Stable Stroke Incidence Rates but Improved Case-Fatality in Dijon, France, From 1985 to 2004

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Background and Purpose—With the progress in stroke prevention, it is important to evaluate the epidemiological trends of strokes over a long period and from a nonselected population-based perspective.

Methods—We estimated changes in incidence, case-fatality rates, severity, risk factors and prestroke use of preventive treatments for first-ever strokes, from a continuous 20-year well-defined population-based registry, from 1985 to 2004.

Results—We recorded 3142 ischemic strokes, 341 primary cerebral hemorrhages and 74 subarachnoid hemorrhages. During the 20-year study, the age at first stroke onset increased by 5 years in men and 8 years in women. Comparing the 1985 to 1989 and the 2000 to 2004 periods, age- and sex-standardized incidences of first-ever strokes were stable except for lacunar strokes whose incidence significantly increased (P=0.01) and for cardioembolic stroke whose incidence significantly decreased (P=0.01). Twenty-eight-day case-fatality rates decreased significantly mainly for lacunar strokes (P=0.05) and for primary cerebral hemorrhages (P=0.03). The proportion of hypercholesterolemia and diabetes significantly increased (P<0.01). In contrast, the proportion of myocardial infarction significantly decreased (P=0.02). Prestroke antiplatelets and anticoagulants treatment significantly increased (P<0.01).

Conclusions—The age- and sex-standardized incidences of first strokes in Dijon have been stable over the past 20 years and were associated with an increase in age at stroke onset, a decrease in case-fatality rates, and an increased use of antiplatelet treatments. (*Stroke*. 2006;37:1674-1679.)

Key Words: cerebrovascular accident ■ epidemiology ■ incidence ■ risk factors ■ stroke

S troke medicine is one major aspect of medicine that has made great progress in prevention, as well as in acute care.¹ It is therefore important to evaluate from reliable populationbased studies any changes in the epidemiology of strokes, similar to declining mortality, for example, observed in cardiovascular disease.² For strokes, the results are not so clear³: there is evidence of both falling and rising case-fatality and mortality rates.^{4,5} We observed similar conflicting data for the incidence of stroke.^{4,6–9}

In Dijon, we have maintained an on-going populationbased stroke registry since 1985 to monitor first-ever stroke occurrence within the population of the city of Dijon.⁷

The aim of this study was to analyze the incidence, case-fatality and severity trends, risk factors and prestroke use of preventive treatments in stroke subtypes from 1985 to 2004.

Materials and Methods

We used the same stroke definition throughout the 20-year period.7

Study Area and Population

According to the census, the population of Dijon was 145 325 inhabitants in 1985 and 150 138 in 2004. The proportion of the

population at risk of stroke \geq 75 years in Dijon, increased by 17% from 1985 to 2004, whereas the proportion of people \geq 85 years increased by 51%.

Case Ascertainment

The major advantage of the stroke Registry of Dijon is its constant method of ascertainment, irrespective of age, without interruption since 1985. A detailed description of the Dijon Stroke Registry has been published.⁷

The collaboration of numerous investigators was required from various departments of the University Hospital, the 3 private hospitals, the 3 private radiology centers, the public and private specialists and general practitioners. Strokes were defined according to World Health Organization (WHO) recommendations¹⁰ and according to the International Classification of Disease.¹¹ The ischemic or hemorrhagic mechanism was identified by CT scans in 90% of the cases in 1985, in 98% of the cases since 1990. MRI examinations took place in 22% of the cases since 2000. A single first-ever stroke or a first-ever stroke preceded by a transient ischemic attack (TIA) was coded as incident. A stroke preceded by a previous stroke was coded as recurrent^{9,12,13} and was not included for the evaluation of incidence, case-fatality and mortality rates for first-ever stroke. If the subject was still alive 28 days after stroke onset, the stroke was considered nonfatal.

Stroke is available at http://www.strokeaha.org

Received September 14, 2005; final revision received March 29, 2006; accepted April 13, 2006.

