Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study

Richard P Sargent, Robert M Shepard, Stanton A Glantz

Abstract

Objective To determine whether there was a change in hospital admissions for acute myocardial infarction while a local law banning smoking in public and in workplaces was in effect.

Design Analysis of admissions from December 1997 through November 2003 using Poisson analysis.

Setting Helena, Montana, a geographically isolated community with one hospital serving a population of 68 140.

Participants All patients admitted for acute myocardial infarction.

Main outcome measures Number of monthly admissions for acute myocardial infarction for people living in and outside Helena.

Results During the six months the law was enforced the number of admissions fell significantly (−16 admissions, 95% confidence interval −31.7 to −0.3), from an average of 40 admissions during the same months in the years before and after the law to a total of 24 admissions during the six months the law was in effect. There was a non-significant increase of 5.6 (−5.2 to 16.4) in the number of admissions from outside Helena during the same period, from 12.4 in the years before and after the law to 18 while the law was in effect.

Conclusions Laws to enforce smoke-free workplaces and public places may be associated with an effect on morbidity from heart disease.

Introduction

Secondhand smoke increases the risk of acute myocardial infarction.[1–7] Smoking also increases the risk of acute myocardial infarction, but this risk falls rapidly after people stop smoking.[8] The effects of secondhand smoke on platelets and the endothelium both occur rapidly (within 30 minutes) and are nearly as large in passive as in active smokers.[1 2 5] The reductions in heart rate variability that occur with two hours of exposure increase the risk of myocardial infarction by around 10%.[9] Ordinances that end smoking in workplaces and public places both eliminate exposure to secondhand smoke and reduce the prevalence of smoking and cigarette consumption.[10] We examined whether enactment of legislation to require smoke-free workplaces and public places might be associated with a decline in hospital admissions for acute myocardial infarction.[11]

Helena, Montana, USA, is a geographically isolated community that imposed such a law from 5 June 2002. Opponents won a court order suspending enforcement of the law on 3 December 2002. This allowed us to examine the association of the ordinance with admissions for myocardial infarction from within Helena (intervention) and from outside Helena, where the ordinance did not apply (control).

Methods

St Peter's Community Hospital serves all heart patients in Helena and the surrounding area, with a total population of 68 140. It is nearly 100 km to the next nearest hospital with cardiology services.

About 90% of the population of Helena live in the 59601 zip code. The 10% remaining live in the 59602 zip code, which includes a residential area immediately adjacent to Helena. Many people who live there work in Helena. We surveyed 500 consecutive patients aged over 18 years old who resided in the 59602 zip code and were admitted to the hospital for all causes. Of the 213 of these patients who were employed outside the home, 192 worked in the 59601 zip code area, so we considered 59602 part of Helena for our analyses. We also included zip codes 59604 and 59624 (post office boxes in Helena). All other 596xx zip codes, 59713, and 59728 were considered "not Helena."

Selection of patients

St Peter's Hospital uses a combination of paper and electronic medical records and computerised billing. We reviewed charts for the months of June to November (the months the ban was in effect) for each year from 1998 to 2003 for patients with a primary or secondary diagnosis of acute myocardial infarction (ICD-9 (international classification of diseases, ninth revision) codes 410.xx). During these months, there were 10 497 admissions for all causes (including acute myocardial infarction) from Helena and 3367 from outside Helena. The attending physician made the diagnosis at the time of discharge, and the hospital billing staff assigned the codes. (Two of the authors (RPS and RMS) were attending physicians for 18 of the 304 admissions included in this study and so assigned the diagnosis. All but three of these patients were treated

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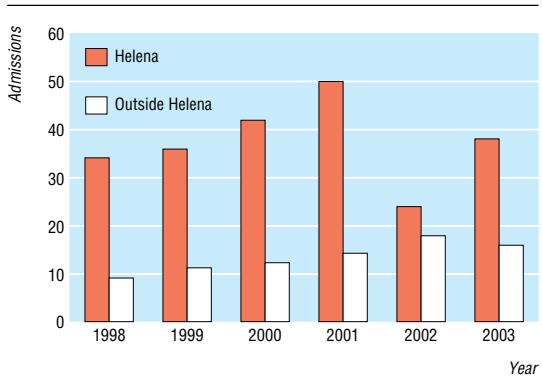

Admissions for acute myocardial infarction during six month periods June-November before, during (2002), and after the smoke-free ordinance (ordinance did not apply outside Helena). The law was implemented on 5 June 2002

before we thought of doing this study. These three patients were also seen by a cardiologist and thus had independent blinded corroboration of the diagnosis.) Data were sorted by primary and secondary diagnoses and by zip code to compare the incidence of acute myocardial infarction in residents with zip codes for the city of Helena and residents of the surrounding areas, where there was no ban.

