

综述

矛盾性失眠脑电特征及治疗的研究进展

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[摘要] 矛盾性失眠 (paradoxical insomnia, Para-I) 又被称为假性失眠 (pseudoinomnia) 或失眠状态错觉 (sleep state misperception, SSM)。这类患者通常主诉患有严重失眠, 但缺乏睡眠紊乱的客观证据, 日间功能受损情况与患者所述的睡眠缺失程度不成比例。高估睡眠潜伏时间 (sleep latency, SL)、低估总睡眠时间 (total sleep time, TST) 是 Para-I 的主要特征。这种睡眠质量的错误评价妨碍了对睡眠障碍的诊断、严重程度及临床疗效的评估。Para-I 的发病机制仍不清楚, 可能与抑郁水平、焦虑水平、人格特征、社会关系质量、大脑结构和功能的特殊改变有关。基于多导睡眠监测 (polysomnography, PSG) 的失眠相关研究发现, 非快速眼动睡眠 (non-rapid eye movement sleep, NREM 睡眠) 和快速眼动睡眠 (rapid eye movement sleep, REM 睡眠) 的改变可能与失眠患者的主客观睡眠不一致程度有关。PSG 是诊断睡眠障碍的重要手段。它可以通过同步监测脑电图 (electroencephalogram, EEG)、肌电图 (electromyogram, EMG)、眼动电图 (electrooculogram, EOG)、口鼻气流、胸腹呼吸运动、血氧饱和度、心电图 (electrocardiogram, ECG)、鼾声等多项参数对睡眠结构及相关的生理行为变化进行分析。近年来, 越来越多的研究开始借助 PSG 对 Para-I 的睡眠 EEG 及治疗进行探索并取得了一定的进展。该文就 Para-I 的脑电特征和治疗的最新进展做一综述, 以期 Para-I 的精准治疗提供新的思路。

[关键词] 矛盾性失眠; 主客观睡眠不一致; 多导睡眠监测; 失眠认知行为治疗

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Research advances in the electroencephalographic characteristics and treatment of paradoxical insomnia

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[Abstract] Paradoxical insomnia (Para-I), also known as pseudoinomnia or sleep state misperception, is a condition in which the patient complains of severe insomnia but has no objective evidence of sleep disorder, and daytime functioning may be disrupted disproportionately to the degree of patient-reported sleep loss. Para-I is characterized by overestimation of sleep latency (SL) and underestimation of total sleep time (TST). Incorrect assessment of sleep quality hinders the diagnosis, evaluation of severity, and assessment of clinical efficacy of sleep disorders. The pathogenesis of Para-I remains unclear, but may be related to factors such as depression, anxiety, personality traits, social relationships and specific changes in brain structure and function. Studies on the polysomnography (PSG) of the patients with insomnia have found that changes in non-rapid eye movement (NREM) and rapid eye movement (REM) sleep may be related to the degree of subjective-objective sleep discrepancy. PSG is a valuable diagnostic tool for sleep disorders. It allows for the analysis of sleep structure and related physiological and behavioral changes by monitoring various parameters, including electroencephalogram (EEG), electromyogram (EMG), electrooculogram (EOG), oro-nasal airflow, thoracic and abdominal respiratory motions, oxygen saturation, electrocardiogram (ECG) and snoring. In recent years, studies have increasingly explored the sleep EEG and treatment of Para-I with PSG, resulting in significant progress. This article reviews the latest advances in the electroencephalographic characteristics and treatment of Para-I, providing new ideas for precise treatment.

[Key words] paradoxical insomnia (Para-I); subjective-objective sleep discrepancy; polysomnography (PSG); cognitive behavioral therapy for insomnia

矛盾性失眠 (paradoxical insomnia, Para-I) 又被称为假性失眠 (pseudoinomnia) 或失眠状态错觉 (sleep state misperception, SSM)。这类患者常主诉患严重失眠, 但无睡眠紊乱的客观证据, 并且患者睡

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眠相关的日间功能受损情况与其所述的睡眠缺失程度不成比例^[1-2]。Para-I 主要特征为高估睡眠潜伏时间 (sleep latency, SL)、低估总睡眠时间 (total sleep time, TST)。这种对于睡眠质量的失实评价严重影响了诊断及治疗的有效实施。目前,临床对其重视还不足,相关发病机制不甚明了,也缺乏较好的治疗方案。本文主要从临床表现、脑电特征及治疗这3个方面对 Para-I 的相关研究以及最新进展做一综述。

