Leksell's posteroventral pallidotomy in the treatment of Parkinson's disease

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Between 1985 and 1990, the authors performed stereotactic posteroventral pallidotomies on 38 patients with Parkinson's disease whose main complaint was hypokinesia. Upon re-examination 2 to 71 months after surgery (mean 28 months), complete or almost complete relief of rigidity and hypokinesia was observed in 92% of the patients. Of the 32 patients who before surgery also suffered from tremor, 26 (81%) had complete or almost complete relief of tremor. The L-dopa-induced dyskinesias and muscle pain had greatly improved or disappeared in most patients, and gait and speech volume also showed remarkable improvement. Complications were observed in seven patients: six had a permanent partial homonymous hemianopsia (one also had transient dysphasia and facial weakness) and one developed transitory hemiparesis 1 week after pallidotomy.

The results presented here confirm the 1960 findings of Svennilson et al., that parkinsonian tremor, rigidity, and hypokinesia can be effectively abolished by posteroventral pallidotomy, an approach developed in 1956 and 1957 by Lars Leksell. The positive effect of posteroventral pallidotomy is believed to be based on the interruption of some striopallidal or subthalamopallidal pathways, which results in disinhibition of medial pallidal activity necessary for movement control.

Key Words • Parkinson's disease • tremor • rigidity • hypokinesia • stereotaxis • pallidotomy • thalamotomy
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FIG. 1. Diagrams showing the classical pallidotomy (A) and Leksell's posteroventral pallidotomy (L) target in relation to the anterior commissure (AC), the posterior commissure (PC), the intercommissural line (IC), the midcommissural point (MC), and the midline of the third ventricle (ML). Note the rectangular disk shape of Leksell's lesion, which was achieved with a bipolar twin electrode setting.

### TABLE 1

Summary of target in 38 patients treated with posteroventral pallidotomy

<table>
<thead>
<tr>
<th>Target</th>
<th>No. of Cases</th>
<th>No. of Operations</th>
</tr>
</thead>
<tbody>
<tr>
<td>rt pallidotomy</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>lt pallidotomy</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>bilat pallidotomy</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>rt pallidotomy &amp; lt thalamotomy</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>lt pallidotomy &amp; rt thalamotomy</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>lt pallidotomy &amp; bilat thalamotomy</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>totals</td>
<td>38</td>
<td>54</td>
</tr>
</tbody>
</table>

In this paper, we report our results and discuss the role of the pallidum in extrapyramidal movement disorders.

### Clinical Material and Methods

**Clinical Material**

Thirty-eight patients with Parkinson's disease who were suffering from severe symptoms of tremor, rigidity, bradykinesia, gait difficulties, dyskinesias, and/or muscle pain were selected for posteroventral pallidotomy. Pure bradykinesia, with or without minor tremor, was often the dominating symptom. The 20 men and 18 women ranged in age from 30 to 80 years (mean 60.3 years). The duration of illness varied from 2 to 20 years (mean 9.4 years). All had received physiotherapy and conventional drug therapy consisting of L-dopa, bromocriptine, anticholinergics, MAO-B inhibitors, or amantadine, but the effect had not been satisfactory.

**Technique of Pallidotomy**

Pallidotomy was unilateral in 34 and bilateral in four patients. In eight patients, thalamotomy had previously been performed on one side and pallidotomy was carried out on the other. Two patients had previously undergone bilateral thalamotomy. Thus, the total number of stereotactic interventions was 54 (Table 1). The interval between the two operations was usually 6 to 14 months, except in the two patients in whom bilateral thalamotomies had been carried out several years before pallidotomy of the left side.

