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RESEARCH REPORT

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NEUROCHEMICAL INTERACTIONS IN THE RESPIRATORY SYSTEM; EVALUATION OF LARYNGEAL REFLEXES IN RESPONSE TO TYPE C FIBER ACTIVATION

Assoc. Prof. Mieczysław Pokorski

The carotid body is a chemosensory organ that is activated by chemical stimuli and reflexly stimulates ventilation. This is usually accompanied by decreased high energy compounds in the chemoreceptor cell. We studied the modulatory action of APT and the neurotransmitters - dopamine and acetylcholine on the chemotransduction process that is linked to the turnover of phospholipids in the carotid body. We compared the effects of exogenous ATP on the activity of phospholipase C degrading phosphatidylinositol-4,5-bisphosphate in the carotid body homogenate. The carotid bodies were preexposed *in vivo* to normoxia and hypoxia. We have found that ATP-stimulates phospholipase C activity in both normoxic and hypoxic carotid bodies. The stimulation in the latter was 3-fold greater, which points to the role of ATP in the hypoxic signal transduction. No such effect was found for the neurotransmitters studied.

The upper airway obturation could be associated with disproportional changes in the motor activities of the phrenic and hypoglossal nerve induced by the large inspiration. In this study a hypothesis according to which nitroglycerine lowers this disproportion has been checked. The study was carried out on the anesthetized, paralyzed and ventilated rabbits. The results showed that:

1. Nitroglycerine increases the hypoglossal nerve activity.
2. Long time hyperventilation does not reduce hypoglossal activity after nitroglycerine.
3. Nitroglycerine abolishes the alcohol action in response to deep and fast breathing.
4. Nitroglycerine reduces time of apneic phase.
5. Nitroglycerine decreases disproportion between hypoglossal and phrenic activities in response to large inspiration.

Intravenous injection of capsaicin (to stimulate pulmonary C-fibres) caused an expiratory apnoea and complete closure of the larynx in cats. In the initial phase of resumed breathing, the post-capsaicin constriction of the larynx was released. With tachypnoea the decrease in tidal volume, coupled with increased respiratory rate, failed to effect minute-ventilation. Bilateral section of the superior laryngeal nerve neither prevented laryngeal constriction in apnoea nor the ventilatory effect of capsaicin.

A hypothesis that gammaaminobutiric acid (GABA) is involved in respiratory depressive response to hypoxia was verified in the guinea pig. The 7% hypoxia was introduced before and after Picrotoxin - GABA antagonist - injection. The time of the occurrence of apnoea was used as an index of method effectiveness. Before Picrotoxin administration it was delayed 3- and more fold. Eventually the breathing depression could be restored by stimulation if the dose of Picrotoxin was adequate. In conclusion: GABAergic system is involved in the creation of post-hypoxic depression of breathing.

STUDIES ON MECHANISMS REGULATING PATTERNS OF THE PHRENIC NERVE ACTIVITY OF AND ACTIVITY NERVES TO ACCESSORY RESPIRATORY MUSCLES; RESPIRATORY-CIRCULATORY CORRELATIONS

Dr. Krystyna Budzińska

In studies on the plasticity mechanism in the central nervous system regulating respiration, it was assumed that nitric oxide is a modulator of transient potentiation of hypoglossal activity which arises after the termination of stimulation of the superior laryngeal nerve. It was shown that transient potentiation after the stimulus is inhibited by NO synthesis substrate, L-Arginine and enhanced by L-NNA, a NO synthase inhibitor. This mechanism is also activated by β-adrenergic agonist, isoprenaline.

We have found that a rapid rise in arterial pressure suppresses activity of the hypoglossal nerve and increases resistance of the upper airway to airflow. We postulate that this mechanism plays a part in prolongation of the episodes of obturation in the sleep apnoea syndrome.

In the experiments performed on rabbits, integrated phrenic nerve activity was recorded in control conditions and following pharmacological blockade of *Nucleus Parabrachialis Medialis* (NPBM). NPBM decreased the respiratory rate due to prolongation of both phases of the respiratory cycle. The results show that NPBM does not form the anatomical structure of the pneumotaxic centre.

SAFETY AND EFFICIACY OF CLADRIBINE (2-CdA) IN MULTIPLE SCLEROSIS

Assoc. Prof. Paweł Grieb

A 2-years long, randomized, double-blind, placebo controlled study of cladribine (2-chloro-2'-deoxyadenosine, 2-CdA) in 85 patients suffering from remitting-relapsing multiple sclerosis has been continued. The dosing of the drug was 5 mg s.c. daily for five consecutive days, courses repeated monthly during the first six months, followed by two additional courses in 3 month intervals. Analysis of the hematological data showed that the treatment is safe and well tolerated. The reduction of lymphocyte counts to 1/3 of the initial values on the average was achieved, while thrombocytopenia was slight and insignificant, and granulocyte count remained normal. The data concerning neurological status of patients are being analysed and will be reported in the future.

BIODEGRADABLE POLYMERS FOR INTRACEREBRAL DRUG DELIVERY

Assoc. Prof. Paweł Grieb

Using transmission and scanning electron microscopy it was shown that a biodegradable block copolymer of lactide and caprolactone (L-Cap) is not toxic toward brain tissue of rats. When implanted into the rat brain, polymer microspheres or strips produced the local tissue response which was not appreciably different from that evoked by the local tissue damage alone. Therefore L-Cap polymers deserve development as vehicles for intracerebral delivery of drugs.

CYTOPROTECTION IN THE CENTRAL NERVOUS SYSTEM

Assoc. Prof. Paweł Grieb

The evidence of free radical-related membrane damage in the brain was found in the rat model of acute circulatory arrest and resuscitation. Idebenon, an antioxidant penetrating blood-brain barrier, significantly alleviated the damage indices, limiting the rise of conjugated dienes and the decreases of the SH groups.

ANTIVIRAL ACTIVITY OF 2-CdA

Assoc. Prof. Paweł Grieb

It was found that 2-CdA (cladribine, 2-chloro-2'-deoxyadenosine) displays a slight (compared with the standard antiviral drug aciclovir) activity against HSV (*Herpes simplex*) virus. The effect is of no clinical value.

OBSTRUCTIVE SLEEP APNOEA AS A CONSEQUENCE OF POSITIVE FEEDBACK BETWEEN SKELETAL MUSCLE TONE AND PHASIC ACTIVITY OF THE NERVES SUPPLYING UPPER AIRWAY MUSCLES

Dr. Wiktor Janczewski
Supported by the State Committee for Scientific Research: grant # 6 P207 028 05

The loss of skeletal muscle tone reduces the magnitude of the reflex response to negative pressure within the upper airway and selectively decreases phasic respiratory activity of the upper airway muscles.

ARACHIDONIC ACID IN CELLULAR PROCESSES OF SIGNAL TRANSDUCTION IN THE CAROTID BODIES

Dr. Robert Strosznajder
Supported by the State Committee for Scientific Research: grant # 6 P207 029 05

In this study the regulation of arachidonic acid (AA) release and its incorporation into membrane glycerolipids of cat carotid bodies (CBs) were investigated. Moreover, the effect of hypoxia and dopamine on these processes was evaluated. It was observed that Ca^{2+} in concentration dependent manner enhanced the release of AA from [^{14}C-arachidonyl]phosphatydylinositol (PtdIns) by about 40-50% through the action of phospholipase C together with diacylglicerol lipase, however, small pool of AA was also liberated by phospholipase A_2. Hypoxia activated AA release, but this effect was statistically insignificant. The study on [^{14}C] AA incorporation into CBs glycerolipids indicated that AA was actively incorporated into PtdIns, then into phosphatidylcholine and subsequently into phosphatidylethanolamine, phosphatidylserine, phosphatidic acid and into polyphosphoinositides. Acute

hypoxia significantly decreases AA incorporation specifically into PtdIns and concomitantly enhances the level of AA radioactivity in diacylglicerol (DAG). The ratio of PtdIns/DAG distinctly decreases in hypoxic CBs. In spite of that hypoxia enhances significantly the level of AA-CoA radioactivity. These results suggest that hypoxia induces inhibition of AA incorporation occurring on the level of acyl-CoA: lysophospholipid acyltransferases. Dopamine may play modulatory role in AA metabolism in normoxic and hypoxic CBs and is not responsible for the hypoxia induced alteration of lipid metabolism in CBs.

European Community Concerted Action:

CANCER AND BRAIN DISEASE CHARACTERIZATION AND THERAPY ASSESSMENT BY QUANTITATIVE MAGNETIC RESONANCE SPECTROSCOPY

Assoc. Prof. Paweł Grieb
Biomed 1 - PL 920432

The project, conducted in cooperation with the Laboratory of Magnetic Resonance and the Neurosurgical Ward, Warsaw-Brodno Hospital, concerns the utilization of magnetic resonance spectroscopy of phosphorous compounds for monitoring the response of human gliomas *in situ* to experimental chemiotherapy with 2-chloro-2'-deoxyadenosine. During the year 1995 the system for spectroscopic data collection based on a Siemens SP-63 (1.5T) MR imager has been implemented, and several control phosphorous spectra have been collected from brains of normal individuals. The accuracy and reproducibility of spectroscopic data is being analysed.

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PARTICIPATION OF INTRA- AND EXTRACELLULAR CALCIUM POOLS IN NMDA NEUROTOXICITY

Prof. Jerzy W. Łazarewicz

The mechanism of NMDA-induced ^{45}Ca release was studied in the hippocampus *in vivo*. Microdialysis of the dentate gyrus demonstrated participation in this effect of the NMDA receptors and Na^{+}/Ca^{2+} exchange, and its inhibition by dantrolene and modulation by ryanodine, which points to the role of Ca^{2+}-induced intracellular Ca^{2+} release. The strongest expression of ^{45}Ca release was in the dentate gyrus, it was moderate in CA1, whereas in the subiculum and in the rabbit hippocampus NMDA induced a decrease in ^{45}Ca release from cells. Comparative analysis was performed of this effect and of a tissue content of its putative participant calbindin D_{28k} (CALB) protein, detected by immunoblotting. The results suggest complex functional relations between NMDA receptors, intracellular Ca^{2+} pools, Ry-R and CALB, and their diversity in various brain regions and species.

MOLECULAR MECHANISMS OF SIGNAL TRANSDUCTION IN AGED BRAIN, ALZHEIMER'S DISEASE AND ISCHEMIA IN CENTRAL NERVOUS SYSTEM

Prof. Joanna Strosznajder

Molecular mechanism of signal transduction in cholinergic and serotonergic neurotransmitter system in aged brain was investigated. Receptor-dependent release of inositol phosphates and mobilization of cytosolic calcium was determined. This study was performed on synaptoneurosomal fraction isolated from cerebral cortex of adult (4-months-old) and aged (27-months-old) rats. Synaptoneurosomal membrane lipids were labelled with 2-[^{3}H]-myo-inositol. It was observed that in adult brain, a non-hydrolysable analog of acetylcholine, carbachol, as well as

serotonin added together with pargyline (each case in the presence of lithium chloride) stimulated significantly inositol phosphate release. This process was more active in aged brain, which mean higher level of radioactivity of liberated inositol phosphates. In the following experiments concerning these receptor activation, the intracellular calcium [Ca_i], using fluorescent indicator Fura-2, was estimated. It was found that activation of cholinergic receptor in adult brain elevated [Ca_i] during 5 min of incubation by approx. 30% above its resting level (180 nM). This receptor-dependent increase of [Ca_i] was completely abolished by TMB-8, the antagonist of inositol-1,4,5-trisphosphate (IP_3) receptor, located in the membranes of endoplasmatic reticulum. However, in aged brains, in spite of enhanced inositol phosphate liberation, resulting from activation of muscarinic receptor-dependent phcspholipase C no changes in [Ca_i] were observed. The results obtained in these studies indicate cholinergic transmission distrurbances in aged brain, involving phospholipase C and inositol phosphate changes. The IP_3 receptor modification in aged brain may be responsible for a lack of this second messenger-dependent calcium signal. The other possible factor is the higher activity of IP_3 degradation in aged brain, as compared to the adult one. In studies concerning the relationship between cholinergic and serotonergic neurotransmitter system,s it was found that the activation of $5\text{-}HT_{1A}$ receptor significantly lowers the muscarinic receptor-dependent calcium signal. This event could be explained by a marked reduction of cholinergic-dependent inositol phosphate release in the cases of enhanced concentration of serotonin.

Our studies on pathological aging and Alzheimer's disease (AD) especially, concentrated on the role of long, hydrophobic amyloid β (Aβ) peptides in the fibrillogenesis and formation of senile plaques. Additionally, the role of amyloid associated proteins in the formation of amyloid fibrils was investigated. Our results suggested that long Aβ peptides, such as Aβ42 can be important in the formation of diffuse (preamyloid) amyloid deposits, but were not sufficient in themselves to lead to Aβ fibrillogenesis and neuritic plaque formation. Our studies, in collaboration with NYU Medical Center in New York, on canine aged model of β-amyloidosis suggested that amyloid peptides forming the diffuse deposits despite their amino acid length and hydrophobicity can not be the only factor responsible for the formation of mature senile plaques. In other experiments the role of apolipoprotein E (apo E) in the formation of senile plaques, in AD was investigated. For this purpose biochemically isolated amyloid β from senile plaques and found that a carboxyl-terminal fragment (residues 216-299) of apo E co-purified. *In vitro* studies showed that similar fragment from recombinant apo E, generated by thrombin cleavage, could form amyloid-like fibrils, which were Congo Red positive. Carboxyl-terminal of apo E, as well as intact apo E have a potential to "seed" amyloid b growth *in vitro*. Amyloid fibril formation *in vitro* can be initiated by the addition of a "seed"

or template. Our results suggested that some Aβ peptides and C-terminal of apo E can act as a "seed" and template in AD. A process mentioned above, responsible for the formation of senile plaques, may be the basis of pathomechanism of AD. The answer to the question of which Aβ peptides and amyloid associated proteins are involved in the process of fibrillogenesis and its further neurotoxicity, will allow us to investigate the action of selected Aβ peptides in particular steps of signal transduction in central nervous system.

In the present year we continued our studies on the properties of Cl^- channels, including $GABA_A$ receptor-dependent ones, 2 and 4 months after 5 min transient cerebral ischemia. It was found that shortened opening time of Cl^- channels, resulting from ischemic insult, was sustained 2 months after and disappeared 4 months after ischemic insult.

Additionally, in experiments concerning ADP-ribosylation of proteins after transient cerebral ischemia we observed an inhibition of the activity of this process and during reperfusion time. The further experiments will explain this unexpected inhibition. Besides, we investigated the action of nitric oxide synthase inhibitors on the enhanced level of putrescine (one of the poliamines) in ischemia-reperfusion injury. The level of other poliamines was not changed. Obtained results showed a lack of correlation between NO/cGMP and putrescine production following transient brain ischemia and reperfusion.

