Stroke and atrial fibrillation: is stroke prevention treatment appropriate beforehand?

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Abstract

Objective—To undertake a pilot study before conducting a large European multicentre prospective study, to determine the proportion of patients with atrial fibrillation who were not receiving antithrombotic treatment before stroke onset, and their characteristics.

Design and patients—The stroke in atrial fibrillation ensemble (SAFE) I study was an observational study conducted in 213 patients with atrial fibrillation consecutively admitted in 1997 to three European centres for an acute stroke or transient ischaemic attack (TIA). It was determined whether they were receiving prior antithrombotic treatment.

Results—Atrial fibrillation was known before stroke in 148 patients (69.5%). Of 213 patients, 34 (16.0%) were receiving anticoagulation treatment before stroke, but only six had an international normalised ratio between 2.0 and 3.5; 65 (30.5%) were receiving antiplatelet treatment; and three (1.4%) were receiving both anticoagulation and antiplatelet treatment. Of 137 patients eligible for oral anticoagulation, 108 (78.8%) did not receive treatment. Of 142 patients eligible for any antithrombotic treatment, 62 (43.7%) were not treated. The logistic regression analysis, assuming anticoagulation treatment as a dependent variable, found digoxin treatment, absence of arterial hypertension, mitral stenosis, and cardioversion as independent factors. Assuming any antithrombotic treatment as a dependent variable, previously known atrial fibrillation, lower age, being a non-smoker, and absence of arterial hypertension were found to be independent factors.

Conclusion—More than half of the patients with atrial fibrillation admitted for acute stroke or TIA were not receiving any antithrombotic treatment beforehand. New onset atrial fibrillation and contraindications account for a minority of non-prescriptions; thus, other reasons should be identified to improve stroke prevention in the community.

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Keywords: stroke; atrial fibrillation; anticoagulant treatment; antiplatelet treatment

nearly 25% of strokes occurring in people over 80 years old.² Moreover, NVAF leads to more severe strokes than other causes,³ with a combined rate of death or significant neurological disability of 71% one year after stroke onset.⁴ The risk of stroke in NVAF is particularly increased in subjects with arterial hypertension, recent congestive heart failure, prior transient ischaemic attacks (TIAs) or stroke, or echocardiographic evidence of left ventricular dysfunction, mitral annular calcification, or left atrial enlargement.^{5–6} Prevention is critical to reduce death and disability in patients with NVAF, because the annual rate of recurrent stroke of any severity is 12% in the absence of treatment.⁷

In both primary and secondary stroke prevention in NVAF patients, it has been shown that the risk of stroke was significantly reduced by warfarin and to a lesser extent by aspirin.^{8–9} The results of these trials were so impressive that most of them were stopped prematurely.¹⁰ However, prescription of such a treatment should take into account high risk patients as defined in the third stroke prevention in atrial fibrillation (SPAF III) study.^{8–11}

Despite these results, day to day practice seems to differ. Indeed, physician surveys suggest that not all appropriate patients with NVAF receive anticoagulation treatment.^{12–22} In a community study conducted in 1994 in the USA, 62% of patients with known NVAF and no contraindications to anticoagulation did not receive anticoagulation, two thirds of them did not receive aspirin, and the degree of anticoagulation treatment was appropriate in only 60% of treated patients.¹⁵ In an Australian centre, of 103 patients with chronic NVAF who were consecutively admitted for various reasons, including 12 strokes and two TIAs, two thirds would have been ideal candidates, but less than 10% were receiving anticoagulation treatment.¹⁸

It is not possible to draw general conclusions from these studies because the health systems differ from country to country, the patterns of anticoagulation differ,²³ most of these studies were conducted before the results of important trials, and previous information campaigns may increase the proportion of patients treated with anticoagulants or aspirin for atrial fibrillation.²⁴

Moreover, little is known about the current use of anticoagulation treatment in patients with atrial fibrillation in continental Europe. Determining the proportion of stroke patients with atrial fibrillation who did not receive appropriate treatment, and the reasons, is

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Non-valvar atrial fibrillation (NVAF) increases the risk of stroke fivefold^{1,2} and accounts for

