Cerebral Aneurysms

AB-14292-99


Object. The occurrence of cerebral aneurysms has been linked to alterations in the extracellular matrix and to matrix-degrading proteases. The purpose of the present study was to determine whether active extracellular matrix remodeling occurs within cerebral aneurysms.

Methods. Aneurysm tissue was collected from 23 patients (two of whom had a ruptured aneurysm and 21 of whom had an unruptured aneurysm) and compared with 11 control basilar arteries harvested at autopsy. Active proteinases capable of gelatin lysis were identified by performing in situ zymography in the presence and absence of a metalloproteinase inhibitor (ethylenediamine tetraacetic acid) and a serine proteinase inhibitor (phenylmethylsulfonyl fluoride). Immunohistochemical analysis was used to localize plasmin, tissue-type (t)-plasminogen activator (PA), urokinase-type (u)-PA, membrandetype (MT1)–matrix metalloproteinase (MMP), MMP-2, MMP-9, and tenascin.

Focal areas of gelatin lysis occurred in most cerebral aneurysm tissue samples (17 of 21), but rarely in control arteries (two of 11) (p = 0.002). Both serine proteinases and MMPs contributed to gelatin lysis; however, the MMPs were the predominant enzyme family. Plasmin (p = 0.04) and MT1-MMP (p = 0.04) were expressed in the aneurysm tissue but were unusual in control tissue. The MMP-2 was also expressed more commonly in aneurysm than in control tissue (p = 0.07). The MMP-9 and t-PA were expressed in both groups; however, different staining patterns were observed between aneurysm and control tissue. Tenascin staining was commonly present in both groups, whereas u-PA staining was rarely present.

Conclusions. Aneurysm tissue demonstrates increased proteolytic activity capable of lysing gelatin and increased expression of plasmin. MT1-MMP, and MMP-2 when compared with normal cerebral arteries. This activity may contribute to focal degradation of the vascular extracellular matrix and may be related to aneurysm formation and growth.

AB-14293-99


Object. Cigarette smoking is associated with aneurysmal subarachnoid hemorrhage (SAH) and subsequent vasospasm. The purpose of this study was to quantify this association.

Methods. Nearly 3500 patients with SAH from North America and Europe have been enrolled in five different multicenter, controlled studies coordinated at the Neuroclinical Trials Center of the Virginia Neurological Institute at the University of Virginia. Among the prospective data gathered were whether the patient smoked at the time of their most recent SAH and the evolution of angiographic vasospasm. The rate of smoking in the patients enrolled in the studies was compared with the expected rate by using a chi-square statistic adjusted for age and gender, in the general population in the United States (U.S.) and Europe. In virtually all age and gender subgroups, and for the combined populations in the five clinical trials, patients with SAH reported current smoking rates 2.5 times higher than expected based on U.S. and European national surveys (p < 0.0001). Cigarette smoking was also associated with younger age at onset of SAH (5–10 years, p = 0.0001) and increased incidence of clinically confirmed vasospasm (p < 0.005).

Conclusions. The findings of a significantly increased representation of current cigarette smokers in the study populations and significant association with younger age at the time of SAH and increased incidence of vasospasm concur with recent reports of smoking as a significant risk factor for ruptured aneurysms and subsequent vasospasm.

AB-14294-99


Background and Objective: In families with two or more relatives with subarachnoid hemorrhage (SAH), other first-degree relatives have an increased risk of SAH. We studied the presence of unruptured intracranial aneurysms in 125 members of 23 families with familial SAH, defined as two or more affected first-degree relatives, in a cross-sectional design. Methods: MR angiography was performed in 116 relatives; CT angiography was performed in the remaining 9 relatives because they had been treated for intracranial aneurysms in the past. Results: Overall, we found 16 aneurysms in 10 of 125 relatives (8%; 95% CI, 4 to 14%). Of the nine patients with previous surgery for ruptured or unruptured intracranial aneurysms, three had new aneurysms. Two factors were associated with a significantly higher risk of intracranial aneurysms: 1) a history of treatment for ruptured or unruptured intracranial aneurysms (relative risk 5.5; 95% CI, 1.7 to 17.8) and 2) having three or more affected relatives (relative risk 3.3; 95% CI, 1.0 to 10.6). Siblings tended to have a higher risk of intracranial aneurysms than did children of SAH patients, although the difference was not significant. Conclusions: Because the yield is high, screening is recommended in first-degree members of families with familial SAH. Repeated screening should be considered in relatives who have been treated for familial intracranial aneurysms.