SIGNAL TRANSDUCTION AND GENE EXPRESSION IN BRAIN PATHOLOGY

Assoc. Prof. Krystyna Domańska-Janik

Developmental stages of oligodendrocyte differentiation with the concomitant changes of plp gene expression in pt-plp mutant rabbits were investigated. The mutation was characterised as a new animal model of Pelizaeus-Marzbacher disease.

An extensive, short-lasting activation of Ca^{2+}-independent form of calcium/calmodulin dependent kinase II (CaMKII) precedes its inhibition and translocation, in accordance with a scheme of the enhanced sequential enzyme autophosphorylation during brain ischemia.

The origin of majority of glutamate release during *in vitro* ischemia was identified as derived from the metabolic compartments. Suggested mechanisms involve a reversal of the amino-acid transporter and an opening of anionic channels localised mainly in neuroglia to the large ions such as glutamate.

METABOLIC CHANGES IN SUBCELLULAR STRUCTURES OF BRAIN CAUSED BY LEAD TOXICITY

Prof. Urszula Rafałowska

The aim of this work was to determine whether prolonged drinking of lead-acetate containing water, which imitates environmental exposure to lead (Pb) affects permeability of blood-brain barrier (BBB) in adult rats and to search for other molecular mechanisms which can be connected with Pb-effects on function of synaptosomes.

We have indicated that lead induces BBB disfunction: enhanced pinocytotic activity of the endothelial cells and opening of interendothelial tight junctions, together with great activation of pericytes. A significant increase of lead level in capillaries and synaptosomes obtained from the brains of rats under chronic toxicity conditions was noted. It was also checked whether Pb by penetrating structures of the brain disturbs some molecular mechanisms responsible for functioning of CNS. It was found that $GABA_B$ affinity (KD) and density of receptor (B_{max}) as well as the level of protein -SH groups in synaptosomes were changed. It was evidenced that Pb-toxicosis decreases concentration of ATP in synaptosomes but simultaneously increases concentration of phosphocreatine and creatine kinase activity.

The findings sugesst existance of the regulatory system maintaining stability of the metabolic processes in synaptosomes in chronic Pb toxic conditions.

NMDA RECEPTOR MODULATION OF EICOSANOID PRODUCTION IN THE HIPPOCAMPUS *IN VIVO*

Prof. Jerzy W. Łazarewicz

Supported by the State Committee for Scientific Research: grant # 4 P05A 042 08

Studies on NMDA receptor-mediated modulation of the eicosanoid production in brain, initiated in the middle of 1995, encompassed pharmacological characteristics of the NMDA-evoked release of prostaglandin D_2 in the rabbit hippocampus *in vivo*, estimated by microdialysis combined with the radioimmunoassay. The recent study concerns relations between prostaglandin D_2 release and a decrease in extracellular Ca^{2+} concentration triggered by NMDA at different concentrations of the agonist, using competitive and uncompetitive NMDA receptor antagonists, a sodium channel blocker and NO synthase antagonist. These experiments are in progress.

ENERGETIC METABOLISM IN BRAINS NERVE ENDINGS UNDER LEAD TOXICITY CONDITIONS

Prof. Urszula Rafałowska

Supported by the State Committee for Scientific Research: grant # 6 P207 093 04

The aim of the performed study was to find out whether prolonged consumption of leaded water, that imitates environmental exposure, affects some energy parameters in nerve endings of adult rat brains. Our results indicate that Pb-toxicosis changes the permeability of blood-brain barrier and causes accumulation of lead inside nerve endings, however the level of Pb in brain or in synaptosomes was found to be much lower than in liver or bones. This influx of Pb to brain did not affect oxygen consumption by synaptic mitochondria and did not change the activity of cytochrom oxidase in synaptic and pericaryonic mitochondria as compared to the control samples. These results indicate that the oxidation chain in synaptic mitochondria under Pb toxicity conditions remains intact. Also, the intracellular water of synaptosomes did not change under chronic Pb toxicity conditions. Compared to the control sample, the concentration of ATP decreased and that of creatine-phosphate (CrP) increased drastically in fractions obtained from Pb^{2-} intoxicated animals with simultaneously increased activity of creatine kinase (CK). It seems likely, that in chronic Pb^{2-} toxicity, the PCr/Cr/CK system constitutes a satisfactory regulatory mechanism of energy processes in nerve endings of the adult rats. However, the possibility remains that the effects may be different in acute Pb^{2-} poisoning and/or in young animals with immature blood-brain barrier.

THE ROLE OF NITRIC OXIDE IN PATHOMECHANISM OF ISCHEMIC BRAIN INJURY

Prof. Joanna Strosznajder

Supported by the State Committee for Scientific Research: grant # 6 P207 027 04

In 1995 several supplementory studies were carried out, on the biphasic activation of nitric oxide synthase (NOS) and formation of cGMP during reperfusion after short 5 min ischemia in gerbils.

Moreover, further experiments on NMDA receptor dependent NO/cGMP liberation in different part of brain were continued.

Submissive studies were concentrated on elucidation of the action of specific inhibitor of neuronal form of NOS 7-Nitroindazole (7-NI) on NOS activity and

cGMP level, and also on the level of putrescine enhanced by ischemia. It was found that 7NI administrated in a dose of 25 mg/kg b.w. 5 min before ischemia protected the brain against accumulation of cGMP and enhancement of NOS activity during ischemia and reperfusion time. This inhibitor had no effect on ischemia-induced putrescine accumulation. Additionally, it was found that short term 5 min ischemia enhances also the level of putrescine during 15 min and 12 h reperfusion period.

The level of the other poliamines such as spermine or spermidine was not changed by ichemia reperfusion. These results suggest the NO/cGMP are not involved in activation of polyamine biosynthesis which occurs on the level of ornithine decarboxylase.

The i.p. administration of guanylate cyclase inhibitor LY-83583 in a dose of 6 mg/kg b.w. 5 min before ischemia protects not only against cGMP accumulation but also against Ca^{2+}-dependent activation of NOS. Our results suggest that above inhibitors ought to be taken into consideration for the treatment of ischemic pathology.

In our further studies the process of NO-dependent ADP rybosylation of protein was studied. The preliminary results indicate the lower activity of rybosylation reaction during ischemia and reperfusion. This study will be continued for better understanding of the effect of ischemia on this process.

PHENOTYPIC DIVERSITY AND MYELIN GENES EXPRESSION IN pt MUTANT

M.Sc. Joanna Sypecka

Supported by the State Committee for Scientific Research: grant # 6 P207 038 04

The aim of study was to establish whether there is any correlation between expression of myelin specific protein genes and observed phenotypic diversity based on severity of neurological symptoms in pt mutant rabbit. The molecular basis of pt disease is a point mutation in exon of plp gene that causes substitution of His^{36} by Glu in PLP molecule. The mutation leads to formation of aberrant myelin and its significant deficiency in CNS. It has been proved that the degree of hypomyelination correlates well with phenotypic diversity and is the highest in the most severely affected animals. Molecular studies indicate that the downregulation of the plp gene expression increases during development in phenotype-dependent manner, i.e. the most significant deficiency is observed in the most severe cases of the disease. It seems that the mutated gene expression and alterations in transport of the formed molecules influence oligodendrocyte functions and lead to secondary inhibition of plp gene itself.

EFFECT OF ISCHEMIA AND EXCITATORY AMINO ACID RECEPTORS ON EXPRESSION AND METABOLISM OF BETA AMYLOID PRECURSOR PROTEIN (b-APP) IN BRAIN (INTERRELATIONSHIP)

Dr. Wanda Gordon-Krajcer
Supported by the State Committee for Scientific Research: grant # 4 P05A 059 08

Effects of rat brain ischemia on expression and metabolism of beta amyloid protein and its precursor (β-APP) was studied considering a role of excitatory amino acids. A Pulsinelli's model of 10-min forebrain ischemia was used, with recovery for up to 7 days. Homogenate proteins of the cortex and different hippocampal regions were electrophoresed and electroblotted. The immunoreactions were made with antibodies to peptide residues homologous to various domains of the β-APP. The results indicate that immunoreactivities with antibodies to all β-APP domains studied increase considerably, especially 7 days after the ischemic insult. Injection of MK-801 for 30 min before ischemia reduces this effect. These results suggest that NMDA receptors participate in the mechanism of changes in the expression and metabolism of β-APP in the brain, and that the signal for these changes is generated rather during than after ischemia.

INTERACTIONS BETWEEN PROTEIN KINASE C AND CALPAINS IN BRAIN ISCHEMIA

Dr. Teresa Zalewska
Supported by the State Committee for Scientific Research: grant # 4 P05A 026 08

Catalytic activity and subcellular localisation of the two isoforms of calpains ("μ" and "m") were investigated in global cerebral ischemia and during development of hypoxic-ischemic encephalopathy in rats. In the control brain only about 10% of total proteolytic, calcium-dependent activity was connected with μ-calpain. Approx. 15% of "μ" and 25% of "m" form activities were localised in plasmatic membranes. It was shown that μ-calpain, a form being activated by μM calcium concentration, is extensively translocated into plasmatic membranes immediately after ischemic insult. Concomitantly, the total enzyme activity is inhibited. These ischemic changes are followed by the time-dependent proteolytic cleavage of PKC and generation of the spectrin- (a specific, endogenous substrate for calpains) - breakdown products in the ischemic brains.

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TRANSPORT OF ENDOGENOUS NEUROTRANSMITTERS AND THEIR PRECURSORS IN BRAIN IN HYPERAMMONEMIA AND INORGANIC MERCURY INTOXICATION

Prof. Jan Albrecht

Hepatic encephalopathy (HE) in the thioacetamide model was shown to stimulate ornithine transport, to inhibit arginine transport, and not to affect lysine transport across the blood-brain barrier (BBB). The results point to the functional heterogeneity of the $\gamma+$ transport system, so far considered to be homogenous and unable to discriminate between the individual basic amino acids. HE treatment with ornithine aspartate differently affected the transport of individual amino acids, which also points to the heterogeneity of the system. Subtle changes in the BBB functioning elicited by HE in the thioacetamide model were reflected in alterations in lectin binding to the endothelial cell membranes of the cerebral capillaries.

Ammonia added *in vitro* in a neurotoxic dose encoutered in acute hyperammonemia *in vivo*, was found to stimulate glutamine (GLN) uptake to the rat cerebral nonsynaptic mitochondria. This phenomenon may contribute to mitochondrial swelling, and subsequently to disturbances of cerebral energy metabolism accompanying hyperammonemia. The result is consistent with a recent hypothesis that GLN mediates ammonia neurotoxicity.

The role of glutamic acid (GLU) in inorganic mercury neurotoxicity was confirmed by showing that, in an organotypic culture of the rat cerebellum, correlated toxic effects of both compounds are attenuated by a noncompetitive NMDA receptor antagonist, dizocilpine.

FURTHER STUDIES ON VASCULAR AND METABOLIC MECHANISMS OF POSTISCHEMIC ENCEPHALOPATHY

Prof. Mirosław J. Mossakowski

Immune cytochemical analysis of endotheline localization in the cellular elements of the blood-tissue interphase in CA1 sector of Ammon's horn of rats, submitted to 10 min global cerebral ischemia was performed. Remarkable increase of its immunoreactivity, with maximum at 24th h following cerebral ischemia was found. It was present in all cellular elements of blood-tissue interphase in particular in microglia and macrophages neighbouring the blood vessels. The results obtained demonstrating postischemic increased content of endotheline may indicate its role in postischemic disturbances of blood supply and thus participation in the development of postresuscitation encephalopathy.

Immune cytochemical analysis of Bax and BcL-2 proteins in the central nervous system of rats submitted to global cerebral ischemia was also performed. Increased content of Bax protein (involved actively in cellular death) and significant decrease of BcL-2 protein (complexes of which are counteracting development of apoptosis) were detected in some neurons of CA1 hypocampal sector and in cerebellar Purkinje cells in 3 h after cardiac arrest lasting 10 min. Bax immunoreactivity was remarkably higher in neurons with features of degeneration. In time intervals longer than 3 h after resuscitation those neurons revealed decreased immunoreactivity of BcL-2, BcL-x in particular. The results presented indicate that differences in expression of some genes from BcL-2 family may play an important role in defining relative sensitivity of particular neuronal population to ischemia and that postischemic changes in the expression of all these proteins studied may participate in the mechanism of the delayed neuronal death.

In the same experimental model of the brain ischemia widespread extracellular deposition of immunocytochemically detectable apolipoproteins E and J in various regions of the central nervous system was found. Most of the deposits were perivascularly located. Apolipoprotein A-1 was not connected with cellular elements. In general extracellular deposits of Apo E, J and A-1 were identical in localization as those of β-amyloid protein precursor described in previous studies. Taking into consideration the probable role of Apo-E in the mechanism of amyloid deposition and fibrilization in the central nervous system a hypothesis concerning some similarities in the pathomechanism of postischemic encephalopathy with those involved in Alzheimer's disease was presented. These observations in conjunction with previous ones concerning deposition of β-amyloid protein precursor and β-amyloid itself resulting from brain ischemia may indicate some pathogenic connections between degenerating process in brain amyloidosis with consequences of temporary severe disturbances in blood brain supply.

In the same model of experimental cerebral ischemia spastic changes of particular segments of the cerebral microcirculation were found and characterized as far as their dynamics, mechanisms and connections with blood-brain barrier disturbances were concerned.

Quantitative isotope methods revealed significant transvascular penetration of methionine appearing two months after ischemic incident. The phenomenon may be indicating of either periodically increased blood-brain barrier permeability or an enhanced active transport of the amino acid, connected with postischemic reparatory processes appearing in the blood-tissue interphase. The late appearance of the above described processes, indicating an active and progressing nature of postischemic changes deserves special attention.

The same was suggested by lectine histochemistry of glucoconjugate receptors in vascular endothelia, neurons and glial cells as well as in neuropile studied during late postischemic survival reaching 12 months after ischemic incident.

In addition to the above studies disturbances in the cerebral blood flow following 10-min experimental cardiac arrest was found. This has been indicated by disappearance of blood flow reaction to the acetylcholine infussion, with its normal response to the activity of NO inhibitor.

The studies on the ischemia-induced blood-brain barrier disturbances, analysis of endothelial intercellular adhesion molecules (ICAM-1) behaviour in the blood vessels of some intracranial brain tumors was performed. Dependence of the observed changes on the nature of neoplastic process and type of vessels were very striking. It has been concluded, that changes in the behaviour of ICAM-1 and perhaps of other adhesion molecules may be a marker of the transport functions in the conditions of injured blood-brain barrier.

In newly introduced compression model of the brain ischemia dynamics of morphological changes, their extent and their intensity as well as degree of blood-brain barrier injury were characterized.

