Hypertension

On a population-wide basis, hypertension is the most common predisposing factor for AF. The relationship between AF and blood pressure (BP) was first convincingly demonstrated by the Framingham Heart Study (FHS), in which hypertensive patients (defined as a systolic BP [SBP] of 160 mm Hg or higher, diastolic BP [DBP] of 95 mm Hg or higher, or use of antihypertensive medications) were significantly more likely to develop AF (odds ratio, 1.5 for men and 1.4 for women) over a 38-year follow-up. Similar to the Manitoba study demonstrated a 1.42-fold increased risk of AF in hypertensive individuals over a 44-year follow-up.

The association between higher SBP and AF has also been noted within a shorter follow-up time of 3 years.

The association between incremental SBP and DBP and risk of AF has been shown in a prospective, population-based study of 2014 Norwegian men who were nondiabetic and nonhypertensive at baseline. Over a 35-year follow-up, the risk of AF onset was increased 1.60-fold (95% CI, 1.15–2.21) with an SBP of 140 mm Hg or higher and 1.50-fold (95% CI, 1.10–2.03) with an SBP of 128 to 138 mm Hg, as compared with an SBP of 108 to 119 mm Hg.

Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. Much focus has been directed towards AF prevention, given the morbidity, mortality, and financial cost to health care systems associated with this arrhythmia. There are a number of common conditions associated with the onset of AF, but not only limited to hypertension, diabetes, or smoking. As we understand the factors associated with incident AF, public health campaigns and targeted patient interventions are warranted to promote blood pressure control, glycemic control in patients with diabetes, smoking cessation to prevent AF, and associated comorbidity. In this narrative review, we consider some of the evidence linking these risk factors with AF. We additionally examine the role of risk factor modification in reducing AF burden. In Part 1 we address the evidence for hypertension, diabetes, and smoking as risk factors for incident AF.

Hypertension

On a population-wide basis, hypertension is the most common predisposing factor for AF. The relationship between AF and blood pressure (BP) was first convincingly demonstrated by the Framingham Heart Study (FHS), in which hypertensive patients (defined as a systolic BP [SBP] of 160 mm Hg or higher, diastolic BP [DBP] of 95 mm Hg or higher, or use of antihypertensive medications) were significantly more likely to develop AF (odds ratio, 1.5 for men and 1.4 for women) over a 38-year follow-up. Similarly, the Manitoba study demonstrated a 1.42-fold increased risk of AF in hypertensive individuals over a 44-year follow-up. The association between higher SBP and AF has also been noted within a shorter follow-up time of 3 years.

The association between incremental SBP and DBP and risk of AF has been shown in a prospective, population-based study of 2014 Norwegian men who were nondiabetic and nonhypertensive at baseline. Over a 35-year follow-up, the risk of AF onset was increased 1.60-fold (95% CI, 1.15–2.21) with an SBP of 140 mm Hg or higher and 1.50-fold (95% CI, 1.10–2.03) with an SBP of 128 to 138 mm Hg, as compared with an SBP of 108 to 119 mm Hg.
These weaknesses were addressed by the multicenter MESA study (Multi-Ethnic Study of Atherosclerosis), which recruited an ethnically diverse cohort and measured BP on 3 separate occasions over a 5-year period to define a “sustained” BP category when 2 or more visits were within the same range. Both sustained prehypertension defined as a BP of 120 to 139/80 to 89 mm Hg and no antihypertensive medication use (hazard ratio [HR], 1.8; 95% CI, 1.004–3.2) and sustained hypertension defined as a BP of 140/90 mm Hg or higher or antihypertensive medication use at 2 consecutive visits (HR, 2.6; 95% CI, 1.6–4.4) were associated with an increased risk of AF. However, one limitation of this study was that incident AF was identified through assessment of discharge summaries and inpatient Medicare data claim records, raising the possibility that patients with
**Antihypertensive agents and incident atrial fibrillation**