The abstracts in this section have been typeset for consistency with journal format but otherwise appear as in the original articles.
Results Among these patients, 68 had a stroke during the follow-up (rate of stroke per year of follow-up 3.2%). In 95 patients with LAD of ≥48 mm, the incidence of stroke (9%) in the severe MR group (moderate or severe, n = 43) was significantly lower than that (25%) of the mild MR group (none, trivial, or mild; n = 52) (chi-square = 3.95, p = 0.047). The relative risk of stroke for increase in MR from mild to severe groups, for every 10 mm increment in LA size, for sex, and for every increase of 10 years of age was 0.45 (95% CI, 0.20 to 0.97), 1.06 (95% CI, 0.75 to 1.49), 0.98 (95% CI, 0.55 to 1.72), and 1.33 (95% CI, 1.04 to 1.71), respectively.

Conclusions In patients with nonhemorrhagic AF, age was an independent predictor of an increased risk of stroke, and MR may be protective against stroke, especially in those patients with LA enlargement.

Epidemiology

AB-14296-99


The insertion (I)/deletion (D) polymorphism of the angiotensin-converting-enzyme (ACE) gene has been associated with an increased risk of myocardial infarction, lacunar stroke, and with an increased intimal-medial thickness in several populations. The aim of this study was to evaluate whether the ACE I/D genotype is associated with stenosis of extracranial arteries and stroke in middle-aged and aged men and women. We studied 388 patients (247 male, 141 female) using Doppler and duplex ultrasound of the extracranial arteries. Patients’ history was obtained by standard questionnaire and by the hospital case records. Genomic DNA was analyzed by polymerase chain reaction (PCR) to identify the I/D polymorphism, with a second insertion specific PCR in samples classified as homozygous DD genotypes to prevent mistyping. The ACE genotype groups (DD 132, ID 164, II 92) were well matched for the basic characteristics. The DD genotype was more common in patients with extracranial artery stenosis ≥50% compared with patients without stenosis (59/147 versus 73/241, odds ratio 1.54, 95%-CI 1.01–2.37), but was not associated with a history of stroke (30/91 versus 102/297, odds ratio 0.94, 95%-CI 0.57–1.54). The association of the DD genotype with extracranial artery stenosis was also present in hypertensive subjects (n = 206, odds ratio 1.76, 95%-CI 0.99–3.17). In the whole group multiple logistic regression analysis revealed that the association of the DD genotype with extracranial artery stenosis was independent of age, gender, hypertension, hyperlipidemia, and diabetes. In conclusion, the ACE DD genotype is a weak risk factor for hemodynamically relevant stenosis of extracranial arteries, but not for stroke.

AB-14297-99


Background The long term health consequences of snoring and sleep apnoea syndrome are still uncertain. This study was conducted to assess the mortality risk associated with snoring and excessive daytime sleepiness (EDS), the two main symptoms of sleep apnoea syndrome, in men.

Methods In 1984 a sample of 3100 men aged 30–69 responded to a postal questionnaire including questions about snoring, EDS, and the prevalence of various diseases (response rate 77.1%). Mortality data for the period 1985–1995 were collected for the complete sample.

