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CEREBRAL BLOOD FLOW
PROGRESS IN BRAIN RESEARCH
ELSEVIER
38

PROGRESS IN BRAIN RESEARCH
VOLUME 35

CEREBRAL BLOOD FLOW

Relationship of Cerebral Blood Flow and Metabolism to Neurological Symptoms

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ELSEVIER PUBLISHING COMPANY
AMSTERDAM / LONDON / NEW YORK
1972

ELSEVIER PUBLISHING COMPANY
335 JAN VAN GALENSTRAAT,
P.O. BOX 211, AMSTERDAM, THE NETHERLANDS

AMERICAN ELSEVIER PUBLISHING COMPANY, INC.
52 VANDERBILT AVENUE, NEW YORK, N.Y. 10017

LIBRARY OF CONGRESS CARD NUMBER 73-168913

ISBN 0-444-40952-1

WITH 225 ILLUSTRATIONS AND 40 TABLES

PRINTED IN THE NETHERLANDS

PROGRESS IN BRAIN RESEARCH

VOLUME 35

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Studies on Cerebral Circulation by the Ultrasonic Doppler Technique — with Special Reference to Clinical Application of the Technique

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INTRODUCTION

The ultrasonic Doppler technique is advantageous as compared with other techniques for the measurement of cerebral blood flow. The changes in the dynamics of cerebral circulation with various circulatory agents can be detected instantaneously and continuously as well as non-operatively by this technique. In addition, it is possible by this technique to measure blood flow individually in each vessel, *i.e.*, internal, external and common carotid arteries and internal and external jugular veins.

This paper deals with the principle and several clinical applications of the ultrasonic Doppler technique in the study of cerebral circulation and the useful characteristics of the method.

PRINCIPLE AND METHOD

The method was discussed by Satomura and Kaneko (1960), Miyazaki (1963) and Miyazaki and Kato (1965).

When an ultrasonic wave impinges upon a blood stream, the frequency of the reflected waves is altered due to Doppler's effect from the moving blood particles and turbulent (agitated) flow, especially from the former. A kind of noise is obtained by composing and demodulating the reflected waves and the direct wave. The frequency of the noise is proportional to the blood flow velocity.

When an ultrasonic wave impinges upon a moving subject, the frequency of the reflected waves (f') is converted as follows:

$$f' = \frac{c + u \cdot \cos\theta}{c - u \cdot \cos\theta} f$$

where f = frequency of direct wave
c = sound velocity
u = velocity of moving subject
θ = angle between the direction of ultrasonic wave and the direction of moving subject

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The frequency of the Doppler beat (fd) is obtained by composing and demodulating the reflected waves and the direct wave. The frequency of the Doppler beat (fd) is demonstrated as follows:

$$fd = f' - f = \frac{c + u \cdot \cos\theta}{c - u \cdot \cos\theta} f \fallingdotseq \frac{2u \cdot \cos\theta}{c} f = \frac{2u}{\lambda} \cos\theta \qquad (c \gg u)$$

λ = wave length

From the above formula, it has been postulated that the moving subject is converted to an audible sound, since the frequency of the Doppler beat is proportional to the velocity of the subject and that the Doppler's effect is theoretically minimum at $\theta = 90°$ and maximum at $\theta = 0°$.

Fig. 1 and Fig. 2 show the block diagram and the frequency characteristics of the

