Autopsy Findings After Intracranial Thrombectomy for Acute Ischemic Stroke
A Clinicopathologic Study of 5 Patients
Nicole S. Yin, BS; Sebastian Benavides, BS; Sidney Starkman, MD; David S. Liebeskind, MD; Jeffrey A. Saver, MD; Noriko Salamon, MD, PhD; Reza Jahan, MD; Gary R. Duckwiler, MD; Satoshi Tateshima, MD; Fernando Vinuela, MD; Paul M. Vespa, MD; Dennis J. Chute, MD; Harry V. Vinters, MD, FCAP, FRCP(C)

Background and Purpose—Endovascular thrombectomy is an increasingly used treatment for arterial occlusion in acute stroke. Various devices (including most extensively the Mechanical Embolus Removal in Cerebral Ischemia [MERCI] Retriever device) have been used for this.

Methods—We review the neuropathologic findings in 5 patients (age range, 59 to 87 years) who died acutely or as late as 38 days after procedures using the MERCI (4 patients) and Penumbra (1 patient) devices were carried out to remove thromboemboli from the middle cerebral artery. Partial recanalization was achieved by thrombectomy in all 5 patients.

Results—All patients showed extensive cerebral infarcts, 3 of 5 with clinical hemorrhagic transformations of the infarct or frank intraparenchymal hemorrhage after thrombectomy; in 1 case, this was judged to be at least partly on the basis of concomitant hypertensive microvascular disease. With 1 exception, basal arteries examined in detail by immunohistochemistry showed prominent, although usually nonocclusive (and generally nonulcerated), atheromata, often with significant luminal stenosis. One patient showed a subintimal dissection with resultant occlusion of the middle cerebral artery.

Conclusions—In this highly selected group of patients, the vascular pathological abnormalities affecting basal arteries were variable, but complicated atherosclerosis was a common finding. Extensive irreversible brain necrosis before therapeutic procedures may have contributed to deaths. (Stroke. 2010;41:938-947.)

Key Words: cerebral infarct ■ intracerebral hemorrhage ■ MERCI device ■ stroke recovery ■ thrombectomy

Endovascular thrombectomy procedures are frequently performed to extract thromboemboli that have occluded major branches of the circle of Willis and proximal arteries emerging from it.1-7 The design, preclinical testing, and practical uses of various devices used to carry out the procedure have been described in several publications as have treatment outcomes.8-15 The retrieved thromboemboli have been carefully studied for their cellular and biochemical components with the hope that the results of such studies may shed light on why thromboembolectomy is effective in only some patients.16

Autopsy findings have not previously been described in patients who have undergone thrombectomy. Analysis of outcomes after thrombectomy shows that mortality is substantial in both those in whom recanalization of an occluded artery has been achieved (mortality rates of 20% to 40%) and those in whom recanalization is not achieved (60% to 80%).16 Patients who undergo autopsy (after any therapeutic procedure) clearly represent a highly selected population with the worst possible outcome. We undertook this study to investigate vascular and brain parenchymal abnormalities in 5 patients who died after thrombectomy.

Subjects and Methods
Subjects included in the study were 5 individuals (4 females, 1 male with an age range of 59 to 87 years; mean age, 75.6 years) who died and underwent autopsy after thrombectomy after acute stroke. The indication for thrombectomy was to restore blood flow to the neurovasculature by removing thrombus. The attending physician used his judgment to offer the treatment option with the greatest potential for a beneficial outcome.
The Mechanical Embolus Removal in Cerebral Ischemia (MERCI) Retriever device had been used in 4 individuals, whereas the fifth patient was treated with a Penumbra device. Patient 2 had an angioplasty performed in the affected left middle cerebral artery (MCA) M1 segment of the artery, after thrombectomy, whereas Patient 4 was given intravenous tissue plasminogen activator in conjunction with thrombectomy. The procedures were performed by G.R.D. (Patients 1 and 2), S.T. (Patients 3 and 5), and F.V. (Patient 4). All physicians were involved since the inception of its use in humans and are highly trained and experienced with the device and were involved in the pivotal trials leading to US Food and Drug Administration clearance of the device.

