

## Intracranial Cerebral Blood Flow in Patients with Chronic Headache in a Sample of Patients in Aswan

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**Abstract:**

**Introduction:** Recent studies indicated that migraine is associated with specific vascular risk profile. However, the functional and structural vascular abnormalities in migraine are rarely addressed.

**objectives:** of the present study is to evaluate cerebral blood flow (CBF) velocities using transcranial Doppler (TCD) during an attack-free period in anterior, middle, and posterior cerebral arteries in migraine patients without aura (MwoA) as well as in chronic tension-type headache (TH) patients who were not receiving prophylactic medication for their headaches, in a sample of patients attended to outpatient clinic in Aswan university hospitals. **Methods:** A total of 50 subjects (32 females, 18 males), Migraine (20), Tension headache group (20), Control group (10), Peak Systolic Velocity (PSV) of middle cerebral artery (MCA), anterior cerebral artery (ACA), posterior cerebral artery (PCA), End diastolic velocity (EDV) Pulsatility index (PI), Resistivity index (RI) were calculated for all groups. **Results:** Mean SD for age in years in MwoA group was  $32.75 \pm 9.6$  and in TH group was  $32.75 \pm 9.6$  and in control group was  $35.20$

$\pm 8.6$ . Also M: F ratio was 3:1 in MwoA, TCD Parameters of Anterior Circulation for MCA show significant difference in PSV between MwoA group and TH group and also between MwoA group and control group but no significant difference between TH group and either group the same was for EDV, in ACA only significant difference in PSV between MwoA group and control group.

**Key Words:** Migraine, tension headache, vascular risk factors, atherosclerosis. Peak systolic velocity.



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## Introduction:

The mechanisms underlying migraine pain still remain elusive, and the role of vascular and neural mechanisms has been discussed and investigated thoroughly, but vascular hypothesis of vasodilatation as a cause for migraine dominated migraine research for most of the 20th century (2).

Pathophysiological mechanisms underlying migraine are not fully clarified, and no physiological biomarkers have been discovered yet. Vascular theory of migraine with studies on changes in arterial diameter during migraine attacks using magnetic resonance angiography found that, During attack, cerebral blood flow (CBF) techniques recorded values as low as 23 mL/100 g per minute when aura symptoms were present (3).

It was reported that TCD is a noninvasive technique that evaluates the (CBF) properties in the cerebral arteries, as well as the cerebrovascular reserve, using a pulsed ultrasonic beam. However, TCD studies in migraine have contradictory results (19). Studies performed on migraine patients have reported abnormalities in hemodynamics (5&9). Some studies have shown increased (CBF) (7), and some reported normal blood flow velocities in migraine patients (6). It was stated that some studies (4, 7) found

alteration of the CBF which was differ in patients suffering migraine with aura than in those suffering from migraine without aura as reported by some researchers (11). Therefore, the optimal solution would be to investigate a single type of migraine (either migraine with or without aura).

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## Objective

Our objective is to study possible vascular flow abnormalities in migraine and TH if and if Migraine and TH have long standing effect on the vascular bed of the brain.

The study was approved by the Local Ethical Committee of Aswan University Faculty of Medicine and all volunteers gave written informed consent before participating in the study.

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## Methods

The study was Case control observational cross sectional study, forty (40) subjects with headache diagnosed according to International Headache Society criteria (1) were participated in this study. A total of 40 subjects (28 females, 12 males) completed the study and were evaluated during a headache-free episode, 20 patients with migraine headache without aura (MwoA) (15 females, 5 males, aged 19 to 50 years with mean age  $32.8 \pm 6.9$  years), 20 patients with chronic tension-type headache (TH)

(13 females, 7 males, aged 27 to 47 years with mean age  $38.5 \pm 5.1$  years). Ten (10) age and sex matched (5 F, 5 M) healthy volunteers (aged 18 to 45 years with mean age  $35.2 \pm 8.5$  years) from staff and nursing members served as controls.

