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Abstracts
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Neglect in the first six hours of stroke

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Objective: To study neglect in hyperacute stroke and its evolution over the first days after onset with respect to penumbra dynamics as assessed by perfusion computed-tomography (P-CT).

Background: Neglect is mostly seen in right middle cerebral artery (MCA) territory infarction and is associated with poor outcome. Its severity and clinical impact may be underestimated in the hyperacute phase when measured by the National Institute of Health Stroke Scale (NIHSS). Decision making for acute thrombolysis is based on this score and patients with low NIHSS may be excluded for such a treatment in spite of severe neglect. A short bedside neglect evaluation is needed along with the NIHSS in order to improve patient selection.

Design/Methods: We developed a 10-minute bedside battery of tests including cancellation, line bisection, figure copying, reading and motor neglect, providing a composite score of neglect (0 = no deficit, 25 = maximum severity) together with a score of anosognosia according to Bisiach. Nine patients (5 women and 4 men, mean age 55.2 years) with first-ever right hemispheric stroke were examined with this battery and P-CT images were obtained, both within 6 hours from symptoms onset. On day 3, the same tests were performed together with diffusion-weighted magnetic resonance imaging (DW-MRI). Both P-CT and DW-MRI were blind-rated to the clinical picture. Regions of interest (ROI) relevant for neglect (i.e. inferior parietal lobule, dorso-lateral frontal cortex, thalamus, striatum, superior temporal gyrus, cingulate gyrus, corpus callosum and subcortical white matter) were examined. A radiological score was defined by the number of ROI involved in the perfusion deficit (P-CT) or in the final infarcted area (DW-MRI).

Results: All patients showed severe neglect manifestations (mean score of 13) on admission, corresponding to large antero-posterior hypoperfusion of the right MCA territory. When at least one ROI included in the penumbra (P-CT) was rescued at day 3 (no diffusion alterations on DW-MRI), the clinical score improved significantly (9.5 points) in comparison to cases where no ROI were rescued (3.7 points, $p < 0.05$). On the other hand, neither the score of anosognosia nor the NIHSS score evolution correlated significantly with radiological score changes.

Conclusions: Our preliminary results show an association between neglect evolution at day 3 and penumbra rescuing, whereas no correlation with NIHSS changes was found. We hypothesise that this scale underestimates neglect severity and may be complemented by our short bedside battery for assessment of right MCA stroke. Our findings also suggest a dissociation between anosognosia and neglect, which deserves further study.

Late salvage of penumbra by mechanical MCA-clot retraction

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Background: The main goal of acute stroke treatment is salvage of the penumbra. Multiple contraindications, including time limits and clotting problems, restrict the number of patients potentially benefiting from iv or ia thrombolysis.

Patient/Methods: A 37-year-old lady with a mechanical heart valve on oral anticoagulation, moderately disabled from a previous non-recanalised left MCA stroke (mRS = 3), was found unresponsive in bed in the morning. On exam, stupor, mutism, bilateral hemiparesis, left hemianopia and left eye deviation was present (NIHSS = 28). After intubation, perfusion-CT and angio-CT were performed according to a standard protocol, and followed by conventional angiography.

Results: Perfusion-CT 210 minutes after awakening showed a minor core area in the depth and a large penumbra in the rest of the right MCA territory, with occlusion of the right MCA on angio-CT. As thrombolysis could not be performed because of an INR of 2.1, the patient underwent mechanical removal of an occlusive thrombus in the right MCA at 6 h 30 after awakening, by a lasso-type device, encircling the proximal end of the clot and retracting it. The patient's Rankin scale decreased from 4 pre-stroke to 3 three months post-stroke. Pathology showed a fresh fibrin-rich thrombus modelling the distal MCA-trifurcation, compatible with an embolus of cardiac origin. Final stroke volume was 0.7 cm^2 in the right (treated) MCA-territory as opposed to 32 cm^2 in the left (untreated and not recanalised for more than 24 hours during the first stroke).

Conclusions: This patient illustrates that late salvage of brain tissue may be possible in patients with unknown onset of stroke if significant penumbra is demonstrated, and that this may be achieved with mechanical recanalisation in patients with prolonged clotting times contraindicating thrombolysis.

Outcome of acute stroke patients without visible occlusion on early arteriography

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Background: The aim of this study was to determine the clinical and radiological outcome of acute stroke patients who showed no arterial occlusion on arteriography and to determine predictors of clinical outcome in these patients.

Methods: We analysed clinical and radiological data of acute stroke patients without arterial occlusion on arteriography within 6 hours of symptom onset.

Results: Out of a patient group of 283 consecutive patients who were screened for intraarterial thrombolysis by arteriography, 28 patients (10%) had no arterial occlusion. The median baseline National Institute of Health Stroke Scale (NIHSS) of these patients was 7. The average time from symptom onset to arteriography was 226 (range 115 to 315) minutes. Presumed stroke aetiology was cardiac embolism in 11 patients (39%), small artery disease in 6 (21%), iatrogenic, following coronary angiography in 1 (4%) and undetermined in 10 patients (36%). After 3 months 21 patients (75%) had a favourable clinical outcome (modified Rankin Scale score [mRS <2]) and 6 patients (21%) a poor outcome (mRS 3 or 4). One patient (4%) died due to myocardial infarction. On follow-up brain imaging, 8 patients (29%) showed a lacunar lesion, 2 (7%) a striatocapsular infarct, 6 (21%) a small or medium-sized anterior circulation infarct, 2 (7%)

multiple small anterior circulation infarcts and 4 (14%) multiple posterior circulation infarcts. In 5 patients (18%) follow-up MRI (n = 4) or CT (n = 1) revealed no ischaemic lesion, one patient (4%) had no follow-up imaging. Initial NIHSS score, clinical stroke syndrome, time to arteriography and suspected stroke aetiology failed to predict clinical functional outcome.

Conclusions: The majority of patients with acute cerebral ischaemia without vessel occlusion on early arteriography develop cerebral infarction on brain imaging. Functional clinical outcome is usually favourable with significant disability or death in only a quarter of the patients.

Epileptic seizures after cerebral ischaemia

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Introduction: Cerebrovascular disease is a well-recognised cause of late onset epilepsy, but the true frequency, risk factors and prognosis are not well established.

Methods: We studied all consecutive patients with cerebrovascular disease and/or epileptic seizures who were seen in our department between January 2000 and June 2003. The diagnosis was established by history, clinical examination, EEG, laboratory findings, ultrasound studies and neuroimaging including MRI. Seizures were classified according to the international classification of epileptic seizures.

Results: Ischaemic cerebrovascular disease was diagnosed in 1029 patients; 1114 patients had one or more epileptic seizures during the study period. Ischaemic disease was identified as the cause of one or more seizures in 121 patients. Sixty-six patients (55%) had only one seizure, 55 patients (45%) went on to develop epilepsy with recurrent seizures during the observational period. In 71 patients the exact time span between the acute ischaemic event and the first seizure could be determined. In 27% of patients the first seizure occurred within the acute phase of the cerebrovascular event (early seizures), in 73% of patients the first seizure occurred more than 2 weeks after cerebral ischaemia (late seizures). Nearly 20% of patients with a major stroke had at least one seizure during the study period as opposed to only 5% after a transient ischaemic attack (TIA) and 2% after a reversible ischaemic neurologic deficit (RIND). Within the group of patients with cardiac emboli as pathogenic mechanism 13% of patients had one seizure and half of them developed epilepsy with recurrent seizures. Focal epileptogenic changes could be seen in the same number of patients with early or late seizures, but 95% of patients with cerebral ischaemia and an epileptogenic focus had at least one seizure, 30% during the acute phase, 70% later during follow-up. High age at onset of cerebral ischaemia and smoking as vascular risk factor lowered the risk for early or late seizures after stroke.

Conclusion: In our present study, 12% of all patients with cerebral ischaemia developed one or more seizures. The data emphasise a higher rate of seizures and epilepsy in patients with major stroke as compared to patients with transient ischaemic events and in patients with focal epileptogenic changes at EEG. On the other hand, old age at onset of ischaemia as well as smoking as vascular risk factor were associated with a lower risk of developing epilepsy after a cerebrovascular event. These results can have major therapeutic implications considering antiepileptic medication at an relatively early stage after stroke in patients at high risk for developing epilepsy after stroke.

Comparison of intra-arterial thrombolysis with conventional treatment in patients with acute central retinal artery occlusion

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Background: Several case series and a recent meta-analysis indicate that intra-arterial thrombolysis (IAT) is effective to treat acute central retinal artery occlusion (CRAO).

Methods: Thirty-seven patients with acute monocular blindness because of unilateral thromboembolic CRAO were treated with IAT using urokinase within six hours of symptom onset. Visual outcome was compared with a control group of 19 patients, also seen within 6 hours, but who did not undergo thrombolytic treatment. In both groups some patients were treated by paracentesis and/or acetazolamide. Predictors of visual outcome were evaluated.