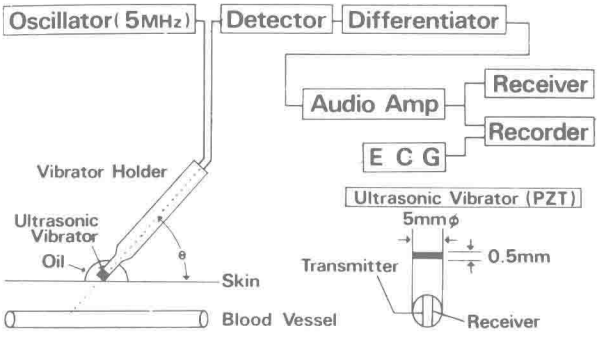

Fig. 1. Block diagram of the apparatus

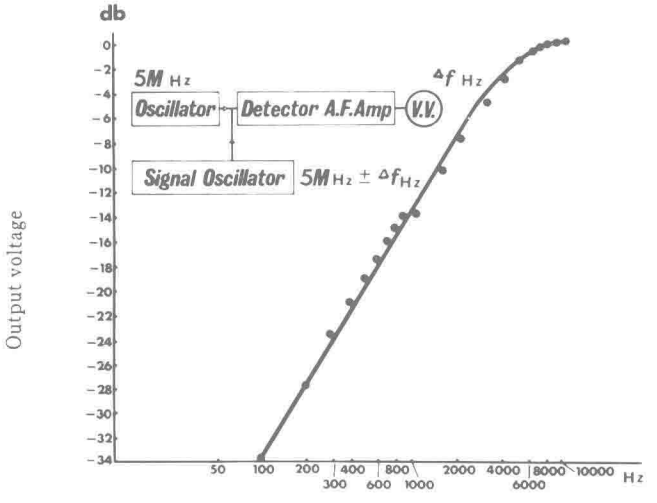

Fig. 2. Frequency characteristics of the apparatus.

apparatus, respectively. The output voltage is proportional to the frequency between about 100 counts/sec and 6000 counts/sec. From these characteristics, the beat due to the Doppler's effect caused by the pulsation of the blood vessels (lower than about 100 counts/sec at arteries) is eliminated.

The rates of cerebral blood flow (ΔCBF), cerebral vascular resistance (ΔCVR) and cerebral oxygen consumption ($\Delta CMRO_2$) are used as a measure of cerebral circulation and metabolism as follows (see Miyazaki, 1966 c; 1968 b).

Rate of cerebral blood flow (ΔCBF) (%) =

$$\frac{CBF' - CBF}{CBF} \times 100 = \frac{A'_{CBF} - A_{CBF}}{A_{CBF}} \times 100$$

Rate of cerebral vascular resistance (ΔCVR) (%) =

$$\frac{CVR' - CVR}{CVR} \times 100 = \left[\left(\frac{A_{CBF}}{A'_{CBF}} \times \frac{MAP'}{MAP}\right) - 1\right] \times 100$$

Rate of cerebral oxygen consumption ($\Delta CMRO_2$) (%) =

$$\frac{CMRO'_2 - CMRO_2}{CMRO_2} \times 100 = \left(\frac{A'_{CBF}}{A_{CBF}} \times \frac{C(A - V)O'_{2'}}{C(A - V)O_2} - 1\right) \times 100$$

where CBF, A_{CBF}, CVR, $CMRO_2$, $C(A\text{-}V)O_2$ and MAP = cerebral blood flow, area of cerebral blood flow pattern, cerebral vascular resistance, cerebral oxygen consumption, cerebral arteriovenous oxygen content difference and mean artery pressure before maneuver, respectively. CBF', A'_{CBF}, CVR', $CMRO'_2$, $C(A\text{-}V)O'_2$ and MAP' = cerebral blood flow, area of cerebral blood flow pattern, cerebral vascular resistance, cerebral oxygen consumption, cerebral arteriovenous oxygen content difference and mean artery pressure after maneuver, respectively.

DYNAMIC OBSERVATIONS OF CEREBRAL CIRCULATORY CHANGE BY VARIOUS CIRCULATORY AGENTS

The change in the pulsatile diameter of the human common carotid artery which synchronizes with the cardiac cycle is so slight as to be negligible (Greenfield *et al.*, 1964; Miyazaki, 1968 a). Therefore, the blood velocity should change in proportion to the blood flow change in the human common carotid artery. From this principle, the changes in the dynamics of the cerebral circulation by various circulatory agents were observed by the ultrasonic Doppler technique as follows.

(*1*) *Effect of low temperatures on cerebral circulation* (Miyazaki, 1966 a)

The blood flow patterns in the internal carotid artery in the same subject observed at different seasons (in March and September) were hemodynamically compared. There were no significant differences between the blood flow patterns of March (room temp., 12 °C) and those of September (room temp., 30 °C), independent of the blood pressure change (Fig. 3). The constancy of the cerebral blood flow at the low