Table 1 Description of the study population

Number of patients	213	Past (but interrupted) treatments (n (%))	
Perugia	38 (17.8%)	Oral anticoagulation	12 (5.6)
Paris	32 (15.0%)	Mean delay since interruption	1.00 year (0–7)
Lille	143 (67.1%)	Reasons: anaemia (1), surgery (1), frequent falls (1), poor observance (2), reduction of atrial fibrillation (2)	
Demography		Aspirin treatment	1 (0.5)
Mean age at stroke onset	77.63 years (range 34–96)	Cardioversion	8 (3.8)
Male sex	98 (46%)	Electrical	2 (0.9)
Atrial fibrillation (n (%))		Pharmacological	8 (3.8)
Previously known	147 (69.5)	Both	1 (0.5)
With mitral stenosis	8 (3.8)	Current treatments just before stroke (n (%))	
With thyrotoxicosis	3 (1.4)	Oral anticoagulation	34 (16.0)
Thrombus on echography	3 (1.4)	INR between 2–3.5	6 (2.8)
Index stroke (n (%))		Aspirin treatment	62 (29.1)
Ischaemic stroke	151 (70.9)	Other antiplatelet drugs	6 (2.8)
Haemorrhagic stroke	18 (8.5)	Any antithrombotic*	99 (40.7)
Haemorrhagic infarct	17 (8.0)	Digoxin	84 (39.4)
TIA	27 (12.7)	β blockers	38 (17.8)
Medical history (n (%))		Calcium channel blockers	53 (24.9)
Previous stroke or TIA	51 (23.9)	ACE inhibitors	56 (26.3)
Previous cerebral haemorrhage	1 (0.5)	Diuretics	80 (37.6)
Previous systemic emboli	5 (2.4)	Nitrates	49 (23.0)
Non-life threatening bleedings	2 (0.9)	Insulin	7 (3.3)
Life threatening bleedings	0 (0.0)	Antiarrhythmics	53 (24.9)
Depression	14 (6.6)	Oral hypoglycaemic drugs	14 (6.6)
Dementia	12 (5.6)	Hormonal treatment (in women)	2 (1.7)
Angina pectoris	58 (27.2)	Antidepressant	10 (4.7)
Congestive heart failure	32 (15.0)	Other cerebroactive drugs	10 (4.7)
Active gastroduodenal ulcer	6 (2.8)	Treatment at discharge (n (%))†	
Peripheral artery disease	16 (7.5)	Oral anticoagulation	74 (41.6)
Alcoholism	11 (5.2)	Aspirin	73 (41.0)
Headache	3 (1.4)	Other antiplatelet drugs	7 (3.9)
Allergy to aspirin	0 (0.0)	Any antithrombotic	154 (86.5)
Risk factors for strokes and other potential causes (n (%))		Digoxin	66 (37.1)
Arterial hypertension	152 (71.4)	β blockers	27 (15.2)
Diabetes mellitus	30 (14.1)	Calcium channel blockers	37 (20.8)
Hyperlipidaemia	54 (25.4)	ACE inhibitors	44 (24.7)
Abnormal haematocrit	30 (14.1)	Diuretics	55 (30.9)
Smoking	24 (11.3)	Nitrates	39 (21.9)
Other high risk cardiopathies	14 (6.6)	Insulin	12 (6.7)
Stenosis of the ICA $\geq$ 50%	28 (13.2)	Antiarrhythmics	46 (25.8)
Familial history of stroke	25 (11.7)	Oral hypoglycaemic drugs	11 (6.2)
CT brain scan (n (%))		Hormonal treatment	2 (1.1)
Multiple infarcts	47 (22.1)	Antidepressants	9 (5.1)
White matter changes	96 (45.1)	Other cerebroactive drugs	21 (11.8)
		In-hospital death (n (%))	35 (16.4)

The total may differ from 100% because of rounding.

*Three patients received anticoagulation and antithrombotic drugs simultaneously.

†Percentages were calculated in survivors at discharge.

ACE, angiotensin converting enzyme; ICA, internal carotid artery; INR, international normalised ratio; TIA, transient ischaemic attack.

necessary to focus future strategies to improve stroke prevention in the community. To address this question, a large prospective multicentre and multinational study is required. Before starting such a study, a pilot study is necessary.

The objective of the stroke in atrial fibrillation ensemble (SAFE) I study was to determine the proportion of patients with atrial fibrillation admitted for acute stroke who were not receiving antithrombotic treatment before stroke and to compare their characteristics with those of patients who did. The reasons why physicians did not prescribe any antithrombotic drug were not evaluated in this study.

