Cerebral Vascular

Disease 7

CEREBRAL VASCULAR DISEASE 7

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14th International Salzburg Conference,
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INTERNATIONAL SALZBURG CONFERENCE ON CEREBRAL VASCULAR DISEASE

founded by

H. Bertha, O. Eichhorn, H. Lechner

organized by

FEDERATION MONDIALE DE NEUROLOGIE WORLD FEDERATION OF NEUROLOGY

Research Group on Cerebrovascular Disease

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 colloqium 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

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EPIDEMIOLOGY, RISK FACTOR MANAGEMENT, AND DECLINING MORTALITY FROM STROKE

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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 contributon 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, increaased 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.

Cigartette 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 susbstantially 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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