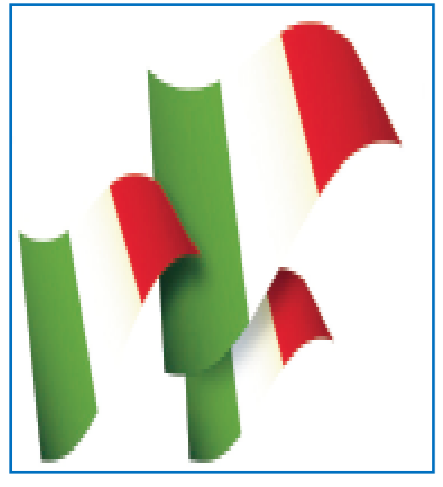


# Increased flow pulsatility in patients with migraine: a possible link between stroke and migraine?

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**Background-** Migraine has been associated with an increased risk for ischemic stroke especially in patients with aura. The mechanisms explaining this association remain poorly understood.. One possible mechanism is the presence of a vascular endothelial dysfunction. A recent study in animal model has shown that an elevated flow pulsatility, inducing increased mechanical deformation or cyclic strain of the arterial wall, leads to an endothelial inflammation and endothelial dysfunction [2].

**Objective-** The aim is to verify the presence of an increased flow pulsatility into cerebral arterial network in migraine with (MA+) and without aura (MA-) during the interictal period of migraine.

**Methods-** We measured the time-delay in milliseconds (ms) between the R-wave of an electrocardiogram and the arterial pulse wave (pulsatile arterial blood) of cerebral microcirculation [1] (R-APWCMtd) on the frontal cortex (Fig. 1,2,3) detected by Near-infrared spectroscopy (NIRS) in 10 patients with MA+ (age  $39.5 \pm 12.2$  years), in 10 with MA- (age  $40.3 \pm 10.2$  years), according to ICHD-3 criteria 2013, during the interictal period of migraine, and in 15 age-, sex- and height-matched healthy control subjects.

