·临床研究·

颅内动脉开窗畸形致缺血性卒中临床研究

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【摘要】 **目的** 总结颅内动脉开窗畸形致缺血性卒中的临床特点，并探讨缺血性卒中与颅内动脉开窗畸形的关系。**方法与结果** 共11例颅内动脉开窗畸形致缺血性卒中患者，头部MRI显示，梗死灶位于脑桥5例（5/11）、内囊后肢3例（3/11）、左侧丘脑1例（1/11）、左侧半卵圆中心1例（1/11）、双侧枕叶合并左侧颈内动脉动脉瘤1例（1/11）；头部MRA显示，开窗畸形发生于基底动脉9例（9/11）、左侧椎动脉1例（1/11）、右侧大脑后动脉1例（1/11）；其中，5例脑桥缺血性卒中系基底动脉（4例）和右侧大脑后动脉（1例）开窗畸形所致，3例内囊后肢缺血性卒中均系基底动脉开窗畸形所致，1例左侧丘脑缺血性卒中系左侧椎动脉开窗畸形所致，1例左侧半卵圆中心缺血性卒中系基底动脉开窗畸形所致，1例双侧枕叶缺血性卒中系基底动脉开窗畸形所致。均予抗血小板、调脂和清除自由基治疗，无一例缺血性卒中复发。**结论** 颅内动脉开窗畸形可以导致局部血流动力学改变，与缺血性卒中密切相关。

【关键词】 卒中； 脑缺血； 血管畸形； 基底动脉

Clinical analysis on ischemic stroke caused by intracranial artery fenestration

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【**Abstract**】 **Objective** To discuss the clinical features of ischemic stroke caused by intracranial artery fenestration, and to explore possible correlation between ischemic stroke and intracranial artery fenestration. **Methods and Results** We retrospectively studied 11 cases of ischemic stroke caused by intracranial artery fenestration from December 2012 to October 2017. Cranial MRI showed the infarcts were located in pons (5 cases, 5/11), posterior limb of internal capsule (3 cases, 3/11), left thalamus (one case, 1/11), left centrum semiovale (one case, 1/11) and bilateral occipital lobes combined with left internal carotid artery (ICA) aneurysm (one case, 1/11). MRA showed fenestration in basilar artery (BA) was found in 9 cases (9/11), fenestration in left vertebral artery (VA) was found in one case (1/11), fenestration in right posterior cerebral artery (PCA) was found in one case (1/11). Five cases of pontine infarction were caused by BA fenestration (4 cases) and right PCA fenestration (one case); 3 cases of internal capsule posterior limb infarction were caused by BA fenestration; one case of left thalamic infarction was caused by left VA fenestration; one case of left centrum semiovale infarction was caused by BA fenestration; one case of bilateral occipital lobes infarction was caused by BA fenestration. All patients were treated by antiplatelet aggregation, lipid regulation and scavenging free radical. No patient recurred ischemic stroke. **Conclusions** Intracranial artery fenestration may results in focal hemodynamic changes, and is closely related to ischemic stroke.

【**Key words**】 Stroke; Brain ischemia; Vascular malformations; Basilar artery

颅内动脉开窗畸形系指颅内动脉局限性血管重复，表现为1支动脉在走行过程中分为2支，平行走行一段距离后再汇合[1]，最常见于椎-基底动脉系统。颅内动脉开窗畸形使血管弯曲增多，导致血流动力学改变，使动脉内膜损害，易在损伤局部形成血栓，从而导致颅内动脉狭窄或脑栓塞[2]。首都医科大学附属复兴医院2012年12月–2017年10月共诊断与治疗11例颅内动脉开窗畸形导致缺血性卒中患者，本研究回顾分析其临床资料，总结其临床特点，并探讨缺血性卒中与颅内动脉开窗畸形之间的关系。

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临床资料

一、病例选择

1. 纳入标准 (1)缺血性卒中的诊断符合《中国急性缺血性脑卒中诊治指南2014》[3],并经头部MRI检查证实。(2)发病急骤,符合脑血管病发病形式。(3)头部MRA显示颅内动脉开窗畸形。(4)本研究经首都医科大学附属复兴医院道德伦理委员会审核批准,所有患者或其家属均知情同意并签署知情同意书。

