·帕金森病及运动障碍性疾病·

脑积水脑室-腹腔分流术后帕金森综合征三例临床分析

李胜德 王琳 关鸿志 魏俊吉 彭斌 万新华

【摘要】 **目的** 探讨脑积水脑室-腹腔分流术后帕金森综合征的临床表现、影像学特点、可能的发病机制和治疗方法。**方法与结果** 共3例脑积水患者均行脑室-腹腔分流术，1例于第2次术后1个月出现帕金森综合征，再次行脑室-腹腔分流术后症状缓解不明显，予左旋多巴和苄丝肼；1例于术后17年出现颈部僵直、动作迟缓，予舒必利后短期内症状明显加重，停药后无改善；1例于术后5个月出现言语减少、动作迟缓，予奥氮平后症状明显加重。3例患者头部CT和(或)MRI均显示侧脑室呈扩大-缩小波动性变化，临床诊断为帕金森综合征(脑室-腹腔分流术后)，予左旋多巴和苄丝肼后症状明显缓解。**结论** 帕金森综合征是脑积水脑室-腹腔分流术后罕见并发症，可能是侧脑室扩大-缩小波动性变化引起黑质纹状体通路损害所致，予多巴胺D2受体阻断剂可因“双重打击”致短期内帕金森样症状加重。多数患者多巴胺能药物治疗有效。

【关键词】 脑积水； 脑室腹膜分流术； 手术后并发症； 帕金森障碍

Parkinsonism following ventriculoperitoneal shunt for treating hydrocephalus: clinical analysis on three cases

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【Abstract】 **Objective** To explore the clinical presentations, imaging features, probable pathogenesis and therapy of parkinsonism following ventriculoperitoneal shunt (VPS) in hydrocephalus. **Methods and Results** There were 3 cases of parkinsonism following VPS in hydrocephalus. Case 1 presented parkinsonism one month after the second ventricular shunt, which was not relieved by another VPS, and was then treated by levodopa and benserazide. Case 2 developed neck rigidity and bradykinesia 17 years after VPS. Symptoms worsened shortly after taking sulpiride and did not improved with sulpiride cessation. Bradykinesia and decreased speech occurred 5 months after VPS in Case 3, and parkinsonism aggravated rapidly on the following day after taking olanzapine. CT and/or MRI of 3 cases showed fluctuating change (enlarging - shrinking) of lateral ventricles. They were diagnosed as parkinsonism following VPS, and responded well to levodopa and benserazide. **Conclusions** Parkinsonism, a rare complication following VPS in hydrocephalus, may result from interruption of nigrostriatal pathways due to ventricular fluctuations. Administration of dopamine D2 receptor antagonist may exacerbate the symptoms of parkinsonism because of "double hit". Most patients are responsive to dopaminergic drugs.

【Key words】 Hydrocephalus; Ventriculoperitoneal shunt; Postoperative complications; Parkinsonian disorders

脑室-腹腔分流术(VPS)是临床治疗脑积水的常用方法，可以有效降低病残率和病死率[1-2]。术后出现帕金森综合征是罕见的手术相关并发症。本文回顾分析3例脑室-腹腔分流术后帕金森综合征患者的临床表现、影像学特点、可能的发病机制和治疗方法，并对相关文献进行复习，以期提高临床医师对该病的认识，如果能够早期积极干预，有可

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能改善患者预后。

临床资料

例1 男性，41岁，主因头痛2年余，饮水呛咳、吞咽困难、构音不清1月余，于2004年10月18日入院。患者2年前无明显诱因出现缓慢加重的全头部胀痛，发作初期呈间断性，逐渐进展为持续性；外院头部CT显示梗阻性脑积水，遂行右侧侧脑室-腹腔分流术，术后头痛症状完全缓解。2个月前再次出现头痛，性质同前；外院头部CT显示梗阻性脑积水，再次行左侧侧脑室-右心房分流术，术后头痛症状完全缓解。1月余前逐渐出现流涎、声音低沉、饮水呛咳、吞咽困难、言语不清等症状；外院头部MRI未见脑室系统扩张。为求进一步诊断与治疗，至我院就诊。入院后体格检查：神志清楚，言语不清、声音低沉，反应迟钝，表情减少，眼球上视受限，颈项强直，四肢肌力正常、肌张力增高，肢体深浅感觉正常，双上肢伴随动作减少、双下肢腱反射活跃，左侧病理征阳性、右侧可疑阳性。实验室检查血清学指标未见明显异常；腰椎穿刺脑脊液检查压力110 mm H_2O（1 mm H_2O = 9.81 × 10^{-3} kPa，80 ~ 180 mm H_2O），常规、生化、细胞学和免疫学指标均于正常水平。肌电图检查神经传导速度（NCV）和瞬目反射未见异常。入院后1周头痛症状加重，尿失禁伴全身颤抖，无法站立和行走，继而出现嗜睡。复查CT显示第三脑室和双侧侧脑室再次扩张，遂行左侧侧脑室-腹腔分流术，术后复查CT显示扩张的侧脑室明显缩小，但临床症状无明显改善。临床诊断为帕金森综合征（脑室-腹腔分流术后），予以左旋多巴200 mg/次（3次/d）+ 苄丝肼50 mg/次（3次/d）以及金刚烷胺100 mg/次（3次/d）口服，连续治疗10 d后临床症状明显改善，可自主进流食，在轻微辅助下站立和行走。患者共住院22 d，出院后失访。