Results During the 10 year follow up period 213 men died, 88 of cardiovascular diseases. Compared with subjects with no snoring or EDS in 1984, men with isolated snoring or EDS displayed no significantly increased mortality. The combination of snoring and EDS was associated with a significant increase in mortality. However, the relative rates decreased with increasing age, and in men aged 60 and above no effect on mortality was found. Men below the age of 60 with both snoring and EDS had an age adjusted total death rate which was 2.7 times higher than men with no snoring or EDS (95% CI 1.6 to 4.5). The corresponding age adjusted hazard ratio for cardiovascular mortality was 2.9 (95% CI 1.3 to 6.7) for subjects with both snoring and EDS. Further adjustment for body mass index and reported hypertension, cardiac disease, and diabetes reduced the relative mortality risk associated with the combination of snoring and EDS to 2.2 (95% CI 1.3 to 3.8) and the relative risk of cardiovascular mortality to 2.0 (95% CI 0.8 to 4.7).

Conclusion Snoring without EDS does not appear to carry an increased risk of mortality. The combination of snoring and EDS appears to be associated with an increased mortality rate, but the effects seems to be age dependent. The increased mortality is partly explained by an association between “snoring and EDS” and cardiovascular disease.

Experimental Pathology

AB-14298-99


Focal cerebral ischemia elicits local inflammatory reaction as demonstrated by the accumulation of inflammatory cells and mediators in the ischemic brain. Interferon-inducible protein-10 (IP-10) is a member of the C-X-C chemokine family that possesses potent chemotactic actions for monocytes, T cells, and smooth muscle cells. To investigate a potential role of IP-10 in focal stroke, we studied the temporal expression of IP-10 mRNA after occlusion of the middle cerebral artery in rats by means of northern analysis. IP-10 mRNA expression after focal stroke demonstrated a unique biphasic profile, with a marked increase early at 3 h (4.9-fold over control; p < 0.01), a peak level at 6 h (14.5-fold; p < 0.001) after occlusion of the middle cerebral artery, and a second wave induction 10–15 days after ischemic injury (7.2- and 9.3-fold increase for 10 and 15 days, respectively; p < 0.001). In situ hybridization confirmed the induced expression of IP-10 mRNA and revealed its spatial distribution after focal stroke. Immunohistochemical studies demonstrated the expression of IP-10 peptide in neurons (3–12 h) and astroglial cells (6 h to 15 days) of the ischemic zone. To explore further the potential role of IP-10 in focal stroke, we demonstrated a dose-dependent chemotactic action of IP-10 on C6 glial cells and enhanced attachment of rat cerebellar granule neurons. Taken together, the data suggest that ischemia induces IP-10, which may play a pleiotropic role in prolonged leukocyte recruitment, astrocyte migration/activation, and neuron attachment/sprouting after focal stroke.

AB-14299-99

Effect of Reduced Cerebral Perfusion Pressure on Cerebral Blood Flow Following Inhibition of Nitric Oxide Synthesis—Rise IR, Kirkeby OJ (Dept of Neurosurgery, Ullevål Hospital, N-0407 Oslo, Norway)—J Neurosurg. 1998;89:448–453.

Object The authors tested the hypothesis in a porcine model that inhibition of nitric oxide synthesis during reduced cerebral perfusion pressure (CPP) affected the relative cerebral blood flow (CBF) and the cerebrovascular resistance.

Methods The CPP was reduced by inducing high cerebrospinal fluid pressure and hemorrhagic hypotension. With continuous blood and intracranial pressure monitoring, relative CPP was estimated using the laser Doppler flowmetry technique in nine pigs that received 40 mg/kg nitro-l-arginine methyl ester (l-NAME) and in nine control animals. The l-NAME caused a decrease in relative CBF (p < 0.01) and increases in cerebrovascular resistance (p < 0.01), blood pressure (p < 0.05), and CPP
(p<0.001). During high intracranial pressure there were no significant differences between the treated animals and the controls. After hemorrhage, there was no significant difference between the groups initially, but 30 minutes later the cerebrovascular resistance was decreased in the control group and increased in the L-NAME group relative to baseline (p<0.05). Combined hemorrhage and high intracranial pressure increased the difference between the two groups with regard to cerebrovascular resistance (p<0.05).

