小脑高场和超高场磁共振成像的研究进展 及在神经退行性疾病中的应用



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[摘要] 人类小脑虽然体积很小,但在运动控制、平衡维持以及认知功能等方面,却发挥着至关重要的作用。多种疾病,尤其 是神经退行性疾病(neurodegenerative diseases, NDs),在发展过程中会累及小脑,严重影响患者日常生活。因此,对NDs患者 小脑的研究,有助于我们理解其病理机制。目前,小脑高场磁共振成像(high field magnetic resonance imaging, HF-MRI)在 NDs中的应用,为小脑结构及功能的改变提供了大量影像学证据。更先进的小脑超高场磁共振成像(ultra high field magnetic resonance imaging, UHF-MRI)允许我们进一步研究小脑的细微结构及功能特点,具有广阔前景,但尚未广泛应用于 NDs 研究 中。本文综述了小脑 HF-MRI及 UHF-MRI 的研究进展及在 NDs 中的应用,分析了小脑 UHF-MRI 成像优势及挑战。未来有望在 小脑 UHF-MRI 赋能下,从小脑角度出发,寻找早期精确诊断 NDs 的神经影像生物标记物。 [关键词] 小脑;神经影像学;磁共振成像;超高场强;脑结构;脑功能;神经退行性疾病

【大键叫】 小脑; 种红影像子; 慨兴派风像; 起同功浊; 脑珀构; 脑功能; 种红赵11 性厌病

Progress in cerebellar high-field and ultra-high-field magnetic resonance imaging and their applications in neurodegenerative diseases

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Abstract Although the cerebellum of humans is quite small in volume, it plays a crucial role in motor control, balance maintenance, and cognitive functions. A variety of diseases, especially neurodegenerative diseases, involve the cerebellum during their progression, severely affecting the daily lives of patients. Therefore, studying the cerebellum in patients with neurodegenerative diseases (NDs) helps us understand their pathological mechanisms. Currently, the application of cerebellar high field magnetic resonance imaging (HF-MRI) in NDs has provided a wealth of imaging evidence for changes in cerebellar structure and function. Advanced ultra-high field magnetic resonance imaging (UHF-MRI) of the cerebellum allows us to further investigate the subtle structures and functional characteristics of the cerebellum, which holds broad prospects, but has not yet been widely applied in the study of NDs. This paper reviews the research progress of cerebellar HF-MRI and UHF-MRI, as well as their applications in NDs, and analyzes the advantages and challenges of cerebellar UHF-MRI. In the future, with the empowerment of cerebellar UHF-MRI, there is hope to identify neuroimaging biomarkers for the early and precise diagnosis of NDs from the cerebellar perspective.

Key words cerebellum; neuroimaging; magnetic resonance imaging; ultra-high-field; brain structure; brain function; neurodegenerative diseases

0 引言

小脑位于颅后窝,由两个小脑半球组成,中央通 过称为蚓部的部分连接,背靠脑干,位于枕叶下方。 尽管人类小脑体积小,但小脑皮层的表面相比大脑 皮层而言折叠得更加紧密,人类小脑表面积几乎是 大脑皮层表面积的80%^[1]。大脑皮质有160亿神经 元,小脑神经细胞数目约其5倍(690亿)^[2]。小脑皮 层整合了多种功能,参与了运动控制、语言及认知情 感处理,因此对我们生活至关重要^[3-4]。小脑深层核 团的功能分区与小脑皮层的运动功能密切相关,通 过与大脑皮层的交互作用,协调运动和认知功能地 实现^[5-7]。小脑可受到多种疾病的影响,如神经退行 性疾病(neurodegenerative diseases, NDs)、精神疾病 和遗传性疾病等^[8-10]。NDs是一类异质性疾病,且尚

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无治愈方法,而越来越多的临床和生物学证据表明小脑与NDs之间存在重要联系^[11]。因此,对这类疾病小脑的研究有助于我们理解其病理机制,制订个性化诊疗方案。磁共振成像(magnetic resonance imaging, MRI)具有非侵入性优势,主磁场强度为1.5 T或3.0 T下的小脑高场 MRI(high field MRI, HF-MRI)在 NDs中的应用^[12-13],为小脑结构及功能的改变提供了大量影像学证据。而主磁场强度大于3.0 T的小脑超高场 MRI(ultra high field MRI, UHF-MRI)允许我们进一步研究小脑的细微结构和功能特征^[14],具有广阔的前景,但尚未广泛应用于 NDs 研究中。本文总结了小脑 HF-MRI和 UHF-MRI 常用技术方法及在 NDs 中的应用,分析了小脑 UHF-MRI 的优势及挑战,以期未来在 UHF-MRI 的赋能下,从小脑的角度出发,寻找早期精确诊断 NDs 的神经影像生物标志物。

1 小脑MRI常用技术方法

1.1 基于体素的形态学测量

小脑体积的评估作为运动和认知表现的潜在指 标,以及NDs进展或治疗效果的可能生物标志,已成为 研究热点[15-16]。基于体素的形态学测量(voxel-based morphometry, VBM)是一种通常基于 3D T1WI 结构 像对脑体积进行测量的分析方法,能够准确量化小 脑不同区域的灰质和白质体积变化^[17]。VBM通常包 括空间标准化、脑分割、统计分析及叠加图生成等步 骤,其中脑分割是关键步骤。空间无偏移幕下模板 (spatially unbiased infratentorial template, SUIT)是小 脑分割最常用的工具。对比基于全脑分割的VBM, 基于 SUIT 的 VBM 在检测小脑体积变化方面具有更 高的敏感度,并可以较好地保留小脑小叶解剖细 节^[18]。然而,SUIT在严重小脑萎缩患者中对部分小 脑小叶的分割准确性可能会降低。ROMERO 等^[19]提 出了一种新的小脑分割方法,对比基于 SUIT 的小脑 分割,在准确性及耗时方面表现更好。近些年,人工 智能的发展促进了对 MRI 影像数据的处理及利用, 利用深度学习可以在VBM中保证数据处理准确度 的前提下显著提高速度^[20]。FABER等^[21]开发的深度 学习管道,展现出高精度的小脑亚区分割,并在后续 对脊髓小脑共济失调患者 VBM 分析中,捕捉到早期 小脑体积改变。同时,VBM在小脑结构 MRI运用中 存在一些挑战。小脑本身较小的体积增加了部分体 积效应的风险,导致原始数据分辨率不够。未来,随 着UHF-MRI的广泛应用,获得空间分辨率更高的图 像^[22],在更小的体素下进行VBM分析有望进一步提 高VBM对小脑结构MRI分析的准确度。

