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Letters

TO THE EDITOR

Bad Air Revisited

The benefits of a respiratory filter researched by Vieira et al. (1) and the constructive editorial by Mentz and O'Brien (2) enhance the attention that has been given to the cardiovascular effects of air pollution.

In the eligibility criteria, patients with "uncontrolled arrhythmia or hypertension" were excluded from the study. Does this refer to the blood pressure recorded during the initial pre-study evaluation and history? Importantly, what were the cutoff points of systolic and diastolic pressures considered to be uncontrolled? The medications used in the treatment of hypertension and heart failure are often similar. Is it possible that specific endpoints and results could be influenced by the individual therapeutic programs? This may have significance, because the authors noted that diesel exhaust had no effect on heart rate variability. This is in contrast to the recent study by Lee et al. (3), which concluded that exposure to ambient fine particulate matter produced remarkably sudden acute changes in nocturnal heart rate variability in patients without known heart disease.

A micron (micrometer) is a unit of length equal to one-millionth (10^{-6}) of a meter. Coarse particles are those considered to be >2.5 μm, and fine particles are 2.5 μm or less. Microfilters can (presumably) remove particulate matter from 2 to 5 μm in diameter.

Initial studies (epidemiological and clinical research) focused their attention on the respiratory and pulmonary effects of particulate matter, gases, and fumes. These studies were unanimous as to the positive causation of lung cancer and various inflammatory diseases of the entire respiratory system and gave additional consideration to less well-defined conditions such as leukemia, birth defects, reproductive problems, hormonal dysfunction, alterations in natural immune defense mechanisms, mutations in chromosomes, and damage to DNA. It became evident that air pollution contributed to excesses of morbidity and subsequent mortality and that toxic substance-induced pathophysiological cardiovascular effects were responsible for these excesses. These included angina pectoris, cardiac arrhythmia, myocardial infarction, progression of atherosclerosis, stroke, heart failure, and sudden cardiac death (4). Ambient air also contains ozone, carbon monoxide, sulfur oxide, nitrogen oxide, hydrocarbons, carbon dioxide, methane, various dusts and pollens, and numerous other chemical pollutants. Unbridled diesel fumes are one of the most toxic substances on earth, injurious to animal and plant life, as well as to all ecological aspects of the environment. Some manufacturers state that their present diesel engines are "clean," but confirmatory studies are sparse or nonexistent. More than 15 years ago, the California Environmental Protection Agency noted that diesel exhaust (spent diesel fumes) contained more than 40 toxic contaminants, all significantly harmful, both individually and even more so in combination.

A personal quest, which began 3 decades ago, eventually resulted in the placement of recirculation air systems in automobiles and the addition of ventilation filters (5). Recirculation systems keep external air from entering the vehicle, and filtration systems capture ambient air particulates and, to some extent, diminish incoming fumes and odors. Both are necessary to decrease the harmful effects to the occupants of motor vehicles. At present, both systems need improvement. Automobile manufacturers should be required to state the type of filter used in the vehicle, as well as the performance of the filter in terms of particle size and effectiveness in fume and odor removal.

What else can the public (whether healthy or afflicted with certain diseases) do besides use personal respiratory filters? When applicable, avoid outdoor jogging, especially in known or expected areas of excess air pollution, and use indoor air "purification" systems. Importantly, do not buy an automobile without a ventilation filter, sometimes called in-cabin air filters. Preferably (in relation to cost) buy a car that has a filter that uses activated charcoal, carbon, carbonite, grape seed, etc., each of which adds additional cleansing of the incoming air.

It is hoped that these recommendations will provide additional personal protection, as afforded by the study of Vieira et al. (1) and commentary by Mentz and O'Brien (2).

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Please note: Dr. RuDusky has reported that he has no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Vieira JL, Guimaraes GV, de Andre DA, Cruz FD, Saldiva PH, Bocchi EA. Respiratory filter reduces the cardiovascular effects associated with diesel exhaust exposure: a randomized, prospective, double-blind, controlled study of heart failure: the FILTER-HF Trial. J Am Coll Cardiol HF 2016;4:55–64.