All patients fulfilled the diagnostic criteria for migraine and chronic tension-type headaches established in the International Headache Society's International Classification of Headache Disorders, second edition (1). We investigate only patients suffering migraine without aura (MwoA).

All patients had a normal neurological examination. Patients with other known systemic diseases were excluded. None of the patients were receiving prophylactic medication for headache or any treatment known to affect CBF. Intake of analgesics, smoking, and consumption of caffeine were forbidden on the day of testing. Heart rate and blood pressure were both measured just before the beginning of the TCD and all participants had normal values.

## Methods

The study was performed in a quiet room by qualified physician to ensure unified technique and results with the subjects lying in a comfortable supine position. All Doppler recordings were performed blinded to the clinical status of the subjects.

Intracranial arteries were insonated through the temporal and transforaminal windows using standardized protocol.

Peak systolic velocities (PSV) of middle cerebral artery (MCA) flow were recorded at depths of 50–60 mm, anterior cerebral artery (ACA) at 50–65 mm, posterior cerebral artery (PCA) at 55–65 mm, vertebral artery (VA) at 50–70 mm, and basilar artery (BA) at 80–100 mm with a 2-MHz probe by the technique described by *Aaslid., 1984 (10)* Peak systolic (V Peak-VP) CBF velocities (cm/s) and Pulsatility index (PI) were determined. Only measurements with the best Signal-to-noise ratio were used, and the highest values for CBF velocities were selected for analysis. All TCD studies were performed with the use of the available TCD apparatus, Viasys/Sonara (Cardinal Health, Madison, WI, USA).

## Statistical analysis:

Data were verified, coded by the researcher and analyzed using IBM-SPSS 21.0 (IBM-SPSS Inc., Chicago, IL, USA) (11). Descriptive statistics: Means, standard deviations, medians, ranges and percentages were calculated. Test of significances: chi-square test was used to compare the difference in distribution of frequencies among different groups. For continuous variables; independent t-test analysis was carried out to compare the means of

normally distributed data. For variables with more than two categories, ANOVA test was used to compare the means of normally distributed data and post-hoc test analysis with Bonferroni corrections was applied. Pearson correlation was used to assess the correlations among variables. A significant P\_value was considered when it is equal or less than 0.05 (11).

## Results

Mean SD for age in years in (MwoA) group was  $32.75 \pm 9.6$  and in TH group was  $32.75 \pm 9.6$  and in control group was  $35.20 \pm 8.6$ . Also M: F ratio was 3:1 in (MwoA) group and nearly equal in TH group regarding disease duration TH was more chronic and both types were commoner in females. Duration of attacks was significantly

prolonged in TH group and highly significant in females **table 1**.

**In table 2**, TCD Parameters of Anterior Circulation for MCA show significant difference in PSV between (MwoA) group and TH group and also between (MwoA) group and control group

**In table 3**, TCD Parameters of Posterior Circulation revealed no significant difference in all parameters in all studied groups.

Only RI was significant in ACA with increased duration of attacks and increased PI in PCA with increased duration of attacks (**tables 4&5**). No significant difference in TCD parameters in relation to sex in all groups (**table 6**)

**Table 1:** Sociodemographic and Disease Characteristics Differences in Cases Vs. Control

Parameter	Migraine (1) (no.=20)	Tension Headache (2) (no.=20)	Control (3) (no.=10)	P-value
<b>Age in years</b>				=0.170*
•Mean SD	32.75 ± 9.6	37.70 ± 6.1	35.20 ± 8.6	
•Median (Range)	33 (19 - 50)	38 (23 - 47)	36 (18 - 45)	
<b>P-value**</b>	1 vs 2 =0.061	2 vs 3 =0.433	1 vs 3 =0.442	
<b>Sex</b>				=0.179***
•Male	5 (25%)	7 (35%)	5 (50%)	
•Female	15 (75%)	13 (65%)	5 (50%)	
<b>Disease Duration</b>				
•< 10 years	15 (75%)	8 (40%)		=0.025***
•≥ 10 years	5 (25%)	12 (60%)		
<b>Attack Duration</b>				
•≤ 24 hours	13 (65%)	1 (5%)		<0.001***
•> 24 hours	7 (35%)	19 (95%)		

\*\*\*TCD Parameters of Anterior Circulation for MCA show significant difference in PSV between (MwoA) group and TH group and also between (MwoA) group and control group



TCD Parameters of Anterior Circulation for MCA show significant difference in PSV between (MwoA) group and TH group and also between (MwoA) group and control group but no significant difference between

TH group and either group the same was for EDV, in ACA only significant difference in PSV between (MwoA) group and control group (*table 2*).