All autopsies were performed over an 89-month period (May 2001 through October 2008). Patient demographics and details of relevant medications and follow-up are provided in Table 1.

### Table 1. Clinical and Pathological Summary of Study Patients

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<tr>
<th>Patient ID</th>
<th>Patient 1</th>
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<th>Patient 3</th>
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<td>Before thrombectomy device</td>
<td>Right MCA and ACA ischemia arterial thrombosis</td>
<td>Left cerebral hemisphere ischemia and occlusion at left M1 segment of the left MCA</td>
<td>Probable cardiogenic embolic occlusion at left MCA bifurcation</td>
<td>Probable embolus secondary to atrial fibrillation</td>
<td>Probable rupture of left ICA plaque with thrombus formation</td>
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<td>MERCI device in right MCA and ACA</td>
<td>MERCI device in left MCA</td>
<td>MERCI device in right MCA</td>
<td>MERCI device and IA tPA in left MCA</td>
<td>Penumbra System in left ICA</td>
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<td>Cerebral edema and herniation</td>
<td>Six thrombus components removed followed by angioplasty of the M1 segment of the left MCA</td>
<td>Developed edema with midline shift and subarachnoid hemorrhage</td>
<td>Massive right MCA infarct and right basal ganglia hemorrhage</td>
<td>Massive left cerebral hemispheric parietal hematoma</td>
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<td>Death 6 days after procedure</td>
<td>Death 1 day after procedure</td>
<td>Hemicraniectomy 11 days after procedure</td>
<td>Bradycardia</td>
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<td>Death 4 days after procedure</td>
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<td>Infarct at autopsy</td>
<td>Massive right cerebral hemispheric infarct in right inferior frontal cortex and temporal lobe</td>
<td>Left cerebral hemisphere ischemic infarct, focally hemorrhagic with marked edema</td>
<td>Massive right cerebral hemispheric infarct</td>
<td>Left cerebral hemispheric infarct: intracerebral hemorrhage with intraventricular extension</td>
<td>Infarct with hemorrhagic transformation in left frontoparietal region</td>
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<td>Cerebral arterial pathology</td>
<td>Severe, calcified, and complicated atheroma in right MCA</td>
<td>Thromboembolus: early organization composed of fibrin/platelet material</td>
<td>Well organized thromboembolus</td>
<td>Significant plaque with fibrous SMA cap in the MCA</td>
<td>Complicated atherosclerotic plaques containing SMA fibrous caps and prominent plaques in left and right MCA and BA</td>
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<td>Circle of Willis: focal atherosclerosis particularly in right proximal MCA</td>
<td>Large atheromatous plaques in left and right vertebral, basilar, posterior and middle cerebral arteries</td>
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<td>Subintimal dissection of the internal elastic lamina in a segment of the right MCA</td>
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NIHSS indicates National Institutes of Health Stroke Scale; Meds, medications; ACA, anterior cerebral artery; TICI, Thrombolysis in Cerebral Ischemia; IA, intra-arterial; tPA, tissue plasminogen activator; ICA, internal carotid artery; BA, basilar artery.
their presenting stroke are provided in the Table. All individuals had presented with a large left or right cerebral hemispheric (MCA territory) infarct; 3 were thought to have originated from a cardiogenic embolus, 1 from in situ thrombosis in the right MCA, and in our most recently encountered patient, there was significant atherosclerosis of the cerebral arteries as well as multiple potential sources of cardiogenic emboli. The most recently studied individual had developed symptoms during an airplane flight, necessitating an emergency landing and transfer to the hospital. Presenting symptoms and medications patients were receiving are listed. All subjects had undergone thrombectomy with heparin flush (5000 u/L) delivered through the guide catheter during the procedure after having experienced their “stroke.” Groin puncture was median 5 hours 10 minutes after the last known well time (range 1 hour 15 minutes to 7 hours 15 minutes).

