Prevalence, Incidence, Prognosis, and Predisposing Conditions for Atrial Fibrillation: Population-Based Estimates

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Atrial fibrillation (AF) is the most common of the serious cardiac rhythm disturbances and is responsible for substantial morbidity and mortality in the general population. Its prevalence doubles with each advancing decade of age, from 0.5% at age 50–59 years to almost 9% at age 80–89 years. It is also becoming more prevalent, increasing in men aged 65–84 years from 3.2% in 1968–1970 to 9.1% in 1987–1989. This statistically significant increase in men was not explained by an increase in age, valve disease, or myocardial infarctions in the cohort. The incidence of new onset of AF also doubled with each decade of age, independent of the increasing prevalence of known predisposing conditions. Based on 38-year follow-up data from the Framingham Study, men had a 1.5-fold greater risk of developing AF than women after adjustment for age and predisposing conditions. Of the cardiovascular risk factors, only hypertension and diabetes were significant independent predictors of AF, adjusting for age and other predisposing conditions. Cigarette smoking was a significant risk factor in women adjusting only for age (OR = 1.4), but was just short of significance on adjustment for other risk factors. Neither obesity nor alcohol intake was associated with AF incidence in either sex. For men and women, respectively, diabetes conferred a 1.4- and 1.6-fold risk, and hypertension a 1.5- and 1.4-fold risk, after adjusting for other associated conditions. Because of its high prevalence in the population, hypertension was responsible for more AF in the population (14%) than any other risk factor. Intrinsic overt cardiac conditions imposed a substantially higher risk. Adjusting for other relevant conditions, heart failure was associated with a 4.5- and 5.9-fold risk, and valvular heart disease a 1.8- and 3.4-fold risk for AF in men and women, respectively. Myocardial infarction significantly increased the risk factor-adjusted likelihood of AF by 40% in men only. Echocardiographic predictors of nonrheumatic AF include left atrial enlargement (39% increase in risk per 5-mm increment), left ventricular fractional shortening (34% per 5% decrement), and left ventricular wall thickness (28% per 4-mm increment). These echocardiographic features offer prognostic information for AF beyond the traditional clinical risk factors. Electrocardiographic left ventricular hypertrophy increased risk of AF 3–4-fold after adjusting only for age, but this risk ratio is decreased to 1.4 after adjustment for the other associated conditions. The chief hazard of AF is stroke, the risk of which is increased 4–5-fold. Because of its high prevalence in advanced age, AF assumes great importance as a risk factor for stroke and by the ninth decade becomes a dominant factor. The attributable risk for stroke associated with AF increases steeply from 1.5% at age 50–59 years to 23.5% at age 80–89 years. AF is associated with a doubling of mortality in both sexes, which is decreased to 1.5–1.9-fold after adjusting for associated cardiovascular conditions. Decreased survival associated with AF occurs across a wide range of ages.
Am J Cardiol 1998;82:2N–9N

Atrial fibrillation (AF) is one of the major cardiac rhythm disturbances that produces substantial excess cardiovascular morbidity and mortality.[1] Because it and its hazards are difficult to control once established, a preventive approach to the problem is required. To prevent AF it is necessary to know the conditions that predispose to its development, the magnitude of the problem in the population, and its prognosis. This article examines the incidence, prevalence, prognosis, and predisposing conditions for AF, based on almost 4 decades of Framingham Study data. It has been established that coronary artery disease, heart failure, valvular heart disease, and hypertension predispose to AF, but most prior studies did not adequately assess the independent contribution of these conditions using multivariate analysis.[1–5]

At each Framingham Study biennial examination, medical histories, physical examinations, and electrocardiograms were routinely obtained to ascertain whether cardiovascular disease was present. The presence of AF was determined from the electrocardiogram routinely obtained at each clinic examination, from hospital records, and from subjects' personal physicians. Potential cardiovascular risk factors were also assessed at each clinic examination, and the presence of myocardial infarction, stroke, heart failure, and valvular heart disease were determined by a panel of 3 physicians using established criteria.[6] Logistic regression based on pooled biennial person-examination data was used to evaluate the association of specified risk factors, echocardiographic features, and cardiac conditions with the subsequent occurrence of

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Framingham Study research is supported by NIH/NHLBI Contract N01-HC-38038 and the Visiting Scientist Program, which is supported by ASTRA USA, Hoechst Marion Roussel and Servier Canada, Inc.

