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MEDICAL RESEARCH CENTRE

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CONTINUATION OF THE STUDIES ON THE FUNCTION OF CAROTID BODIES AND ON THE ROLE OF VAGAL C FIBERS IN REFLEX APNOEA

Prof. Mieczysław Pokorski

The studies on the effects on the hypoxic respiratory response and the carotid body ultrastructure of the phospholipase C inhibitor phenylmethylsulphonyl fluoride (PMSF) were continued in anesthetized cats. PMSF was administered intraperitoneally. The experiments were carried out before and in defined time periods after the administration of PMSF. Double-control experiments were performed on non-injected and medium-injected cats. The results demonstrate that the stimulatory hypoxic respiratory response was abolished 7 days after the PMSF injection. This was accompanied by profound degenerative changes in the carotid body parenchyma, most notable in the chemoreceptor cells. These results indicate that inhibition of phospholipase C has a profound inhibitory effect on carotid body function. It is concluded that degeneration of phosphoinositols is involved with the transduction of a hypoxic signal in the carotid body. (M. Pokorski)

The occurrence of prompt and constant apnoea induced by capsaicin administration into the right side of circulation in cats, as well as the depression of tidal volume in resumed breathing, rely on preserved vagal pathways. Capsaicin challenge to the left ventricle or to the common carotid artery evokes variable apnoeic spells occurring independently of reserved or damaged vagal feedback. Chemoreflex provoked by intracarotid capsaicin injection is coupled with hyperventilatory response, undergoing beyond the vagal loop. (M. Szereda-Przestaszewska and B. Wypych)

In the follow up studies on the role of ATP in phospholipids degeneration by phospholipase C, the influence of this nucleotide as a potential regulator of phospholipase activity in the brain cortex was tested. The results indicate that ATP acting extracellularly through a purinergic receptor(s) activates PtdIns $(4,5)P_2$ degradation and release of $Ins(1,4,5)P_3$. ATP acting directly on PLC inhibits in a receptor-independent manner phosphoinositide degradation and protects against liberation of lipid-derived second messengers. (R. Strosznajder)

STUDY OF NEURAL AND NEUROCHEMICAL MECHANISMS OF RESPIRATORY PATTERN CONTROL

Dr. Krystyna Budzińska

Study was undertaken to investigate the role of phospholipids pathway in short term potentiation (STP) of hypoglossal nerve activity evoked by stimulation of superior laryngeal nerve. This phenomenon consists in persistence of the effect of stimulation during some time after cessation of stimulation. In the experiments on rabbits a precursor of phospholipids, Citicoline, was used and the effects of SLN stimulation before and after Citicoline treatment were studied. It was found that Citicoline increases both the induction and maintenance of potentiation of hypoglossal nerve activity due to superior laryngeal nerve stimulation. It suggests that phospholipids pathway plays a role in modulation of short-term potentiation of hypoglossal nerve activity. The mechanism of respiratory STP differs from that of hippocampal LTP. (K. Budzińska and E. Wojtal)

A degree of compensation after gradual and entire denervation of the diaphragm muscle in the respiratory system was studied. The experiments were performed on anesthetized and spontaneously breathing cats. Parameters of ventilation, activities of the both phrenic nerves and EMG of the diaphragm were evaluated. It was found that unilateral phrenicotomy was well compensated by non-paralysed part of the diaphragm muscle. While after the paralysis of diaphragm (the dominant of the respiratory muscle), efficiency of the respiratory system was diminished. This was expressed in a decrease the minute ventilation and augmentation the respiratory drive. (B. Sokołowska)

Changes in the inflation Hering-Breuer reflex following pharmacological blockade of the nucleus trigemini sensibilis: Experiments were performed in anesthetized, paralyzed, unilaterally vagotomized, and artificially ventilated rabbits. Tracheal pressure, expiratory carbon dioxide concentrate, and integrated activity of the phrenic nerve and medullary respiratory neurons were recorded. Before and after the blockade of the nucleus trigemini sensibilis with 2 μl polocaine injection, the inflation Hering-Breuer reflex was induced by clamping the trachea at the peak of inflation. Before the blockade of the nucleus trigemini sensibilis, average expiratory time following the trachea clamping increased by 120% compared with control expiration. After the blockade, average expiratory time following the trachea clamping increased by 304% compared with its respective control. This large increase in the duration of expiration following the blockade of the nucleus trigemini sensibilis speaks for the contribution of this structure to the inhibition of expiration. (H. Gromysz)

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

Dr. Robert Strosznajder

Supported by the State Committee for Scientific Research: grant # 6 P207 029 05

The results of this study demonstrate that hypoxia caused a significant decrease in incorporation of [1-^{14}C] AA specifically into PtdIns. Hypoxia had no significant effect on the level of AA radioactivity in DAG as compared to control but significantly enhanced the level of AA-CoA radioactivity. In the normoxic SCG, DA significantly decreased AA incorporation into PtdCh, PtdIns and DAG. Moreover, DA decreased the level of AA-CoA radioactivity. Dopamine had no effect on AA incorporation into phospholipids of hypoxic SCG.

ARACHIDONATE METABOLISM IN THE BRAIN

Dr. Robert Strosznajder

The results of this study indicate that: (1) arachidonate is able to cross at a relevant rate BRB and BBB; (2) in all brain regions except retina, optic tract and hippocampus, blood barriers have a transport capacity for AA significantly higher than that for docosahexaenoate and palmitate; (3) aging does not affect influx into retina and other structures of rat central nervous system of the arachidonate.

ROLE OF THE LARYNGEAL VALVULAR MECHANISM AND OF THE THORACIC EXPIRATORY MUSCLES IN REFLEX APNOEA EVOKED BY STIMULATION OF CHEMOSENSITIVE LUNG C-FIBRES

Beata Wypych

Supported by the State Committee for Scientific Research: grant # 4 P05A 096 10

Serotonin is an endogenous autacoid contained within the lungs. When injected into the right side of circulation in cats evokes pulmonary chemoreflex. Its respiratory component consists of prompt apnoea, followed by rapid shallow breathing. Arrest of the respiratory airflow during apnoea is coupled with simultaneous inhibition of inspiratory and expiratory motoneurones of the respiratory muscles.

The reported experiments have shown that during post-serotonin apnoea the time of electrical silence in the phrenic neurogram (inspiratory activity) corresponded exactly with the duration of apnoea. In resumed breathing peak neural activity of the phrenic nerve was apparently increased.

Electrical activity of the expiratory muscles of the rib cage, namely: triangularis sterni and internal intercostal was inhibited during apnoea, phrenic alike. With renewed respiratory movements electrical discharges of both muscles appeared to

be decreased, compared to the baseline values. This generalized depression was sustained for few minutes.

Vagal input seemed to contribute merely to the intensity of respiratory effector's changes.

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PRECLINICAL AND CLINICAL STUDIES OF IMMUNOSUPPRESSIVE AND CYTOSTATIC PROPERTIES OF CLADRIBINE (2-CdA)

Assoc. Prof. Paweł Grieb

The rat heart allotransplant model was used to study a possible synergism between cyclosporine A (CsA) and cladribine (2-CdA). Wistar rats were used as donors, and August rats as recipients. 2-CdA alone was marginally effective in prolonging graft survival, but its combination with CsA markedly increased the time to graft rejection, compared to CsA alone.

The mechanism of 2-CdA-induced thrombocytopenia was studied in three monkeys *Papio anubis* receiving repeated doses 0,2 mg/kg of the drug, either once a week, of q 5 every five weeks, for six months. The only sign of drug-related toxicity was a marked abnormality in bone marrow histology, indicating disturbances at the late stages of megakariocytopoesis and similar in appearance to that seen in idiopathic thrombocytopenia in humans. Cladribine-induced thrombocytopenia seems to be bone-marrow dependent.

The multicenter study of cladribine efficacy in relapsing or refractory low-grade non-Hodgkin's lymphomas has been published. The study involved 94 patients, objective responses (complete or partial remissions) were observed in 51% cases.

Two-year open study of cladribine treatment of relapsing-remitting multiple sclerosis has been concluded. The drug was given subcutaneously (5 mg/day) or orally (10 mg/day) q 5. Six monthly treatments were supplemented with additional two given at three-month intervals. Side effects were mild. In seven patients the treatment appeared highly active in reducing relapse rate, while in the remaining three the disease activity appeared unchanged.

BIOCOMPATIBILITY OF BLOCK COPOLYMER OF d,l-LACTIDE AND ε-CAPROLACTONE (L-Cap) WITH BRAIN TISSUES

Assoc. Prof. Paweł Grieb

The samples of the L-Cap biodegradable polymer (supplied by dr M. Bero and collaborators from the Center of Polymer Chemistry in Zabrze, Poland) were im-

planted into brains of rats and adjacent tissue response was followed with the use of transmission and scanning electron microscopy. Tissue reactions were indistinguishable from those to trauma alone, indicating that the said polymer may be safely used for drug delivery to the brain.

PLASMA CHOLESTEROL IN CEREBRAL MALIGNANCIES

Assoc. Prof. Paweł Grieb

The data of fasting plasma total cholesterol levels of 52 patients with malignant gliomas, 56 patients with cerebral metastatic tumors and 50 patients with peripheral malignancies with no cerebral involvement were compared. It was found that hypocholesterolemia characteristic of peripheral malignancies (known as "tumor-associated hypocholesterolemia") is infrequent in brain tumors.

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

Grant BIOMED 1 - PL 920432 - Assoc. Prof. Paweł Grieb

The techniques of proton and phosphorous magnetic resonance spectroscopy (^{1}H- and ^{31}P-MRS) were implemented on the 1.5 Tesla tomograph (Siemens Magnetom SP-63), including reproducible postprocessing of the spectra. The data were collected from the brains of healthy volunteers and from tumor areas of patients with gliomas. The proton spectra contained three prominent peaks assigned to N-acetylaspartate (NAA, "neuronal marker"), choline compounds (Cho) and creatine/phosphocreatine (Cr+PCr). The spectra from gliomas showed markedly lower NAA/Cho and NAA/Cr+PCr peak ratios compared to the normal brain areas. The phosphorous spectra were obtained from muscles and brains of healthy individuals. The phosphorous spectrum from a large intractable gliomas has been recorded and the patient entered chemotherapy with cladribine (2-CdA) and lovastatin (the inhibitor of cholesterol synthesis). The later case will be followed by ^{31}P-MRS to find out whether changes in phosphorous spectrum occur during treatment.

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INTRACELLULAR CALCIUM POOLS AND CALCIUM-DEPENDENT EXCITOTOXIC MECHANISMS

Prof. Jerzy W. Łazarewicz

Studies characterising intracellular Ca^{2+} pools in brain neurones were focused on learning the features of NMDA evoked Ca^{2+} induced Ca^{2+} release (CICR) *via* ryanodine receptors (RYRs). As recently demonstrated in our laboratory, in microdialysis experiments of the rat dentate gyrus *in vivo* combined with measurements of $^{45}Ca^{2+}$ efflux, application of NMDA to dialysis medium induces $^{45}Ca^{2+}$ release, 50 μM ryanodine stimulates this effect by 1890%, whereas 250 μM ryanodine and 100 μM dantrolene inhibits it. It is known from *in vitro* experiments that 10^{-2} M caffeine activates RYRs, and 10^{-4}–10^{-3} M caffeine potentiates Ca^{2+}-evoked stimulation of RYRs. However, it is not clear whether caffeine applied *in vivo* in pharmacological doses can modulate intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$), also effects of caffeine on NMDA-evoked CICR has not been described. The aim of this study was to establish the effect of caffeine applied *in vivo* in high pharmacological doses on NMDA-evoked $^{45}Ca^{2+}$ release in rat dentate gyrus. A method of caffeine application has been elaborated, which assured its stable concentration in brain and prevents its draining during microdialysis. The experiments have demonstrated that i.p. application of 40 mg/kg of caffeine followed by constant infusion of 80 μg/kg/min does not influence NMDA-evoked $^{45}Ca^{2+}$ release in the dentate gyrus *in vivo*. Application of caffeine induced by itself a brief stimulation of $^{45}Ca^{2+}$ release. This effect will be characterised in subsequent studies.

Microdialysis has been also utilised in attempts to demonstrate NMDA-evoked CICR in the rabbit hippocampus *in vivo*. As has been proved in our previous studies, in the rabbit hippocampus, in contradistinction to the rat, NMDA application leads to the inhibition, instead of activation, of $^{45}Ca^{2+}$ release. Since a simultaneous decrease in extracellular Ca^{2+} concentration was observed, together with activation of processes dependent on the increase in $[Ca^{2+}]_i$, such as release of taurine, phosphoethanolamine and prostanoids to dialysate, one may assume that NMDA induces increase in $[Ca^{2+}]_i$ in the rabbit hippocampal neurones *in vivo*. However, it is not clear to what extent intracellular Ca^{2+} mobilisation, and particularly CICR

participate in this effect. Thus, we studied the effect of 100 μM dantrolene and 50 μM ryanodine on NMDA-evoked changes in $^{45}Ca^{2+}$ efflux and in the release of taurine, phosphoethanolamine and prostaglandin D_2 to dialysate. The results did not show any significant effect of these RYRs modulators on $^{45}Ca^{2+}$ efflux and on other indicators of NMDA-evoked changes in $[Ca^{2+}]_i$ in the rabbit hippocampus. These data are indicative of a low activity of the NMDA-evoked CICR in rabbit hippocampus.

In studies on the potential role of Ca^{2+}- and excitatory amino acids in postischemic degeneration of CA1 hippocampal neurones, we assessed the anti-ischemic neuroprotective potential of two novel antagonists of the glycine$_B$ site of NMDA receptor. Body temperature was measured during the postischemic period, since NMDA antagonists are known to induce hypothermia, which is neuroprotective by itself. In comparison, the action of a well known neuroprotector NBQX, which is an AMPA antagonist was studied. To our knowledge the mechanism of NBQX-evoked neuroprotection has not been associated with brain hypothermia. The antagonists were administered to Mongolian gerbil at 30 mg/kg i.p., 15, 30 and 45 min after bilateral common carotid occlusion for 3 min. Spontaneously regulated rectal body temperature was measured for up to 3 h and after 24 h. Histopathological evaluation of CA1 damage was performed after 14 days. In untreated animals ischemia resulted in loss of almost 90% of CA1 pyramidal neurones, which was completely prevented by NBQX and reduced to 50% by glycine$_B$ antagonists. All these substances induced hypothermia. A decrease in rectal body temperature after glycine$_B$ antagonists was early and brief, whereas after NBQX a maximal hypothermia was observed later, 3 h after ischemia, and persisted for 24 h. These results point to a notable neuroprotective potential of glycine$_B$ antagonists after brain ischemia. However, this effect, similarly to neuroprotection evoked by other NMDA antagonists, may be associated with hypothermia induced by these agents. Unexpected finding of a delayed and prolonged postischemic hypothermia after NBQX administration may entirely explain NBQX-evoked neuroprotection. Our results did not provide arguments in favour of the role of NMDA and AMPA receptors in the mechanism of selective delayed damage to CA1 neurones after ischemia.

MECHANISM OF POSTISCHEMIC NEURONAL DAMAGE: CORRELATION OF EARLY CHANGES IN KINASES AND PROTEASES WITH INDUCTION OF CELL DEATH

Assoc. Prof. Krystyna Domańska-Janik

Growing evidence indicates that delayed neuronal injury after mild and transient ischemic insult is triggered by an activation of the constitutive program of cell death – apoptosis. Inhibition of this hypothetical chain of events before reaching

"the point of no return" should rescue affected neurones from ischemic irreversible degeneration.

Internucleosomal cleavage of DNA which could be detected as a typical laddering pattern after DNA electrophoresis, is one of the most accepted hallmarks of apoptosis. In our experiments we were not able to detect any signs of this typical DNA change at any time point during reperfusion (up to 96 h) after 5 to 20 min of cerebral ischemia in gerbil hippocampus, striatum or cerebral cortex. However, taking into consideration that oligonucleosomal fragmentation appears only transiently and asynchronously in the affected tissue, this negative result does not exclude the occurrence of apoptotic death after cerebral ischemia. Therefore, a more sensitive (though less specific) method of the detection, for example *in situ* TUNEL single cells labelling, should be used in our further experiments.

We have further investigated early and ischemia-specific changes in intracellular (post-receptor) signal transduction in the brain structures where delayed neuronal damage is observed. Special attention was given to the interactions between serine/threonine kinases which are transiently activated and then down-regulated after ischemia and calpain-specific proteolysis. Calpains are cysteine proteases, activated during ischemia and supposedly involved in the mechanism of apoptosis. The results indicate that calpains could be responsible for the previously observed postischemic PKC degradation and decreased catalytic activity in gerbil hippocampus. Using partially purified enzymes *in vitro* we have demonstrated that particular isoforms of cPKC (α, β and γ) react differently and specifically on two isoforms (μ and m) of brain calpains. This reaction, with transient appearance of the catalytically active PKC degradation product: PKM, resembles the response previously observed after ischemia in hippocampus *in vivo*.

Moreover, the ischemia-induced changes correlate well with the altered expression of mRNA for γPKC in gerbil hippocampus. Using RT-PCR technique we have demonstrated an early induction followed by a marked depression of this isoform of PKC up to 96 h after ischemia. The mRNA content for the other cPKC isoforms (α and β) have not changed during this period of time. Considering the supposed neuronal and postsynaptic localization of γPKC our results indicate an exceptional sensitivity of this cellular compartment to ischemic injury.

The other investigated serine/threonine kinase, previously shown to be extensively modified during and after ischemia, was calcium-calmodulin dependent kinase II (CaMKII). Experiments performed on gerbil hippocampus revealed that inhibition of NMDA receptor by MK801 as well as blocking of voltage-dependent Ca^{2+} channels by nimodipine, both result in attenuation of ischemia-evoked changes in CaMKII activity *in vivo*. The observed effect was not additive and involved both of the previously described, diverse phases of this kinase reaction: an early activation/translocation and subsequent enzyme down-regulation.

PLP GENE MUTATION AND ITS PLEIOTROPIC EFFECTS ON MYELINATION IN "PT" RABBIT

Assoc. Prof. Krystyna Domańska-Janik

Spontaneous pt mutation in rabbits consists in a point transversion of a single nucleotide in plp gene. The cellular/regional expression of myelin-specific proteins (PLP, MBP, CNP, MAG and MOG) was investigated by immunohistochemistry in developing brains of 14 and 42 days-old control and mutant rabbits. Parallely, the expression of plp gene was evaluated by *in situ* hybridization (ISH) technique. The results showed a substantial reduction in the expression of almost all myelin-associated proteins investigated, especially pronounced in the younger, 14-days old pt rabbits.

The most severe depletion, on both the transcriptional (mRNA) and translational (protein) level, was noticed for PLP as a primary molecular target of the mutation. Immunoreactivity of myelin-specific glycoproteins (MAG and MOG) was, in spite of its reduction in pt myelin sheaths, slightly elevated in oligodendrocyte cell bodies. The ISH confirmed, in accord with our previous data (Northern blots), that PLP mRNA transcript was primarily unaffected by pt mutation and then progressively down-regulated along maturation. This is in line with our hypothesis that suppression of myelination program in pt rabbits does not appear before the developmental expression of PLP (about 10-14 day *post partum*). Accumulation of abnormal PLP protein would affect the pt-oligodendrocyte function and lead to generalised hypo- and dysmyelination in mutant rabbit CNS.

