

Cerebral Vascular Disease 7

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CEREBRAL VASCULAR DISEASE 7

Proceedings of the World Federation of Neurology
14th International Salzburg Conference,
September 28 – October 1, 1988

Editors:

J.S. Meyer, H. Lechner, M. Reivich and J.F. Toole



重医附一院



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INTERNATIONAL SALZBURG CONFERENCE ON CEREBRAL VASCULAR DISEASE

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organized by

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Welcoming address

Prof. H. Lechner, M.D., Chairman

May I welcome you on behalf of the Organizing Committee on the Salzburg Conference. My welcome is addressed to all our members but on behalf of the Local Committee I would especially like to address our guests from different parts of the world who have come to take part in our conference. I also wish to give special thanks to our sponsors: Bayer Leverkusen, Heilmittelwerke Wien, Hoechst Wiesbaden, Solco Basel, Boehringer Ingelheim and Sandoz Wien.

May I remind you that the present conference is taking place in the same building and conference rooms as the first Salzburg Conference which took place in 1960 in the Mozart Room. The organizational basis for this conference was begun in 1959 at the Marseille Symposium dealing with the topic 'Brain and Ischemia' and at this meeting Prof. Meyer, Prof. Loeb, Prof. Agnoli, Prof. van der Drift and myself decided to organize a biannual conference, which should include all aspects of Neuroscience and should serve as a platform for interaction and informational exchange. On this organizational basis the first Salzburg Conference was arranged in 1960 by Prof. Bertha, Dozent Eichhorn and myself. The members who have attended regularly formed the Board of the Founding Members, which later in cooperation with the World Federation of Neurology became the Executive Board of the Salzburg Conference which meets annually. From the beginning the published Proceeding of the Salzburg Conference became well-known throughout the world and there are few scientific libraries in the world, where you cannot find the various volumes of all the Salzburg Conferences.

Before ending this address of welcome I wish to thank all who have collaborated with me and given their support to fulfill the idea of the founders of the Salzburg Conference namely to create a forum for international exchange of research on Cerebrovascular Diseases.

Prof. Dr. H. Lechner
Head of the Department of Neurology
and Psychiatry, Graz, Austria

Introduction remarks to members attending the World Federation of Neurology Research Group on Cerebrovascular Disease's 14th International Salzburg Conference

Professor John S. Meyer, M.D., Chairman

Fellow members, welcome to the 14th biennial meeting of the Salzburg Conference on Cerebrovascular Disease. These Conferences were originally founded 28 years ago, in 1960, by the late Professor Hans Bertha, who was then Professor and Chairman of the Department of Neurology and Psychiatry of the University of Graz. His able associates, who worked as the organizing secretaries of the Congress, were the late Otto Eichhorn and our genial and ubiquitous host, Professor Helmut Lechner. The Salzburg Congress was organized to communicate new achievements in research concerned with the physiology of the cerebral circulation and its metabolism together with the pathology, pathogenesis, diagnosis, prevention and treatment of cerebrovascular disease and stroke. The Salzburg Conferences have always been designed to be a meeting place or forum for the international interchange of ideas on an interdisciplinary basis between scientists and practitioners interested in all aspects of the cerebral circulation and its disorders. The twin organizing secretaries, Lechner and Eichhorn, did such a good job achieving these goals that the two of them were affectionately known under the pseudonym, 'Lechhorn', which conveniently named both of them and did not put a strain on the recent memory of the senior scientists! Unfortunately, and indeed sadly, both Professor Bertha and Otto Eichhorn have died, but on a happier note, we still have Professor Helmut Lechner, who ably succeeded Bertha as Chairman of Neurology and Psychiatry at the University of Graz. Fortunately, Lechner has sustained the level of excellence in organizing these international meetings in Salzburg which has led to international scientific acclaim.