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Vascular Risk Factors

We collected vascular risk factors according to the same criteria over the 20 years⁷: hypertension (HT) if \geq 160/095 mm Hg, diabetes mellitus (DM) if the fasting plasma glucose level \geq 7.8 mmol/L, (or if treated with insulin or oral hypoglycemic agents), hypercholesterolemia if the total cholesterol level \geq 6.0 mmol/L, history of tobacco abuse (>1 cigarette per day, current or former habit), TIA in the previous month, previous myocardial infarction, angina, and peripheral vascular disease.

Atrial fibrillation was diagnosed from ECG or Holter recordings. Two-dimensional echocardiography was performed to detect possible cardioembolic sources. Hypertrophy of left ventricle was noted in 18.55% ($\pm 6.47\%$) with no significant variations on echocardiography performed in 35% of the cases in 1985, 45% in 1990, 65% in 1995 and 82% in 2004.

Carotid and vertebral ultrasonography as well as standard blood and urine tests were routinely performed. There was no change in the prevalence of cervical artery occlusion, which remained around 6.14% ($\pm 3.25\%$), dissection of cervical arteries which remained around 5.75% ($\pm 2.25\%$) and stenosis of the cervical carotid $\geq 70\%$ which remained around 7.8% ($\pm 3.75\%$) on ultrasonography, that was performed in 85% of the cases in 1985, 95% in 1990 and close to 98% in 2004.

Use of prestroke preventive treatment was recorded at stroke onset: treatment of blood HT, DM, cardiac arrhythmia, and the use of antiplatelets and anticoagulants. In 1985, treatment with lipidlowering drugs was not requested and was not recorded in our files.

Handicap Scores at 1 Month

The degree of handicap was estimated according to the Rankin Scale at $1 \mbox{ month}.$

Diagnosis of Stroke Subtypes and Classification

The diagnosis of stroke subtypes was always performed on clinical and cerebral imaging.⁷ We included (1) ischemic strokes (IS) from atheroma of large arteries; (2) ischemic strokes from lipohyalinosis of small arteries, so-called lacunar infarct (LI); (3) ischemic strokes from cardiac embolism (CE) originating from atrial fibrillation (AF), or valve disease, patent foramen ovale or spontaneous intracavitar thrombus; (4) primary cerebral hemorrhagic (PCH) and (5) subarachnoid hemorrhagic (SAH) stroke. When it was difficult to differentiate between IS, LI and CE strokes, medical staff meetings were held to classify the difficult cases in 1 of the 3 groups.

Data Processing and Statistical Methods

The age- and sex-specific incidence rates were calculated. The standardized rates were calculated by the direct method with the French (census of 1999), European and the SEGI World⁹ populations. We assumed a Poisson distribution for the annual number of events to calculate 95% CI for the incidence.⁹ Exponential regression was performed to test time trends in stroke incidence after age and sex adjustment to the population of Dijon.

Case-fatality rates were based on survival after 28 days and trends were evaluated with linear regression. Univariate analyses with Student *t* test were performed for continuous variables. A *P* value <0.05 was considered significant. We used multivariate regression to identify the independent class variables (age, sex, stroke subtype, HT, DM, hypercholesterolemia, previous TIA, AF, myocardial infarction, peripheral vascular disease and time periods) that may influence 28-day case-fatality (dependent variable).

Changes in prestroke risk factors and medication were ascertained and evaluated according to linear regressions when comparing the trends over 20 years, and according to proportion tests when comparing the first and last periods.

Results

Clinical Features Among the 3691 Stroke Patients

We registered 1920 IS, 725 LI, 497 CE stroke, 341 PCH, 74 SAH and 134 ischemic strokes of an undetermined mecha-

nism. In the latter group there was no variation in fatal and nonfatal cases during the 20 years.

The absolute number of strokes increased by 11%, with 875 cases during the first 5-year period (1985–1989) and 982 the last 5-year period (2000–2004).

The mean age at stroke onset significantly increased from 66.0 years in 1985 to 71.1 in 2004 (P=0.01) for men and from 67.8 years to 75.6 years (P=0.01) for women.