1.2 弥散张量成像

弥散张量成像(diffusion tensor imaging, DTI)技术可以探测活体组织内水分子弥散特征,通过特定算法实现无创性对脑白质纤维束成像,并且能在微

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观水平上测量和评估脑白质纤维束的方向、位置和 微结构的完整性[23]。MACHADO-RIVAS等[24]开发的 新算法实现了DTI对孕妇胎儿小脑上脚和小脑中脚 白质束的可靠成像,并允许对发育变化特征进行描 述。AL-ARAB等^[25]对脊髓共济失调2型患者小脑上 脚及小脑中脚弥散指标的研究,表明DTI是追踪脊 髓共济失调2型患者脑白质神经退行性变进展和评 估其严重程度的敏感工具。尽管传统DTI是重建小 脑通路最常用方法,但其在连接性研究中存在局限 性。在包含多种纤维方向的区域,DTI的各向异性指 标会降低,导致对这些区域数据的解释变得困难^[26]。 DTI技术为揭示小脑白质的微观结构及其功能提供 了新的视角,尤其在神经发育及NDs研究中发挥了 重要作用,但目前仍存在样本量小、技术优化不足等 问题。其他更具前景的技术,如神经轴突定向弥散 和密度成像(neurite drientation dispersion and density imaging, NODDI)相比DTI,展现出更敏感的识别多 发性硬化脑微观结构改变的能力[27],未来需要进一步 探究其对NDs小脑微观结构改变的实践性。

1.3 功能磁共振成像

功能磁共振成像(functional magnetic resonance imaging, fMRI) 是基于检测血氧水平依赖(blood oxygenation level dependent, BOLD)信号的变化。 fMRI可分为任务态 fMRI(task-state fMRI, ts-fMRI) 和静息态 fMRI(resting-state fMRI, rs-fMRI),常用的 分析指标包括功能连接(functional connectivity, FC)、 低频振幅和局部一致性等。与脑结构成像改变相 比,功能成像改变可以在早期被检测到,用作生物标 志物被认为更敏感^[28]。近年来,fMRI的研究极大推 动了对小脑功能的理解,小脑在运动协调、认知功能 和情绪调节中的作用已被逐步揭示。针对小脑的不 同功能区,任务下BOLD激活差异为理解小脑在运 动和认知任务中的多重角色提供了证据[29]。小脑皮 层的颗粒层和深部核团在执行复杂运动任务时表现 出显著激活,而不同认知任务的激活模式则反映了 小脑与大脑皮层网络间的功能整合^[30]。JUNG等^[31]通 过UHF-MRI结合皮层层级分析,发现BOLD响应在 微血管水平与皮层结构紧密关联,这为解析小脑特 定功能区域提供了更高的时空分辨率[31]。然而,小脑 fMRI研究也面临挑战,包括生理噪声、运动伪影的影 响及神经血管耦合的非线性特性^[32]。另外,BOLD信 号容易受到个体差异和年龄因素的影响[33],需要更 精细的分析方法来提高信号的准确性。fMRI常用的 数据驱动分析方法,如独立成分分析(independent component analysis, ICA),在去除生理噪声方面展现 出良好的效果^[32]。改进的独立向量分析相比ICA,对 评估孤独症谱系患者脑功能网络改变更佳[34],但耗时 更长。综上所述,fMRI技术在小脑功能研究中取得 了显著进展,未来研究仍需进一步优化fMRI数据处

理及分析,降低生理噪声、运动伪影带来的影响。

2 小脑HF-MRI的优势及挑战

HF-MRI 被广泛用于小脑成像研究,但其在解析 小脑复杂结构上仍存在对比度和分辨率不足的问题, 特别是对于小脑小叶的识别,这对研究小脑的精细结 构产生了限制。与常规HF-MRI相比,UHF-MRI具有 显著的成像优势(图1)。

在 UHF-MRI 中, 信噪比(signal-to-noise ratio, SNR) 随着磁场强度的增加而提高,使得图像空间分辨率 得到了显著提高[20],即在一个体素内达到更小尺寸。 随着设备硬件技术的进步,开发针对脑部成像的 UHF-MRI,进一步提高了脑部影像的空间分辨率^[35]。 MAROUES 等^[36]使用 7.0 T MRI 对 3 名健康被试者进 行小脑成像,观察到小脑皮层的颗粒层及分子层等微 小结构,表明近显微分辨率的体内小脑成像在7.0T MRI下是可行的。更高的空间分辨率使得研究者能 够更准确地观察到小脑皮层及其深层核团的解剖特 点^[36]。UHF-MRI的空间分辨率的提高,能够区分相 邻的神经结构^[37],这在HF-MRI中往往是难以实现 的。AGOSTINELLI等^[38]绘制的小脑7.0TMRI影像 与组织学匹配的图谱,有助于研究者及临床工作者精 准识别与疾病相关的细胞群。这种对许多神经和精神 疾病影响的解剖区域的详细观察,可能为患有这种病 理状况的患者的诊断和随访提供新的影像标志物[38], 如7.0 TMRI提高了对多发性硬化患者中小脑结构的 勾画及病变特征的显示^[39-40]。PRIOVOULOS等^[41]证 明,使用运动校正的7.0TMRI,在临床上可接受的采 集时间内,对小脑皮质层和小脑皮层进行体内成像 以及定量测量是可行的。此外,相较于HF-MRI, UHF-MRI磁敏感效应增强,从而有利于提高磁敏感 加权成像(susceptibility weighted imaging, SWI)及 fMRI对比度^[20, 42]。BOILLAT等^[43]通过7.0T下fMRI 技术,成功地揭示了小脑中高度有序的全身映射图 谱,这一发现加深了对小脑功能解剖学的理解,为未 来研究提供了重要参考,尤其是在感觉运动功能及 疾病相关领域。

UHF-MRI虽然提供了更高的空间分辨率和信号 灵敏度,但在成像上依然存在一些技术挑战:射频场 (B1+)在小脑区域产生的介电效应减弱了脉冲信 号[44],这种负面影响在超高场环境下更明显。一项研 究结果显示,小脑专用射频-接收线圈提供的信噪比 增益可以改善小脑成像质量,但代价是视野减小[49]。 患者的舒适度也是一个需要考虑的因素,主磁场场 强升高导致射频特定吸收率增加,可能导致患者局 部体温升高,存在一定安全风险^[46]。UHF-MRI设备 通常需要更长的成像时间,不自主运动增加者,可能 不能配合全程扫描,从而影响图像的质量和可靠 性[47]。在高场强环境下,成像对运动更敏感,更容易 出现运动伪影[47]。成像时间的延长及也可能限制了 临床应用的普及。

