负荷和 VA-ECMO 在心源性休克的治疗中的应用。

## 2. 病理生理机制

### 2.1. 心源性休克的病理生理机制

心源性休克的病理生理呈现“心功能恶化→血流动力学紊乱→微循环障碍→炎症激活→多器官衰竭”的链式反应，其基本血流动力学触发因素是尽管前负荷充足，却无法维持足够的每搏输出量，导致心输出量减少和组织灌注不足。心源性休克中的外周血管收缩和液体潴留是早期维持器官灌注的重要的代偿机制，但最终因加剧了前负荷和后负荷的不匹配，导致血流动力学状态恶化，进而因舒张功能障碍而加重淤血。心指数降低、肺毛细血管楔压和中心静脉压升高，以及交感神经和神经激素激活(内源性儿茶酚胺释放、全身血管阻力增加和肾素－血管紧张素－醛固酮系统激活)共同构成了经典所谓“冷”(指外周灌注)和“湿”(指充盈压)心源性休克表型的病理生理基础[12] [13]。

### 2.2. VA-ECMO 治疗的作用机制

VA-ECMO 在心源性休克治疗中扮演着重要角色。它通过将静脉血引出体外，经过氧合器进行氧合和二氧化碳去除，然后通过动脉回输到体内，从而提供有效的循环支持和部分呼吸支持。这种体外循环支持方法能够部分或完全替代心脏和肺的功能，帮助维持全身器官的灌注和氧合[5] [14]。

在心源性休克患者中，VA-ECMO 的应用可以显著改善患者的血流动力学状态，提高心输出量和血压，从而改善组织灌注。这对于因心脏泵血功能衰竭而导致的低血压和器官灌注不足的患者尤为重要。VA-ECMO 不仅能够提供循环支持，还能通过氧合器改善患者的氧合状态，减少组织缺氧和代谢性酸中毒的发生[5]。

此外，VA-ECMO 还可以作为桥接治疗手段，帮助患者度过急性期，等待心脏功能恢复或进行进一步的治疗，如心脏移植或植入长期心室辅助装置。这种桥接治疗对于心源性休克患者尤为重要，因为它可以维持患者的生命体征，为后续治疗争取时间[15]。

### 2.3. 左心室卸负荷治疗的作用机制

左心室卸负荷是指通过减少左心室的前负荷和后负荷，降低心室壁的应力，从而减轻心脏负担，提高心脏输出量，改善组织灌注[16]。左心室卸负荷的治疗作用机制包括以下几个方面：1) 降低心室壁应力与氧耗，通过机械辅助装置(如主动脉内球囊反搏 IABP、体外膜肺氧合 ECMO 或左心室辅助装置 LVAD)

或药物(如血管扩张剂)减少左心室收缩时需克服的后负荷(主动脉压力)，从而降低心室壁张力及心肌氧耗。这有助于缓解心肌缺血，改善能量代谢失衡[10] [16]。2) 改善血流动力学，机械装置通过部分或完全替代左心室的泵血功能，直接增加心输出量和全身灌注，同时降低左心室舒张末压(LVEDP)，减轻肺循环淤血和肺水肿，改善终末器官的血液供应[17]。3) 促进心肌顿抑恢复在急性心肌损伤(如心肌梗死或心脏手术后)中，心室卸负荷通过减少心肌做功，为受损心肌提供“休息”机会，促进心肌细胞代谢修复和收缩功能的恢复，减少不可逆损伤[18]。4) 阻断恶性循环与心室重构，慢性心力衰竭时，心室卸负荷通过降低心脏前后负荷，减少神经内分泌系统(如交感神经和肾素–血管紧张素系统)的过度激活，抑制心室扩张和病理性重构，延缓心力衰竭进展[19]。

## 3. 左心室卸负荷的必要性、方法及临床决策

### 3.1. 必要性

当 VA-ECMO 支持下左心室后负荷增加时，衰竭的左心室无法维持心室–动脉耦合，会出现左心室扩张恶化、左心室舒张末压升高、冠状动脉灌注减少等一系列恶性循环。这不仅会进一步加重心肌缺血缺氧，导致心肌细胞损伤和凋亡，还会引发心律失常，增加患者的死亡风险。此外，左心室扩张还会导致肺循环瘀血，引起肺水肿，进一步影响气体交换，加重患者的呼吸功能障碍。因此，进行左心室卸负荷对于改善心源性休克患者的预后具有重要意义，可以有效减轻左心室的容量负荷和后负荷，恢复心室-动脉耦合，改善冠状动脉灌注，保护心肌细胞[20]-[22]。

