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# 血管内支架结合 GDC 栓塞治疗基底动脉顶端宽颈动脉瘤

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【摘要】 目的 报道应用血管内支架结合电解可脱卸弹簧圈栓塞治疗 1 例未破裂的基底动脉顶端宽颈动脉瘤的经验及结果。方法 将球囊膨胀型血管内支架跨动脉瘤瘤颈部位置入并准确释放后,微导管超选进入动脉瘤内填塞弹簧圈。结果 支架成功的置入,支架近端在基底动脉,远端在大脑后动脉近段,动脉瘤得到次全栓塞(90%以上),载瘤动脉及毗邻的侧支血管保持通畅,患者恢复满意。结论 血管内支架结合弹簧圈栓塞治疗基底动脉顶端宽颈动脉瘤是可行的,支架植入对侧支血管血流无明显影响。

【关键词】 脑动脉瘤;基底动脉;血管内治疗;电解可脱卸弹簧圈;支架

**Intraluminal stenting and endosaccular coiling for a wide-necked basilar tip aneurysm.** LIU Jianmin, HUANG Qinghai, XU Yi, et al. Department of Neurosurgery, Changhai Hospital, Second Military Medical University, Shanghai 200433, China.

【Abstract】 **Objective** To report our experience and result of intraluminal stenting and endosaccular coiling for an unruptured wide-necked basilar tip aneurysm. **Methods** Balloon-expandable stent was deployed across the aneurysmal neck through endovascular approach. Microcatheter was superselectively introduced into aneurysmal sac for GDC packing. **Results** The procedure was technically successful. The proximal segment of stent was deployed in basilar artery, and distal segment in left PCA. The aneurysm was subtotally (more than 90%) occluded with patency of parent artery and adjacent perforating arteries. **Conclusions** Endovascular stenting combined with endosaccular coiling is an alternative method for treating basilar tip wide-necked aneurysm, and deployment of stent yields no adversary effect on flow of branch vessels.

【Key words】 Cerebral aneurysm; Basilar artery; Endovascular therapy; Detachable coil; Stent

由于发出较多功能重要的穿支血管,手术夹闭容易造成损伤而且操作空间较小,因此多数学者认为,与手术夹闭相比血管内治疗,对于基底动脉顶端动脉瘤有着明显的优势<sup>[1]</sup>。但该部位的动脉瘤往往累及基底动脉末端、大脑后动脉起始段,单纯的弹簧圈(GDC)栓塞可导致载瘤动脉受影响并容易复发,特别是宽颈和大型动脉瘤<sup>[2]</sup>。我们首先应用血管内支架结合电解可脱卸弹簧圈(GDC)栓塞治疗 1 例基底动脉顶端的未破裂宽颈动脉瘤,获得了满意的效果。现报道如下。

## 材料与方法

### 一、病例资料

患者男,22 岁,因头痛、头晕 4 个月行头颅 CT 检查提示为鞍区占位,MRA 及普通 DSA 提示为基

底动脉顶端大型动脉瘤转入我院。全脑血管造影检查见基底动脉顶端动脉瘤,椭圆形,体积约 13.5 mm × 11 mm × 11 mm,瘤颈宽约 7 mm,左侧大脑后动脉显影良好,起始段已成瘤颈的一部分,左小脑上动脉自瘤壁发出,右侧大脑后动脉未显影,左侧后交通动脉发育良好(图 1、2)。右侧颈内动脉造影显示后交通动脉发育良好,右大脑后动脉呈胚胎型。测量基底动脉远端直径约 2.4 mm,左大脑后动脉近端直径约 2.2 mm,其余血管未见明显异常。

### 二、手术经过

全麻后,全身肝素化,将 ENVOY 导引导管送至椎动脉内(C1 水平),进行路图(roadmapping)后,在 Prowler-14 微导管保护下将 ATW 微导丝在导引导管内推进至载瘤动脉,将 ATW 导丝远端送至左侧大脑后动脉 P2 段,退出微导管,根据测量的血管直径,选用 BX 支架 2.5 mm × 18 mm,将 BX 支架送至动脉瘤段,瘤颈两端各 5~6 mm(图 3),缓慢充盈

