·神经调控前沿理论与临床转化·

非侵入性电磁神经调控治疗创伤后应激障碍认知损害研究进展

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【摘要】 创伤后应激障碍是严重创伤性事件引起的精神障碍，主要临床症状包括侵入性回忆、回避行为、认知功能和情绪障碍、过度警觉，与包括杏仁核、海马和前额叶在内的脑网络异常模式密切相关。目前尚无有效治疗方法，药物治疗和心理疗法效果有限，患者依从性差，且常伴不良反应或症状残留。非侵入性电磁神经调控技术因安全性高、可重复性强、患者接受度高等优势，成为改善创伤后应激障碍认知损害的研究热点。本文综述经颅电刺激和经颅磁刺激在创伤后应激障碍认知损害中的应用进展，总结其与心理疗法联合应用的潜在价值，以期为神经调控用于创伤后应激障碍认知损害的临床治疗提供新的思路。

【关键词】 应激障碍，创伤后； 认知障碍； 电刺激疗法； 经颅磁刺激； 综述

【中图分类号】 R741；R651

Progress on non-invasive electrical and magnetic neuromodulation therapy for cognitive impairment in posttraumatic stress disorder

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【Abstract】 Posttraumatic stress disorder (PTSD) is a severe mental disorder triggered by traumatic events. Its core clinical symptoms include intrusive memories, avoidance behaviors, negative alterations in mood and cognitive disorder, and hyperarousal. These symptoms are closely associated with aberrant neural circuitry involving the amygdala, hippocampus and prefrontal cortex. However, current pharmacological and psychological interventions are constrained by limited efficacy, poor compliance, and undesirable side effects. Given the high safety, strong reproducibility, and high patient acceptance, non-invasive electrical and magnetic neuromodulation techniques have emerged as a research focus for improving cognitive impairment in PTSD. This paper reviews the advancements in applying transcranial electrical stimulation (TES) and transcranial magnetic stimulation (TMS) to alleviate cognitive impairment in PTSD, and discusses the potential value of their combined use with psychological therapies. It aims to provide new perspectives for the clinical application of neuromodulation technique in treating cognitive impairment in PTSD.

【Key words】 Stress disorders, post-traumatic; Cognition disorders; Electric stimulation therapy; Transcranial magnetic stimulation; Review

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创伤后应激障碍(PTSD)指个体目睹或经历极端创伤性事件后出现的严重精神障碍[1],其核心可能是过度恐惧反应或恐惧抑制障碍[2]。在西方国家,其终生患病率为2%~7%[3];我国流行病学调查数据显示,其终生患病率约0.68%(95%CI:0.37%~1.25%),1个月患病率约0.09%(95%CI:0.04%~0.17%)[4]。创伤后应激障碍的主要症候群包括侵入性回忆、回避行为、认知功能和情绪障碍、过度觉醒等[5],并可能波及家庭和社会关系,导致广泛的情感和经济负担;此外,还常出现广泛的认知损害,包括对安全性、信任、自尊、亲密关系和控制感的系统性误判,这些由创伤诱发的认知损害持续影响情绪调节和行为决策,患者难以准确评估自身与外界,从而形成过度警觉、回避行为、错误归因和自我怀疑,是创伤后应激障碍的核心症状之一[6]。因此,探究改善创伤后应激障碍患者认知功能的系统治疗方法对有效减轻症状乃至治愈疾病具有重要临床意义和社会价值。传统治疗方法主要包括药物治疗和心理疗法,首选心理疗法为认知加工疗法(CPT)、长时间暴露、认知重组、眼动脱敏与再加工(EMDR)、认知行为疗法(CBT)等。2022年更新的《澳大利亚创伤后应激障碍预防与治疗指南第三版》[7]推荐心理疗法为首要治疗方法,但也强调某些情况下仍考虑药物治疗。《澳大利亚创伤后应激障碍预防与治疗指南第三版》[7]推荐文拉法辛、帕罗西汀、氟西汀作为备选治疗药物。然而,心理疗法依从性较差[8],治疗后可能残留部分症状[9],长期效果欠佳[7],且患者自身状态与心理咨询师特质等可能影响疗效[10];药物治疗则可能引发头晕、呕吐等不良反应[11]。因此,探寻一种治疗有效的新方法成为当务之急。神经调控技术是一种通过侵入性或非侵入性技术,利用电、磁、声、光等多种物理刺激方式和化学手段,靶向特定神经核团或神经网络节点,调节异常神经活动的技术[12]。本文聚焦经颅电刺激(TES)和经颅磁刺激(TMS),从作用机制、目标脑区、刺激参数等方面综述非侵入性电磁神经调控技术在创伤后应激障碍认知损害中的基础与临床研究现状及发展前景,以期为疾病的治疗提供新的视角。

