7. Jahrestagung der Zerebrovaskulären Arbeitsgruppe der Schweiz
7e Réunion du Groupe suisse de travail pour les maladies cérébrovasculaires

Abstracts
Zürich-Oerlikon, 16. Januar 2003

Metabolic changes after $H_2{}^{15}O$-PET examination using acetazolamide in a patient with Moyamoya disease: a case report with review of further cases

C. Muroi, Y. Yonekawa, N. Khan, A. Pangalu, E. Keller

Zurich

Introduction: Moymoya disease is a unique cerebrovascular disease with a much higher incidence in Japanese than in Caucasians. Most cases appear to be sporadic, but the incidence of familial cases has been increasing. $H_2{}^{15}O$-PET examinations using acetazolamide have been considered to be indispensable for evaluating pre- and postsurgical cerebral haemodynamics. We report on a patient with severe postoperative ischaemic complications after $H_2{}^{15}O$-PET examination.

Case report: The patient showed a severe and prolonged metabolic acidosis after $H_2{}^{15}O$-PET examination using acetazolamide, which took place 13 hours before operation. She showed multiple postoperative infarctions on the not-operated side and died due to untreatable ICP elevations and brain herniation.

Discussion: The history of this patient with prolonged acidosis is analysed and the role of metabolic changes induced by $H_2{}^{15}O$-PET examination with acetazolamide challenge is focused. Although the precise mechanisms leading to multiple cerebral infarctions remain speculative, occurring metabolic acidosis may be an event we have to pay attention to in the perioperative treatment of patients with Moyamoya disease. Based on this case, we analysed the clinical records of further patients with Moyamoya disease. Patients who underwent surgery within 48 hours after $H_2{}^{15}O$-PET examination showed a significant lower intra- and postoperative pH and HCO_3^-. On the basis of our cases, we recommend that a time interval of more than 48 hours should be kept between the $H_2{}^{15}O$-PET with acetazolamide challenge and surgery.

Re-occlusion of carotid artery after endarterectomy due to heparin-induced thrombocytopenia with thrombosis

S. Hartmann, W. Korte, C. Fretz, W. Nagel, B. Weder

St. Gallen

Heparin-induced thrombocytopenia (HIT) is an immune-mediated prothrombotic drug reaction caused by platelet-activating antibodies that recognise complexes of platelet factor (PF) 4 and heparin. HIT is – beside massive bleeding – probably the most dangerous side effect of heparin exposure; it is estimated that up to 8% of patients exposed to unfractionated heparin will develop heparin/PF4 antibodies with or without a significant fall in platelet count.

We report a 78-year-old female, with a symptomatic high-grade left internal carotid artery (ICA) stenosis who has been treated with unfractionated heparin because of recurrent transient ischaemic attacks. A few hours after ICA endarterectomy, the patient showed focal seizures and a monoparesis of the right arm. Magnetic resonance (MR)-angiography indicated re-thrombosis of left ICA. A rapid fall of platelet count was detected, suggesting the possibility of HIT with thrombosis (HITT). The presence of anti-heparin/PF4 antibodies was established (ELISA). Heparin was immediately stopped, a hirudin derivative was started and the patient recovered after surgical revision of ICA.

In conclusion, HIT is an immune-mediated phenomenon that can occur after exposure to heparin or heparin-like substances. Since these antibodies can activate platelets, trigger platelet rich thrombi and thus thrombocytopenia, it can lead to potentially life-threatening conditions. Therefore, regular platelet counts during heparin therapy are necessary. Antibody assays are time consuming and some are technically demanding, but new fast and reliable assays are becoming available. If HIT is suspected, immediate cessation of heparin and all heparin-like substances is mandatory and alternate anticoagulation has to be installed.

Aggravation of ischaemic injury by *tissue*-plasminogen activator (t-PA): experimental evidence and underlying mechanisms

D. M. Hermann, E. Kilic

Zurich

Introduction: Thrombolysis is currently the only efficacious treatment of acute ischaemic stroke. Recently, concerns have been raised that the thrombolytic *tissue*-plasminogen activator (t-PA) may have detrimental side effects. Since such effects may be of major clinical relevancy, we conducted a series of animal experimental studies.