Results: Visual improvement was more likely with IAT ($p = 0.01$), and the chances to regain a visual acuity of >0.6 were significantly better ($p = 0.04$). In the IAT group 8 of 37 patients (22%) regained a visual acuity of >0.6, in the control group none of the 19 patients. Younger patients were more likely to regain some vision with ($p = 0.012$) or without IAT ($p = 0.026$). Three patients suffered minor treatment-related cerebral ischaemic events, two TIAs and one minor stroke. However, there were no haemorrhagic complications.

Conclusions: This series of patients with CRAO demonstrates that IAT enhances the chances of visual improvement when compared to a group with conventional treatment only. Furthermore, younger patients have better chances to achieve some visual recovery.

Long-term rupture and stroke risks in patients with dissecting aneurysms due to spontaneous dissection of the cervical internal carotid artery

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Introduction: Stroke prevention in patients with dissecting aneurysm due to spontaneous dissection of the cervical internal carotid artery (ICAD) is controversial. We performed an observational study in such patients to assess the long-term risk of rupture of the dissecting aneurysm and stroke.

Methods: Twenty-three patients (9 women, 14 men; mean age 45 ± 7 years) with 25 ICADs causing 23 dissecting aneurysms were included. Follow-ups were done after 3, 6 and 12 months and annually thereafter. The first three follow-ups (3, 6 and 12 months) included clinical and colour duplex sonography (CDS) investigations, the subsequent annual follow-ups were done either clinically or by a structured telephone interview.

Results: Two patients underwent an endovascular therapy of the aneurysms: in the first case the aneurysm was treated with balloon occlusion of the ICA followed by extracranial-intracranial bypass surgery, in the second case the aneurysm was occluded. Antithrombotic therapy included aspirin and warfarine. Median follow-up was 6.9 (range 0.3–13.8 years) for all patients who were conservatively treated, and 6.5 and 14.1 years, respectively, for the 2 patients with endovascular therapy. Antithrombotic treatment had been stopped for various reasons in 7 patients (median 4 years, range 0.3–11.5). One patient had no follow-up, because he died from a malignant MCA infarction after 14 days.

Conclusion: Our data indicate that dissecting aneurysms treated with antiplatelet agent, oral anticoagulant or no treatment have a benign long-term outcome with a low risk of rupture or stroke, which suggests that conservative therapy is appropriate in most cases. One observational study suggested that low-dose aspirin is the best approach due to the low risk of cerebral embolism and the absence of aneurysm rupture. On the other hand, case reports describe the successful endovascular therapy of such aneurysms.

Perfusion-CT in acute occipital stroke

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Objective: To correlate perfusion-CT with clinical findings and prognosis in acute ischaemic stroke.

Background: Localisation, size of core and penumbra, and prognosis in acute stroke may be aided by new imaging techniques

such as diffusion-perfusion MRI or perfusion-CT (PCT). The latter has only been explored and shown to be accurate in acute middle cerebral artery territory strokes, but no data are available for the posterior cerebral artery (PCA) territory.

Methods: In 2002 and 2003, 10 patients admitted to our stroke unit with symptoms and signs of PCA-territory stroke underwent PCT and angio-CT (ACT). The 4 patients with symptoms starting >24 hours before admission and one patient with TIA were excluded from the present analysis.

Results: PCT was performed between 121 and 720 minutes after last well time. Abnormalities in the occipital lobe of one (n = 4) or two (n = 1) PCA territories were seen in all 5 patients, corresponded to the visual field deficits and were confirmed by MRI. As plain CT was normal in 3 of these 6 PCA territories in the acute phase, diagnostic sensitivity of CT was increased. Additional lesions in the cerebellum (1), thalamus (1) and inferomesial temporal lobes (2) were not detected, as they lie outside the territory of standard PCT. ACT showed occlusion of P2 or P3 in 83% of affected territories. Spontaneous resolution of visual field defects over 7 days was more frequent in patients with a significant penumbra than in those without (100 vs 33%).

Conclusions: In patients with acute PCA territory stroke, a standard protocol PCT increases diagnostic sensitivity for the occipital lobe, may miss thalamic and inferomedial temporal ischaemia, and may predict spontaneous resolution of visual field deficits.

Venous CT-angiography in deep cerebral venous thrombosis

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Objective: To evaluate venous CT-angiography (vCTA) as an alternative to venous vMRA to diagnose deep cerebral venous thrombosis (DCVT).

Background: DCVT is a treatable condition, which may be suspected in patients with unusual headaches, decreased level of consciousness and bilateral thalamic lesions on neuroimaging. Diagnostic confirmation by venous Magnetic Resonance Imaging Angiography (vMRA), conventional angiography or autopsy is usually required.

Design/Method: Six patients with suspected DCVT underwent vCTA on a spiral CT scanner and 5 had simultaneous vMRA. In the patients in whom the diagnosis of DVCT was confirmed by vMRA, follow-up vCTA and vMRA were obtained after 3 months. In a patient who died and who was unable to undergo vMRA because of a pace-maker, autopsy and post-mortem MRI were done. In order to calculate sensibility and specificity for occlusion of deep cerebral veins of vCTA as compared to vMRA, 56 named deep cerebral veins in the 5 acute and 2 follow-up paired vCTAs and vMRAs were assessed by a blinded neuroradiologist.

Results: In 3 of the 6 patients, DCVT was confirmed by vMRA or autopsy. vCTA of these 3 patients showed thrombi or absent venous flow in different compartments of the deep venous system. Follow-up by vCTA and vMRA (2 patients) or autopsy (one patient) showed partial recanalisation. The blinded comparison of deep cerebral veins on vCTA and vMRA showed a high degree of accuracy for vCTA.

Conclusions: vCTA seems to be a highly accurate method to diagnose, characterise and follow-up patients with suspected deep cerebral venous thrombosis.

Pseudothrombozytopenie als Verzögerungsgrund für eine intravenöse Thrombolyse bei ischämischem Hirnschlag

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Einleitung: Die Pseudothrombozytopenie ist mit einer Prävalenz von 1:1000 relativ selten und tritt unabhängig von der Präsenz oder Absenz anderer Krankheiten auf. Bei Kontakt des Blutes dieser Patienten mit Ethylendiamintetraessigsäure (EDTA), dem üblichen Antikoagulans bei hämatologischen Untersuchungen, kommt es zur Agglutination von Blutplättchen und damit zu artifiziell tiefen Thrombozytenzahlen bei maschineller Zählung. Verantwortlich für dieses Phänomen ist ein Antikörper, der gegen Epitope auf dem Glykoprotein IIb der Thrombozyten gerichtet ist. Normalerweise sind diese Epitope nicht exponiert, werden aber durch die Interaktion der EDTA mit den Glykoproteinen IIb-IIIa freigelegt. Andere Antikoagulantien, wie zum Beispiel Citrat, bewirken normalerweise keine Freilegung dieser Epitope und führen somit nicht zu einer Plättchenagglutination. EDTA-Agglutinine sind somit für den Patienten ungefährlich.

Fallbeschreibung: Wir beschreiben einen 34jährigen Patienten, der wegen akut aufgetretenem sensomotorischem Hemisyndrom rechts begleitet von einer Sprachstörung zugewiesen wurde. Die notfallmässigen Abklärungen ergaben die Diagnose eines akuten, ischämischen, linkshemisphärischen Hirnschlages. Die geplante Durchführung einer iv-Lysetherapie wurde durch eine schwere Thrombozytopenie von 13 000 Thrombozyten/µl im routinemässig mit einem EDTA-Röhrchen angefertigten Blutbild zunächst vereitelt. Nachdem eine erneute Bestimmung mit einem Citrat-Röhrchen Thrombozytenwerte in unteren Referenzbereich ergeben hatte, konnte die Diagnose einer Pseudothrombozytopenie gestellt und die Lysetherapie noch knapp im Zeitfenster durchgeführt werden.

Schlussfolgerung: Die Kenntnis der Pseudothrombozytopenie erscheint bei zunehmender Verbreitung der Lysetherapie auch für Nicht-Hämatologen wichtig. Insbesondere bei der iv-Lyse von Patienten mit ischämischem Schlaganfall ist die für die Abklärung zur Verfügung stehende Zeit knapp, so dass diese Therapie bereits durch Verzögerungen von wenigen Minuten vereitelt werden kann.

Intracerebral bleeding due to pseudoxanthoma elasticum in a 38-year-old man

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Introduction: This case report describes an unusual cause of stroke.

Patient: A 38-year-old male with mild hypertonia and no further vascular risk factors was admitted to the hospital with right hemiparesis and dysarthria. The CT-scan showed a bleeding in the left basal ganglia. In MRI-scans an extensive leukoaraiosis was noticed. Further diagnostics including laboratory examinations (vasculitis, infectious diseases, haemophilia, CADASIL) and cerebral angiography were not conclusive.

Two years later the patient developed a pontine ischaemia remaining unclear as well. In an ophthalmological examination angioid streaks of the retina which occur in several collagen disorders were detected. The following dermatological evaluation resulted in the diagnosis of pseudoxanthoma elasticum (Grönblad-Strandberg-Touraine syndrome), a disorder of collagen fibres which causes fragility of blood vessels and can result in both bleeding and ischaemia of several organs.

An intracranial haemorrhage as a first manifestation of pseudoxanthoma elasticum is very uncommon.