球囊( 10 atm )以释放支架,在透视下撤回球囊,将 ATW 导丝原位保留。选用另一微导管 Rapid Transit 并在其头端蒸汽下塑一小弯,以 Transend EX platinum 微导丝支撑将微导管从支架网眼( 近流入道处 )超选进入动脉瘤内( 图 4 ),先后以 GDC-18 12×30、8×30、8×30 ,GDC-10 7×25、3×8、3×8 栓塞动脉瘤( 图 5 ),术后造影显示载瘤动脉通畅,大脑后动脉和小脑上动脉显影良好,动脉瘤得到次全栓塞( 90 % 以上 ),瘤内仅有少量造影剂残留( 图 6 )。术后给予低分子肝素皮下注射 2 d ,抵克力得( 噻氯吡啶 250 mg ,每天 2 次 ),肠溶阿司匹林片 300 mg ,每天 1 次。

结 果

手术操作较为顺利。支架植入后,远端位于左侧大脑后动脉内( 接近左侧 P1 与 P2 段交界处 ),近端位于基底动脉远端( 左小脑上动脉开口近端 5 mm 左右 )。造影见瘤内仍有造影剂充填,但较术前有明显滞留。先后填入 6 枚 GDC 后,动脉瘤得到次全填

塞( 90 % 以上 )。即刻造影见小脑上动脉、大脑后动脉及基底动脉血流通畅,动脉瘤内仅有少量造影剂残留。术后 24 h 作 CT 检查,未见载瘤动脉及毗邻血管供血区域内的新发梗死灶。

术后患者恢复良好,次日即可正常活动,无任何神经系统功能障碍。并于术后 3 d 出院。

讨 论

因为基底动脉远端以及大脑后动脉起始段( P1 段 )上发出许多重要的穿支血管,分别供应脑干、丘脑、后穿支等,加上手术的可操作空间较小,仍有部分患者基底动脉顶端动脉瘤手术治疗效果较差。相反,由于基底动脉顶端动脉瘤的指向与基底动脉的走行往往相一致,使血管内进路易达到治疗部位,因此,血管内治疗被认为是后循环动脉瘤治疗较佳的选择。Gruber 等<sup>[2]</sup>报道手术夹闭组恢复良好率为 75 % ,而血管内治疗组为 95 % 。随着栓塞材料及栓塞技术的不断发展,后循环动脉瘤血管内治疗的效果得到不断的提高,使手术相关残死率进一步降

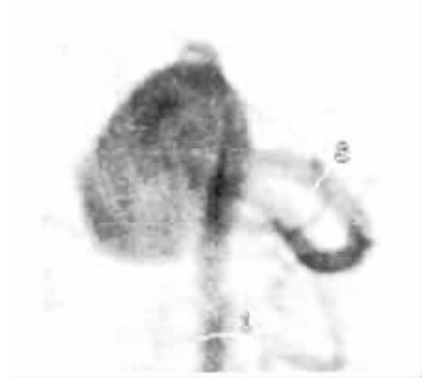


图 1 血管造影显示基底动脉顶端大型动脉瘤



图 2 3D-DSA 示宽颈大型动脉瘤,瘤颈累及 P1 与 BA 远端,SCA 自瘤体上发出( 箭头 )



