Hemodynamic and metabolic disturbances in patients with intracranial dural arteriovenous fistulas: positron emission tomography evaluation before and after treatment

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In patients with intracranial dural arteriovenous fistulas (AVFs), clinical symptoms and angiographic findings vary. The relevance of disturbed venous drainage to clinical symptoms and prognosis has been recognized. However, the roles of cerebral hemodynamics and metabolism, which are impaired by shunt flow or disturbed venous drainage, have not been fully evaluated. The authors studied the cerebral hemodynamic and metabolic status in 10 patients with intracranial dural AVFs using positron emission tomography (PET) scanning. Ten patients with dural AVFs underwent a PET study before treatment. The regional cerebral blood flow (rCBF), regional oxygen extraction fraction (rOEF), regional cerebral metabolic rate of oxygen (rCMRO2), and regional cerebral blood volume (rCBV) were measured using the 15O-labeled gas inhalation steady-state method. The PET parameters that were obtained were analyzed and compared with the patients' neurological and angiographic findings. In six of the 10 patients, a PET study was also performed after treatment. Before treatments, all four patients with cerebral symptoms showed a severe reduction in rCBF and a mild elevation in the rOEF. The areas showing reduced rCBF corresponded with areas in which retrograde venous drainage into the cortical veins and delayed parenchymal circulation were seen on angiograms. In another two patients with occlusion of the affected sinus and/or retrograde drainage into the cortical veins, mild abnormalities were demonstrated in rCBF mapping. In the remaining four patients, all four PET parameters except rCBV were within normal limits and venous flow was not impaired on the angiograms. In four patients who underwent surgical excision or transvenous embolization of the affected sinus, the cerebral hemodynamics and metabolism were improved, as were the clinical symptoms. In two patients who underwent transarterial embolization of the feeding vessels only or craniotomy, no hemodynamic improvement was achieved. Our results indicate that hemodynamic insufficiency detected by the PET study corresponded well with cerebral symptoms and angiographic findings of retrograde venous drainage into the cortical veins and delayed parenchymal circulation, but not with sinus occlusion or arterial blood supply. Eradication or prevention of retrograde venous drainage from the affected sinus into the cortical veins should be a treatment goal in patients with dural AVFs.

KEY WORDS • dural arteriovenous fistula • positron emission tomography • cerebral hemodynamics • cerebral metabolism

Intracranial dural arteriovenous fistulas (AVFs) are relatively uncommon lesions. With advances in neuroradiological technology, reports of dural AVFs have increased and their clinical features have been clarified. In patients with dural AVFs, the severity of their symptoms ranges from benign tinnitus to comatose status. Similarly, angiographic findings in this disease are complex, consisting of combinations of arteriovenous shunts, sinus occlusions, retrograde venous drainage into the cortical veins, venous aneurysms, and impaired parenchymal circulation. The clinical presentation and prognosis are thought to be determined by the pattern of venous drainage rather than by the arterial blood supply. On serial angiograms, the abnormality of venous drainage can be judged and divided into some types, but it is difficult to evaluate these findings quantitatively and to base selection of treatment on them. Moreover, it remains unclear which angiographic findings are most important or indicative of a critical situation. The cerebral hemodynamics and metabolism may be impaired to various degrees, depending on the degree of shunt flow or disturbed venous drainage. However, the roles of cerebral hemodynamics and metabolism have not been fully evaluated in patients with intracranial dural AVFs, although they are thought to be the key not only for an understanding of the pathophysiology of the disease but also for the selection of appropriate treatment.

We measured cerebral hemodynamics and oxygen metabolism in 10 patients with dural AVFs using positron emission tomography (PET) scanning and analyzed the correlations among hemodynamic/metabolic status, clinical symptoms, and angiographic findings. In six of the 10 patients, a PET study was also performed after intravascular and/or direct surgery to evaluate the efficacy of treatment with respect to improved cerebral hemodynamics and metabolism.
Clinical Material and Methods

Patient Population

Over the last 3 years, we performed PET studies in 10 patients with dural AVFs to evaluate their cerebral hemodynamics and metabolism before any treatment. Table 1 displays a summary of the patients’ clinical and angiographic findings. There were seven men and three women in the group; their ages ranged from 51 to 70 years (mean 60.2 years). The patients’ clinical symptoms ranged from disorientation to consciousness disturbance. One patient (Case 4) had a history of intraventricular hemorrhage.

