REPORT
ON SCIENTIFIC ACTIVITIES
1988

POLISH ACADEMY OF SCIENCES
MEDICAL RESEARCH CENTRE

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1988

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

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Head: Prof. Witold Karczewski

PHYSIOLOGICAL AND PATHOPHYSIOLOGICAL ASPECTS OF RESPIRATORY AND CARDIOVASCULAR CONTROL

I. Functional organization of the respiratory neural network

1. It has been demonstrated that electrostimulation of certain areas of motor cortex evokes a biphasic response in phrenic motoneurons, consisting of a short-latency excitation and a subsequent inhibition. Such a pattern of response is similar to that evoked through the superior laryngeal nerve reflex arch. However, the interaction of cortical and reflex (SLN) stimulations suggests the use of different pathways in medulla and cervical spinal cord for the transmission of information from these sources.

2. An oligosynaptic inhibitory pathway from motor nucleus of trigeminal nerve to phrenic motoneurons, and a polisynaptic pathway from vagus nerve to motor nucleus of trigeminal nerve have been identified. The results suggest the involvement of the nucleus of trigeminal nerve in the central control of respiratory rhythm.

3. Stimulation of peripheral chemoreceptors with almitrine and cyanide in a guinea pig evokes respiratory responses which depend on the degree of anesthesia. Inspiratory stimulation has been observed only in lightly anesthetized animals. In deeper anesthesia respiratory depression has been observed, which presumably is a consequence of synergistic depressant action of general anesthesia and almitrine or cyanide on the central level.

4. Each of the roots of hypoglossal nerve and ansa cervicale display the neural activity synchronized with, and similarly responsive to chemical and nervous stimuli as the activity of phrenic nerve. However, the threshold of excitability of hypoglossus and ansa cervicale in response to carbon dioxide is substantially higher than that of phrenic nerve, and transient hyperventilation immediately inhibits respiratory activities there. This observation may be relevant to the mechanism of apnea in consequence of upper airway collapse during sleep.

5. It has been found, that the occurrence and magnitude of provoked deep breaths depend on the instantaneous balance between stimulatory and

inhibitory afferentation arriving through the vagi, and that the above mentioned balance may be different for phrenic and superior laryngeal nerves.

6. The studies of influence of vagal and laryngeal reflexes on the activities in XII and VII cranial nerves, and on the upper airway resistance have been conducted. A decrease of the upper airway resistance during reflex apnea related to the activation of the slowly-adapting pulmonary mechanoreceptors has been shown. During swallowing a complete closure of upper airway is followed by a ten second period of a marked decrease of the upper airway resistance. Electrical stimulation of XII cranial nerve also produces a marked decrease in resistance.

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

1. Intravenous application of serotonin enhances respiratory rhythm and increases laryngeal expiratory resistance in animals with chronically vagotomized lungs. These changes are not related to carotid bodies. Changes in respiratory rhythm are, however, markedly reduced and laryngeal expiratory resistance decreases, following cervical vagotomy. It seems that afferent va. gal pathways, originating from larynx some of them presumably serotonergic, substantially influence the central respiratory effects of serotonin.

2. Experiments with High Frequency Ventilation led to the conclusion that, contrary to the widespread opinion, phrenic apnea occurring during such ventilation is not related to vagal afferentation, as it occurs also in vagotomized animals. It is likely that apnea during high frequency ventilation may be an effect of activation of the respiratory muscle receptors.

3. Investigations of a neurological deficit appearing in the sequela of subarachnoid haemmorhage and cerebral vasospasm led to a method of prediction of the degree of the deficit based on the intraskin immuno-test (Immunoskin Test — SEVAC). The prognostic value of the test is consequential to the quantitation of the immunoreactivity to blood extravasation, and it is suggestive of the importance of immunoreactivity as the pathomechanism of SAH-related vasospasm. Histopathological changes in walls of brain arteries leading to, and/or associated with SAH have also been investigated.

See the List of Publications: a) 19, 20, 32, 41, 42, 61, 62, 70, 71, 75; b) 21, 38, 54, 69.

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ADAPTATION TO PHYSICAL EXERCISE AND CHANGES IN ENVIRONMENT

I. Factors determining carbohydrate tolerance and sensitivity of skeletal muscle to insulin

It was found that physical exercise increases sensitivity to insulin — measured in vitro as the insulin concentration inducing half maximal effect on lactate production and glycogen synthesis — not only in the red muscle (the rat soleus) but also in the white muscle (the rat epitochlearis). Moreover, in the latter both endurance and „sprint" efforts produced similar effects, whereas in the former only endurance exercise was effective. These findings provide an evidence that an increase in the muscle insulin sensitivity of all types of muscle fibers plays an important role in the exercise-induced improvement of glucose tolerance and that various types of exercise can be used in therapy and prevention of carbohydrate intolerance (The study was performed in cooperation with the Department of Biochemistry, Oxford University, U.K.).

It was demonstrated in rats that a diet enriched with fat containing a high percentage of saturated fatty acids (animal fat) impairs glucose tolerance and muscle sensitivity to insulin. A diet rich with fat containing polyunsaturated fatty acids (sunflower oil) induced an opposite effect. Physical exercise did not change the insulin action on skeletal muscles of rats fed the fat enriched diets.