Diffusion-weighted imaging and apparent diffusion coefficient maps to distinguish stroke due to large artery atherosclerosis from cardioembolic stroke

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Background: Recent diffusion-weighted imaging (DWI) MR studies have indicated a link between DWI findings and the underlying pathogenetic mechanism of stroke. However, DWI lesion characteristics in an unselected population of patients with stroke either due to cardioembolism or due to large artery atherosclerosis have been compared so far.

Patients and Methods: We studied 83 consecutive patients with stroke either due to cardioembolism (CE; n = 43) or due to large artery atherosclerosis (LAA; n = 40) according to the TOAST classification who underwent DWI within 7 days after symptom onset. Blinded to the patients' clinical data, we compared both groups regarding the

following DWI lesion characteristics: number and distribution of (a) all hyperintense DWI lesions and (b) only those hyperintense DWI lesions with hypointense appearance on ADC maps.

Results: The mean number of lesions with increased signal intensity on DWI was 4.65 (SD 4.87) in the LAA group and 3.09 (SD 4.74) in the CE group (p = 0.011). Multiple (≥2) lesions were seen in 28 patients (70.0%) with LAA and in 21 patients (48.8%) with CE (p = 0.041). Multiple lesions including border zone areas more often occurred in LAA (n = 13, 32.5%) than in CE (n = 5, 11.6%; p = 0.020). Multiple lesions in either both anterior circulations or anterior plus posterior circulation were present in 5 patients (12.5%) with LAA and in 4 patients (9.3%) with CE. The mean number of hyperintense DWI lesions with hypointense appearance on ADC maps was 3.20 (SD 3.98) in the LAA group and 2.05 (SD 2.74) in the CE group (p = 0.036). Multiple lesions fulfilling these criteria occurred in 23 patients (57.5%) with LAA and 15 patients (34.9%) with CE (p = 0.036). Using these criteria, significant group differences in lesion patterns were observed only for multiple lesions within the same arterial territory, excluding border zone areas, which occurred in 11 patients (27.5%) with LAA and 4 patients (9.3%) with CE (p = 0.030). Multiple lesions in either both anterior circulations or anterior plus posterior circulation were rare and only occurred in CE (n = 3; 7%) but not in LAA patients.

Conclusion:

1. Patients with LAA strokes more often had multiple hyperintense DWI lesions and a higher number of lesions than patients with CE strokes.
2. Only if hyperintense DWI lesions were restricted to those with hypointense appearance on ADC maps, involvement of either both anterior circulations or anterior plus posterior circulation suggested CE.
3. If only acute DWI lesions (i.e. those with hypointense appearance on ADC-maps) were considered, hyperintense DWI lesions in border zone areas did not indicate stroke due to LAA.

Increase of tissue factor pathway inhibitor (TFPI) in stroke patients: a new mechanism of Ancrod?

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Introduction: Ancrod is a venom from a Malayan viper, which is believed to exert its action through degradation of fibrinogen and fibrin, essentially providing a fibrinolytic effect. Recently, Ancrod has been evaluated as a potential acute treatment strategy for strokes. Unfortunately this trial (the ESTAT trial) did not provide sufficient data to suggest that early treatment of stroke with Ancrod is helpful. Lipoprotein(LP)(a) has been shown to be a risk factor for atherosclerosis and atherous thromboses as well as strokes. We therefore evaluated whether any change in Lp(a), which shares structural similarities with plasminogen, might have had any influence on the outcome of Ancrod treated patients since we have shown in the past that various elements and treatments can modulate Lp(a)-concentrations. Also, Lp(a) has been shown to form complexes with tissue factor pattern inhibitor, a potent anticoagulant, suggesting that any potential modulation of Lp(a) might influence fibrinolytic and coagulant pathways.

Methods: We compared total cholesterol, LDL, Lp(a), plasmin inhibitor and tissue factor pathway inhibitor activity in patients treated in the ESTAT trial at our study site. In all, 15 patients received Ancrod and 15 patients continuously received placebo for 72 hours and were followed with clinical and laboratory parameters over a 5-day period. Assays were performed according to the suggestions of the manufacturers and were all commercially available.

Results: Ancrod significantly reduced plasmin inhibitor (1.06 vs 0.81, p <0.001) and TFPI activity (1.71 vs 2.14, p = 0.003) but increased Lp(a) (1.73 vs 1.05, p = 0.003) in the verum group as compared to baseline at 144 h. There were no significant differences in total cholesterol, triglycerides, HDL- and LDL-cholesterol between the verum and placebo group as compared to baseline.

Discussion: The above results indicate that Ancord is not only active as a direct fibrinolytic, but also as an indirect fibrinolytic as well as an indirect anticoagulant both by decreasing plasmin inhibitor and increasing tissue factor pathway inhibitor activity from baseline. This increase in TFPI activity is observed despite the increase in Lp(a), indicating that complexing of TFPI by Lp(a) does not carry a limiting effect for TFPI activity. The reason for the increase in Lp(a) remains to be elucidated and is not obvious from the current results.

Conclusion: As shown by the results from this double-blind randomised trial of Ancrod treatment in acute stroke, we conclude that Ancrod not only has direct fibrinolytic activity but also indirect fibrinolytic activity (by decreasing plasmin inhibitor) as well as indirect anticoagulant activity (by increasing TFPI activity). The significance of the changes observed in Lp(a) concentration remains to be elucidated.

BISS: Bürgerspital-Inselspital-Stroke-Study. Management and outcome of stroke in a general ward and a university hospital based stroke unit

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Background: The beneficial effects of dedicated stroke units (SU) compared to general wards (GW) are well established. However, some GWs have started to improve their management applying SU guidelines and treatment protocols. The aim of the present study was to compare the acute stroke management in a Swiss GW with an active protocol with that of an SU at a university hospital.

Methods: 266 consecutive stroke patients were prospectively included over a period of 13 months. 103 patients were treated in a GW and 163 patients were admitted to an SU. Demographic data, vascular risk factors, presenting clinical and radiological characteristics and co-morbidities were evaluated. Outcome measures were modified Rankin scale score (mRS) at 4 months, mortality, length of hospitalisation (LOH) and discharge to a nursing home.

Results: Patients treated in GW were older (75.47 years vs 61.02 years, p = 0.000) and had more co-morbidities (Charlson-Index 1.62 vs 0.58, p = 0.000) compared to patients of the SU. While in the GW all patients had a CT scan at presentation, SU patients were more likely to undergo MRI (p = 0.000), ultrasound examination of the carotid arteries (p = 0.000) and the heart (p = 0.000), angiography (p = 0.000) and 24-h ECG (p = 0.000). LOH was similar in both hospitals. In-hospital and 4-month mortality rates were lower in SU (6.2% / 9.8% vs 13.5% / 20.4%). At follow-up, SU patients were less disabled (p = 0.005) and were less likely to be discharged to a nursing home (p = 0.002). However, in a univariate analysis of variance corrected for age and co-morbidity, the outcome of patients treated in the SU and the GW were not different. There remained only a nonsignificant trend that SU patients were less likely discharged to a nursing home (p = 0.058). In a multivariate regression analysis, co-morbidity was a stronger predictor of outcome than the hospital, in which the patient was treated.

Conclusions: Patients treated in an SU and in a GW differ significantly regarding age and co-morbidities. There was a non-significant trend that SU patients were less likely discharged to a nursing home than patients from the GW; otherwise, in a multivariate regression analysis, outcome of patients from the SU and GW did not differ. Co-morbidity and age were stronger predictors than the hospital, in which the patient was treated. Therefore, co-morbidities, measured with the Charlson-Index should more often be included in stroke research.

Transient ablation of tinnitus by unilateral stroke of Heschl's gyrus: evidence for a centrally maintained unilateral tinnitus system depending on primary auditory cortex

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Objective: To clarify the pathogenesis of chronic unilateral tinnitus in a patient with transient relief by a stroke involving contralateral temporal cortex.

Background: Generation of aberrant central pathways and cortical plasticity are suspected in chronic tinnitus. Understanding these processes may lead to more effective treatments.

Methods: A 67-year-old patient with a mechanical heart valve experienced an acute right sensory hemisyndrome and mild fluent aphasia. He also noticed disappearance of chronic, non-pulsatile 6000 Hz tinnitus in his right ear that had appeared shortly after an acute moderate hearing loss 7 years earlier. Evolution of the tinnitus was observed and MRI, fMRI, audiograms, brainstem auditory evoked potentials (BAEP) and auditory middle evoked potentials (AMEP) were performed in the acute and chronic phases of stroke.

Results: Tinnitus reappeared on day 4 post-stroke and progressively regained its usual features. Diffusion-MRI showed three distinct ischaemic lesions in the left middle cerebral artery territory, with one being related to the auditory system: (1) primary auditory cortex (Heschl's gyrus), (2) posterior insula and (3) part of the parietal lobe. Audiograms showed a chronic right cochlear nerve deficit and BAEP revealed no brainstem pathology. AMEP amplitudes were decreased on the left during the acute phase and back to normal at 9 months. Comparison of fMRI without and with tinnitus at rest (silence) revealed no specific auditory system activity but increased bilateral amygdala activity with reappearance of tinnitus.