MECHANISM AND ULTRASTRUCTURAL FEATURES OF LEAD TOXICITY

Prof. Urszula Rafałowska

I. The kinetics of citrate transport across the membrane of the synaptic mitochondria obtained from synaptosomal fraction isolated from brains of control and chronically Pb-intoxicated rats were evaluated. The transport of citrate to synaptic mitochondria is a saturable process which obeys Michaelis-Menten kinetics. A decrease of transport velocity and affinity of citrate into mitochondria was noted under chronic Pb^{2+}-toxicity conditions, which may be due to a decrease of proteinaceous -SH groups. Decreased citrate transport may affect the Krebs cycle and subsequently cause changes in energy processes in synaptic mitochondria.

II. The effects of acute lead toxicity on the energy metabolism and regulatory mechanisms in brain synaptosomes were investigated. Oxygen consumption increased in Pb intoxicated synaptosomes as compared to the control but the cytochrome oxidase activity did not change. Intracellular concentration of ATP increased significantly in synaptosomes obtained from the brains of lead-treated

rats as compared to control, whereas the levels of ADP and AMP increased only slightly.

The level of phosphocreatine (CrP) was found to be higher and activity of creatine kinase (CK) increased drastically under lead-toxicity condition, but the Na-K-ATPase activity decreased. Taken together, the results suggest that under acute lead-toxicity condition, both the respiration and the PCr/CK system, are activated to maintain adequate ATP concentration.

III. Myelin changes in brains of chronically lead-poisoned animals were studied at the biochemical and ultrastructural level. Myelin obtained from chronically Pb-poisoned rats was found to be morphologically changed. Lead caused drastic disturbances of the lamella structure in axonal myelin sheaths. Searching for mechanisms responsible for these morphological changes, we have examined the effects of Pb^{2-} on the level of myelin phospholipids and peroxidation processes. While peroxidation was unchanged, an increase in the content of phosphatidylethanolamine and increased phosphatidylphenolamine/phosphatidylcholine ratio were noted. These effects together with the decreased level of protein-SH groups may be the reason of changes in organisation of the lipid-proteins sheaths of myelin under lead-toxicity conditions.

MODULATION OF EICOSANOID RELEASE IN THE HIPPOCAMPUS *IN VIVO* BY NMDA RECEPTORS

Prof. Jerzy W. Łazarewicz

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Studies have been continued, using microdialysis method, on the mechanisms of NMDA-induced prostaglandin (PG) D_2 release in the rabbit hippocampus *in vivo*. Additional experiments confirmed a dose-effect relation which indicates that this phenomenon is NMDA receptor-mediated. However a bell-shaped curve of NMDA-evoked PG D_2 release, with its maximum at 0.5-1.0 mM NMDA, differs from the NMDA-induced decrease in $^{45}Ca^{2-}$ efflux, which depends on NMDA concentration from 0.1 to 5 mM. Inhibition of the NMDA-evoked PG D_2 release by competitive NMDA receptor antagonists APV and CPP also confirms the role of NMDA receptors, although resistance of this effect to inhibition by a noncompetitive NMDA receptor antagonist MK-801 needs explanation. NMDA-evoked PG D_2 release and decrease in $^{45}Ca^{2-}$ efflux were not altered by tetrodotoxin, which excludes the role of a non-specific NMDA-induced depolarisation. Notable individual differences between rabbits in sensitivity to the MK-801 inhibition of NMDA-induced PG D_2 release and a decrease in $^{45}Ca^{2-}$ efflux were observed.

To explain the above observations we examined the role of intracellular calcium mobilisation in the mechanism of NMDA-evoked PG D_2 release in the rabbit

hippocampus. Since none of the parameters under study was affected by dantrolene or ryanodine, it seems that calcium from the ryanodine pool is not involved significantly in NMDA-induced phenomena. Comparative studies on the role of this calcium pool in the regulation of PG D_2 production by NMDA receptors in the rat dentate gyrus, which is particularly reach in ryanodine receptors, are in progress.

A previously constructed quartz fibre optical probe, an attachment to an existing spectrofluorimeter, has been improved and adapted for measurements of intracellular Ca^{2+} concentration in the hippocampal slices *in vitro* with fura-2. We continue effort to adapt this probe for intracellular Ca^{2+} measurements in deep brain structures *in vivo*.

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

Dr. Wanda Gordon-Krajcer

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Effects of rat brain ischemia on the expression and metabolism of beta amyloid protein and its precursor (β-APP) was studied with a focus on the 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 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 brain, and that the signal for these changes is generated rather during than after ischemia.

To elucidate the direct effect evoked by excitatory amino acids on the processing of β-APP and amyloid accumulation in the rabbit hippocampus, 1 mM NMDA was applied *via* transhippocampal microdialysis probe for 20 min. Separation of proteins from the hippocampal tissues neighboring microdialysis probe and superfusates by electrophoresis was followed by their Western blot analysis using antibodies against some domains of β-APP.

The results indicate that application of NMDA to the hippocampus induces changes in the pattern of β-APP fragments and accumulation of βA (amyloidogenic fragment) and other domains of β-APP. These results are consistent with the results of "*in vitro*" experiments performed to elucidate the role of NMDA receptors in modulation of β-APP processing and release of different β-APP fragments from rat

hippocampal slices to the superfusion medium. To detect the role of NMDA receptors in modulation of β-APP processing, adult rat hippocampal slices were superfused with NMDA containing media and immunoreactivity of soluble β-APP derivatives was measured in superfusates. Hundred μM and particularly 250 μM NMDA induced a release of amino-terminal β-APP derivatives and a fragment of βA, which was dose-dependent, and prevented by antagonists of NMDA receptors and Ca^{2+}-free medium. Release of carboxyterminal fragments of β-APP was not detected. These data indicate that stimulation of NMDA receptors induces cleavage of β-APP.

EFFECT OF DEPHOSPHORYLATION *IN VITRO* ON CONFORMATION OF PROTEIN TAU IN PAIRED HELICAL FILAMENTS (PHF)

Dr. Wanda Gordon-Krajcer

In order to determine whether the high phosphate content of tau in paired helical filaments (PHF) is a result of inefficient phosphatase system in Alzheimer's disease (AD) or of limited access to phosphorylation sites, we compared the effects of alkaline phosphatase (E. coli) in native PHF, amorphous PHF (formic acid-treated) and fetal human tau. Samples were incubated with 20 IU phosphatase/ml for 30 min to 24 h and analyzed by SDS-PAGE and immunoblotting with a panel of phosphorylation-sensitive (six) and -insensitive (two) antibodies. Fetal tau was efficiently dephosphorylated at all four P-Ser but not P-Thr epitopes (Thr181 and Thr231) and its mobility was markedly improved. Native PHF underwent a partial dephosphorylation at the N-terminus but were unaffected at P-Thr epitopes ot the C-terminal half of tau. The mobility of native PHF remained unchanged. In contrast, amorphous PHF were dephosphorylated much more efficiently and their mobility increased. The efficiency of dephosphorylation at the N-terminal half exceded that in fetal tau. The C-terminal epitopes, however, were still less reactive with phosphatase than that in fetal tau. The results suggest that the C-terminal epitopes of tau in PHF have limited access to phosphatases and may remain phosphorylated in their presence.

INTERACTIONS OF PROTEIN KINASE C AND CALPAINS IN CEREBRAL ISCHEMIA

Dr. Teresa Zalewska

Supported by the State Committee for Scientific Research: grant # 4 P05A 026 08

Increasing evidence suggests that excessive activation of calcium-dependent neutral proteases – calpains, could play a key or contributory role in the pathology of cerebral ischemia and are implicated in a certain forms of apoptosis. Calpains

(EC 3.4.22.17), are calcium-activated, non-lysosomal, neutral cysteine proteases proposed to participate in many important intracellular processes, such as turnover of cytoskeletal proteins and regulation of kinase activities. Two isoforms of calpain exist in brain tissue. One form, m-calpain requires milimolar calcium concentration for activation *in vitro*, while the other form, μ-calpain is optimally active at calcium concentration in the micromolar range. In addition to calcium concentration, the proteolytic activity is also regulated by the endogenous inhibitor – calpastatin. The behaviour of both calpains isoforms as well as calpastatin was investigated in rat subjected to acute postdecapitative global ischemia. Acute ischemia up to 15 minutes duration resulted in gradual, time-dependent decrease of total μ-calpain activity (to 60% of control values) and in the elevation of calpastatin activity (by 28%). The observed down-regulation of total μ-calpain activity coincided with its extensive translocation to the particular fraction. The proteolytic activity associated with this fraction increased after ischemia to 300% of control. In the case of m-calpain the only observed effect was its translocation and, in consequence, the increase of activity in particular fractions (to 200% of control). Such a redistribution of the enzyme activity toward plasma membranes, observed for the first time, seems to be a general response of calpains to ischemic conditions. These results fit well the generally accepted mechanism of autolytic calpain activation at the plasma membranes, where the preferred substrates could be targeted in the presence of calcium. Our suggestion of the initial postischemic activation of μ-calpain was further confirmed by the observed increase of the 76 kDa activated isoform of μ-calpain, and decrease of its 80 kDa inactive precursor (Western blotting). An additional evidence for the early post-ischemic activation of calcium-dependent proteolysis is the accumulation of products resulting from calpain-catalyzed proteolysis of specific substrates: cytoskeletal protein – fodrin and regulatory enzyme, protein kinase C. The above findings suggest that ischemia induces two divergent, but interrelated effects on calpain activity. One involves rapid activation of the proteolytic process, as was detected in the present study by the extensive calpain association with the particulate fraction and the substantial cleavage of calpain substrates (fodrin and PKC). The second involves gradual, time-dependent down-regulation of the total μ-calpain activity with a concomitant increase of calpastatin activity. The ability of tissue to keep these two activatory and inhibitory processes in balance might determine the final outcome of the ischemic episode.

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MOLECULAR MECHANISMS OF BRAIN AGING, ALZHEIMER DISEASE AND ISCHEMIA EVOKED ALTERATION OF SIGNAL TRANSDUCTION

Prof. Joanna Strosznajder

Objectives: The regulatory mechanism of signal transduction in purinergic, serotonergic cholinergic and glutaminergic system in adult and aged brain were investigated.

Analysis of second messengers release and their role in the regulation of Ca^{2-} ions in brain were carried out. In evaluation of calcium ion homeostasis in aged brain, attention was focused on endoplasmic reticulum CaATP-ase specifically inhibited by tapsigargine. In the studies on pathological brain aging biochemical characteristics of Down syndrome (DS) preamyloid and amyloid in neuritic plaques were compared. Moreover, in cooperation of Department of Pathology, New York University Medical Center, we have participated in the studies on novel meningocerebrovascular amyloidosis.

For the first time, micropreparative gel electrophoresis was used for the separation of low-molecular-weight, highly insoluble amyloid peptides. Studies on ischemic encephalopathy focused on the role of nitric oxide (NO) in the activation of free radicals cascade and membrane lipid peroxidation and other biochemical processes in brain cortex and cerebellum.

Looking for neuroprotective agents that may have clinical therapeutic potential our research has focused on NO synthase inhibitor, 7-nitroindazole. A new technic of ion-exclusion chromatography was elaborated for the separation of nitrate and nitrite, a stable metabolites of nitric oxide.

Results: It was found that ATP is an important regulator of phospholipase C. Acting through purinergic receptor it activates phosphatidylinositol 4,5 bisphosphate (PIP2), whereas acting directly it inhibits in an allosteric manner PIP2 and phosphatidylinositol (PI) hydrolysis by PLC. Moreover in both cases ATP inhibits conversion of IP3 to IP2 playing an important role in regulation the level of IP3 that liberates Ca ions from endoplasmic reticulum. Simultaneously with IP3 the other second messenger, diacylglycerol (DAG) is formed that activates protein kinase C. Degradation of PIP2 is the early events in signal transduction processes.

Hydrolysis of other phospholipids by PLC may be involved in maintaining the signal. A significantly lower Ca^{2+}-activated degradation of phosphatidylethanolamine (PE) and phosphatidylcholine (PC) in brain cortex as compared to phosphoinositides hydrolysis was observed. Brain aging had no significant effect on these processes. However, in aged brain, cholinergic and serotonergic receptor-dependent IP3-regulated Ca^{2+} release from endoplasmic reticulum is significantly decreased or eliminated, probably as a result of IP3 receptor alteration. Our results suggested that higher activity of endoplasmic reticulum Ca^{2+} ATP-ase and more active Ca^{2+} uptake into endoplasmic reticulum in aged brain may be responsible for the lower cytosolic Ca^{2+} concentration in aged brain comparing to adult and for the alteration of Ca^{2+} ions homeostasis.

Essential alterations in the mechanism of regulation of basal- and NMDA receptor-dependent cGMP level were observed in aged brain. This second messenger through activation of specific kinase is involved in the regulation of Ca channel(s) or IP3 formation and action. In aged brain significantly lower level of cGMP was found. Activation of phosphodiesterase is responsible for the degradation and lower basal level of cGMP in aged brain. Elimination of NMDA receptor-dependent cGMP formation in hippocampus and cerebellum is related to alteration of guanylyl cyclase and NMDA receptor protein by NO, produced in higher amount in aged brain comparing to adult.

In the studies on the arachidonate transport through the blood-brain and the blood-retina-barrier during aging it was found that arachidonate is able to cross at relevant rate BRB and BBB. In all brain regions except retina, optic tract and hippocampus BBB has a transport capacity for AA significantly higher than for other polyunsaturated fatty acids. Aging does not affect influx into CNS of this second messenger, which is rapidly incorporated into microcapillary and brain lipids. A simple diffusion transport across the BRB and BBB for AA is postulated.

A novel meningocerebrovascular amyloidosis associated with a novel transthyretin mutation at codon 18 where aspartate is replaced by glycine was described.

In continuation of the studies on the pathogenesis of postischemic encephalopathy, the molecular action of nitric oxide (NO) was evaluated. NO-activated brain cortex lipid peroxidation was observed in the early phase of reperfusion. It was found that this gas mediator is able to transmit signal into cerebellum through activation of guanylate cyclase. NO enhanced significantly the level of cGMP also in cerebellum. An inhibitor of NO-synthase which appears to have some selectivity to nNOS, markedly decreased ischemia-evoked biochemical alteration, but has negligible protective effect on ischemia-induced cell death in CA1 layer of hippocampus.

A new method of ion exclusion chromatography was elaborated for the determination of stable NO metabolites in the blood and brain.

ROLE OF HB-GAM CYTOKINE IN AMYLOID β FIBRILISATION IN AGED BRAIN AND ALZHEIMER DISEASE AND ITS ROLE IN NEURONAL DEGENERATION IN CENTRAL NERVOUS SYSTEM

Prof. Joanna Strosznajder

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The distribution of heparin binding growth associated molecule (HB-GAM) in the cerebral amyloidoses of Alzheimer's disease (AD) and Down's syndrome (DS), was investigated immunochemically.

Antibodies to BH-GAM, a cytokine which plays an important role in brain development and maturation showed strong immunoreactivity with senile plaques in AD and DS. It was found that anti-HB-GAM reacted with preamyloid lesions but only when markers of dystrophic neurites were present, suggesting that the presence of HB-GAM in Aβ lesions is a marker of neuronal injury.

MOLECULAR MECHANISMS OF BIOCHEMICAL TRANSFORMATION OF NONTOXIC AMYLOID β INTO TOXIC FORM AND THE ROLE IN SIGNAL TRANSDUCTION IN BRAIN

Prof. Joanna Strosznajder

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The effect of different forms of amyloid β 1-40 and Aβ 25-35 on growth and proliferation of different types of cells in culture (neuroblastoma, glioblastoma, embryonic cells and HeLa-60) was investigated. Preliminary studies have dealt with the effects of Aβ on DNA integrity and Poly ADP-ribose synthase polymerase (PARP). The action of nontoxic Aβ 1-28, and neurotoxic Aβ 25-35 on basal and cholinergic receptor-mediated intracellular Ca^{2+} concentration was evaluated in brain cortex synaptoneurosomes using Fura-2 fluorescence probe.

AMYLOID β PEPTIDES IN THE NORMAL AND DISEASED STATE

Dr. Maciej M. Lalowski

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An attempt has been made to identify the composition of amyloid β peptides within the cerebellum of Down's syndrome, where diffuse plaques (preamyloid) are very abundant and mature (compact) neuritic plaques are sparse. The composition of DS preamyloid was characterized and compared to amyloid in the neuritic plaques and leptomeninges in the same patients. Aβ17-42 or p3 was found to be a

major Aβ peptide of DS cerebellar preamyloid. This 26-residue peptide was also present in very low quantities (1%) in neuritic plaques. Preamyloid can now be defined biochemically as lesions in which a major Aβ peptide is p3. In addition, *in vitro* studies have clearly demonstrated that Aβ17-42 can form amyloid-like fibrils that are Congo red positive; however it does so much less well than either Aβ1-40 or Aβ1-42. Under the conditions used, this highly hydrophobic peptide tended to form mostly amorphous aggregates. It correlated well with the known, largely nonfibrillar nature of diffuse lesions and might, in part, explain the lack of clinical signs of cerebral dysfunction associated with this lesions. p3 (Aβ17-42) arises from the larger precursor AβPP, by the combined action of α-secretase at the Aβ N-terminus and the γ-secretase. This α-secretase pathway was initially called "nonamyloidogenic", because it involves the cleavage with Aβ domain, precluding the formation of longer Aβ1-40/42,43 peptides. The studies clearly document for the first time that this pathway is not necessarily "nonamyloidogenic". Alterations of AβPP processing which favor the production of p3 versus other Aβ peptides may not necessarily benefit AD patients because this peptide appears to be the major constituent of at least some initial Aβ preamyloid deposits.

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SYNERGISM OF SELECTED NEUROTRANSMITTERS AND THEIR PRECURSORS IN THE PATHOMECHANISM OF HEPATIC ENCEPHALOPATHY

Prof. Jan Albrecht

The uptake of [^{14}C]glutamate (GLU) to nonsynaptic mitochondria isolated from rat cerebral hemispheres was measured in the presence of potential amino acid modulators. L-aspartate (ASP) virtually abolished GLU uptake at ASP/GLU ratio of 1:1, as predicted from the involvement of the GLU/ASP carrier. L-arginine (L-ARG) inhibited GLU uptake in a dose dependent manner: A significant effects was already noted at L-ARG/GLU ratio of 1:2. Of the different L-ARG metabolites added in 25-fold excess, ornithine, putrescine, or ammonia had no effect, whereas creatine and the NO generator – sodium nitroprusside increased the uptake by 73% and 57%, respectively. D-ARG was three times less effective in inhibiting GLU uptake than L-ARG. Glutamine inhibited the uptake by only 14% when added in 10-fold excess of GLU, whereas none of the following L-amino acids: histidine, tyrosine, phenylalanine, proline, leucine, isoleucine, tryptophan, glycine, methionine, valine, serine, tyrosine, taurine, alanine or cysteine, affected the uptake when added in 10-25-fold excess. An inhibition of the uptake of similar magnitude to that in the presence of GLN was noted with a 25-fold excess of dicarboxylate carrier ligands, α-ketoglutarate and phenylsuccinate. The results suggest that L-ARG may function as an endogenous modulator of cerebral mitochondrial GLU transport.

