POLISH ACADEMY OF SCIENCES
MEDICAL RESEARCH CENTRE

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MEDICAL RESEARCH CENTRE

Editors
W. Dziedzic, K. Szeliga-Radwańska

Scientific Consultant
H. Kaciuba-Uściłko, Professor of Physiology

Polish Academy of Sciences
Medical Research Centre
3 Dworkowa Str., 00-784 Warsaw – Poland

Telephones 49 64 93
49 69 73
Fax 48-12 49 69 73

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Obituary Professor in Ordinary Dr. hab. med. Adam Kunicki (1903–1989)

Prof. Dr. hab. med. Adam Kunicki was born in 1903 in Frysztat. He received the degree of doctor of medicine in 1928 at the Jagiellonian University in Kraków. He then joined the staff of the Neurological-Psychiatric Clinic of Prof. Plitz in Kraków for two years and worked in the Department of Neurology in Łódź. In 1931 he spent a six-month neurological training period with Clovis-Vincent in Paris. In 1936 he started work in Warsaw in the first Department of Neurosurgery in Poland headed by Dr. Jerzy Choróbski. During this period he published his first paper on brain neoplasms, surgery of the hypophysis and intracranial hypotension. After the war A. Kunicki returned to Kraków and organised in 1945 the second Department of Neurosurgery in Poland. In 1948 he was appointed docent of neurosurgery and in 1951 professor at the newly organised Department and Clinic of Neurosurgery of the Medical Faculty at the Kraków University. It is here that the first centre of neuroradiology was opened, directed by Docent Dr. Spett. Furthermore Professor Kunicki organised and equipped the Laboratory of Histopathology of Neoplasms of the central nervous system, where future neuropathologists were trained. At the beginning of the '50-ies he travelled to various neurosurgical centres in the USA and Canada and to Moscow and Leningrad. The achievements of world neurosurgery which he had the opportunity to become acquainted with contributed to the development of the Neurosurgical Clinic in Kraków which during this period achieved European standards. In recognition of his merits in the development of neurosurgery in Poland he was named Professor in Ordinary in 1956. In the '60-ies he became Chief Consultant in the field of neurosurgery and was for many years chairman of the Polish Society of Neurosurgeons. Some of his students were appointed as head of departments of neurosurgery organised at this time, and later of neurosurgical clinics in Szczecin (doc. dr A. Liszka) and in Gdańsk (doc. dr J. Chmielewski).

During the period of his activity at the Kraków Clinic of Neurosurgery Professor Kunicki published 51 papers presenting a wide spectrum of surgical

treatments of brain and spinal cord tumours. Particularly noteworthy among these are those concerning operative treatment of pineal gland tumours, curability of subcortical glia tumour and the morphological criteria of malignangy of these neoplasms. A number of his papers dealt with the transdural modification of the Frazier operation for the treatment of neuralgia of the trigeminal nerve and more careful diagnostic techniques in neurosurgery such as: positive fractionated pneumoencephalography, ventriculography and cerebral arteriography. Professor A. Kunicki – experienced clinician and specialist in neurological semiology – never omitted a penetrating neurological analysis of each patient and always exacted the same accuracy from his assistants.

In 1970 Prof. A. Kunicki was offered the position of Director of the Medical Research Centre of the Polish Academy of Sciences in Warsaw and at the same time he was appointed head of the Department of Neurosurgery of the Academy. In the five-year research plan he included themes connected with the development and control of brain oedema in cranio-cerebral trauma and neurosurgical operations. The subject was further extended to include studies on the mechanism of control of intracranial hypertension. For the wider dissemination of the results of this research A. Kunicki appointed a Comission of Pathophysiology of Intracranial Pressure (ICP) at the Medical Division of the Academy of Sciences. Its activity led to an extended collaboration of three Polish centres and two foreign ones (the Netherlands and the USSR). The results of research of the Department of Neurosurgery of the Polish Academy of Sciences and method of ICP measurement, at first intraventricular, then supradural and finally lumbal-subarachnoid, with the use of computer technique and supplementary tests, found practical implementation in some centres at home and in the USSR. In his final perod of clinical activity Prof. Kunicki was Vicechairman of the World Federation of Neurosurgical Societies, and head of the Committee of Neurosurgical Sciences of the Polish Academy of Sciences. He was a full member of the Polish Academy of Sciences and Honorary Chairman of the Polish Society of Neurosurgeons. He was also Corresponding Member of the working Groups on Neuropathology of the World Federation of Neurology, Honorary Member of the British, Czechoslowak and Bulgarian Societies of Neurosurgeons, Corresponding Member of the American and Italian Societies of Neurosurgeons. As professor emeritus he continued to support the clinical ICP research and the studies of the Commission on the Pathology of Intracranial Hypertension.

Prof. Kunicki received high State awards for his scientific and organisational work.

He was one of the founders of Polish neurosurgery, a talented neurosurgical operator, he educated two generations of Polish neurosurgeons, and also was a clearsighted researcher.

Eugenisz Mempel and Zbigniew Czernicki
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Z. Bujacz, secretary, **J. Lewicka,** secretary

RESEARCH REPORT

DEPARTMENT OF NEUROPHYSIOLOGY
3 Dworkowa Str. 00-784 Warsaw
Telephone: 49 58 29

Head: Assoc. Professor Mieczysław Pokorski

MECHANISMS OF RESPIRATORY RESPONSES TO CHEMICAL AND HUMORAL STIMULI

(Assoc. Professor Mieczysław Pokorski)

Intracellular mechanisms of the signal transduction have been studied in the cat carotid body in vitro. We found that the natural chemical stimuli low PO_2, low pH and high CO_2 activated the enzyme phospholipase C, which hydrolyses the phosphoinositides of the plasma membrane to produce lipid-dependent second messengers. Activation of the phosphoinositide cascade seems to be a key element of chemotransduction, which effects reflex ventilatory responses mediated by the carotid body.

In other in vitro experiments on the cat carotid body it was found that the amino acid taurine has a modulatory effect on the pharmacological carotid body stimulation by canide – an inhibitor of the mitochondrial electron transport. The release was augmented by taurine. The studies implicate the transmembrane carrier-mediated taurine transport system in the transduction of the cyanide stimulus in the carotid body.

In in vivo experiments on the guinea pigs the mechanisms of the central hypoxic ventilatory depression have been studied. We have tested a hypothesis that the inhibitory GABA receptor complex plays a role in these mechanisms. A part of the complex sis the benzodiazepine receptor, which was blocked with the specific benzodiazepine antagonist annexed. It was found that the benzodiazepine system has a modulatory effect on the hypoxic ventilatory response. The exact determinants of the annexed effect require further studies. In the experiments concluding the studies on the role of endogenous opiates in the control of respiration, it was found that opiates do not underlie the blunted hypoxic ventilatory response in animals adapted to chronic hypoxia.

MECHANISMS OF AUTOMATIC AND BEHAVIORAL CONTROL OF RESPIRATION

(Assoc. Professor Krystyna Budzińska)

To study cortical contribution to the control of respiration the analysis of short- and long-term effects of transcranial magnetic stimulation on respiratory activity of diaphgram and thoracic muscles was performed. It was found that contraction of all studied muscles facilitated the magnitude of the response to magnetic stimulation. Short-term respiratory effects were found to be depended on the level of CNS arousal. Cortical stimulation influenced the mechanisms controlling both amplitude and respiratory timing and it caused the modification of the respiratory pattern according to the state of arousal in a given moment of the respiratory cycle.

In studies on the role of motor nucleus trigeminal nerve in the control of respiration it was found that the anatomical site of the neurones of mylohyloideus nerve is localized in the caudal part of motor nucleus of trigeminal nerve (n. Vmt.). From the other side, electrophysiological studies have shown that electrical stimulation or pharmacological blockade of this particular part of n. Vmt. provokes an inhibition or a release of phrenic nerve activity. The results suggest that neurones of mylohyloideus nerve that control upper airways muscle give collateral to the brain stem respiratory neurons and in this way take part in the central regulation of respiration.

Precise localization of the phrenic nucleus in the spinal cord and spatial distribution of the cell bodies of separate C_4, C_5 and C_6 phrenic roots in rabbits were studied with the technique of retrograde axonal transport of horse-radish peroxidase. The morphological features of phrenic motoneurones were described. Longitudinal limits of phrenic nucleus extend from the C_3–C_4 border to rostral half of C_6. The motoneurons forming C_4 and C_5–C_6 roots were spatially separated, however the dendrites were extensively distributed within phrenic nucleus and also extend beyond it. Comparison of the results on cats with those on rabbits shows that spatial organization of phrenic nucleus in the rabbit is similar to that in the cat but it is shifted a half of the segment rostrally.

THE ROLE OF IMMUNE MECHANISMS IN SUBARACHNOID HEMORRHAGE PATHOLOGY

(Assoc. Professor Mirosław Ryba)

The investigations concerning the contribution of humoral and cellular immunity in the pathogenesis of the late neural deficits in patients with subarachnoid hemorrhage from reptured intracranial aneurysms have been continued.

In aneurysmal walls surgically removed during aneurysm clamping immunoglobulin IgM – complement C3 complexes have been found in endothelium. In peripheral blood lymphocytes from these patients a depressed response to concanavalin A, the mitogen specific for suppressor cells, was found. On the basis of these findings a hypothesis was proposed, according to which intracranial aneurysms develop as the result of a preexisting chronic autoimmune process; rupture of the aneurysm triggers acute humoral and cellular attack against brain tissues, facilitated by apparent dysfunction of suppressor cells.

Clinical experiments with the use of immunosuppressant cyclosporine A to prevent the development of late neural deficits in patients suffering from subarachnoid hemorrhage were continued. Thirty three patients were treated with this drug. It appears that cyclosporine A is effective, but its well known hepato- nephro- and neurotoxicity demands the search for alternative means of producing transient immunosuppression in patients with aneurysmal subarachnoid hemorrhage.

MODELING OF THE OBSTRUCTIVE SLEEP APNEA MECHANISM

(Professor Andrzej Kukwa)

Despite numerous theoretical and clinical considerations mechanism of the obstructive sleep apnea (OSA) is still unclear. The fibrooptic studies revealed that already during the initial phase of the sleep (depending on the severity of the symptoms) the lumen of the upper airway diminishes consecutively. The sleep of these patients is poorly diversified and does not reach the deep (REM) phase. It proves that there must be another different from muscular mechanism of the upper airway obstruction.

Apparently, the most probable cause of it is disfunction of neuro-muscular coordination which was earlier suggested by Remmers at al. and Broulliette and coworkers.

Our clinical observations also proved such a possibility and allowed us to hypothesize that within collapsing of the upper airway lumen can be two phases distinguished. The initial – when it comes to gradual narrowing of the upper airway diameter up to the complete closure of it and the secondary phase – of consecutive occurrence of apneic episodes with variable duration (apneic episodes are separated by a series of deeper and faster breaths).

These observations became our theoretical basis for an animal model of the OSA mechanism. For this purpose it was necessary to study the respiratory activity in the nerves which, as it was proved earlier, demonstrate this kind of activity. Apart from phrenic (PhN) nerve it was hypoglossal and in facial nerves.

The study was performed on rabbits with an average weight of 4 kg. The animals were anesthetized and artificially ventilated under halothane anesthesia.

The activity of PhN (branch C_4) one of the branches of the facial and hypoglossal nerve trunk was monitored.

During the experiment CO_2 concentration and gasometry of the blood were monitored. We have completed 9 full records of the experiments including two where the monitored activity of the facial nerve branch had an expiratory character. The respiratory activity of the facial and hypoglossal nerves were monitored only when expiratory CO_2 concentration exceeded 5%. The withdrawn of the respiration (ventilation for 10–15 s) resulted in a gradual increase of the respiratory activity in all the neurones; proportionally the highest in the hypoglossal and slightly lower in the facial nerve. A small increase of the amplitude was perceived in the PhN. Sudden increase of the volume or frequency or both resulted in an instant decrease in the amplitude of the above mentioned respiratory activities.