### 3.2. 方法

#### 3.2.1. 主动脉内球囊反搏(IABP)

主动脉内球囊反搏(IABP)可以作为减少 PCWP 的初始选择，其和 VA-ECMO 的联合应用在近二十年间已形成临床实践模式，其主要目的是借助脉动血流生成与降低心脏后负荷达成协同治疗。当前，该策略在心脏术后康复、移植过渡期以及难治性心脏骤停等 VA-ECMO 适应证中，依旧被视作改善预后和推动撤机的关键干预举措。多项观察性研究表明，联合治疗能使生存率显著提高，特别是对左心室扩张患者的撤机成功率有显著影响[23]-[27]。所以，部分学者提议对所有 VA-ECMO 患者常规使用 IABP，然而该策略尚未得到广泛认可[24] [25] [27]。近期临床数据存在分歧：一方面大规模分析未明确支持将联合应用作为临床规范[28]-[30]，另一方面最新研究表明其能够提高生存率[31]。

从理论上讲，在 VA-ECMO 期间使用 IABP 可使平均系统阻抗、收缩压和左心室应力降低 10%~15% [32]。因此，在以下情形中联合应用或许具有必要性：左心室无射血状态：在外周或中央 VA-ECMO 支持期间左心室持续无射血，且存在左心室血栓风险；替代机械循环支持受限：其他机械辅助设备不可行或不适用时[33]-[35]。此外，临床研究显示联合应用可降低中心静脉压、肺毛细血管楔压(PCWP)、左心室容积以及胸片肺水肿表现[36] [37]，可能改善 30%以上 VA-ECMO 治疗中并发的肺水肿，甚至对过渡至长期左心室辅助装置(LVAD)后的预后有益[37]。值得关注的是，IABP 在 VA-ECMO 期间对左心室的卸载作用较为有限：临床经验与计算机模拟表明 PCWP 降低幅度通常不超过 5 mmHg，这提示其血流动力学益处可能受到多种因素的制约。这种有限效应与机械性卸载设备的直接干预(如 Impella)形成反差，需要结合患者的个体化评估来选择策略[38]。

从机制角度而言，联合应用能够增强冠状动脉及移植血管的血流，但此结论大多基于实验环境，并且显著取决于 VA-ECMO 的配置(如插管位置和导管方向) [39]-[41]。尽管部分实验对 IABP 可能给脊髓和脑血流带来负面影响提出质疑(尤其是在心脏功能严重受损时) [39] [42] [43]，但联合应用仍被证实可改善中央与外周 VA-ECMO 患者的心肌氧供与氧平衡[44]，不过其对微循环的改善作用尚不清晰[38] [45]。

#### 3.2.2. 经皮左室辅助装置(pLVAD)

经皮左心室辅助装置(pLVADs)在需要更强减负效果时使用，它借助将血液从左心室分流至主动脉的方式，减轻左心室的容量负荷，降低左心室的后负荷，同时增加冠状动脉的灌注量。相较于主动脉内球囊反搏，pLVADs 在左心室卸负荷方面成效更为显著，然而其操作相对复杂，成本也较高。当前临床上所应用的 pLVADs 主要涵盖 Impella 装置与 TandemHeart 装置。pLVADs 不仅能够有效地减轻左心室负荷，还能提供一定程度的循环支持，从而改善患者的血流动力学状态[16] [20] [21]。

Impella 属于一种经主动脉置入的左心室辅助装置，其设计为基于导管的微型轴流泵，能够将血液从左心室泵送至升主动脉。Impella® 2.5 每分钟最多可提供 2.5 升的血流量，适用于心源性休克的经皮短期机械支持[46]。不过，对于严重心源性休克患者，尤其是那些有多器官功能衰竭先兆且需要机械通气的患者而言，可能无法提供充足的血流量[47] [48]。

mpella® 2.5 及更大的 CP 型号已在多例成人患者中作为外周静脉－动脉体外膜肺氧合(VA ECMO)期间左心室卸负荷的辅助装置使用[49] [50]。所有可用于左心室支持的 Impella®装置，即 2.5 型、CP 型以及外科使用的 5.0 型，均已与 VA ECMO 联合应用，在成人患者中显著降低了右心房压、肺毛细血管楔压(PCWP)、左心室容积和肺水肿程度，并在儿童患者中增加了肺血流量和右心室功能[49]-[52]。此外，一项大型回顾性分析表明，Impella®与 VA ECMO 联合使用可提升生存率，并能更优地过渡到康复阶段或接受其他支持治疗[50]。因此，Impella®设备应被视为在 VA-ECMO 期间的一种强大的左心室卸载设备，这一点也得到了模拟实验的支持，表明 Impella®流量可以最大降低 PCWP 10 毫米汞柱和左心室容积 20% [53]。

