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Acute Exposure to Air Pollution Triggers Atrial Fibrillation

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Abstract

Objective—The aim of the present study is to evaluate the association of air pollution with the onset of atrial fibrillation (AF).

Background—Air pollution in general and more specifically particulate matter has been associated with cardiovascular events. Although ventricular arrhythmias are traditionally thought to convey the increased cardiovascular risk, AF may also contribute.

Methods—Patients with dual chamber implantable cardioverter defibrillators (ICDs) were enrolled and followed prospectively. The association of AF onset with air quality including ambient $PM_{2.5}$, black carbon, sulfate, particle number, NO_2, SO_2, and O_3 in the 24 hours prior to the arrhythmia was examined utilizing a case-crossover analysis. In sensitivity analyses, associations with air pollution between 2 and 48 hours prior to the AF were examined.

Results—Of 176 patients followed for an average of 1.9 years, 49 patients had 328 episodes of AF lasting ≥ 30 seconds. Positive but nonsignificant associations were found for $PM_{2.5}$ in the prior 24 hours, but stronger associations were found with shorter exposure windows. The odds of AF increased by 26% (95% CI 8% to 47%) for each 6.0 μg/m^3 increase in $PM_{2.5}$ in the 2 hours prior to the event (p=0.004). The odds of AF was highest at the upper quartile of mean $PM_{2.5}$.

Conclusion—Particulate matter was associated with increased odds of AF onset within hours following exposure in patients with known cardiac disease. Air pollution is an acute trigger of AF, likely contributing to the pollution-associated adverse cardiac outcomes observed in epidemiological studies.

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Keywords

Air pollution; Atrial fibrillation; Particulate matter; Traffic

INTRODUCTION

Air pollution has been linked to overall mortality in epidemiological investigations (1–4) and most evidence points to increased cardiovascular disease as the primary driver.(5–8) Although air pollution is composed of multiple pollutants; in most studies fine particular matter ($PM_{2.5}$) is consistently associated with these cardiac events which include sudden cardiac death, heart failure, and myocardial infarctions.(2,4–9) The relative risk of daily cardiovascular mortality has been shown to be increased approximately 0.4% to 1.0% with a 10 μg/m^3 increase in mean 24 hour $PM_{2.5}$.(10) In addition, stroke has been associated with air pollution in several studies, with increased risks ranging from 0.4% to 18% with a 10 μg/m^3 increase in $PM_{2.5}$ on the day of the event.(11–15) $PM_{2.5}$ is produced by direct emissions from local and regional sources such as motor vehicles plus secondary particles from upwind fossil fuel burning.(4) Black carbon (BC), particle number count (PNC), and NO_2 are also produced predominantly by motor vehicles, while SO_4 and ozone are secondary pollutants produced in atmospheric reactions of emissions from upwind sources.

These associations between air pollution and health are generally observed in large population-based studies evaluating epidemiological evidence for death, or hospital admission data for myocardial infarctions, heart failure and stroke. This literature also includes patient based studies on ventricular arrhythmias, usually in susceptible populations with low left ventricular ejection fractions and implantable cardioverter defibrillators (ICD).(4,16–20) Based on these investigations, it is generally assumed that the majority of air pollution's cardiac effects are on the ventricle, and the cardiovascular complications of air pollution are mediated through ventricular arrhythmias, sudden death and worsened heart failure. However, a previous retrospective study by our group pointed to a possible association between atrial fibrillation (AF) with rapid ventricular responses and air pollution. That study was limited by the large proportion of these patients with single chamber ICDs which will only detect AF episodes that caused a rapid ventricular response.(21) Other studies have hinted at an association with AF, in that air pollution has been associated with some electrophysiological predictors of AF,(22) and also with atrial premature beats.(23,24)

The morbidity and mortality associated with AF is substantial. AF is responsible for more hospitalizations and longer hospital stays than any other arrhythmia, and may also lead to stroke, congestive heart failure, myocardial infarction and death.(25–31) Thus, AF, even if asymptomatic, is a very important cause of morbidity and mortality.(29,32) Whether air pollution contributes to AF has been difficult to detect, in large part because many episodes of AF are asymptomatic or minimally symptomatic. Therefore, individuals do not present for emergency treatment, making this arrhythmia unsuitable for population based studies. Even in the case of clinically recognized AF, the specific time of onset of these events is often not well defined. Electrocardiac monitoring studies are generally limited to short term monitoring for atrial arrhythmias and thus a true association may be missed. In this study, we evaluate the role of acute air pollution exposures as triggers of AF in a sample of high risk patients with *continuous* monitoring of atrial arrhythmias over an extended period of follow-up. Associations with traffic related and other air pollutants in the 24-hours prior to an AF event are evaluated, as well as alternative air pollution exposure windows relative to AF onset.

METHODS

Patient population

Subjects were recruited between September 2006 and March 2010 from patients followed at the Tufts Medical Center Cardiac Arrhythmia Center. Inclusion criteria included prior implantation of a dual (atrial and ventricular) chamber ICD and residential zip codes within a 50-kilometer radius of the Harvard Supersite air quality monitoring station. Exclusions included age younger than 18 years, chronic AF, lack of follow-up at Tufts Medical Center, terminal illness, or inability to give informed consent. The study protocol and informed consent were approved by the Institutional Review Board at Tufts Medical Center and the Harvard School of Public Health.

At the initial visit all patients completed an interviewer-administered questionnaire, including socio-demographic characteristics, medical history, detailed medication, lifestyle, and smoking history. They were measured for height and weight. A comprehensive past and current medical history, based on the National Cardiovascular Disease Data ICD Registry form was filled out by study coordinators based on review of medical records.

