Acta Neurochirurgica 38, 187–194 (1977)

ACTA
NEUROCHIRURGICA

Department of Neurosurgery, National Hospital of Norway, Rikshospitalet, Oslo, Norway (Head: Prof. Dr. med. T. Hauge)

Precerebral Arterial Blood Flow Pattern in Intracranial Hypertension With Cerebral Blood Flow Arrest

By

H. Nornes, B. Angelsen, and **K.-F. Lindegaard**

With 6 Figures

Summary

A pulsed echo Doppler flowmeter was used to confirm cerebral flow arrest in patients with intracranial hypertension resulting in a cerebral perfusion pressure of zero or close to zero. The cerebral angiograms showed non-filling. The term brain tamponade is used for this condition. The patients had a mean arterial blood pressure at the time or recording ranging from 70 to 160 mmHg. Under these conditions the transcutaneous recording of internal carotid artery flow velocity immediately distal to the carotid bifurcation showed a reverberating flow pattern with marked pulsations. The forward flow in systole was counterbalanced by the retrograde flow during diastole resulting in a net flow of zero. This flow pattern is basically caused by a combination of intracranial vascular obstruction, the arterial blood pressure at the site of measurement, and finally the compliance of the distal arterial segment as well as the intracranial compliance. This non-invasive method is of potential value in the determination of intracranial flow arrest.

Key words: Cerebral blood flow arrest, brain tamponade, pulsed echo Doppler flowmeter, reverberating flow.

The paradox of a dead brain in a living body has been known for many years [14]. It is now widely accepted in modern medicine that the time of death of a person is the moment when there is incontrovertible evidence of total brain destruction with complete and irreversible cessation of all the functions of intracranial nervous tissue. The certification of death due to total brain destruction is based on neurological examination [7, 14, 15], isoelectric EEG, and the demonstration of arrest of the cerebral blood flow (CBF) [2, 5, 6, 12, 13, 14, 15].

The effect of increased intracranial pressure (ICP) on the CBF

is determined by the fact that cerebral perfusion pressure (CPP) equals the difference between the systemic arterial blood pressure (ABP) and the ICP [3, 6, 10, 12]. This is the fundamental principle for the development of ischaemic brain damage in intracranial hypertension [6]. Riishede *et al.* [12] were the first to demonstrate in angiograms the nonfilling of cerebral arteries due to increased intracranial pressure. This method is now considered essential for the

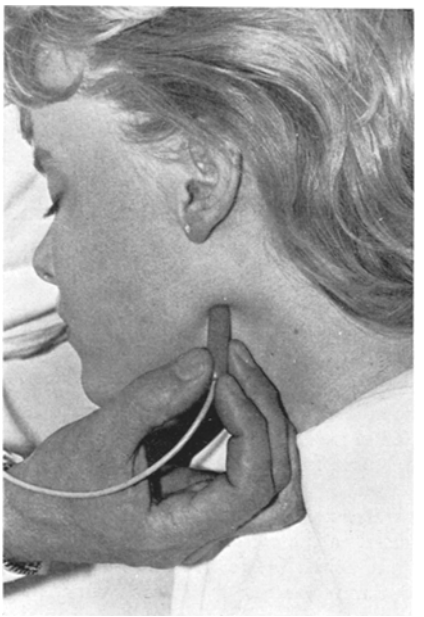

Fig. 1. Ultrasound probe position below mandibular angle for the detection of signals from the internal and external carotid arteries

certification of death in patients with a beating heart [14, 15]. Brock *et al.* [2] have discussed the use of radioactive tracers for determining the death of a person with reference to organ transplantation. They consider lasting absence of clearance of tracer deposited within the brain to demonstrate total absence of flow through it and thus to prove cerebral death. We have previously described the findings of zero CPP and arrest of internal carotid artery (ICA) blood flow following extreme intracranial hypertension [10]. The term brain tamponade [15] is descriptive of this picture.