All patients underwent superselective angiography to identify patterns of arterial blood supply and venous drainage. Occlusion of the sinuses and delays in parenchymal circulation time were also investigated angiographically.

Hemodynamic and Metabolic Status Before Treatment

Positron Emission Tomography Study. We used a fourring seven-plane PET scanner (Headtome IV; Shimadzu, Japan) that has a transaxial resolution of 6.5 mm and an axial resolution of 4.5 mm full width at half maximum in clinical use. A transmission scan was obtained for attenuation correction of positron annihilation gamma-rays in the brain. Regional cerebral blood flow (rCBF), the regional cerebral metabolic rate of oxygen (rCMRO2), the regional oxygen extraction fraction (rOEF), and regional cerebral blood volume (rCBV) were measured after the patients inhaled 15O-labeled carbon dioxide, oxygen, and carbon monoxide, with arterial activity corrected through a radial artery catheter. During PET scanning, the patient was firmly immobilized by placing a plastic collar (Philadelphia Cervical Collar Co., Westville, NJ) around the neck and by using a head restraint. The patient’s ears were plugged and eyes were covered. Before each group of PET measurements was obtained, the patient’s head position was checked according to its alignment relative to six reference points. Blood gas levels were measured with a blood gas autoanalyzer (280 Blood Gas System; Ciba-Corning Diagnostics Corp., Medfield, MA). Blood pressure was monitored throughout the examinations.

Oval regions of interest (ROIs) (2 × 5 cm) were selected in the cortex of the frontal, temporal, parietal, and occipital lobes. Smaller ROIs (2 × 3 cm) were placed at the thalamus.

Control values of PET parameters were obtained from hemispheric ROIs in five healthy volunteers and were expressed as the mean ± standard deviation (SD) as follows: 36.3 ± 5.7 ml/100 g/minute for CBF; 2.59 ± 0.37 ml/100 g/minute for CMRO2; 2.47 ± 0.48 ml/100 g for CBV, and 0.42 ± 0.05 for rOEF.

Results of the PET Study. The results of the PET study are shown in Table 1. Impaired cerebral hemodynamics in the whole cerebrum was the finding in Cases 1 and 2; six patients had regional impairment. The patients in Cases 1 to 4 had significantly decreased rCBF, mildly-to-severely increased rOEF, and severely increased rCBV values. Of these four patients, all but one (Case 1) had a normal rCMRO2 value. The patient in Case 5 had mildly increased rCBF and a mildly decreased rOEF in the occipital lobe. In the patient in Case 6, rCBF was mildly decreased but the rOEF was normal. The patients in Cases 7 and 8 had only regional elevations of rCBV in the patients in Cases 9 and 10 all PET findings were within normal limits.

Correlation of Clinical and Angiographic Findings With the Results of the PET Study

The patients in Cases 1 to 4 presented with clinical symptoms ranging from disorientation to consciousness disturbance (Group A). Angiograms obtained in these patients showed retrograde venous drainage into the cortical veins. In the areas that exhibited reduced rCBF on PET studies, parenchymal circulation delay was seen.

