

## Central motor conduction time in patients with periventricular lucencies

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### Abstract

Central motor conduction time and regional cerebral blood flow were measured before and 20 min after intravenous injection of 17 mg/kg acetazolamide in 10 patients with periventricular lucencies (PVL) and 10 age-matched healthy controls. Central motor conduction time was measured using a magnetic stimulator and regional cerebral blood flow was measured by stable xenon computed tomography method. The central motor conduction time was significantly longer in the patients with PVL than in the healthy controls and was shortened significantly by the intravenous injection of acetazolamide in the patients with PVL. The blood flow not only in the periventricular white matter but also in the cerebral cortex and the cerebral white matter was significantly lower in the patients with PVL than in the healthy controls. The intravenous injection of acetazolamide increased significantly the regional cerebral blood flow except in the PVL areas. The prolongation of the central motor conduction time may be at least partly related with decreased blood flow in the cerebral cortex and cerebral white matter.

**Keywords:** Central motor conduction time; Magnetic stimulation; Periventricular lucencies; Cerebral blood flow; Xenon computed tomography; Acetazolamide

### 1. Introduction

Periventricular lucencies (PVL) on computed tomography (CT) scan (Gupta et al., 1988) or periventricular hyperintensity on magnetic resonance imaging (Zimmerman et al., 1986; Gerald and Weisberg, 1986; Kertesz et al., 1988) are considered to be mainly due to demyelination and gliosis (Sze et al., 1986; Révész et al., 1989; Leifer et al., 1990; Van Swieten et al., 1991). Cerebral blood flow in patients with PVL has been reported to be decreased (Kobari et al., 1990a; Tachibana et al., 1990; Kobari et al., 1990b; Meguro et al., 1990; Kawamura et al., 1991; De Reuck et al., 1992a; De Reuck et al., 1992b; Terayama et al., 1992) but there are no reports on central motor conduction time and/or on the effect of acetazolamide on regional cerebral blood flow in patients with PVL. Therefore, we measured central motor conduction time and regional cerebral blood flow before and after intravenous injection of acetazolamide.

### 2. Materials and methods

Ten patients with PVL on CT scan (mean age, 58.6 years) who visited our outpatient clinic and 10 age-matched healthy controls (mean age, 57.4 years) were studied. The research received prior approval by the appropriate institutional review body and informed consent was obtained from each subject or patient. Patients with hydrocephalus, muscle weakness or stroke were excluded. The patients with PVL were admitted to our hospital and head magnetic resonance imaging, echocardiography and blood viscosity were studied on the patients. Cortical infarcts and lacunes were not present on the head magnetic resonance imaging. The average score of the mini-mental state examination (Folstein et al., 1975) was 25 points in the patients with PVL.

Central motor conduction time was measured using a SMN-1100 magnetic stimulator and a round coil with a diameter of 17 cm (Nihon Kohden, Tokyo, Japan). Magnetic stimulation was performed at the head with muscles relaxed and with muscles mildly contracted and at the neck. Muscle potentials were recorded from bilateral opponens pollicis muscles and abductor digiti minimi muscles

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using a Synax 1100 evoked potential recorder (NEC San-ei, Tokyo, Japan). The latencies of the right opponens pollicis muscle evoked by magnetic stimulation of the brain (counter-clockwise coil current seen from the above) with the coil centered at the vertex and evoked by magnetic stimulation of the neck with the coil centered at the sixth cervical spine were measured.

The regional cerebral blood flow was measured using the stable xenon CT method (Touho et al., 1990; Johnson et al., 1991; Kashiwaki et al., 1992). The basal ganglia section and the midbrain section parallel to the orbitomeatal line were selected as the region of interest in the head. The subjects inhaled room air followed by a mixture of 30% xenon and 50% oxygen for 3 min. Serial scanning was performed once before xenon inhalation, three times

in the wash-in process and five times in the wash-out process of 5 min. The serial scanning program consisted of a total of 18 scans consisting of 9 serial scans on each section. The xenon concentration in the end-tidal expired gas was continuously recorded by the thermoconductivity method. We used the xenon delivery and analysis system (AZ-7000 model, Anzai Sogyo, Tokyo, Japan) and CT equipment (PreSage, Yokogawa Medical Systems, Tokyo, Japan).