一、创伤后应激障碍脑网络损伤机制

创伤后应激障碍与过度恐惧反应或者恐惧抑制障碍相关[2]。基于恐惧回路的关键结构,Rauch等[13]提出包含杏仁核、前额皮质(PFC)、海马在内的创伤后应激障碍神经回路模型(图1),并认为其间异常的相互作用是潜在发病机制。

脑区间神经网络的信息传递依靠神经节律,神经节律由神经元节律性同步电活动引发[14],其特定频率与认知功能密切相关;神经节律共同反映神经元网络功能特征,并在认知过程中得以体现[15]。认知功能相关神经节律包括与探索性学习和导航规划、工作记忆相关的θ节律(4~8 Hz)[16-17]以及与注意加工、多种认知功能及协调参与复杂认知功能的神经元活动相关的γ节律(30~150 Hz)[18]。杏仁核在恐惧记忆形成和表达过程中发挥核心作用,其神经节律和特定中间神经元调节恐惧记忆的维持和消退[19-20],在生理状态下杏仁核θ节律强度在恐惧记忆编码过程中明显增加[21],并参与恐惧记忆的维持[22];恐惧消退学习过程中,杏仁核表达钙结合蛋白(CaBP)的中间神经元同步活动表现为6~12 Hz节律,与恐惧相关节律(3~6 Hz)存在竞争,选择性抑制已编码的恐惧记忆。而创伤后应激障碍患者的竞争作用丧失,恐惧消退无法进行,导致恐惧记忆复发[23]。海马在创伤后应激障碍相关认知和记忆调节中发挥关键作用,背侧海马参与恐惧消退、情景编码和情景依赖性检索[24]。海马体积缩小[25]、功能低下及其与脑默认网络(DMN)等功能连接异常可导致恐惧记忆过度泛化、情景记忆检索损害、侵入性记忆增多及自我感削弱等,并减弱其与前额皮质协同调节情绪和记忆的能力[26]。同时,杏仁核和海马体存在不同的传入和传出投射,这些投射对于空间记忆、情景记忆、陈述性记忆和情感记忆的形成和动机过程至关重要[27]。前额皮质是调节恐