**Table 2: TCD Parameters of Anterior Circulation in Migraine, Tension Headache cases and controls**

Parameter	Migraine (1) (no.=20)	Tension Headache (2) (no.=20)	Control (3) (no.=10)	P-value*
<b>MCA (mean ± SD)</b>				
• PSV	163.20 ± 38.3	121.90 ± 15.2	98.70 ± 9.8	<b>&lt; 0.001</b>
<b>P-value**</b>	<b>1 vs 2 &lt;0.001</b>	<b>2 vs 3 =0.030</b>	<b>1 vs 3 &lt;0.001</b>	
• EDV	80.15 ± 13.9	59.45 ± 10.1	50.20 ± 4.2	<b>&lt; 0.001</b>
<b>P-value**</b>	<b>1 vs 2 &lt;0.001</b>	2 vs 3 =0.086	<b>1 vs 3 &lt;0.001</b>	
• PI	1.41 ± 0.2	1.38 ± 0.1	1.18 ± 0.2	= 0.711
<b>P-value**</b>	1 vs 2 =0.905	2 vs 3 =0.488	1 vs 3 =0.429	
• RI	0.76 ± 0.2	0.77 ± 0.2	0.61 ± 0.2	= 0.076
<b>P-value**</b>	1 vs 2 =0.488	<b>2 vs 3 =0.048</b>	<b>1 vs 3 =0.036</b>	
<b>ACA (mean ± SD)</b>				
• PSV	127.05 ± 4.6	109.60 ± 6.8	92.10 ± 6.5	<b>= 0.003</b>
<b>P-value**</b>	<b>1 vs 2 =0.033</b>	2 vs 3 =0.078	<b>1 vs 3 =0.001</b>	
• EDV	65.80 ± 3.5	53.45 ± 4.5	50.90 ± 6.1	<b>= 0.049</b>
<b>P-value**</b>	<b>1 vs 2 =0.037</b>	2 vs 3 =0.720	<b>1 vs 3 =0.040</b>	
• PI	2.07 ± 0.2	1.58 ± 0.2	0.91 ± 0.1	<b>= 0.007</b>
<b>P-value**</b>	1 vs 2 =0.090	2 vs 3 =0.064	<b>1 vs 3 =0.002</b>	
• RI	0.89 ± 0.1	0.76 ± 0.1	0.60 ± 0.1	<b>= 0.035</b>
<b>P-value**</b>	1 vs 2 =0.144	2 vs 3 =0.156	<b>1 vs 3 =0.011</b>	

\*\*\* Significant difference in PSV between (MwoA) group and control group

TCD Parameters of Posterior Circulation revealed no significant difference in all parameters in all studied groups (*table 3*).