It was proved that both the thyroid hormone deficit and excess impair glucose tolerance in the rat. In the experimental hypothyroidism-induced by surgical thyroidectomy + propylthiouracil treatment for 30 days — responsiveness of lactate production to insulin in the soleus muscle was abolished, whilst sensitivity of glycogen synthesis to insulin remained intact. In the experimental hyperthyroidism — produced by s.c. injections of triiodothyronine (T_3) for 3 days — responsiveness of glycogen synthesis to insulin was drastically diminished and sensitivity of lactate formation to this hormone was unchanged. Physical exercise increased only the glycogen synthesis response to insulin in hypothyroid rats and the sensitivity of lactate formation to this hormone in hyperthyroid animals. It was concluded that thyroid hormones interact with insulin in skeletal muscles, most likely at a post-receptor side. They stimulate glucose utilization in oxidative processes with a concomitant inhibition of glycogen formation.

II. Mechanisms controlling lipid metabolism

The study on metabolic effects of prolonged, controlled hyperadrenalinemia-induced by s.c. implantation of retard adrenaline (A)-releasing tablets

to rats was continued in cooperation with the Institute of Functional Pathology, University of Graz (Austria). Within the first day of hyperadrenalinemia a pronounced decrease in the activity of lipoprotein lipase (LPL) in skeletal and heart muscles was demonstrated. Activity of this enzyme returned to normal values after 48 h of hyperadrenalinemia. The results suggest that adrenaline contributes to the control of the plasma triacylglycerol (TG) uptake by muscles affecting the key enzyme (LPL) responsible for the plasma TG hydrolysis. Activity of this enzyme was also examined in rats with experimentally induced hypo- and hyperthyroidism. The results obtained so far indicate that thyroid hormones diminish LPL activity in the muscle tissue. An inverse correlation was ascertained between the muscle TG content and LPL activity.

III. Metabolic reactions to various types of physical exercise

In cooperation with the Human Performance Laboratory Ball State University, Muncie, Indiana (USA) an interrelationship between the muscle and blood lactate (LA) concentrations was investigated in men during graded exercise. A threshold increase was demonstrated in the muscle LA content preceding a rise in blood LA and hydrogen ion (H^+) concentrations-commonly known as the anaerobic threshold (AT). The data provide an evidence that AT — results from the nonlinearly increasing rate of LA production in working muscles.

Thermoregulatory, metabolic and hormonal responses to repeated bouts of prolonged exercise (50% $\dot{V}O_2max$, 30 min), separated by 30 min rest periods were studied in men.

In spite of the rest periods and water loss replacement core body temperature and majority of metabolic and hormonal changes showed an increase with time, similar to that occurring during continuous exercise. Significant correlations were found between body temperature, metabolic rate, blood glucose, lactate or FFA concentrations and changes in blood levels of hormones involved in the control of exercise metabolism such as: catecholamines, insulin, growth hormone, glucagon.

In cooperation with the Department of Physiology, University of Kuopio (Finland) body temperature and metabolic effects of active warming-up were studied in men exercising at low ambient temperature (5° C). During the exercise preceded by 10 min warming-up body temperature was higher and energy cost of work lower in comparison with identical exercise preceded by 30 min rest at low ambient temperature.

The effect of moderate dehydration on body temperature, and exercise metabolism was investigated in dogs. In dehydrated dogs the exercise-induced increases in rectal and particularly in muscle temperature were enhanced. It was associated with an increased concentration of the plasma FFA and cortisol levels. No significant changes were found in the contents of muscle metabolites (glycogen, lactate, adenine nucleotides and creatine phosphate).

In cooperation with the Institute of Gerontology, N.I.H. in Baltimore (USA), relationships between serum testosterone concentration at rest or after physical exercise and the aerobic capacity, age and body mass were analyzed in healthy men. It was proved that the aerobic capacity is a dominant factor determining the serum testosterone level at rest and the magnitude of this hormone increase during exercise.

IV. Mechanisms of thermoregulation

The studies were continued on sweating kinetics in human subjects in response to endogenous and exogenous heat load. They provided some new data concerning a relation between effectiveness of thermoregulation and sex, cardio-respiratory fitness, state of hydration and kind of clothing. The investigations emphasized an importance of the sweating kinetics in the initial period of heat loading for effectiveness of thermoregulation.

In cooperation with the State University of California in Riverside (USA) it was demonstrated that in dehydrated dogs a rapid restoration of thermoregulatory function after drinking water results from reflex activation of thermoregulatory mechanisms, initiated by receptors located in the mouth, esophagus, and stomach.

V. Factors modifying the dynamics of circulatory responses to physiological stimuli

The effect of aging on the cardiovascular response to posture changes was studied in healthy men from 20 to 65 years. There was no significant influence of aging on the dynamics of changes in stroke volume (measured by reography) but the magnitude of several indices characterizing the transient heart rate responses to standing up was decreasing with age of the subjects.

The validity of stroke volume measurements by reography was confirmed by comparing the data obtained with this method with those measured using the dopler echo-cardiography in the supine position. Continuing the studies on cardiovascular responses to static exercise it was demonstrated that a long-term intensive endurance training (competitive field hockey) results in a reduced increase in heart rate and enhanced stroke volume response to the static hand-grip.

Durnal variations in the cardiovascular and plasma catecholamine response to hand-grip were analyzed in healthy human subjects.

VI. Studies on the risk factors of the coronary heart disease (CHD)

Dynamics of changes in several CHD risk factors such as: impaired glucose tolerance, hypercholesterolemia, overweight, and cigarette smoking were analyzed in coronary patients 15-16 years after their myocardium infarction.

The data failed to show any significant relationship between the risk factor score and the incidence of severe complications of the disease.

An evidence was provided that a 2-week low intensity endurance training leads to an improvement of working ability, reduction of the serum triacylglycerol (TG) and total cholesterol concentrations with an elevation of the HDL-cholesterol level in middle age men with CHD risk factors.