2. 排除标准 (1)头部影像学显示不清,诊断有争议。(2)颅内开窗畸形为偶然发现,无临床症状。(3)不能配合完成检查。

3. 一般资料 选择2012年12月–2017年10月在首都医科大学附属复兴医院神经内科住院治疗的颅内动脉开窗畸形致缺血性卒中患者11例,男性9例,女性2例;年龄34~82岁,平均56.33岁。临床表现为构音障碍2例,头晕4例,双下肢无力1例,左侧肢体无力3例,右侧肢体无力3例,共济失调2例,双眼黑蒙1例,复视1例。11例患者的临床资料详见表1。

二、影像学表现

1. 头部MRI检查 扩散加权成像(DWI)显示,梗死灶位于脑桥5例(5/11),内囊后肢3例(3/11),左侧丘脑1例(1/11),左侧半卵圆中心1例(1/11),双侧枕叶未见明显梗死灶1例(1/11;图1,表1)。

2. 头部MRA检查 MRA显示,开窗畸形发生于基底动脉(BA)共9例,其中远端1例(1/11),近端8例(8/11),且1例(例9)合并左侧颈内动脉动脉瘤;发生于左侧椎动脉(VA)1例(1/11);右侧大脑后动脉(PCA)1例(1/11;图2,表1)。

3. 缺血性卒中与颅内动脉开窗畸形的关系 本组11例患者中4例脑桥缺血性卒中系基底动脉开窗畸形所致、1例脑桥缺血性卒中系右侧大脑后动脉开窗畸形所致,3例内囊后肢缺血性卒中均系基底动脉开窗畸形所致,1例左侧丘脑缺血性卒中系左侧椎动脉开窗畸形所致,1例左侧放射冠缺血性卒中系基底动脉开窗畸形所致,1例双侧枕叶缺血性卒中系基底动脉开窗畸形所致(表1)。

三、治疗及转归

本组有1例(例9)双眼黑蒙持续20~30 min后好转,予阿司匹林100 mg/d口服抗血小板和尼莫地平30 mg/次、3次/d口服抗血管痉挛治疗,其后未再出现黑蒙症状;余10例均予100 mg/d口服抗血小板、阿托伐他汀20~40 mg/晚口服调脂和依达拉奉30 mg/次、2次/d静脉滴注清除自由基治疗,其中有3例(例4、例5、例10)予生理盐水2000~3000 ml/d补液治疗。本组患者共住院9~14 d,平均11.98 d,出院时6例(例1、例3、例5、例8、例9、例11)日常生活不受影响;5例(例2、例4、例6、例7、例10)转至外院继续康复治疗,随访的2例(例2、例4)患者于发病2~6个月后可自主行走,日常生活无需他人照料,失访3例(例6、例7、例10)。本组无一例缺血性卒中复发,均未再次复查头部MRI和MRA。

典型病例

患者(例3) 男性,46岁,因左侧肢体无力1 d,于2015年1月15日入院。患者于1 d前无明显诱因自觉左侧肢体不灵活,左手持物不稳,辨距不良,不能准确抓取物体,行走数步后向左侧偏斜,不影响正常生活和工作,未予特殊处理;次日(1月15日)晨起自觉症状加重,表现为左手持日常生活用品费力,伴麻木感,口角向右侧歪斜,吐字略不清,额纹对称,行走数步后需休息,右侧肢体正常,无头晕、头痛、意识障碍、肢体抽搐,为求进一步诊断与治疗,遂至我院就诊。否认高血压、冠心病、糖尿病病史,吸烟史30年、20支/d,偶饮酒,无其他不良嗜好,否认家族遗传性疾病病史。入院后体格检查:血压150/90 mm Hg(1 mm Hg = 0.133 kPa),神志清楚,言语略含糊,定向力、记忆力、理解力和计算力基本正常;双侧瞳孔等大、等圆,直径约3 mm,对光反射灵敏,各向眼动充分,无眼震、复视,双侧额纹对称,双眼闭目有力,无露睫毛、露白,口角向右侧歪斜,伸舌居中,无舌肌萎缩、肌束颤,悬雍垂居中,咽反射正常,转头、耸肩动作对称,咀嚼有力;左侧肢体肌力4$^+$级,右侧5级,肌张力均正常;左侧指鼻试验轻度欠稳准、跟-膝-胫试验欠稳准,右侧共济运动正常,双侧痛觉和深感觉正常;左侧Babinski征阳性、右侧阴性,脑膜刺激征阴性;听诊颈动脉和锁骨下动脉(SCA)均未闻及血管杂音。实验室检查:血清低密度脂蛋白胆固醇(LDL-C)为3.87 mmol/L(0~3.12 mmol/L),余指标均未见异常。影像学检查:头部MRI显示右侧内囊后肢新发梗死灶(图1b),头部MRA显示基底动脉近端开窗畸形(图2a)。予阿司匹林100 mg/d口服抗血小板、阿托伐他汀20 mg/晚口服调脂和依达拉奉30 mg/次、2次/d静脉滴注清除