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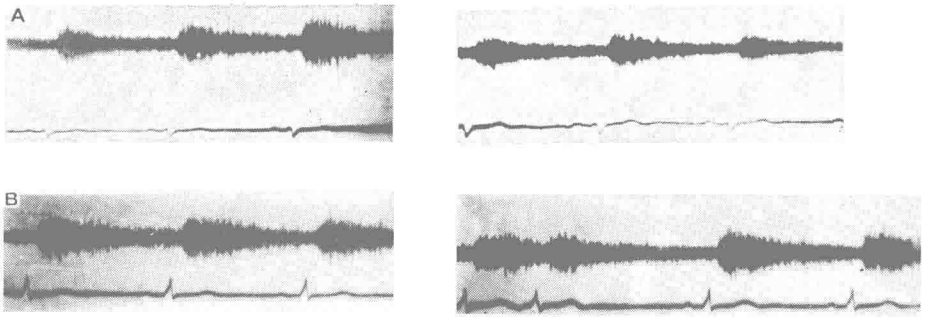

Fig. 3. Effect of low temperature on the blood flow patterns in internal carotid artery in the same subject. Left, indicates blood flow patterns of September and right, of March. (A) no change in blood pressure between two months. (B) markedly increased blood pressure in March (170/80–240/110).

temperature may well be based on the homeostasis of the extra- and intra-cranial circulation.

(*2*) *Effect of induced hypertension on cerebral circulation* (Miyazaki, 1966 a)

The blood flow patterns in the internal carotid artery before and after the administration of vasopressor drugs (adrenalin and noradrenalin) were hemodynamically compared.

The cerebral blood flow was markedly increased after the administration of adrenalin (Fig. 4). This phenomenon would mainly be due to increased cardiac output. On the other hand, two types of the cerebral blood flow were noted after the administration of noradrenalin (Fig. 5). One was a decrease in the blood flow accompanied by an increase in the blood pressure. The other was an increase in the

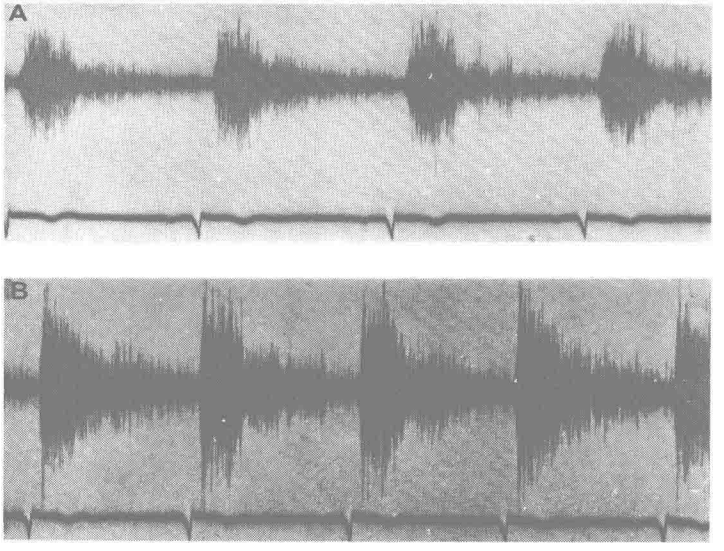

Fig. 4. Effect of induced hypertension by administration of adrenalin on the blood flow patterns in internal carotid artery. (A) control (B.P. 140/50). (B) after administration (B.P. 180/60).

