
**Granulocytes, Platelet Activating Factor, and Stroke**

*To the Editor:* Saito et al report increased leukotriene-like immunoreactivity (C4 and D4) in gerbil brain following bilateral common carotid occlusion and reduced leukotriene levels as a result of busulfan-induced granulocytopenia. Their results support the view that granulocytes may be involved in the pathogenesis of cerebral infarction.

In addition to being the source of leukotrienes, granulocytes, particularly basophils, produce platelet-activating factor (PAF), which is a potent inducer of platelet activation, thrombosis, and ischemia. Apart from the platelet-mediated effects, PAF has also been shown to cause direct neuronal damage, cerebral vasoconstriction, and cerebral hypoperfusion, possibly mediated by specific PAF binding sites in the brain. In addition, we have reported an increased sensitivity to PAF-induced platelet activation in patients with acute cerebral infarction. We therefore suggest that the generation of platelet-activating factor may be another mechanism by which granulocytes participate in the pathogenesis of cerebral infarction.

**References**


**Pulsed Doppler Assessment of Arterial Obstructive Disease**

*To the Editor:* The article by Drs. Rautenberg and Hennerici in *Stroke*1 raised these questions for the authors in my mind:

1. How were the asymptomatic patients selected?
2. What were the features of the symptomatic transient ischemic attacks (TIAs)?
3. Were blood pressures obtained in both arms, and were there significant inequalities?
4. In those patients who received surgery to bypass the innominate obstruction, did the TIAs stop?
5. Finally, is it correct that some of the asymptomatic patients underwent surgery?

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**Reference**


The following is in reply:

*To the Editor:* We appreciate the letter by Dr. Perron. His five questions are answered as follows:

1. The asymptomatic patients were investigated because of carotid bruits, risk factors for atherosclerosis, or coexisting peripheral arterial disease or coronary artery disease (cf. reference 4).

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Pulsed Doppler assessment of arterial obstructive disease.

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