The Salzburg Conference is a closed conference. Attendance is based on priorities of excellence attained by the manuscripts submitted and judged by peer review of the Scientific Advisory and Executive Committees. The membership does not wish to exclude anyone, but does intend to fulfill the intention of the founders, which is to provide adequate time for real discussion and interchange of ideas between participants. This requires that the number attending be limited so that we may sit together and talk to one another in the manner of a colloquium and symposium rather than a coliseum. It also means that the number of papers must be limited to allow ample time for discussion. This, we believe, is a desirable format that should be preserved. Results of the scientific presentations and discussions are made available to a wider audience through publication of the proceedings as promptly as can be achieved by your editorial committee, working with *Excerpta Medica* as the publisher.

On behalf of the four secretaries of the World Federation of Neurology Research Group on Cerebrovascular Disease, Professors Helmut Lechner of Austria, Carlo Loeb of Italy, Lindsay Simon of the United Kingdom, and myself representing the United States of America, on behalf of our local organizing committee and on behalf of our sponsors, welcome again to this beautiful and historic city and, it is hoped, to a worthwhile, educational and instructive 14th Salzburg Conference.

John Stirling Meyer, M.D.
 Director, Cerebrovascular
 Research Laboratories,
 VA Medical Center, Houston and
 Professor of Neurology
 Baylor College of Medicine

Contents

EPIDEMIOLOGY AND RISK FACTORS FOR STROKE

Epidemiology, risk factor management and declining mortality from stroke

P.A. Wolf 3

Incidence of risk factors and types of stroke in patients with multi-infarct dementia

J.S. Meyer, K.L. McClintic, R.L. Rogers, P. Sims and K.F. Mortel 7

Carotid artery disease in cerebrovascular asymptomatic volunteers - Correlations with risk factors, CBF and CT findings

K. Niederkorn, H. Lechner, S. Horner, R. Schmidt, F. Fazekas and G. Schneider 13

Magnetic resonance signal abnormalities in young cerebrovascular asymptomatic patients with low-output cardiac disease

R. Schmidt, H. Lechner, F. Fazekas, H. Offenbacher, B. Dusleag, K. Niederkorn and S. Horner 17

Age-related alteration of cerebral enzymatic antioxidant system

G. Benzi 23

Risk factors for stroke in Copenhagen

G. Boysen, J. Nyboe, M. Appleyard, P.S. Sørensen, J. Boas, F. Somnier, G. Jensen and P. Schnohr 25

Strokes in westcentral India: A prospective case-control study of 'risk factors' in cerebral infarction

P.M. Dalal, K.P. Dalal, S.V. Rao and B.R. Parikh 29

Sepivac: A community based study of stroke incidence in USL No. 6 of Umbria, Italy

S. Ricci, P. Rucireta, M.G. Celani, G. Guercina, E. Duca, R. Scaroni, N. Caputo, C. Chiurulla, R. Vitali, F. La Rosa, L. Seppoloni, M. Paolotti and R. Ferraguzzi 35

Relation between risk factors and type of lesion in a sequential series of 250 stroke patients

A. Capon and F. Gregoire 41

Leuko-araiosis, age and cerebrovascular risk factors in a geriatric population: A clinical-CT study

D. Inzitari, M. Cadelo, G. Pracucci, M. Mascalchi and F. Bianchi 45

Acute stroke with extremely high blood pressure

M. Britton and A. Carlsson 49

Common carotid artery occlusion: Hemodynamic features

V. Zbornikova and C. Lassvik 51

Arterial disease risk factors and angiographic evidence of atheroma of the carotid arteries	
<i>A. Schneidau, M.J.G. Harrison, C. Hurst, H.C. Wilks and T.W. Meade</i>	55
The significance of clinically 'silent' signs for carotid stroke	
<i>J.H.A. van der Drift</i>	59