1 Para-I 的临床表现

1.1 概念演变

1990 年睡眠障碍国际分类 (International Classification of Sleep Disorders, ICSD) 第1版^[3]中对主客观睡眠不一致的现象进行了讨论,并将其命名为 SSM; 2005 年 ICSD 第2版^[2]取消了 SSM 的相关描述,在慢性失眠障碍中提出 Para-I 这一亚型,并提出了定量诊断的参考标准: TST \geq 6.5 h 且睡眠效率 (sleep efficiency, SE) $>$ 85%。但这一标准存在较多的争议,因此在 2014 年出版的 ICSD 第3版中仅在慢性失眠障碍中保留了 Para-I 的概念。

1.2 伴发疾患和人格特点

由于缺乏一致的定量诊断标准,研究^[4-6]中报告的失眠患者中 Para-I 的患病率数据差异较大 (9.2%~50%)。这种主客观睡眠不一致的现象在其他睡眠、精神障碍等疾病中也有报道,如阻塞性睡眠呼吸暂停 (obstructive sleep apnea, OSA)^[7-8]、创伤后应激障碍 (post-traumatic stress disorder, PTSD)^[9]、边缘型人格障碍 (borderline personality disorder)^[10]、双相障碍 (bipolar disorder)^[11]、抑郁症 (depressive disorder)^[12] 等。目前 Para-I 与这些疾病共病的相关研究较少。

失眠的心理学相关研究^[13]发现 Para-I 患者的焦虑、抑郁水平显著高于普通人群。焦虑被认为是高估 SL 的独立预测因素^[14],抑郁在低估 TST 和失眠严重程度之间起到完全中介作用^[15]。另有研究^[16]发现,Para-I 组 ($n=28$) 在明尼苏达多相人格测验 (Minnesota Multiphasic Personality Inventory, MMPI) 中的人际关系敏感、抑郁、焦虑、敌对、偏执和精神病性因子得分明显高于非 Para-I 组 ($n=30$) 和睡眠良好组 ($n=10$); 一项中年社区居住人群睡眠状况的研

究^[17]中也报告了社会关系质量对低估 TST 的影响。但在更大样本量 ($n=249$) 的研究^[18]中并没有发现 Para-I 与非 Para-I 患者在 MMPI 各因子得分上的差异。

此外,影像学研究^[19]发现,Para-I 患者存在尾状核、壳核和丘脑的形状改变,而非 Para-I 患者表现为丘脑、杏仁核和海马萎缩。这些结果提示 Para-I 可能具有独特的病理生理机制。分析 Para-I 患者客观睡眠质量对于深入了解 Para-I 的发病机制及治疗至关重要,但由于研究方法的限制,这些研究未对其进行深入分析。

2 睡眠脑电特征

多导睡眠监测 (polysomnography, PSG) 是睡眠障碍诊断的重要手段,它可以同步监测脑电图 (electroencephalogram, EEG)、肌电图 (electromyogram, EMG)、眼动电图 (electrooculogram, EOG)、口鼻气流、胸腹呼吸运动、血氧饱和度、心电图 (electrocardiogram, ECG)、鼾声等多项参数。近年来越来越多的研究开始借助 PSG 对 Para-I 患者的睡眠 EEG 进行分析,从客观睡眠质量的角度探索 Para-I 可能的发病机制及有效的治疗措施。

2.1 睡眠结构

非快速眼动睡眠 (non-rapid eye movement sleep, NREM 睡眠) N3 期^[20]和快速眼动睡眠 (rapid eye movement sleep, REM 睡眠)^[21-22]结构的改变可能与 Para-I 有关。

N3 期睡眠,又称为“深度睡眠”或“慢波睡眠”,被认为是睡眠深度最深的阶段,通常占青年至中年成人总睡眠时间的 10%~20%,并随年龄增加而减少^[23]。YOON 等^[20]回顾了 150 例失眠患者的自我报告和客观睡眠指标,将患者分为低估 TST ($n=115$) 和高估 TST ($n=35$) 组。结果发现低估 TST 组 N3 期睡眠更少 ($P=0.002$),同时低估 TST 组患者报告了更高的匹兹堡睡眠质量指数 (Pittsburgh sleep quality index, PSQI) 评分 ($P<0.001$)、更长的 SL ($P=0.001$) 以及更大主客观 SL 差异 ($P=0.001$)、更高的疲倦感和早醒 ($P=0.029$ 、 $P=0.038$); 多元回归分析显示, N3 期睡眠时间、PSQI 和早醒与失眠患者 TST 的主客观差异显著相关,这意味着这些患者可能没有足够的深睡眠,可能更容易将睡眠状态感知为清醒从

