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Hyperaemia Prior to Acute Cerebral Swelling in Severe Head Injuries: the Role of Transcranial Doppler Monitoring

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Summary

Acute cerebrovascular congestion after a closed head injury is significantly related to intracranial hypertension. As an indirect method of cerebral blood flow measurement, transcranial doppler sonography (TCD) provides a rapid and noninvasive assessment of cerebral haemodynamics, including hyperaemic conditions.

TCD examinations was serially performed in 35 patients with severe head injury with intact cerebral circulation; i.e. the mean flow velocity (MFV) patterns of the middle cerebral artery (MCA) did not show signs of cerebral circulatory arrest such as systolic spike, to and fro, or no flow. The results showed that the MFV of the MCAs and ipsilateral extracranial internal carotid arteries (ICAs) in 9 of these patients increased sharply and pulsatility index (PI) decreased during 48–96 hours after the injury. This was soon followed by patterns of high intracranial resistance, consistent with elevated intracranial pressure (ICP) in monitored patients and acute brain swelling on repeated computed tomographic (CT) scans. The correlation between increased MFVs, decreased PIs, and cerebral haemodynamic changes leading to acute brain swelling is discussed.

The number of patients who ended with severe disability, vegetative state, or death was 66% in this group of 9 patients, compared to only 34% for the 35 patients overall with severe head injury. Though the morbidity and mortality rates largely depend on the primary injury, the presence of acute cerebral swelling aggravate the grave course in these patients. And the ability of TCD to monitor the hyperaemic state prior to oedema should lead us to adjust the therapy in order to minimize the secondary insult related to intracranial hypertension.

Keywords: Cerebral hyperaemia; transcranial Doppler; severe head injury; cerebral oedema.

Introduction

Diffuse cerebral swelling after a closed head injury is mainly due to cerebral hyperaemia and subsequent increase in cerebral blood volume, and not due to brain oedema[4, 5, 6, 13, 18, 20, 21]. This acute cerebrovascular congestion or hyperaemia is significantly related to intracranial hypertension and unfavourable outcome[5, 6, 13].

Several methods had been introduced to measure this hyperaemic state, such as by measuring Hunsfield Units on computed tomography (CT)[5, 6, 13, 21], or by studying the cerebral blood flow[5, 18, 19, 21].

The recent development of transcranial doppler (TCD) sonography has provided a rapid and noninvasive assessment of cerebral haemodynamics especially blood velocity and pulsatility in the basal cerebral arteries[1, 2, 9]. Aaslid *et al.*[2] and Hennerici *et al.*[11] recommended the use of mean flow velocity (MFV) values to discriminate normal from abnormal, since it is less dependent on systemic cardiac factors. These measured velocities are proportional to flow in most circumstances[1, 2, 3, 8, 17]. It therefore serves as a continuous index of cerebral blood flow in the measured vessels. Recently, Shigemori *et al.*[19] using TCD sonography found that increase of MFV in TCD recording is strongly related to the development of diffuse cerebral swelling.

Here we would like to present 9 cases of severe brain injury in whom acute increase of MFV and decrease of pulsatility index (PI) values preceded the acute cerebral swelling as clinically assessed and proved by CT examination and/or intracranial pressure (ICP) monitoring. The main purpose of the present study was to examine the value of TCD monitoring in predicting haemodynamic phenomena following severe head injury.