In cooperation with the Institute of Gerontology, N.I.H. in Baltimore (USA) interrelations between physical activity, age, body mass, type of fat distribution and the CHD risk factors such as: hypertension, hyperlipidemia, and impaired glucose tolerance were investigated using the multivariate regression analysis. It was found that overweight is of primary importance for development of the above mentioned CHD risk factors.

VII. Dynamic studies of medullary interstitial electrolites

To provide a biophysical basis for estimation of electrolyte concentration in the renal medulla from measurements of tissue electrical admittance (reciprocal impedance), in vivo and in vitro recordings of both impedance (Z) and phase angle (φ) were performed. Using these data, the real impedance component, equivalent to resistance (R), the imaginary component, equivalent to capacitive reactance (X_c) as well as X_c/R ratio were calculated.

In vivo studies

In aneasthetized rats platinum/iridium needle electrodes were placed in the renal medulla and Z and φ measurements were taken using a Hewlett-Packard impedance meter and measuring frequency of 0.5 — 1 — 3.5 — 10 — 20 — 50 kHz. With increasing frequency (f), X_c showed a steep decrease while R fell progressively but slowly. Electrolyte concentration in the renal medullary interstitium was experimentally depressed using furosemide, hypertonic mannitol infusion, or slow gradual bleeding of the animals. An increase in medullary electrolytes was accomplished by an infusion of hypertonic NaCl solution. Both R and X_c changed in the direction opposite to tissue ion concentration.

Incerasing the measuring frequency above 3.5 kHz did not improve the resolution of the method for estimation of tissue electrolyte concentration from measurement of impedance. Nor was resistence (R) a more sensitive index of tissue ions compared to total impedance.

In vitro studies

For evaluation of the role of electrolyte concentration versus fractional interstitial volume as determinants of tissue impedance, studies employing a biological model of renal medullary tissue were performed.

An array of rat blood cell suspensions of increasing hematocrit (0 — 75%) was prepared. The cells were suspended in NaCl solutions of increasing concentration. In such a model electrolyte concentration and fractional volume of the interstitial fluid (100 — Ht) could be varied independently. It was shown

that Z, φ, R and X_c values were critically dependent on the measuring frequency. R changes depended both on the ion concentration and (100 — Ht) value. Alterations of X_c reflected mostly electrode polarisation which decreased rapidly with increasing frequency. The adequacy of probable equivalent circuits for description of the tissue-electrode complex and cell suspension--electrode complex was analysed.

As a whole, the data confirm that electrolyte concentration in the interstitium of the renal medulla can be continuously monitored as electrical impedance (or admittance).

See the List of Publications: a) 4, 21, 22, 27, 29, 30, 40, 45, 54, 55, 65, 66, 67, 80, 90; b) 2, 5, 10, 11, 12, 13, 22, 23, 34, 39, 46, 59, 63.

Cardiovascular Laboratory
Head: Prof. Krystyna Cedro-Ceremużyńska

1. Acute phase of myocardial infarction (MI) is known to evoke profound humoral and metabolic response. To investigate whether this response also influences peroxidation of lipids, blood level of malondialdehyde (MDA) was measured in patients in acute phase of MI (n=15) and in healthy controls (n=8). Values of MDA (μM) increased from 3.44±0.84 on the first day of MI to 4.20±1.12 on the 3rd day. In healthy subjects, MDA level amounted to 2.20±0.27. No apparent relation was found between the increased MDA and the course of MI as estimated by clinical, biochemical and echocardiographic criteria. Since activated leukocytes might contribute to enhancement of peroxidative processes reflected by increased MDA, the study was undertaken to investigate aggregability of leukocytes in patients in acute phase of MI and in healthy controls. This study is in progress.

2. The role of adrenergic stimulation in initiating phosphorylation of myosin light chains in both normal and hypertrophic hearts is controversial. Thus, the study was undertaken to follow an effect of adrenergic agonist (isoproterenol 10^{-6} M) and antagonist (propranolol 2×10^{-6} M) upon phosphorylation of protain 19000D, corresponding to myosin light chains. The experiments were carried out on the rat isolated myocytes from normal and hypertrophic (pressure overload) hearts. In hypertrophic hearts phosphorylation of myosin light chains was increased (by 27+7%, n=6) as compared to controls. This process was stimulated by isoproterenol in both normal and hypertrophic hearts (by 67±5%, n=6 and 65+10%, n=6, respectively) and completely inhibited by propranolol, indicating involvement of beta adrenergic receptors in phosphorylation of myosin light chain proteins in both normal and hypertrophic hearts, at least in the applied experimental model.

See the List of Publications: a) 25, 26, 43; b) 19.

Laboratory of Experimental Surgery
Head: Assoc. Professor Maciej Borkowski

EVALUATION OF THE STATE OF PERIPHERAL BLOOD CIRCULATION IN PATIENTS WITH VASCULAR DISEASES

The results of earlier investigations on the effects and effectiveness of PGE_1 (Prostin VR) infusion in the treatment of ischaemic diseases were supplemented by clinical observations.

Experimental investigations on rabbits showed that transcutaneous electric stimulation (TES) an adjunctive method in the ischaemic limb disorder treatment affects blood coagulation. The anticoagulative effect of TES can improve rheological blood parameters in the result of electrotherapy in thrombotic diseases.

Rheongiographic investigations of blood flow changes in the upper limbs of patients with Raynaud's disease or syndrome have led to the elaboration of a methodology yielding accurate and trustworthy results. Observations and suggestions were conveyed to the producer of the electroimpedance rheograph.

In the experiments performed on rabbit the possibility of incised venous vessel wall anastomosis with the application of a cw Nd:Yag laser beam of 10.6 μm wavelength conducted through an optical fibre was demonstrated. A full, non-occlusive anastomosis was achieved.

