

自主神经系统在术后恶心呕吐中的作用机制与干预研究进展

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摘要

术后恶心呕吐(postoperative nausea and vomiting, PONV)是围术期最常见并发症之一, 可引发电解质紊乱、误吸、伤口裂开等不良后果, 影响患者术后快速康复, 增加医疗成本。PONV发生机制复杂, 已有研究表明自主神经系统(autonomic nervous system, ANS)可能在其发生发展过程中起到了重要作用。心率变异性(heart rate variability, HRV)是一种无创, 可动态反应自主神经功能状态的生理指标, 在PONV预测和风险分层中具有潜在价值。本文就ANS在PONV发生中的自主神经机制、HRV在PONV预测中的应用, 以及基于ANS调节的防治措施等方面进行综述, 旨在为临床提供参考。

关键词

自主神经系统, 术后恶心呕吐, 机制, 心率变异性, 预测, 干预

Mechanisms and Intervention Research Progress of the Autonomic Nervous System in Postoperative Nausea and Vomiting

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Abstract

Postoperative nausea and vomiting (PONV) is one of the most common perioperative complications

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and can lead to adverse consequences such as electrolyte disturbances, aspiration, and wound dehiscence, thereby compromising enhanced postoperative recovery and increasing healthcare costs. The pathogenesis of PONV is complex, and accumulating evidence suggests that the autonomic nervous system (ANS) may play an important role in its initiation and progression. Heart rate variability (HRV) is a noninvasive physiological metric that can dynamically reflect autonomic function and has potential value in PONV prediction and risk stratification. This review summarizes the autonomic mechanisms of ANS involvement in PONV, the application of HRV in predicting PONV, and preventive and therapeutic strategies based on ANS modulation, with the aim of providing a reference for clinical practice.

Keywords

Autonomic Nervous System, Postoperative Nausea and Vomiting, Mechanisms, Heart Rate Variability, Prediction, Intervention

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1. 引言

术后恶心呕吐(postoperative nausea and vomiting, PONV)被定义为术后 24 h 内发生的恶心或呕吐症状, 发生率居高不下, 在高风险人群中甚至可达 80% [1], 一直以来都是影响患者术后康复和舒适度的难题。PONV 本身虽不致命, 但频繁的恶心呕吐却会导致一系列严重的后果, 例如呕吐物误吸引发吸入性肺炎或是术后切口裂开出血, 尤其是甲状腺这类手术, 严重者可危及生命[2]。因此, 如何有效预测和预防以及干预 PONV 显得尤其重要。但 PONV 发生机制复杂, 目前尚未研究透彻。既往主要聚焦于中枢神经机制、外周感受通路、神经递质及受体以及个体易感因素如年龄、性别、吸烟史、遗传等展开研究[3]-[5]。近年来, 越来越多的证据表明自主神经系统(autonomic nervous system, ANS)与 PONV 的发生密切相关, 可能是关键通路[6]。ANS 广泛支配着心血管、呼吸、消化等全身多个脏器, 对机体应激反应的调节和内环境稳态的平衡起着至关重要的作用[7]。手术与麻醉会扰乱自主神经平衡, 使患者更易出现 PONV。而心率变异性(heart rate variability, HRV)能客观反应自主神经状态[8], 可为 ANS 与 PONV 的关联提供可靠依据。因此, 从自主神经角度探索 PONV 关键机制, 并寻求预测以及干预手段, 对完善 PONV 防治策略具有重要意义。

2. ANS 在 PONV 中的作用机制

2.1. ANS 概述

ANS 是外周神经系统的重要组成部分, 包括了交感神经系统(sympathetic nervous system, SNS)、副交感神经系统(parasympathetic nervous system, PNS)和肠神经系统(enteric nervous system, ENS)。SNS 起源于脊髓胸腰段(T1-L2), 迷走 PNS 起源于颅神经核及骶髓(S2-S4) [9], 而 ENS 分布于胃肠壁内, 可独立或在 SNS 和 PNS 的调节下控制肠道运动与分泌[10]。ANS 不受人体意识控制, 却相互制衡并且精细地调控人体各脏器的功能, 调节机体内环境稳态和对应激反应[11]。

