Cerebral Aneurysms

AB-14613-00

Objective. MR angiography may be an appropriate tool to screen for unruptured intracranial aneurysms. Feasibility, test characteristics, and interobserver agreement in evaluation of MR angiograms were assessed by members of the MARS (Magnetic resonance Angiography in Relatives of patients with Subarachnoid hemorrhage) Study Group.

Subjects and methods. We screened 626 first-degree relatives of a consecutive series of 193 patients with subarachnoid hemorrhage examined at two institutions. We used MR imaging and MR angiography (three-dimensional time-of-flight imaging at both institutions and additional three-dimensional phase-contrast imaging at one institution). Three observers independently assessed the MR angiograms. Conventional angiography was performed in relatives with possible or definite aneurysms. Interobserver agreement in evaluation of MR angiograms was poor (k = 0.2; claustrophobia, n = 5). The positive predictive value of MR angiography was 100% (95% CI, 79–100%) for “definite” aneurysms and 58% (95% CI, 28–85%) for “possible” aneurysms. Sensitivity of MR angiography was estimated at 83% (95% CI, 65–94%) for aneurysms and 58% (95% CI, 28–85%) for “possible” aneurysms. Interobserver agreement in the evaluation of MR angiograms was poor (k = 0.3), probably because different diagnostic strategies used by individual observers resulted in different use of the assessment category “possible aneurysm.”

Conclusion. MR angiography is a feasible screening tool for detection of intracranial aneurysms. Positive predictive value, sensitivity, and specificity are acceptable when at least two neuroradiologists independently assess MR angiograms.

AB-14614-00

Background: The brain is rich in creatine kinase-BB isoenzyme activity (CK-BB), which is not normally present in cerebrospinal fluid (CSF). Results of previous studies have shown that CK-BB can be detected in the CSF of patients with aneurysmal subarachnoid hemorrhage (SAH), but whether CK-BB levels correlate with patients’ neurologic outcomes is unknown.

Objective: To evaluate the relationship between CSF CK-BB level and outcome after SAH.

Design: Prospective observational cohort.

Setting: University-affiliated tertiary care center.

Patients: Convenience sample of 30 patients seen for cerebral aneurysm clipping.

Interventions: We sampled and assayed CSF for CK isoenzymes a median of 3 days after SAH in 27 patients, and at the time of unruptured aneurysm clipping in 3 patients.

Main Outcome Measures: Without knowledge of CK results, we assigned the Glasgow Outcome Scale score early (≤1 week) and late (>2 months) after surgery.

Results: Higher CSF CK-BB levels were associated with higher Hunt and Hess grades at hospital admission (Spearman rank correlation, r = 0.69; P < .001), lower Glasgow Coma Scale scores at hospital admission (P = -0.72; P < .001), and worse early outcomes on the Glasgow Outcome Scale (r = -0.64; P < .001). For patients with a favorable early outcome (Glasgow Outcome Scale score, 3–5), all CK-BB levels were less than 40 U/L. With a cut-off value of 40 U/L, CK-BB had a sensitivity of 70% and a specificity of 100% for predicting unfavorable early outcome (Glasgow Outcome Scale score, 1–2). Having a CK-BB level greater than 40 U/L increased the chance of an unfavorable early outcome, from 33% (previous probability) to 100%, whereas a CK-BB level of 40 U/L or less decreased it to 13%. Similar findings were obtained when considering late outcomes.

Conclusion: The level of CSF CK-BB may help predict neurologic outcome after SAH.

AB-14615-00

OBJECTIVE: An intracranial aneurysm is an important acquired cerebrovascular disease that can cause a catastrophic subarachnoid hemorrhage. Despite modern therapy, most patients die or are left disabled as a direct result of a severe initial hemorrhage. The development of more effective treatment strategies depends on understanding the fundamental biology of cerebral aneurysms. The purpose of the present study is to determine whether inflammation or immunological reactions occurs in cerebral aneurysms.

METHODS: Aneurysm tissue was collected at the time of microsurgical repair from 23 unruptured and 2 ruptured aneurysms (25 patients) and compared with 11 control basilar arteries harvested at autopsy. Immunohistochemistry was used to localize complement (C3c, C9), immunoglobulins (IgG, IgM), vascular cell adhesion molecule-1, macrophages and monocytes (CD68), T lymphocytes (CD3), and B lymphocytes (CD20).

RESULTS: Complement (C3c, P < 0.0001; C9, P = 0.0017), immunoglobulin (IgG, P = 0.0013; IgM, P = 0.031), vascular cell adhesion molecule-1 (P = 0.0022), macrophages (CD68, P = 0.004), and T lymphocytes (CD3, P = 0.0004) were all frequently present in the wall of aneurysm tissue but were rarely identified in control basilar arteries. A few B lymphocytes (CD20, P = 0.41) were found in aneurysm tissue, but none were found in the basilar arteries.