Patients and methods

PATIENTS

SAFE I was an observational study conducted in 213 consecutive patients admitted to three European university centres (Perugia, Italy; Paris Sainte-Anne Hospital, France; Lille R Salengro Hospital, France), between 1 January and 31 December 1997. All patients, of any age and sex, admitted to the centres during the study period were eligible to enter the study,

provided they were admitted for an acute stroke (ischaemic or haemorrhagic) or TIA, and had atrial fibrillation either on the admission ECG or on a previous occasion. The characteristics of the study population are given in table 1.

METHODS

At admission all patients underwent non-contrast computed tomography (CT), routine biological tests, 12 lead ECG, and chest radiography. The international normalised ratio (INR) values were determined at admission. Patients with ischaemic stroke or TIA underwent cervical Doppler ultrasonography. All patients with an ischaemic stroke or TIA, and most patients with haemorrhagic stroke, underwent a transthoracic echocardiography. Magnetic resonance angiography, magnetic resonance imaging, conventional angiography, and transoesophageal echocardiography, were performed in selected patients. Ischaemic stroke was defined as clinical signs of focal disturbance of cerebral function lasting longer than 24 hours or leading to death, with no apparent cause other than of vascular origin,

and no sign of relevant primary intracerebral haemorrhage on CT scan or at necropsy. Haemorrhagic stroke was defined as clinical signs of focal disturbance of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than vascular, and evidence of a relevant primary intracerebral haemorrhage on CT scan or at necropsy. Patients with haemorrhagic changes within the infarct were classified in the haemorrhagic infarct group. TIAs were defined as episodes of focal cerebral dysfunction, presumably ischaemic in origin, lasting less than 24 hours and followed by return to normality, without any relevant lesion on CT scan other than of ischaemic origin. Medical history was determined from all available records (letter from their general practitioner (GP) or telephone call) and sources (patient, family, or GP).

We collected the following data: age; sex; previous stroke or TIA; presence of arterial hypertension (defined as systolic blood pressure ≥ 160 mm Hg, or diastolic blood pressure ≥ 90 mm Hg, or treatment with antihypertensive drugs before stroke onset); diabetes mellitus (defined as fasting serum glucose concentration ≥ 120 mg/dl (6.7 mmol/l), or current use of antidiabetic drugs); hyperlipidaemia (defined as fasting serum triglycerides concentration ≥ 150 mg/dl (1.71 mmol/l), or fasting cholesterol serum concentration ≥ 230 mg/dl (6.0 mmol/l), or current hypolipidaemic treatment); history of peripheral artery disease with intermittent claudication; alcoholism (defined as a mean alcohol consumption ≥ 300 g/week); cigarette smoking (≥ 10 cigarettes/day or cessation less than five years earlier); high risk cardiopathies as defined according to the TOAST (trial of org 10172 in acute stroke treatment) criteria²⁵; and significant stenosis of the internal carotid arteries defined as $\geq 50\%$ narrowing of the lumen documented by Doppler ultrasonography, B mode echotomography, magnetic resonance, or conventional angiography. Treatments at entrance and at discharge were also recorded.

STATISTICAL ANALYSES

The first step of the statistical analysis determined the percentage of subjects who were receiving oral anticoagulation treatment, antiplatelet treatment, both, or neither. In patients receiving anticoagulation treatment, those whose INR value at admission was between 2.0–3.5 were considered as being

properly treated, those with an INR value < 2 were considered as undertreated, and those with an INR value > 3.5 were considered as overtreated.

The second step comprised a bivariate analysis comparing variables between patients who were receiving oral anticoagulation treatment before stroke and patients who were not. We used the χ^2 test with Yates correction or Fisher's exact test when appropriate and the odds ratio (OR) method with 95% confidence interval (CI) to compare qualitative factors between groups; the unpaired *t* test was used to compare quantitative variables. Variables were demographic details, length of time of atrial fibrillation, characteristics of the index stroke, medical history, risk factors for stroke, potential causes of stroke other than atrial fibrillation, CT findings, and past or current treatments. The same analysis was also performed between patients who were under any antithrombotic treatment (oral anticoagulants or antiplatelet agents) before stroke and patients who were not.