2. Mentz RJ, O'Brien EC. Air pollution in patients with heart failure: lessons from a mechanistic pilot study of a filter intervention. J Am Coll Cardiol HF 2016;4:65–7.

3. Lee MS, Eum KD, Rodrigues EG, et al. Effects of personal exposure to ambient fine particulate matter on acute change in nocturnal heart rate variability in subjects without overt heart disease. Am J Cardiol 2016;117:151–6.

4. Brook RD, Rajagopalan S, Pope CA 3rd, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation 2010;121:2331–78.

5. RuDusky BM. Your Car Can Be Hazardous to Your Health. Bloomington, IN: Author House, 2002:35–73.

REPLY: Bad Air Revisited

We thank Dr. RuDusky for taking an interest in our work. Our study was intended to address the role of a respiratory filter intervention during controlled pollution exposure in patients with heart failure (HF). In 2007, HF was associated with 39.4% of all hospitalizations due to cardiovascular diseases in Brazil, and it may be responsible for 6.3% of all causes of deaths in South America (1). Regarding hypertension, it is a leading risk factor for cardiovascular disease and a significant cause of morbidity and mortality as long as it remains uncontrolled (2). A large body of evidence indicates that patients with hypertension are characterized by endothelial dysfunction (3). We excluded volunteers with uncontrolled hypertension because it could play an important role as a confounder and selection bias, especially in a small sample of patients with HF. Blood pressure (BP) was recorded during the initial pre-study evaluation and history and also at the beginning of each session. Uncontrolled hypertension was defined as an average systolic BP ≥140 mm Hg or an average diastolic BP ≥90 mm Hg, among those with diagnosed hypertension and who are currently using BP-lowering medication. As outlined in the article, patients with HF were receiving optimal medical therapy, and 2 volunteers from the control group were receiving beta-blocker therapy.

Although our neutral findings of heart rate variability (HRV) could be explained by the optimal beta-blocker therapy in the HF group, it is noteworthy that diesel exhaust exposure (DE) also did not affect HRV in the control group. This suggests that the use of cardiovascular therapies might not be the primary explanation for the absence of an effect of air pollution on autonomic function. There are several methodological differences between the recent study by Lee et al. (4) and ours that can explain these contradictory findings. Lee et al. (4) assessed lagged nocturnal effects of fine particulate matter (PM2.5), whereas we conducted an experimental short-term study with controlled DE exposure that provided a precisely defined PM2.5 concentration in a regulated environment. We cannot state whether longer-term air pollution exposure could affect the HRV in optimally treated HF patients.

The epidemiologic association between air pollution exposure and exacerbation of cardiovascular disease is well established, yet the mechanisms underlying the increased risk of cardiovascular events are incompletely understood. Increasing concern relating to the health effects of air pollution has led many individuals to use facemasks to reduce personal exposure (5). There is, therefore, a need to consider approaches that can reduce effects of ambient air pollution exposure on both a societal and a personal level. Reduction of traffic emissions involves economic and political difficulties. The pioneering demonstration that a simple filter intervention can reduce the adverse effects of pollution in patients with HF could provide an inexpensive strategy for preventing HF decompensation. Given the worldwide prevalence of exposure to traffic-related air pollution, we speculate that patients with uncontrolled hypertension may benefit from the filter intervention as well.

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REFERENCES

1. Bocchi EA, Arias A, Verdejo H, et al. The reality of heart failure in Latin America. J Am Coll Cardiol 2013;62:949–58.

2. Sakhuja A, Textor SC, Taler SJ. Uncontrolled hypertension by the 2014 evidence-based guideline: results from NHANES 2011-2012. J Hypertens 2015;33:644–51; discussion 652.

3. Gkaliagkousi E, Gavriilaki E, Triantafyllou A, Douma S. Clinical significance of endothelial dysfunction in essential hypertension. Curr Hypertens Rep 2015;17:85.