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POLISH ACADEMY OF SCIENCES
MEDICAL RESEARCH CENTRE

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POLISH ACADEMY OF SCIENCES
MEDICAL RESEARCH CENTRE
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RESEARCH REPORT

STUDIES ON THE FUNCTION OF THE NERVOUS SYSTEM AND ON MECHANISMS CONTROLLING BASIC FUNCTIONS OF THE ORGANISM

Department of Neurophysiology
Head: Prof. Witold Karczewski

PHYSIOLOGICAL AND PATHOPHYSIOLOGICAL ASPECTS OF RESPIRATORY AND CARDIOVASCULAR CONTROL

I. Functional organization of the respiratory neuronal network

1. It has been demonstrated that apneustic breathing elicited by a focal pontine lesion or a hemisection at the level of rostral pons can be abolished by a midline section of the brainstem („split-brainstem preparation"). The intact half of the brainstem starts to generate normal respiratory volleys (with slightly reduced amplitude), whereas in the lesioned half respiratory activity disappears; it reappears, however, after an injection of naloxone or stimulation of the vagal input. This implies that separation of the lesioned subgenerator from the „normal" one may be advantageous. In the „split-brainstem preparation" apneustic breathing cannot be elicited. It has been shown also that „split-medulla preparation" is capable of generating respiratory volleys under conditions of increased central or peripheral respiratory drive.

2. A local midline lesion placed on the ventral side of the medulla at the level of the intermediate chemosensitive zone abolishes high frequency oscillations in respiratory motoneurones. Respiratory rhythmogenesis is not abolished by such lesions.

3. Studies on the respiratory effects of arterial chemoreceptor stimulation (NaCN) in guinea-pigs have shown that there is a „classical" response (deep inspiration followed by rapid and shallow breathing) in normoxic, hyperoxic and lightly anaesthetized animals. However, hypoxic and deeply anaesthetised guinea-pigs (the latter even during hyperoxia) respond to cyanide injection with a respiratory arrest. This effect has not been described in other animal species. Both types of responses were abolished by blocking the carotid body chemoreceptors.

4. Stimulation of the raphe nucleus at the level of the medulla reduced the amplitude of inspiratory activity but did not affect the respiratory frequency. Information from raphe neurons reaches respiratory motoneurones via solitary tract nucleus (NTS).

5. The responses of the mylohyoid nerve (known as crucial in protecting the upper airway patency) to vagal input stimulation were studied in rabbits. Lung inflation and electrical stimulation of the vagus enhanced by mylohyoid

n. activity (with concomitant inhibition of phrenic n. activity). Ketamine and 20% ethanol reduced the activity of the mylohyoid nerve and elicited apneustic breathing pattern.

II. **The study of physiological and clinical correlations in the respiratory system**

1. The mechanisms of the apneustic pattern of respiration were investigated. The study was done on the previously described model of apneustic respiration induced by administration of the anesthetic agent ketamine (this type of respiration consists of irregular, long inspiratory phases interrupted by grossly unchanged expiratory phases). An important problem posed in the study was to find out whether the known normalization of ketamine-induced apneustic respiration due to hypoxic stimulus is associated with a change in the responsiveness of central inspiratory drive to hypoxia. We found that ketamine diminished that responsiveness (measured as the rate of rise of integrated phrenic nerve activity), but at the same time increased the total magnitude of the central inspiratory drive (measured as the area under the envelope of integrated phrenic nerve activity). The results also point to the disorders of the off-switch mechanism as the underlying cause of apneustic respiration. These disorders may be alleviated by increased chemical drive. These studies underscore the importance of central input and integration of peripheral chemical drive in the function of the off-switch mechanism, and may lead to some modification of current concepts regarding the functional organization of the central generator of respiratory rhythm.

2. Deprivation of vagally mediated afferentation from the lungs (as a result of bilateral vagotomy in the rib cage) in animals breathing spontaneously through the larynx had comparable effects to classical vagotomy performed in the neck; breath frequency decreased due to about doubling of the inspiratory time duration, and total volume increased with a net effect of unappreciable changes in lung ventilation. This deprivation, however, increased significantly the inspiratory flow, decreased the expiratory pressure gradient above and below the larynx as well as the laryngeal (chiefly expiratory) resistance. Preservation of laryngeal afferentation seems to be of importance in central respiratory responses (as monitored by integrated activity of the phrenic and laryngeal inferior nerves) to increasing external resistances of the respiratory tract.

3. The studies have been continued on the role of the autonomic nervous system in the phenomenon of neurological deficit due to cerebral vessel constriction in patients suffering of a subarrachnoideal hemorrhage. A beneficial effect of steroids was found in this setting. The mechanism of this effect may involve the influence of steroids on the interrelationship between the cholinergic and adrenergic part of the system.

See the List of publications:
7, 30, 31, 38, 50, 51, 53, 54, 103, 122, 132, 133

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ADAPTATION TO PHYSICAL EXERCISE AND CHANGES IN ENVIRONMENT

1. Metabolism and its control

— In cooperation with Department of Biochemistry, University of Oxford (U.K.) the effects af acute physical exercise of different characteristics and 5-week physical training on the soleus muscle insulin sensitivity were investigated in rats. Muscle sensitivity to insulin, determined in vitro, was expressed as the hormone concentration in the incubation medium (from 1.0 to 10.000 $\mu U.ml^{-1}$) inducing half of the maximum stimulation of lactate production and glycogen synthesis. Effectiveness of training was assessed by measuring activities of hexokinase and 2-oxogluterate dehydrogenase in the skeletal muscle.

Insulin sensitivity of glucose transport to the muscle cells (estimated indirectly by measuring lactate production and glycogen synthesis) was increased significantly within a few hours after completing endurance treadmill exercise, while it showed a decreasing tendency following a short-term intensive exercise. Endurance physical training caused an increase in the soleus muscle insulin sensitivity. The effect was greater and lasted for a longer time (up to 3 days) than that produced by the acute endurance effort. The „sprint" type training did not affect sensitivity of soleus muscle to insulin.

The data indicate that the observed changes in the muscle sensitivity to insulin may contribute to the improved glucose tolerance occuring after the endurance type of physical activity.

— Continuing the study on the influence of prolonged, sustained hyperadrenalinemia dynamics of changes in glucose and the soleus muscle sensitivity to insulin was analyzed in rats implanted sc with adrenaline (A) releasing, retard tablets for up to 5 days.

At 6 h after implantation of the A-retard-tablet it was found that plasma glucose and fatty acid concentrations increased and insulin concentration decreased compared with values obtained from placebo-tablet-implanted rats. Administration of glucose load demonstrated an impaired glucose tolerance in vivo, and incubation of soleus strips in vitro showed a decreased sensitivity of the rates of glycolysis and glucose transport to insulin. Thereafter gradual normalization of blood gucose and insulin levels as well as of glucose tolerance and the soleus muscle insulin sensitivity was demonstrated. Moreover, after 5 days of hyperadrenalinemia the sensitivities of glucose transport in the skeletal muscle (as measured by 3—10-methyl ($U^{14}C$) glucose incorporation), and glycolysis were significantly increased in comparison with sham-operated rats. Using the adenosine-receptor agonist (2-chloro-adenosine) and antagonist (8-phenyl-theophyline) it was proved that both the initial decrease in the muscle insulin sensitivity and its further increase were somehow related to the effect

of adenosine, which is known to act as a local inhibitor of muscle insulin sensitivity.