Central motor conduction time and regional cerebral blood flow were measured before and 20 min after intravenous injection of 17 mg/kg acetazolamide. Statistical analysis was performed using the Mann-Whitney's *U*-tests for comparison between the patients and the healthy controls and using Wilcoxon signed-ranks tests for compari-

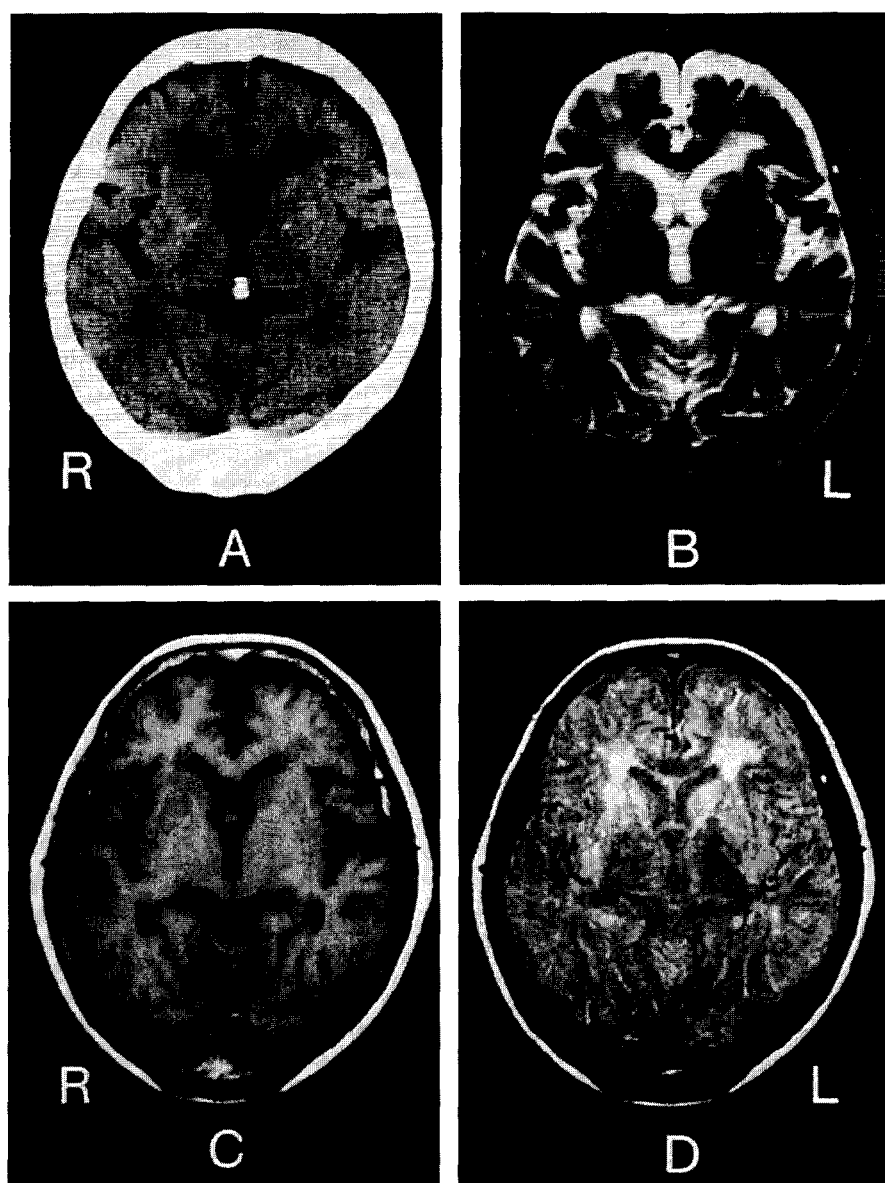


Fig. 1. Computed tomography and magnetic resonance imaging in a patient with periventricular lucencies. A: CT. B:  $T_2$ -weighted image. C:  $T_1$ -weighted image. D: proton density image. Mild periventricular lucencies are present.

son between before and after the intravenous injection of acetazolamide.

### 3. Results

Fig. 1 shows CT scan and magnetic resonance imaging in a patient with PVL. Eight of our patients had mild PVL like Fig. 1 and the remaining 2 patients had moderate PVL. All of our patients had hypertension.

Fig. 2 shows the potential of the right opponens pollicis muscle evoked by head magnetic stimulation. Table 1 shows the mean and standard deviation of the central motor conduction time. Before the intravenous injection of acetazolamide, the central motor conduction time was significantly longer in the patients with PVL than in the healthy controls. After the intravenous injection of acetazolamide, the central motor conduction time was shortened significantly in comparison with that before the intravenous injection of acetazolamide.