CONCLUSION: Extensive inflammatory and immunological reactions are common in unruptured intracranial aneurysms and may be related to aneurysm formation and rupture.

AB-14616-00
OBJECTIVE: Associations among various factors and the occurrence of hydrocephalus after aneurysmal subarachnoid hemorrhage (SAH) were evaluated retrospectively in 897 patients enrolled in the North American study of triazolazid mesylate.

METHODS: Patients were assessed for hydrocephalus in a blinded fashion. Assessment of hydrocephalus was made on the basis of 3-month follow-up computed tomographic studies or, for those without a 3-month follow-up scan, on the basis of the latest computed tomographic studies obtained at least 10 days after SAH. Criteria indicating the occurrence of hydrocephalus were the presence of significantly enlarged temporal horns or prior placement of a ventricular shunt. Univariate analysis was performed to assess relationships among various factors and hydrocephalus. Factors statistically associated with the occurrence of hydrocephalus were analyzed further using logistic regression analysis.

RESULTS: Overall, 25.9% of the 897 patients developed hydrocephalus. Statistically significant associations among the following factors and hydrocephalus were observed (P value; risk coefficient): 1) severity of 3-month post-SAH Glasgow Outcome Scale (0.0001; 2.60); 2) increased ventricular size at admission (0.0001; 2.78); 3) neurological grade severity at admission (0.0274; 1.26); 4) preexisting hypertension (0.0284; 1.66); 5) alcoholism (0.0066; 2.30); 6) female sex (0.0056; 0.49); 7) increased aneurysm size (0.0239; 0.56); 8) pneumonia (0.0299; 1.78); 9) meningitis (0.0290; 5.86); and 10) intraventricular hemorrhage at admission (0.0414; 1.64).

CONCLUSION: Hydrocephalus seems to have a multifactorial etiology. Knowledge of risk factors related to the occurrence of hydrocephalus may help guide neurosurgeons in the long-term care of patients who have experienced aneurysmal SAH.

Clinical

AB-14617-00


In order to define the patterns of neurological involvement in Behcet’s disease and to assess prognostic factors, 558 files of the neuro-Behcet out-patient clinic were reviewed. Those patients without any evidence of objective neurological involvement as well as the patients with other possible explanations for the neurological picture, and cases not fulfilling the criteria for Behcet’s disease were excluded. The remaining 200 cases (155 male, 45 female) were evaluated: 162 had parenchymal CNS involvement (brainstem or ‘brainstem +’ involvement in 51%, spinal cord involvement in 14%, hemispheric involvement in 15% and isolated pyramidal signs in 19%) while 38 had secondary or non-parenchymal CNS involvement. In the first group the most common findings were pyramidal signs, hemiparesis, behavioural changes and sphincter disturbance, whereas in the second group the syndrome of raised intracranial pressure due to dural sinus thrombosis was the main clinical manifestation. In 60% of the cases with parenchymal involvement, CSF was hypercellular and/or had an elevated protein level, whereas in cases with non-parenchymal involvement the CSF was usually normal except for the elevated pressure. In more than half of the patients with parenchymal involvement, MRI showed brainstem and/or basal ganglion lesions. Forty-one per cent of the cases had a course with at least one attack and remission, another 28% also had attack(s) but showed secondary progression, 10% had primary progression and 21% had silent neurological involvement. Survival analysis was performed in patients who had at least a 3-year duration of neurological disease. Parenchymal involvement, elevated protein and/or pleocytosis in the CSF, ‘brainstem +’ type involvement, primary or secondary progressive course and relapse during steroid tapering were all associated with a poorer prognosis.

AB-14618-00

Stroke During Sleep: Epidemiological and Clinical Features—Bornstein NM (Stroke Unit, Dept of Neurology Tel-Aviv Sourasky Medical Center, 6 Weizmann St, Tel-Aviv 64239, Israel) Gur AY, Fainshtein P, Korczyn AD—Cerebrovasc Dis. 1999;9:320–322. Copyright © 1999 S. Karger AG, Basel.

Stroke during sleep is an unexplored area of vascular neurology and its pathogenesis; clinical significance and prevention still remain uncertain. The aim of our study was to determine the epidemiological and clinical patterns of ischemic stroke occurring during sleep. Consecutive patients (n=1822) with acute ischemic stroke recorded in the Tel Aviv Stroke Register were studied. Stroke during sleep was determined whenever focal neurological deficit was verified to have occurred while the patient had been asleep. The comparisons between patients with stroke during sleep and while awake were performed using the t test with Bonferroni correction and the χ² test for age, sex, vascular risk factors (i.e. ischemic heart disease, myocardial infarction, atrial fibrillation, arterial hypertension, hyperlipidemia, diabetes mellitus, peripheral vascular disease, smoking), vascular distribution (carotid versus vertebrobasilar) and severity of stroke (mild, moderate or severe). Data regarding the onset of stroke (during sleep or while awake) were available for 1,671 patients. A minority of strokes occurred during sleep (n=311, 18.6%), and stroke during sleep was severer (χ²=11.9, p<0.002). No significant differences were found in terms of age, sex and vascular distribution between the two groups. None of the vascular risk factors was found to be more frequent in stroke during sleep. Strokes occurring during sleep were found to be severer than those with onset while awake. However, no specific clinical patterns of risk factor profiles could be identified in these patients. Hemodynamic factors may play an important role in the occurrence of stroke during sleep, and this issue should be further investigated.