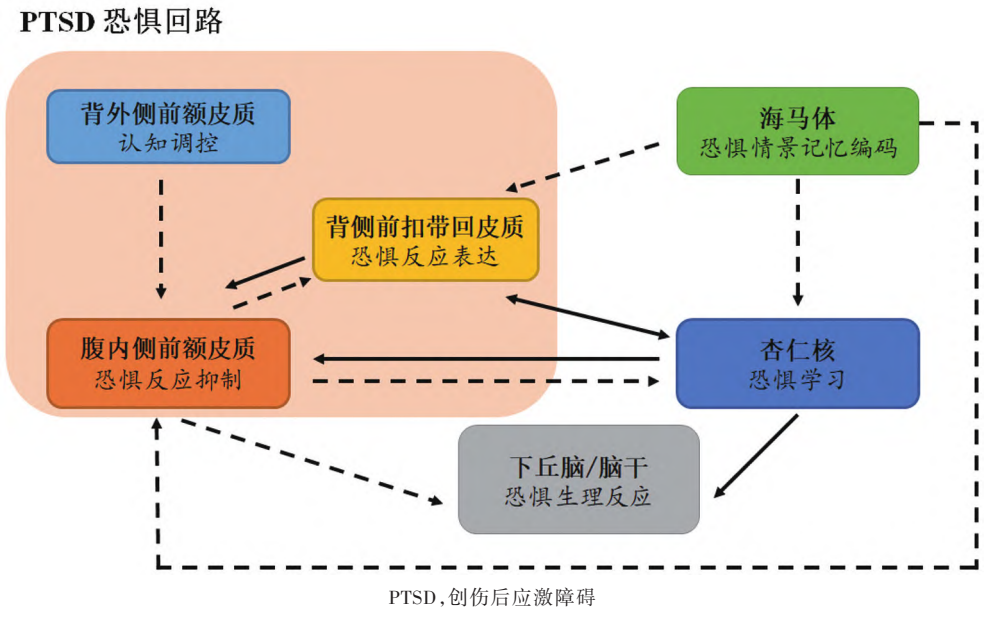

图1 创伤后应激障碍相关脑区间病理生理学机制联系（实线表示区域间连接，虚线表示区域间连接中断）

Figure 1 The pathophysiological connectivity among brain regions related to PTSD.

惧的重要脑区，包含多个亚区，恐惧记忆形成与消退过程中内侧前额皮质（mPFC）负责评估威胁信息并与下游脑区进行信息传递，抑制杏仁核驱动的恐惧反应并促进恐惧消退[28]。腹内侧前额皮质（VMPFC）与杏仁核相连，参与情绪处理、记忆和执行控制[29]，而背外侧前额皮质（DLPFC）通过与腹内侧前额皮质等前额叶区域的功能耦合间接影响杏仁核的活动。这些前额皮质亚区对杏仁核的直接或间接抑制作用损害可能导致创伤后应激障碍患者的过度恐惧反应和情绪调节障碍[30]。上述研究表明，杏仁核调节习得性恐惧过程，并接受来自前额皮质和海马的投射，创伤后应激障碍患者前额皮质和海马损伤，对杏仁核的控制减弱，表现为杏仁核对恐惧刺激的高反应性[31]，表明创伤后应激障碍患者异常行为的神经机制可能依赖多脑区神经节律的协同与竞争，为靶向干预提供了理论依据。

二、经颅电刺激研究进展

经颅电刺激属于非侵入性神经调控技术，通过在头皮表面施加低强度电流，改变大脑皮质神经元的跨膜电位，从而调节神经元兴奋性和神经可塑性。近年研究显示，经颅电刺激对感知、情绪、认知、记忆、运动等神经功能均有积极作用[32-34]。常见形式包括经颅直流电刺激（tDCS）、经颅交流电刺激（tACS）、经颅随机噪声刺激（tRNS）和经颅微电流刺激（CES）。

1. 经颅直流电刺激 tDCS通过在头皮放置成对的有源电极，持续施加低强度、恒定直流电，诱导阳极下方神经元去极化或阴极下方神经元超极化，改变细胞膜静息电位，从而调节目标皮质神经元兴奋性[35]。tDCS刺激停止后的持续作用可能涉及突触可塑性改变，尤其依赖N-甲基-D-天冬氨酸受体（NMDAR）活动[36]，与长时程增强（LTP）机制类似。脑源性神经营养因子（BDNF）/酪氨酸蛋白激酶B（TrkB）信号转导通路对维持该后效应至关重要[37]。tDCS阳极可以显著增加前额皮质氧合血红蛋白含量[38]，增强背外侧前额皮质及其连接网络的局部脑血流量[39]，共同调节大脑皮质兴奋性。尽管通常认为tDCS阳极增强皮质兴奋性、阴极抑制兴奋性，但认知任务期间阴极刺激的抑制效应可能不足以完全抵消任务诱导的激活强度[40-41]，主因tDCS对认知功能的复杂影响涉及多个脑区的协同作用[42]。此外，tDCS在改善认知功能方面的效应呈显著的非线性特征，其疗效受个体差异（如解剖结构、神经生理状态）等多种复杂因素的影响[43-44]。临床研究显示，tDCS在改善社会认知功能（如共情和情绪识别）及其相关情绪性记忆等领域颇具研究潜力，成为创伤后应激障碍干预研究的重点方向（表1）[45-48]。一项基于巴甫洛夫恐惧条件反射范式的研究对健康受