表1 11例颅内动脉开窗畸形致缺血性卒中患者的临床资料

Table 1. Clinical data of 11 patients with ischemic stroke caused by intracranial artery fenestration

Case	Sex	Age (year)	Position of infarcts	Position of fenestration	Clinical manifestation
1	Male	82	Left pons	Proximal end of BA	Dysarthria, weakness of both legs
2	Male	39	Left posterior limb of internal capsule	Distal end of BA	Dizziness, weakness on the right side
3	Male	46	Right posterior limb of internal capsule	Proximal end of BA	Weakness on the left side
4	Female	34	Left centrum semiovale	Proximal end of BA	Weakness on the right side
5	Male	66	Right pons	Proximal end of BA	Dizziness, ataxia
6	Male	67	Left pons	Proximal end of BA	Dysarthria, weakness on the left side
7	Female	75	Left thalamus	Left VA	Weakness on the right side
8	Male	81	Right pons	Right PCA	Dizziness, ataxia
9	Male	61	Bilateral occipital lobes (left ICA aneurysm)	Proximal end of BA	Episodic amaurosis
10	Male	62	Left posterior limb of internal capsule	Proximal end of BA	Weakness on the left side
11	Male	54	Left and middle pons	Proximal end of BA	Dizziness, diplopia

BA，basilar artery，基底动脉；VA，vertebral artery，椎动脉；PCA，posterior cerebral artery，大脑后动脉；ICA，internal carotid artery，颈内动脉

自由基治疗，患者共住院11 d，出院时左侧肢体肌力5⁻级，日常生活不受影响，嘱3个月内复查头部MRI和MRA，期间不适随时就诊。随访1年，未见缺血性卒中复发，未复查头部MRI和MRA。

讨　论

动脉开窗畸形是一种血管变异，可发生于人体任何动脉，颅内动脉以椎-基底动脉系统开窗畸形最为常见，开窗一般较短且常发生于动脉近端[4]。开窗畸形动脉呈单一起源，在走行过程中分为2支，共用或不共用同一外膜，平行走行一段距离后再汇合。动脉开窗畸形的机制目前尚不清楚，一般认为的胚胎学机制是，胚胎发育第5周时基底动脉由纵行且成对的神经动脉融合而成，神经动脉如果在某一点停止融合，基底动脉即发育为开窗畸形[5]。椎动脉开窗畸形的机制较基底动脉开窗畸形更为复杂，可能与部分原始椎动脉残留或再通以及发育时丛状血管吻合不完全有关[6]。

英文文献采用“Fenestration”和“Duplication”描述双血管变异，目前通常以“Fenestration”特指开窗畸形（或译为有孔），而“Duplication”译为双起源动脉或局限性血管重复[7]。局限性血管重复系指两支独立血管在走行过程中始终分开，最终与不同血管汇合，这也是一种先天性发育异常，是胚胎发育第32～40天原始血管退化不完全所致[8]。动脉开窗畸形的病理改变是管壁肌层发育不完全，伴某些节段性弹力纤维缺失、某些节段正常，所有节段均由共同的血管外膜包绕；目前尚无关于局限性血管重复的病理学研究，一般认为血管结构是正常的[9]。

颅内动脉开窗畸形通常无特征性临床表现，但部分学者认为，椎-基底动脉开窗畸形与脑干缺血性卒中相关[10]。胡卫东等[4]的尸体解剖研究显示，颅内动脉开窗畸形是2支动脉共用1个动脉内膜，不易分离，且易造成管壁狭窄，部分血栓形成于开窗畸形血管壁，提示血流动力学改变和开窗部位形成湍流口。颅内动脉开窗畸形使血管弯曲增多、折角变锐、血流速度不均匀，从而导致血流动力学改变，造成管壁内膜损害，故易发生动脉粥样硬化，且易在损害局部形成血栓并导致动脉狭窄或栓塞[2]。