An analysis of a short- and long-term 8.5 years observations has led to the elaboration of optimal surgical method for the arteriovenous fistula formation and evaluation of their suitability in patients undergoing protracted dialysis treatment.

See the List of Publications: a) 2, 28; b) 3, 4, 9, 35.

STUDIES ON THE STRUCTURE AND BIOLOGICAL PROPERTIES OF THE NERVOUS TISSUE

Department of Neuropathology
Head: Assoc. Professor Irmina B. Zelman

DYNAMICS OF PROGRESSION OF ISCHEMIC ENCEPHALOPATHY FOLLOWING CLINICAL DEATH: EVALUATION OF BLOOD-BRAIN BARRIER CHANGES, IMMUNOLOGICAL RESPONSE AND EARLY BIOCHEMICAL ABNORMALITIES

A variety of techniques have been employed to evaluate the effects of 10- min global brain ischemia following clinical death, on the blood-brain barrier permeability to serum proteins. The studies revealed increased permeability to a number of proteins, through this barrier which was noted immediately after reanimation and often persisted for two weeks following the ischemic incident. The character and topography of the changes indicated the contribution of acute post-ischemic hyperperfusion and systemic venous stasis to the damage and pointed to its distinctly biphasic character. A number of serum proteins were found to accumulate in neuronal cytoplasm, being indicative of nerve cell membrane damage. Moreover, ischemia led to changes in the distribution of two astroglia — specific proteins: the glial fibrillary acidic protein (GFAP) was increased in the regions of extravasation, while S-100 protein was decreased in the areas of profound damage of nerve cells.

A short-term (5-min) global brain ischemia model was employed to evaluate early („premorphobiological") biochemical changes that could be of functional relevance and/or contribute to subsequent morphological damage. The studies revealed a rapid decrease of ouabain binding sites in the ischemia vulnerable CA_1 region of hippocampus (see the following section), but not in the frontal cortex. This result is interpreted to manifest region-selective impairment of the sodium-potassium pump. An other observation pointing to early disturbances of the transport function of nerve cell membranes was an equally rapid decrease of iodipine binding sites, reflecting the decrease in the number of voltage-depended Ca^{2+} channels. These early changes in cell membrane function may be casually related to atterations in the concentration of prostaglandins D_2 and F_{2a} and tromboxane B_2, as observed shortly after reanimation.

The intriguing question of selective vulnerability of pyramidal neurons of the hippocampal CA_1 region to the short-term cerebral ischemia, manifested by the so-called „delayed neuronal death" has been dealt with in ultrastructural studies. Changes in the ultrastructure of cerebral capillaries and glia have been pointed out as contributory (albeit not essential) pathogenetic fa-

ctors of ischemic damage. A noteworthy observation was that the post-ischemic hyperactivation of neurons is not accompanied by ultrastructural changes in synapses of CA_1 neurons, irrespective of their topographic location or neurotransmitter system involved. However, marked changes in inhibitory interneurons were noted. Hence, bioelectric hyperactivity of pyramidal neurons of CA_1 region may be, at least in part, related to their impaired inhibition.

Ultrastructural distribution of adelynate cyclase activity in the CA_1 hippocampal region was examined, with emphasis on the association of this enzyme with different synaptic membranes. The enzyme activity was detected both in postsynaptic membranes and in dendrites showing no synaptic junctions. The tests were performed on control brains and were aimed at adapting the procedure to investigate enzyme changes during ischemia.

PATHOMECHANISM OF CEREBRAL ANOXIA AS STUDIED IN TISSUE CULTURE

Investigations on the reaction of the hippocampus, grown in organotypic culture in vitro, to anoxia led to clear distinction between the early changes; characterized by damage of neuronal mitochondria and swelling of astrocytic cytoplasm and late, advanced changes including disintegration of neurons and fibrillary reaction of astroglia. A calcium channel blocker nimodipine exerted a dose-dependent cytoprotective effect with regard to the „late" changes only, which is consistent with earlier observations made on cerebral ischemia in vivo. In technical terms the result with nimodipine adds credente to the ultrastructural criteria employed for distinguishing between the „early" and „late" changes.

CEREBRAL BLOOD VESSELS IN NORM AND PATHOLOGY

Studies complementing earlier investigations on pial blood vessels supplying cerebral hemispheres, disclosed variations between different animal species with regard to vegetative innervation. Studies have been undertaken on morphological changes following neurogenic vasospasm accompanying subarachnoid hemorrhage. Organic character of the vascular changes was emphasized; their nature will form the subject of more detailed investigations.

TRANSPORT AND METABOLISM OF GLUTAMATE PRECURSORS IN THE ASTROGLIA AND OTHER METABOLIC COMPARTMENTS OF THE CENTRAL NERVOUS SYSTEM:
Effects of hepatic encephalopathy (HE)

The effects of a number of neurotransmitters (amino acids and catecholamines) on the release of newly taken up, radiolabelled glutamine from astroglia-enriched- and synaptic fractions was investigated. Of all the compounds tested, only glutamate stimulated glutamine release and the effect

was confined to astroglia, which is consistent with the „glutamate acceptor" and „glutamine donor" function of astrocytes in the CNS. The effect of glutamate was sodium-depended and it was not mimicked by either of the glutamate receptor agonists: N-methyl-D-aspartate, quisqualate or kainate, indicating a carrier-mediated mechanism. In relation to the in vivo conditions, the results indicate that glutamate, released from the nerve endings, may control back transport of its precursor from astrocytes to neurons.