— Using the above described model of prolonged, sustained hyperadrenalinemia dynamics of some metabolic changes occurring under this conditions in blood, liver and three types of skeletal muscles both at rest and after physical exercise were examined. It was found that in the initial stage (12 h) hyperadrenalinemia produced: a) marked decrease in glycogen content in liver and skeletal muscles, accompanied by hyperglycemia, elevated blood FFA concentration, lowering of muscle creatine phosphate (CrP) with accumulation of lactate in skeletal muscles. Continuation of hyperadrenalinemia was associated with gradual rebuilding of glycogen stores and normalization of other metabolic variables except the plasma FFA that remained enhanced. Throughout 72 h of hyperadrenalinemia more rapid depletion of glycogen from the liver and skeletal muscles was found during physical exercise comparing with sham operated animals, coinciding with marked reduction of working ability.

— In order to assess a relationship between body composition glucose tolerance and whole body insulin sensitivity changes in blood glucose and insulin concentrations after the oral glucose load were compared in body builders, untrained, healthy subjects as well as in obese men having lean body mass similar to that in body builders. The study demonstrated an improvement of glucose tolerance accompanied by an increase in insulin sensitivity with the training-induced muscle hypertrophy. Enhanced body adiposity was found to be associated with opposite changes.

— In cooperation with the Institute of Physiology, University of Giessen (FRG) studies were carried out on work performance, thermoregulation and muscle metabolism in thyroidectomized goats. It was found that:

  a) Thyroid hormone deficiency resulted in a markedly diminished work efficiency of goats exercising on a treadmill at an ambient temperature of 30° C.

  b) The close relationship between the exercise-induced increase in core temperature and the magnitude of evaporative heat loss, characteristic for intact animals, was nearly completely abolished after thyroidectomy.

  c) Muscle glycogen utilization and lactic acid accumulation during exercise were enhanced in thyroidectomized animals in spite of the lower work rate and shorter duration of exercise in comparison with euthyroid goats.

— Continuing investigations on the role played by thyroid hormones in the control of energy metabolism the effects of the hormone deficit or excess on the skeletal muscle contents of high energy phosphates as well as of carbohydrate substrates and metabolites were analysed in dogs.

— In cooperation with the Department of Physiology, University of Kuopio (Finland) an influence of warming-up on the thermoregulatory response to graded physical exercise was evaluated in long-distance skiers. The pro-

cedure was found to increase the rate of sweating, thus limiting exercise hyperthermia.

2. Exercise tolerance in cardiac patients

— An effect of weight carrying on cardiovascular responses to dynamic exercise (walking) was studied in patients with coronary heart disease. It was demonstrated that an addition of the isometric component, which did not increase the total energy cost of exercise, caused a considerable enhancement of ischemic symptoms, with concomittant augmentation of the plasma noradrenaline response.
— Relationships between the degree of coronary insufficiency and hemodynamic responses to a standard exercise test were evaluated in coronary patients several years after myocardial infarction. In patients with severe ischemic changes in the exercise ECG a markedly lower cardiac output in relation to oxygen uptake was demonstrated. The degree of cardiac ischemia was expressed as the ratio of ST-segment depression to exercise oxygen uptake or to a percentage of the subjects' maximal heart rate.
— Results of a standard exercise test performed by coronary patients at sea level and at 950 m above it were compared. In the latter conditions the ischemic symptoms were much more pronounced, cardiac output was lower, and blood lactate concentration was higher than in control studies at sea level at the some work intensity.

3. Extracellular electrolites in the kidney

A methodological basis was created for the long term studies of the electrolyte environment of the renal medulla under different functional conditions. This included:

— Development of the method for the renal blood flow measurement in the rat. In essence, the method consists of continuous automatic recording of the renal vein outflow within a renal vein — jugular vein shunt system incorporating a pump controlled by a sensor of blood level in a side branch. It was shown that measurement per se did not interfere with functional integrity of the kidney. In particular, normal autoregulation of the renal blood flow was observed in response to reductions in renal perfusion pressure obtained by suprarenal aortic constriction. The glomerular filtration rate was not affected. Blood flow values were comparable with those reported from studies employing non-invasive methods, such as p-aminohippurate clearance or electromagnetic flowmetry.
— Extensive studies were perfomed to verify that measurements of electrical admittance of the renal medulla reflect sodium chloride concentration in that tissue area. To extend the scope of calibration curves, slices of the rabbit kidney cortex, outer medulla, inner medulla and papilla were equilibrated with solutions of 50 to 250 mmolar NaCl. It was shown that for all kidney zones tissue electrical admittance was linearly related to tissue so-

dium concentration. For the inner medulla and papilla the slopes were identical; the calibration curve was slightly steeper for the outer medulla and much flatter for the cortex. The data showed that electrical admittance is a reliable index of tissue NaCl concentration but values measured in different kidney zones cannot be directly compared.

See the List of publications:
6, 8, 9, 10, 11, 14, 24, 36, 44, 47, 48, 49, 60, 61, 65, 91, 92, 98, 105, 110, 124, 125, 134, 140, 141, 154, 164, 165, 166.

Cardiovascular Laboratory
Head: Assoc. prof. Krystyna Cedro-Ceremużyńska

CONTROL MECHANISMS OF CARDIOVASCULAR SYSTEM IN DEFINED PATHOLOGICAL CONDITIONS

Biochemical and ultrasutructural changes in the nonischaemic area of infarcted myocardium were earlier reported by numerous authors, including ourselves (Cardiovasc. Res., 1970, 4, 168). To investigate further the mechanism of these alterations, the content of lipid peroxidation products (malondialdehyde, conjugated dienes, fluorescent end-products) was measured in the ischaemic and nonischaemic areas of pig heart subjected to LAD ligation for 20 min, as well as in the left ventricular myocardium of sham operated pigs. As expected, the content of peroxidation products was significantly higher in ischemic as compared to nonischaemic myocardium. Values of lipid peroxidation products in non-ischaemic muscle were significantly higher than those detected in the intact heart of sham operated pigs. It is concluded that free radicalmediated membrane lipid peroxidation is involved in the mechanism of injury in „healthy" portion of infarcted heart. (Submitted to J. Mol. Cell. Cardiol).

Content of malondialdehyde (MDA) as an index of the intensity of membrane lipid peroxidation was measured in rabbit heart homogenate containing xanthine oxidase and ferrous ions as hydroxyl radical generating system. Prostacyclin and its stable analogue (Iloprost) inhibited MDA production equally to mannitol, supporting our earlier suggestions (Pharmacol. Res. Comm., 1986, 18, 321) that elimination of active oxygen metabolites might be involved in the mechanism of cytoprotective action of prostacyclin (subbmited to Biochem. Biophys. Res. Comm.). In in vitro system, it has been found that serum HDL inhibits membrane lipid peroxidation (Abstract of World Congress of Cardiol, 1986, Washington).