本组有3例内囊后肢梗死患者，内囊后肢受前循环和后循环共同供血，前循环供血动脉是豆纹动脉和前脉络膜动脉，后循环供血动脉是丘脑膝状体动脉[11]，因此认为，椎-基底动脉狭窄是内囊后肢缺血性卒中的重要原因[12]。最新的大宗病例研究显示，颅内动脉开窗畸形经MRA检出的阳性率为0.85%[13]。本组有10例患者存在椎-基底动脉开窗畸形，并伴有其支配区新发梗死灶，占我院同期新发缺血性卒中患者的2.27%（10/440），高于文献报道[12-13]，提示颅内动脉开窗畸形增加缺血性卒中的风险。

颅内动脉开窗畸形常伴其他血管异常，以动脉瘤最为常见，其次为头颈部异常，如Klippel-Feil综

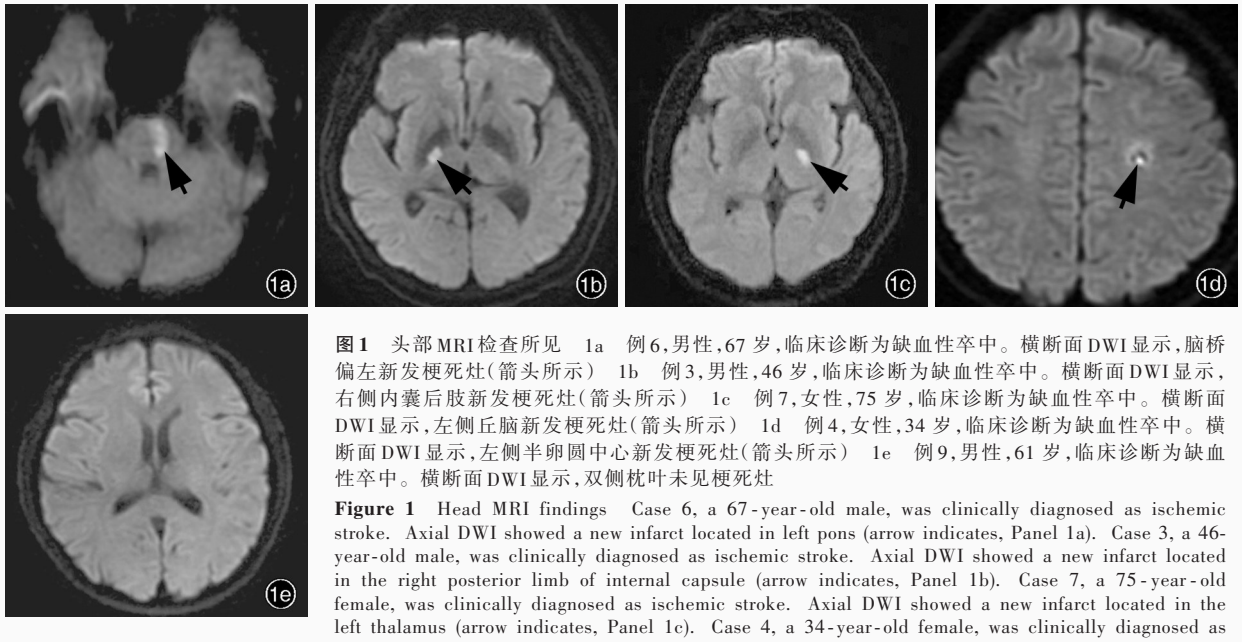

图1 头部MRI检查所见 1a 例6，男性，67岁，临床诊断为缺血性卒中。横断面DWI显示，脑桥偏左新发梗死灶（箭头所示） 1b 例3，男性，46岁，临床诊断为缺血性卒中。横断面DWI显示，右侧内囊后肢新发梗死灶（箭头所示） 1c 例7，女性，75岁，临床诊断为缺血性卒中。横断面DWI显示，左侧丘脑新发梗死灶（箭头所示） 1d 例4，女性，34岁，临床诊断为缺血性卒中。横断面DWI显示，左侧半卵圆中心新发梗死灶（箭头所示） 1e 例9，男性，61岁，临床诊断为缺血性卒中。横断面DWI显示，双侧枕叶未见梗死灶

Figure 1 Head MRI findings Case 6, a 67-year-old male, was clinically diagnosed as ischemic stroke. Axial DWI showed a new infarct located in left pons (arrow indicates, Panel 1a). Case 3, a 46-year-old male, was clinically diagnosed as ischemic stroke. Axial DWI showed a new infarct located in the right posterior limb of internal capsule (arrow indicates, Panel 1b). Case 7, a 75-year-old female, was clinically diagnosed as ischemic stroke. Axial DWI showed a new infarct located in the left thalamus (arrow indicates, Panel 1c). Case 4, a 34-year-old female, was clinically diagnosed as ischemic stroke. Axial DWI showed a new infarct located in the left centrum semiovale (arrow indicates, Panel 1d). Case 9, a 61-year-old male, was clinically diagnosed as ischemic stroke. Axial DWI showed bilateral occipital lobes had no infarcts (Panel 1e).