<table>
<thead>
<tr>
<th>Trial</th>
<th>Year</th>
<th>Study population (n) and inclusion criteria</th>
<th>Trial drug</th>
<th>AF event rate in hypertensive population, n</th>
<th>Odds ratio (95% CI)</th>
<th>Trial outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAPPP</td>
<td>1999</td>
<td>4041; age, 75–66 y; men, 54.9%; DBP ≥100 mm Hg or both</td>
<td>Captopril vs β-blockers</td>
<td>30</td>
<td>3.02</td>
<td>1.05 (0.96–1.22)</td>
</tr>
<tr>
<td>STOP‑2</td>
<td>1999</td>
<td>4401; age, 70–84 y, men, 51%; SBP ≥180 mm Hg or DBP ≥105 mm Hg or both</td>
<td>Conventional vs newer antihypertensive</td>
<td>207</td>
<td>114 (4401)</td>
<td>1.14 (0.95–1.37)</td>
</tr>
<tr>
<td>TRACE</td>
<td>2000</td>
<td>790; mean age, 68 y; men, 71%; reduced LV systolic function (≤36%); following MI; sinus rhythm</td>
<td>1–2 mg/d trandolapril vs placebo</td>
<td>113</td>
<td>63 (235)</td>
<td>0.45 (0.26–0.76)</td>
</tr>
<tr>
<td>GISSI‑3</td>
<td>2000</td>
<td>17,749; age, 68 y; men, 71%; LVH on ECG</td>
<td>Lisinopril + nitrates; lisinopril</td>
<td>385</td>
<td>221 (150)</td>
<td>0.45 (0.26–0.76)</td>
</tr>
<tr>
<td>SOLVD</td>
<td>2000</td>
<td>186; mean (SD) age, 65.7 (11.7) y; men, 89.8%, white race, 100%; severe LVSD</td>
<td>5–20 mg/d enalapril vs placebo</td>
<td>45</td>
<td>150</td>
<td>0.22 (0.11–0.41)</td>
</tr>
<tr>
<td>VAL‑HeFT</td>
<td>2000</td>
<td>2205; median age, 63 y; men, 79.94%; ≥18 y, at least 3-month history of HF, NYHA II–IV symptoms</td>
<td>Valsartan or placebo</td>
<td>228</td>
<td>177</td>
<td>0.49 (0.38–0.62)</td>
</tr>
<tr>
<td>LIFE</td>
<td>2000</td>
<td>8851; hypertensive with LVH on ECG</td>
<td>Atenolol vs losartan</td>
<td>375</td>
<td>221 (150)</td>
<td>0.67 (0.55–0.83)</td>
</tr>
<tr>
<td>CHARMA</td>
<td>2000</td>
<td>14,939; age, 65 (11) y; men, 78%; HF with reduced LVEF (≤40%)</td>
<td>Candesartan with a target dose of 32 mg/d vs placebo</td>
<td>233</td>
<td>217 (150)</td>
<td>0.54 (0.26–1.09)</td>
</tr>
<tr>
<td>HOPE</td>
<td>2000</td>
<td>18,001; age, 55 y; men, 74%; without known HF/LV systolic dysfunction</td>
<td>Ramipril</td>
<td>421</td>
<td>220 (150)</td>
<td>0.92 (0.68–1.24)</td>
</tr>
<tr>
<td>VALUE</td>
<td>2000</td>
<td>39056; age, 55 y; men, 51%; AF or atrial flutter</td>
<td>Valsartan, 80–160 mg/d vs amlodipine, 5–10 mg/d</td>
<td>229</td>
<td>177</td>
<td>0.843 (0.713–0.997)</td>
</tr>
</tbody>
</table>

**Abbreviations:** ACEI, angiotensin‑converting enzyme inhibitor; ALLHAT, Antihypertensive and Lipid‑Lowering Treatment to Prevent Heart Attack Trial; CAPPP, CAPtopril Prevention Project; CHARM, Candesartan in Heart failure: assessment of Reduction in Mortality and morbidity; ECG, electrocardiogram; GISSI‑3, Gruppo Italiano per lo studio della sopravvivenza nell’Infarto miocardico; HOPE, Heart Outcomes Prevention Evaluation Study; VALUE, Valsartan Antihypertensive Long‑term Use Evaluation; others, see Table 1.
no cardiovascular risk factors were present (HR, 1.06; 95% CI, 0.98–1.15) compared with the presence of cardiovascular risk factors (HR, 1.14; 95% CI, 1.05–1.23).

One FHS offspring study report has noted that pulse pressure (HR, 1.26 per 20‑mm Hg increment; 95% CI, 1.12–1.43; \( P < 0.001 \)) but not mean arterial pressure (\( P = 0.39 \)) led to an increased risk of AF in a model adjusted for age, sex, and clinical risk factors.