Continuing investigations on the mechanism of beneficial effect of propranolol in hypertrophic cardiomyopathy, the influence of this drug upon the processes of protein degradation was studied in hypertrophic rat myocardium in vitro. Propranolol inhibited activity of neutral proteases and stimulated acid and alkaline proteases. Stimulatory effect of propranolol upon the activity of

the latter enzymes, together with earlier detected inhibition of protein biosynthesis might influence protein turnover in hypertrophic cardiomyopathy (submitted to Bas. Res. Cardiol.).

See the List of publications:
13, 40, 41, 64, 114, 143.

Laboratory of Experimental Surgery
Head: Assoc. prof. Jerzy Borkowski

CLINICAL AND EXPERIMENTAL STUDIES ON NEUROREGULATION OF PERIPHERAL CIRCULATION

The purpose of the work was to investigate the usefulness of the method based on the measurement of skin impedance for the assessment of the effects of transcutaneous electric stimulation in patients with Raynaud's disease and syndrome.

The investigations were carried out with patients, in whom thermographic investigations had previously shown a positive therapeutic reaction to electrostimulation. The stimulation was carried out with a rectangular pulse current (1 ms width) with the frequency of 100 Hz and was applied for 40 min. The voltage was selected individually; the criterion being the maximum signal strength which did not cause pain or fibrilation. The values fell within the range of 10—40 V with 3—35 mA current.

The measurement of skin tissue impedance to alternating current (module of impedance — MI) was carried out by the bipolar method with selected current frequencies in the range from 100 to 100000 Hz using copper and silver electrodes of 5 cm^2 surface area placed on both forearms within the effective range of electrostimulation. One of the forearms was subjected to stimulation, while the other one served as a control. The measurements were carried out immediately and 10 min after the stimulation. Before stimulation the MI values for different frequencies of the measuring current ranged from 1230 to 140 ohm, the difference between the stimulated and the control forearm being less than 5%. After stimulation a decrease in MI in the stimulated area was recorded in all patients, averaging 39% (in the range from 52 to 27%). In the control forearm the differencies before and after the stimulation procedure were less than 2%.

In spite of the imperfections connected with the bipolar measurement method, it was demonstrated that in patients with Raynaud'a disease and syndrome, changes in blood circulation resulting from electrostimulation can be assessed by impedance measurements. The decreased MI is an evidence of increased blood flow in the stimulated area.

A comparison of the results of impedance measurements with the data obtained in thermographic investigations carried out in the same patients have shown that the information obtained using these two methods is identical.

STUDIES ON THE STRUCTURE AND BIOLOGICAL PROPERTIES OF THE NERVOUS TISSUE

Dapartment of Neuropathology
Head: Assoc. prof. Irmina B. Zelman

I. Pathogenetic mechanism of ischemic encephalopathy and attempts at its prevention

1. Counteraction of ischemic brain damage by prostacyclin PGI_2 and a calcium channels blocker — nimodipine.

The studies with prostacyclin focused on the effects of the compound on the ultrastructural changes of CNS following 20-min complete ischemia. Examination comprised the different types of cells in the motor and sensory cortex and the fat from frontal and parietal lobes. Prostacyclin appeared to reduce the ultrastructural postischemic damage when administered both before and in the course of ischemia, but also during reperfusion. The cytoprotective effect was most pronounced with regard to astrocytes and capillaries and less so to neuronal elements. A hypothesis has been put forward relating cytoprotection by PGI_2 to prevention of cytotoxic brain edema. In another study, PGI_2 was observed to counteract selective damage of neurons of Amon's horn CA_1 sector, resulting from moderate ischemia. However, the protective effect became apparent only on administration in the early stage of ischemia.

The assumption that nimodipine, a calcium channel blocker, may be a valuable cytoprotectant against ischemia was based on the hypothesis relating nerve cell damage to excess calcium influx. The effect of nimodipine was directly tested in two experimental models: a) moderate cerebral ischemia in Mongolian gerbil, leading to selective damage of neurons of Amon's horn CA_1 sector and b) anoxia in organotypic nervous tissue culture in vitro. The drug was found to protect the CA_1 neurons against ischemia, however, the effect was even more dependent upon the administration protocol than in the case of PGI_2 or indomethacin. Complete prevention of the changes was only noted on repeated, extended to 24 h administration of nimodipine. Nimodipine ws also found to prevent anoxic changes in vitro. This effect primarily involved astrocytes and consisted in reduction of cytoplasmic swelling. This result also provides evidence in favour of the long disputed presence of calcium channels in the astrocytes. Further investigations on the protective activity of nimodipine will include the model of complete cerebral ischemia in the rabbit, of which the relevant biochemical parameters of neurotoxicity (extracellular concentrations of excitatory amino acids, calcium etc.) have been established in cooperation with the Department of Neurochemistry.

2. Mechanism of selective damage of hippocampal neurons in moderate cerebral ischemia

Electron microscopic analysis of neurons of Ammon's horn CA_1 sector in

the period from cessation of ischemia up to the cell desintegration (5th day after ischemia) revealed that the maturation of the pathological changes is a biphasic process. It starts with an apparent functional hyper-activation of neurons in the first 24 h after ischemia and is followed by progressive degeneration with the onset on the second day after ischemia. The degenerative changes are clearly distinct from typical ischemic changes in that they primarily involve the endoplasmic reticulum with relatively good preservation of mitochondria.

3. Pathophysiological characteristics of an experimental model of clinical death

The investigations deal with the pathophysiological evaluation of clinical death in the rat, in a model allowing reanimation and long-term survival. A detailed analysis of cerebral blood flow in the conditions of 5—10 min clinical death confirmed complete brain ischemia in the period of cardiac arrest on the one hand, and characteristic dynamic of CNS reperfusion on the other hand. The results indicate that complete recovery of bioelectric activity of cerebral cortex is possible only in animals which underwent postischemic hyperperfusion.

Preliminary immunological examination of animals that survived 6 months after 10-minute clinical death revealed the presence of antineuronal antibodies in their sera, which may participate in the pathomechanism of postischemic brain damage.

II. **The role of glia in physiological and pathological processes of CNS**

Studies with astrocyte-enriched cultures prepared from the neonatal rat cortex revealed the presence of astrocytes of functional receptors to execitatory amino acids and the inhibitory amino acid GABA. Exposure to excitatory amino acids resulted in an increase in both the breakdown of membrane inositol phospholipids and calcium flux. Quisghalate receptors were identified as prevalent subclass of glutamate receptors. GABA was found to inhibit calcium flux and to abolish the stimulatory effect of glutamate, indicating some form of interaction between the glutamate and GABA receptors coupled to the calcium channels (in collaboration with the Brain Research Group, Open University, Milton Keynes, UK).

