ARKINSON’S disease (PD) is a common neurodegenerative disease with a prevalence of 85 to 187 cases per 100,000 population. Stereotactic radiofrequency (RF) pallidotomy is effective in alleviating the symptoms of idiopathic (I)PD, but the long-term efficacy as well as the safety of this procedure remain to be fully determined. Radiofrequency has long been favored as the technique of choice for functional lesion making in pallidotomy. It consistently generates well-circumscribed lesions and allows electrical stimulation for localization and monitoring of temperature and impedance during the procedure. The advent of magnetic resonance (MR) imaging has allowed better visualization of targets in the basal ganglia without the need of invasive techniques such as ventriculography. The superior soft-tissue contrast produced by MR imaging, with delineation of blood vessels and multiplanar imaging capability, allows planning of safer trajectories for RF electrodes. High rates of success have recently been reported with the use of stereotactic RF pallidotomy and MR imaging localization in reducing bradykinesia, rigidity, and tremor, with low morbidity resulting. The histopathological and MR imaging appearance as well as the parameters of localization and lesioning for RF pallidotomies have been reported. However, delayed infarction after RF pallidotomy has not been previously described. We report on three patients in our series who suffered MR imaging–documented delayed infarctions adjacent to the RF lesions.

**Delayed internal capsule infarctions following radiofrequency pallidotomy**

**Report of three cases**

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The authors report on a series of patients with idiopathic Parkinson’s disease (IPD) who underwent stereotactic radiofrequency (RF) pallidotomies, three of whom suffered delayed postoperative strokes. These three belonged to a group consisting of 42 patients with medically intractable IPD in whom 50 pallidotomies were performed. All three patients had significant previous vascular disease and were in a high-risk group for cerebral infarction. A postoperative magnetic resonance (MR) image was obtained immediately after the pallidotomy was performed to document the placement of the RF lesion and to rule out any hematoma. The delayed strokes occurred on postoperative Days 10, 51, and 117 in patients with previous vascular disease (Group 1, 11 patients). No strokes occurred in the group with the vascular disease risk factor (Group 2, 11 patients) or in the group with no risk factors for vascular disease (Group 3, 20 patients). This observation is statistically significant (p < 0.05). The T2-weighted MR images showed the lesions as high-intensity signals extending to the posterior limb of the internal capsule ipsilateral to the pallidotomy site. The poststroke T1-weighted images obtained in two patients showed persistent contrast enhancement of the RF lesion and no enhancement around the stroke lesion. Clinically and radiographically, these discrete new lesions represent delayed infarctions, suggesting that RF lesioning can induce delayed injury in adjacent tissue. Patients with previously identified vasculopathy may be at risk for delayed capsular infarction following RF pallidotomy.

**KEY WORDS • Parkinson’s disease • stroke • magnetic resonance imaging • stereotactic surgery • radiofrequency lesion**

**Case Material and Background**

**Patient Population**

Three patients with delayed stroke were part of a series of 42 patients who underwent a total of 50 pallidotomies that were consecutively performed between November 1993 and July 1996 at the University of California at Los Angeles (UCLA) Medical Center (Table 1). Thirty-two men and 10 women comprised the patient series. Their ages at the time of pallidotomy ranged from 34 to 82 years, with a mean age of 62 years. Retrospectively, these 42 patients were placed in one of three groups depending on their vascular disease history and risk factors. Group 1 included 11 patients who had a documented history of...
vascular disease. This definition included symptomatic coronary artery disease, peripheral vascular disease, and cerebrovascular disease, as well as clinically silent but radiographically significant disease. For instance, nine of the 11 patients were deemed to have had vascular disease based on clinically silent infarctions or deep white matter disease that was noted incidentally in the official report of the preoperative MR image. (All readings for the 42 patients reviewed had been independently dictated by UCLA staff neuroradiologists prior to this study.) Group 2 consisted of 11 patients who had known vascular disease risk factors such as hypertension, diabetes, alcohol abuse, and tobacco use, but who were without symptomatic or radiographically significantly disease (alcohol abuse was defined as more than six drinks per day, and tobacco use was deemed significant if the patient was an active smoker or had smoked regularly within 5 years of the operation). The 20 patients in Group 3 had no vascular risk factors and no history of vascular disease (Table 2).

