Air Pollution and the Heart: Congested Roads and Blocked Arteries

Theodore K. Fenske, MD, FRCPC, FACC, FCCP is a Clinical Professor, Division of Cardiology, University of Alberta, Clinical Staff, Royal Alexandra Hospital, Edmonton, Alberta.

Presented at the Cardiology Rounds, Royal Alexandra Hospital, November 2008.

Peter's case

Peter is a 54-year-old businessman with a family history of premature coronary artery disease and a personal history of hypertension, dyslipidemia, non-insulin-dependent diabetes mellitus and obesity. He leads a sedentary existence, spending > 3 hours each day in his vehicle driving to his office and to various meetings and events. He is already on proven effective risk reduction therapies, including antihypertensive, lipid-lowering and oral hypoglycemic medications.

In addition to closer attention to diet and regular exercise, what else can Peter do to help reduce the risk of developing CVD?

For the correct answer, see page 44.

Nostalgic comments of "the good old days" ran high as antique-car hobbyists and auto aficionados gathered to help mark the centennial for the Model T Ford this year. Affectionately called the "Tin Lizzie," the Model T was the first mass-produced vehicle in the world. During the 19 years of its production, there were > 15 million turned out, establishing Ford as the best-selling automobile brand in the US and the Model T as the car of the century. Unfortunately, however, the popular phrase "it's all good" does not apply to Ford's legacy. In fact, Henry Ford's dream to "put America on wheels" has turned out to be more of a nightmare. Not only are tailpipe emissions playing a prominent role in accelerating global warming, but they are adding significantly to the modifiable risk factors that drive coronary heart disease in our rapidly-growing, high-risk population.

The scope of the air quality problem

It has long been known that air pollution can adversely affect human health. A number of catastrophic events from the last century, such as the Meuse valley fog of 1930, have taught us the unfortunate lesson that extreme levels of air pollution cause a direct increase in the death toll. In addition to the well-attested respiratory concerns caused by increased exposure to air pollution, mounting evidence over the past 15 years has drawn attention to the damaging effects of air pollution on the CV system. The landmark Harvard Six City Study demonstrated that in a cohort of > 8,000 adults living in various cities in the US and followed for 16 years, the greatest number of CV deaths occured in the most polluted cities. The recently published Women's Health Initiative established a strong statistical association between fine particulate air pollution and death from coronary heart disease. In Coachella Valley, California, a detailed review of the daily death counts in relation to air pollution levels indicated that an increase in

It has long been known that air pollution can adversely affect human health.

particulate matter concentration of 10 ug/m3 was associated with a 1% increase in total mortality. A similar linear relationship has been shown between mean annual air pollution exposure and increasing carotid artery intima-media thickness, as well as direct CV mortality.

Air pollution consists of both particulate matter and gaseous components. Ambient particles are subdivided on the basis of aerodynamic diameter (AD) into coarse particles (AD between 2.5 um and 10 um) which arise from windblown dust and wildfires, fine particles (AD < 2.5 um) which derive from refineries and tailpipe emissions and ultrafine particles (AD < 0.1 um) which also derive predominately from tailpipe emissions. Environment Canada records air quality levels in specially-equipped stations throughout the country and routinely records levels of fine particulate matter (PM < 2.5), as well as, gas levels including carbon monoxide, nitrogen dioxide, sulphur dioxide and ozone. The US and Canada use similar estimation methodologies to compile the data on atmospheric emissions in reference to standardized norms in order to arrive at an hourly index of air quality (see Environment Canada website for details).

Tailpipe emissions account for a large proportion of the pollution that we inhale on a daily basis. In both North America and Europe, direct emissions from personal vehicles and freight transport have been steadily rising. Rapid urbanization and increasing time spent in congested traffic means that exposure is increasing even where pollution levels are reported to be falling. In China, where one-fifth of the globe's population reside, the rapid economic growth has gone hand in hand with a doubling in transportation use between the years 1990 and 2003 and there are predications that the number of motorized vehicles could increase by 15-fold over the next 30 years. Since ambient air pollution indices worsen with rising temperatures, air quality may become an even greater issue as global warming continues.

The mechanisms of air pollution-induced cardiac injury

The mechanisms of air pollution-induced CV compromise can be considered in two separate groups:

1 Triggering acute vascular injury
2 Accelerating chronic atherosclerotic disease

Epidemiological studies correlating the day-to-day variations in atmospheric pollution with daily variations in death and disease have shown that elevated levels of ambient aerosols (including ozone, sulphur dioxide, nitrogen dioxide and carbon monoxide) have been associated with increased cardiac admissions to hospital and acute MI. The amount of time people spend on motorcycles and in their cars has been consistently linked to an increased risk of acute MI. Case-crossover studies have shown that heavy traffic exposure can trigger a MI within the hour in susceptible persons. A controlled provocative study involving 20 men with established coronary

artery disease demonstrated how diluted diesel exhaust inhalation caused more marked ST depression during exercise testing than when subjects breathed filtered air. This study illustrated the profound adverse effect tailpipe emissions can have on coronary perfusion in susceptible individuals.