Methods: Male C57BL/6j mice were submitted to focal brain ischaemia, as induced by middle cerebral artery thread occlusions. At defined time points before, during and after vascular occlusion, t-PA was *i.v.* administered at various doses (0.2–10 mg/kg). Brain perfusion was measured by laser Doppler flowmetry (LDF) and cerebral blood flow (CBF) autoradiography. Brain injury was assessed histochemically 24 hours after reperfusion onset.

Results: Our results confirm previous findings that *i.v.* t-PA increases brain injury in non-embolic focal ischaemia. In addition, our data show that t-PA treatment is followed by a secondary hypoperfusion. Both the decrease in blood flow and increase in injury were efficaciously prevented by heparin (200 IU/kg). Heparin alone, on the other hand, neither influenced brain perfusion nor tissue damage. Besides, the NMDA receptor antagonist MK-801 (0.2 mg/kg) attenuated t-PA-mediated injury, but did not have any effect when given alone at this rather low dose. The effects of MK-801 were independent of haemodynamic changes.

Discussion and conclusions: The present studies confirm that the beneficial effects of t-PA may be compromised by unfavourable changes and furthermore reveal possible mechanisms involved in this so-called "t-PA toxicity". Our data point towards the necessity to develop add-on treatments, preventing both the haemodynamic deficits and injury increase. In the future, concerted efforts will be required in this field.

Endothelin B receptor null mutation prevents subarachnoid haemorrhage-induced cerebral vasospasm in the rat in vivo

J. Fandino, R. M. Rapoport, C. E. Geriepy, M. Yanagisawa, M. Zuccarello

Aarau

Background and purpose: Support for endothelin (ET)-1 mediation of subarachnoid haemorrhage (SAH) induced vasospasm is partly derived from ET_A receptor antagonist inhibition of spasm. While these findings suggest the lack of ET_B receptor involvement, this possibility remains. To investigate this possibility, we utilised the spotting lethal (sl) rat, which carries a naturally occurring deletion in the ET_B receptor gene that prevents functional receptor expression and also the tissue-specific ET_B transgene that allows normal enteric nervous system development.

Methods: Vertebrobasilar angiography was performed to determine baseline, followed by injection of autologous arterial blood into the cisterna magna. Follow-up angiography was performed on day 2 post SAH. Basilar artery spasm magnitude was calculated as percent change (mean ± SE) by comparison of pre- and post-SAH angiograms.

Results: SAH induced 13.1 ± 0.9% spasm in +/+ (wild type) rats (n = 3), which was similar to the spasm magnitude in Wistar rats (14.2 ± 1.0%; n = 14). In contrast, SAH did not elicit spasm in sl/+ (0.2 ± 0.5%; n = 5) and sl/sl rats (–3.8 ± 1.2%; n = 6). Control saline injections did not induce spasm. Mortality following SAH and angiography in +/+, sl/+ and sl/sl rats was high at 75, 72 and 55%, respectively, and was 25% in Wistar rats.

Conclusions: These results demonstrate that ET_B receptors are essential for SAH-induced spasm of the rat basilar artery.

Perfusion-CT assessment of ischaemic penumbra within 3 hours and before intravenous thrombolysis of hyperacute stroke

M. Reichhart, M. Wintermark, G. Van Melle, B. Yersin, P. Maeder, R. Meuli, J. Bogousslavsky

Lausanne

Background: Ischaemic penumbra can be measured by perfusion-/diffusion-weighted imaging, but this is usually not feasible within 3 hours. We studied the feasibility and prognostic accuracy of penumbra assessment by perfusion CT before thrombolytic treatment (TT) and within 3 hours of stroke onset.

Methods: Penumbra and vessel patency were assessed on admission by perfusion CT and CT-angiography, respectively. Regional cerebral blood volume threshold of 2.5 ml/100 g was used to distinguish the penumbra (higher) and the infarct (lower). The Lausanne Stroke Index (LSI) was calculated by dividing the volume of the penumbra by that of the penumbra + infarct.