Conclusions: Findings in this patient suggest that (1) a peripheral cochlear lesion may lead to tinnitus that is maintained by central mechanisms, (2) central representation of tinnitus may be unilateral, (3) primary auditory cortex may play an essential role in centrally maintained tinnitus and (4) a unilateral lesion of the primary auditory cortex is sufficient to (transiently) abolish centrally maintained tinnitus that is perceived contralaterally.

Neuroprotective role of metabotropic glutamate receptor 5 in NMDA-mediated excitotoxicity in the cerebral cortex of mice

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Background and Purpose: Glutamate hyperstimulation mediated by NMDA (N-methyl-D-Aspartate) receptors may lead neurons into the cell death program. This mechanism is considered to be one of the causes of neuronal damage after ischaemic stroke as well as some cerebral degenerative diseases. Recent in vitro studies showed that metabotropic glutamate receptors (mGluRs) could play a role in this type of excitosis. We here studied the involvement of the mGluR5 receptor in an in vivo model of NMDA-dependent excitosis.

Methods: Small volumes of the NMDA receptor agonist quinolinic acid were injected in the somatosensory cortex (SI) of wild type (n = 8) and mGluR5 knock-out (KO) mice (n = 8). Four wild type animals received an intraperitoneal injection of MPEP, an mGluR5 specific inhibitor, 30 minutes prior to excitotoxin injection, and 4 KO mice received an intraperitoneal injection of the NMDA receptor antagonist MK-801 again 30 minutes prior to excitotoxin injection. After a five-day survival period, Nissl and cytochrome oxidase stains of SI were used to measure the lesion volume in consecutive slices.

Results: Cortical lesions in mGluR5 KO mice were three times larger than those induced in wild type mice. The lesion volume could be significantly reduced by intraperitonal injection of the NMDA receptor antagonist MK-801 prior to the quinolinic injection. This indicates that the increase in lesion size in KO mice was dependent on NMDA receptor activation and not due to a general fragility of the system. Intraperitoneal injection of MPEP in wild type mice 30 minutes before excitotoxin injection resulted in a two-fold increase of the lesion volume compared with wild type mice given quinolinic acid alone. This demonstrated that the increased lesion volume in KO mice was not due to a developmental difference between the wild type and knockout mouse strains and suggests that the role of mGluR5 in protecting against NMDA-mediated excitotoxicity requires coincident activation of its receptor.

Conclusion: mGluR5 activation interferes with NMDA-mediated neurotoxicity and can therefore offer new approaches to neuroprotection in acute stroke and degenerative brain diseases.

Microembolic signals in carotid endarterectomy: are counts more important than the nature of the embolic material?

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Objectives: Stroke is the most common complication of carotid endarterectomy (CEA). Although both embolism and haemodynamic compromise can lead to perioperative brain ischaemia, embolism is considered the more common mechanism. We investigated the nature of microembolic signals (MES) in patients undergoing CEA by applying a new automated system and we assessed their association with perioperative ischaemic complications.

Methods: 75 patients, 39 with asymptomatic (A) and 36 with symptomatic (S) carotid stenosis >70%, were monitored over both middle cerebral arteries preoperatively (30 min), intraoperatively and immediately postoperatively (60 min) using a Multi-Dop T2 ultrasound device (DWL, Germany). MES were identified according to standard criteria and analysed by an off-line automated system to predict their gaseous or solid nature. This system is based on the wavelet transform of MES combined with bigated TCD. CEA was performed in all patients under loco-regional anaesthesia.

Results: A total of 2649 signals were detected and classified. All MES detected preoperatively were solid. MES are mainly solid during the dissection ($p = 0.003$) and clamping ($p = 0.04$) stages of the CEA and postoperatively ($p < 0.0001$). During declamping, MES are predominantly gaseous ($p < 0.0001$). MES are more often detected in S compared to A patients preoperatively ($p = 0.01$), during dissection ($p = 0.04$) and postoperatively ($p = 0.05$). MES detection in the preoperative period and during dissection were associated with intraoperative ischaemic complications: 4 TIAs, 3 brain infarcts and one syndrome of hyperperfusion. The total counts of solid emboli during the whole operation ($p < 0.002$), the counts of all emboli regardless of their nature during the whole operation ($p < 0.005$), during the dissection ($p = 0.025$) and clamping ($p = 0.025$) were higher in these complicated patients compared to those who had an uncomplicated CEA. In addition, our showed that a threshold of >7 solid emboli was associated to perioperative brain ischaemia ($p = 0.002$).

Conclusions: High loads of solid microemboli (>7) during the total CEA are a strong predictor of perioperative brain ischaemia.

Acute autonomic dysfunction contralateral to strokes: a prospective study of 100 consecutive cases

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Background: Acute autonomic dysfunction, sometimes mentioned in acute stroke, has not been studied prospectively.

Rationale: To study autonomic dysfunction on the hemibody contralateral to the lesion in the acute phase of stroke.

Methods: 100 consecutive patients (median age ± IQR, 74 ± 21, range 19–93, 51 women 80 ± 17 and 49 men 70 ± 17) were studied prospectively in the acute phase of stroke for dysautonomic dysfunction (DD). Changes in cutaneous temperature, colouration, diaphoresis, pain or oedema were noted in the first three days post stroke. Associations of DD with topography (cortical pre- and/or post-central, corona radiata, basal ganglia, internal capsule, thalamus, brainstem), age, sex, ischaemic or haemorrhagic aetiology and presence of sensorimotor deficits or ataxia were performed using chi-square statistic and logistic regression analysis.

Results: DD was significantly associated with the presence of a lesion in the following localisations: postcentral cortex (p = 0.034), internal capsule (p = 0.003) or the basal ganglia (p = 0.001) and negatively with the presence of a lesion in the brainstem (p = 0.001). Logistic regression analysis including all studied topographic variables shows that brainstem was significantly associated with a decreased risk to develop autonomic dysfunction (OR = 0.095, 95% CI 0.011–0.801, p 0.031) and internal capsule with a risk for more severe dysautonomia (OR = 3.3, 95% CI 1.04–10.1, p = 0.04). More severe autonomic dysfunction with oedema of the hand was observed in association with internal capsule (p = 0.001) or basal ganglia (p = 0.003) lesions, while a negative association was seen when located in the brainstem (p = 0.007). DD was not associated with the ischaemic or haemorrhagic nature of the lesion, its side, age, sex, hyper- or hypotonic paresis, hyper- or hyporeflexia. DD was found in association with sensory deficits (p = 0.0001) and ipsilateral hyperkinesia (p = 0.004).

Conclusion: Acute DD is more prone to be seen in hemispheric lesions involving sensory pathways from cortex to internal capsule. DD is significantly absent with isolated thalamus and brainstem lesions.

Epidemiology of aphasia due to first ischaemic stroke: a population-based study

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Objective: To estimate the incidence of first ischaemic stroke as well as frequency and severity of post-stroke aphasia in the canton Basle-City.

Methods: In a one-year prospective population-based study among the permanent residents of Basle-City (188 015 inhabitants, census 2002) all cases of acute ischaemic stroke were recorded. Multiple sources of information were used, including the stroke register of the local University Hospital, the register of the external neurorehabilitation unit, the records of speech therapists within all hospitals, regular mailings to all hospitals as well as to all physicians practising in Basel-City and taking care of nursing home residents. The diagnosis of acute ischaemic stroke was based on clinical assessment and corresponding neuroimaging findings. In all in-hospital patients the diagnosis of aphasia was made by a neurologist or a speech therapist.

Results: We identified 262 patients with acute first ischaemic stroke. The overall incidence rate amounted to 1.39 (95% confidence interval [CI] 1.22 –1.56) per 1000 inhabitants. Incidence rate increased with advancing age from 0.17 (95%-CI 0.02–0.3) for individuals <45 years old to 10.0 (95%-CI 7.5–12.6) among those aged 85 years or older. Post-stroke aphasia was diagnosed in 78 cases, yielding a 30% prevalence of aphasia. The risk of suffering from post-stroke aphasia increased 4% (2–7%) with each year of age of stroke patients (p <0.01 for age trend). Women had a higher risk of having aphasia after first ischaemic stroke than men (odds ratio 1.82; 95%-CI 1.04–3.18). However, after adjustment for age the difference between the sexes was no longer statistically significant (odds ratio 1.53; 95%-CI 0.86–2.72). Aphasia was mild in 35 (45%), moderate in 22 (28%) and severe in 21 (27%) patients, respectively.

Conclusion: The results of this prospective epidemiological study provide quantitative and qualitative data on aphasia due to first ischaemic stroke and may be useful for resource allocation and planning rehabilitation processes.

Blood pressure changes after acute ischaemic stroke: influence of the degree of vessel recanalisation in patients treated with intra-arterial thrombolysis

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Background: A transient elevation of arterial blood pressure is frequently observed in patients with acute ischaemic stroke. The underlying mechanisms of this blood pressure elevation are poorly understood. The main reason might be a locally disturbed autoregulation of cerebral blood flow within the ischaemic penumbra. If cerebrovascular autoregulation is lost, perfusion becomes a linear function of blood pressure. In this case, poststroke hypertension may aim to enhance the perfusion in the penumbra in order to save the tissue jeopardised by ischaemia. From this we derive the hypothesis that recanalisation and reperfusion of the penumbra will reverse the blood pressure elevation more rapidly than this occurs with persistent occlusion of the cerebral vessel.