Reprints are not available.

0002-9149/98/$19.00
PII S0002-9149(98)00583-9

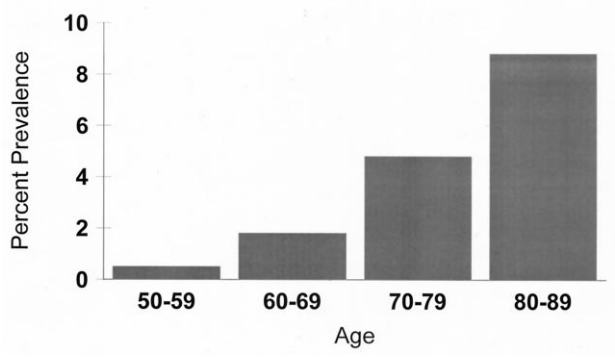

FIGURE 1. Prevalence of atrial fibrillation by decade of age. Framingham Study, subjects aged 50–89 years. (Adapted with permission from *Stroke*.[20])

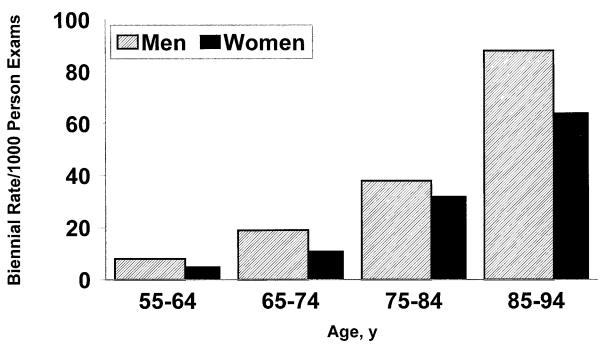

FIGURE 2. Incidence of atrial fibrillation, Framingham Heart Study, 38-year follow-up. (Adapted with permission from *JAMA*.[7])

AF. Over 38 years of follow-up, there were 562 cases of AF arising anew in 2,090 men and 2,641 women at risk of its initial occurrence.

PREVALENCE

AF is the most common serious sustained cardiac rhythm disturbance. It is estimated that 2.2 million US citizens have the condition.[7] The prevalence of AF doubles with each advancing decade of age >50 years and reaches almost 10% in octogenarians (Figure 1).[8] In the Framingham Study cohort, the age-adjusted prevalence of AF was found to be higher in men than in women at all ages on all biennial examinations.[9] The male to female age-adjusted ratio averaged 1.7. Persons with loud heart murmurs, a myocardial infarction, and heart failure had a substantially higher prevalence of AF.

National Hospital Discharge survey data have indicated an increase in AF prevalence between 1982 and 1993 in adults above and below age 65 years.[9] Based on additional systematically obtained general population-based data from the Framingham Study, the prevalence of AF in persons aged 65–84 years was examined over a span of 22 years. It was determined that the prevalence of AF increased significantly over that period in men, but not in women (Table I). Between 1968 and 1989, the prevalence in men tripled from 3.2% to 9.1%.[9] Multivariate analysis indicated that this increase in AF in men persisted when age, valve disease, and prior myocardial infarction were taken into account.[9] Thus, the basis for the increasing prevalence of AF is unexplained. One possibility is that it is due to the recently improved survival after myocardial infarction. In an aging US population, this increasing trend in the prevalence of AF is ominous because of the likelihood that it will be accompanied by an increased prevalence of stroke.