The third step comprised a logistic regression analysis assuming anticoagulation before stroke (quoted 1 when present and 0 when absent) as dependent variable, the independent variables included in the analysis being selected from the bivariate analysis with a 0.25 level as a screening criterion.²⁶ Another logistic regression analysis was performed assuming any antithrombotic treatment (oral anticoagulants, antiplatelet agents, or both) before stroke (quoted 1 when present and 0 when absent) as dependent variable. Collinearity between variables (defined as $r > 0.6$) was excluded.

Patients were regarded as eligible for oral anticoagulation treatment when they had known atrial fibrillation, no recent systemic or cerebral bleeding, no alcoholism, no history of falls, and no known cognitive dysfunction leading to difficulties to manage anticoagulant treatment. Patients were regarded as eligible for antiplatelet treatment when they had known atrial fibrillation, no past gastrointestinal bleeding, no recent gastrointestinal ulcers, and no allergy to antiplatelet drugs. Age was not regarded as a contraindication for anticoagulation treatment because there was no upper age limit in the inclusion criteria of most trials conducted in patients with NVAf. Data were analysed using the SPSS/Macintosh package.

Table 2 Comparison between patients with and without anticoagulation treatment at admission

	Oral anticoagulation		Odds ratio	95% confidence intervals	<i>p</i> Value
	Yes (<i>n</i> = 34)	No (<i>n</i> = 179)			
Age (years)*	74.9	78.1			0.0645
Atrial fibrillation previously known	30 (88.2)	118 (65.9)	3.88	1.30 to 11.55	0.0096
Mitral stenosis	4 (11.8)	4 (2.2)	5.83	1.38 to 24.72	0.0239
Ischaemic stroke	17 (50.0)	134 (74.9)	0.34	0.16 to 0.71	0.0034
Haemorrhagic stroke	12 (35.3)	6 (3.4)	15.73	5.34 to 46.29	0.0001
Previous stroke or TIA	8 (23.5)	43 (24.0)	0.97	0.41 to 2.31	0.9508
Arterial hypertension	20 (58.8)	132 (73.7)	0.51	0.24 to 1.09	0.0777
Previous cardioversion	4 (11.8)	4 (2.2)	5.83	1.38 to 24.72	0.0239
Digoxin treatment	21 (61.8)	63 (35.2)	2.97	1.39 to 6.36	0.0037

Differences were considered significant at $p < 0.05$. Other variables listed in table 1 did not differ between both groups.

*Mean values and *t* test. For other variables: values (%) and χ^2 test, with Yates' correction or Fisher's exact test when appropriate.

Table 3 Comparison between patients with and without antithrombotic treatment (oral anticoagulant or antiplatelet drugs) at admission

	Any antithrombotic		Odds ratio	95% confidence intervals	p Value
	Yes (n = 99)	No (n = 114)			
Age (years)*	76.6	78.71			0.0870
Atrial fibrillation previously known	84 (84.9)	64 (56.1)	4.38	2.25 to 8.50	0.0001
Ischaemic stroke	63 (63.6)	88 (77.2)	0.52	0.28 to 0.94	0.0298
Haemorrhagic stroke	14 (14.1)	4 (3.5)	4.53	1.43 to 14.32	0.0054
Previous stroke or TIA	28 (28.3)	23 (20.2)	1.56	0.83 to 2.94	0.1667
Arterial hypertension	63 (63.6)	89 (78.1)	0.49	0.27 to 0.90	0.0201
Smoking	5 (5.1)	19 (16.7)	0.27	0.10 to 0.74	0.0075
Past oral anticoagulation	10 (10.1)	2 (1.8)	6.29	1.34 to 29.62	0.0084
Digoxin treatment	47 (47.5)	37 (32.5)	1.88	1.08 to 3.29	0.0253
Antiarrhythmic treatment	31 (31.3)	22 (19.3)	1.91	1.01 to 3.59	0.0431

Differences were considered significant at $p < 0.05$. Other variables listed in table 1 did not differ between both groups.

*Mean values and t test. For other variables: values (%) and χ^2 test, with Yates' correction or Fisher's exact test when appropriate.