DYNAMICS OF DEMENTIVE PROCESSES IN ALZHEIMER'S DISEASE AS CORRELATED WITH FOCAL BRAIN ATROPHY AND MORPHOLOGICAL AND IMMUNOHISTOCHEMICAL CHARACTERISTICS OF SENILE AND DEGENERATIVE CHANGES IN THE CENTRAL NERVOUS SYSTEM

Assoc. Prof. Irmina B. Zelman

In patients with clinically diagnosed Alzheimer's disease interdependence between appearance and intensity of dementia on one side and disease duration on

the other side was established. Speach disturbances were also correlated with the intensity of generalized atrophy of the neocortex and hippocampus. The obtained results are indicating that general cortical lesions of the neocortex but not selective abnormalities in speach centres are responsible for speach disorders in the Alzheimer's disease.

In the brains of patients with Alzheimer's disease lack of correlation between cortical accumulation of senile plaques and intensity of structural lesions of the frontal lobe white matter was found. In this context it seems probable that injury of the white matter in Alzheimer's disease is not connected directly with primary brain degeneration leading to dementia, but it can play role in its intensification.

In the group of patients who died due to the brain insult in 31% of cases congophilic angiopathy and senile plaques spread in the neuropil were found. Congophilic angiopathy was significantly more frequent in patients with arterial hypertension and coronary disease. No correlation was found between congophilic angiopathy and nature of vasogenic brain damage (infarction versus intracerebral hemorrhage).

Ultrastructural studies of myelin in the course of aging process in pt mutant rabbits are indicating of lack of the primary myelin changes characteristic for senile process. They are showing that progressing with age myelin abnormalities are resulting from severe damage of myelin-oligodendroglia complex charakteristic for this mutation and progressing with age.

It has been shown that squalenic neuropathy in rats (continuation of previous studies) results of primary structural damage of myelin sheath but not Schwann cells of the peripheral nerves.

STRUCTURAL CHARACTERISTICS OF HIV-INFECTION OF THE CENTRAL NERVOUS SYSTEM

Prof. Mirosław J. Mossakowski

The main task of the study was further collection of neuropathological material of AIDS cases in adults and their characteristics as far as HIV-specific processes, opportunistic infections and neoplastic growths were concerned.

Material collected from 1987 increased by 25 new cases in 1995, reaching the number of 100 cases.

In the reported period analysis of cases with HIV-specific syndromes was of particular interest with their differentiation into inflammatory (HIV-encephalitis) and degenerative (HIV-leucoencephalopathy) forms. While comparing our own

material with larger collections from several countries it was found that separate HIV-leucoencephalopathy occurred relatively more seldom and mixed forms combining features of HIV-encephalitis and HIV-leucoencephalopathy appeared more frequently. Leptomeningeal inflammatory process, with typical multinuclear cells was found as a phenomenon accompanying parenchymal inflammatory changes. Independent vascular involvement in the form of HIV-angitis was found in several cases. In general, HIV-specific processes were present in 27.9% of our material, being in 13% of cases the only pathological brain process. In the remaining cases HIV pathology was accompanied by oportunistic infections and neoplastic proliferation. Incidence of HIV-specific syndromes in our material was almost identical with that in American collection, rather than in Italian-Austrian series and twice as frequent comparing with Swiss collection.

HIV-accompanying processes, such as vacuolar myelopathy were strikingly seldom. As far as opportunistic infections are concerned much higher incidence of cytomegalic and papova viral infections, and smaller frequency of toxoplasmosis was noticed in our material. Relatively more common comparing with foreign collections was aspergillus infection. Number of descriptive diagnosis of micronodular encephalitis, without etiological morphological indication was relatively small, despite of the fact that diagnosis was based mostly on histopatological examination.

NEURONAL AND NEUROGLIAL NEOPLASMS OF CNS. MORPHOLOGICAL AND IMMUNOHISTOCHEMICAL CHARACTERISTICS

Prof. Halina Kroh

Due to the scarce frequence of this group of tumors the study is limited to small number of gangliogliomas. Biopsy material involves two types of neoplasm: classic and anaplastic, classified according to the features of glial elements. Neuronal component is difficult to evaluate as regards the origin of changes such as atypia, hamartrosis or neoplasia. The characteristics of neuronal changes seem to be without influence on the evaluation of the type or the degree of anaplasia. Immunohistochemical properties of neoplasms display duplicating, intrinsic pattern of initial cells.

MECHANISM OF AMMONIA-INDUCED TAURINE (TAU) RELEASE FROM MÜLLER GLIA CULTURED *IN VITRO*

Prof. Jan Albrecht

Supported by the State Committee for Scientific Research: grant # 6 P207 059 05

It was found that ammonium ions stimulate taurine (TAU) release from cultured rabbit Müller cells in a dose-dependent manner, at the doses ranging from physiological to those accompanying hyperammonemic coma. The release of TAU evoked by ammonium ions was correlated with the increase of intracellular cAMP, and decreased or abolished by pharmacological treatments inhibiting cAMP synthesis. Stimulation of TAU correlated with cAMP accumulation was also observed following treatment with inhibitors of various protein kinases, a high-magnesium calcium-free medium, and modulators of intracellular pH. A hypothesis has been put forward that Müller cells react with cAMP-dependent TAU release to non-specific changes in cell shape, volume, and/or organization of cytoskeleton. Thus, the response may reflect the osmoregulatory or neuromodulatory functions of TAU.

THE ASSESSMENT OF CYTOTOXICITY OF 2-CHLORODEOXYADENOSINE (2-CdA) AND 2-BROMODEOXYADENOSINE (2-BdA) TOWARD ANAPLASTIC GLIOMAS

Prof. Mirosław J. Mossakowski

Supported by the State Committee for Scientific Research: grant # 4 4344 92 03

Electron-microscopic studies with organotypic cultures have shown that the exposure of a normal tissues of hippocampus and cerebellum of young rats to 2-CdA, concentrations up to 10 micromole for up to two weeks does not produce any damage of cellular structures. However, the exposure of organotypic cultures of malignant gliomas to the same concentrations of 2-CdA leads to progressive mitochondrial damage. Since micromolar concentrations of 2-CdA cannot be achieved through systemic administration (because bone marrow toxicity), a development of local intracerebral delivery system for this drug is indicated.

ELABORATION OF EXPERIMENTAL MODEL OF ALZHEIMER'S DISEASE

Assoc. Prof. Ryszard Pluta
Supported by the State Committee for Scientific Research: grant # 6 P207 051 05

It was synthesized 476.2 mg of β-amyloid peptide. For the first time a direct evidence is provided that the increase level of circulating soluble human β-amyloid peptide increases cerebral blood flow in normal rat. Our data showed that in the rat brain, global cerebral ischemia can produce extracellular, perivascular accumulation of apolipoprotein E and apolipoprotein J and their increased intracellular immunoreactivity. Perivascular deposits of apolipoprotein E/apolipoprotein J colocalized with β-amyloid protein procursor that have been disclosed by us previously in this model.

REGULATION OF CEREBRAL MICROCIRCULATION DURING REPERFUSION FOLLOWING FOCAL CEREBRAL ISCHEMIA

Assoc. Prof. Ewa Koźniewska-Kołodziejska
Supported by the State Committee for Scientific Research: grant # 4 P05A 088 09

The aim of the study is to investigate the regulation of cerebral microcirculation shortly after reperfusion and in the phase of delayed hypoperfusion following focal cerebral ischemia in the rat. The study comprises: 1. The effect of inhibition of the baseline release of nitric oxide NO on cerebral microcirculation and modulation of this response by an inhibition of the superoxide anion production; 2. The reactivity of cerebral microcirculation of acetylcholine, papaverine and blood gases; 3. Participation of thromboxane A_2 in the phenomenon of delayed hypoperfusion.

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MORPHOLOGICAL AND MORPHOMETRICAL ANALYSIS OF THE CNS STRUCTURES DURING DEVELOPMENTAL PERIOD IN NORMAL AND PATHOLOGICAL CONDITIONS. SUMMARY OF INVESTIGATIONS ON THE DEVELOPMENT OF HUMAN HIPPOCAMPUS IN PERINATAL PERIOD

Prof. Maria Dąmbska

We have studied the development of hippocampal structures in the prenatal and postnatal period. The results obtained from hippocampal cortex (archicortex) were compared with the inferotemporal neocortex and with the dorsal motor vagal nucleus of the medulla. The numerical density of neurons, cell perikarya and nuclear cross-sectional area, ratio of nucleus to perikaryon area were measured. The results showed considerable differences in the rates of neuronal development. The present data completed previous observations demonstrating that CA2 neurons are most advanced in developmental process. CA1 neurons which mature slowly approached the rates of neocortical development. The sequence of cell maturation in layer III and V of the inferotemporal cortex was similar to a certain degree to that observed in CA1 sector, with its higher intensity during postnatal period. The differences in the neuronal maturation of the dorsal vagal motor nucleus and cortex reflect the functional differences between these structures.

In the second part of the investigations on the recognization of developmental abnormalities two cases with malformations were found. Their examination revealed the way by which the recent developmental disturbances may cause appearance of another brain malformations. An infant, who was one of the cases, died of multiorganic malformations and brain dysgeneses, consisting of early disturbances in neuronal migration, represents may be a new cerebro-oculo-cutaneous syndrome.

ULTRASTRUCTURAL AND IMMUNOHISTOCHEMICAL STUDY OF DISORDERS APPEARED DURING CNS DEVELOPMENT. GENE EXPRESSION. EFFECT OF VINCRISTINE NEUROTOXICITY

Assoc. Prof. Danuta Maślińska

Programmed cell death (apoptosis) regulates cell homeostasis in the body. Some environmental agents (eg. plant alkaloids) may induce or inhibit apoptosis in the tissue and disturb the cell balance. During apoptosis typical morphological and biochemical changes are observed but in the CNS this process is not quite clear. In the present study, the new technique of labelling *in situ* the DNA fragmentation characteristic for apoptosis was employed, and the results show, that vincristine - a widely used drug, induces apoptosis in the brain of the treated animals. Additionally, in the affected brain cells the ultrastructural changes characteristic for apoptosis have been also found. These observations lead to the conclusion that apoptosis may be an essential mechanism of vincristine neurotoxic effect on the CNS during chemiotherapy.

The study performed on autopsy human brains showed, that the plant cyclic peptides of poisoning mushrooms (*Amanita phalloides*) had similar effect as vincristine alkaloids. In the brain of children (2-12 years old) accidentally intoxicated with those toxins the strong expression of proteins AP-1, c-jun and p53 were found. All the proteins appeared in the cell at the initial stage of apoptosis. In the neurons expression of those proteins was co-localized with expression of amyloid beta protein. The results may confirm the previous suggestion that some fragments of amyloid beta protein participate in the brain neuron degeneration.

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INFLUENCE OF GLOBAL CEREBRAL ISCHEMIA ON THE IMMUNO-ELECTRON REACTIVITY OF 5-HYDROXYTRYPTAMINE AND ENDOTHELIN IN HYPOTHALAMO-NEUROHYPOPHYSIAL SYSTEM AND HIPPOCAMPUS IN THE RAT BRAIN

Assoc. Prof. Barbara Gajkowska

Immuno-electron microscopic demonstration of subcellular distribution and a role of 5-Hydroxytryptamine (5-HT) and endothelin in the brain of the rat hypothalamo-neurohypophysial system and hippocampus after ischemia was studied.

The experiments were performed on adult Wistar rats. The animals were subjected to experimental 10 min global, cerebral ischemia. For immunocytochemical studies, using post-embedding immuno-gold technique, the animals were sacrificed from 10 min to 24 hours following ischemia. Immunolabelling was performed with 10 nm gold-antibody complexes for 5-HT and endothelin.

5-HT. Our results indicate that ischemia leads to stimulation of serotonin system in hypothalamo-neurohypophysial system and hippocampus. The ischemia-induced release of 5-HT affects directly neurones, and neurotransmitter system contributes to excitotoxic damage.

The subcellular distribution and a role of the **endothelin** in the rat hypothalamo-neurohypophysial system and hippocampus after ischemia were examined.

In hypothalamo-neurohypophysial system endothelin-like immunoreactivity was enhanced after ischemia suggesting that endothelin plays a role in endocrine regulation acting as hypothalamic neuromodulator controlling secretion of the posterior pituitary hormones.

Electron-microscopic immunocytochemical evaluation of localization of endothelin in hippocampus revealed that endothelin is present not only in endothelial cells of hippocampal microvessels but also in astrocytes, microglia and macrophages and in some axonal endings. The most pronounced changes in the endothelin-like

immunoreactivity was found 24 hours after ischemia. Our results suggest that ischemia-induced increased content of endothelin may play an important role in the pathogenesis of post-ischemic tissue abnormalities.

INTRANUCLEAR INCLUSIONS IN CORTICAL NEURONS OF RATS AFTER EXPERIMENTAL CARDIAC ARREST

Assoc. Prof. Michał Walski

The study concerned examination of the nuclei of neurons derived from the cerebral cortex of rats that underwent 10-min experimental cardiac arrest followed by reanimation of the animals that survived from 2 weeks up to 10 months. The animals were divided into two groups: the first included these specimen that survival from 2 to 6 weeks; the second those that survived 8 weeks to 10 months. In the neurons of animals of the first group we observed irregular vesicular structures and bundles of parallel fibrils 8 nm in diameter which were surrounded by low density chromatin. In the second group we often observed paracrystalline form resembling "plaited ropes" and tubulous structures linked by microfibrillar netting. These structures were analysed with a gonyometer. The tubulous and paracrystalline structures were observed in the neuronal nuclei present in the zones of perivascular fibrosis. Post ischaemic karyoskeleton rebuilding is probably caused by the selective faulty expression of genes.

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COMPENSATIVE CRANIOSPINAL SYSTEM CHANGES DUE TO HEMODYNAMIC AND CEREBROSPINAL FLUID FLOW DISTURBANCES

Prof. Zbigniew Czernicki

Based on the mathematical formula, developed in the Department, a new method to evaluate CSF content in cerebral ventricles, basal cisterns and subarachnoid spaces of the brain convexity separately was introduced into the clinical practice.

While comparing the influence of calcium channel blockers - Dotarizine and Flunarizine - on the cerebral vessels reactivity at different CO_2 concentrations it was shown that during hyperventilation Dotarizine causes larger cerebral vessels dilation than Flunarizine. It was also found that during anoxia Dotarizine causes an increase of the cerebral blood flow and considerable vascular dilation in comparison with anoxia alone.

Potential mapping technique in focal brain lesions documented on CT or MRI scans was developed considering its diagnostic and prognostic application in the clinical practice.

A method of physiological loadings in the diagnosis of the subarachnoid spaces tightness was developed and applied in the clinical practice.

Using the EEG cerebral mapping method the creation of the secondary, so-called mirror-like, epileptic foci in cerebral hemispheres in the course of epilepsy was documented which is a progress in comparison with traditional EEG examination. This technique has practical implications in the diagnosis of epileptic foci and qualification for surgical treatment.

Somatosensory evoked potentials (SEP) in patients treated with thalamotomy were studied preoperatively and in the early and remote time postoperatively. Characteristic SEP changes corresponding to the postoperative functional improvement in the treated patients were found. They reflect reorganization of the critical input paths after thalamotomy.

