Cerebral Aneurysms

Ab-14403-99


Object. Previous reports on the results of treatment for aneurysmal subarachnoid hemorrhage (SAH) have been based only on activities of daily living after discharge, whereas resumption of work has received insufficient attention. Most Japanese work under a lifetime employment system, and it is best for those who have recovered from SAH to return to work for their previous employer. The present study was conducted to determine the extent to which discharged patients who have suffered an SAH resume their former occupations in Japan, focusing on those between 40 and 49 years of age, who usually have a strong desire to return to work.

Methods. The participants consisted of 193 patients with SAH. Based on the results of telephone interviews or written questionnaires, their work status at 1 year after onset was analyzed.

The work resumption rates for patients with Hunt and Kosnik neurological Grades 1 or 2 on admission were higher than for those with Grades 3 or 4 (p=0.015) and lower for patients with basilar artery aneurysms than for those with aneurysms at other sites (p=0.028). With regard to premorbid occupation, the work resumption rates were high (80%) for professionals and engineers, many of whom were public servants, or teachers at junior or senior high schools. The resumption rates were also high for primary industry workers (80%), but lowest (20%) for professional drivers (p=0.04–0.001). The work resumption rate was lower for women than for men (p=0.01).

Conclusions. These findings indicate that resumption of work is determined not only by medical factors, but also by social factors including gender, type of occupation, employment system, and socioeconomic background.

Ab-14404-99


OBJECTIVE: The goal of this study was to document the influence of the treatment method (early surgery versus early endovascular treatment) on the development of chronic shunt-dependent hydrocephalus in a series of 242 patients treated within 7 days after aneurysmal subarachnoid hemorrhage (SAH).

METHODS: The following parameters were prospectively recorded in a computerized database and retrospectively analyzed for association with chronic shunt-dependent hydrocephalus: 1) Hunt and Hess grade, 2) Fisher computed tomographic grade, 3) incidence of repeat SAH, 4) aneurysm location, and 5) treatment method (early surgery versus early endovascular treatment).

RESULTS: Forty of 187 patients (21.4%) who survived the SAH and its neurological and/or medical sequelae underwent definitive shunting for treatment of chronic hydrocephalus. The rate of shunt dependency was positively correlated with a higher Hunt and Hess grade (P<0.001), a higher Fisher computed tomographic grade (P=0.003), the occurrence of intraventricular hemorrhage (P<0.001), repeat SAH (P<0.003), and aneurysms arising at the anterior communicating artery (P<0.001).

CONCLUSION: The results of the present study indicate that the treatment method used does not affect the risk of the later development of chronic shunt-dependent hydrocephalus (early surgery, 23.2% [29 of 125]; early endovascular treatment, 17.7% [11 of 62]; P=0.45).

Clinical

Ab-14405-99

Migraine and Stroke in Young Women: Case-Control Study—Chang CL, Donaghy M, Poulter N (Cardiovascular Studies Unit, Dept of Clinical Pharmacology, Imperial College School of Medicine, London W2 1PG, England), and World Health Organisation Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception—BMJ. 1999;318:13–18.

Objective To investigate the association between migraine and ischemic or haemorrhagic stroke in young women.

Design Hospital based case-control study.

Setting Five European centres participating in the World Health Organisation Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception.

Subjects 291 women aged 20–44 years with ischaemic, haemorrhagic, or unclassified arterial stroke compared with 736 age and hospital matched controls.

Intervention Questionnaire.

Main outcome measure Self reported history of headaches.

Results Adjusted odds ratios associated with a personal history of migraine were 1.78 (95% confidence intervals, 1.14 to 2.77), 3.54 (1.30 to 9.61), and 1.10 (0.63 to 1.94) for all stroke, ischaemic stroke, and haemorrhagic stroke respectively. Odds ratios for ischaemic stroke were similar for classical migraine (with aura) (3.81, 1.26 to 11.5) and simple migraine (without aura) (2.97, 0.66 to 13.5). A family history of migraine, irrespective of personal history, was also associated with increased odds ratios, not only for ischaemic stroke but also haemorrhagic stroke. In migraineous women, coexistence of oral contraceptives or a history of high blood pressure or smoking had greater than multiplicative effects on the odds ratios for ischaemic stroke associated with migraine alone. Change in the frequency or type of migraine on using oral contraceptives did not predict subsequent stroke. Between 20% and 40% of strokes in women with migraine seemed to develop directly from a migraine attack.