AB-14619-00


The reliability of the National Institutes of Health Stroke Scale (NIHSS) for use by trained neurologists in clinical trials of acute stroke has been established in several hospital-based studies. However, it also has the potential for application in community-based settings and to be used by nonneurologists: issues which have not been explored before. Hence, we aimed to determine the reliability of the NIHSS when administered by research nurses within the existing North Eastern Melbourne Stroke Incidence Study. Using the NIHSS, thirty-one consecutively registered stroke patients were assessed by 2 neurologists and 1 of 2 trained research nurses. The interrater reliability of observations was compared using weighted and unweighted kappa statistics and intraclass correlation coefficients (ICC). There was a high level of agreement for total scores between the 2 neurologists (ICC=0.95) and between each neurologist and research nurse (ICC=0.92 and 0.96). While there was moderate to excellent agreement among neurologists and research nurse (weighted κ=0.4) for the majority of the NIHSS items, there was poor agreement for the component ‘limb ataxia’. Overall, agreement between nurse and neurologist for individual items was not significantly different from agreement between neurologists. It appears that in both hospital and community settings, trained research nurses can administer the NIHSS with a reliability similar to stroke-trained neurologists. This ability could be used to advantage in large community-based trials and epidemiological studies.

AB-14620-00

Our aim was to determine if anticardiolipin antibodies are an independent risk factor for ischemic stroke and to determine their influence on stroke type and clinical outcome. We prospectively studied 194 consecutive patients with ischemic stroke admitted within 48 h of stroke. A control group consisted of 100, age and sex matched, healthy individuals. Neurological and functional status was assessed on admission, at 30 days, and at 1 year. IgG anticardiolipin antibodies were significantly more frequent in stroke patients (25.3%) than controls (6%, p<0.05). A multivariate analysis suggested that anticardiolipin antibodies are an independent risk factor for ischemic stroke in addition to hypertension and atrial fibrillation (RR = 2.94, p<0.05). Elevated IgG anticardiolipin antibodies were associated with cognitive impairment as measured by the Mini Mental State Examination at 30 days and at 1 year. IgG anticardiolipin antibodies did not correlate with stroke recurrence, or mortality at 30 days or 1 year.

**Epidemiology**

**AB-14621-00**


Although a number of studies suggest an association between stroke and depression, few have examined the relation between magnetic resonance imaging (MRI)-identified lesions and depression among community-dwelling older adults. This cross-sectional study sought to assess the association between MRI infarcts in the basal ganglia and non-basal-ganglia areas, potential functional consequences of these lesions, and depressive symptomatology in 3,371 US men and women aged 65 years or older who participated in the Cardiovascular Health Study between 1992 and 1994. By using multiple linear regression models, the authors found that after adjustment for age, gender, and stroke history, Center for Epidemiologic Studies Depression Scale scores were independently associated with non-basal-ganglia lesions (p=0.04) but were not independently associated with basal ganglia lesions (p=0.11). When measures of physical disability and cognitive impairment were added to the models, these measures displaced MRI-identified infarcts in their association with depressive symptoms. In additional models, hemispheric location and size of the basal ganglia lesion were found to have no relation to depression levels. These results suggest that the functional consequences of cerebrovascular disease may be the causal pathway by which basal ganglia and non-basal-ganglia lesions are associated with depressive symptomatology.

**AB-14622-00**


The atherogenicity of homocyst(e)ine—H(e)—emerged from many studies showing an association between moderately elevated levels and vascular occlusive disease. The aim of this study was to evaluate whether high homocyst(e)ine levels were associated with carotid atherosclerosis. Carotid atherosclerosis was defined as an intimal media thickness of internal and carotid bifurcation of at least 2 mm on the near and far walls as determined by B-mode ultrasonography. The study population included 91 patients: group 1 (61% males, mean age 64±10 years, 57% with history of hypertension) with ultrasound evidence of carotid atherosclerosis and 100 with normal carotid walls—group 2 (36% males, mean age 52±15 years, 27% with history of hypertension). Homocyst(e)ine levels (μmol/L) were determined by high-performance liquid chromatography with a fluorescent detector. Body mass index, dyslipidemia, smoking, diabetes, serum creatinine, plasma follic acid and vitamin B12 were not significantly different in the two groups. Homocyst(e)ine levels (μmol/L) were significantly higher in patients with carotid atherosclerosis than in those with normal arteries (11.7±6.5 μmol/L, 95% CI 10.4–13.1 vs 8.0±4.4 μmol/L, 95% CI 7.2–8.9, p<0.0001). By multiple regression analysis H(e) levels were positively correlated with male gender (p<0.02), age (p<0.001), and negatively with folate acid (p<0.0001). By logistic regression the independent predictors of carotid atherosclerosis were male gender (OR 2.65), hypertension (OR 2.55), age (≥10 years, OR 2.15) and H(e) levels (≥1 μmol/L, OR 1.11).