Oxygen consumption was measured polarographically in fractions enriched in astrocytes or neurons and in synaptosomes derived from rats in which two subsequent stages of acute hepatic encephalopathy (HE) were induced by thioacetamide treatment. A decrease of oxygen consumption was noted in astrocytes from animals with coma, well in agreement with the decrease of CM-RO_2. In contrast, at the same stage the oxygen consumption in neurons was increased, whereas synaptosomes remained unaffected. The results confirm the view that astrocytes are the cells of which the metabolism is primarily affected in HE.

Two brain enzymes participating in the arginine to GABA pathway: arginase and ornithine aminotransferase (OAT), were assayed in synaptosomes and astrocytes of rats with hepatic encephalopathy (HE) induced by thioacetamide. In synaptosomes, both enzymes showed and enhanced activity in advanced HE, suggesting increased synthesis of the neurotransmitter GABA. This could lead to increased GABA-ergic neurotransmission supposed to contribute to HE. In astrocytes only OAT showed and increase, possibly reflecting metabolic activation of these cells.

The cerebral pyruvate carboxylase — the major CO_2 fixing enzyme of the brain and an astroglial marker — was assayed in different stages of acute hepatic encephalopathy (HE) produced in the same thioacetamide model. In advanced HE there was a marked drop of the enzyme activity, coinciding with decreased brain α-KG and increased brain ammonia. In the late recovery period from HE, a durable stimulation of the enzyme was noted, accompanied by increased α-KG and transiently decreased brain ammonia. The results indicate that pyruvate carboxylase activity may contribute to changing ammonia detoxicating potential of the brain during HE, in agreement with the concept linking the fixation of CO_2 to the Krebs cycle with ammonia metabolism.

Histological, ultrastructural and immunohistochemical studies were performed on blood vessels adjacent to cerebral and spinal cord gliomas induced by ethylnitrosourea (ENU). The vessels contacting spinal cord tumours, but not those associated with cerebral tumours, were observed to be partly displaced from the neoplastic tissue by peritumoral edema. The disrupted tumour--vessel contact appears to be responsible for impaired maturation of the vessels, as revealed by persistent anti-Factor VIII staining.

III. Pathogenetic mechanisms of selected endo- and exogenous degenerative processes of the CNS, with special attention to the action of specific neurotoxins

The studies have concentrated on the effects of MPTP (1-methyl-4-phenyl-1, 2,3,6-tetrahydropyridine) — a model neurotoxin inducing symptoms of Parkinson's disease. Electron microscopic investigations revealed that, when administered into mice, MPTP leads to selective damage of dopaminergic neurons of pars compacta substantiae nigrae with consecutive alteration of their axonal endings in putamen, expressed among others by a significant reduction of number of their granular synaptic vesicles. These abnormalities were accompanied by remarkable fibrogliosis, specially intense in putamen. The results of these studies indicate that MPTP selectively damages this part of nigrostriatal system, which begins in pars compacta substantiae nigrae and ends in non--lymbic part of the striatum, preserving that one which, starting in area A_{10} of substantia nigra, innervates neurons belonging to limbic portion of striatum, represented by nucleus accumbens and nucleus tuberculi olfactorii. When added to organotypic nerve tissue culture, MPTP brings about selective degene-

ration of dopaminergic neurons and their synaptic endings. The structural changes developed already after the first dose of MPTP in cultures treated for three or four days. This acute response of substantia nigra cells in in vitro conditions was in contrast with the results reported for the animal model of Parkinson's disease induced by prolonged exposure to MPTP.

Studies on the effect of MPTP on the respiration of isolated neurons (microdiver technique), revealed that the neurons of the nigrostriatal pathway are selectively vulnerable to the toxin. Within this pathway striatal neurons manifested more rapid and pronounced inhibition than substantia nigra neurons. Ascorbinic acid — a powerful antioxidant — markedly attenuated the MPTP-induced decrease of striatal dopamine content, which is consistent with the view of MPP^+ being the toxic metabolite of MPTP.

Studies on prolonged subacute manganese intoxication revealed the changes in the axons and myelin sheaths to selectively develop in substantia nigra and striatum, which confirms a particular vulnerability of these structures to the metal.

Electron microscopic evaluation of glia before-, during, and after myelination, in pt rabbit revealed disturbances in the differentiation and maturation of oligodendrocytes. Extended period of proliferation of these cells and myelination gliosis are likely to be related to the mitotic activity of these cells during myelination. Disturbances of maturation were found to involve astroglia as well. Immature astrocytes showed features of hypertrophy in the period preceding myelination and excess arborization of processes before and during myelination. These observations point to the primary character of astroglial reactions as well as their possible interference with ensheathment of nerve fibres in pt mutant.

See the List of Publications:
1, 2, 3, 4, 5, 16, 27, 32, 42, 52, 55, 57, 58, 59, 73, 81, 82, 83, 84, 85, 86, 87, 88, 97, 99, 100, 101, 102, 111, 112, 113, 115, 116, 117, 129, 135, 136, 137, 138, 144, 145, 155, 162.

Department of Neurochemistry
Head: Assoc. prof. Jerzy Łazarewicz

I. EFFECTS OF ISCHEMIA, HYPEROXIA AND NEUROTOXINS IN THE LEVEL AND TRANSPORT OF CALCIUM AND NEUROTRANSMITTERS IN THE CENTRAL NERVOUS SYSTEM

The brain dialysis in vivo was applied in studies on the effect of complete brain ischemia of extracellular concentration of calcium and neuroactive amino acids and blood-brain barrier permeability in the rabbit hippocampus, supplemented with pathophysiological and morphological measurements. It was shown that 15-min ischemia induced biphasic changes of extracellular calcium concentration (a decrease proceeded by a transient elevation). The level of

calcium normalized after 45 min recirculation. Brain ischemia induced a rapid increase of extracellular glutamate, aspartate and taurine concentrations and delayed long-lasting release of phosphoethanolamine. The permeability of blood-brain barrier to fluoresceine was elevated. A coincidence in time of the normalization of extracellular glutamate and calcium concentrations with EEG activity reappearance was noted which may suggest a causal relationship between those events.

In cooperation with the Institute of Neurobiology, University of Göteborg (Sweden) it was demonstrated that kainic acid when applied in the dialysis medium to rabbit hippocampus in vivo stimulates calcium influx to neurons and activates synaptosomal calcium uptake in vitro. The specific features of kainate-avoked Ca fluxes suggest the involvement of voltage-independent ionic channels. Further studies exhibited also the receptor — mediated drop of calcium and increase of glutamate, aspartate taurine and phosphoethanolamine in ectracellular space of the rat striatum. These effects were not modified by cortico-striatal deafferentiation but were significantly attenuated by the excitotoxic lesion to the striatum, which suggests post-synaptic, dendrosomatic site of kainate effects. In the collaboration with the Pavlov Institute of Physiology, USRR Academy of Sciences, Leningrad, (USRR), in vivo micospectrofluorimetric studies were performed with chlorotetracycline — calcium chelate probe, which exhibited kainate-evoked release of calcium bound to cortical membraneous structures, closely resambling effects observed in initial phase of brain anoxia. Results of studies with kainate exhibited numerous similarities with ischemic disturbances which suggests the involvement of excitatory amino acid neurotransmitters in the pathomechanism of ischemic brain damage.