NEUROIMAGING, TRANSCRANIAL DOPPLER AND CEREBRAL BLOOD FLOW STUDIES

Positron emission tomography and memory functions in healthy aged volunteers and in patients with various amnesic syndromes	
<i>G. Pawlik, J. Kessler, V. Holthoff, J. Rudolf, K. Wienhard, R. Wagner and W.D. Heiss</i>	65
Conjugate eye deviation after hemispherical stroke: Clinico-anatomical correlations	
<i>C.C. Tijssen, J.A.M. van Gisbergen and B.P.M. Schulte</i>	71
RCBF-CO ₂ reactivity correlation: A measure of cerebrovascular reserve in health and disease	
<i>Y. Tsuda, A. Hartmann and H. Matsuo</i>	73
In vivo measurement of cytosolic free calcium during cerebral ischemia and reperfusion	
<i>M. Reivich, D. Uematsu and J.H. Greenberg</i>	79
The impact of internal carotid artery occlusion and of the integrity of the circle of Willis on cerebral vasomotor reactivity - A transcranial Doppler study	
<i>R.W.M. Keunen, R.G.A. Ackerstaff, D.F. Stegman and B.P.M. Schulte</i>	85
Three-dimensional transcranial doppler scanning in stroke patients	
<i>K. Niederkorn, S. Horner, R. Schmidt, F. Fazekas, W.M. McKinney and H. Lechner</i>	89
Transcranial doppler determination of hemispheric dependency	
<i>A. Krajewski, J.W. Norris, N.M. Bornstein, L.G. Chadwick and M. Ichise</i>	93
Noninvasive measurement of regional cerebral blood flow and neuropsychological assessment in dementia of Alzheimer's type	
<i>K. Broich, A. Hartmann, S. Adam and H.J. Biersack</i>	97
Cerebral blood flow tomography by SPECT in cerebro vascular disease	
<i>N.A. Lassen</i>	103
Role of SPECT (HM-PAO, TC99mRC) and ultrasound (TCD) in the instrumental diagnostic approach to the comprehension of reversible ischemic symptoms	
<i>W. Liboni, P. Baggio, G. Chianale, G. Marta, G. Castellano and G. Cornaglia</i>	105
Regional glucose metabolism in acute head injury as determined by positron emission tomography	
<i>A. Alavi, F. Fazekas, W. Alves, G. Spielman, T. Gennarelli and M. Reivich</i>	111

Comparison of positron emission tomography (PET), magnetic resonance imaging (MRI) and computerized tomography (CT): Brain imaging in the investigation of acute head trauma

A. Alavi, W. Alves, F. Fazekas, R. Zimmermann, D. Hackney,
L. Bilaniuk, T. Langfitt, J. Powe, M. Kushner, M. Reivich and
T. Gennarelli 113

The pseudobulbar (suprabulbar) palsy revisited: A clinical-computed tomography study

C. Loeb, C. Gandolfo, C. Caponnetto and M. Del Sette 115

Serial MRI in acute ischemia: A correlation with CT, SPECT and clinical parameters

F. Fazekas, H. Lechner, H. Offenbacher, R. Schmidt, K. Niederkorn,
F. Payer, H. Valetitsch and G. Schneider 119

3D-MR angiography of atherosclerotic carotid and vertebral artery disease

S. Felber, P. Ruggieri, G. Laub, F. Aichner, J. Willeit, G. Birbamer
and F. Gerstenbrand 123

Magnetic resonance imaging in ischemic stroke

J.L. Marti-Vilalta, J. Pujol and A. Arboix 127

A comparison of CT, MRI and PET in normal aging and dementia of the Alzheimer type

F. Fazekas, A. Alavi, J.B. Chawluk, R.A. Zimmermann and
M. Reivich 131

IBZM: a potential CNS D-2 dopamine receptor imaging agent: Preliminary results in monkey and humans

A. Alavi, H. Kung, M.P. Kung, S. Pan, J. Billings, R. Kasliwal,
M. Reivich and J. Reilly 135

Comparison of FDG-PET and image-guided P-31 MR-spectroscopy in brain tumors

W.D. Heiss, W. Heindel, K. Herholz, J. Bunke and J. Jeske 137

Clinical applications of proton magnetic resonance spectroscopy

J.W. Berkelbach van der Sprenkel, P.C. van Rijen, P.R. Luyten,
J.A. den Hollander and C.A.F. Tulleken 143