**Pallidotomy Technique**

The target coordinates were calculated for the Leksell frame (Elekta Instruments, Inc., Atlanta, GA) from 1-mm sagittal, coronal, and axial MR slices. A midsagittal plane was used to determine the anterior and posterior commissures, and then coronal and axial images were obtained perpendicular and parallel to this anterior–posterior commissure line. The pallidotomy target was defined on the coronal slice passing through the mammillary body and was just lateral to the internal capsule and above the lateral fissure. The appropriateness of this target was confirmed by comparing it to the point defined by Laitinen, et al.,

<table>
<thead>
<tr>
<th>Pallidotomy Site(s)</th>
<th>No. of Patients</th>
<th>No. of Pallidotomies</th>
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<tr>
<td>rt side only</td>
<td>13</td>
<td>13</td>
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<td>21</td>
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<td>rt side × 2</td>
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<tr>
<td>lt side × 2</td>
<td>1*</td>
<td>1</td>
</tr>
<tr>
<td>bilat in 1 op</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>bilat in 2 ops</td>
<td>3†</td>
<td>6</td>
</tr>
<tr>
<td>bilat in 2 ops &amp; rt side</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>total</td>
<td>42</td>
<td>50</td>
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* First pallidotomy was performed at another hospital.
† Performed 6 to 9 months apart.

The lesions were made by means of the same RF generator and electrode used for electrical stimulation. Before the final lesion was made, a thorough neurological examination was performed by the neurologist using a probe set at a temperature of 50°C. This examination included testing of the visual field, speech, motor, and the extrapyramidal system. When no undesired effects were observed, the temperature was increased to 80°C and maintained for 60 seconds to make a permanent lesion. As many as three lesions were made at 2-mm intervals in the trajectory of the electrode to produce an oval lesion.

**Case Reports**

**Case 1**

History. This patient was a 61-year-old right-handed man who had suffered from IPD for 10 years and whose medical history included myocardial infarctions in 1975 and 1984. On both occasions, he required coronary artery bypass graft surgery. He underwent his first RF pallidotomy with intraoperative microelectrode recording in the right pallidum in July 1995; the procedure produced good alleviation of symptoms initially.
Examination. The patient presented again with levodopa-induced severe dystonia of his left extremities as well as progression of tremor, rigidity, and bradykinesia on the right side. An MR image revealed that the RF lesion on the right had significantly regressed in size and was located slightly anterior to the ideal target in the pallidum.

Operation. The patient underwent bilateral RF pallidotomies without intraoperative microelectrode recordings in February 1996 to enlarge the lesion in the posterior direction on the right and to create a new lesion in the left pallidum to address the progression of symptoms on the right side.

Postoperative Course. The patient did well in the immediate postoperative period with improvement of symptoms on both sides to the point at which he was able to discontinue taking his medications. However, 10 days postsurgery he presented with acute right-sided hemiparesis and facial droop and his speech was slurred.

Operation and Postoperative Course. Because the patient’s symptoms were more severe on the right side, she underwent a left pallidotomy without intraoperative microelectrode recording. The patient did well in the immediate postoperative period; the procedure produced dramatic alleviation of her symptoms on the right side. However, 51 days postsurgery she presented with acute right-sided weakness and facial droop; her speech was slurred.

She was admitted for treatment of stroke and underwent transthoracic echocardiography and carotid duplex studies, the results of which were both negative. Her lesion was localized on MR imaging to the posterior limb of the left internal capsule adjacent to the pallidotomy site (Fig. 2). She was treated with aspirin and discharged to a rehabilitation facility. With intensive physical and speech therapy her weakness and dysarthria partially resolved.

Case 3

History. This patient was a 64-year-old right-handed man suffering from IPD with bilateral tremors intractable to medical therapy. Aside from the IPD, the patient had no other known medical problems. However, on his presurgical MR image he was noted to have clinically silent deep white matter disease indicative of ischemia.

