Painful Tic Convulsif Caused by a Brain Tumor
Undiagnosed Preoperatively

To the Editor.—Painful tic convulsif, a term coined by Cushing in 1920, consists of ipsilateral concurrent trigeminal neuralgia and hemifacial spasm.1 Fifty-five cases have been reported2 since Cushing's initial report. In 7 of the 55 cases, painful tic convulsif is reported to be caused by a brain tumor.2 We describe a patient treated for ipsilateral trigeminal neuralgia and hemifacial spasm who was found to have an undiagnosed brain tumor discovered during microvascular decompression of the 5th and 7th cranial nerves.

A 32-yr-old man was suffering from daily transient hemifacial spasm. After treatment with nimodipine hydrochloride and cloroazepam for a presumed psychosomatic disorder without relief for 2 yr, he experienced severe lancinating pain in his right maxilla after dental surgery. Diagnosis of trigeminal neuralgia was made, and he was treated with pramipexol and dichlofenac sodium with temporary relief. After 1 yr, his pain increased in severity, and he was referred to the department of neurology in our hospital. The neurologic findings showed intermittent twitching of his right eyelid and right corner of the mouth. The pain affected the region innervated by the third branch of the right trigeminal nerve without distinct trigger points. Audiometric examination, taste, and equilibrium were normal, and other cranial nerves were normal.

He was treated with 600 mg/day carbamazepine with temporary relief. To investigate the cause of his neuralgic attacks, magnetic resonance imaging (MRI) was performed but showed no brain tumor. Because of the atypical nature of the facial pain accompanied by neuralgic attacks, he was referred to our pain clinic. We performed a series of ten stellate ganglion blocks over 2 weeks under the diagnosis of atypical facial pain. His pain decreased from 10 of 10 to 0 on a visual analog scale a week after the initiation of treatment with the block. Neurosurgery was consulted regarding microvascular

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decompression (Jannetta’s procedure) for his hemifacial spasm and neuralgic attacks. During the surgery, an epidermoid cyst, 1 cm in diameter, was found in the cerebellar pontine angle, compressing the 5th and 7th cranial nerves, and was removed. His symptoms disappeared immediately after the operation, and his postoperative course was favorable.

The anesthesiologist, when asked to care for patients with tic convulsif, should be aware that the symptoms can be caused by a brain tumor, and thus, more thorough neurologic examination, involving MRI, should be considered.

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References

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rPF4 Does Not Cause Pulmonary Hypertension in Humans

To the Editor—Kurrek et al. reported that recombinant human platelet factor 4 (rPF4), a peptide foreign to the lamb, produced acute pulmonary hypertension when infused in awake unheparinized animals. They demonstrated the hypertension was caused by release of thromboxane A2. They likened this response to the acute pulmonary hypertension caused by heparin-prostamine complex in the lamb and rarely in humans by activation of complement by the classic pathway. Based on these two unrelated observations with differing mechanisms, they caution about the use of rPF4 as a heparin antagonist in humans. This seems farfetched.

rPF4 is not a foreign peptide if administered to humans and would not be expected to stimulate release of thromboxane A2. If heparin-rPF4 complexes activate complement as does heparin-prostamine complex, the likelihood of adverse response is not different from that now accepted with prostamine. So far, recombinant human rPF4 has been administered in doses of 0.5–5.0 mg/kg intravenously to 18 patients to reverse heparin anticoagulation after cardiac catheterization. No changes in pulmonary artery pressure were observed.

In 12 patients after coronary artery bypass graft, we studied reversal by rPF4 of heparin-induced anticoagulation after cardiopulmonary bypass. Intravenous bolus administration of rPF4 in doses from 1 to 7.5 mg/kg failed to alter pulmonary artery pressure (table 1). We do not believe rPF4 represents a special hazard for acute pulmonary hypertension based on the observations of Kurrek et al.

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References

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Table 1. rPF4 and Human Pulmonary Artery Pressure

<table>
<thead>
<tr>
<th></th>
<th>PA SBP (mmHg)</th>
<th>PA DBP (mmHg)</th>
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<tbody>
<tr>
<td>Before CPB</td>
<td>25 ± 6</td>
<td>14 ± 5</td>
</tr>
<tr>
<td>After CPB</td>
<td>35 ± 15</td>
<td>17 ± 6</td>
</tr>
<tr>
<td>5 min</td>
<td>32 ± 9</td>
<td>19 ± 5</td>
</tr>
<tr>
<td>10 min</td>
<td>32 ± 9</td>
<td>19 ± 5</td>
</tr>
<tr>
<td>30 min</td>
<td>28 ± 8</td>
<td>17 ± 4</td>
</tr>
</tbody>
</table>

CPB = cardiopulmonary bypass; PA SBP = pulmonary artery systolic pressure (mean ± SD); PA DBP = pulmonary artery diastolic pressure (mean ± SD).

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