图 3 支架跨瘤颈放置

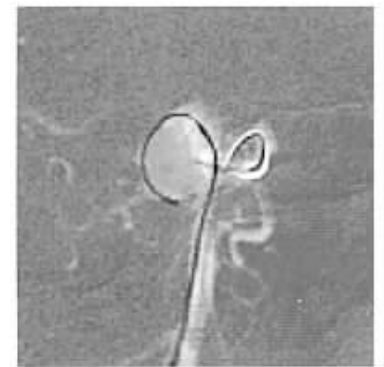


图 4 微导管通过支架网眼进行瘤内弹簧圈栓塞 万方数据

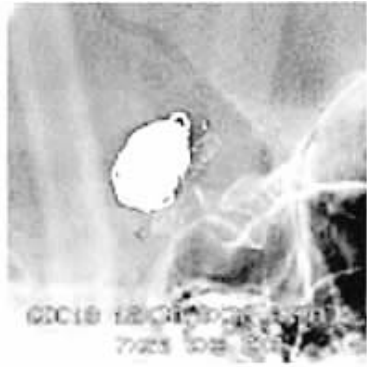
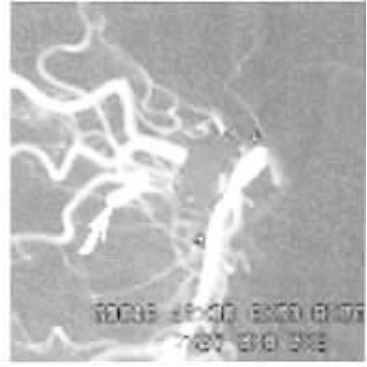


图 5、6 GDC 栓塞后形态及造影,显示载瘤动脉通畅( 箭头所指为 SCA )



低<sup>[3]</sup>。

本例患者动脉瘤位于基底动脉顶端,动脉瘤颈累及大脑后动脉起始段以及基底动脉远端,小脑上动脉自动脉瘤体上发出。由于瘤颈较宽,采用单纯的弹簧圈或暂时性瘤颈辅助技术(remodeling 技术或双导管技术)栓塞治疗难度极大,弹簧圈必将突入载瘤动脉,而在局部形成血栓或血流减慢都将可能导致脑干或丘脑缺血,引起严重的神经功能障碍。Bavinzski 等<sup>[4]</sup>发现该类动脉瘤往往需要多次的血管内栓塞治疗。采用血管内支架将为后续的弹簧圈栓塞治疗提供足够的支撑作用,并且能够保持弹簧圈在瘤内的稳定性以及载瘤动脉的通畅。同时支架的植入改变血流动力学,减少血流对动脉瘤的冲击,促进瘤内血栓并防止弹簧圈紧缩。这也正是本例患者采用血管内支架结合 GDC 栓塞治疗的理论依据。

但目前颅内支架的使用仍有较多的顾虑,血管内支架在冠状动脉以及外周血管疾病的诊治中得到广泛应用,但尚无在基底动脉顶端的应用经验可资借鉴。我们已应用球囊膨胀型冠脉支架成功地治疗了大脑中动脉、椎基底动脉以及颅内颈内动脉处的病变<sup>[5-7]</sup>。虽然支架输送过程中有一定的难度,但所有患者均能成功的将支架输送至治疗部位。而且在大脑中动脉以及基底动脉内植入的支架均覆盖重要的穿支血管开口,但术后的临床及影像学随访均未发现有侧支血管闭塞或神经功能障碍残留。因此,我们认为血管内支架在本例患者中的应用不致引起严重的不良后果。事实上本例患者在术后 3 d 即可出院,而且无任何神经系统功能障碍。

在对弹簧圈栓塞治疗动脉瘤的长期结果影响因素的研究中发现,动脉瘤瘤颈大小与影像学结果之间存在明显的相关性<sup>[8]</sup>。瘤颈越大,即时的栓塞效果以及长期随访结果越差。但 Bavinzski 等<sup>[4]</sup>的结果发现,中期结果与瘤颈大小无明显关系,而与动脉瘤大小有关。无论瘤颈大小,小型动脉瘤的结构最稳定。大型或巨大型动脉瘤无论术后即刻结果如何,长期随访仍有 60% 病例可出现动脉瘤复发或弹簧圈紧缩而需要再次治疗。本例患者无论从瘤颈大小或瘤体大小看,均属难治性动脉瘤。在动物实验以及临床应用研究中均证实,血管内支架植入后通过对动脉瘤内血流动力学的改变,可防止弹簧圈的紧缩并促进瘤内血栓进一步形成。我们的前期结果中也发现血管内支架结合 GDC 栓塞治疗颅内动脉