INCIDENCE

The incidence of AF also doubles with each successive age decade beyond 50 years, so that almost 10% of persons who reach age 80 years of age can expect to acquire this serious cardiac rhythm disturbance (Figure 2). The incidence in men is substantially greater than in women at all ages, but with a closing gap with advancing age. After adjusting for age and other risk factors predisposing persons to AF, men were 50% more likely than women to develop the rhythm disturbance. Adjusting for cardiovascular risk factors and other predisposing cardiac conditions, the risk of AF increased 2-fold for each advancing decade of age.[8] The incidence of both chronic and transient AF increased similarly with age, with a male predominance (Figure 3).

CLINICAL CHARACTERISTICS

Persons who develop AF are usually elderly, more likely than age-matched controls to have diabetes, left ventricular hypertrophy, echocardiographic abnormalities, coronary artery disease, valvular heart disease, heart failure, and to have already suffered a stroke.[8] About a third of women and 20% of men have valvular heart disease, 28% of men and half as many women have myocardial infarctions, and about 25%

TABLE I Secular Trends in Prevalence of Atrial Fibrillation: Framingham Subjects, Aged 65–84 Years (1968–1989)

	Age-Adjusted Percent Prevalence					
	1968–1970	1971–1973	1975–1977	1979–1981	1983–1985	1987–1989
Men	3.2	5.3	6.5	7.8	7.5	9.1*
Women	2.8	3.3	4.3	4.3	3.9	4.7†

*$p < 0.002$.
†$p = 0.60$.
Adapted from *Am Heart J*.[9]

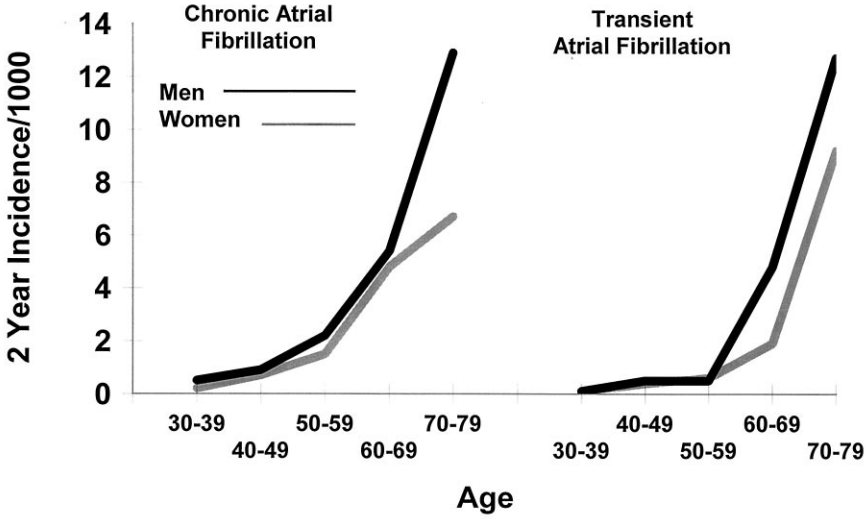

FIGURE 3. Incidence of atrial fibrillation (AF) by age and sex: transient versus chronic. (Adapted with permission from *Am Heart J.*[1])

of both sexes have heart failure. Patients with AF were not observed to be more obese or to imbibe more alcohol.

CARDIAC PRECURSORS

The recognized cardiac conditions associated with AF are various types of valvular heart disease, acute myocardial infarction, myocarditis, hypertrophic cardiomyopathy, congenital heart disease, pericarditis, hypertensive cardiovascular disease, and heart failure. In the Framingham Study, the most common cardiac precursors of AF were heart failure, myocardial infarction, and valvular heart disease.[8] These cardiac conditions accounted for 20% of the AF incidence in men and 31% of its occurrence in women.[8]

Of these cardiac conditions, heart failure imposed the greatest risk of AF, with a 4.5-fold increased risk in men and a 5.9-fold increased risk in women (Table II). Valve disease was associated with a 1.8-fold increase in men and a 3.4-fold increase in women. Taking other risk factors and cardiac conditions into account, myocardial infarction was significantly associated with AF only in men, increasing their risk by 40% (Table II).