In addition, individual components of BP, including SBP, pulse pressure, and even arterial pressure, have been evaluated regarding the risk of AF. The Cardiovascular Health Study demonstrated that risk of incident AF increased with every 10‑mm Hg rise in SBP (HR, 1.11; 95% CI, 1.05–1.18), and it was marginally lower if no cardiovascular risk factors were present (HR, 1.06; 95% CI, 0.98–1.15) compared with the presence of cardiovascular risk factors (HR, 1.14; 95% CI, 1.05–1.23). One FHS offspring study report has noted that pulse pressure (HR, 1.26 per 20‑mm Hg increment; 95% CI, 1.12–1.43; \( P < 0.001 \)) but not mean arterial pressure (\( P = 0.39 \)) led to an increased risk of AF in a model adjusted for age, sex, and clinical risk factors.

### TABLE 3

**Studies relating to diabetes and incident atrial fibrillation**

<table>
<thead>
<tr>
<th>Study</th>
<th>Population size, n</th>
<th>Population characteristics</th>
<th>Diabetic population</th>
<th>Follow-up duration</th>
<th>Risk of AF with DM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group Health in United States</td>
<td>1410 newly recognized AF cases and 2203 controls</td>
<td>Age, 30–84 y; case-control study from Group Health cohort; median age, 74 and 68 y in case and control groups, respectively; men, 35.4% and 45.2% in case and control groups, respectively; white race, 93.4% and 88.6% in case and control groups, respectively</td>
<td>Treated DM, 17.9% and 14.1% in case and control groups, respectively; patients with untreated DM excluded from analysis</td>
<td>3 y: 10/1/2001–12/31/2004</td>
<td>OR, 1.07; 95% CI, 0.75–1.51 for treated diabetes &lt;5 y OR, 1.51; 95% CI, 1.05–2.16 for &gt;5 but ≤10 y OR, 1.64; 95% CI, 1.22–2.20 for &gt;10 y OR, 1.03; 95% CI: 1.01–1.06 for each year treated diabetes duration</td>
</tr>
<tr>
<td>ARIC (^{11})</td>
<td>13025</td>
<td>Mean (SD) age, 57 (5.7) y; men, 44.1%; African-American, 22.9%</td>
<td>pre-diabetes, 51.4%; diabetes, 14.9%</td>
<td>14.5 y: 1990–2007</td>
<td>HR, 1.13; 95% CI, 1.07–1.20 in those with diabetesHR, 1.05; 95% CI, 0.96–1.15 in those without diabetes</td>
</tr>
<tr>
<td>NAVIGATOR (^{16})</td>
<td>8943 patients with impaired glucose tolerance</td>
<td>Median (Q1, Q3) age at screening, 63 (58, 68) y; men, 48.7%; white race, 82.9%</td>
<td>T2DM at baseline, 2.7%</td>
<td>16.4 y: 1993–2011</td>
<td>HR, 1.37; 95% CI, 1.03–1.83 for DM in multivariate-adjusted model HR, 1.09; 95% CI, 0.93–1.27 for DM and 1% rise in HbA1c level</td>
</tr>
<tr>
<td>HS (^{11})</td>
<td>34 720</td>
<td>Mean age in patients without T2DM at baseline, 52.8 (48.9–58.7) y; mean age in patients with T2DM at baseline, 55.5 (50.0–62.1); all female health professionals; age, ≥45 y; no cardiovascular risk, cancer, or AF</td>
<td>T2DM at baseline, 2.7%</td>
<td>16.4 y: 1993–2011</td>
<td>HR, 1.37; 95% CI, 1.03–1.83 for DM in multivariate-adjusted model HR, 1.09; 95% CI, 0.93–1.27 for DM and 1% rise in HbA1c level</td>
</tr>
<tr>
<td>ACCORD (^{16})</td>
<td>10082</td>
<td>Mean (SD) age, 62.2 (6.8) y; men, 61.4%; white race, 64.9%; patients with DM; HbA1c level ≥7.5; age range, 40–79 y; at least 1 additional cardiovascular risk factor</td>
<td>Intensive therapy targeting HbA1c ≤6.0%; standard therapy targeting HbA1c ≥7.0–7.9%</td>
<td>Median, 4.68 y</td>
<td>Incident rate of AF: 5.9/1000 person-years in the intensive-therapy group; 6.3/1000 person-years in the standard-therapy group (( P = 0.52 ))</td>
</tr>
</tbody>
</table>
| VALUE \(^{11}\)           | 15 245             | Mean age across group, 65–67 y; men, 57%, Caucasian race, 91% | 3 groups: non-diabetic, diabetic at baseline, diabetes developed during study | Mean, 4.2 y | Adjustable multivariate HR, 1.38; 95% CI, 1.05–1.80 for new-onset AF in new-onset DM 
Adjustable multivariate HR, 1.66; 95% CI, 1.13–2.44 for persistent AF in new-onset DM |
| Niigata Preventive medicine study \(^{16}\) | 28 449             | Japanese community; mean (SD) age, 59.2 (11) y; men, 34%, diabetics, 12% | Metabolic vs no metabolic syndrome as per NCEP-ATP III and AHA-NHLBI | Mean, 4.5 y | Metabolic syndrome: HR, 1.44; 95% CI, 1.09–1.90 for impaired glucose tolerance (NCEP-ATP III)HR, 1.35; 95% CI, 1.06–1.73 for impaired glucose tolerance (AHA-NHLBI) |