Based on the results of previous studies a new method was developed in order to assess an influence of the type of cognitive activity, the test design and timing of the spinal registration on the blood flow velocity changes (transcranial Doppler sonography).

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DEGENERATIVE AND REGENERATIVE CHANGES IN IMMATURE AND SENILE MUSCLE (EXPERIMENTAL STUDIES AND CONGENITAL MYOPATHIES)

Prof. Anna Fidziańska-Dolot

Morphological and immunohistochemical studies of the muscle biopsies were performed in 3 cases of the fatal infantile form of spinal muscular atrophy (SMA). Fetal-like pattern of the muscles was confirmed by the classical histological methods as well as by desmin and neonatal myosin studies.

In addition, a new finding such as massive muscle cell elimination by apoptosis was observed. The ultrastructural features of this phenomenon were essentially the same as those found in human embryogenesis. Affected muscle cells die rapidly with characteristics of programmed cells death. The ability of immature muscle fibres to undergo apoptosis stimulated us to study experimentally injured neonatal rat skeletal muscle. As a noxious agents there were used bupivacaine, notexin and cold.

All those injurious stimuli caused apoptosis in neonatal rats and necrosis in rats older than 7 days of age. Dying immature muscle cells bore the same ultrastructural characteristics as apoptotic cells in other tissues. The ability of neonatal muscle to undergo apoptosis or necrosis in response to injury is modulated by the stage of its maturity.

Clinical, immunohistochemical, cytogeneic and immunoblot studies of a 5-year old girl with clinical symptoms of Duchenne muscular dystrophy and X:22 translocation showed that the translocated gene is still expressed in truncated dystrophin protein associated with the plasma membrane.

IMMUNO-ELECTROPHYSIOLOGICAL CORRELATION IN AMYOTROPHIC LATERAL SCLEROSIS (ALS) AND ALS-RELATED SYNDROMES

Prof. Irena Niebrój-Dobosz

The aim of this study was to find relationships between the presence of antibodies against gangliosides in serum and cerebrospinal fluid and the block of conductivity in ALS and ALS-related syndromes. In the relatively large group of 78 ALS patients, 24 cases of ALS-related diseases no such a relation was ascertained. Elevated titer of antibodies against gangliosides was present in neuropathies, the congenital ones including. No correlation, however, was found between the presence of the conductivity block and the antibody titer. The present findings argue, therefore, against the opinion, that the presence of conductivity block is a phenomenon differentiating the aquired and congenital neuropathy. The present study confirmed the lack of diagnostic or prognostic value of anti-ganglioside determinations in ALS and ALS-related syndromes. Presence of circulating antigangliosides is not a consequence of higher content of the determined also related antigens. The conducted morphological studies indicate necessity of caution in treating the MRI results as indicators of the white matter changes in ALS.

CLINICAL, GENETICAL AND ELECTROPHYSIOLOGICAL CHARACTERISTIC OF SPINAL MUSCULAR ATROPHY

Assoc. Prof. Katarzyna Rowińska-Marcińska

The studies concerning clinical and genetical analysis of the spinal muscular atrophy have been accomplished. Examination of 590 cases from 445 families revealed various course of the disease. A correlation between the age of the first symptoms and the age of the immobilization has been found as well as a correlation between the sex of the patients and the prevalence and the course of the disease. Additionally, the fenomenon of the double motor unit discharges has been analysed in spinal muscular atrophy and amyotrophic sclerosis cases. The method for quantitative evaluation of the fenomenon was elaborated. According to our data discharges of the MU occur frequently in peripheral neuron lesion, especially in ALS. The origin of the fenomenon in peripheral neuron lesion is not clear. There are arguments, supported by clinical and experimental studies, for motoneuron as well as for peripheral origin of double discharges. One may suspect that shape

evaluation may contribute to the discussion on double potential origin. It seems, important that voluntary activated double discharges have been observed mainly in the motor unit with signs of reinnervation often in the muscle with preserved force.

CLINICAL, IMMUNOLOGICAL AND IMMUNOGENETIC CORRELATIONS IN POLISH PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOPATHIES

Prof. Irena Hausmanowa-Petrusewicz

Idiopathic inflammatory myopathies are heterogeneous group of diseases of unknown etiology. Various environmental and immunogenetic factors play a role in the induction of humoral and cellular response and chronic inflammation of the muscle. In our series we found myositis specific autoantibodies (MSA) in 19% of patients: antisynthetases, anti-SRP, anti-Mi-2 and myositis associated antibodies: anti-PM-Scl and anti-Ku.

Antisynthetases were found in patients with severe myositis and interstitial lung disease, anti-Mi-2 in patients with benign dermatomyositis with good response to therapy and anti-SRP was associated with very severe myositis, unresponsive to aggressive immunosupressive therapy.

We found significantly increased frequency of DQA1 and DRB1 0301 alleles in patients with idiopathic inflammatory myopathies as compared to published control population.

CLINICO-GENETIC CHARACTERISTIC OF MUSCULAR DYSTROPHY

Prof. Irena Hausmanowa-Petrusewicz

The limb-gridle muscular dystrophy is a heterogeneous group of dystrophies which presently is submitted to reversion due to the progress of molecular genetics. Application of DNA tests and evaluation of quantity and quality of muscle dystrophin enables to separate it from the group of cases diagnosed previously as limb-gridle dystrophy, some number of cases of Becker dystrophy or even Duchenne dystrophy. Till now the number of such cases is 18/50. Moreover, the new molecular methods permit to recognize the dystrophinopathies also in affected females - such cases were previously considered as indicative for limb-gridle dystrophy. These findings are extremely important for genetical counceleing in affected families.

MORPHOLOGICAL CHANGES IN THE SURAL NERVE IN CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY

Dr. Hanna Drac

Chronic inflammatory demyelinating polyneuropathy (CIDP) is believed to be caused by not precisely known immunodeciated damage to peripheral nerves. Morphological examination of sural nerve in the nine cases of CIDP revealed a heterogeneity of morphological picture. Mixed changes (demyelination and axonal degeneration) were noticed more frequently than demyelination. Active demyelinating lesion was sporadically encountered but remyelination and onion bulb formations were noninfrequently visible. Mononuclear cells dispersed in the endoneurium were present but mononuclear cell infiltrations were absent. Subperineurial and endoneurial oedema was present in most of cases. Unmyelinated fibers were involved in pathological process in some cases. Sural nerve biopsy is supportive but not fundamental for the diagnosis of CIDP.

N-CAM AS A MARKER OF MUSCLE CELL IMMATURITY

Prof. Anna Fidzianska-Dolot

Supported by the State Committee for Scientific Research: grant # 4 S405 005 06

Continuing the previous studies on neural cell adhesive molecules (N-CAM) it was shown that in adult human muscle N-CAM is concentrated near to neuromuscular junctions and on satellite cells but it is nearly undetectable in nonsynaptic portions of myofibres. N-CAM is abundant on myotubes and intramuscular nerves in embryonic muscle but is lost as development proceeds. After denervation N-CAM appears along the entire length of muscle fibres. In addition N-CAM is observed on the surface of regenerating muscle cells.

These data suggest that N-CAM immunohistochemistry may be considered as a conventional technique in the diagnosis of neuromuscular diseases.

CLINICO-GENETIC CORRELATION IN RECENTLY RECOGNIZED DYSTROPHINOPATHIES

Prof. Irena Hausmanowa-Petrusewicz
Supported by the State Committee for Scientific Research: grant # 4 P05B 113 08

Atypical dystrophinopathies were demonstrated in a group of 48 patients with Becker dystrophy. The dystrophin abnormalities were examined using immunocytochemical staining and quantification by Western blot. The dystrophin changes were expressed as mosaic pattern and/or drop of amount of dystrophin and its reduced molecular weight.

In respect of those changes a pronounced interfamilial variability was stated (no intrafamilial variability!). In some cases the changed pattern of dystrophin was associated only with cardiac involvement or myoglibinuria without evident muscle symptoms.

ANGIOGENESIS AND MYELINATION OF THE CENTRAL NERVOUS SYSTEM

Prof. Janina Rafałowska
Supported by the State Committee for Scientific Research: grant # 4 S402 071 06

Developmental failures of blood vessels within central nervous system.

Basing on the literature and own material the author considered some causes of development of vessel malformation: aneurysms, teleangiectasis, arterio-venous malformations and cavernous angiomas, paying attention to difficulties in classifying developmental vescular malformations and choice of terminology. Presumable etiopathogenesis of some malformations with particular regard to a role of viral oncogenes is taken into account.

Coexistance of various vascular malformations within the brain.

Two cases of basilar artery aneurysm accompanied by different development failures of blood vessels were presented. In both cases anomaly in formation of brain base vessels, angioma consisted of different size thin-walled vessels, and arterio-venous angioma within brain stem were stated. Besides, conglomerates of abnormal vessels were found. The variability of vascular malformations seems to point to a long-lasting action of pathogenetic factor during ontogenesis. An attempt was made to refer particular developmental abnormalities to a proper stage of ontogenesis.

MOTOR CONTROL IN PATIENTS WITH CHILDHOOD SPINAL MUSCULAR ATROPHY

Prof. Irena Hausmanowa-Petrusewicz

Supported by the State Committee for Scientific Research: grant # 6 6372 92 03

The simultaneous EMG activity of lower limbs and lower trunk muscles (12 muscles together) was recorded with surface electrodes in 19 children with SMA and control group of age-matched healthy children. EMG data were compared with manual testing of muscle strength.

The electromyographic examination of children with spinal muscular atrophy performed during 6 different motor tasks revealed an excessive coactivated activity both in agonist and antagonist muscles during volitional single ankle and postural motor task.

The extension of coactivated activity correlated with the degree of muscle weakness. The same kind of EMG examination performed in dystrophic children revealed similar motor control changes but not so pronounced and less correlated with muscle weakness.

The abnormal motor control in SMA children was confirmed by transcranial magnetic stimulation.

The excessive irradiation getting to many unexpected muscle groups might be beneficial for postural control but certainly is impairing some volitional motor activities.

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RESPONSES OF THE SYMPATHO-ADRENAL SYSTEM TO PHYSIOLOGICAL STIMULI - MODIFYING FACTORS, INTERACTIONS WITH OTHER HORMONES

Prof. Krystyna Nazar

Changes in blood glucose, insulin, noradrenaline (NA) and adrenaline (A) concentrations in response to oral glucose load (70 g) were studied in 65 men aged 18-60 years. The initial levels of plasma A and NA were found to increase with age ($r=0.39$, and 0.37), whilst the glucose-induced elevation in plasma catecholamines did not correlate with age, body mass, or changes in blood concentrations of glucose and insulin.

It was demonstrated that in young men 3-day bed-rest impairs glucose tolerance, which is accompanied by an increase in insulin secretion. Basal activity of the adreno-medullary system, and responses of plasma catecholamines to physiological stimuli were reduced, whereas the increase in plasma renin activity, induced by orthostatic manoeuvre, was exaggerated.

During graded exercise at high ambient temperature plasma NA concentration at heavy loads was found to increase more than at thermoneutral conditions, which was accompanied by decreased psychomotor performance.

FACTORS AFFECTING LIPID AND CARBOHYDRATE UTILIZATION IN SKELETAL MUSCLES *IN VITRO* AND *IN SITU*

Assoc. Prof. Leszek Budohoski

The relationship between fatty acid uptake from the incubation medium and the concentration of a monomeric form of this compound (unbound to albumin) was evaluated in the incubated soleus muscle. It was shown that both, at constant con-

centration of albumin (concentration of unbound fatty acids increases proportionally to the total concentration of fatty acid) as well as at the constant ratio [FA]/[albumin] (concentration of unbound fatty acid unchanged) palmitate uptake increased with the increased concentration of the total fatty acids. Results obtained suggest that a different mechanism than passive diffusion, for fatty acid uptake may exist. Furthermore, the effect of thyroid hormones deficit on fatty acid uptake by incubated muscle was evaluated. No differences in palmitic acid uptake between thyroidectomized and control rats were found. The rate of intramuscular TG synthesis was increased in thyroidectomized rats at low palmitate concentration. At higher concentrations (1.5-2.0 mm) the rate of this process was diminished in this group of animals.

Sensitivity of solues muscle to insulin was evaluated in hypothermic rats. It was shown that under this condition, the rates of lactate production and glycogen synthesis as well as the sensitivity of both processes to insulin were increased in comparison with normothermic rats of matched age and body mass. However, the rate of glucose transport was diminished by hypothermia, and this process was not sensitive to insulin.

RENAL MEDULLARY ADMITTANCE (ELECTROLYTE CONCENTRATION) DURING EXPERIMENTAL CHANGES IN RENAL PERFUSION PRESSURE (RPP)

Prof. Janusz Sadowski

The effect of RPP on medullary electrolyte concentration was studied in anesthetized rats. RPP was changed by suprarenal aortic constriction. Tissue electrical admittance (Y) and medullary blood flow (MBF) were measured simultaneously using an integrated flow-and-admittance probe, and cortical blood flow (CBF) by laser-Dopper probe on kidney surface. For RPP range 80-120 mm Hg a 20% pressure change altered Y by 5%. RPP decrease from 120 to 100 mm Hg reduced CBF by 5% and MBF by 25%, suggesting good blood flow autoregulation in the cortex and poor in the medulla. A further RPP decrease to 80 mm Hg reduced CBF and MBF by 15% (poor autoregulation of the former and, paradoxically, better regulation of the latter). The data indicate a modest role of RPP in the control of electrolyte gradient in rat kidney. Unidirectional changes of Y and RPP suggest importance of NaCl delivery to Henle's loop, depending on the actual rate of glomerular filtration.

EFFECT OF LOW-CARBOHYDRATE DIET ON PHYSIOLOGICAL RESPONSES TO EXERCISE IN MEN AND WOMEN

Prof. Hanna Kaciuba-Uściłko
Supported by the State Committee for Scientific Research: grant # 4 S404 028 07

The aim of this study is to evaluate the effects of 3-day ketogenic diet (<5% carbohydrates) on anaerobic and aerobic capacity, psychomotor performance, as well as on metabolic and neurohormonal responses to exercise of various characteristics. It was demonstrated that ketogenic diet impairs ability to perform exercise, in which glycolysis predominates in energy supply (anaerobic capacity). In each type of exercise (supramaximal, graded to maximum, prolonged) an increase in blood lactate level was reduced. Neurohormonal responses to supramaximal and graded exercise were modified by the ketogenic diet, e.g. catecholamine and growth hormone release to blood was increased, whereas insulin and testosterone secretion was diminished. The choice reaction time, measured during graded exercise of increasing intensity, was found to be shortened after the ketogenic diet, which indicates improved psychomotor performance.