Results

Of 213 patients with atrial fibrillation who were admitted for stroke or TIA, 34 (16.0%) were receiving anticoagulation treatment before stroke, and 99 (46.5%) were receiving any antithrombotic drugs, including three patients who had both anticoagulation and antiplatelet treatment. One hundred and fourteen (53.5%) were not receiving any antithrombotic treatment before their stroke. Of 34 patients who were receiving anticoagulant treatment, only six (17.6%) were properly treated, while 15 (44.1%) were undertreated, and three (8.8%) were overtreated. In 10 patients, INR values were not determined because of obvious non-compliance, long delays between stroke onset and admission, and recent interruption of treatment for medical reasons (anaemia or surgery). On the other hand, at discharge, of 178 survivors, 154 (86.5%) received anticoagulant or antiplatelet treatment.

Atrial fibrillation was previously known in 148 (69.5%) patients and the mean delay between the onset of atrial fibrillation and stroke ranged from 0–22 years. Of these 148

patients, 118 (79.7%) were not receiving oral anticoagulation treatment and 64 (43.2%) did not receive any antithrombotic drug. Of 137 patients eligible for oral anticoagulation (as previously defined), 108 (78.8%) were not receiving such treatment; of 142 patients eligible for any antithrombotic treatment, 62 (43.7%) were not receiving oral anticoagulation or antiplatelet treatment.

In the bivariate analysis comparing variables between patients who were receiving oral anticoagulation treatment before stroke and patients who were not, those receiving such treatment before stroke were more likely to have haemorrhagic stroke, previously known atrial fibrillation, mitral stenosis, to have had cardioversion, and to be currently receiving digoxin treatment; they were also less likely to have ischaemic stroke, but they did not differ in regard to history of previous stroke or TIA and arterial hypertension (table 2). Patients receiving any antithrombotic treatment before stroke were more likely to have haemorrhagic stroke, previously known atrial fibrillation, to have been receiving (interrupted) anticoagulants, and to be currently receiving digoxin or any other antiarrhythmic treatment; they were less likely to be admitted for ischaemic stroke, to have arterial hypertension, and to be smokers, but they did not differ in regard to history of previous stroke or TIA (table 3).

The logistic regression analysis assuming anticoagulation treatment before stroke as dependent variable found digoxin treatment, absence of arterial hypertension, mitral stenosis, and history of cardioversion as independent variables (table 4). The logistic regression analysis assuming any antithrombotic treatment before stroke as dependent variable found known atrial fibrillation, lower age, being a non-smoker, and absence of arterial hypertension as independent variables (table 5).

Table 4 Logistic regression analysis with oral anticoagulation (1 if present, 0 if absent) as dependent variable

	β	SE	p
Digoxin	1.0366	0.4030	0.0101
Arterial hypertension	-0.8906	0.4156	0.0321
Mitral stenosis	1.7183	0.8069	0.0332
Cardioversion	1.5885	0.7922	0.0450

Candidate variables were chosen as variables associated with current anticoagulation treatment at admission in the bivariate analysis, with a value of $p < 0.25$, and consisting of clinical variables assessable before stroke onset: age (years), male sex, previously known atrial fibrillation, mitral stenosis, previous systemic emboli, depression, peripheral artery disease, arterial hypertension, hyperlipidaemia, other high risk cardiopathies, cardioversion, digoxin, β blockers, calcium channel blockers, and centre = Paris (all quoted 0 = no and 1 = yes). Colinearity between variables (defined as $r > 0.6$) was excluded. Overall prediction of the model 84.98%.

β , regression coefficient; SE, standard error of the mean; constant = -1.7602.

Table 5 Logistic regression analysis with any antithrombotic drug at admission (1 if present, 0 if absent) as dependent variable

	β	SE	p
Known atrial fibrillation	1.6735	0.3742	0.0000
Age	-0.0411	0.0172	0.0169
Smoking	-1.3355	0.5726	0.0197
Arterial hypertension	-0.6934	0.3446	0.0442

Candidate variables were chosen as variables associated with current anticoagulation at admission in the bivariate analysis, with a value of $p < 0.25$, and consisting of clinical variables assessable before stroke onset: age (years), previously known atrial fibrillation, previous stroke or TIA, previous systemic emboli, non-life threatening bleedings, arterial hypertension, hyperlipidaemia, smoking, past oral anticoagulation, cardioversion, digoxin, diuretics (all quoted 0 = no and 1 = yes). Colinearity between variables (defined as $r > 0.6$) was excluded. Overall prediction of the model: 70.42%.

