A 66-year-old man on rivaroxaban (Xarelto) for atrial fibrillation experienced a pontine hemorrhage. He survived the acute hospitalization and was discharged to a rehabilitation facility in a quadriparetic state. Over the 4 months following the hemorrhage, he developed a tremor, first in the lower and then in the upper extremities, which prompted his referral to the Movement Disorders clinic.

On examination of visual tracking, there was no horizontal movement of the left eye and no adduction of the right eye. He had distal more than proximal, left more than right, and lower more than upper limb weakness, ranging from 3/5 in the left leg to 4/5 in the right arm, with depressed deep tendon reflexes. In the left upper extremity, he had a tremor that was present at rest, with posture, and with action. The movements met the description of Holmes tremor in the Consensus Statement of the Movement Disorder Society on Tremor. The movements lacked the jerky quality of myoclonus, the randomness of chorea, or the sustained twisting quality of dystonia, nor was there any pulling toward a null position as in dystonic tremor. There was also head tremor with activation of the left shoulder girdle and neck muscles. When he held the left arm outstretched, supporting it against gravity, and the examiner shoved the limb out of position, the limb oscillated for several cycles before settling into position again. Pointing (finger-chin-finger) was ataxic, and there were mild athetoid movements of the fingers. A similar resting tremor was present in (finger-chin-finger) was ataxic, and there were mild athetoid movements of the fingers. A similar resting tremor was present in

Higher cuts, at the level of the midbrain, show a striking goggle-like appearance of T2 hyperintensity representing anterograde degeneration of axons while sparing surrounding structures, has been imaged in a case of Holmes tremor. It is well-known that mesencephalic lesions may cause a low-frequency tremor present at rest, with posture, and with action (Holmes tremor). The term “rubral” for such tremors implies a role for the red nucleus (RN), but the critical structure may instead be cerebellar efferents, some of which synapse in the parvo cellular RN, whereas others wrap around the RN en route to thalamus. The question is difficult to resolve because it would be rare for a mesencephalic lesion to affect a substantial portion of the cerebellothalamic fibers while sparing the RN.

Our patient’s Holmes tremor was caused by a pontine lesion some distance away from the midbrain and is thus a “cerebellar outflow tremor.” Its remarkable feature is the magnetic resonance imaging, which shows a midbrain lesion, presumably representing edema associated with anterograde degeneration of axons damaged in the pons. To our knowledge, this is the first case in which a midbrain lesion limited to cerebellar efferents, while sparing surrounding structures, has been imaged in a case of Holmes tremor.

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Figure 1: (A) Pontine hemorrhage. (B) Goggle-like appearance in midbrain representing anterograde axonal edema in which cerebellothalamic fibers surround red nuclei.
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