AN INTEGRATED PROBE FOR SIMULTANEOUS RECORDING OF BLOOD FLOW AND TISSUE ION CONCENTRATION IN RAT RENAL MEDULLA

Prof. Janusz Sadowski
Supported by the State Committee for Scientific Research: grant # 4 P05A 013 08

The relation between electrolyte concentration in renal medullary interstitium and medullary blood flow has never been elucidated, due to an absence of suitable methods. An integrated probe was constructed for simultaneous continuous recording of tissue electrical admittance, a measure of total ion concentration (by a method earlier developed in this laboratory), and medullary blood flow (using a laser-Doppler flow meter). In order to limit inevitable damage of a part of renal tissue, the size of the integrated probe was reduced by using the metal cannula housing the laser-Doppler light fiber as one of two admittance electrodes. This was of critical importance since rat kidney weight equals ~1 g and that of the medulla ~30 mg. Insertion of such intergated probe caused only a minor reduction of the kidney's glomerular filtration rate. The validity of admittance measurement in the modified system was confirmed in *ex vivo* studies of medullary tissue slices.

CONSTRUCTION AND SOFTWARE DESIGN OF A DEVICE ENABLING CONTINUOUS 24-h MONITORING OF HAEMODYNAMIC HEART ACTIVITY USING IMPEDANCE CARDIOGRAPHY - REOMONITOR

Dr. Gerard Cybulski
Supported by the State Committee for Scientific Research: grant # 8 S506 013 05

The new ambulatory monitoring, universal 4-channel recorder enabling simultaneous collection of ECG and central haemodynamics signals was constructed. The system is based on 80C552 family controller with build-in analogue to digital converters and 20MB PCMCIA (type II) FLASH MEMORY CARD is used as data storage. The communication with the system is performed via specialized keys and the small, built-in LCD. We designed and constructed the miniaturized, tetrapolar, current impedance cardiography device with built-in one channel of ECG, which was applied as a detector of the central haemodynamics signals. The device enables the full disclosure of the collected data, which can be used in off-line, beat-to-beat evaluation of cardiac output, stroke volume, ejection time, pre-ejection period and heart rate.

The device can be used in determination of haemodynamic efficiency in healthy subjects (in sports medicine) or in patients during normal heart work, ischemia or arrythmia events, for supplementary diagnosis or evaluation of medical therapy.

TRIGLYCERIDE METABOLISM IN SKELETAL MUSCLES

Prof. Hanna Kaciuba-Uściłko
Supported by Polish/USA Maria Skłodowska-Curie Joint Fund II,
grant MZ/HHS-92-103

In cooperation with Department of Physiology, State University of N.Y. in Syracuse, U.S.A., the studies on muscle lipid metabolism were continued.

The obtained data confirmed our previous results that the rate of triacylglycerol (TG) synthesis is higher in red (soleus, red portion of gastrocnemius) than in mixed (plantaris) or white (white portion of gastrocnemius) muscle types. Further, it depends on fatty acid (FA) availability, being significantly higher during 2 mM than 0.5 mM palmitate perfusion. The new findings of the present study are that the fractional degradation of intramuscular TG is: a/ fairly high representing a large turnover of the stored energy substrate; b/ similar in all muscle types, suggesting a similar cellular control process in TG turnover; and c/ dependent on extracellular

FA supply, which establishes TG synthesis rate for lipid deposition. The absolute rate of TG degradation was lower in the white portion of gastrocnemius than in other muscles. This is to be expected since white muscle fibres relay less on lipid oxidation than the red ones.

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TWENTY YEARS CLINICAL OUTCOME OF PATIENTS AFTER THE FIRST MYOCARDIAL INFARCTION IN RELATION TO MEDICAL OR SURGICAL TREATMENT

Dr. Ewa Wójcik-Ziółkowska

Retrospective analysis of clinical outcome of 81 patients 20 years after the first myocardial infarction (MI) showed that during the observation 23.5% patients died, including 52.7% cases of cardiac death. Among survivors 79% were medically treated (group 1), and 21% (group 2) underwent surgical treatment (CABG or PTCA). In comparison with group 1, in group 2: incidence of recurrent MI were two times greater, hypertension, and hyperlipidemia occurred more often ($p<0.01$ and $p<0.01$ respectively). There was also more frequent incidence of angina pectoris with ST-segment depression in ECG, and severe ventricullar arrhythmias ($p<0.01$). Seventy percent of patients from group 2 had >3 vessel disease. Indices of left ventricular function, etimated by echocardiography, were similar in the two groups. It is concluded that unstable course of coronary artery disease, even in the late period after MI, is predictive of serious cardiac events and the most important indication for surgical intervention.

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EARLY CAPTOPRIL TREATMENT PREVENTS RISE OF ANGIOTENSIN II IN ACUTE MYOCARDIAL INFARCTION

Prof. Krystyna Cedro-Ceremużyńska

Activation of the renin-angiotensin-aldosterone (RAA) system following acute myocardial infarction (AMI) has been associated with serious arrhythmias, extension of myocardial damage and development of heart failure. Blockade of RAA by angiotensin converting enzyme inhibitors (ACEI) early in AMI have led to suppression of arrhythmias and reduction in mortality as shown by ISIS-4 trial. The notion that ACEI exert their effect by blocking production of angiotensin II (AII) is based primarily on experimental studies and requires confirmation in AMI patients. In 63 patients with AMI enrolled into ISIS-4, randomly treated with ACEI (Captopril 6.25 up to 50 mg bid) or placebo, serum renin, AII, aldosterone were estimated on day 1 and 3. ACEI led to suppression of AII ($p<0.05$), had no effect on aldosterone, and increased (reflexly?) plasma renin activity ($p<0.01$). These results show that suppression of circulating AII, both proischemic and proarrhythmic hormone, may contribute to benefits related to ACEI therapy in AMI.

L-ARGININE INCREASES EXERCISE CAPACITY IN PATIENTS WITH STABLE ANGINA

Prof. Krystyna Cedro-Ceremużyńska

Endothelium-dependent vasodilator responses mediated by L-arginine/NO are impaired in various forms of cardiovascular diseases including atherosclerosis and ischemic heart disease (IHD). An attempt to restore these responses with L-arginine was successful in some experimental and clinical settings suggesting that reduced intracellular availability of L-arginine may be involved in this defect. Endothelial

dysfunction may adversely influence adaptation of the coronary blood flow to physical activity. We investigated whether L-arginine improves exercise tolerance in patients with stable angina. L-arginine (6 g/day) or placebo were administered to 25 randomized patients in a double-blind study. Exercise test (Marquette Case 12 treadmill system) with 12 lead ECG was performed according to Bruce protocol. Total exercise time to maximum ST depression increased, and the total duration of exercise was prolonged after L-arginine as compared with placebo ($p<0.02$ and $p<0.05$, respectively).

Increased exercise tolerance in L-arginine supplemented patients may be causally related to improvement of vasodilator responses depending on L-arginine/NO in vascular endothelium.

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PHENOTYPICAL CHARACTERIZATION OF CELLS EXTRAVASATING TO SKIN FOLLOWING LOCAL CYTOKINE APPLICATION

Prof. Waldemar L. Olszewski

Extravasation of immune cells into tissue takes place both in physiological (immune surveillence) and pathophysiological conditions (inflammation, cancer or graft). Process of leucocyte (granulocytes, lymphocytes, macrophages) extravasation is regulated by locally produced cytokines. In the studies on the role of cytokines in immune cell extravasation, the effect of either exogenous recombinat cytokines or cytokines produced endogenously and released to tissues was examined. In the latter situation, macrophages are the main source of the cytokines. In the studies performed in 1995, the kinetics of the extravasation and cell migration through tissues to afferent lymphatics, following blood injection into the tissues, was examined, using a dog model. It was found that following injection of FITC-labelled erythrocytes the increased tissue permeability for these cells was noted and their concentration in lymph increased by 10-50 fold. However, only 3-5% of the total number of injected erythrocytes could be found in lymph within 4 h. Calciparin and Polopirin S increased both lymph outflow and lymph erythrocyte and leucocyte concentrations. In the next part of the study, an analysis of commercial monoclonal antibodies specific for human immune cell antigens cross-reacting with dog cells was performed. Such a screening allows to select the proper antibodies for further phenotypic determination of immune cells extravasating to skin.

THE KINETICS OF RECIPIENT BONE MARROW RECONSTITUTION FOLLOWING VASCULARIZED BONE MARROW TRANSPLANTATION

Prof. Waldemar L. Olszewski

Vascularized bone marrow transplantation in orthotopic hind-limb graft to lethally irradiated syngeneic recipients repopulates recipients bone marrow cavities much faster than i.v. infusion of bone marrow cell (BMC) suspension. We have shown previously (*Transpl Proc* 1994, 26, 3319) that hemopoietic cells from transplanted hind-limb home to bone marrow of syngeneic recipients. The question arises what is the distribution of transplanted bone marrow cells in allogeneic recipients. To study the kinetics of migration of hemopoietic cells from the graft to the recipient, tibia bone marrow cells of BN rats were labelled with ^{51}Cr and injected i.v. into BN rats. After 24 hrs hind-limb containing ^{51}Cr labelled BMC was amputated and anastomosed to femoral vessels of 8 Gy irradiated or nonirradiated Lewis recipients. After another 24 hrs, the distribution of released cell bound radioactivity was measured in all tissues. For the control, ^{51}Cr labelled BMC in suspension were infused i.v. into other groups of rats. Our results show slight differences in allogeneic BMC homing in the recipients in comparison with syngeneic cells. Decreased accumulation of allogeneic BMC may be related to the allogeneic lymphocyte cytotoxicity (ALC).

THE MECHANISM OF THE RELEASE OF HEMATOPOIETIC CELLS FROM BONE MARROW FOLLOWING TRAUMA

Prof. Waldemar L. Olszewski

In the study on the mechanism of regulation of immune response following blood transfusion, normovolemic WAG rats were transfused intravenously 3 times with 2 ml of syngeneic heparinized plasma, every other day. No significant changes in WBC and bone marrow (BM) cell numbers/femur were noted after plasma transfusion (PT). There was, however, a significant decrease in the number of spleen (SPL) cells/g tissue on the day 3 following PT. Analysis of bone marrow showed no significant changes in the percentage of cells of erytroid, myeloid and lymphoid lineage during the entire observation period. Monoclonal antibody analysis revealed, however, a significant decrease in the percentage of $OX7^-$ stem cells in BM on days 3 and 7 after PT. A slight decrease in the percentage of BM $OX12^+$ B cells was also observed. Moreover, there was a significant drop in the percentage of $OX6^+$ class II-positive cells in bone marrow on days 3, 7 and 14 following PT. Functional

studies demonstrated a significant decrease in the responsiveness of blood (B) lymphocytes to PHA, ConA and PWM on day 14 after PT. The responsiveness of splenocytes to PHA was diminished on days 3 and 14 and to PWM on day 3 following PT. A reduction in the responsiveness of BM lymphocytes to PHA was observed on day 14 and to PWM on days 3 and 7 after PT. The results demonstrated that transfusions of syngeneic plasma evoked a decrease in the percentage of $OX7^-$ stem cells and $OX6^+$ class II-positive cells in bone marrow and reduced the responsiveness of B, SPL and BM lymphocytes to mitogens.

THE METHODS FOR CONTROLLING THE PROCESS OF CELLULAR REJECTION IN SKIN, LIVER AND IN BLOOD CIRCULATION

Prof. Waldemar L. Olszewski

The aim of the present study was to generate the monoclonal antibody (mAb) for skin Langerhans cells in a dog model. A great deal of effort has been directed by many authors to eliminate such cells from allografts. Only a limited effect was achieved by treatment with total body irradiation, cyclophosphamide or monoclonal anti-MHC class II antibodies. The major problem with raising mAb against dendritic cells (DC) is due to difficulties in obtaining the sufficient numbers of DC from skin or afferent lymph. Our model of experimental chronic lymphedema in the dog appeared to be useful for raising sufficient numbers of DC. Mice were immunized with enriched to 50% on metrizamide gradient DC population, and their splenocytes were fused with myeloma cells. Thirty hybridomas were isolated and screened for specific anti-DC mAb using immunocytochemical and cytometric analysis. Two clones producing mAb specific for DC were selected. We found these mAbs were not cytotoxic for DC, however, they reduced DC-lymphocyte clustering in a 4 h assay by 50%. MAbs used in allo-MLR profoundly inhibited allostimulation in a 6 day assay. The suppressive effect of mAbs in a 3 day assay with PHA was less pronounced. Preliminary results indicate that two specific for skin DC mAbs were obtained. These antibodies affect DC functions *in vitro*.

It has been postulated that the state of chimerism might favour graft acceptance and ultimately lead to donor-specific unresponsiveness. The process of donor cell migration from the graft to various tissues of the recipient has been well demonstrated after different types of transplantation. The question arises whether donor-specific transfusion, a therapy prolonging organ allograft survival, causes chimerism, and whether the chimerism coincides with tolerance to donor specific alloantigens.

LEW (RT11) rats were immunized with 1 ml of BN (RT1n) blood (DST). Seven days after DST donor mononuclear cells were identified in recipient blood, spleen and lymph nodes by flow cytometry using monoclonal antibody anti-donor MHC class I OX27. FITC-labelled BN lymphocytes were injected into DST-pretreated LEW rats, and 6 h later labelled cells were identified in recipient spleen, lymph nodes and liver using immunochemistry (mAb anti-FITC).

Seven days after DST we observed the presence of donor cells in recipient spleen (7.52±0.51%) and blood (4.47±1.0%) but not in the lymph nodes. Six hours after injection of FITC-labelled lymphocytes into DST-pretreated rats, only a few labelled cells in donor spleen and lymph nodes were observed, whereas accumulation of FITC-labelled debris in the liver was seen. This observation points to the acute elimination of donor lymphocytes by DST-pretreated recipient. DST-treated rats become temporarily chimeras but remain reactive to donor alloantigens. It seems that induction of chimerism is not enough to induce tolerance to donor alloantigens.

THE EFFECT OF LYMPH AND LYMPH CELLS FROM HUMAN SKIN ON PROLIFERATION OF TUMOR CELLS

Dr. Irena Grzelak

The subject of the studies which are carried out in cooperation with Tissue Culture Department, Norwegian Radium Hospital, Oslo, is the influence of lymph and lymph cells from human skin on proliferation of tumor cells. The question to answer is, how local tissue environment affects the growth of tumor cells which proliferate, at least at the initial stage, in normal humoral and cellular environment characteristic for the tissue in which they grow. In our previous study on the effect of human skin lymph on tumor cell proliferation, we have found that lymph fluid had both an inhibitory and stimulatory effect on tumor cell growth. In this study, the effect of lymph cells (lymphocytes, Langerhans cells) from human skin on growth of established tumor cell lines (B and T cell leukaemia, melanoma, sarcoma, epithelial carcinoma, epidermoid carcinoma, transformed keratinocytes) was examined in 72-hour cultures. It was found that the proliferation of tumor cells was decreased in the presence of lymph cells. Blocking of HLA ABC class I antigens with monoclonal anti-HLA ABC class I antibody did not affect the proliferation rate of tumor cells cultured in the presence of skin lymph cells.

