the recovery stage of stroke and were living relatively normal lives despite neurological impairment. Twenty-one of the 24 patients had experienced strokes between 4 and 10 AM. None were taking any other drugs. Throughout the period of our study, all patients were requested to maintain their routine daily schedules.

We conducted our study using oral nitrendipine (Nantong Pharmaceutical Factory, Hebei, China) and tested its efficacy in relation to the circadian changes of blood pressure using different therapies. The trial was carried out in the following order: blood pressure (both systolic and diastolic) was measured from the right brachial artery at 2-hour intervals over a 24-hour period beginning at 8 AM. At the same time, all hypertensive patients were given 10 mg nitrendipine T.I.D. daily over 1 week, followed by another 24-hour period of blood pressure measurements. Finally, a new therapy was instituted as follows: 20 mg nitrendipine was administered once daily in the early morning over a 1-week period, followed by the same investigation of blood pressure described above.

As reported by Millar-Craig et al., the circadian change of blood pressure was expected during the first stage of our trial without nitrendipine. During the second stage of the trial, the previous therapy could have reduced the blood pressure at any time in the twelve 2-hour intervals each day, but the daily rhythm of circadian change was only slightly different from that without nitrendipine. In the third stage, with the new regimen, there was a varied pattern of circadian change of blood pressure, which avoided the profound nocturnal fall and reduced the elevated blood pressure in the morning hours. This regimen not only decreased hypertension in the peak hours of blood pressure, but also helped stabilize blood pressure during the circadian period. We conclude that this new regimen may be superior to previous therapies in the prevention of ischemic stroke onset.

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Pure Lemniscal Sensory Deficit Caused by Pontine Hemorrhage

To the Editor:

The literature rarely describes strokes producing pure lemniscal sensory deficit.1-4 The patient described by Gravelleau et al5 is the only such case that we know. This patient subsequently developed dysarthria, motor weakness, and impairment of other sensory modalities. We report another case of pontine hemorrhage in which selective impairment of the lemniscal sensory modality was the only clinical manifestation throughout the course.

A hypertensive 67-year-old woman developed a sudden left-sided tingling sensation and gait difficulty. She was an alert woman with normal cranial nerves, motor functions, and reflexes. There was decreased touch and vibration sense in the left half of her body that worsened in the lower extremity. Position sense was impaired in the left fingers and toes. Temperature and pinprick were normally perceived, and stereognosis and graphesthesia were normal. There was ataxia on finger-to-nose and heel-to-shin tests in the left limbs. She could stand alone, but needed assistance in walking because she tended to veer to the left.

Routine laboratory tests were normal. Brain computed tomographic scan showed a small hemorrhage in the right pontine tegmentum, which was more clearly identified by magnetic resonance imaging 2 days after onset of the stroke.

She improved rapidly after admission. A day later, vibration sense was normally perceived in the left side of her face. Two days later, decrease in position sense was restricted to the left toes and she could walk unaided. Two weeks later, she still showed mild impairment of vibration and position sense in the left lower extremity. Slight weakness of left foot dorsiflexion was noted. A selective impairment of medial lemniscal sensory function was associated with a small hemorrhage in the pontine tegmentum that corresponded to the anatomic location of the medial lemniscus. The pyramidal tract and spinothalamic sensory pathways were spared. Although rare, cases with strategically situated, small strokes that produce a selective impairment of either medial lemniscal5-6 or spinothalamic sensory modality5,6 undoubtedly help delineate the anatomic localization of the human sensory pathway.

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