Results: TT was initiated within 148 ± 30 minutes in 35 patients, while perfusion CT was performed within 114 ± 45 minutes of stroke onset in 34 patients. A penumbra was found in 31 (91%) patients (mean LSI, 66 ± 23%) and ICA/MCA occlusion in 27 (79%). Fourteen patients (40%) had a favourable outcome (3-month NIHSS ≤1) with arterial reopening in over half of the patients (57%; positive predictor of outcome, P = 0.001); in 12 of these, a strong correlation was found between admission penumbra (mean LSI, 73 ± 18%) and 24-h NIHSS improvement (70.5 ± 16.5%; p <0.001), whereas the volume of the perfusion CT-predicted infarct corresponded exactly to the final infarct size.

Conclusion: Ischaemic penumbra can be measured by perfusion CT before thrombolysis and within 3 hours of stroke onset, and allows early prediction of 24-hour NIHSS improvement and final infarct size in patients with arterial reopening.

Intra-arterial thrombolysis with urokinase in acute basilar artery occlusion: clinical and radiological predictors of outcome and recanalisation

M. Arnold, K. Nedeltchev, R. W. Baumgartner, L. Remonda, G. Schroth, T. J. Loher, F. Stepper, M. Sturzenegger, H. P. Mattle

Berne, Zurich

Background: This study aimed to define predictors of recanalisation and clinical outcome in patients with acute basilar artery occlusion (BAO) treated with local intra-arterial thrombolysis (LIT).

Methods: At presentation, vascular risk factors, the severity of the neurological deficit by the National Institute of Health Stroke Scale (NIHSS) and radiological findings were assessed. Outcome was evaluated by the modified Rankin scale (mRS) after 3 months and was quantified as good (mRS 0–2), poor (mRS 3–5) or dead (mRS 6).

Results: Forty consecutive patients with a median baseline NIHSS of 18 were included. The average time from symptom onset to treatment was 329 minutes (range 138–660). Outcome was good in 14 patients (35%), poor in 9 (23%) and 17 patients (42%) died. Two symptomatic cerebral haemorrhages (5%) occurred. Recanalisation of the BA was achieved in 32 patients (80%); it was partial in 24 (60%) and complete in 8 patients (20%). A multivariate logistic regression analysis showed that a high baseline NIHSS score (p = 0.002) was an independent predictor of poor outcome or death. Recanalisation was associated with a good outcome (p = 0.007) and was more frequent in patients treated within 6 hours of symptom onset (p = 0.011) and in patients with a dense BA sign on initial CT scan (p = 0.022). In an univariate model tetraplegia (p = 0.002) and coma (p = 0.004) were associated with a poor outcome or death.

Conclusions: The results of this study indicate that a low baseline NIHSS and vessel recanalisation are associated with a good outcome. When LIT is initiated early and when a dense BA is visible on CT, recanalisation is more likely to be successfully achieved.

Pre- and in-hospital delays from stroke onset to intra-arterial thrombolysis

K. Nedeltchev, M. Arnold, C. Brekenfeld, J. Isenegger, L. Remonda, G. Schroth, H. P. Mattle

Berne

Background and purpose: Thrombolysis for treatment of acute ischaemic stroke should be administered as fast as possible after symptom onset. The aim of this study was to examine in our tertiary care centre the time intervals preceding intraarterial thrombolysis (IAT) in order to accelerate and optimise the management of acute stroke.

Methods: Between January 1, 2000 and April 30, 2002, 597 patients with acute stroke were admitted to our stroke centre. 148 patients underwent diagnostic arteriography and 100 (16.8%) received IAT. For all of them, we prospectively recorded and analysed the time of symptom onset, admission, CT and/or MRI scan, diagnostic arteriography and, if performed, IAT.