Our observations prove the initial assumption that collapsing of the upper airway during sleep occurs as a consequence of the neuromuscular discoordination mechanism.

DEPARTMENT OF NEUROCHEMISTRY
3 Dworkowa Str. 00-784 Warsaw
Telephone: 49 58 97

Head: Professor Jerzy Łazarewicz

AMINO ACID NEUROTRANSMITTERS AND BRAIN NEURONAL INJURY

(Professor Jerzy Łazarewicz)

Our previous results indicated that the NMDA receptors play a major role in ischemia-evoked calcium redistribution in the hippocampus. The present study was aimed to evaluate effects of maximal activation of the NMDA receptors on changes in extracellular calcium concentration in the hippocampus in vivo and to elucidate some of the NMDA receptor regulation. Using a method of the hippocampal microdialysis it was found that NMDA induces a dose-dependent, up to 60%, decrease in extracellular calcium concentration in the hippocampus. This effect was sensitive to several NMDA receptor antagonists and to 7-Cl-kynurenate, that is a blocker of glycine modulatory site on the NMDA receptors, but was potentiated by glycine and D-serine, positive modulators of these receptors. Glycine and D-serine alone did not influence the steady state calcium level in the hippocampus. These results confirm a potential primary role of the NMDA receptors/channels in calcium influx to the hippocampal neurons. They also illustrate the role of glycine in the in vivo modulation of the NMDA receptor activity. Endogenous glycine supports normal ongoing NMDA receptor neurotransmission, but an increase of extracellular glycine concentration may potentiate activation of the NMDA channels during abnormal stimulation of the NMDA receptors in different pathological conditions including brain ischemia.

Strychnine is a customary tool used to differentiate glycine binding sites on the NMDA receptors from the classical strychnine-sensitive glycine inhibitory receptors operating Cl^- channels. It was shown in studies performed on the cortical and hippocampal membranes, that glycine, unlike GABA, induces a negligible rise in the ^{36}-Cl^- influx into membrane vesicles. Moreover, strychnine, like classical GABA antagonists: bicuculline and picrotoxin, inhibits GABA-evoked ^{36}-Cl^- influx to membranes. These results indicate a relatively small specificity of strychnine and point to its limited applicability in functional studies on regulation of the NMDA receptors.

Previously it was shown that stimulation of the NMDA receptors results in the Ca^{2+} – and phospholipase A_2-mediated arachidonate release from cultured neurons. We focused our attention on the role of NMDA receptors in ischemia-evoked arachidonate release in neurons. Studies performed in the collaboration with Dr Wróblewski (FGIN, Georgetown University, Washing-

ton DC, USA) demonstrated that incubation of cerebellar granule cells in the glucose- and oxygen-deprived medium results in the arachidonate release. These incubation conditions have been considered an adequate in vitro model for studies on ischemic metabolic disturbances in neurons. The observed effect was inhibited by NMDA receptor antagonists and phospholipase A_2. The results, even though they were obtained from the in vitro model experiments on the cultured neurons, illustrate possible participation of the NMDA receptors in the release of arachidonate in the ischemic brain. It was found in preliminary control experiments that the NMDA receptor mediated, Ca^{2+} and phospholipase A_2 dependent arachidonate release operates also in situ, in the hippocampal neurons, that are rich in the NMDA receptors.

ROLE AND SIGNIFICANCE OF BIOACTIVE LIPIDS IN SIGNAL TRANSDUCTION PATHWAY IN NORMOXIC, ISCHEMIC AND AGED BRAIN

(Professor Joanna Strosznajder)

The studies concerned the role of biactive lipids in signal transduction pathway in normoxic, ischemic and aged brain.

In the studies on brain aging the significant changes in regulation of phospholipase activity by Ca^{2+} ions were observed. Our results have shown that exclusively membrane bound enzymes involved in degradation of pohosphatidylinositol express significantly higher activity in the presence of endogenous Ca^{2+} in aged brain as compared with adult. These enzymes are much less susceptible for further regulation by Ca^{2+} ions as compared to the enzyme activity from adult brain. Aging was without any effect on membrane bound phospholipase C, acting against bisphosphophosphatidylinositol, and on the activity of cytosolic enzymes. These results suggests that the aging-induced changes in Ca^{2+} ions redistribution may be responsible for modification of Ca^{2+} dependent membrane bound phospholipases and subsequently for the higher level of diacylglycerol, fatty acids and lysophospholipids in the brain. Taking into consideration very important role of arachidonic acid in the signal transduction pathway particular attention was put on the cholinergic and serotonergic receptor mediated arachidonic acid uptake into membrane lipids and subsequently on its release.

The involvement of pre- and postsynaptic receptors and the effect of brain aging was included in the studies.

Activation of both pre- and postsynaptic receptors leads to liberation of Ca^{2+} dependent arachidonic acid pool through phospholipase A_2 exclusively in adult but not in aged brain. The significant differences between the receptors mediated events were observed.

Dynamics of arachidonate release by serotonergic receptor is significantly different as compared to cholinergic receptor mediated event. A small transient

pool of AA is released by serotonin (5-HT) – μM during a very short time in the presence of endogenous Ca^{2+} ions probably elevated by IP_3 formed simultaneously during activation of phosphoinositides degradation by 5-HT2 receptor. Presynaptically a very small pool of AA release is potentiated by 50 Mm KCL. Opposite to 5-HT receptor muscarinic cholinergic receptor (Mach) mediated AA release depends on the presence of high Mm Ca^{2+} concentration, high agonist concentration and long 30–60 min time of its action. Higher than 10 μm concentration of 5-HT and prolonged time of the agonist action stimulate AA uptake into lipids, probably through 5-HT_1 receptor.

Serotonin activates specifically AA uptake into phosphatidylinositol by GTP binding protein and aging eliminates this process. Uptake of AA is not regulated by ACh receptor. The specific and selective effect of aging on receptor mediated AA uptake and release may be a very important event responsible for disturbances of neurotransmission and memory in aged brain. The results on serotonergic receptor mediated events justify application of 5-HT receptor antagonist-ketanserine as a protective agent against ischemic induced metabolic changes.

Ketanserine applied 15 min before 10 min global ischemia significantly prolonged survival time of this type of ischemia and decreased phospholipase C activity acting on phosphatidylinositol, thus protecting brain against accumulation of diacylglycerol (DAG). The effect of ketanserine on enzymes responsible for AA release was negligible. Application of the 5-HT_{1A} receptor agonist ought to protect the brain against arachidonic acid accumulation during ischemia.

In the studies on the protection and regulation of signal transduction pathway during ischemia the Platelet Activating Factor, PAF and the antagonist of its receptor – gingolide – BN 52021 were used. It was observed that PAF significantly activates phosphoinositide cycle in the brain and that the antagonist of PAF receptor protects the brain against release and action of DAG, IP_3, and AA. The studies on the role of PAF and sphingosine in the normoxic and ischemic brain were carried out in cooperation with the Department of Biochemistry in Perugia and the Department of Hormone Research of Weizmann Institute in Israel.

ROLE OF HISTAMINE IN THE NEUROTRANSMITTERS TRANSPORT MODULATION, METABOLISM AND FUNCTION OF CAPILLARIES AND ACTIVITY OF CALMODULIN IN CENTRAL NERVOUS SYSTEM

(Assoc. Professor Urszula Rafałowska)

The mechanisms of histamine action and transport of its precursor histidine were investigated in the rat brain synaptosomes which underwent the ADP-Fe/ascorbate-induced peroxidation. Peroxidation impaired histidine uptake by 40% and its release by 25% of the maximal uptake. Simultaneously, there was a marked decrease of the synaptosomal histamine content, to about

30% of contro values. Activity of the two histamine-metabolizing enzymes histidine decarboxylase and histamine N-methyl-transferase were drastically lowered by 40 and 60% of the control values respectively. Pretreatment of rats with glucocorticoid analog dexamethasone (1 mg/kg of body weight) did not influence significantly the effects invoked by peroxidation on histamine levels and the activity of enzymes but impaired the histidine transport. These results indicate that the iron-dependent peroxidation decreases both neuronal pool of histamine and its turnover, which may affect the central nervous system function.

In other experiments rats were submitted to normobaric hyperoxygenation. Their brain synaptosomes were isolated and uptake as well as release of histamine precursor-histidine, histamine level and histamine metabolizing enzymes activities were measured. In hyperoxic synaptosomes this uptake was inhibited by about 20%. After 1-hour hyperoxia, a tendency towards an increase of histamine level with a significant increase of histidine decarboxylase and histamine methyltransferase activity were found. Four-hour hypoxia caused a decrease of both the histamine level and the activities of the enzymes measured. The changes were reversed by 1-hour posthyperoxic recovery except for histidine uptake which remained inhibited.

All these findings indicate that the mechanism of histamine uptake as well as activities of histamine metabolizing enzymes in the brain synaptosomal fraction are highly sensitive to free radical formation under conditions of normobaric hyperoxia of rats.

MODULATION OF SIGNAL TRANSDUCTION IN CNS PATHOLOGY – THE INVOLVEMENT OF CALCIUM, PHOSPHOLIPID-DEPENDENT PROTEIN KINASE (PKC)

(Assoc. Professor Krystyna Domańska-Janik)

PKC has been proposed as a link which couples the short-term responses initiated by neurotransmitters and second messengers with the long-term alternation in the cell function. We have demonstrated the dual effect of transient (6 min) gerbil brain ischemia on PKC, monitored by immunoelectroblotting technique. The response involved initial translocation/activation toward membranes with the subsequent enzyme down-regulation to about 50% of control within 3–6 hours of recovery. The above PKC changes might contribute to the irreversible neuronal degeneration resulting in the selective cell loss as estimated by the decrease in lipid-bound sialic acid (ganglioside) concentration 3 and 7 days after ischemia.

Continuing the study on the postreceptor amplification of cAMP-signaling system in the response to brain ischemia we have demonstrated a rapid decrease in the agonist binding to A_1 – adenosine receptor, followed by functional,

reversible impairment of A_1 – adenosine signal transduction to adenylate cyclase. The results implicate that physiological neuroprotective action of adenosine might be impaired by the ischemia due to PKC-dependent inactivation of the inhibitory GTP binding protein (Gi) coupled to A_1 receptor and adenylate cyclase system.

Further biochemical characterization of the dysmyelinating mutant pt rabbit was focused on the abnormal protein turnover reported previously. Calcium activated neutral proteinase (CANP) was estimated by its catalytic activity and immunoblot analysis. Two forms of CANP, one activated by calcium in μmolar concentration (μ-CANP) and the other with low calcium sensitivity (m-CANP) were found in myelin and premyelin fractions. The developmental pattern and the higher enzyme expression in pt mutant myelin implicates their involvement in the myelin formation, turnover and in generation of the charge isomers of myelin basic protein.

The role of adenosine in myocardial hypertrophy (aortic construction and pharmacologically – induced hypertension in rats) has been studied in isolated heart model. The observed decrease of interstitial as well as effluent levels of adenosine suggests that hypertrophy results in an enhancement of the adenosine degradation and/or uptake mechanisms, which may have a significant influence upon interstitial adenosine concentrations achieved under various experimental conditions.

DEPARTMENT OF NEUROPATHOLOGY
3 Dworkowa Str. 00-784 Warsaw
Telephone: 49 54 10

Head: Assoc. Professor Irmina B. Zelman

THE MECHANISM OF ISCHEMIC CENTRAL NERVOUS SYSTEM DAMAGE: LATE EFFECTS AND THE ROLE OF ENDO- AND EXOGENOUS EXCITOTOXINS

(Professor Mirosław J. Mossakowski)

The studies were conducted with the use of 2 experimental models: 1) – global CNS ischemia in the course of cardiac arrest in the rat and 2) – short-term forebrain ischemia in the Mongolian gerbil, being also supplemented with the analysis of relevant clinical material.