Material and Methods

Patients

54 patients with severe head injury (Glasgow coma scale 8 or less after non surgical resuscitation) were admitted to our emergency unit during four-year-period (April 1988 to March 1992). All patients were evaluated using TCD sonography starting from admission, and then every 12 hours during the critical period and then daily until it was not needed anymore. Among these, 19 patients were admitted with an already compromised cerebral perfusion pressure, in whom TCD showed patterns of intracranial circulatory arrest[10] i.e. systolic spike, to and for, or no flow. This group were excluded from further evaluation. The other 35 patients had their first TCD evaluation between 2 to 6 hours after the trauma, and they were considered as the basis of our study. Among them, 9 patients (26%) were found to have abnormally elevated flow velocity of the middle cerebral arteries, more than 100 cm/second, during their course of treatment, and are presented here. There were 5 men and 4 women and their ages ranged from 4 to 49 years (mean 24 years). Glasgow Coma Scale (GCS) scores after non surgical resuscitation was 5 in 7 patients, 7 and 3 in the other 2 patients. Table 1 details the clinical condition, CT findings, and outcome of these 9 patients.

Examination Technique

TCD studies were performed using a TC2-64 transcranial doppler (EME, Uberlingen, Germany). Transtemporal windows were used for insonation of the middle cerebral arteries (MCAs) in all patients. Simultaneous measurement of distal portion of the extracranial internal carotid artery, high in the neck, were also done in all cases. The measured MFV were displayed in centimeters per second (cm/sec.). Gosling's pulsatility index[8] (peak systolic velocity - end diastolic velocity /mean velocity) for each measurement was also calculated. Doppler intensity was set at 50% or 75% of Spatial Peak Temporal Average (SPTA) for transtemporal insonation, as recommended by the Bioeffect Committee of the American Institute of Ultrasound in Medicine[9]. Carotid arteries in the neck were insonated using 10% or 25% SPTA. TCD examinations were all done during stable haemodynamic conditions and not within one hour after previous ICP therapy. CT scan was performed on admission and then repeated serially at regular intervals and when clinically indicated. ICP was monitored in 5 of these patients, using an epidural fluid filled-device (Nihon Kohden Corporation, Japan), or a fiberoptic device (Camino Laboratories, California, USA). In two patients, carotid angiography was performed at the time when their TCD showed acute increase of MFV.

Results

TCD evaluation of this groups of 9 patients showed that MFVs on admission was 57.8 ± 6.4 cm/sec (mean ± SE). It then quickly increased and peaked during 48 to 96 hours after the injury at 132.4 ± 5.9 cm/sec (mean ± SE). The course of this change for each case is shown in Fig. 1 a.

Table 1

Case	Diagnosis	Admission CT	GCS	Pupillary signs	ICP init.	ICP max.	Outcome
1. M 21	severe DAI	bilat. frontal contusion, rt. temporal thin SDH	5	anisocoric; light reflex bilateral absent	25	35	vegetative
2. M 20	severe DAI	interpedunc. high density; rt. IVH	7	rt. side midriasis; deviation to right	–	–	mild disability
3. F 49	severe DAI	high density spot at corpus callosum and medulla; interhemispheric SAH	3	anisocoric; light refl. bilaterally sluggish; corneal refl. absent	20	80	dead
4. M 4	severe DAI	SAH at lt. Sylvian, ambient, and interhemispheric. HDA at corpus callosum	5	light refl. absent bilaterally; decerebrate posture	22	60	dead
5. F 37	Rt. SDH	rt. SDH; mild midline shift, lt. frontal contusion and ICH	5	isocoric; light reflex suggish bilaterally	25	100	dead
6. M 6	severe DAI	mildly oedematous, spotty HDA at pons, basal gangl.	5	isocoric; light refl. absent bilaterally	–	–	severe disability
7. M 19	severe DAI	multiple small HDA, interhemispheric SAH, Rt. IVH	5	both midriasis; bilat. light reflex absent	–	–	dead
8. F 34	severe DAI	HDA at interpeduncular, lt. Sylvian, and rt. ambient cisterns	5	anisocoric, light reflex rt. absent	–	–	mild disability
9. F 23	severe DAI	lt. parietal spotty HDA	5	anisocoric, light reflex bilateral sluggish	26	29	mild disability

GCS Galsgow coma scale; *ICP* intracranial pressure; *DAI* diffuse axonal injury; *SDH* subdural haematoma; *IVH* intraventricular haematoma; *SAH* subarachnoid haemorrhage; *HDA* high density area; *ICH* intracerebral haematoma.