We studied patients' charts if there was a primary or secondary discharge or emergency room diagnosis of acute myocardial infarction. Acute myocardial infarction was the primary diagnosis for 283 cases. Selection criteria were onset of symptoms in the study area, a primary diagnosis of acute myocardial infarction, and no recent procedure that could have precipitated acute myocardial infarction. We excluded eight cases because onset of symptoms occurred outside the study area and one because the patient died in the emergency room three days after angioplasty. The charts of three patients were reviewed because of multiple admissions in any 60 day period. Of a total of five such admissions, one was excluded because there was no chemical evidence (raised troponin I concentrations or creatine phosphokinase activity) for a new event. We therefore included 274 admissions with a primary diagnosis in the analysis.

We reviewed 71 cases with a secondary diagnosis of acute myocardial infarction. To be included, patients had to have chemical evidence (raised troponin I concentrations or creatine phosphokinase activity) at the time of admission or within the first 24 hours, onset of symptoms inside the study area, and no recent procedure that could have precipitated acute myocardial infarction. In the analysis we included 30 admissions with a secondary diagnosis and excluded 41.

Admissions for acute myocardial infarction during six month period (June to November) when smoking ban was enforced and equivalent months in years before and after ban, according to areas with (Helena) and without enforcement*

	Helena	Not Helena
Ordinance year (2002)	24	18
Other years†	40	12.4
Difference (95% CI)	–16 (–31.7 to –0.3)	5.6 (–5.2 to 16.4)
Helena difference–not Helena difference (95% CI)	–21.6 (–40.6 to –2.6)	

*All comparisons done assuming Poisson distribution.
†Average number of admissions during six month period for years other than 2002.

In all cases, we accepted the attending physician's diagnosis of acute myocardial infarction, and all attending physicians (other than the authors) were blinded to the study. In the three cases included after the study was started a consulting cardiologist, who was blinded to the study, confirmed the diagnosis, according to the medical record. We did not change any diagnosis. We excluded or included cases according to the criteria noted above.

We reviewed charts of patients from outside the study area to determine whether onset of symptoms was in or out of the study area and included them if the patient's symptoms started in the study area. Twenty six patients in the primary acute myocardial infarction group had out of area zip codes; 14 were included. Eight patients with a secondary diagnosis of acute myocardial infarction had zip codes out of the area. We included three patients with a diagnosis of primary myocardial infarction (for example, primary diagnosis of cardiogenic shock with secondary diagnosis of acute myocardial infarction) whose symptoms had started in the study area.

Overall we selected 354 admissions for review, and 304 met the inclusion criteria.

Statistical methods

We tested the hypothesis that the law was associated with changes in the total number of admissions for acute myocardial infarction in the six months of June to November (when the law was in effect). We compared the number of admissions during the six months the law was in effect (in 2002) with the average number of admissions during the same six months in the years before (1998-2001) and after (2003) the law.

Results

During the six months the smoke-free law was in effect (June-November 2002, figure), there was a significant drop in the number of admissions for acute myocardial infarction by −16 admissions (95% confidence interval −31.7 to −0.3) in Helena. During the same six months in the years before and after the law the average number of admissions was 40 compared with a total of 24 admissions during the six months of the law (table). There was a non-significant increase of 5.6 admissions per month (−5.2 to 16.4) from outside Helena during the same period, from 12.4 before the law compared with 18 during the law. The changes inside and outside Helena were significantly different during these months (table).

Discussion

During the implementation of a smoke-free law that applied to public places and workplaces we observed a significant drop in admissions for acute myocardial infarction. This is the first study to report such an association. Like any initial report, further research is desirable to confirm the finding. The observations that admission rates fell in the area where the law was implemented but not outside the area, suggests that smoke-free laws not only protect people from the long term dangers of secondhand smoke but that they may also be associated with a rapid decrease in heart attacks.

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Strengths of study

An important aspect of this study is that it was done in one isolated place with a single hospital that dealt with all admissions for acute myocardial infarction. In most other places that have implemented smoke-free policies, there are several hospitals with people moving across jurisdictional boundaries for work, housing, and health care. These factors "smear out" the effect of any smoke-free policies in both space and time. Data from California, however, could be interpreted as supporting our results. Death rates from heart disease fell faster in California than elsewhere in the United States during the California tobacco control programme,[12] which, while including a tax increase and media campaign (including the promotion of smoke-free environments), focused on creating smoke-free workplaces and public places.[13] The fraction of the population covered by smoking restrictions rapidly increased as a result of the campaign,[14 15] and there was a parallel reduction in deaths from heart disease.[12] Helena's small size and isolation were important contributing factors to our ability to detect a change in admission rates.

Weaknesses of study

Helena's small size, however, can also be an important limitation of the study as the total number of acute myocardial infarctions we observed was small. The statistical approach to analysis, with the Poisson distribution, does not account for the secular trend of increasing admissions over time (figure) and biases the results towards the null. Despite these small numbers and conservative statistical analysis, however, we were able to detect a significant change associated with the smoke-free law.

This is a "before and after" study that relies on historical controls (before and after the period that the law was in effect), not a randomised controlled trial. Because this study simply observed a change in the number of admissions for acute myocardial infarction, there is always the chance that the change we observed was due to some unobserved confounding variable or systematic bias.