End-of-procedure Thrombolysis in Cerebral Infarction score was 2 in all patients. Patients survived over a range of 1 to 38 days after thrombectomy. One patient (Patient 3, the longest postthrombectomy survivor in this series) had undergone hemi-craniectomy 12 days after the stroke to relieve rising intracranial pressure.

In all autopsies, brains were removed and processed using conventional techniques. Complete autopsy was carried out on 4 of the 5 patients. Before sectioning of each brain, the circle of Willis and its major branches were dissected from its base and cross-sectioned. Multiple segments from major branches of the circle of Willis were studied by routine histology and/or immunohistochemistry (see subsequently). Digital images of fixed brain slices were retained.

Routine paraffin sections of arteries from all cases were stained with hematoxylin and eosin as well as the elastica van Gieson (EVG)
method (Diagnostic Biosystems, Pleasanton, Calif) to highlight elastica and intimal thickening. Immunoperoxidase immunohistochemistry was performed on sections from selected blocks using primary antibodies to smooth muscle actin (SMA, to show smooth muscle cells) and CD68 (to demonstrate macrophages). Primary antibodies for these studies were from Dako (Carpinteria, Calif) and used at a primary dilution of 1:100 (both SMA and CD68). Digital photographs of EVG-stained arteries were subject to the computer programs Adobe Photoshop CS2 Version 9.0 and Image J 1.38X allowing for quantification of percentage stenosis of atherosclerotic segments.

Results

Clinicopathologic features for all patients are summarized in the Table. Figures representing radiographic and neuropathologic findings are intentionally presented sequentially from the earliest patient encountered to the most recent using the numbering system given in the Table. The atheromatous plaque in all examined arteries showed varying collections of SMA-immunoreactive smooth muscle cells and CD68-immunoreactive macrophages, smooth muscle cells often forming a subintimal cap over collections of macrophages. None showed ulceration of the plaque or intraplaque hemorrhage (Figures 1, 3, 6, 8C–E, and 11). Lumina of MCA segments examined from Patients 2, 4, and 5 were patent. Atheroemboli were not found in the distal circulation of any case, although rare small arteries showed occlusion by platelet–fibrin thromboemboli. The estimated degree of arterial stenosis varied from virtually none to 75%, mean 45%. Patient 3, thought to have experienced a cardiogenic embolus (Figures 5 and 6), showed advanced organization of the right MCA thromboembolus with ingrowth of smooth muscle cells into the occlusive material, consistent with extended (38-day) survival. Figures 4, 7, and 9 illustrate relevant neuroimaging studies (pre- and/or postthrombectomy) for Patients 3, 4, and 5, respectively; Figures 2, 3A, 5, 8A–B, and 10 illustrate brain parenchymal and vascular abnormalities. Patient 1 was the only individual to have sustained a subintimal dissection of the right MCA (Figure 1). This dissection occurred over a comparatively nonatherosclerotic portion of that artery. Arterial segments from 4 of the 5 patients (the exception being Patient 3) showed significant complicated atherosclerosis with varying degrees of atherosclerotic stenosis of arterial segments that had been traversed during thrombectomy.

All brains examined showed hemispheric infarcts, which were consistent in appearance (grossly and microscopically) with the clinical onset of stroke symptoms and corresponding neuroimaging studies (Figures 2, 4, 5, and 7 through 10). In Patients 1, 2, and 4, the infarcts were associated with massive, fatal cerebral edema. In Patient 3 (who survived the procedure by >1 month), edema had subsided and the right cerebral hemispheric infarct showed cystic cavitation. In Patient 5, an extensive, substantially hemorrhagic cortical infarct was noted in the left cerebral hemisphere (Figure 10), whereas an acute hematoma in the left basal ganglia was attributed to significant hypertensive arteriopathy.