The pallidal target was defined with stereotactic computerized tomography (CT) using a Stereoadapter, and surgery was performed with the Stereoadapter and a Stereoguide without ventriculography. Fifteen minutes before surgery, the patients received 50 mg of pethidine hydrochloride (Petidin) injected intramuscularly and 0.5 mg of atropine sulphate (Atropin) injected subcutaneously. Local anesthesia of 1% prilocaine hydrochloride with adrenaline (Citanest) was used for bone fixation of the Stereoguide and the scalp incision. The pallidal target lay 2 to 3 mm in front of the midcommissural point and 18 to 21 mm lateral to the midline of the third ventricle; in the first three patients it was 3 mm below the intercommissural (IC) line and in the others it was 6 mm below the IC line. Through a frontal burr hole placed 2 cm from the midline, a 1.8-mm thick monopolar electrode† with 2 mm of uninsulated tip was introduced to the target under impedance monitoring. The final lesion site was based on the patient's reactions to electrical stimulation.

Electrical stimulation of the target area was carried out with the monopolar electrode. The stimulus parameters were: rectangular 1-msec monophasic pulses, 60 and 6 Hz, intensity 2 to 8 mA. The threshold responses to pallidal stimulation generally required higher current

* The Laitinen stereotactic devices manufactured by Issal AB, Vallentuna, Sweden.
† Laitinen-Wiksell monopolar electrode manufactured by Comair AB, Stockholm, Sweden.
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TABLE 2
Threshold responses on electrical stimulation of the posteroventral pallidum and side effects*

<table>
<thead>
<tr>
<th>Response</th>
<th>No. of Cases</th>
<th>Side Effect†</th>
</tr>
</thead>
<tbody>
<tr>
<td>increased muscle tension</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>visual phenomenon</td>
<td>12</td>
<td>homonymous scotoma (6)</td>
</tr>
<tr>
<td>tremor increase</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>tremor decrease</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>nausea</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>paresthesia</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>muscular jerks</td>
<td>2</td>
<td>hemiparesis (2)</td>
</tr>
<tr>
<td>speech arrest</td>
<td>2</td>
<td>dysphasia (1)</td>
</tr>
<tr>
<td>anxiety</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>none</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

*Stimulus parameters: 1-msec monophasic square wave pulses, 60 Hz, intensity 2 to 5 mA. Some patients had multiple threshold responses.
†Clinical side effects of permanent lesions. Number of cases is given in parentheses.

intensity than to ventrolateral-ventralis intermedius (VL-VIM) thalamic stimulation. Threshold responses are shown in Table 2. Radiofrequency thermo lesions were produced with a lesion generator,‡ 72° to 82°C for 60 to 90 seconds at several layers of depth so that the dorsoventral length of the lesion was 6 to 7 mm and the transverse diameter about 6 mm.

Assessment of Motor Performance

The motor performance was assessed by a writing and drawing test and a gait test 1 day before and 1 day after surgery. The patients wrote letters of the alphabet, drew figures with the right and then the left hand, and walked as fast as possible around a 50-m circle clockwise and then counterclockwise (Fig. 2). The times for performing these tasks were recorded. We have recently added to the battery of tests the Purdue Pegboard Test and Stroop's Color Word Test, but because these tests were administered in only a limited number of patients, their results are not given in this paper.

Results

The clinical results are shown in Table 3. The follow-up period ranged from 2 to 71 months (mean 28 months). All patients were alive when this manuscript was prepared.

Neurological Function

Tremor. Tremor was present preoperatively in 32 patients. It sometimes diminished or became slow and irregular immediately after introduction of the electrode to the target. Often it diminished further during the stimulation procedures prior to thermo lesioning. In 14 patients, the tremor diminished markedly or disappeared completely during thermo lesioning. Often, its frequency fell from 6 or 7 Hz to approximately 4 Hz. Following the procedure, the remaining tremor occurred on a periodic basis and gradually disappeared. In six patients, relief of tremor was poor; in one of these, tremor improved in the arm but not in the leg. In the patients with poor tremor relief, electrical stimulation had given a visual response. In order to avoid visual field defect, the lesion was reduced in size and was placed more dorsal than initially aimed. An excellent or good long-lasting tremor relief was obtained in 26 (81%) of the 32 patients.

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\[\text{lent or good long-lasting tremor relief was obtained in } 26 \text{ (81%) of the 32 patients.}\]
Rigidity. Relief of rigidity was accomplished in more patients than improvement in tremor. It was rated excellent or good in 35 patients (92%). Muscular hypotonia, a common finding after VL-VIM thalamotomy, was never observed after pallidotomy.