Operations and Postoperative Course. Because the patient’s symptoms were worse on the right side, he initially underwent a left RF pallidotomy without intraoperative microelectrode recording; the procedure produced good results. A right RF pallidotomy with intraoperative microelectrode recording was performed 7 months later. This procedure also resulted in dramatic improvement in the patient’s rigidity and left-sided tremor. The patient became much more independent in his activities of daily living; however, he developed acute left-sided weakness with gait disturbance and dysarthria 117 days after the second pallidotomy.

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He was admitted for presumed stroke and underwent transthoracic echocardiography and carotid duplex study. The echocardiogram did not show any thrombus and the duplex study did not show any significant stenosis. The diagnosis of a right posterior limb capsular infarction adjacent to the pallidotomy site was confirmed on MR imaging (Fig. 3). Tests for a hypercoagulable state, including prothrombin, partial thromboplastin time, fibrinogen, D-dimer, protein C, protein S, antithrombin III, homocysteine, anticardiolipin antibody, and lupus anticoagulant studies, were all within normal limits. He was started on a course of aspirin. The patient’s dysarthria mostly resolved during his hospital stay, but he continued to have some residual left-sided weakness at the time of his discharge.

Results

The volume of the left RF lesion (Fig. 1A) in the first patient (Case 1), visualized on MR imaging obtained within 1 hour of his bilateral pallidotomies, measured 0.9 ml with 2.97 ml of gadolinium (Gd) enhancement. The RF lesion did not impinge on the internal capsule. Comparison with the poststroke MR image (Fig. 1C) shows an area of low signal intensity in the internal capsule superior to the left RF lesion. This low-intensity signal area does not enhance with Gd and is consistent with an infarction. Persistent Gd enhancement can be seen around the RF lesions. The T2-weighted image (Fig. 1B) demonstrates the 7-mm posterior extent of the infarction in the posterior limb of the internal capsule.

The prestroke MR image (Fig. 2A) obtained in the second patient (Case 2) within 1 hour of the pallidotomy demonstrates a well-demarcated RF lesion in the left pallidum measuring 0.96 ml with 3.62 ml of Gd enhancement and no impingement on the internal capsule. The poststroke proton density–weighted MR image (Fig. 2C) shows a new area of high signal intensity in the posterior limb of the left internal capsule that did not exist in the prestroke MR image (Fig. 2B).

The RF lesion in the third patient (Case 3), visualized on MR imaging obtained within 1 hour of the second pallidotomy, measured 1.61 ml with 3.51 ml of Gd enhancement (data not shown). The RF lesion did not impinge on the internal capsule (Fig. 3B). The poststroke MR image (Fig. 3C) shows a discrete new high-intensity signal area posterior to the right RF lesion in the posterior limb of the internal capsule. Comparison of the prestroke MR study (Fig. 3B) with that obtained poststroke (Fig. 3C) reveals a new discrete area of high signal intensity on the T2-weighted image. This new area appears as a low-intensity signal lesion on the T1-weighted image (Fig. 3A) and does not enhance after administration of contrast material. There is persistent enhancement of the RF lesions on both sides.

The delayed strokes in the three patients occurred on postoperative Days 10, 51, and 117, respectively. No strokes occurred in Group 2 (11 patients) or 3 (20 patients). All three of the strokes occurred in patients with a history of vascular disease (Group 1, 11 patients). The fact that all three strokes occurred in Group 1 patients is statistically significant according to the chi-square test, with a probability value of 0.0287.

Discussion

The pallidotomy technique used in our patients, with MR imaging localization, electrophysiological guidance, and RF lesioning, is comparable to methods used by other groups. The immediate postpallidotomy MR images document the appropriate placement and size of these lesions. No impingement of the RF lesions into the internal capsule was seen in the three patients with delayed strokes, and these lesions conformed to the expected size based on RF parameters. However, comparison of the pre- and poststroke MR images in these patients shows the development of a new low-intensity signal area on the T2-weighted image in the posterior limb of the internal capsule adjacent to the RF lesion. Unlike the RF lesions, which display persistent ring enhancement for up to several months, these low-intensity signal lesions do not enhance with Gd. These lesions exhibit high-intensity signals with irregular borders on T2-weighted images, which is consistent with infarctions associated with edema in the late acute stage. The lesions correlate anatomically with the patients’ hemiparesis in all three cases.