瘤的短中期随访中可有血栓进一步形成<sup>[5]</sup>。本例患者采用血管内支架技术,除防止弹簧圈突入载瘤动脉并提高动脉瘤的栓塞程度目的外,还期望能使动脉瘤内进一步血栓形成并避免动脉瘤复发,但这有待于进一步的随访。

目前对未破裂基底动脉顶端动脉瘤的自然病史尚未完全清楚。该例患者为未破裂动脉瘤并已出现占位效应,其临床症状可能是发生破裂的先兆。在瘤颈放置血管内支架明显改善瘤内血流动力学(可见造影剂的滞留)的基础上,进一步采用弹簧圈行瘤内栓塞,虽然未能达到即刻致密栓塞的效果,但可促进动脉瘤内血栓形成,逐渐达到治愈目的。对未破裂动脉瘤效果尚佳。而且支架为瘤颈内膜的覆盖提供了可能,支架植入后瘤颈部位的内膜化将明显加速。在基底动脉支架植入后使血流动力学发生改变后将可能避免血流的水锤效应。

本例患者的结果提示,血管内支架结合 GDC 栓塞治疗基底动脉顶端宽颈动脉瘤是安全、可行的,有助于促进动脉瘤解剖愈合,而且对侧支血管内血流无明显影响。

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# 血管内支架结合GDC栓塞治疗基底动脉顶端宽颈动脉瘤

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患者男, 69岁, 诉头痛, 口齿不清、流涎3个月, 伴双下肢无力. CT平扫表现: 桥脑左侧见不规则形团状稍高密度影, 密度不均. 诊断: 桥脑区占位性病变? 出血? 磁共振成像(MRI)平扫表现: 桥前池区桥脑左前方类圆形异常信号影, 边界大部清楚, 约2.4 cm×1.4 cm大小, 其内信号不均, T2WI中心以低信号为主, 见条状高信号, 周围环绕高信号; TIWI不均匀高信号, 其内见条状低信号, 病灶与基底动脉分界不清, 相应层面基底动脉未见显示. 脑干受压变形. 诊断: 脑基底动脉巨大动脉瘤.
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目的 观察结扎新西兰大白兔双侧颈总动脉导致基底动脉分叉处动脉瘤的形成, 探讨该过程中动脉分叉处内皮细胞的形态和功能变化. 方法 将15只新西兰大白兔分成实验组和对照组. 实验组大白兔结扎双侧颈总动脉代偿性地提高基底动脉的血流, 对照组行假手术. 在结扎颈总动脉后的3个月内, 通过多普勒监测基底动脉的血流. 1.5、3个月后, 获取兔基底动脉分叉部, 采用电子显微镜和免疫组织化学的方法观察分叉部内皮细胞的形态和功能. 结果 在第1次手术后的0、1、4、7、14、28、35、42、49、56、70、84 d后, 采用多普勒测量观察到实验组基底动脉血流量较对照组显著增加. 在实验组中, 首次结扎的1.5个月后的β-catenin仅表达在内皮细胞核, 3个月后的兔的基底动脉分叉处出现动脉瘤的早期阶段, 并未见有β-catenin的表达. 电镜下观察到结扎的1.5个月后的内皮细胞由梭形变成柱形, 3个月后的内皮细胞间出现裂缝, 并出现细胞核聚集. 结论 内皮细胞的损伤诱发了脑动脉瘤的形成.
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基底动脉瘤是颅内动脉瘤的一种, 约占颅内动脉瘤的2%. 小的动脉瘤可无临床症状, 大的动脉瘤可表现为占位效应, 导致邻近脑组织受压, 累及脑干、脑神经等重要结构. 基底动脉瘤破裂可导致急性蛛网膜下腔出血、脑内血肿以及出血后血管痉挛所致的脑梗死改变. 因此, 在动脉瘤未破裂之前诊断此病尤为重要. 本文回顾分析我院2000年3月~2003年3月共17例基底动脉瘤诊断, 现总结如下.
4. 外文期刊 [Watanabe, A, Imamura, K, Ishii, R Endosaccular aneurysm occlusion with Guglielmi detachable coils for obstructive hydrocephalus caused by a large basilar tip aneurysm. Case report.](#)  
The authors present the case of a 60-year-old man with obstructive hydrocephalus caused by a large basilar artery tip aneurysm, in whom direct surgical clipping of the aneurysm neck was considered hazardous. After endosaccular aneurysm occlusion, his symptoms (headache, intellectual impairment, and gait disturbance) and ventricular dilation immediately improved without placement of a ventricular shunt. To the authors' knowledge, this is the first case of its kind treated solely endovascularly.
5. 学位论文 王轩 脑淋巴引流阻滞对SAH继发性脑缺血损伤的影响及吡哆醇的缓解作用 2007  
研究背景:  
急性脑血管病是人类三大致死性疾病之一, 其中蛛网膜下腔出血(subarachnoidhemorrhage, SAH)的发病率仅次于脑梗塞和脑出血, 位居第三位. 绝大多数SAH系脑动脉瘤破裂所致, 其病死率高达25%以上, 存活下来的患者约1/3因神经功能缺损而需依赖他人生活, 给社会和家庭带来沉重负担. 再出