例2 女性，35岁，因反复恶心、呕吐15个月，烦躁3个月，缄默、肢体僵直2个月，于2015年2月26日入院。患者17年前颅脑创伤致脑积水于外院行右侧侧脑室-腹腔分流术，术后正常生活16年。15个月前开始反复出现恶心、呕吐，时为喷射状呕吐，呕吐物为胃内容物，外院予甘露醇治疗（具体剂量不详）后症状缓解，头部CT和（或）MRI显示侧脑室扩大，未予特殊处理。2个月前出现颈部前屈僵硬，言语减少，动作迟缓，外院诊断为抑郁症、自身免疫性脑炎，予舒必利对症治疗（具体剂量不详），治疗10 d后出现吞咽困难、流涎、面无表情、肢体僵直、少动、无法行走，遂停药，症状仍持续加重。为求进一步诊断与治疗，至我院就诊。入院后体格检查：神志清楚，缄默，可配合闭眼、张口等简单指令，动作幅度小、动作缓慢，四肢肌力正常、肌张力增高，可见缓慢自主活动，肢体深浅感觉正常，四肢腱反射活跃，双侧病理征阳性，颈项强直，无疼痛回避，共济运动和姿势步态检查不配合。实验室检查数次腰椎穿刺脑脊液压力200 ~ 260 mm H_2O，常规、生化、细胞学和免疫学指标均于正常值范围。头部CT和（或）MRI显示侧脑室呈扩大-缩小波动性变化（图1）。临床诊断为帕金森综合征（脑室-腹腔分流术后），予以左旋多巴400 mg/次（3次/d）+ 苄丝肼100 mg/次（3次/d）口服，症状逐渐缓解，可自主言语、遵嘱活动肢体、自主进食。患者住院25 d，出院后10个月随访，药物剂量减至左旋多巴100 mg/次（3次/d）+ 苄丝肼25 mg/次（3次/d），临床症状持续改善，可自主行走、器械锻炼；出院后19个月随访时，继续服用左旋多巴 + 苄丝肼（药物剂量维持不变），运动功能基本恢复，无头痛、恶心、呕吐和肢体僵直等症状。

例3 女性，18岁，主因头痛4年，言语减少、动作迟缓1个月，于2015年12月23日入院。患者4年前车祸伤后出现头部胀痛、视物模糊、全身乏力，偶有恶心、呕吐，呈非喷射状呕吐；外院头部CT显示脑积水，由于患者临床症状不明显，未予特殊处理。症状进行性加重，6个月前症状明显加重，外院头部CT显示梗阻性脑积水，行右侧侧脑室-腹腔分流术，术后症状缓解。1个月前出现反复头痛、言语减少、全身颤抖、动作迟缓，外院诊断为抑郁症，予奥氮平2 mg后全身颤抖加重、言语不能、无法进食、行走不能，无意识障碍，停药后症状仍进行性加重。为求进一步诊断与治疗，至我院门诊就诊。体格检查：神志清楚，不能对答，双眼上视眼震，颈项强直，四肢肌力检查不能配合、肌张力增高，可见缓慢自主运动，共济运动和感觉系统检查不能配合，病理征阴性。头部CT显示侧脑室呈扩大-缩小波动性变化（图2）。临床诊断为帕金森综合征（脑室-腹腔分流术后），予以左旋多巴100 mg/次（3次/d）+ 苄丝肼25 mg/次（3次/d）口服，连续治疗1个月后可自主进食，肢体活动增多。患者2个月后随访时，停用左旋多巴，可于辅助下行走，言语自如；3个月后随访时，能够生活自理。

