A brother of the propositus had an acute myocardial infarct at the age of 40. The latter died 2 years later from mesenteric thrombosis. Because the association between hereditary protein S deficiency and arterial thrombosis is controversial,2,3 we are reporting another case with this same association in which other members of the family were affected by arterial thrombotic diseases.

A 44-year-old man was admitted to our hospital a few hours after noticing dizziness, left facial sensory symptoms, a hoarse voice, and deglutary troubles. Past medical history was unremarkable for vascular risk factors or thrombotic episodes. On examination, neurological deficits were compatible with a left lateral medullary syndrome. Further investigation, including chest x-ray, electrocardiography, and echocardiography, were normal. Cranial computed axial tomography at the earlier stages of the clinical picture was normal. On Doppler examination, a left vertebral artery was not found, but other extracranial arteries were normal. A total occlusion of the left vertebral artery was confirmed by arteriography.

Family history was noteworthy for venous thrombosis (Figure 1). A brother of the propositus had an acute myocardial infarct at the age of 45, and a second brother had an acute stroke at the age of 40. The latter died 2 years later from mesenteric thrombosis.

The patient submitted to a protocol for the biologic diagnosis of thrombosis currently in use in our hospital. Results from platelet count, coagulation screening, hemogram, protein C, fibrinogen, plasminogen, and antithrombin III testing were all normal. No increase of plasminogen activity inhibitors of the lupus type or of plasminogen, and antithromboin III testing were all normal. No increase of immunoglobulin (Ig) G or IgM anticardiolipin antibodies was found. However, total and free protein S levels were low at 49% and 29%, respectively. The criterion used for laboratory diagnosis of protein S antigen was that the plasma concentration of protein S antigen should be lower than the lower limit of the range established for healthy volunteers (<70%). Normal values in our laboratory ranged from 80% to 110%, based on the plasma of apparently healthy individuals.

The relationship between protein S deficiency and arterial thrombosis is controversial and uncommon.2-4 Isolated cases of cerebral arterial thrombosis and familial protein S deficiency, without the vascular risk factors, have been described in young adults.1-5-6 Sacco et al3 thought that protein S deficiency could be responsible for arterial thrombosis when other additional risk factors for vascular disease were present. However, neither our patient nor the other patients referred to in previous works5,6 showed such risk factors. Like others,2,6 we believe that this familial coagulopathy can be determined as an unequivocal risk factor for arterial thrombotic disease only by means of epidemiological studies.

**References**


**Nitrendipine May Protect Hypertensive Patients From Ischemic Stroke Onset**

*To the Editor:*

Increased frequency of ischemic stroke onset in the morning hours has been reported,1-4 and the mechanism of this circadian variation as it relates to frequency has been demonstrated to result from the circadian change of blood pressure; that is, the blood pressure falls in sleep and rises or fluctuates during the morning hours, probably causing the higher incidence of ischemic stroke in the morning hours. For this reason, we suggest that hypertensive patients at high risk of stroke should be treated during these vulnerable periods to control or avoid the nocturnal fall and morning fluctuation of blood pressure. Such therapy might prevent the triggering of ischemic stroke in hypertensive patients.

Twenty-four hypertensive patients with ischemic stroke (15 men and nine women, mean age 61.42 [range 52-72] years) and 20 hypertensive patients with transient cerebral blood infusion disorders (13 men and seven women, mean age 58.75 [range 47-70] years) were included in our study. These infarcted patients were in...
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.

## References


## Pure Lemniscal Sensory Deficit Caused by Pontine Hemorrhage

To the Editor:

The literature rarely describes strokes producing pure lemniscal sensory deficit. The patient described by Gravelleau et al. 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 lemniscal or spinothalamic sensory modality undoubtedly help delineate the anatomic localization of the human sensory pathway.

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Nitrendipine may protect hypertensive patients from ischemic stroke onset.

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