血和继发脑缺血是导致 SAH患者严重预后的两个主要并发症。目前对脑动脉瘤的闭塞术和血管内栓塞术取得了很大的进步, SAH 后再出血率已显著降低, 但其总体预后并没有显著改善。因此, 深入探讨SAH后继发脑缺血损伤的机制, 寻找有效防治措施是改善SAH预后的关键环节, 具有十分重要的现实意义。

数十年来, SAH 后脑部主要动脉血管发生的痉挛和管腔狭窄, 即所谓脑血管痉挛 (cerebral vasospasm, CVS) 一直是研究的焦点问题。近年来发现 SAH 后 CVS 出现的时程和严重程度与继发性脑缺血损伤并不呈完全平行的关系, 除CVS外, SAH后脑微血管痉挛和微循环调节功能异常等因素也与继发性脑缺血的发生有关。神经元的死亡可通过坏死 (necrosis) 和凋亡 (apoptosis) 两种不同形式发生。哺乳动物细胞凋亡是受 Bcl-2蛋白家族、凋亡蛋白酶因子 1 (apoptosis protein activating factor, Apaf-1)、caspase (胱天肽酶) 蛋白家族等调控的。caspase 家族成员归属于不同的凋亡通路, 其中 caspase-3 与凋亡密切相关。在线粒体对细胞凋亡的调控中, Bcl-2 家族的蛋白质对线粒体内一些促凋亡因子的释放具有调控功能, 因此在决定细胞生死中也具有十分重要的作用。通过大分子物质示踪等技术在不同种属动物包括人类的研究已经证实, 脑内和蛛网膜下腔中的大分子物质可以被引流入颅外淋巴管和淋巴结。吡哆醇为维生素 B 族化合物, 是机体内一种重要辅酶, 它以 5'-磷酸吡哆醛的形式参与了氨基酸、脂类等代谢和免疫反应等过程。临床上对淋巴滞留性脑病患者采用大剂量吡哆醇或泛酸治疗能明显减轻患者症状, 说明吡哆醇能够改善大分子物质引流, 但其机理不清。SAH继发脑缺血发生后, 大量血浆蛋白等大分子物质可通过受损的血脑屏障进入脑组织中; 由细胞受损而产生的细胞裂解产物和缺血代谢瀑布产生的大量肽类等大分子物质在脑组织中急剧增加。上述大分子物质在脑组织中的积聚直接使神经细胞进一步受损, 或通过脑组织渗透压升高而引起脑水肿甚至脑疝, 导致患者神经功能缺损乃至死亡。这些在蛛网膜下腔和脑组织内积聚的大分子物质被清除的程度如何, 将对 SAH的最终结局产生极为重要的影响。然而, 目前国内外其他学者对SAH继发脑缺血损伤的研究尚没有涉及大分子物质清除这一重要问题。