Arrhythmias

Patients were followed with either a clinic visit or by telephone every three months from study enrollment until June 30, 2010. At these encounters, ICD data which included the arrhythmia logbook and electrograms were downloaded directly or with trans-telephonic transmission and printed. All ICDs were dual chamber and capable of recording the date, time and real time electrograms of atrial and ventricular events. In addition to documenting the arrhythmia, the ICD characterizes each event as atrial or ventricular and as sustained or nonsustained, and records the total time of each episode. Heart rate detection and treatment rates for ventricular arrhythmias are programmed by the physician according to the specific needs of the patient. In general, treatment for ventricular arrhythmia begins at heart rates above 160 beats per minute (bpm). In addition to documenting rapid ventricular episodes, these devices also document and store electrograms of rapid atrial arrhythmias, even if the ventricular rate remains low.

Arrhythmias documented by the ICD were later reviewed and interpreted by an electrophysiologist (MSL) blinded to air quality. These arrhythmias were characterized as ventricular, sinus tachycardia, AF, atrial arrhythmia other than AF, or not an arrhythmia. Sinus tachycardia is characterized by gradual onset and a 1 to 1 atrial to ventricular association. Acute onset atrial arrhythmias were subdivided into AF and atrial arrhythmias other than AF. These arrhythmias generally do not have 1 to 1 atrial to ventricular association; typically the atrial rate is much faster than the ventricular rate. Irregular rapid atrial electrograms were classified as AF (Figure 1).

We restricted our analyses to clinically relevant AF defined as those lasting 30 seconds or longer as recommended by the American Heart Association, the American College of Cardiology and the Heart Rhythm Society. (27,33,34) To minimize clustering of events and overrepresentation of clustered events, if time periods between two successive AF episodes were shorter than 60 minutes, only the first episode of the arrhythmia was included in the analysis. Because of the possibility of pro-arrhythmia from the implantation of the ICD, all events occurring within 6 weeks of implant of the ICD device were excluded. Patients with less than 90 days of follow-up were excluded from analysis.

Air quality and weather data

Air quality was measured hourly at a Harvard Supersite monitor located on the roof of Countway Library 5 Km from Tufts Medical Center. Fine particle mass ($PM_{2.5}$, particulate matter ≤ 2.5 microns in aerodynamic diameter) was measured by Tapered Element Oscillating Microbalance (TEOM, Rupprecht and Patashnick, East Greenbush, NY), black carbon (BC) by aethalometer (McGee Scientific, Berkeley, CA), particle number count (PNC, number of particles per cm^3) by condensation particle counter (TSI Inc, Shoreview, MN), and sulfate (SO_4) by sulfate particulate analyzer (Thermo Fisher Scientific, Waltham, MA). Gaseous air pollutants in the Boston metropolitan area also were measured hourly and averaged over three sites for sulfur dioxide (SO_2), three sites for nitrogen dioxide (NO_2), and two sites for ozone (O_3), all operated by the Massachusetts Department of Environmental Protection.(35) Meteorological data (daily mean temperature, dew point, and barometric pressure) was obtained from the hourly surface observations of the National Weather Service at Logan Airport (East Boston).

Statistical Analysis

A case-crossover design allowed for investigation of the acute effects of exposure to air pollution.(36) The subject's exposures before the time of the event (case period) is compared with the distribution of exposure estimated from separate control periods and the matched sets are analyzed using conditional logistic regression, thus each patient serves as his or her own control.(9,37–40) For these case-crossover analyses, only individuals who had AF in follow-up could be included for analysis.

Case periods were defined by the time prior to onset of each confirmed arrhythmic event, rounded to the nearest hour. Control periods (3–4 per case) were selected using a bidirectional time-stratified approach by matching on weekday and hour of the day within the same calendar month.(41) Hourly pollution concentrations and weather conditions were then matched to the case and control time periods for analysis. With this approach, the odds ratio from the logistic regression model is a consistent estimator of the incidence rate ratio. Average air pollution for the 24-hours prior to the AF, modeled as a continuous variable and in quartiles, was evaluated. Odds of AF associated with air pollution were estimated with conditional logistic regression, adjusted for temperature and dew point, and reported as percent increased odds (odds ratios minus one times 100) for an interquartile range (IQR) increase in exposure. The IQR reflects the range of exposures that the individual study subjects are likely to experience. Exposure–response relationships were examined based on estimated odds of arrhythmias versus quartiles of air pollution. In sensitivity analyses, associations with mean air pollution for the prior 2, 6, 12, and 48 hours were examined. Associations restricted to those patients less than the median distance (26 km) from the air pollution monitoring site, and associations restricted to patients with 20 or fewer AF events were also explored. Data management and all statistical analyses were conducted using SAS software version 9.1.3 (SAS Institute, Cary, NC, 2008).

RESULTS

Patient Population

Of the 1143 subjects screened, 843 were excluded, some for more than a single reason. The majority of the exclusions were for single chamber ICDs (n=502), living outside the 50-kilometer radius of the Harvard Supersite air quality monitoring station (n=345) and/or chronic AF (n=84). Of the 300 eligible patients, 200 were enrolled (66%) and 176 were followed for at least 90 days. The median distance of patients' residence to the air pollution monitor was 20 km. Patients were 70% male, 93% Caucasian, had a mean age of 65 years (range 26 to 89 years) and a mean BMI of 29 kg/m^2 (range 16 to 57) (Table 1). The mean

left ventricular ejection fraction was 31.4% (range 10% to 70%). Indications for implantation of an ICD were primary prophylaxis in 111 subjects (63%), secondary prophylaxis in 33 subjects (19%), and syncope which clinically appeared arrhythmic, but without documentation of an arrhythmia in 32 subjects (18%). Twenty-nine percent of the population had a prior history of paroxysmal AF. Most patients were on standard pharmacologic therapy for cardiomyopathies which included beta blocking agents (93%), angiotension converting enzyme inhibitors or angiotension receptor blockers (84%), anti-platelet agents (68%) and statins (72%).

