the ischemic hemispheres.

Kletter found that although atherosclerosis did occur in the STA, there was an increase in elastic fibers in the STA wall with aging (as opposed to other vessels) enabling it to dilate despite atherosclerosis. Kletter also reported failure to create a patent vascular anastomosis to a vessel on an ischemic spinal cord. He did show, in contrast, that pedicled muscle grafting can vascularize the spinal cord.

Collice (Milan, Italy) described a rat carotid artery model for surgeons seeking facility in microvascular technique.

Participants

The following were among those who presented papers at the Third International Symposium:

- Dr. M. Hausmann, Max-Planck-Institute, Cologne, West Germany; Dr. W. Heus, Hirnrinde-Laboratorium, University Clinic, Vienna, Austria; Dr. J. Astrep, Dept. of Clinical Physiology, Bispebjerg Hospital, Copenhagen, Denmark; Dr. G. Austin, Loma Linda U. Medical School, California; Dr. J. Fein, Dept. of Neurological Surgery, Albert Einstein College of Medicine, Bronx, N.Y.; Dr. Norman Chater, Dept. of Neurological Surgery, Davies Medical Center, U. of California, San Francisco; Dr. M. Mizukami, Inst. of Brain and Blood Vessels, Mikosyo Memorial Hospital, Gunma, Japan; Dr. P. Weinstein, Dept. of Neurological Surgery, U. of California, San Francisco.

- Also Dr. F. Mere, Dept. of Neurological Surgery, U. of Pecs, Pecs, Hungary; Dr. D. Piepras, Dept. of Neurological Surgery, Mayo Clinic, Rochester, Minnesota; Dr. G. Khodadad, U. of Cincinnati Medical School, Ohio; Dr. H. Herrschel, Neurolog. Klinik and Max-Planck-Institute; Dr. O. Reichman, Div. of Neurological Surgery, Loyola U., Chicago, Ill.; Dr. H. Kikuchi, Dept. of Neurological Surgery, Kitano Hospital, Osaka, Japan; Dr. H. Hapman, Dept. of Neurology, Munich U., W. Germany; Dr. M. Dujovny, Dept. of Neurology, U. of Pittsburgh, Pa.

- Also Dr. U. Itö, Dept. of Neurological Surgery, Tokyo Medical U., Tokyo, Japan; Dr. R. Meyermann, Dept. of Neuroradiology, Nervenklinik, Göttingen, W. Germany; Dr. P. Confotti, Inst. di Neurochirurgia, Napoli, Italy; Dr. G. Kletter, Neurochirurg. U. Klinik, Vienna, Austria; Dr. G. Allen, Dept. of Neurosurgery, The Johns Hopkins U., Baltimore, Md.; Dr. C. Banister, Dept. of Neurological Surgery, North Manchester General Hospital, Manchester, England; Dr. L. Auer, Neurochirurg. U. Klinik, Graz, Austria; Dr. K. Holbach, Neurochirurg. U. Klinik, Bonn, W. Germany; Dr. M. Heilbrun, Div. of Neurological Surgery, Utah U., Salt Lake City.

- Also Dr. J. Ausman, Dept. of Neurological Surgery, U. of Minnesota, Minneapolis; Dr. R. Deruty, Hôpital Neurologique, Lyon, France; Dr. R. Crowell, Dept. of Neurological Surgery, Harvard Medical School, Boston, Mass.; Dr. V. Olteanu-Nerbe, Neurochirurgie U. Klinik, Munich, W. Germany; Dr. M. Collice, Ospedale Maggiore, Milano, Italy.

Summary of Eighth International Salzburg Conference on Cerebral Vascular Disease,
Salzburg, Austria, September 22–25, 1976

Prepared by John Stirling Meyer, M.D.

Chairman of the Conference: David H. Ingvar, M.D.

Organizing Secretaries: O. Eichhorn, M.D.; H. Lechner, M.D.