研究目的:

- 1、进一步阐明脑淋巴引流途径在病理条件下脑组织大分子物质引流和维持脑组织内环境稳定中的重要作用。
- 2、明确SAH后脑淋巴引流途径变化特征, 阐明脑淋巴引流途径在SAH继发脑缺血损伤中起到的内源性保护作用。
- 3、从改善脑内物质的淋巴引流角度寻找有效防治SAH继发脑缺血损伤的药物。

研究方法:

1、选用健康成年 Wistar大鼠, 雌雄不拘, 随机分为正常对照组、SAH 组、SAH+CLB 组、SAH+CLB+ 吡哆醇组、SAH+CLB+ 生理盐水组。采用颈淋巴管结扎和淋巴结摘除法制作大鼠脑淋巴引流阻滞 (CLB) 模型, 应用枕大池二次注血法建立大鼠SAH模型。于模型制作结束后12h内, 监测有关生理指标等变化。于第二次注血后48h在体观察基底动脉 (BA) 管径变化, 测定BA对乙酰胆碱 (Ach) 的反应性, 同时进行组织病理学观察基底动脉形态和管径, 并对脑膜微循环的动态变化进行观察。

2、对上述分组动物, 于术后48h, HE染色观察大脑皮层和海马的形态结构变化; PI 染色观察大脑皮层和海马细胞核的形态结构变化, 采用TUNEL 荧光标记法检测大脑皮层和海马神经元凋亡。取新鲜大脑皮层和海马, 采用SABC免疫荧光技术检测caspase-3 和 Bcl-2 的蛋白表达; 利用RT-PCR技术检测 caspase-3 和Bcl-2的mRNA表达。

研究结果:

1、脑池注血的动物除血压一过性升高外, 动脉血气等生理指标维持在正常范围内; SAH 组、SAH+CLB 组、SAH+CLB+生理盐水组出现ICP升高和脑灌注压 (CPP) 下降; BA 管腔明显痉挛狭窄, 管壁增厚, 且对Ach反应性降低; 脑膜微动、静脉明显痉挛, 血流多呈泥沙样流动, 甚至可见血流郁滞、摆动等明显异常, 以 SAH+CLB组、SAH+CLB+溶媒组更为严重。吡哆醇可减轻CLB对SAH所致ICP升高、CPP下降、BA 痉挛和脑膜微循环异常的变化。

2、(1) HE染色可见SAH组大鼠部分神经元脱落, 皱缩, 核固缩、深染, SAH+CLB组脑组织及毛细血管壁明显水肿, 组织间隙增宽, 内含大量水肿液, 脑血管壁水肿, 神经元部分稀疏, 细胞皱缩、变形, 核染色质致密浓缩、边集, 部分核碎裂, 部分神经元周围有空泡形成。吡哆醇组上述表现较SAH+CLB组有一定程度的减轻。(2) PI染色可见SAH组细胞部分凋亡, 细胞核呈波状或折线样, 部分染色质出现浓缩状态, 个别细胞核的染色质高度凝聚、边缘化, 呈新月形, SAH+CLB 组大鼠有大量细胞凋亡, 且多属于凋亡晚期; 染色质高度凝聚、边缘化, 新月形, 核碎裂, 产生凋亡小体。吡哆醇组凋亡细胞明显减少。(3) TUNEL 法原位细胞凋亡检测显示SAH组散在较多的 TUNEL 阳性细胞, SAH+CLB 组可见大量TUNEL阳性细胞, 凋亡细胞多集中在大脑皮层、海马、基底节区、脉络丛、室管膜等部位, 而以海马、室管膜更为明显。吡哆醇组可见散在 TUNEL 阳性细胞。(4) 免疫荧光技术检测到SAH组有较多的 caspase-3 免疫阳性细胞, SAH+CLB组有大量caspase-3免疫阳性细胞分布, 吡哆醇组 caspase-3 免疫阳性细胞表达较SAH+CLB组减少。SAH组可见较多的 Bcl-2免疫阳性细胞, SAH+CLB组可见少量Bcl-2免疫阳性细胞分布, 吡哆醇组 Bcl-2 免疫阳性细胞表达较SAH+CLB组明显增多。(5) RT-PCR检测结果显示SAH组大脑皮层和海马caspase-3基因mRNA表达明显高于正常对照组, SAH+CLB组caspase-3 基因mRNA表达更高, 吡哆醇组caspase-3基因的mRNA表达则较SAH+CLB 组明显减少。SAH 组 Bcl-2 基因mRNA表达明显低于正常对照组, SAH+CLB组Bcl-2基因mRNA 表达则更低, 吡哆醇组caspase-3基因的mRNA表达则较SAH+CLB组明显增高。