For the 176 patients with at least 90 days of follow-up, the mean follow-up time was 1.9 years. In the course of the study 9 individuals (of the 200 enrolled) died, including 7 individuals followed for at least 90 days. Two of these subjects had AF during follow-up. The total time under observation was 340 person-years.

Arrhythmias

During the study period 328 incident AF with a duration of ≥ 30 seconds were observed among 49 patients (Table 1). The mean number of AF episodes among these 49 patients was 6.7 (median 4, range 1 to 65, and IQR 7). Patients with AF were more likely to have a past medical history of AF or percutaneous coronary intervention for revascularization but were similar in other respects to those without AF. Patients with AF were more likely to have a past medical history of AF or percutaneous coronary intervention for revascularization but were similar in other respects to those without AF.

Air quality

Daily mean air pollution concentrations during the study period were modest (Table 2). Mean $PM_{2.5}$ was 8.4 $\mu g/m^3$ with only one day above the EPA standard of 35 $\mu g/m^3$ (maximum 52.4 $\mu g/m^3$). The traffic related air pollutants, i.e. $PM_{2.5}$, BC, and NO_2, were highly correlated (Pearson correlation > 0.5), although the strongest correlation (r=0.82) was between $PM_{2.5}$ and SO_4, an indicator of secondary, transported fine particles (Table 2). PNC was negatively correlated with $PM_{2.5}$ and SO_4, but positively correlated with SO_2 and NO_2 (indicators of primary emissions).

Air Quality and AF

Increased odds were found between mean $PM_{2.5}$ in the prior 24 hours and AF (Table 3). The strongest increased odds was a 14% increase (95% Confidence Interval (CI) −3% to 34%) for each 5.0 $\mu g/m^3$ increase in 24-hour mean $PM_{2.5}$. Stronger increased odds were found with shorter exposure periods before the AF event (Figure 2) with the strongest odds in the 2 hour moving average (26%; 95% CI 8% to 47%; Table 3) and effectively no association with mean over the previous 48 hours (Figure 2). A similar pattern was observed for BC, NO_2 and PNC in that increased relative odds were also found for 2-hour moving averages compared to the 24 hour moving average. In quartile analysis there was an apparent exposure response effect in that the highest risk of AF was in the highest quartile of $PM_{2.5}$ (Figure 3).

In sensitivity analyses restricted to the 25 patients (174 AF events) living closer than 26 km from the air pollution monitoring site, a stronger association was found with air pollution in the prior 2 hours for $PM_{2.5}$ (42% increased odds compared to 26% in the whole population), BC (22% increased odds compared to 16% in the whole population), NO_2 (28% increased odds compared to 18% in the whole population) (Figure 4). In analysis restricted to the 25 subjects with no history of AF prior to study enrollment (124 episodes of AF) the associations with $PM_{2.5}$ and BC were similar and stronger for NO_2 (Figure 5).

To ensure that patients with many events were not driving the positive association, the analysis was repeated with the exclusion of those individuals with > 20 AF episodes (3 individuals). Among these 46 patients (204 AF events), the increased odds of air pollution were either similar or of greater magnitude for $PM_{2.5}$ (28%; 95% CI 4% to 59%), BC (26%; 95% CI 7% to 49%), NO_2 (48%; 95% CI 19% to 85%), and PNC (44%; 95% CI 1% to 105%). There was still no association with SO_4, SO_2, or O_3.

DISCUSSION

Fine particulate matter was an acute trigger of AF in this prospective study. This study is unique for its ability to capture all (including asymptomatic) atrial arrhythmias in a population at high risk for subclinical and clinical cardiac events. In addition, this study is able to specify the precise time of AF onset, and match time of onset with hourly ambient air pollution measurements. In many epidemiological studies the time window for cardiovascular effects of pollution is over days to years. However, we observed evidence of a shorter time window of increased exposure of 2 hours which was associated with an acutely increased risk. This short time window may be due to the nature of AF mechanism compared to other cardiovascular endpoints such as myocardial infarction or congestive heart failure or to the more precise timing of the events as is present in current ICD data collection.

Atrial fibrillation may be triggered by changes in autonomic tone,(42–45) inflammation and oxidative stress,(46–48) atrial ischemia,(49) and atrial pressures changes.(27,49,50) Acute alterations in sympathetic and parasympathetic tone and reduced heart rate variability is well documented in air pollution studies in humans,(51–53) and animals.(54,55) Increases in particulate air pollution have also been linked to inflammation.(56–58) Cardiac ischemia is worsened by air pollution.(59,60) And finally increases in right ventricular and thus likely right atrial pressures are seen with increases in particulate matter.(61) Thus, it biologically plausible that particulate air pollution can acutely trigger AF.

In addition to the strongest odds found with $PM_{2.5}$, BC was associated with AF in the two hour moving average prior to AF. $PM_{2.5}$ incorporates pollution from a multiple sources, both direct emissions from motor vehicles and other local and regional sources and secondary particles from upwind fossil fuel combustion.(51,53,62) BC is mainly a marker of local and regional vehicular emissions. Other pollutants associated with vehicular emissions, PNC and NO_2 were also associated with AF, but did not reach statistical significance. Pollutants not associated with motor vehicular pollution (SO_4, SO_2, O_3) were not associated with AF.