RISK FACTORS

Noncardiac causes of AF that have been reported include thyrotoxicosis, alcohol abuse, severe infections, and pulmonary pathology. Adjusting only for age, cigarette smoking in women, and diabetes, hypertension and electrocardiographically demonstrated left-ventricular hypertrophy (ECG-LVH) in both sexes were significant AF predictors (Table III). Women who smoked were 40% more likely to develop AF; those who were diabetic had a 2-fold increased risk; those with hypertension had a 70% greater risk; and those with ECG-LVH had almost a 4-fold increased risk. In men, diabetes increased risk 70%, hypertension 80%, and ECG-LVH by 3-fold. After adjusting for other associated conditions, as well as age and sex, diabetes and hypertension remained significant predictors of AF, but with somewhat decreased odds ratios (Table III). In both age-adjusted and risk factor–adjusted analyses, neither obesity nor

TABLE II Risk of Developing Atrial Fibrillation Associated with Specified Cardiac Conditions: 38-Year Follow-Up of Framingham Study Subjects, Aged 55–94 Years

Cardiac Conditions	Age-Adjusted Odds Ratio		Risk Factor–Adjusted Odds Ratio	
	Men	Women	Men	Women
MI	2.2*	2.4*	1.4†	1.2
Heart failure	6.1‡	8.1‡	4.5‡	5.9‡
Valve disease	2.2‡	3.6‡	1.8*	3.4‡

MI = myocardial infarction.
*p <0.01.
†p <0.05.
‡p <0.001.
Adapted from *JAMA.*[7]

TABLE III Risk Factors for Development of Atrial Fibrillation: Framingham Study, 38-Year Follow-Up*

Risk Factors	Age-Adjusted OR		Risk Factor–Adjusted OR	
	Men	Women	Men	Women
Cigarettes	1.0	1.4†	1.1	1.4
Diabetes	1.7‡	2.1§	1.4†	1.6‡
ECG–LVH	3.0§	3.8§	1.4	1.3
Hypertension	1.8§	1.7§	1.5‡	1.4†
BMI	1.03	1.02	—	—
Alcohol	1.01	0.95	—	—

BMI = body mass index; ECG–LVH = echocardiographic left ventricular hypertrophy; OR = odds ratio.
*2-year pooled logistic regression; †p <0.05; ‡p <0.01; §p <0.001.
Adapted from *JAMA.*[7]

alcohol intake was a substantial or significant risk factor for AF.

ECHOCARDIOGRAPHIC PREDICTORS

Although structural heart disease is often present when AF appears, the echocardiographic precursors of the condition were not reported before its investigation in the Framingham Study.[10] The echocardiographic findings in persons who developed AF were evaluated, and the risk of future AF in those who had the abnormalities was investigated in 1,924 subjects 50–94 years of age. Persons with AF had larger left atrial, left ventricular end-diastolic and end-systolic dimensions; greater ventricular septal and left ventricular posterior wall thickness; more left ventricular mass/height and lower percent fractional shortening; and a higher prevalence of mitral annular calcification than persons without AF (Table IV). Using Cox proportional hazards modeling, the association of these echocardiographic features with subsequent development of AF was quantified after adjustment for age, sex, hypertension, coronary artery disease, heart failure, diabetes, and valve disease. Left atrial size, left ventricular fractional shortening (inverse), and the sum of left ventricular and posterior wall thickness were demonstrated to be independent echocardiographic predictors of AF. For each of these echocardiographic predictors, AF risk increased in a continuous graded fashion (Figure 4). Those with ≥2 of the highest risk-quartile measurements for these features in combination had a 17% risk of AF compared with 3.7% when none was present. Evidently these echocardiographic features can be used to provide predictive information beyond that provided by the cardiovascular risk factors and clinical conditions known to predispose to AF.

