

Influence of Smoking on Endothelial Function

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ABSTRACT. Smoking impairs endothelial function in passive smokers as well as in active smokers. The aim of our study was evaluation of endothelial function using noninvasive ultrasonography (flow-mediated dilatation (FMD) in nonsmoker, passive smoker and smoker population. We investigated 32 normotensive, normocholesterolemic male subjects without clinical evidence of diabetes mellitus and coronary artery disease CAD, mean age (34±9), of them –12 age-matched nonsmoker subjects, 6 passive smokers, and 14 active smokers.

Our investigation revealed significantly lower FMD in smokers than in nonsmokers. Endothelial function was significantly impaired in passive smokers compared to nonsmokers and did not significantly differ from endothelial function of smokers. © 2008 Bull. Georg. Natl. Acad. Sci.

Key words: *endothelial dysfunction, flow-mediated dilatation, passive smoking.*

Smoking is one of the most important risk factors for cardiovascular events. Several mechanisms might account for the smoking-induced alterations in endothelial function. Cigarette smoke contains a large number of oxidants that cause oxidative stress of endothelial cells [1,2]. Smoking is associated with a direct toxic effect on human endothelial cells [3,4], reduces endothelial prostacyclin production [5] and increases leukocyte adhesion to endothelial cells [6]. Alternatively, smoking increases endothelial angiotensin II production, which reduces nitric oxide activity that might contribute to endothelial dysfunction in smokers [7]. Increased platelet aggregation [8] and serum fibrinogen [9], as well as decreased serum plasminogen levels [10], known to occur in smokers, might also impair endothelial function in smokers. It has been shown that environmental tobacco smoke causes arterial damage in passive smokers that might be related to enhanced degradation of nitric oxide secondary to the formation of oxygen-derived free radicals [11].

Brachial artery ultrasonography is a reliable non-

invasive marker of endothelial function, which allows repetitive dynamic assessment of both arterial diameter and velocity (2). Flow mediated dilatation (FMD) of the brachial artery in response to occlusion is proven to be one of the effective measurements of endothelial dysfunction [12].

The aim of our study was evaluation of endothelial function using the aforementioned noninvasive method in nonsmoker, passive smoker and smoker population.

Methods. We investigated 32 normotensive, normocholesterolemic male subjects without clinical evidence of diabetes mellitus and CAD, mean age (34±9), of them –12 age-matched subjects with no exposure to active or passive smoking (group I), 6 nonsmoking passive smokers (exposure to environmental tobacco smoke for ≥ 1 hour per day for ≥ 2 years) (group II), 14 active smokers (≥1 packs per day for ≥ 2 years) (group III). Patients with borderline hypertension (SBP>/=, DBP>/=) were included in the study. The diameter of the brachial artery was measured from 2D ultrasound images. The brachial artery was scanned in longitudinal section. Three cardiac

cycles were analyzed for each scan, and measurements were averaged. After the baseline measurements, a pneumatic tourniquet was inflated below the elbow to at least 50 mm Hg above the systolic pressure; forearm cuff occlusion was maintained for 4.5 minutes and the diameter of the artery was measured at 1 minute after cuff deflation. Endothelium-dependent peripheral FMD was expressed as the percent change of brachial artery diameter 1 minute after forearm occlusion release, with baseline resting diameter used as a reference.

Results. FMD was significantly lower in smokers (2.7% +/- 2.1%), than in nonsmokers (7.4% +/- 3.2%) ($p = 0.0002$). Endothelial function was impaired in passive smokers (FMD=3.8 % +/-2.7 %) compared to nonsmokers ($p=0.03$) and did not significantly differ from endothelial function of smokers ($p=0.33$) (Fig.).

Discussion. FMD is considered to be a sensitive marker for endothelial dysfunction, as it has been shown to be mediated mainly by nitric oxide. Increase of blood flow velocity during reactive hyperemia induces increase of wall shear stress with subsequent local

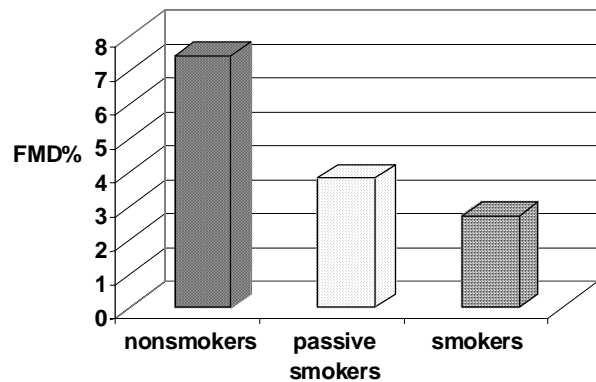


Fig. Influence of passive and active smoking on FMD

hyperproduction of endothelial nitric oxide.

Our results are in agreement with previous studies that endothelial dysfunction is significantly impaired in passive smokers as well as in smokers. It should be noted that the extent of endothelial dysfunction did not differ significantly in both groups, which perhaps must be taken into consideration while conducting preventive measures in aforementioned population groups.

სამედიცინო მეცნიერებანი

მოწვევის გავლენა ენდოთელურ ფუნქციაზე

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თბილისის სახელმწიფო სამედიცინო უნივერსიტეტის შინაგან სნეულებათა დეპარტამენტი

(წარმოდგენილია აკადემიკოს ვ. ოკუჯავას მიერ)

მოწვევა იწვევს ენდოთელური ფუნქციის დარღვევას არა მხოლოდ აქტიურ, არამედ პასიურ მწვევლებშიც. ავტორთა კვლევის მიზანს წარმოადგენდა ენდოთელური ფუნქციის შესწავლა არაინვაზიური მეთოდის —ულტრასონოგრაფიის გამოყენებით [ნაკადით განპირობებული დილატაცია (ნგდ) მწვევლ და არამწვევლ პოპულაციაში]. გამოკვლეულ იქნა 32 კაცი ნორმალური სისხლის წნევისა და სისხლში ქოლესტერინის ნორმალური მაჩვენებლებით. აქედან 14 იყო აქტიური, 6 პასიური მწვევლი, ხოლო 12 არ იყო მწვევლი. გამოკვლეულ პირებს არ აღენიშნებოდათ გულსისხლძარღვთა დაავადებებისა და დიაბეტის კლინიკური ნიშნები. მწვევლებში არამწვევლებთან შედარებით ნგდ მაჩვენებლები იყო მნიშვნელოვნად დაბალი. პასიურ მწვევლებში არამწვევლებთან შედარებით ენდოთელური ფუნქცია იყო მნიშვნელოვნად დარღვეული და არასარწმუნოდ განსხვავდებოდა მწვევლების ენდოთელური ფუნქციისაგან.

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