The spatial proximity of the infarctions to the pallidoto-
Delayed infarctions following RF pallidotomy are not uncommon. In a recent series of 50 patients undergoing RF pallidotomy, three patients (6%) developed delayed strokes, two of which were subclinical and not noted in follow-up evaluations. These delayed infarctions may be unrecognized because of suboptimum follow-up time and the presence of additional risk factors such as vascular disease. It is well known that the nonuniform composition of brain matter can cause distortions in RF lesioning and render certain areas of the brain more susceptible to thermal injury. White matter, gray matter, cerebrospinal fluid, and blood have different electrical resistances with varying degrees of heat dissipation. The higher vascularity of gray matter also makes it more resistant to ischemia. These factors modulating the spread of energy within brain matter may explain why the observed secondary injuries from RF lesioning occurred in the internal capsule white matter. Conversely, gray matter infarctions may be subclinical and therefore not noted in follow-up evaluations.

The use of microelectrode recording has recently been advocated by several authors to localize the globus pallidus internus better during RF pallidotomies. It is not yet clear whether this additional step has led to more accurate RF lesion placement with better outcome. No higher morbidity rate has been associated with microelectrode recording in our series, which is not surprising because our protocol does not require additional penetrations of brain matter to accomplish the recording. Of the three patients presented in this study, only one had undergone a pallidotomy with microelectrode recording preceding the delayed stroke. The question of whether repeating an RF pallidotomy on the same side increases the risk of delayed infarction is not settled. One did undergo repeated RF pallidotomy on the same side. However, the other two patients in our series who underwent RF pallidotomy revisions have not had strokes, despite a history of vascular disease in one of them.

In recently published series of RF pallidotomies a complication rate of 1.4 to 7% is reported, depending on how the rate is calculated. Reported morbidities include transient and permanent motor and visual deficits resulting from hemorrhage, abscess, and suboptimally placed RF lesions. Our rate for symptomatic delayed stroke after pallidotomy procedures is 6% (three of 50 patients). This rate may be an underestimate of the true incidence because the progression of RF lesions in patients over an extended period of time is unknown. Also, a proportion of delayed infarctions may not cause obvious neurological deficit or are considered coincidental, and some may be unrecognized because of suboptimum follow-up review.

Cases of delayed stroke after RF pallidotomy have not been previously reported, although a recent report by an Emory University group did mention incidentally a probable capsular infarction near the surgical lesion in a patient who had undergone pallidotomy 3 weeks previously. Another group has recently identified this complication in their series (M Hariz, personal communication, 1997). Careful reviews of RF pallidotomy experiences by other groups will be needed to show whether these delayed infarctions are isolated observations or represent a real complication resulting from RF procedures. It remains to be seen whether a direct relationship exists between vascular disease history and delayed infarction after RF pallidotomy. Further investigation of this possible relationship is warranted because our experience indicates that patients with a history of vascular disease may have a higher risk of stroke after RF pallidotomy. Multivariate analysis applied to a larger patient series and long-term follow-up review may be helpful because our limited analysis in this study with the chi-square test only shows that all three delayed strokes occurring in Group 1 were not due to chance alone. Neurosurgeons as well as

**Fig. 3.** Case 3. A: Axial T1-weighted poststroke MR image with Gd enhancement showing the RF lesions in the pallidum. The more recent RF lesion on the right shows a larger ring-enhancing pattern. The 33-day-old left RF lesion has a small outer rim of low-intensity signal around the enhancing center. B: Axial T2-weighted prestroke MR image obtained within 1 hour of the right pallidotomy, documenting that there was no impingement on the internal capsule. C: Axial T1-weighted poststroke MR image enhancement showing the new discrete high-intensity signal area in the posterior limb of the right internal capsule posterior to the RF lesion 117 days after right pallidotomy (arrow). Note the diffuse deep white matter disease.
neurologists and referring primary physicians will need to combine their efforts and be vigilant about this possible complication.

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