Conclusions. These results suggest that nitric oxide synthesis inhibition affects the autoregulatory response of the cerebral circulation after cardiovascular compensation has taken place. Nitric oxide synthesis inhibition enhanced the undesirable effects of high intracranial pressure during hypovolemia.

AB-14300-99

The effects of the anti-inflammatory cytokine, IL-10, on brain injury following permanent focal ischemia were determined. Rats subjected to occlusion of the right middle cerebral artery (MCAO) were administered IL-10 (1 µg) centrally into the lateral ventricle 30 min and 3 h post MCAO or systemically into the tail vein (5 or 15 µg/h) starting 30 min post MCAO for 3 h. Brains were removed 24 h later and infarct size was measured. IL-10 administered centrally significantly (P<0.01) reduced infarct size by 20.7%±6.0 compared to vehicle. Systemic IL-10 administration at 5 and 15 µg/h significantly (P<0.05) decreased infarct size (40.3%±14.0 and 30.7%±13.7, respectively). These studies indicate that an anti-inflammatory therapeutic approach using IL-10 can provide neuroprotection in ischemic stroke.

AB-14301-99

Determination of circulating activated platelets may be helpful to estimate the prognosis and to stratify therapies in arterial vascular disorders including stroke. We used flow cytometry and phase contrast microscopy to study whether the fraction of platelets expressing p-selectin and CD63 and the fraction of platelets with shape change are increased in patients with acute and previous cerebrovascular ischemia.

The proportion of platelets expressing activation dependent antigens was higher in patients with acute (n=24; p-selectin: 8.23±4.21%; CD63: 3.53±2.53%) and with previous cerebrovascular ischemia (n=46; 3.86±1.98%; 2.80±1.79%) as compared to age- and sex-matched control subjects (n=35; 2.17±0.96%; 1.79±0.75%; p≤0.005, respectively). In patients with previous ischemia, there was no difference between treatment with aspirin (n=25) or phenprocoumon (n=21). Hypertension, diabetes mellitus and smoking were not associated with increased antigen expression (analysis of variance). The fraction of discoid platelets and platelet counts were not significantly different between groups.

Our results indicate increased expression of platelet neoantigens in acute and to a less degree in previous cerebrovascular ischemia. Ongoing platelet activation after cerebrovascular ischemia despite therapy with aspirin or phenprocoumon indicates that new anti-platelet drugs may be of benefit for these patients. Flow cytometry appears to be a useful tool to assess platelet function in cerebrovascular ischemia.

AB-14302-99

We compared the frequencies of signs of old intracerebral hemorrhages on brain magnetic resonance imaging scans in 66 patients with ischemic stroke, 69 with myocardial infarction, and 86 with peripheral arterial disease (a total of 221 patients). Magnetic resonance imaging scans were independently assessed by two investigators without knowledge of clinical or laboratory data. In 31 patients (14%) we found local cerebral hemosiderin deposits. In 24 patients they were clinically silent. Hemosiderin deposits were significantly more frequent in patients with ischemic stroke (26%) than in patients with myocardial infarction (4%) or peripheral arterial disease (13%). Hemosiderin deposits were associated with cerebral white matter lesions (odds ratio, 5.3; 95% confidence interval, 2.5–12.4). The odds ratios were higher in patients with severe cerebral white matter lesions. Our findings support the hypothesis that cerebral vessels of patients with ischemic stroke are more prone to rupture than those of patients with other manifestations of atherosclerotic disease, which may explain the higher incidence of intracerebral hemorrhages when these patients are treated with oral anticoagulants. The microhemorrhages were associated with cerebral white matter lesions, which suggests that they are another manifestation of cerebral small-vessel disease.

AB-14303-99

Context—The relative importance of hemodynamic factors in the pathogenesis and treatment of stroke in patients with carotid artery occlusion remains controversial.