The studies on neurotoxic effects of normobaric hyperoxia exhibited significant activation of lipid peroxidation in nerve endings with concomitant inhibition of GABA uptake, whereas the spontaneous and depolarization-evoked GABA release, ^{45}Ca uptake and synaptosomal membrane potential remained unchanged. The specific high sensitivity of GABA uptake system in synaptoplasmatic membranes to free radical attack is suggested. It was found in collaborative studies with the Institute of Occupational Health in Helsinki (Finland) that sulfides, industrial toxins with predilection to CNS, when administered in vivo to rats, not only inhibit synaptosomal respiration (which was reversed by heme) but also a decrease in GABA and dopamine uptake and release as well as depolarization evoked by veratridine, in heme-insensitive manner. These results suggest that neurotoxicity of sulfides may be partially evoked by their direct inhibitory effect on neurotransmission.

II. METABOLISM OF ACIDIC LIPID PROTEIN-LIPID INTERACTION AND PROTEASES ACTIVITY IN THE ISCHEMIC BRAIN AND DURING DYSMYELINATION OF C.N.S.

The in vivo studies on polyphosphatidylinositols in the brain was continued. Ischemic insult 1 min and 10 min and also agonist of cholinergic recep-

tor — carbachol induced 50% degradation of phosphatidylinositoldiphosphate (PIP_2). Concomitantly 1 min ischemia and carbachol produced increase of all inositolphosphates and among them inositoltriphosphate. After 10 min ischemia only the increase of inositol-mono-1-phosphate was found as a results of ischemically induced activation of inositoldiphosphate and inositoltriphosphate phosphatases. Cholinergic receptor antagonist atropine and gammabutyrolacton applicated 10 min before ischemia protected brain agonist ischemically induced changes of membrane phosphoinositides. These compounds protected the brain agonist accumulation of two second messengers fusogenically acting diacylglycerol and inositoltriphosphate liberating intracellular calcium (Wikieł, Halat, Strosznajder). The results indicated the involvement of cholinergic receptor in PIP_2 degradation induced by ischemia. In vitro studies were performed for the determination of phosphatidylinositol degradation by the enzyme from the brain submitted to ischemia.

Ischemia (10 min) induced activation of enzymes degradating PI by about 60% mainly phospholipase C exclusively in synaptosomes. Atropine and gammabutyrolactone diminished ischemically activated phospholipase C activity. Results indicated that not only PIP_2 but also PI degradation is connected with the stimulation of cholinergic receptor during ischemia (Wikieł, Strosznajder). The other acidic lipids gangliosides play an important role in the regulation of neuron function. The effect of GM_1 gangliosides on Ca transport and Ca^{2+} binding to synaptosomes was studied. A significant inhibition by gangliosides of the depolarization-induced calcium uptake and changes in calcium binding was observed. The results suggest that gangliosides are potent modulators of synaptosomal calcium fluxes and equilibration in the membrane (Domańska-Janik, Noremberg, Łazarewicz). The modulatory effect of free fatty acids and lysophospholipids on calcium dependent neutral proteases (CANP) was investigated. Low calcium ion requiring CANP is more strongly inhibited by unsaturated fatty acids than mCANP high calcium ion requiring formed.

Lysophospholipids 10^{-5}—10^{-3} M have an exclusively inhibitory effect on uCANP. Calpastatin decreases the activity of mCANP in the presence of polyunsaturated fatty acid while in the presence of lysophospholipids it does not exert any depressive effect on enzyme activity. The results indicate that fatty acids and lysocompounds liberated in different physiological and pathological conditions may modulate calcium-activated neutral protease (Zalewska, Strosznajder).

In the studies on hexose monophosphate pathway HMP activity it was observed that mild hypoxia (7% O_2 in N_2 during 2 h) decreased HMP activity to 30% of control level whereas during first hour of reoxygenation the activity increased up to 200% of the control.

The changes in HMP activity in CNS may be involved in modulation of lipid and other macromolecules synthesis in CNS. In the studies on neurological mutant paralytic tremor „pt" rabbit, the reduced amount of myelin to 25—30% of control and the disturbances in myelin lipid-protein relationship was obser-

ved. The formation of protein-lipid complex in „pt" rabbit myelin decreased by about 10—15% as compared to control.

In protein-lipid complex there was a decrease by about 34% of protein and by about 16% of acidic phospholipids.

The changes in lipid-protein relationship in „pt" rabbit myelin and in the formation and composition of protein-lipid complex may be responsible for the disturbances of myelin formation and compaction in „pt" rabbit.

See the List of Publication:
12, 22, 23, 33, 34, 35, 70, 71, 72, 104, 108, 109, 126, 127, 147, 148, 149, 150, 151, 152, 158, 159, 160.

Department of Neurosurgery
Head: Prof. Eugeniusz Mempel

I. EFFECT OF DIHYDROBENZPERIDOL ON SOMATOSENSORY EVOKED POTENTIALS IN PATIENTS WITH EXTRAPYRAMIDAL DISEASES

The effect of small, single doses of dihydrobenzperidol (DHB) on the registration of somatosensory evoked potentials (SEPs), disturbed by miogenic artefacts was studied in patients with extrapyramidal disorders. The examinations were performed in two groups of 10 patients subjected to thalamotomy: a) with parkinsonian syndromes and b) with different forms of muscular dystonia. The cortical SEPs were studied before and after thalamotomy, thalamic and cortical SEPs during surgery.

The results indicate that DHB improves significantly the recordings of both cortical and thalamic SEPs. In parkinsonians, more than in dystonic patients, an increase of amplitude and latency of all components of cortical SEPs was observed in preoperative recordings. The greatest effect of DHB was demonstrated in the negative and late latency components of the potential, which indicates that cortical associative connections were concerned. In the postoperative recordings of the SEPs there was a clear influence of DHB on the increase in the early components P_{18} — N_{22} of the potential (by about 30%). The stereotactic operation does not damage the region of the thalamus, which is responsible for the specific components P_{18} — N_{22} — P_{29} of the SEP. On the other hand, oscillatory components between P_{29} — N_{38} — P_{44}(which occur during the afferent voley of cerebellar stimuli to the thalamus) observed in parkinsonians before surgery, disappear after the operation. The presence of these components correlates with the clinical observation of parkinsonian tremor. Their absence in the postoperative SEP recording is of good prognostic value when evaluating the effects of surgery.

II. DISTURBANCES OF THE VOLUME-PRESSURE RELATIONS IN DIFFERENT STATES OF INTRACRANIAL PATHOLOGY — DIAGNOSIS AND TREATMENT

Simulation and clinical studies on the optimalisation of diagnostic infusion tests resulted in the implementation of the so-called Optimal Infusion Test (OIT). The OIT combines the features of the bolus and constant infusion tests, however, with substantially reduced volumes infused, ICP increases and duration of the procedure. A special computer — controlable infusion pump has been designed, enabling the automation of the OIT procedure.