#### 3.2.3. 房间隔分流

通过制造房间隔缺损来减轻左心室(LV)的负担此种左向右分流的方式，已有相关报道显示其对左心室减压与负荷状态有着显著的改善作用，相关实验数据也为该结论提供了支撑[54]-[59]。然而，在技术层面，创建尺寸适宜的缺损存在较大难度，这一问题可能致使左心室减负不充分，甚至引发左心室无射血、心腔内血栓形成以及主动脉根部血栓等不良状况。另一种解决策略是运用专用经皮装置构建特定尺寸的房间隔缺损[60] [61]，当该缺损不再有使用需求时，可借助导管置换术，利用专用封堵器将其关闭[62]。需要着重指出的是，房间隔造口术作为最早在 VA-ECMO 期间用于左心室减压的技术之一，依然属于高技术含量的操作，目前主要在专科中心以及儿科患者群体中应用[56]。

经房间隔穿刺置管的左心室减压方法已在各类患者中成功实施，相关报道可追溯至 20 世纪 90 年代[55] [60] [63]-[69]。但通过 ECMO 静脉端引流的血流量主要取决于导管和插管的尺寸[65] [68]，并且能够通过独立泵对流量进行控制，在撤机时也可进行暂时夹闭操作。实践结果显示，使用 22F 导管能够显著降低肺毛细血管楔压(PCWP)，降低幅度可达 4~17 mmHg，从而实现有效的左心室减压[64]。若将引流管经二尖瓣置入无射血功能的左心室，能够确保心室内的血液循环，降低心腔内血液淤滞以及血栓形成的风险。此外，少数实验和临床研究提出，可以通过腋动脉或股动脉入路，采用主动脉导管进行左心室引流[70]-[72]。另一种技术是采用经肺动脉导管进行左心室引流[73]-[75]。需要注意的是，肺动脉引流可能会导致肺血流减少，进而引发肺缺血，因此需要对肺动脉血流和/或呼气末二氧化碳进行持续监测。这些技术创新为不同临床场景下的左心室管理提供了多样化的解决途径，但在具体实施过程中，需要严格评估适应症以及风险收益比[76]。

#### 3.2.4. 外科手术

对于需要外科手术的患者，左心室卸载的直接手术方式，常需经右肺上静脉或肺动脉置入心尖引流管或减压插管，此类操作多需施行正中胸骨切开术或开胸术[77]-[80]。当前文献所报道的临床经验相对匮

乏，故而可能难以作为常规方法应用于特定心脏外科中心的临床实践。相较于经皮穿刺技术，外科减压技术采用的插管口径更大(可显著提升静脉引流与左心室减压效果)，然而该术式存在较高的出血风险[81]。尽管死亡率和并发症发生率较高，仍有学者提议将心脏切开术后减压作为中心静脉插管的短期辅助手段[77]。

当下最新建议倡导在启动 VA-ECMO 时，同期植入临时左心室辅助装置，并进行适当的左心室减压[82]。基于此，可考虑采用大口径左心室心尖减压装置，至少应配备 32-F 引流导管。VA-ECMO 回路的静脉引流可借助经皮静脉插管完成，而主动脉通路则通过主动脉上 10 mm Dacron 人造血管构建。该配置方案可通过回路管道上的可调节钳夹和血流计，对左心室减压量和右心室引流量进行个性化调控。当右心室功能与肺功能充分恢复后，仅需拔除股静脉引流导管，便可在床旁将 VA-ECMO 系统转换为更具持续性的临时左心室辅助装置[83]。

### 3.3. 临床决策的选择

VA-ECMO 虽能支持心脏恢复或为替代疗法争取时间，但由于其侵入性，超过数周后的疗效有限。其核心临床挑战在于左心室超负荷，该并发症可引发肺水肿、心肌应激及致命性风险。当前通过 IABP、Impella®心室辅助装置等辅助治疗进行左心室减压，特定病例需结合外科干预(如左心室引流术或房间隔造口术)。值得注意的是，心室超负荷的病理机制涉及后负荷增加与氧供需失衡，最新临床指南建议通过实时超声监测联合血流动力学参数优化减压策略。针对 LV 减压策略的临床选择，目前主要依据血流动力学目标分级实施：当需将 PCWP 降至 20 mmHg 以下时，IABP 可提供约 5 mmHg 的初级减压[38] [53]；若需强化卸载，Impella®装置因兼具微创性与显著效果已成为首选经皮方案[50] [84]，其效果与技术要求更高的经皮房间隔造口术或左心房引流相当[38]，而后者更多应用于特定解剖条件或联合 ECMO 植入场景；对于已接受开胸手术患者，外科辅助减压(如左室引流)则展现独特优势[21]。值得注意的是，计算机模拟平台的最新进展为精准预测不同减压策略的血流动力学效应提供了量化依据，特别是对 Impella®转速调节、房间隔造口直径与外科引流管径等关键参数的动态优化，显著提升了临床决策的个体化水平[38] [85]-[87]。这些干预措施的核心目标在于打破 LV 过载的恶性循环——通过降低室壁应力、改善冠脉灌注及减少肺毛细血管渗漏，为心肌功能恢复创造时间窗，同时规避血栓形成、肺出血等致命并发症。