A strength of this study was the ability to precisely define the time of onset of AF. Because AF may be asymptomatic or of short-duration, it is important to accurately and precisely define the time of onset of these events. Misalignment of air pollution exposures with acute cardiac events has been shown to produce an underestimate of the association and can mask true associations.(63) Another strength of this study is the hourly pollution measurements obtained in the Boston area, allowing for more accurate determination of association of air quality and arrhythmias. These precise hourly measurements allowed for examination of acute triggering (as little as 2 hour moving averages) of AF, in contrast to most previous studies which have relied on integrated calendar day mean air pollution data.

AF is responsible for more hospitalizations and longer stays than any other arrhythmia, and may lead to stroke, congestive heart failure and myocardial infarction.(25–28) AF is the most common arrhythmia in the United States and most other countries.(27,64) The morbidity and mortality of AF is largely based on the risk of embolic events, the induction

of heart failure and myocardial ischemia, and not on whether or not it causes arrhythmic symptoms. Given the known associations of AF and adverse cardiac outcomes,(25–29) and the results of this study it is likely that at least a portion of air pollution's adverse cardiovascular effects are mediated by AF. Therefore, it is quite likely that AF contributes to the morbidity and mortality that has been associated with air pollution.

Most concerning is that the higher odds of AF were observed at air quality levels well under ambient air quality standards. The Environmental Protection Agency (EPA) 24 hour standard for $PM_{2.5}$ is 35 μg/m^3.(65) In the current study the maximum $PM_{2.5}$ was 52.4 μg/m^3 and the 75th percentile was10.2 μg/m^3. In fact, in only one day did $PM_{2.5}$ exceed 35 μg/m^3 in Boston during the study time-frame. The average $PM_{2.5}$ during the study period was less than 60% of the current EPA annual average standard of 15 μg/m^3. The exposure-response shown in this study suggests that in cities with higher levels of pollution, the risk of AF may be even greater. Thus, these results raise important public health implications in that even low levels of air pollution such as that seen in Boston are associated with an increased occurrence of AF episodes.

Prior data linking air pollution to atrial arrhythmias are limited by a short duration of monitoring or incomplete monitoring of atrial arrhythmias. Incomplete arrhythmia monitoring is demonstrated by a sub-analysis of an earlier study of Boston ICD patients which reported associations of air pollution with atrial arrhythmias.(21) In this study, most patients had a single chamber ICD (ventricular) and thus only rapid AF such as would activate the ventricular arrhythmia zone were recorded. Consequently, these episodes were a highly selected subset of AF and likely were a minority of the atrial arrhythmia episodes.

Other studies on the association of atrial arrhythmias and air pollution utilize short and intermittent cardiac monitoring. For example, in a 24 week-30 minute/week Holter study of 32 adults in Ohio, 5-day moving averages of $PM_{2.5}$, sulfate and ozone were associated with an increased odds of supraventricular ectopy.(23) In a 4 week-24 hour/week Holter study of 57 German men with coronary artery disease, elevated concentrations of particulate matter and NO_2 in the prior 24-hours were associated with increased risk for atrial runs.(24) In another study monitoring PR prolongation and P wave complexity, possible predictors of AF, were associated with $PM_{2.5}$ in the prior 24 hours.(22) No prior study to our knowledge has utilized such an extended time period of follow-up with complete monitoring for all AF as was done in the current study.

Limitations

As in many air pollution studies there are limitations to single site air quality monitoring. Single site ambient monitoring may lead to exposure misclassification. Exposure misclassification is less likely with $PM_{2.5}$ because of the regional spatial homogeneity of this pollutant. Other pollutants with less spatial homogeneity may have weaker associations because of exposure misclassification. To overcome this issue we also analyzed only patients residing within 26 km of the monitoring station. In this analysis the effect of $PM_{2.5}$, BC and NO_2 were strong but with wider confidence intervals. The association with sulfate, PNC, SO_2 and O_3 remained weak and far from statistical significance.

Given the number of statistical tests performed (7 pollutants with 5 different lag times) it is possible that some of our results may be due to chance. However, the strongest associations were seen for the set of air pollutants indicative of motor vehicle exhaust. These associations were strongest for the 2 hour window prior to the AF and decreased with increasing lag times. There was also a dose dependent association with $PM_{2.5}$ in that higher pollutants levels had an increased odds of AF. For these reasons we believe these results present a coherent, biologically plausible evidence of a true association which is not due to chance.

A significant proportion of the study population (29%) had episodic AF prior to their enrollment. The prior history of AF is surely one of the strongest risk factors for AF; thus these individuals are at particularly high risk for AF. Yet, it appears that air pollution may initiate a specific episode in these individuals. In analysis restricted to those patients without a prior history of AF the results were unchanged compared to the entire group (Figure 5). Patients in this study were at higher risk of AF because of their underlying heart disease and reduced left ventricular ejection fraction. However, AF is the most common arrhythmia in the United States and age is one of the strongest risk factors, yet it is unknown whether these results are relevant to other populations also at risk for AF.

This study evaluated the acute onset of AF, not the duration or total time in AF. The total duration of AF may be more important than the number of episodes of AF. This study also focused on AF and not on ventricular arrhythmias.

CONCLUSIONS

In this high risk population, ambient air pollution increased the occurrence of AF episodes. Importantly, the air pollution-associated increase in AF demonstrated an exposure response risk of AF with worsening air quality, even in a city with air quality which is within the EPA mandated levels. Since AF is associated with stroke and cardiovascular disease, it is likely that AF contributes to the adverse outcomes of air pollution seen in epidemiologic studies.