Investigations were continued on the role of gamma-glutamyltranspeptidase (GGT) — an enzyme involved in the brain glutamine and glutamate transport, in the pathomechanism of hepatic encephalopathy (HE). Three questions have been addressed: 1) Is the stimulation of the enzyme activity by HE due to direct ammonia interaction with the tissue? 2) Are there any regional differences in the response of the enzyme? 3) Whether and how does the response change during ontogenesis. To this end, the effects of hyperammonemia of varying intensity and duration on the enzyme activity were measured in homogenates or capillaries of different brain regions of young (18-day) and adult (6-week) rats. The results pointed to the lack of simple dependence of the enzyme response on the duration or severity of hyperammonemia, indicating the possible involvement of other factors than ammonia in the enzyme activation during HE. The stimulation of GGT varied in different brain regions and metabolic compartments, and the effects in young and adult brain regions were frequently contrasting.

Studies on the effect of thioacetamide-induced HE and on the in vitro treatment with ammonia on the glutamate dehydrogenase activity in nonsynaptic cerebral mitochondria revealed the enzyme, to be resistant to either treatment. The next enzyme in the row involved in glutamate metabolism — α-ketoglutarate dehydrogenase — showed diversified responses to HE or ammonia, depending upon α-ketoglutarate concentration in the incubation mixture. The marked decrease of the enzyme activity by HE noted at physiological substrate concentration and the high vulnerability to ammonia of the high affinity form of the enzyme, both point to the involvement of α-ketoglutarate dehydrogenase in the pathomechanism of HE.

IMMUNOMORPHOLOGICAL CHARACTERIZATION OF BULK ISOLATED ASTROGLIAL FRACTIONS AND GLIAL CELLS IN DIFFERENT REGIONS OF THE CENTRAL NERVOUS SYSTEM

A fraction enriched in astrocytes, isolated with a method routinely used in this Laboratory, was demonstrated to react with antibodies against two astrocytic marker proteins: glial fibrillary acid protein (GFAP) and glutamine synthetase (GS). This result confirms the presence in the fraction of astrocytes with well preserved cell membranes and as such justifies the use of the fraction in studies on the metabolic properties of these cells. A preliminary attempt has been made to isolate, a fraction enriched in astrocytic pro-

cesses from the rat brain. As a starting material served „synaptoneurosomes" — a suspension containing intact association of pre- and postsynaptic parts of nerve endings. Hypoosmotic treatment of synaptoneurosomes and subsequent low speed centrifugation rendered a fraction enriched in glial cell surface antigens (M_1, Leu M_7). Electron microscopic analysis of this fraction revealed presence of „finger-like" structures resembling released fragments of astrocytic processes. Immunomorphological analysis of organotypic cultures of newborn rat cerebellum, grown in vitro for up to 5 weeks, was performed with the use of the avitin-biotin complex (ABC) method. GFAP- but also vimentin-positive cells were observed throughout, confirming the presence of vimentin not only in fibroblasts but in astrocytes as well. In consistence with earlier observations in situ, the neurofilaments (NF) antigen marking the nerve cell processes was only noted in young cultures (up to 14 DIV). Astrocytes localized around tumors showed an intense reaction to antibodies against the alpha-l-antichymotrypsin, thus confirming earlier observations made on experimental tumors.

NEUROTOXIC ACTION OF QUINOLINIC ACID (QUIN) IN VIVO AND IN VITRO

Intracerebral and intraventricular administration of QUIN into the rat was found to produce typical neuronal necroses only in dorsal hippocampus, but unspecific neuronal alterations in the ventral hippocampus, lateral septal nuclei and cerebral cortex, rarely in striatum. The distribution and intensity of the changes were related to the QUIN dose and the site of administration, but unspecific neuronal changes were more widespread following intraventricular QUIN application. Ultrastructural studies on the QUIN-induced neuronal necroses revealed severe degenerative changes of axonal endings and their synaptic vesicles, pointing to the so far neglected possibility that QUIN may acts presynaptically. Disturbances of calcium homeostasis have been suggested to contribute to the QUIN-induced damage. Intraventricular administration of QUIN elicited a marked decrease of the substance P in frontal cortex and striatum, but not in hippocampus and midbrain. However, it remained without any effect on the Mg^{2+}-dependent binding of a glutamate surtogate — D-(^{3}H)-aspartate.

In hippocampus, grown in organotypic culture in vitro, QUIN produced early postsynaptic damage mainly in pyramidal neurons. Apart from QUIN-specific dendritic changes severe destruction of the perikaryal organelles and nuclei, typical for irreversible cell injury were observed. The process of neuronal destruction was observed to progress further after withdrawal of QUIN from the nutrient medium, similarly as it was disclosed in in vivo conditions. The response of glia consisted of distinctly increased number of intermediate filaments and reactive changes of fibrous astrocytes. It was shown that zinc ($ZnCl_2$) which added alone to the hippocampal cultures me-

dium (in relatively small concentrations similar to those observed in vivo) induced ultrastructural changes mimicking those produced by excitatory amino acids, when applicated simultaneously with QUIN attenuated neurotoxic effect of the latter. It is postulated that zinc exerts a cytoprotective effect against QUIN neurotoxicity via interaction on the level of NMDA receptors.

NEUROTOXICITY OF SERA FROM ALS PATIENTS

Antibodies against nervous tissue were detected in sera derived from ALS (amyotrophic lateral sclerose) patients. These antibodies are likely to be responsible for the citotoxicity of ALS sera observed after intraventricular administration into rabbit brain. These observations point to the contribution of immunological mechanisms to the pathogenesis of ALS.