The mean age was significantly higher in women than in men (76.2 versus 70.1 for IS, P < 0.01; 75.5 versus 71.5 for LI, P < 0.01; 82.0 versus 77.3 for CE, P < 0.01) except for PCH (72.5 versus 71.4, P = 0.83) and SAH (58.2 versus 52.8, P = 0.90).

Distribution of Standardized Incidence Rates of First-Ever Strokes by 5-Year Periods, by Age and Sex

According to standardized rates from European (Table 1a) and world (Table 1b) populations, the stroke incidence rates were higher in men than in women (P<0.01), for every period over the 20 years. The trends over the 20 years in both men and women were not significant.

If we compare the first 5-year period to the last one according to European population (Table 1a), we observe no change between these 2 periods. The incidence rates of strokes in men ($P^*=0.58$) and in women ($P^*=0.60$) remained stable. The trends of strokes over 85 years of age ($P^*=0.28$) and <85 years of age ($P^*=0.58$) for these 2 periods were unchanged. In contrast, by comparing the first and the last periods, we found that though the rise in the incidence of IS from atheroma of large arteries was not significant ($P^*=0.09$), the incidence of LI rose significantly ($P^*<0.01$), whereas the incidence of CE strokes decreased significantly ($P^*<0.01$). There was no change for PCH and SAH incidence.

Distribution of Handicap

Comparing the first (1985) and the last (2004) periods, Rankin scores at 1 month did not have the same distribution and amplitude: the proportion of Rankin consistent with spontaneous walking (score from 0 to 3), increased after 65 years (P=0.01), whereas the proportion of Rankin from 4 to 5 in these patients fell (P=0.01).

Distribution of 28-Day Case-Fatality Rates by 5-Year Periods, by Age and Sex

The 28-day case-fatality rates (Table 2) significantly decreased for overall strokes during the 20 years (P=0.03). The case-fatality rates decreased in men aged <75 years (P=0.02), in women aged <75 years (P=0.02) and 65 years (P<0.01). According to stroke subtypes, case-fatality rates significantly decreased for LI (P=0.05) and for PCH (P=0.03).

In multivariate regression analyses, hemorrhagic strokes, the first 5-year period, HT, previous myocardial infarction and age over 85 years had a significant negative effect on case-fatality at 28 days. In contrast, sex, hypercholesterolemia, diabetes, history of TIA, AF, tobacco and peripheral artery disease had no effect.

	1985–1989	1990–1994	1994–1999	2000–2004	Relative Incidence (first and last period)	Р	Average Rate of Annual Variation Incidence	Р
	Rates (95% CI)		Rates (95% CI)	Rates (95% CI)	RI (95% CI)	Value*	Variation (95% CI)	, Value**
Any first ever stroke								
Men	130.57	134.77	109.64	124.45	0.96 0.4		-0.0071	0.48
	(117.99–143.15)	(122.27–147.26)	(98.50-120.79)	(112.49–136.40)	(0.84-1.10)		(-0.0233-0.0091)	
Women	73.18	80.24	68.00	80.09	1.04	0.60	0.0023	0.81
	(65.68-80.69)	(72.53-87.95)	(60.76–75.23)	(72.17-88.02)	(0.92-1.17)		(-0.0143-0.0189)	
Age $>$ 85 y	1119.76	1357.70	1078.06	996.69	0.89	0.28	-0.0108	0.49
	(941.16–1298.37)	(1173.19–1542.22)	(932.94–1223.18)	(857.16–1136.23)	(0.71–1.12)		(-0.0358-0.0142)	
Age $<\!\!85$ y	87.54	90.55	75.42	90.08	1.03	0.58	-0.0018	0.86
	(80.82–94.27)	(83.84–97.25)	(69.25-81.59)	(83.31–96.85)	(0.93–1.14)		(-0.0194-0.0158)	
Overall	97.87	103.22	85.45	99.14	1.00	0.97	-0.0029	0.76
	(90.97–104.76)	(96.33–110.11)	(79.16–91.73)	(92.30–105.99)	(0.92-1.10)		(-0.0195-0.0136)	
Stroke subtypes								
lschemic stroke								
Macroatheroma	50.13	58.51	41.33	54.77	1.12	0.09	-0.0013	0.94
	(45.11–55.15)	(53.29–63.72)	(36.95–45.71)	(49.65–59.90)	(0.99–1.26)		(-0.0323-0.0297)	
Microatheroma	16.97	16.25	21.05	23.39	1.35	< 0.01	0.0255	0.07
	(14.07–19.88)	(13.48–19.02)	(17.90-24.20)	(19.98–26.80)	(1.13–1.60)		(0.0114-0.0396)	
Cardioembolic	13.62	11.37	8.98	9.60	0.68	< 0.01	-0.0279	0.08
	(11.31–15.94)	(9.27-13.48)	(7.13–10.83)	(7.68–11.52)	(0.51–0.91)		(-0.04450.0114)	
Primary intracerebral								
hemorrhage	9.08	10.31	8.66	9.01	1.17	0.31	-0.0090	0.67
	(6.86–11.29)	(8.03–12.58)	(6.65–10.67)	(7.01–11.01)	(0.89–1.55)		(-0.0445-0.0264)	
Subarachnoid								
hemorrhage	2.53	1.90	3.37	2.12	0.84	0.60	0.0018	0.95
	(1.31–3.75) (0.87–2.93)		(2.00-4.75)	(1.04-3.21)	(0.41-1.73)		(-0.0542-0.0579)	