Results: The mean time to arrival in the emergency room was 99 minutes for patients who were admitted directly (Berne patients), 127 minutes for those who were referred from community hospitals without a CT scanner (non-Berne/-CT patients) and 210 minutes for patients from hospitals with imaging facilities (non-Berne/+CT patients). The mean delay from symptom onset to treatment was 234 minutes for Berne patients, 269 minutes for non-Berne/-CT and 302 minutes for non-Berne/+CT patients. The patients from the last group needed longer to receive IAT than did patients who were admitted directly (p = 0.002) or who were transferred from a hospital without a CT scanner (p = 0.03).

Conclusions: This prospective study indicates that direct referral without prior imaging at community hospitals saves time until IAT. In addition, our in-hospital delay preceding IAT is longer than the delays reported for intravenous thrombolysis and leaves room for improvement.

Transient amnesia and bilateral anterior corpus callosum infarcts

P. Michel, J. Ghika, Ph. Maeder, J. Bogousslavsky

Lausanne

A 62-year-old hypertensive nurse woke up with rotational vertigo and gait instability, lasting for 45 minutes, immediately followed by an episode of transient global amnesia lasting for 16 hours. No other abnormalities were found on acute examination than profound anterograde and retrograde amnesia going back for about 1 year.

Cranial CT 6 hours and EEG 24 hours after onset of the symptoms were normal. MR-imaging at 48 and 72 hours showed two small lesions on diffusion and T_2-weighted images, localised in a mirror-like fashion in the anterior corpus callosum, compatible with acute ischaemic lesions in the territory of the pericallosal arteries (anterior cerebral artery). Temporal lobes and limbic structures were normal. No arterial occlusion or stroke aetiology was found on MR-angiography, extra- and intracranial Doppler studies, transthoracic echocardiography and testing for hypercoagulable states. A tendency for bruising and metrorrhagia since adolescence was also present in two of her daughters and was explained by a genetic disturbance of platelet aggregation (different from von Willebrand's disease). Despite a complete recovery on neuropsychological exams, the patient developed a moderate prolonged depression since.

Conclusions: This case shows unusual ischaemic MRI finding in a patient with transient global amnesia and suggests a role of the anterior corpus callosum in memory storage and retrieval.

Perfusion-CT guided intravenous thrombolysis after 3 to 6 hours: feasibility and safety study

P. Michel, M. Wintermark, F. Vingerhoets, R. Meuli, J. Bogousslavsky

Lausanne

Purpose: This study's purpose is to show the feasibility and safety of using a newly developed perfusion-CT-software [1] to select patients with a favourable penumbra/core-ratio for intravenous thrombolysis in the 3- to 6-hour window after onset of acute ischaemic stroke.

Design: To be included, the patients need to show a minimal penumbra size for a given infarct (core) size on the perfusion-CT, according to a linear progressive cut-off table. This cut-off model was designed for maximal potential benefit: the smaller the core size is (and thus the smaller the theoretical bleeding risk), the smaller the lower limit of the penumbra needs to be for inclusion. The maximal upper size for inclusion is a core of 30% of the MCA territory (where penumbra must be at least 60%). The trial will be stopped after 4 symptomatic haemorrhages or after all 12 patients are enrolled.

Planned sample size: 12.

Population studied: Patients 18 to 80 years with acute carotid territory stroke giving informed consent, with a NIHSS severity of 6–22 and a CT-hypodensity of less than 30% of the MCA-territory.

Interventions: Treatment modalities, exclusion criteria and blood pressure management are identical to the ones in the NINDS rt-PA for acute ischaemic stroke study [2], except for the 3- to 6-hour window.

Outcome measures: The primary endpoints are symptomatic intracranial haemorrhages at 24 hours and mortality at 7 and 90 days. Secondary endpoints are independence at 90 days (mRS 0–2), Barthel index at 90 days, NIHSS at 24 hours, 7 and 90 days, final infarct size and recanalisation rate at 24 hours by angio-CT.

Statistical methods: Rates of primary outcomes are compared with the rates obtained in the NINDS (0–3 hours) and ECASS (0–6 hours) [3] studies as well as with our own experience of the 35 most recent patients thrombolysed within 3 hours.