2.2. PONV 概述

现代手术与麻醉技术取得了非常大的进步, 但是术后并发症如 PONV 却仍难以避免, 给患者带来痛

苦, 增加医疗负担。PONV 是指术后 24 h 内发生的恶心和(或)呕吐症状[1] [12]。恶心是一种主观上的不适, 想吐的感觉, 但无内容物排除。而呕吐则是一系列肌肉运动将胃内容物强行从口腔排出的过程[13]。一般情况下 PONV 发生率在 20%~30%, 高风险人群可达 80%。既往研究表明 PONV 的危险因素包括患者自身因素、麻醉因素以及手术因素。患者自身因素中不吸烟的年轻女性, 有 PONV 或晕动病史的人更容易发生 PONV, 此外焦虑或者肥胖也可能与 PONV 相关, 但目前证据并不充分。麻醉因素中全身麻醉 PONV 发生率高于椎管内麻醉和区域神经阻滞, 吸入麻醉药和阿片类药物可增加 PONV 风险。手术因素中, 手术时间越长 PONV 发生率越高, 不同手术方式对患者 PONV 影响不同, 腹腔镜手术、妇科手术、甲状腺手术等被认为是 PONV 的高风险手术[2] [4] [14]。

PONV 的发生机制复杂, 涉及多个环节和通路。恶心是一种由高级中枢产生的不适感觉, 常发生在呕吐之前。而呕吐则是一种复杂反射, 包括传入、整合和传出[14] [15]。延髓外侧网状结构背外侧区域存在一个相关结构被称作呕吐中枢, 是呕吐网络的重要组成部分[16], 可接收第四脑室腹侧极后区的化学感受触发区(chemoreceptor trigger zone, CTZ)、高级神经中枢(大脑皮层对视觉、嗅觉和情绪的影响)、前庭器官以及内脏感觉神经末梢的神经冲动[15] [17]。经中枢整合后, 由迷走、交感及膈、肋间神经等传出, 引发胃肌松弛、逆蠕动、唾液分泌、膈肌与腹肌收缩, 将胃内容物排出[18] [19], 整个过程中涉及多种神经递质和受体, 如 5-羟色胺、乙酰胆碱和 P 物质等[15]。

2.3. ANS 参与 PONV 的机制细节

2.3.1. 迷走神经在 PONV 中的核心作用

迷走神经作为第 10 对脑神经, 是 PNS 中分布最广, 功能最复杂的神经, 是连接消化道和中枢的重要通路, 在调节胃肠道功能和恶心呕吐反射中发挥着核心作用[20], 其在 PONV 的反射弧中, 既承担了感觉传入, 又承担了运动传出部分, 可能是关键通路之一[15]。

迷走神经的传入纤维起源于消化道、心血管和呼吸系统等多个内脏器官[21], 可将各种刺激信号, 如麻醉药刺激胃黏膜释放 5-羟色胺或是手术操作造成的胃肠道牵拉、扩张、膨胀以及化学刺激、炎症反应等传递至脑干的孤束核(nucleus tractus solitarius, NTS)。孤束核是呕吐中枢的关键组成部分, 它接收来自外周(如迷走神经和舌咽神经)和中枢如 CTZ 的多种传入信号, 并整合这些信息[15] [17] [22], 进而激活呕吐中枢的其他核团, 如背侧迷走神经核(dorsal motor nucleus of the vagus, DMNV)和疑核(nucleus ambiguus), 最终通过迷走神经传出纤维和其他运动神经, 支配胃肠道的肌肉和腺体, 当呕吐中枢下达指令时, 迷走神经传出迅速引起胃张力降低、幽门和食管下括约肌松弛, 以及小肠发生逆蠕动, 将内容物推向胃部, 引发恶心和呕吐[19] [23]。胃肠道蠕动减慢或胃排空延迟是 PONV 的常见诱因, 而迷走神经在调节胃肠道运动中起主导作用。麻醉药物中阿片类药物, 可以抑制胃肠道蠕动, 增加胃内压, 从而间接激活迷走神经传入通路[24]-[26]。

研究显示, 阻断迷走神经传入通路(如高度选择性迷走神经切断术)可显著降低许多致吐刺激引发呕吐的概率, 这说明迷走传入是诱发呕吐反射的重要途径[6]。而在临床实践当中可以发现, 迷走神经过度兴奋可能会让恶心呕吐症状即时发作, 例如眼科手术, 术中牵拉眼球能够诱发眼心反射, 呈现显著心率下降和恶心的症状[27], 而在脊椎麻醉的过程中, 由于显著抑制了交感神经, 导致迷走神经相对亢进, 部分患者会突然出现恶心症状, 同时合并心动过缓与血压降低等自主神经紊乱表现, 通过静脉注射阿托品等抗胆碱能药物阻断迷走神经传导之后, 可迅速缓解症状[28] [29], 这些临床观察所得到的结果充分证实, 恶心呕吐的严重程度跟迷走神经的兴奋程度存在一定的相关性。但迷走神经张力降低, 交感神经占优, 可能会导致胃排空延迟, 增加胃内容物对胃壁的刺激, 从而诱发恶心, 故迷走神经活性的异常, 无论是过度兴奋还是抑制, 都可能影响胃肠道的正常功能, 从而增加 PONV 的风险。