PREVENTION, DIAGNOSIS, COURSE, TREATMENT AND CLINICO-PATHOLOGICAL OBSERVATIONS IN CEREBRO-VASCULAR DISEASES

The Scandinavian multicenter trial of hemodilution in acute ischemic stroke: final results and conclusions

K. Asplund 155

A prospective study of 1000 first stroke patients admitted to a community-based primary care hospital: The Lausanne stroke registry, Lausanne, Switzerland

J. Bogousslavsky, G. van Melle and F. Regli 161

- Survival and recurrence following reversible ischemic attacks. A 10-year follow-up study
S. Passero, P. Fiori Nastro and N. Battistini 165
- Multinational stroke mortality data at the baseline of the WHO MONICA project
K. Asplund, J. Tuomilehto, K. Kuulasmaa, J. Torppa 167
- Early prognostic indicators in the functional recovery and outcome of stroke
W. Grossmann, J. Kick, M. Hallen and G. Paal 171
- Basilar artery disease: Clinical outcome and long-term follow-up
S. Biedert, U. Schulz, G. Zech-Uber, H. Betz, R. Reuther 177
- Neurological outcome in survivors from acute completed ischemic stroke: Effects of age, severity and previous strokes
D. Kidron and E. Melamed 183
- Cerebral blood flow during normal state and during regional cerebral ischemia before and after administration of a calcium antagonist
A. Hartmann, C. Dettmers, T. Rommel, A. Nierhaus, R. Reddelin and Y. Tsuda 187
- Fast-acting inhibitor of plasminogen activator in acute cerebral infarction
G. Schwendemann, M. Brockmann and T.W. Stief 191
- The effect of intravenous glycerol on deep brain circulation in patients with acute intracerebral hemorrhage
F. Gotoh, Y. Fukuuchi, T. Shinohara, S. Takashima, Y. Terayama, J. Kawamura and K. Takahashi 193
- CBF and CBF reactivity to acetazolamide before and after carotid surgery
F. Chollet, P. Celsis, M. Clanet, B. Guiraud-Chaumeil, A. Rascol and J.P. Marc-Vergnes 197
- The neurological aspects of selective embolization of cerebral arteriovenous malformations
J. de Jonge, A.A.W. Op de Coul, A.C.M. Leyten and T.G. Tjan 201
- Brain damage markers in the CSF of patients with stroke
J. Matias-Guiu, I. Bonaventura, A. Ruibal and J.M. Martinez-Vazquez 205
- Trace element alterations associated with cerebral infarction
H. Duflou, W. Maenhaut and J. De Reuck 209
- Antiphospholipid antibodies and stroke: Clinical and radiological features
V.L. Babikian, G.K. Call, B. Norrving, P.B. Gorelick, W.M. Feinberg, R. Kelley, V.E. Pochay and R.T. Canoso 213
- Binswanger's disease (subcortical arteriosclerotic encephalopathy): Clinicopathological and neuroimaging aspects
A. Agnoli, M. Feliciani and G. Fabbri 217