Fig. 3 shows an actual record of xenon CT in a patient with PVL and Fig. 4 shows an actual record of xenon CT in a healthy control. Before the intravenous injection of acetazolamide, the blood flow not only in the PVL areas

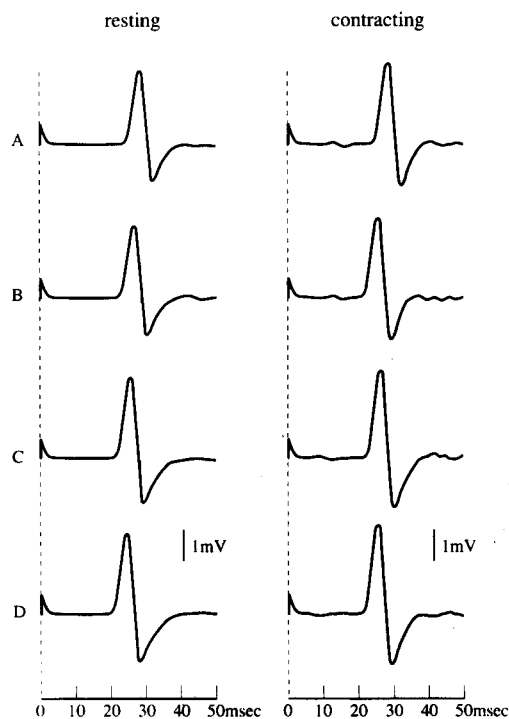


Fig. 2. Potential of the right opponens pollicis muscle evoked by head magnetic stimulation. A,B: patient with periventricular lucencies. C,D: healthy control. A,C: before intravenous injection of acetazolamide. B,D: after intravenous injection of acetazolamide. Before the intravenous injection of acetazolamide, the central motor conduction time was longer in a patient with PVL (A) than in a healthy control (C). After the intravenous injection of acetazolamide (B), the central motor conduction time is shortened in comparison with that before the intravenous injection of acetazolamide (A).

Table 1

The central motor conduction time before and after the intravenous injection of acetazolamide (ms, mean  $\pm$  standard deviation)

	Patients with periventricular lucencies		Healthy controls	
	Before	After	Before	After
Resting muscle	11.3 $\pm$ 1.6 <sup>a,b</sup>	9.4 $\pm$ 1.5 <sup>a</sup>	9.3 $\pm$ 1.3 <sup>b</sup>	9.2 $\pm$ 1.4
Contracting muscle	10.7 $\pm$ 1.4 <sup>c,d</sup>	8.1 $\pm$ 1.2 <sup>c</sup>	7.9 $\pm$ 1.1 <sup>d</sup>	7.9 $\pm$ 1.2

<sup>a,b,d</sup>  $p < 0.01$ . <sup>c</sup>  $p < 0.05$ . The significant difference is present between the same letters.

but also in the cerebral cortex and the cerebral white matter in the patient with PVL was decreased in comparison with that in the healthy control. After the intravenous injection of acetazolamide, the blood flow in the PVL areas did not change but the blood flow in the cerebral cortex and the cerebral white matter increased in comparison with that before the intravenous injection of acetazolamide.

Table 2 shows the mean and standard deviation of the regional cerebral blood flow before the injection of acetazolamide and the increase rate in the blood flow by the intravenous injection of acetazolamide. Before the intravenous injection of acetazolamide, the blood flow in the cerebral cortex, the cerebral white matter and the periventricular white matter was significantly lower in the patients with PVL than in the healthy controls. The intravenous injection of acetazolamide did not increase the blood flow in the PVL areas but increased the blood flow in the other areas. Blood viscosity did not show any significant difference between the patients with PVL and the healthy controls.

Table 2

The regional blood flow (ml/100 g/min) before the injection of acetazolamide and the increase rate (%) in the blood flow by the intravenous injection of acetazolamide (mean  $\pm$  standard deviation)

	Patients with periventricular lucencies		Healthy controls	
	Blood flow	Increase rate	Blood flow	Increase rate
Frontal cortex	41.8 $\pm$ 6.7 *	62 $\pm$ 9	62.8 $\pm$ 8.5	54 $\pm$ 8
Temporal cortex	44.1 $\pm$ 7.2 *	64 $\pm$ 8	65.2 $\pm$ 8.2	58 $\pm$ 9
Parietal cortex	43.3 $\pm$ 6.4 *	65 $\pm$ 9	59.5 $\pm$ 7.9	55 $\pm$ 8
Occipital cortex	42.7 $\pm$ 8.1 *	64 $\pm$ 7	57.3 $\pm$ 7.6	57 $\pm$ 7
Frontal white matter	21.4 $\pm$ 5.9 *	25 $\pm$ 9	26.6 $\pm$ 6.4	42 $\pm$ 8
Temporal white matter	23.1 $\pm$ 5.8 *	48 $\pm$ 7	28.1 $\pm$ 6.7	52 $\pm$ 7
Parietal white matter	22.5 $\pm$ 6.1 *	42 $\pm$ 8	27.7 $\pm$ 6.3	44 $\pm$ 9
Occipital white matter	21.8 $\pm$ 6.2 *	38 $\pm$ 7	26.2 $\pm$ 7.2	46 $\pm$ 8
Periventricular white matter	18.6 $\pm$ 5.2 *	0 $\pm$ 9 **	26.8 $\pm$ 6.9	41 $\pm$ 7

\*  $p < 0.01$ , \*\*  $p < 0.001$  compared with healthy controls.