Trial status: The trial was activated on November 19, 2002. Enrollment expected over 9 months, follow-up 3 months for each patient. Patients screened to date in the first 5 days (november 22, 2002): 2. Patients enrolled to date: 1.

References

1 Wintermark M, Reichhart M, Thiran J-P, Maeder P, Chalaron M, Schnyder P, et al. Prognostic accuracy of cerebral blood flow measurement by perfusion computed tomography, at the time of emergency room admission, in acute stroke patients. Ann Neurol 2002;51: 417–32.

2 Anonymous. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med 1995;333: 1581–7.

3 Hacke W, Kaste M, Fieschi C, von Kummer R, Davalos A, Meier D, et al. Randomised double-blind placebo-controlled trial of thrombolytic therapy with intravenous alteplase in acute ischaemic stroke (ECASS II). Second European-Australasian Acute Stroke Study Investigators. Lancet 1998; 352:1245–51.

Antithrombotic drugs for carotid artery dissection, a Cochrane review, update

Ph. Lyrer, S. Engelter

Basel

Background: Extracranial internal carotid artery dissection can lead to occlusion of the artery and hence cause an ischaemic stroke. It is the underlying stroke mechanism in approximately 2.5% of all strokes and the second leading cause of stroke in patients younger than 45 years of age. Antithrombotic agents (heparin, oral anticoagulants or antiplatelet drugs) may prevent arterial thrombosis in eICAD, but these benefits may be offset by increased bleeding.

Objectives: To determine whether antithrombotic drugs (antiplatelet drugs, anticoagulation) are effective and safe in treatment of patients with extracranial internal carotid artery dissection and which is the better treatment.

Methods: We searched the Cochrane Stroke Group Trials Register (last searched 3rd of October 2002). In addition we performed comprehensive searches of the Cochrane Controlled Trials Register (Cochrane Library Issue 3, 2002), Medline (January 1966 to June 2002) and EMBASE (January 1980 to June 2002) and checked all relevant papers for additional eligible studies. Selection criteria: randomised controlled trials, controlled clinical trials assessing the efficacy of antiplatelet drugs or anticoagulants in the treatment of extracranial carotid artery dissection. For non-randomised trials, case series (studies) that reported on antithrombotic treatment with at least 4 patients were eligible. All trials and studies were assessed for eligibility. Data from all eligible studies were extracted independently by two reviewers. Disagreements were resolved by discussion. Data on the primary outcome measures were extracted systematically. These were: all deaths, vascular deaths and disability. Secondary outcomes were: first stroke occurrence, stroke recurrence, any stroke during reported follow-up, extracranial haemorrhage and intracranial haemorrhage. The first choice treatment was taken for analyses.

Results: No randomised trials and 64 case series (including 893 treated patients) were identified. No reliable comparisons of antiplatelet drugs or anticoagulants with control were available. 26 eligible studies including 327 patients (who either received antiplatelet drugs or anticoagulants) were included in the analysis. There was no significant difference in odds of death comparing antiplatelet drugs with anticoagulants, odds ratio ([OR] 1.59, 95% CI 0.22–11.59). There was also no significant difference in the odds of being alive but disabled (OR 1.7, 95% CI 0.64–4.51). Few intracranial haemorrhages (0.5%) were reported for patients on anticoagulants, none for patients on antiplatelets.

Conclusions: There were no randomised trials comparing either anticoagulants or an-

tiplatelet drugs with control. There is, therefore, no evidence to support their routine use for the treatment of extracranial internal carotid artery dissection. There were also no randomised trials that directly compared anticoagulants with antiplatelet drugs and the reported non-randomised studies did not show any evidence of a significant difference between the two. We suggest that a randomised trial including at least 1400 patients in each treatment arm with this condition is clearly needed.

Antiplatelet therapy for preventing stroke after carotid endarterectomy – a Cochrane review

S. Engelter, Ph. Lyrer

Basel

Background: Antiplatelet drugs are effective and safe in a wide variety of patients at high risk of vascular ischaemic events. Among patients undergoing vascular surgical procedures, these agents significantly reduce the risk of graft or native vessel occlusion. In this context we wished to examine their effects in patients after carotid endarterectomy (CEA).

