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**Internal carotid artery: relationship among arterial caliber,
haematocrit and cerebral vascular resistance,
investigation in eighty healthy men by echodoppler**

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Key words: Carotid artery, Cerebral vascular Resistance, Haematocrit, Echodoppler

SUMMARY

Eighty healthy male subjects (age range: 47 to 87) with hematocrits of 48% or greater and no vascular or hematologic pathology were studied. In each subject, the following values were determined: hematocrit, blood pressure (measured in both arms by sphygmomanometry), and the calibers and blood flow *velocities* of both internal carotid arteries. For the last 2 measurements, doppler ultrasound was used to examine the extracranial portion of the arteries before, 20 minutes after, and 7 days after euvolemic hemodilution.

Following hemodilution, all the subjects demonstrated a statistically significant increase of the diastolic blood flow *velocity* of the two carotid arteries ($p < 0.01$). This corresponded to a statistically significant reduction of the hematocrit ($p < 0.01$). When the data were compared, no statistically significant relationships were observed among arterial caliber, side (left or right), blood pressure, and age.

After 7 days, the results were practically unchanged. The precise correlation between the decrease of the hematocrit and the increase of the diastolic blood flow *velocity* of the internal carotid artery indicates that the latter is a valid index of cerebral vascular resistance. Thus, measurement of the diastolic blood flow velocity is a valid method for the assessment of cerebral blood flow, since the hematocrit is the single most important factor in the determination of blood viscosity.

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INTRODUCTION

It is well known that cerebral blood flow is directly proportional to perfusion pressure and inversely proportional to cerebral vascular resistance. Perfusion pressure is defined as the difference between the mean arterial pressure and the sum of the venous and intracranial pressures. However, the cranium is a rigid structure, and under physiological conditions both the venous and intracranial pressures are virtually negligible (5 mm Hg and 10 cm of H₂O, respectively). Thus, for all practical purposes, perfusion pressure may be considered equal to mean arterial pressure (Prencipe, 1978).

Cerebral vascular resistance reflects a combination of the factors that oppose the flow of blood. Since intracranial pressure is negligible, blood viscosity and arterial caliber must be taken into consideration. Blood viscosity is only slightly influenced by variations of temperature and the lipoprotein concentration of plasma, but is markedly affected by the number of red blood cells. This factor is reflected by the hematocrit.

The influence of arterial caliber on cerebral vascular resistance is difficult to assess. Kanzow (1969) has estimated that the resistance between the aorta and the middle cerebral artery constitutes 20% of the total resistance. Furthermore, a reduction of caliber of at least 80% would be necessary to affect the cerebral blood flow.

With respect to physiological stimuli that can alter cerebral vasomotor tone, it is known that a diminution of arterial pressure accompanies vasodilation and that an increase accompanies vasoconstriction. Hence, since an increase of the cerebral vascular resistance is proportional to the increase of arterial pressure, cerebral blood flow remains unchanged, even in the face of wide variations of arterial pressure. This is considered an autoregulatory phenomenon (Agnoli, 1969).

The hematocrit plays a particularly important role. It is not by chance that patients with polycythemia have been noted to have a greater than 50% reduction of cerebral blood flow (Prencipe, 1978). Furthermore, following weekly phlebotomy to a hematocrit of 45%, an increase of cerebral blood flow has been observed in patients with polycythemia, both with an elevated and a normal erythrocyte mass (Humphrey, 1979). Thomas (1979) has reported that a reduction of the hematocrit from a mean of 53.6% to 45.5% has been shown to increase cerebral blood flow by 73% and to reduce whole blood viscosity by 30%.

There also seems to be a relationship between cerebral infarct size and hematocrit (Harrison, 1981). Moreover, it has been observed that the incidence of cerebral infarct increases from 6.5% in subjects with a hematocrit less than 30% to as much as 63.5% in subjects with a hematocrit greater than 51% (Tohgi, 1981).

Hemodilution appears to be the only method that can increase mean cerebral blood flow up to 50% (Thomas, 1977).

By determining of the diastolic blood flow velocity of the internal carotid

artery, doppler ultrasonography permits the evaluation of variations of resistance to blood flow to the brain.

Diastolic blood flow velocities in the internal carotid artery, which supplies a low-resistance territory, have been reproducibly reported. Risberg (1980) has already correlated diastolic velocity in the internal carotid artery with hemispheric blood flow. He has reported that higher values of diastolic velocity accompany higher blood flow values.

The purpose of this study is to confirm the relationship among the diastolic velocity of the internal carotid artery, the hematocrit, and the cerebral blood flow. Furthermore, we want to confirm that a reduction of the hematocrit (and thus a reduction of cerebral vascular resistance) corresponds to an increment of the carotid diastolic blood flow velocity and that a reduction of the hematocrit is therefore related to an increase of cerebral blood flow.