Major general autopsy findings included the following: Patient 1 had neurogenic pulmonary edema and pleural effusions; Patient 3 had moderate coronary artery atherosclerosis, evidence of ischemic cardiomyopathy with a left ventricular aneurysm, and diffuse alveolar damage; Patient 4 had coronary artery disease with past stenting of several arteries and atherosclerosis of the abdominal aorta; Patient 5 had severe generalized and coronary atherosclerosis with ischemic changes in the myocardium, a history of bioprosthetic aortic valve replacement, and cardiomegaly with left ventricular and biatrial dilatation. The cerebral abnormalities, especially infarct with edema and/or cerebral hemorrhage,
were deemed major factors contributing to death in all cases, but especially in 3 individuals.

**Discussion**

To our knowledge, this is the first report describing the vascular and neuropathologic abnormalities in a group of patients who died after endovascular thrombectomy procedures, all carried out to extract thromboemboli from MCA segments. The authors recognize that this small series represents a highly select group of patients who contrast in striking terms with the numerous patients who have received benefit from innovative thromboembolectomy procedures.1–15 In a large multicenter trial of 164 patients who underwent thrombectomy within 8 hours of stroke symptom onset, 57.3% of treated patients showed successful recanalization in “treatable” arteries and 69.5% recanalized vessels after adjunctive therapy.18 Favorable clinical outcomes were observed in 36% with an overall mortality rate of 34%. Outcome may to some extent be dependent on the nature of the material occluding an affected artery, a detailed morphological analysis of which has previously been published.18 Further detailed analysis may well shed light on molecular components within a thromboembolus that render a patient more or less likely to have a good outcome.

Systemic disease, usually complications of atherosclerosis involving the coronary and other arteries (including the aorta) and pulmonary lesions, almost certainly contributed to death in all patients. In at least 3 of 5 patients, large MCA territory infarcts with associated mass effect from edema were judged to have significantly contributed to fatal outcomes. In every case, microscopic features of cerebral infarcts were consistent with their having occurred at the time of symptom onset. These 5 patients did not survive their strokes; 3 developed hemorrhages after the procedure. Patient 2 had a small subarachnoid hemorrhage, likely caused by the procedure. Patient 4 had a large hemorrhage. In Patients 4 and 5, significant parenchymal hemorrhage was superimposed on ischemic lesions in the same cerebral hemisphere. In Patient 4, the parenchymal hemorrhage

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**Figure 3.** Patient 2. A, Circle of Willis dissected from base of the brain. Note patchy, focally accentuated atherosclerosis in major branches of the circle of Willis. B, Transverse section of the moderately atherosclerotic left MCA bifurcation (EVG stain). C, Section of left MCA bifurcation, parallel to that shown in Figure 3B, immunostained with primary antibody to SMA. Note “cap” composed of SMA-immunoreactive cells overlying atheromatous material. D–E, Parallel sections of the M2 segment of the left MCA stained with EVG (D) and primary antibodies to SMA (E). Arrows (in E) indicate 2 dense layers composed of smooth muscle cells. F, Section of basilar artery from the same patient immunostained with primary antibodies to CD68; arrow highlights dense accumulation of macrophages in the atheroma.

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**Figure 4.** Patient 3. A, T2 axial MRI scan (obtained 3 weeks postthrombectomy) shows right-sided craniectomy and extensive right MCA territory infarct. B, Gradient recalled echo axial MRI image shows an extensive right MCA territory infarct and a right basal ganglia hemorrhage (arrows).
hemorrhage had extended into the lateral ventricle, parenchymal hematomas Berger Type 2 and a subarachnoid hemorrhage, due to the tissue plasminogen activator, whereas Patient 5 developed a hemorrhagic infarct Type 1, unlikely due to the procedure. The ganglionic hematoma observed in this patient was deemed in part to be on the basis of concomitant hypertensive cerebral microvascular disease. The first patient in our series had experienced a subintimal arterial dissection in a severely atherosclerotic segment of her MCA, although the dissection occurred in a portion of the arterial wall that did not show significant atheroma. The immediate causes of death for Patients 1 and 5 were the families’ decisions to withdraw care. All care was withdrawn except for the ventilator for Patient 3, and comfort care protocols were initiated for the remaining patients.