CLINICAL AND EXPERIMENTAL CELL TRANSPLANTATION

Prof. Waldemar L. Olszewski
Supported by the State Committee for Scientific Research: grant # 6 P207 011 05

Transplantation of hepatocytes is a method of future in the therapy of metabolic defects. The reaction of the recipient on cellular transplants of cells isolated from organs has not been throughly studied, either in allogeneic or autogeneic systems. The processes usually not observed during or after organ grafting take place. We have noticed that intravenous hepatocyte transplants are acutely rejected, whereas liver organ grafts may survive even without therapeutic immunosuppression. Our studies have shown that transplanted hepatocytes are destroyed by own granulocytes, both after intravenous as well as after subcutaneous transplantation. Granulocytopenia attenuates the rejection process. *In vitro*, the cytotoxic effect is mediated by granulocytes and monocytes. Recipient sera may also have a toxic effect. These are original observations, throwing some new light on the process of cellular grafting.

LOCAL IMMUNE RESPONSE IN THE LIVER TO PROLIFERATING TUMOR CELLS AND IMMUNOMODULATION FOR PREVENTION OF LIVER METASTASIS FORMATION

Assoc. Prof. Barbara Łukomska
Supported by the State Committee for Scientific Research: grant # 4 P05B 020 08

The study was designated to investigate the cell adhesion molecules expossed by normal and tumor bearing liver and to evaluate the in vitro adhering capacity of liver lymphocytes (LAL) to the normal liver tissue and to the primary and metastatic hepatic tumors. The analysis of distribution of adhesion molecules in the normal liver revealed expression of ICAM-1, ICAM-2, ICAM-3, VCAM-1 and LFA-3 on sinusoidal cells. Sinusoidal cells of primary tumors showed higher expression of adhesion molecules compared to the normal liver. No adhesion molecules were observed on adenocarcinoma metastatic cells, the expression of ICAM-1, ICAM-2, VCAM-1 and LFA-3 was identified on capillary blood vessels present at the periphery of secondary tumor lesions in low density. There were many more lymphocytes adhered *in vitro* to the primary than secondary tumors or normal liver tissue. Anti-CD-11a and anti-CD11a/18F(ab)$_2$ fragments of MoAbs (specific for ICAMs ligands) caused inhibition of LAL binding to the liver tissue

by 45%, inhibition of LAL adherence by other integrins was less effective. In conclusion, lower density of adhesion molecules results in less binding of liver lymphocytes to the normal liver and metastatic foci. CD-11a/18 is an important pathway of lymphocyte adhesion to the sinusoidal lining cells, although some other organ specific molecular structures can also be involved.

HUMAN IMMUNE PERITONEAL CELLS IN PERITIONITIS AND DIGESTIVE TRACT TUMORS

Dr. Urszula Kubicka

Supported by the State Committee for Scientific Research: grant # 4 S402 128 07

Little is known about involvement of peritoneal cells in local immune response to cancer of digestive tract in human peritoneum. In our previous studies, the phenotypical characterization of free peritoneal cells in patients undergoing elective cholecystectomy was performed. This population comprised of 45% of macrophages and over 50% of T cells, mostly with cytotoxic/suppressor phenotype. The aim of the present study was to analyze the population of free peritoneal cells in patients with digestive tract tumors. Especially, its cellular composition and anti-bacterial and anti-tumor cytotoxic activity was examined. Moreover, the effect of cytokine stimulation on peritoneal cells and accumulation of cells in peritoneal cavity has been studied. There was a preferential accumulation of cells of myelomonoidal lineage in peritoneal cavity of patients with colon adenocarcinoma. The percentage of CD3 T lymphocyte decreased compared with non-tumor control. Within the CD3 T cells, the increase of activated T lymphocytes ($CD25^+$, $CD54^+$, $CD71^+$) was seen. There was also an increase in the expression of adhesion molecule CD54 on peritoneal macrophages compared with control patients. Changes in the proportions of various subsets of free immune cells in peritoneal fluid in patients with colon cancer suggest that even in the absence of penetration of tumor through the serosa there is an immune alert in peritoneum in patients with cancer. The standarization of the results obtained from our study may allow to evaluate the immune response of the host to the tumor developing in abdomen.

THE MECHANISM OF HOMING OF CYTOTOXIC IN LIVER SINUSOIDS - THE ROLE OF THIS POPULATION IN LOCAL REACTION AGAINST NEOPLASMATIC CELLS

Sergiusz Durowicz, M.D.

Supported by the State Committee for Scientific Research: grant # 4 P05A 020 09

It is well known that the specific lymphocyte population with high cytotoxic activity against neoplasmatic cells marginates in liver sinusoids. It plays an important role in in vivo tumor cell elimination. The question arises what is the molecular mechanism of lymphocyte trapping in liver sinusoids. To study this problem, we have examined the influence of trypsin, lidocaine, sodium azide, neuramidase, and anti-asialo GM1 anti-serum on lymphocyte trapping in rat liver sinusoids. It seems that these factors may influence receptor-depending lymphocyte trapping. Our preliminary studies suggest that lidocaine, sodium azide and AAGM1 anti-serum diminish lymphocyte trapping in liver sinusoids. At the same time, to study the influence of methastatic tumor on lymphocyte trapping, we have elaborated a model of adenocarcinoma coli metastases into the rat liver. The aim of our further investigations is to estimate the influence of blocking of the respective molecular adhesion molecules on lymphocyte trapping in liver sinusoids.

A NEW MECHANISM OF CYCLOSPORIN A ACTIVITY IN MIGRATION OF ALLOGRAFT REJECTION - INHIBITION OF HOST LYMPHOCYTE MIGRATION TO THE GRAFT

Michał Maksymowicz, M.D.

Supported by the State Committee for Scientific Research: grant # 4 P05A 083 09

The studies of the last half a year were devoted to the problem of the effect of Cyclosporin A treatment on expression of adhesion molecules (LFA1/ICAM1, VLA4/VCAM1, CD2, CD44, ELAM1) on blood mononuclear and lymph cells in the rat model. Cytocentrifuged smears of blood and lymph cells were stained with monoclonal antibodies against the above mentioned adhesion molecules. These experiments represent preliminary phase of the study which will be carried out using flow cytometry.

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AMINAL STUDY OF "ORAL TOLERANCE"

Assoc. Prof. Andrzej Lipkowski

A protein given orally induces immunological tolerance to antigens generated from this digested protein. This phenomenon known as oral tolerance is under extensive study as a potential treatment for autoaggressive diseases. Experimental allergic encephalomyelitis (EAE) is the animal model of autoagressive diseases such as multiple sclerosis. The aim of our study was to evaluate the usefulness of spinal cord hydrolizates as a potencial specific immunosupressive agents in multiple sclerosis treatment. The preliminary study on rats with EAE showed that animals treated with bovine spinal cord hydrolizate have diminished number of inflammatory infiltrates. This study was accomplished under scientific cooperation with the Industrial Chemistry Research Institute, Warsaw.

INTERACTION OF OPIOID RECEPTORS WITH OTHER NEUROTRANSMITTER SYSTEMS IN PAIN SIGNAL TRANSDUCTION

Assoc. Prof. Andrzej Lipkowski

In recent years major receptor systems involved in pain transmission have been characterized. These receptors form a mosaic of elements with complex networks of cross-interactions. The endogenous neuropeptide opioid system plays a major role in suppression of pain signal transmission in both central and peripheral nervous system. Nevertheless, other neuropeptides, in addition to pain modulation, play a role in mediation of variety of somatomotor, autonomic and other neural functions, including modulation of side effects of opioids. Therefore, we believe that

using selected neuropeptides or their analogues or antagonists as additives to opioids could be one of the effective approaches of increasing effectiveness of pain treatment with possible reduction of side effects. We have targeted our study on the interaction of opioid system with neuropeptides, substance P and neurotensin. Substance P antagonists or neurotensin itself produce very weak antinociceptive effects. We were able to show that the co-injection of these substances with opioids results in an elevation of antinociception produced by low doses of opioids without any visible neurological disfunction. In addition, we have observed that the co-injection of substance P antagonists delayed the development of opioid tolerance. This study was accomplished under scientific cooperation with the Departments of Anesthesia and Pharmacology, New England Medical Center, Boston, USA.

STRUCTURE-ACTIVITY RELATIONSHIPS STUDY OF OPIOID PEPTIDES

Assoc. Prof. Andrzej Lipkowski
Supported by the State Committee for Scientific Research: grant # 6 6337 92 03

In order to overcome the undesirable and toxic side effects of opioids, currently used as drugs, we have targeted our structure-activity relationship (SAR) studies of endogenous opioid peptide analogues at the design of new generations of antinociceptive drugs with various receptor selectivity, pharmacokinetic and pharmacodynamic profiles. Using a combination of computer assisted design, synthetic chemistry, and multiple pharmacological assay methods we have elaborated general topographical models of ligands for delta, mu and kappa opioid receptors. Using this model were able to synthetize a number of selective opioid ligands, including alkaloid-peptide hybrids, which are under extensive animal study as potential antinociceptive drugs. This study is accomplished under scientific cooperation with the Department of Anesthesia, Massachusetts General Hospital, Boston, and Department of Chemistry, University of Arizona, Tucson, USA.

TOPOGRAPHICAL TERMS OF ACTIVITY AND SELECTIVITY OF TACHYKININ LIGANDS

Anna Minich, M. Chem.

Supported by the State Committee for Scientific Research: grant # 4 P05F 003 08

The mammalian tachykinins: substance P, neurokinin A and neurokinin B interact with at least three types of tachykinin receptors: NK_1, NK_2, NK_3. These compounds possess a variety of biological functions including pain transmission, vasodilation smooth muscle contraction, secretion of saliva, bronchoconstriction activation of immune system and neurogenic inflammation.

There is a perspective, that selective analogues of NK receptors, which possess antagonistic properties can find application as analgesic antiinflammatory and antirheumatic drugs.

Therefore I have studied structure-activity relationships of tachykinins.

In the project, I intend to achieve this goal through molecular modelling and chemical synthesis of model NK-receptor antagonists.

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

Scope and the subject profile:
physiology, neurosciences and experimental surgery, including transplantology.
Present holdings:
books - monographic and serial volumes (Polish and foreign) - 17.317
periodicals, newspapers (number of titles) - 166
Reference aids:
catalogues
- alphabetical: books, periodicals and microfishes
- subject: books
main card-files
- bibliographical list of papers published by scientists of the Medical Research Centre, Polish Academy of Sciences from 1967.
Users:
scientific workers of the MRC, interlibrary loans available for all scientific institutes in Poland and abroad.

Bibliography of library: a list of new books and current periodicals is prepared weekly. On the basis of the Scientific Citation Index a report of citations of papers published by MRC scientists in 1995 was prepared. It contains 554 citations.

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MEDIPAN is a manufacturer of a specialistic equipment for medical service needs. Syringe and volumetric infusion pumps represent the basic assortment of the Plant. They are both constructed and produced there.

In 1995 MEDIPAN continued to carry out intensive development works which have effected in the modernization of the pumps produced so far.

As regards the model 610-2 double-syringe microprocessor infusion pump, constructed and put into production in 1994, the stress was laid on its programming system. Due to some improvements the range of the pump's functional abilities has been considerably enlarged. Furthermore, it can employ now several dozen of different types and volumes of syringes.

Greater number of provisions against the incorrect programming or operation of the pump, as well as the possibility of "conversation" with the pump during the process of its working parameters programming, allow the medical personnel to feel more comfortable in their work and to protect the patients against an accidental infusion. Research works concerning the extension of the possibilities of co-working of model 610-2 syringe pump and model 606 volumetric pump with a computer were carried out. Also the project of a special system of collaboration of infusion pumps with a computer was elaborated.

INTERNATIONAL COOPERATION

VISITING SCIENTISTS

Department of Neurochemistry

Mario Alberghina	Department of Biochemistry, University of Cathania, Italy
Hanna Pappius	Neurological Institute, McGill University, Montreal, Canada

Department of Neuropathology

Igor B. Barskov	Brain Institute, Medical Academy of Sciences, Moscow, Russia
Lia Nikolaishvili	I.S. Beritashvili Institute of Physiology, Tbilisi, Georgia (long term visit)
Roger F. Butterworth	Neuroscience Reserch Unit, McGill University Montreal, Canada

Department of Neurosurgery

Siergiej Jeoczijan Valery Łoszakov	Burdenko Institute of Neurosurgery, Medical Academy of Sciences, Moscow, Russia
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Nina Kuridze	I.S. Beritashvili Institute of Physiology, Tbilisi, Georgia (long term visit)

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Department of Endocrinology

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Gabriel Eilon	Hitachi Chemistry Research Centre, Irvine, USA

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	Department of Pharmacology and Toxicology, National Centre of Sciences Research, Habana, Cuba
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Department of Neurochemistry

Krystyna Domańska-Janik	Laboratory of Experimental Brain Research, University of Lund, Sweden
	Institute of Experimental Neuropathology, Austrian Academy of Sciences, Vienna, Austria
Jerzy W. Łazarewicz	Institute of Anatomy and Cell Biology, University of Göteborg, Sweden
Joanna Strosznajder	Institute of Biochemistry, University of Catania, Italy
	Institute for Chemistry Research, University of Kyoto, Japan
	Faculty of Pharmaceutical Sciences, Teikyo University, Tokyo, Japan

Teresa Zalewska	Department of Pharmacology, University of Arizona, Tucson, Arizona, USA (long term visit)

Department of Neuropathology

Hanna Borkowska	International Graduate School in Neurosciences, University of Tampere, Finland (long term visit)
Lidia Faff	Max-Delbrück Centre for Molecular Medicine, Berlin, Germany (long term visit)
Elżbieta Kida	Institute for Basic Research in Developmental Disabilities, New York, USA (long term visit)
Stanisław Krajewski	La Jolla Cancer Research Foundation, Cancer Research Centre, La Jolla, USA (long term visit)

Laboratory of Developmental Neuropathology

Maria Dąmbska	Institute for Basic Research in Developmental Disabilities, New York, USA (long term visit)

Laboratory of the Ultrastructure of the Nervous System

Barbara Gajkowska	Laboratoire de Biologie et Ultrastructure de Noyau de l'Institute de Recherches Scientifiques sur le Cancer, Villejuif, France

Department of Neurosurgery

Stefan Piechnik	Cambridge Centre of Brain Repair, University of Cambridge, United Kingdom (long term visit)

Neuromuscular Unit

Małgorzata Kozłowska	Department of Human Genetics, Faculty of Medicine, University of Newcastle, United Kingdom (long term visit)

Department of Applied Physiology

Leszek Budohoski	Department of Biochemistry, University of Oxford, United Kingdom (twice)
Jolanta Chwalbińska-Moneta	Department of Clinical Physiology, Karolinska Institute, Huddinge, Sweden
Gerard Cybulski	Centre for Biological and Medical Systems of Imperial College, London, United Kingdom (long term visit)
Ryszard Grucza	Department of Physiology, University of Kuopio, Finland
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Barbara Kruk	Department of Physiology, University of Kuopio, Finland