Conclusions Migraine in women of childbearing age significantly increases the risk of ischaemic but not haemorrhagic stroke. The coexistence of oral contraceptive use, high blood pressure, or smoking seems to exert a greater than multiplicative effect on the risk of ischaemic stroke associated with migraine.

Ab-14406-99


There are limited data, mainly clinical and radiological, on small centrum oval infarcts (SCOIs). From a consecutive series of 159 autopsy brains we identified 12 cases which on gross pathological examination harboured a total of 21 SCOIs. In the majority of lesions histology revealed a significant component of incompletely infarcted brain. Clinicopathological data suggested that the underlying mechanism was likely to have been cardioembolic in 3 cases, and possibly embolic from heart or aortic arch in a further 5. Two cases were due to ipsilateral
carotid artery atheroma (i.e. 10 of 12 cases had possible embolic sources). The majority of lesions appeared to lie in arterial borderzones. The combined data suggest that SCOIs are pathologically and pathogenetically heterogeneous, and therefore that the term ‘lacune’ is inappropriate because this implies intrinsic small vessel disease as the underlying cause. Clinically, potentially treatable cardiac and large vessel pathology should be excluded.

AB-14407-99

Background—Exposure to risk factors such as hypertension or hypercholesterolemia decreases the bioavailability of endothelium-derived nitric oxide (NO) and impairs endothelium-dependent vasodilation. Recently, a circulating endogenous NO synthase inhibitor, asymmetric dimethylarginine (ADMA), has been detected in human plasma. The purpose of this study was to examine the relationship between plasma ADMA and atherosclerosis in humans.

Methods and Results—Subjects (n = 116; age, 52 ± 1 years; male:female ratio, 100:16) underwent a complete history and physical examination, determination of serum chemistries and ADMA levels, and duplex scanning of the carotid arteries. These individuals had no symptoms of coronary or peripheral artery disease and were taking no medications. Univariate and multivariate analyses revealed that plasma levels of ADMA were positively correlated with age (P < 0.0001), mean arterial pressure (P < 0.0001), and Σ glucose (an index of glucose tolerance) (P = 0.0006). Most intriguingly, stepwise regression analysis revealed that plasma ADMA levels were significantly correlated to the intima-media thickness of the carotid artery (as measured by high-resolution ultrasonography).

Conclusions—This study reveals that plasma ADMA levels are positively correlated with risk factors for atherosclerosis. Furthermore, plasma ADMA level is significantly correlated with carotid intima-media thickness. Our results suggest that this endogenous antagonist of NO synthase may be a marker of atherosclerosis.

AB-14408-99

OBJECTIVE—To test the hypothesis that genetic susceptibility to diabetic nephropathy is associated with an increased familial risk of vascular disease, we have examined the causes and rates of death of parents of individuals with at least 1 type 1 diabetes complicated by diabetic nephropathy compared with the causes and rates of death of parents of control subjects with diabetes uncomplicated by nephropathy.

RESEARCH DESIGN AND METHODS—Individuals with at least a 14-year duration of type 1 diabetes complicated by diabetic nephropathy were identified and matched for age, sex, and duration of diabetes to control subjects. A total of 118 parents and 118 matched control subjects were identified and approached to obtain information on parental age and cause of death. For parents who had died, the cause of death was ascertained from the death certificate.

RESULTS—Kaplan-Meier curves showed that parents of subjects with nephropathy (PN) had reduced survival compared with parents of diabetic subjects without nephropathy (PC) (log rank test P < 0.05). There was an excess of all vascular deaths and, in particular, strokes in the parents of subjects with nephropathy (PN: 20 of 103 deaths, 19% vs. PC: 3 of 66 deaths, 4%; Fisher’s exact test P < 0.01).

CONCLUSIONS—Parents of diabetic patients with nephropathy have reduced survival. This seems to be largely explained by an increase in vascular deaths and, in particular, a four-fold increase in the number of strokes. This supports the hypothesis that a common hereditary risk factor predisposes to both vascular death and diabetic renal disease.

Epidemiology
AB-14409-99

Background—A single base pair mutation in the prothrombin gene has recently been identified that is associated with increased prothrombin levels. Whether this mutation increases the risks of arterial and venous thrombosis among healthy individuals is controversial.