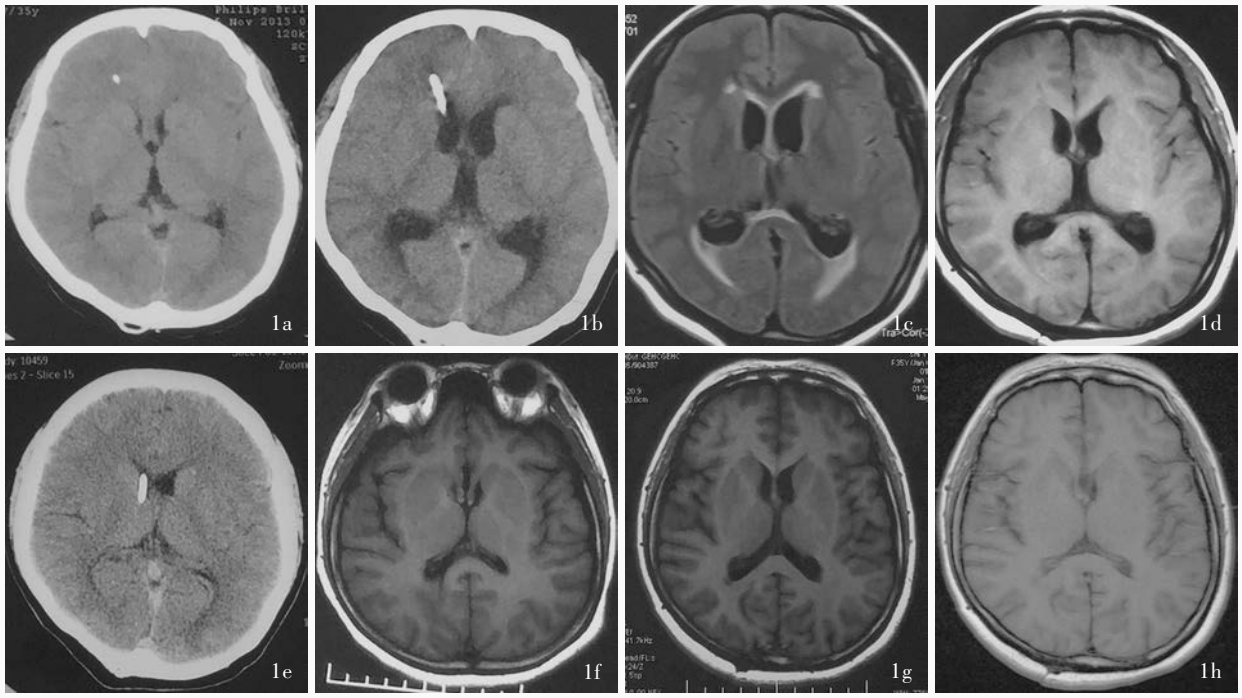

图1 头部影像学检查所见 1a 横断面CT(2013年11月5日)显示双侧侧脑室明显缩小 1b 横断面CT(2013年12月24日)显示双侧侧脑室扩大 1c 横断面FLAIR成像(2014年1月13日)显示双侧侧脑室扩大 1d 横断面T_1WI(2014年5月20日)显示双侧侧脑室扩大 1e 横断面CT(2014年11月27日)显示双侧侧脑室明显缩小 1f 横断面T_1WI(2014年12月17日)显示双侧侧脑室较图1e扩大 1g 横断面T_1WI(2015年1月21日)显示双侧侧脑室较图1f扩大 1h 横断面T_1WI(2015年3月5日)显示双侧侧脑室较图1g缩小

Figure 1 Head imaging findings Axial CT (November 5, 2013) showed significant volume reduction of bilateral lateral ventricles (Panel 1a). Axial CT (December 24, 2013) showed the volume of bilateral lateral ventricles was enlarged (Panel 1b). Axial FLAIR (January 13, 2014) showed enlargement of bilateral lateral ventricles (Panel 1c). Axial T_1WI (May 20, 2014) showed enlargement of bilateral lateral ventricles (Panel 1d). Axial CT (November 27, 2014) showed significant volume reduction of bilateral lateral ventricles (Panel 1e). Axial T_1WI (December 17, 2014) showed bilateral lateral ventricles was larger than Panel 1e (Panel 1f). Axial T_1WI (January 21, 2015) showed bilateral lateral ventricles was larger than Panel 1f (Panel 1g). Axial T_1WI (March 5, 2015) showed bilateral lateral ventricles was smaller was larger than Panel 1g (Panel 1h).