EXPERIMENTAL CEREBRAL ISCHEMIA

- Characteristics of neurological deficits and behavioral impairments of rats induced by bilateral incomplete cerebral ischemia
G. Nemeth, A. Cintra, G. Mayer, K. Fuxe and S. Hoyer 223
- Cerebral blood flow, tissue PO_2 , and somatosensory evoked potentials after intravascular occlusion of the middle cerebral artery in baboons
C. Dettmers, A. Hagendorff, A. Nierhaus, F. Brassel, A. Hartmann, U. Schlegel, M. Hoffstadt, C. Schul and J. Grote 229
- Some aspects of circulatory disturbances following cerebral ischemic insults
T.S. Nowak, M. Seida, S. Tomida, J. Ikeda, K. Vass, R. Pluta, F. Joo, H.G. Wagner, S. Xu and I. Klatzo 239
- Neuronal function after sequential cerebral arterial occlusion in rabbits: Deafferentation vs cortical ischemia
A. Wakayama, R. Graf, G. Rosner and W.D. Heiss 243
- Middle cerebral artery occlusion in hypertensive rats: Effect of antihypertensive treatment
B.B. Johansson and C. Nordborg 247
- Polyamine metabolism in reversible cerebral ischemia of mongolian gerbils
W. Paschen, G. Röhn, J. Hallmayer and G. Mies 251
- Cerebral circulation and metabolism in dogs with experimental cerebral embolism, effect of thrombolysis and flunarizine, a calcium entry blocker
J. Weyne, G. de Ley and G. Demeester 255
- Cerebral endothelial plasma membrane alterations in acute hypertension
S. Nag 259
- The effects of inhibition of thromboxane A_2 synthetase on the interaction between platelets and the cerebral vessel wall in experimental middle cerebral artery occlusion
F. Gotoh, Y. Fukuuchi, T. Amano, K. Tanaka, J. Kawamura, T. Yamawaki, N. Ito, K. Obara, K. muramatsu and K. Takahashi 263
- Subcellular distribution of putrescine in reversible cerebral ischemia of rat brain
G. Röhn, W. Paschen, M. Kocher, U. Oschlies and K.A. Hossmann 269
- Ischemic modulation of cerebro-cortical membranes
K. Kumami, A. Villacara, T. Yamamoto, B.B. Mrsulja and M. Spatz 273
- Difference of flow-pressure relationships between hypotension induced by exsanguination and by trimethaphan infusion in monkey brain
Y. Shinohara, M. Yamamoto, S. Takagi, M. Haida, H. Ohsuga, R. Taniguchi and Y. Kamezu 277
- Therapeutic efficacy of tissue plasminogen activator in experimental cerebral embolization utilizing autologous arterial white thrombus
M. Takigami, T. Uede and K. Hashi 281

Postischemic recovery of glucose and energy metabolism is different in brain cortex and hippocampus in aged rats

S. Hoyer and K. Betz

285

Marginal ischemia in the rat: a model with bilateral carotid artery occlusion studied with ^{31}P and ^1H NMR spectroscopy

J.W. Berkelbach van der Sprenkel, C.J.A. v. Echteld, A.L. Benabid, M. Decorps and C.A.F. Tulleken

289

List of contributors

297

Index of authors

303

**EPIDEMIOLOGY AND RISK FACTORS FOR
STROKE**

STROKE
EPIDEMIOLOGY AND RISK FACTORS FOR

EPIDEMIOLOGY, RISK FACTOR MANAGEMENT, AND DECLINING MORTALITY FROM STROKE

P.A. WOLF

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In the past 15 years a dramatic decline in death from stroke has occurred in most industrialized nations. In the United States the decline of 50% in stroke mortality, a 5% annual decrement, represents an acceleration of the 1% annual decline from 1915 to 1970. This accelerated decline supports the influence of modifiable environmental factors in stroke occurrence. Reduction in stroke mortality occurred in both sexes, in blacks and whites, and in all regions of the United States (1). Furthermore, death rates attributable to stroke have declined in the face of falling total death rates. In fact, the diminution in stroke death has been a major contributor to the decline in total cardiovascular diseases, providing further substantiation that the stroke decline is real and not an artifact of death certification or coding practices.

This momentous decline in death rates from stroke could come from decreased incidence of stroke, improved survival of stroke patients, or from a combination of the two. Evidence supporting the role of declining incidence of stroke came from the community based study of Rochester, Minnesota where stroke incidence declined over time coincident with improved control of hypertension (2). Age-adjusted stroke mortality rates declined significantly between 1971 to 1980 in Allegheny County, Pennsylvania USA (3). These data from Allegheny County were consistent with findings in the National Hospital Discharge Survey which also showed a decreasing case-fatality rate from stroke from 1970 to 1983 (4).