综上,UHF-MRI在高场强下获得的SNR 增益通 常用于提高时间空间分辨率,对研究小脑有巨大潜 力。然而,一旦达到一定亚毫米级分辨率,成像运动 伪影更加敏感,单纯减小体素尺寸可能不会如预期 那样提高图像质量。介电效应也制约了 UHF-MRI 下小脑的研究及应用,虽然已有研究证明高介电常 数材料可以提高小脑f-MRI成像质量[48],但其在常规 序列的实践能力还需要进一步验证。未来,进一步 发展小脑专用射频-接收线圈及成像序列,有望释放 小脑UHF-MRI的潜力。

3 小脑MRI在神经退行性疾病中的应用

3.1 阿尔茨海默病

阿尔茨海默病(Alzheimer's disease, AD)是痴呆 症最常见的形式,它会逐渐降低认知和社会情感功 能。既往对于AD的研究主要集中于大脑和海马,但 最近的神经病理学和神经影像学研究进展强调了小 脑在AD中的重要作用[49]。CHEN等[12]利用VBM分 析,发现小脑体积存在萎缩,且临床痴呆评分



比。1A~1C:31岁健康女性3.0TMRI 小脑成像图。扫描序列为T1磁化准备 快速梯度回波成像(T1-mprage),参数: TR 1900 ms, TE 2.5 ms, 层厚 1.0 mm; 1D~1F:34岁健康女性7.0TMRI小脑 成像图。扫描序列为T1-mprage,参数: TR 5000 ms, TE 2.0 ms, 层厚 0.6 mm。 Fig. 1 Comparsion of 3.0 T and 7.0 T MRI for cerebellum. 1A-1C: Image of 3.0 T MRI for cerebellum in a 31-year-old healthy female. Scanning sequence is T1 magnetization-prepared rapid acquisition gradient echo (T1-mprage). Parameter: TR 1900 ms, TE 2.5 ms, thickness 1.0 mm; 1D-1F: Image of 7.0 T MRI for cerebellum in a 34-year-old healthy female. Scanning sequence is T1-mprage. Parameter: TR 5000 ms, TE 2.0 ms, thickness 0.6 mm.

(clinical dementia rating, CDR)量表得分不同 AD 患 者中小脑的萎缩程度不同。在CDR 量表得分为0~ 0.5的AD患者中,小脑Crus I和右侧小叶VI较对照 组显著萎缩;而在CDR量表得分为0.5-1的AD患者 中,小脑多个区域包括双侧小叶 I~VI、IX、Crus I、 左侧小叶Ⅲa、Ⅲb和蚓部较对照组显著萎缩^[12]。 CHEN 等^[50]使用机器学习的方法构建了整合小脑放 射组学和结构连接模型,小脑模型在区分轻度认知 障碍(mild cognitive impairment, MCI)和认知正常 (normal cognition, NC)及预测 NC 到 MCI 的转变方 面优于海马模型,其中关键的预测因素包括右侧小 叶Ⅲ、左侧小叶Ⅰ和Ⅱ的纹理特征以及蚓部Ⅰ和Ⅱ 的网络特征,这些特征与AD的认知能力下降有关。 以上说明,AD患者中小脑结构改变存在一定分布特 点,并随着病症进展加重,提取这些特征有助于早期 识别和预测AD临床前阶段的进展情况。

3.2 帕金森病

帕金森病(Parkinson's disease, PD)的特征是异 质性运动和非运动症状,由涉及中枢神经系统各个 部分的神经退行性变引起,预计全球发病率估计将 从2015年的约700万翻倍,到2040年将增加到约 1300万^[51]。近期的神经病理学和神经影像学研究进 展表明小脑在PD中的扮演了重要角色[13]。此前的研 究显示,PD患者中小脑双侧小叶 I~IV、VI、Crus I、 Crus II、VIIb、VIIa、VIIb、右侧小叶 V 和蚓部存在萎缩, 并且萎缩模式对小脑内和小脑-皮层之间的FC产生 了影响^[52]。近期,KERESTES等^[53]使用VBM分析发 现,与对照组相比,临床分期为早期的PD患者双侧 小脑前叶中的小叶V较大,而临床分期为中晚期的 PD患者双侧小脑后叶中的小叶II体积较小,并且随 着疾病分期的升高,小叶WI体积逐渐降低,提示PD 患者病程中小脑运动区和非运动区的萎缩存在分 离。另外,LIU等⁶⁴利用fMRI发现PD患者小脑功能不 对称,提示PD的进展具有不对称性。此外,SHEN等^[5] 利用 fMRI 发现震颤为主型 PD (tremor-dominant PD, TDPD) 患者感觉运动网络(sensorimotor network, SMN)内部的动态功能网络连接(dynamic functional network connectivity, DFNC)减低, 而小脑-SMN的 DFNC 增加,提示了一动态补偿的机制。综上,PD 患 者中小脑的萎缩随着病程进展而加重,并且小脑不 同区域的萎缩存在分离的现象,与小脑功能分区有 关,此外还伴随着功能的不对称改变。

3.3 脊髓侧索硬化

脊髓侧索硬化 (amyotrophic lateral sclerosis, ALS)是一种累及小脑的进行性神经元变性疾病^[56]。 ALS病理改变与某些基因的重复扩增有关,如9号染 色体开放阅读框 72 (chromosome 9 open reading frame 72, C9ORF72)和共济失调蛋白-1 (ataxin-1, ATXN1)基因^[57]。一项关于 ALS 患者基因型相关小

脑特征的研究发现,散发性ALS患者的小脑萎缩局 限于小脑小叶 I~V, C9ORF72 ALS 患者的小脑蚓 部、小叶I~IV、V、WA/B、IX存在广泛萎缩,而 ATXN2 ALS 患者则没有表现出明显的小脑萎缩^[16]。 此外,BEDE等^[16]在C9ORF72 ALS患者小脑DTI中 观察到小脑小叶I~IV、V的各向异性分数降低、轴 向弥散率和径向弥散率增加,而在散发性ALS中,观 察到小脑小叶 I~IV、V、IX和 Crus I、Crus II的各向 异性分数降低以及小叶 I~IV、V和VI的径向扩散率 增加。BARRY等^[58]在基于UHF-MRI的fMRI研究中 发现,感觉运动皮质和双侧小脑小叶VI之间的FC出 现中断。此外,另一项研究发现,散发性和遗传性 ALS 患者均出现大脑-小脑 FC 降低及中断。而 ABIDI 等^[59]利用 fMRI 发现 ALS 患者纹状体-小脑及 顶叶-小脑FC增加。以上说明,ALS患者中小脑结构 及功能出现进行性改变,且在不同类型患者中出现