See the List of Publications: a) 1, 3, 14, 31, 33, 34, 35, 36, 37, 38, 39, 49, 50, 51, 52, 53, 58, 59, 60, 63, 72, 76, 77, 78, 79, 85, 88, 89; b) 1, 25, 26, 27, 28, 29, 30, 31, 32, 33, 41, 42, 43, 44, 45, 50, 51, 60, 61, 64, 70, 71, 77, 78.

Department of Neurochemistry
Head: Assoc. Professor Jerzy Łazarewicz

1. DISORDERS OF NEUROTRANSMISSION IN THE PATHOGENESIS OF ISCHEMIC BRAIN INJURY

The nature of calcium ionophores which are involved in ischemia-induced Ca^{2+} shift to brain neurons was studied by continuous microdialysis of the rabbit hippocampus in vivo. This method which allows measurement of changes in extracellular concentrations of calcium and amino acids and in the blood-brain barreir (BBB), the permeability was also used for local drug application. It was found that 2-amino-5-phosphonovalerate, which is a competitive antagonist of NMDA-sensitive glutamate receptors, strongly inhibits the ischemia--evoked calcium shift to the cellular compartment, without affecting the leakage of BBB. Nimodipine, the dihydropyridine blocker of voltage-sensitive calcium channels (VSCC), when applied locally had a negligible effect on ischemia-evoked calcium fluxes, whereas, when administered systemically, exerted its protective effects on calcium homeostasis and BBB through the vascular rather than parenchymal site of action. These results indicate that channels coupled to NMDA receptors are mainly responsible for the fatal ischemic shift of calcium to brain neurons, whereas VSCC which are susceptible to dihydropyridine inhibition seem to be less important in that process.

Investigations on the role of free radical processes in the functional impairment of synaptic endings demonstrated the possible involvement of pe-

roxidation in disturbances of neurotransmission which develop in various pathological conditions including brain ischemia. It appeared that accumulation of peroxidation products in synaptosomes isolated after ischemia followed by recirculation in normoxic conditions, is maximal during 20-min resuscitation but still remains elevated 24 h after ischemia. Even brief exposure of recovering animals to hyperoxia significantly potentiated postischemic peroxidation. Exposure of the control rats to prolonged hyperoxia also an increase in the amount of peroxidation products in synaptosomes, accompanied by pronounced morphological changes. Prolonged hyperoxia, which stimulated dopamine uptake reduced the uptake of histidine in rat brain synaptosomes and reversibly disturbed the content of histamine and the activity of enzymes involved in the synthesis and degradation of histamine.

2. THE SIGNIFICANCE OF BIOACTIVE LIPID AND PROTEIN IN SIGNAL TRANSDUCTION (Assoc. Professor Joanna Strosznajder)

Regulation of the second messenger molecules liberation upon activation of receptor(s) connected with inositol phospholipid degradation and the interaction of this signal transduction system with adenyl-cyclase activity was studied. Moreover, the significance of membrane lipid in agonist-receptor binding and the activity of calcium dependent neutral proteases in brain was investigated.

It was found that carbachol (stabile analog of acetylcholine) in the presence of Ca^{2+} ions activates liberation of arachidonic acid through the action of phospholipase A_2. GTP binding protein regulate receptor mediated phospholipase A_2 activity, exclusively in the brain plasma membrane. Cholesterol, an important lipid component in the plasma membrane of eukaryotes modulate the characteristics of some receptor(s) system. It was found that the growth factor-like action of cholesterol may be explained by its modulation of enzyme activities involved in the synthesis and breakdown of polyphosphoinositides which is followed by a cascade of reactions ultimately leading to the initiation of DNA synthesis and cell proliferation. These studies indicate also that the other membrane lipid component dolichol modulates $GABA_A$ neurotransmission system. Higher level of dolichol in synaptosomes decreased GABA uptake and the affinity of agonist into $GABA_A$ receptor.

The interaction between signal transduction system, which transmits its signals through the breakdown of inositol phospholipids, and the other system coupled with adenyl cyclase activity was observed upon activation of alpha adreno; H_1 histominergic receptors and also in the case of phorbol ester action. The activation of protein kinase C may be responsible for the stimulatory response of adenylate cyclase system in this condition and also during phobol ester action. Signal transduction processes can be modified by calcium dependent neutral proteases (CANP)s. The present studies demonstrated subcellular distribution of Calpain I and II activity (enzymes stimulated

by μM and mM Ca^{2+} concentration respectively). Brain hypoxia changed the intracellular distribution of CANPs (3 fold rise in synaptosomes) without affecting the total calcium dependent proteolytic activity. Moreover, hypoxic insult enhance susceptibility of membrane protein on proteases action.

See the List of Publications: a) 8, 9, 10, 46, 47, 48, 60, 69, 73, 82, 83, 86; b) 6, 8, 17, 20, 50, 51, 60, 61, 62, 65, 66, 67, 68, 72, 73, 75, 76.

Department of Neurosurgery
Head: Prof. Eugeniusz Mempel

NEUROBIOLOGICAL ADAPTATIVE MECHANISMS

1. Correlation between the volume-pressure relations and the CT images in the brain injury and tumor patients (Assoc. Prof. Zbigniew Czernicki)

The numerical analysis of CT images was applied for the evaluation of intracranial reserve. Fiveteen patients with severe head injury and 15 patients with cerebral glioma were examined. The method was clinically verified and was found very usefull, especially in the studies leading to the evolution of the pathological changes in the same patient.

The studies on the infusion test optimisation were continued. Two different methods were clinically tested:
1) the sinusoidal infusion test;
2) the computer controlled closed-loop infusion test.
Both the methods were compared with the constant rate infusion test and both were found to be less invasive. The closed-loop infusion test has the widest clinical applicability.