Hepatic encephalopathy (HE) is characterized by symptoms pointing at disturbances in glutamatergic neurotransmission in the brain, particularly in the striatum. The binding parameters of ligands specific for kainate (KA) and AMPA receptors and for different recognition sites in the N-methyl-D-aspartate (NMDA) receptor complex were assessed in rats with acute HE induced with a hepatotoxin, thioacetamide. The KA-, AMPA-, and NMDA-displaceable [^{3}H]glutamate binding, and the binding of: 1) [^{3}H]dizocilpine, 2) N-[1-(2-thienyl)cyclohexyl]-[^{3}H]piperidine (TCP) and 3) the coactivator site agonist [^{3}H]glycine was assayed in purified membranes of the cerebral cortex, hippocampus and striatum. In HE rats, B_{max} of KA-displaceable glutamate binding was significantly increased in the cerebral cortex, diminished in the striatum, and unchanged in the hippocampus, whereas

B_{max} of AMPA-displaceable glutamate binding was increased in the cerebral cortex, decreased in the hippocampus, but unchanged in the striatum. In HE rats, B_{max} of NMDA-displaceable glutamate binding was increased in the cerebral cortex and hippocampus but slightly decreased in the striatum. In this region the binding affinity was also slightly increased. In HE, B_{max} of [^{3}H]dizocilpine binding was unchanged in the striatum and cerebral cortex but substantially decreased in the hippocampus. B_{max} of [^{3}H]TCP binding was decreased in the cerebral cortex and striatum but increased in the hippocampus. The different responses of these two phencyclidine site antagonists to HE may be indicative of a conformational change within the ion channel and/or the presence of microdomains reacting differently to extrinsic factors. HE did not affect glycine binding but potentiated the maximal stimulation of [^{3}H]dizocilpine binding by glycine in the cerebral cortex. The results emphasize the brain region- and domain-specificity of the responses of the NMDA receptor complex, and the brain region-specificity of the responses of KA and NMPA receptors to HE.

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TISSUE PATHOGENETIC MECHANISMS OF POSTISCHEMIC ENCEPHALOPATHY

Prof. Andrzej Kapuściński

The photochemical model of focal brain ischemia characterized by illumination of the brain surface with light generated by the air cooled 250 W halogen lamp transmitted by the lightpipe after i.v. injection of 3% bengal rose has been used. Bengal rose reacting with light produces alterations in capillary endothelial cells with deposition of thromboaggregates occluding capillary vessels developing focal tissue ischemia. In the above experimental model of rats after 1, 4 and 7 days of survival the following data were evaluated: extension of necrotic area, region of penumbra, microscopic tissue abnormalities, advancement of brain edema, immunocytochemical reaction for GFAP and condition of injured blood-brain barrier. Model of compression focal brain ischemia allows to receive reproducible results of morphological changes, their extent and intensity. Compression with the measurable power, precisely delineated brain surface and permanent duration of pathology produces reversible tissue ischemia independent of configuration of the arterial capillary network in the pia mater. In two above experimental models changes in localization of sugar receptors in the network of rat's brain capillaries were evaluated by means of lectin technique. Two lectins have been used localizing following sugar residues: β-D-galactose and N-acetyl-galactosamine and neuraminic acid. Changes in localization of examined glycoconjugates appeared after 1 day of the animal survival and characterized by weaker staining reaction. After 4 days of the animal survival necrotic areas appeared surrounded by phagocytes and reactive astrocytes indicated exclusively β-D-galactose receptors. Both types of receptors indicated a significant decrease of histochemical reaction in the whole network of brain capillaries and especially the staining reaction disappeared or was very weak in neocortex. Comparing the results using both experimental models it was evident that photochemical ischemia produces more subtle histochemical changes. In the experimental model of brain ischemia after injection of 0.05 ml of air into left common carotid artery in rats combined with occlusion of this vessel, significant elevation of brain lactate level was observed. Mild hypothermia (28-31^{0}C, 4 h) reduced cerebral intracellular acidosis significantly. The possibility of a therapeu-

tic application of mild hypothermia as neuroprotective action against development of postischemic tissue changes was discussed. In an other experimental model of cerebral ischemia in rats after injection of 10 pmol of endothelin-1 into the right lateral brain ventricle, there was also observed a significant increase of brain lactate. Coenzyme Q10 reduced lactate acidosis to the normal level 24 h after initiation of ischemia. The beneficial effect of Co Q10 can be ascribed to the improvement of cellular respiration leading to the elimination of free radicals. Co Q10 can play an important role in prevention and treatment of ischemic insults. In the experimental model of cardiac arrest a significant decrease of norepinephrine and epinephrine levels was observed in the rat brain during ischemia and in the early period after resuscitation. The plasma catecholamine level increased in the postresuscitation period, concomitant to the normalization of circulatory system and development of postischemic active hyperemia.

OPPORTUNISTIC INFECTIONS AND HIV-SPECIFIC CHANGES IN THE BRAIN IN POLISH AIDS CASES

Assoc. Prof. Irmina B. Zelman

The frequency of opportunistic infections and their relation to HIV-specific pathology was analysed morphologically in 127 brains of patients who died in the course of AIDS in 1987-1996 years (116 males, 10 females, mean age 39 years and one 11-month-old child). Routine histological methods were applied, in some cases supplemented with histochemical and immunohistochemical stainings.

HIV-specific pathology (HIV-encephalitis, HIV-encephalopathy or both) was found in 36% of cases, whereas opportunistic infections amounted to 76 cases (60%). A characteristic feature of cases with opportunistic pathology was coexistence of two or more opportunistic infections in the same brain, their coexistence, with HIV-specific changes and/or with primary or secondary malignant lymphomas (in one case with primary Kaposi sarcoma). In 40 brains (53% of cases with opportunistic infections) single infection was found, in the remaining 36 cases (47%) the pathological process was not uniform. In 9 cases two different opportunistic infections were disclosed, in 20 cases infections were associated with HIV-specific changes, and in 4 cases – with malignant lymphomas. Three cases were characterised by coexistence of opportunistic infections, HIV-specific changes and malignant lymphomas. Among opportunistic infections toxoplasmosis appeared to be most often associated with HIV-specific changes. The high frequency of this coexistence may be explained by the invasion of blood-born cells of monocyte/macrophage lineage latently infected with HIV into brain parenchyma. The most frequent, in our material, was CMV infection (24% of cases), followed by toxoplasmosis (16,5%). Progressive multifocal leukoencephalopathy (PML) and cryptococcosis were ob-

served in the same number of cases (10%). Brain aspergillosis, a casuistic rarity in AIDS material, was found in 3 cases. Only in limited number of cases (4,7%) nodular encephalitis without etiological conotation was diagnosed.

The characteristic neuropathological feature was the difference in the topography and structural expression of the same type of opportunistic infections in particular cases, reflecting the different pathways of infection, different periods of its evolution and intensity of disturbances in patients immunological reactivity, additionally influenced by specific therapeutic management.

DISORGANIZATION OF CORTICAL STRUCTURE AND BRAIN GLIOMAS

Prof. Halina Kroh

Among biopsy material obtained from 63 patients during temporal lobectomy due to intractable epilepsy, the tumoral tissue consisted of various types of astrocytomas (38), oligodendrogliomas (8), oligoastrocytomas (5), anaplastic gliomas (2), dysembryoplastic neuroectodermal tumors (6) and gangliogliomas (4). Beside the tumor mass, the white matter contained dispersed neurons in 10 cases, disordered cortical lamination was present in 3 cases and additional microtumors of glial, neuronal or non-defined character of type different than the main tumor mass in 6 cases.

Cortical dysplasia and neuronal heterotopias belong to the disorders of abnormal cellular migration. In cases of temporal gliomas the coexistence of microtumors in the cortex and white matter and neuronal nests typical for the composed type of DNT corresponds to the disturbances observed also in our material.

The three types of additional migration disturbances associated with gliomas of temporal lobes suggest initial common heterotopic origin of changes which progressed along various pathways. (H. Kroh and E. Matyja)

INTRACRANIAL HEMANGIOPERICYTOMAS

Prof. Halina Kroh

The aim of the study was controversial origin of intracranial hemangiopericytomas. All examined biopsies of meningeal tumors (8) presented typical immunohistochemical features (9 antibodies). Two tumors exhibited additional histological features, found usually in meningiomas. Histological differences stressed non-univocal origin of meningeal hemangiopericytomas from vascular pericytes or meningeal cap cells.

CLINICAL AND PATHOLOGICAL EXAMINATION OF DIFFERENT FORMS OF BRAIN AMYLOIDOSIS WITH OR WITHOUT DEMENTIA

Dr. Maria Barcikowska

The aim of our project was an examination of so called normal aging and neurodegenerative changes (PrP or β-amyloid positive amyloidosis) in human and animal brains. Several clinical investigations were done to resolve heterogeneity of Alzheimer's disease (AD) patients. The problem of the presence of parkinsonian syndrome among AD patients and the differentiation, based on neuropsychological and neurological assessment, of Pick's disease in the cohort of AD patients with frontal symptoms were a matter of our investigations. The most characteristic of AD pathology involvement – limbic system dysfunction was discussed due to hippocampal volume diagnosis in the MRI examination. Hippocampal volume is believed to be the most sensitive factor in the correlation with the level of neuropsychologically diagnosed dementia. Aphatic, language disturbance in the group of AD patients varied with the development of a generalized dementia level, which was the conclusion of our study. Language functioning in AD patient clinically diagnosed is an interesting prognostic tool in the assessment of dementia in the brain with Alzheimer's type of amyloidosis. The apolipoprotein E allels as the only known risk factor for AD was characterized in the group of AD patients and within their first grade relatives treated in outpatient clinic; results being not different from those that were published before in other countries. The clinicopathological study of a case with Diffuse Lewy Body disease was done and it was the first such case in the Polish literature. Another case reported by us: a case of PrP positive amyloidosis clinically diagnosed as the olivo-ponto-cerebellar atrophy was published. An unusual sporadic case of Creutzfeldt-Jakob disease was characterised by an abundance of PrP-immunopositive kuru and multicentric plaques but without any known mutation in the PRNP gene. An immunohistochemical search for β-amyloid deposits was also done in the very old animal brains. Positive results were visualized in dogs, monkey's and (for the first time) in goat brains. The β-amyloid deposits were labelled by using 4G8 mAb (against β-amyloid residues) also in post trauma brains of fetuses and also within young and old brains. These last two preliminary investigation will be continued during next years.

ELABORATION OF EXPERIMENTAL MODEL OF ALZHEIMER'S DISEASE

Assoc. Prof. Ryszard Pluta

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Brains form patients with Alzheimer's disease contain amyloid plaques which are composed of beta-amyloid peptide and are considered to play a causal role in

the neuropathology of this disease. The origin of beta-amyloid peptide in brain parenchyma and vessels of Alzheimer's disease patients is not known. Our study examined the permeability of the blood-brain barrier to beta-amyloid peptide in rats subjected to single or repeated episodes of global cerebral ischemia followed by i.v. injections of human synthetic beta-amyloid-(1-42)-peptide. These experiments were performed to test the hypothesis that the blood-brain barrier has the capacity to control blood-brain transport and extra- and intracellular accumulation of circulating beta-amyloid peptide. Rats receiving beta-amyloid peptide after ischemia demonstrated multifocal and widespread accumulation of beta-amyloid peptide in hippocampus, cerebral cortex and occasionally in white matter. Beta-amyloid peptide penetration involved arterioles, veins and venules. Neuronal, glial and pericyte bodies were observed filled with beta-amyloid peptide. Beta-amyloid peptide deposition ranged from numerous small immunopositive dots to regular diffuse plaques. The latter were often observed without any visible connection with blood vessels. Thus, for the first time, direct evidence is provided that soluble human beta-amyloid peptide crosses the open blood-brain barrier and enters the brain parenchyma from the circulation.

THE ROLE OF APOLIPOPROTEIN E IN THE PATHOGENESIS OF β-AMYLOID

Dr. Elżbieta Kida

Supported by the State Committee for Scientific Research: grant # 4 P05B 054 10

The involvement of apolipoprotein E (apo E) in the pathogenesis of Alzheimer's disease was first disclosed by Namba et al. in 1991, based on the finding of apo E in both β-amyloid deposits and neurofibrillary tangles. Studies conducted afterwards by Alan Roses and his collegues revealed a genetic link between apo E and sporadic and familial Alzheimer's disease. It was found that the frequency of apo E allele ε4 is distinctly higher (2-3 times) in patients with Alzheimer's disease than in controls. Later on it was shown that Alzheimer's disease patients carrying apo E ε4 allele demonstrate higher β-amyloid load within the central nervous system, higher number of senile plaques showing Aβ 40, higher neurofibrillary tangle counts and more severe damage of the cholinergic system. However, the precise mechanisms underlying apo E involvement in the pathogenesis of Alzheimer's disease, as well as in other β-amyloidoses, remain only hypothetical. It is postulated that apo E may directly influence Aβ fibrillogenesis, the resolution of Aβ, phosphorylation of tau protein, the function and integrity of the cholinergic system as well as regeneration of neuritic processes.

One of the major biological functions of apo E is lipids transport, as well as their local distribution and mobilization in normal and injured tissues, important for membrane integrity and regeneration. Following the experimental damage of

both peripheral and optic nerve, apo E expression increases more than 200-folds. These observations inclined us to initiate the studies aimed to elucidate of whether similar increase in apo E expression occurs in the process of β-amyloidosis in the areas of specific tissue damage: within senile plaques containing swollen and structurally damaged neuritic processes, so called dystrophic neurites. These neuritic processes reveal the presence of APP, precursor protein for Aβ, the main component of β-amyloid. The studies were performed on postmortem brain tissues collected from Alzheimer's disease, Down syndrome, and control cases without dementia, but with β-amyloid deposits. It was demonstrated that extracellular deposits of apo E within senile plaques containing dystrophic neurites correlate with the presence of extracellular Aβ deposits in these areas. This finding suggests that apo E appears extracellularly within senile plaques containing dystrophic neurites secondarily to Aβ. Moreover, apo E within these lesions showed topographic differences, and was the most abundant in these regions that showed an early and severe damage of afferent terminals such as the marginal zone of the dentate gyrus. At the same time, however, there was either none, or only a weak immunoreactivity to apo E within the cytoplasm of dystrophic neurites. It is well documented that nerve cells reveal apo E receptors and are able to internalize apo E containing lipoproteins. Thus, the scarcity or even absence of apo E within dystrophic neurites disclosed at present might indicate that the processes of tissue regeneration undergoing with apo E may be altered in β-amyloidosis which might further aggravate structural alterations and lead to further dysfunction of the central nervous system.

These studies are continued at present in tissue culture conditions, by using both organotypic cultures of rat cerebellum, hippocampus and striatum as well as neuroblastoma cell line SY5Y. Our preliminary data suggest, that subtoxic doses of excitatory amino acids as well as of various Aβ forms (Aβ 40, 42, 17-42) applied to culture medium produce an increased tissue expression of apo E.

THE ROLE OF Bcl-2 AND Bax IN CNS DEVELOPMENT AND IN THE PATHOMECHANISM OF ALZHEIMER'S DISEASE AND DOWN SYNDROME

Dr. Elżbieta Kida

Bcl-2 and Bax, two proteins associated with genetically determined programmed cell death, were examined immunocytochemically in the developing human cortex and in Alzheimer's disease and Down syndrome brains. These studies showed the immunolocalization of these protein in developing human brain, their association with the apoptosis, maturation and terminal differentiation of the central nervous system structures, and their putative involvement in the pathogenesis of developing Down syndrome brain. Disclosed differences in immunoreactivity to Bcl-2 in

Alzheimer's disease and Down syndrome brain tissues in comparison with controls allowed us to suggest that Bcl-2 represents one of the factors engaged in the pathogenesis of structural lesions in both these disorders.

IMMUNOHISTOCHEMICAL CHARACTERIZATION OF SENILE PLAQUES IN THE BRAIN STEM

Dr. Elżbieta Kida

Immunohistochemical characterization of senile plaques in the brain stem revealed that early, diffuse Aβ deposits in this brain region do not show, similarly to the neostriatum, apo E immunoreactivity. By immunoelectron microscopy it was shown that diffuse plaques in the brain stem, like in other brain regions previously studied, contain Aβ in a fibrillar form.

IMMUNOHISTOCHEMICAL AND GENETIC STUDIES OF JUVENILE FORM OF NEURONAL CEROID LIPOFUSCINOSIS

Dr. Elżbieta Kida

Immunohistochemical and genetic studies of juvenile form of neuronal ceroid lipofuscinosis allowed immunolocalization of a protein product of the CLN3 gene in the central nervous system, as well as identification of new mutations in this gene in atypical cases.

REGULATION OF CEREBROCORTICAL MICROCIRCULATION DURING REPERFUSION AFTER FOCAL CEREBRAL ISCHEMIA

Dr. Ewa Koźniewska-Kołodziejska

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The study which was planned to be carried out this year comprised endothelium-dependent regulation of cerebral microcirculation during early reperfusion (10-30 min) and during delayed hypoperfusion (2-3 hours after reperfusion) following focal cerebral ischemia (suture occlusion of the middle cerebral artery) of 30 or 90 min duration. The experiments performed until now helped to establish the time relation between the duration and degree of ischemia and the development of delayed hypoperfusion. It has been found that delayed hypoperfusion does not develop when ischemia lasts less than 3 hours and is not followed by hyperfusion. The decrease in microflow which is seen when NO synthesis is blocked in control rats does not occur in the early reperfusion after 30 min ischemia and, in fact an increase of microflow is observed instead. This result can not be, at present, interpreted as the abolishing of the normal response. It is more likely that the increase of

microflow following inhibition of NO synthesis may result from the increase of blood pressure associated with the administration of NO synthase inhibitor and the lack of normal autoregulation of flow due to the ischemic insult. This study is in progress and was not yet published.

IMPAIRMENT OF THE ENDOTHELIUM-DEPENDENT REGULATION OF CEREBRAL CIRCULATION AFTER CARDIAC ARREST-INDUCED GLOBAL CEREBRAL ISCHEMIA

Dr. Ewa Koźniewska-Kołodziejska

This study concerns participation of NO in the maintenance of basal cerebral blood flow and cerebrovascular reactivity to NO-dependent (acetylcholine), endothelium-dependent (ATP) and endothelium-independent (papaverine) mediators in rats who were subjected to 10 min global cerebral ischemia (cardiac arrest) 7 days before the study. It has been found that neither basal cerebral blood flow nor the participation of NO in the maintenance of basal cerebral blood flow in these rats is different from what has been observed in control. However, lack of the response of cerebral circulation to acetylcholine and decreased sensitivity to papaverine in the rats subjected to cardiac arrest suggests a selective impairment of vascular reactions.