TABLE IV Echocardiographic Characteristics of a Framingham Study Sample (Subjects Aged 59–90 Years) According to Incident Atrial Fibrillation Status

	Incident Atrial Fibrillation	
	Present	Absent
Left atrial dimension (mm)	42.1	39.5
Left ventricular end-diastolic dimension (mm)	49.8	48.3
Left ventricular end-systolic dimension (mm)	30.5	28.8
Ventricular septal wall thickness (mm)	10.2	9.0
Left ventricular posterior thickness (mm)	9.8	8.9
Left ventricular mass/height (g/m)	133.0	107.1
Fractional shortening (%)	37.1	38.6
Mitral annual calcification (%)	24.7	11.9

Adapted from *Circulation*.[10]

TABLE V Relative Risk of a Stroke in Persons with Atrial Fibrillation and Other Cardiovascular Conditions According to Age: Framingham Study

	Risk Ratios*			
	50–59 yrs	60–69 yrs	70–79 yrs	80–89 yrs
Hypertension	3.5	3.2	2.5	1.7
Coronary disease	2.9	2.0	1.7	0.7
Cardiac failure	3.9	2.4	2.2	1.7
Atrial fibrillation	4.0	2.6	3.3	4.5

*$p < 0.001$ or < 0.01, adjusted for other stroke risk factors.
Adapted from *Stroke*.[20]

CARDIOVASCULAR SEQUELAE

AF has been shown to be associated with increased risk for cardiovascular morbidity or mortality.[11–14] Epidemiologic and clinical studies have generally indicated that AF constitutes a major independent risk factor for stroke, with a 3–5-fold increased risk after adjusting for other risk factors (Table V).[15–20] The impact was substantial at all ages. With increasing age, the effects of hypertension, coronary artery disease, and heart failure on stroke incidence decreased, whereas the influence of AF did not diminish. In contrast to other cardiovascular contributors to the occurrence of strokes, the percentage of strokes attributable to AF increased with age (Tables V and VI). For persons ages 80–89 years, AF was responsible for

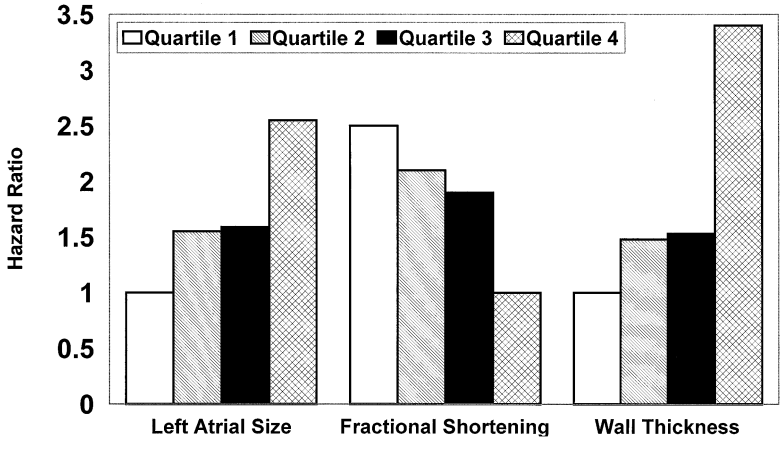

FIGURE 4. Hazard ratios according to quartile of echocardiographic predictors. (Adapted with permission from *Circulation*.[10])

TABLE VI Attributable Risk of Stroke for Atrial Fibrillation by Age: Framingham Study

	Age Group (yrs)			
	50–59	60–69	70–79	80–89
Attributable (%)	1.5	2.8	9.9	23.5
Events occurring with the condition (%)	6.5	8.5	18.8	30.7

Adapted from *Stroke*.[20]

23.5% of strokes that occurred in the Framingham Study.[20] In that age group, almost 31% of strokes were accompanied by AF (Table VI).

Cardiac conditions such as coronary artery disease and heart failure often coexist with AF and may exist at a subclinical level, making it possible that these underlying conditions are responsible for the excess occurrence of strokes rather than the AF itself. However, in the Framingham Study, men with overt coronary artery disease who also had AF more than doubled their high risk of stroke, and in women there was nearly a 5-fold increased hazard (Figure 5). Likewise, in those who had overt heart failure, AF imposed a further 2-fold increase in risk of a stroke.