表1 经颅电刺激改善创伤后应激障碍认知损害的临床研究

Table 1. Clinical studies on TES improving cognitive impairment in PTSD

文献来源	研究对象	刺激靶点	刺激参数	范式或评估量表	疗效
van't Wout等[45](2016)	健康人群	VMPFC	阳极2 mA	巴甫洛夫恐惧条件反射、消退和回忆范式	对早期消退回忆无显著影响，加速晚期消退学习
Asthana等[46](2013)	健康人群	左侧DLPFC	阴极1 mA，12 min	巴甫洛夫恐惧条件反射、消退和回忆范式	抑制恐惧记忆巩固
Ahmadizadeh等[47](2019)	PTSD患者	DLPFC	2 mA，20 min×10次，30 s内强度自零缓慢升高至2 mA	CAPS等	改善认知功能，未改善回避行为
Han等[48](2022)	PTSD患者	F3(阳极)/F4(阴极)	2 mA，20 min×10次	CAPS等	基线认知功能较高患者获益更佳
van't Wout-Frank和Philip[56](2021)	PTSD退伍军人	VMPFC	2 mA，25 min×6次，30 s内强度自零缓慢升高至2 mA	战争相关虚拟现实驾驶任务	提出tDCS联合VRET方案，推进居家治疗
van't Wout-Frank等[57](2024)	慢性PTSD退伍军人	VMPFC	2 mA，25 min×6次，30 s内强度自零缓慢升高至2 mA	战争相关虚拟现实驾驶任务	治疗1个月后症状减轻，社交能力改善；tDCS联合VRET方案获得短期和长期疗效
Philip等[58](2024)	慢性PTSD退伍军人	VMPFC	2 mA，25 min×6次，30 s内强度自零缓慢升高至2 mA	战争相关虚拟现实驾驶任务	tDCS联合VRET方案获得短期和长期疗效
Eyraud等[59](2024)	慢性PTSD患者	左侧DLPFC	2 mA(20 min/次、2次/d+间隔20 min)×5 d	创伤剧本暴露	联合DLPFC刺激无益

PTSD，posttraumatic stress disorder，创伤后应激障碍；VMPFC，ventromedial prefrontal cortex，腹内侧前额皮质；DLPFC，dorsolateral prefrontal cortex，背外侧前额皮质；CAPS，Clinician-Administered Posttraumatic Stress Disorder Scale，临床管理创伤后应激障碍量表；tDCS，transcranial direct current stimulation，经颅直流电刺激；VRET，virtual reality exposure therapy，虚拟现实暴露疗法

试者在恐惧消退学习阶段进行前额皮质刺激，结果并未影响消退的早期回忆，但加速消退学习的后期进程[45]；在恐惧记忆巩固期对左侧背外侧前额皮质进行阴极tDCS刺激，可抑制恐惧记忆巩固[46]。另一项随机对照试验显示，针对背外侧前额皮质予以10次的tDCS虽可改善创伤后应激障碍患者认知功能，但对回避行为无明显疗效，提示其治疗效果具有症状特异性[47]。此外，tDCS的疗效还与患者基线认知功能密切相关，基线认知功能较好者改善效果更佳[48]。

2. 经颅交流电刺激　tACS通过在头皮放置成对的有源电极，施加低强度的正弦波交流电(电流0.50～2 mA，频率0.10～100 Hz)，于特定频率下与内源性神经节律相互作用，进而调节神经网络[49-50]，其核心作用机制在于神经夹带，即通过施加与目标脑区固有节律频率相近的交流电，使局部神经网络的节律活动与刺激频率同步化[51]。这种神经夹带效应可重塑神经网络中节律的时间和空间结构，进而调节依赖该频段节律的认知功能或行为过程[52]。动物模型研究显示，靶向腹侧海马CA1区的tACS可以有效促进创伤后应激障碍模型小鼠的恐惧消退，消除创伤记忆[53]。临床上，tACS主要用于认知功能障碍患者(如轻度认知障碍或痴呆)和老年人群的认知功能改善[54]。对于创伤后应激障碍认知损害的疗效尚待更多高质量研究进一步评估，优化刺激参数(如频率、强度、持续时间)，并深入探究其作用机制。