DR. P. O. YATES (Manchester, England) reported on quantitative neuronal measurement profiles in senile dementia and advised caution in correlating regional blood flow changes and dementia. Regional blood flow may be normal despite abnormal brain function and vice versa. He stressed the importance of the strategic localization of cerebral infarctions necessary to produce dementia. With new biochemical methods for identification of ribonucleoproteins, cytometric analysis permits quantitative estimation of nuclear and nucleolar volume in diseased brains. In senile dementia 17% to 50% of the Nissl material is lost in a diffuse manner, although some neurons may remain normal, particularly in the thalamus, locus ceruleus and some cranial nerve nuclei. There is a similar loss of Nissl material with age but those patients with clinical signs of dementia showed much greater changes.

Dr. L. Gustafson (Lund, Sweden) presented a series of 17 patients with the clinical diagnosis of "presenile dementia" studied during life with regional cerebral blood flow (rCBF). Diagnosis at autopsy was Alzheimer's disease in five, Pick's type of frontotemporal degeneration in four, multi-infarct dementia in four, Jakob-Creutzfeldt disease in three and postencephalitic dementia in one. There was significant correlation of the reduction of flow of fast and slow compartmental values with atrophy of gray and white matter. There was good regional anatomical correlation of pathological changes with flow values but poor correlation for the medial aspects of the hemispheres.

Dr. F. I. Perez (Houston, Texas) reported on statistical correlation of psychometric tests with reduced rCBF in a series of patients with Alzheimer's disease (AD) and multi-infarct dementia (MID). In AD frontal, temporal and parietal reductions of flow were marked while in MID the temporal and parietal zones in the middle cerebral artery territory tended to be more reduced. In AD both performance and memory IQ were significantly reduced whereas in
MID memory IQ was more strikingly reduced. CSF cyclic AMP levels were reduced in AD and this reduction correlated with memory and performance IQ values.

There was general agreement in the many papers presented at this meeting that in dementia there is bilateral reduction of rCBF.

Dr. John Marshall (London) reported on stability of rCBF measurements in dementia and found that the second measurement was considerably lower than the first, which he attributed to the patient's becoming less activated as he becomes accustomed to the test situation.

Dr. N. T. Mathew (Houston) correlated ventricular size measured by computer-assisted tomography (CT scan) in patients with various types of dementia. Ventricular enlargement was maximum in communicating hydrocephalus. There was no difference in the degree of ventricular enlargement between MID and AD but ventricular size increased with age in both groups. In metabolic encephalopathies with dementia it was noted that cortical atrophy and ventricular enlargement also occurred. In dementia associated with état lacunaire, the lacunae were usually too small to be recognizable in CT scans.

Dr. W. Dekoninck (Montignies le Tilleul, Belgium) and Dr. S. Hoyer (Heidelberg, Germany) reported abnormal patterns of CBF and glucose as well as oxidative metabolism in dementia. CBF and metabolism were usually more reduced in multi-infarct dementia. Cerebral amino acid metabolism was also reported abnormal.

Dr. Ladurner and associates (Graz, Austria) reported low levels of rCBF in both hemispheres in dementia. Although the left hemisphere showed a tendency to be more reduced than the right in their series, this was not significant. Others, particularly from Scandinavian laboratories, concluded that flow in the dominant hemisphere tends to be more reduced than in the non-dominant hemisphere.

Dr. J. S. Meyer and associates (Houston) reported usefulness of noninvasive measurement of rCBF by 133Xe inhalation in patients with MID and AD (in whom the diagnosis had been confirmed by CT scans) compared to normal control subjects. In normal controls there was a progressive reduction of gray matter rCBF throughout both hemispheres with advancing age from 23 to 62. In AD and MID the initial slope index of rCBF (which correlated best with gray matter flow) was reduced compared to controls but in AD the reduction of CBF was greater, more diffuse, and affected both mean flow values and gray flow values.

Dr. J. Risberg (Lund) reported similar experience in dementia using the noninvasive inhalation method of measuring rCBF for 32 regions. He found significant correlation between rCBF values measured by carotid injection of 133Xe and the noninvasive inhalation method and noted bilateral reduction of rCBF in dementia although in some cases there was a greater reduction in the left hemisphere.