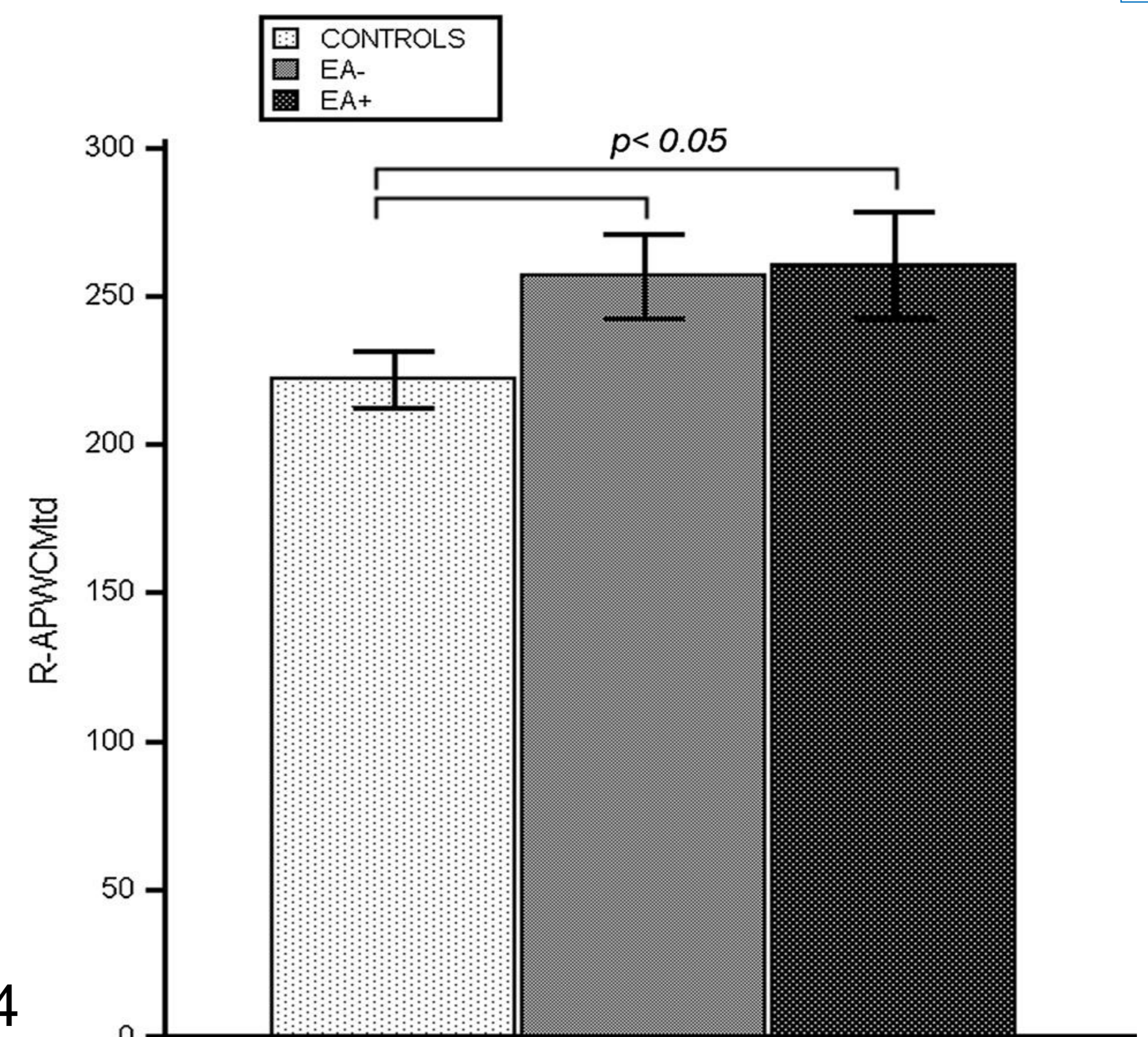
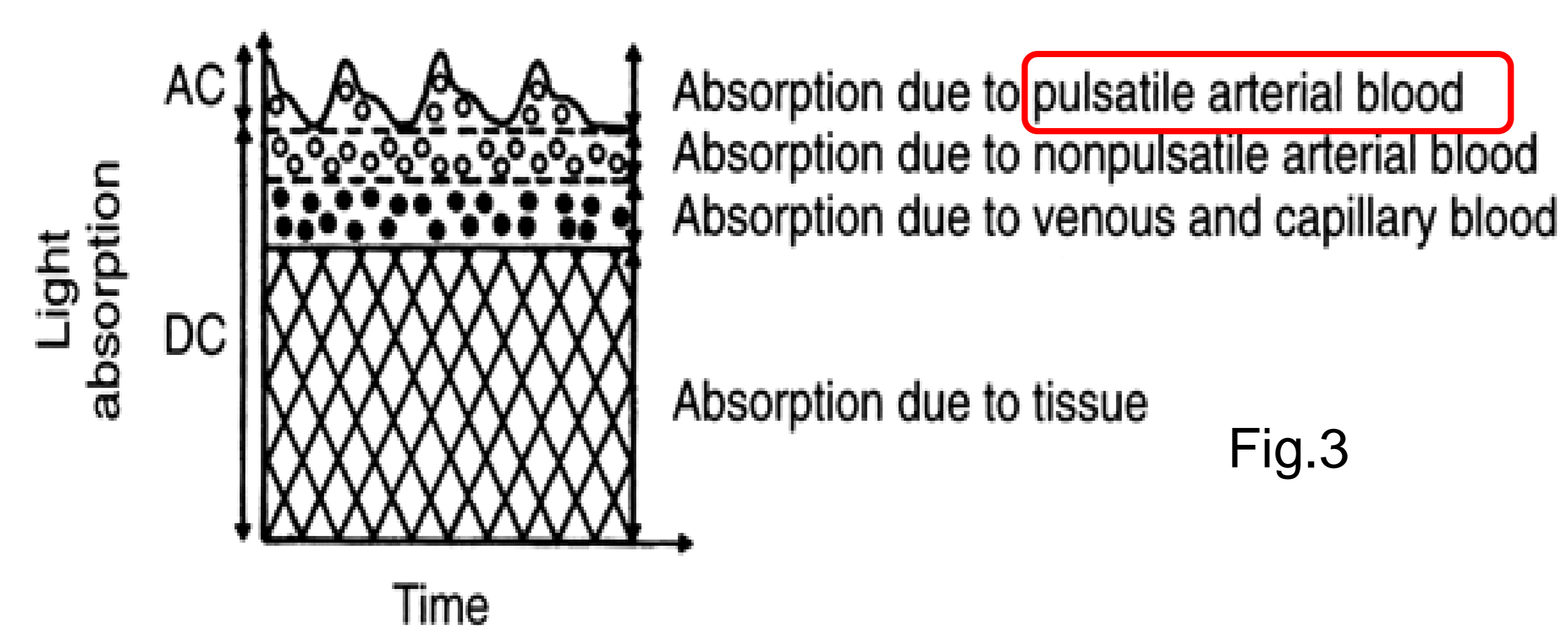