Józef Langfort	Department of Medical Physiology, The Panum Institute, University of Copenhagen, Denmark
Krystyna Nazar	Department of Physiology and Pharmacology, School of Medicine, University of Nottingham, United Kingdom
	Department of Biochemistry, University of Oxford, United Kingdom
	Laboratory of Physiology CNRS, University of Lyon, France
	Department of Physiology, College of Medicine, State University of New York, Syracuse, N.Y., USA

Department of Surgical Research and Transplantation

Irena Grzelak	Norwegian Radium Hospital, Oslo, Norway (twice)
Urszula Kubicka	Indian National Science Academy, Madras, Pondicherry, India (twice)
Barbara Łukomska	Centre National de Recherche Scientifique, Bordeaux, France (twice)
Waldemar L. Olszewski	St. Bartolomew's Medical School, London, United Kingdom (twice)
	Indian National Science Academy, Madras, Pondicherry, India (twice)
	Norwegian Radium Hospital, Oslo, Norway

Iwona Tyszer	Indian National Science Academy, Madras, Pondicherry, India (twice)
Marzenna Zaleska	Indian National Science Academy, Madras, Pondicherry, India (twice)

Department of Endocrinology

Tomasz Bednarczuk	Thyroid Eye Research Program, Allagheny-Singer Research Institute, Pittsburgh, USA (long term visit)
Andrzej Lipkowski	Massachusetts General Hospital, Harvard Medical School, Boston, USA
	FERRING Research Institute, Malmö, Sweden
Aleksandra Misicka	FERRING Research Institute, Malmö, Sweden

PARTICIPATION IN INTERNATIONAL MEETINGS

One Day Symposium on Advances in Lymphology, Taramani, Madras, India, February 25, 1995: *U. Kubicka, W.L. Olszewski*

IEE Colloquium on Signal Processing in Cardiography, London, UK, March 1, 1995: *G. Cybulski*

First Congress of the European Pediatric Neurology Society (EPNS), Eilat, Israel, March 19-23, 1995: *A. Fidziańska, I. Hausmanowa-Petrusewicz, A. Kamińska*

International Council of Military Sports: Symposium 1995 "Physical Fitness, Sport and Health in the Armed Forces", Warsaw, Poland, March 20-24, 1995: *J. Chwalbińska-Moneta*

"British-Polish Science Day", British Council, London, UK, March 29, 1995: *M.J. Mossakowski*

International Scientific Advisory Committee for the "Evaluation Program for Reminyl (sabeluzole) in Alzheimer's Disease", New York, USA, March 30, 1995: *M. Barcikowska*

2nd Neuroscience Conference in Memory of Doctor Lenas Katikouras, Thessaloniki, Nicosia, Cyprus, March 31 - April 1, 1995: *I. Hausmanowa-Petrusewicz*

2nd Israeli-Polish Peptide Symposium, Rehovot, Israel, April 4-11, 1995: *A. Lipkowski, A. Misicka*

Meeting of the German Cardiac Society, Mannheim, Germany, April 19-23, 1995: *K. Cedro-Ceremużyńska*

Meeting of the Hungarian Society of Cardiology, Budapest, Hungary, May 2-7, 1995: *K. Cedro-Ceremużyńska*

XXX Congress of the European Society for Surgical Research, Amsterdam, The Netherlands, May 10-13, 1995: *M. Maksymowicz, W.L. Olszewski*

International Conference on Neurocutaneous Syndrome (Phacomathosis), Warsaw, Poland, May 12-13, 1995: *M. Dąmbska, I. Kuchna*

2nd International Workshop on "Thyroid Hormone Resistance", Padua, Italy, May 12-13, 1995: *T. Bednarczuk*

3rd International Congress on "Mechanisms and Management of Heart Failure", Geneva, Switzerland, May 20-21, 1995: *K. Cedro-Ceremużyńska*

"Tumor Microenvironment - Progression, Therapy and Prevention", Tiberias, Israel, May 14-18, 1995: *U. Kubicka, B. Łukomska, W.L. Olszewski*

Polish-Israeli Neurological Conference, Warsaw, May 17-18, 1995: *J. Albrecht, M. Barcikowska, I. Hausmanowa-Petrusewicz, E. Salińska, J. Strosznajder, J. Sypecka*

International Symposium "Frontiers in Opioid Research and Application", Warsaw, May 17-19, 1995: *I. Maszczyńska, A. Misicka, B. Zabłocka, T. Zalewska*

International Symposium on Cold and Heat Stress in Humans, Tatranske Matliare, Slovakia, May 18-21, 1995: *H. Kaciuba-Uściłko, K. Nazar*

International Symposium on Neuromuscular Diseases in honor of Professor Irena Hausmanowa-Petrusewicz, Warsaw, Poland, May 25-26, 1995: *H. Drac, A. Fidziańska, A. Kamińska, M.J. Mossakowski, I. Niebrój-Dobosz, J. Rafałowska, K. Rowińska, H. Strugalska, E. Zalewska*

The Symposium on Adrenergic Systems 100 Years after Discovery of Adrenaline, Cracow, Poland, May 26, 1995: *K. Cedro-Ceremużyńska, J. Langfort, M. Pokorski, E. Wojtal*

XXth Meeting of the European Group of Lymphology, Titisee, Germany, May 26-27, 1995: *W.L. Olszewski*

IV International Symposium on "Molecular and Physiological Aspects of the Systemic Regulation", Cracow, Poland, June 6-7, 1995: *B. Gajkowska, M.J. Mossakowski*

Joint Third Central European FACS/CAS Users' Meeting and Fifth CEQUAL Sendaround Meeting, Balatonfüred, Hungary, June 12-14, 1995: *W.L. Olszewski*

Meeting on "Molecular, Cellular and Clinical Aspects of Angiogenesis", Nato Advanced Study Institute, Porto Carras, Halkidiki, Greece, June 16-27, 1995: *G. Szczęsny*

Fifth Meeting of the European Neurological Society, Munich, Germany, June 17-21, 1995: *I. Hausmanowa-Petrusewicz, R. Pluta*

14th American Peptide Symposium, Columbus, Ohio, USA, June 18-23, 1995: *A. Lipkowski, I. Maszczyńska, A. Misicka*

II World Congress of Medical Polonia, Częstochowa, Poland, June 20-24, 1995: *J. Jurkiewicz*

Symposium: "Moderne Verfahren zur Optimirung der Hirntumorbehandlung", Munich, Germany, June 22-24, 1995: *J. Andrychowski, J. Bogucki, Z. Czernicki*

16th International Lectin Meeting "Interlac 16", Toulouse, France, June 26-30, 1995: G. Szumańska

Advanced School of Neurochemistry, 2nd Biennial Course on "From Signal Transduction to Gene Expression", Okazaki, Japan, June 28 - July 2, 1995: *R. Strosznajder*

XVII International Symposium on Cerebral Blood Flow and Metabolism, Cologne, Germany, July 2-6, 1995: *R. Pluta*

15th Biennial Meeting of International Society for Neurochemistry, Kyoto, Japan, July 2-7, 1995: *H. Borkowska, M. Samochocki, J. Strosznajder*

3rd Joint German-Polish Neurosurgical Symposium "Traumatic Lesions of the CNS", Seeheim-Jungenheim, Germany, July 6-8, 1995: *J. Andrychowski, Z. Czernicki, P. Marszalek*

Fourth IBRO World Congress of Neuroscience, Kyoto, Japan, July 9-14, 1995: *L. Faff, M. Samochocki, J. Strosznajder*

3rd International Neurotrauma Symposium, Toronto, Canada, July 22-27, 1995: *Z. Czernicki*

9th International Congress of Immunology, San Francisco, USA, July 23-29, 1995: *S. Durowicz*

4th International Congress on Amino Acids, Vienna, Austria, August 7-11, 1995: *J. Albrecht, W. Hilgier*

23rd Meeting of the Federation of the European Biochemical Society, Basel, Switzerland, August 13-18, 1995: *M. Chalimoniuk*

13e Congres Francais d'Endocrinologie, Nancy, France, August 31 - September 2, 1995: *B. Gajkowska*

The 11th European Congress of Multiple Sclerosis of the European Committee for Treatment and Research in Multiple Sclerosis (ECTRIMS), Jerusalem, Israel, September 3-6, 1995: *P. Grieb*

The Meeting of European Neuroscience, Amsterdam, The Netherlands, September 3-7, 1995: *M. Chalimoniuk, B. Dąbrowska-Bouta, B. Gajkowska, W. Gordon-Krajcer, I. Koładkiewicz, M. Pokorski, L. Strużyńska, E. Salińska, J. Sypecka, M. Śmiałek*

XV European Congress of Pathology, Copenhagen, Denmark, September 3-8, 1995: *U. Kubicka*

3rd Symposium of Poland-Japan Society for Exchange in Surgery "Tumors, transplants, infections - surgical and immunological aspects", Warsaw, Poland, September 4, 1995: *Z. Czernicki, J. Jurkiewicz, M.J. Mossakowski, W.L. Olszewski*

Symposium "Peptidomimetics. Design, Synthesis and Structure", Spa, Belgium, September 4-8, 1995: *A. Lipkowski, A. Misicka*

Symposium on "Cerebral Vasospasm: New Aspects on Pathomechanism and Therapy", Giessen, Germany, September 8-9, 1995: *J. Andrychowski*

First International Symposium "Müller Cells: Metabolism, Membrane Transport and Cellular Interactions", Leipzig, Germany, September 8-10, 1995: *J. Albrecht, L. Faff*

First FEPS Congress, Maastricht, The Netherlands, September 9-12, 1995: W. Janczewski

1995 Autumn School "Microneurography in Cognitive Neuroscience", San Feliu de Guixols, Spain, September 9-14, 1995: *U. Jernajczyk*

First Congress of the European Federation of Neurological Societies, Marseille, France, September 9-14, 1995: *M. Barcikowska, I. Hausmanowa-Petrusewicz*

International Conference "Computers in Cardiology", Vienna, Austria, September 10-13, 1995: *G. Cybulski, W. Niewiadomski*

Second International Congress of Polish Neuroscience Society, Cracow, Poland, September 13-16, 1995: *J. Albrecht, M. Barcikowska, H. Borkowska, K. Budzińska, M. Chalimoniuk, B. Dabrowska-Bouta, M. Dąmbska, K. Domańska-Janik, L. Faff, W. Gordon-Krajcer, I. Koładkiewicz, J. Krzysztoń, I. Kuchna, A. Lipkowski, M. Łałowski, J. Łazarewicz, E. Łuczywek, D. Maślińska, M.J. Mossakowski,*

A. Pfeffer-Baczuk, R. Pluta, M. Pokorski, E. Salińska, M. Samochocki, A. Stafiej, J. Strosznajder, L. Strużyńska, J. Sypecka, J. Waśkiewicz, E. Wojtal, B. Zabłocka, T. Zalewska, E. Ziemińska

Association of European Young Medical Scientists "Minimal Invasive Surgery", Gdańsk, September 14-16, 1995: *W.L. Olszewski*

Annual Congress of the European Respiratory Society, Barcelona, Spain, September 16-20, 1995: *K. Budzińska, W. Janczewski, U. Jernajczyk, M. Pokorski, M. Szereda-Przestaszewska*

MEDICON'95, VII Mediterranean Conference on Medical and Biological Engineering, Jerusalem, Israel, September 17-21, 1995: *G. Cybulski*

Seventh International Helminthological Symposium, Helminth-Helminthoses-Environment, Kosice, Slovak Republic, September 19-22, 1995: *M. Walski*

European Brain Bank Network for Neurobiological Studies in Neurology and Psychiatric Disorders (EBBN), Plenary Meeting "Brain Research and Society", Barcelona, Spain, September 21-24, 1995: *H. Kroh*

XV International Congress of Lymphology, Sao Paulo, Brazil, September 25-26, 1995: *H. Gałkowska, B. Łukomska, W.L. Olszewski*

Symposium "New Frontiers in Cell and Molecular Biology" organized under personal auspices of the Director General of UNESCO, Federico Mayor Zaragoza, Warsaw, Poland, October 2-7, 1995: *R. Strosznajder*

IV International Congress of Phlebology and Lymphology, Buenos Aires, Argentina, October 3-7, 1995: *B. Łukomska, W.L. Olszewski*

7th Congress of the Polish Society for Organ Transplantation (ESOT'95), Vienna, Austria, October 3-7, 1995: *M. Maksymowicz*

"Gamma Knife Radiosurgery", Warsaw, Poland, October 14, 1995: *D. Horsztyński, P. Marszałek*

The X International Congress of Electromyography and Clinical Neurophysiology, Kyoto, Japan, October 15-19, 1995: *I. Hausmanowa-Petrusewicz, K. Rowińska-Marcińska*

3rd International Conference on Stroke "Heart and Brain", Prague, Czech Republic, October 18-21, 1995: *R. Pluta*

36th European Neuromuscular Centre (ENMC) International Workshop "Familial Desmin-Related Myopathies and Cardiomyopathies - From Myophathology to Molecular and Clinical Genetics", Naarden, The Netherlands, October 20-22, 1995: *A. Fidziańska-Dolot, A. Kamińska*

7th Kongress der Gesellschaft Deutschsprachigen Lymphologen "Lymphologica 95", Bochum, Germany, October 27-28, 1995: *W.L. Olszewski*

International Conference "Dehydration, Rehydration and Exercise in the Heat", Nottingham, UK, November 1-5, 1995: *H. Kaciuba-Uścilko, K. Nazar*

International Conference "From Snoring to Sleep Apnea Syndrome: Therapeutic Approach", Lyon, France, November 9-10, 1995: *W. Jamczewski*

2nd East-West European Congress of Angiology "Cerebral Revascularisation", Warsaw, Poland December 7-8, 1995: *Z. Czernicki, P. Marszałek, W. Sapieja*

38th European Neuromuscular Centre (ENMC) International Workshop: Spinal Muscular Atrophy Trial Group, Naarden, The Netherlands, December 10-12, 1995: *I. Hausmanowa-Petrusewicz*

The 5th International Conference on Non-Invasive Cardiology, Tel Aviv, Israel, December 17-21, 1995: *E. Wójcik-Ziółkowska*

SCIENTIFIC DEGREES

DOCTOR'S DEGREE

Magdalena Jaskłowska-Englisz

The effect of donor-specific blood transfusion on survival of intravenous lymphocyte graft

Department of Surgical Research and Transplantation

Anna Maria Noszczyk

Effect of retinoids on the molecular mechanisms of skin ageing

on leave of absence to the Department of Dermatology, Medical School in Warsaw

Robert Strosznajder

Phosphoinositol breakdown in the mechanism of carotid chemireceptor transduction

Department of Neurophysiology

SCIENTIFIC MEETINGS

ORGANIZED BY THE MEDICAL RESEARCH CENTRE

International Symposium "Frontiers in Opioid Research and Application", Warsaw, May 17-19, 1995.