In addition to the 1 patient who appeared to have a procedure-related MCA dissection, 1 other (the longest survivor among the 5) showed complete occlusion of the MCA segment with advanced organization of the thromboembolus, consistent with its having developed at or around the time of the presenting stroke and the endovascular procedure. Other arterial segments examined in detail (from Patients 1, 2, 4, and 5) showed variably advanced, usually complicated atheromata (confirmed by immunohistochemistry) with stenosis, although no evidence of acute intraplaque hemorrhage or intimal ulceration. The lumens in such vessels were patent. Atheroemboli, sometimes encountered in the distal arteries of patients with severe atherosclerosis in the cervical arteries, were not found in any of these patients despite extensive sampling of brain parenchyma.

Figure 5. Patient 3. A–C, Panels represent selected coronal slices of a fixed brain showing the right MCA territory infarct undergoing cystic cavitation (arrows in B–C). Arrow in A shows extension of infarct into the right basal ganglia with hemorrhagic transformation. D, A “saddle thromboembolus” in the right MCA bifurcation (arrows).

Figure 6. Patient 3. Representative cross-section of right MCA (A from section stained with hematoxylin and eosin; B from a parallel section immunostained with primary antibody to SMA) showing a well-organized thromboembolus in a relatively nonatherosclerotic artery. B highlights ingrowth of smooth muscle cells into the thromboembolus.
The presence of substantial intracranial atherosclerosis in these cases has important implications for patient selection and further device development. Thrombectomy devices are designed to retrieve or aspirate thrombi from relatively normal arterial beds rather than severely atherosclerotic arteries. The loops of the MERCI Retriever can snag and unwind on fixed atheroma with risk of impaired thrombus removal, local vessel dissection, and remote vessel traction injury. The Penumbra system is too large and bulky to pass easily through stenotic atherosclerotic arteries.

Figure 7. Patient 4. A, Axial sections of cerebral hemispheres taken 3 hours after stroke symptom onset but immediately before thrombectomy. Diffusion-weighted images show acute infarct in the left MCA territory (panels at left). The perfusion-weighted image abnormalities (right panels) are considerably larger than those shown on the diffusion-weighted images. B, Large left parieto-occipital intraparenchymal hematoma (postthromboembolectomy) extending into the lateral ventricles.
Figure 8. Patient 4. A–B, Coronal sections of a fixed brain show a large left MCA territory subacute infarct with associated edema and left to right shift of midline structures. There is also extensive intraventricular hemorrhage, especially in the right lateral ventricle. C–D, Cross-sections of the left MCA (C stained with EVG, D stained immunohistochemically using primary antibody to SMA) show relatively mild atheroma with a smooth muscle cell cap (arrows in D). At another level (E), the MCA shows significant atheroma with estimated 55% to 60% stenosis of the lumen. None of the lumina shows a thromboembolus. (E from a section stained with EVG.)

Figure 9. A, Patient 5. T2 and fluid-attenuated inversion recovery images (obtained at the time of the patient's admission to hospital, 1:00 PM, prethrombectomy) show old infarct in the right occipital lobe. B, Fluid-attenuated inversion recovery images obtained prethrombectomy (at left) and 7 hours later (at right, approximately 5 hours postthrombectomy) show a new infarct in the left MCA territory.
segments, limiting its ability to aspirate more distally located thrombi. When the target occlusive lesion is known to consist substantially of in situ atherosclerosis, primary angioplasty and stenting—cracking, stretching, and maintaining radial force on the plaque—could be a treatment option rather than embolectomy.23 In the 5 cases in this series, the interventional team believed at the time of treatment that the vascular occlusions were largely thrombi with only minimal in situ atherosclerosis. All patients had normal neck arteries according to angiography. Patients 2, 4, and 5 had atrial fibrillation and came in with a normal international normalized ratio, leading to the presumption that the source of the clots was cardiac. Patient 5 also had a heart valve replacement, increasing the likelihood of a cardiac embolus. A workup for Patient 1 was not done due to the severity of the patient’s stroke, but she had a history of severe ischemic heart disease and previous myocardial infarctions, indicating that the source of clot was presumably cardiac. Patient 3 had ischemic cardiomyopathy with congestive heart failure; her stroke was likely caused by a cardiogenic embolus. The untoward outcome in these 4 cases that actually harbored moderate or extensive local atherosclerosis highlights the need for improved methods to diagnose target occlusion composition pretreatment such as making intravascular ultrasound catheter tips small and navigable enough for deployment in the intracranial circulation.