**Table 3:** TCD Parameters of Posterior Circulation in Migraine, Tension Headache cases and controls

Parameter	Migraine (1) (no.=20)	Tension Headache (2) (no.=20)	Control (3) (no.=10)	P-value*
<b>PCA (mean ± SD)</b>				
• PSV	100.85 ± 25.6	87.85 ± 9.7	83.10 ± 4.8	= 0.069
<b>P-value**</b>	1 vs 2 =0.066	2 vs 3 =0.577	<b>1 vs 3 =0.041</b>	
• EDV	49.53 ± 3.5	44.65 ± 2.3	38.90 ± 4.2	= 0.108
<b>P-value**</b>	1 vs 2 =0.245	2 vs 3 =0.246	<b>1 vs 3 =0.038</b>	
• PI	2.03 ± 0.3	1.72 ± 0.2	0.96 ± 0.1	<b>= 0.043</b>
<b>P-value**</b>	1 vs 2 =0.364	2 vs 3 =0.071	<b>1 vs 3 =0.013</b>	
• RI	0.83 ± 0.2	0.76 ± 0.1	0.78 ± 0.2	= 0.601
<b>P-value**</b>	1 vs 2 =0.328	2 vs 3 =0.842	1 vs 3 =0.547	
<b>VA (mean ± SD)</b>				
• PSV	80.50 ± 4.3	75.00 ± 3.1	74.00 ± 4.5	= 0.467
<b>P-value**</b>	1 vs 2 =0.294	2 vs 3 =0.875	1 vs 3 =0.311	
• EDV	40.45 ± 2.5	38.20 ± 1.6	39.00 ± 2.4	= 0.688
<b>P-value**</b>	1 vs 2 =0.394	2 vs 3 =0.804	1 vs 3 =0.653	
• PI	1.23 ± 0.2	0.96 ± 0.2	0.91 ± 0.1	= 0.390
<b>P-value**</b>	1 vs 2 =0.244	2 vs 3 =0.875	1 vs 3 =0.258	
• RI	0.61 ± 0.1	0.56 ± 0.1	0.54 ± 0.1	= 0.530
<b>P-value**</b>	1 vs 2 =0.356	2 vs 3 =0.834	1 vs 3 =0.336	
<b>BA (mean ± SD)</b>				
PSV	98.60 ± 5.8	88.25 ± 4.1	80.80 ± 5.6	= 0.092
<b>P-value**</b>	1 vs 2 =0.136	2 vs 3 =0.378	<b>1 vs 3 =0.039</b>	
EDV	49.15 ± 3.1	44.45 ± 2.8	43.80 ± 2.7	= 0.391
<b>P-value**</b>	1 vs 2 =0.235	2 vs 3 =0.893	1 vs 3 =0.269	
PI	1.94 ± 0.3	1.78 ± 0.2	1.25 ± 0.2	= 0.295
<b>P-value**</b>	1 vs 2 =0.650	2 vs 3 =0.237	1 vs 3 =0.123	
RI	0.76 ± 0.1	0.76 ± 0.1	0.74 ± 0.1	= 0.978
<b>P-value**</b>	1 vs 2 =0.959	2 vs 3 =0.838	1 vs 3 =0.871	

Regarding Correlation between TCD parameters and Duration of disease and attacks in (MwoA) only RI was significant in ACA with increased duration of attacks and increased PI in PCA with increased

duration of attacks, in TH group also RI and PI was significant in PCA with increased duration of attacks and increased PI in PCA with increased duration of attacks. Increased



RI in both BA and MCA with increased duration of headache (**tables 4&5**).

**Table 4:** Correlation between TCD parameters (PSV, PI, and RI) and Disease Duration (Migraine Group)

		Disease Duration/years		Headache Duration/hours	
		r*	P-value**	r	P-value
MCA	• PSV	-0.228	> 0.05	-0.148	> 0.05
	• PI	0.029	> 0.05	-0.275	> 0.05
	• RI	0.251	> 0.05	0.065	> 0.05
ACA	• PSV	-0.072	> 0.05	0.035	> 0.05
	• PI	-0.158	> 0.05	-0.268	> 0.05
	• RI	-0.172	> 0.05	<b>-0.312</b>	<b>&lt; 0.05</b>
PCA	• PSV	-0.121	> 0.05	-0.060	> 0.05
	• PI	-0.067	> 0.05	<b>0.365</b>	<b>&lt; 0.05</b>
	• RI	0.284	> 0.05	0.080	> 0.05
VA	• PSV	-0.232	> 0.05	-0.115	> 0.05
	• PI	-0.250	> 0.05	-0.225	> 0.05
	• RI	-0.263	> 0.05	-0.165	> 0.05
BA	• PSV	-0.132	> 0.05	-0.063	> 0.05
	• PI	-0.230	> 0.05	-0.321	> 0.05
	• RI	-0.231	> 0.05	-0.253	> 0.05

\*\*\*RI was significant in ACA with increased duration of attacks and increased PI in PCA with increased duration of attacks, in TH group also RI and PI was significant in PCA with increased duration of attacks and increased PI in PCA with increased duration of attacks.

