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自主神经在功能性胃肠病病理生理过程中的作用与研究

宋艳惠，杨　渊，李　存，盛　鑫

摘　要： 自主神经在中枢神经系统与肠神经系统中起着双向调节作用,影响胃肠道运动、消化、吸收功能,心率变异性(HRV)作为评估自主神经功能常见指标在功能性胃肠病(FGIDs)中进行了越来越多的研究。本文主要对自主神经在FGIDs病理生理过程中的作用及临床应用进行综述,以期为FGIDs的研究和临床治疗提供新的思路。

关键词： 自主神经；　心率变异性；　功能性胃肠病

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Role of autonomic nerves in the pathophysiological process of functional gastrointestinal disorders　*SONG Yanhui, YANG Yuan, LI Cun, et al. (Tongji Hospital, Tongji Medical College of Huazhong University of Science and Technology, Wuhan* 430030, *China)*

Abstract: Autonomic nerves play a bidirectional regulatory role in the central nervous system and enteric nervous system, influencing gastrointestinal motility, digestion, and absorption. Heart rate variability has been increasingly studied as an index to assess autonomic function in functional gastrointestinal disorders (FGIDs). This article reviews the role of autonomic nerves in the pathophysiological process of FGIDs and related clinical applications, with the aim to provide new ideas for the research and clinical treatment of FGIDs.

Key words: Autonomic nerve; Heart rate variability; Functional gastrointestinal disorder

功能性胃肠病(functional gastrointestinal disorders,FGIDs)主要表现为慢性或复发性腹痛、腹泻、便秘、腹胀、恶心和呕吐、胸骨后疼痛不适等。成人FGIDs依据器官分类,如食管相关疾病、胃十二指肠疾病等[1]。FGIDs影响全球超过40%的人口,研究最多的肠易激综合征及功能性消化不良的患病率分别为4. 1%和7. 2%[2]。患者反复就诊及检查、大量药物使用甚至手术、生活质量及工作能力下降,给社会生产力及卫生资源带来沉重负担[3]。FGIDs又称脑-肠互动异常,其病理生理机制尚未完全明确,自主神经系统是脑-肠相互作用中的重要组成部分,介导中枢神经和肠神经系统之间的双向通信[4]。中枢自主神经网络接受外周自主神经的传入信息,信息在中枢处理后,反馈信息通过自主神经传送至外周靶器官,调节胃肠道动力、敏感性、通透性、炎症及免疫功能等,进而引起FGIDs的胃肠道症状[5]。

1　自主神经参与功能性胃肠病的病理生理过程

自主神经系统(autonomic nervous system,ANS)参与维持胃肠道各种功能的稳态,其包含交感和副交感神经系统。副交感神经系统包括迷走及盆神经。支配胃肠道的迷走神经传入纤维周围突终止于胃肠道壁的各层结构内,形成各种类型的神经末梢,中枢突上行后主要终止于孤束核[6,7],孤束核发出纤维投射于臂旁核、中央杏仁核、室旁丘脑核、下丘脑内外、脑室旁区等脑区[8],这些脑区与控制食物摄取、情绪和行为反应、疼痛处理密切相关。中枢神经系统整合迷走神经传入的机械及化学信息,并向迷走神经传出纤维起点的迷走神经背侧运动核(dorsal motor nucleus of the vagus nerve,DMVN)和交感神经传出纤维起点的脊髓中间外侧柱发出下行纤维,从而调节自主神经活动[9,10]。调节胃肠道的交感神经节前纤维来自脊髓中间外侧柱,主要发出4种类型的节后纤维支配胃肠道,包括血管收缩纤维、运动抑制纤维、分泌抑制纤维和稀疏地支配黏膜、非括约肌、淋巴组织的纤维[11]。

1.1　自主神经参与胃肠动力紊乱　呕吐、腹泻、便秘和其他许多FGIDs的胃肠道症状由胃肠道动力紊乱造成,自主神经在胃肠运动中起着重要作用,迷走神经激活和/或交感神经抑制可增强胃肠运动。在动物中,经皮迷走神经刺激(transcutaneous vagus nerve stimulation,VNS)已被发现通过抑制交感神经并增强迷走神经改善胃肠运动障碍[12]。Shi等人和Huang等人各实施一项随机对照试验,评估了便秘型IBS患者在4周VNS后相较于对照组血清TNF-α和IL-6水平降低、迷走神经张力增高、腹部疼痛及便秘较前明显改善,并通过相关性分析认为VNS通过增强迷走神经活动,调节肠道运动及敏感性,从而改善便秘及腹痛症状[13,14]。

1.2　自主神经参与内脏高敏感　在功能性胸

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作者单位:(华中科技大学同济医学院附属同济医院,湖北 武汉 430030)