This paper presents results obtained with a non-invasive blood flowmeter to show the reverberating flow in precerebral arteries under such conditions.

Methods

A pulsed echo Doppler flowmeter operating at 6 MHz was used [1]. This instrument allows the determination of blood flow velocity non-invasively. The depth at which the echo is reflected can be set with high accuracy by varying the receiving gate. The instrument is also able to discriminate with regard to flow direction, and the true zero-line can easily be obtained electronically. The flow velocity at each point is determined by the Doppler frequency shift of each pulse and presented in the records as the average velocity across the vessel lumen. The

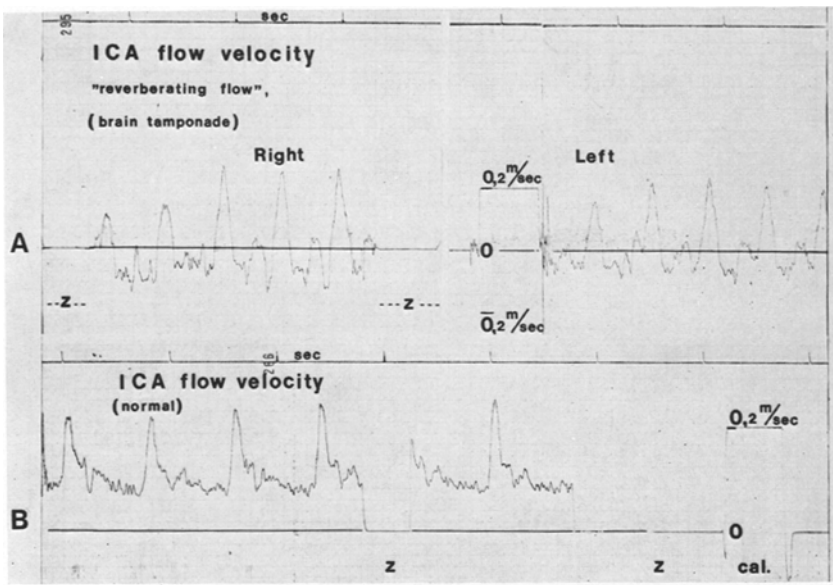

Fig. 2. Pulsatile internal carotid artery (ICA) flow velocity in a 48 years old woman with subarachnoid haemorrhage. No net forward flow. Lower tracing (B) showing normal ICA flow velocity (from control series). Z zero control

flowmeter has a bandwidth of approximately 25 Hz. The angle of the ultrasound beam to the vessel was kept at about 45°. If the inner diameter of the vessel is known the volume flow can be calculated by multiplying lumen area (A) by the time-average velocity (V). In this study flow velocity alone was considered. Pulsatile flow velocity as well as electronically integrated mean flow velocity could be recorded on an Elema ink jet recorder. The ICP was recorded with an epidural pressure transducer [10] in three patients and in conjunction with ABP measurements this was used for the estimation of zero perfusion pressure. In two patients, not subjected to Doppler flow studies, electromagnetic blood flow measurements on the ICA were made as previously described [10] and data are presented for comparison.

Four channel cerebral angiography was used in all cases to confirm cessation of CBF, each series covering 18 seconds.

Material

A control series of 15 healthy adults was used for testing the pulsed echo Doppler technique on the precerebral arteries. The study was conducted in 11 patients (age 29 to 58 years, average 44) with extremely high intracranial pressure due to subarachnoid haemorrhage from a saccular aneurysm (6), head injury (3), and intracranial tumour (2). The arterial blood pressure (ABP) at the time of recording ranged from 70 to 160 mmHg mean.