Objectives: The objective of this review was to evaluate whether antiplatelet agents are beneficial after endarterectomy of the internal carotid artery.

Methods: We searched the Cochrane Stroke Group Trials Register (last search October 1, 2002). In addition we performed comprehensive searches of the Cochrane Controlled Trials Register (Cochrane Library Issue 3, 2002), Medline (January 1966 to September 2002) and Embase (January 1980 to September 2002), and checked all relevant papers for additional eligible studies. We selected randomised, controlled, unconfounded trials comparing antiplatelet agents with control after carotid endarterectomy in symptomatic or asymptomatic carotid stenosis of different degrees. Antiplatelet medication had to be started within a range of three months before carotid endarterectomy and three months after surgery. Treatment duration had to be at least 30 days. Follow-up should be at least three months. Two reviewers selected trials for inclusion, assessed trial quality and extracted data independently from each other and blinded for the authorship. From each trial we extracted, first the number of patients originally allocated to each treatment group, and, second the number of patients who met the criteria for each outcome (intention-to-treat analysis). We calculated a weighted estimate of the odds for each outcome event across studies using the Peto odds ratio method.

Results: Six trials involving 907 patients were identified. For "stroke (all causes)" the Peto odds ratio of 0.58 with a 95% confidence interval (CI) of 0.27–0.98 indicated a statistically significant benefit in favour of antiplatelet drugs (p = 0.04). Concerning "death (all causes)", "vascular death", "stroke or vascular death", "serious vascular events", "dependency", "myocardial infarction", "major extracranial haemorrhage", "local haemorrhage requiring surgery", "restenosis", TIA or "amaurosis fugax", neither any benefit nor any hazard of antiplatelet drugs could be shown. For the outcome events "intracranial haemorrhage", "ischaemic stroke" and "occurrence or progression of contralateral stenosis", data were either too sparse for meaningful analyses or not available at all.

Conclusions: Although the data were limited, the estimates of treatment effects among patients undergoing carotid endarterectomy were similar to those observed in high-risk vascular patients in general. The beneficial effect of antiplatelet drugs in reducing the outcome "stroke of any cause" might be due to chance. Alternatively, it may indicate that among patients after carotid endarterectomy, antiplatelet agents are more efficacious for reducing stroke than for other vascular outcome events. There was no evidence for any special safety concerns, especially regarding bleedings.

Carotid plaque analysis: comparison between visual and GSM evaluation

I. Mayor, S. Momjian, P. Lalive, R. Sztajzel

Genève

Recent studies have demonstrated that besides degree of carotid stenosis, plaque morphology may play an important prognostic role in the occurrence of cerebrovascular events. Hypoechoic and heterogeneous plaques in particular seem to carry an increased risk of subsequent stroke. Most of these trials emphasising the relationship between plaque echostructure and stroke risk are based on high resolution ultrasound using a visual method of classification, considered thus far as subjective. More recently, several studies have suggested that the computerised measurement of the grey-scale median (GSM) may evaluate carotid plaque echogenicity more objectively and accurately than the visual method. However, this technique, although very suitable for research purposes, requires a separate equipment, is time consuming and therefore difficult to apply in daily clinical practice. We sought to compare these two types of evaluations in order to determine how valuable and reliable the visual approach is and how confident we can be when assessing our diagnosis on this method.

Patients and methods: We studied 68 consecutive patients, with 86 carotid bifurcation plaques, 30 symptomatic and 56 asymptomatic, causing 30 to 99% stenosis on duplex scanning. We assessed the grey-scale median (GSM) of these plaques and compared it to the visually evaluated echogenicity using the five-type classification system with vessel lumen and adventitia as reference structures. Plaque heterogeneity was also studied in a subgroup of 47 patients with 60 carotid stenoses by comparing visual analysis with the GSM method (difference between the GSMs of the most echogenic and the most echolucent areas within each plaque).