2.3.2. 交感神经在 PONV 中的作用

交感神经目前与 PONV 之间的联系证据尚不充分。但在某些情况下, 交感神经兴奋被认为可能加剧 PONV 风险。例如疼痛、焦虑可使交感肾上腺素能通路活跃, 儿茶酚胺分泌增多[7], 已有研究表明, 儿茶酚胺神经递质参与了 PONV 的发生[14] [30], 且交感神经过度活跃后会导致胃肠道血流减少、蠕动抑制和胃排空延迟, 引发内脏不适和恶心[31]。故交感神经本身可能并不直接触发呕吐反射, 但可通过儿茶酚胺、胃肠动力抑制等来降低 PONV 阈值, 或是交感神经兴奋增加了术中及术后阿片类药物用量, 从而导致 PONV 发生率增加。有研究者指出降低交感神经张力的药物如右美托咪定, 术中持续泵注可减少 PONV 发生[32] [33], 似乎也证实了这点。

2.3.3. 自主神经失衡触发 PONV 的分子通路与细胞反应

自主神经系统在 PONV 中的作用不仅限于迷走神经和交感神经的宏观调节, 还包括更加具体的分子通路和神经元兴奋性改变。手术过程中的牵拉、胃肠道扩张、麻醉药物刺激以及炎症反应, 都会促使肠嗜铬细胞释放 5-羟色胺, 从而激活迷走传入神经末梢上的 5-HT₃ 受体并启动致吐信号, 这些致吐信号会传导至延髓孤束核和化学感受触发区进行处理[34]。5-HT₃ 受体作为配体门控阳离子通道被激活后会引引起钠离子和钙离子的内流[35]。这种离子内流会导致传入神经元发生去极化并增强放电活动, 显著提高脑干呕吐网络对外周刺激的敏感性[36]。P 物质与 NK1 受体、多巴胺与 D₂ 受体、组胺与 H₁ 受体以及乙酰胆碱与 M₁ 受体等多条信号通路也都参与了延髓致吐环路的整合调控[37]。其中 P 物质与 NK1 通路被认为和持续性或延迟性呕吐关系极为密切[38]。故围术期释放的炎症介质、内脏灌注的变化以及胃肠运动的异常都可能进一步降低迷走传入神经元的激活阈值, 从而增强脑干中枢对致吐刺激的反应能力[39] [40]。由此可见术后恶心呕吐并不是单纯由某一种神经递质或受体驱动的, 更可能是由外周刺激、迷走传入敏化、脑干整合增强以及自主神经输出失衡共同参与的级联过程。

3. HRV 在 PONV 预测中的价值与局限

3.1. HRV 与 ANS 的关系

HRV 是指连续心跳周期(R-R 间期)之间微小时间间隔的波动, 是评估 ANS 对心脏调节功能的一种非侵入性、量化指标[41]。HRV 的分析能够反应交感神经和副交感神经(主要是迷走神经)对心脏活动的动态平衡调节。高水平的 HRV 往往意味着自主神经反应灵敏、心脏节律调节能力强, 通常与较高的迷走神经(副交感)活动相关; 相反, HRV 降低则提示自主神经功能受损或交感神经持续占优势, 心率缺乏正常的波动性[42]。HRV 的分析方法目前大体可分为两种, 分别是线性和非线性分析法, 其中线性分析法由于计算较为容易, 故被广泛应用于临床, 包括时域和频域分析[43]。时域分析适用于评价长时程 ANS 活动状态, 常用指标包括全部窦性心搏 NN 间期的标准差(standard deviation of NN intervals, SDNN)、全部相邻 NN 间期长度之差的均方根值(root mean square of successive differences, RMSSD)等。SDNN 可反映总体 HRV, 即交感神经和副交感神经共同作用下的心脏自主神经调节能力, 故较高的 SDNN 通常表示自主神经调节能力较强。RMSSD 参数主要反映高频变异性, 与迷走神经活性密切相关, 较高的 RMSSD 通常表示迷走神经张力较高。频域分析主要用于反应短时程 ANS 活动状态, 最常用时间窗为 5 min。频域分析常用指标包括总功率(total power, TP)、高频(high frequency, HF)、低频(low frequency, LF), 以及 LF/HF 的比值。TP 与时域中 SDNN 类似, 可反应自主神经系统整体的活性, HF 主要由迷走神经活动介导, 与呼吸相随的窦性心律周期变化相关, 故 HF 功率被视为迷走神经活性的指标。LF 准确地说受交感神经和迷走神经的双重调节作用, 但也有研究者认为在一定条件下主要反映交感神经活性。LF/HF 比值则常被用于衡量交感和副交感的平衡状态, 比值升高提示交感神经活性相对增强, 降低则提示迷走神经活性相