TABLE 1a. Standardized Overall Incidence According to European Population per 100 000 per year (95% CI) of First Strokes, From 1985 to 2004, Stratified by Sex, Age, and Pathological Type

*1985-1989 vs 2000-2004; **over 20 years.

Distribution of Premorbid Risk Factors and Use of Preventive Treatments, by 5-Year Periods

Table 3 reports the 3 significant results on prestroke risk factors. Comparing the period between 1985 and 1989 and the period between 2000 and 2004, the prevalence of hypercholesterolemia and diabetes significantly increased ($P^* < 0.01$), whereas the prevalence of previous myocardial infarction significantly decreased ($P^* < 0.02$). Because of the increasing number of missing data for blood hypertension and smoking, changes in their prevalence could not be estimated.

For prestroke medication, we noted a significant increase of the use of antiplatelets agents and anticoagulants in AF ($P^* < 0.01$), with no change for treated HT.

Discussion

The distribution of stroke subtypes in Dijon was similar to those observed in other Western countries.^{13–15} Previous reports^{4,6–8,16} have observed no significant changes in stroke incidence rates during the last 2 decades. Dijon may be included in this group. However, a marked decrease had been observed in other studies.¹⁷ Recently, the Oxford study¹³ reported a significant decrease in the incidence rates during a 20-year period but using a register at 2 time points only. But for us, the most encouraging result concerns the increased age at first stroke onset.

We must analyze the reasons for this discrepancy. The incidence rates were stable in Dijon even though the absolute number of strokes significantly increased in both men and women, and the population at risk >85 years increased by 51% as observed elsewhere.¹³ Our results may reflect a balance between the increasing number of strokes and elderly persons in the Dijon population, the efficiency of prestroke preventive treatment, demonstrated by the rise in use of antiplatelet and anticoagulant therapy, and the decrease in previous myocardial infarction, as noted elsewhere.^{13–16}

We can explain the rise of LI incidences rates by the rise in the prevalence of diabetes and hypercholesterolemia. In contrast, the rise in the use of antiplatelets may explain the decrease in the prevalence of previous myocardial infarction. The issue of uncontrolled hypertension in Dijon cannot be discussed in detail because the frequency of missing data increased during the study period. However, HT has been shown to be difficult to control even in selected populations as well as during therapeutic trials.¹⁸