通信作者:盛　鑫,E-mail:479290356@qq. com

痛、功能性消化不良、肠易激综合征等FGIDs中，腹痛、腹胀、腹部不适与胃肠动力的关联性不大，可用内脏高敏感性解释。自主神经可通过胃肠道免疫失调、炎症和屏障功能受损引起肥大细胞低度活化和炎性介质的分泌增多，可以改善对肠道黏膜和肌间神经丛接收器的敏感度，从而导致内脏的高敏感[15]。迷走神经通过3种方式发挥免疫抗炎作用：(1)迷走神经传入纤维-孤束核-下丘脑室旁核，从而涉及下丘脑-垂体-肾上腺素轴(HPA)的神经内分泌通路，肾上腺释放的皮质类固醇激素使促炎细胞因子分泌减少，进而发挥全身抗炎作用；(2)胆碱能相关抗炎通路(cholinergic anti-inflammatory pathway，CAP)，通过肠内神经元迷走神经传出纤维间接作用于附近巨噬细胞的α7-烟碱型乙酰胆碱受体(α7nAChR)，抑制其释放促炎细胞因子如TNF-α、IL-1β等，快速且准确特异地发挥局部免疫抗炎作用[16]；(3)脾交感神经抗炎通路，迷走与脾交感神经相互作用，抑制促炎因子的释放，但目前这条通路具体机制尚不明确[17]。同时由于中枢ANS与疼痛感知网络区域有很大重叠，包括下丘脑、杏仁核、岛叶皮质、前扣带回等，迷走神经具有疼痛抑制作用的另一潜在机制可能是其将信息通过孤束核传至中枢ANS如臂旁核等，进而产生下行疼痛抑制信号[18]。研究表明更强的副交感神经活动与更低的疼痛感知相关[19,20]，低强度VNS通过激活迷走神经传入A_β纤维减少内脏疼痛[21]，而迷走神经切除术增加疼痛[22]。交感神经具有抗炎和促炎的双重作用，由于交感神经、肠神经系统、黏膜免疫功能之间存在复杂的相互作用，其对炎症效应介导的具体途径需更多研究阐明。在动物模型中发现通过α_2肾上腺素受体介导的促炎作用和通过β_3肾上腺素受体介导的抗炎作用的证据[23]。交感神经也参与内脏高敏性，CAN对其下行抑制减弱，使得内脏痛觉阈值降低[24]。在慢性内脏疼痛的大鼠模型中发现α_2肾上腺素受体激动剂和α_1肾上腺素受体拮抗剂，可以降低内脏超敏反应，并增强疼痛。化学交感神经切除术和手术交感神经切除术导致慢性内脏超敏反应的丧失[25]。

2 心率变异性(heart rate variability，HRV)在功能性胃肠病中的应用

评估自主神经功能的方式多种多样，包括HRV、压力反射敏感性(baroreflex sensitivity，BRS)、Ewing试验、瞳孔测量、血/尿儿茶酚胺测定、交感神经皮肤反应等，其中HRV虽然理论上评估心脏自主神经功能，但已证实心脏自主神经与胃肠道自主神经具有相关性[26,27]，被用作胃肠道ANS的间接标记物[28,29]，由于其客观、无创、廉价、便捷的特性已广泛用于FGIDs患者自主神经功能的评估。

2.1 HRV的相关概念 心率变异性(HRV)是指相邻R-R间期之间的间隔变化。HRV分析包括3种分析方法：时域、频域、非线性域分析[30]。时域分析主要是通过分析RR间期的统计指标得出ANS的总张力变化，常用的时域指标有平均正常RR间期标准差(SDNN)、相邻RR间期的均方根(RMMSD)、相差大于50 ms相邻RR间期占RR间期总数的百分比(pNN50)以及相邻RR间期之差的均方根(RMMD)等。频域分析方法将RR间期时间序列通过时频分析的方法得到频谱信息，临床主要意义是记录交感神经及迷走神经功能变化，常用的频域分析指标有总功率(TP)、超低频功率(ULF)、极低频功率(VLP)、低频功率(LP)和高频功率(HF)。非线性域分析方法常用分析指标有熵和庞加莱图等[31]。3种分析方法各有优缺点，时域指标计算简单，但不易区分副交感和交感系统的活动；频域指标计算方法虽略为复杂，但弥补了时域指标不能区分交感和副交感的不足；非线性指标因其代表的临床意义不明，尚未广泛使用。