对增强, LF/HF 指标在临床研究中被广泛应用[44]-[48]。

3.2. HRV 与 PONV 发生的关联研究

HRV 作为自主神经功能的客观量化工具, 已经被广泛应用于心血管、内分泌、精神压力等领域相关研究[49]-[51]。既往研究表明 HRV 降低与心源性猝死等心血管不良预后密切相关[52]-[54], 慢性应激、焦虑抑郁患者也常常表现为 HRV 下降[55] [56], 均提示自述神经系统紊乱与身心健康的破坏以及疾病的发生相关。在围手术期间, 麻醉药物、手术操作以及患者生理状态变化均会显著影响自主神经活动, 故 HRV 可用于监测镇痛深度和应激水平[49]。全麻诱导后常出现 HRV 总体下降[57] (交感副交感均被抑制), 术中强烈疼痛刺激会引起 HRV 中低频成分上升、HF 降低(交感优势) [58]。故既往大量研究聚焦于 HRV 与低血压、心动过缓以及疼痛应激的关系[59]-[62]。由于自主神经系统在恶心呕吐发生中的关键作用, 越来越多的研究者开始探索 HRV 与恶心呕吐的关联。一项随机研究[63]比较了七氟烷麻醉和丙泊酚麻醉对术后 HRV 恢复及 PONV 的影响, 结果显示七氟烷组受试者术后早期 PONV 的发生频率和严重程度显著高于丙泊酚组(尤其在术后 30 分钟内)。两组术前术后的 HRV 频域参数比较发现, 丙泊酚组术后 HRV 基本恢复接近术前水平, 而七氟烷组术后总功率、LF 和 HF 功率均较术前明显降低, 且未能在短时间内恢复。也就是说, 接受七氟烷麻醉的受试者术后自主神经活动仍处于抑制状态, 而这一状态可能与 PONV 更高的发生率有关。虽然该研究未发现两组 HRV 指标在术后组间有显著差异, 但在七氟烷组内部, HRV 下降的患者往往出现了 PONV。这一结果提示, 麻醉方式可通过影响自主神经稳定性而间接影响 PONV 的发生。目前所有有关 HRV 与 PONV 的研究中, 均是监测术前、术中或者术后某一个单一时间点的 HRV 指标或者不用时间点的变化来探索 HRV 与 PONV 的关系, 且研究结果不尽相同, 但是在了一项癌症化疗后恶心相关研究[64]中, Morrow 等研究者以心搏间期连续差值的标准差(standard deviation of successive differences, SDSA)动态评估了 24 名卵巢癌女性受试者在抗癌化疗后的自主神经变化(SDSA 是 HRV 时域指标, 可反映迷走神经活性)。发现 SDSA 先升高再下降, 但是患者首次恶心几乎全部发生在 SDSA 峰值之后的下降期, 提示迷走神经可能让机体进入更易出现恶心的准备状态, 但触发恶心还需要额外刺激。这提醒我们 PONV 的发生可能不仅仅是取决于某一单一时点的 HRV, 很可能与围术期迷走和交感神经平衡的动态变化轨迹有关, 也许采用连续或多时间点 HRV 指标来构建 PONV 风险预测模型会有意外的发现。

总体而言, HRV 作为自主神经功能的客观指标, 为理解 PONV 提供了新的视角, 但不同手术麻醉模式下其与 PONV 的关系相当复杂, 未来仍需要更深入的研究来统一结论, 以期能够提前发现自主神经活动异常的患者, 提前采取针对性的措施。