Model studies have focused on the changes in the cerebral vascular bed. Scanning electron microscopic observation revealed that global CNS ischemia results in profound ultrastructural alterations in the cerebral microvessels, including vessel construction, endothelial surface changes and formation of microthrombi. The changes become apparent in the early postischemic phase and recede after one week. Transmission electron microscopic evaluation of the same material disclosed the presence of both reactive and regressive changes, the former pointing to the increased potential for transendothelial transport. The use of horse radish peroxidase as a marker allowed to distinguish two phases of the blood-brain barrier damage. The first phase, related to venous stasis, appears immediately after ischemia, whereas the second phase shows a late onset (6 h after ischemia). In addition, global cerebral ischemia was observed to lead to marked changes in the immunoreactivity of the vasoactive intestine peptide (VIP) throughout the cerebral tissue. The changes included the vascular bed, manifesting postischemic disturbances of the vascular reactivity.

Electron microscopic studies performed in the same model with the use of the gold staining technique revealed abundant accumulation of calcium deposits in the synaptic clefts and postsynaptic structures of hippocampus, which is consistent with the notion that calcium participates in the ischemic damage. The early postischemic period was also found to be accompanied by a marker increase in the cerebral PGI_2 level. However, this increase was too low to provide physiologically meaningful compensation for the dramatic rise of other prostaglandins known to promote vasoconstriction and platelet aggregation. The early recirculation period following global ischemia is manifested by a biphasic rise of the cerebral inositol trisphosphate (IP3) level, in good agreement with the postulated ischemia-induced increase of the glutamate-glutamate receptor interaction.

Complementary studies performed on the autopsy material of cardiac arrest cases, as well as studies on the short-term forebrain ischemia model have concentrated on the astrocytic changes. Taken together, the results have pointed to the primary character of the astrocytic damage, not related to neuronal changes. Specifically, the astrocytes showed a decreased immunoreactivity against the astroglia-specific protein GFAP. The astrocytic reaction has also been analyzed in a different set of clinical material, comprising temporal lobes resected from patients with many-year temporal epilepsy. Here, the astrocytes showed an increased immunoreactivity against both GFAP and S-100 – one other glia-specific marker protein.

In studies on the organotypic culture of the rat hippocampus, the excitotoxic aspect of ischemia was simulated by addition of an NMDA receptor agonist – quinolinic acid. The ischemia-specific tissue damage induced by this compound was in a large degree prevented by the administration of a calcium channel blocker- verapamil.

The effect of premature birth and perinatal asphyxia on a number of cerebral antigens in the pontocerebellar system was evaluated in newborns. The changes observed included a decreased immunoreactivity against the carbohydrate epitopes Leu-M1/FAL and Leu7/HNK-1, the neuron-specific enolase (NSE) and the S-100 protein.

CENTRAL NERVOUS SYSTEM AGING AS A BASIS OF DEMENTIVE SYNDROMES

(Assoc. Professor Irmina B. Zelman)

The investigations have focused on a thorough immunocytochemical characterization of the human autopsy material, and were complemented with model studies on the organotypic culture of the rat hippocampus, and on the "pt" rabbit, which is a dysmyelination mutant.

The pathophysiological correlates of amyloidal changes in cerebellum and their correlation with amyloid deposits in other brain structures have been analyzed in the autopsy material derived from 100 patients who died because of stroke at the age of 80 years and above. Cerebral amyloid deposits were observed in 72 cases. While in 31 of these cases the deposits also involved cerebellum, there was not a single case with a selective cerebellar involvement. There was no correlation between the abundance of amyloid deposits and the clinical symptoms of dementia, cerebeller disturbances, or with diabetes or hypertension in the period preceding stroke. As opposed to the cerebral amyloid, the cerebellar amyloid showed no immunoreactivity against the "tau" antigen and PHF ("paired helical filaments"), pointing to the absence of neurofibrillary degeneration.

The expression of ubiquitin and the tau protein was compared in the neurofibrillary tangles (NFT) within the locus coeruleus (l.c.) and nucleus

centralis superior (n.c.s.), in different diseases (Alzheimer disease, Parkinson dementia, senile dementia). Regardless the syndrome, ubiquitin expression has occured later and involved a smaller fraction of NFT in l.c. than in n.c.s., whereas tau-1 immunoreactivity has been less frequent in n.c.s.

A comparative analysis of the expression of a microglial marker, ferritin, was carried out on autopsy material derived from Alzheimer disease, Parkinson disease and senile cases. The most profound differences were noted in substantia nigra, where abundant proliferation of immunoreactive microglia clearly distinguished Alzheimer disease from Parkinson disease cases.

The effects of two excitotoxins: glutamate (GLU) and quinolinic acid (Quin) on the ultrastructural features and the expression of tau protein were analyzed in an organotypic, differentiated culture of the rat hippocampus. GLU, but not Quin, induced filament bundles in the cultures. However, both Quin and GLU enhanced the tau immunoreactivity, which indicates the effect to be mediated by the NMDA class of GLU receptors.

The development of early senile changes in the brains of 3–5 years old normal, healthy rabbits and in the pt mutants with disturbed myelination were compared. The mutation enhances and accelerates the changes without affecting their pattern. Changes in the myelin sheaths and myelinated fibres were the only features that distinguished the involution process in the pt mutant. It appears that in healthy rabbits, the process commences too early for the myelin changes to become manifested.

EFFECTS OF ENDO- AND EXOGENOUS NEUROTOXINS ON THE TRANSPORT AND METABOLISM OF AMINO ACID NEUROTRANSMITTERS AND THEIR PRECURSORS IN DIFFERENT METABOLIC COMPARTMENTS OF THE BRAIN

(Assoc. Professor Jan Albrecht)

Disturbances of amino acid neurotransmission and the pathomechanism of hepatic encephalopathy.

Acute hepatic encephalopathy (HE) induced in rats with a hepatotoxin, thioacetamide (TAA), resulted in a decrease of synaptosomal uptake of two metabolic precursors of glutamate and GABA: glutamine and ornithine, but caused an increased uptake of other precursor – 2-oxoglutarate (2-OG). These uptake changes were opposite in their direction, and possibly controlled by the changes in the cerebral contents of the respective compounds. Since all three precursors are astroglia-derived, the uptake changes are thought to represent an adaptive response of the glutamatergic or GABAergic nerve endings to altered astrocytic functioning.

Excessive loading of the rat brain with ammonia in three experimental models: simple hyperammonemia, TAA-induced HE and in vitro treatment

with ammonium ions, was observed to decrease the activities of malate dehydrogenase and aspartate aminotransferase – the two mitochondrial enzymes involved in the synthesis of the neurotransmitter pool of glutamate. The effects were selectively manifested in mitochondria derived from the nerve endings. All the three treatments have led to the inhibition of pyruvate carboxylase – an enzyme responsible for the replenishment of the Krebs cycle constituents participating in glutamate synthesis. The enzyme activity, and thus the inhibitory effect, were confined to the nonsynaptic mitochondrial fraction, largely enriched in astrocytic mitochondria. This result points to the disturbances of the metabolic communication between the nerve endings and astrocytes as a major cause of disturbances of amino acidergic neurotransmission in HE.

The depolarization (high potassium) – induced release of the L-glutamate analogue – D-aspartate was measured in hippocampal slices derived from rats with simple hyperammonemia, TAA-induced HE and in „normal" slices treated with ammonium ions. HE inhibited the release, whereas hyperammonemia produced no effect, and in vitro treatment with ammonia resulted in an increase of the release. Hence, impairment of synaptic glutamate exocytosis is the phenomenon that distinguishes HE related to toxic liver failure from the direct influence of ammonia, and emphasizes the role of other factors than ammonia in the pathophysiological mechanism of HE.

TAA-induced HE was observed to increase both spontaneous and depolarization-evoked release of newly loaded inhibitory neurotransmitter GABA from the cortical, but not the cerebellar or striatal slices. An increased release of GABA in the cerebral cortex may contribute to the endogenous benzodiazepine-mediated enhancement of GABAergic tone, which is thought to be partly responsible for the pathophysiological mechanism of HE.

Pretreatment of cerebellar granule cell cultures for 24 h with elevated concentrations of ammonium ions enhanced the release of endogenous L-aspartate, an amino acid of well confirmed excitatory properties. This result adds L-aspartate to the list of the amino acid neurotransmitters of potential significane in the pathomechanism of HE.

Neurotoxicity of inorganic mercury.

Studies have been initiated on the puzzling phenomenon of neurotoxicity related to the systemic administration of inorganic mercury salts – compounds known for their inability to penetrate the blood-brain barrier. Intraperitoneal administration of a single dose of mercuric chloride was observed to lead to a rapid and irreversible inhibition of Na/K-ATPase activity in rat cerebral capillaries. The impairment of the capillary sodium pump is likely to contribute to distrubances of ion homeostasis in the brain and as such may be considered as a cause of "indirect neurotoxicity" of mercuric chloride.

LABORATORY OF DEVELOPMENTAL NEUROPATHOLOGY
3 Ludwika Pasteura Str., 02-093 Warsaw
Telephone: 22 96 27

Head: Professor Maria Dąmbska

THE COMPARISON OF BRAIN DEVELOPMENT IN NORMAL CONDITIONS AND DISTURBED BY SELECTED DAMAGES AND PATHOLOGIC PROCESSES

(Professor Maria Dąmbska)

The investigations were carried out on human autopsy material and experimental animal models.

1. The morphometric analysis performed in hippocampus of newborns at term allowed to evaluate the maturing neuronal population at this age and to compare the cases estimated as normal without pathologic changes with the results obtained from children who died because of perinatal hypoxia. The susceptibility of neurons in sectors CA1 and CA4 was found as independent of their immaturity. The proliferation of astrocytes with positive reaction for glial fibrillary acidic protein (GFAP) in temporal lobe was found to be increased in the brains of newborns with perinatal hypoxic encephalopathy.

The CNS malformations were studied in the cortex and in spinal cord. The anomaly consisting of deep disorganization of cortical structure was considered as a result of circulatory disturbances including an early damage of cortico-pial barrier. The spinal cord malformation was described as a background of post-irradiation myelopathy.

2. The experimental study concerned the effect of vincristine administration, the known antimitotic and neurotoxic drug, on central nervous system of young rabbits. The regions lacking of blood-brain barrier were examined. In many neurons the death of "apoptotic type" was observed. In Peyer's patches, where apoptosis appears in normal conditions, this phenomenon was found more frequently after vincristine administration. These results show that apoptosis plays a particular role in the antineoplastic action of vincristine and may be responsible for many side effects observed during the treatment.

LABORATORY OF THE STRUCTURE OF THE NERVOUS SYSTEM
3 Dworkowa Str., 00-724 Warsaw
Telephone: 49 54 20

Head: Professor Jerzy Borowicz

STUDIES ON THE RAT HYPOTHALAMO-HYPOPHYSEAL SYSTEM FOLLOWING SUSTAINED CLINICAL DEATH (IN THE LATE PERIOD OF 6–10 MONTHS)

(Professor Jerzy Borowicz)

The studies concerned remote cerebral changes, particularly in the hypothalamic secretory nuclei obtained from rats after global ischemia, due to 5 and 10 min clinical death. Striking differences in the intensity of the changes were noted, depending on duration of the ischemic incident. It has been also observed that in the structures of neurosecretory system the phenomenon of maturation of pathological processes takes place, being usually connected with other cerebral functional systems. In the previous studies concerning short periods after reanimation the appearance of relatively insignificant pathological changes was noted in neurosecretory nuclei. These changes subsequently resolved, so that ultrastructural appearance was normal or almost normal. The presently conducted electron-microscopic studies carried out on brains of the rats which remained alive for 3 to 6 and 9 months after an ischemic incident revealed intensive pathological processes in neurons; the majority of them were irreversible. They were evident as destruction of nuclear and cytoplasmic skeleton of neurons, apoptosis as manifestation of the process of coagulative necrosis of the cell, of Ca^{++} ions (being a well known indicator of necrobiotic cellular processes).

Moreover, similarly as in previous years, immunocytochemical studies were conducted on the active neuropeptides, including VIP, and their localization in neurosecretory system of the rat in various phases, after sutained clinical death. Their participation in this neurohormone release was shown. The observed changes indicate significant and persisting disorders of neurosecretory processes.