This study confirmed homocyst(e)ine is associated with carotid atherosclerosis. Consequently the authors recommend H(e) levels be screened in all patients at risk for atherosclerosis.

**AB-14623-00**


**Background**—Recent evidence from an animal model of stroke, the stroke-prone spontaneously hypertensive rat, implicated the gene encoding atrial natriuretic peptide (ANP) as a possible candidate contributing to the likelihood of experiencing a stroke. The purpose of the present study was to investigate the role of ANP in the pathogenesis of cerebrovascular accidents in humans.

**Methods and Results**—We investigated 2 previously known markers at ANP, G1837A, and T2238C, for their possible association with the occurrence of stroke. This was the largest matched case-controlled sample studied thus far: the sample was drawn from a large prospective study (the Physician’s Health Study). When assuming a dominant mode of inheritance, a statistically significant positive association was observed for the 1837A allele, indicating an odds ratio of 1.64 (95% confidence interval, 1.01 to 2.65) for stroke. This observation led to the discovery of a new molecular variant in exon 1, G664A, which was responsible for a valine-to-methionine substitution in the proANP peptide. This mutation, which was in linkage disequilibrium with the G1837A marker, was associated with the occurrence of stroke (odds ratio, 2.0; 95% confidence interval, 1.17 to 3.19; P=0.01).

**Conclusions**—Our findings suggest that molecular variants of the ANP gene may represent an independent risk factor for cerebrovascular accidents in humans. The strong parallelism to the experimental data obtained in the stroke-prone animal model provides assurance for the relevance of our observation.

**AB-14624-00**


Patients with a lacunar stroke syndrome may have cortical infarcts on brain imaging rather than lacunar infarcts, and patients with the clinical features of a small cortical stroke (partial anterior circulation syndrome, PACS) may have lacunar infarcts on imaging. The aim was to compare risk factors and outcome in lacunar syndrome (LACS) with cortical infarct, LACS with lacunar infarct, PACS with cortical infarct, and PACS with lacunar infarct to determine whether the clinical syndrome should be modified according to brain imaging.

As part of a hospital stroke registry, patients with their first ever stroke from 1989 to 1998 were ascertained by stroke physicians who assigned a clinical classification using clinical features only. A neuroradiologist classified recent clinically relevant infarcts on brain imaging as cortical, posterior cerebral artery territory or lacunar.
infarct volume, neurological severity and PAF binding to platelets of patients with acute cerebral ischemic stroke—Adusnky A (dept of geriatric medicine, sheba medical center, tel hashomer, israel 52621), hershkowitz m, atar e, bakoun m, poreh a—neurol. res. 1999;21:645–648.

Ab-14625-00

infarct volume, neurological severity and PAF binding to platelets of patients with acute cerebral ischemic stroke—Adusnky A (dept of geriatric medicine, sheba medical center, tel hashomer, israel 52621), hershkowitz m, atar e, bakoun m, poreh a—neurol. res. 1999;21:645–648.

Studies show that platelet activating factor (PAF) is involved in the cerebrovascular response to ischemia, and that its binding to platelets may change in stroke victims. The purpose of this study was to determine whether binding of PAF to platelets of stroke patients could serve as an index for determining the volume of ischemic strokes and severity of neurological presentation. Thirteen stroke patients and 21 healthy controls were studied. The neurological severity of these stroke patients was evaluated by the Scandinavian stroke scale. Infarct volume was assessed by planimetric measures of brain CT. PAF binding to platelets was determined by use of radiolabelled PAF. (3H)PAF binding to platelets of stroke patients was lower than in controls ((149.58 ±46.11 and 50.03), p<0.01). platelet thrombus formation correlated with weight (r=0.45, p=0.04) and HDL cholesterol (r=0.41, p=0.01). In multiple regression analysis only HDL cholesterol showed significant correlation with platelet thrombus formation (p=0.03). Platelet aggregation and circulating prothrombotic factors did not correlate with platelet thrombus formation. A comparison between normal and hypercholesterolemic subjects revealed enhanced thrombus area (0.026±0.20 vs 0.045±0.039 mm²; p=0.04), resting CD62 expression (6±7% vs 15±10% positive platelets, p=0.02), and platelet aggregation (16.7±5.2 vs 21.7±6.7 ohms, p=0.04) in hypercholesterolemic subjects. Our results demonstrate that HDL cholesterol is a significant independent predictor of ex vivo platelet thrombus formation.