Hypokinesia. In 35 patients (92%), the surgery was very effective for bradykinesia; this could be seen during the operation. Mechanical introduction of the electrode to the target region often improved the speed of arm and leg movements on the contralateral side of the body. During electrical stimulation this effect seemed to increase further, and during thermoablation movements in all patients became fast and precise. Bradykinesia disappeared equally in the arm and the leg. During motor testing 20 to 24 hours after surgery, speed and precision in the contralateral hand had improved greatly; there was also some ipsilateral improvement. Figure 3 shows the effect of pallidotomy on the speed of writing and walking in 16 consecutive patients; for comparison, similar test results for 16 consecutive thalamotomy patients are shown.

Gait. The improvement in gait was similar to that of hypokinesia (Fig. 3 and Table 3). Initiation of a step quickened, and balance improved. The associated movements reappeared on the side contralateral to pallidotomy. Tilting of the body toward the side contralateral to surgery and body inattention, often seen after thalamotomy, were not observed in any patient with pallidotomy.

Speech. The volume of speech and articulation often improved after pallidotomy (Table 3). Speech volume after left pallidotomy improved in a 54-year-old woman who had undergone bilateral thalamotomy 17 and 10 years earlier, respectively, and who had developed complete aphonia. Another 55-year-old female schoolteacher with severe right-sided rigidity, hypokinesia, and whispering aphonic speech regained a fairly good speech volume, allowing her to return to her profession. Two patients had transient postoperative dysphasia. One, a 73-year-old woman, had transitory facial weakness, dysphasia, and right homonymous scotoma after left pallidotomy. A 64-year-old man had a successful left pallidotomy without complications, but 1 week later he experienced a sudden minor stroke with slight dysphasia, right hemiparesis, and right visual field defect.

Dystonia and Muscle Pain. Pallidotomy had a very good effect on involuntary movements induced by L-dopa and on painful muscular spasms (Table 3). The dystonic movements disappeared more or less completely in all but three patients. In these patients, relief of tremor was poor.

General Condition. All patients tolerated the pallidotomy well. Neither bilateral pallidotomy nor pallidotomy combined with contralateral thalamotomy appeared to create stress for the patients. They were permitted to stand immediately after surgery, have a meal, and leave the hospital on the following day. Even the eldest patient, an 80-year-old man whose gait and balance prior to surgery were extremely poor, could walk home 20 hours after left pallidotomy. No psychological side effects were observed. In most patients, antiparkinsonian medications could gradually be reduced by 50% to 75%.

Lesion Control

Postoperative stereotactic CT was performed 3 to 12 months later in 13 patients to confirm the size and site...
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of the lesions. The lesions lay in the posteroventral region of the pallidum, very close to the amygdala and the internal capsule, and ranged in size from 21 to 160 cu mm, with a mean of 95 cu mm (Fig. 4).

**Long-Term Results**

The follow-up period ranged from 2 to 71 months (mean 28 months). Thirty-four patients (89%) were clearly improved, and tremor, rigidity, and bradykinesia were more or less completely abolished on the side contralateral to pallidotomy. Bradykinesia had greatly improved, and the movement pattern often returned to normal. This improvement did not appear to diminish during the follow-up period. All four patients who had undergone bilateral pallidotomy showed considerable improvement, although in one there was unilateral recurrence of the symptoms 8 weeks after the second operation. One patient had been bedridden for 3 years; after her second pallidotomy, she began to walk with assistance. Her condition has remained stable for 4 years.

Three patients had a partial or total recurrence of symptoms 1 to 8 weeks after pallidotomy. In these patients, postoperative CT study showed the lesions to be smaller and more dorsally placed than in the other patients.