DEPARTMENT OF NEUROSURGERY
58 Białobrzeska Str., 02-325 Warsaw
Telephone: 22 36 43

Head: Professor Eugeniusz Mempel

BRAIN MAPPING IN EPILEPTIC PATIENTS WITH SUSPECTED LESIONS IN FRONTAL REGIONS OF THE BRAIN

(Professor Eugeniusz Mempel)

Brain mapping by the BEAM method was done in a group of 22 epileptic patients with large and small generalized seizures, in whom bilateral discharges dominate in the eeg records from the fronto-temporal regions of both hemispheres. The results of examination allowed in eight cases to detect epileptogenic foci variously localized in the frontal lobes not withstanding the normal results of computer tomography of the brain. It was also possible to visualize spatial propagation of epileptic discharge waves in the brain hemispheres. Brain mapping proved to be the important supplementary examination to eeg and computer tomography of the brain.

Brain mapping was made in epileptic alternating motor-sensory seizures in school children. Computer analysis performed on a representative cases of such seizures, first left, and after 16 years right-sided, of discharges in the frequency and temporal-spatial domains demonstrated the presence of a mirror epileptic focus of enlarged dimensions. These results could not be obtained by neurological examinations, brain computer tomography and magnetic resonance of the brain, the latter method showing normal results.

INFLUENCE OF CHANGES IN INTRACRANIAL VOLUME-PRESSURE RELATION AND CEREBRAL CIRCULATION ON INTRACRANIAL VOLUME COMPENSATION

(Assoc. Professor Zbigniew Czernicki)

The study concerned two problems:

1) Application of physiological loading for evaluation of the intracranial state;
2) Determination of changes in intracranial volume-pressure relations as a function of resistance of the cerebro-spinal outflow.

The analysis of changes in the harmonic components of the arteriogenic wave of intracranial pressure proved particularly useful for evaluation of intracranial

volume compensation. For this evaluation the modified complex High Frequency Centroid method of analysis was applied. The behavior of intracranial pressure under the influence of posture and Quekensteadt's test served as an exponent of what is called the rapid component of the intracranial compensation. Autoregulative efficiency was examined by application of carbogen for respiration. The resulting increase in the cerebral flow was found to be dependent on the current volume-pressure relations. Comparison of the Transcranial Doppler (TCD) results and autoregulation tests allowed to establish a correlation between the two methods.

It was demonstrated that changes in the resistance of the cerebrospinal fluid outflow depend on the intensity of infusion. An increase of this intensity, causing a rise in the intracranial pressure evoked a nonlinear fall of the cerebrospinal fluid outflow resistance. This depression of the outflow resistance value persisiting for about 60 min, even when the infusion intensity is reduced causes serious clinical implications.

INVESTIGATIONS ON THE LATERALISATION OF BRAIN HEMIPSHERES

(Assoc. Professor Jadwiga Szumska)

The time of information transmission concerning verbal and nonverbal visual stimuli between the brain hemishpheres was studied. Twenty two subjects were examined under experimental conditions by applying the method of evoked potential recording from ten points of the brain cortex. It was proved that the interhemispherical transfer amounts to 6–12 ms, occuring in the earlier phase of processing information on stimuli and only in the posterior parts of the brain.

Higher nerve functions were examined in individuals of advanced age (above the 60th year of life). Patients (n = 38) with Alzheimer and Parkinson's disease as well as a control group of healthy individuals were examined by means of a special set of neuropsychological tests. Deep disturbances of memory processes, learning, language and visual-spatial processes were found in patients with Alzheimer's disease.

A group of dyslectic children was examined basing on their performance of motor tasks and the results were compared with those obtained in the age-matched children without dyslexia. The rate of performance of the tasks was distinctly affected by coexisting distrubances of sound perception.

NEUROMUSCULAR UNIT
1a Banacha Str., 02-097 Warsaw
Telephone: 659 75 06

Head: Professor Irena Hausmanowa-Petrusewicz

THE ROLE OF DISTURBED MATURATION OF MUSCLE FIBERS IN PATHOGENESIS OF NEUROMUSCULAR DISEASE

(Professor Irena Hausmanowa-Petrusewicz)

With light microscopy and ME it was shown that the reaction of a muscle cell to injury depends on maturation of this cell. The mature muscle reacts to injury with necrosis whilst the immature with apoptosis.

These two types of muscle cell death are different in respect to the structure and to the disappearance of debris. For the first time in human myology the phagocytic ability of the muscle fibers has been proved. These findings change the previously accepted statement according to which the fetal muscle is resistant to injury.

In congenital dystrophy (expressed clinically as arthrogryposis) the fetal defect of immature muscle fibers was shown. This defect was related to longstanding immunological lesion of capillaries.

Longlasting follow-up of the case of nemaline myopathy permitted to find out: 1) the changes in clinical phenotype – the features of facio-scapulo-humeral syndromes appear in the patient after many years; 2) the repeated biopsies allowed to state the decreased numbers of rods and normal growth of diameter of muscle fibers as well as their normal architecture. It seams that for the first time in clinical myology the pattern of muscle adaptive phenomenon has been demonstrated.

TRANSMISSION ABNORMALITIES

(Professor Barbara Badurska)

The coexistence of two autoimmunological deseases such as myasthenia and multiple sclerosis was demonstrated and the immunological peculiarities of the patients with both these diseases were analyzed.

Using the technique of stimulated SF EMG the abnormalities in neurotransmission in myasthenia with thymoma and without thymoma were shown to have the same characteristics.

The occurrence of very rare Eaton-Lambert syndrome in children was demonstrated.

SPINAL MUSCULAR ATROPHY
(Professor Irena Hausmanowa-Petrusewicz)

The studies on DNA in the childhood SMA were continued. The aim of them was to explain the fact that some families with chronic childhood SMA do not mapp to locus on the chromosome 5 and to explain the male preponderance in benign the SMA group. Patients suffering from SMA were examined by polymyographic tests. It was found out how they perform the multijoint patterned movement and a single-joint volitional task. In the SMA patients, as compared to healthy controls, electrical activity appeared also in muscles not involved normally in a given task. This coactivation, pronounced in SMA, might have a compensatory meaning.

The occurrence of complex (satellite) potentials in the SMA patients was evaluated and their dependence on reinnervation and terminal sprouting was demonstrated.

PRODUCTS OF DYSTROPHIC GENE
(Professor Irena Niebrój-Dobosz)

The interrelationships between phenotypes of Duchenne dystrophy (DMD) and Becker dystrophy (BMD), DNA changes as well as muscle dystrophin were examined. The pilot results are as following: 1) in 50–60% of the tested patients with clinical diagnosis of DMA or BMD the DNA deletion was found; 2) the size of deletion does not correlate with severity of clinical state; 3) the BMD turned out to be much more common than it was used to estimate and it was found to have very variable clinical expression; 4) some cases diagnosed till now as limb-girdle dystrophy turned out to be the BMD cases; 5) due to DNA and dystrophin testing combined with the cytogenetic tests it was possible to recognize the girls with DMD (cases with autosomal translocation or manifesting carriers); 6) in our series the unique pedigree of DMD with deletion but with presence of normal dystrophin was found.

Parallel to clinical studies the experimental work on the dystrophic mice – mds – showed:

Except clinical and genetic studies the biochemical analysis of the experimental model of muscular dystrophy in mdx mice was performed. A deficiency of the main calcium binding protein – calsequestrin – appeared to be present. The consequence of it may be the free calcium content elevation in the muscle cell followed by the increased activity of Ca^{++}-activated protease and muscle necrosis. Using an immunocytochemical method the calcium transporting ATPase in Duchenne's dystrophy was examined. Both in Duchenne's dystrophy, as well as limb-girdle dystrophy the activity of Ca^{++} ATPase appears to be

decreased in terminal cisternae of sarcoplasmic reticulum but increased in sarcolemma and T-system.

The electrophysiological examination of dystrophic muscles showed occurrence of complex (satellite) potentials that correlated with variability of fibres diameter and presence of regenerating fibres.

As exceptional casuistic a case suspected of dystrophy with myoglobulinuria should be stressed. It turned-out to be an inborn defect of H-subunits of LDM.

It is most probably the first case of myopathy connected with this kind of genetical error.

DEPARTMENT OF APPLIED PHYSIOLOGY
17 Jazgarzewska Str., Warsaw
Telephone: 40 40 47

Head: Professor Hanna Kaciuba-Uścilko

INTERRELATIONSHIP BETWEEN THE NEURO-ENDOCRINE FUNCTION AND ENERGY METABOLISM

(Professor Krystyna Nazar)

Stimulation of the sympathetic nervous system (SNS) by insulin is considered as one of the mechanisms promoting development of hypertension in obese hyperinsulinemic patients. The aim of this study was to find out whether plasma noradrenaline (NA) and hemodynamic responses to oral glucose (75 g) in obsese hypertensive (OH) patients differ from those in healthy controls. As a reference stimulus activating SNS orthostatic test was used. Ten male OH patients (age: 38.7 + 2.7 years, BMI: 31.0 + 2.5 and mean blood pressure, MBP: 113 + 3.0 mm Hg) and ten healthy subjects (age: 32.1 = 11.7 years, BMI: 24.7 = 2.8, MBP: 95 + 2 mm Hg) participated in this study. Comparing with controls, OH patients showed significantly greater overall responses of plasma insulin and NA to glucose load calculated as the incremental 2h area under the curve. It was accompanied by a significant increase in systolic, diastolic and mean blood pressure, which did not occur in healthy subjects. In both groups similar increases in heart rate, stroke volume (measured by impedance cardiography), and cardiac output were found. Only in controls there was a significant decrease in the calculated peripheral vascular resistance. The maximal post-glucose plasma NA levels positively correlated ($r = 0.79$, $p < 0.01$) with those obtained during the active orthostatic test (8th min of standing), which suggests that the exaggerated plasma NA response to glucose in OH patients reflects their unspecific SNS hypereactivity.

Continuing the studies on factors modifying neuroendocrine responses to physical exercise an effect of ketogenic low carbohydrate (5%) diet was followed up in young healthy men performing a supramaximal (30s Wingate test) effort. A marked enhancement of the plasma catecholamine response to this kind of exercise, along with the large increase of the plasma beta-hydroxybutyrate and a decreased plasma insulin level as well as a smaller elevation of blood lactate concentration were demonstrated in the subjects on the ketogenic as compared to the normal diet.

Another series of experiments was carried out in order to find out whether a negative shift in subjects' mood alters physiological responses to exercise. For this purpose 20 young men performed graded exercise (50, 100, 150 W) on two occasions: 1) when they came to the Lab. for the first time and were submitted to IQ quiz before exercise, 2) when they were familiarized with the Lab. and rested

before exercise. The subjects' mood was assessed using the Profile of Mood State questionaire and their plasma free and total catecholamine levels were determined. In comparison with 2nd session, in the 1st one the subjects showed higher scores of tension, anger, depression, confusion and global mood before exercise. They also had elevated plasma free and total noradrenaline (NA) with no difference in adrenaline (A) level. A significant correlation was ascertained between the global mood scores and the plasma NA ($r = 0.46$) measured before and after IQ quiz. During exercise there was a parallel increase in free and total NA and both attained significantly higher values in the 1st session. The increases in the plasma free A tended also to be greater in the 1st session, whilst the plasma total A remained virtually unchanged. This suggest that the exercise induced increases in the plasma fee A are, at least partly, caused by the hormone liberation from its sulfaconjugated form.

In the studies on dogs it was proved that even a mild dehydration can markedly enhance the plasma catecholamine response to treadmill exercise.

Metabolic and hormonal changes induced by 8-weeks low calorie diet (1000 Kcal/day) with or without phosphate supplementation were studied in 30 obese women. It was proved that phosphate supplementation (in the form of tablets containing calcium, potassium and sodium phosphates in proportion 21:4:1), enhances the resting metabolic rate and prevents a decrease in the plasma triiodothyronine (T_3) concentration in dieting women. It does not affect the plasma concentrations of cholesterol, triacylglycerol, free fatty acid, glucose, catecholamine, cortisol, insulin, growth hormone and testosterone.