AB-14627-00

biphasic opening of the blood-brain barrier following transient focal ischemia: effects of hypothermia—huang zg, xue d, preston e, Karbalai h, buchan am (alberta stroke program, office of stroke research, rm 1162, foothills hospital, 1403 29th st nw, calgary, ab, canada T2N 2T9)—can j neuro sci. 1999;26:298–304.

Objective: Tracer constants (Ks) for blood-to-brain diffusion of sucrose were measured in the rat to profile the time course of brain-blood barrier injury after temporary focal ischemia, and to determine the influence of post-ischemic hypothermia.

Methods: Spontaneously hypertensive rats were subjected to transient (2 hours) clp occlusion of the right middle cerebral artery. Reperfusion times ranged from 1.5 min to 46 hours, and i.v. 3H-sucrose was circulated for 30 min prior to each time point (1h, 4h, 22h, and 46h: n=5–7 per time point). Ks was calculated from the ratio of parenchymal tracer uptake and the time-integrated plasma concentration. Additional groups of rats (n=7–8) were maintained either normothermic (37.5°C) or hypothermic (32.5°C or 28.5°C) for the first 6 hours of reperfusion, and Ks was measured at 46 hours.

Results: Rats injected after 1.5–2 min exhibited a 10-fold increase in Ks for cortical regions supplied by the right middle cerebral artery (p<0.01). This barrier opening had closed within 1 to 4 hours post-reperfusion. By 22 hours, the blood-brain barrier had re-opened, with further opening 22 and 46 hours (p<0.01), resulting in edema. Whole body hypothermia (28°C–29°C) during the first six hours of reperfusion prevented opening, reducing Ks by over 50% (p<0.05).

Conclusions: Transient middle cerebral artery occlusion evokes a marked biphasic opening of the cortical blood-brain barrier, the second phase of which causes vasogenic edema. Hypothermic treatment reduced infarct volume and the late opening of the blood-brain barrier. This opening of the blood-brain barrier may enhance delivery of low permeability neuroprotective agents.

AB-14628-00


Object: investigation into a potential treatment for the acute period following onset of spontaneous subarachnoid hemorrhage (SAH) is hampered by the lack of a standardized experimental model. For that purpose the authors elaborated on a small-animal model in which computer-controlled intracranial blood infusion is used and investigated whether this model can reliably reproduce acute neuronal injury after SAH.

Methods. Whole autologous blood (blood-infused group) or isotonic saline (control group) was infused into the cisterna magna or olfactory cistern of rats. The infusions decreased exponentially during a 5-minute period. Throughout the infusion period, intracranial pressure (ICP) was monitored. Neuronal injury was quantified by observing tissue immunoreactivity to a 70-kDa heat shock protein (HSP70) and comparing this with the tissue’s reaction to hematoxylin and cosin staining. On Days 1, 3, and 5, the CA1, CA3, and dentate gyrus regions of the hippocampus were analyzed, respectively.

During saline infusion ICP increased within seconds beyond 80 mm Hg and afterward decreased in accordance with the infusion rate. During the infusion of blood, the same initial pressure peak was found,
but the ICP remained increased beyond this pressure level throughout the 5-minute infusion period. The HSP70 immunoreactivity in the saline-infused group was found only on Day 1 in the CA1 region and the dentate gyrus, but not in the CA3. After injection of whole blood, there was HSP70-positive staining in the CA1, CA3, and dentate gyrus regions throughout the observation period.

Conclusions. The controlled cisternal infusion of blood caused neuronal injury that resembled that of previous experimental models that produce SAH by rupture of intracranial vessels with endovascular techniques. Unlike those experiments, the intracisternal infusion technique presented by the authors provides more standardized bleeding with regard to ICP, the volume of subarachnoid blood, and the extent of acute cellular injury.

Imaging

AB-14629-00

Background: Duplex ultrasonography and magnetic resonance angiography (MRA) are becoming competitive alternatives to angiography for determining the degree of internal carotid artery (ICA) stenosis. Varying reports have been published regarding the suitability of each technique for grading ICA disease. This retrospective study compared the merits of these three modalities for measuring ICA stenosis.

Methods: One hundred and eleven patients being considered for carotid endarterectomy underwent intra-arterial digital subtraction angiography (DSA) via arch injection. Duplex imaging was performed in all patients and MRA in 50. The degree of carotid stenosis estimated by the three modalities was compared.