Study aim: To evaluate the influence of vessel recanalisation achieved with intra-arterial thrombolysis in patients with acute ischaemic stroke on the blood pressure changes during the first 12 hours after treatment.

Methods/Patients: We retrospectively analysed blood pressure course in 102 consecutive patients with acute ischaemic stroke, who were admitted to our stroke unit between 1/2000 and 8/2002 and treated with intra-arterial thrombolysis. Blood pressure parameters were recorded on admission, within one hour after thrombolysis and 12 hours after treatment. Determination of recanalisation degree classified according to thrombolysis in myocardial infarction (TIMI) grades was based on cerebral arteriography performed by a neuroradiologist immediately after intra-arterial thrombolysis. We compared the group of patients with sufficient recanalisation (TIMI 2 and 3) with the group with insufficient recanalisation (TIMI 0 and 1).

Results: Overall, there was a significant decrease in systolic, diastolic and mean arterial blood pressure between admission and 12 hours after thrombolysis (p <0.001). Compared to those with sufficient recanalisation, patients with insufficient recanalisation showed a significantly higher systolic and mean arterial blood pressure 12 hours after thrombolysis (p = 0.003, 0.011 respectively). In the patient group not having received any antihypertensive treatment since admission (n = 88) sufficient recanalisation led to a significantly larger decline in systolic arterial blood pressure between admission and 12 hours after thrombolysis (p = 0.050).

Conclusion: These results suggest that elevated blood pressure levels after acute ischaemic stroke are inversely associated with the degree of recanalisation and may be a pathophysiological response to maintain or enhance perfusion of hypoperfused brain

tissue with impaired cerebral autoregulation. Prospective studies are needed to confirm this hypothesis.

A prospective study of hyperkinesias ipsilateral (IH) to acute stroke

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Background: Hyperkinesia ipsilateral to the lesion (IH), i.e. contralateral to hemiparesis is poorly studied.

Rationale: To study clinical associations of IH in acute stroke.

Methods: We prospectively studied 100 consecutive patients in their acute phase of stroke for IH, a syndrome defined by stereotypic increase of purposeless spontaneous activity, such as manipulations of objects, repetitive body manipulation or apparent purposeless exercising. We verified the association of IH with topography of strokes (cortical pre- and/or post-central, corona radiata, basal ganglia, internal capsule, thalamus, brainstem), age, sex, ischaemic or haemorrhagic aetiology and presence of sensory deficits or ataxia using chi-square tests and logistic regression analysis.

Results: IH showed significant associations with cortical stroke ($p = 0.022$), either precentral ($p = 0.009$) or postcentral ($p = 0.005$), as well as with lesions of the corona radiata ($p = 0.008$), internal capsule ($p = 0.046$), but surprisingly not in the basal ganglia or brainstem. Logistic regression analysis showed no significant topographic prediction of IH. Neither the ischaemic or haemorrhagic nature of lesion, age, changes in tone or reflexes nor ataxia were associated with the presence of IH, whereas being a woman (p 0.0034), the presence of contralateral sensory deficits ($p = 0.0001$) or contralateral autonomic dysfunction ($p = 0.004$) were significantly associated with IH.

Conclusion: IH is associated with cortical strokes, contralateral sensory deficits and dysautonomia, but not with strokes in basal ganglia, probably sharing similar sensory mechanisms with hyperkinesia due to loss of proprioception such as seen in pseudoathetosis, sensory alien hand or hyperconcern rather than hyperkinesis due to basal ganglia lesions.

Clinical characteristics in patients with stroke due to patent foramen ovale versus those with cardioembolic stroke

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Objective: Patent foramen ovale (PFO) is accepted as stroke aetiology in patients with otherwise cryptogenic stroke. Little is known of possible differences in clinical characteristics between patients with stroke due to PFO and cardioembolic stroke (CE).

Methods: All consecutive patients with stroke due to PFO documented by echocardiography as the sole identifiable stroke mechanism and those with CE stroke and a high risk source according to the TOAST criteria were ascertained for 9 years. We compared age, sex, modifiable vascular risk factors and clinical stroke syndromes (according to the OCSP classification) of both groups.

Results: Among 300 identified patients 125 patients had stroke due to PFO (30 with atrial septal aneurysm) and 175 patients had CE stroke. PFO patients (mean age ± SD: 53 ± 15.1 years) were significantly younger than CE patients (73 ± 11.4 years, $p < 0.0001$). The sex ratio did not differ between the PFO group (64% men) and the CE group (55% men) ($p > 0.1$). Hypertension (71 vs 29%) and diabetes (21 vs 6%) were more often present in CE patients than in PFO patients (both $p < 0.001$). In turn, smoking occurred more often among PFO patients (45%) than among CE patients (32%; $p < 0.05$). The most frequent stroke syndrome was partial anterior circulation syndrome in both groups. Interestingly, posterior circulation syndromes occurred more often among PFO patients (31%) than among CE patients (19%; $p < 0.05$), while total anterior circulation syndromes were more frequent in the CE group (21%) than in the PFO group (12%; $p < 0.05$).

Conclusion: Patients with strokes due to PFO were younger, were more often smokers and had less often hypertension or diabetes than patients with stroke due to major cardioembolic sources. PFO stroke patients more often had posterior circulation syndromes and less often total anterior circulation syndromes than patients with cardioembolic strokes.

Neuroprotection against permanent focal cerebral ischaemia by a cell-permeant and protease-resistant JNK inhibiting peptide

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Background: We have evaluated the neuroprotective effect of D-JNKI1, a cell permeant and protease-resistant peptide inhibitor of the c-Jun-N-terminal kinase inhibitor in permanent focal cerebral ischaemia in the mouse. This compound has recently been shown to be a potent neuroprotectant in transient focal cerebral ischaemia (Borsello et al., in press).

Methods: Male ICR-CD1 mice were subjected to permanent suture occlusion of the left middle cerebral artery. Regional cerebral blood flow was monitored in all animals. Three hours after ischaemia onset animals were treated with an intra-cerebro-ventricular injection of either D-JNKI1 or vehicle. Infarct volumes and neurological scores were measured 24 hours after ischaemia. A subset of animals was tested on a RotarodTM treadmill before and 24 hours after ischaemia. Data are presented as mean ± SD. Parametric data were compared with Student's T-test, neurological scores with the Mann-Whitney test.

Results: With D-JNKI1 administration 3 hours after ischaemia, there was a reduction in infarct volume from 161 ± 28 mm^3 ($n = 12$) to 85 ± 27 mm^3 ($n = 9$), ($p < 0.00001$). Neurological outcome was improved: median neurological score was reduced from 2.00 in vehicle treated animals to 1.25 in D-JNKI1 treated animals ($p < 0.05$). The performance on the Rotarod treadmill was also better in the D-JNKI1 treated group (72 ± 48% of baseline, $n = 6$) than in vehicle treated permanent ischaemia group (15 ± 10%, $n = 6$, $p < 0.05$).

Conclusions: These data suggest that JNK is mediating neuronal death induced by permanent occlusion of the middle cerebral artery in vivo and that D-JNKI1 is a potent neuroprotectant not only in mild, but also in severe cerebral ischaemia, with an extended therapeutic window.

Intra-arterial thrombolysis in ischaemic stroke: how many patients can be treated in routine clinical practice?

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Background: The short treatment window of 3 hours for intravenous thrombolysis (IVT) limits the number of stroke victims who can be treated. In multicentre surveys 1.6 to 6.8% of patients have received IVT and individual hospitals where special referring systems for stroke patients had been created to accelerate admission thrombolysed up to 18% of their patients. Intraarterial thrombolysis (IAT) can be applied up to 6 hours after symptom onset. Therefore, the question arises how many additional patients can be treated with IAT compared to IVT in routine clinical practice.

Methods: We prospectively collected data on indications advising for or against diagnostic arteriography and IAT of consecutive acute ischaemic stroke patients aged 16 to 80 years who had been admitted from July 1, 2001 to June 30, 2002 to our stroke centre.

Results: During the study period 230 patients (= 100%) aged 16 to 80 years with acute ischaemic stroke were admitted to the Inselspital Berne. The mean age was 62.2 ± 13.8 years. 39.6% were women. 151 patients (65.7%) were admitted directly and 79 (34.3%) referred from community hospitals. 76 patients (33.0%) arrived within 3 hours and 154 (67.4%) within 6 hours. Arteriography was performed in 74 patients (32.2%), IAT in 46 (20.0%) and IVT within 3 hours in 2 patients (0.9%). Of the patients with a NIHSS score on admission of ≥4 we were able to treat 42%. Patients referred from a

community hospital qualified significantly more often for angiography and IAT than direct admissions (58.2 vs 23.3% and 41.8 vs 15.2%, p <0.001).

81 patients presenting within 6 hours did not undergo arteriography and thrombolysis, mostly because of mild (n = 44) or rapidly improving neurological deficits (n = 13). 75 patients (36.6%) presented after 6 hours. 25 had strokes during sleep, 23 ignored their symptoms and 18 had fluctuating symptoms with uncertain time of stroke onset.