MECHANISMS INVOLVED IN THE MUSCLE INSULIN SENSITIVITY

(Assoc. Professor Leszek Budohoski)

Continuing the previous investigations on the muscle insulin sensitivity it was found that:

1. Improvement of the muscle insulin sensitivity after physical exercise depends on an inhibition of adenosine action, since this effect is abolished by 2-chloro-adenosine (adenosine receptor agonist).
2. An influence of adenosine on the sensitivity of glucose transport to insulin in myocytes is mediated by A^1 – adenosine receptors. These receptors do not affect an interaction of insulin receptors with the hormone, but their activation causes an increase in the tissue c AMP content (in collaboration with the Department of Pharmacology and Therapeutics, University of Leicester, Great Britain).
3. A short-term (15 min) electrostimulation of sciatic nerve of the rat effectively improves sensitivity to insulin of the soleus muscle both the intact and previously tenotomized. The latter condition was proved to impair the sensitivity of lactate production and glycogen synthesis to insulin.

In collaboration with the Department of Anatomy University of Toronto (Canada) it was demonstrated for the first time that insulin controls glucose transport and its oxidation in pre-miocytes from the tissue culture (line L-6).

CORONARY HEART DISEASE IN WOMEN

(Dr. Ewa Wójcik-Ziółkowska)

There are noc clear-cut diagnostic criteria of coronary heart disease (CHD) in women because of a number of false positive results of the standard diagnostic tests. On the other hand, incidents of myocardial infarcts in women increase progressively. Thus, an attempt was made to compare a time-coure of CHD, 5 years after myocardial infarction in 12 women (aged approx. 60 years) with that in 12 men (aged approx. 50 years). There were no significant differences between these two sex-groups in the results of the 24h ECG, evaluate by the Holter method, in the results of exercise tests or echo-cardiography. It should be noted, however, that the plasma level of triacyloglycerol (TG) was higher in women than in men, while the latter showed lower values of HDL-cholesterol.

In a group of 104 women (aged 35–60 years) with clinically suspected CHD complex examinations have been started including besides the standard clinical examinations, an estimation of hormonal and lipid profiles.

HORMONAL MODULATION OF THE RENAL CORTICO-MEDULLARY ELECTROLYTE GRADIENT: ROLE OF PROSTAGLANDINS

(Professor Janusz Sadowski)

In vitro studies have shown that renal prostaglandins inhibit ADH dependent stimulation of NaCl reabsorption in the thick ascending limb of Henle's loop and could thereby dissipate the cortico-medullary electrolyte gradient. In the present work we attempted to reproduce this effect in vivo.

Changes in salt transport were detected by estimating total electrolyte concentration in the medullary interstitium surrounding the loops. This was done by continuous recording of tissue electrical admittance (reciprocal impedance) using needle electrodes placed in the medulla (methodology developed in this laboratory). Total renal blood flow, glomerular filtration rate and parameters of renal excretion were measured simultaneously.

Experiments with male Wistar rats anaesthetized with Inactin showed that inhibition of prostaglandin synthesis with i.v. Indomethacin, 15 mg/kg.h, significantly increased electrolyte concentration in the medullary interstitium,

indicating increasing salt transport in medullary loop segments. The renal blood flow and glomerular filtration rate did not change. There was a net increase of the tubular reabsorption of solutes, mainly electrolytes, without any change in free water reabsorption.

Additional experiments involved measurement of tissue electrical conductance carried out in vitro on pieces of inner medulla obtained from kidneys removed directly after experiments. The "standard conductance" so measured was significantly higher in kidneys of Indomethacin treatet rats compared to non-treated control animals. The medullary tissue concentration of non-ionic solutes was virtually equal in both groups. The standard conductance was significantly below control in another group of animals in which medullary extracellular electrolytes had been "washed out" by a slow intrapelvic infusion of istotonic mannitol solution.

The data suggests very strongly that in anesthetized rats renal medullary prostaglandins inhibit NaCl reabsorption in loops of Henle and thereby dissipate the corctico-medullary electrolyte gradient in the kidney. Inhibition of prostaglandin biosynthesis acts in the opposite direction.

CARDIOVASCULAR LABORATORY
3 Dworkowa Str., 00-784 Warsaw
Telephone: 49 73 55

Head: Professor Krystyna Cedro-Ceremużyńska

EDRF AND PLATELET-VESSEL WALL INTERACTION

(Professor Krystyna Cedro-Ceremużyńska)

There is an increasing evidence that nitric oxide (NO), synthetized in vascular endothelium and in platelets by NO synthase, influences vascular tone, down regulates platelet function and platelet-vessel wall interaction both in vitro and in vivo. We have investigated the effect of a NO synthase inhibitor, N^G-mono-methyl-L-arginine (L-NMMA, 100 mg/kg i.v.) on the platelet-endothelial cell interaction in the rabbit arteries ex vivo using scanning electron microscope (SEM). The effect of L-NMMA was examined on an intact endothelium and on that damaged by arterial constriction. The infusion of L-NMMA increased systemic blood pressure and decreased carotid blood flow, however, it did not change the appearance of an intact endothelium and it did not result in platelet activation on the intact endothelial cells. In contrast, SEM of the endothelial areas damaged by constriction showed extensive platelet adhesion and aggregation on subendothelium. These morphological changes were not detected in control animals with intact or damaged by arterial constriction endothelium. The results show that under physiological conditions, an inhibition of NO synthase alone does not result in platelet activation in vivo. However, when combined with endothelial injury it may lead to platelet activation and thrombosis. The results obtained in collaboration with te Dept. Pathology, Child Health Center, in Warsaw confirm the role of NO-synthase as regulatory mechanism promoting hemostasis and preventing thrombosis.

PLATELET ACTIVITY IN ACUTE MYOCARDIAL INFARCTION (MI). CLINICAL INVESTIGATION

(Professor Krystyna Cedro-Ceremużyńska)

Platelet hyperactivity in acute MI is well documented but its relation to the course of this desease remains to be established. We have investigated a relation of the platelet activity (adhesion and aggregation to collagen, measured by the methods of Mant and Born, respectively) to the intensity of rhythm disturbances (Lown grading) evaluated by Holter monitoring in 42 patients during the first

24h of acute MI diagnosed by clinical, biochemical and ecg criteria. Platelet aggregation was not significantly different in the patients with AMI and in the controls (healthy, age-matched subjects, $n = 16$). Platelet aggregation in the patients with a stable rhythm and in those with complex arrhythmias did not differ significantly. The platelet adhesion was significantly increased in the patients with AMI as compared controls ($p < .001$) and it was significantly ($p < .01$) higher in patients with complex arrhythmias (Lown 3–4b) than in those with a stable rhythm (Lown 0–2).

Platelet adhesion, the earliest observable platelet response in contact with collagen, is considered as an indicator of platelet interaction with the vessel wall. It may contribute to formation of intracoronary aggregates and to potentiation of arrhythmias.

However, it may also result from more advanced endothelial injury and more intense humoral response to MI in the patients with threatening arrhythmias. Although increased platelet adhesiveness and its relation to the rhythm disturbances in AMI have been documented by this study, causal relation of these findings remains to be established.

The above described study was performed in collaboration with Dept. Cardiology, Postgraduate School, Grochowski Hospital, Warsaw.

LABORATORY OF EXPERIMENTAL SURGERY
Praski Hospital
67 Solidarności Str., 03-401 Warsaw
Telephone: 19 16 94

Head: Assoc. Professor Maciej Borkowski

DIAGNOSTIC STUDIES AND THERAPY IN PATIENTS WITH PERIPHERAL, VASCULAR DISEASES

(Assoc. Professor Maciej Borkowski)

Remote effects of peripheral, vascular surgery have been examined in patients during at least one year following the vascular transplantation of various types and the aortal aneurism operation. This study includes clinical examinations, reography and UDP blood flow measurements.

DEPARTMENT OF SURGICAL RESEARCH AND TRANSPLANTATION
5 Chałubińskiego Str., 02-004 Warsaw
Telephone: 21 49 23

Head: Professor Waldemar Olszewski

NEURO-ENDOCRINE REGULATION OF CAPILLARY TRANSPORT OF PROTEINS AND CELLS INTO THE TISSUE SPACE AND LYMPHATICS

(Professor Waldemar Olszewski)

The phenotypic characterization of cells migrating from skin to afferent lymph in humans under normal conditions and during chronic lymph stasis was performed. It was found that afferent lymph from patients with lymph stasis contains an increased number of cells (mean 12×10^6 ml). Compared to normal lymph there was less T, and more B cells, whereas the percentage of Langerhans cells was unchanged.

The effect of increased venous pressure on cell migration from peripheral lymph node was studied in dogs. It was found that increased venous pressure resulted in a decrease of lymphocyte output (10-times) with the increase of erythrocyte output (29-times) into the efferent lymph. The difference between lymphocyte and erythrocyte release from lymph node suggests that immunoregulatory but nit hydrostatic factors are involved in regulation of lymphocyte traffic through lymph node.

TRANSPLANTATION OF ALLOGENEIC CELLS AND ORGANS

(Professor Waldemar Olszewski)

The aim of the study was to examine whether donor specific transfusion (DST) and Cyclosporine A (CsA) treatment, prolonging organ allograft survival, would also prolong the survival time of lymphocyte allografts. It was found, that DST treated rats rejected hyperacutely allogeneic lymphocytes. This indicates that DST protects the organ allograft but the recipient remains fully reactive to donor antigens. Presumably the beneficial effect of DST on organ allograft was brought about by elimination of passenger cells. It was observed that sera from DST-treated rats accelerated the destruction of i.v. injected donor lymphocytes despite of low titers of cytotoxic antibodies.

The kinetics of the repopulation of bone marrow cavities and lymphoid organs in irradiated rats receiving syngeneic hind limb graft or bone marrow

cells i.v. was studied. Recipients were exposed to 8.0 Gy of γ irradiation. Faster recovery of bone marrow and lymphoid tissues were observed in recipients of bone marrow rats after limb grafts compared to the group receiving 60×10^6 bone marrow cells i.v.

REGULATORY FUNCTIONS OF LYMPHOID CELLS
(Professor Waldemar Olszewski)

Previous studies have shown that normal rat liver sinusoids are populated by blood mononuclear cells with high natural cytotoxic activity against tumor cells. Twenty four percent of these cells revealed azurophilic granules characteristic for large granular lymphocytes (NK cell). The predilection of this cell population to home in the liver after an intravenous injections was examined. The results suggest that subpopulation of mononuclear cells, isolated from liver sinusoids and enriched in NK cells on Percoll gradient, homes preferentially to the liver, while subpopulation depleted in NK cells migrates mainly to lymphoid tissues.

The selective migration of lymphocytes to rat liver sinusoids was also investigated on a liver through-portal vein perfusion model. It was found that there is physiological retention of live mononuclear cells in the liver sinusoids, with the capacity of 10^6 cells/g of organ. More syngeneic than allogeneic cells were retained. Syngeneic serum in the perfusion system facilitated retention of allogeneic cells.

The activity of human NK cells in long lasting (4 weeks) bone marrow cultures as well as IL2 and GM-CSF effect on NK progenitor cells in the presence of autologous stromal cells were studied. Cytokine-dependent differences in generation of different subpopulations were noted. Generation of cells, morphologically characterized as LGL and displaying cytotoxic activity against K562 and Daudi cells was observed only in the presence of r-IL2. Most of these cells were CD56 positive. The results point to the presence in bone marrow of CD33 progenitor cells which, in the presence of IL2, can differentiate into nature CD56 positive NK cells.

Distribution of the donor alloantigen (splenocytes) and antidonor alloserum in the prospective organ recipient was examined. The high level of alloserum was observed in recipient's serum, liver and spleen 3 days after infusion. A large fraction of donor lymphocytes was eliminated in the presence of alloserum. Alloserum – donor lymphocyte complexes accumulated in the spleen and liver but not in the lymph nodes. It seems that high levels of alloserum persisting in recipient's serum may block donor antigens in serum. Accumulation of donor lymphocyte – alloserum complexes circulating in the spleen and liver points to the active elimination of alloantigen.