Three possible mechanisms may be operative in the decline of case-fatality rates for stroke: (1) in recent years care of acute stroke has improved; (2) stroke events in recent years are less severe and life-threatening than formerly; or, (3) milder cases that previously went undetected are now routinely diagnosed as stroke. It may be useful to examine each of these possible mechanisms. (1) Acute stroke care has improved: Data suggest that improved medical care is responsible for no more than marginal improvement in stroke survival. (2) Strokes are milder: Hospital case-fatality rates in Allegheny County, Pennsylvania decreased significantly from 19.6 to 11% from 1971 to 1980 (3). This decline in death rates coincided with a reduction in the severity of stroke. Fewer stroke patients were comatose and this reduction in prevalence of coma was thought to be responsible for more than 80% of the decline in the case-fatality rate. (3) Increased diagnostic sensitivity and accuracy: It is possible that small cerebral infarctions or hemorrhages, with mild or minimal neurologic deficits are now diagnosed as definite stroke through the routine use of computerized tomographic scan. The widespread availability of a diagnostic test for stroke, particularly for stroke in the elderly may be particularly important here. In Allegheny County the advent of computerized tomographic scan was accompanied by a twofold increase in survivorship of stroke patients. At present there is evidence to support the contention that both declining incidence and falling

case-fatality rates are contributing to the decline in death from stroke in the United States, and presumably in other western nations.

Recently identified risk factors for stroke

Maternal Death from Stroke

At follow-up a cohort of Swedish men born in 1913, after 18.5 years, 57 of the original 789 men had sustained a stroke. Hypertension was found to be a potent risk factor for stroke even after other significant variables were taken into account (5). Abdominal obesity, increased plasma fibrinogen level, and maternal history of death from stroke were independently related to stroke incidence. Although family history of stroke is commonly perceived as a marker of increased stroke risk, confirmation in prospective epidemiologic study has heretofore been lacking. This finding of a greater prevalence of maternal death from stroke among stroke victims needs to be confirmed and stroke occurrence in parents needs to be more precisely documented to include all stroke events, nonfatal as well as fatal.

Cigarette smoking

Several large scale prospective studies have clearly linked cigarette smoking to stroke incidence (6,7). Cigarette smokers, men and women, had more than double the stroke risk of nonsmokers in the Framingham Study cohort. Even after other cardiovascular risk factors, (systolic blood pressure level, total serum cholesterol, left ventricular hypertrophy by electrocardiograph, obesity, and glucose intolerance), were taken into account, cigarette smoking increased stroke incidence by 40% in men and 60% in women. There was a similar significant relationship between cigarette smoking and the specific stroke subtype of atherothrombotic brain infarction (6). In a cohort of female nurses, risk of stroke in light smokers (those smoking less than 15 cigarettes per day), was more than twice that of nonsmokers (7). Heavy smokers, (25 cigarettes/day) had nearly a 4-fold increased stroke risk when compared with nonsmokers. This significantly increased stroke risk persisted after other risk factors including alcohol consumption, oral contraceptive and postmenopausal estrogen use and other standard risk factors were taken into account.

Insights into the mechanism by which cigarette smoking predisposes to stroke may be gained by examination of stroke risk following smoking cessation. Incidence of stroke in smokers fell substantially within 2 years after quitting the cigarette habit and in 5 years risk of stroke fell to the level of a nonsmoker (6). This suggests that cigarette smoking acts by precipitating stroke in susceptibles rather than by promoting atherosclerosis.

Of considerable interest, relative risk of subarachnoid hemorrhage also showed a dose-response relationship from 4-fold in light smokers to 9.8-fold in smokers of 25 or more cigarettes daily (7). The association between cigarette smoking and subarachnoid hemorrhage from aneurysm was also found in men (as well as women) in Framingham (8) and New Zealand (9) in case control analyses.

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