**Table 5:** Correlation between TCD parameters (PSV, PI, and RI) and Disease Duration (Tension Headache Group)

		Disease Duration/years		Headache Duration/hours	
		r*	P-value**	r	P-value
MCA	• PSV	-0.088	> 0.05	-0.073	> 0.05
	• PI	-0.194	> 0.05	-0.076	> 0.05
	• RI	<b>-0.405</b>	<b>&lt; 0.05</b>	-0.166	> 0.05
ACA	• PSV	0.040	> 0.05	0.068	> 0.05
	• PI	0.065	> 0.05	-0.248	> 0.05
	• RI	-0.188	> 0.05	-0.215	> 0.05
PCA	• PSV	0.150	> 0.05	0.237	> 0.05
	• PI	-0.031	> 0.05	<b>-0.429</b>	<b>&lt; 0.05</b>
	• RI	0.146	> 0.05	<b>-0.536</b>	<b>&lt; 0.05</b>
VA	• PSV	-0.055	> 0.05	0.037	> 0.05
	• PI	-0.080	> 0.05	-0.253	> 0.05
	• RI	0.020	> 0.05	<b>-0.326</b>	<b>&lt; 0.05</b>
BA	• PSV	0.185	> 0.05	0.295	> 0.05
	• PI	-0.103	> 0.05	0.008	> 0.05
	• RI	<b>-0.330</b>	<b>&lt; 0.05</b>	-0.140	> 0.05

\*\*\* Increased RI in both BA and MCA with increased duration of headache P value < 0.05.

**Table 6:** Correlation between TCD parameters (PSV, PI, RI) and Sex

		Migraine			Tension Headache		
		Male	Female	P-value*	Male	Female	P-value
<b>MCA</b>	• <b>PSV</b>	178.2 ± 20.9	158.2 ± 9.2	= 0.326	124.9 ± 6.1	120.3 ± 4.5	= 0.554
	• <b>PI</b>	1.34 ± 0.4	1.43 ± 0.3	= 0.863	1.13 ± 0.2	1.52 ± 0.1	= <b>0.039</b>
	• <b>RI</b>	0.62 ± 0.2	0.61 ± 0.1	= 0.979	0.67 ± 0.2	0.83 ± 0.3	= 0.136
<b>ACA</b>	• <b>PSV</b>	132.4 ± 8.3	125.3 ± 5.6	= 0.518	114.7 ± 15.8	106.9 ± 6.7	= 0.554
	• <b>PI</b>	2.26 ± 0.6	2.01 ± 0.2	= 0.635	1.61 ± 0.4	1.57 ± 0.3	= 0.923
	• <b>RI</b>	0.81 ± 0.1	0.92 ± 0.1	= 0.479	0.71 ± 0.1	0.78 ± 0.1	= 0.597
<b>PCA</b>	• <b>PSV</b>	93.8 ± 11.0	103.2 ± 7.1	= 0.508	89.0 ± 5.7	87.2 ± 6.2	= 0.836
	• <b>PI</b>	1.87 ± 0.3	2.08 ± 0.4	= 0.695	1.51 ± 0.3	1.83 ± 0.2	= 0.430
	• <b>RI</b>	0.84 ± 0.1	0.83 ± 0.1	= 0.983	0.70 ± 0.1	0.80 ± 0.1	= 0.400
<b>VA</b>	• <b>PSV</b>	69.0 ± 11.5	84.3 ± 4.1	= 0.121	75.1 ± 5.2	74.9 ± 4.1	= 0.974
	• <b>PI</b>	1.47 ± 0.5	1.15 ± 0.2	= 0.446	0.79 ± 0.1	1.05 ± 0.2	= 0.391
	• <b>RI</b>	0.60 ± 0.1	0.61 ± 0.1	= 0.929	0.55 ± 0.1	0.56 ± 0.1	= 0.927
<b>BA</b>	• <b>PSV</b>	91.2 ± 12.2	101.1 ± 6.7	= 0.502	87.1 ± 8.2	88.9 ± 4.7	= 0.861
	• <b>PI</b>	2.14 ± 0.4	1.87 ± 0.4	= 0.650	1.24 ± 0.2	2.07 ± 0.2	= <b>0.015</b>
	• <b>RI</b>	0.86 ± 0.2	0.72 ± 0.3	= 0.341	0.60 ± 0.1	0.85 ± 0.1	= <b>0.011</b>