New onset of AF has been reported to be associated with an imminent hazard of a stroke,[19,21,22] suggesting that recent-onset AF is responsible for many ischemic stroke events.[22,23] However, it has also been suggested that acute stroke might precipitate transient AF.[24] In the Framingham Study, 115 of 656 initial strokes occurred in association with AF. Of these, 26 had their AF discovered for the first time on admission or shortly thereafter. Because 92% of persons presenting with newly discovered AF at the time of acute strokes continued to have this rhythm, it seems likely that AF was the precipitant, rather than the consequence, of the strokes.[25]

Investigations of the time course of embolic complications in AF indicate that the risk of stroke in persons with AF appears highest during the early months after the initial diagnosis of the rhythm disturbance.[22,25] In addition to the substantial risk of thromboembolism associated with AF, those who have had ≤1 embolic event are at high risk of further emboli.[26] The Framingham Study found that recurrence after an initial stroke occurred early and sooner in persons with AF.[22] Also, ischemic strokes associated with AF were nearly twice as likely to be fatal as strokes unassociated with AF. Stroke recurrences were also more frequent, and functional deficits were more likely to be severe among survivors.[27]

Recent studies suggest that an increased risk of thromboembolism exists in persons with AF who have left atrial enlargement or decreased left ventricular systolic function.[28,29] Thus, these persons who are at increased risk of developing AF also appear to be at increased risk of thromboembolic events.

AF is also associated with an increased risk of coronary death and it may precipitate the onset of heart failure. Having both coronary artery disease and AF was found in the Framingham Study to adversely influence the prognosis regarding total mortality and stroke.[1]

MORTALITY

AF has a significant impact on longevity, approximately doubling all-cause and cardiovascular mortality rates.[13,14] Although the cardiovascular morbidity associated with AF is well documented, it has not been clearly established whether or not AF itself results in excess mortality. The truncated survival observed with AF could reflect the increased mortality of the cardiovascular conditions with which it is often associated. However, population-based data from the Framingham Study suggest that AF confers an excess risk of mortality from cardiovascular and all causes, even taking into account the influence of the associated cardiovascular conditions. The risk imposed by subsets of persons with AF remains controversial. Some investigators report that AF in patients with a myocardial infarction imposes excess mortality, whereas others have found no relationship.[30–33] Likewise, it is also uncertain whether AF independently

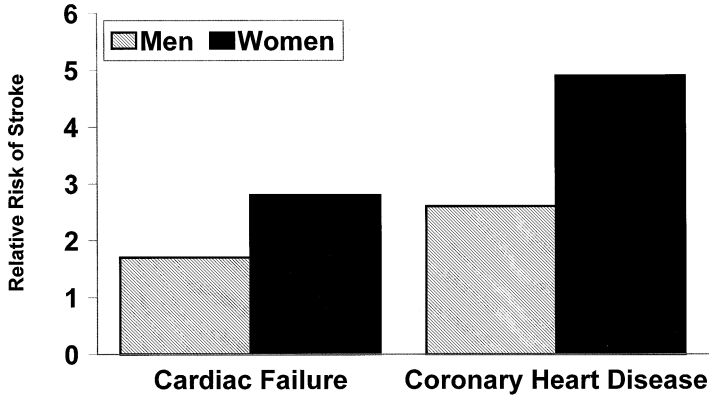

FIGURE 5. Age-adjusted relative risk of stroke for men and women with atrial fibrillation in the presence of cardiac failure and coronary artery disease. (Adapted with permission from *Stroke*.[20])

influences mortality in heart failure or stroke, with some finding (and others refuting) an independent contribution.[27, 34–38] More extensive data from insurance applicants found that applicants had increased mortality if they had AF in the setting of mitral stenosis or coronary artery disease.[14] A number of cohort studies have examined the issue in male air force recruits, male civil servants, and population-based samples.[4,5,13,39–42] One study found an insignificant 1.9-fold excess mortality, but most found that AF conferred an excess risk of death ranging from a 1.3-fold adjusted relative risk to a 2.6-fold unadjusted relative risk. However, the limitation of most studies has been their lack of time-dependent multivariate analysis.