Methods and Results—In a prospective cohort of 14 916 men, we determined the prevalence of the G20210A prothrombin gene variant in 833 men who subsequently developed myocardial infarction, stroke, or venous thrombosis (cases) and in 1774 age- and smoking status-matched men who remained free of thrombosis during a 10-year follow-up (control subjects). Gene sequencing was used to confirm mutation status in a subgroup of participants. Overall, carrier rates for the G20210A mutation were similar among case and control subjects; the relative risk of developing any thrombotic event in association with the 20210A allele was 1.05 (95% CI, 0.7 to 1.6; P = 0.8). We observed no evidence of association between mutation and myocardial infarction (RR = 0.9, P = 0.4) or stroke (RR = 1.1, P = 0.8). For venous thrombosis, a modest nonsignificant increase in risk was observed (RR = 1.7, P = 0.08) that was smaller in magnitude than that associated with factor V Leiden (RR = 3.0, P < 0.001). Nine individuals carried both the prothrombin mutation and factor V Leiden (5 controls and 4 cases). One individual, a control subject, was homozygous for the prothrombin mutation.

Conclusions—In a large cohort of US men, the G20210A prothrombin gene variant was not associated with increased risk of myocardial infarction or stroke. For venous thrombosis, risk estimates associated with the G20210A mutation were smaller in magnitude than risk estimates associated with factor V Leiden.

AB-14410-99

Objective: To identify possible contributors to the seasonal variation in stroke mortality. Background: Stroke and respiratory disease mortality rates were calculated from vital statistics and census data for the United States from 1938 to 1988. State-specific average temperatures by month were derived from data obtained from the National Climatic Data Center for the United States from 1938 to 1988. Methods: Each time series was decomposed into a trend, a seasonal effect, and a residual effect. Cross-correlation was used to assess the relationship between the residual time series. Results: There is a strong and consistent seasonal pattern of high stroke and respiratory disease mortality in the colder winter months. Stroke mortality was significantly and independently both positively associated with respiratory disease mortality and inversely associated with temperature. The sharp initial increases in both respiratory disease and stroke mortality in the late fall and early winter are synchronous, and the amplitudes are strongly associated, except for a saturation effect with extreme respiratory disease amplitudes. Conclusions: Seasonal change in stroke mortality is associated with seasonal variation in both respiratory disease and temperature. Respiratory disease and temperature may influence stroke mortality non spuriously by affecting stroke case fatality, incidence, or both.
Experimental Pathology

AB-14411-99


Background. Prolonged hypothermic circulatory arrest (HCA) causes neurologic injury. However, the mechanism of this injury is unknown. We hypothesized that HCA causes nitric oxide production to result in neuronal necrosis. This study was undertaken to determine whether the neuronal nitric oxide synthase inhibitor 17477AR reduces necrosis after HCA.

Methods. Thirty-two dogs underwent 2 hours of HCA at 18°C. Nitric oxide synthase catalytic assay and intracerebral microdialysis for nitric oxide production were performed in acute nonsurvival experiments (n=16). Sixteen animals survived for 72 hours after HCA: Group 1 (n=9) was treated with 17477AR (Astra Arcus), and Group 2 (n=7) received vehicle only. Animals were scored from 0 (normal) to 5 (coma) for neurologic function and from 0 (normal) to 100 (severe) for neuronal necrosis.

Results. Administration of 17477AR reduced nitric oxide production in the striatum by 94% (HCA alone), 3.65±2.42 μmol/L; HCA and 17477AR, 0.20±0.14 μmol/L (citrine). Dogs treated with 17477AR after HCA had superior neurologic function (62.22±29.82 for group 1 versus 141.86±61.53 for group 2, p=0.019) and significantly reduced neuronal necrosis (9.33±4.67 for group 1 versus 38.14±2.23 for group 2, p<0.00001) compared with untreated HCA dogs.

Conclusions. Our results provide evidence that neuronal nitric oxide synthase mediates neuronal necrosis after HCA and plays a significant role in HCA-induced neurotoxicity. Pharmacologic strategies to inhibit neuronal nitric oxide synthase after the ischemic period of HCA may be clinically beneficial.