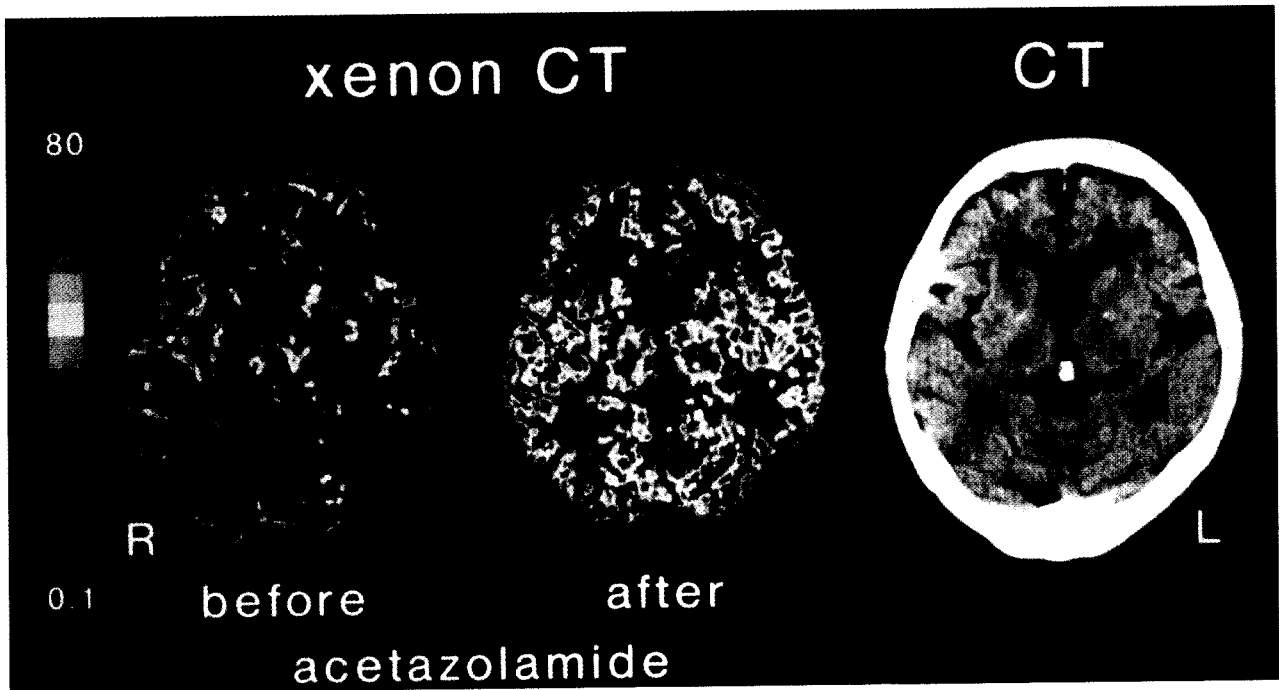


Fig. 3

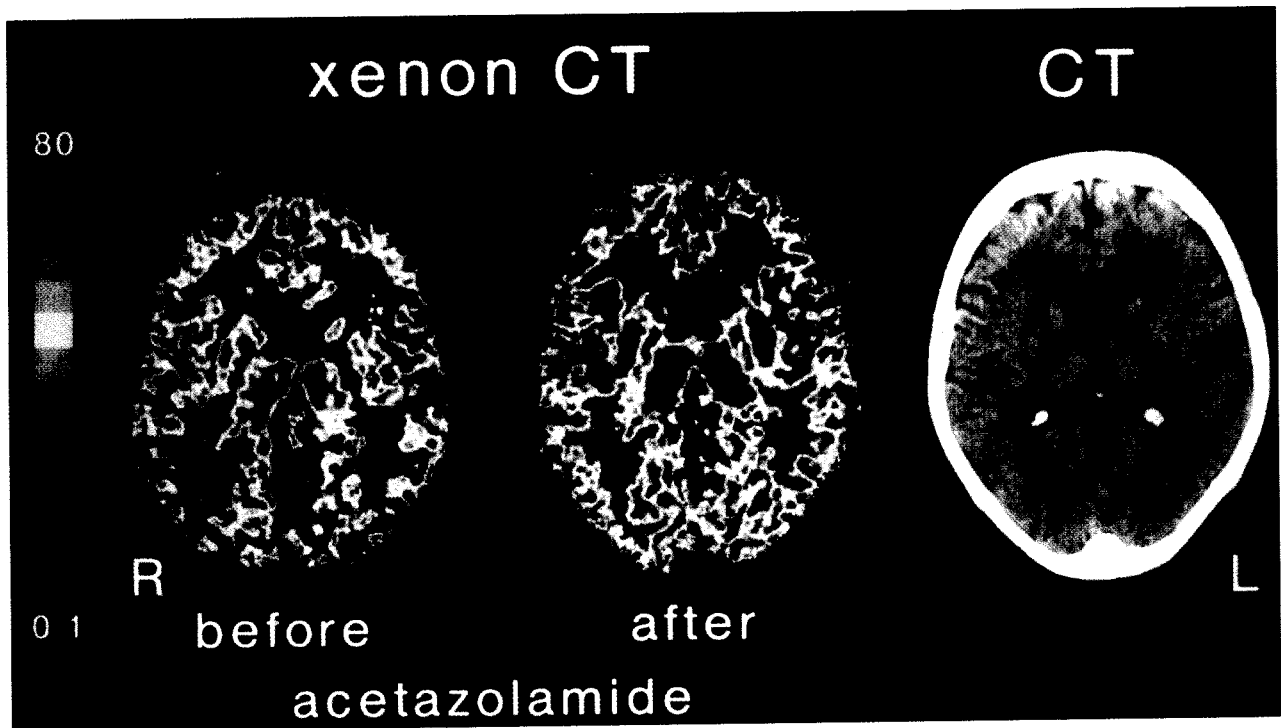


Fig. 4

Fig. 3. Xenon computed tomography in a patient with periventricular lucencies. Before the injection of acetazolamide, the blood flow not only in the PVL areas but also in the cerebral cortex and the cerebral white matter is decreased. The blood flow in the PVL areas was not changed by the injection of acetazolamide.

Fig. 4. Xenon computed tomography in a healthy control.

#### 4. Discussion

Hypertension-caused diffuse arteriosclerosis is considered to be the primary factor in the pathogenesis of PVL in the elderly (Gupta et al., 1988; Van Swieten et al., 1991; Yao et al., 1992). In the present study, the central motor conduction time was prolonged and the blood flow in not only the PVL areas but also in the cerebral cortex and the cerebral white matter was decreased in the patients with PVL in comparison with the healthy controls. This suggests that (1) arteriosclerosis is present not only in the PVL areas but also in the cerebral cortex and the cerebral white matter in the patients with PVL or that (2) the metabolism is decreased not only in the PVL areas but also in the cerebral cortex and the cerebral white matter in the patients with PVL (Turc et al., 1994).

Head magnetic stimulation is considered to stimulate the dendrite of the motor neuron in the cerebral cortex (Thompson et al., 1990) and neck magnetic stimulation is considered to stimulate the brachial plexus (Eisen and Shtybel, 1990). The central motor conduction time measured by magnetic stimulation is the conduction time from the