3.3. 围术期混杂因素对 HRV 的干扰及 PONV 预测准确性的影响

尽管现有研究表明 HRV 在 PONV 风险预测中展现出一定应用前景, 但在围术期环境下, HRV 信号极易受到多重干扰因素影响, 因此临床解读 HRV 预测结果时需谨慎[65]。麻醉药物本身就可直接影响自主神经系统功能平衡, 无论是吸入性麻醉剂、丙泊酚或阿片类药物均会导致 HF、LF 及 LF/HF 等参数的变化[66], 这提示围术期 HRV 的改变可能并不完全反映 PONV 易感性的真实变化。循环容量状态是另一重要干扰源, 失血、低血容量状态、静脉回流减少及液体治疗差异均可通过压力反射机制和交感神经系统代偿性激活改变 HRV, 尤其伴随低血压或心动过缓时, 更容易与迷走兴奋相关的 PONV 信号相混淆[67]。此外, 血管活性药物的应用同样显著影响自主神经输出状态, 如去氧肾上腺素、麻黄碱、去甲肾上腺素及抗胆碱类药物均可能干扰 HRV 指标的生理学解读[68]。除上述因素外, 机械通气参数设置、呼吸频率变化、体位改变、疼痛刺激、焦虑情绪、体温波动及术后躁动等均可导致 HRV 参数波动[69]-[71],

进而降低其对 PONV 的预测特异性。现有研究在采样时程、分析方法、伪差处理策略及指标选择等方面存在明显异质性, 这亦是导致当前 HRV 预测 PONV 的结论缺乏一致性的重要原因[61][72]。因此, 未来研究需建立标准化围术期 HRV 采集时间窗和分析流程, 并在统计模型中充分校正麻醉深度、循环状态及血管活性药物应用等关键混杂因素, 以提高 HRV 在 PONV 风险分层中的临床适用性。

4. 自主神经调节的干预措施

4.1. 动物实验证据

动物模型为阐明自主神经与呕吐反射的机制提供了重要证据。由于常用实验动物(如鼠类)不存在呕吐反射, 研究者常采用貂、犬等具备呕吐生理反应的动物。既往一项经典研究在雪貂模型中, 电刺激胃窦黏膜, 通过迷走传入通路可触发显著的胃运动变化和呕吐反射[73]。另一项动物实验研究发现, 与正常犬相比, 迷走神经切断后的犬, 其对血管加压素引起的胃慢波异常与呕吐或恶心样行为的效应明显减弱[74], 这提示迷走神经(尤其是迷走传出神经通路)参与其中。这些动物研究均奠定了胃肠-迷走-延髓中枢通路在恶心呕吐发生中的理论基础, 也为自主神经干预措施提供了方向。

4.2. 外科手术

在不同外科手术中均可能刺激到迷走神经而诱发恶心呕吐, 尤其是某些特定操作, 例如眼科手术牵拉眼球引发的动眼迷走反射(眼心反射), 术者应提前告知麻醉医生并预防性使用抗胆碱药物等, 以免出现心率骤降和恶心呕吐。还有研究基于动物实验证据探索了迷走神经离断术对于 PONV 的影响。例如一项观察性队列研究[6]纳入 1187 例行迷走神经离断术(食管切除、胃切除)患者和 2036 例非迷走神经切断术的手术患者作为对照, 结果发现迷走神经离断组 PONV 发生率仅 11.9%, 显著低于保留迷走神经的对照组 28.7%, 多因素逻辑回归分析表明迷走神经切断术是 PONV 的显著相关因素之一。虽然不能为了防止呕吐而切断迷走神经, 但是基于动物和临床试验, 我们未来可以探索可逆性的短效的靶向迷走神经阻滞用于高危 PONV 患者的围术期管理。

4.3. 药物干预

在围术期 PONV 的药物预防与治疗中, 5-HT₃ 受体拮抗剂(5-HT₃ receptor antagonists, 5-HT₃RA)是临床应用最广泛、证据最充分的一线止吐药物, 代表性药物包括昂丹司琼、格拉司琼、托烷司琼、雷莫司琼及帕洛诺司琼等[1][75][76]。多项国际共识指南均将 5-HT₃RA 列为 PONV 多模式管理的基础用药, 强调其既可作为单药用于中低风险人群的预防, 也可与糖皮质激素、NK1 受体拮抗剂等联合用于高危患者的综合预防, 也亦适用于术后出现恶心呕吐后的补救治疗[75][77]。其在 PONV 管理中之所以占据核心地位, 关键在于该类药物的作用位点与致吐信号的传递通路高度吻合, 围术期多种刺激(手术应激、麻醉药物、胃肠道牵拉或化学刺激等)可诱导胃肠道肠嗜铬细胞释放 5-羟色胺(5-HT), 5-HT 通过激活胃肠道迷走传入末梢的 5-HT₃ 受体, 启动迷走传入放电并将致吐信息上传至脑呕吐中枢, 且 5-HT₃ 受体在延髓相关结构(如化学感受触发区及孤束核 NTS)亦有较高表达, 参与致吐的关键环节。因此, 昂丹司琼等 5-HT₃RA 通常被认为可在外周(迷走传入末梢)与中枢(化学感受触发区/NTS)两端同时发挥阻断作用, 抑制迷走兴奋信号的产生与上传, 并削弱脑干呕吐网络的整合输出, 从而实现稳定的止吐效果, 这也说明了迷走神经通路在恶心呕吐中的关键作用[15][36][78]。