No significant difference in TCD parameters in relation to sex in all groups (**table 6**)

## Discussion

Transcranial Doppler testing of cerebrovascular changes is a reliable and interesting for the diagnostic evaluation and management of migraine patients. It offers the opportunity to noninvasively monitoring of cerebral blood flow parameters and therefore, represents a valuable tool for vascular research in migraine. However, TCD studies in migraine have contradictory results. Studies conducted during attack-free episodes have shown either increased (**14 & 7**) or normal blood flow velocities in

migraine patients (**17& 6**). It was reported that CBF velocities in MCA on the headache

side during a migraine attack was reduced.(**17**). While another study (**18**) reported no velocity changes at the headache and non- headache side MCAs during an acute attack (**19**). It is argued that the increased flow velocity displayed by TCD is a hallmark of migraine (**20**).

Our study found significant difference between (MwoA) group and both TH and control groups regarding the PSV and EDV

which was against **Ozkalayci et al., (2018)** who found no significant velocity differences in migraine patients during headache –free episode when compared to controls **(19)**. But in agreement with **Arjona et al., 2007** who observed that mean blood flow velocity in the MCA of patients with (MoaW) is higher (8 cm/sec) than in patients with episodic tension headache **(13)**. In a similar study **(19)** it was also reported an increased basilar artery CBF velocities in patients with chronic tension-type headache, and no difference was present in other examined arteries. But we found no significant changes between all groups regarding Posterior circulation TCD parameters, these changes can be explained by the vasodilator activity of neurotransmitters such as nitric oxide and calcitonin gene-related peptide, contributing to central sensitization. Others suggested either downstream vasodilatation (arteriolar vasodilatation theory) **(14&15&16&17)** especially in arterioles or a lower diameter in the insonated part of these vessels **(17 and 7)**. Our results showed no other significant changes in CBF velocities which could be explained by that, the neuronal activation is more prominent than vascular effects. In contrast to our results an increased CBF velocities in the MCA, ACA and PCA as well as decreased PI in patients

with TH were reported, while no difference was present in patients with (MwoA) **(20)**, while **Fiermonte et al., 1999**, demonstrated lower PI in migraine **(21)**, This decreased PI is usually an indirect upstream vascular resistance, which in turn would cause an increase in mean blood flow velocity inside the insonated segments **(23)**.

In agreement with our results, no difference in cerebrovascular reactivity in the anterior circulation among patients with migraine with or without aura and controls, it was found, also no difference in the posterior circulation between patients with migraine without aura and controls **(23)**.

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## Conclusion

Transcranial Doppler testing of cerebrovascular changes is a reliable and interesting for the diagnostic evaluation and management of migraine patients. Our result were in agree with some studies and some of them were against, as regarding if Migraine and TH have long standing effect on the vascular bed of the brain Only RI was significant in ACA and increased PI in PCA with durations more than 10 years and attacks more than 24 hours, extensive studies are needed to clarify standards for future studies.

One of the limitations of our study is that we did not have another group of patients with secondary headache.

### **Abbreviations:**

CBF	Cerebral blood flow
TCD	Transcranial Doppler
MwoA	Migraine without aura
TH	Tension headache
PSV	Peak systolic volume
MCA	Middle cerebral artery
ACA	Anterior cerebral artery
PCA	Posterior cerebral artery
EDV	End diastolic volume
PI	Pulsatility index
RI	Resistance Index
BA	Basilar artery

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