Objective—To test the hypothesis that stage II cerebral hemodynamic failure (increased oxygen extraction measured by positron emission tomography [PET]) distal to symptomatic carotid artery occlusion is an independent risk factor for subsequent stroke in medically treated patients.

Design and Setting—Prospective, blinded, longitudinal cohort study of patients referred from a group of regional hospitals between 1992 and 1996.

Patients—From 419 subjects referred, 81 with previous stroke or transient ischemic attack in the territory of an occluded carotid artery were enrolled. All were followed up to completion of the study, with average follow-up of 31.5 months.

Main Outcome Measures—Telephone contact every 6 months recorded the subsequent occurrence of all stroke, ipsilateral ischemic stroke, and death.

Results—Stroke occurred in 12 of 39 patients with stage II hemodynamic failure and in 3 of 42 patients without (P=.005); stroke was ipsilateral in 11 of 39 patients with stage II hemodynamic failure and in 2 of 42 patients without (P=.004). Six deaths occurred in each group (P=.94). The age-adjusted relative risk conferred by stage II hemodynamic failure was 6.0 (95% confidence interval [CI], 1.7–21.6) for all stroke and 7.3 (95% CI, 1.6–33.4) for ipsilateral stroke.

Conclusions—Stage II hemodynamic failure defines a subgroup of patients with symptomatic carotid occlusion who are at high risk for subsequent stroke when treated medically. A randomized trial evaluating surgical revascularization in this high-risk subgroup is warranted.

Neurosonology

AB-14304-99
Background and Objective: A clear association among snoring, sleep apnea, and increased risk of stroke has been shown by previous studies. However, the possible role played by sleep apnea in the pathogenesis of cerebrovascular disease is subject to debate. To evaluate the influence of hemodynamic changes caused by obstructive sleep apnea syndrome (OSAS), we investigated cerebrovascular reactivity to hypercapnia in patients with OSAS. Methods: The study was performed at baseline and after 1 night and 1 month of nasal continuous positive air pressure (n-CPAP) therapy, with patients in the waking state (8:00 to 8:30 AM and 5:30 to 6:00 PM) with transcranial Doppler ultrasonography. Cerebrovascular reactivity was calculated with the breath-holding index (BHI). Results: In the baseline condition, compared with normal subjects, patients with OSAS showed significantly lower BHI values in both the morning (0.57 versus 1.40; p<0.0001) and the afternoon (1.0 versus 1.51; p<0.0001). Cerebrovascular reactivity was significantly higher in the afternoon than it was in the morning in both patients (p<0.0001) and controls (p=0.05). In patients, the BHI returned to normal values, comparable with those of control subjects, after both 1 night and 1 month of n-CPAP therapy. Conclusions: These findings suggest an association between OSAS and diminished cerebral vasodilator reserve. This condition may be related to the increased susceptibility to cerebral ischemia in patients with OSAS, particularly evident in the early morning.

AB-14305-99
Hypoechoic Plaque at US of the Carotid Artery: An Independent Risk Factor for Incident Stroke in Adults Aged 65 Years or Older—Polak JF (Dept of Radiology, Brigham and Women’s Hospital, 75 Francis St, Boston, MA 02115), Shemenski L, O’Leary DH, Lefkowitz D, Price TR, Savage PJ, Brant WE, Reid C—Radiology. 1998;208:649–654.

METHODS: To investigate the association between incident (first) stroke and the echogenicity of internal carotid arterial plaque at ultrasonography (US).