### TABLE 1
Clinical and angiographic findings and results of PET study in 10 patients with dural AVFs*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs.)</th>
<th>Sex</th>
<th>Clinical Manifestation</th>
<th>Pre-exam Bleeding</th>
<th>Site of Dural AVF</th>
<th>Occlusion of Affected Sinus</th>
<th>RV Drain</th>
<th>Delayed Circulation†</th>
<th>Region of Impaired Hemodynamics</th>
<th>PET Measurements in Region W/ Impaired Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55, M</td>
<td></td>
<td>consciousness dist</td>
<td>no</td>
<td>SSS, rt TSS</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>whole cerebrum</td>
<td>rCBV (ml/100 g) = 5.26, rCBF (ml/100 g/min) = 24.6, rOEF = 1.98, rCMRO2 (ml/100 g/min) = 0.51</td>
</tr>
<tr>
<td>2</td>
<td>64, M</td>
<td></td>
<td>memory dist</td>
<td>no</td>
<td>SSS, lt TSS</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>whole cerebrum</td>
<td>rCBV (ml/100 g) = 5.32, rCBF (ml/100 g/min) = 24.0, rOEF = 2.70, rCMRO2 (ml/100 g/min) = 0.51</td>
</tr>
<tr>
<td>3</td>
<td>64, M</td>
<td></td>
<td>disorientation</td>
<td>no</td>
<td>straight sinus</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>bilat thalamus</td>
<td>rCBV (ml/100 g) = 5.02, rCBF (ml/100 g/min) = 24.1, rOEF = 2.68, rCMRO2 (ml/100 g/min) = 0.50</td>
</tr>
<tr>
<td>4</td>
<td>51, M</td>
<td></td>
<td>memory dist</td>
<td>yes</td>
<td>lt TSS</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>lt temporal</td>
<td>rCBV (ml/100 g) = 5.25, rCBF (ml/100 g/min) = 23.5, rOEF = 2.74, rCMRO2 (ml/100 g/min) = 0.54</td>
</tr>
<tr>
<td>5</td>
<td>70, F</td>
<td></td>
<td>tinnitus</td>
<td>no</td>
<td>lt TSS</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>lt temporal</td>
<td>rCBV (ml/100 g) = 4.33, rCBF (ml/100 g/min) = 43.4, rOEF = 2.76, rCMRO2 (ml/100 g/min) = 0.37</td>
</tr>
<tr>
<td>6</td>
<td>56, M</td>
<td></td>
<td>tinnitus</td>
<td>no</td>
<td>lt TSS</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>lt occipital</td>
<td>rCBV (ml/100 g) = 4.17, rCBF (ml/100 g/min) = 30.1, rOEF = 2.76, rCMRO2 (ml/100 g/min) = 0.43</td>
</tr>
<tr>
<td>7</td>
<td>55, F</td>
<td></td>
<td>tinnitus</td>
<td>no</td>
<td>rt TSS</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>rt occipital</td>
<td>rCBV (ml/100 g) = 4.11, rCBF (ml/100 g/min) = 40.8, rOEF = 3.12, rCMRO2 (ml/100 g/min) = 0.41</td>
</tr>
<tr>
<td>8</td>
<td>70, M</td>
<td></td>
<td>dizziness</td>
<td>no</td>
<td>lt TSS</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>lt temporal</td>
<td>rCBV (ml/100 g) = 4.46, rCBF (ml/100 g/min) = 32.2, rOEF = 2.74, rCMRO2 (ml/100 g/min) = 0.46</td>
</tr>
<tr>
<td>9</td>
<td>61, F</td>
<td></td>
<td>tinnitus</td>
<td>no</td>
<td>rt TSS</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>none</td>
<td>rCBV (ml/100 g) = 3.31, rCBF (ml/100 g/min) = 41.1, rOEF = 3.01, rCMRO2 (ml/100 g/min) = 0.44</td>
</tr>
<tr>
<td>10</td>
<td>56, M</td>
<td></td>
<td>tinnitus</td>
<td>no</td>
<td>marginal sinus</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>none</td>
<td>rCBV (ml/100 g) = 3.42, rCBF (ml/100 g/min) = 33.9, rOEF = 2.75, rCMRO2 (ml/100 g/min) = 0.45</td>
</tr>
</tbody>
</table>

* Dist = disturbance; RV drain = retrograde venous drainage into cortical veins; SSS = superior sagittal sinus.
† At region with retrograde flow into cortical vein.
‡ Increased values lie outside of mean ± 2 SD of healthy controls.
§ Decreased values lie outside of mean ± 2 SD of healthy controls.
TABLE 2