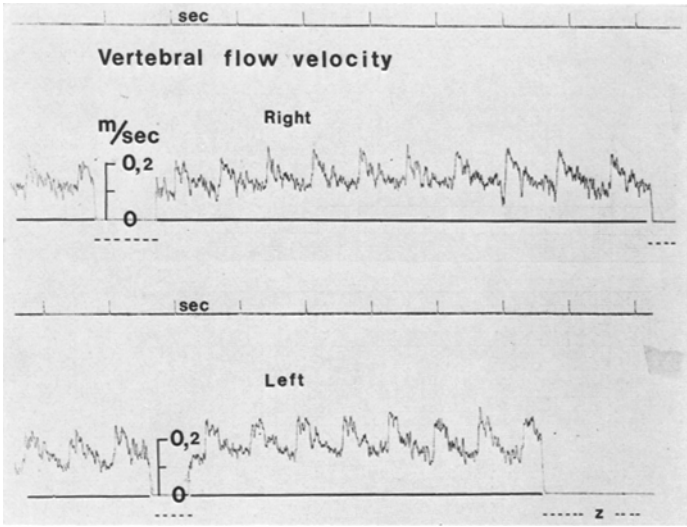

Fig. 3. Vertebral artery blood flow velocity in a 32 years old healthy man (control series). Probe position at atlanto-occipital level

Control series. The internal and external carotid arteries are closely related immediately distal to the bifurcation in the neck. The two arteries could be identified in all individuals on the basis of vessel anatomy and their specific flow velocity patterns. Fig. 1 shows the probe position distal to the mandibular angle, and Fig. 2, lower tracing, gives an example of normal ICA flow with comparatively high end-diastolic flow velocity. This is in contrast to the low diastolic flow in the external carotid artery (ECA) which is due to the different conductivity characteristics of the two arterial systems which will be discussed elsewhere.

Satisfactory recordings from both carotid systems bilaterally were obtained in all individuals studied and the findings were easily reproducible. Attempts were made to register vertebral artery flow

velocity either at the atlanto-occipital level or at the vessel entrance into the vertebral column at the level of the sixth cervical vertebra. An example is given in Fig. 3. However, in only 3 of the 15 persons studied was it possible to obtain acceptable records from one or both vertebral arteries.

Intracranial flow arrest series: Satisfactory recordings were made bilaterally in all patients from the carotid arteries, and a typical blood flow velocity pattern in the ICA was observed in all 11 patients

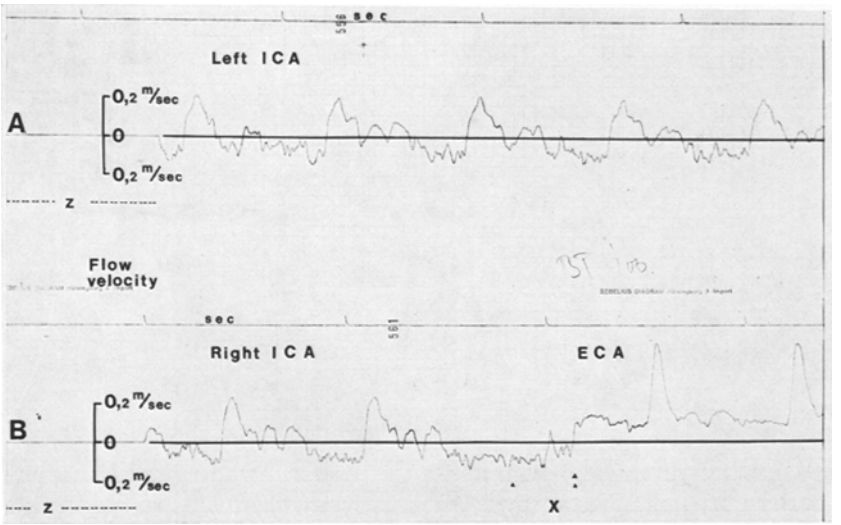

Fig. 4. Bilateral ICA flow velocity showing no net flow in a 56 years old woman (subarachnoid haemorrhage). Lower tracing from X shows the normal external carotid artery (ECA) flow pattern