Our data were from billing records for people who reached the hospital. We reviewed death records for Lewis and Clark County but did not include them because of concern about the accuracy of the assigned causes of death.

The criteria for identifying acute myocardial infarction changed during the study period. In March 1999 St Peter's began using troponin I concentration for diagnosis. To test whether this change could have affected the results, we conducted a regression analysis, including a variable indicating whether troponin I concentration was used; this variable did not approach significance. The change in diagnostic approach does not seem to affect our results.

We did not make any direct observations to measure how much exposure to secondhand smoke was reduced during the months when the law was in force. We do not know the prevalence of smoking in venues covered by ban, though the city-county health department reported that all but two businesses complied.[16]

What is already known on this topic

Secondhand smoke causes acute (within 30 minutes) changes in platelet and vascular endothelial function and reductions in heart rate variability that all increase the risk of an acute myocardial infarction

Epidemiological studies have shown that people living or working in an environment polluted with secondhand smoke have a 30% increase in risk of acute myocardial infarction

Smoke-free workplace and public place laws rapidly reduce exposure to secondhand smoke

What this paper adds

In the six months in which a law to ban smoking in the workplace and in public places was enforced in an isolated community, admissions to the local hospital for acute myocardial infarction fell compared with the same months in the years before and after the law was in effect

Smoke-free laws may be associated with a rapid effect on morbidity from heart disease

Relation to other studies

Researchers have predicted that smoke-free laws would be associated with a reduced incidence of acute myocardial infarction through a combination of reduced exposure to secondhand smoke and encouraging smokers to quit[11] (38% of the patients with acute myocardial infarction in the study were current smokers, 29% were former smokers, and 33% had never smoked at the time of admission). While both of these effects are probably occurring, we do not have large enough sample size to estimate their relative contribution to our results. Several mechanisms, including increased platelet activation,[1 2 5] inhibition of vascular endothelium,[17] impairment of coronary artery dilatation capacity,[18–21] decreases in antioxidant substances especially ascorbic acid,[22] aortic stiffening,[23] and impaired heart rate variability,[9] all of which could increase the likelihood of an acute coronary event, have been measured within minutes to hours of exposure to secondhand smoke. Even occasional exposure to secondhand smoke has been associated with an increased risk for acute coronary syndromes.[24]

The effect associated with the smoke-free law may seem large but is consistent with the observed effects of secondhand smoke on cardiac disease. Secondhand smoke increases the risk of a myocardial infarction by about 30%[3 4]; if all this effect were to occur immediately, we would expect a fall of $-0.30\times40.5=-12.2$ in admissions during the six months the law was in effect, which is within the 95% confidence interval for the estimate of the effect (a drop of -32.2 to -0.8 admissions). Creation of smoke-free environments, as required by the law, would also reduce the risk of acute myocardial infarction among those smokers who stop smoking or reduce consumption.

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Commentary: How acute and reversible are the cardiovascular risks of secondhand smoke?

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Could eating in a smoky restaurant precipitate an acute myocardial infarction in a non-smoker? As unlikely as this sounds, a growing body of scientific data suggests that this is possible. In this context, the results of the observational study in Helena, MT are provocative: hospital admissions for acute myocardial infarction declined by about 40% during the six months in which a comprehensive local ordinance on clean air was in effect, and rebounded after the ordinance was suspended.[1]

Given the small size and observational design of the study, these findings might be discounted or even disregarded altogether. However, the study focuses attention on an interesting subset of literature on secondhand smoke and its consequences. We now have a considerable amount of epidemiological literature and laboratory data on the mechanisms by which relatively small exposures to toxins in tobacco smoke seem to cause unexpectedly large increases in the risk of acute cardiovascular disease.[2–7]

Secondhand smoke causes coronary heart disease

Exposure to secondhand smoke increases the risk of fatal and non-fatal coronary heart disease in non-smokers by about 30%.[2 5 8 9] Because coronary heart disease is a leading cause of death in many countries, even relatively small increases in risk from this one factor can result in a large population burden of disease attributable to exposure to tobacco smoke.[10 11] While the substantial cardiovascular risks posed by active smoking are now almost universally accepted, the tobacco industry and some other observers continue to question the idea that secondhand smoke can cause cardiovascular disease and death.[12–15] Notwithstanding the substantial clinical and experimental evidence regarding the adverse cardiovascular effects of exposure to secondhand smoke, some have argued that an association between low level environmental exposures and health outcomes should be more critically evaluated, particularly when the relative risk for the exposure is below 2.0.[14 15] In addition, the risk of coronary heart disease associated with the typical self reported level of exposure to secondhand smoke (for example, that of a non-smoker living with a smoker) can seem disproportionate. It is more than one third of the risk associated with smoking 20 cigarettes a day, even though the measured exposure to tobacco smoke among non-smokers is only about 1% of the exposure from smoking 20 cigarettes a day.[2 4 5 16]

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