而造成主客观睡眠不一致。

一些研究在REM睡眠中也发现了Para-I患者睡眠深度的改变。STEPHAN等^[21]进行的一项连续唤醒试验比较了Para-I患者($n=10$)与睡眠良好者($n=20$)在NREM/REM睡眠时主观的睡眠深度。他们在受试者进入NREM/REM睡眠10 min后对受试者进行唤醒,在唤醒后询问受试者唤醒前的睡眠感受并要求他们对睡眠深度进行评分。结果发现Para-I患者较睡眠良好者更容易将睡眠状态感知为清醒;两者在NREM睡眠时感受的睡眠深度相似,但Para-I患者REM睡眠的主观睡眠深度显著低于睡眠良好者。另有研究^[22]发现,Para-I患者REM睡眠时微觉醒次数显著增加。RIEMANN等^[24]提出的“REM睡眠不稳定”假说认为REM睡眠时觉醒增加可能会使睡眠状态更容易被感知为清醒并被存储和回忆。REM睡眠微觉醒次数的增加可能使Para-I患者更容易感到睡眠中断与清醒。

2.2 睡眠脑电活动

另有多项研究对睡眠EEG的成分进行了更为复杂的分析,进一步探索了主客观睡眠不一致可能的机制。循环交替模式(cyclic alternating pattern, CAP)、睡眠纺锤波(sleep spindle)密度、高频脑电功率、大脑半球神经元电活动的不对称、1/f斜率(1/f slope)下降等被认为可能与Para-I的主客观睡眠不一致有关。

CAP是在NREM睡眠时出现的一种周期性脑电活动,与脑岛、基底前脑、中扣带回等脑区活动有关,代表着睡眠的不稳定性及高警觉水平^[25-26]。每个CAP循环包括一个A相(A1、A2、A3亚型)和一个B相。A1亚型主要出现在深睡眠或浅睡眠向深睡眠转化的过程中,而A2或A3亚型常见于深睡眠向浅睡眠转化过程中^[27]。PARRINO等^[28]发现Para-I患者N1和N2期CAP出现率增高,A2亚型占比增加,提示Para-I患者可能存在更多深睡眠向浅睡眠转化的过程,睡眠更不稳定、睡眠深度更浅。

睡眠纺锤波是NREM睡眠时的特征性脑电波形,主要在N2期睡眠中出现。BENBIR ŞENEL等^[29]研究发现,Para-I患者($n=20$)睡眠纺锤波的持续时间和频率与睡眠良好者($n=20$)相似,但密度明显降低。睡眠纺锤波的密度被认为与抵抗外部干扰的能力有关,密度越高抗干扰能力越强^[30]。睡眠纺锤波密

度降低可能使Para-I患者在睡眠过程中更容易感受到外部环境刺激,从而将睡眠状态感知为清醒。

EEG频谱分析的相关研究^[31]发现,低估TST的失眠患者睡眠EEG的 β/δ 功率值更高,而高估TST的失眠患者则较低。XU等^[32]对年轻(18~40岁)失眠患者的EEG频谱的功率与TST主客观差异的相关性进行了进一步的探索,结果发现年轻失眠患者额叶 β/δ 功率值及唤醒次数与TST主客观不一致的水平呈正相关。高频EEG活动的增加被认为与皮层的过度觉醒有关^[33]。这些研究结果提示,Para-I患者TST的低估可能与皮层过度觉醒有关。

此外,ST-JEAN等^[6]在功率谱分析的基础上比较了失眠患者($n=31$)以及睡眠良好者($n=19$)的大脑左右半球间额叶、顶叶和中央区脑电活动不对称性。结果发现,与睡眠良好者相比,Para-I患者($n=14$)左额叶区域失活,而右额叶区域过度激活。PROVENCHER等^[34]的研究进一步比较了失眠患者($n=43$)以及睡眠良好者($n=19$)的大脑半球内脑电活动的不对称性,发现右侧顶-枕区域(P4/O2) δ 不对称与失眠患者SL和TST的主客观差异有关。