with arrested cerebral circulation confirmed later by non-filling in the angiograms. Fig. 2, upper tracing, shows marked flow pulsations but the net flow is zero. The tracing areas above and below zero line counterbalance each other within each cycle thus demonstrating that the forward flow of blood in systole is equal to the backward flow in diastole. This represents a marked contrast to the normal ICA flow. By changing the probe angle to the skin surface, the external carotid artery flow was easily detected as shown in Fig. 4, lower tracing right. A patient, not incorporated in this series, had a subarachnoid haemorrhage leading to death within a few hours. Intracranial pressure was measured with an EDP transducer and ICA flow was recorded with an implanted electromagnetic flowmeter probe in

the neck. Fig. 5 is presented for comparison and shows mean forward flow approaching zero. Diastolic flow is negative.

Vertebral artery flow velocity was succesfully recorded in only 2 of the 11 patients. This was due to the difficulties in reliably locating and recording flow from this artery. Fig. 6 shows the vertebral artery flow pattern in a patient with complete arrest of intracranial blood flow after a head injury with brain laceration and haemorrhage.

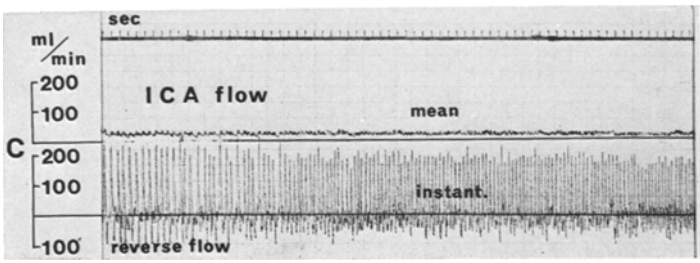

Fig. 5. From a 33 years old man with current subarachnoid haemorrhage from a saccular aneurysm. Mean forward ICA flow approaching zero within 40 seconds. Recorded with an electromagnetic flow probe. Note marked flow pulsations (instant flow) and the reverse (negative) flow in diastole. Death a few hours later

Discussion

Energy must be delivered to the system at the right rate and pattern to maintain normal blood flow through an artery. When blood is made to flow through an artery a certain pressure is needed at the entrance. When the pressure is raised within an elastic vessel the vessel walls become deformed. This requires energy which is converted to potential energy stored in the vessel wall. This energy stored during systole is used for the transport of blood in diastole and was described by Frank [4] as the Windkessel effect. In the described clinical situation, usefully termed brain tamponade, the resistance to flow is practically infinite and net blood flow is zero. The internal carotid artery has no branches before its entry into the skull and with brain tamponade only minute negligible flow through extradural branches can be maintained. However, the vessel segment from the neck to the cerebrum possesses a certain compliance or distensibility, which is a measure of the ability of a hollow structure to change its volume. It is generally given by the ratio of volume change to pressure change.

The main determinants of the flow velocity pattern observed in this study are the arterial blood pressure at the inlet (site of measure-

ment) and the compliance of the distal arterial segment. However, in this situation with an intracranial blockade due to increased ICP, the intracranial compliance obviously plays an important role. This is sometimes seen in serial angiograms or cineangiographic studies where the end of the contrast plug at about dura level shows a back and forth movement.

The hindrance to intracranial filling and flow causes the energy stored in the vessel wall to reverse blood flow in the postsystolic

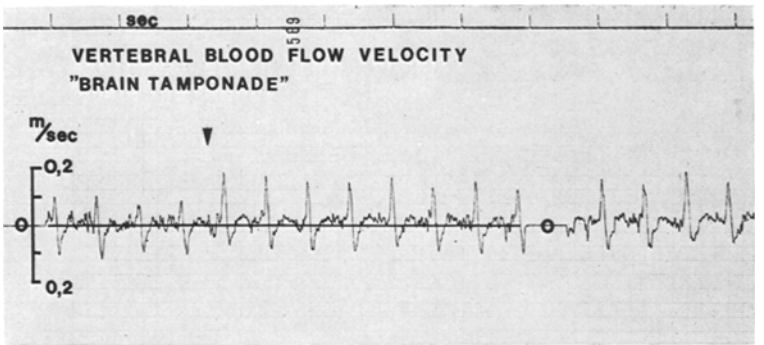

Fig. 6. Vertebral artery blood flow velocity in a 28 years old man with brain tamponade due to head injury with brain laceration and edema. No net forward flow

period when the systemic blood pressure is falling. The term reverberating flow is descriptive of such flow conditions.