Summary of treatment and clinical course in 10 patients with dural AVFs

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Treatment</th>
<th>Neurological Symptom Posttreatment</th>
<th>Findings on Follow-Up Angiography</th>
<th>Complications During Follow-Up Period</th>
<th>Final Outcome</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TAE, TVE of the rt TSS</td>
<td>improved</td>
<td>occlusion of the SSS</td>
<td>venous hypertension, renal failure</td>
<td>dead</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>TAE, excision of the SSS</td>
<td>disappeared</td>
<td>none</td>
<td></td>
<td>excellent</td>
<td>28</td>
</tr>
<tr>
<td>3</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
<td>intracerebral hemorrhage†</td>
<td>poor</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>TAE, excision of the lt TSS</td>
<td>disappeared</td>
<td>cured</td>
<td>none</td>
<td></td>
<td>excellent</td>
</tr>
<tr>
<td>5</td>
<td>craniotomy</td>
<td>improved</td>
<td>unchanged</td>
<td>acute subdural hemorrhage</td>
<td>poor</td>
<td>31</td>
</tr>
<tr>
<td>6</td>
<td>TAE, excision of the lt TSS</td>
<td>disappeared</td>
<td>cured</td>
<td>none</td>
<td></td>
<td>excellent</td>
</tr>
<tr>
<td>7</td>
<td>TAE, isolation of the rt TSS</td>
<td>disappeared</td>
<td>cured</td>
<td>none</td>
<td></td>
<td>excellent</td>
</tr>
<tr>
<td>8</td>
<td>TAE</td>
<td>unchanged</td>
<td>recanalization</td>
<td>none</td>
<td></td>
<td>fair</td>
</tr>
<tr>
<td>9</td>
<td>observation</td>
<td>improved</td>
<td>unchanged</td>
<td></td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>observation</td>
<td>improved</td>
<td>unchanged</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*NA = not applicable; TVE = transvenous embolization of the affected sinus.
† Occurred after PET study but before any treatments.

Of the six patients who had the mild symptoms of tinnitus and dizziness (Group B), two had mildly increased or decreased rCBF. In the patient in Case 5, rCBF was mildly increased in the left occipital lobe; retrograde drainage from the affected sinus into the cortical veins was seen but there was no delay in parenchymal circulation time. The patient in Case 6 showed mildly decreased rCBF and increased rCBV; his transverse–sincipital sinus (TSS) was occluded on the left side but he manifested no retrograde venous drainage into the cortical veins and no delayed parenchymal circulation.

Positron emission tomography parameters were compared between Groups A and B using the Bonferroni modified t-test. Using mean values (±SD) we found that the rCBV value in Group A (5.21 ± 0.13 ml/100 g) was significantly higher than that in Group B (3.97 ± 0.48 ml/100 g) (p < 0.01); the mean rCBF value in Group A (24.1 ± 0.5 ml/100 g/minute) was significantly lower than that in Group B (36.9 ± 5.5 ml/100 g/minute) (p < 0.01), the rOEF value in Group A (0.52 ± 0.02) was significantly higher than that in Group B (0.43 ± 0.03) (p < 0.01); and the mean rCMRO value in Group A (2.53 ± 0.36 ml 100 g/minute) was lower than that in Group B (2.86 ± 0.17 ml/100 g/minute), although the difference was not statistically significant.

Treatment and Clinical Course

Table 2 shows the treatment and clinical course of the 10 patients. The patients in Cases 2, 4, 6, and 7 underwent excision or isolation of the affected sinus following transcatheter arterial embolization (TAE) of the feeding arteries. Their neurological symptoms disappeared after the treatment and their dural AVFs were confirmed to be cured on follow-up angiograms. The patient in Case 1 underwent sinus embolization with platinum coils following the TAE; he recovered consciousness but his condition worsened again 6 months after the treatment. Follow-up angiograms revealed occlusion of the superior sagittal sinus. The patient died of renal failure 15 months after the initial treatment. The patient in Case 8 underwent TAE alone; follow-up angiograms revealed recanalization of the feeding vessels. This patient was treated conservatively because his venous drainage was not impaired.