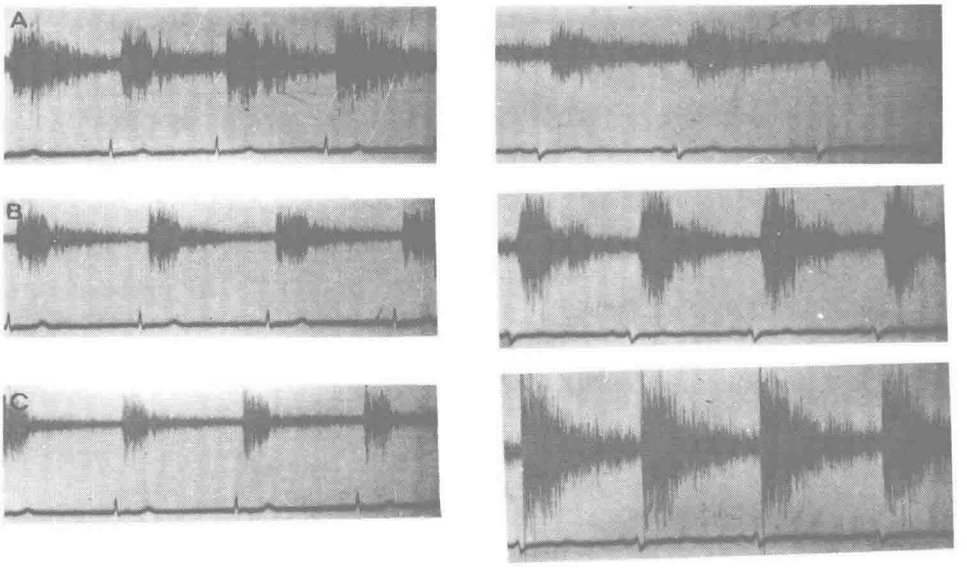

Fig. 5. Effect of induced hypertension by administration of noradrenalin on the blood flow patterns in internal carotid artery. Left, indicates blood flow patterns in a case of decreased blood flow and right, increased blood flow. (A) control, (B, C) after administration. Increasing rate of blood pressure moderate in B and severe in C.

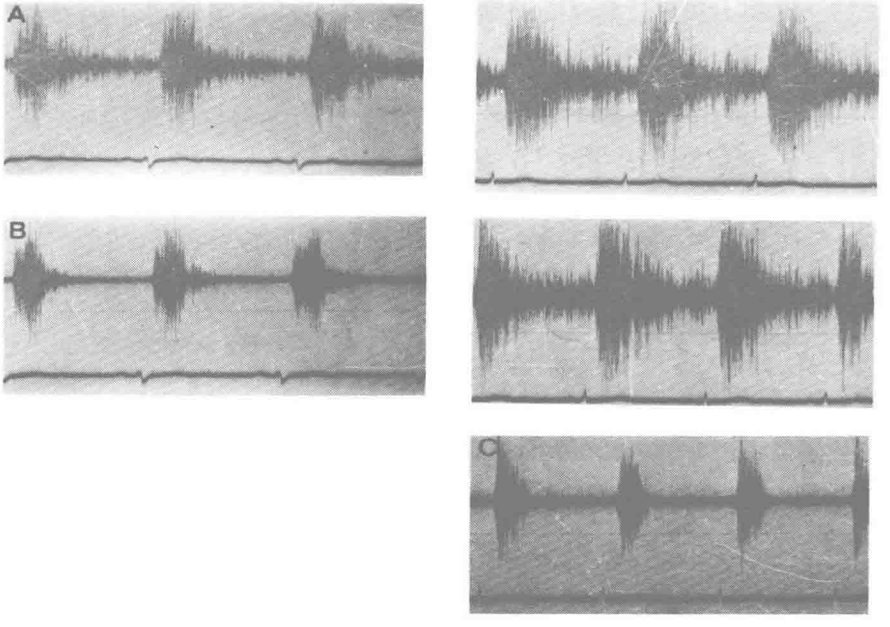

Fig. 6. Effect of induced hypotension on the blood flow patterns in internal carotid artery. Left, indicates blood flow patterns in severe cerebral arteriosclerosis and right, in mild cerebral arteriosclerosis. (A) control, (B) after antihypertensive drug, (C) after antihypertensive drug plus postural change. Decreasing rate of blood pressure moderate in B and severe in C.

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blood flow. The latter type was prone to occur in the aged. This may mainly be due to the difference in the reactivity of the cerebral vessels to noradrenalin.

Thus, the effect of an induced hypertension on the cerebral circulation seems to be dependent on the hemodynamic correlation between the mechanism of hypertension and the reactivity of the cerebral vessel.

(*3*) *Effect of induced hypotension on cerebral circulation* (Miyazaki, 1965 a)

The blood flow patterns in the internal carotid artery were hemodynamically investigated during an induced hypotension by the intravenous infusion of an antihypertensive drug (hexamethonium) and during postural hypotension (supine→upright position) (Fig. 6).

In patients with severe cerebral arteriosclerosis, a decrease in the cerebral blood flow was observed by the intravenous infusion of the antihypertensive drug. On the other hand, in patients with mild cerebral arteriosclerosis, no such decrease was observed by the intravenous infusion of the antihypertensive drug. However, when both the intravenous infusion and the postural change were combined, a definite change in the cerebral blood flow was observed.

The findings suggest the presence of a trend for greater cerebral vascular insufficiency in patients with severe cerebral arteriosclerosis than in patients with mild cerebral arteriosclerosis due to disorder of the cerebral circulatory homeostasis (autoregulation).