PREVENTIVE IMPLICATIONS

In addition to intrinsic cardiac causes such as heart failure, coronary disease, and valve disease, risk factors for cardiovascular disease such as hypertension and impaired glucose tolerance also significantly predispose to AF. Hence, decreasing the risk of cardiovascular events that induce AF will also have the additional benefit of directly decreasing the incidence of AF.

The treatment of AF with anticoagulants or antiarrhythmic medications can diminish the symptoms and thromboembolic events with which it is associated.[43–46] However, these remedies are frequently contraindicated in the elderly and their use is often associated with significant morbidity. Identification of high-risk subgroups of AF patients by means of clinical features and echocardiography may permit drug treatment to be given to those at highest risk of a stroke. Ideally, the safest and most effective approach for the prevention of the thromboembolic sequelae of AF is to prevent its occurrence by modification of the cardiovascular disease risk factors and prevention of structural heart disease.

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EPIDEMIOLOGY AND INCIDENCE OF ATRIAL FIBRILLATION: DISCUSSION LED BY WILLIAM KANNEL, MD

Edward Pritchett, MD (Durham, NC): I want to ask a question about the offspring of the people being followed in the cohort. Are you following them, are they starting to have atrial fibrillation (AF), and is there a familial component to AF as there is a familial component to Alzheimer's disease? Alzheimer's disease is a disease of aging, as is AF. Can we learn anything from your cohort?

William B. Kannel, MD (Framingham, MA): Yes, we are following the cohort. We now have 16 years of follow-up on them. As you know, they entered the study at the same age as their parents were 40 years previously, and we're beginning to track the AF prevalence and other risk factors to see if there's familial aggregation. We're also getting some DNA so we can get more finite indications of genetic susceptibility. Thus far, we can't answer your question as to whether having a family member with AF predisposes you to the condition. We have a couple of identical twins, and interestingly enough, 2 of them developed AF, which was immediately followed by AF in the other twin, but that's just anecdotal based on 1 set of identical twins.

James A. Reiffel, MD (New York, NY): I've got 2 questions. One is, does anyone have any thoughts as to why the incidence is higher in men than in women? And the other is, your group and others have reported that there is a mortality risk from AF, even when adjusted for other factors. The question is, what's the mechanism? Is it inadequate rate control and tachycardiac-induced heart failure? Is it the stroke risk? Is it pulmonary emboli? Is it depression from the AF? Do we have any data from your population as to what the mechanisms may be?

Dr. Kannel: Regarding the male dominance, we can only guess. We suspect it may be due to the higher incidence of coronary disease in the men. They're surviving, and surviving longer, allowing them to develop AF and heart failure. Perhaps that's the reason for the excess incidence in men.

Your second question related to the excess mortality aside from cardiovascular disease. We haven't yet sorted that out. It is possible that having AF concomitant with another condition makes you more susceptible to an early death. Even if you have cancer plus AF induced by failure, you'd probably die earlier than if you didn't have it, or even pulmonary emphysema or chronic obstructive pulmonary disease. So it may be that kind of an issue, of hastening mortality from another condition because AF adds to the problem.

Brian Olshansky, MD (Maywood, IL): I have a question about the risk of developing AF in patients with congestive heart failure. What is the relationship in time? Is it possible that AF is actually causing heart failure instead of the other way around?

Dr. Kannel: Well, it's always possible that the patient may have had subclinical left ventricular dysfunction that we are unable to detect, but insofar as a clinically overt disease, the clinically overt disease followed the presence of AF by a minimum of 2 years or longer. But you're quite right. It's only in the last 10 years that we've been doing routine echocardiograms on the whole cohort and are able now to pick up subclinical left ventricular dysfunction. We also find that risk factors—multivariant risk factors—that predict heart failure also predict AF, and some of that is due to the existing risk factors that accompany both conditions, but we seem to be finding an excess of AF in heart failure patients, adjusting for the known risk factors and other conditions. The mechanism? I'll leave that to you fellows to sort out.

