

外科患者非心脏手术术后低血压的研究进展

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摘要

术后低血压是外科患者恢复期常见并发症, 与不良临床结局, 例如心肌损伤、器官功能障碍及死亡风险显著相关。尽管采用了先进的血流动力学监测和目标液体导向治疗方案, 但我们的管理仍然是被动的, 当术后低血压发作时, 我们才能进行干预。这篇文献综述旨在阐述其定义、机制、预后及预测模型进展。

关键词

术后低血压, 低血压病理生理学机制, 低血压预后, 预测模型

Advances in Research on Postoperative Hypotension in Non-Cardiac Surgeries in Patients Undergoing Surgery

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Abstract

Postoperative hypotension is a common complication in the recovery period of surgical patients, which is significantly associated with adverse clinical outcomes, such as myocardial damage, organ dysfunction, and increased risk of death. Despite the use of advanced hemodynamic monitoring and targeted fluid management protocols, our current management remains reactive, with interventions

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only being initiated when hypotension occurs. This literature review aims to elucidate the definition, mechanisms, prognosis, and advancements in predictive models for this condition.

Keywords

Postoperative Hypotension, Pathophysiological Mechanisms of Hypotension, Prognosis of Hypotension, Prediction Model

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1. 术后低血压的定义和流行病学特征

术后低血压是外科患者恢复期常见的血流动力学并发症,与不良临床结局(如心肌损伤、器官功能障碍和早期死亡率)密切相关。尽管术中低血压已被广泛研究[1] [2],但术后低血压的相关研究相对较少,但其危害可能更显著,因其通常持续时间更长且未被及时治疗[3]。

术后低血压的定义主要基于平均动脉压(MAP)的绝对阈值和持续时间,但缺乏统一的全球标准。不同研究使用的阈值存在差异:绝大多数研究定义术后低血压为 MAP 低于 65 mmHg [4] [5],此外,定义还需结合临床,如 MAP 降至 55~75 mmHg 范围时,需评估是否伴随器官灌注不足或需要血管活性药物治疗[6]。整体而言,65 mmHg 是术后低血压最常见的临界值,但其应用需结合患者风险因素或手术类型[4] [5]。值得注意的是,低血压的定义尚未充分标准化,导致实践中存在模糊性[7] [8]。

流行病学数据显示,术后低血压在手术患者中具有较高的流行率,并与多种不良结局显著关联。在非心脏手术患者中,约 40% 的患者在普通病房恢复期间至少经历过一次 MAP 低于 65 mmHg 的事件。若采用更宽松的阈值(如 MAP < 75 mmHg 持续 4 小时以上),流行率可升至 48%。术后低血压也可能发生在未经历术中低血压的患者中(即术中 MAP > 65 mmHg) [9],且在体质脆弱或心血管风险高的患者(如年龄 ≥ 60 岁接受中高风险手术者)中更易发生[10] [11]。

2. 术后低血压的病理生理机制

术后低血压(postoperative hypotension, POH)的病理生理机制复杂且异质性强,涉及多因素驱动。基于现有文献,术后低血压并非单一实体,而是由多种生理和病理过程相互作用的结果,主要围绕血流动力学紊乱、血管调节失调和基础疾病状态展开,而非单纯的低血压本身直接导致器官损伤[12]。

术后低血压的发生常与血管舒张(vasodilatation)或其他生理紊乱直接相关。血管舒张可能由多种因素引发,包括麻醉残留效应(如丙泊酚等药物导致血管扩张)、炎症反应或神经源性调控异常。麻醉药物可通过抑制血管平滑肌收缩力,增加外周血管顺应性,从而降低平均动脉压(MAP) [12]。此外,术后炎性细胞因子(如 IL-6 或 TNF- α)水平升高也可能介导血管舒张,导致血压下降。这些紊乱不仅在术中发生,术后仍持续存在,从而驱动术后低血压[12]。然而,驱动机制的具体类型(如血管舒张、血容量减少或心脏泵功能受损)需根据患者个体情况识别,以指导治疗决策。

术后低血压的病理生理基础具有显著异质性,不同患者可能涉及不同的机制组合。压力-输出-阻力三角形(pressure-output-resistance triangle)模型强调低血压可能源于心输出量减少(如心功能减弱)、外周阻力降低(如血管舒张)或血压调节系统的综合失衡[13]。等压曲线(iso-pressure curve)概念有助于可视化血

压变化, 帮助理解低血压的发生阈值及其对器官灌注的影响[13]。血流动力学金字塔(haemodynamic pyramid)框架则将血压变化分层为上游(如心输出量)和下游(如器官特异性阻力)机制, 强调术后低血压的异质性源于不同层级因素的交互作用。这些模型突显术后低血压病理生理的复杂性, 其机制可能包括血管张力失调、心脏泵功能障碍或容量状态改变, 同时与患者术前基础状态高度相关。

术后低血压还受到手术类型和患者共病的显著影响。心脏手术尤其可能诱导急性内皮功能障碍, 破坏血管张力和凝血平衡[14]。在特定人群(如高龄或神经系统疾病患者)中, 神经源性直立性低血压的病理生理机制可能参与术后低血压过程[14]。术后肌钙蛋白水平升高与低血压的强关联提示其机制可能更侧重于炎症和心源性因素, 而非直接神经源性通路。