Symposium was held at new facilities of Medical Research Center, Polish Academy of Sciences in Warsaw. It was sponsored by Medical Research Centre, under the joint auspicies of Polish Scientific Committee (KBN) and Department of Anesthesiology, New England Medical Center, Boston, USA. The object of the symposium was to illustrate the various aspects of opioid research and to show how different approaches are integrated together in this field. More than hundred scientists working with different aspects of opioids participated in the symposium. The program comprised twenty main lectures, given by distinguished scientists, the poster session, and the round table session. The program covered a variety of topics related to opioid research, from basic chemical synthetic and modelling study, through pharmacological study of structure-activity relationships, to medical application in anesthesiology. The abstracts of the symposium were published in a new medical journal "Medical Science Monitor". Selected papers will be published in full in international journal "Analgesia".

3rd Symposium of Poland-Japan Society for Exchange in Surgery: "Tumors, Transplants, Infections - Surgical and Immunological Aspects", Warsaw, Poland, September 4-6, 1995.

The main subjects of the Symposium were immunology of gastrointestinal tumors, transplants and infections. An important achievement of Japanise scientists was presentation of laparoscopic surgery of stomach and large bowel tumors, based on topographical and immunohistochemical studies, while of Polish scientists - pointing to the role of immune cells in local immune reactions to tumor antigens. Totally, 55 participants took part in the Symposium (18 from abroad). They presented 24 papers (1 from MRC).

Symposium: "Cytokines in Inflammatory Processes, Tissue Injury, Anti--Tumor Reactions, Rejection of Allografts", Warsaw, Poland, March 24, 1995.

Symposium was organized by the Department of Surgical Research and Transplantation, Medical Research Centre, Polish Academy of Sciences. The number of participants was 129 (1 from abroad). The number of presentations was 17 (2 presented by scientists from MRC).

Teaching Conference: "Treatment of CSF Flow Disturbances", Pułtusk, Poland, March 30-31, 1995.

Teaching Conference was organized by the Department of Neurosurgery Medical Research Centre, Polish Academy of Sciences, and the Commission of Intracranial Pressure and Cerebral Hemodynamic Disturbances of PASci. The Conference had a workshop character connected with round table discussion. Four discussion sessions were organized. 100 national and 12 participants from abroad attended the Conference.

Scientific Conference: "Current situation of ICP monitoring, new methods and trends", Wrocław, Poland, September 21-24, 1995.

Scientific Conference was organized together with the Polish Neurosurgical Society Board as an independent session of the Polish Neurosurgical Society Symposium on the 30th anniversary of the Wrocław Medical School Neurosurgery Clinic. Fourty national and 7 participants from abroad attended the Conference. There were 10 papers presented.

PUBLICATIONS

1. Barcikowska M: Neuropathology of aging brain. *Folia Med Lodziensis* 1995, 22, 55-72 (in Polish).

2. Barcikowska M: Significance of Meynerts's nucleus and its connections with temporal lobe cortex in Alzheimer's disease. In: *2nd Spring School in Neurobiology on: Temporal Lobes - Morphology, Functions and their Disorders*. Eds: A Grabowska, K Kosmal, DM Kowalska. IBO, Warsaw, 1995, pp. 27-33 (in Polish).

3. Barcikowska M, Chodakowska A, Klimowicz I, Liberski PP: A case of radionecrosis mimicking metastatic tumor of the cerebral hemisphere. *Folia Neuropathol* 1995, 33, 55-57.

4. Barcikowska M, Kida E: Ferritin-positive microglia in Alzheimer's and Parkinson's disease brain. *Folia Med Lodziensis* 1995, 22, 141-153.

5. Barcikowska M, Kwieciński H, Liberski PP, Kowalski J, Brown P, Gajdusek DC: Creutzfeldt-Jakob disease with Alzheimer type Aβ-reactive amyloid plaques. *Histopathology* 1995, 26, 445-450.

6. Bednarczuk T, Nauman A, Januszewski S: Type II iodothyronine 5'-deiodinase activity in brain regions of adult rats, following experimentally induced complete cerebral ischemia. *Pol J Endocrinol* 1995, 46, 181-189.

7. Bielecki K, Włodarczyk A, Durowicz S: Selected difficult problems in large bowel surgery. In: Ogilve Syndrome. Ed.: K Bielecki. CMKP, Warsaw, 1995, pp. 17-26 (in Polish).

8. Boręsewicz A, Karwatowska-Prokopczuk E, Lewartowski B, Herbaczyńska-Cedro K: A protective role of nitric oxide in isolated ischaemic reperfused rat heart. *Cardiovasc Res* 1995, 30, 1001-1008.

9. Brzustowicz LM, Wang CH, Matseoane D, Kleyn PW, Vitale E, Das K, Penchaszadeh GK, Munsat TL, Hausmanowa-Petrusewicz I, Gilliam TC: Linkage disequilibrium and haplotype analysis among Polish families with Spinal Muscular Atrophy. *Am J Hum Genet* 1995, 56, 210-215.

10. Budzińska K, Romaniuk JR: The role of raphe and tractus solitarius neuronal structures in the modulation of respiratory pattern in rabbits. *Acta Neurobiol Exp* 1995, 55, 155-164.

11. Chomicki J, Jurkiewicz J, Zabołotny W, Czernicki Z, Cervos-Navarro J: Effect of dotarizine on CO_2-dependent cerebrovascular reactivity. *Acta Neurochir (Wien)* 1995, 136, 186-188.

12. Czarnowski D, Langfort J, Pilis W, Górski J: Effect of a low-carbohydrate diet on plasma and sweat ammonia concentration during prolonged nonexhausting exercise. *Eur J Appl Physiol* 1995, 70, 70-74.

13. Czernicki Z, Berdyga J, Stepińska G, Jurkiewicz J: Evaluation of craniospinal system condition using standarized volume-pressure loadings. *Acta Neurochir (Wien)* 1995, 133, 191-194.

14. Czernicki Z, Suzuki R, Nakagawa K, Hirakawa K, Endo S: Acetazolamide produced blood flow velocity changes measured by laser Doppler in gerbils with reduced CBF. *Acta Neurochir (Wien)* 1995, 138, 81-83.

15. Dąmbska M: The vascularization of the developing human brain. *Folia Neuropathol* 1995, 33 (4), 189-193.

16. Dąmbska M, Maślińska D, Muzylak M: The features of peripheral nerve lesion in young and adult rabbits after vincristine administration. *Folia Neuropathol* 1995, 33, 21-24.

17. Dębicki G, Ryba M, Grieb P, Walski M, Januszewski S: Will the limitation of the free-radical damage improve the effects of reanimation? *Medical Science Monitor* 1995, 1, (1), 13-15 (in Polish).

18. Dębicki G, Ryba M, Grieb P, Walski M, Januszewski S: An experimental model of acute respiratory failure, reanimation and post-resuscitation syndrome. *Medical Science Monitor* 1995, 1, 211-214 (in Polish).

19. Dobrowolski L, Kompanowska-Jezierska E, Sadowski J: Modulation of renal medullary ionic hypertonicity by prostaglandins: data from tissue admitance studies in the rat. *J Physiol (London)* 1995, 485, 827-834.

20. Dobrzyński B, Kaczorowski K, Lewicki R, Golińska A, Nazar K: Assessment of physical fitness and capacity in boys and girls. In: *Current Problems of Sports in Children and Adolescents*. Warsaw, 1995, pp. 261-265 (in Polish).

21. Dobrzyński B, Kaczorowski K, Lewicki R, Nazar K: Maximal anaerobic power in trained and untrained boys and girls. In: *Current Problems of Sports in Children and Adolescents*. Warsaw, 1995, pp. 255-258 (in Polish).

22. Dobrzyński B, Kaczorowski K, Lewicki R, Nazar K: Physical fitness and capacity in boys aged 15-19 yrs training ski running. In: *Current Problems of Sports in Children and Adolescents*. Warsaw, 1995, pp. 258-261 (in Polish).

23. Domańska-Janik K, Zabłocka B: Modulation of signal transduction in rat synaptoneurosomes by platelet-activating factor. *Mol Chem Neuropathol* 1995, 25, 51-67.

24. Durlik M, Łukomska B, Cybulska E, Olszewski WL: Cyclosporine changes the rejection pattern of different tissues in vascularized hind limb allograft. *Polish J Immunol* 1995, 20, 365-367.

25. Durowicz S, Sadowska-Ryffa D, Cybulska E, Wojewódzka U, Olszewski WL: Liver lymphocytes and their role in immune reactions. *Polish J Immunol* 1995, 20, 369-373.

26. Dziduszko J, Horsztyński D: Clinical usefulness of somatosensory evoked potentials in evaluation of cervical discopathia. *Reumatologia* 1995, 33, 1, 35-42 (in Polish).

27. Fersten E: Temporal lobe participation in language and perceptive functions. In: *2nd Spring School in Neurobiology on: Temporal Lobes - Morphology, Functions and their Disorders*. Eds: A Grabowska, K Kosmal, DM Kowalska. IBO, Warsaw, 1995, pp. 107-116 (in Polish).

28. Fersten E, Szatkowska I, Łuczywek E, Herman A, Grabowska A: The influence of medial temporal structures lesion on the sensoric information memory. *Studia Psychologiczne* 1995, 32, 79-94 (in Polish).

29. Fidziańska A, Kamińska A: Neural cell adhesion molecule (N-CAM) as a marker of muscle tissue alterations. *Folia Neuropathol* 1995, 33, 126-128.

30. Fidziańska A, Morrone A, Pergoraro E, Ryniewicz B, Ilnicka A, Zammarchi E, Hoffman EP: An X autosome translocation stabilizes truncated dystrophin: implications for lack of truncated dystrophins in Duchenne muscular dystrophy. *Neuropediatrics* 1995, 26, 3, 163-167.

31. Fidziańska A, Ryniewicz B, Barcikowska M, Goebel HH: A new familial congenital myopathy in children with desmin and dystrophin reacting plaques. *J Neurol Sci* 1995, 131, 88-95.

32. Gajkowska B, Gadamski R, Mossakowski M: Immuno-electron microscopic demonstration of GABA and glutamate synapses in Mongolian gerbils hippocampus after ischemia. *Folia Neuropathol* 1995, 33, 5-19.

33. Gajkowska B, Gajewska A, Dul B, Kochman K: Immunocytochemical study of preoptic area LHRH-synthetizing neurons in the young and old female rat. *Neuroendocrinol Lett* 1995, 17, 4, 265-270.

34. Gajkowska B, Mossakowski MJ: Effect of ischemia on the serotonin neuron system in rat hippocampus: an immunocytochemical study. *Neuroendocrinol Lett* 1995, 17, 57-62.

35. Gajkowska B, Mossakowski MJ: Localization of endothelin in the blood-brain interphase in rat hippocampus after global cerebral ischemia. *Folia Neuropathol* 1995, 33, 221-230.

36. Gajkowska B, Śmiałek M, Porada D, Piotrowski P: Neurotoxic effect of sodium tellurite in the rat temporal lobe. Rapid communication. *Acta Neurobiol Exp* 1995, 55, 221.

37. Gajkowska B, Śmiałek M, Porada D, Piotrowski P: Evaluation of neurotoxic action of sodium tellurite on the temporal lobe structures in the rat. In: *2nd Spring School in Neurobiology on: Temporal Lobes - Morphology, Functions and their Disorders*. Eds: A Grabowska, K Kosmal, DM Kowalska. IBO, Warsaw, 1995, pp. 107-116 (in Polish).

38. Gajkowska B, Viron A: 5-hydroxytryptamine in hypothalamo-neurohypophysial system after ischemia: an immunogold study on the rat. *Neuroendocrinol Lett* 1995, 17, 3, 221-226.

39. Gałkowska H, Olszewski WL, Wojewódzka U: Involvement of cytokines and adherence molecules in skin immune cell cooperation - implications for transplantation. *Polish J Immunol* 1995, 20, 353-357.

40. Gałkowska H, Wojewódzka U, Olszewski WL: Factors affecting spontaneous dendritic cell-lymphocyte clustering in skin afferent lymph. In: *Dendritic Cells in Fundamental and Clinical Immunology*. Eds: Banchereau and Schmitt. Plenum Press Publ. Corp., *Advances in Experimental Medicine and Biology*, 1995, 378 (2), pp. 405-407.

41. Gałkowska H, Wojewódzka U, Olszewski WL: Cytokines and adherence molecules involved in spontaneous dendritic cell-lymphocyte clustering in skin afferent lymph. *Scand J Immunol* 1995, 42, 324-330.

42. Garcia-Barcina M, Łukomska B, Gawron W, Winnock M, Vidal-Vanaclocha F, Bioulac-Sage P, Balabaud C, Olszewski WL: Expression of cell adhesion molecules on liver-associated lymphocytes and their counter-receptors on sinusoidal lining cells in patients with benign or malignant liver disease. *Am J Pathol* 1995, 146, 1406-1413.

43. Gołębiowski M, Barcikowska M, Pfeffer A: The usefulness of CT scanning in clinical observation of the patients with Alzheimer's disease. *Polski Przegląd Radiologiczny* 1995, 60, 61-64 (in Polish)

44. Greenleaf JE, Kruk B, Nazar K, Falęcka-Wieczorek I, Kaciuba-Uścikko H: Glucose infusion into exercising dogs after confinement: rectal and active muscle temperatures. *Aviat Space Environ Med* 1995, 66, 1169-1174.

45. Grieb P, Skierski J, Jagielski J, Ryba M, Mossakowski MJ: DNA-protein flow cytometry of dissociated cultures of human anaplastic gliomas. Pattern of proliferation and differentiation, and the effect of a new cytostatic drug cladribine (2-CdA). *Folia Neuropathol* 1995, 33, 145-152.

46. Grieb P, Stelmasiak Z: Treatment of multiple sclerosis with a new immunosuppressant cladribine (2-CdA). *Neurol Neurochir Pol* 1995, 1, 69-76 (in Polish).

47. Grieb P, Stelmasiak Z, Solski J, Nowicki J, Jakubowska B, Ryba M: The effect of repeated treatments with cladribine (2-chlorodeoxyadenosine) on blood counts in multiple sclerosis patients. *Arch Immunol Ther Exp* 1995, 43, 323-327.

48. Grochowicz PM, Hibberd AD, Bowen KK, Clark DA, Willenborg DO, Cowden WB: Castanospermine alone and in combination with CsA - effect on heart allograft survival, synergism and tolerance induction. *Polish J Immunol* 1995, 20, 349-351.

49. Grzelak I, Zaleska M, Olszewski WL: The effect of allogeneic blood transfusions on hemopoiesis and immune responsiveness. *Polish J Immunol* 1995, 20, 359-363.

50. Hausmanowa-Petrusewicz I, Błaszczyk M, Jabłońska S: Coexistence of scleromyositis associated with PM-Scl antibody and myasthenia. *Neuromusc Disord* 1995, 5, 2, 145-147.

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