Berndt Lüderitz, MD (Bonn, Germany): I have a question on life expectancy. Would you consider this parameter for your data? I mean, the life expectancy increased tremendously within the last 10 years in Europe. For instance, it's 79.5 for women and 73 for men, and my question is, must we expect more AF patients in our office for these reasons, since AF increases with age, of course; and second, how is life expectancy influenced by AF, based on your data?

Dr. Kannel: Well, there are 2 issues. One is the secular trend, and there is an increasing prevalence over time. Some of that's undoubtedly due to the aging of the cohort, but the data I showed you were age-adjusted and in a specific age group, so we can't attribute the increasing prevalence to an aging population. Yes, it's clear that because of the aging of the population, we're going to see a higher prevalence of AF. We have not calculated survival curves, although we could do this, and undoubtedly AF will influence survival. Anything that increases the mortality rate 2-fold has to influence survival. But an aging population of increased size is a major concern as a public health issue for this condition, because, we're going to be seeing a lot more AF, either because of the coexisting conditions that produce it or due to the "age effect." But remember, age also reflects the exposure to the predisposing conditions, so it may be reflecting not age per se, but the length of time you're exposed to hypertension, diabetes, coronary disease, and other conditions.

Michael Reiter, MD (Denver, CO): If I understand your data correctly, you've combined paroxysmal and chronic AF. Do you have any data separating the 2

conditions? It would be interesting to know whether there are important differences in the predictors for chronic versus paroxysmal AF, and also, it would be very interesting to know whether the risk of stroke is different for paroxysmal and chronic AF.

Dr. Kannel: We looked at this some years ago and we could discern no markers for one that are distinct for the other. The risks for chronic AF for all of the outcomes were quite similar for transient and chronic AF. Another interesting question would be to know how often and how soon does paroxysmal AF merge into chronic AF. One of the difficulties in sorting out the effects of each is that as they merge from one into the other, they tend to come and go before eventually settling into the chronic, sustained variety. I agree, it would probably be worth a serious attempt to sort out any differences that may be present.

Rodney H. Falk, MD (Boston, MA): There's an astonishing amount of data that has come out of Framingham that's taught us a vast amount. One of the intriguing things is that, at least based on the Framingham definition of lone AF, there is a significant increase in morbidity and mortality that hasn't been shown in some other studies, but your definition, or your older definition, of lone AF didn't take into account hypertension in the absence of cardiomegaly or left ventricular hypertrophy on electrocardiography. Now that you've incorporated echocardiography into your cohort, are you changing your definition of lone AF, and are there any data on that?

Dr. Kannel: I've always found it very difficult to define that entity—lone AF—particularly if you look at the relation of hypertension, not only to AF but to every other cardiac condition. What you really mean when you say lone AF is AF in the absence of major cardiac conditions. Most patients who are labeled lone AF have a little hypertension and have a little of all the other predisposing conditions, so it's very difficult to sort this out. The difference between our data and the data you quote is that our population was considerably older than the Mayo Clinic population, and the average age of the lone AF patients was much greater than theirs, and that may explain some of the differences in the findings. I think the best we can do is examine what is the outlook for AF that occurs in the absence of overt major cardiovascular conditions, and in our data, lone AF seems to carry a substantial excess risk.

John Camm, MD (London, England): I've seen some data from the United Kingdom where AF is much less prevalent in Afro-Caribbean and Asian communities, but where it is present, it's related much more to hypertension in the Afro-Caribbean, much more to ischemic heart disease in the Asian, and it's a much more balanced relationship in the white population. Those data relate to the city of Birmingham.

Dr. Kannel: I think hypertension is an underrated predisposing condition. We estimate that about 14% of all the AF in the population can be directly attributable to hypertension as commonly defined. The Framingham population is almost exclusively white, so evaluation of racial differences would not be possible.