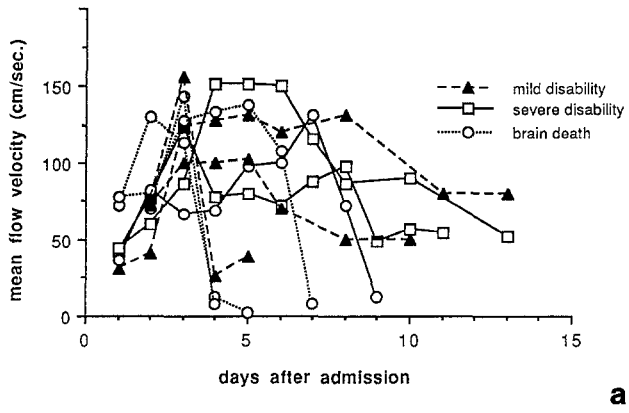

Fig. 1 a. Changes of mean flow velocities of middle cerebral arteries. Abnormally high flow velocities, showing hyperaemic condition, were noted from the second day of admission. They then decreased as oedema has developed. In four cases (open circle) they ended up with cerebral circulatory arrest/brain death

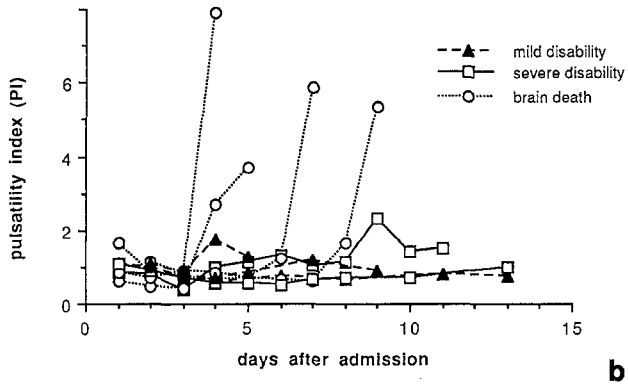

Fig. 1 b. Changes of pulsatility indices. They decreased until the third admission day, then began to increase as oedema has developed, creating a TCD patterns of high intracranial resistance in 7 cases before ended up with brain death in 5 cases

The MFVs of the distal extracranial portion of the internal carotid artery, high in the neck, simultaneously increased to about 72–82 cm/second. Its normal range is 36.3 ± 8.6 cm/second[14].

The pulsatility index (PI) value was 1.0 ± 0.09 (mean ± SE) on admission and soon went down to 0.65 ± 0.06 (mean ± SE) when the MFVs reached their peak values. The course of this change for each case is shown in Fig. 1 b.

These high MFVs were soon followed by patterns of high intracranial resistance, i.e. diastolic flow velocity decreased and systolic peaks became more spiky, in 6 cases, and patterns of cerebral circulatory arrest – leading to brain death – in one case, consistent with elevated ICPs on monitored patients.

Repeat CT scans were taken within 24 H after the presence of this high resistance patterns, and acute swelling was noted from the absence or compression of cerebral cisterns in these 7 cases (Fig. 2). This pattern of high intracranial resistance, observed in 6 patients, quickly worsened into patterns of cerebral circulatory arrest which led to brain death in 3, and normalized in the other 3 cases (case 1, case 8, case 9). Case 1, with severe bilateral frontal contusion, ended up in a permanent vegetative state, while cases 8 and 9 were discharged two months later with mild disability.

The other two patients, case 2 and case 6, whom the high flow velocities on TCD were soon normalized and never had any pattern of high intracranial resistance, showed more favourable pictures regarding oedema on their repeat CTs and on their TCD monitoring. Case 2 was discharged with mild disability, and case 6, probably related to primary contusion of the brainstem, was bed-ridden with severe neurological deficit. The result of ICP monitoring in 4 patients, and all data related to TCD monitoring are listed at Table 2.