Constant = 2.4254.

Discussion

Our study has shown that in consecutive patients with stroke and atrial fibrillation, only one sixth were receiving anticoagulant treatment before their stroke and less than half received either anticoagulant or antiplatelet treatment.

Our study was hospital based and does not provide any information about patients with atrial fibrillation who did not have a stroke

within the study period, and about patients with stroke leading to death before hospitalisation. Conversely we cannot exclude that some patients with TIA were not recognised, not admitted to a stroke unit, or admitted to cardiological wards. However, despite any recruitment bias, this study was conducted in patients we should focus on—that is, those who have atrial fibrillation and have experienced a cerebrovascular event. Their major demographic characteristics did not differ from those of stroke patients included in community based registries.²⁷

Our study was observational and did not try to determine why patients were not treated according to guidelines. The aim was to determine the proportion of patients who were not treated according to the guidelines, even if the reasons were good—for example, having a non-recognised atrial fibrillation. This information is important because if a reason for undertreatment is underrecognition of atrial fibrillation, information campaigns directed at recognising atrial fibrillation in the community may be necessary. This is why we also included patients whose atrial fibrillation was not known before stroke onset, and who were not candidates for antithrombotic treatment.

We cannot exclude the possibility that patients who received neither anticoagulation nor antiplatelet treatment may have had contraindications. However, this seems unlikely for two reasons: the rate of non-prescription of antithrombotic treatment seems to be too high to be explained just by contraindications; and at discharge 154 patients were receiving anticoagulants or antiplatelet treatment, leading to an 86.5% rate of prescription in survivors. The 13.5% rate of non-prescription, which included transient or permanent contraindications such as very large infarcts, haemorrhagic infarcts, or primary intracerebral haemorrhages, is probably much higher than it should have been theoretically before stroke.

Even in patients who received oral anticoagulation treatment, the INR values were not always within the target range. Because of the study design, we do not know the interval between the last dose and the time of blood sampling for INR, but in some patients the delay between stroke onset and admission may have skewed the results towards an overrepresentation of patients with low INR values.

Four variables were associated with prescription of oral anticoagulation: digoxin treatment; absence of arterial hypertension; presence of mitral stenosis; and history of cardioversion. The relation with the lack of arterial hypertension may reflect a reluctance on the part of practitioners to give oral anticoagulation treatment to hypertensive patients, or at least to patients with non-stabilised arterial hypertension. This is part of many recommendations,¹³ but the best approach is probably to lower blood pressure after the acute stage, and provide oral anticoagulation treatment for patients with atrial fibrillation, because arterial hypertension is associated with an increased risk of ischaemic stroke in these patients.¹⁰ Although there is, to our knowledge, no

randomised trial proving that oral anticoagulation treatment is safe and effective in reducing the risk of stroke in patients with atrial fibrillation and mitral stenosis, it has been recommended in these patients for many years in light of the findings of the Framingham study which showed the high risk of stroke in such patients.²⁸ Therefore, it is not surprising that mitral stenosis is associated with a higher rate of oral anticoagulation treatment in patients with atrial fibrillation.

Another explanation for the higher rate of anticoagulation treatment in patients with mitral stenosis may be that these patients are more likely to be followed by a cardiologist, as well as patients with digoxin treatment or cardioversion. Cardiologists may be less reluctant to use oral anticoagulation treatment than GPs, but this hypothesis has to be evaluated. The type of atrial fibrillation, either paroxysmal, persistent or permanent, was not taken into account in our study as far as most trials.⁷ It is not possible to determine if the type of atrial fibrillation influences the prescription of oral anticoagulant treatment, but the pooled data¹⁰ suggest that patients with paroxysmal NVAF have the same risk of stroke as patients with chronic NVAF. History of stroke or TIA was surprisingly not associated with a higher rate of anticoagulation treatment, despite the results of the European atrial fibrillation trial.⁷ As reported for myocardial infarction,²⁹ it seems that such an event does not modify therapeutic attitudes concerning vascular risk factors.