Similar observations on reverberating blood flow were made by using the same recording technique on the proximal part of the common carotid artery in patients with marked stenosis or occlusion of the external and internal carotid arteries at the bifurction [9].

The question arises of whether this method should be considered reliable for the diagnosis of brain tamponade. Further studies are essential before this can be answered because of particular problems with vertebral flow recording in this technique. The necessity for a four-channel vessel investigation as in the angiographic verification of a complete cessation of intracranial blood flow must also be applied to this method. However, our experience so far is that the method provides valuable information for the timing of the final angiography and is an additional safeguard in the determination of permanent cessation of cerebral blood flow. Furthermore, the findings demonstrate that blood flow studies should not, if possible, be limited to the registration of volume blood per unit time since the study of how blood is being transported may add valuable information.

References

1. Angelsen, B. A. J., Analog estimation of the maximum frequency of doppler spectra in ultrasonic blood velocity measurements. The Division of Engineering Cybernetics, The Norvegian Institute of Technology, Trondheim. N. 76-21-W. 1976.
2. Brock, M., Schürmann, K., Hadjidimos, A., Cerebral blood flow and cerebral death. Acta Neurochir. (Wien) *20* (1969), 195—209.
3. Cappelen, Chr. Jr., Hall, K. V., Electromagnetic blood flowmetry in clinical surgery. Acta chir. scand. Suppl. *368* (1967), 1—27.
4. Frank, O., Die Grundform des arteriellen Pulses. Biol. *37* (1899), 483—526.
5. Heiskanen, O., Cerebral circulatory arrest caused by acute increase of intracranial pressure. Acta Neurol. scand. Suppl. *7* (1963).
6. Langfitt, T., Kassel, N. F., Nonfilling of cerebral vessels during angiography: Correlation with intracranial pressure. Acta Neurochir. (Wien) *14* (1966), 96—104.
7. Lindgren, S., Petersen, I., Zwetnow, N., Prediction of death in serious brain damage. Acta chir. scand. *134* (1968), 405—416.
8. Nornes, H., The role of the circle of Willis in graded occlusion of the internal carotid artery in man. Acta Neurochir. (Wien) *28* (1973), 165—177.
9. Nornes, H., Common carotid artery blood flow velocity in patients with distal occlusive disease. In preparation.
10. Nornes, H., The role of intracranial pressure in arrest of haemorrhage in patients with ruptured intracranial aneurysm. J. Neurosurg. *39* (1973), 226—234.
11. Peronneau, P., Deloche, A., Bui-Mong-Hung *et al.*, Debitmetrie Ultrasonore—Développements et applications expérimentales. Europ. Surg. Res. *1* (1969), 147—156.
12. Riishede, J., Ethelberg, S., Angiographic changes in sudden and severe herniation of brain stem through the tentorial incisure. Arch. Neurol. Psychiat. *70* (1953), 399—409.
13. Tönnis, W., Frowein, R. A., Wie lange ist Wiederbelebung bei schweren Hirnverletzungen möglich. Mschr. Unfallheilk. *66* (1963), 169—190.
14. Walker, A. E., The death of a brain. Johns Hopkins Med. J. *124* (1969), 190—201.
15. Wawersik, J., Kriterien des Todes unter dem Aspekt der Reanimation. Der Chirurg *39* (1968), 345—348.

Author's address: H. Nornes, M.D., Department of Neurosurgery, National Hospital of Norway, Rikshospitalet, Oslo, Norway.