**Side Effects**

Despite electrical stimulation and repositioning of the electrode because of visual stimulation response, six patients had a central homonymous visual field defect after the 42 pallidotomies (14%); one patient also had transitory facial weakness and dysphasia. Figure 5 shows a hemianoptic defect in the lower left quadrant which was presumably due to a heat lesion of the dorsolateral aspect of the optic tract. The defect was perceived as a small shadow or spot in the lower central visual field and was easily noticed by the patient, but could not be detected in a clinical visual confrontation test. In five patients, the visual field defect on the left side did not present a severe disturbance, but in one patient, the small central defect on the right side was very troublesome because it made reading difficult. The visual field defect appears to be permanent.

The above-mentioned patient with a right visual field defect also had a transitory facial weakness on the right side and dysphasia. A late postoperative stroke with slight hemiparesis occurred in another patient 6 days after pallidotomy.

**Discussion**

**Effects of Thalamotomy**

In Sweden, with a population of 8.5 million, 60 to 70 thalamotomies are performed each year for the treatment of parkinsonian tremor. In most cases, there is permanent resolution of contralateral tremor and L-dopa-induced choreoathetosis. The effect of thalamotomy on rigidity is usually favorable, but may be of shorter duration. Thalamotomy often worsens bradykinesia. Laitinen and Vilkki, who used the Purdue Pegboard Test to measure the speed and precision of their parkinsonian patients, showed that thalamotomy increased bradykinesia. The deterioration was more marked after left than after right thalamotomy. The deteriorating effect was most clear in tests for simultaneous bimanual performance, where the operated hand lagged behind the nonoperated hand. In the reaction-time tests, the initiation of movement had become slow. It is also well known that in right-handed patients left thalamotomy causes transitory verbal deterioration, while right thalamotomy causes a visuospatial impairment.
Painful muscle cramps are often not improved by thalamotomy, and gait disturbances rarely improve. This is also demonstrated in Fig. 3. When walking, the patient often tilts toward the side contralateral to thalamotomy, and this side lags behind the other. Balance and speech disturbances may result from unilateral thalamotomy and are an almost constant finding after bilateral thalamotomy. Thus, although thalamotomy usually has a dramatic effect on tremor, this effect is mainly cosmetic and social. Neurologists have been aware of this and, in many countries, only a few parkinsonian patients are referred for thalamotomy.

Effects of Pallidotomy

Our study confirmed the finding of Svennillon, et al.,30 that Leksell’s posteroventral pallidotomy procedure had a positive and long-lasting effect on all three cardinal symptoms of Parkinson’s disease: tremor, rigidity, and bradykinesia. Their careful study showed that in their whole series, with the anterodorsal pallidotomies included, lasting remission of tremor had been observed in 67 (82%) of 81 patients, and the success rate for rigidity was 79%. Follow-up review ranged from 1 to 5 years. Although Svennillon, et al., did not define the degree of improvement for hypokinesia, they stated that “The most obvious gain was improved mobility, in terms of strength, range, speed, and precision.” In the same text they stated: “Tremor, hypokinesia, and rigidity are relieved to an equal extent by operation.” Of their last 20 patients, who had been operated on in the posteroventral pallidum, 19 (95%) had “complete relief of rigidity and tremor.”30 It is not clear why this operation seems not to have been performed after that. Discussions between Lars Leksell and one of us (L.V.L.) finally encouraged us to attempt treatment of parkinsonian akinesia with posteroventral pallidotomy. We now have no doubt that the results following Leksell’s pallidotomy are excellent in regard to cardinal symptoms of Parkinson’s disease: tremor, rigidity, and hypokinesia. If the symptoms are unilateral, the movement pattern, speed, and precision often return to normal. The operation also appears to benefit hyperkinesias induced by L-dopa and the painful muscle cramps that often occur in parkinsonian patients, as well as greatly improving gait and diminishing balance problems. Speech volume may also improve after pallidotomy. Muscular hypotonia with postoperative tilting toward the contralateral side, a common finding after thalamotomy, was never observed after pallidotomy. In two patients who in addition to parkinsonian akinesia suffered from anxiety, surgery relieved this psychiatric symptom.