The effect of CyA, an immunosupressive drug preventing allograft rejection, on in vivo migratory properties of the rat lymphocytes from thoracic duct and

mesenteric lymph nodes was studied. Only few CyA-treated thoracic duct lymphocytes could be retrieved from the thoracic duct. Evidently less radioactivity of CyA-treated lymphocytes were detected in blood, spleen, liver and mesenteric lymph nodes, compared to untreated animals. CyA-treated mesenteric lymph node lymphocytes accumulated at a lower rate in blood, spleen and mesenteric lymph nodes but more in lungs. The results indicate that CyA does affect the migratory properties of lymphocytes.

The proliferative function of normal human immune peritoneal cells and specific role of peritoneal macrophages in peritoneal lymphocyte responsiveness to mitogens were investigated. It was found that peritoneal macrophages were responsible for the regulation of proliferative response of perineal cell population, since blocking with monoclonal antibodies against macrophage (a-CD68) and class II antigens (anti-HLA-DR) caused a decrease of their proliferative response to mitogen.

Resident skin dendritic cells seem to play a dominant role in the rejection of skin allograft. These cells are required for T-dependent immune responses and can bind T resting cells. To assess the role of dendritic cell – lymphocyte interactions in the development of immune response in skin, the spontaneous binding of autologous lymphocytes by dendritic cells from the dog afferent lymph, as well as the effect of immunosuppressive and anti-inflammatory drugs on binding were studied. Only prednisolone caused the significant decrease of dendritic cell – lymphocyte clustering in vitro. It appears that controlling of cell clustering by local drug administration may be helpful in mitigation of skin immune reactions.

IMMUNE RESPONSE AFTER TRAUMA

(Professor Waldemar Olszewski)

The influence of operative trauma (transplantation of hind limb) on hemopoiesis was studied in the Lewis rats. No differences in the number of cells of erythroid and myeloid lines were observed 10 days after the transplantation. In the thymus and lymph nodes accumulation of macrophages in enlarged sinusoids was seen. After 30 days the number of cells in bone marrow decreased compared to values noted after 10 days, while histological pictures of lymphatic organs were normal. Results suggest that normal hemopoiesis in rats – recipients of syngeneic hind limb grafts may be caused by stimulation by cytokines released from traumatized tissue (mainly IL1).

The effect of human skin lymph on proliferation of various human tumor cell lines was examined. T, B, melanoma and sarcoma cell lines were used. Considerable variations in reactivity of different tumor cells to the presence of lymph in culture medium was observed. Generally, low concentrations (5–20%) stimulated proliferation of most cell lines, whereas higher concentrations

(40–80%) had an inhibitory effect. Preincubation of tumor cells with lymph (1–3 days) had the same inhibitory effect as its continuous presence in media during assay. Similar effects of lymph and serum (with the same as lymph protein concentrations) on almost all tumor cell lines indicate that the substances transported from plasma, beside of some locally produced humoral factors (IL1), play a regulatory role in lymph. Since blocking of lymph IL1 activity with anti-IL1 antibody resulted in an increase of tumor cell proliferation, it seems that the monokines may be involved in the control of tumor growth in skin environment.

MENTAL HEALTH DEPARTMENT
12 Żeromskiego Str., 90-711 Łódź
Telephone: 33 64 57

Head: Assoc. Professor Zygfryd Juczyński

CRITERIA OF THE EFFECTIVENESS OF TREATMENT OF ALCOHOL DEPENDENT PATIENTS

(Assoc. Professor Zygfryd Juczyński)

Total abstinence is certainly the best and the simplest predictor of a change in a person's relations to alcohol, but it is not the only measure of the effectiveness of treatment. The purpose of this study was to identify such predictors among different variables, which would be closely linked with the effectiveness of treatment, and more importantly, which would allow to anticipate the after treatment changes. Effectiveness of treatment (dependent variable), has been estimated during the last two years including (besides relation to alcohol) an estimate of the health state and the level of psychological and social adjustment of an individual.

In this study 220 men at the age of 40 years from Łódź were examined. They have been treated in the past for alcohol dependence. The follow-up assessment data was available for 25% of the examined subjects. Multiple regression analyses were used to assess the value of the measures as predictors of the outcome status. Twenty medical, psychological and social variables, based on catamnestic data, were included into these analyses. It was revealed that: 1. the number of hospitalizations; 2. duration of intervals between drinking incidents; 3. employment stability; 4. type of treatment and 5. marital stability, are the best predictors of treatment effectiveness.

These five predictors explain 65% of the total variance of criterion variable. The likehood of remission from alcohol dependence, amelioration of health state, psychological and social adjustment are very important for those subjects, who were only once or never hospitalized because of alcoholism, had relatively long (several months) intevals between drinking bouts, continued outpatient treatment after hospitalization and had relative employment and marital stability.

THE LIBRARY
75 Rolna Str., 02-813 Warsaw
Telephone: 43 34 94

Head: Krystyna Marczakowska

The library constitutes one Department of the Medical Research Center and acts as an information source for scientists.

Library structure: main library with affiliated special Library in Łódź. Scope and the subject profile: physiology, neurosciences and experimental surgery including transplantology.

Present holdings:
books – monographic and serial volumes (Polish and foreign) – 17525
periodicals, newspapers (number of titles) – 625

Reference aids:
catalogues – alphabetical: books, periodicals and microfiches
– subject: books
main card-files – bibliographical list of papers published by scientists of the Medical Research Center Polish Academy of Sciencef from 1967.

Users:
scientific workers of the Medical Research Center, interlibrary loans available for all scientific Institutes in Poland and abroad.

Bibliography of library: a list of new books and current periodicals prepared weekly. On the basis of the Science Citation Index a report of citations of papers published by MRC Scientists in 1989–1990 was prepared. It contains 1180 citations.

MEDIPAN – Scientific Instruments Department
7/11 Wiktorska Str., 02-287 Warsaw
Telephone: 48 22 62

Head: Andrzej Lasek

MEDIPAN Pilot Plant is a producer of a special equipment for the Institute of Experimental and Clinical Medicine (MRC) of the Polish Academy of Sciences as well as the medical equipment for other medical Service units.

Infusion pumps of different functional groups represent the basic assortment of the Plant produced for medical service needs. This year 290 pumps were produced by MEDIPAN.

Additionally the Plant manufactures cytologic centrifuges and a device for exercise testing consisting of a physiotest, bicycle ergometer as well as a printer for result recording.

MEDIPAN infusion pumps can be classified into two basic functional groups:

1. Syringe pumps models 610 AS and 610 BS as well as microprocessor-controlled model 611 syringe pump.

The models mentioned above are designed for long lasting intravenous injections of small quantities of highly concentrated drugs.

2. Peristaltic pumps for intravenous drips, manufactured so far in 4 models: 601 SP, 602 SP, 605 SP and 6050. They are characterized by functionality and ergonomy of manipulating elements.

Each model of pump differs from the others in a range of functions that can be performed. Model 601 SP enables setting up a flow rate, dosing volume, indication of a currently dosed volume as well as legible signalization of alarm states. The model 602 SP represents a simplest pump, which enables setting only a flow rate volume. In 1991 further modernization of the pumps produced so far was continued. Besides, both economical and technological foundations of the infusion drip pump – model 604 have been elaborated. This newest model, when constructed, will meet all the requirements of the European Community norms.

INTERNATIONAL COOPERATION

VISITING SCIENTISTS

Department of Neuropathology

Taljaard J.J.F.	Research Unit for the Neurochemistry of Mental Diseases University of Stellenbosch, Faculty of Medicine, Tygeberg, South Africa
Haugvicova R.	Institute of Physiology, Czechoslovak Academy of Science, Prague, Czecho-Slovakia
Gannushkina I.	Institute of Neurology, Academy of Medical Sciences, Moscow, EUN
Victorow I.	Institute of Brain Research, Academy of Medical Sciences, Moscow, EUN

Department for Surgical Research and Transplantation

Ahonen J.	Clinic of Transplantation, Univeristy of Helsinki, Finland
Mito M. Mito H.	Clinic of Surgery, University of Asahikawa, Asahikawa, Japan
Siranath D.	TATA Cancer Institute, Bombay, India
Sinha A.K.	Leprosy Control Unit, Tarapur, India
Husien A.	Institute of Tropical Medicine, Cairo, Egipt

Department of Neurochemistry

Tosic M.	Laboratoire de Neurochimie, Service de Pediatrie CHUV, Losanne, Switzerland
Matthieu M.	Centre Hospitalier, Universitaire Vaudois, Losanne, Switzerland

De Nechaud B.	Biochimie Cellulaire, College de France, Paris, France
Pylova S.	Institute of Reanimatology, Medical Academy of Sciences, Moscow, EUN

Department of Neurophysiology

Paulev P.E. McCord K.	Department of Medical Physiology, University of Copenhagen, Copenhagen, Denmark

Department of Applied Physiology

Newsholme E.	Department of Biochemistry, University of Oxford, Oxford, United Kingdom
Porta S.	Institute of Functional Pathology, University of Graz, Graz, Austria
Raccota R.	Lab. of Physiology, Department of Biology, University of Mexico, Mexico

VISITS ABROAD

Department of Neuropathology

Hilgier W.	School of Medicine, Department of Emergency, Wright State University, Dayton, Ohio, USA (long term visit)
Kida E. Januszewski S. Pluta R.	Institute for Basic Research and Developmental Disabilities, Staten Island, New York, USA (long term visit)
Krajewski S.	Department of Neuropathology, University of Düsseldorf, Germany
Ratajczak M.Z.	Department of Pathology and Medicine, University of Pennsylvania, Philadelphia, USA (long term visit)
Renkawek K.	Institute of Neurology Catholic University of Nijmegen, Netherlands
Weinrauder H.	Centre de Neurochimie CNRS, Strasbourg, France

Department of Neurochemistry

Gordon-Majszak W.	Institute for Basic Research and Developmental Disabilities, New York, USA (long term visit)
Puka M.	Institute of Neurobiology, University of Göteborg, Sweden
Salińska E.	Institute for Basic Research and Developmental Disabilities, New York, USA

Waśkiewicz J.	Department of Neurochemistry and Biochemistry, University of Oklahoma, Norman, USA (long term visit)
Wikieł H.	Roswell Park Cancer Institute, Buffalo, New York, USA (long term visit)
Zalewska T.	Muscle Biology Group, College of Agriculture, University of Arizona, Tucson, Arizona, USA (long term visit)

Department of Neurophysiology

Karczewski W. Czerwosz L.	Department of Medicine Charring Cross Hospital, Medical School, London, United Kingdom
Pokorski M.	Department of Medical Physiology, University of Copenhagen, Denmark
Romaniuk J.	School of Medicine, Case Western Reserve University, Cleveland, Ohio, USA (long term visit)
Szereda-Przestaszewska M.	Department of Physiology, University of Zurich, Switzerland

Department of Applied Physiology

Brzezińska Z.	Faculty of Medicine, University of Göteborg, Sweden
Cybulski G.	Centre for Biological and Medical Systems, London, United Kingdom (long term visit)
Grucza R.	Laboratory of Work Physiology, Medical Faculty Pitié-Salpetriere, CNRS, Paris, France
	Institute of Physiology, Department of Medicine, University of Bochum, Germany
	Max-Planck Institute of Physiology and Medicine, Bad Nauheim, Germany

Grucza R. Kruk B.	Department of Physiology, University of Kuopio, Finland
Langfort J. Kaciuba-Uściłko H. Nazar K.	Department of Pathophysiology, University of Graz, Austria
Kaciuba-Uściłko H.	Institute of Physiology Justus-Liebig University, Giessen, Germany
	Institute of Cardiology and Sports Medicine, Kōln, Germany
Krzemiński K.	Department of Health Sciences, Boston University, USA (long term visit)
Nazar K.	Department of Physiology, Medical Faculty Grange Blanche, CNRS, University of Lyon, France
	Laboratory of Work Physiology Medical Faculty Pitie-Salpetriere, CNRS, Paris, France
Sadowski J.	Institute of Physiology, University of Heidelberg, Germany
Żernicka E.	Department of Pharmacology and Therapeutics, University of Leicester, United Kingdom