Results: There was good correlation between subjectively graded MRA and DSA images (r=0.87, P<0.001, n=82 carotids) but poor correlation for objective estimates. MRA tended to underestimate the degree of stenosis (bias −4.5% per cent) compared with DSA, but showed good correlation with duplex ultrasonography estimates (r=0.86, P<0.001, n=87 carotids). Both non-invasive modalities produced high values of sensitivity and specificity in estimating stenoses of greater than 70 per cent. MRA was less sensitive for distinguishing between severe stenosis and complete occlusion.

Conclusion: This study did not resolve the debate regarding the method of choice as both MRA and duplex ultrasonography were accurate for imaging carotid stenoses.

AB-14630-00

Background: Cerebral angiography is associated with a small but definite risk of neurological complications with an unknown incidence of clinically silent embolism. We assessed the neurological complication rate compared with the frequency of silent embolism after angiography.

Methods: We used diffusion-weighted magnetic resonance imaging (MRI) before and after angiography to assess embolic events. 100 consecutive angiographies (66 diagnostic and 34 interventional procedures) were done on 91 patients. Patients underwent neurological assessment before, immediately after, and 1 day after angiography.

Findings: Before angiography, no abnormalities were seen on diffusion-weighted MRI. Diffusion-weighted MRI showed 42 bright lesions in 23 patients after 23 procedures (17 diagnostic, six interventional) in a pattern consistent with embolic events. There was no new neurological deficit after any angiographic procedure. After diagnostic angiography in patients with a history of vasculopathy, the frequency of lesions was significantly higher than in patients without vascular risk factors (12 [44%] of 27 vs five [13%] of 39 patients, P=0.03). In diagnostic angiography, the appearance of lesions was significantly correlated with whether vessels were difficult to probe (P=0.01), amount of contrast medium needed (P<0.01), fluoroscopy time (P<0.01), and use of additional catheters (P=0.02).

Interpretation: After diagnostic and interventional cerebral angiography, embolic events are more frequent than the apparent neurological complication rate. In diagnostic procedures, the incidence of embolism is closely related to a vascular risk profile.

Neurosonology

AB-14631-00

The localization of atherosclerotic lesions is influenced by hemodynamic factors, namely, shear stress and tensive forces. The present study investigated the relationships between shear stress and circumferential wall tension and between these hemodynamic factors and the intima-media thickness (IMT) of the common carotid artery in healthy men. Fifty-eight subjects were studied. Shear stress was calculated as blood viscosity × blood velocity/inner diameter. Circumferential wall tension was calculated as blood pressure × inner radius. Blood velocity, inner diameter, and IMT were measured by high-resolution echo-Doppler. Mean shear stress was 12.6±3.3 dynes/cm² (mean±SD; range, 4.8 to 20.4) and was inversely related with age, blood pressure, and body mass index (BMI). Mean circumferential wall tension was 3.4±0.6×10⁴ dynes/cm (range 2.4 to 5.6) and was directly associated with age and BMI. IMT was inversely associated with shear stress (r=0.55, P<0.0001) and directly associated with circumferential wall tension (r=0.43, P<0.0001). Shear stress and circumferential wall tension were inversely correlated (r=0.66, P<0.0001). In multiple regression analysis, shear stress and (marginally) cholesterol were independently associated with IMT, whereas circumferential wall tension, age, and BMI were not. These findings confirm that common carotid shear stress varies among healthy individuals and decreases as age, blood pressure, and BMI increase. Our findings also demonstrate that circumferential wall tension is directly associated with wall thickness, age, and BMI and that shear stress is associated with common carotid IMT independent of other hemodynamic, clinical, or biochemical factors.

AB-14632-00

There is evidence suggesting that among other factors, an alteration in cerebral hemodynamics plays a relevant role in the occurrence of strokes in patients with carotid disease. The purpose of this study was to investigate patterns of cerebrovascular reactivity in patients with internal carotid occlusion and severe contralateral carotid stenosis and their relationship with symptomatology. Using transcranial Doppler ultrasound, cerebrovascular reactivity to hypercapnia in middle cerebral arteries was evaluated with the breath-holding index (BHI) in 42 patients with internal carotid occlusion and severe contralateral carotid stenosis and in 40 control subjects. A significant decrease of BHI on the occluded side was observed in symptomatic patients with respect to asymptomatic ones (0.12±0.1 vs. 0.75±0.4, P<0.0001) and with respect to the control
group (1.11 ± 0.1, P < 0.0001). The difference was also significant between asymptomatic patients and controls (P < 0.0001). Breath-holding values on the stenotic side were significantly higher (P < 0.0001) in asymptomatic patients (1.01 ± 0.2) with respect to symptomatic ones (0.39 ± 0.1). A significant difference (P < 0.0001) was also present between controls and symptomatic patients. The pattern of cerebrovascular reactivity in patients with severe bilateral carotid steno-occlusive disease seems to be strictly dependent on the presence of previous symptoms. Further studies are needed to investigate whether the study of cerebral hemodynamics in patients with bilateral carotid artery disease is important for planning therapeutic strategies.