## 4. 最新研究进展与未来展望

多项研究表明，对于急性心肌梗死合并心源性休克患者，VA-ECMO 联合左心室卸负荷治疗可显著提高患者的生存率和预后。在国内的一项回顾性研究中发现，IABP 在 VA-ECMO 后用于 LV 减负与 CS 患者的院内死亡率降低相关[88]。同时，还有一项回顾性研究纳入了 92 例急性心肌梗死合并心源性休克患者，其中 46 例接受 VA-ECMO 联合 IABP 治疗，46 例仅接受 VA-ECMO 治疗，结果显示联合治疗组患者的 30 天生存率显著高于单独 VA-ECMO 组[89]。此外，另一项研究也发现，VA-ECMO 联合 pLVAD 治疗可显著改善患者的血流动力学状态，降低左心室舒张末压，提高心功能指标[90]。在一项国际多中心随机试验中，对 STEMI 合并心源性休克患者进行了随机分组，接受 Impella CP 加标准治疗或标准治疗，主要终点是在 180 天死亡率。结果显示，接受 Impella CP 治疗的患者在 180 天内的死亡率显著降低，且严重并发症的发生率也有所减少[91]。随机的 DanGer Shock 试验结果显示，在选定的有经验的中心，对于无缺氧性脑损伤的 ST 段抬高型心肌梗死相关心源性休克患者，接受 Impella CP 治疗的患者 180 天死亡率显著低于仅接受标准治疗的患者[92]。

目前对于左心室卸负荷联合 VA-ECMO 治疗心源性休克的适应证尚不明确，需要进一步的研究来确定最佳的治疗时机和患者选择标准。不同的病因、病情严重程度、心功能状态等因素可能影响治疗效果。

因此，需要开展更多的临床研究，明确适应证，制定个体化的治疗方案。不同的左心室卸负荷装置具有各自的优缺点，如何根据患者的具体情况选择最合适的装置，以及如何优化装置的使用方法，是当前面临的重要问题。例如，IABP 操作相对简单，但卸负荷效果有限；pLVAD 卸负荷效果显著，但操作复杂，价格较高。因此，需要进一步研究不同装置的临床效果和安全性，优化装置的选择和使用方法。出血、血栓形成、感染等并发症的发生严重影响了患者的预后，需要进一步研究有效的并发症预防和管理策略。尽管目前已经有了一些预防和管理并发症的方法，但其效果仍不理想。因此，需要开展更多的研究，探索新的预防和管理策略，降低并发症的发生率和死亡率。

随着对心源性休克病理生理机制的深入理解，未来有望制定更加个体化的治疗策略，根据患者的具体病因、病情严重程度、心功能状态等因素，选择最合适的左心室卸负荷方法和 VA-ECMO 治疗方案。个体化治疗策略可以提高治疗效果，降低并发症发生率，改善患者的预后。研发更加安全、有效、简便的左心室卸负荷装置和技术，如微型轴流泵、经皮左心室辅助装置等，有望进一步提高治疗效果，降低并发症发生率。新型装置和技术的研发需要多学科的合作，包括医学、工程学、材料科学等。同时，需要开展更多的临床研究，验证新型装置和技术的安全性和有效性。开展多中心随机对照研究，比较不同左心室卸负荷方法联合 VA-ECMO 治疗心源性休克的疗效和安全性，为临床实践提供更高级别的证据。多中心随机对照研究可以提高研究的样本量和代表性，减少偏倚，提高研究结果的可信度。同时，可以为制定临床指南和规范提供科学依据。

## 5. 总结

左心室卸负荷联合 VA-ECMO 治疗心源性休克在改善患者血流动力学状态、提高生存率和预后方面显示出良好的应用前景。然而，目前仍面临适应证选择、装置优化和并发症管理等挑战，需要进一步的研究和探索。未来，随着个体化治疗策略的制定、新型装置和技术的研发以及多中心随机对照研究的开展，左心室卸负荷联合 VA-ECMO 治疗有望为心源性休克患者带来更好的治疗效果和预后。

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