**Abbreviations:** ACCORD, Action to Control Cardiovascular Risk in Diabetes Study; AHA-NHLBI, American Heart Association – National Heart, Lung and Blood Institute; ARIC, Atherosclerosis Risk in Communities; DM, diabetes mellitus; HbA1c, glycated hemoglobin A1c; NAVIGATOR, Nateglinide and Valsartan in Impaired Glucose Tolerance Outcomes Research, NCEP-ATP III, National Cholesterol Education Program – Adult Treatment Panel III; T2DM, type 2 diabetes mellitus; others, see TABLES 1 and 2.
Smoking and incident atrial fibrillation

Table 4

<table>
<thead>
<tr>
<th>Study</th>
<th>Study period</th>
<th>Follow-up, y</th>
<th>Sample size, n</th>
<th>Population characteristics</th>
<th>AF cases, n</th>
<th>Group studied</th>
<th>Adjusted HR (95% CI)</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>FHS</td>
<td>1968–1971, 1981–1984, 1984–1987</td>
<td>1968–1971, 1981–1984, 1984–1987</td>
<td>Mean, 1.00 (0.7–1.4)</td>
<td>Current smokers</td>
<td>38 4764</td>
<td>Mean age, 60.9 y (range, 45 to 79 y); women, 45%</td>
<td>1.81 (1.0–2.2)</td>
<td>Smoking has no significant relationship with AF.</td>
</tr>
<tr>
<td>Rotterdam Study</td>
<td>1987–2002</td>
<td>2004–2012</td>
<td>Mean (SD), 15.2±2.1</td>
<td>Current smokers</td>
<td>190</td>
<td>Mean age, 59.2 y; mean age in smokers, 57 y; hospital-based cohort in Japan</td>
<td>1.89 (1.0–3.3)</td>
<td>Smoking was independently associated with the incidence of AF.</td>
</tr>
<tr>
<td>LKH</td>
<td>1990–1993</td>
<td>1990–1993</td>
<td>Age ≥ 55 y; women among current &amp; former smokers, 59% &amp; 45.8%</td>
<td>Current smokers</td>
<td>371</td>
<td>Age ≥ 55 y; women among current &amp; former smokers, 50% &amp; 48.5%</td>
<td>1.49 (1.1–1.9)</td>
<td>Smoking was associated with increased risk of atrial fibrillation.</td>
</tr>
<tr>
<td>Shinken Database</td>
<td>1987–2002</td>
<td>1990–2002</td>
<td>Mean age, 60.9 y (range, 45 to 75 y); men, 49%</td>
<td>Current smokers</td>
<td>152</td>
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<td></td>
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</tr>
<tr>
<td>Current smoker Brinkmann index ≥ 800</td>
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<td>190</td>
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</tr>
</tbody>
</table>
with a higher incidence of AF. A subgroup analysis of the VALUE Trial (an international multicenter trial to assess the incidence of cardiac events in hypertensive patients taking valsartan or amlodipine) similarly showed that patients who developed diabetes during the follow-up of 4.2 years had a higher risk of incident AF (relative risk [RR], 1.38; 95% CI, 1.05–1.80) and were more likely to have persistent AF (HR, 1.66; 95% CI, 1.13–2.44).