MATERIAL AND METHODS

We have examined 80 healthy male subjects (age range: 47 to 87) with nonvascular or hematologic pathology. Their hematocrits were 48% or greater. In each subject, the following parameters were determined: hematocrit, blood pressure in both arms, caliber and blood flow velocities of Dx and Sx internal carotid arteries 1 cm after their origin. The carotid artery measurements were performed before and 20 minutes after phlebotomy of 350 cc of blood, which was immediately followed by the intravenous reinfusion of 350 cc of saline solution. These parameters were reassessed after 7 days.

We chose male subjects because of the difficulty of finding enough women with hematocrits high enough to tolerate phlebotomy. A Compur M1100 minicentrifuge was used to determine the hematocrit. A mercury sphygmomanometer was used to measure blood pressure in both arms. When the two measurements were discordant, the higher value was used. An Acuson 128XP echodoppler ultrasound unit was used to measure vessel caliber and blood flow velocity.

RESULTS

Following euvoletic hemodilution, all the subjects demonstrated a statistically significant reduction of the hematocrit ($p < 0.01$). This was accompanied by a statistically significant increase of the diastolic velocity of the internal carotid arteries ($p < 0.01$). No statistically significant relationships were observed between these data and the arterial caliber, although the arterial caliber was inversely proportional to the systolic and diastolic blood flow velocities in all the subjects.

Variations of blood pressure had minimal effects, which were not statistically significant. Therefore, the systolic velocity did not influence the results of this study. Statistically significant relationships were not observed among variations of diastolic blood flow velocity, hematocrit, side, and age. Even after 7 days, the mean

values of the hematocrit and diastolic blood flow velocity were lower. The difference with the initial values was still statistically significant ($p < 0.01$).

Table 1 shows the mean hematocrits and the diastolic blood flow velocities of the right and left internal carotid arteries at the beginning of the study, 20 minutes following euvolemic hemodilution, and 7 days later. Figures 1 and 2 demonstrate the variations of hematocrit in the right and left carotid arteries, respectively, 20 minutes and 7 days following hemodilution. Figure n. 3 illustrates the variations of the hematocrit in all the subjects 20 minutes and 7 days following phlebotomy and hemodilution.

DISCUSSION AND CONCLUSIONS

The results of this study confirm the importance of the hematocrit in the cerebral blood flow. In fact, the statistically significant increase of diastolic blood flow velocity as measured by doppler ultrasound following a statistically significant reduction of the hematocrit by hemodilution corresponds to a reduction of the

TABLE 1 - The mean haematocrits and the diastolic blood flow velocities of the right and left internal carotid arteries at the beginning of the study, 20 minutes following euvolemic haemodilution and 7 days later.

	Basal	20 min	7 days
HCT %	52.1 ± 1.2	47.4 ± 2.1	46.9 ± 2.2
RIGHT I.C. D.V. (cm/sec)	9.9 ± 1.9	14.5 ± 1.8	16.0 ± 2.4
LEFT I.C. D.V. (cm/sec)	12.7 ± 2.8	17.0 ± 2.9	18.3 ± 2.3

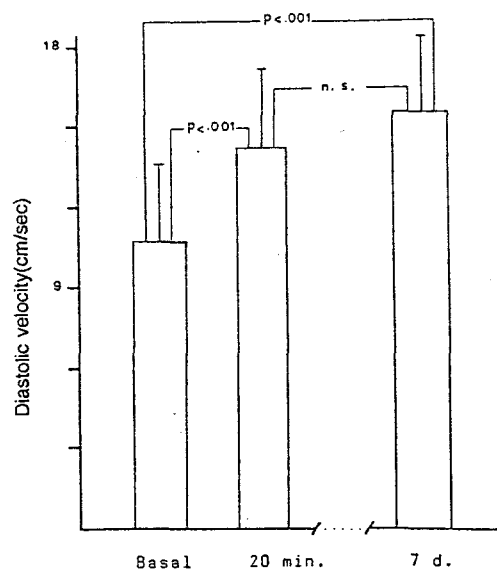


Fig. 1 — Variations of diastolic velocity after haemodilution (right internal carotid).