Experiments on cats were carried out to study the effects of hemodilution on disturbed pressure-volume relations. The model with the epidural baloon was used, and slow and fast volume changes were applied. The results indicate, that hemodilution decreased the disturbances in the measured Visual Evoked Potentials (VEPs) in animals with developing edema, but practically had no effect on VEPs in the process of herniation.

Continuing the study on distant effects of head trauma, 26 new patients have been examined, giving a total of 106 cases. In about 30% of cases with subarachnoid bleeding, increased outflow resistance (R_0) was observed in the early period after trauma (2 weeks to 3 months). In all cases but one, R_0 normalised in the test carried out later on, but it is in the group of patients with temporarily increased R_0 that were found most of the cases with head aches, inability to work and other complaints.

III. INVESTIGATIONS OF HIGHER NERVOUS ACTIVITES AFTER FOCAL BRAIN DAMAGES

1. An estimation of the process of verbal material learning and the influence of interference factors in patients with brain damages;
2. Memory investigations in patients with speech comprehension disorders

Ad. 1. Comparative investigations (before, immediately after and a long time after the operation), concerning learning of verbal material and its retrieval after interference were performed in 40 patients subjected to thalamotomy and in 20 patients after amygdalotomy and (or) hyppocampotomy. The results demonstrate that the disorders in learning of verbal material are independent of the etiology of the disease process (involuntary movements, epilepsy), the localization of the brain damage (thalamus, nucleus amygdalae, cornu Ammonis) and the damaged hemisphere (left, right). The learning difficulties are caused by short-term memory impairments. It is also evident that interfering factors disturb retrieval processes independently of the damage localization and period of the investigation.

Ad. 2. Memory investigations were performed in 17 patients with speech comprehension disorders after left hemisphere damages. Visual pattern series were

applied (pictures, objects, inscriptions). Patients were able to identify the visual material and remembered real objects for a long time. These results seem to be important for rehabilitation purposes.

See the List of Publications:
15, 16, 39, 46, 78, 79, 80, 153

Laboratory of Developmental Neuropathology
Head: Prof. Maria Dąmbska

THE COMPARISON OF NORMAL BRAIN DEVELOPMENT WITH ITS DISTURBANCES PROVOKED BY SELECTED DAMAGING FACTORS AND PATHOLOGIC PROCESSES

Two main problems were studied: the first one consisted in the estimation of late effects of perinatal white matter damage on further maturation of the CNS. Brains of newborns and children were neuropathologically examined. It was stated that perinatal pathology resulted in two types of white matter damage: classical periventricular ischemic infarcts and more diffuse oedema resulting in colliquative necrosis. The examination of cases with several years of survival allowed to observe the final effect of these changes and to reconstitute the clinical history of cases with diplegic forms of cerebral palsy. The diffuse white matter damage leading even to hydranencephaly was also investigated in six cases of congenital toxoplasmosis. The role of proliferation of subependymal glia in this process was described.

The second problem concerned the incidence and topography of disseminated intravascular coagulation (DIC). High incidence of DIC in CNS was found in cases with this syndrome appearing in other organs. The topographic distribution of microthrombi was particularly high in of the vessels of the periventricular white matter which suggests their contribution to the lesions of this structure.

In addition experimental studies on toxical effects of cytostatic drugs on immature brains were performed. Pathologic changes occurring after administration of vincristine to rabbits some days after their birth (fibryllary tangles in nerve cells and axons and myelin damage) were similar to those observed in adult animals after an intrathecal administration of this drug.

See the List of Publications:
17, 18, 19, 20, 21, 28, 43, 63, 66, 67, 74, 75, 76, 128, 139, 142, 163.

Laboratory of the Ultrastructure of the Nervous System
Head: Prof. Jerzy Borowicz

CHANGES IN NEUROSECRETORY SYSTEM FOLLOWING BRAIN ISCHEMIA

Studies of ultrastructural changes in secretory nuclei (n. supraopticus and n. paraventricularis) and hypophyseal neural lobe were performed after complete brain ischemia. Ischemia was evoked according to the method of Korpaczew et al. In this model, constriction of the vascular bundle of the heart led to cardiac arrest, cessation of blood circulation and clinical death- Release of constriction of the heart vascular bundle after 2.5 min and application of external heart massage allowed for return of the heart function, blood circulation and respiration within 1—3 min. For ultrastructural studies, material was taken immediately and three days after experimentally evoked ischemia. Material from rats not subjected to ischemia served as a control.

Immediately after ischemia, the great majority of hypothalamic secretory neurons exhibited ultrastructural features indicating their increased activity. This was manifested by hypertrophy of granular endoplasmic reticulum, well developed Golgi complex and an abundance of secretory vesicles. Also observed were neurons showing degenerative features, so called „dark neurons", in the vicinity of which typical astroglial cells were present. Three days after cessation of ischemia majority of neurons were normal. The results of these studies indicate that complete ischemia of short-duration evokes ultrastructural changes which are reversible. Immediately after the experiment the majority of axons did not differ from the control ones. On the other hand, a small population (about 10%) showed features, which may indicate enhanced processes of secretion. All axons observed contained swollen mitochondria, characteristic of ischemic changes. It is important to note that three days after the experiment the number of axons showing degenerative features increased. These results indicate disturbances of axonal transport of neurosecretory material from the hypothalamic secretory nuclei to the neurohypophysis and an inhibition of secretion of neurohypophysial hormones. In this experimental model, ultrastructural changes in the gigantocellular region of the medullary reticular formation were also studied. In first experimental group, ultrastructural changes affecting the gigantic neurons were observed and consisted of a swelling of mitochondria. Ultrastructural changes persisted and were more pronounced in the second experimental group. Gigantic neurons had, in general, a tendency for Golgi complex and granular endoplasmic reticulum enlargement. In the nuclei of these neurons, coiled bodies and filamentous spindle-shaped inclusions were occacionally observed „Dark" gigantic neurons exhibiting degenerative features were also observed. These features suggest changes in the activity of gigantic neurons after ischemia. These changes were accompanied by edematous changes of astroglial cells prominent in the perivascular tissue.

See the List of Publications:
25, 69, 161.