IMPAIRMENT OF THE ENDOTHELIAL CONTROL OF CEREBRAL CIRCULATION DURING CHRONIC HYPERTENSION IN RATS

Dr. Ewa Koźniewska-Kołodziejska

In the study of the disturbances of cerebrovascular endothelium in genetic hypertension (spontaneously hypertensive rats) no abnormalities were observed as far as NO-dependent regulation of cerebral blood flow and cerebral vascular resistance is concerned. It appeared, however, that vasodilator prostaglandins do not participate in the regulation of basal cerebral blood flow and cerebral vascular resistance in spontaneously hypertensive rats to the same extent as in normotensive animals. The prostaglandin which seems to be responsible for the increase of basal cerebral vascular resistance in spontaneously hypertensive rats and for the maintenance of their basal cerebral blood flow at the level comparable to that in normotensive rats is PGH_2. The next important finding is that, in contrast to normotensive animals, the interaction between NO and prostaglandins is not essential for the maintenance of basal cerebral flow and cerebral vascular resistance in spontaneously hypertensive rats.

REGULATION OF CEREBRAL CIRCULATION DURING CHRONIC HYPONATREMIA

Dr. Ewa Koźniewska-Kołodziejska

The study was performed on male and female rats subjected to chronic hyponatremia induced with intraperitoneal administration of 5% glucose solution associated with subcutaneous administration of antidiuretic hormone. In previously published study we have reported that chronic hyponatremia induced in this way impairs cerebral blood flow exclusively in female rats and is associated with high mortality. On the basis of this year study we can conclude that chronic hyponatremia induced with vasopressin impairs endothelium-dependent, receptor-mediated responses in cerebral circulation in both genders but it has more pronounced impact on endothelium-dependent regulation of cerebral circulation in female than in male rats. Decrease of CBF (and increase of CVR) observed in hyponatremic female rats might be due to the impairment of endothelial production of NO. It is possible that these changes in cerebral vascular resistance and flow represent vasopressin-induced vasoconstriction which is unmasked in hyponatremic female (but not male) by a withdrawal of endothelium-dependent relaxing factor.

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CNS DEVELOPMENT IN NORM AND PATHOLOGY: CLINICAL, NEUROPATHOLOGICAL AND QUANTITATIVE CONSIDERATIONS

Prof. Maria Dąmbska

The maturation of neurons from hippocampal sectors CA1-CA4 and the development of vascularization during the second half of pregnancy were examined using morphometric methods. Fetal brains at 28, 32, 36, 40 weeks of developmental age were used. The examined parameters were: neuronal and vascular density, classification of neurons according to profile area of cell bodies, ratio of the nuclear to pericaryon area, distribution of vessels according to their size and area occupied by vessels.

It was found that not only the maturation of neuron but also the development of vascular net differ in the examined sectors. The vascular density was found to be lowest in CA1 sector, where the neuronal density is the highest until birth. Our results suggest the role of vascularization of developing Ammons horn in its susceptibility to hypoxic lesions.

Astroglial reactivity in the periventricular white matter at parietal level in newborns of 28-40 weeks of development was estimated by qualitative and quantitative examination by use of reaction with glial fibrillary acidic protein (GFAP). It was found that the early astroglial reaction coincides with the predilection of this area to perinatal brain damage.

The members of our group cooperate in the study concerning immunolocalization of two proteins Bcl-2 and Bax in developing, normal and Down syndrome brains. The results indicate that both may be involved in the final differentiation and maturation of neurons.

ULTRASTRUCTURAL AND IMMUNOHISTOCHEMICAL STUDIES OF THE BRAIN DEVELOPMENT DISORDERS. APOPTOSIS AND GLIAL REACTION IN PERINATAL PATHOLOGY INCLUDING GENETIC DISORDERS OF THE DEVELOPING BRAIN

Assoc. Prof. D. Maślińska

Cells participating in phagocytotic clearance have been studied. It is believed, that in the brain microglia is the main cell responsible for phagocytosis. During development microglia undergoes morphological transformation which is characteristic for its maturation within microenvironment of the nervous tissue. The aim of the study was to examine localization of the microglial cells in the brain during its development. Study was performed on the fetus brain at 14-40 weeks of gestation. The results provided evidence that localization of microglia is clearly related to the development of the brain structures. However morphological forms of these cells did not suggest the role of microglia to be confined to phagocytosis.

Moreover, phagocytotic clearance has been studied following indication of apoptosis in the brain of rabbits intoxicated by vincristine. No activation of microglia was observed in the brain of these animals. Numerous apoptotic bodies were engulfed and digested by different types of parenchymal cells including astroglia, oligodendroglia, microglia, pericytes and occasional macrophages. Our results lead to the conclusion that in the brain as in other organs a mechanism of phagocytotic clearance following apoptosis is different than after necrosis, which is always concomitant with inflammation and transformation of resting microglia to phagocytotic forms of the cell.

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LATE EFFECTS OF ISCHEMIA ON VASCULAR-TISSUE INTERFACE IN BARRIER-COMPETENT AND BARRIER-FREE REGIONS OF RAT BRAIN. ULTRASTRUCTURAL, HISTOCHEMICAL, AND IMMUNOCYTOCHEMICAL INVESTIGATIONS

Assoc. Prof. Barbara Gajkowska

The aim of the present studies was to investigate the effects of 10 min total brain ischemia on the vascular-tissue interface in barrier-free and barrier-competent regions of rat brain. The experiments were performed on cerebral cortex, hippocampus, and the hypothalamo-hypophysial system of rat brain. The investigations were focused on the late period after animal resuscitation after the ischemic insult (24 h – 12 months).

Ultrastructural and morphological observations documented the presence of a significant number of phagocytes in the perivascular area in the period 6-12 months after ischemia, when compared with control age-matched rats and with the animals studied at shorter times after ischemia. Two subpopulations of phagocytes were identified: 1/ the cells which remained adjacent to the capillary wall and separated from the brain parenchyma by basement membrane, and 2/ the cells which crossed the basement membrane. In the latter case, the phagocytes contained an increased number of phagolysosomes. Concomitantly, the number of microglial cells in neuropil increased. The morphological features of microglia, i.e. numerous lysosomes and lipofuscin granules and frequent mitoses, indicated their increased activity.

In conclusion, the number of phagocytic cells remains increased during a long period after ischemia, a finding which can be interpreted as a morphologic manifestation of increased phagocytosis and enzymatic degradation of the damaged neuronal elements.

Histochemical studies were performed to further characterize the subpopulations of brain phagocytic cells. The new method of identification of thiamine pyrophosphatase as a marker of cell membrane of activated phagocytes, has been successfully implemented. The electron-dense product of the histochemical reaction was deposited on the surface of the phagocytes and cytoplasmic projections of the putative brain phagocytes.

In parallel, the post-embedding immunocytochemistry for electron microscopy was performed. The monoclonal antibodies specific for endothelin 1, 2, and 3 labeled with 15 nm colloidal gold particles were employed. The observations were performed between 24 h and 12 months after ischemia to determine dynamics of the development of post-ischemic alterations in different parts of rat brain. The regions where the blood-brain barrier was competent were compared with those of an incompetent barrier. The results indicate that ischemia leads to the protracted elevation of endothelin activity in multiple neural elements of the CA1 hippocampal region and in the hypothalamo-hypophysial system of rat brain. The shrunken neurons, astrocytes, perivascular phagocytes and microglia of the hippocampus were strongly labeled with the anti-endothelin antibodies 6 and 12 months after ischemia. It is conceivable that elevated endothelin expression stimulates cerebral blood perfusion and thus is likely to aggravate post-ischemic encephalopathy.

Expression and distribution of endothelin 1, 2, and 3 were investigated also in the hypothalamo-neurohypophysial system of the rat before and after ischemia. It was found that ischemia stimulates the endothelin expression in some neurons of the supraoptic (SO) and paraventricular (PV) nuclei, astrocytes, endothelium, pericytes, perivascular macrophages and microglia. Strong endothelin immunoreactivity was observed as long as 6 months after ischemia. Twelve months after ischemia enhanced endothelin immunoreactivity was detectable only in phagocytes, and the immunolabeling of other cell types was comparable to the control. It can be speculated that endothelin in SO and PV neurons may affect and regulate hormonal synthesis and release (oxytocin and vasopressin) in the hypothalamo-neurohypophysial system. This hypothesis is reinforced by the fact that endothelin immunoreactivity could be observed in the axonal endings of neurohypophysis, co-localizing with vasopressin and oxytocin. Strong endothelin activity was detected also in pituicytes in neurohypophysis, and in endothelium and macrophages up till 6 months after ischemia. It is therefore likely that endothelin released after an ischemic insult may penetrate to the circulation acting as a classical hormone.

Additionally, the following morphological studies were performed:

1/ ultrastructural assessment of the influence of ethanol in combination with calcium antagonists, verapamil and nifedipine, on rat myocardium. It was shown that verapamil enhances ethanol cardiomyopathy, whereas the deleterious effects of ethanol were reversed by nifedipine.

2/ ultrastructural changes in the optic and ischiadic nerves were examined in EAE rats;

3/ studies on the ultrastructure of blood vessels in the superficial layers of the cerebral cortex of anoxic rabbits treated with calcium antagonist, dotarizine;

4/ transmission and scanning electron microscopy studies upon efficacy of a novel photochemical method for induction of cerebral ischemia;

5/ effects of squalene on the ultrastructure of the optic and ischiadic nerve.

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THE DIAGNOSIS OF INTRACRANIAL HOMEOSTASIS DISTURBANCES: A/ HEMODYNAMIC DISTURBANCES IN EFFECT OF CEREBRAL ISCHEMIA OF DIFFERENT DEGREES; B/ COMPREHENSIVE DIAGNOSTICS OF CEREBROSPINAL FLUID CIRCULATION DISTURBANCES; C/ ANALYSIS OF ELECTRO-PHYSIOLOGICAL DATA – EEG AND EVOKED POTENTIALS IN THE WAY OF MAPPING IN PATIENTS WITH FOCAL BRAIN LESIONS

Prof. Zbigniew Czernicki

In animals subjected to global ischemia distinct cerebrovascular reactivity patterns were found in the different brain areas, on the basis of CT scans morphometrical data, characteristic indices for hydrocephalus and cerebral atrophy were established, new method of the non-valve ventriculo-lumbar drainage in patients with obstructive hydrocephalus was introduced in the clinical practice.

In the experimental part of the cerebrovascular reactivity research, further studies concerning Ca^{2+} channel bloker – Dotarizine – were continued. In the present series of experiments the influence of Dotarizine administered orally in a chronic way was studied. It was found that Dotarizine prevents cerebral vasoconstriction due to hyperventilation and causes the increase in vascular dilatation under anoxic conditions. In the other experimental series regional differences of cerebrovascular reactivity are investigated. In the experiments with global ischemia in rabbits it was found that following global ischemia the basilar artery reactivity remained undisturbed while middle cerebral artery reactivity was considerably disturbed.

In the clinical studies cerebrovascular reactions due to endovascular procedures were investigated. Using transcranial Doppler sonography the constriction of a vessel subjected to such a procedure is caused by mechanical irritation of the vessel and lasts up to 48 hours.

In the evaluation of patients with brain contusion the time onset of secondary intracerebral hematomas in the primary contusion foci was investigated. Repeated CT examinations showed that hemorrhagic transformation could occur until 72 hours following trauma.

In clinical studies concerning the disturbances of CSF circulation, a multistage method of the low-pressure hydrocephalus diagnosis was improved. The diagnostic

stage of morphometric analysis of CT images was further developed and values of the indices characteristic for hydrocephalus and cerebral atrophy were calculated. Additionally, for the particular cases of obstructive hydrocephalus a new non-valve ventriculo-lumbar drainage method was developed and clinically applied. A method of linear connection of the valve systems was also developed in order to treat overdrainage syndrome particularly in severely ill patients and patients with serious operative contraindications.

Electrophysiological studies included EEG mapping and somatosensory evoked potentials (SEP) in cases of focal brain lesions, brainstem and cerebellar lesions, and cervical discopathias. SEP examinations in patients with focal brain lesion showed that the decrease of early amplitude components and the disturbances of potential propagation are important lesion indicators of prognostic value. In patients with brain stem lesions, specific late potential components were found. SEP mapping showed that the area of cortical excitation is wider on the side of the brainstem compression and the cortical excitation persists longer on the conralateral side. In cases of cervical discopathia latency evaluation in SEP examinations appear to be a prognostic indicator of the degree of radicular compression.

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STRUCTURE OF NORMAL AND DISEASED SKELETAL MUSCLE AND PERIPHERAL NERVE

Prof. Anna Fidziańska-Dolot

The project concentrated on:

1/ Investigation of apoptosis in normal fetal human muscle, in neonates with acute form of spinal muscular atrophy (SMA) as well as in experimentally induced apoptosis in neonatal rat skeletal muscle. It was shown that apoptosis in fetal life is a two-step process, regulating both the number and the size of muscle cells. It is suggested that in pathological conditions, such as SMA in children or experimentally injured neonatal rat skeletal muscles, immature muscle fibers react to noxious stimuli with apoptosis.

2/ Ultrastructural studies of experimentally induced muscle regeneration in senile rats. The results clearly showed that senile muscle was able to regenerate in response to injury. The regeneration proces in senescence, however, was slower, distempered and markedly impaired as compared to young adult animals.

3/ Age-related muscle stiffness in rats. It is suggested that the muscle stiffness is not due to a reflex response, but depends mainly on non-reflex factors, chiefly on large overgrowth of non-elastic connective tissue replacing degenerated active muscle fibers.

4/ Ultrastructure of sural nerve in patients with chronic demyelinating polyneuropathy. The changes were observed to be similar to those seen in Guillain-Barré syndrome. The presence of onion-bulb structures indicated chronic character of the pathological process.

Additionally, clinical and morphological characteristics of congenital myopathies as well as detailed clinical and morphological analysis of central core, rod and myotubular myopathy were described.

STRUCTURAL AND FUNCTIONAL CHARACTERISTICS OF DYSTROPHIC SYNDROMES (NONTYPICAL DYSTROPHINOPATHIES EXCEPTED)

Assoc. Prof. Irena Niebrój-Dobosz

Comparative biochemical and immunochemical studies in Duchenne's dystrophy and its experimental model in mdx mice do not support the opinion of the basic role of lack of dystrophin in provoking the dystrophic process. Electrophysiological studies indicate that a typical primary myopathy, such as Duchenne's dystrophy, is characterized by changes in motoneurons, either because of their degeneration, or as a cause of the morbid process itself. Genetic studies indicate on the possibility of the detection of the mutation of dystrophin gene in RFLP polymorphism and CA sequence analyses allow to detect the dystrophin gene mutation both in Duchenne's dystrophy and Becker's dystrophy. Molecular genetics techniques, used in the examinations of limb girdle dystrophy patients, detected mutations in the gene for CANP3, localized on the 15 q chromosome. An enigmatic mechanism is supposed to be present in part of these patients. Analysis of the appearance of IgG intrathecal synthesis in ALS patients, conducted in the years 1994-1995 has been completed pointing to the autoimmune character of the disorder in some of these patients.

CHANGES IN THE MOTOR UNIT IN NEUROMUSCULAR DISEASES

Assoc. Prof. Katarzyna Rowińska-Marcińska

Analysis of MUAP registered in cases of myopathic and neurogenic muscle lesions allowed to propose a terminology for potentials with multiple picks. All non simple potentials that is potentials with more then 5 turns and/or more then 4 phases are defined as "irregular" potentials and limit the term "complex" to potentials consisting of at least one satellite accompanying the main potential component.

Examination of Double MU discharge shape revealed a modification of the second component which may appear either substantially reduced or irregular or otherwise dissimilar to the first one. Correlation between the internal interval, second component amplitude and jitter has been noted.

In conclusion we proved that precise analysis of the potential irregularity is helpful in differentiating slow and fast progressing process.

In addition retrospective comparative study of the electrophysiologic changes in CBS and CIDP polyneuropathy have been performed. In all cases nerve stimulation data fulfil the diagnostic electrophysiologic criteria for demyelinated inflammatory polyneuropathy. However, individual differences in electrophysiologic features and in the course of the disease prohibit the use of nerve stimulation data for differentiating between GBS and CIDP cases.

GENETICO-CLINICAL CORRELATION IN SMA

Prof. Irena Hausmanowa-Petrusewicz

The revision of prognosis in early-onset childhood SMA (type I) revealed that in this form, up to 10% children can survive over 4 years. Some children survive even up to 20 yrs being however unable to seat. This means that the prognostics *quo ad vitam* in this group is not entirely negative.

Analysis of a cohort of 573 patients demonstrated that in some chronic cases of SMA creatine kinase (CK) may be quite high. It was also shown that childhood SMA is a gender-influenced disease. There were significantly more males in the late-onset group III, and in no female the disease started after the age of 8 yrs.

Electrophysiological analysis of EMG records in different forms of SMA indicated the capacity of preserved motoneuron to reinnervate the denervated muscle fibers in SMA III and the inability of motoneuron to extend its innervation field in SMA I. This would explain the absence of compensation in this form of SMA.

MUSCLE PATHOLOGY IN GENERAL IMMUNOLOGICAL PROCESSES

Prof. Irena Hausmanowa-Petrusewicz

Idiopathic inflammatory myopathies (IIM) are a heterogenous group of diseases of unknown etiology. It is through that genetic factors and environmental agents, which may induce cellular and humoral immune response and chronic muscle inflammation, play a role in the pathogenesis.

In our series of 84 patients with IIM we found myositis specific antibodies (MSA): antisynthetases, anti-SRP, anti-Mi-2 and myositis associated antibodies: anti-PM-Scl and anti-Ku. Antisynthetases were associated with severe myositis and interstitial lung disease, anti-Mi-2 was a marker of classic, benign dermatomyositis with good response to therapy and anti-SRP was found in the case of very severe myositis, nonresponsive to aggressive immunosuppressive therapy.

We found increased frequency of DQA1*0501 and DRB1*0301 alleles in patients with IIM.

Summary: MSA were found less frequently in Polish patients with IIM compared to North American and Japanese populations. MSA can be useful in diagnostics of IIM because of its specifity for distinctive diseases, and helpful in differentation, prognosis and choice of therapy. DQA1*0501 and DRB1*0301 alleles are present more frequently in patients with IIM.

N-CAM AS A MARKER OF MUSCLE AND NEUROMUSCULAR JUNCTION IMMATURITY IN CHILDREN WITH CONGENITAL MYOPATHIES

Prof. Anna Fidziańska-Dolot

Supported by the State Committee for Scientific Research: grant # 4 S405A 005 06

Clinical and morphological studies performed during the last year allowed us to diagnose a rare congenital myopathy, a cardiomyopathy with cytoplasmic inclusions (cytoplasmic body myopathy). Immunocytochemical studies of the biopsied muscle suggested that the basic component of inclusions was smooth muscle actinin with only a small amount of sarcomeric actinin. A changed composition of Z-line in muscle is a probable cause of excessive proliferation of actinin and production of Z-line material.