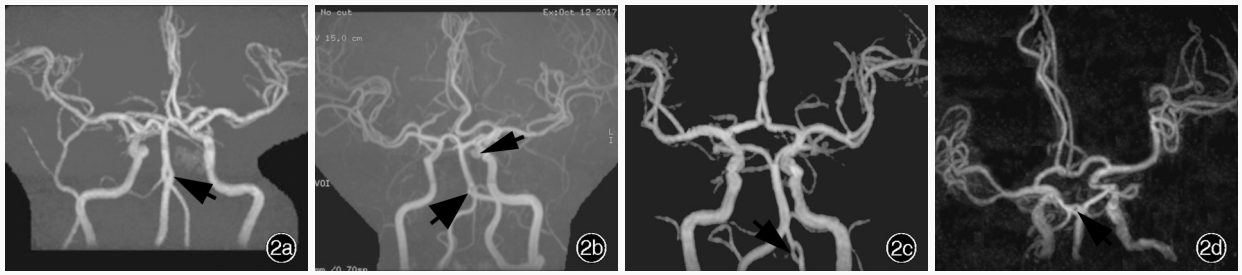

图2 头部MRA检查所见 2a 例3，男性，46岁，临床诊断为缺血性卒中。MRA显示，基底动脉近端分为2支，平行走行一段距离后汇合，提示基底动脉开窗畸形（箭头所示） 2b 例9，男性，61岁，临床诊断为缺血性卒中。MRA显示，基底动脉近端开窗畸形（粗箭头所示）和左侧颈内动脉C2段动脉瘤（细箭头所示） 2c 例7，女性，75岁，临床诊断为缺血性卒中。MRA显示，左侧椎动脉V4段末端开窗畸形（箭头所示） 2d 例8，男性，81岁，临床诊断为缺血性卒中。MRA显示，右侧大脑后动脉P1段开窗畸形（箭头所示）

Figure 2 Head MRA findings Case 3, a 46-year-old male, was clinically diagnosed as ischemic stroke. MRA showed proximal BA was divided into two branches, which converged after a period of parallel running, suggesting a BA fenestration (arrow indicates, Panel 2a). Case 9, a 61-year-old male, was clinically diagnosed as ischemic stroke. MRA showed fenestration of the proximal BA (thick arrow indicates), and C2 segment aneurysm of the left ICA (thin arrow indicates, Panel 2b). Case 7, a 75-year-old female, was clinically diagnosed as ischemic stroke. MRA showed fenestration of the end of V4 segment of left VA (arrow indicates, Panel 2c). Case 8, a 81-year-old male, was clinically diagnosed as ischemic stroke. MRA showed fenestration of the P1 segment of right PCA (arrow indicates, Panel 2d).

合征（颈椎融合综合征）等[6]，或颅内动脉变异，如椎动脉入横突孔水平变异[14]。基底动脉开窗畸形常发生于基底动脉近段靠近椎-基底动脉交界处，且基底动脉近段开窗畸形与该处动脉瘤明显相关[8]，动脉瘤好发于动脉开窗畸形分叉部和窗口支分叉近端内侧[15]。有文献报道，颅内动脉开窗畸形近端动脉瘤的形成与Willis环动脉瘤的发生机制相似，是由于动脉中膜损害，血流在局部形成湍流而冲击血管，在血管分叉部易形成囊状动脉瘤[5]。针对颅内动脉开窗畸形与颅内动脉瘤血流动力学的相关性，段玉霞等[16]进行数值模拟分析，发现开窗处是旋涡状高剪切力区，表明此处血流动力学压力升高；血流冲击时，动脉瘤颈受剪切力最大，瘤体次之，瘤顶最小。本组例9患者存在左侧颈内动脉C2段动脉瘤，开窗畸形发生于基底动脉近端，但二者在解剖学上无明显相关性，是对上述文献的进一步补充。

颅内动脉开窗畸形合并颅内动脉瘤或其他颅内血管异常时，对神经介入科、神经外科的治疗具有提示作用[17]。近年来，后循环动脉瘤倾向于血管内介入治疗[18]，颅内动脉开窗畸形的存在，使颅内