3.4 多发性硬化

异质性结果。

多发性硬化(multiple sclerosis, MS)是最常见的 累及中枢神经系统的慢性炎性脱髓鞘疾病100,小脑参 与了疾病的早期阶段^[61]。GALBUSERA等^[62]基于小 脑 7.0 T MRI,发现 MS 患者小脑皮层在早期已出现 了微观结构改变。MEIJBOOM 等^[63]利用 VBM 分析 发现,小脑体积随着病程进展减少。一项对MS患者 小脑特征的研究显示,小脑小叶 I~IV、下蚓部灰质体 积减小、小脑上脚病损体积增加以及小脑小叶WIb、 Crus II 中灰质体积减小与身体残疾和认知功能障碍 相关^[64]。RUGGIERI等^[65]发现MS患者小脑所有小叶 的体积较对照组显著减小,并与DTI 定量指标各向 异性分数呈正相关,提示感觉运动小脑萎缩以及小 脑传入和传出连接的破坏如何导致 MS 患者的身体 残疾。SCHOONHEIM 等^[66]发现与复发缓解型 MS 患 者相比,继发进展型MS患者表现出更严重的认知障 碍和小脑损伤,并且继发进展型MS患者认知障碍与 小脑FC减弱之间有关。另外一项研究表明,MS患 者小脑 FC 减低、小脑体积减小与临床表现加重之间 存在关联^[67]。总之,小脑参与了MS病程发展,并与 临床表现密切相关。

3.5 亨廷顿病

亨廷顿病(Huntington's disease, HD)是一种显性 遗传性NDs,临床特征主要为舞蹈样动作、认知障碍 和精神行为异常三联征^[68],其全球发病率和患病率分 别约为每10万人年0.38例和每10万人2.71例^[69]。一 项VBM分析显示,HD患者小脑体积与统一亨廷 顿病评定量表(Unified Huntington's Disease Rating Scale, UHDRS)存在显著负相关^[70]。另外一项研究表 明,HD患者小脑灰质体积减小与生活质量降低显著 相关^[71]。PADRON-RIVERA等^[72]发现HD患者小脑 右侧Crus I、双侧Crus II以及左侧小叶VIIb和VIIIa出 现显著变性。而另外一项研究显示,与健康对照组相比,平均发病年龄在10~20岁之间的青少年型HD (juvenile HD, JHD)患者的小脑体积增大,这可能是由于代偿机制^[73]。一项病例报告,展示了一名JHD 患者小脑体积在3年的随访中未见显著减小,而其他 区域脑体积出现减小伴随临床症状加重,提示小脑 的代偿机制^[74]。此外,基于HD患者rs-fMRI的荟萃 分析显示小脑右侧小叶和VI和Crus I脑功能活动增加,印证了小脑代偿机制的假说^[75]。综上,HD患者的 小脑功能及结构改变出现异质性结果,可能是因为 不同类型HD对小脑影响程度不同以及早期病程中 的小脑代偿功能在进展过程中被耗竭。此外小脑功 能的增加或者降低都会对临床表现产生影响。

综上所述,小脑参与了多种 NDs 病理机制及病 程发展,对不同 NDs 及不同病程阶段的影响具有重 叠及差异性,并且与临床表现密切相关。因此,对小 脑的结构及功能的研究有助于 NDs 诊疗。然而, NDs 患者小脑结构与功能 MRI 的改变有待被精确量 化,这些改变与临床及神经病理改变之间相关性及 因果关系仍需进一步探究。同时,以上大多研究是 基于小脑 HF-MRI 开展,更具前景的小脑 UHF-MRI 在 NDs 中尚未广泛应用。未来,在小脑 UHF-MRI 下 对 NDs 进行大样本、多中心的研究,有望精确量化这 类疾病小脑的改变,帮助对其早期精确地诊断。

4 总结及展望

总之,小脑与其他脑区关联密切,对运动控制和 认知情绪至关重要。越来越多的神经病理和神经影 像学证据表明,小脑在NDs中扮演着重要角色。目 前为止,MRI在脑的研究中已经得到了广泛的应用, 但人们更关注于大脑成像研究,对小脑成像研究相 对较少。此外,小脑成像研究主要是基于HF-MRI开 展,而UHF-MRI在小脑成像研究中的潜力未被充分 释放。人们对NDs中小脑结构与功能改变的认知仍 然有限。未来,随着技术的不断创新发展,小脑 UHF-MRI有望进一步得到优化及广泛应用。数据分 析方法的创新也将驱动小脑影像研究的发展,如深 度学习技术可以帮助精确识别和处理复杂的成像数 据。基于MRI的小脑成像的未来趋势将朝着更高场 强、更高精度、更深入的研究的方向发展。研究应重 点关注多模态影像学结合人工智能研究、纵向队列 研究、新技术应用和临床应用,以更好地理解小脑在 NDs中的作用,寻找早期精确诊断的神经影像生物 标志物,制订个性化诊疗策略。

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作者贡献声明:刘晨构思并设计了本文的方向 和框架,并对稿件重要内容进行了修改,获得重庆市 中青年医学高端人才工作室项目、重庆市中青年医 学高端人才项目以及国家自然科学基金项目的资 助;胡瑜伟查阅文献并初步构思本综述的内容,起 草、撰写并修改本稿件;杨先菲、李从伟、欧沛灵、石 琳锋负责查找小脑磁共振成像相关文献,并对稿件 重要内容进行了修改;全体作者都同意最后的修改 稿发表,同意对本综述的所有方面负责,确保本综述 的准确性和诚信。

参考文献[References]