In contrast to thalamotomy, pallidotomy does not create stress in the patient. This difference is still more specific when the size of the lesions is compared. The mean size of the pallidal lesions, 95 cu mm, has been more than three times that of thalamotomy (mean 26 cu mm).11

FIG. 6. Map of the frontal section of the brain 2 mm anterior to the midcommissural point (adapted from Schaltenbrand and Wahren25). The white ring indicates the presumed lesion area in the posteroventral pallidum, lateral and dorsal to the optic tract (o). Abbreviations: n = substantia nigra; s = subthalamic nucleus; t = ventrolateral thalamus; ml = midplane of the third ventricle; il = intercommissural line.

Localization of Lesion

Postoperative CT studies showed that the lesions lay in the posteroventral region of the pallidum, close to the pallidum externum and the internal capsule, very close to the optic tract, and just dorsal to the amygdala complex (Figs. 4 and 6). Their vicinity to the optic tract could present a serious risk of contralateral homonymous hemianopsia. Six (14%) of 42 pallidotomies were associated with this side effect, but all six were followed by excellent improvement of tremor, rigidity, and bradykinesia. Therefore, it is crucial to place the lesion as close to the optic tract as possible, but still avoid lesioning it. Although we exercised care when using electrical stimulation to locate the lesion, and thus avoided the optic tract, central homonymous hemianopsia occurred in six patients. When stimulation resulted in an optic response, the electrode was either withdrawn in a dorsal direction or, in more recent cases, repositioned 2 mm more lateral. Visual field defects were not detected by perimetry in Leksell’s 20 patients who in 1956 and 1957 underwent posteroventral pallidotomy.30 This was not mentioned in Guiot’s analysis8 of 138 patients, who had been operated on in the central part of the pallidum.
Pallidum in Movement Control

that lesions in the substantia nigra lead to bradykinesia and tremor. In the parkinsonian brain, the dopamine level in the substantia nigra, putamen, and caudate nucleus is low. Dopamine deficiency in the nigra neostriatum may lead to increased inhibition in the globus pallidus, possibly via GABA (γ-aminobutyric acid)-ergic mechanisms, which then inhibits the initiation of movement. It is well documented that L-dopa therapy often abolishes this inhibition and re-establishes a normal movement pattern.

It is also well known that the globus pallidus plays an important role in the physiology of movement. In the monkey, cells in the lateral portion of the pallidum re-establish a normal movement pattern.

Several clinical and experimental studies have shown that lesions in the substantia nigra lead to bradykinesia and tremor. In the parkinsonian brain, the dopamine level in the substantia nigra, putamen, and caudate nucleus is low. Dopamine deficiency in the nigra neostriatum may lead to increased inhibition in the globus pallidus, possibly via GABA (γ-aminobutyric acid)-ergic mechanisms, which then inhibits the initiation of movement. It is well documented that L-dopa therapy often abolishes this inhibition and re-establishes a normal movement pattern.

It is also well known that the globus pallidus plays an important role in the physiology of movement. In the monkey, cells in the lateral portion of the pallidum re-establish a normal movement pattern.

In 1958, Carpenter, et al., showed that experimental cerebellar tremor in the monkey could be abolished by pallidal lesions. Obrador and Diesssen and Wycis and Gildenberg reported that pallidotomy and ansotomy in man abolished not only parkinsonian resting tremor but also cerebellar intention tremor and choreoathetosis. We have been able to confirm the findings of Svennison, et al., that posteroventral pallidotomy has a positive effect on parkinsonian resting tremor. We have also shown that L-dopa-induced choreoathetosis can be abolished by stereotactic lesions in this part of the pallidum. The classical pallidotomy lesions placed in the anterodorsal and medial areas of the pallidum, as reported in 1958 by Cooper and Bravo, influenced rigidity, but much less so tremor and still less so bradykinesia. How can we interpret these contradictory findings?