2. Evaluation of an experimental model of Parkinson disease in cats (Prof. Eugeniusz Mempel)

The model of Parkinson disease for electrophysiological investigations has not been documented yet in cats.

Two series of investigations were performed. In the first series microinjections of 6-hydroxydopamine (6-OHDA) were applied in three experimental versions: a) both sided damages of pars compacta substantia nigra, b) both sided damages of substantia nigra and globus pallidus, c) both sided damages of substantia nigra and partially caput nuclei candati.

In the second series of investigations intracarotid infusions of l-methyl--4-phenyl-1,2,3,6 tetrahydropyridine (MPTP) were applied to establish the hemiparkinsonism model.

The abtained results confirmed behaviorally and electrophysiologically (emg) that both models of parkinsonism and hemiparkisonism in cats are suitable for a short term electrophysiological investigations. The changes persisted for 3-5 weeks in the first kind of investigations and 6 weeks in the second.

Further studies concerning a dose of neurotoxin, a lesion range in the dopaminergic structures, as well as enzymatic determinations and especially measurements of monoaminooxydaze A and B content in the cerebral tissue are necessary to establish a chronic model of parkinsonism in cats.

3. Effect of different damages in the left and right brain hemispheres on the fluency of words (Assoc. Prof. Jadwiga Szumska)

During clinical studies the retrieval abilities of words stored in the long--term memory were examined. There were three categories of words: names of animals, words beginning with the letter „k" and „sharp" objects. The number of correct words in each category provided in one minute was calculated.

Two groups of patients (20 subjects after amygdalotomy and/or hyppocampotomy and 20 subjects after thalatomy) were examined two-three weeks after the surgery and the results were compared with those obtained in the preoperation period. It is suggested that the normal word fluency requires an undisturbed activity of structures in medial left temporal lobe and left and right thalamus.

See the List of Publications: a) 74.

Laboratory of Developmental Neuropathology
Head: Prof. Maria Dąmbska

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

The influence of vincristine on the maturing nervous system was investigated. This antimitotic drug was given intraperitoneally to rats and rabbits in their early neonatal period. The development of behavioral response to haloperidol was disturbed in rats after treatment with vincristine. Ultrastructural dose-depending changes in the nerve and glial cells and axons were observed in those animals. In young rabbits after vincristine administration characteristic bundles of neurofilaments were observed. The changes were of chronic type and persisted during four months. After vincristine administration the blood-brain barrier (BBB) was not penetrable for horse-radish

peroxydase but the perivascular lesions indicated that this drug penetrated the BBB in small amounts.

The morphometric investigations were performed on Ammon's horn of small infants who died without brain lesions and with pre-and perinatal central nervous system damage. The area CA_1 and CA_2 were damaged in cases of pathological pregnancy in anamnesis, whereas after pathologic delivery only CA_1 demonstrated neuronal lesions.

See the List of Publications: a) 5, 6, 7, 84.

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

HISTOCHEMICAL AND IMMUNOCYTOCHEMICAL EVALUATION OF THE HYPOTHALAMO-HYPOPHYSEAL NEUROSECRETORY SYSTEM OF THE RAT FOLLOWING CEREBRAL ISCHEMIA

Electron-microscopic investigations and some cytochemical and immunocytochemical studies of hypothalamic secretory nuclei — n.supraopticus, n. paraventricularis — as well as the hypophyseal neural lobe were performed in rats subjected to complete cerebral ischemia. Ischemia was evoked according to the method of Korpaczev et al. and lasted 5 min. Electron-microscopic obesrvations using a conventional method carried out two and four weeks after evoking cerebral ischemia revealed an increased number of polymorphic lysosomes, lipid droplets as well as filamentous and tubular elements of the cytoskeleton. Synaptic endings exhibited a normal structure. An increased number of lysosomes was observed in the pituicytes of the hypophyseal neural lobe, while an abundance of morphotic elements of cytoskeleton and microvesicles was present in nerve terminals. Studies applying the reaction with alcian blue and tanic acid allowed to follow various forms of lysosomes in the pericaryons, processes and synaptic endings of the neurons. Considerable attention was paid to the cytoskeleton, the structure of which differed from that of the control material. An increased number of neurotubules was accompanied by accumulation of neurofilaments both in pericaryons as well as in the processes of secretory neurons. These findings may indicate that pathological changes persist two weeks after reanimation and even have a tendency to increase in scope. They are probably irreversible.

Using the oxalate-pyroantimonate technique ultrastructural localization of calcium (Ca^{2+}) was determined in the nucleus supraopticus, the nucleus paraventricularis and the hypophyseal neural lobe after complete cerebral ischemia. An increased calcium accumulation was observed in mitochondria

of neurons two weeks after inducing of ischemia. Calcium precipitate was also found in vesicles, in cytoplasm of neurons and their swollen dendritic processes. Abundant calcium precipitate occurred in synaptic vesiclles and within synaptic cleft. It was present between disjuncted lamellae of the myelin sheath in profiles of myelinated axons located in neuropil. Diffuse precipitate was visible in the cytoplasm of pituicytes, as well as in microvesicles of the neurohypophysis. An evaluation of relationships between calcium accumulation and neuronal damage and identification of reversible as well as irreversible stages of these processes may help to understand the pathophysiology of ischemia.

Using immunocytochemical technique ultrastructural localization of vasopressin was studied in the axons of the rat neurohypophysis 48 hours after 15 min-lasting clinical death. The observations revealed an abundance of vasopressin-positive axons in the rat neurohypophysis. These results may indicate the physiologically normal level of vasopressin or may be indicative of an inhibition of the hormone release under these experimental conditions.