CLINICO-GENETIC CORRELATION IN RECENTLY RECOGNIZED DYSTROPHINOPATHIES

Prof. Irena Hausmanowa-Petrusewicz

Supported by the State Committee for Scientific Research: grant # 4 P05B 113 08

DNA tests and qualitative and quantitative evaluation were performed in 47 families with Becker muscular dystrophy. A dystrophinopathy was described with DNA deletion but with abortive clinical expression – some times skeletal muscles are spared and the only symptoms are cardiac failure or CK-emia. These findings are not only a contribution to the new aspect of X-linked dystrophy, but they are also of practical significance. The molecular tests are helpful in establishing diagnosis in difficult and atypical cases. It is conceivable that in the "premolecular" area the atypical cases were misdiagnosed, and the frequency of this variety was underestimated.

Clinical and laboratory examinations of females – obligatory carriers of DMD, and rechecking the group of the limb-girdle dystrophy revealed that the clinical expression of X-linked dystrophy in also possible in females. This finding appears to be of great importance in that it changes the presently accepted criteria of genetical counselling.

The results underscore the need to recheck the diagnoses of LGMD made before dystrophin testing was introduced.

ANGIOGENESIS AND MYELINATION IN THE CENTRAL NERVOUS SYSTEM

Prof. Janina Rafałowska

Supported by the State Committee for Scientific Research: grant # 4 S402 071 06

Examinations of human fetal and neonatal spinal cord in hematoxilin-eosin (HE) staining and with antibodies to myelin basic protein (MBP) and glial fibrillary acidic protein (GFAP) were completed. For visualisation of blood vessels *Ulex europeus* lectin was used. Astrocytic reactivity and increase of vascularisation within white matter of the spinal cord during myelination were found.

Cervical segment of the spinal cord in 1, 3, 6, 9, 12, 15 days-old Wistar rats was analysed. Astrocytic reactivity was noted in the course of rat spinal cord myelination, similarly to human spinal cord. On the 15th day after birth rat spinal cord myelination was still very weak.

Specimens od rats lumbo-sacral spinal cord aged from 1 day to 15 days were taken for electron microscopy examination. Estimation of blood vessels was performed. Numerous microvilli on luminal surface of blood vessels endothelium were observed.

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HEMODYNAMIC, METABOLIC AND NEUROHORMONAL RESPONSES TO PHYSIOLOGICAL STIMULI: EFFECT OF PHYSICAL ACTIVITY AND ENVIRONMENTAL FACTORS

Prof. Krystyna Nazar

The investigation was continued on factors modifying psychomotor performance during physical exercise and on the influence of low ambient temperature on multiple choice reaction time (RT) in young well fit men during graded exercise until volitional exhaustion. The study showed an improvement of psychomotor performance at 2°C in comparison with results obtained at 22°C. In the cold, RT was shortened at submaximal work-loads and its increase occurred at higher exercise intensity than under the thermoneural conditions. The results suggest a higher level of arousal with reduction of central fatigue during work in cold environment. In subsequent series of experiments in the same subjects an effect of caffeine ingestion (5 mg/kg b.m.) on exercise-induced changes in RT were studied at neutral (22°C) and low (2°C) ambient temperatures. A beneficial effect of caffeine on psychomotor performance was found only during submaximal loads of the exercise performed at 22°C.

The activity of the autonomic heart innervation was evaluated on the basis of heart rate variability (HRV) analysis in the supine and standing position in 19 young untrained subjects and 6 elite swimmers. The data suggest that intensive training causes a reduction of the sympathetic nervous system activity in the supine position with an enhancement of this system reactivity to the orthostatic stimulus.

In cooperation with the Outpatient Cardiac Unit for Diagnosis and Therapy at our Institute the usefulness of HRV indices in examination of autonomic heart innervation was analyzed in elderly patients with coronary heart disease. The simple HRV indices, such as eg. maximal increase in the R-R interval during respiratory cycle, appeared to be highly sensitive to changes in the parasympathetic activity in response to orthostatic manoeuvre.

The dynamics of stroke volume and cardiac output responses to graded exercise were studied in healthy untrained subjects using an impedance rheography with an own original program of computerized continuous recording and analysis of ECG

and rheographic signals. The study demonstrated the usefulness of this method in evaluation of the hemodynamic changes during exercise tests.

In our previous study it was documented that during graded exercise plasma concentration of growth hormone (hGH) shows a nonlinear increase with a threshold at the exercise load close to the blood lactate (LA) threshold. The aim of the present investigation was to find out whether this response depends on the working muscle mass. The time course of exercise-induced blood LA and hGH changes was followed in young subjects performing graded exercise with both legs (ET1), one leg (ET2), and one arm (ET3). The blood LA threshold could be detected only during ET1, whilst in the remaining tests the blood LA concentration increased linearly with exercise intensity. The general patterns of exercise-induced changes in the plasma hGH were similar in all tests. The threshold occurred at lower exercise loads during ET2 or ET3 than during ET1, but at similar blood LA levels. The data indicate that the magnitude of plasma hGH response to exercise depends on the muscle mass engaged in exercise, and suggest a relation between hGH threshold and exercise-induced acidosis.

The effect of low ambient temperature and caffeine ingestion (5 mg/kg) on the time course of blood LA changes during graded exercise was examined in healthy, well fit subjects. It was found that cold exposure (2^0C) does not influence the blood LA threshold, and that caffeine shifts the threshold towards higher work intensities only in thermoneutral conditions (22^0C). Both in the cold and thermoneutral environments caffeine increased the blood LA levels at high exercise loads. This study indicates that caffeine does not increase endurance working ability in the cold.

The effect of caffeine on supramaximal working ability was investigated in cooperation with the Department of Physiology, University in Kuopio (Finland). It was demonstrated that caffeine increased the mean power and blood (LA) concentration attained during the 60s "jumping test" but did not affect the maximal power output. The results indicate that caffeine increases the anaerobic capacity.

A new modification of anaerobic threshold determination was elaborated basing on changes in heart rate (HR) during graded treadmill exercise. This modification, including mathematical transformation of a relationship between HR and work load, increases sensitivity of the anaerobic threshold detection in comparison with the commonly used Conconi test.

NEURO-HORMONAL CONTROL OF LIPID AND CARBOHYDRATE METABOLISM IN SKELETAL MUSCLE

Assoc. Prof. Leszek Budohoski

An influence of catecholamines on insulin dependent glucose utilisation by the soleus muscle was studied *in vitro* in rats treated for 7 days with β-adrenergic block-

ing agent (propranolol), after adrenalectomy or adrenodemedullation. It was shown that prolonged β-adrenergic blockade diminishes the responsiveness of lactate production and glycogen synthesis to insulin, however the sensitivity of both these processes to insulin was not affected. In muscles dissected from adrenalectomized animals the responsiveness of lactate production to insulin was decreased, whilst a small, significant improvement of the sensitivity of this process to insulin was shown. The rate of glycogen synthesis was not affected. In adrenodemedullated rats neither sensitivity nor responsiveness of lactate production to insulin was affected, whereas the rate of glycogen synthesis was significantly enhanced at all insulin concentrations. The sensitivity of the latter process to insulin was unchanged. It is suggested that catecholamines may take part in the control of muscle glucose utilisation due to their effect on fatty acid availability.

The effect on muscle glucose utilization and its sensitivity to insulin of muscle glycogen depletion in rats caused by food deprivation for up to 72 h was examined in the soleus muscle *in vitro*. It was shown that prolonged starvation reduces the basal blood glucose concentration *in vivo* and enhances the rate of lipid mobilization, as indicated by an increased plasma level of fatty acids and β-hydroxybutyrate. A decrease in intramuscular glycogen content caused an enhancement of glucose utilisation by the soleus muscle, both stimulated and non-stimulated by insulin. A strong negative correlation was found between the glycogen content and the basal rate (1 μU/ml INS) of lactate production, glucose transport and glycogen synthesis. A significant positive correlation was found between the intramuscular glycogen content and the sensitivity of lactate production, glucose transport and glycogen synthesis to insulin. The data demonstrated that food deprivation with an accompanying decrease in muscle glycogen content significantly modifies the muscle glucose utilisation measured under basal conditions as well as the sensitivity of glucose transport, lactate production and glycogen synthesis to insulin. The observed changes are negatively correlated with the soleus muscle glycogen content.

In the continued investigations on the relationship between the fatty acid (FA) uptake from the medium and the concentrations of a monomeric form of this compound (unbound to albumin, AUFA) a perfused rat's hindlimb preparation was used. By perfusing muscles with various concentrations of palmitate (1.0-2.0 mM) and different concentrations of albumin (4%, 6%, 8%) we were able to prove that the rate of incorporation of palmitic acid into the intracellular triacylglycerol pool depends directly on the concentration of the albumin unbound portion of fatty acids.

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

Our previous study showed that low-carbohydrate (L-CHO) diet, inducing moderate ketosis, shifts lactate threshold (LT) towards higher exercise loads and elevates post-exercise plasma catecholamine levels as compared to the values after the control (M) mixed diet. The purpose of the present investigation was to examine the effect of L-CHO diet on the time-course of plasma adrenaline (A), noradrenaline (NA), testosterone (T), and growth hormone (HGH) changes during incremental exercise performed until volitional exhaustion. The data showed that (1) during incremental exercise plasma concentrations of catecholamines as well as of HGH and T show nonlinear increases with thresholds, that under normal dietary condition, are close to or exceed the lactate threshold, (2) reduced CHO intake enhances activity of the sympathetic nervous system and lowers the threshold of its activation, (3) after L-CHO diet plasma growth hormone concentration is increased, while that of testosterone is diminished at rest and during exercise, however, the thresholds of both hormones are elevated similarly as the LA threshold. The above findings indicate that dietary modifications not only influence blood hormone levels but cause also changes in the relationship between the endocrine response to exercise and its intensity.

In a further investigation the effect of L-CHO diet on exercise thermoregulation was evaluated in men and in women in two phases of menstrual cycle. The data obtained indicate that carbohydrate restriction increases heat load during heavy exercise without changing the magnitude of body temperature response due to accelerated rate of sweating. These changes were more pronounced in men than in women, particularly in the follicular phase of the menstrual cycle.

LIPID METABOLISM IN SKELETAL MUSCLE OF PERFUSED RAT HINDLIMB

Assoc. Prof. Leszek Budohoski

Supported by the State Committee for Scientific Research: grant # 6 P04C 033 10

Optimal conditions allowing the measurement of intracellular triacylglycerol fatty acid and phospholipid pools in the perfused rat's hindlimb were established. The rates of incorporation of fatty acid into triacylglycerol and phospholipid pool as a function of the perfusion time and of the total concentration of palmitic acid in the perfusion medium were measured. An attempt was made to work out an experimental protocol allowing estimation of intracellular fatty acid content. Skeletal muscles of different fibre type composition as well as an adipose tissue were examined.

The results obtained will be compared with the reference data obtained at the Department of Biochemistry in Oxford.

THE EFFECT OF INSULIN-LIKE GROWTH FACTOR ON SKELETAL MUSCLE SENSITIVITY TO INSULIN

Assoc. Prof. Leszek Budohoski

Supported by the State Committee for Scientific Research: grant # 4 P05B 103 10

The effect of physical exercise of various intensity and duration on the IGF I and IGF II concentration in plasma and skeletal muscles of different fibre types was studied. All samples were collected and are now assayed at the Department of Pediatrics, John Radcliff Hospital in Oxford.

The effect of IGF I on the sensitivity of lactate production, glucose transport and glycogen synthesis to insulin was measured in the rat soleus muscle *in vitro*. A possible modulation of the observed effects by adenosine receptor agonists eg. 2-chloroadenosine was tested.

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

Dr. Gerard Cybulski

Supported by the State Committee for Scientific Research: grant # 8 S506 013 05

A new ambulatory monitoring system consisting of an universal 4-channel recorder enabling simultaneous collection of ECG and central hemodynamic signals was constructed. The system is based on 80C552 family controller with built-in analogue to digital converters and 20MB PCMCIA (type II) FLASH MEMORY CARD is used for data storage. The communication with the system is performed via specialised keys and a small, built-in LCD. We designed and constructed a miniaturised, tetrapolar, current impedance cardiography device with built-in one channel of ECG, which was applied as a detector of the central hemodynamic signals. The device enables a full disclosure of collected data. The specialised Windows based graphic user interface allows for data presentation in the normal and tape mode. Additionally, full disclosure data (1 hour recording/A4 page) and selected strips may be hard copied on a laser or jet printer. The system allows for off-line, beat-to-beat automatic evaluation of cardiac output, stroke volume, ejection time, pre-ejection period and heart rate. The system could be used for evaluation of cardiac contractility and hemodynamic characteristics of cardiac muscle in both research and sports medicine. Further clinical examinations could allow the use of this device as a supplementary diagnostic tool for cardiologists.

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ENDOTHELIN AND URODILATIN IN THE CONTROL OF RENAL HEMODYNAMICS AND RENAL EXCRETION

Prof. Janusz Sadowski

The role of endogenous endothelins (ET) in the control of cardiovascular system and renal function and ET interaction with urodilatin (URO) were studied in anesthetized female Wister rats. The ET activity was blocked using PD 145065 (later briefly PD), a non-selective antagonist of ET_A and ET_B receptors, given i.v. at 5 $mg \cdot kg^{-1}$ body wt. URO was infused i.v. at 0.1 $nmol \cdot min^{-1} \cdot kg^{-1}$ body wt., alone or after ET receptor blockade.

PD decreased mean arterial blood pressure (MBP) from 114±4 to 109±4 mm Hg and the renal blood flow (RBF) from 6.6±0.3 to 5.8±0.4 $ml \cdot min^{-1}$ ($P<0.02$) and increased renal vascular resistance (RVR) from 17.7±1.2 to 20.1±2.1 mm $Hg \cdot min \cdot ml^{-1}$. A decrease in MBP in the absence of changes in HR, as observed after PD, indicated a reduction of the total peripheral vascular resistance (TPR). The present data indicate that under our experimental conditions the systemic vasculature was under a vasoconstrictor tonus of ET. In contrast to the decrease of TPR, RVR tended to increase after the inhibition of ET receptors. This suggests that under our experimental conditions endogenous endothelins act as renal vasodilators, in contrast to their prevailing vasoconstrictor effect on systemic vasculature as a whole.

Renal excretory function was not affected by the inhibition of endogenous ET, however, it is interesting that the high variability of sodium excretion in animals under control conditions was clearly abolished by PD. These data suggest that endogenous endothelins may moderately influence the tubular sodium reabsorption.

After pre-treatment with PD, infusion of URO decreased MBP more than did URO alone: 15±3% vs. 7±2% ($P<0.05$). RVR and HR did not change after URO alone but decreased with URO given to PD treated rats (19±5% and 14±3%, respectively, $P<0.01$). Thus, the blockade of ET receptors prevents the usual attenuation of URO effects by ET.

During URO infusion the increase in urine flow and solute excretion were more rapid after PD treatment than that observed without blockade of ET receptors. These data raise the possibility that endogenous renal ET may diminish the responsiveness of the renal excretory function to natriuretic peptides. Probably, both peptide

families affect the same tubular sites and possibly antagonise the effects of each other.

It is concluded that in anesthetised surgically prepared rats endogenous ET can cause renal vasodilatation, in contrast to constriction of systemic vasculature. The enhancement by ET blockade of vascular systemic and renal effects of URO supports the ET interaction with natriuretic peptides in the control of cardiovascular and renal function.

ROLE OF RENAL MEDULLARY CIRCULATION IN MODULATION OF THE CORTICO-MEDULLARY ELECTROLYTE GRADIENT

Prof. Janusz Sadowski

Supported by the State Committee for Scientific Research: grant # 4 P05A 013 08

Responses of the renal medullary blood flow and medullary total ion concentration to increased filtered load of NaCl: data from simultaneous recording.

Total interstitial ion concentration (tissue electrical admittance) and local tissue blood flow (laser-Doppler probe) were simultaneously recorded in the renal medulla of 18 anesthetized Wistar rats under control conditions and after increasing the filtered NaCl load by an infusion of 5% NaCl into the aorta just opposite the left renal artery. Both parameters were measured using the integrated sensor of an own design, previously shown to be little invasive. The renal perfusion pressure was maintained constant at about 105 mm Hg using a suprarenal screw-controlled snare placed on the aorta.

An infusion of 5% NaCl increased the interstitial ion concentration (tissue admittance) from 938±31 to 1004±33 μS (+7%). Unexpectedly, the renal medullary blood flow, renal cortical blood flow (laser Doppler probe on kidney surface) and the glomerular filtration rate (clearance of tritiated inulin) were not significantly altered. During hypertonic NaCl infusion the urine flow increased 3-fold, sodium excretion increased 4-fold, and urine osmolality decreased from 950±97 to 770±52 mosm/kg H_2O (all changes significant).

The data indicate that salt loading did not change the renal hemodynamics, probably due to an equilibration of two opposing influences: of extracellular volume expansion (tending to increase hemodynamic parameters) and of activation of the tubulo-glomerular feedback mechanism (tending to suppress the glomerular filtration and reduce renal blood flow). In the absence of any increase in the medullary blood flow (no wash out of tissue ions), increased delivery of NaCl to the ascending limb of Henle's loop resulted in an increased NaCl reabsorption in this segment, increased supply of salt to the interstitium and a moderate elevation of medullary ionic hypertonicity (tissue admittance).

The ultimate change was not probably influenced by modulatory action of hormonal and autacoid systems, which requires further investigations.

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ADAPTATION TO EXERCISE IN PATIENTS WITH HEART FAILURE: RELATION OF HEMODYNAMIC PARAMETERS TO CORONARY INSUFFICIENCY AND HEART RATE VARIABILITY

Dr. Ewa Wójcik-Ziółkowska

Diminished exercise capacity of patients with heart failure (HF) is due to abnormal left ventricle function and coexisting disorders of skeletal muscle metabolism. However, the mechanism of low exercise tolerance in HF patients is not fully recognized. No correlation was ascertained between resting parameters of left ventricle function, such as systolic and diastolic diameters, ejection fraction, filling pressure, and exercise capacity or HF degree. The widely used NYHA classification of HF based on subjective symptoms has been questioned.

Objective estimation of the left ventricle function during exercise is of great importance for evaluation of the disease progression and monitoring of therapy. Moreover, exercise testing helps to establish recommendations for the patients' daily life activity and in some cases for physical training. The beneficial effect of low intensity training has been described in some recent reports.