dendrite of the motor neuron to the brachial plexus. Therefore, not only the conduction velocity of the pyramidal tract but also the excitability of the motor neurons may affect the central motor conduction time. Ylikoski et al. (1993) reported that leukoaraiosis could explain some of the intellectual impairment in the elderly, especially that of slowing of distinct motor and attentional functions, as well as slowing of mental processing. Because we excluded the patients with muscle weakness in the present study, the prolongation of the central motor conduction time suggests a subclinical impairment in the conduction velocity of the pyramidal tract and/or the excitability of the motor neurons.

The intravenous injection of acetazolamide improved the central motor conduction time and increased the blood flow except for the PVL areas in patients with PVL. This suggests that the prolongation of the central motor conduction time is at least partly related with the decreased blood flow in the cerebral cortex and the cerebral white matter, but it may or may not be related with the decreased blood flow in the PVL areas.

Acetazolamide, which is a carbonic anhydrase inhibitor, is thought to dilate cerebral blood vessels by increasing the carbon dioxide level (Vorstrup et al., 1984; Bickler et al., 1988; Ringelstein et al., 1992; Frankel et al., 1992). In the present study, the intravenous injection of acetazolamide did not increase the blood flow in the PVL areas. This suggests that cerebrovascular dilatory reserve capacity is decreased in the PVL areas.

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