Dr. N. Lassen (Copenhagen, Denmark) indicated that although CBF and metabolism were reduced in both hemispheres in dementia, the reduction is greater in the dominant hemisphere for speech.

Dr. Larsen and associates (Copenhagen), using 250 measurements of rCBF in right-handed subjects, compared the effects of speech and listening on rCBF compared to the blindfolded, ear-covered, resting state. During speech the rCBF of frontal and mouth areas of both hemispheres increased as it did in Wernicke's area in the temporal lobe (monitoring one's own speech). During listening there was an increase in premotor and temporal areas bilaterally. In both speech and listening the dominant hemisphere showed more regional changes in rCBF while in the non-dominant hemisphere the changes were more diffuse.

Dr. Roland (Copenhagen) showed that sensory discrimination, auditory discrimination and selective movements of the fingers produced highly regional increases of rCBF in expected anatomical areas of cortical representation.

Dr. E. Ott and associates (Graz) reported that in aphasic patients with brain tumor there was a reduction of rCBF in the posterior frontal and temporal regions (rather than an increase) during speech. In patients with aphasia due to cerebral infarction and hemiplegia there were also abnormal patterns of rCBF increase during attempts to speak or move the paralyzed limbs.

Dr. W. D. Heiss and associates (Vienna, Austria) found good correlation of rCBF values in aphasic subjects. In motor aphasia the reduction of CBF was predominantly in the lip and tongue area of the motor cortex and in sensory aphasia the CBF reduction was predominantly in the temporal regions.

Dr. A. Hartmann (Heidelberg) reported on measurements of rCBF and CSF pressure (CSFP) in patients with normal pressure hydrocephalus (NPH) and dementia. Monitoring CSF by lumbar catheter showed good correlation with simultaneous ventricular pressure measurements. In 9 of 14 cases there were intermittent pressure elevations and plateau waves. In all cases rCBF was reduced and five cases were improved by ventriculostriatal shunt with a maximum increase of rCBF in the distribution of the anterior cerebral artery; clinically, gait improvement was greater than dementia improvement.

Dr. David Ingvar (Lund) commented that the regional reduction of CBF in communicating hydrocephalus was in the prefrontal areas, the reverse of the usual pattern, which may account for the hypokinesis and dementia characterized by lack of spontaneous activity.

Dr. Collignon (Leuwen, Belgium) reviewed the neuropsychological aspects of NPH in 208 patients reported in the literature, including 10 patients of his own group. Ataxia, gait apraxia, constructional apraxia, incontinence, anosognosia and memory impairment were among the most common symptoms.

Dr. H. van der Drift (Wassenaar, Netherlands) reported on 275 cases of stroke with recovery (reversible ischemic neurological deficit) in patients with verteobasilar insufficiency. In 40 who died there was pathological confirmation at autopsy. The pathological substrate was infarction despite recovery from a neurological deficit present for two to six weeks.

Dr. A. C. Klassen and associates (Minneapolis, Minnesota) reported on the value of monitoring electrocardiogram (ECG) and respiration patterns in a stroke intensive care unit. Bilateral cerebral infarction was the commonest cause of phasic respiration and generally carried a poor prognosis. The ECG was abnormal in 82% of their stroke patients; this was classified as due to pre-existing heart disease and neurogenic origin secondary to the stroke. The early detection and treatment of cardiac dysrhythmias,
Dr. F. Torres (Minneapolis) reported on the predictive value of the EEG in a prospective study of 104 patients with cerebral atherosclerosis followed for 101 months in which 12 had a stroke. There was no significant correlation between EEG patterns and prognosis for stroke.

Dr. H. Herrschaft and colleagues (Kölín-Merheim, Germany) correlated serial detailed neurological and psychological assessments with measurements of rCBF in patients with cerebral infarction. After three weeks there was a significant global increase of rCBF by 12% but little or no focal increase in the zones of maximal reduction and infarction. This global CBF increase correlated with improvement in the state of consciousness, some improvement of the motor and sensory deficits and improvement of the psychological test performance and EEG.