AB-14633-00


Object. The effect of increased intracranial pressure (ICP) on cerebral venous blood flow has been the subject of very few clinical and experimental studies. The authors assessed the usefulness of venous transcranial Doppler (TCD) ultrasonography as a noninvasive monitoring tool for predicting raised ICP.

Methods. Serial venous TCD studies of the basal vein of Rosenthal and the straight sinus (SS) were prospectively performed in 30 control volunteers and 25 patients with raised ICP. Correlations with ICP data were calculated using a multivariate regression model. Venous blood flow velocities (BFVs) in the basal vein of Rosenthal showed, within a certain range, a linear relationship between mean ICP and maximal venous BFV (r = 0.645; P < 0.002). Moreover, a linear relationship was found for maximal venous BFVs in the SS and mean ICP (r = 0.928; P < 0.0003).

Conclusions. Venous TCD studies may provide an additional noninvasive monitoring tool for raised ICP and give further insights into the cerebral venous hemodynamics present during raised ICP.

Pharmacology / Therapeutics

AB-14634-00


AB-14635-00


BACKGROUND AND PURPOSE: Advances in thrombolytic therapy, brain imaging, and neurointerventional techniques provide new therapeutic options for acute stroke. Intra-arterial thrombolysis has proved to be a potent therapeutic tool. To show that this procedure can be performed in community hospitals, we describe our experience with a group of 11 patients treated for middle cerebral artery occlusions.

METHODS: Twenty-two patients seen during a period of 1 year with clinical findings of acute major-vessel stroke met screening criteria and were evaluated under an institutional review board-approved protocol. After CT scanning, 17 of those patients met strict criteria, gave informed consent, and underwent angiography. Eleven patients had M1 and M2 middle cerebral artery occlusions and received local thrombolytic therapy with urokinase. Recanalization efficacy, complications, and outcome data were compiled.

RESULTS: The average score on the National Institutes of Health Stroke Scale was 22.2 at the onset of treatment and 12.5 after therapy, with 91% of patients showing neurologic improvement. Complete (TIMI 3) recanalization occurred in 73% of cases and partial recanalization (TIMI 2) in 18%. At the 90-day follow-up evaluation, 56% of patients had good outcomes (modified Rankin score, 0 to 1). One intracranial hemorrhage occurred.

CONCLUSION: Intra-arterial thrombolysis can be performed in a community hospital by radiologists with interventional and neuroradiologic skills given appropriate institutional preparation.

AB-14636-00

Use of Factor IX Complex in Warfarin-Related Intracranial Hemorrhage—Boulis NM (Rm 2128, Box 0338, 1500 E Medical Center Dr, Ann Arbor, MI 48109), Bobek MP, Schmaier A, Hoff JT—Neurosurgery. 1999;45:1113–1119.

OBJECTIVE: Anticoagulation-treated patients presenting with intracranial hemorrhage, including subdural hematoma, epidural hematoma, subarachnoid hemorrhage, and intracerebral hemorrhage, require urgent correction of their coagulopathy to prevent worsening hemorrhage and to facilitate surgical intervention when necessary. In this study, we compared the use of fresh frozen plasma (FFP) with that of Factor IX complex concentrate (FIXCC) to achieve rapid correction of warfarin anticoagulation.

METHODS: Patients admitted to a tertiary care center with computed tomography-proven intracranial hemorrhage and a prothrombin time of more than 17 seconds were considered for inclusion in the study protocol. Complete data sets were obtained for eight patients randomized to treatment with FFP and five patients randomized to treatment with FIXCC. The prothrombin time and International Normalized Ratio were measured every 2 hours for 14 hours. Correction of anticoagulation was defined as an International Normalized Ratio of ≤ 1.3.

RESULTS: A difference in repeated International Normalized Ratio measurements during the first 6 hours of correction was observed between the FIXCC and FFP groups (P < 0.03). The rate of correction was greater (P < 0.01) and the time to correction was shorter (P < 0.01) for the FIXCC-treated group. No difference in neurological outcomes was detected between groups, but a higher complication rate was observed for the FFP-treated group.

CONCLUSION: The use of FIXCC accelerated correction of warfarin-related anticoagulation in the presence of intracranial hemorrhage.

Surgery

AB-14637-00

Surgical Patent Foramen Ovale Closure for Prevention of Paradoxical Embolism-Related Cerebrovascular Ischemic Events—Dearani JA...
Background—The role of surgical closure of patent foramen ovale (PFO) for cerebral infarction (CI) or transient ischemic attack (TIA) resulting from paradoxical embolism is unclear, and its effect on recurrence is unknown. Our objective was to determine the outcome of surgical closure of PFO in patients with a prior ischemic neurological event, define the rate of CI or TIA recurrence after PFO closure, and identify risk factors for these recurrences.