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Abbreviations

AF	Atrial fibrillation
BC	Black Carbon
bpm	Beats per minute
ICD	Implantable Cardioverter Defibrillator
NO_2	Nitrogen Dioxide
O_3	Ozone
$PM_{2.5}$	Particulate Matter less than 2.5 μm aerodynamic diameter
PNC	Particle Number Count
SO_2	Sulfur Dioxide
SO_4^{2-}	Sulfate
$\mu g/m^3$	micrograms per cubic meter

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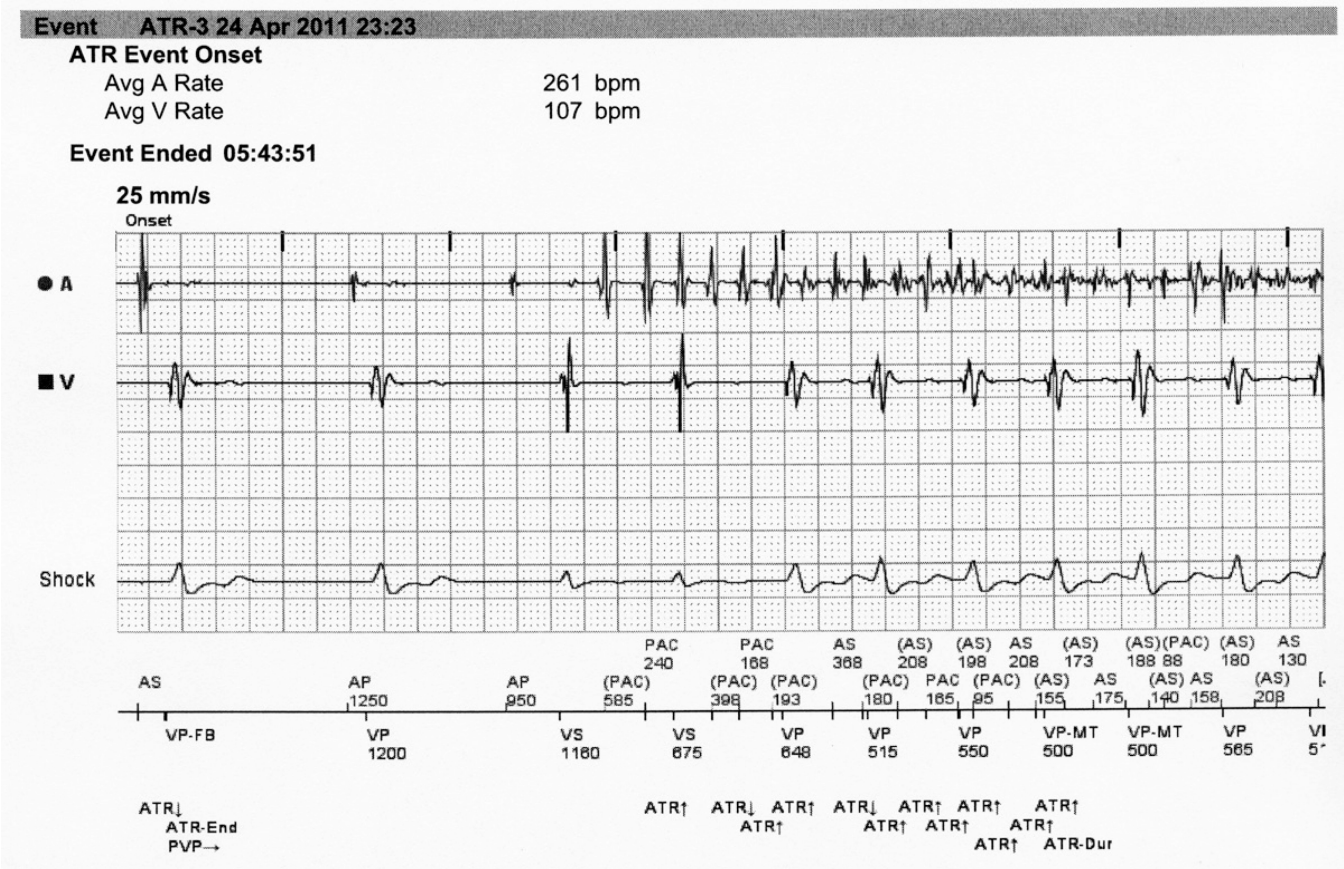

Figure 1. Electrocardiogram of atrial fibrillation

Intracardiac electrogram documenting the initiation of a disorganized atrial arrhythmia. Note the defibrillator documented time and date of arrhythmia onset as well as the intracardiac electrogram of both the atrium and the ventricle. The rapid and irregular atrial rate is typical of atrial fibrillation.

