Passive smoking affects acutely the microcirculation of healthy never-smoking volunteers

Authors:
V Linardatou1, E Karatzanos1, D Delis1, S Pagoni2, C Kourek1, N Panagopoulou1, A Leventakis1, I Vasiliadis3, S Nanas1. 1Clinical Ergospirometry, Exercise & Rehabilitation Laboratory, School of Medicine, NKUA - Athens - Greece, 2General Hospital of Athens G. Gennimatas, Pathology Dpt - Athens - Greece, 3Sotiria Regional Chest Diseases Hospital, 1st Respiratory Medicine, NKUA - Athens - Greece.

Topic(s):
Tobacco

Citation:

Background: The effects of cigarette smoking exposure on vascular health, particularly related to incidents of cardiovascular events, are well established. Cigarette smoke is responsible for preventable deaths worldwide. The negative effects of passive smoking exposure on the cardiovascular system are notably similar in significance to those caused by active smoking. Cigarette smoking is associated with acute alterations in microcirculation which include reduced blood flow. Acute effects of passive smoking on microcirculation have not been sufficiently studied. Near-Infrared Spectroscopy (NIRS) combined with vascular occlusion technique (VOT) is a reliable, clinical research tool for the assessment of microcirculation.

Aims: The aim of this study was to detect alterations in peripheral microcirculation of non-smokers after passive exposure to cigarette smoke.

Methods: Sixteen healthy (7?, 9?), never-smoking volunteers (age: 34±9 years) were exposed to 30 minutes passive smoking. All participants were not regularly exposed to an environment polluted by cigarette smoke. Exclusion criteria were also black skin, body mass index greater than 30 kg/m2, history of respiratory, cardiovascular disease and anemia. Measurements were made in smoke-free, temperature-controlled (21 to 22 °C) environment before and after exposure to passive smoking. Smoke concentration was monitored with a real-time particle monitor. Microcirculatory parameters were measured with NIRS combined with VOT. The optode of NIRS was placed on the thenar muscle. A pneumatic cuff was placed above probe on the upper arm and rapidly inflated (to 50 mmHg above each individual’s systolic blood pressure) to induce arteriovenous occlusion for 3’. After the occlusion and the transient ischemia, the cuff was quickly deflated. StO2 baseline, Oxygen consumption rate (StO2 decrease slope), Oxygen reperfusion rate (StO2 increase slope) and the time needed for StO2 to return to baseline level after reactive hyperemia (time to hyperemia end) were assessed. O2 consumption rate reflects the metabolic activity of the muscle and the adequacy of microcirculation to provide necessary oxygen to the tissue. Oxygen reperfusion rate and time to hyperemia end are indexes of endothelial function. The values refer to mean ± SD.

Results: Baseline StO2 didn’t change significantly after passive smoking (79.6±6.4 to 79±8%, p=0.53). O2 consumption rate decreased after 30min exposure to passive smoking from 12.8±4.2 to 11.3±2.8 units/min (p=0.04) and reperfusion rate decreased from 5.6±1.8 to 5±1.7 units/sec (p=0.04). There was not a significant difference on time to hyperemia end (138.2±26.5 to 142.1±34.6 sec, p=0.64).

Conclusions: Our results suggest that acute exposure to passive smoking deteriorates microcirculatory function in healthy never-smoking subjects. Repeated exposure and its effects on endothelium and microcirculation need further investigation.