讨 论

脑室-腹腔分流术后并发症5年累积发生率约32%,主要为分流失败和感染[2]。术后帕金森综合征是其罕见并发症,迄今全球仅有28例报道[3-20],男性17例,女性11例;发病年龄7~68岁,平均30岁;术前均表现为头痛、恶心、呕吐等颅内高压症状;脑积水病因以中脑导水管狭窄致梗阻性脑积水多见,脑室-腹腔分流术后上述症状改善;帕金森综合征可出现于单次或多次脑室-腹腔分流术后,13例发生于单次术后3个月至17年,其中8例(8/13)发生于单次术后1年内,15例发生于多次术后(距末次手术1天至4个月),其中8例(8/15)发生于末次术后1周内;临床表现为面部表情减少、流涎、言语减少、声音低沉、吞咽困难、动作迟缓、肢体僵直、翻身困难;体格检查可见肢体震颤、肌张力增高、姿势步态不稳、严重时无法独立行走,亦可出现反应迟钝、认知功能障碍,多伴上视麻痹和眼震,可伴Parinaud综合征,腱反射活跃,部分患者病理征阳性;头部影像学检查多提示侧脑室呈扩大-缩小波动性变化。

本组3例患者均因梗阻性脑积水行脑室-腹腔分流术,术后出现典型帕金森综合征表现,头部影像学均显示侧脑室呈扩大-缩小波动性变化,称为脑室震荡。例1因梗阻性脑积水曾行两次脑室-腹腔分流术,第2次术后1个月并发帕金森综合征,表现为表情减少、动作迟缓、肢体僵直,延髓症状考虑与肌张力增高有关,同时伴双眼上视受限,再次行脑室-腹腔分流术后症状无缓解;例2颅脑创伤致脑积水,予脑室-腹腔分流术后17年出现言语减少、肢体僵直,服用舒必利后短期内症状明显加重,停药无缓解;例3车祸伤致脑积水,予脑室-腹腔分流术后5个月出现言语减少、动作迟缓,服用小剂量奥氮平后症状明显加重,肢体僵直,无法行走。3例患者的临床表现、病程进展和影像学特点均与文献报道相

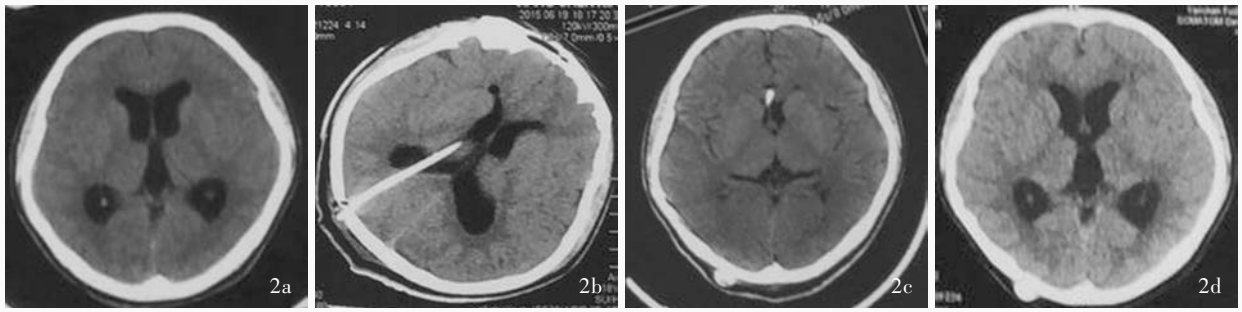

图2 头部横断面CT检查所见 2a 2014年5月2日显示双侧侧脑室扩大 2b 2015年6月19日显示双侧侧脑室明显扩大 2c 2015年6月24日显示双侧侧脑室明显缩小 2d 2015年10月8日显示脑室-腹腔分流术后双侧侧脑室明显扩大

Figure 2 Axial CT findings Axial CT (May 2, 2014) showed enlargement of bilateral lateral ventricles (Panel 2a). Axial CT (June 19, 2015) showed the significant enlargement of bilateral lateral ventricles (Panel 2b). Axial CT (June 24, 2015) showed significant volume reduction of bilateral lateral ventricles (Panel 2c). Axial CT (October 8, 2015) showed obvious enlargement of bilateral lateral ventricles after VPS (Panel 2d).