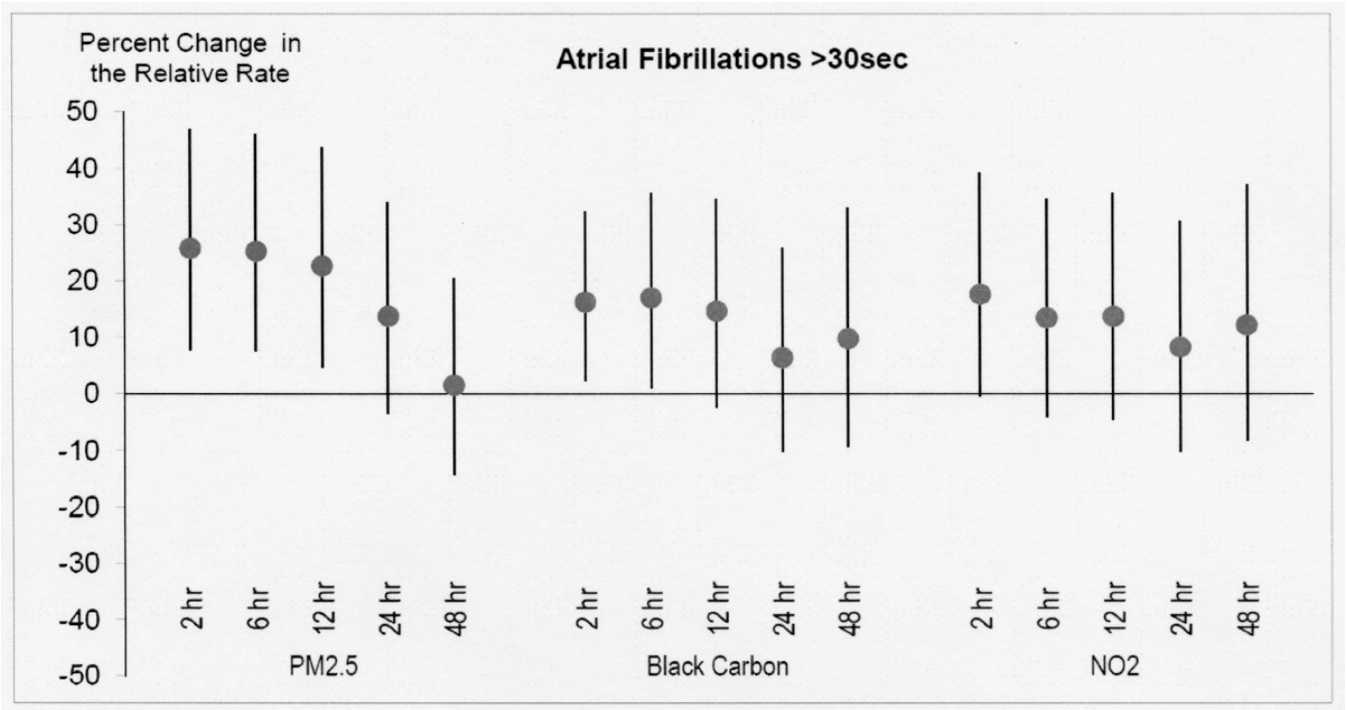

Figure 2. Risk of atrial fibrillation with air pollution

Percent increase in the occurrence (odds) of AF episodes associated with increase in air pollution ($PM_{2.5}$, black carbon, and NO_2) as a function of moving average in hours prior to event. Stronger increased odds were found with shorter exposure periods before the AF event with the strongest odds in the 2 hour moving average.

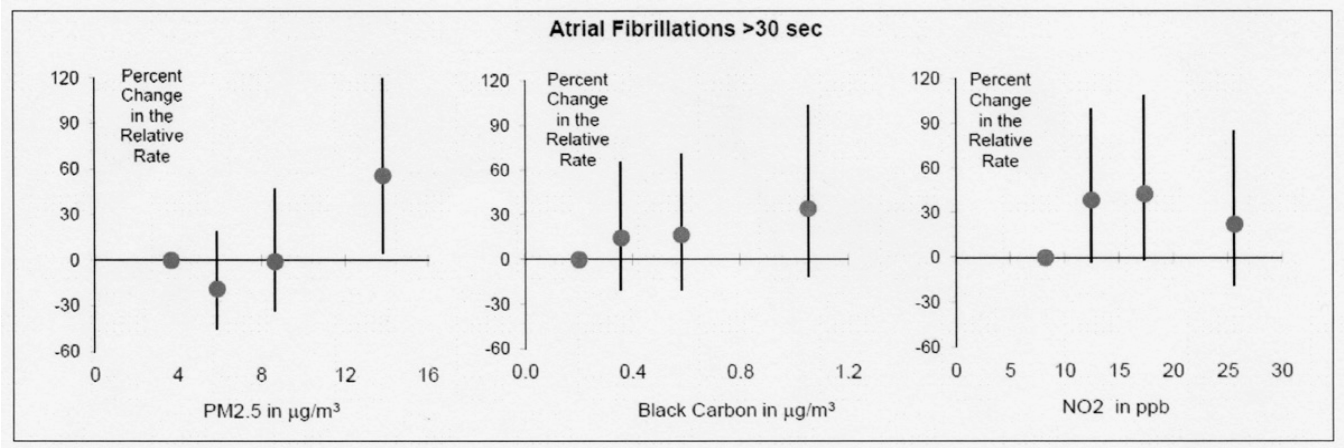

Figure 3. Risks of atrial fibrillation relative to quartile of pollution in the 2 hours prior to the arrhythmia

This figure models the predictors as quartiles and plots each quartile at the median concentration for that quartile. Percent increased occurrence (odds) of AF for 2-hour moving averages of air pollution ($PM_{2.5}$, black carbon and NO_2) versus quartile medians. There was an apparent exposure response effect in that the highest risk of AF was in the highest quartile of $PM_{2.5}$.