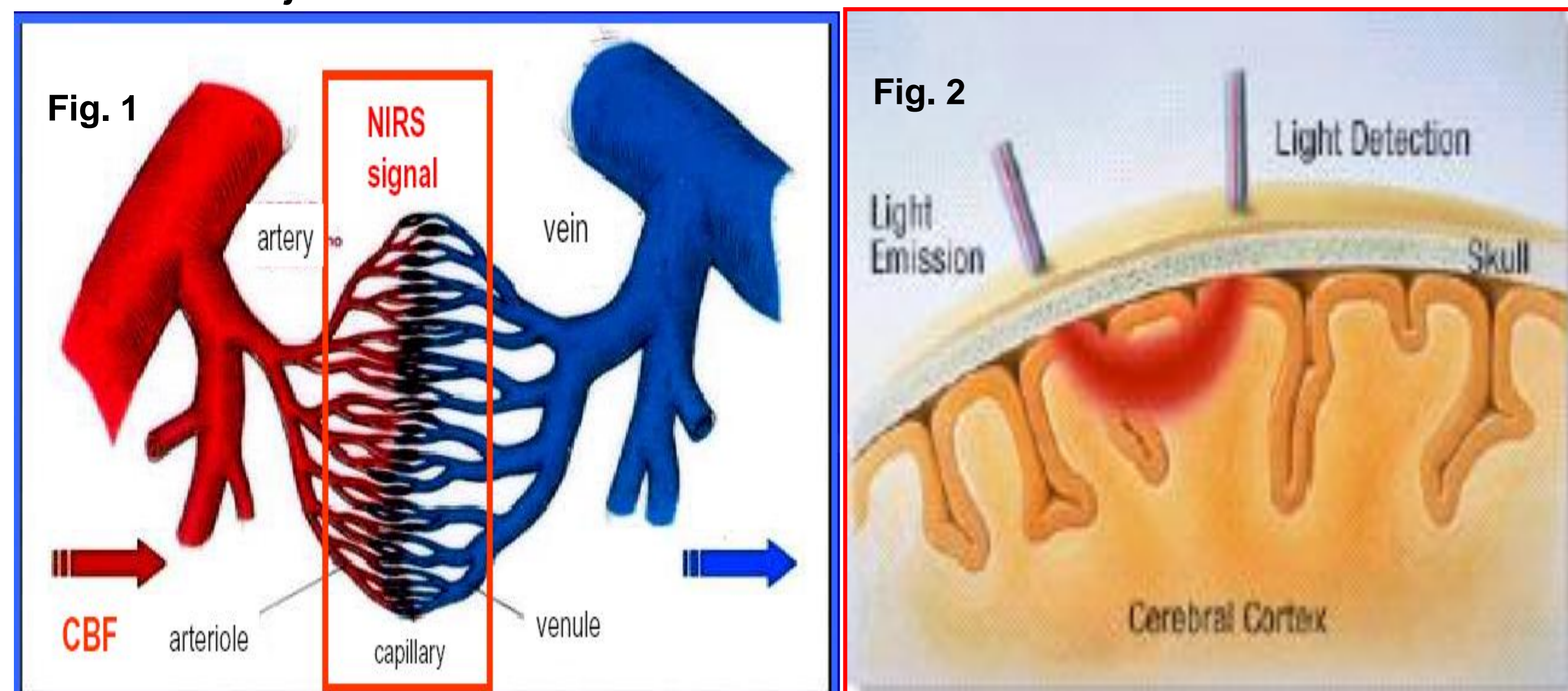
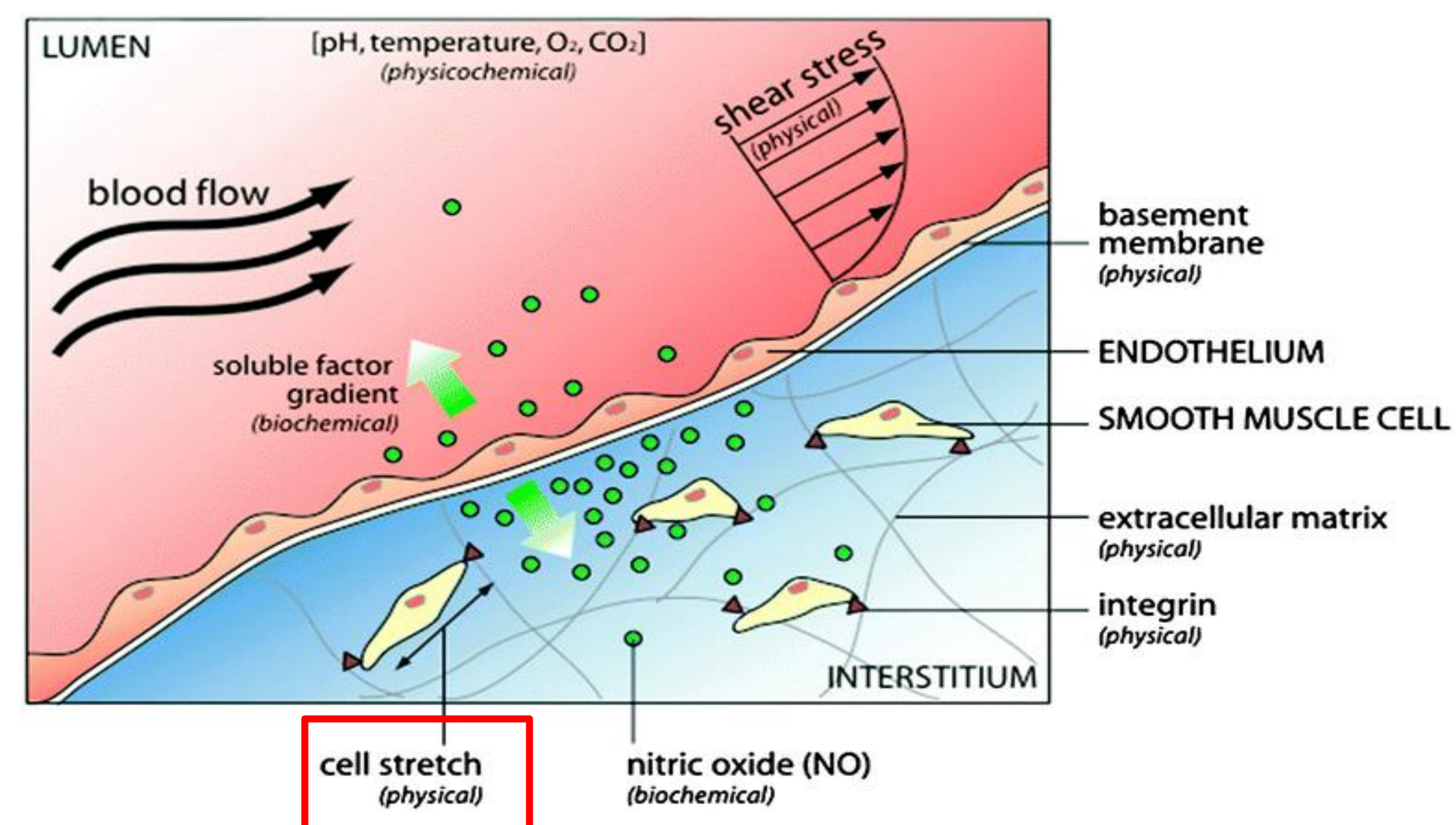