MATERIALS AND METHODS: A cohort of 4,886 individuals who, at baseline, were 65 years of age or older and without symptoms of cerebrovascular disease was followed up for an average of 3.3 years. Baseline clinical findings were from color Doppler and duplex US studies of the carotid arteries and a record of traditional risk factors: age, sex, presence of diabetes mellitus, pack-years of cigarette smoking, presence of hypertension, elevated systolic and diastolic blood pressures, elevated low-density lipoprotein cholesterol level. Results: Incident strokes, excluding hemorrhagic strokes and strokes of cardiac origin, were seen in 104 individuals (2.1%) at risk. Age- and sex-adjusted odds ratios for incident stroke were significant for hypoechoic plaque (odds ratio, 2.53; 95% CI, 1.42, 4.53). After controlling for risk factors in a Cox proportional hazards model, the relative risk (RR) of incident stroke was 1.72 (p=0.015) for hypoechoic plaque and 2.32 (p=0.004) for internal carotid arterial narrowing of at least 50%. In addition, hypoechoic plaque (RR, 2.78; CI, 1.36, 5.69) and 50%–100% stenosis (RR, 3.08; CI, 1.28, 7.41) were associated with ipsilateral, nonfatal stroke.

CONCLUSION: In asymptomatic adults aged 65 years or older, the risk of incident stroke was associated with two US features: hypoechoic internal carotid arterial plaque and an estimated internal carotid arterial stenosis of 50%–100%.

Pharmacology / Therapeutics

AB-14306-99

PURPOSE: To evaluate the accuracy and clinical utility of the Outpatient Bleeding Risk Index for estimating the probability of major bleeding in outpatients treated with warfarin. The index was previously derived in a retrospective cohort of 556 patients from a different hospital (derivation cohort).

SUBJECTS AND METHODS: We enrolled 264 outpatients starting warfarin (validation cohort) to validate the index prospectively. All patients were identified upon hospital discharge, and physician estimates of the probability of major bleeding were obtained before discharge in the validation cohort.

RESULTS: Major bleeding occurred in 87 of 820 outpatients (6.5%/yr). The index included four independent risk factors for major bleeding: age 65 years or greater; history of gastrointestinal bleeding; history of stroke; and one or more of four specific comorbid conditions. In the validation cohort, the index predicted major bleeding: the cumulative incidence at 48 months was 3% in 80 low-risk patients, 12% in 166 intermediate-risk patients, and 53% in 18 high-risk patients (c index, 0.78). The index performed better than physicians, who estimated the probability of major bleeding no better than expected by chance. Of the 18 episodes of major bleeding that occurred in high-risk patients, 17 were potentially preventable.

CONCLUSIONS: The Outpatient Bleeding Risk Index prospectively classified patients according to risk of major bleeding and performed better than physicians. Major bleeding may be preventable in many high-risk patients by avoidance of over-anticoagulation and nonsteroidal anti-inflammatory agents.

AB-14307-99

PURPOSE: Recently developed interventional radiologic techniques, such as embolization with platinum coils, may induce thrombus formation within an aneurysm. The aim of the present study was to investigate the frequency of microemboli distal to untreated and treated cerebral aneurysms.

METHODS: Among a total of 110 patients treated with platinum coil embolization, 35 patients (27 women and eight men, aged 50±10 years) who were at high risk of ischemic complications underwent emboli detection with a transcranial Doppler sonographic monitoring system. All patients were studied before and after coil embolization. The aneurysms were located at the internal carotid artery (n=14), the basilar artery (n=10), the middle cerebral artery (n=7), or the vertebral artery (n=4). Twenty-nine (85%) of 35 patients were monitored within 6 hours of the completion of treatment.

RESULTS: Microemboli distal to the aneurysm were not detected in any of the patients before treatment. Microemboli were detected in 11 patients (31%) after embolization (mean, 16.2±21 per hour; range, 1–74 per hour). Microemboli were detected in five (71%) of seven patients in whom ischemic complications occurred after treatment, but in only six (21%) of 28 asymptomatic patients. This difference was statistically significant. The rate of occurrence of emboli in patients with ischemic complications (23±30 emboli per hour) was higher than in asymptomatic patients (10±7 emboli per hour), but this difference was not statistically significant.