3. 经颅直流电刺激与心理疗法的结合　近年tDCS的应用方式进一步创新。有研究采用tDCS联合长期暴露疗法治疗创伤后应激障碍，结果显示，治疗后临床管理创伤后应激障碍量表5(CAPS-5)评分显著降低，初步证明居家tDCS联合家庭暴露疗法的可行性[55]。tDCS联合虚拟现实暴露疗法(VRET)在缓解症状方面展现出良好应用前景，有研究对创伤后应激障碍患者予以靶向腹内侧前额皮质的tDCS联合VRET，治疗后1个月症状明显减轻[56]，社交和职业能力显著提高[57]，长期随访显示，其复发率低且药物依赖性小[58]。然而，另一项随机对照试验对创伤后应激障碍患者予以靶向背外侧前额皮质的tDCS联合创伤剧本暴露疗法，并未显著改善整体症状[59]。表明tDCS的疗效存在一定异质性，可能受刺激靶点、患者基线神经生理状态及具体症状维度等因素的影响。总体而言，现有证据表明，tDCS与心理疗法结合在创伤后应激障碍的治疗中具有临床潜力，但其疗效受多重因素的影响，存在显著异质性，尚待通过优化刺激靶点和个体化刺激参数提高疗效(表1)[56-59]。

三、经颅磁刺激研究进展

经颅磁刺激通过对置于头皮表面的刺激线圈予以瞬时强电流，在线圈周围产生快速变化磁场，该磁场可穿透颅骨并在大脑皮质感应出电流，引发靶区神经元去极化，调节大脑皮质兴奋性[60]。根据

表2 经颅磁刺激改善创伤后应激障碍认知损害的临床研究总结

Table 2. The summary of clinical studies on TMS improving cognitive impairment in PTSD

文献来源	研究对象	刺激靶点	刺激参数	范式或评估量表	疗效
Cohen等[61](2004)	PTSD患者	右侧DLPFC	10 Hz	PCL、CAPS、HAMD	改善核心症状
Nam等[62](2013)	PTSD患者	右侧PFC	1 Hz×3周	CAPS	改善再体验症状
Isserles等[63](2013)	PTSD患者	mPFC	20 Hz,120% MT,2 s“开”/20 s“关”,3次/周,共4周	CAPS-2	促进恐惧消退,改善侵入性症状
Isserles等[64](2021)	PTSD患者	mPFC	18 Hz,100% MT,2 s“开”/20 s“关”,3次/周,共4周	DSM-5	未证实其可有效促进恐惧消退或减轻整体症状
Fryml等[65](2019)	PTSD患者	左侧或右侧前额皮质	10 Hz,120% MT,5 s“开”/10 s“关”,共5周	CAPS	rTMS联合长期暴露疗法安全可行
Kozel等[66](2018)	PTSD患者	右侧DLPFC	1 Hz,110% MT,30 min连续	CAPS、PCL等	rTMS联合CPT改善症状效果更佳
Seybert等[67](2021)	PTSD患者	右侧DLPFC	20 Hz,100% MT,2 s“开”/28 s“关”,5次/周,共6周	PCL-5、CAPS-5等	对难治性PTSD有效

PTSD,posttraumatic stress disorder,创伤后应激障碍;DLPFC,dorsolateral prefrontal cortex,背外侧前额皮质;PFC,prefrontal cortex,前额皮质;mPFC,medial prefrontal cortex,内侧前额皮质;MT,motor threshold,运动阈值;PCL,Posttraumatic Stress Disorder Checklist,创伤后应激障碍自评量表;CAPS,Clinician-Administered Posttraumatic Stress Disorder Scale,临床管理创伤后应激障碍量表;HAMD,Hamilton Depression Rating Scale,汉密尔顿抑郁量表;DSM-5,Diagnostic and Statistical Manual of Mental Disorders Fifth Edition,美国精神障碍诊断与统计手册第5版;rTMS,repetitive transcranial magnetic stimulation,重复经颅磁刺激;CPT,cognitive processing therapy,认知加工疗法

刺激脉冲的模式不同,经颅磁刺激主要分为重复经颅磁刺激(rTMS)、深部经颅磁刺激(dTMS)、单脉冲经颅磁刺激(sTMS)和成对脉冲经颅磁刺激(pTMS)。近年来,创伤后应激障碍成为经颅磁刺激治疗领域的研究热点(表2)[61-67]。