非周期性脑电信号(1/f斜率)被认为是评价神经突触兴奋/抑制平衡的可靠神经生理学指标^[35],功率与频率成反比是其主要特征。ANDRILLON等^[36]对1/f斜率进行分析发现,失眠患者($n=347$)在N2和N3期睡眠中的1/f斜率较睡眠良好者($n=89$)显著降低,尤其在Para-I患者($n=59$)的N3期睡眠中该指标的下降更明显。这意味着,失眠患者的N2和N3期睡眠存在兴奋/抑制平衡受损,且在Para-I患者的N3期睡眠中更显著。

3 治疗

3.1 药物治疗

Para-I目前仍无共识性治疗方案。失眠的相关研究^[37]中发现,苯二氮草类/非苯二氮草类受体激动剂在改善客观睡眠的同时具有改善主观睡眠的效果,如佐匹克隆可增加NREM睡眠时间、减少SL高估,但TST低估可能增加。但苯二氮草类药物的长期使用可能会降低睡眠时的抗外部干扰能力,使CAP中A2和A3亚型增加,导致觉醒次数增加^[38]。因此,在选择治疗药物时应综合评估患者的主观和客观睡眠情况。此外,一些小规模的临床研究^[39]及个案报告^[40]报

道了喹硫平和奥氮平等非典型抗精神病药物对 Para-I 的疗效。KHAZAIE 等^[40]比较了利培酮和奥氮平治疗 Para-I 的效果,发现两者均可以改善睡眠质量,且奥氮平疗效更好。

3.2 心理治疗

近年来研究发现失眠认知行为治疗 (cognitive behavioral therapy for insomnia, CBTI)^[41-44]、基于正念的失眠治疗 (mindfulness-based therapy for insomnia, MBTI)^[45]等可能是改善 Para-I 患者主客观睡眠不一致的有效手段。

CBTI 可以显著改善失眠患者 SL、TST 的主客观差异^[41-43],而且主客观睡眠差异大的失眠患者的 CBTI 的疗效显著优于差异小的患者^[44]。一项研究^[44]比较了 TST 低估组 ($n=16$) 和准确/高估组 ($n=20$) CBTI 的疗效,结果发现 CBTI 可显著改善两组 TST 的主客观差异,同时还可以减少低估组 SL 的主客观差异。此外,他们的研究还发现在实施睡眠限制时,低估组在第 1 周时即观察到 TST 主客观差异显著减少,而准确/高估组在第 4 周时才出现显著变化。这一结果表明睡眠限制可能是改善低估 TST 的有效手段。

MBTI 可有效减少 SL 的主客观差异,从而改善睡眠质量和失眠症状。SHAIF 等^[45]的研究将 113 名老年失眠患者 (50~80 岁) 随机分配到 MBTI ($n=55$) 或睡眠卫生教育 (a sleep hygiene, education, and exercise programme, SHEEP) ($n=58$) 组。干预后,两组 SL 的高估显著降低,并且研究发现特质正念的增加可能是 MBTI 组高估 SL 改善的重要机制。

此外,有研究报道了持续气道正压通气 (continuous positive airway pressure, CPAP) 对 OSA

患者的主客观睡眠差异作用。CPAP 不仅可以使低估 TST 组的低估减少,同时可以使高估 TST 组的高估减少^[46]。在不伴有 OSA 的 Para-I 患者中,CPAP 是否具有这种改善作用目前仍不清楚。

4 总结与展望

Para-I 是一种复杂的失眠障碍,可能是多因素共同作用的结果。Para-I 睡眠 EEG 的研究加深了我们对于这一疾病客观睡眠情况的了解,未来可以考虑结合 EEG 分析、心理学、神经功能影像学等方法进一步探究 Para-I 的发病机制。此外,Para-I 目前尚无共识性的治疗方案,一些药物和心理治疗 (CBTI、MBTI 等) 可能具有改善 SL、TST 主客观差异的效果;对于共病 OSA 患者,CPAP 治疗具有较好的疗效^[46],在不伴有 OSA 的 Para-I 患者中是否具有改善效果有待进一步研究。

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