	1985–1989	1990–1994	1994–1999	2000–2004	Relative Incidence (first and last period)	P Value*	Average Rate of Annual Variation Incidence	P Value**	
Any first ever stroke									
Men	88.16	88.45	72.85	82.82	0.96	0.58	-0.0078	0.43	
	(79.24–97.08)	(79.90–96.99)	(65.06-80.63)	(74.41–91.23)	(0.84–1.10)		(-0.0233-0.0078)		
Women	48.47	50.78	44.18	53.20	1.04	0.60	0.0032	0.74	
	(42.86-54.08)	(45.43-56.14)	(39.02–49.35)	(47.32–59.09)	(0.92-1.17)		(-0.013-0.0194)		
Age $>$ 85 y	1119.76	1357.70	1078.06	996.69	0.89	0.28	-0.0108	0.49	
	(941.16–1298.37)	(1173.19–1542.22)	(932.94–1223.18)	(857.16–1136.23)	(0.71–1.12)		(-0.0358-0.0142)		
Age $<\!\!85$ y	60.43	60.36	51.15	61.37	1.03	0.58	-0.0023	0.82	
	(55.46-65.40)	(55.65-65.06)	(46.74–55.57)	(56.44-66.29)	(0.93-1.14)		(-0.0197-0.0152)		
Overall	65.73	66.84	56.29	66.04	1.00	0.97	-0.0031	0.74	
	(60.70–70.75)	(62.07-71.62)	(51.84–60.74)	(61.09–70.99)	(0.92-1.10)		(-0.0193-0.0131)		
Stroke subtypes									
Ischemic stroke									
Macroatheroma	34.54	37.97	27.55	37.08	1.12	0.09	-0.0017	0.92	
	(30.80–38.29)	(34.36-41.58)	(24.41-30.69)	(33.31–40.85)	(0.99–1.26)		(-0.0312-0.0278)		
Microatheroma	11.44	10.64	13.77	15.63	1.35	< 0.01	0.0254	0.09	
	(9.34–13.55)	(8.71–12.57)	(11.57–15.98)	(13.18–18.09)	(1.13–1.60)		(0.0094–0.0413)		
Cardioembolic	7.98	6.74	5.28	5.74	0.68	< 0.01	-0.0268	0.09	
	(6.52–9.44)	(5.39-8.08)	(4.09-6.48)	(4.49–7.00)	(0.51–0.91)		(-0.04410.0094)		
Primary intracerebral									
hemorrhage	6.57	7.07	5.75	5.74	1.17	0.31	-0.0119	0.26	
	(4.87-8.26)	(5.41-8.73)	(4.33–7.18)	(4.36-7.11)	(0.89–1.55)		(-0.0268-0.0030)		
Subarachnoid									
hemorrhage	1.97	1.49	2.60	1.67	0.84	0.60	0.0021	0.95	
	(1.01–2.93)	(0.67-2.31)	(1.52-3.68)	(0.81–2.53)	(0.41–1.73)		(-0.0520-0.0561)		

TABLE 1b. Standardized Overall Incidence According to World Population per 100 000 per year (95% CI) of First Strokes, From 1985 to 2004, Stratified by Sex, Age, and Pathological Type

*1985-1989 vs 2000-2004; **over 20 years

Therefore, lack of control of HT may, hypothetically, explain the absence of any decrease in stroke incidence.

The other reason for the discrepancy between Dijon and Oxford¹³ may be that the proportion of diabetes was stable in Oxford¹³ but significantly increased in Dijon. Specific data concerning the incidence of LI were not available in Oxford¹³ or in Söderham,¹⁶ but were recorded in the Germany study.¹⁴ It is known that the prevention of stroke in diabetic patients is not very effective.¹

In Dijon, like in most studies, case-fatality rates were slightly higher in women than in men.^{9,17} Mean case-fatality rates were rather lower in Dijon (14.5%) than in Belluno¹⁹ (33%). Case-fatality rates in older men and in younger women also declined in Dijon as was the case in other populations^{8,9,13,16} but not in all cases.^{5,20,21} The determinants of better case-fatality rates were LI, lower systolic and diastolic blood pressure, younger age and the last time period.