1.重复经颅磁刺激 rTMS是一段时间内以特定参数持续向大脑特定区域施加磁脉冲序列的过程[68],其疗效受多种参数的影响,包括刺激强度、频率、持续时间及线圈位置、类型[69]。θ短阵快速脉冲刺激(TBS)是一种特殊的rTMS[70],以30~50 Hz的三脉冲高频爆发为单位,并以5 Hz频率重复呈现,形成持续性θ短阵快速脉冲刺激(cTBS)或间歇性θ短阵快速脉冲刺激(iTBS)两种方式,抑制或增强皮质兴奋性[71]。rTMS已用于多种神经系统变性疾病和精神障碍疾病的治疗,在改善认知功能方面前景广阔。临床研究显示,对阿尔茨海默病患者左侧背外侧前额皮质予以rTMS,可以有效改善认知记忆功能[72];精神分裂症患者接受规范化rTMS后,注意力、工作记忆及执行功能等多个认知域显著改善,且6个月随访时仍具有稳定的认知获益[73],突显了rTMS的长期神经调控潜力。有研究以右侧背外侧前额皮质为刺激靶点行rTMS,可显著改善创伤后应激障碍核心症状如再体验症状等[61]。另有研究对创伤后应激障碍患者右侧前额皮质予以低频(1 Hz)rTMS,可显著减轻再体验症状,降低CAPS评分,且耐受性良好[62]。

2.深部经颅磁刺激 dTMS通过特殊设计线圈产生穿透力更强的磁场,短暂性磁脉冲用于诱导更深脑区的靶向神经元去极化。相较于rTMS的传统“8”字线圈(可刺激硬膜下皮质靶标,深度0.70 cm),dTMS的刺激深度达4 cm[74]。临床研究显示,短期创伤暴露后立即靶向内侧前额皮质予以dTMS,可显著减轻创伤后应激障碍患者侵入性症状[63]。然而,2021年的一项多中心随机对照试验采用相似的干预时序却未能证实其促进恐惧消退、减轻整体创伤后应激障碍症状的有效性[64],推测可能与刺激线圈类型有关[75]。现有证据表明,dTMS作为短期创伤暴露的联合手段均未在恐惧消退过程中表现出稳定且显著的增效作用,其在创伤后应激障碍的治疗中表现出显著的疗效异质性。

3.单脉冲和成对脉冲经颅磁刺激 sTMS和pTMS在神经科学研究和临床应用中各具特点。sTMS采用单脉冲刺激,主要用于基础神经电生理检测,对评估神经传导功能具有重要意义。在创伤后应激障碍治疗中,sTMS主要承担刺激靶点定位功能,为后续rTMS治疗提供精准定位依据。相比之下,pTMS采用成对脉冲连续刺激模式,通过在极短时程(通常间隔1~200 ms)施加两个不同强度磁脉冲,可以更深入探究皮质内抑制与易化机制。该项技术通过调节磁脉冲间隔产生不同神经生理效应。

4.重复经颅磁刺激与传统疗法的联合应用 相较于单纯rTMS,rTMS联合循证心理治疗在创伤后应激障碍中展现出潜在的协同增效作用,其初步疗效和安全性已得到多项研究的证实[65-67]。一项针对

美国退伍军人的研究显示，靶向左侧或右侧前额皮质的rTMS与长期暴露疗法相结合，具有良好的安全性和临床可行性[65]。另一项针对美国退伍军人的随机对照试验显示，相较于假刺激和认知加工疗法，接受靶向右侧前额皮质的rTMS联合认知加工疗法的受试者创伤后应激障碍症状显著减轻[66]。对于精神药物和心理治疗无效的难治性创伤后应激障碍患者，联合经颅磁刺激、认知加工疗法和精神药理学的综合治疗可使症状获得显著改善[67]。上述研究表明，rTMS与心理治疗联合应用可以实现个体化神经调控与行为干预的深度整合，为优化创伤后应激障碍的治疗策略提供了新的范式。其核心作用机制为，rTMS直接调节创伤后应激障碍相关特定脑网络的兴奋性和可塑性，进而启动或增强大脑对后续心理治疗的响应，使治疗诱导的神经可塑性变化更直接、有效[76]，这种神经-行为协同干预模式具有推广至多种精神疾病的潜力。尽管如此，目前研究仍存在一定局限性，多数临床试验样本量有限，长期随访数据不充分，尚无标准的rTMS刺激参数，尚待进一步探究。