Department for Surgical Research and Transplantation

Grzelak I.	Norwegian Radium Hospital, Oslo, Norway
Łukomska B.	Laboratoire de Interactions Cellulaire, University of Bordeaux, France
	Thanjavur Medical College, Thanjavur, India
	Transplantation Center INSA, Bombay, India
	Medizinische Hohschule, Hannover, Germany

Olszewski W.	Department of Surgery, School of Medicine, University of Pittsburgh, USA
	Institute for Surgical Research, University of Munich, Germany
	Medizinische Hohschule, Hannover, Germany
	Thanjavur Medical College, Thanjavur, India
	Transplantation Center INSA, Bombay, India
	Norwegian Radium Hospital, Oslo, Norway

Department of Neurosurgery

Czernicki Z.	Clinic of Neurosurgery, University of Bonn, Germany
Mempel E. Jurkiewicz J.	Neurochirurgische Klinik Kantonsspital, Aarau, Switzerland

Laboratory of Ultrastructure of Nervous System

Gajkowska B.	Cancer Research Center, CNRS, Villejuif, France
Loesch A.	Department of Anatomy and Developmental Biology, University College London, United Kingdom (long term visit)
Walski M.	Link Analytical Limited, High Wycombe, Bucks, United Kingdom

Neuromuscular Unit

Hausmanowa-Petrusewicz I.	Baylor College of Medicine, Texas Medical Center, Houston, Texas, USA
Sieradzan K.	Department of Anatomy and Developmental Biology, University College, London, United Kingdom (long term visit)

Cardiovascular Laboratory

Cedro-Ceremużyńska K.	Wellcome Research Laboratory, Beckenham, United Kingdom
	St. Raphael Medical Foundation, Mediolan, Italy

Mental Health Department

Jancz M.	Department of Psychology, University of Sydney, Australia (long term visit)

PARTICIPATION IN INTERNATIONAL MEETINGS

Conference "Sciences, Technology and Innovation Policies", Vienna, Austria, March 2–6.
W. Karczewski

2nd International Congress on the Immune Consequences of Trauma, Shock and Sepsis, Munich, Germany, March 6–9.
M. Krynicki

Congress LYMPHOLOGICA 91, Hannover, Germany, March 7–10.
P. Bryla

Second Austrian Neuroscience Winter Meeting, Kitzbuhel, Austria, March 21–24.
H. Kroh, G. Szumańska

4th Intersciences World Conference on Inflammation, Antirheumatics, Analgesics, Immunomodulators, Geneva, Switzerland, April 15–18.
J. Strosznajder

Congress International Society of Lymphology, Madrid, Spain, April 26–27.
W. Olszewski

XXVI Congress of the European Society for Surgical Research and VIII. Tripartite Meeting with the Society University Surgeons and the Surgical Research Society Salzburg, Austria, May 8–11.
W. Olszewski, I. Grzelak, M. Durowicz, M. Jasklowska-Englisz, A. Namysłowski

Meeting on Alzheimer's Disease Research, Greenwich, Connecticut, USA, May 17–18.
D. Maślińska

International Congress of Pathophysiology, Moscow, EUN, May 28–June 1.
A. Kapuściński, I. Hausmanowa-Petrusewicz

4th International Symposium on the Biology, Immunology and Surgery of the Greater Omentum, Utrecht, Netherlands May 30–June 1.
U. Kubicka

Symposium: Phospholipids and Signal Transmission, Wiesbaden, Germany, May 29–June 2.
K. Domańska-Janik, M. Samochocki, E. Salińska

2nd European SMA Workshop, Stratford-upon-Avon, Warwickshire, United Kingdom, June 7–9.
I. Hausmanowa-Petrusewicz

11th European Immunology Meeting (also 22nd Meeting of the SSJ), Helsinki, Finland, June 9–12.
I. Grzelak, M. Jaskłowski, A. Namysłowski, D. Sadowska-Szablisty

Eighth International Symposium on Intracranial Pressure "ICP and Craniospinal Dynamics", Rotterdam, Netherlands, June 16–20.
J. Berdyga

9th European Congress of Neurosurgeons, Moscow, EUN, June 23–28.
E. Mempel, Z. Czernicki

Giornate Nazionali di Angiologia 1991, Milano, Italy, June 23–29.
W.L. Olszewski

International Symposium on Arterial Chemoreception, Neurobiology and Cell Physiology of Chemoreception, Chieti, Italy, June 24–28.
M. Pokorski

Regional Meeting of the International Union of Physiological Sciences, Prague, Czecho-Slovakia, June 30–July 5.
M. Szereda-Przestaszewska, H. Kaciuba-Uścilko, K. Nazar, J. Sadowski, G. Cybulski, E. Kompanowska-Jezierska, L. Dobrowolski

Third European Conference on Myasthenia Gravis – EURO-MYASTHENIA III, Oxford, United Kingdom, July 3–5.
M. Strugalska-Cynowska

VIIth International Workshop on Natural Killer Cells, Stockholm, Sweden, July 4–7.
W. Olszewski, B. Łukomska, D. Sadowska-Szablisty

Satellite Symposium Neurobiology of Essential Fatty Acids, Cairns, Australia, July 10–12.
J. Łazarewicz, J. Strosznajder

XIII Congress International Society for Neurochemistry, Sydney, Australia, July 15–19.
J. Łazarewicz, J. Strosznajder

5th Congress of the International Psychogeriatric Association (IPA), Rome, Italy, August 18–23.
E. Kida, M. Barcikowska

12th Meeting of the International Society for Heart Research, Leuven, Belgium, September 9–14.
K. Herbaczyńska-Cedro

Oxford Conference on Control of Breathing and its Modelling Perspective – 5th Meeting, Tokyo, Japan, September 14–19.
M. Pokorski

36 Jahrestagung der Deutschen Gesellschaft für Neuropathologie und Neuroanatomie, Düsseldorf, Germany, September 16–18.
H. Kroh, S. Krajewski

2nd International Symposium on Hypoxia, Berlin, Germany, September 19–21.
J. Łazarewicz, J. Waśkiewicz

First Annual Congress of European Respiratory Society, Brussels, Belgium, September 21–26.
M. Pokorski, M. Głogowska, K. Budzińska

First Hungarian Neuropathological Conference and 6th Hungarian-Polish Neuropathological Symposium, Budapest, Hungary, September 26–28.
M.J. Mossakowski, M. Dąmbska, D. Maślińska, I. Kuchna, H. Weinrauder, E. Kida, M. Barcikowska

XIII International Congress of Lymphology, Paris, France, September 29–October 5.
B. Łukomska, H. Gałkowska, W. L. Olszewski

5th Congress of European Society for Organ Transplantation, Maastricht, Netherlands, October 7–10.
W. Olszewski

3rd International Symposium on Experimental and Clinical Neurobiology, Stara Lesnå, The High Tatras, Czecho-Slovakia, November 25–29.
J. Łazarewicz, J. Strosznajder

Arbeitstagung der "Intrakraniellen Druck, Hirnödem und Hirndurchblutung", Berlin, Germany, November 7–10.
Z. Czernicki

2nd Psychosozialer Krebskongress "Psychosoziale Versorgungsangebote in der Onkologie", Heidelberg, Germany, November 20–23.
Z. Juczyński, J. Szamburska

European Conference on SMA, Baarn, Netherlands, November 22–24.
I. Hausmanowa-Petrusewicz

Second Paneuropean Congress of Neurology, Satellite Symposium "D-5 Spinal Muscular Atrophies", Vienna, Austria, December 7–12.
I. Hausmanowa-Petrusewicz

24th Danube Symposium for Neurological Sciences, Vienna, Austria, December 6–7.
M. Dąmbska, B. Gajkowska, G. Szumańska

2nd Congress of the Paneuropean Society of Neurology, Vienna, Austria, December 7–14.
R. Pluta

SCIENTIFIC DEGREES

DOCTOR'S DEGREES

Joanna Majkowska

Changes in the rat brain after clinical death, pathophysiological and morphological characteristics

(Department of Neuropathology)

SCIENTIFIC MEETINGS ORGANIZED BY THE MEDICAL RESEARCH CENTRE

Practical course of staining with monoclonal antibodies. January 30–31, sponsored by DAKOPATTS, Dennmark

21st course of the basic microsurgical techniques. May 15–17, sponsored by ETHICON

Practical course of staining with monoclonal antibodies. June 11–12, sponsored by DAKOPATTS, Denmark

Symposium of Transplantation Immunity Section of the Polish Immunological Society. June 13, Warsaw

Symposium of Experimental Surgery Section of the Polish Surgeons Society. June 14, Warsaw

Symposium: Antibiotics, anti-viral and anti-fungal drugs in surgery and transplatology. October 30, Warsaw

20th course of the basic microsurgical techniques. November 12–14, sponsored by DAVIS and GECK

LIST OF PUBLICATION

Original works

1. Albrecht J.: Durable inhibition of rat cerebral capillary Na^+/K^+-ATPase after in vitro administration of mercuric chloride. *Toxicol. Lett.* 1991, 59, 133–138.

2. Albrecht J., Norenberg M.D.: Aluminium chloride stimulates NaCl-dependent release of taurine and gamma-aminobutyric acid in rat cortical astrocytes. *Neurochem. Int.* 1991, 18, 125–129.

3. Albrecht J., Simmons M., Dutton G.R., Norenberg M.D.: Aluminum chloride stimulates the release of endogenous glutamate, taurine and adenosine from cultured rat cortical astrocytes. *Neurosci. Lett.* 1991, 127, 105–107.

4. Badurska B., Ryniewicz B., Kowalski J.: Epileptic seizures in myasthenic children. *Neurol. Neurochir. Pol.* 1991, 25, 326–331 (in Polish).

5. Baker M.A., Doris P.A., Turlejska E.: Influence of hydration state on hormonal responses to exercise in dogs. *J. Physiol. Pharmacol.* 1991, 42, 305–316.

6. Budzińska K., Głowicki K., Karczewski W.A., Kulesza J., Ryba M.: The respiratory response to magnetic stimulation of the cortex in the unanaesthetized baboon. *Acta Neurobiol. Exp.* 1991, 51, 125–128.

7. Calabretta B., Sims R.B., Valiteri M., Caracciolo D., Szczylik C., Venturelli D., Ratajczak M., Beran M., Gewirtz A.M.: Normal and leukemic hematopoietic cells manifest differential sensitivity to inhibitory effects of c-myb antisense oligodeoxynucleotides: An in vitro study relevant to bone marrow purging. *Proc. Natl. Acad. Sci. USA* 1991, 88, 2351–2355.

8. Ceremużyński L., Kłoś J., Barcikowski B., Herbaczyńska-Cedro K.: Calcium channel blocker prevents stress-induced activation of renin and aldosterone in conscious pig. *Cardiovasc. Drugs Ther.* 1991, 5, 643–646.

9. Chomicz L., Walski M.: Ultrastructure of oncospheral envelopes of Diorchis elisae (Skrjabin, 1914) Spassky et Frese, 1961 (Cestoda Hymenolepididae). *Parasitology Research* 1991, 77, 550–553.

10. Czernicki Z.: Scientific achievements of Professor Adam Kunicki MD. Neurol. Neurochir. Pol. 1991, 25, 557–558.

11. Czernicki Z.: Cerebral blood flow and microcirculation changes following brain injury. *Neurol. Neurochir. Pol.* 1991, 5, 665–670 (in Polish)

12. Czernicki Z., Grochowski W., Uchman G., Razumowsky A.: Occlusion of superior sagittal sinus caused by meningioma and intracranial volume-pressure relations and brain edema. *Neurol. Neurochir. Pol.* 1991, 5, 580–586 (in Polish).

13. Czernicki J., Jurkiewicz J.: Disturbances of intracranial pressure-volume relations. *Neurol. Neurochir. Pol.* 1991, 5, 671–677 (in Polish).