The aims of the present study were: 1/ to compare exercise capacity of patients with HF after myocardial infarction (MI) and those with HF without MI, relating the obtained results to NYHA classification, 2/ to investigate hemodynamic responses to exercise in those patients, 3/ to determine the relationship between the ischemic threshold and the degree of heart insufficiency. Thirty five men (mean age 64 years) with HF symptoms were examined. This group included 25 patients after MI (MI+) and 10 patients without MI (MI-). Twenty two patients were qualified to class II and 13 to class III according to NYHA classification. In all patients cardiac enlargement was documented by X-ray (thoracic ratio > 0.55) and echocardiography at rest showed left ventricle dysfunction (ejection fraction < 50%). There were no differences between M+ and M- groups in the left ventricular function indices at rest. The patients were submitted to the symptom limited graded treadmill exercise test. Oxygen uptake (VO_2) and cardiac output (CO) were measured using Sensor Medics 2900 with supplementary NICO equipment. Besides, ECG was monitored, heart rate (HR) was recorded continuously and blood pressure was measured every 2 min.

No relationships were found between indices of left ventricle function at rest and the hemodynamic responses to exercise. In most cases, the estimated exercise capacity of patients and their classification according to NYHA criteria showed discrepancy. Exercise tolerance was similar in MI+ and MI- groups (approx. 60% of predicted VO_2max). Cardiac output increases during exercise were lower than normal values by approx. 30%. The symptoms of hemodynamic insufficiency (fall of blood pressure) or symptoms of exhaustion in most cases preceded the exercise ischemic threshold.

The results of the exercise test appeared to be helpful for establishing individual recommendations concerning permissible work loads in the daily activity of patients, irrespectively of the cause of HF and left ventricular function parameters at rest.

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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 (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 capacity in patients with stable angina. L-arginine (6 g/day) or placebo were administered to 22 randomized patients in a double-blind study. An exercise test (Marquette Case 12 treadmill system) with 12 lead ECG was performed according to the Bruce protocol.

In comparison with placebo, in the patients treated with L-arginine,
1/ exercise time to maximal ST segment depression increased ($p<0.002$)
2/ sum of maximal ST segment depression decreased ($p<0.04$)
3/ maximal workload (METS) increased ($p<0.006$).

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

LACK OF ANTIOXIDANT EFFECT OF CAPTOPRIL IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

Prof. Krystyna Cedro-Ceremużyńska

Captopril, an angiotensin converting enzyme (ACE) inhibitor, exerts a number of effects independent of ACE inhibitory properties including cardioprotection, attenuation of reperfusion-induced arrhythmias, endothelium-dependent vasodilator activity. It has been suggested that at least some of these effects are dependent on superoxide radical scavenging ability of the SH group in the captopril molecule. *In vitro* studies on the anti-free radical activity of captopril brought conflicting results.

Evaluation of the clinical value of the antioxidant properties of captopril seems of importance in view of common use of this drug in the acute phase of myocardial infarction (MI). We investigated, therefore, the effect of captopril upon the indexes of oxidative stress in the patients with acute MI, measuring plasma lipid peroxides and leucocyte oxygen free radical production. Of 63 patients with acute MI who entered ISIS-4, 33 were randomly assigned in a double-blind fashion to oral captopril (50 mg twice daily) and 30 to placebo.

The values of plasma lipid peroxides and leucocyte free radical production measured before and on day 3, 7, 14 of captopril/placebo treatment did not differ between the groups. The results provide no evidence for the antioxidant effect of captopril in the clinical setting of MI.

INITIAL ADRENERGIC RESPONSE IN ACUTE MYOCARDIAL INFARCTION INFLUENCES THE MAGNITUDE OF THE LEFT VENTRICULAR INJURY

Prof. Krystyna Cedro-Ceremużyńska

It has been well established that excessive response of the sympathetic nervous system in acute myocardial infarction (MI), manifested by increased blood and/or urinary catecholamines is associated with complicated clinical course and poor prognosis. Our early experimental studies have documented that catecholamines released into the blood after acute coronary occlusion, exert arrhythmogenic and cardiotoxic effects, increase infarct size and promote ischemia distant from the area of infarction. The question as to whether the intensity of sympathoadrenal activation at the onset of MI may influence the extent of myocardial damage in the clinical setting, remains to be answered.

The aim of this study (substudy of ISIS-4) was to investigate the relation between blood catecholamine concentration at the onset of MI and the magnitude of the left ventricular injury assessed by two-dimensional echocardiographic examination (ECHO). The study group consisted of 42 patients with acute MI. Catecholamines were measured by HPLC on day 1 of MI, ECHO was performed on day 3 and 14. Left Ventricular Wall Motion Score (LVWMS) was calculated. Blood adrenaline concentration on day 1 correlated positively with the magnitude of myocardial injury determined on day 3 and 14 ($p<0.032$ and $p<0.014$, respectively). Similar relationship has been found between blood noradrenaline level on day 1 and the degree of left ventricular damage assessed on day 3 and 14 ($p<0.021$ and $p<0.012$, respectively). The results suggest that the magnitude of catecholamine release in the early phase of MI contributes to the ultimate left ventricular injury.

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PHENOTYPES OF IMMUNE CELLS EXTRAVASATING TO SKIN

Prof. Waldemar L. Olszewski, Dr Hanna Gałkowska

Phenotypic characteristics of inflammatory cells in skin with lymph stasis.

Tissues with lymph stasis belong to the so called "immunologically priviledged site". They are prone to infections and tumor development (lymphangiosarcoma), presumably due to limited elimination of foreign antigens and lymphocyte recirculation. In group of 30 patients with lymph stasis all revealed inflammatory changes in skin. Main histological features were hyperkeratosis, acanthosis, increased density of Langerhans cells (CD1a+), DR+ keratinocytes, perivenular infiltrates of DR+, CD69+, CD4+ cells. Advancement of changes depended on the stage of lymph stasis. What was the etiological factor responsible for the inflammatory changes remains unclear. Immunohistochemical evaluation of skin biopsies points to bacterial infection as a causative factor.

Bacterial flora of tissue fluid, lymph and lymph nodes in lymph stasis.

Bacteriological cultures performed in a group of 38 patients with lymph stasis revealed presence of isolates in tissue fluid in 40% of cases, in lymph in 35% and nodes in 35%. The dominant bacterial strain was *Staph. epidermidis*. In the control group of 20 healthy volunteers no isolates were found. Bacteria present in tissues with lymph stasis may be responsible for the observed inflammatory changes in skin.

Preventional treatment with benzathine penicillin in skin inflammation of patients with lymphedema.

Forty five patients with lymphedema of the lower limbs were included in an open clinical trial. The duration of lymphedema before initiation of therapy was 7 months to 40 years. Recurrent episodes of infection occurred in benzathine penicillin treated group during one year follow-up in only 4 of 45 patients (9%). These data, although evaluated without a placebo group, suggest that long-term benzathine penicillin administration decreases the frequency of infection in patients with lymphedema.

KINETICS OF REPOPULATION OF BONE MARROW CAVITIES AND LYMPHOID ORGANS AFTER VASCULARIZED AND IN-SUSPENSION SYNGENEIC AND ALLOGENEIC BONE MARROW TRANSPLANTATION

Prof. Waldemar L. Olszewski, Assoc. Prof. Barbara Łukomska

A large proportion of BMCs transplanted as in-bone VBM graft or in-suspension into nonirradiated or lethally irradiated LEW rats accumulates in the recipients BM cavities. A portion of the BMCs accumulates in SPL and MLN. The mechanism of homing of BMCs to LO remains unclear. Presumably the hemopoietic fraction migrates to the BM cavities, whereas the lymphoid fraction homes to SPL and lymph nodes. Thirty to three hundred times less BMC released from the in-bone VBM graft, compared to i.v. injected BMC graft, bring about complete repopulation of LNs and SPL. It can be inferred that spatial functional relationship of hemopoietic and stromal cells in the VBM Tx with intact BM, results in physiologically regulated release to recipient's circulation of stem cells and hemopoietic cytokines. They probably mediate the rapid and complete repopulation of BM lymphoid organs. It was found that the BMCs homing to BM cavities do not reveal tendency to migrate out from BM and subsequently populate LO, in a fashion typical for lymphocytes. This seems logical since BMCs can function, proliferate and produce hemopoietic cytokines, only in spatial contact with BM stromal cells. The lymphoid cells possess a constitutive trait to recirculate between the LO, where they find the most convenient functional environment. Irradiation of the recipient evidently facilitated homing of BMCs to BM and LO in recipients of in-bone VBM Tx. It was not so expressed in rats receiving BMCs in-suspension. Presumably, the transplanted BM stromal cells together with BMCs, releasing hemopoietic cytokines, are responsible for the high homing rate of the transplanted BMCs. The rate of "homing of allogeneic BMCs in recipient BM, SPL and MLN is evidently lower than of syngeneic BMC, both after the in-bone and in-suspension grafting. Irradiation of the allogeneic recipient does not abrogate this phenomenon. Pretreatment of recipient with AAGM1 abrogates the allogeneic BMC cytotoxicity phenomenon, pointing to the NK cells as mediators of the allogeneic effect. A higher allogeneic effect after in-suspension than in-bone VBM Tx indicates that BMC transplanted with autologous stromal cells are less sensitive to the NK attack.

IMMUNE EFFECTS OF BLOOD TRANSFUSION

Prof. Waldemar L. Olszewski, Dr. Irena Grzelak

Transfusions of blood and its components are associated with the numerous alterations in the immune responsiveness of the recipient. The exact mechanism by which blood transfusions induce a state of the reduced immune responsiveness

remains unclear. Blood cells as well as humoral plasma components may play a role in this phenomenon. The effects of whole syngeneic and allogeneic blood transfusions as well as of syngeneic plasma on hematopoietic and immune systems have been reported previously. In the present study the effect of allogeneic plasma transfusion (ATP) on hemopoiesis and immune responsiveness was examined in a rat model. Normovolemic WAG rats were transfused intravenously 3 times with 2 ml of allogeneic (AUG) heparinized plasma, every other day, under light ether anesthesia. Three, 7 and 14 days after the last transfusion, blood from the vena cava, as well as spleens (SPL), mesenteric lymph nodes (MLN) and femur bone marrow (BM) were examined. We have found that transfusion of allogeneic plasma, almost doubling the circulating plasma volume, caused a significant decrease in WBC and splenocytes number/g tissue on day 3 after APT, and MLN cells number/g tissue on day 3 and 14 after transfusion. No significant changes in the percentage of cells of erythroid, myeloid and lymphoid lineage occurred during the entire observation period. No changes were also seen in the percentage of $OX7^-$ stem cells in blood, SPL, MLN and BM after APT. There was, however, a significant decrease in the percentage of $OX6^-$, MHC class II-positive cells in blood on days 3 and 7, in SPL on day 7, in MLN on days 3 and 14 and in BM on days 3, 7 and 14 following APT. The responsiveness of blood SPL and BM lymphocytes to PHA, ConA and PWM stimulation was significantly reduced almost during the entire observation period. Surprisingly, the responsiveness of MLN lymphocytes to mitogen stimulation was significantly increased. Results suggest that transfusion of allogeneic plasma strongly affects the immune reactivity in blood and lymphoid compartments, supplying an excess of plasma regulatory factors, as cytokines, shed receptors for cytokines and blockers of their receptors, soluble histocompatibility antigens and various proteins with blocking properties. Although present in low concentrations, they are very efficient in down-regulating immune processes in the blood circulation.

CELLULAR IMMUNE REACTION IN SKIN, LIVER AND PERITONEAL CAVITY IN NORMAL CONDITIONS AND IN ALLOGRAFTS

Prof. Waldemar L. Olszewski

The influence of biologically active factors on lymphocyte trapping in liver sinusoids.

Rat liver retains in sinusoids a population of blood cells, enriched in $3.2.3^-$, $CD8^-$ and MHC class II^- cells, displaying a high cytotoxic activity. We investigated the effect of gadolinium chloride (affecting liver Kupffer cells), xylocaine (stabilizing cell membrane potential), trypsin (influencing cellular receptors), sodium azide (affecting cell oxidation processes), and neuraminidase (removing of the sialic acid residues) on trapping of blood lymphocytes in the liver. It was found that the

number of lymphocytes retained in sinusoids of control liver was $1.07x10^6$ cells/g tissue. Administration of gadolinium chloride, trypsin and neuraminidase dit not evoke significant changes in the number of blood cells trapped in liver sinusoids, compared to control group ($1.08x10^6$, $0.8x10^6$ and $1.29x10^6$ cells/g tissue, respectively). In contrast, administration of xylocaine and sodium azide significantly diminished the number of trapped lymphocytes ($0.5x10^6$ and $0.3x10^6$ cells/g tissue, respectively).

Cyclosporin A decreases recruitment of lymphocytes in heart allograft.

It was found previously that thoracic duct lymphocytes pretreated with CsA reveal decreased migratory capacity to heart allograft. Studies of the mechanism of this phenomenon have shown that CsA decreases expression of L-selectin on lymphocytes. L-selectin is responsible for adhesion of lymphocytes to endothelial cells and subsequently extravasation to graft tissue.

Differences in rejection kinetics of heart and lymphocyte allograft after DST.

DST prolongs heart allograft survival time, at the same time accelerates lymphocyte allograft rejection. The question arises what may be the role of NK cells in this paradoxal two-directional process. Administration of anti-asialo-GM1 antisera specifically inhibiting NK cell cytotoxicity, resulted in slowing down the rejection of i.v. transplanted allogeneic lymphocytes.

CLINICAL AND EXPERIMENTAL CELL TRANSPLANTATION

Prof. Waldemar L. Olszewski

Supported by the State Committee for Scientific Research: grant # 6 P207 011 05

Rejection of organs, tissue fragment and cell allografts proceed along different pathways. Organ and tissue fragment grafting has been invented by humans and the process of recognition and rejection has not been evolutionarily developed. In contrast cells (bacterial, viral, fungal, maternal during pregnancy) penetrate tissues and are transported by the blood stream to the lymphoid organs. There, a process of natural elimination starts following the recognition of non-self antigens on the trapped cells. A similar process presumably develops after cell transplantation. Transplanted cells are either locally attacked by granulocytes and macrophages and destroyed or are carried by the blood to the lymphoid organs where they undergo a natural process of elimination. The results of our experiments substantiating this concept are presented.

The allogeneic cells transplanted intravenously or intratissueally are recognized by recipient immune cells and rapidly eliminated. This process constitutes a limitation to clinical transplantation of cells like Langerhans islets, hepatocytes, endocrine and neural cells. The mechanism of fast elimination remains unclear. In this study the kinetics of the elimination of intravenously injected allogeneic splenocytes were

eliminated for the most part within 6 hours. There were no significant differences in elimination between the allogeneic and control syngeneic nonlymphoid cells, however the recovery rate of these cells was extremely low. It seems that nonlymphoid cells are nonspecifically eliminated irrespective of whether they are syngeneic or allogenic. Their surface, uncovered with intercellular adhesion molecules, may nonspecifically attract granulocytes which express enzymatic cytotoxicity.

THE PATHOMECHANISM OF POSTTRAUMATIC EDEMA OF EXTREMITIES

Prof. Waldemar L. Olszewski

Supported by the State Committee for Scientific Research: grant # 4 P05C 037 10

The subject of the 1996 investigative programme was the role of the bacteriological factor in the development of the lymphangiographic and histological changes in lymphatics and lymph nodes. Sewen strains of bacteria: *Staphylococcus epidermidis*, *Staph. hominis*, *Staph. sciuri*, *Acinetobacter*, *E. coli*, *Corynebacterium*, *Streptococcus*, were injected s.c. into the paw of mongrel dogs, weekly, for a period of 6 weeks. Lymphangiographic pictures revealed presence of obliterative changes in afferent lymphatics. On histology, inflammatory infiltrates were seen in the lymphatic wall and accumulation of histiocytes in cortical and medullary parts of lymph nodes. Obliterative changes of small skin lymphatics were observed. Most pronounced were the changes in animals injected with *Staph. epidermidis*.

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

In our previous studies we have found out that metastatic adenocarcinoma cells in the liver did not express adhesion molecules. Low expression or lack of adhesion molecules on tumor cells can hamper killing of metastatic cells by infiltrating lymphocytes. Tumor cells can activate supressor cells which in turn may inhibit the responsiveness of other subsets of lymphocyte by secretion of immunosuppressive cytokines.

The present study was designed to investigate the effect of metastatic adenocarcinoma of the liver on the cytotoxic activity of peritoneal liver sinusoidal lymphocytes and to evaluate the activity of cytokines released by these cells. We showed that the level of cytotoxicity of peritumoral liver sinusoidal lymphocytes against RAJI cells was significantly lower than that of lymphocytes isolated from

the normal liver tissue. There was no difference between the level of IL-4, IL-10, IL-13, TGF-β1 (cytokines expected to contribute to the suppression of immune response), released by peritumoral liver sinusoidal lymphocytes cultured *in vitro* in the presence of K-562 cells and IL-2, in comparison to lymphocytes isolated from the normal liver tissue. In conclusion, adenocarcinoma metastatic foci induce protective immunity inhibiting cytotoxic activity of lymphocytes within the tumor environment. This defect seems not to be related to the secretion of immunosuppressive cytokines by peritumoral liver sinusoidal lymphocytes.

HUMAN IMMUNE PERITONEAL CELLS IN PERITONITIS AND DIGESTIVE TRACT TUMORS

Dr. Urszula Kubicka

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

In our previous studies on peritoneal fluid cells we found that normal peritoneal cavity contains a specific cell population consisting mainly of macrophages and T cytotoxic/suppressor cells. The question arises whether the neoplastic process developing in the gastrointestinal tract evokes changes in cellular composition of the peritoneal fluid. Preliminary observations indicate that colon tumor stimulated recruitment of myelomonoidal cells, whereas gastric cancer mainly elicited lymphocytes. The process of recruitment of immune cells is regulated by cytokines. We found significant expression in peritoneal macrophages of intracellular TNFα (+++/++++) (68.5% – gr I – adenocarcinoma, and 67.7% – gr II – adenocarcinoma of colon), and IL-1β (++/+++) (50.7% – gr I and 60.9% – gr II). There was only weak expression of these cytokines in controls (+/++) (TNFα – 26.3% and IL-1β – 0.0%). In small lymphocyte-like cells the expression of cytokines was slightly more pronounced in patients of gr I (TNFα – 39.7% and IL-1β – 52.9%) in comparison with gr II (TNFα – 38.7% and IL-1β – 28.0%) and controls (TNFα – 20.6% and IL-1β – 0.0%), IL-8 was expressed in a few peritoneal cells. Intracellular TNFα accumulated mainly close to the cell surface, whereas IL-1β was uniformly distributed in the cytoplasm. The presence of intracellular cytokines in peritoneal cells of cancer patients correlates with the previously reported expression of lymphocyte activation antigens on peritoneal cells of tumor patients, and points to an activation process in peritoneal cavity.