Several factors observed in Dijon may account for the fall in case-fatality rates: the rise in LI incidence associated with the decrease in the incidence of severe CE stroke, the rise in minor strokes, associated with the decrease in strokes with severe handicaps. This reduction may be also a cohort effect^{13,16} because life expectancy in the general population was also improving. Our study has some interesting points. There were no changes in the ethnic mix and economic status of the population of Dijon, or in the organization of the healthcare system during the 20-year study. The strength of our population-based register is that uniform registration and diagnostic criteria established by the WHO¹⁰ have been strictly applied over the study period to ensure that secular trends in the incidence of stroke were not affected by changes in diagnostic practices or incomplete ascertainment of cases. Another interesting point of our study was the long-term involvement by the same investigators, and the same research team during the 20-year period. There was no age limit for patients.

In Dijon, we think that the ascertainment was exhaustive because the incidence of minor handicaps (Rankin from 0 to 3) increased, meaning that we were able to identify minor cases. Consistency and reliability were also ensured by the high percentage of diagnostic investigations performed on each patient. CT plus MRI scans were performed in almost 100% of the patients in Dijon.

But our study has some limits with the missing data on systolic and diastolic blood pressure and smoking.

It is the first population-based study, which investigated stroke incidence, and case-fatality rates on the basis of

	1985–1989	1990–1994	1995–1999	2000-2004	Average Percentage of Annual Variation	<i>P</i> Value
Sex						
Men	17.8% (14.6%–21.7%)	12.5% (9.7%–15.9%)	13.0% (10.1%–16.5%)	9.7% (7.3%–12.8%)	-0.0048 (-0.00770.0018)	0.09
<65	8.4% (4.9%-14.4%)	3.4% (1.3%-8.8%)	7.0% (3.4%–14.1%)	3.7% (1.6%-8.7%)	-0.0021 (-0.0065-0.0023)	0.45
<75	13.1% (9.5%–17.9%)	8.8% (5.9%-12.9%)	7.4% (4.7%–11.5%)	3.1% (1.5%-6.4%)	-0.0063 (-0.00790.0047)	0.02
>75	24.4% (18.8%-31.2%)	17.2% (12.6%–23.3%)	19.7% (14.8%–26.0%)	16.6% (12.4%-22.2%)	-0.0041 (-0.0090-0.0008)	0.24
Women	17.7% (14.5%–21.6%)	20.3% (17.1%–24.1%)	14.8% (11.9%–18.4%)	10.3% (7.9%–13.3%)	-0.0056 (-0.01060.0005)	0.16
<65	13.7% (7.4%–24.8%)	9.5% (4.4%-20.0%)	6.8% (2.9%–15.6%)	3.4% (1.1%-10.2%)	-0.0067 (-0.00750.0060)	< 0.01
<75	17.0% (11.8%–24.1%)	9.8% (6.2%-15.3%)	7.8% (4.5%–13.3%)	2.4% (0.9%-6.4%)	-0.0091 (-0.01180.0065)	0.02
>75	18.0% (14.1%-22.9%)	25.7% (21.3%-30.7%)	18.2% (14.4%-22.9%)	13.9% (10.6%–18.0%)	-0.0040 (-0.0130-0.0050)	0.48
Overall	17.8% (15.4%–20.5%)	16.6% (14.4%–19.1%)	13.9% (11.8%–16.3%)	10.0% (8.3%-12.1%)	-0.0052 (-0.00690.0035)	0.03
Stroke subtypes						
Ischemic stroke						
Macroatheroma	11.9% (9.2%–15.2%)	11.6% (9.1%–14.6%)	12.4% (9.7%–15.8%)	7.6% (5.6%–10.3%)	-0.0024 (-0.0058-0.0010)	0.30
Microatheroma	6.9% (3.8%-12.4%)	4.1% (1.9%–9.0%)	3.4% (1.6%-6.9%)	2.4% (1.0%-5.8%)	-0.0028 (-0.00410.0015)	0.05
Cardioembolic	25.8% (19.5%-33.7%)	32.4% (25.1%–41.1%)	23.1% (16.8%–31.4%)	19.6% (13.7%–27.5%)	-0.0056 (-0.0143-0.0030)	0.33
Primary intracerebral						
hemorrhage	42.6% (32.1%-54.8%)	39.4% (30.1%-50.4%)	34.0% (24.7%-45.6%)	24.5% (17.0%-34.6%)	-0.0119 (-0.01590.0079)	0.03
Subarachnoid hemorrhage	23.0% (9.3%–50.3%)	7.7% (1.1%–43.4%)	23.3% (10.3%-47.9%)	26.1% (10.6%–55.5%)	0.0050 (-0.0115-0.0215)	0.61