A number of studies have sought to investigate the relationship between glycemic control and the incidence of AF. For instance, the ARIC Study (Atherosclerosis Risk in Communities) showed a linear relationship between glycated hemoglobin $A_1c$ (HbA$_1c$) levels and AF incidence (HR, 1.13; 95% CI, 1.07–1.20 per 1% rise). The NAVIGATOR study showed that for a 1.0-mmol rise in fasting glucose levels there was a 33% increase in the risk of AF (HR, 1.33; 95% CI, 1.11–1.59). A community-based case control study known as the Niigata Preventive Medicine Study not only demonstrated that impaired fasting glucose was associated with a higher incidence of AF (HR, 1.35; 95% CI, 1.06–1.73; $P=0.001$), but also showed that individuals with metabolic syndrome (defined by National Cholesterol Education Program Third Adult Treatment Panel criteria) had a higher risk of AF onset (HR, 1.88; $P=0.001$).

A number of hypotheses have been postulated to explain the link between diabetes and AF, including diabetic microangiopathy, abnormal sympathetic tone, diabetic cardiomyopathy, and metabolic fluctuation. It is also pertinent to consider whether adequate glycemic control may reduce the risk of AF. Unfortunately, data from 10,082 diabetic patients from the ACCORD trial (Action to Control Cardiovascular Risk in Diabetes) showed that intensive glycemic control (HbA$_1c$ <6.0%) compared with standard glycemic control (HbA$_1c$ 7.0%–7.9%) led to no significant difference in the incidence of AF (5.9 per 1,000 patient-years and 6.37 per 1,000 patient-years, respectively, $P=0.52$).

In summary, diabetes increases the risk of incident AF (11%–37%) of incident AF. This risk includes prediabetes, metabolic syndrome, and treated diabetes. The risk of AF increases with poor glycemic control and longer duration of diabetes. Intensive glycemic control does not offer significant advantages at least in the short term.

Smoking A number of studies have explored the relationship between smoking and development of AF, in particular, the influence of duration and quantity of tobacco on AF risk. For instance, an analysis of 15,221 patients diagnosed with AF from the Shinken database revealed that smokers were more likely to develop AF, with an incidence rate of 9.0 and 5.0 per 1,000 patient-years for smokers and nonsmokers, respectively. There was additionally no difference in the risk of AF between men and women ($P=0.195$). The Manitoba follow-up study similarly demonstrated an increased risk of AF in smokers (RR, 1.37; 95% CI, 1.00–1.87). Furthermore, the ARIC study showed that both current (HR, 2.05; 95% CI, 1.71–2.47) and former smokers (HR, 1.32; 95% CI, 1.10–1.57) had an increased risk of AF, as compared with individuals who had never smoked. Those with the longest smoking history (≥675 cigarette-years) had the highest risk of AF (RR, 2.10; 95% CI, 1.74–2.53) compared with nonsmokers. Those who quit smoking had a marginally lower risk of AF (HR, 0.88; 95% CI, 0.65–1.17), as compared with current smokers, although the difference was nonsignificant ($P=0.38$).

What are the potential mechanisms? Smoking leads to an increased risk of AF by inducing oxidative stress, inflammation, and atrial fibrosis. Further research to consider the threshold at which these pathophysiological changes are reversible to return AF risk to baseline is warranted.

In summary, smoking is not only a risk factor for AF but also for conditions that can predispose to heart failure and subsequent development of AF. Discontinuation of smoking may reduce the further risks of AF. More research is recommended to evaluate the impact of e-cigarettes and passive smoking on AF, as well as the impact of smoking cessation on reducing the risk of stroke, myocardial infarction, chronic kidney disease, and all-cause mortality.

Conclusion It is clear that hypertension, diabetes, and smoking predispose to the onset of AF, which is particularly important when considering that these risk factors are modifiable. Thus, strategies to promote BP and glycemic control as well as smoking cessation must shape public health strategy.

ARTICLE INFORMATION

CONFLICT OF INTEREST GYHL is a consultant for Bayer/Janssen, BMS/Pfizer, Medtronic, Boehringer Ingelheim, Novartis, Veeva, and Daiichi-Sankyo, as well as a speaker for Bayer, BMS/Pfizer, Medtronic, Boehringer Ingelheim, and Daiichi-Sankyo. No fees are directly received personally. Other authors have no conflict of interest to declare.


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