术后低血压的病理生理常与患者脆弱状态协同作用。衰弱患者的心血管储备降低、血管顺应性下降, 易受手术应激影响, 导致术后低血压风险增加。不同器官对低血压的敏感性也存在差异, 如 $\text{MAP} \leq 55 \text{ mmHg}$ 与急性肾损伤强相关, 但对心脏或脑的影响机制可能不同[15]。

总之, 术后低血压的病理生理机制以血管舒张、血流动力学异质性和基础疾病交互为特征, 机制多样且依赖于患者个体状态。未来研究应聚焦于识别这些机制驱动的具体生理紊乱, 以优化个体化监测和治疗策略。

3. 术后低血压与临床预后的关联

大量研究表明, 术后低血压与心血管事件风险增加呈独立相关, 且这种关联具有剂量依赖性(即低血压深度和持续时间均影响风险) [16]。心血管事件风险: 心肌损伤: 术后低血压显著增加心肌损伤风险。单中心观察队列研究发现, $\text{MAP} \leq 65 \text{ mmHg}$ 的术后低血压独立预测心肌损伤(术后高敏肌钙蛋白 $\text{T} \geq 50 \text{ ng/L}$), 调整后风险比(HR)为 1.52 (98.4% CI 1.17~1.96); 当阈值降至 $\text{MAP} \leq 55 \text{ mmHg}$ 时, 风险进一步升高(HR 2.02, 98.4% CI 1.50~2.72) [17]。Cox 模型证实, 低血压深度(非仅持续时间)是主要驱动因素—— $\text{MAP} < 65 \text{ mmHg}$ 暴露 635 分钟以上时风险显著增加[18], 提示严重程度比持续时间更具临床影响。死亡风险: $\text{MAP} \leq 65 \text{ mmHg}$ 与 30 天全因死亡风险(HR 1.68, 98.4% CI 1.02~2.77)及 90 天死亡风险升高相关。分级分析显示, 低血压暴露时间越长风险梯度越显著(如 $\text{MAP} < 75 \text{ mmHg}$ 超 635 分钟时风险骤增), 且该关联在调整术中低血压后仍成立。

术后低血压也可导致多器官功能障碍, 但器官敏感性差异显著: 肾脏损伤: 与急性肾损伤(AKI II/III 期)强相关。 $\text{MAP} \leq 55 \text{ mmHg}$ 时 AKI 风险 HR 为 1.68 (98.4% CI 1.02~2.77) [19]; 肾脏移植患者中, 术后收缩压 $\leq 90 \text{ mmHg}$ 与移植植物功能延迟独立相关, 提示肾灌注压不足是关键机制。

4. 风险因素和预测模型

机器学习模型在术后低血压预测中展现出显著潜力。随机森林(RF)模型在预测剖宫产术中低血压时表现出优异的性能, 准确率高达 90%, 优于其他预测模型[20]。梯度提升模型则基于临床特征预测麻醉后监护室(PACU)低血压, 但其应用需结合临床判断以提高临床接受度。此外, 基于动脉波形分析的机器学习模型能够通过实时算法提前 5~15 分钟预警低血压事件, 为临床主动干预提供关键时间窗[21]。低血压预测指数(HPI)作为商用预测工具, 通过分析动脉波形特征预测平均动脉压(MAP) $< 65 \text{ mmHg}$ (持续 ≥ 1 分钟)的事件, 在非心脏手术中有效降低了低血压的持续时间和严重程度。

列线图(Nomogram)模型为个体化预测提供了可视化工具。例如, 针对宫颈癌患者脊椎硬膜外麻醉后低血压风险开发的列线图模型, 整合了基础心率和灌注变异指数(PVI)等参数, 其校准曲线显示预测准确性优异; 决策曲线分析进一步证实该模型在 20%~75%的临床阈值范围内具有实用价值[21]。联合风险模型代表了预测范式的前沿方向。研究表明, 整合多基因风险评分(PRSs)、肠道微生物组评分与传统风险因

素,可显著提升慢性疾病的预测性能。这一策略提示,构建融合生理、遗传和微生态等多维度的预测模型,有望进一步优化术后低血压的风险分层与早期干预策略。

5. 总结与展望

术后低血压普遍存在(非心脏手术患者中达 40%),且独立于术中低血压,其持续时间长、治疗延迟的特性加剧了心肌损伤、肾衰竭及死亡风险,尤其在脆弱人群中危害显著。其病理生理机制呈多因素驱动(血管舒张、心功能抑制、容量失衡),需通过“压力-输出-阻力三角”等模型个体化解析,而非单一因素干预。低血压深度(MAP $\leq$ 55 mmHg)比持续时间对预后的影响更关键,尤其肾脏是低血压敏感靶器官。术后低血压是影响外科患者康复的关键可干预因素。未来需通过跨学科合作,融合机制研究、技术创新及临床验证,构建“预测-预警-干预”闭环管理体系,最终改善患者结局。机器学习模型(如 HPI)和列线图已实现早期预警,但需结合临床判断优化接受度,多维度联合模型代表未来精准预测方向。

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