Discussion

High flow velocity during TCD measurement has two possible interpretations, an absolute increase of cerebral blood flow (CBF) or a decrease of the vessel diameter without changes of the absolute CBF (compensatory increase of MFV during arterial spasm). Weber *et al.*[22] and Lindegaard *et al*[14] excluded the possibility of intracranial arterial spasm by using the ratio of MCA flow velocity to that of distal extracranial portion of ipsilateral internal carotid artery (ICA) high in the neck. Our data on our 9 patients showed that the MFV of both, the MCAs and ipsilateral ICAs, simultaneously increased sharply during the first 3 days after trauma and they reached more than twice above their initial values. Weber *et al.*[22] assumed a spasm of MCA if the ratio of blood flow velocity in the MCA (VMCA) to the blood flow velocity in the ICA (VICA) exeeded 3 (normal value 1.7 ± 0.4). In our cases, the highest value of this ratio was 2, observed in case 8, while the mean value for all cases was 1.91. Therefore, acute cerebrovascular hyperaemia, rather than spasm, was assumed to occur in these patients. Angiography was performed in case 1 and case 2, during their peak of MFVs, and revealed no arterial spasm.

Rozsa *et al.*[20] studied traumatic brain swelling seen on CT using as measurement the Hunsfield Unit, and found that in the first hours and days after head injury, diffuse swelling was caused more frequently by cere-

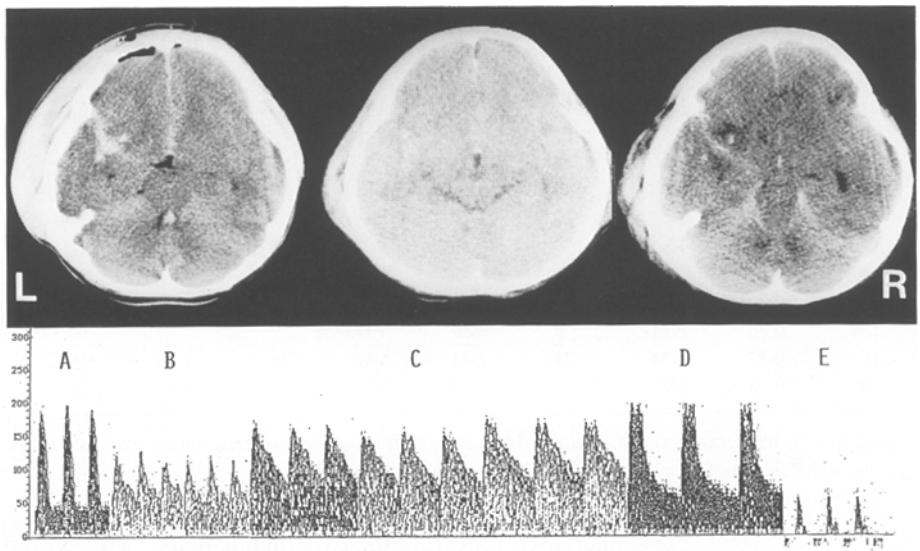

Fig. 2 a. Upper: CT scans of case 4, on admission (left), during the presence of high flow velocity on TCD (middle), and after high resistance pattern appeared on TCD (right). Note that the ambient cistern was initially tight, then loose, and finally obstructed during acute brain swelling. Lower: Results of TCD monitoring in case 4. The high resistance pattern seen on admission (*A*) was improved after non-surgical resuscitation i.e. controlled ventilation and mannitol administration (*B*), then changed into high flow velocity pattern (*C*) on the third admission day, followed by high resistance pattern (*D*) on the fifth day, before quickly progressed into cerebral circulatory arrest (*E*) on the same day. ICP increased progressively from 20 to 60 mmHg when this D pattern was observed. *MFV* Mean flow velocity (cm/s); *PI* pulsatility index; *ICP* intracranial pressure (mmHg)