- SERENO M I, DIEDRICHSEN J, TACHROUNT M, et al. The human cerebellum has almost 80% of the surface area of the neocortex[J]. Proc Natl Acad Sci U S A, 2020, 117(32): 19538-19543. DOI: 10.1073/ pnas.2002896117.
- [2] BEURIAT P A, CRISTOFORI I, GORDON B, et al. The shifting role of the cerebellum in executive, emotional and social processing across the lifespan[J/OL]. Behav Brain Funct, 2022, 18(1): 6 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/35484543/. DOI: 10.1186/s12993-022-00193-5.
- [3] RUDOLPH S, BADURA A, LUTZU S, et al. Cognitive-affective functions of the cerebellum[J]. J Neurosci, 2023, 43(45): 7554-7564. DOI: 10.1523/JNEUROSCI.1451-23.2023.
- [4] CAREY M R. The cerebellum[J/OL]. Curr Biol, 2024, 34(1): R7-R11
 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/38194930/. DOI: 10.1016/
 j.cub.2023.11.048.
- [5] FENG S X, HUANG Y Y, LI H H, et al. Dynamic effective connectivity in the cerebellar dorsal dentate nucleus and the cerebrum, cognitive impairment, and clinical correlates in patients with schizophrenia[J/OL]. Schizophr Res, 2024, 271: 394-401 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/38729789/. DOI: 10.1016/j.schres.2024. 05.003.
- [6] CAJIGAS I, MORRISON M A, LUCIANO M S, et al. Cerebellar deep brain stimulation for the treatment of movement disorders in cerebral palsy[J]. J Neurosurg, 2023, 139(3): 605-614. DOI: 10.3171/2023.1. JNS222289.
- [7] NIE L L, JIANG Y C, LV Z X, et al. Deep cerebellar nuclei functional connectivity with cerebral cortex in temporal lobe epilepsy with and without focal to bilateral tonic-clonic seizures: a resting-state fMRI study[J]. Cerebellum, 2022, 21(2): 253-263. DOI: 10.1007/s12311-021-01266-3.
- [8] YANG Y P, LI J L, LI T, et al. Cerebellar connectome alterations and associated genetic signatures in multiple sclerosis and neuromyelitis optica spectrum disorder[J/OL]. J Transl Med, 2023, 21(1): 352 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/37245044/. DOI: 10.1186/ s12967-023-04164-w.
- [9] KLAUS J, STOODLEY C J, SCHUTTER D J L G. Neurodevelopmental trajectories of cerebellar grey matter associated with verbal abilities in males with autism spectrum disorder[J/OL]. Dev Cogn Neurosci, 2024, 67: 101379 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/38615557/. DOI: 10.1016/j.dcn.2024.101379.
- [10] MITIAGIN Y, BARZILAI A. Ataxia-telangiectasia mutated plays an important role in cerebellar integrity and functionality[J]. Neural Regen Res, 2023, 18(3): 497-502. DOI: 10.4103/1673-5374.350194.
- [11] LIU G, YANG C, WANG X, et al. Cerebellum in neurodegenerative diseases: advances, challenges, and prospects[J/OL]. iScience, 2024, 27(11): 111194 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/39555407/. DOI: 10.1016/j.isci.2024.111194.
- [12] CHEN Y, SPINA S, CALLAHAN P, et al. Pathology-specific patterns of cerebellar atrophy in neurodegenerative disorders[J]. Alzheimers Dement, 2024, 20(3): 1771-1783. DOI: 10.1002/alz.13551.
- [13] LI T B, LE W D, JANKOVIC J. Linking the cerebellum to Parkinson disease: an update[J]. Nat Rev Neurol, 2023, 19(11): 645-654. DOI: 10.1038/s41582-023-00874-3.
- [14] MORMINA E, PETRACCA M, BOMMARITO G, et al. Cerebellum and neurodegenerative diseases: beyond conventional magnetic resonance imaging[J]. World J Radiol, 2017, 9(10): 371-388. DOI: 10.4329/wjr.v9.i10.371.
- [15] LONG J Y, QIN K, WU Y, et al. Gray matter abnormalities and associated familial risk endophenotype in individuals with first-episode bipolar disorder: evidence from whole-brain voxel-wise meta-analysis[J/OL]. Asian J Psychiatr, 2022, 74: 103179 [2024-09-23]. https://pubmed.ncbi.nlm. nih.gov/35691059/. DOI: 10.1016/j.ajp.2022.103179.
- [16] BEDE P, CHIPIKA R H, CHRISTIDI F, et al. Genotype-associated cerebellar profiles in ALS: focal cerebellar pathology and cerebro-cerebellar connectivity alterations[J]. J Neurol Neurosurg Psychiatry, 2021, 92(11): 1197-1205. DOI: 10.1136/jnnp-2021-326854.
- [17] SI S Q, BI A, YU Z Y, et al. Mapping gray and white matter volume abnormalities in early-onset psychosis: an ENIGMA multicenter voxel-based morphometry study[J]. Mol Psychiatry, 2024, 29(2): 496-504. DOI: 10.1038/s41380-023-02343-1.
- [18] DIEDRICHSEN J. A spatially unbiased atlas template of the human cerebellum[J]. Neuroimage, 2006, 33(1): 127-138. DOI: 10.1016/j.

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neuroimage.2006.05.056.