AB-14412-99

Pretreatment With Intravenous FGF-13 Reduces Infarct Volume and Ameliorates Neurological Deficits Following Focal Cerebral Ischemia in Rats—Yao DL (Dept of Pharmacology, Human Genome Sciences, 9410 Key West Ave, Rockville, MD 20850), Masonic K, Petullo D, Li YL, Lincoln C, Wibberley L, Alderson RF, Antonaccio M—Brain Res. 1999;818:140–146. Copyright © 1999 Elsevier Science B.V.

Fibroblast growth factor-13 (FGF-13), novel member of FGF family has recently been molecularly cloned as a result of high throughput sequencing of an ovarian cancer cell, hippocampal, and kidney cDNA libraries. The human gene encodes for a protein with a molecular weight of 22 kDa that is most homologous to FGF-8 (70% similarity). In the current study, we tested the effects of intravenously administered FGF-13 in a model of permanent focal cerebral ischemia in Sprague–Dawley rats. FGF-13 or the vehicle was administered systematically via the tail vein 30 min prior, and 30 min and 24 h after the occlusion of the left middle cerebral artery (MCAo). Animals were weighed and evaluated behaviorally prior to and at 24 and 48 h after MCAo. The volume of cerebral infarct and swelling were determined using an image analysis system (BioQuant) and cresyl violet stained sequential sections from the forebrain region. Histopathology was evaluated to compare the therapeutic effects. We found a 63% reduction in infarct volume in FGF-13- vs. vehicle-treated animals (infarct volume was 21.9±3.8% in vehicle- and 8.1±1.6% in FGF-13-treated rats, p=0.0016) and a moderate inhibition of brain swelling by FGF-13. These results support an important role for NO in ischemic neurotoxicity and indicate that neuronal NOS inhibition may be clinically beneficial.

AB-14414-99


Object. Ultra-early hematoma evacuation (<4 hours) after intracerebral hemorrhage (ICH) may reduce mass effect and edema development and improve outcome. To test this hypothesis, the authors induced lobar hematomas in pigs.

Methods. The authors infused 2.5 ml of blood into the frontal cerebral white matter in pigs weighing 8 to 10 kg. In the treatment group, clots were lysed with tissue plasminogen activator (tPA), 0.3 mg and aspirated at 3.5 hours after hematoma induction. Brains were frozen in situ at 24 hours post-ICH and hematomal and perihematomal edema volumes were determined on coronal sections by using computer-assisted morphometry. Hematoma evacuation rapidly reduced elevated cerebral tissue pressure from 12.2±1.3 to 2.8±0.3 mm Hg. At 24 hours, prior clot removal markedly reduced hematoma volumes (0.40±0.10 compared with 1.26±0.13 cm3, p<0.005) and perihematomal edema volumes (0.28±0.05 compared with 1.46±0.24 cm3, p<0.005), compared with
unevacuated control lesions. Furthermore, no Evans blue dye staining of perihematomal edematous white matter was present in brains in which the hematomas had been evacuated, compared with untreated controls.

Conclusions. Hematomas were quickly and easily aspirated after treatment with tPA, resulting in significant reductions in mass effect. Hematoma aspiration after fibrinolysis with tPA enabled removal of the bulk of the hematoma (>70%), markedly reduced perihematomal edema, and prevented the development of vasogenic edema. These findings in a large-animal model of ICH provide support for clinical trials that include the use of fibrinolytic agents and ultra-early stereotactically guided clot aspiration for treating ICH.

AB-14415-99

Background. Cigarette smoking accelerates atherosclerosis and restenosis after vascular reconstruction. The mechanisms by which smoking alters vessel structure after injury are unclear. This study examined the effects of cigarette smoking on endothelial regeneration, an important component of arterial remodeling.

Materials and methods. Adult male rats were subjected to balloon injury of the thoracic aorta and exposed to mainstream cigarette smoke via a Griffith-type smoking machine for 2 weeks. Control groups included rats which were restrained in the machine but not smoked and a group not utilizing the machine. Aortic reendothelialization was determined using Evans’s blue staining of the arterial surface. Serum levels of nitric oxide were measured to determine if smoke exposure altered this potential endothelial cell mitogen.

Results. Cigarette smoking increased aortic endothelial regeneration (78.4±4.6% vs 59.2±2.1%, P<0.05) and was associated with an increase in serum nitric oxide level (59.9±7.1 μM vs 28.5±1.8 μM, P<0.05). Daily restraint alone in the smoking machine had no effect on endothelial regeneration.

Conclusions. This is the first report on the effects of smoking on endothelial regeneration and demonstrates that smoking increases reendothelialization after large vessel injury and serum levels of nitric oxide, an EC mitogen.