THE MECHANISM OF HOMING OF CYTOTOXIC CELLS 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

Recent studies strongly suggest that liver plays an important immunoregulatory role in general and local immunity. Local immune function of liver seems to be associated with a specific population of lymphocytes (LC), most probably of blood origin, which marginate in hepatic sinusoids. They seem to be responsible for destruction of malignant cells which have reached liver *via* the bloodstream. In our previous studies phenotypical and functional characterization of this cell population in normal liver was performed. In the present study we investigated whether the presence of colon cancer metastases (evoked by inoculation of CC531 colon adenocarcinoma cells into the portal vein of syngeneic WAG rats) had influence on cell types trapped in liver sinusoids. *In vivo* studies on LC trapping in liver give inconsistent results due to competitive homing in lymphoid organs. To avoid this problem, the process of LC retention in liver was studied in an extracorporeal rat liver perfusion model with peristaltic pump, oxygenator and heat-exchanger. Metastatic and control W/WAG livers were washed out from sinusoidal marginating cells prior to and 1hr after perfusion with syngeneic blood (pre- and postperfusion wash-out, PRE-W and POST-W, respectively) and the retrieved cell population was identified with monoclonal antibodies.

Results: Monoclonal antibody analysis showed a prevalence in PRE-W from liver with metastases compared to control PRE-W, of helper/inducer W3/25$^+$ (39.3 vs 32.2%, respectively), class II$^+$ OX6$^+$ (32 vs 21.7%), NK cells 3.2.3$^+$ (25.3 vs 10%), monocyte/macrophage ED1$^+$ (29.9 vs 14.5%), whereas fewer of T cells OX19$^+$ (26 vs 31.7%). POST-W cells from metastatic livers displayed the same characteristic as PRE-W, with an evidently higher than in normal liver percentage of W3/25$^+$ (38.9 vs 33.2%), OX6$^+$ (26.8 vs 15.6%), ED1$^+$ (32.2 vs 18.1%) and 3.2.3$^+$ (33.2 vs 16.6%) cells. In conclusion, rat liver with metastases of colon adenocarcinoma retains in sinusoids a population of blood cells enriched in CD4$^+$, NK, monocyte/macrophage and MHC class II$^+$ cells.

A NEW MECHANISM OF CYCLOSPORIN A ACTIVITY IN PREVENTION 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

Treatment of allograft recipient with cyclosporine A (CsA) causes prolongation of graft survival, decreased infiltration of the graft with recipient immune cells, and selective inhibition of donor T cell proliferation by blocking cytokine synthesis. Previously we showed that CsA affects the *in vivo* lymphocyte migratory properties. The question arises whether the down-regulation of expression of adhesion molecules by CsA could be responsible for the decreased migration? Thoracic duct lymphocytes (TDL) harvested from WIS rats treated with CsA (10 mg/kg for 7d) were labeled with 51Cr and injected i.v. into WIS rats with 4d AUG heart transplant (HTx). 24h later accumulation of TDL radioactivity in AUG HTx and recipient organs was measured and expressed as % of injected dose per gram of tissue. FACS analysis of CD11a, CD18, CD44, CD54, CD62L, CD8 and CD4 expression on TDL, PBL, MLN and spleen cells of WIS rats treated with CsA was performed. Results were compared with control non-CsA treated rats. The level of allograft radioactivity after infusion of CsA-pretreated TDL was 0.46±0.2% of injected dose per gram of tissue, compared to 1.1±0.8% in control. CD62L expression on CsA-pretreated cells was decreased as compared to control (34.4, 53.3, 41.9 vs 54.8, 65.0, 56.0 % of positive TDL, PBL, MLN, respectively, $p<0.05$). Expression of adhesion molecules CD11a/CD18, CD44 did not differ from the control. Decreased expression of L-selectin on CsA-pretreated lymphocytes may limit their accumulation in the allograft.

PATHOMECHANISM OF PROTRACTED WOUND HEALING

Dr. Hanna Gałkowska

Supported by the State Committee for Scientific Research: grant # 4 P05C 030 10

Phenotypical analysis of infiltrating cells in wounds with protracted healing was performed in 3 groups of wounds: a/ varicous ulcer of lower leg, b/ infected surgical upper abdominal midline wound, c/ normal surgical wound. Epidermis, dermis and subcutaneous tissue were evaluated. In group a/ biopsies were taken from the margin of ulcer. There was lack of germinal layer of epidermis, keratinocytes were DR^-, $CD44^-$, no $CD1a^-$ cells (Langerhans cells) were observed. In dermis, the dominating cells were $CD68^-$ macrophages, followed by CD45RO cells. All infiltrating cells including fibroblasts and endothelial cells were DR^-, $CD44^-$. In group b/ only 2-3 layers of keratinocytes were seen. They were DR^-, $CD44^-$. There were

no CD1a$^-$ cells. In dermis high density of CD68$^-$ and DR$^-$ cells was observed. Endothelial cells were DR$^-$ and only slightly factor VIII$^-$. In group c/ activated keratinocytes expressing DR and numerous CD1a$^-$ cells could be seen within the first 24 h. In dermis high concentration of CD68$^-$ cells was observed. After 3 and 7 days the CD68$^-$ cells were less numerous, being replaced by CD3$^-$8$^-$, CD45RO lymphocytes. Taken together, rapid activation of keratinocytes and recruitment of CD1a$^-$ and CD68$^-$ cells can be seen in fresh wounds. In wounds with protracted healing, the structure of epidermis remains abnormal, with few CD1a$^-$ cells. In dermis CD68$^-$ macrophages dominate, abnormal structure of blood capillaries is seen.

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STUDY OF ORAL TOLERANCY INDUCTION USING BOVINE SPINAL HYDROLYSATE IN ANIMAL MODEL OF EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS

Assoc. Prof. Andrzej W. Lipkowski

Specific protein given orally is known method of induction of tolerance of immunologic response to this antigen. This method is reviewed recently by some authors as a possible tool in the treatment of autoagressive diseases. Experimental allergic encephalomyelitis (EAE) is the respected animal model of autoagressive disease as multiple sclerosis. The aim of this year study was the evaluation of the effect of bovine spinal hydrolysate upon the course of EAE in the rat. Experimental allergic encephalomyelitis was evoked by intra-dermal injection of spinal cord homogenate of guinea pig with complete Freund's adjuvant and *Mycobacterium phlei*. In the group of animals pretreated with bovine spinal cord hydrolysate before immunization the number of animals with first relapse was lower in comparison to untreated ones. Antibodies level anti-MBP as well as a number of inflammatory infiltrates were lower in the treated animals. These pilot data indicate that oral treatment with spinal cord protein hydrolysate modulates clinical course in the rat and might have some clinical implication for search multiple sclerosis treatment in human. The study was done with close collaboration with Industrial Chemistry Research Institute in Warsaw.

INTERACTION BETWEEN OPIOIDS AND OTHER NEUROTRANSMITTERS IN PAIN TRANSMISSION PROCESSES

Assoc. Prof. Andrzej W. Lipkowski

Endogenous opioid system plays a major role in suppresion of pain signal transmission in both central and peripheral nervous system. There is also a growing evidence that other neuromodulators and neurotransmitters also play role in the transmission and suppression of pain signals. In particular, multidisciplinary experimental approaches have demonstrated that NMDA system may participate in the integration of nociceptive information. Spinal NMDA system, in addition to pain modulation, may play a role in mediation of variety of neural side effects

including neurotoxicity. Recently, endogenous peptide, histogranin, has been isolated and characterized as endogenous NMDA antagonist. Our study of intrathecal interaction of histogranin with opioids showed that histogranin itself has little effect on reducing of thermal acute nociceptive stimuli. However, in combination with opioid peptide, biphalin, strongly enhanced and prolonged duration of antinociceptive effect of biphalin was observed. This program was accomplished under scientific cooperation with the Department of Anesthesia, Massachusetts General Hospital, Boston, USA.

STRUCTURE-ACTIVITY RELATIONSHIPS OF OPIOID LIGANDS

Assoc. Prof. Andrzej W. 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 studies of endogenous opioid peptide and their peptide mimetics on a design of the new generation of antinociceptive drugs with various receptor selectivity, pharmacokinetic and pharmacodynamic profiles. Using a combination of computer assisted design, conformational and topographical considerations, synthetic chemistry, and multiple pharmacological assay methods we have elaborated a series of compounds with new, unusual biological and pharmacological profiles. The most promising avenues for new drug discovery are new series of opioid peptide analogues and alkaloid-peptide hybrides. We were able to construct common topographic model of opioid peptides and alkaloids. Basing on this model we were able to construct hybrides in which dynorphin peptide fragments hybridized with naltrindol, delta selective alkaloid shifted its primary receptor selectivity to kappa opioid receptors.

The program was accomplished under scientific cooperation with the Department of Anesthesia, New England Medical Center, Boston, and Department of Chemistry, University of Arizona, Tucson, USA.

DEPARTMENT OF ENDOCRINOLOGY
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STUDY ON INTERACTIONS BETWEEN T LYMPHOCYTE DERIVED FROM PERIHERAL BLOOD AND ORBITAL TISSUES WITH EXTRACELLULAR MATRIX PROTEINS IN PATIENTS WITH THE AUTOIMMUNE THYROID DISEASE AND OPHTHALMOPATHY ACCOMPANYING GRAVES' DISEASE

Prof. Janusz Nauman

Supported by the Medical Research Centre Intramural Grant No. 32
and by the State Committee for Scientific Research Grant No. 4 P05B 047 09

We have studied putative role of interactions between T lymphocytes and extracellular matrix proteins (ECMP) in the pathogenesis of the thyroid-associated ophthalmopathy. The experimental system based on the culture of T cells with ECMP, namely: collagen I (Col I), collagen IV (Col IV), fibronectin (FN), laminin (LM) and elastin (EL) has been developed. Two different types of the assay were established: the test of peripheral blood mononuclear cells (PBMC) proliferation in response to protein antigens and the test based on costimulation of PBMC by antigens and CD3. We have studied interactions of T cells and ECM proteins in 40 subjects including healthy control subjects, patients with active Graves' ophthalmopathy and with stable eye disease, patients with Graves' disease or Hashimoto thyroiditis without eye involvement, and patients with nonautoimmune thyroid disorders (multinodular goitre, single thyroid nodules). Preliminary results seem to confirm the notion that impaired T cells – ECM proteins interactions may play a pathogenic role in the thyroid associated-ophthalmopathy as we observed significantly higher stimulation index values in the group of patients with an active eye disease in response to collagen I. On the other hand, ELISA assays confirmed high prevalence of anti-collagen I autoantibodies in sera of patients with stabilized ophthalmophathy. These autoantibodies may play a role of marker of orbital fibrosis and scarring in the clinical course of the disease.

The study was performed in cooperation with The Thyroid-Eye Disease Research Center (where some of experiments were carried on by Dr. Tomasz Bednarczuk), Allegheny Singer Research Institute, Medical College of Pennsylvania, Pittsburgh, PA, USA, Department of Endocrinology, University Medical School of Warsaw, Department of Immunology, Transplantation Institute of the University, Medical School of Warsaw.

ANTIPEROXIDASE AND ANTITHYROGLOBULIN AUTOANTIBODIES IN PATIENTS WITH MYASTHENIA, INSULIN-DEPENDENT DIABETES MELLITUS, RHEUMATOID ARTHRITIS, SYSTEMIC SCLEROSIS AND SYSTEMIC LUPUS ERYTHEMATOSUS

Prof. Janusz Nauman

Prevalence of antiperoxidase and antithyroglobulin autoantibodies in patients with autoimmune disorders not affecting the thyroid gland was studied using ELISA technique with purified human antigens. We studied 121 subjects, among them 33 patients with myasthenia gravis (MG), 16 patients with insulin dependent diabetes mellitus (IDDM), 28 patients with rheumatoid arthritis (RA), 25 patients with progressive systemic sclerosis (SS), 19 subjects with systemic lupus erythematosus (SLE) and 30 age- and sex-matched healthy control subjects. Antiperoxidase autoantibodies were found in as many as 84.8% of patients with myasthenia, 37.5% of patients with IDDM, 53.6% of patients with RA, 36.8% of patients with SLE and 32% of patients with SM. On the other hand, antithyroglobulin autoantibodies were found in 36.8% of patients with MG, 12.5% of patients with IDDM, 64.3% of patients with RA, 26.3% of patients with SLE and 32% of subjects with SS. Autoantibodies titers were usually low and were correlated to each other. The results confirm the notion that markers of the autoimmune thyroid disease are frequently present in autoimmune disorders apparently not involving the thyroid gland.

The study was performed in cooperation with Dr. Barbara Czarnocka Ph.D., S.Sci, of Department of Biochemistry, Medical Center of Postgraduate Education, Warsaw.

THE EFFECTS OF IODINE DEFICIENCY ON THYROID HORMONE METABOLISM IN THE PLACENTA

Tomasz Bednarczuk, M.D.

Supported by the State Committee for Scientific Research: grant # 4 P05B 129 10

The study was performed to determine the influence of iodine deficiency on the thyroid hormone metabolism in the placenta to answer the question whether placenta could play a preventive role against the consequences of not sufficient iodine supply during pregnancy. The influence of iodine deficiency on placental enzymatic activity has never been investigated in humans.

The thyroid hormones level, iodine concentration in the urine of mothers and newborns, and the placental deiodinases activity were evaluated in the following age-matched groups: (a) Iodine deficiency group (IDG) consisting of 7 women with urine iodine concentration below the normal range (100 μg/g creatinine); (b) Control group (CG) consisting of 12 women with normal urine iodine concentra-

tion, receiving iodine supplementation of 150 μg daily during second and third pregnancy trimester.

The following results directly pertaining to these questions were obtained:

1. Total serum T3 concentration was significantly higher in iodine deficient mothers (1.48 vs. 0.83 nmol/L, p=0.0067). Total serum T3 concentration in children was not affected by iodine deficiency.

2. In mothers receiving iodine supplementation during pregnancy urine iodine concentrations (means iodine urine concentration 415 μg/g creatinine) were significantly higher (Wilcoxon test; p=0.0001) than in mothers without the supplementation (43.71 μg/g creatinine). Noteworthy, newborn urine iodine excretion in the IDG (123.3 μg/g creatinine) was not significantly different than in the control group (145.5 μg/g creatinine).

3. Total 5-deiodination activity and the activity of 5-deiodinase type III were not significantly different in the investigated groups.

4. Total 5'-deiodination activity and the activity of 5'-deiodinase type II were significantly lower in the iodine deficiency group (p=0.47, p=0.0225 respectively). The negative correlation was found between total 5'-deiodination activity and total serum T3 concentration in mothers (r=-0.51, p=0.0248).

Our data suggest the existence of few mechanisms protecting the fetus against iodine deficiency. Apart from already described maternal thyroxine transport through placenta the mechanisms comprise fetal thyroid stimulation (increased TSH level in newborns found in our material), an active iodine placental uptake (newborn urine iodine excretion in the IDG within the normal range) and modification of placental thyroid hormones deiodination in conditions of decreased iodine availability.

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 subject profile:

physiology, neurosciences and experimental surgery, including transplantology

Present holdings:

books – monographic and serial volumes (Polish and foreign) – 15 394
periodicals, newspapers (number of titles) – 159

References 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 1996 was prepared. It contains 832 citations.

MEDIPAN –Scientific Instruments Department
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MEDIPAN is a manufacturer of specialistic equipment for medical service. Syringe and volumetric infusion pumps are the basic assortment. All the products have been designed in the Department.

In 1996 the activities have focused on adaptation of infusion pumps to functioning in collaboration with computer systems. Technical basis has been elaborated of a system which will enable:

– remote control of infusion,
– receipt of feedback information from the pump,
– rapid diagnosis.

Preliminary steps towards designing a new single-syringe pump have been undertaken.

INTERNATIONAL COOPERATION

VISITING SCIENTISTS

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Department of Endocrinology

Tomasz Bednarczuk	Thyroid Eye Research Program, Allegheny-Singer Research Institute, Pittsburgh, USA

PARTICIPATION IN INTERNATIONAL MEETINGS

XXth Anniversary of Organ Transplantation in Poland "Progress in Transplantation", Warsaw, Poland, January 25-26, 1996: *W. Olszewski, M. Maksymowicz*

Workshop in honor of Lewis P. Rowland, Padova, Italy, February 1, 1996: *I. Hausmanowa-Petrusewicz*

European Association of Neurosurgical Societies Winter Meeting, Taormina, Italy, February 21-25, 1996: *Z. Czernicki*

Symposium on Recent Advances in Diagnosis and Therapy of Neuromuscular Diseases, Prato, Italy, March 21-24, 1996: *I. Hausmanowa-Petrusewicz, I. Niebrój-Dobosz*

13th International Symposium on Arterial Chemoreceptors, Santiago, Chile, March 25-29, 1996: *M. Pokorski, M. Walski*

International Celebratory Thyroid Meeting, Dublin, Ireland, March 26-28,1996: *J. Nauman*

31st European Society for Surgical Research Congress, Southampton, United Kingdom, March 31-April 3, 1996: *W.L. Olszewski, B. Łukomska, H. Gałkowska, M. Jaskłowska-Englisz, I. Grzelak, G. Szczęsny, M. Maksymowicz*

Spring Meeting of Clinical Molecular Genetics Society, Newcastle upon Tyne, United Kingdom, April 2-3, 1996: *M. Kozłowska*

The 17th Annual International Gravitational Physiology Meeting, Warsaw, Poland, April 14-19, 1996: *H. Kaciuba-Uściłko, K. Nazar, E. Żernicka*

3rd International Dead Sea Symposium on Advances in Diagnosis and Treatment of Cardiac Arrhythmias, Tel Aviv, Israel, April 17-19, 1996: *K. Cedro-Ceremużyńska*

International Congress on Sudden Infant Death Syndrom, Košice, Slovakia, April 18-20, 1996: *M. Szereda-Przestaszewska*

6th Alzheimer Europe Annual Meeting, Warsaw, Poland, April 18-20, 1996: E. Łuczywek, M. Barcikowska

Symposium of International Society of Lymphology, Tuscon, USA, April 19-21, 1996: *W. Olszewski*

5th European Congress of Neuropathology, Paris, France, April 22-28, 1996: *A. Kapuściński, M. Dąmbska, H. Kroh, R. Pluta*

IV International Symposium of Jagiellonian Medical Research Centre: Eicosanoids, Aspirin and Asthma, Cracow, Poland, May 1-3, 1996: *K. Cedro-Ceremużyńska*

Brainstorming – "Ornithine Aspartate (OA) and treatment of hepatic encephalopathy", Chelleford, United Kingdom, May 2-4, 1996: *J. Albrecht*

International Conference "The Molecular and Free Radical Basis of Wide Spectrum Diseases", Łódź-Płock, Poland, May 7-8, 1996: *H. Kaciuba-Uściłko, K. Nazar, M. Walski*

Symposium "Acute Pain", Heraklion, Greece, May 9-11, 1996: *P. Jakubowski, D. Kosson*

World Meeting of Polish Physicians, Chicago, USA, May 8-12, 1996: *J. Jurkiewicz*

First Annual Conference of Lymphology Society of India, Thanjavur, India, May 15, 1996: *W. Olszewski*

Polish-Finnish Symposium: "Progress in Clinical Neurophysiology", Warsaw, Poland, May 15-17, 1996: *I. Hausmanowa-Petrusewicz, E. Zalewska*