Methods and Results—We retrospectively analyzed 91 patients (58 men, 33 women) with ≥1 prior cerebrovascular ischemic event who underwent surgical PFO closure between April 1982 and March 1998. The presence of a PFO with a right-to-left shunt was confirmed with transesophageal echocardiography. Mean age was 44.2 ± 12.2 years. The index event was a CI in 59 and a TIA in 32; a Valsalva-like episode preceded the event in 15 patients. Deep venous thrombosis was documented in 9 patients, and a hypercoagulable state was identified in 10. Surgical closure was performed with transcatheter occlusion by either direct suture (n = 82) or patch closure (n = 9). Limited incisions were used in 18.7% of patients. There was no operative mortality. Morbidity included transient atrial fibrillation (n = 11), pericardial drainage for effusion (n = 4), exploration for bleeding (n = 3), and superficial wound infection (n = 1). Follow-up totaled 176.3 patient-years, and mean follow-up was 2.0 years. No one had a CI, and 8 had a TIA during follow-up, with 1 caused by temporal arteritis. Transesophageal echocardiography demonstrated all closures to be intact in these patients. The overall freedom from TIA recurrence during follow-up was 92.5 ± 3.2% at 1 year and 83.4 ± 6.0% at 4 years. Having multiple neurological events before PFO closure was the only significant risk factor for TIA or CI recurrence after closure by univariate analysis (P = 0.05); the small number of post-PFO closure cerebral ischemic events precluded multivariate analysis.

Conclusions—Surgical closure of PFO can be performed with minimal morbidity and mortality. PFO closure may decrease the risk of recurrent stroke or TIA and may avoid lifelong anticoagulation in the young adult if there is no other indication. Recurrent cerebrovascular ischemic events after surgery should prompt further evaluation to identify causes other than paradoxical embolism.

Items of Interest


Epidemiology and Significance of Atrial Fibrillation—Ryder KM, Benjamin EJ (The Framingham Heart Study, 5 Thuber St, Framingham, MA 01702-6334)—Am J Cardiol. 1999;84:131R–138R. Copyright © 1999 by Excerpta Medica, Inc.


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Unilateral Focal Lesions in the Rostral Medulla Influence Chemosensitivity and Breathing Measured During Wakefulness, Sleep, and Exercise—Morrell MJ (National Heart and Lung Institute, Imperial College School of Medicine, Royal Brompton Hospital, Sleep and Ventilation Unit, Sydney St, London SW3 6NP, UK), Heywood P, Moosavi SH, Guz A, Stevens J—J Neurol Neurosurg Psychiatry. 1999;67:637–645.


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Alcohol, Stroke and Coronary Heart Disease: Are There Anti-Oxidants and Pro-Oxidants in Alcoholic Beverages That Might Influence the Development of Atherosclerotic Cardiovascular Disease?—Puddey IB (Dept of Medicine and Western Australian Heart Research Institute, Univ of Western Australia, Royal Perth Hospital, PO Box X2213, Perth, Western Australia 6000), Croft KD—Neuroepidemiology. 1999;18:292–302. Copyright © 1999 S. Karger AG, Basel.


NIH Announces Web-Based Audioteleconference on Advances in Prevention, Acute Management, and Rehabilitation of Stroke

The National Institutes of Health (NIH) Clinical Center and National Institute of Neurological Disorders and Stroke (NINDS) announce that a web-based continuing medical education program for physicians, Advances in the Prevention, Acute Management, and Rehabilitation of Stroke, will take place on Tuesday, March 14, 2000, from 8:00 PM until 10:00 PM EST. Physicians will receive 2 CMEs for their participation in the program.

Participants will listen to a presentation by NIH researchers via a premier telephone service while simultaneously viewing support materials on their own computers. They will also have the opportunity to interact directly with the presenting researchers during telephone question and answer periods. The objective of the program is to offer participants information about the most current treatment strategies for prevention and acute management of stroke and to provide them with information about promising advances in stroke rehabilitation. The faculty includes Thomas J. DeGraba, MD, Head, Clinical Stroke Research Unit, Stroke Branch, NINDS; Steven Warach, MD, PhD, Chief, Section of Stroke Diagnostics and Therapeutics, Stroke Branch, NINDS; and Leonardo G. Cohen, MD, Chief, Human Cortical Physiology Section, Medical Neurology Branch, NINDS.

The site for program registration is www.frontlinemeded.org and the deadline is March 7, 2000. Participants should have access to an Internet connection with Web browser and the capacity of being on the Internet and telephone at the same time. Participants will also need Adobe Acrobat Reader. It can be downloaded free of charge (see instructions at the registration site listed above). The program is fully supported by the NIH Clinical Center. There is no charge for participation, but registration is limited.
Abstracts of Literature
Askiel Bruno and Engin Y. Yilmaz

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