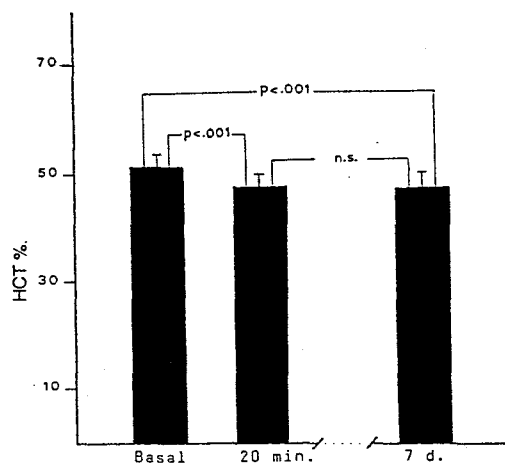


Fig. 2 — Variations of diastotic velocity after haemodilution (left internal carotid).

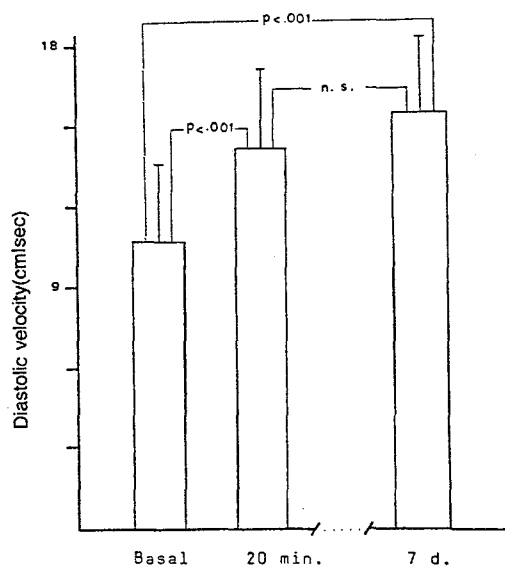


Fig. 3 — Variations of HCT values after haemodilution.

cerebral vascular resistance. This is equivalent to an increase of the cerebral blood flow. The results demonstrate that variations of the diastolic blood flow velocity of the internal carotid arteries are correlated almost exclusively with the reduction of hematocrit, even when one considers that there were no statistically significant variations of blood pressure following hemodilution. Furthermore, in comparison with the cerebral vascular resistance (and thus, cerebral blood flow), even statistically significant changes of the blood pressure would not influence the phenomenon of autoregulation.

It may thus be affirmed that an increase of diastolic blood flow velocity corresponds to an increase of blood flow. Important information about cerebral blood flow may be derived from a simple, noninvasive, and reproducible method. Doppler ultrasonography is useful for the assessment of large groups of patients at risk for whom an increase of the cerebral vascular resistance (and thus a diminution of the diastolic blood flow velocity correspond to a reduction of blood flow.

The results of this study justify the increasingly accepted role of hemodilution in certain pathological situations of blood hyperviscosity. Such condition may antagonize the transport and exchange of O₂ from the blood to tissues at the level of the microcirculation (Menil *et al.*, 1983). These may damage the cerebral tissues (Bartoli *et al.*, 1989). Furthermore, such damage has been observed to increase the risk of deep venous thrombosis (Bartoli *et al.*, 1989).

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REFERENCES

- Agnoli A. 1969. L'autoregolazione della circolazione cerebrale. *Rec Progr Med* 46: 29.
- Bartoli V, Mannini L, Fantoni F, Lupi C. Rapporti tra viscosità ematica e patologia ischemica d'organo. *Res Clin Lab* 19 (Suppl 1). 1989: 4151.
- Menil EW. 1983, Rheology of blood and flow in the microcirculation. *J Appl Physiol.* 18: 255.
- Harrison MG, 1981. effect of haematocrit on carotid stenosis and cerebral infarction. *The Lancet* 18, juli, 114-115.
- Humphrey PR, 1977. Cerebral blood flow and viscosity in relative polycythaemia. *The Lancet* oct. 27, pg. 873-877.
- Kanzow E, 1969. On the location of the vascular resistance in the cerebral circulation in cerebral blood flow. *Cerebral blood flow* pg. 96, Ed. Springer Verlag.
- Menil...
- Prencipe M, 1978. Moderne acquisizioni sulla fisiopatologia della circolazione cerebrale e sulle ischemie cerebrali. Ed Scientifiche Biomedica Foscama, Roma.
- von Reutern GM, Budinggen HJ, 1992. Sonografia doppler extra ed intracranica. Centro scientifico ed, Torino.
- Risberg J, Smith P, 1980. Prediction of Hemispheric Blood Flow from carotid velocity measurements. *Stroke*, vol II, 4: 68-73.
- Tohgi H, 1981. Importance of the haematocrit as a risk factor in cerebral infarction. *The Lancet* juli 23, pg 161-163.
- Thomas DJ, 1977. Effect of haematocrit on cerebral blood flow in man. *The Lancet*, nov 5, 941-943.

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