Conclusions: Every third patient underwent diagnostic arteriography and every fifth received IAT. In the hours 3 to 6 after stroke onset we treated as many patients as in the first 3 hours. The routine clinical use of IAT instead of IVT in our tertiary care centre resulted in a substantial increase in patients who received thrombolysis for their ischaemic stroke.

D-Dimer-Bestimmung zur Unterscheidung kardioembolisch und nicht kardioembolisch bedingter Hirnschlagursachen

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Für eine individuelle Sekundärprävention nach Hirnschlag ist die Unterscheidung zwischen kardioembolischer (CE) und nichtembolischer (nCE) Ursache wichtig. Die Diagnose eines kardioembolischen Hirnschlages setzt den technisch aufwendigen Nachweis einer möglichen kardialen Emboliequelle voraus. Bei allen Schlaganfällen spielen Hämostaseprozesse eine Rolle. Je nach Ätiologie bestehen Unterschiede im Ausmass und zeitlichen Ablauf der Gerinnungsaktivierung. Wir untersuchten, ob sich solche Unterschiede diagnostisch nutzen lassen.

Methode: Bei konsekutiven Patienten im Alter zwischen 16 und 80 Jahren mit erstmaligem ischämischem Hirnschlag wurde innerhalb von max. 12 h nach Symptombeginn bei der ersten Blutentnahme Blut für spätere Gerinnungsanalysen abgenommen. Die weitere Abklärung und Therapie wurde durch die Studie nicht beeinflusst. Nach Spitalentlassung klassierte ein Neurologe, der für die Laborresultate «geblindet» war, die Hirnschlagursache nach den TOAST-Kriterien.

Resultate: Zwischen 16.7.01 und 17.6.02 wurden 100 Patienten eingeschlossen: CE 55 (cardioembolic possible 41, probable 14) und nCE 39 (large artery disease 20, small vessel occlusion 6, evaluation negative 13). Ausgeschlossen wurden 6 Patienten mit unvollständiger Abklärung. CE Patienten waren signifikant älter (mean 65.6 vs 59.0 years, p = 0.002) und hatten leicht schwerere Schlaganfälle bei knapp signifikant höheren NIHSS-Scores (10.5 vs 7.6, p = 0.048). Es fanden sich bei CE signifikant höhere D-Dimere (mean 1128 vs 487 µg/l, p <0.001). Der Unterschied blieb nach Korrektur für Alter und NIHSS bestehen. Alle Patienten mit CE hatten D-Dimere >290 ug/l (Sens. 100%, Spez. 39%). Bei einem max. Zeitintervall von 6 h zwischen Symptombeginn und Blutentnahme steigt die Spez. auf 47% und bei 3 h auf 55%. Für TAT besteht ein Trend zu höheren Werten für CE bei einem max. Zeitintervall von 6 h (p = 0.07). Für F1+2 (p = 0.64) und Fibrinogen (p = 0.82) fanden sich keine Unterschiede.

Schlussfolgerung: Bei keinem der Patienten mit D-Dimeren <290 µg/l konnte eine kardioembolische Hirnschlagursache festgestellt werden. D-Dimere, wenn innerhalb weniger als 12 Stunden nach Symptombeginn gemessen, stellen somit einen wichtigen diagnostischen Parameter dar, um zwischen CE und nCE bedingtem Hirnschlag zu unterscheiden. Dies ist insbesondere bei jenen Patienten wertvoll, die bisher trotz aufwendiger Abklärungen als kryptogen bezeichnet werden mussten.

Seasonal variations in hospital admissions due to aneurysmal subarachnoid haemorrhage

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Introduction: In clinical practice, the occurrence of aneurysmal subarachnoid haemorrhage (SAH) often coincides with a particular time of season. Our objective was to examine seasonal variations in hospital admissions due to aneurysmal SAH.

Methods: The study population consisted of 489 patients with aneurysmal SAH who were admitted at the Department of Neurosurgery, University Hospital of Zurich, Switzerland, between 1st January 1996 and 31st December 2002. Statistical significance of seasonal variation was determined by applying Roger's r test.

Results: Statistically significant seasonal variation was only found among patients younger than 60 years, showing a first peak in spring and a second lower peak in fall (Roger's r = 6.89, p <0.05). A borderline significance was found in men younger than 60 years (Roger's r = 5.96, p = 0.051). A trend was observed in patients presenting with Fisher grade 1–2 (Roger's r = 5.70, p = 0.058).

Conclusions: Previous studies from different countries have shown significant seasonal variations, with the peak period for aneurysmal SAH differing widely. There appears to be some link between aneurysmal SAH and time of season or variations in weather conditions. Further investigations are desirable to evaluate which weather or climatic parameters correlate with SAH.

Rôle du Duplex-couleur transcrânien dans le diagnostic et le pronostic des AVC ischémiques sylviens

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Introduction: Plusieurs études récentes ont permis de démontrer l'utilité diagnostique du Duplex-couleur transcrânien dans la phase aiguë de l'AVC; peu de travaux toutefois se sont intéressés à sa valeur sur le plan pronostique. L'objectif de notre travail est d'évaluer grâce au Duplex-couleur transcrânien la corrélation entre le flux résiduel au niveau de la cérébrale moyenne et l'étendue de la lésion visualisée à la neuroimagerie.

Patients et méthodes: Notre série comprend 26 patients (12 hommes et 14 femmes) admis dans notre Hôpital en raison d'un déficit neurologique suspect d'AVC sylvien et évoluant depuis moins de 6 heures. Tous les patients bénéficient d'une exploration par Duplex-couleur transcrânien, d'un CT-scan cérébral (19 avec séquences angiographiques), et d'une une IRM cérébrale (20 patients), pratiquée dans les 7 premiers jours.

Le signal Doppler perçu au niveau de l'artère cérébrale moyenne (ACM), pouvant être normal, amorti ou absent est confronté aux résultats des examens neuroradiologiques.

Résultats: Douze patients présentent un signal normal de l'ACM; l'Angio-CT, réalisé seulement chez 7 patients, confirme la perméabilité de cette artère. L'IRM est normale dans 2 cas et chez un patient, n'ayant pas pu avoir d'IRM, le CT-scan est normal à 3 jours de l'événement aigu. L'IRM montre une lésion autre que sylvienne dans 3 cas, une lésion de type lacunaire dans 3 cas également et une lésion du territoire partiel sylvien chez 3 patients.

Sept patients présentent un signal amorti de l'ACM; l'Angio-CT réalisé chez 3 patients montre dans 2 cas une occlusion proximale de l'ACM et chez un patient une ACM perméable. L'IRM montre dans tous les cas une lésion partielle du territoire sylvien.

Quatre patients présentent un signal d'occlusion de l'ACM, confirmé à l'Angio-CT. L'IRM cérébrale montre une lésion de tout le territoire sylvien chez 2 patients et une lésion partielle dans 2 cas.

Trois patients sont exclus en raison de l'absence de fenêtre temporale.

Conclusion: Un signal Doppler amorti ou d'occlusion de l'ACM prédit de manière systématique une lésion ischémique intéressant tout ou une partie du territoire sylvien. En revanche, un signal normal témoignant de la perméabilité de l'ACM ne permet pas de prédire avec certitude l'étendue de la lésion. Ces résultats suggèrent un rôle potentiel du Duplex-couleur transcrânien dans l'approche diagnostique de l'AVC aigu ainsi que dans le processus décisionnel d'une intervention thérapeutique de type fibrinolyse.

Prognostic value of clinical and laboratory findings among stroke patients receiving intravenous thrombolysis

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Objective: To evaluate the prognostic value of initial clinical and laboratory findings as well as safety among ischaemic stroke patients receiving intravenous recombinant tissue plasminogen activator in a single stroke centre independently from clinical trials.

Methods: Initial clinical characteristics (i.e. demographic data, NIH stroke scale score [NIHSSS], time to thrombolysis, risk factors, blood pressure, temperature, antiplatelet pretreatment) and early laboratory results (i.e. levels of glucose, c-reactive protein and creatinine, INR, platelet count) were compared with the outcome variables "poor outcome" (i.e. mRS >2 including death) after 3 months or "intracranial haemorrhage" (symptomatic plus asymptomatic). As data source, we used the prospectively recorded thrombolysis register, which is part of the local stroke data base.

Results: We identified 84 patients, who received rt-PA between June 1998 and August 2003. Outcome was favourable in 48 (57%) patients, while 36 (43%) had a poor outcome, including 16 (19%) patients who died. Higher NIHSSS ($p < 0.001$), older age ($p < 0.05$), history of hypertension ($p < 0.05$) or coronary heart disease ($p < 0.01$) as well as increased creatinine levels ($p < 0.05$) were significantly associated with "poor outcome", while none of the other tested variables showed such an association (each $p > 0.05$). Interestingly, in patients aged 80 years or older 7/11 (64%) had a favourable outcome, which did not differ significantly from those <80 years old (41/73, 56%; $p > 0.05$). "Intracranial haemorrhage" occurred in 16 patients (19%). None of the tested variables were significantly associated with the occurrence of symptomatic or asymptomatic intracranial haemorrhages.