Dr. M. Reivich (Philadelphia, Pennsylvania) mentioned similar experience with 15 patients with unilateral cerebral infarction having serial rCBF measurements. Maximum reduction of bilateral hemispheric blood flow occurred on the 7th day and by the 14th day had returned to or was above baseline levels.

Dr. V. C. Haschinski (Toronto, Canada) studied nocturnal EEGs, EMGs and eye movements on polygraphic records in 33 stroke patients for prognostic value. He found that prognosis correlated best with the state of consciousness. The alert patients with stroke generally fared well; the comatose patient had a poor prognosis. Patients with normal REM sleep or stage 2 sleep had a good prognosis. Normal stage 2 sleep (sleep spindles, K. complex and no more than 20% delta present) was the best prognostic index, since 32 of 33 patients showing stage 2 sleep had an excellent prognosis.

Several papers from Graz dealing with platelet aggregation and blood viscosity as risk factors in stroke patients indicated that increased platelet aggregation of itself is seldom the sole risk factor in cerebral infarction.

Drs. Lenzi, Jones, McKenzie and Moss (Siena, Italy and London) reported regional measurement of cerebral oxygen uptake and blood flow in patients with cerebral infarction using the noninvasive Oxygen 15 inhalation technique developed by Ter-Pogossian in St. Louis. The authors reported an imbalance between regional oxygen uptake and perfusion (decreased) in patients with TIAs. In severe regional infarction the CMRO 2 is reduced to a greater extent than the regional blood flow. In patients with multi-infarct dementia, there is a reduction of both CMRO 2 and rCBF in the infarcted zones.

Dr. M. Reivich and associates (Philadelphia) reported on local cerebral glucose metabolism in the cat. Regional glucose utilization of the cerebral cortex is normally 5.4 mg/100 g brain/min and that of white matter is 1.7 mg/100 g brain/min. In cerebral infarction there is a central zone of reduced glucose consumption, a bordering zone of increased glucose metabolism and a remote reduction of CMRGl in the opposite hemisphere. In the zone of bordering increased glucose metabolism, there is regional hyperemia. CMRGl may be increased to levels as high as 13.1 mg/100 g brain/min. The critical rCBF below which glucose consumption becomes enhanced (presumably due to lactate production) is 25 ml/100 g brain/min. This method has been used successfully in humans.

Drs. J. de Reuck and H. van der Eecken (Ghent, Belgium) described the angioarchitecture of intracerebral hemorrhage by postmortem injection techniques and were able to identify bleeding points in the hematomas. In hypertensive hemorrhage the feeding arteries showed abnormal changes but in trauma were not abnormal. Intracerebral hemorrhage had an avalanche effect on normal bordering vessels, causing them to contribute to the bleeding. This was true of secondary brain stem arterial hemorrhages.

Dr. J. C. Gautier (Paris) discussed the clinical syndrome of thalamic hemorrhage based on personal experience of five cases examined during life and correlated with the findings at autopsy. Patients may survive for months to ten years. Almost all cases were hypertensive and had slight to moderate hemiparesis. Sensory loss and oculomotor disturbances were surprisingly often absent. Dementia was never present. With the advent of CT scanning, evaluation for surgical removal of hemorrhages of the basal ganglia capsule and thalamus was now feasible.

Dr. P. Pilz (Salzburg) described dissecting aneurysm of the middle cerebral artery with the "string sign" on angiography as a cause of massive cerebral infarction in the young. This may be associated with fibromuscular dysplasia or, more rarely, oral contraceptives.

Dr. K. Hashi (Osaka, Japan) and Mr. L. Symon (London) summarized advances in the past decade for treatment of ruptured aneurysm. These included timing of operation as judged by clinical classification, use of the operating microscope, development of the tandem bar aneurysm clip, and use of the CT scan and magnification angiography for defining detail of the aneurysm, its site and locus of rupture. Mr. Symon reported that in experimental cerebral infarction the evoked response failed when rCBF fell below 20 ml/100 g brain/min but infarction did not occur until rCBF fell below this. Using the Astrup K+ electrode he found the flux of potassium bore no relation to the disappearance of the evoked response. The K flux occurred when rCBF fell below 10 to 12 ml/100 g brain/min. GABA uptake by synaptosomes from cerebral biopsy specimens became impaired when CBF fell below 25 ml/100 g brain/min.