AB-14417-99

Background and purpose: In childhood-onset moyamoya disease, the angiographic disease process of stenoocclusive lesions is progressive, and cerebral infarctions often develop as a result of ischemia. Our purpose was to determine how the severity of stenoocclusive lesions in the anterior and posterior circulations affects the distribution of cerebral infarction in patients with childhood-onset moyamoya disease.

Methods: In 69 patients with childhood-onset moyamoya disease, angiograms were reviewed for stenoocclusive lesions, and CT scans, MR images, or both were reviewed for the sites and extent of cerebral infarction. The relationship between the angiographic and CT/MR findings was examined.

Results: The prevalence and degree of stenoocclusive lesions of the posterior cerebral artery (PCA) significantly correlated with the extent of lesions around the terminal portion of the internal carotid artery (ICA). The prevalence of infarction significantly correlated with the degree of stenoocclusive changes of both the ICA and PCA. Infarctions tended to be distributed in the anterior borderzone in less-advanced cases, while in more advanced cases lesions were additionally found posteriorly in the territory of the middle cerebral artery, the posterior borderzone, and the PCA territory.

Conclusion: Our results indicate that progressive changes of the anterior and posterior circulations are associated with the distribution of cerebral infarction, culminating in a patchily disseminated or honeycomb pattern of infarction on CT and MR studies in late stages of the disease.

AB-14418-99

Background: Incidental foci of signal loss suggestive of past microbleeds are a frequent finding on gradient-echo T2*-weighted MRI of patients with nontraumatic intracerebral hemorrhage and have been associated with bleeding-prone microangiopathy. If and to what extent such lesions may also occur in the normal population is unclear.

Objective: To determine focal hypointensities in asymptomatic elderly individuals and their relation to other clinical and morphologic variables.

Methods: T2*-weighted MRI of the brain was performed in a consecutive series of 280 participants (mean age 60 years, range 44 to 79) of the Austrian Stroke Prevention Study. This cohort consisted of randomly selected individuals without history or signs of neuropsychiatric disorder.

Results: Past microbleeds ranging from one to five foci of signal loss were seen in 18 (6.4%) individuals. They were strongly associated with higher age, hypertension, and lacunes (p<0.001), and extensive white matter damage was more frequently noted (p=0.02). Hypertension was present in all individuals with focal hypointensities in the basal ganglia and infratentorially but in only 5 of 10 volunteers with microbleeds limited to cortico-subcortical sites (p=0.04).

Conclusions: MRI evidence of past microbleeds may be found even in neurologically normal elderly individuals and is related, but not restricted, to other indicators of small vessel disease. The predictive potential of this finding regarding the risk of intracerebral bleeding requires further investigation.

Objectives—The aim of this study was to use transcranial Doppler ultrasonography to investigate cerebral vascular reactivity to hypercapnia in the middle cerebral arteries of patients with carotid occlusion with different outcomes. Patients and methods—Cerebrovascular reactivity to hypercapnia was calculated with the breath-holding index (BHI). Patients with unilateral carotid occlusion were divided as follows: asymptomatic (20 patients), transient ischemic attack (TIA) (20 patients), minor (20 patients) and major stroke (14 patients). Values of BHI homolateral to the carotid occlusion were compared with those of 25 healthy subjects and 34 stroke patients without significant carotid stenosis. Results—BHI values were comparable in healthy controls, non stenotic stroke patients and asymptomatic occluded patients. BHI values of patients with asymptomatic occlusion were significantly lower than those of the above-mentioned groups (P<0.0001). Moreover, the reduction of BHI was significantly associated with the extent of the neurological impairment. In fact, BHI values were significantly higher in TIA than in minor and major stroke (P<0.0001) and in minor than in major stroke patients (P<0.02). Finally, we found that a BHI value homolateral to carotid occlusion of 0.69 can be considered the cut-point for distinguishing between symptomatic and asymptomatic patients. Conclusion—Prospective studies are needed to demonstrate if the presence of this threshold value may help in selecting a subset of patients with asymptomatic carotid occlusion or with transient or mild neurological deficit with the highest probability of benefiting from surgical therapy.
Impaired hydroxylation of S-warfarin in in-vitro expression systems. We protected the brain from ischemic damage in the acute stage.