Conclusion: Although stroke severity and age were important prognostic variables, the beneficial effects of thrombolysis and its safety were also present in patients being 80 years or older. The association of increased creatinine levels with poor functional outcome deserves further analysis.

Cervical artery dissections above age 65

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Objective: To describe features of patients above age 65 with cervical artery dissections and to compare them to younger patients with dissections and to other elderly patients with stroke.

Background: Cervical arterial dissections usually occur in patients below age 60 and may be suspected if unilateral head and neck pain, minor preceding trauma or a Horner's syndrome is present.

Design/Method: The clinical and radiological data of all patients admitted to our stroke unit between January 2000 and November 2003 with a symptomatic cervical artery dissection on MRI and MRA were reviewed. Clinical and epidemiological characteristics of patients above age 65 were compared to patients below 65 and to elderly patients without dissection.

Result: Between January 2001 and October 2003, 3 patients aged 72, 76, 80 were diagnosed with retinal and cerebral stroke due to carotid (2) and vertebral (1) artery dissection. They represent 4.2% of all dissections and 0.4% of all patients above 65 admitted to the stroke unit during this period. The vertebral artery dissection was related to a minor trauma with a lateral C2 fracture. Significant atherosclerosis was found in one patient. One carotid siphon dissection was found by chance during an MRA study done for a scientific protocol. As compared to the younger patients with dissections, absence of pain and Horner's syndrome was noted in all 3 patients. As compared to a random sample of 100 patients above age 65, a similar number of vascular risk factors was present. All 3 patients were discharged home and were free of recurrences on oral anticoagulation after 3 months.

Conclusion: Cervical arterial dissections above age 65 are at risk of being missed because of their rarity and less characteristic clinical features. Their pathogenesis seems to be heterogeneous and not specifically related to atherosclerosis.

Perfusion-CT in transient hemispheric deficits

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Objective: To describe the prevalence and patterns of perfusion-CT abnormalities in patients with transient hemispheric dysfunction.

Background: Transient ischaemic attacks (TIA), transient global amnesia (TGA), migraine with hemispheric neurological deficits (MIG), partial onset seizures (EPI) and psychogenic deficits (PSY) can be difficult to differentiate from each other and from acute stroke.

Design/Method: Over a 2-year period, 46 patients with transient hemispheric dysfunction underwent perfusion-CT during or briefly after the resolution of the clinical deficit. Final diagnosis was made on clinical grounds, EEG (EPI, TGA), and delayed neuroimaging in all TIA and MIG. 3 patients were excluded due to uncertain final diagnosis and one due to movement artifacts during PCT.

Results: Focal hypoperfusion, found in 2/9 patients with TIA, was not predictive of new lesions on follow-up imaging. In 10 patients with EPI, focal hypoperfusion in the postictal period was found in one patient and hyperperfusion in two; both had a focal abnormality on EEG. One EPI patient with an extensive right MCA hypoperfusion after a secondarily generalised seizure was judged to have acute stroke and underwent iv-thrombolysis; MRI confirmed small ischaemic cortical lesions and recovery was complete. Focal hyperperfusion was also found in 1/9 patients with MIG in whom MRI was normal. All patients with TGA had normal PCTs although one had a minor stroke of the corpus callosum on MRI.

Conclusions: Focal hypoperfusion on PCT during or after transient hemispheric neurological deficits can be seen in TIAs and focal onset seizures, whereas hyperperfusion may occur in focal seizures or in migraine with aura. These patterns may help identify a specific diagnosis and treatment in transient hemispheric neurological deficits.

Subarachnoid haemorrhage due to ruptured dolichoectatic intracranial arteries: 6 cases

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Objective: To describe clinical, radiological and pathologic features of 6 patients with subarachnoid haemorrhage (SAH) due to spontaneous rupture of a dolichoectatic basilar artery.

Background: Dolichoectasia of intracerebral arteries may remain asymptomatic or cause compressive or ischaemic symptoms. There are only 11 well-described cases of SAH due to spontaneous rupture (for review see: Cerebrovasc Dis 2003;16:292).

Design/Method: We reviewed medical records of 5 major hospitals over a 5-year period (1998–2002) and found 6 cases with a median age of 70 (range 57–81). All but one had dolichoectasia documented by invasive or non-invasive angiography and 2 underwent autopsy.

Results: 5 of 6 patients were previously symptomatic from brainstem compression or ischaemia, with time between onset of symptoms and SAH varying between 3 months and 4 years. The sixth patient had a SAH as the first manifestation and died after a second SAH. Rapidly progressive symptoms before rupture were present in most, and thrombus formation was found in 5/6 patients. All patients had hypertension and most had significant leukoaraiosis. Basilar artery dolichoectasia with fusiform aneurysms were present in all 6 patients and thought to be the site of rupture. Additional dolichoectasia was found in the anterior circulation in 3 patients, unruptured saccular aneurysms in one and arteria lusoria in one.

Conclusions: We describe 6 elderly patients with SAH due to rupture of dolichoectatic basilar arteries. Multiple intracerebral vascular pathologies were present in most patients. Symptoms of aneurysm growth or clot formation within the dolichoectasia preceded its rupture in most cases and outcome was invariably fatal.

Prospective assessment of acute tone changes in acute stroke

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Background: Tone changes in the acute phase of stroke have never been studied prospectively.

Rationale: To study acute changes in tone contralaterally to the lesion during the first days of stroke in 100 consecutive patients with acute stroke.

Methods: 100 consecutive patients (median age ± IQR 74 ± 21, range 19–93, 51 women 80 ± 17, 49 men 70 ± 17) were studied prospectively in the acute phase of stroke for changes in tone according to Ashworth's scale and hypotonia. Associations of acute change in tone with topography (cortical pre- and/or post-central, corona radiata, basal ganglia, internal capsule, thalamus, brainstem), age, sex, ischaemic or haemorrhagic aetiology and presence of sensory deficits or ataxia were performed using chi-square tests and logistic regression analysis.

Results: Acute hypertonia in upper extremity (but not of the lower extremity) was significantly associated with strokes located in precentral cortex ($p = 0.042$), internal capsule ($p < 0.001$) and basal ganglia ($p = 0.021$). Logistic regression analysis shows that only internal capsule location is protective from hypertonia in the upper limb (OR = 0.15, 95% CI 0.03–0.80, $p = 0.026$). There was no association between hypertonia and the ischaemic or haemorrhagic nature of the lesions, age, sex, side of lesion, presence of autonomic dysfunction, sensory deficits or ataxia. A significant association was found with ipsilateral hyperkinesia ($p = 0.010$) and acute hyperreflexia ($p = 0.001$). Acute severe transient hypertonia was seen in 3 patients with parietal strokes and 2 with lenticulostriate artery strokes. Acute hypotonia limited to the upper extremity was also significantly associated with strokes in precentral cortex ($p = 0.042$), internal capsule ($p = 0.0001$) and basal ganglia ($p = 0.021$). Logistic regression analysis showed significant association between hypotonia of both upper and lower extremity and internal capsule lesions (OR 6.3, 95% CI 1.24–32.7, $p = 0.026$ and OR 9.63 95% CI 1.10–83.73, $p = 0.040$);there was no association between hyper- and hypotonia and autonomic dysfunction, ataxia, while a significant association was found with sensory deficits ($p = 0.032$), hyperreflexia ($p = 0.001$) and ipsilateral hyperkinesias ($p = 0.010$).

Conclusion: Acute hyper- and hypotonia seem to represent two facets of lesions in similar location along the descending motor pathways. The presence of sensory deficits seems to be determinant for developing acute changes in tone and hyporeflexia.

Extracranial-intracranial bypass in haemodynamically relevant ischaemic cerebrovascular disease

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Introduction: EC-IC bypass procedure has been the accepted technique to augment cerebral blood flow in patients with occlusive cerebrovascular disease since its introduction in 1967. Since the cooperative EC-IC bypass study in 1985, the number of bypass procedure has declined. The absence of documented haemodynamic status was one of the major flaws in this study. We present our clinical experience with EC-IC bypass procedure in selected patients with haemodynamically relevant ischaemic cerebrovascular diseases.

Patients and methods: 61 patients underwent EC-IC bypass surgery due to occlusive cerebrovascular diseases between 1998 and 2002. These included 33 patients with single or multiple vessel occlusive diseases. In the other 28 patients Moyamoya angiopathy was diagnosed. H_2O^{15}-PET examination with acetazolamide challenge was performed in 42 patients pre- and postoperatively. Clinical outcome based on a modified clinical outcome scale was followed up to 12 months (range 3–37 months).

Results: The COS was as follows: COS 1: cessation of preoperative ischaemic symptoms n = 34 (57.6%), COS 2: decreased frequency of ischaemic symptoms, no further stroke n = 17 (28.8%), COS 3: same frequency of ischaemic symptoms, no further stroke n = 1 (1.7%), COS 4: higher frequency of ischaemic symptoms, further stroke n = 4 (6.8%), COS 5: death n = 3 (5.1%). PET findings: improvement in baseline cerebral blood flow n = 28 (66.7%), unchanged n = 14 (33.3%). Improvement in cerebrovascular reserve capacity (CVR) n = 27 (64.3%), unchanged n = 24 (33.3%), worsened n = 1 (2.4%). 63.5% of patients without further stroke showed improved CVR at 12 months after surgery.