一致，但是由于临床医师对该病的认识不足，先后诊断为神经变性病、自身免疫性脑炎、抑郁症等而予相应治疗，其中例2和例3服用抗精神病药后临床症状迅速加重，此前的文献未见类似报道。

脑室-腹腔分流术后帕金森综合征的发病机制至目前尚未完全阐明，研究者们围绕黑质纹状体通路提出不同假设[21]。此类患者FLAIR成像显示中脑导水管周围呈高信号，^{18}F-多巴胺（^{18}F-DOPA）PET显像显示纹状体放射性摄取降低，提示可能存在纹状体直接损伤、黑质纹状体通路损伤和黑质致密部损伤[6-7,10-12]。脑室-腹腔分流术后可以出现分流管功能障碍[4,6-7,12-13,19]，导致脑脊液引流异常，脑脊液淤积和过度引流可以导致侧脑室呈扩大-缩小波动性变化。脑室-腹腔分流术后帕金森综合征的可能机制包括以下几方面：（1）扩大的侧脑室和第三脑室直接压迫和改变毗邻的尾状核头部和基底节结构[8]，造成轴突损伤、脑白质小血管受压变形、毛细血管显著减少，继发神经胶质细胞反应性改变[22]。（2）颅内高压导致间脑和中脑上部黑质纹状体投射纤维剪切力增高、扭曲和缺血改变[6,10]，脑组织间隙压力增高导致代谢产物蓄积[22]。（3）在此基础上侧脑室扩大-缩小波动性变化对侧脑室旁黑质纹状体产生机械性冲击力，造成上述结构扭曲、变形和缺血改变，投射纤维受到机械性破坏[13]。（4）脑组织牵张-收缩循环改变组织物理特性，使其对压力变化更加敏感，脑室扩大-缩小波动速度超过代偿机制时脑组织损伤[4]。（5）反复行脑室-腹腔分流术，可以造成侧脑室过度引流，继而出现逆向跨小脑幕压力差，造成中脑结构破坏和中脑功能障碍，甚至导致纹状体丘脑联系和皮质纹状体通路损伤[13]。（6）脑积水可以造成纹状体突触后膜多巴胺D2受体缺失或表达下调[23]，加重临床症状。总之，侧脑室扩大-缩小波动性变化主要影响黑质纹状体通路功能，从而出现相应临床症状。

本组例2和例3服用抗精神病药后帕金森样症状迅速加重，可以用“双重打击”机制解释。脑室-腹腔分流术后侧脑室扩大-缩小波动性变化形成对黑质纹状体通路的第1次打击；抗精神病药对多巴胺D2受体有拮抗作用，形成对多巴胺能投射通路的第2次打击，从而迅速加重临床症状。综合文献报道的28例患者和本组3例患者，出现典型帕金森样症状前，患者多有头痛、呕吐、嗜睡、言语减少、动作迟缓和反应迟钝等临床表现，此种非特异性症状类似脑炎、精神病或抑郁症，若予多巴胺D2受体阻断剂，更易加重帕金森综合征。

脑室-腹腔分流术后帕金森综合征的危险因素目前尚未阐明，考虑与分流管功能异常有关。据文献报道，分流管功能异常发生率为8%～64%[24]，可发生于分流管近端、分流管瓣膜和远端腹腔内，病因包括堵塞、中断和过度引流等。脑室-腹腔分流术后帕金森综合征患者侧脑室呈扩大-缩小波动性变化，推测分流管存在可逆性引流不畅，如分流管近端靠近脉络丛或腹腔大网膜包裹等，而脑脊液蛋白定量和可调压分流瓣与分流失败的关系尚不确定，但任何导致分流管功能障碍的因素均应警惕。分流管远端远离脉络丛可以减少分流失败的概率[24]，其他危险因素尚待进一步研究。

根据上述发病机制，参考帕金森病治疗原则，多巴胺能药物如左旋多巴、多巴胺受体激动剂和金刚烷胺、抗胆碱能药物均可用于脑室-腹腔分流术后

帕金森综合征的治疗[21]。文献报道的28例患者中22例予药物治疗[6-7,10,12,15-16,18-20]，其中，单纯应用左旋多巴或左旋多巴联合其他药物治疗16例，13例（13/16）治疗有效；金刚烷胺、苯海索、溴隐亭或屈昔多巴联合治疗6例，2例（2/6）症状改善[16,20]。服用左旋多巴的患者，药物剂量不同，最高1500 mg/d[10]，治疗后2周至2个月临床症状明显缓解。对于多巴胺能药物治疗无效的患者，Curran和Lang[7]认为与传导通路受损部位有关，传导通路分为黑质纹状体通路、纹状体丘脑通路和皮质纹状体通路，当黑质纹状体通路相对正常、纹状体丘脑通路和皮质纹状体通路损伤时，多巴胺能药物治疗无效。^{18}F-DOPA PET显示纹状体放射性摄取正常，提示黑质纹状体通路无明确损害，推测多巴胺能药物治疗无效。