四、经颅电刺激与经颅磁刺激的比较

尽管经颅电刺激和经颅磁刺激在创伤后应激障碍等精神障碍中均展现出治疗潜力，但二者在工程属性与临床适用性上存在本质区别。(1)刺激精度与空间特异性：两种技术各有侧重，经颅磁刺激依靠快速磁场变化在皮质局部诱发出聚焦电场，具有更高的空间分辨率(3～5 cm)，可实现对特定皮质功能区的相对精准调控；经颅电刺激施加的电流在穿过高电阻的颅骨过程中发生明显衰减与弥散分布，使其空间靶向精度受限[77]，更适用于对较大范围的皮质网络进行整体调节。(2)刺激深度：两种技术表现出不同的发展路径，传统经颅磁刺激的有效刺激范围多局限于硬膜下皮质，深度约为0.70 cm，对深部脑区的直接调控能力有限，dTMS通过改良线圈设计，增强对背外侧前额皮质等关键脑区的调控[74]；经颅电刺激诱发的电场主要集中于电极下方，靶向皮质表面[78]，但新兴技术如高密度经颅电刺激[79]、时间干涉刺激[80]，通过优化电极排布或采用场干涉策略显著提高对深部脑区进行靶向刺激的潜力。(3)设备成本和临床推广性：经颅电刺激设备轻便、成本较低，操作简便，可用于常规门诊乃至经规范培训的家庭环境[81]，具有良好的推广潜力；经颅磁刺激设备昂贵，且对操作人员的专业资质、头部线圈的精确定位及治疗室的物理空间均有特定要求，一定程度上限制其临床普及[82]。(4)安全性与耐受性：两种技术整体安全性均较高[83]，但亦有一定的不良反应，经颅电刺激过程中，患者可感受到轻微头皮刺痛和灼烧感[84]，整体耐受性较高；经颅磁刺激过程中也存在局部疼痛、头痛或不适感等轻微不良反应[85]，但高频或不当操作则存在诱发癫痫的风险，但发生率极低。(5)患者依从性：经颅电刺激因其刺激强度温和、体验舒适，更易被患者接受，适合日频次、长达数周(2～3周)的长期干预[57]；经颅磁刺激则需严格掌握禁忌证(如颅内动脉瘤、金属支架植入等)，且要求患者具有一定的配合度，对于伴明显情绪困扰或注意障碍的创伤后应激障碍患者可能构成挑战，影响疗效，通过治疗前充分沟通、心理教育及治疗中支持性互动，可有效提高治疗依从性[86]。总之，经颅电刺激以其低成本、高安全性，更适用于早期探索、大规模筛查或面向皮质网络层面的广域调控；经颅磁刺激则凭借其更强的空间聚焦能力，有望成为实现靶向治疗的工具。未来，两种技术通过与多模态建模、联合刺激策略及心理治疗耦合方式深度融合，有望为创伤后应激障碍的个体化神经调控提供更优的“工程-临床”一体化解决方案。

五、小结与展望

由于药物匮乏和心理治疗的局限，神经调控技术在神经精神疾病的治疗中日益受到关注，其中，无创电磁刺激作为非侵入性神经调控技术，因具有广泛的神经调控能力以及风险低、患者接受度高等优势，在改善创伤后应激障碍认知损害的临床试验中展现出良好的应用前景。但现有研究样本量有限、长期随访数据不充分、疗效存在异质性，最优刺激参数和刺激靶点等刺激策略尚待进一步明确。在神经调控直接调节神经活动的基础上联合心理疗法可将个体化神经调控与行为干预深度整合，为优化创伤后应激障碍认知损害的治疗提供新思路。未来研究除扩大样本量外，还可聚焦多脑区、跨通路的脑网络协同作用，并引入更多研究手段开展深入的临床研究。总之，非侵入性电磁神经调控技术对创伤后应激障碍认知损害的作用机制和临床疗效尚待进一步探究，随着基础实验验证和临床数据深度分析以及多技术集成和参数优化，其临床应用潜力必将进一步增大，有望成为改善创伤后应激障碍认知损害的有效方法。