STUDIES ON TRANSPLANTATION AND EXPERIMENTAL SURGERY

Department of Surgical Research and Transplantation
Head: Prof. Waldemar Olszewski

CHARACTERIZATION OF „PASSENGER" LEUKOCYTES IN NORMAL HUMAN SKIN

The mechanism of cell migration from blood capillaries to the prenodal lymphatic vessels is poorly understood. The studies on the ability of certain cell populations to extravasate in the non-lymphoid tissues may lead to further elucidation of that process. Peripheral afferent lymph consist of lymphocytes and macrophage-like cells with the appearence of veiled cells. Eighty percents of macrophage-like cells binds OKM1 monoclonal ab, 100% is OKIa1+ and M710+ (cytoplasmic marker for human macrophages), 70% is OKT6+, 80% -OKT9+ (α-transferrin receptor), and 100% is slightly OKT4+. No IL1 could be detected by means of anti-IL-1 antibody either on the membrane or in the cytoplasme. Among the small mononuclear cells 60% were OKT4+ and 18% OKT8+. Ten % of cells attached antibody againts epithelial membrane antigen. Some cells reacted with the moab against factor VIII. In functional tests macrophage — like veiled cells were more effective in antigen presentation, the AMLR and MLR as compared to blood monocytes. The results show that lymph migrating macrophage-like veiled cells are active in stimulation of autologous and allogenic mixed lymphocyte cultures. This indicates that they may be responsible for immunogenicity of skin allografts.

STUDIES ON THE ACTIVATION OF CIRCULATING BLOOD MONOCYTES AFTER OPERATIVE TRAUMA

Operative trauma results in a decreased response of blood mononuclear cells in AMLR (autologous mixed lymphocyte response). In the present studies the influence of exogenous IL-2 on the reconstruction of AMLR was investigated. The studies were performed in 8 patients undergoing cholecystectomy. It was found that addition of IL-2 (produced by the MLA114 line) to the AMLR cultures performed after the operation restored the AMLR responce to the properative level. An operative trauma caused the decrease in % of OKT4+ cells on day +1 after the operation. The low level of OKT4+ cells persisted until day 5. The normalization of the OKT4+ cell level correlated with restoration of AMLR response. The results suggest that the decreased level of AMLR in the early postoperative period may be caused by the decreased number of IL-2 producing OKT4+ cells and/or by a defect in the production of IL-2 by these cells.

STUDIES ON THE DISTRIBUTION OF ADOPTIVELY TRANSPLANTED DONOR LYMPHOCYTES RESPONSIBLE FOR PROLONGATION OF HEART GRAFT SURVIVAL

The studies on the kinetics of solid grafts rejection versus cellular one were carried out. Accumulation of 51 Cr-labelled allogeneic lymphocytes, in the spleen and lymph nodes of the recipient was found lower than in the syngeneic controls. Concurrently, the radioactivity of protein-bound 51 Cr in serum and kidneys was increased, which suggests the in vivo damaging of allogeneic lymphocytes. Recipient's splenectomy brought about an increased accumulation of allogeneic lymphocytes in the LN for the first 6 h after injection. Intravenous administration of anti- asialo-GM1-antibody (serum bloking NK function) partly inhibited the process of elimination of allogeneic lymphocytes. Donor lymphocytes and alloantiserum against donor lymphocytes given to the recipient 11 days and 10 days, respectively prior to inoculation of donor splenocytes, did not inhibit the rate of their elimination when compared with the untreated control. In contrast, pretreatment of heart graft recipients with donor lymphocytes and alloserum 11 and 10 days before grafting, respectively, caused prolongation of the graft survival time above 40 days. These results show that the enhancement protocol effective in prolongation of the allograft survival does not prevent elimination of i.v. transplanted allogeneic lymphocytes. This may suggest that the mechanism by which i.v. injected splenocytes are rejected in the allogeneic recipients differs from that of rejection of solid tissue grafts.

REJECTION PATTERN OF NERVE ALLOGRAFTS-CHANGES IN GRAFT AND HOST CELL DETERMINANTS

Two processes develop simultaneously in rejecting nerve allografts: Wallerian degeneration and infiltration by host immune cells. In the first, graft Schwann cells become activated and host mo are attracted by degraded myelin, in the other, host immune cells are recruited due to the allogeneic signal. The use of monoclonal antibodies allowed to descriminate between the two processes and study their kinetics during acute nerve rejection and in course of treatment with CsA. We found that epineural sheets of normal BN nerves contained Ia+(OX6+) and ED1+(mo,dendritic) dendritic-like cells. Schwann cells turned to be Ia- and ED1-. Seven days after syngeneic transplantation Schwann cells become ED1+ but remained Ia-. Treatment of syngeneic transplants with CsA did not inhibit ED1 expression. In acutely rejecting grafts Schwann cells became ED1+ and Ia+. Graft bed and epineurium were infiltrated with Ia+, ED1+ and some OX8+ cells. Treatment with CsA reduced number of infiltrating Ia+ and ED1+ cells. Schwann cells remained mostly Ia- but were still ED1+. Expression of ED1 determinants on Schwann cells follows Wallerian degeneration, whereas that of Ia determinants is the result of allogeneic rejection.

AN INFLUENCE OF ACUTE AND CHRONIC VENOUS STASIS ON THE KINETICS OF THE PLASMA PROTEIN AND CELL TRANSPORT INTO THE TISSUE FLUIDS AND LYMPH

An influence of acute and chronic venous stasis on the kinetics of lymph production was examined in dogs and human volunteers. The question was whether the rapid increase of venous pressure in capillary system will be followed by an increase in capillary filtration estimated as a net capillary fluid flux. Experiments in dogs showed that acute venous stasis decreased interstitial fluid pressure (from — 1,2 mm Hg to — 1,8 mm Hg). The decrease of lymph flow in 50% of the cases was observed. Venous hypertension did not influence the lymph pressure. The changes in non migrating cells (erythrocytes) correlating with lymph flow were observed, whereas, leukocytes remained unchanged. Differential count revealed a decrease in lymphocytes and monocytes (from 78% to 41%), veiled cells (from 16 to 9%) and increase of polymorphonuclear cells (from 26 to 50%). The studies in volunteers showed that acute venous stasis provoked an increased lymph pressure with concomitant decrease of pulse frequency, amplitude and lymph output which correlated with venous hypertension caused by femoral vein ligation. Chronic venous stasis (seven days) caused in dogs a slight increase of interstitial fluid pressure. The concentration of leukocytes in chronic venous stasis was higher than in acute one, as a result of an increased level of polymorphonuclear cells and veiled cells. In volunteers — chronic venous stasis cause an increased lymph flow. The lymphatic pulse was unstable due to secondary changes in the vessels. A kinetics of penetration of different antibiotics to lymph and blister fluid was also examined. It was found that antibiotics with a high protein serum binding activity (cyprofloxacin, flukloxacillin) penetrate well to tissues and after high doses their concentration in lymph and blister fluid remains within minimal inhibitory concentration. However, the degree of penetration to tissues of antibiotics with the high serum protein binding activity (flukloksacyline, 96% binding) is only 20%, while that of antibiotics with low serum protein binding activity (gentamycine, 0% binding) is 100%. The results show that to establish the proper therapeutic doses of antibiotics the degree of their binding to serum proteins should be considered.

The study on stimulatory activity of lymph fluid was also performed. It was shown that human lymph contained interleukin 1 (IL-1). IL-1 activity was not present in serum. It seems that IL-1 is not produced by migrating mononuclear lymph cells but rather by epidermal cells. IL-1 present in the lymph may modulate not only the local response in the skin but also in the regional lymph node.