文献报道的28例患者中10例行脑室-腹腔分流术[3-4,7-9,11,13-14,17,20]，其中6例直接手术治疗[3-4,7,9,14,17]，4例在药物治疗无效后改行手术治疗[5,8-9,20]，其中仅1例术后再予药物治疗后症状改善，余3例效果欠佳；3例重新置入分流管[4,7-8]，其中1例术后予左旋多巴后症状缓解，余2例效果欠佳；4例行第三脑室造瘘术[5,11,13,17]，症状均改善，其中3例未联合药物治疗。Okawa等[13]认为，早期行第三脑室造瘘术联合积极的多巴胺序贯治疗，可能减少并发症。

本组3例患者均采用以左旋多巴为主的治疗，均于药物治疗后1个月临床症状明显改善，其中例2左旋多巴剂量增至1200 mg/d时方逐渐症状改善。例2和例3病情平稳后减少药物剂量或停用左旋多巴，症状无加重或复发，例1失访。复习相关文献，脑室-腹腔分流术后出现帕金森综合征表现时，推荐左旋多巴药物，可以联合多巴胺受体激动剂、金刚烷胺或抗胆碱能药物，若左旋多巴疗效不佳可考虑第三脑室造瘘术或行^{18}F-DOPA PET评价黑质纹状体通路功能。禁止使用多巴胺D2受体阻断剂。

随着脑室-腹腔分流术治疗脑积水病例数的增加[1-2]，术后帕金森综合征病例数亦逐渐增加。患者出现帕金森样症状和侧脑室扩大-缩小波动性变化时，应考虑脑室震荡对黑质纹状体通路的影响，早期予左旋多巴治疗，能够改善临床症状，提高患者生活质量，应注意避免应用多巴胺D2受体阻断剂。

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The 23rd World Congress of Neurology

Time: September 16–21, 2017

Venue: Kyoto, Japan

Abstract submission deadline: April 5, 2017

Website: www.2017.wcn-neurology.com

The 23rd World Congress of Neurology (WCN 2017) will take place in Kyoto, Japan on September 16–21, 2017, cohosted by the Japanese Society of Neurology (JSN), Societas Neurologica Japonica, and Asian and Oceanian Association of Neurology (AOAN). The theme of WCN 2017 will be "Defining the Future of Neurology".

Founded originally in 1902, the JSN has evolved into a large society with 8579 members. Initially a combined neurology and psychiatry association, the current JSN separated in 1959 and continued to flourish ever since. It was the 12th WCN meeting held at Kyoto in 1981 that greatly contributed to the development of JSN and AOAN. Therefore, WCN 2017 which is being held at the very same venue would be a very historic meeting which will again serve as a springboard to strongly advance the Asia Initiative of World Federation of Neurology (WFN) for worldwide advancement of neurology in both scientific and clinical aspects, thus "Defining the Future of Neurology". You can participate in very active discussions and cutting-edge lectures by the world's top scientists and neurologists including three Nobel laureates as well as hear all the advances of scientific and clinical neurology. Gene therapy and stem/induced pluripotent stem (iPS) cell medicine are such examples on the one hand and brain-machine-interface, information technology and robotics in care and rehabilitation on the other. Neurology related to environmental and disaster medicine will also attract many neurologists particularly in rapidly developing countries.

Abstract topics include: ataxia and cerebellar disorders, autoimmune disorders, autonomic nervous system disorders, channelopathies, child neurology, central nervous system (CNS) infections, dementia, epilepsy, functional disorders and behavioral neurology, headache, health economics & outcomes, history of neurology, metabolic and mitochondrial diseases, motor neuron disease, movement disorders, multiple sclerosis (MS) & demyelinating diseases, neuro-critical care, neuroepidemiology and environmental, neuroethics, neurogenetics, neuroimaging, neuromuscular disorders, neurooncology, neuroophthalmology, neurootology/vestibular disorders, neurophysiology, neurorehabilitation, nutritional disorders or intoxications, pain, palliative care, sleep disorders, sports neurology, stem cells and gene therapy.

The Local Organizing Committee of JSN in close collaboration with the WFN looks forward to welcoming you in Kyoto for WCN 2017.