TABLE 2. Sex, Age, and Stroke Subtypes Adjusted 28-day Case-Fatality Rates From 1985 to 2004

continuous ascertainment over a 20-year period, identifying every stroke subtype thanks to at least 98% verification by neuroimaging. Our study provided evidence that preventive strategies did reduce the incidence of CE strokes at community level but not LI, which is induced by rising diabetes and hypercholesterolemia and possibly by the lack of optimal treatment of HT. The encouraging results concerned the important effect of prevention which delayed stroke onset, and the decrease in case-fatality at 28 days in both men and women.

TABLE 3. Prestroke Risk Factors and Medications in Patients With Stroke

	1985–1989		89	1990-1994		1995–1999		2000-2004							
	n	%	Missing	n	%	Missing	n	%	Missing	n	%	Missing	<i>P</i> *	Trend**	P**
Prestroke risk factor:															
Hypercholesterolemia	109	(11.7%)		135	(12.5%)		231	(22.9%)		329	(28.7%)		< 0.01	0.0122	0.04
Blood Hypertension history	607	(65.3%)		663	(61.6%)		625	(61.9%)		734	(64.1%)		0.57	-0.0006	0.76
Diabetes	97	(10.4%)		136	(12.6%)	21 (2.0%)	159	(15.7%)	24 (2.4%)	200	(17.5%)	10 (0.8%)	< 0.01	0.0048	< 0.01
Previous TIA	234	(25.2%)		234	(21.7%)		215	(21.3%)		255	(22.3%)		0.12	-0.0018	0.33
Previous atrial fibrillation	213	(22.9%)		312	(29.0%)	24 (2.2%)	293	(29.0%)	27 (2.7%)	265	(23.1%)	7 (0.6%)	0.91	0.0001	0.97
Previous myocardial infarction	206	(22.2%)		217	(20.1%)	26 (2.4%)	261	(25.8%)	23 (2.3%)	205	(17.9%)	7 (0.6%)	0.02	-0.0014	0.73
Peripheral vascular disease	112	(12.1%)		104	(9.7%)	25 (2.3%)	130	(12.9%)	24 (2.4%)	117	(10.2%)	6 (0.5%)	0.17	-0.0004	0.80
Prestroke medication:															
Treated blood hypertension						17 (1.6%)			28 (2.8%)			7 (0.6%)			
1 drug	239	(25.7%)		285	(26.5%)		285	(28.2%)		321	(28.0%)		0.24	0.0017	0.08
2 drugs	206	(22.2%)		304	(28.2%)		259	(25.6%)		240	(21.0%)		0.51	-0.0012	0.76
1 or 2 drugs	445	(47.9%)		589	(54.7%)		544	(53.9%)		561	(49.0%)		0.62	0.0005	0.91
Antiplatelets agent	48	(5.2%)		169	(15.7%)	19 (1.8%)	319	(31.6%)	30 (3.0%)	312	(27.2%)	6 (0.5%)	< 0.01	0.0164	0.11
Anticoagulants for atrial fibrillation	24	(2.6%)		70	(6.5%)	29 (2.7%)	65	(6.4%)	28 (2.8%)	88	(7.7%)	5 (0.4%)	<0.01	0.0030	0.11

*1985-1989 vs 2000-2004; **over 20 years

Thus, there is still an opportunity for considerable health gains in stroke care in a general population and for further evaluations.

Acknowledgments

We would like to thank the University Hospital of Dijon, the Faculty of Medicine of Dijon, the Burgundy University, the Regional Agency of Hospitalization, the Regional Council of Burgundy, Inserm and the Institut de Veille Sanitaire, Grant Barry and Philip Bastable for rewriting the text.

None.

Disclosures

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