- [19] ROMERO J E, COUPÉ P, GIRAUD R, et al. CERES: a new cerebellum lobule segmentation method[J/OL]. Neuroimage, 2017, 147: 916-924 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/27833012/. DOI: 10.1016/j.neuroimage.2016.11.003.
- [20] FISCH L, WINTER N R, GOLTERMANN J, et al. Deepmriprep: voxel-based morphometry (VBM) preprocessing via deep neural networks[EB/OL]. 2024: arXiv: 2408.10656. http://arXiv.org/abs/2408.10656.
- [21] FABER J, KÜGLER D, BAHRAMI E, et al. ČerebNet: a fast and reliable deep-learning pipeline for detailed cerebellum sub-segmentation[J/OL]. Neuroimage, 2022, 264: 119703 [2024-09-23]. https://pubmed.ncbi.nlm. nih.gov/36349595/. DOI: 10.1016/j.neuroimage.2022.119703.
- [22] BURKETT B J, FAGAN A J, FELMLEE J P, et al. Clinical 7-T MRI for neuroradiology: strengths, weaknesses, and ongoing challenges[J]. Neuroradiology, 2021, 63(2): 167-177. DOI: 10.1007/s00234-020-02629-z.
- [23] TOURNIER J D. Diffusion MRI in the brain Theory and concepts[J/OL]. Prog Nucl Magn Reson Spectrosc, 2019, 112/113: 1-16 [2024-09-23]. https: //pubmed.ncbi.nlm.nih.gov/31481155/. DOI: 10.1016/j.pnmrs.2019.03.001.
- [24] MACHADO-RIVAS F, AFACAN O, KHAN S, et al. Tractography of the cerebellar peduncles in second- and third-trimester fetuses[J]. AJNR Am J Neuroradiol, 2021, 42(1): 194-200. DOI: 10.3174/ajnr.A6869.
- [25] AL-ARAB N, HANNOUN S. White matter integrity assessment in spinocerebellar ataxia type 2 (SCA2) patients[J]. Clin Radiol, 2024, 79(1): 67-72. DOI: 10.1016/j.crad.2023.10.020.
- [26] WIEGELL M R, LARSSON H B, WEDEEN V J. Fiber crossing in human brain depicted with diffusion tensor MR imaging[J]. Radiology, 2000, 217(3): 897-903. DOI: 10.1148/radiology.217.3.r00nv43897.
- [27] SEYEDMIRZAEI H, NABIZADEH F, AARABI M H, et al. Neurite orientation dispersion and density imaging in multiple sclerosis: a systematic review[J]. J Magn Reson Imaging, 2023, 58(4): 1011-1029. DOI: 10.1002/jmri.28727.
- [28] PINI L, JACQUEMOT C, CAGNIN A, et al. Aberrant brain network connectivity in presymptomatic and manifest Huntington's disease: a systematic review[J]. Hum Brain Mapp, 2020, 41(1): 256-269. DOI: 10.1002/hbm.24790.
- [29] BATSIKADZE G, DIEKMANN N, ERNST T M, et al. The cerebellum contributes to context-effects during fear extinction learning: a 7T fMRI study[J]. Neuroimage, 2022, 253: 119080. DOI: 10.1016/j.neuroimage. 2022.119080.
- [30] THÜRLING M, HAUTZEL H, KÜPER M, et al. Involvement of the cerebellar cortex and nuclei in verbal and visuospatial working memory: a 7 T fMRI study[J]. Neuroimage, 2012, 62(3): 1537-1550. DOI: 10.1016/j.neuroimage.2012.05.037.
- [31] JUNG W B, IM G H, JIANG H Y, et al. Early fMRI responses to somatosensory and optogenetic stimulation reflect neural information flow[J/OL]. Proc Natl Acad Sci U S A, 2021, 118(11): e2023265118 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/33836602/. DOI: 10.1073/ pnas.2023265118.
- [32] CABALLERO-GAUDES C, REYNOLDS R C. Methods for cleaning the BOLD fMRI signal[J/OL]. NeuroImage, 2017, 154: 128-149 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/27956209/. DOI: 10.1016/ j.neuroimage.2016.12.018.
- [33] D'ESPOSITO M, DEOUELL L Y, GAZZALEY A. Alterations in the BOLD fMRI signal with ageing and disease: a challenge for neuroimaging[J]. Nat Rev Neurosci, 2003, 4(11): 863-872. DOI: 10.1038/nm1246.
- [34] JING J L, KLUGAH-BROWN B, XIA S Y, et al. Comparative analysis of group information-guided independent component analysis and independent vector analysis for assessing brain functional network characteristics in autism spectrum disorder[J/OL]. Front Neurosci, 2023, 17: 1252732 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/37928736/. DOI: 10.3389/fnins.2023.1252732.
- [35] FEINBERG D A, BECKETT A J S, VU A T, et al. Next-generation MRI scanner designed for ultra-high-resolution human brain imaging at 7 Tesla[J]. Nat Methods, 2023, 20(12): 2048-2057. DOI: 10.1038/ s41592-023-02068-7.
- [36] MARQUES J P, VAN DER ZWAAG W, GRANZIERA C, et al. Cerebellar cortical layers: in vivo visualization with structural high-field-strength MR imaging[J]. Radiology, 2010, 254(3): 942-948. DOI: 10.1148/radiol.09091136.
- [37] VACHHA B, HUANG S Y. MRI with ultrahigh field strength and high-performance gradients: challenges and opportunities for clinical neuroimaging at 7 T and beyond[J/OL]. Eur Radiol Exp, 2021, 5(1): 35 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/34435246/. DOI: 10.1186/ s41747-021-00216-2.
- [38] AGOSTINELLI L J, SEAMAN S C, SAPER C B, et al. Human brainstem and cerebellum atlas: chemoarchitecture and cytoarchitecture paired to MRI[J]. J Neurosci, 2023, 43(2): 221-239. DOI: 10.1523/ JNEUROSCI.0587-22.2022.
- [39] LOUAPRE C, TREABA C A, BARLETTA V, et al. Ultra-high field 7 T imaging in multiple sclerosis[J]. Curr Opin Neurol, 2020, 33(4): 422-429. DOI: 10.1097/WCO.0000000000839.
- [40] FARTARIA M J, O'BRIEN K, ŞOREGA A, et al. An ultra-high field study of cerebellar pathology in early relapsing-remitting multiple sclerosis using MP2RAGE[J]. Invest Radiol, 2017, 52(5): 265-273. DOI: 10.1097/RLI.0000000000338.