(*4*) *Effect of arrhythmia on cerebral circulation* (Miyazaki, 1966 a)

The blood flow patterns in the internal carotid artery were investigated in patients with auricular fibrillation and premature beat (Fig. 7). Normal blood flow patterns were generally observed in cases of either auricular fibrillation or premature beat, *i.e.*, effective systole. However, in the presence of conspicuous arrhythmia such as severe auricular fibrillation or premature beat accompanied by a very short coupling,

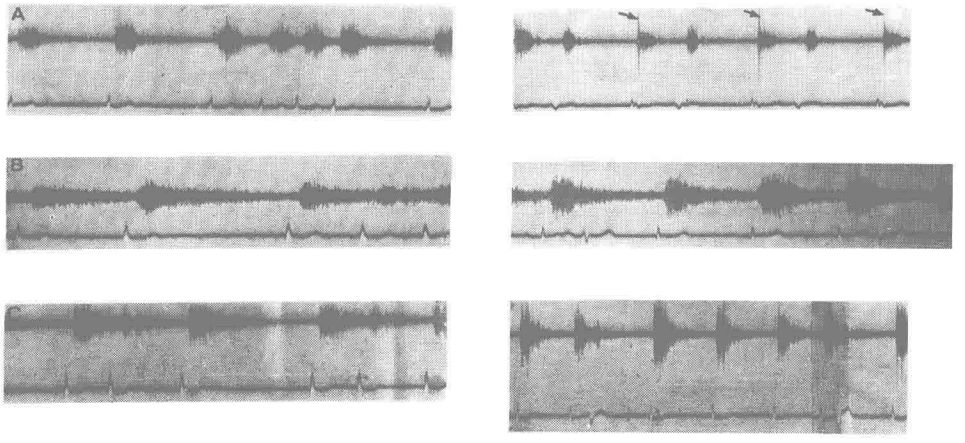

Fig. 7. Effect of arrhythmia on the blood flow patterns in internal carotid artery. Left, indicates blood flow patterns in auricular fibrillation and right, in premature beat. (↓) indicates the Doppler beat due to the pulsation of blood vessel.

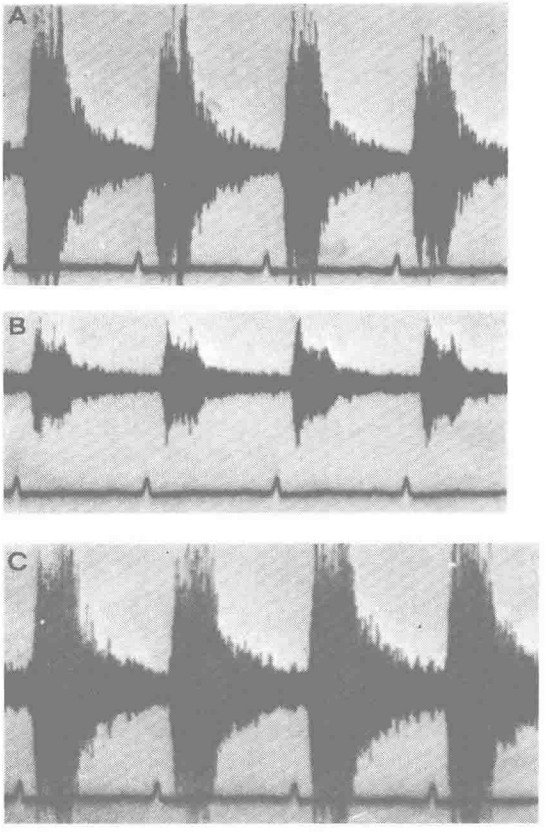

Fig. 8. Effect of aminophylline on the blood flow patterns in internal carotid artery, 81 years, B.P. 250/110. (A) control (B) during administration (C) after administration.

the cerebral blood flow was frequently decreased or completely ceased, *i.e.*, ineffective systole. The findings appear to be related to the elucidation of the mechanism of the Adams–Stokes syndrome.

(*5*) *Effect of circulatory drugs on cerebral circulation* (Miyazaki, 1966 b)

The ultrasonic Doppler technique is most appropriate for the investigation of the effect of cerebral circulatory drugs. For example, the effect of theophylline ethylenediamine and nicotinic acid on cerebral circulation were as follows.