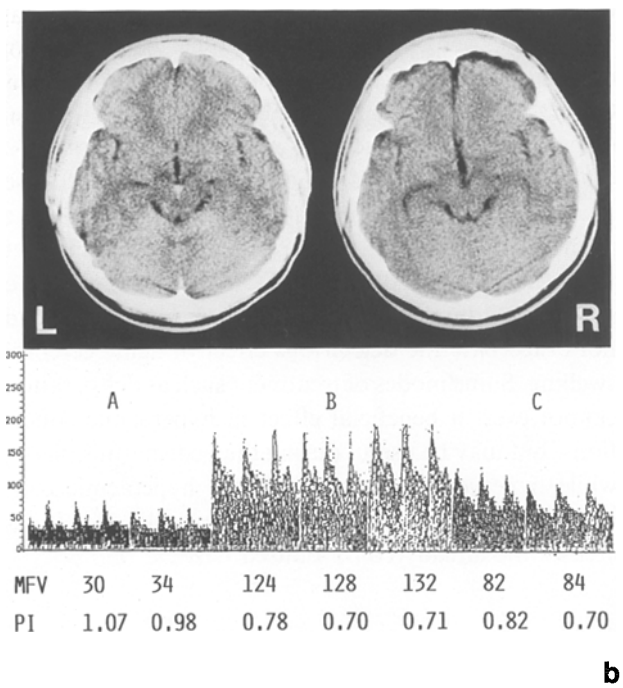

Fig. 2 b. Upper: CT scans of case 2; on admission (left) and when the high flow velocity was observed on TCD monitoring (right). High intracranial resistance pattern had never been observed in this case, which was discharged with only mild disability. Lower: Results of TCD monitoring in case 2. High flow velocity pattern appeared from the third until the seventh admission day (*B*), and then gradually normalized (*C*). High resistance pattern had never been observed during its course. Abbreviations see Fig. 2 a

brovascular congestion. McHedlishvili[15, 16], using experimental brain injury in dogs, found that circulatory changes play a crucial role in the course of brain oedema development, particularly in the major cerebral arteries and pial arterial networks. Resistance to blood flow decreases as the pial networks dilates during pre-oedematous period following brain trauma, followed by constriction as soon as oedema has developed. In our cases, the mean PI value decreased to about 63% of its initial value, suggesting changes of vascular resistance. Concerning cerebral vascular resistance, a very important fact explained by the Poiseulle formula[1] said that "flow resistance is inversely proportional to diameter in the fourth power". This means that a modest increase of 10% in vessel diameter will result in about 32% decrease in resistance. As much of the resistance within the cerebral circulation is found within the arterioles, the decrease of PIs in our cases is believed to be related to the dilatation of cerebral arterioles, including pial arterial network, during the pre-oedematous period.

Any incremental increase in cerebral arterial blood volume gives an additional volume to the total intracranial volume i.e. increases the intracranial pressure. This and the vascular constriction observed by McHed-

Table 2. *Transcranial Doppler Sonography*

Case	Admission			At peak MFVs			Following peak MFVs			End result		
	MFV	PI	ICP	MFV	PI	ICP	MFV	PI	ICP	MFV	PI	ICP
No. 1	42	0.87	25	124	0.36	20	*72*	*1.31*	*35*	98	1.15	20
No. 2	30	1.07	NM	124	0.78	NM	80	0.82	NM	84	0.70	NM
No. 3	72	0.60	20	130	0.45	20	*12*	*2.70*	*80*	*2*	*3.69*	*60*
No. 4	78	1.64	22	138	0.76	15	*106*	*1.32*	*20*	*8*	*5.89*	*60*
No. 5	36	0.85	25	132	0.60	25	*78*	*1.43*	*45*	*12*	*5.37*	*100*
No. 6	44	1.03	NM	152	0.56	NM	86	0.70	NM	52	1.0	NM
No. 7	70	1.15	NM	144	0.86	NM	*8*	*7.89*	*NM*			
No. 8	76	1.10	NM	156	0.87	NM	*26*	*1.75*	*NM*	38	1.29	NM
No. 9	72	0.99	26	100	0.78	25	*72*	*1.30*	*29*	72	0.9	20