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Figure 10. Patient 5. A, Axial section of a fixed left cerebral hemisphere shows a large acute hematoma in the left basal ganglia (arrows), the site of severe hypertensive microvascular disease. B (left cerebral hemisphere), E extensive left MCA territory subacute infarct. Arrow indicates a prominently hemorrhagic segment of the infarct. Note relative preservation of the left ACA territory (bottom right of the photograph).

Figure 11. Patient 5. Parallel segments of the left MCA (A–D) stained with EVG (A, C) and primary antibody to SMA (B, D) show extensive atheroma of the artery.
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Disclosures

None.

References

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Abstract

急性缺血性卒中颅内取栓术后尸检发现：
5例患者的临床病理学研究

Autopsy Findings After Intracranial Thrombectomy for Acute Ischemic Stroke: A Clinicopathologic Study of 5 Patients

Nicole S. Yin, BS; Sebastian Benavides, BS; Sidney Starkman, MD; David S. Liebeskind, MD; Jeffrey A. Saver, MD; Noriko Salamon, MD, PhD; Reza Jahan, MD; Gary R. Duckwiler, MD; Satoshi Tateshima, MD; Fernando Vinuela, MD; Paul M. Vespa, MD; Dennis J. Chute, MD; Harry V. Vinters, MD, FCAP, FRCP(C)

背景和目的：血管内取栓术越来越多地被用来治疗急性卒中的动脉闭塞。各种装置（包括广泛使用的脑缺血机械取栓装置 [MERCI]）已在此方法中应用。

方法：回顾5例患者（年龄范围59至87岁）的神经病理学发现，运用MERCI（4例患者）和Penumbra（1例患者）装置从大脑中动脉取出血栓的操作后，他们在短时间内或长达38天后死亡。所有5例患者通过取栓实现了局部血管的再通。

结果：所有患者均显示出大面积的脑梗死，3例取栓后伴有梗死处出血转化或脑实质出血；其中1例出血的产生据推测至少部分是基于合并的高血压微血管病。除1例外，通过脑底部动脉免疫组织化学的仔细检查显示，突出的但通常是非闭塞的（一般无溃疡）粥样瘤，常伴有明显的管腔狭窄。1例患者显示有血管内膜夹层及由此产生的大脑中动脉闭塞。

结论：在这组高选择性的患者中，影响脑底部动脉的血管病理学异常是多样的，但复合性动脉粥样硬化是最普遍的发现。治疗操作前广泛的不可逆的脑坏死也可能导致死亡。

关键词：脑梗死，大脑内出血，MERCI装置，卒中恢复，取栓术

(Stroke. 2010;41;938-947. 林森 译 张苏明 校)

表 患者的临床和病理小结

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<td>尸检中梗死发现</td>
<td>左侧大脑半球外侧皮质下和颞叶大面积脑梗死</td>
<td>左侧大脑半球脑梗死，局灶出血伴明显水肿</td>
<td>左侧大脑半球大面积脑梗死</td>
<td>左侧大脑半球大面积脑梗死</td>
<td>左侧颞叶脑梗死伴出血转化</td>
</tr>
<tr>
<td>大脑动脉病理改变</td>
<td>左侧 MCA 严重钙化的复合性闭塞</td>
<td>双侧 MCA 和 BA 存在明显的伴 SMA 分支的复合性动脉粥样硬化病变</td>
<td>组织结构完整的血栓微栓塞</td>
<td>大脑中动脉存在伴 SMA 纤维帽的明显斑块</td>
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</tr>
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表中数据为文献摘要中的部分内容。