动脉瘤血管内介入治疗难度增大，手术时还须避免部分重复的血管损伤，若手术、栓塞、血栓形成等原因造成开窗畸形的2支动脉中1支堵塞，也可能导致严重的缺血性卒中[1]。此外，常见并发症还包括动脉瘤破裂，严重影响预后[19]。但亦有学者得出相反结论，认为局限性血管重复可发挥保护作用，其中一支狭窄或堵塞时，另一支仍能发挥供血作用[9]。

本研究采用3.0T MRA检测颅内动脉开窗畸形，具有典型影像学特征[4]。既往文献多以数字减影血管造影术（DSA）诊断颅内动脉开窗畸形，为创伤性检查，且辐射较大、检查时间较长、费用较昂贵。目前3.0T MRA可以清晰显示颅内动脉开窗畸形的部位、走行、形态、狭窄或扩张、动脉瘤及其与之毗邻关系，三维时间飞跃（3D-TOF）MRA还可以减少因快速注射对比剂给患者带来不适感和对比剂过敏的风险，且费用相对较低、操作方便、检查时间短，更能为人们所接受，降低颅内动脉检查的门槛，适用于脑血管病的初筛和诊断，有助于提高颅内动脉开窗畸形的检出率，可以作为诊断颅内动脉开窗畸形的首选方法[20]，但是对于存在疑惑的病例，仍应采用DSA明确诊断[21]。

本组例9患者临床表现为双眼黑蒙，持续20～30分钟后视力逐渐恢复，考虑为椎-基底动脉短暂性脑缺血发作（TIA）。患者既往常出现双侧额部疼痛，发作前多次出现左侧视野大片黄色光斑，持续约10分钟，考虑为典型偏头痛，故双眼黑蒙不排除基底动脉型偏头痛。其发病原因可能是，颅内动脉开窗畸形使局部血流动力学改变，出现局部脑缺血，从而增加偏头痛的风险，或者是血管异常导致局部血流动力学改变，酸性代谢产物积聚，导致颅内小动脉扩张，同时出现颅外动脉反应性扩张，从而导致头痛发作[22]。

综上所述，颅内动脉开窗畸形可以导致局部血流动力学改变，与缺血性卒中密切相关。

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Fifth European Stroke Organization Conference

Time: May 22–24, 2019

Venue: Milan, Italy

Website: http://eso-conference.org/2019/

The 5th European Stroke Organization Conference (ESOC) will take place in Milan, Italy, on May 22–24, 2019. ESOC 2019 will build on the enormous success of the last four European Stroke Organization (ESO) Conferences. ESOC is Europe's leading forum for discussing and disseminating the latest advances in stroke care.

Over 1800 abstracts were submitted to ESOC 2018 in Gothenburg. In the large clinical trials sessions, results from 10 major randomized controlled trials (RCTs) were presented, many of which with accompanying high impact publications. Our delegate numbers continue to grow year on year and we are confident ESOC 2019 will be the largest yet.

One of the highlights of ESOC 2018 was the presentation of the "European Action Plan 2018–2030" which builds on the experience and the format of the previous Helsingborg Declarations. This document was written by ESO in cooperation with the patient organization Stroke Alliance for Europe (SAFE), with the involvement of the World Health Organization (WHO).

ESOC 2019 will see presentations of major clinical trials, state-of-the-art talks by renowned clinicians and researchers and receive updates on the latest guidelines. We will be joined by the Italian Stroke Organization.

Fourth Festival of Neuroscience of British Neuroscience Association 2019

Time: April 14–17, 2019

Venue: Dublin, Ireland

Website: http://meetings.bna.org.uk/bna2019/

In April 14–17, 2019, at the Convention Centre Dublin (CCD), the British Neuroscience Association (BNA), in partnership with Neuroscience Ireland (NI) and the British Society for Neuroendocrinology (BSN), will host its fourth Festival of Neuroscience.

The first Festival (BNA2013 in London) set the template for a completely novel forum, where other organizations with an interest in brain research were invited to join the BNA to create a cross-disciplinary and celebratory neuroscience event, bringing together fundamental research with clinical expertise and public engagement as well. Subsequent Festivals (BNA2015 in Edinburgh, BNA2017 in Birmingham) confirmed the success and popularity of this innovation; each attracted 1150–1500 delegates, a remarkable thirty partner organisations have taken part to date, and each has created a genuinely diverse and stimulating mix of neuroscientific interests.