研究结论:

- 1、阻断脑淋巴引流途径可加重SAH后脑血管痉挛和基底动脉的病理变化。
- 2、阻断脑淋巴引流途径可降低SAH后脑部微循环灌注量, 加重微血管痉挛, 降低脑微血管流速。
- 3、吡哆醇干预对脑淋巴引流阻断所致的SAH后脑血管痉挛和微循环异常具有一定缓解作用。
- 4、阻断脑淋巴引流途径将加重SAH所致脑水肿。
- 5、阻断脑淋巴引流途径可以通过下调Bcl-2表达, 上调caspase-3 表达来加重 SAH继发性脑缺血损伤。
- 6、吡哆醇干预对脑淋巴引流阻断所致的SAH继发性脑缺血损伤具有一定缓解作用。

## 6. 外文期刊 [Groff, MW, Adams, DC, Kahn, RA, Kumbhar, UM, Yang, BY, Bederson, JB Adenosine-induced transient asystole for management of a basilar artery aneurysm. Case report.](#)

Advances in anesthetic and surgical management, such as induced deep hypothermic circulatory arrest and application of temporary clips, have improved outcome for patients with basilar artery aneurysms. Nonetheless, these techniques are associated with significant risks. The authors report a case in which three transient periods of cardiac asystole were induced during basilar artery aneurysm surgery. Adenosine-induced asystole facilitated the safe clipping of the aneurysm by producing consistent periods of profound hypotension and collapse of the aneurysm without the need for temporary clipping. This technique provided unencumbered identification of perforating arteries, precise definition of the local anatomy, and an ideal environment for the safe placement of the aneurysm clip.

## 7. 外文期刊 [Lanzino, G, Wakhloo, AK, Fessler, RD, Hartney, ML, Guterman, LR, Hopkins, LN Efficacy and current limitations of intravascular stents for intracranial internal carotid, vertebral, and basilar artery aneurysms.](#)

OBJECT: Results of previous in vitro and in vivo experimental studies have suggested that placement of a porous stent within the parent artery across the aneurysm neck may hemodynamically uncouple the aneurysm from the parent vessel, leading to thrombosis of the aneurysm. For complex wide-necked aneurysms, a stent may also aid packing of the aneurysm with Guglielmi detachable coils (GDCs) by acting as a rigid scaffold that prevents coil herniation into the parent vessel. Recently, improved stent system delivery technology has allowed access to the tortuous vascular segments of the intracranial system. The authors report here on the use of intracranial stents to treat aneurysms involving different segments of the internal carotid artery (ICA), the vertebral artery (VA), and the basilar artery (BA). METHODS: Ten patients with intracranial aneurysms located at ICA segments (one petrous, two cavernous, and three paraclinoid aneurysms), the VA proximal to the posterior inferior cerebellar artery origin (one aneurysm), or the BA trunk (three aneurysms) were treated since January 1998. In eight patients, stent placement across the aneurysm neck was followed (immediately in four patients and at a separate procedure in the remaining four) by coil placement in the aneurysm, accomplished via a microcatheter through the stent mesh. In two patients, wide-necked aneurysms (one partially thrombosed BA trunk aneurysm and one paraclinoid segment aneurysm) were treated solely by stent placement; coil placement may follow later if necessary. No permanent periprocedural complications occurred and, at follow-up examination, no patient was found to have suffered symptoms referable to aneurysm growth or

thromboembolic complications. Greater than 90% aneurysm occlusion was achieved in the eight patients treated by stent and coil placement as demonstrated on immediate postprocedural angiograms. Follow-up angiographic studies performed in six patients at least 3 months later (range 3-14 months).