Dr. W. S. Fields (Houston) reported on a cooperative controlled trial of platelet suppressive therapy using aspirin in a series of 178 patients with TIAs in the carotid territory. The incidence of TIAs was significantly decreased but not abolished by aspirin. The incidence of cerebral infarction and the mortality rate were not apparently significantly altered in the relatively small series studied.

Dr. A. Agnoli and colleagues (Rome) reported on a randomized series of 10 severely ill patients with acute stroke treated with sodium pentothal to test whether suppression of cerebral metabolism during the acute stage of cerebral ischemia improved survival. Six patients survived and four died. Results were considered better than in the untreated group.

Dr. K. M. Kogure (Miami, Florida) summarized his work on controlled hypoxia in rats and concluded that hypoxia may impair membrane function resulting in cAMP changes before neurotransmitter levels are affected.

Dr. V. Hossmann (Kölín-Merheim) showed that in experimental hypertensive encephalopathy damage to the blood-brain barrier occurred in areas of "breakthrough" with increased regional flow.

Dr. O. B. Paulson (Copenhagen) showed evidence that
Sanguineous Cerebrospinal Fluid in Recanalized Cerebral Infarction

TADAYOSHI IRINO, M.D., MAMORU TANEDA, M.D., AND TAKAO MINAMI, M.D.

SUMMARY To clarify the causal relationship between spontaneous recanalization of the occluded cerebral artery and development of hemorrhagic infarction, 15 patients with internal carotid or middle cerebral arterial axis occlusion were submitted to consecutive lumbar punctures and follow-up cerebral angiography. Consequently, six of seven recanalized patients had sanguineous cerebrospinal fluid (CSF) on the second or third day after ictus, while only one of eight non-recanalized patients had bloody CSF.

It was strongly suggested that recanalization might have an intimate relationship with the development of hemorrhagic infarction.

Atirial fibrillation was present on the ECG in six patients (table 1). Fifteen patients who had no previous stroke had sudden onset of severe hemiplegia and disturbance of consciousness. Fibrinolytic agents were not used in the treatment of these patients.

Angiography was first performed within 24 hours after the onset, using 7 ml of 60% Amidotrizoate. Angiography also was performed on the second or third day in order to study the occluded artery. When recanalization was not demonstrated, additional angiography was performed between the fourth and seventh day.

CSF was obtained by lumbar puncture on admission and then at one to three-day intervals in the first week; the appearance and pressure of the CSF were noted. When the CSF was bloody, it was placed in three test tubes and the red cell counts were compared with each other to eliminate a traumatic tap. Sanguineous CSF always contained more than 1,500 red blood cells per cubic millimeter. The appearance of the CSF was classified as clear, xanthochromic or sanguineous. The results were compared in two groups: recanalized and non-recanalized patients.

Results

In two patients (Cases 1 and 2) with internal carotid arterial occlusion and five patients (Cases 4, 5, 6, 7 and 8) with middle cerebral arterial axis occlusion, follow-up angiography showed clearing of the carotid arterial tree within three days after the stroke. In one patient (Case 3) with internal carotid arterial occlusion, the angiograms demonstrated middle cerebral arterial occlusion on the third day and no occlusion on the fifth day. In the remaining seven patients recanalization did not occur (table 1).

The CSF was bloody or xanthochromic between the second and fourth day after the onset in seven patients. Six of them were recanalized within a week after the onset, while...
Summary of Eighth International Salzburg Conference on Cerebral Vascular Disease:
Salzburg, Austria, September 22-25, 1976
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Stroke. 1977;8:19-22
doi: 10.1161/01.STR.8.1.19
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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