Results: The mean GSM value of the plaques increased with the plaque type (type 1, 44; type 2, 55; type 3, 100; type 4, 109; type 5; 159). The difference of echogenicity between the five types of plaques was statistically significant ($p < 0.02$). The overlap between the distribution of the GSM values of each group was small. The correlation between the plaque type and the mean GSM of the carotid plaque in each group was rho = 1 (Spearman; $p < 0.05$). Visual evaluation tended to be more accurate for moderate and severe (50–99%) than for mild stenosis (30–49%). Regarding plaque heterogeneity, we found a good concordance between visual analysis and heterogeneity assessment by the GSM method.

Conclusion: Our results suggest that the visual evaluation of plaque echogenicity and heterogeneity based on the five-types classification correlates well with the computerised measurement of the GSM. Furthermore, assessment of reference structures may also contribute to improve the interobserver agreement. The visual evaluation and classification of carotid plaque therefore remains a valuable method in daily clinical practice.

Detection of emboligenic dysrhythmia for secondary stroke prevention

D. Jabaudon, R. Sztajzel

Clinique Neurologie,
Hôpitaux Cantonaux Universitaires, Genève

Atrial fibrillation (AF) constitutes the single most frequent cause of cardiogenic stroke and underlies at least 10% of all strokes. Despite the prevalence of this disorder, AF remains underdiagnosed, as it usually causes no symptoms and often displays an intermittent pattern that may not appear on a single ECG recording. Identification of AF as the genuine cause of a stroke is, however, essential, as this often implies the introduction of anticoagulant therapy for secondary prophylaxis.

In this study, we prospectively examined the impact of prolonged ECG recording techniques (i.e. Holter and R-test) in determining the aetiology of a stroke and identifying AF.

In 180 consecutive patients admitted to our clinic with an acute neurological deficit, we performed a 24-hour ECG recording

(Holter), as well as an 8-day outpatient ambulatory ECG event recording (R. test). Additional work-up included intra- and precerebral Doppler ultrasound, MR imaging and, in selected cases, echocardiography.

The contribution of long-term ECG recording in the detection of cardioembolic strokes will be discussed, as well as the input of these ambulatory techniques in determining stoke aetiology in various subpopulations of patients.

Symptomatic carotid dissection associated with spontaneous aortic dissection, case report

T. Eugster[a], Ph. Lyrer[b], L. Gürke[a], P. Stierli[a]

[a]*Aarau,* [b]*Basel*

Einleitung: Die symptomatische Dissektion der Aorta mit Einbezug der Arteria carotis ist eine akut lebensbedrohende Erkrankung. Wir stellen einen 54jährigen Patienten vor mit symptomatischer Dissektion der A. carotis bei Typ-A-Dissektion der Aorta.

Verlauf: Der primär kreislaufstabile und neurologisch unauffällige Patient erhielt einen Ersatz der aszendierenden Aorta. Postoperativ soweit bei unruhigem Patienten beurteilbar neurologisch unauffällig. Am 6. postoperativen Tag bei installierter oraler Antikoagulation Auftreten eines Schlaganfalles mit Plegie der linken Hand. Die Duplexuntersuchung zeigte die bereits präoperativ bekannte Dissektion der Carotis mit zerebraler Minderperfusion aufgrund einer Einengung des wahren Lumens. Die Indikation zur Operation wurde gestellt.

Diskussion: Anhand des hier vorgestellten Falles wird unter Berücksichtigung der Literatur die Therapie der Carotisdissektion bei diesen speziellen Indikationen diskutiert.

Anger in, anger out: experience and expression of anger in acute stroke

L. Urben, J. Bogousslavsky

Centre Hospitalier Universitaire Vaudois, Lausanne

Objective: To recognise experienced and expressed anger in acute stroke, to study its relationship to clinical and epidemiological parameters and to other behavioural changes post-stroke, and to gain further insight into the mechanisms of anger and aggressiveness in humans.