The patients in Cases 3 and 5 had impaired venous drainage; they experienced intracranial bleeding and their status is currently poor. The patient in Case 3 bled after examination. Although the patient in Case 5 underwent craniotomy, retrograde venous drainage into the cortical veins persisted.

The patients in Cases 9 and 10 had low-flow arteriovenous shunts and normal hemodynamics. They are being monitored on an outpatient basis. Their symptoms have slightly improved without any intervention and they are in good condition.

Hemodynamic and Metabolic Status After Treatment

The patients in Cases 1, 2, 4, 5, 6, and 8 underwent follow-up PET study 3 to 6 months after treatment. All four patients who had undergone surgical excision or transvenous embolization of the affected sinus showed increased rCBF and decreased rCBV; the rOEF was normalized in the patients in Case 2, 4, and 6 and remained elevated in the patient in Case 1 who died 15 months after treatment.

In the two patients who underwent craniotomy (Case 5) or transarterial embolization alone (Case 8), the hemodynamics were not improved after treatment.

Illustrative Case

Case 4

This 51-year-old man experienced a sudden onset of headache.

Examination. A precontrast computerized tomography scan demonstrated an intraventricular clot at the left trigone. His acoustic cognition was remarkably disturbed. He was able to memorize things he read but not those he heard. Selective angiography showed dural AVFs at the left TSS, which was fed by the left ascending pharyngeal, middle meningeal, and occipital arteries and drained in retrograde fashion via the vein of Labbé and other cortical veins (Fig. 1 upper left). The greater part of the left TSS was not opacified on internal carotid angiograms. The parenchymal circulation at the left temporal lobe and cerebellar hemisphere was remarkably delayed (Fig. 1 upper
right). Positron emission tomography scanning revealed a marked decrease in rCBF (23.5 ml/100 g/minute) and an increase in rCBV (5.25 ml/100 g) in the left temporal lobe. An increase in the rOEF (0.54) was noted in the same area, although the rCMRO$_2$ value was within normal limits (2.74 ml/100 g/minute) (Fig. 2 left).

**Treatment.** The patient underwent transarterial embolization of the feeding arteries, and shunt flow through the dural A VFs decreased markedly. On a PET scan obtained after embolization, a significant increase in rCBF (37.2 ml/100 g/minute) was noted; however, the patient’s rCBV (4.3 ml/100 g) and rOEF (0.51) values remained high (Fig. 2 center). He underwent excision of the affected TSS 2 weeks after embolization. Intraoperatively, the vein of Labbé appeared as a red vein and was cut.

**Postoperative Course.** Postoperatively, the patient’s acoustic cognition was restored and he was able to return to his job as general manager of a hotel. Four months after the surgery, follow-up angiograms showed no opacification of the dural AVF (Fig. 1 lower left) and no delay in parenchymal circulation (Fig. 1 lower right). A postoperative PET study showed that the rCBV (3.4 ml/100 g) was normalized (Fig. 2 right).

**Discussion**

**Clinical Manifestations of Dural AVFs and Cerebral Hemodynamics**

The clinical manifestations of dural AVFs range from benign tinnitus to diffuse cerebral dysfunction.$^{1,13}$ Cerebral symptoms are thought to depend more on the pattern of venous drainage than on the site of the AVF or the arterial supply.$^{1,7,12,17}$ Impaired venous drainage results in cerebral hemodynamic insufficiency, but there are few reported studies on the cerebral hemodynamics or metabolism
in patients with dural AVFs. Two-dimensional cerebral hemodynamic and metabolic studies are considered useful for assessing the corresponding lesion and neurological functions.