- [41] PRIOVOULOS N, ANDERSEN M, DUMOULIN S O, et al. High-resolution motion-corrected 7.0-T MRI to derive morphologic measures from the human cerebellum in vivo[J/OL]. Radiology, 2023, 307(2): e220989 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/36648348/. DOI: 10.1148/radiol.220989.
- [42] BAE Y J, KIM J M, SOHN C H, et al. Imaging the substantia nigra in parkinson disease and other parkinsonian syndromes[J]. Radiology, 2021, 300(2): 260-278. DOI: 10.1148/radiol.2021203341.
- [43] BOILLAT Y, BAZIN P L, VAN DER ZWAAG W. Whole-body somatotopic maps in the cerebellum revealed with 7T fMRI[J/OL]. Neuroimage, 2020, 211: 116624 [2024-09-23]. https://pubmed.ncbi. nlm.nih.gov/32058002/. DOI: 10.1016/j.neuroimage.2020.116624.
- [44] SCHICK F. Whole-body MRI at high field: technical limits and clinical potential[J]. Eur Radiol, 2005, 15(5): 946-959. DOI: 10.1007/s00330-005-2678-0.
- [45] PRIOVOULOS N, ROOS T, IPEK Ö, et al. A local multi-transmit coil combined with a high-density receive array for cerebellar fMRI at 7 T[J/OL]. NMR Biomed, 2021, 34(11): e4586 [2024-09-23]. https://pubmed.ncbi. nlm.nih.gov/34231292/. DOI: 10.1002/nbm.4586.
- [46] WINTER L, SEIFERT F, ZILBERTI L, et al. MRI-related heating of implants and devices: a review[J]. J Magn Reson Imaging, 2021, 53(6): 1646-1665. DOI: 10.1002/jmri.27194.
- [47] PLATT T, LADD M E, PAECH D. 7 tesla and beyond: advanced methods and clinical applications in magnetic resonance imaging[J]. Invest Radiol, 2021, 56(11): 705-725. DOI: 10.1097/RLI.00000000000820.
- [48] VAIDYA M V, LAZAR M, DENIZ C M, et al. Improved detection of fMRI activation in the cerebellum at 7T with dielectric pads extending the imaging region of a commercial head coil[J]. J Magn Reson Imaging, 2018, 48(2): 431-440. DOI: 10.1002/jmri.25936.
- [49] YANG C, LIU G D, CHEN X, et al. Cerebellum in Alzheimer's disease and other neurodegenerative diseases: an emerging research frontier[J/OL]. MedComm, 2024, 5(7): e638 [2024-09-23]. https://pubmed.ncbi.nlm. nih.gov/39006764/. DOI: 10.1002/mco2.638.
- [50] CHEN Y N, QI Y W, HU Y Y, et al. Integrated cerebellar radiomic-network model for predicting mild cognitive impairment in Alzheimer's disease[J/OL]. Alzheimers Dement, 2024 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/39535490/. DOI: 10.1002/alz.14361.
- [51] JANKOVIC J, TAN E K. Parkinson's disease: etiopathogenesis and treatment[J]. J Neurol Neurosurg Psychiatry, 2020, 91(8): 795-808. DOI: 10.1136/jnnp-2019-322338.
- [52] O'CALLAGHAN C, HORNBERGER M, BALSTERS J H, et al. Cerebellar atrophy in Parkinson's disease and its implication for network connectivity[J]. Brain, 2016, 139(Pt 3): 845-855. DOI: 10.1093/brain/ awv399.
- [53] KERESTES R, LAANSMA M A, OWENS-WALTON C, et al. Cerebellar volume and disease staging in Parkinson's disease: an ENIGMA-PD study[J]. Mov Disord, 2023, 38(12): 2269-2281. DOI: 10.1002/mds.29611.
- [54] LIU Y J, YUAN J Y, TAN C L, et al. Exploring brain asymmetry in early-stage Parkinson's disease through functional and structural MRI[J/OL]. CNS Neurosci Ther, 2024, 30(7): e14874 [2024-09-23]. https://pubmed. ncbi.nlm.nih.gov/39056398/. DOI: 10.1111/cns.14874.
- [55] SHEN B, YAO Q, LI W, et al. Dynamic cerebellar and sensorimotor network compensation in tremor-dominated Parkinson's disease[J/OL]. Neurobiol Dis, 2024, 201: 106659 [2024-09-23]. https://pubmed.ncbi. nlm.nih.gov/39243826/. DOI: 10.1016/j.nbd.2024.106659.
- [56] CHIPIKA R H, MULKERRIN G, PRADAT P F, et al. Cerebellar pathology in motor neuron disease: neuroplasticity and neurodegeneration[J]. Neural Regen Res, 2022, 17(11): 2335-2341. DOI: 10.4103/1673-5374.336139.
- [57] RENTON A E, CHIÒ A, TRAYNOR B J. State of play in amyotrophic lateral sclerosis genetics[J]. Nat Neurosci, 2014, 17(1): 17-23. DOI: 10.1038/nn.3584.
- [58] BARRY R L, BABU, ANTERAPER S A, et al. Ultra-high field (7T) functional magnetic resonance imaging in amyotrophic lateral sclerosis: a pilot study[J/OL]. Neuroimage Clin, 2021, 30: 102648 [2024-09-23]. https:// pubmed.ncbi.nlm.nih.gov/33872993/. DOI: 10.1016/j.nicl.2021.102648.
- [59] ABIDI M, MARCO G D, GRAMI F, et al. Neural correlates of motor imagery of gait in amyotrophic lateral sclerosis[J]. J Magn Reson Imaging, 2021, 53(1): 223-233. DOI: 10.1002/jmri.27335.
- [60] CHATAWAY J, WILLIAMS T, LI V, et al. Clinical trials for progressive multiple sclerosis: progress, new lessons learned, and remaining challenges[J]. Lancet Neurol, 2024, 23(3): 277-301. DOI: 10.1016/S1474-4422(24)00027-9.
- [61] MAXWELL D L, ORIAN J M. Cerebellar pathology in multiple sclerosis and experimental autoimmune encephalomyelitis: current status and future directions[J/OL]. J Cent Nerv Syst Dis, 2023, 15: 11795735231211508 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/ 37942276/. DOI: 10.1177/11795735231211508.
- [62] GALBUSERA R, PARMAR K, BOILLAT Y, et al. Laminar analysis of the cerebellar cortex shows widespread damage in early MS patients: a pilot study at 7T MRI[J/OL]. Mult Scler J Exp Transl Clin, 2020, 6(4): 2055217320961409 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/ 33149930/. DOI: 10.1177/2055217320961409.

(下转第211页)

detecting and quantitation of enlarged perivascular spaces on MRI[J]. J Magn Reson Imaging, 2022, 57(1): 11-24. DOI: 10.1002/jmri.28369.

- [32] CAI K, TAIN R, DAS S, et al. The feasibility of quantitative MRI of perivascular spaces at 7 T[J]. J Neurosci Methods, 2015, 256: 151-156. DOI: 10.1016/j.jneumeth.2015.09.001.
- [33] DEIKE K, DECKER A, SCHEYHING P, et al. Machine learning based perivascular space volumetry in Alzheimer disease[J]. Invest Radiol, 2024, 59(9): 667-676. DOI: 10.1097/rli.000000000001077.
 [34] MORTAZAVI M M, GRIESSENAUER C J, ADEEB N, et al. The
- [34] MORTAZAVI M M, GRIESSENAUER C J, ADEEB N, et al. The choroid plexus: a comprehensive review of its history, anatomy, function, histology, embryology, and surgical considerations[J]. Childs Nerv Syst, 2014, 30(2): 205-214. DOI: 10.1007/s00381-013-2326-y.
- [35] SUN A, WANG J. Choroid plexus and drug removal mechanisms[J/OL]. AAPS J, 2021, 23(3): 61 [2024-08-09]. https://pubmed.ncbi.nlm.nih. gov/33942198/. DOI: 10.1208/s12248-021-00587-9.
- [36] EIDE P K, VALNES L M, PRIPP A H, et al. Delayed clearance of cerebrospinal fluid tracer from choroid plexus in idiopathic normal pressure hydrocephalus[J]. J Cereb Blood Flow Metab, 2020, 40(9): 1849-1858. DOI: 10.1177/0271678x19874790.
- [37] CHOI J D, MOON Y, KIM H J, et al. Choroid plexus volume and permeability at brain MRI within the Alzheimer disease clinical spectrum[J]. Radiology, 2022, 304(3): 635-645. DOI: 10.1148/radiol.212400.
- [38] CHRISTENSEN J, LI C, MYCHASIUK R. Choroid plexus function in neurological homeostasis and disorders: The awakening of the circadian clocks and orexins[J]. J Cereb Blood Flow Metab, 2022, 42(7): 1163-1175. DOI: 10.1177/0271678X221082786.
- [39] FOKKINGA E, HERNANDEZ-TAMAMES J A, IANUS A, et al. Advanced diffusion-weighted MRI for cancer microstructure assessment in

body imaging, and its relationship with histology[J]. J Magn Reson Imaging, 2024, 60(4):1278-1304. DOI: 10.1002/jmri.29144.