Fig.4

**Discussion-** In young, healthy adults, the aorta has a high distensibility and first-generation arteries are relatively stiff. This abrupt transition from the aortic distensibility (low impedance) to the stiff (high impedance) branch vessels represents an impedance mismatch that represents a protective mechanism that limits the transmission of excessive pulsatile energy into the cerebral microcirculation. The longer R-APWCMtd may lead to the reduction of the impedance mismatch and thereby may facilitate the transmission of excessive pulsatile energy or flow pulsatility into the cerebral microcirculation [1]. We may speculate that an elevated flow pulsatility, inducing increased mechanical deformation of the arterial wall, may lead to an endothelial dysfunction into cerebral arterial network.



**Conclusion-** In conclusion, in our migraine patients, a longer R-APWCMtd is associated with migraine and indicates an increased distensibility of the wall of cerebral arterial network. The increased distensibility leads to an increased flow pulsatility into the cerebral arterial network that may lead to an endothelial dysfunction .

We excluded secondary headaches by appropriate laboratory and imaging diagnostic tests. The cases and controls were free from major cardiovascular risk factors and migraine prophylactic medications.

**Results** -The patients with migraine had a significantly longer R-APWCMtd than the control subjects  $F = 13.4$ ,  $p < 0.001$ : MA+:+38.3 ms; MA-:+34.7 ms (Fig. 4).

In the multiple regression analysis, the R-APWCMtd was significantly associated with migraine (coefficient of determination  $R^2 = 0.50$ , multiple correlation coefficient 0.71,  $p < 0.0001$ ) but not with age, gender, height, migraine attack frequency and disease duration.

This condition, obtained in migraineurs without major cardiovascular risk factors, may represent one possible mechanism underlying the increased risk of ischemic stroke especially in patients with MA+.

1. Viola S & Viola P, Buongarzone MP, Fiorelli L, Litterio P. The increased distensibility of the wall of cerebral arterial network may play a role in the pathogenic mechanism of migraine headache. *Neurol Sci* 2014.
2. Li M, Tan Y, Stenmark KR, Tan W. High Pulsatility Flow Induces Acute Endothelial Inflammation through Overpolarizing Cells to Activate NF- $\kappa$ B. *Cardiovasc Eng Technol*. 2013;4:26-38.