Recommendations for the use of oral anticoagulation treatment in patients with NVAF state no upper age limit.³⁰ In clinical studies, the mean age of patients was lower than 70 years, and only 20% were over 75 years. Many physicians may remain uncertain about the safety of oral anticoagulation treatment in the elderly and argue that drug trials were conducted in younger patients. However, the SPAF III study, by determining groups of high risk patients, has clearly shown the benefit of anticoagulation treatment in patients older than 75 years, especially women.¹¹ Despite this result, a recent study has shown that anticoagulants are underused in elderly patients with atrial fibrillation, particularly in women older than 75 years.³¹ The results of the stroke prevention in reversible ischaemia trial (SPIRIT)³² may probably lead to an increased reluctance on the part of physicians to use anticoagulants in the elderly. However, it should be borne in mind that SPIRIT was conducted in patients with non-cardioembolic strokes and that the level of anticoagulation treatment was much higher.

Four variables were associated with prescription of any antithrombotic treatment: known atrial fibrillation; lower age; being a non-smoker; and absence of arterial hypertension. The relation with lower age and the lack of arterial hypertension may also reflect a reluctance on the part of practitioners to give hypertensive or elderly patients antithrombotic treatments. The prescription of antithrombotic treatment was influenced by the knowledge of atrial fibrillation, although prescription of oral

anticoagulation was not. However, 64 of 114 patients (56.1%) without any antithrombotic treatment had a known atrial fibrillation. The statistical relation between being a non-smoker and receiving any antithrombotic treatment remains unexplained.

Conclusion

The SAFE I study has shown that more than half of those patients admitted for acute stroke or TIA who had atrial fibrillation were not receiving any antithrombotic treatment before their stroke. Advanced age and arterial hypertension were associated with a lower rate of prescription. The reasons for such undertreatment remain to be determined.

To address this question, a large prospective multicentre and multinational study, including referral and non-referral, university and non-university hospitals, is required. This will be the aim of SAFE II. SAFE II may include other important social or medical variables which should be taken into account when prescribing anticoagulation treatment, such as the patient's level of education, being examined by a cardiologist over the previous 12 months, the number of medical contacts over the previous 12 months, having cognitive decline, living alone, or living in a rural, suburban or urban area.

One of the most likely reasons for undertreatment may be the failure of the conclusions from clinical trials to translate into change in clinical practice. If this is true, information campaigns will be required. In terms of public health, they may be as useful as new trials. In this context, GPs and cardiologists have a crucial role in detecting atrial fibrillation and instituting adequate treatment, because they are more likely to see these patients before they have a stroke.

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IMAGES IN CARDIOLOGY

The Vineberg procedure

This 66 year old white man had had an inferior myocardial infarction and coronary artery bypass surgery (CABG) in 1980. He underwent five vessel CABG in 1980 for refractory angina with severe three vessel coronary artery disease. He received several saphenous vein grafts. The left internal mammary artery (LIMA) was sutured directly into the posterior left ventricular wall using the Vineberg technique.

He had been angina free since surgery in 1980; however, a decrease in exercise tolerance prompted exercise echocardiographic stress testing in 1999. The resting echocardiogram showed mild inferior hypokinesis. He exercised for 6 minutes 47 seconds on the standard Bruce protocol, stopping for fatigue. There were no ECG changes suggestive of ischaemia. Echocardiography following exercise showed inferior dyskinesis and anteroseptal hypokinesis with normal function of the posterolateral wall.

Coronary angiography demonstrated a 60% distal left main stenosis and occlusions of the proximal left anterior descending (LAD), left circumflex, and right coronary arteries. The saphenous vein graft to the obtuse marginal

branch of the circumflex was occluded. The saphenous vein grafts to the LAD, diagonal, and posterior descending branch of the right coronary artery (PDA) were patent. There was high grade disease of the saphenous vein graft to the PDA. The LIMA to the myocardium was patent (A). A magnified view during LIMA angiography (B) showed collateral filling of the obtuse marginals (curved arrow) and PDA (straight arrow).

Promulgated by the Canadian surgeon Arthur Vineberg, implantation of the LIMA directly into the myocardial wall was performed in the 1960s. With the introduction of LIMA to coronary artery anastomosis toward the end of the decade, the Vineberg procedure was largely abandoned. This case demonstrates that 19 years following the Vineberg procedure, this patient still had a patent LIMA graft providing brisk collateral filling of the obtuse marginal and PDA. This graft is performing well, providing adequate blood flow to the posterolateral wall. The patient is currently feeling well continuing his antianginal medical regimen.

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