MFV mean flow velocity; *PI* pulsatility index; *ICP* intracranial pressure; italic characters: high resistance patterns; underlined characters: patterns of cerebral circulatory arrest / systolic spike; *NM* not monitored

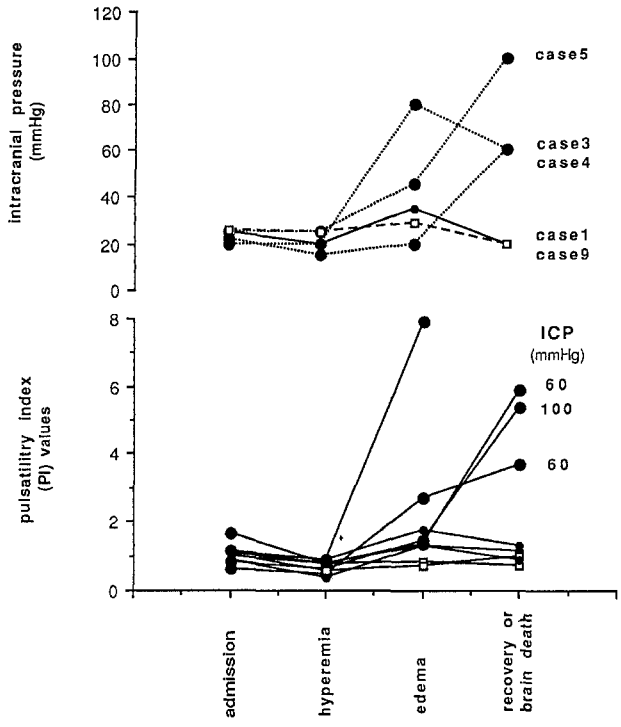

Fig. 3. Pattern of changes of TCD pulsatility (lower graph) for all 9 cases, and changes of ICP in 4 monitored cases (upper graph). Note the similar trends in the fluctuation of ICP and TCD pulsatility during different phases of intracranial haemodynamics. They tend to stay low during hyperaemia, and increased after oedema has developed. Their normalization leads to recovery (open box and small solid circle), regardless of neurological status, and their progressiveness leads to intracranial circulatory arrest and brain death (solid large circle)

lishvili[16] caused the increase of PI values after oedema has already developed. The results of ICP monitoring in 4 patients revealed that it fluctuated in a similar trend as the changes of PI values on TCD (Fig. 3). ICP increased following the hyperaemic phase, while at the same time the majority of TCD showed patterns of high intracranial resistance. We assumed this TCD pattern to be related to a critical value of the cerebral perfusion pressure (CPP) as also observed by Hassler *et al.*[10]. A further drop in CPP will change this pattern into cerebral circulatory arrest, i.e. systolic spike, to and fro, or no flow, as exhibited by 4 out of our 9 cases.

The number of patients who ended up with severe disability, vegetative, or dead was high, 6 out of 9 cases or 66%, in this group with abnormally high MFVs, compared with its reference group, which is only 12 out of 35 cases or 34%. Although the outcome of these severely head injured patients largely depends on the primary injury of the neural structures, this indirect measurement of CBF using TCD should lead us to accurately adjust the treatment in order to prevent and not exacerbate the deleterious effect of acute cerebral swelling. Some modes of treatment such as dehydration cannot exert a beneficial effect in hyperaemic conditions, but may be useful during the oedematous phase, while hyperventilation, suitable for hyperaemic conditions, is dangerous in oedematous swelling for it decreases the already compromised CBF.

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