Merck European Thyroid Symposium, Warsaw, Poland, May 16 - 18, 1996: *J. Nauman*

First Congress of Euroacademy for Multidisciplinary Neurotraumatology, Salzburg, Austria, May 17-18, 1996: *Z. Czernicki*

21st Congress of European Group of Lymphology, Roma, Italy, May 23-25, 1996: *W. Olszewski,. B. Łukomska*

II European Kidney Research Forum, Bergamo-Baveno, Italy, May 24-27, 1996: *J. Sadowski, L. Dobrowolski, E, Kompanowska-Jezierska, A. Walkowska*

I Annual Congress of the European College of Sport Science, Nice, France, May 28-31, 1996: *H. Kaciuba-Uściłko, K. Nazar*

Central European CODMAN MEDOS Symposium, Prague, Czech Republic, May 29-30, 1996: *J. Jurkiewicz*

The Second International Workshop on Maturation Phenomenon in Cerebral Ischemia, Neuronal Recovery and Plasticity, Tokyo, Japan, June 1, 1996: *R. Pluta*

I International Conference on Apoptosis in Skeletal and Cardiac Muscles, Padova, Italy, June 1-4, 1996: *A. Fidziańska, A. Kamińska*

V International Symposium of Polish Network of Cellular and Molecular Biology UNESCO/PAN: "Molecular and Physiological Aspects of Systemic Regulation", Cracow, Poland, June 4-5, 1996: *B. Gajkowska*

10th Nord-Baltic Conference on Biomedical Engineering, Tampere, Finland, June 9-13, 1996: *I. Hausmanowa-Petrusewicz, K. Rowińska-Marcińska, G. Cybulski, E. Zalewska*

Neurochirurgische Konference im Neurochirurgische Klinic Aarau, Aarau, Switzerland, June 10-14, 1996: *J. Jurkiewicz*

XI Meeting of European Society for Neurochemistry, Groningen, The Netherlands, June 15-20, 1996: *J. Strosznajder, J. Łazarewicz, J.Albrecht, B. Gajkowska, M. Śmiałek, T. Zalewska, K. Domańska-Janik, B. Kwiatkowska--Patzer, J. Sypecka, B. Zabłocka*

South Pacific Symposium on Iodine Deficiency, Diabetes and Environment, Pepeete, Tahiti, June 18-20, 1996: *J. Nauman*

Workshop of European Neuromuscular Center, Naarden, The Netherlands, June 21-23, 1996: *I. Hausmanowa-Petrusewicz*

International 14-th Puijo Symposium "Physical Activity, Diet and Cardiovascular Diseases – a Fresh Beyond Old Facts", Kuopio, Finland, June 24-28, 1996: *R. Grucza*

Varian Course of High Performance Liquid Chromatography, Zug, Switzerland, July 6-13, 1996: *B. Głód*

24th Meeting of the Federation of European Biochemical Societes (FEBS '96), Barcelona, Spain, July 7-12, 1996: *L. Budohoski, M. Łałowski*

40th Annual Meeting of Society for Research on Hydrocephalus and Spina Bifida, Utrecht, The Netherlands, July 10-13, 1996: *J. Jurkiewicz*

International Symposium on Pharmacology of Cerebral Ischemia, Marburg, Germany, July 21-24, 1996: *E. Kołodziejska*

International Narcotic Research Conference, Los Angeles, USA, July 21-26, 1996: *A. Lipkowski*

XVIIth International Carbohydrate Symposium, Milano, Italy, July 21-26, 1996: *Z. Bartoszewicz*

5th International Conference on Alzheimer's Disease and Related Disorders, Osaka, Japan, July 22-29, 1996: *R. Pluta, M. Barcikowska-Litwin, E. Kida*

XI Congress of International Society for Developmental Neurosciences, Tampere, Finland, July 30-August 4, 1996: *J. Albrecht, B. Dąbrowska-Bouta, K. Domańska-Janik, J. Sypecka, D. Maślińska*

Tenth International Symposium on Pharmacology of Thermogulation, Memphis, USA, August 16-22, 1996: *R. Grucza*

XVI International Congress of the Transplantation Society, Barcelona, Spain, August 25-30, 1996: *W. Olszewski, B. Łukomska, E. Jaskłowska-Englisz*

Danube Symposium on Neurological Sciences, Krems, Austria, August 28-30, 1996: *M.J. Mossakowski*

European Thyroid Symposium, Amsterdam, The Netherlands, August 30 - September 4, 1996: *J. Nauman*

8th International Symposium on Cells of the Hepatic Sinusoid, Bordeaux, France, September 1-5, 1996: *B. Łukomska, S. Durowicz*

3rd World Stroke Congress and 5th European Stroke Conference, Munich, Germany, September 1-5, 1996: *E. Koźniewska*

XV Martin Day of Respiration, Martin, Slovakia, September 2-3, 1996: *M. Szereda-Przestaszewska*

17th Congress of the Polish Anatomical Society with International Participation, Gdańsk, Poland, September 3-6, 1996: *M. J. Mossakowski, R. Pluta*

8th International Congress of the Czech and Slovak Neurochemical Society, Martin, Slovakia, September 4-7, 1996: *J. Łazarewicz, K. Domańska-Janik*

II International Congress: "Phagocytes, biological and clinical aspects", Pavia, Italy, September 4-7, 1996: *M. Walski, D. Maślińska*

European Respiratory Society Congress, Stockholm, Sweden, September 7-11, 1996: *K. Budzińska, M. Szereda-Przestaszewska, B. Wypych*

International Conference on Recent Advances in Neurotraumatology, Rimini-Riccione, Italy, September 8-11, 1996: *J. Jurkiewicz*

XX Congress of the Polish Physiological Society, Wrocław, Poland, September 8-11, 1996: *Z. Brzezińska, G. Cybulski, J. Chwalbińska-Moneta, I. Falęcka-Wieczorek, R. Grucza, H. Kaciuba-Uściłko, B. Kruk, H. Krysztofiak, K. Krzemiński, K. Nazar, A. Ziemba, L. Budohoski, E. Żernicka, A. Dubaniewicz, J. Sadowski, E. Kompanowska-Jezierska, B. Bądzyńska, L. Dobrowolski, A. Walkowska, K. Budzińska, L. Czerwosz, E. Koźniewska, M. Szereda-Przestaszewska*

V Conference of German Society of Neuropharmacology and Neurotoxicology, Magdeburg, Germany, September 13-14, 1996: *J. Albrecht*

XXIII Congress of the Polish Society of Sports Medicine, Cracow, Poland, September 15-18, 1996: *G. Cybulski, J. Chwalbińska-Moneta, R. Grucza, H. Kaciuba-Uściłko, H. Krysztofiak, B. Kruk, K. Krzemiński, K. Nazar, A. Ziemba, W. Niewiadomski*

The Second National Network Conference on Lymphedema: The Problem and the Challenge, San Francisco, USA, September 19-22, 1996: *W. Olszewski*

Neurosurgery Symposium: "Veranderte Therapiekonzepte", Giessen, Germany, September 25-26, 1996: *Z. Czernicki, J. Andrychowski, J. Bogucki*

International Symposium: Exercise in Prevention, Diagnosis and Therapy of Metabolic Disorders (Devoted the memory of the late Prof. dr med. Stanisław Kozłowski), Warsaw, Poland, September 25-27, 1996: *M. J. Mossakowski, E. Wójcik-Ziółkowska, K. Nazar, H. Kaciuba-Uściłko, R. Grucza, J. Chwalbińska-Moneta, B. Kruk, A. Ziemba, G. Cybulski, H. Krysztofiak, B. Tomczak, E. Szczęsna*

III International Symposium of the Cell Transplant Society, Miami, USA, September 29-October 2, 1996: *W. Olszewski, M. Jaskłowska-Englisz, M. Durlik, S. Durowicz*

4th Central European FACS/CAS Users' Meeting, Portoroze, Slovenia, September 29-October 2, 1996: *S. Janczewska, M. Poreda*

4th International Lymphology Symposium of Brussels, Brussels, Belgium, October 3-5, 1996: *W. Olszewski*

4th International Workshop on Dendritic Cells: Dendritic cells in fundamental and clinical immunology, Venice, Italy, October 5-10, 1996: *H. Gałkowska*

2nd Australian Peptide Conference; From Discovery to Therapeutics, Fraser Island, Australia, October 6-11, 1996: *A. Misicka-Kęsik*

Alzheimer's Disease – 12th International Conference, Jerusalem, Israel, October 8-11, 1996: *R. Pluta*

8th European Congress of Clinical Neurophysiology, Munich, Germany, October 9-11, 1996: *E. Zalewska*

Polish-Czech-Slovak Symposium of Neurosurgery, Polanica Zdrój, Poland, October 10-12, 1996: *W. Sapieja*

Symposium of Royal Society of Medicine "Postprandial lipid metabolism", London,United Kingdom, October 12-17, 1996: *E. Żernicka*

Conference of the All-German Neuropharmacological and Neurotoxicological Society, Magdeburg, Germany, September 13-14, 1996: *J. Albrecht*

Symposium of American Society of Neurology, Miami, USA, October 13-16, 1996: *I. Hausmanowa-Petrusewicz*

III Hungarian Congress on Alzheimer's Disease, Budapest, Hungary, October 17-19, 1996: *M. Barcikowska-Litwin*

An International Symposium: " Apoptosis and its Role in Immunity", Warsaw, Poland, October 20, 1996: *D. Maślińska, M. Muzylak*

10th International Brain Edema Symposium, San Diego, USA, October 20-23, 1996: *Z. Czernicki, R. Pluta*

8th European Congress on Clinical Neurophysiology, Münich, Germany, November 9-10, 1996: *I. Hausmanowa-Petrusewicz, E. Zalewska*

II Congress of European Federation of Neurology Societies, Roma, Italy, October 31 - November 3, 1996: *I. Hausmanowa-Petrusewicz, P. Grieb*

69th Annual Meeting of The American Thyroid Association, San Diego, USA, November 14-17, 1996: *J. Nauman, T. Bednarczuk*

15th Annual Sciences Meeting of the American Pain Society, Washington, USA, November 14-17, 1996: *I. Maszczyńska*

26th Meeting of Society for Neuroscience, Washington, USA, November 16-21, 1996: *M. Łałowski*

Symposium of Polenov Institute of Neurosurgery, St. Petersburg, Russia, November 27-30, 1996: *Z. Czernicki, J. Jurkiewicz*

VIIth Conference of Polish Histamine Research Society: "Biogenic Amines and Related Biologically Active Compounds", Ustroń, Poland, November 28-30, 1996: *M. Maślińska*

International Symposium "Reanimatology on the Eve of the 21st century", Moscow, Russia, December 2-4, 1996: *R. Pluta*

36th American Society for Cell Biology, Annual Meeting, San Francisco, USA, December 7-11, 1996: *W. Gordon-Krajcer*

SCIENTIFIC DEGREES

DOCTOR'S DEGREE

Lidia Strużyńska (Department of Neurochemistry MRC PASci)
Metabolic and functional changes in synaptosomes and capillaries from rat brain evoked by toxic lead effects

Jacek Ignacy Kiljański (Department of Endocrinology MRC PASci)
Orbital antigens involved in the pathogenesis of the thyroid-associated ophthalmopathy

Joanna Sypecka (Department of Neurochemistry MRC PASci)
Pleiotropic effect of PLP gene mutation in pt rabbit

Adam Gołąbek (Department of Neuropathology MRC PASci)
Characterization of apolipoproteins binding to the amyloid β-peptide and their role in the fibrillogenesis in vitro

Jadwiga Janas (Institute of Cardiology, Warsaw)
Identification and characterization of specific, tissue peptidase that inactivates endothelin-1

HABILITATIONS

Maria Barcikowska-Litwin (Department of Neuropathology MRC PASci)
Amyloid deposits within neuropil and neurones with neurofibrillary tangles in Alzheimer's disease and ageing

Ewa Matyja (Department of Neuropathology MRC PASci)
Endogenous amino acids neurotoxicity and its modulation in tissue culture system

Ryszard Marek Pluta (National Institutes of Health, Bethesda, MD 20892, USA)
Cerebral vasospasm after subarachnoid hemorrhage: pathophysiology and new method of prevention and treatment

SCIENTIFIC MEETINGS
ORGANIZED BY THE MEDICAL RESEARCH CENTRE

International Symposium "Exercise in prevention, diagnosis, and therapy of metabolic disorders" (devoted to the memory of the late Prof. dr med. Stanisław Kozłowski), Warsaw, Poland, September 25-27, 1996.

Symposium was organized by the Department of Applied Physiology, Medical Research Centre, Polish Academy of Sciences and Polish Scientific Society of Obesity and Metabolism in cooperation with the Institute of Sport and Servier-Polska (Honorary President – Prof. Mirosław J. Mossakowski, MD, PhD, DSci, Dhc).
One hundred ninety one scientists participated in the Symposium (171 from Poland and 21 from abroad).
The main topic of the meeting was the clinical application of exercise as single or supplementary means of prevention and therapy of metabolic ailments. The lectures of the first two days included also basic research on exercise metabolism. The third day was devoted mainly to the exchange of experience on exercise testing and prescription in patients with obesity, diabetes and other metabolic or endocrine disorders. The Symposium provided an opportunity for bridging the physiological, biochemical, pharmacological and clinical interests.
The Program of the first two days included 15 lectures delivered by leading specialists in the field of physiology and biochemistry of exercise from USA, Sweden, Denmark, Canada, Czech republic and Great Britain. Besides 11 oral communications and 46 posters were presented. On the third day so called "Polish Day" 7 lectures were delivered (in Polish) and then a round table discussion was organized. During the discussion experts from leading Polish clinical centers reached on the importance of obesity treatment for prevention and therapy of coronary heart disease and hypertension.

Conference "Skin infections – resistance, long lasting treatment with antibiotics", Warsaw, Poland, November 16, 1996.

Symposium was organized by Surgical Research and Transplantation Department of Medical Research Centre. Number of participants – 103.
Conference was devoted to the inflammatory processes of human skin – phenotypic characterization of infiltrating lymphocytes and macrophages, cytokin production, adhesion molecule expression, reactivity of infiltrating cells to mitogens. The main interest was focused on the regulatory role of tissue fluid cytokines on tumor cell proliferation. Six invited lectures and oral communications were presented.

Conference "Advances in hydrocephalus treatment", Pultusk, Poland, December 6-7, 1996.

The conference was organized in participation of Neurological Sciences Committee of Polish Academy of Sciences. There were 83 participants including 8 foreign guests. Twenty three lectures including 7 invited ones were given during 3 scientific sessions and round table discussion. Among them 8 were presented by the workers of the Neurosurgery Department.
It is a great achievement of the Conference to agree upon the necessity of wider application of endoscopic method in the treatment of certain types of hydrocephalus in the clinical practice. Its importance in hydrocephalus treatment in children was emphasized. Prof. Marchel, Roszkowski, Kojder and Majchrzak presented a common view that endoscopic method in the treatment of obstructive hydrocephalus should be the first procedure offered to the patients. Implantation of shunt systems should be undertaken only in cases when endoplasmic ventriculostomy was failed or there are contraindications for ventriculostomy. The other important topic of the Conference was a new, non-invasive method in the diagnosis of normal pressure hydrocephalus by using the so-called hydrocephalus index developed in the Department of Neurosurgery. The accuracy of differential diagnosis between normal pressure hydrocephalus and cerebral atrophia by application of hydrocephalus index can be compared to results of the infusion test.
The device for the computerized tomography images transfer on long distance was presented during the Conference for the first time in Poland. Such a teleradiological system facilitating the cooperation between centres isolated from one another was installed in the Province Hospital in Ciechanów and the Neurosurgery Department of Medical Research Centre of Polish Academy of Sciences.

IIIrd Neurochemical Conference "Biochemistry, pathophysiology and molecular biology of neurotransmission and signal transduction in the nervous system", Warsaw, Poland, December 17, 1996

This domestic scientific conference was organized by Department of Neurochemistry together with Polish Neuroscience Society and Neurochemical Commision of the Committee of Neurological Sciences, Polish Academy of Sciences. Totally 113 participants took part in the conference. The program comprised 1 opening lecture, 38 oral presentations during sessions untitled: "Neuropharmacology and neuroprotection", "Regulation of gene expression and CNS pathology", "Biochemical basis of CNS dysfunction: effects of neurotoxic compounds" and "Receptors and intracellular signal transduction". Moreover 12 posters were presented in the session "Varia". The Abstracts book of the conference has been published.

Conference "Hepatocytes and liver transplantation in liver failure" and Conference "Mobilization of cholesterol from tissues". Both conferences were integrated under the common title: "Hepatocyte and liver transplantation in liver failure", Warsaw, Poland, December 21-22, 1996.

Presented themes were concerned with hepatocyte isolation, their transplantation to different tissues (spleen, under skin, intraperitoneally, intravenously, liver), mechanism of their destruction by macrophages and granulocytes of recipient, the tolerance after liver transplantation, and also the success rate for hepatocyte and liver transplantation in cholesterol metabolism failure due to the lack of LDL receptors.
Eight invited lectures and 16 oral communications were presented. Prof. N. Miller – Great Britain, Dr. M. Winnock – France, Dr. S. Winnock – France, Dr. F. Bocardo – Italy were among invited speakers.

Polish-Italian Symposium "The role of lymphatic system in immunity", Warsaw, Poland, December 23, 1996.

Symposium was organized by Surgical Research and Transplantation Department of Medical Research Centre. Number of participants – 40. Invited speakers – Prof. Corradino Campisi and Dr. Francesco Bocardo from Italy.
The Symposium was devoted to the results of investigations on local immunity disorders in tissue deprived of lymphatic tissue. The kinetic of Staphylococcus epidermidis, Acinetobacter, E. coli, Corynebacteriae infections in lymphedematous tissue was discussed. Surgical models for lymphedema development in animals were presented.

PUBLICATIONS

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4. Albrecht J, Matyja E: Glutamate: a potential mediator of inorganic mercury neurotoxicity. *Metabol Brain Dis* 1996, 11, 2, 175-184.

5. Andrychowski J, Nauman P, Czernicki Z: Podskroniowy dostęp operacyjny do tętniaków górnego odcinka tętnicy podstawnej mózgu. Modyfikacja ze zdjęciem łuku jarzmowego. Uwarunkowania anatomiczne. Korzyści wynikające z dostępu [Subtemporal approach to aneurysms of the upper part of the basilar artery. Modification of the surgical approach with zygomatic arch removal. Anatomical considerations and potential advantages of the approach]. *Przegląd Lekarski* 1996, 53, Suppl 1, 111-113 (in Polish).

6. Andrychowski J, Nauman P, Czernicki Z, Bojanowski K: Zastosowanie obciążeń fizjologicznych i testu infuzyjnego w ocenie szczelności przestrzeni płynowych u chorych hospitalizowanych z powodu pourazowego płynotoku nosowego. Opis metody [The physiological loadings and infusion test as a new diagnostic method in follow-up of patients with nasal liquorrhea, treated conservatively]. *Neurol Neurochir Pol* 1996, 30, Suppl. 4, 565-569 (in Polish).

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COMMUNICATIONS

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