8. 外文期刊 [Roebroek, RM, ter, Berg-JW, van, der-Laan-RT De novo formation of a megadolichobasilar artery associated with bilateral fusiform aneurysms of the internal carotid artery \(letter\)](#)

9. 外文期刊 [Nagasawa, S, Kawanishi, M, Tada, Y, Kawabata, S, Ohta, T Simulation of therapeutic parent artery occlusion for basilar head aneurysms. Hemodynamic effect of occlusion sites and diameters of collateral arteries.](#)

We simulated parent artery occlusion therapy for basilar head aneurysms to elucidate the hemodynamic changes induced by different occlusion sites and diameters of the posterior communicating arteries (PCoM) as collateral pathways. A vascular model of the vertebrobasilar system with a basilar head aneurysm was constructed. Four types of occlusion were simulated: Basilar artery occlusion distal to (Type A), between (Type B) and proximal to (Type C) the superior cerebellar arteries, and bilateral vertebral artery occlusion (Type D). Glycerol solution was perfused into the model, and the half-life of the dye injected into the aneurysm was calculated and regarded as an index of stagnant flow in the aneurysm. The half-life was increased significantly and nonlinearly after parent artery occlusion, depending on the occlusion site and the ratio of two PCoM diameters (diameter ratio). Intra-aneurysmal stagnation developed markedly in Type A and Type B in the diameter ratio higher than 0.70 and considerably in Type C in the ratio higher than 0.80. Additional P1 occlusion of the posterior cerebral artery enhanced the stagnation in Type A and B. Since the results are consistent with the published clinical data, the simulation study will be useful for speculating the efficacy of the therapeutic occlusion.

10. 外文期刊 [Hosoya, T, Adachi, M, Yamaguchi, K, Haku, T, Kayama, T, Kato, T Clinical and neuroradiological features of intracranial vertebrobasilar artery dissection.](#)

BACKGROUND AND PURPOSE: We sought to determine the clinical and neuroradiological features of intracranial vertebrobasilar artery dissection. METHODS: The clinical features and MR findings of 31 patients (20 men and 11 women) with intracranial vertebrobasilar artery dissections confirmed by vertebral angiography were analyzed retrospectively. The vertebral angiography revealed the double lumen sign in 11 patients (13 arteries) and the pearl and string sign in 20 patients (28 arteries). RESULTS: The patients ranged in age from 25 to 82 years (mean, 54.8 years). Clinical symptoms due to ischemic cerebellar and/or brain stem lesions were common, but in 3 cases the dissections were discovered incidentally while an unrelated disorder was investigated. Headache, which has been emphasized as the only specific clinical sign of vertebrobasilar artery dissection, was found in 55% of the patients. Intramural hematoma on T1-weighted images has been emphasized as a specific MR finding. The positive rate of intramural hematoma was 32%. Double lumen on 3-dimensional (3-D) spoiled gradient-recalled acquisition (SPGR) images after the injection of contrast medium was identified in 87% of the patients. The 3-D SPGR imaging method is considered useful for the screening of vertebrobasilar artery dissection. CONCLUSIONS: Intracranial vertebrobasilar artery dissection is probably much more frequent than previously considered. Such patients may present not only minor symptoms. Neuroradiological screening for posterior circulation requires MR examinations, including contrast-enhanced 3-D SPGR. Angiography may be necessary for the definite diagnosis of intracranial vertebrobasilar artery dissection because the sensitivity of the finding of intramural hematoma is not satisfactory.

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