CONCLUSION: Microemboli were detected significantly more often in patients who suffered from cerebral ischemia after coil embolization of an intracranial aneurysm. This observation supports the definition of a high-risk group of patients with incomplete embolization or with a large-diameter, broad-neck aneurysm. The early detection of microemboli after treatment may be an indicator for excessive intraaneurysmal thrombus formation and could influence the decision for prophylactic treatment with heparin or aspirin.
Surgery

AB-14308-99

Background. Improved techniques in cerebral and myocardial protection have made replacement of the chronically aneurysmal ascending thoracic aorta a safe and effective procedure. We hypothesized that patients with severe ascending or aortic arch atherosclerosis were at greater risk for operative complications during ascending aortic replacement because of the diffuse nature of their atherosclerotic process.

Methods. We retrospectively analyzed the records of 17 patients who received ascending aortic replacement during elective coronary artery bypass grafting (CABG) because of the intraoperative finding of severe atherosclerosis. All 17 patients underwent tube graft replacement of the ascending aorta under hypothermic circulatory arrest and retrograde cerebral perfusion before coronary artery bypass grafting. The outcomes for these patients were compared with those of a control group of 89 consecutive patients who underwent replacement for ascending aortic aneurysm.

Results. The hospital mortality rate for replacement of the ascending thoracic aorta for severe atherosclerosis was 23.5% (4/17) versus 2.25% (2 of 89) for the control group (p=0.006). The incidence of cerebrovascular accident in the atherosclerotic group was 17.6% (3/17) and 3.37% (3/89) for the control group (p=0.051). Nine of 17 atherosclerotic patients (52.9%) had operative morbidity. Only 20.2% (18 of 89) of the control patients had nonfatal postoperative complications.

Conclusions. The severely atherosclerotic ascending aorta is a marker of diffuse atherosclerosis. Despite improved techniques of myocardial and cerebral protection, we have been unable to duplicate our success with ascending thoracic aneurysm repair. Preoperative screening of the ascending aorta by chest computed tomography may be appropriate in select high-risk patients to determine operability.

AB-14309-99

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Main outcome measures: Regional, district, and age-sex operation rates as three year average 1993–6 (use) compared with respective estimates of need for carotid endarterectomy among those who presented with symptomatic carotid disease—transient ischaemic attack or minor stroke.

Results: The operation rate more than doubled between 1991–2 and 1995–6, from 35 to 89 per million population, compared with an estimated level of need in the region’s general population of 153 per million population (transient ischaemic attack 77, minor stroke 76). The ratio of use to need was 0.47 (95% confidence interval 0.4 to 0.54); district ratios were 0.28 (0.19–0.38) to 0.81 (0.62 to 1.06). The annual use:need ratio rose over the three years 1993–6 from 0.38 to 0.59. Use:need ratios were lower in elderly and female patients. Providers were keen to develop guidelines for referral and to increase access to diagnostic facilities; purchasers were more reluctant, given the limited impact of this intervention on the incidence of stroke and the relatively high cost of the operation.

Conclusion: Although treatment rates increased in Wessex there is still unmet need. Further research is needed to determine the referral pathways of patients with symptomatic carotid disease for diagnosis and operation and to evaluate strategies to improve access to diagnostic facilities.

Items of Interest

College of American Pathologists Conference XXXI on Laboratory Monitoring of Anticoagulant Therapy: The Clinical Use and Laboratory Monitoring of Low-Molecular-Weight Heparin, Danaparoid, Hirudin and Related Compounds, and Argatroban—Laposata M (Div of Laboratory Medicine, Massachusetts General Hospital, Gray Bldg, Rm 235, 32 Fruit St, Boston, MA 02114), Green D, Van Cott EM, Barrowcliffe TW, Goodnight SH, Sosolik RC—Arch Pathol Lab Med. 1998;122:799–807.


Prevention of Stroke in Patients With Nonvalvular Atrial Fibrillation—Hart RG (Dept of Medicine [Neurology], Univ of Texas Health Science Center, 7703 Floyd Curl Dr, San Antonio, TX 78284-7883), Sherman DG, Easton JD, Cairns JA—Neurology. 1998;51:674–681. © 1998 by the American Academy of Neurology.
Abstracts of Literature
Askiel Bruno and Alfredo M. Lopez-Yunez

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