There is experimental and clinical evidence indicating that all parkinsonian symptoms, at least tremor and bradykinesia and presumably also rigidity, develop in or are mediated by the pallidum. As discussed above, dopamine deficiency presumably leads to inhibition in the dorsomedial part of the pallidum, where the initiation of the movements is blocked. Additional stereotactic lesions in the dorsomedial pallidum would therefore impair, not improve, the initiation of movement. Recently, we have observed in several parkinsonian patients small localized calcifications in the anterodorsal pallidum (unpublished data), which may be further evidence that this portion of the pallidum is not healthy in patients with Parkinson’s disease. Stereotactic lesions in the lateral part of the posteroventral pallidum seem to block pallidal inhibition and re-establish the initiation of movement; simultaneously, parkinsonian tremor and rigidity also disappear. It seems likely that the lesions interrupt some striopallidal or striosubthalamosomal pallidial pathways and re-establish more or less normal functioning in the medial pallidum and pallidofugal pathways.

It has been documented in large clinical series that choreoathetosis and hemiballism can be abolished by lesions in the pallidum or in the ansa lenticularis. These two movement disorders may be similar to each other: post-apoplectic hemiballism often begins with slight choreic movements of the hand which, within a few days, increase and expand to massive rhythmic movements of the proximal part of the extremity. Hemiballism is believed to arise from lesions in the subthalamosomal pallidial pathways or in the subthalamic nucleus, which cause disturbed balance in the pallidum and release rhythmic movements. The L-dopa-induced choreoathetosis often resembles spontaneous chorea. Our present study supports the concept that these rhythmic movement disorders (resting tremor, choreoathetosis, and hemiballism) can be abolished by lesions placed in the posteroventral pallidum. It is unclear whether intention (cerebellar) tremor can also be abolished by similar lesions.

Our clinical observations are in accordance with previous laboratory and clinical findings that all parkinsonian symptoms may develop in, or at least are mediated by, pathological mechanisms in the dorsal and medial parts of the pallidum internum. Leksell’s group and our study have shown that stereotactic lesions in the posteroventral pallidum can abolish them. Therefore, the common theory of the origin of the parkinsonian tremor may need to be revised. Hassler and Riechert believed that parkinsonian rigidity only was mediated by the pallidothalamic pathways, whereas the tremor was of cerebellar origin; this may not be entirely true. We are convinced that the parkinsonian...
tremor can be completely abolished by stereotactic lesions in the posteroventral pallidum. The effect seems to be long-lasting. The fact that a number of our patients did not have complete tremor relief was most likely due to the very small size and the dorsal placement of the lesion, which was caused by visual reactions during electrical stimulation. In the future, it may be possible to modify the shape of the lesions so that the pathological pathways, whatever they are, can be effectively cut without damage to the optic tract.

The responses to electrical stimulation of the posteroventral pallidum differ clearly from those observed at thalamic stimulation. The current intensity in the posteroventral pallidum must be twice as high as that in the VL-VIM thalamus to result in objective or subjective reactions. To avoid optic tract responses, we recently changed the laterality of the target from 20 to 22 mm; current intensities needed for subjective or objective responses exceeded three times those of thalamic stimulation. However, tremor and akinesia usually began to diminish after stimulation.

Leksell's pallidotomy has in many aspects been much more effective than thalamotomy. The greatest difference between the two approaches is that pallidotomy abolishes akinesia whereas thalamotomy increases it. Because of the increased risk for visual field defect, we still use thalamotomy in patients in whom the major problem is tremor of the arm. But when pallidotomy can be performed without risk for homonymous hemianopsia, it may become a valuable treatment for all types of movement disorders in drug-resistant Parkinson's disease.

Conclusions

We have been able to confirm the findings of Sven-nilson, et al., that Leksell's posteroventral pallidotomy has a very good long-lasting effect on all parkinsonian symptoms: tremor, rigidity, and particularly bradykinesia. The best indications for pallidotomy consist of incapacitating and drug-resistant bradykinesia and muscle pain, particularly in the legs; tremor; rigidity; and the L-dopa-induced choreoathetosis react equally well to that treatment. The close vicinity of the pallidal lesions to the optic tract implies a great risk for central homonymous hemianopsia. Other side effects occur very rarely.

Acknowledgment

Our idea to revisit the pallidum was born during and after many fruitful discussions with Professor Lars Leksell, 1907–1986. We are deeply grateful to this great man and friend.

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