抗胆碱能药物可减少前庭和迷走对呕吐中枢的刺激, 从而预防 PONV。此类药物应用最广泛的是东莨菪碱透皮贴片[79]-[81]。东莨菪碱是外周和中枢的抗胆碱药, 作为竞争性拮抗剂阻断副交感神经节后纤维的乙酰胆碱 M 受体, 通过抑制迷走神经对胃肠道和平衡器官的过度激活, 对恶心呕吐具有良好预防作

用[82]。研究表明,术前使用东莨菪碱贴片可将术后 PONV 的发生率显著降低,相对危险度下降约 30%~40%,效果接近或优于单一昂丹司琼等药物[81]。

全身麻醉期间交感神经过度激活(应激、疼痛)也被认为可能是触发 PONV 的因素之一。因此一些镇静镇痛药,如 α_2 肾上腺素能受体激动剂右美托咪定、可乐定等可通过抑制交感神经减少儿茶酚胺释放,使血流动力学更平稳,并且减少术中或术后的阿片类药物使用,从而间接降低 PONV 发生率。多项随机对照试验和 Meta 分析[83]-[85]一致表明静脉持续泵注低剂量右美托咪定可显著降低术后恶心呕吐发生率,并减少术后镇痛药物需求。 β 受体阻滞剂中尤其是超短效的艾司洛尔,是一种心脏选择性 β_1 -肾上腺素能受体拮抗剂,也广泛用于围手术期交感神经的阻滞,控制术中心率。有随机对照研究发现围手术期使用艾司洛尔也可显著降低术后恶心呕吐和术后疼痛,有助于患者更早出院[86][87]。

4.4. 经皮穴位电刺激

经皮穴位电刺激(transcutaneous electrical acupoint stimulation, TEAS)是在特定穴位(如内关穴)贴附电极,给予低频电脉冲刺激[88]。内关穴(双侧腕内内侧横纹上约 2 寸处)是传统中医治疗呕吐的要穴[89]。TEAS 作为围术期 PONV 预防的重要方法,可通过激活脑-肠轴、调节自主神经功能和抗炎等多种机制来降低 PONV 发生率[90][91]。一项随机研究[92]在妇科腹腔镜手术患者中比较了 TEAS 组和假刺激对照组,结果显示 TEAS 使术后 24 小时 PONV 发生率从对照组的 67.4%降低至 42.9%,绝对减少约 24.5%。Wang [93]等在宫腔镜手术患者中,使用腕带式 TEAS 干预,并指导受试者术后持续佩戴 24 h,研究显示 TEAS 组术后 24 h 内 PONV 发生率较对照组降低约 26%。TEAS 的优势在于安全简便、非侵入且不良反应极少,患者佩戴腕部电刺激贴片并不影响手术操作和术后活动,依从性良好。但 TEAS 的最佳刺激时间和频率目前并未明确,有待更进一步的研究。

5. 小结

术后恶心呕吐的发生机制与自主神经系统密切相关,迷走神经介导的胃肠-脑干通路被认为是 PONV 的主要诱发途径之一,但其余自主神经相关机制目前尚未明晰。心率变异性作为自主神经活动的客观指标,在 PONV 预测方面展现出前景,但现有研究方法及指标并未统一,研究结果不尽相同,未来需要进一步大规模研究明确 HRV 各指标的监测时点(连续或单一)以及预测价值。对于 PONV 自主神经干预,传统的药物干预已取得非常好的效果,再辅以穴位电刺激等非药物疗法进行多模式预防及治疗,尽管 PONV 的发生率已经显著降低,但是据统计目前仍有 30%,随着对自主神经机制认识的深化,未来可能开发出更新的神经调控技术(如精准可逆的迷走神经调节装置)来进一步降低 PONV 的发生。

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