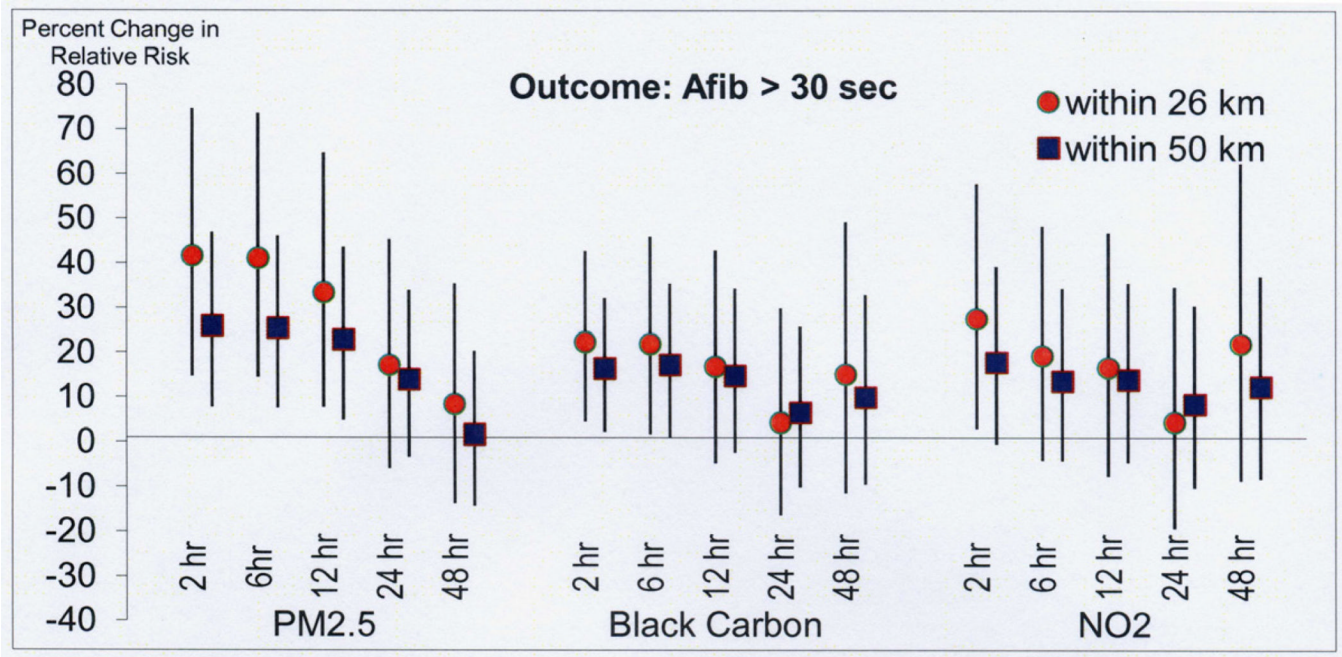

Figure 4. Risk of atrial fibrillation with air pollution in patients living within 26 km of the air pollution monitoring site

In analyses restricted to the 25 patients (174 AF events) living closer than 26 km from the air pollution monitoring site, a stronger association was found with air pollution in the prior 2 hours for $PM_{2.5}$ (42% increased odds compared to 26% in the whole population), BC (22% increased odds compared to 16% in the whole population), NO_2 (28% increased odds compared to 18% in the whole population).

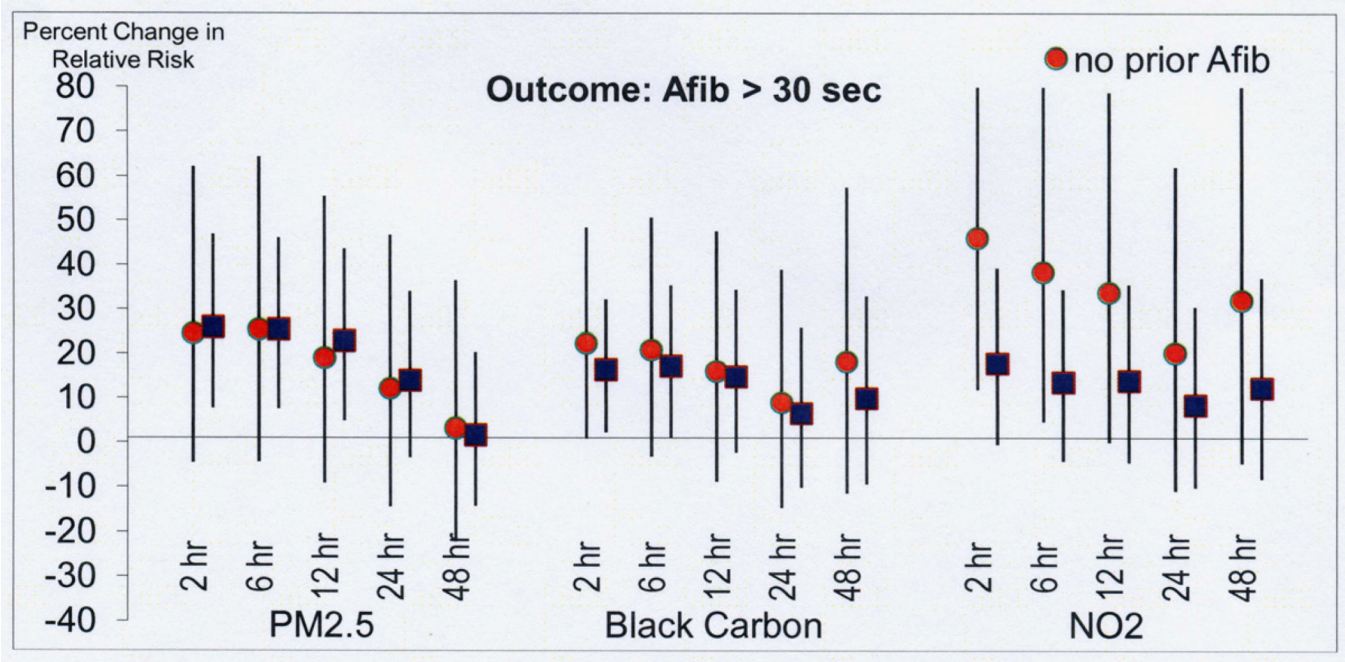

Figure 5. Risk of atrial fibrillation with air pollution in patients without a prior history of atrial fibrillation

In analysis restricted to the 25 subjects with no history of AF prior to study enrollment (124 episodes of AF) the associations with $PM_{2.5}$ and BC were similar and stronger for NO_2 compared to all patients.