The triggering of acute vascular injury by air pollutants is thought to be mediated by either a prothrombotic effect, by autonomic deregulation, or by a combination of both mechanisms. Experimental data has demonstrated that ultrafine PM may act as prothrombotic factors, increasing the risk of platelet activation and clot formation. Simultaneous analysis of ambient air pollution levels and coagulation parameters in volunteers have indicated that higher air pollution levels are associated with increased plasma viscosity and a shorter prothrombin time. As well, there is evidence suggesting that air pollution can affect the autonomic control of the heart, which may increase the risk of sudden death. Several studies indicate that decreased heart rate variability, a marker of cardiac risk, can be seen with air pollution exposure.

Air pollution is thought to accelerate chronic vascular disease by inciting endothelial dysfunction mediated by oxidative stress and vascular inflammation. Endothelial dysfunction has been shown to occur in healthy volunteers after as little as two hours of inhaling a mixture of concentrated ambient particles plus ozone. Studies done in the Los Angeles basin demonstrated that long-term exposure to air pollution was associated with a significant increase in carotid artery intima-media thickness, such that for every 10 ug/m3 increase in fine PM exposure (PM 2.5), there was a 6% increase in intima-media thickness. It has been shown that these air pollutants are fine enough to be inhaled deeply into the alveoli, where they can generate reactive oxygen species and in turn can trigger pulmonary inflammation. The inflammatory cascade initiated in the lungs can result in a release of inflammatory mediators into the systemic circulation, as evidenced by documented increases in C-reactive protein levels. As well, ultra fine PM has been shown to translocate into the bloodstream and can be found in remote organs, including the heart, ideally positioned to induce direct toxic CV effects via vascular inflammation.

Improving air quality

The Beijing Olympics provided an opportunity to trial the implementation of a number of air quality improvement strategies. In the months preceding the games, the Chinese government took action to reduce the amount of air pollution in Beijing by limiting both private and government vehicle volume, instituting driving restrictions for truck drivers, providing higher quality fuels for public use, halting construction activities in the city and temporarily closing chemical, steel and manufacturing plants, as well as reducing power generation.

While the ideal solution to counter the adverse effects of air pollution on the CV system is to reduce the sources of pollution, realistically, city smog is not going to blow away anytime soon. But that is not to say we cannot endorse some small steps in the right direction. The established strategies to reduce exposure to air pollutants, such as keeping car travel to a minimum, avoiding travel during peak rush hours, refraining from idling, keeping our vehicles well-tuned, car pooling, trip chaining and preferentially making use of public transit are obvious and should rank highly. Improved engine efficiency and changes in fuel sources, as well as, switching from gas-guzzling

FAQ

How can exposure to tailpipe emissions be reduced if car travel is essential?

Avoid car travel during peak rush hours, particularly in the heat of the day, since there is a direct correlation between increasing air pollution levels and higher temperatures. Shifting your workday an hour later and returning to dine with your family in style at 7 p.m or 8 p.m. could reduce both air pollution exposure and road frustration levels.

More on Peter's case

Peter is in a high-risk category for developing CVD. Aggressive risk reduction therapies, such as he is taking, are certainly warranted, but insufficient on their own. Heightened attention to lifestyle factors remains a critical ingredient to health maintenance. In addition to following more closely a prudent diet and taking part in daily aerobic exercise, Peter can help to reduce his risk of CVD by reducing his time spent driving his car. A brief discussion outlining ways that he can reduce exposure to tailpipe emissions and emphasizing the importance of active transportation would be in his long-term best interest.

Hummers to electric or hybrid smart cars are promising short-term solutions. As well, teleconferencing and e-mail can keep people in timely contact with one another without the carbon production inherent with driving to meetings and not to be overlooked on the list is opting for active transportation. We have allowed oil to displace food as the predominant energy source for moving ourselves about and this is an error—both for the health of our planet and our bodies. Changing our travel mode to active walking or bicycling instead of passively driving, would not only improve air quality, but would get us as a society on the right road to improving fitness levels and controlling weight.

Conclusion

Air pollution is unequivocally linked to adverse CV outcomes in the general population, with unfavourable effects even seen below existing air quality standards. In addition to ensuring that our patients' BP, glucose levels and lipids are under control, we need to raise concern about their exposure to air pollution and include smog on their list of modifiable risk factors. With our smoking patients we need to emphasize that air pollution may be cumulative if not synergistic with the cigarettes injury on the blood vessels and for our other high-risk patients, they should be urged to take precautions and limit exposure on days with high levels of pollution to help prevent the triggering effects of air pollution. Lastly, as a profession of influence, we would do well to model environmental stewardship by minimizing our own motor vehicle use, opting instead for alternative transportation modalities like the bus or the bicycle. Perhaps 30 years from now, when we celebrate the bicentennial of the bicycle, the world will be on pedal-powered wheels and our cars will join the Model T Ford as museum exhibits only.

For references, please contact **cme@sta.ca**

Take-home message

- Air pollution is an independent risk factor for developing CVD
- Tailpipe emissions can cause endothelial dysfunction and accelerate atherosclerosis
- Reducing exposure to air pollutants can help reduce the risk of CV events
- Active transportation, such as walking or bicycling, can significantly improve both our air quality and our fitness levels