First, the hemodynamic change in the internal carotid artery was investigated before, during and after the intravenous administration of theophylline ethylenediamine (250 mg) dissolved in a 5% glucose solution (20 ml). It was found that the effect of the drug on the cerebral circulation was dependent on the administration time, *i.e.*, cerebral vasoconstriction during administration and cerebral vasodilation after administration (Fig. 8). Theophylline ethylenediamine has been most widely used clinically for the treatment of cerebral vascular diseases. Two theories, however, have been presented as to the effect of the drug, *i.e.*, cerebral vasoconstriction and cerebral vasodilation. This experimental conflict as to the effect of theophylline

ethylenediamine on the cerebral circulation may be partly derived from the above pharmacological characteristics of the drug.

Next, the hemodynamics of the extra- and intra-cranial arteries, *i.e.*, the external carotid artery and the internal carotid artery, were observed before, during and after the intravenous administration of nicotinic acid (20 mg) dissolved in a 5% glucose solution (20 ml). A contrasting circulatory effect between the extra- and intra-cranial arteries was observed dependent on the facial blushing in general, *i.e.*, when the facial blushing is conspicuous, there were observed an increase in the blood flow and a

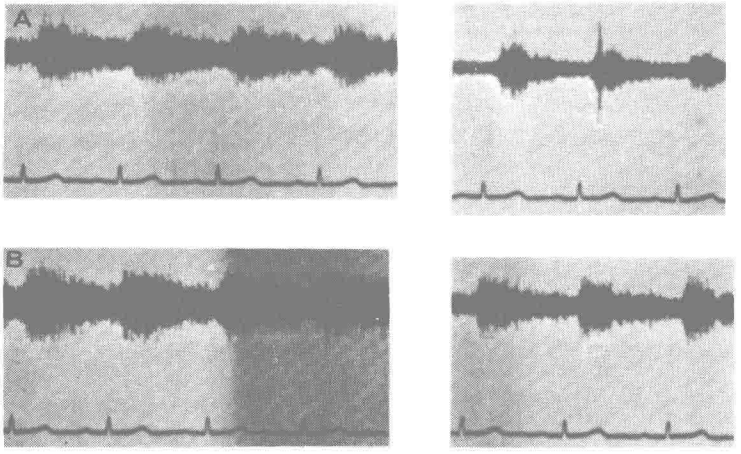

Fig. 9. Effect of nicotinic acid on cerebral circulation. A case with facial blushing (hemiplegic old man 60 years, B.P. 120/60). Left, indicates blood flow patterns in the internal carotid artery and right, in the external carotid artery. (A) control, (B) after administration.

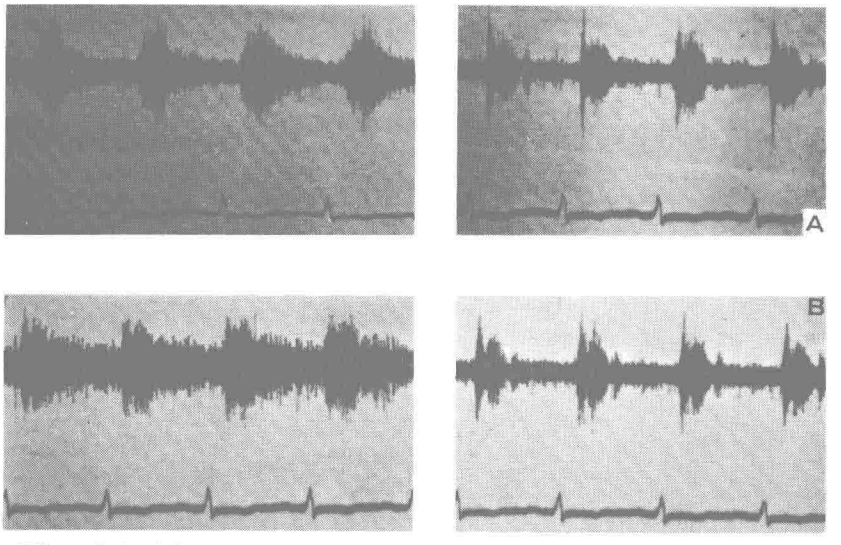

Fig. 10. Effect of nicotinic acid on cerebral circulation. A case without facial blushing (hypertensive old man 61 years, B.P. 210/80). Explanation of the patterns is the same as in Fig. 9. This case shows mild increase of blood flow in internal carotid artery without facial blushing.