See the List of Publications:
29, 37, 62, 68, 89, 90, 93, 94, 95, 96, 106, 118, 119, 120, 121, 146.

OTHER RESEARCH WORKS

Mental Health Department
Head: dr Zygfryd Juczyński

1. THE CAUSES AND DETERMINANTS OF MALADJUSTMENT OF CHILDREN AND YOUTH

The model of mental health accepted in the investigations is based on two elements namely: the ability of an individual to constant development and the dynamic adjustment to the changes of social, psychological and biological environment. The mental health has a multidimensional character, it is a process depending on a relationship between an individual and his environment.

In the investigations a functional approach underlining its explorative function is preferred. The conducted research have an interdisciplinary (medical, psychological and social) as well as longitudinal character. The subjects of the investigations are the children and youths of 6—20 years of age. Series of investigating instruments, in majority standarized, have been elaborated and adapted for the preliminary examinations. After introduction of necessary changes basic investigations have started.

2. AN INFLUENCE OF PERSONALITY AND PSYCHOSOCIAL CONDITIONS ON THE COURSE OF TREATMENT OF SOME MALIGNANT TUMOURS

In the course of treatment of the neoplastic disease the psychosocial factors determining the forms and degree of patient's activity play a considerable part. The attitude against illness and the functioning in the patient's role is determined by a series of paramedical variables.

The program of investigations has aimed to identify the psychosocial factors, permitting to predict the treatment prognosis. In preliminary investigations a group of 28 patients was examined with diagnosis of breast, collum uteri, and large intestine cancer. The results pointed out the attention on frequent defense mechanism (denial), connected with intensified anxiety.

3. THE PROBLEM OF THE STABILIZATION OF PERSONS TAKING SOME DEPENDING DRUGS AT YOUNG AGE

Before starting basic investigations it is necessary to concentrate on construction of the research program, including the principles and hypotheses. The main attention has been paid to elaboration of the questionnaire for medical and environmental examinations. After the preliminary investigations some indispensable corrections have been introduced.

See the List of Publications:
45, 46, 107, 123, 130, 131, 156, 157

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Grzelak I.	Det Norske Radiumhospital, Norway (long term visit)
Kubicka U.	FEBS Summer School, God, Hungary
Lukomska B.	Immunology Dept., Karolinska Institutet, Sweden, (long term visit)
Orlewska E.	FEBS Summer School, Jonian Village, Greece
Plachta J.	FEBS Summer School, God, Hungary

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PARTICIPATION IN INTERNATIONAL SCIENTIFIC MEETINGS IN 1986

XXVII International Symposium of Psychopharmacology
January 1986, Jesenik, Czechoslovakia
Rydzyński Z.

INTERVISC Conference
2—5 Feb. 1986, Libic, Czechoslovakia
Dąmbska M.

4th Intern. Congress of Child Neurology
13—16 March 1986, Jerusalem, Israel
Renkawek K.

Symposium on Lymphoedema
17—18 March 1986, Norwegian Radium Hosp., Oslo, Norway
Olszewski W.L.

European Stem Cell Meeting, Innsbruk, Austria
1—3 April, 1986
Ratajczak M.

Soviet-Polish Symposium on Cerebral Ischemia, Tbilisi, USSR
21—24 April 1986
Czernicki B., Gadamski R., Gajkowska B., Gordon-Majszak W., Jurkiewicz J., Łazarewicz J., Mossakowski M., Pluta R., Renkawek K., Strosznajder J.

The Mainz Symposium of The European Society of Cardiology
27—29 April 1986, Mainz, GFR
Cedro-Ceremużyńska K.

Deutsche Gesellschaft für Neurochirurgic
4—7 May 1986, Bonn, GFR
Czernicki Z., Jurkiewicz J.

XXI Congress of the European Society for Surgical Research
6—10 May 1986, Nancy, France
Olszewski W.L., Romaniuk A.

International Symposium on Effector Mechanisms in Tumor Immunity
13—15 May 1986, Roma, Italy
Łukomska B.

2nd Advanced Seminar in Neurosurgical Research „Regeneration and recovery processes in the nervous system"
4—6 June 1986, Venecja, Italy
Czernicki Z.

International Symposium on Cerebral Ischemia and Hemorheology
18—21 June 1986, Tegernsee, GFR
Czernicki Z.

VI Intern. Congress of Immunology
6—11 July 1986, Toronto, Canada
Gałkowska M., Grzelak I.

XXX Intern. Congress of Physiology IUPS
8—10 July 1986, Vancouver, Canada
Budohowski L.

IX Meeting of the International Microsurgical Society
27—29 July 1986, Brescia, Italy
Grochowicz P., Olszewski W.L.

2nd European Congress on Cell Biology
5—11 July 1986, Budapest, Hungary
Renkawek K.

XI Intern. Congress of the Transplantation Society
3—8 August 1986, Helsinki, Finland
Grochowicz P., Grzelak I., Olszewski W.L., Orlewska E., Romaniuk A., Wąsowska B.

17th FEBS Meeting of European Biochemical Society
24—29 August 1986, West Berlin
Budohowski L., Strosznajder J.

VI Conference of European Neurochemistry Society
2—5 September 1986, Prague, Czechoslovakia
Albrecht J., Gordon-Majszak W., Hilgier W., Kosicka B., Wikieł H., Wysmyk-Cybula U.

Erwin Riesch Symposium „Pathology of Cerebrospinal Microcirculation
2—5 September 1986, West Berlin
Dąmbska M., Gajkowska B., Iwanowski L., Kamionowska M., Kroh H., Maślińska D., Renkawek K., Zelman I.

Third Intern. Symposium on Myelination and Demyelination
13—17 September 1986, Varna, Bulgaria
Renkawek K., Taraszewska A., Weinrauder H.

5th Annual Congress Societas Europea Pneumologica
3—6 September 1986
Czerwosz L., Głogowska M., Grieb P., Janczewski W., Romaniuk J.

Xth Intern. Congress of Neuropathology
7—12 September 1986, Stockholm, Sweden
Dąmbska M., Iwanowski L., Kamionowska M., Kroh H., Mossakowski M., Maślińska D., Renkawek K., Zelman I.

Xth Intern. Congress of Cardiology
14—18 September 1986, Washington, USA
Cedro-Ceremużyńska K.

3rd Intern. Course on Physiological Biochemistry of Exercise and Training
1—4 October 1986, Athens, Greece
Kaciuba-Uściłko H., Nazar K.

19th Danube Symposium of Neurological Sciences
9—11 October 1986, Heidelberg, GFR
Dąmbska M., Mossakowski M., Renkawek K.

7th Intern. Symposium of Pedopsychiatry
13—18 October 1986, Suzdal, USSR
Rydzyński Z.

Symposium for Cinical and Experimental Immunology
24—27 October 1986, Halle, DDR
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5th Congress of Bulgarian Psychiatric Association
November, Sofia, Bulgaria
Rydzyński Z.

Experts' Round Table Meeting on Intracranial Hypertension
23—25 November 1986, West Berlin
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