**Conclusion**—We conclude that an elevation in homocysteine concentration is associated with an acute impairment of vascular endothelial dysfunction, which is mediated through oxidative stress mechanisms and can be inhibited by the antioxidant vitamin C.

**Methods and Results**—We studied 17 healthy volunteers (10 male and 7 female) aged 33 (range 21 to 59) years. Brachial artery diameter responses to hyperemic flow (endothelium dependent), and glyceryltrinitrate (GTTN, endothelium independent) were measured with high resolution ultrasound at 0 hours (fasting), 2 hours, and 4 hours after (1) oral methionine (L-methionine 100 mg/kg), (2) oral methionine preceded by vitamin C (1 g/day, for 1 week), and (3) placebo, on separate days and in random order. Plasma homocysteine increased (0 hours, 12.8±1.4; 2 hours, 25.4±2.5; and 4 hours, 31.2±3.1 μmol/L, P<0.001), and flow-mediated dilation fell (0 hours, 4.3±0.7; 2 hours, 1.1±0.9; and 4 hours, −0.7±0.8%) after oral L-methionine. There was an inverse linear relationship between homocysteine concentration and flow-mediated dilatation (P<0.001). Pretreatment with vitamin C did not affect the rise in homocysteine concentrations after methionine (0 hours, 13.6±1.6; 2 hours, 28.3±2.9; and 4 hours, 33.8±3.7 μmol/L, P=0.27), but did ameliorate the reduction in flow-mediated dilation (0 hours, 4.0±1.0; 2 hours, 3.5±1.2 and 4 hours, 2.8±0.7%, P=0.02). GTN-induced endothelium independent brachial artery dilation was not affected after methionine or methionine preceded by vitamin C.

**Conclusions**—We conclude that an elevation in homocysteine concentration is associated with an acute impairment of vascular endothelial function that can be prevented by pretreatment with vitamin C in healthy subjects. Our results support the hypothesis that the adverse effects of homocysteine on vascular endothelial cells are mediated through oxidative stress mechanisms.

**Methods**—Patients with a daily warfarin dose requirement of 1.5 mg or less (low-dose group, n=36), randomly selected patients with a wide range of dose requirements from an anticoagulant clinic in north-east England (clinical control group, n=52), and 100 healthy controls from the community in the same region were studied. Genotyping for the CYP2C9*2 and CYP2C9*3 alleles was done by PCR analysis. Case notes were reviewed to assess the difficulties encountered during the induction of warfarin therapy and bleeding complications in the low-dose and clinic control groups.

**Findings**—The odds ratio for individuals with a low warfarin dose requirement having one or more CYP2C9 variant alleles compared with the normal population was 6.21 (95% CI 2.48–15.6). Patients in the low-dose group were more likely to have difficulties at the time of induction of warfarin therapy (5.97 [2.26 –15.82]) and have increased risk of major bleeding complications (ratio 3.68 [1.43–9.50]) when compared with randomly selected clinic controls.

**Interpretation**—We have shown that there is a strong association between CYP2C9 variant alleles and low warfarin dose requirement. CYP2C9 genotyping may identify a subgroup of patients who have difficulty at induction of warfarin therapy and are potentially at a higher risk of bleeding complications.
circulatory arrest with retrograde cerebral perfusion technique was exclusively applied in the most recent 76 patients.

Results. There were 50 (20%) early deaths and 37 (19%) late deaths. Postoperative stroke was found in 26 (11%) patients of which 13 (50%) died. Mutual predictive factors for postoperative mortality and stroke were earlier series, preoperative chronic renal failure, ruptured aneurysm, arch clamping during procedure, and using partial cardiopulmonary bypass. Among 129 patients operated on during the most recent 5 years, early mortality and incidence of stroke decreased to 14.7% and 6.9%, respectively.

Conclusions. Results of operations for arteriosclerotic aneurysms of the transverse aortic arch in 246 patients during a period of 17 years have been improving but are still not satisfactory.

Items of Interest


Diagnosis and Treatment of Ischemic Stroke—Alberts MJ (Box 3392, Duke Univ Medical Center, Durham, NC 27710)—Am J Med. 1999;106:211–221. Copyright © 1999 by Excerpta Medica, Inc.


Abstracts of Literature
Askiel Bruno and Engin Y. Yilmaz

Stroke. 1999;30:1494-1500
doi: 10.1161/01.STR.30.7.1494

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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