Methods: Among patients of the prospective Lausanne Emotion in Acute Stroke Study, we identified those with a total score of ≥4 in the items "angry", "aggressive", "rebellious" and "revolted" of the Emotional Behaviour Index (EBI), an observational scale administered by the nursing personnel on 4 consecutive days after admission. Additionally, patients reported anger in a semi-directed questionnaire and were divided into three subgroups: (a) expressed anger alone (EAA: positive EBI), (b) internally experienced anger alone (IAA: positive questionnaire) and (c) expressed and internally experienced anger (EIA: positive EBI and questionnaire). Patients were evaluated with short versions of the Hamilton depression (HDRS) and anxiety (HDAS) scales. The association of anger with epidemiological data, lesion location, type and severity of neuro- and neuropsychological deficits, and mood disorders was assessed by uni- and multivariate analyses.

Results: We identified 58/277 patients (21%) (EAA: 17, IAA: 34, EIA: 7; mean age: 67.5 ± 12.3). Epidemiological data, except sex (women: 59% vs 43%, $p < 0.03$), were similar in patients presenting anger and the rest of the cohort. No correlation was found to lesion location. Only 17% of IAA patients also had EAA and conversely, only 30% of those with EAA had IAA. Eight EAA patients (47%) presented dysphasia compared to 6 (18%) IAA patients ($p = 0.01$). The severity of the neurological deficit on admission (NIHSS) was positively correlated with the EBI score (Spearman $r = 0.47$; $p < 0.05$). All subgroups of patients with anger had a more severe neurological deficit on admission (EAA: 11 ± 2.8, IAA: 7.5 ± 1.7, EIA: 8 ± 2.2, controls: 6 ± 1.6; Mann-Whitney $p < 0.05$). Anger was associated with depressive mood (HDRS ≥8; 14 vs 5.5%, $p = 0.034$) and anxiety (HDAS ≥4; 29 vs 12%, $p = 0.012$).

Conclusion: Anger is frequent in acute stroke patients but the association between internally experienced and expressed anger is low. Both an observational scale and self-reports are necessary for its assessment. Anger, though distinct from a catastrophic reaction, is linked to the severity of the neurological deficit and related to early post-stroke depressive mood and anxiety.

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

T. Karapanayiotides[a], P. Ruchat[a], G. Darbellay[b], V. Kemeny[a], B. Piechowski-Jozwiak[a], J. M. Vesin[b], P. A. Despland[a], J. Bogousslavsky[a], G. Devuyst[a]

[a]*Lausanne CHUV,* [b]*EPFL*

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 (G) or solid (S) 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 S during the dissection [median (percentiles), S:3(1,6), G:0(0,1); Wilcoxon signed rank $p = 0.003$] and clamping [S:1(1,3), G:1(0,1); $p = 0.04$] stages of the CEA and postoperatively [S:2(1,5), G:0(0,0); $p < 0.0001$]. During declamping, MES are predominantly gaseous [S:1(0,2), G:5(2,8); $p < 0.0001$]. MES are more often detected in S compared to A patients preoperatively (42 vs 23%; $p = 0.01$), during dissection (69 vs 51%; $p = 0.04$) and postoperatively (86 vs 72%; $p = 0.05$). MES detection in the preoperative period (OR:1.7, 95% CI: 1.1–2.6) and during dissection (OR:1.9, 95% CI: 1.3–3.1) were associated with intraoperative ischaemic complications (TIAs: 4, brain infarcts: 3). The total counts of solid emboli during the whole operation [37(18.75,129.5) vs 6(1,14); $p < 0.002$], the counts of all emboli regardless of their nature during the whole operation [49.5(25,138) vs 15(6,28); $p < 0.005$], during the dissection [10(2,19) vs 0.5(0,5); $p = 0.025$] and clamping [6(1,18) vs 1(0,3); $p = 0.025$] were higher in these 7 patients compared to those who had an uncomplicated CEA.

Conclusions: High loads of solid microemboli during the total CEA is a strong predictor of perioperative brain ischaemia while loads of solid emboli during specific stages of the operation are not. High total loads of microemboli regardless of their nature are equally strong predictors of ischaemia.