- [40] CHEN L, HUANG L, ZHANG J, et al. Amide proton transfer-weighted and arterial spin labeling imaging may improve differentiation between high-grade glioma recurrence and radiation-induced brain injury[J/OL]. Heliyon, 2024, 10(11): e32699 [2024-08-09]. https://pmc.ncbi.nlm.nih. gov/articles/PMC11219995/ DOI: 10.1016/j.heliyon.2024.e32699.
- [41] JABEHDAR MARALANI P, CHAN R W, LAM W W, et al. Chemical exchange saturation transfer MRI: What neuro-oncology clinicians need to know[J/OL]. Technol Cancer Res Treat, 2023, 22: 15330338231208613 [2024-08-09]. https://pmc.ncbi.nlm.nih.gov/articles/ PMC10594966/. DOI: 10.1177/15330338231208613.
- [42] KAMAGATA K, ANDICA C, TAKABAYASHI K, et al. Association of MRI indices of glymphatic system with amyloid deposition and cognition in mild cognitive impairment and Alzheimer disease[J/OL]. Neurology, 2022, 99(24): e2648-e2660 [2024-08-09]. https://pmc.ncbi.nlm. nih.gov/articles/PMC9757870/. DOI: 10.1212/wnl.000000000201300.
- [43] SHEN T, YUE Y, BA F, et al. Diffusion along perivascular spaces as marker for impairment of glymphatic system in Parkinson's disease[J/OL]. NPJ Parkinsons Dis, 2022, 8(1): 174 [2024-08-09]. https://pmc.ncbi.nlm.nih. gov/articles/PMC9772196/. DOI: 10.1038/s41531-022-00437-1.
- [44] GAO M, LIU Z, ZANG H, et al. A histopathologic correlation study evaluating glymphatic function in brain tumors by multi-parametric MRI[J]. Clin Cancer Res, 2024, 30(21): 4876-4886. DOI: 10.1158/ 1078-0432.
- [45] SHANG P, ZHENG R, WU K, et al. New insights on mechanisms and therapeutic targets of cerebral edema[J]. Curr Neuropharmacol, 2024, 22(14): 2330-2352. DOI: 10.2174/1570159x22666240528160237.



(上接第199页)

- [63] MEIJBOOM R, YORK E N, KAMPAITE A, et al. Patterns of brain atrophy in recently-diagnosed relapsing-remitting multiple sclerosis[J/OL]. PLoS One, 2023, 18(7): e0288967 [2024-09-23]. https://pubmed.ncbi. nlm.nih.gov/37506096/. DOI: 10.1371/journal.pone.0288967.
- [64] BONACCHI R, MEANI A, PAGANI E, et al. The role of cerebellar damage in explaining disability and cognition in multiple sclerosis phenotypes: a multiparametric MRI study[J]. J Neurol, 2022, 269(7): 3841-3857. DOI: 10.1007/s00415-022-11021-1.
- [65] RUGGIERI S, BHARTI K, PROSPERINI L, et al. A comprehensive approach to disentangle the effect of cerebellar damage on physical disability in multiple sclerosis[J/OL]. Front Neurol, 2020, 11: 529 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/32695059/. DOI: 10.3389/ fneur.2020.00529.
- [66] SCHOONHEIM M M, DOUW L, BROEDERS T A, et al. The cerebellum and its network: disrupted static and dynamic functional connectivity patterns and cognitive impairment in multiple sclerosis[J]. Mult Scler, 2021, 27(13): 2031-2039. DOI: 10.1177/1352458521999274.
- [67] TOMMASIN S, IAKOVLEVA V, ROCCA M A, et al. Relation of sensorimotor and cognitive cerebellum functional connectivity with brain structural damage in patients with multiple sclerosis and no disability[J]. Eur J Neurol, 2022, 29(7): 2036-2046. DOI: 10.1111/ene.15329.
- [68] WALKER F. Huntington's disease[J/OL]. Lancet, 2007, 369: 218-228 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/17240289/. DOI: 10.1016/ S0140-6736(07)60111-1.
- [69] MEDINA A, MAHJOUB Y, SHAVER L, et al. Prevalence and incidence of Huntington's disease: an updated systematic review and

meta-analysis[J]. Mov Disord, 2022, 37(12): 2327-2335. DOI: 10.1002/mds.29228.

- [70] GALVEZ V, RAMÍREZ-GARCÍA G, HERNANDEZ-CASTILLO C R, et al. Extrastriatal degeneration correlates with deficits in the motor domain subscales of the UHDRS[J/OL]. J Neurol Sci, 2018, 385: 22-29 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/29406908/. DOI: 10.1016/j.jns.2017.11.040.
- [71] JUNCA E, PINO M, SANTAMARÍA-GARCÍA H, et al. Brain, cognitive, and physical disability correlates of decreased quality of life in patients with Huntington's disease[J]. Qual Life Res, 2023, 32(1): 171-182. DOI: 10.1007/s11136-022-03220-0.
- [72] PADRON-RIVERA G, DIAZ R, VACA-PALOMARES I, et al. Cerebellar degeneration signature in Huntington's disease[J]. Cerebellum, 2021, 20(6): 942-945. DOI: 10.1007/s12311-021-01256-5.
- [73] TERESHCHENKO A, MAGNOTTA V, EPPING E, et al. Brain structure in juvenile-onset Huntington disease[J/OL]. Neurology, 2019, 92(17): e1939-e1947 [2024-09-23]. https://pubmed.ncbi.nlm.nih.gov/ 30971481/. DOI: 10.1212/WNL.00000000007355.
- [74] SANTOS-LOBATO B L, ROCHA J S S, ROCHA L C. Case report: Cerebellar sparing in juvenile Huntington's disease[J/OL]. Front Neurol, 2023, 13: 1089193 [2024-09-23]. https://pubmed.ncbi.nlm.nih. gov/36712421/. DOI: 10.3389/fneur.2022.1089193.
- [75] ZHANG S R, LIN J Y, CHENG Y F, et al. Aberrant resting-state brain activity in Huntington's disease: a voxel-based meta-analysis[J/OL]. Front Neurol, 2023, 14: 1124158 [2024-09-23]. https://pubmed.ncbi. nlm.nih.gov/37064205/. DOI: 10.3389/fneur.2023.1124158.

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