Our PET results for 10 patients with dural AVFs indicated that clinical symptoms correlated well with impaired hemodynamic parameters, especially the reduction in rCBF and the elevation in rOEF. In all four patients with cerebral symptoms (Group A), an obvious reduction in rCBF and mild elevation in rOEF values were shown at the site corresponding to the symptoms. In contrast, six patients who presented with pulsatile tinnitus or dizziness without cerebral symptoms (Group B) had either mildly decreased, mildly increased, or normal rCBF and none of these patients had an increased rOEF. There were statistically significant differences in rCBF, rOEF, and rCBV values between Group A and Group B patients. Hemodynamic and metabolic evaluation is very useful to assess the cerebral function in patients with dural AVFs.

The most catastrophic event in the clinical course of a dural AVF is intracranial hemorrhage. Patterns of venous drainage have been identified as predictors of risk of intracranial hemorrhage. In five of our patients (Cases 1–5), retrograde venous drainage via the cortical veins was shown on serial angiograms. Of these five patients, four had decreased rCBF and one (Case 5) had mildly increased rCBF. Intracranial bleeding was documented in three patients (Cases 3, 4, and 5). Our findings coincide with those reported by others; retrograde drainage through the cortical veins is indicative of a high risk of bleeding. On the other hand, risk of intracranial bleeding cannot be inferred from rCBF and/or rOEF values.

**Correlation Between Cerebral Hemodynamic/Metabolic Status and Angiographic Findings**

Our results indicate that the cerebral hemodynamic and metabolic status closely corresponds with angiographic findings. In patients in whom venous drainage was not impaired by arteriovenous shunts (Cases 7–10), the rCBF, rCMRO₂, and rOEF values were within normal limits. Of these four patients, two had mildly increased rCBV and two had normal rCBV values. In Case 6, angiograms showed sinus occlusion and no retrograde drainage through the cortical veins; rCBV was increased and rCBF was mildly decreased. In Cases 1 to 5, angiograms showed retrograde venous drainage into the cortical veins; in all but one patient (Case 5), rCBF was decreased and the rOEF was mildly or markedly increased. In Case 5 there was no angiographic evidence of obviously delayed parenchymal circulation; the patient’s collateral venous drainage route may have been well developed. The angiograms obtained in Cases 1 to 4 showed delayed parenchymal circulation but the rCMRO₂ was within normal limits in all but one patient (Case 1).

Among the angiographic findings in patients with dural AVFs, retrograde venous drainage into the cortical veins and delayed parenchymal circulation are important predictors of the hemodynamic status; sinus occlusion is not.

**Efficacy of Treatments With Respect to Hemodynamics**

Dural AVFs have been treated by excision, isolation or transvenous embolization of the affected sinus, transarterial embolization of the feeding arteries, and radiosurgery. We found that the angiographic findings that are most important for indicating hemodynamic impairment are retrograde venous drainage into the cortical veins and delayed parenchymal circulation. Therefore, the goal of treatment should be eradication or prevention of retrograde venous drainage from the affected sinus into the cortical veins. When a dural AVF is treated by transarterial embolization, the obliterated dural AVF often reappears on follow-up angiography. We presented Case 4 as an illustrative case. This 51-year-old man underwent surgical excision of the affected sinus following transarterial embolization and, although the fistula was shown to be almost completely obliterated on subsequent angiograms, during surgery the vein of Labbé was confirmed to drain arterial blood. Thus, angiographic findings of obliteration of a dural AVF do not guarantee a permanent cure. We recommend radical treatment—surgical excision or transvenous embolization of the affected sinus—to improve the cerebral hemodynamics and symptoms in patients with severe impairment of venous return. In our illustrative case, the PET study performed before surgery revealed elevated rCBV; the rCBV in the temporal lobe was fully normalized after the excision of the dural AVF.

**Conclusions**

We conclude that hemodynamic and metabolic measurements as well as evaluation of the neurological symptoms and angiographic assessment are useful to evaluate the efficacy of treatment in patients with dural AVFs.

**Acknowledgments**

We thank Mr. Miyake, Mr. Sago, Mr. Oka, Mr. Ohmiya, and Miss Azabu for PET measurements, and Miss Iga for laboratory assistance.

**References**

Hemodynamics and metabolism in dural AVFs


Manuscript received July 25, 1996. Accepted in final form January 7, 1997.

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