Table 1

Patient Population (176 subjects followed for at least 90 days).

	All subjects N		Subjects with AF>30 sec N	
Age (years)	176	65.4 (26–89)	49	67.9 (35–88)
Gender (male)		123 (70%)		33 (67%)
Race				
White		164 (93%)		47 (96%)
Black		8 (5%)		2 (4%)
Other		4 (2%)		0 (0%)
BMI (kg/m^2)	158	29.0 (16–57)	40	27.8 (19–45)
Structural heart disease	176		49	
Ischemic		91 (52%)		30 (61%)
Nonischemic		52 (30%)		10 (20%)
Other		33 (19%)		9 (18%)
Left ventricular ejection fraction (%)	174	31.4 (10–70)	49	31.4 (10–70)
History of revascularization				
CABG	175	45 (26%)	49	15 (31%)
PCI	176	29 (17%)	49	13 (27%) *
History of congestive heart failure	176	96 (55%)	49	31 (63%)
CHF class I		19 (11%)		8 (16%)
II		31 (18%)		10 (20%)
III		46 (26%)		13 (27%)
IV		0		0
Indications for implant	176		49	
Primary prophylaxis		111 (63%)		31 (63%)
Secondary (cardiac arrest or sustained VT)		33 (19%)		7 (14%)
Syncope apparently arrhythmic		32 (18%)		11 (23%)
Co-morbidities				
Pulmonary Disease	176	31 (18%)	49	13 (27%)
Diabetes	174	47 (27%)	49	13 (27%)
Hypertension	172	98 (57%)	48	27 (56%)
History of AF	175	51 (29%)	49	24 (49%) *
Medications	170		47	
ACE-I or ARB		142 (84%)		41 (87%)
Beta blocker		158 (93%)		43 (91%)

	All subjects N		Subjects with AF>30 sec N	
Antiarrhythmic agents: (Amiodarone, sotalol, or others not including beta blockers)		28 (17%)		10 (21%)
Platelet Aggregation Inhibitors		115 (68%)		31 (66%)
Statin		123 (72%)		38 (81%)
Smoking				
Current	175	24 (14%)	49	5 (11%)
Former	175	85 (49%)	49	25 (51%)
Never	175	66 (38%)	49	19 (39%)
Lived with Smoker	175	111 (64%)	49	32 (65%)
History of sleep apnea	169	25 (15%)	45	7 (16%)

* P< 0.05 between those with and without atrial fibrillation in follow-up.

Table 2

Summary statistics and Pearson's correlation coefficient of daily mean air pollutant concentrations and meteorological variables in Boston, USA, during the study period from September 1, 2006 until June 30, 2010.

	Summary statistics					Pearson correlation coefficient			
	# Days	Mean	25th	50th	75th	$PM_{2.5}$	SO_4^{2-}	BC	PNC
Particles									
$PM_{2.5}$ (μg/m^3)	1213	8.4	5.3	7.2	10.2	1	0.82	0.64	−0.17
SO_4^{2-} (μg/m^3)	964	2.2	1.0	1.6	2.6		1	0.51	−0.30
BC (μg/m^3)	1315	0.59	0.36	0.52	0.76			1	0.04
PNC (1,000/cm^3)	1318	14.9	10.1	14.6	18.6				1
Gases									
SO_2 (ppb)	1399	3.2	1.8	2.7	4.0	0.22	0.13	0.23	0.58
NO_2 (ppb)	1399	16.1	11.9	15.2	19.2	0.37	0.24	0.59	0.47
O_3 (ppb)	1399	24.8	17.5	24.3	31.5	0.18	0.22	−0.21	−0.39
Weather									
Temperature (C)	1398	10.6	3.4	11.0	18.3	0.31	0.37	0.24	−0.74
Dew point temperature (C)	1398	3.5	−3.7	4.1	11.9	0.34	0.41	0.35	−0.72

Table 3

Percent change in risk of ICD detected arrhythmias associated with each interquartile range (IQR) increase in mean air pollution in the 2 and 24 hours prior to arrhythmic event, adjusted for temperature and dew point.

	Hours Prior	IQR	% change	(95% CI)	p-value
24 Hour Moving Average					
Particulates					
$PM_{2.5}$ (μg/m^3)	24	5.0	14%	(−3% , 34%)	0.12
BC (μg/m^3)	24	0.39	6%	(−10% , 26%)	0.47
PNC (1000/cm^3)	24	8.4	12%	(−19% , 56%)	0.49
SO_4 (μg/m^3)	24	1.6	0%	(−14% , 15%)	0.96
Gases					
NO_2 (ppb)	24	7.3	8%	(−10% , 31%)	0.41
SO_2 (ppb)	24	2.1	6%	(−13% , 29%)	0.54
O_3 (ppb)	24	14.0	18%	(−12% , 56%)	0.27
2 Hour Moving Average					
Particulates					
$PM_{2.5}$ (μg/m^3)	2	6.0	26%	(8% , 47%)	0.004 **
BC (μg/m^3)	2	0.49	16%	(2% , 32%)	0.021 *
PNC (1000/cm^3)	2	10.9	24%	(−4% , 61%)	0.099
SO_4 (μg/m^3)	2	1.7	2%	(−10% , 16%)	0.71
Gases					
NO_2 (ppb)	2	10.0	18%	(−1% , 39%)	0.056
SO_2 (ppb)	2	2.2	4%	(−10% , 20%)	0.59
O_3 (ppb)	2	17.0	3%	(−20% , 31%)	0.83