利益冲突 无

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·小词典·

中英文对照名词词汇(三)

局灶知觉损害性发作
focal impaired awareness seizure(FIAS)

局灶知觉性发作 focal aware seizure(FAS)

抗癫痫发作药物 antiepileptic seizure medicine(ASM)

控制性皮质撞击 controlled cortical impact(CCI)

眶额皮质 orbitofrontal cortex(OFC)

扩散张量成像 diffusion tensor imaging(DTI)

扩散张量纤维束示踪成像
diffusion tensor tractography(DTT)

蓝斑 locus coeruleus(LC)

酪氨酸蛋白激酶B tyrosine protein kinase B(TrkB)

酪氨酸激酶结构域 tyrosine kinase domain(TKD)

立体定向脑电图 stereo-electroencephalography(SEEG)

临床管理创伤后应激障碍量表
Clinician-Administered Posttraumatic Stress Disorder Scale (CAPS)

颅脑创伤 traumatic brain injury(TBI)

卵泡刺激素 follicle stimulating hormone(FSH)

脉冲发生器 impulse generator(IPG)

慢皮质电位 slow cortical potential(SCP)

美国脊髓损伤协会
American Spinal Injury Association(ASIA)

美国精神障碍诊断与统计手册第5版
Diagnostic and Statistical Manual of Mental Disorders Fifth Edition(DSM-Ⅴ)

美国食品与药品管理局
Food and Drug Administration(FDA)

弥漫性软脑膜胶质神经元肿瘤
diffuse leptomeningeal glioneuronal tumor(DLGNT)

迷走神经刺激术 vagus nerve stimulation(VNS)

10米步行试验 10 Meter Walk Test(10MWT)

明尼苏达心力衰竭生活质量问卷
Minnesota Living with Heart Failure Questionnaire (MLHFQ)

难治性癫痫 refractory epilepsy(RE)

难治性精神分裂症 treatment-resistant schizophrenia(TRS)

难治性强迫症
treatment resistant-obsessive compulsive disorder(TR-OCD)

难治性心绞痛 refractory angina pectoris(RAP)

难治性抑郁症 treatment-resistant depression(TRD)

脑机接口 brain-computer interface(BCI)

脑默认网络 default mode network(DMN)

脑深部电刺激术 deep brain stimulation(DBS)

脑源性神经营养因子
brain-derived neurotrophic factor(BDNF)

内侧颞叶癫痫 mesial temporal lobe epilepsy(mTLE)

内侧前额皮质 medial prefrontal cortex(mPFC)

内侧前脑束 medial forebrain bundle(MFB)

内囊前肢 anterior limb of the internal capsule(ALIC)

颞叶癫痫 temporal lobe epilepsy(TLE)

纽约心脏协会 New York Heart Association(NYHA)

皮质脊髓束 corticospinal tract(CST)

皮质-纹状体-丘脑-皮质
cortico-striato-thalamo-cortical(CSTC)

匹兹堡睡眠质量指数 Pittsburgh Sleep Quality Index(PSQI)

胼胝体下回 subcallosal gyrus(SCG)

胼胝体下扣带回 subcallosal cingulate(SCC)

前边缘皮质 prelimbic cortex(PLC)

前额皮质 prefrontal cortex(PFC)

5-羟色胺 5-hydroxytryptamine(5-HT)

青少年肌阵挛性癫痫 juvenile myoclonic epilepsy(JME)

丘脑底核 subthalamic nucleus(STN)

丘脑底核后部 posterior subthalamic area(PSA)

丘脑腹中间核 ventral intermediate nucleus(Vim)

丘脑前核 anterior nucleus of the thalamus(ANT)

丘脑下脚 inferior thalamic peduncle(ITP)

丘脑中央中核-束旁核复合体
center median-parafascicular complex(CM-Pf)

全面性癫痫 general epilepsy(GE)

全面性强直-阵挛发作
generalized tonic-clonic seizure(GTCS)

全面性阵发性快速活动
generalized paroxysmal fast activity(GPFA)