14. Dąmbska M.: Neuropathological study of the development nervous system. In: *Postępy Neurologii.* Ed.: CMKP, Warszawa, 1991, pp. 74–81 (in Polish).

15. Dąmbska M., Maślińska D., Majdecki T.: Premature infant with Touberous Sclerosis. Morphological and immunohistochemical study. *Neuropatol. Pol.* 1991, 29, 2, 75–82.

16. Drac H.: Congenital hypo- and hypermyelinating neuropathy. *Neuropatol. Pol.* 1991, 29, 133–146.

17. Drac H., Babiuch M., Wiśniewska W.: Morphological changes in peripheral nerves with age. *Neuropatol. Pol.* 1991, 29, 49–67.

18. Durlik M., Łukomska B., Morzycka-Michalik M., Olszewski W.L.: Bone marrow reconstitution in irradiated rats receiving syngeneic hind limb graft. *Eur. Surg. Res.* 1991, 23, S. 1, 30.

19. Durowicz S., Olszewski W.L.: Trapping of lymphocytes in liver sinusoids. *Eur. Surg. Res.* 1991, 23, S. 1, 77.

20. Dziduszko J.: Neurinoma of the cranio-cervical region. *Pol. Tyg. Lek.* 1991, 27–29, 515–516 (in Polish)

21. Dziduszko J., Żarski S., Horsztyński D.: Examination of spinal evocate potentials during stimulation of peripherical nerves in cases of haernia lumbalis. *Reumatologia* 1991, 29, 2–6 (in Polish).

22. Emeryk B., Rowińska K., Michalska T.: Facilitation phenomenon in the electrophysiological diagnosing of myasthenia gravis. *Neurol. Neurochir. Pol.* 1991, 25, 156–162 (in Polish).

23. Faff-Michalak L., Albrecht J.: Aspartate aminotransferase, malate dehydrogenase, and pyruvate carboxylase activities in rat cerebral synaptic and nonsynaptic mitochondria: Effects of in vitro treament with ammonia, hyperammonemia and hepatic encephalopathy. *Metabol. Brain Dis.* 1991, 6, 4, 187–197.

24. Faff-Michalak L., Wysmyk-Cybula U., Albrecht J.: Different responses of rat cerebral mitochondrial 2-oxoglutarate dehydrogenase activity to ammonia and hepatic encephalopathy in synaptic and nonsynaptic mitochondria. *Neurochem. Int.* 1991, 19, 573–579.

25. Fersten E., Szeląg E., Łuczywek E., Szumska J.: Cerebral functional asymmetry in face perception in patients with focal brain damage. *Neurol. Neurochir. Pol.* 1991, 4, 463–468 (in Polish).

26. Fidziańska A.: Skeletal muscle diseases. In: *Basic of Pathology*. Eds. J. Groniowski, S. Kruś. PZWL, Warsaw, 1991, 872–894 (in Polish).

27. Fidziańska A., Badurska B., Jaramowska D., Retka W.: Neonatal form of nemaline myopathy with Intramuscular Nerve Immaturity. *Int. J. Prenatal and Perinatal Studies* 1991, 3, 69–76.

28. Fidziańska A., Glinka Z., Kamińska A., Pope F.M.: Type III collagen deficient EDS IV producing muscular hypotonia with abnormal muscle fibroblasts. *Neuropediatrics* 1991, 22, 228–232.

29. Fidziańska A., Goebel H.H.: Human ontogenesis. 3. Cell death in fetal muscle. *Acta Neuropathol. (Berl.)* 1991, 81, 572–577.

30. Fidziańska A., Kamińska A.: Apoptosis: a basic pathological reaction of injured neonatal muscle. *Pediatr. Pathol.* 1991, 11 3, 421–429.

31. Fidziańska A., Kamińska A., Glinka Z.: Muscle death. Ultrastructural differences between muscle cell necrosis and apoptosis. *Neuropatol. Pol.* 1991, 29, 19–28.

32. Gajkowska B.: The ultrastructural morphology and cytochemistry of the neurosecretory system of the old rat after experimentally evoked clinical death. *J. Hirnforschung* 1991, 32, 4, 213–225.

33. Gajkowska B., Markiewicz D., Kobuszewska-Faryna M.: Ultrastructure of hypothalamo-hypophyseal system of the rat with Morris Hepatoma 7777 after treatment with farmorubicin. *Neuropatol. Pol.* 1991, 29, 3–4, 207–220.

34. Gajkowska B.: Viron A.: Ultrastructural evidence for endogenous vasoactive intestinal peptide-like immunoreactivity in neurohypophysis. *Neuroendocrinol. Lett.* 1991, 13, 5, 387–391.

35. Gordon-Majszak W., Gajkowska B.: Biochemical and morphological changes in the rat brain synaptosomes after exposure to normobaric hyperoxia in vivo. *Exp. Pathol.* 1991, 42, 3, 189–215.

36. Groniowski J., Walski M., Celary-Walska R., Groniowski M.: Ultrastructural alterations of pulmonary surfactant in rat lungs after inhibition of protein synthesis by puromycin. *Folia Histochem. Cytobiol.* 1991, 29, 2, 59–67.

37. Grucza R., Smorawiński J., Cybulski G., Niewiadomski W., Kahn J.F., Kapitaniak B., Monod H.: Cardiovascular response to static handgrip in trained and untrained men. *Eur. J. Appl. Physiol.* 1991, 62, 337–341.

38. Grzelak I., Łukomska B., Sadowska-Szablisty D., Olszewski W.L., Dąbrowski M., Rużyłło W.: Immunophenotypic and functional characterization of pheripheral blood mononuclear cell populations in patients with dilated cardiomyopathy. *Arch. Immunol. Ther. Exp.* 1991, 39, 33–40.

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40. Grzelak I., Olszewski W.L., Rowiński W.: The effect of moderate operative trauma on the blood lymphocyte reactivity-immunomodulatory effect of interleukin-2. *Arch. Immunol. Ther. Exp.* 1991, 39, 133–138.

41. Hausmanowa-Petrusewicz I.: Neuromuscular disease. In: *Textbook of Internal Medicine*. Ed.: T. Orłowski. PZWL, Warsaw, 1991, pp. 554–564 (in Polish).

42. Hausmanowa-Petrusewicz I.: Muscular atrophies. How many types? In: *Amyotrophic lateral sclerosis*. Ed. L.P. Rowland. Raven Press, N.Y., 1991, pp. 157–168.

43. Hausmanowa-Petrusewicz I.: Advances in neuromyology. *Postępy Nauk Medycznych* 1991, 4, 205–209 (in Polish).

44. Hausmanowa-Petrusewicz I., Rowińska K., Kopeć A.: Chronic acquired demyelinating motor neuropathy. *Acta Neurol. Scand.* 1991, 84, 40–45.

45. Herbaczyńska-Cedro K., Lembowicz K., Pytel B.: N^G-monomethyl--L-arginine increases platelet deposition on damaged endothelium in vivo. A scanning electron microscopic study. *Thrombosis Res.* 1991, 64, 1–9.

46. Hilgier W., Benveniste H., Diemer N.H., Albrecht J.: Decreased glusoce utilization in discrete brain regions of rat in thioacetamide-induced hepatic encephalopathy as measured with [^{3}H]-deoxyglucose. *Acta Neurol. Scand.* 1991, 83, 353–355.

47. Hilgier W., Haugvicova R., Albrecht J.: Decreased potassium-stimulated release [^{3}H] D-aspartate from hippocampal slices distinguishes encephalopathy related to acute liver failure from that induced by simple hyperammonemia. *Brain Res.* 1991, 567, 165–168.

48. Iglesias J.R., Kroh H., Matyja E., Aruffo C.: Experimental CNS tumors in rats. Testing field for a computerized classification and tumor grading. *Exp. Pathol.* 1991, 43, 25–31.

49. Jakubowska T., Iwańska B., Sobczyk W., Łuczywek E.: Early symptoms of cortical defects children which subacute sclerosing panencephalitis. *Approche Neuropsychologique de apprentissages chez l'enfant.* 1991, 3, 35–38.

50. Jaskłowska-Englisz M., Olszewski W.L.: Donor specific transfusions prolong organ allograft survival but accelerate elimination of i.v. transplanted allogeneic lymphocytes. *Eur. Surg. Res.* 1991, 23, S. 1, 37.

51. Jaskłowska-Englisz M., Olszewski W.L.: Allogeneic lymphocytes transplanted i.v. failed to home to lymph nodes and spleen of recipients treated with donor specific transfusion. *Patologia Polska* 1991, 42, 91.

52. Jurkiewicz J., Czernicki Z., Pawłowski G.: Analysis of the correlations of volume-pressure relations and visual evoked potentials during disturbances of intracranial volume compensation. *Neurol. Neurochir. Pol.* 1991, 5, 567–573 (in Polish).

53. Jurkiewicz J., Czernicki Z., Pawłowski G.: Changes of intracranial volume-pressure relations and visual evoked potentials produced by stabilized and compensated additional volume. *Neurol. Neurochir. Pol.* 1991, 5, 574–579 (in Polish).

54. Jurkiewicz J., Costabile G., Czernicki Z., Hess K., Probst Ch.: The usefulnes of somatosensory evoked potentials in the diagnosis of low-pressure hydrocephalus. *Neurol. Neurochir. Pol.* 1991, 5, 559–566 (in Polish).

55. Kaciuba-Uściłko H., Grucza R.: Thermoregulation. In: *Applied and Clinical Physiology for Medical Students*. Ed. E. Szczepańska-Sadowska. Warsaw Medical School, 1991, pp. 257–284 (in Polish).

56. Kaciuba-Uściłko H., Kruk B., Szczypaczewska M., Opaszowski B., Stupnicka E., Bicz B., Nazar K.: Metabolic, body temperature and hormonal responses to repeated periods of prolonged cycle-ergometer exercise in men. *Eur. J. Appl. Physiol.* 1991, 64, 26–31.

57. Kapuściński A.: Changes of concentration of cyclic AMP in rat brain and plasma in the clinical death model. *Neuropatol. Pol.* 1991, 29, 95–101.

58. Krajewski S., Wechsler W.: Development of subrenal capsule tumors following transplantation of RN6 multicell spheroids. *J. Neuro-Oncology* 1991, 10, 219–231.

59. Kroh H.: Ganglion cells and their satellites in experimental trigeminal schwannoma in rat. *Neuropatol. Pol.* 1991, 29, 109–117.

60. Kroh H., Cervós-Navarro J.: Alpha-l-antichymotrypsin in human glioblastoma multiforme cells and its relation to GFAP staining. *Clinical Neuropathol.* 1991, 10, 181–186.

61. Kroh H., Ruzikowski E.: Multiple schwannomas of cauda equina in the course of peripheral schwannomas. Case report. *Neuropatol. Pol.* 1991, 29, 201–205.

62. Kruk B., Cherdrungsi P., Pekkarinen H., Hänninen O.: Effect of glucose ingestion on thermoregulatory responses to intermittent exercise in man. *Biology of Sport* 1991, 8, 177–186.

63. Kruk B., Pekkarinen H., Manninen K., Hänninen O.: Comparison in men of physiological responses to exercise of increasing intensity at low and moderate ambient temperatures. *Eur. J. Appl. Physiol.* 1991, 62, 353–357.

64. Krynicki M.: The influence of neuropaptides of central and peripheral nerve system on activity of immune system. *Immunologia Polska* 1991, 16, 213–228.

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81. Namysłowski A., Olszewski W.L.: Methods of "natural" prolongation on organ allografts survival. *Immunologia Polska* 1991, 16, 229–238.

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88. Olszewski W.L.: Lymph pressure and flow in limbs. In: *Lymph stasis: Pathophysiology, diagnosis and treatment*. Ed. CRC Press, Boca Raton, 1991, pp. 109–155.

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91. Olszewski W.L.: Injury of lymphatics. In: *Lymph stasis: Pathophysiology, diagnosis and treatment*. Ed. CRC Press, Boca Raton, 1991, pp. 287–292.

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