2.2 功能性胃肠病的HRV指标特点 FGIDs患者相对于健康对照存在HRV异常。研究将IBS患者依据粪便类型、腹痛严重程度、病程长短、有无焦虑或抑郁分组时，发现腹泻型IBS[32]、腹痛严重[33]、病程长、合并焦虑抑郁患者[34]存在HRV的异常。在IBS患者中发现腹痛与HRV中的TP和LP呈正相关[35]。IBS患者HRV的最新元分析中发现，IBS相对于健康对照HF降低，提示副交感神经活动减低或交感神经功能增强[36]。Polster等人对IBS患者的HRV比较发现白天、站立试验期间的HRV显著低于对照组，而夜间、仰卧位期间则无明显差别，解释为当ANS面临更多“挑战”时，IBS无法像对照组有效地应对心理或生理应激，更容易显现出异常的HRV；并对IBS患者进行主成分分析及正交偏最小二乘方判别分析，发现年龄较大女性及腹泻患者更容易出现异常HRV[37]。同时HRV与胃肠道症状严重程度、病程长短相关[38]。由于压力诱导的下丘脑-垂体-肾上腺素轴及ANS的功能障碍在FGIDs中得到公认，越来越多的研究评估了FGIDs患者应对应激源时的自主神经功能变化，如Cheng等人在校正了性别、年龄、体重指数、当前焦虑状态等因素后发现IBS患者较健康对照在应对内脏应激源-乙状结肠镜检查期间HF减低及LF/HF增高不显著，病程长的IBS患者更明显，提示IBS的ANS在应对内脏应激源时表现迟钝[39]。Kano等人与Cheng得到相似的结果，IBS患者病程越长，LF/HF增高越不显著，提示这些患者的ANS“钝化”[18]。而Tillisch等人发现IBS患者在直肠乙状结肠扩张期间交感神经活动增强，副交感神经活动减低，且差异主要见于男性[40]。Walker等人的一项前瞻性研究发现，在串行减法任务等应激状态下腹痛持续组女性相较于腹痛缓解组女性SDRRI及HF显著减低，即年轻女性、持续腹痛患者更易出现HRV异常，提示副交感神经功能减退[41]。Kursat等人发现，功能性消化不良(FD)相较于健康对照组RMMSD、SDNN、pNN50均减低，提示副交感神经张力下降[42]。Guo等人对FD患者胃排空功能及HRV测定，发现50.9%的患者有近端胃排空延迟，其HF明显减低，并

相对于餐后胃排空正常的FD患者LF/HF明显增高，认为近端胃排空延迟可能是由于餐后迷走神经活动减少导致的交感迷走神经平衡中断所致，改善迷走神经活动可能是治疗FD患者的一种有效的治疗方法[43]。Hoshikawa等人未发现反刍综合征患者的评估迷走神经指标RMSSD与健康对照存在区别，但由于其只纳入10例患者，可能犯二类错误[44]。

2.3　HRV在FGIDs治疗中的应用　FGIDs的治疗有药物治疗和非药物治疗。药物治疗包括止泻、解痉等，是目前FGIDs的主要治疗方案。非药物的补充治疗包括认知行为疗法（CBT）、肠道靶向催眠疗法、身心疗法、正念疗法、益生菌等[45,46]。一项对便秘型肠易激综合征患者实施的随机对照研究发现，接受8周CBT治疗后的试验组胃肠道症状、焦虑、抑郁程度明显低于对照组，且HRV中的HF明显升高、LF/HF比低于对照组，24周随访时症状持续改善[47]。Huang等人评估了便秘型IBS患者在4周VNS后相较于对照组腹部疼痛及便秘较前明显改善，且HRV中的HF升高、LF/HF比低于对照组，得出VNS通过调节自主神经改善胃肠道症状[14]。对于功能性消化不良患者研究发现应用CBT治疗[48]及VNS均可有效改善症状[49,50]。CBT、身心疗法等治疗改善胃肠道具体机制尚不完全清楚，Winggassen等人认为心理治疗通过改变疾病特定的认知、行为、焦虑的中介作用改善FGIDs的症状[51]。

自主神经的评估指标可作为FGIDs补充治疗疗效评价的客观指标及预测疗效指标。虽然补充治疗证实可改善患者胃肠道症状、心理健康和生活质量，但由于需要接受合格训练的医师及患者承担高昂费用，限制了上述治疗在临床的普及，因此识别哪些患者能从中更多或更少收益尤为重要，Jarrett等人发现CBT在改善交感神经张力较高的IBS患者腹痛方面的效果可能较差[52]。Davydov等人将IBS患者随机分为步行组或瑜伽组，评估2周治疗前后的胃肠道症状、HRV、BRS，报告BRS可作为步行/瑜伽治疗疗效预测指标，低BRS患者瑜伽后腹痛严重程度显著降低，但对于步行组，腹痛程度的降低与较高BRS相关[53]。FGIDs研究结果的评估主要是对患者进行量表评估，具有一定的主观性且回顾性，Magdalena等进行一项元分析，纳入在补充治疗前后均进行HRV评估的6项研究，报告补充治疗后改善了胃肠道症状和自主神经功能，并建议将HRV作为评估疗效的客观指标[46]。

3　总结与展望

自主神经参与FGIDs的病理生理过程，FGIDs患者存在自主神经功能障碍。HRV与胃肠道症状、严重程度、病程等相关，且被报道可用于补充治疗疗效客观评估及预测指标。未来研究可在以下方面扩展和深入：（1）深入自主神经参与FGIDs发病机制的具体途径研究，为FGIDs提供更多治疗手段；（2）增加治疗的前瞻性研究，为自主神经评估指标作为监控和预测治疗效果的生物标记提供更多证据支持。

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