Conclusion: In our experience EC-IC bypass surgery in a selected group of patients with haemodynamically relevant cerebral ischaemia has proven to be beneficial. H_2O^{15}-PET examination using acetazolamide challenge remains indispensable in selection of these surgical candidates and proves to be valuable in evaluation of the postoperative perfusion status.

Cerebrovascular risk factors in the Swiss population

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Background and Purpose: Identification of the population at risk remains the best approach to reduce the burden of cardiovascular morbidity and mortality. We examined the prevalence of common vascular risk factors (RF) and their combinations in a large cohort of healthy Swiss persons.

Methods: Overall 4458 persons (1741 men and 2717 women, mean age 57.8 ± 15 years) underwent measurements of blood pressure (BP), total cholesterol (TC), blood glucose, body mass index (BMI) and smoking habits. Hypertension (HT), hypercholesterolaemia (HCh), diabetes mellitus (DM), overweight (OW), obesity (OB) and tobacco use (SM) were defined according to established international standards.

Results: OW was the most prevalent RF (50%), followed by HT (47%), HCh (33%), SM (13%) and DM (1.6%). The proportion of persons without RF was 16.8%, with 1 RF 35%, 2 RF 28.6%, 3 RF 3.4% and 4 RF 0.8%. OW was more prevalent in men than in women (53 vs 41%, $p = 0.02$). More men than women aged 41–50 years and 51–60 years had HT (49 vs 36%, $p = 0.01$, and 52 vs 42%, $p = 0.02$). The prevalence of HCh and DM did not show any sex-related differences. HT, OW and HCh were not only the most common single risk factors, but were also most likely to aggregate with each other.

Conclusions: The majority of Swiss people have one or two vascular risk factors. OW and HT are by far most common and are likely to aggregate with each other. A small modification of these two factors would reduce the incidence of stroke and myocardial infarction significantly.

Cardiac fibroelastoma: a rare cause or an innocent bystander in cryptogenic stroke?

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Background: Fibroelastoma (FE) is the third most common primary cardiac neoplasm with a predilection to cardiac valves that has the potential for embolisation in different vascular territories including the cerebral arteries. However, the data on the role of FE in ischaemic stroke are scarce. Therefore we assessed the frequency of FE in patients with first-ever cryptogenic stroke included in the Lausanne Stroke Registry (LSR).

Methods: We reviewed 754 first-ever cryptogenic stroke patients admitted to our community-based primary care centre between 1987 and 2001 and selected those with FE confirmed by transoesophageal

echocardiography (TEE). In all cases we assessed demographics, clinical findings, topography of infarcts, TEE findings and clinical outcome.

Results: Four patients with FE were identified (0.5% of cases) (2 men; mean age: 36.7; range: 33–42); 3 were smokers and one had a history of migraine. 3 patients had headaches at the onset of stroke. Two patients were somnolent, all had hemiparesis, one had transient upper extremity hemibalism, one had motor dysphasia and 3 had hemisensory deficit. Infarcts were localised in the anterior circulation territory and involved anterior superficial middle cerebral artery territory in two (in one anterior choroidal artery territory was additionally affected) and complete middle cerebral artery territory in the other two cases. In 2 patients FE was detected on mitral and in 2 on aortic valves. A thorough diagnostic work-up was negative except two cases in which PFO coexisted and paradoxical embolism was considered as a cause of stroke. In the other 2 patients FE was considered the only potential source of cerebral embolism. All patients received oral anticoagulants.

Conclusions: Valvular FE was rare and the causal relationship with ischaemic stroke was speculated in only 0.2% in our cryptogenic stroke patients. Considering the long life expectancy of young stroke survivors, the utility of long-term anticoagulation in cases with FE remains to be elucidated.

Unusual bilateral extracranial carotid and vertebral arteries dissection after chiropractic manipulation of the cervical spine

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Background: Spinal manipulation is an independent risk factor for vertebral artery dissection. However, dissection of the four extracranial arteries following spinal manipulation have very rarely been reported.

Patient and methods: A 35-year-old woman was admitted (27.11.03) for an acute aphasia (<24 hours) associated with headache. She had a history of cervical spine manipulation performed by an osteopath practitioner for neck pain two days before. The examination revealed a subcortical-type aphasia, mild central facial palsy with frontal signs, without Horner's syndrome. Whereas MR imaging confirmed acute and multiple deep (caudate, putamen), insular and frontal opercular ischaemic infarcts (DWI), there were not only signs of dissection of left internal carotid artery, but also of the contralateral carotid and of both vertebral arteries on MRA. She was treated with immediate heparinisation. Skin biopsy for investigation of a connective tissue disease was performed (results available in Jan. 2004).

Results: Whereas multiple cervical arteries spontaneous dissection involving three vessel account for less than 2% of cases in the largest published series, we found only one previously published case report of all four vessels dissection following chiropractic manipulation of the cervical spine, without evidence of an underlying collagen vascular disease.

Conclusions: Dissection of all four extracranial arteries may occur after chiropractic manipulation of the cervical spine.

Predictors of disabling fatigue in "non-disabling" stroke

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Background: Poststroke fatigue (PSF) is a frequent and complex phenomenon whose predictors are difficult to size.

Methods: We prospectively studied 90 patients with a first non-disabling stroke (NIHSS ≤3 at 6 m) in order to identify the relevant variables associated with PSF. At 6 months follow-up, patients were evaluated with the Fatigue Assessment Instrument (FAI), Hamilton Depression and Hamilton Anxiety Rating Scales, disability scales (Barthel, Rankin), NIHSS and a detailed neuropsychological battery. Personal data (age, sex) as well as MRI findings were also collected. Stepwise multiple regression analyses with FAI fatigue severity score as outcome measure were conducted.

Results: Being younger (<47) ($z = -2.08$, $p = 0.038$), severity of neurological impairment ($z = 1.94$, $p = 0.05$), left laterality of the lesion ($z = 1.82$, $p = 0.069$) as well as degree of anxiety (2.82, $p = 0.005$) were the best predictors of fatigue (FAI >2.63 corresponding to the median). When considering only the patients with a severe fatigue syndrome (FAI >4), the persisting predictors were left lesion, younger age and anxiety. With reference to anatomical aspects, we found that, in subjects with a lesion involving the cortex, an impairment in executive functions, divided attention and phasic alert as well as a left hemisphere damage and younger age were significant factors. For patients with lesions sparing the cortex, the degree of anxiety was the only statistically significant predictor ($z = 3.18$, $p = 0.001$).

Conclusion: Our data show that it is possible to highlight relevant factors associated with PSF, which may vary according to the lesion topography. While mood dysfunction is critical in patients without cortical involvement, specific cognitive impairment may be more important in the cases with cortical damage.

Ruptured aneurysms: the experience of an ISAT centre since randomisation stop

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Background: The International Subarachnoid Aneurysm Trial (ISAT) stopped randomisation in May 2002 and the results were published in October 2002. The department of Radiology and Neurosurgery of the CHUV participated in this study from 1999 on, as the only centre in Switzerland. This report illustrates the experience at the CHUV in treating ruptured aneurysms since the publication of ISAT.

Results: Over the last 12 months 45 ruptured aneurysms were treated in our departments: 33 aneurysms were in the anterior and 2 in the posterior circulation. Patients presented with a WFNS score of 1 in 34%, 2 in 27%, 3 in 7%, 4 in 14% and 5 in 18%. Overall 35 (77%) were treated by surgical and 10 (23%) by endovascular approaches. One aneurysm needed a combined surgical and endovascular treatment. Eight ruptures were associated with an intraparenchymal haematoma with mass effect. However, the main reason for performing surgical clipping was an angioanatomy unfavourable for endovascular treatment.

Conclusion: Despite the results of ISAT, in the daily practice a significant number of ruptured aneurysms still are best treated by surgical clipping. We strongly believe that aneurysm treatment is best performed when the management is decided for each patient based upon the careful analysis of the important angioanatomical features.

Prevalence of intracranial aneurysms and fibromuscular dysplasia in spontaneous carotid artery dissection

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Background and Objective: Patients with spontaneous dissection of the cervical internal carotid artery (ICAD) have been reported to have an increased prevalence of both intracranial aneurysms and fibromuscular dysplasia (FMD). This association has been used as argument for the presence of a common underlying arteriopathy of yet unknown origin in ICAD. The aim of this study was to determine the prevalence of intracranial aneurysms and FMD in patients with spontaneous ICAD.

Methods: A hundred and sixty-seven consecutive patients with ICAD who underwent catheter (CA) or MR (MRA) angiography, or both of the cerebral arteries were investigated for the presence of intracranial aneurysms and FMD.

Results: Ninety CA (with ± MRA in 19 cases) and 77 MRA alone were performed. No intracranial aneurysm was found, whereas 11 patients (6.6%) had signs of FMD.

Conclusions: The prevalence of FMD but not intracranial aneurysms is increased in patients with ICAD suggesting that different pathomechanisms are responsible for the development of FMD and ICAD compared to intracranial aneurysms.