See the List of Publications: a) 15, 16, 81, 87.

STUDIES ON TRANSPLANTATION AND EXPERIMENTAL SURGERY

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

I. STUDIES ON THE MECHANISM OF IMMUNE PROTEIN CAPILLARY TRANSPORT INTO TISSUE SPACE AND LYMPHATICS

The capillary transport of molecules regulating local immune response was studied in venous hypertension, hyperthermia and in combination of both, as well as after sympathetic denervation. Water immersion hyperthermia combined with acute venous hypertension enhanced capillary transport of immune proteins and leukocytes to higher degree than hyperthermia or venous hypertension separately. Sympathetic denervation of dog hind-limb vessels had no early effect on capillary permeability for proteins, however, a decreased extravasation of blood borne cells was observed. These findings suggest that it is possible to manipulate the capillary transport without evoking inflammatory reaction, thus, control local immunity by regulating tissue influx of immunoglobulins and leukocytes.

In another study, the cellular composition of the efferent lymph was studied in patients with postinflammatory lymph stasis. An increase of B lymphocytes and a decrease of CDI+ Langerhans cells and IL-2R+ cells was observed. In contrast, accumulation of CDI+ Langerhans cells in epidermis was observed. In contrast, accumulation of CD1+ Langerhans cells in epidermis was observed. Studies on humoral factors in the lymph revealed presence of IL-I inhibitor in afferent lymph. No such inhibitor activity was so far been found in the plasma.

II. STUDIES ON THE INFLUENCE OF DONOR SPECIFIC TRANSFUSIONS ON THE ALLOGENEIC GRAFT SURVIVAL

The effect of allogeneic donor specific blood transfusion (DST) on the heart graft survival in BN to LEW rat strain combination was studied. Single DST one week and double DST one and two weeks before transplantation brought about statistically significant prolongation of cardiac graft survival (MST 14.2 ±2.0 and 17.0 ±2.8 days, respectively). Treatment of recipients with low doses of CsA (2.5 and 5 mg/kg b.w.) resulted in the prolonged graft survival (MST 16.5 ±2.3 and 22.2 ±5.8 days, respectively). Synergistic effect of CsA in a dose of 5 mg/kg b.w. and a single DST was observed with the MST 32.2 ±6.9. However, CsA did not act synergistically with two DST (MST 15.6 ±7.7). In addition, the effect of sera from DST recipients on MLC in vitro was studied. It was found that those sera (WIS), after absorption

with donor erythrocytes and splenocytes, were able to suppress the reactivity of WIS splenocytes in a mixed lymphocyte culture with AUG or LEW responding cells.

III. ALLOGENEIC SKIN TRANSPLANTATION FOR CLINICAL PURPOSES

The antigen presenting capacity of the dog Langerhans cells was studied. The rabbit anti-dog immune serum against Langerhans cells, isolated from afferent lymph, was raised. The obtained sera displayed high cytotoxic and agglutinating titer against dentritic cells in vitro and were specific for T6 and class II antigens, as was shown by blocking with monoclonal antibodies. The sera reduced PHA triggered proliferation of lymph cells by decreasing the stimulatory capacity of dendritic cells. An important question arising from the obtained results is whether the anti-Langerhans cell immune sera may modulate the rejection process of skin allograft.

Further studies on characterization of the liver sinusoids cytotoxic cells were carried out. These cells were found to display high cytotoxicity against NK-sensitive (K-562, YAC-1) and NK-resistant (L-5178y, P-815) tumor cell lines. This remained in contrast with peripheral blood cytotoxic cells which were active only against NK-sensitive lines. Liver sinusoids mononuclear cells were found enriched in 0X8 positive cells (40%) and cells rich in azurophilic granules. Cells isolated from the liver sinusoids did not respond to mitogens in culture and possesed a suppressive activity for autologous peripheral blood cells culture. In order to study the origin of those cells, the syngeneic liver graft was performed from female to male LEW rats and the frequency of Y chromosome was estimated on the washed-out lymphocytes. It was established that the liver natural cytotoxic cells are not of liver origin but sequester from the blood stream.

Human peritoneal exudate cells were characterized using morphological, cytochemical, and phenotypic (monoclonal antibodies) criteria. Cells were obtained intraoperatively from patients undergoing cholecystectomy. Peritoneal cell population contained 45% monocytes/macrophages, 9% granulocytes, 2% free mesothelial cells, 2% of B lymphocytes and 42% of T lymphocytes. It was not possible to visualize IL-2R or transferrin receptors. It suggests lack of activated cells in the peritoneal cell population.

IV. CELLULAR MECHANISMS OF REJECTION AND ACCEPTANCE OF ALLOGRAFTS

It has been shown, that allogeneic lymphocytes, when transplanted intravenously from AUG to WIS rat strain, are eliminated within 6 hours whereas syngeneic lymphocytes do not undergo rejection. In contrast, the elimination of nonlymphoid cells such as epithelial cells, fibroblasts, or chondrocytes is not dependent on their allogeneicity. In the present study the kine-

tics of elimination of allogeneic lymphocytes in lymphoid or nonlymphoid compartments were investigated. Allo- and syngeneic lymphocytes were injected intravenously and intramuscularly. Elimination of allogeneic lymphocytes after i.v. injection was faster than that of syngeneic lymphocytes, whereas elimination of lymphocytes injected i.m. was independent of their allo- and syngeneity. Elimination of allogeneic lymphocytes in recipients with induced immunological enhancement was also studied. Recipient rats, prior to lymphocyte injection, were pretreated with alloserum and donor cellular antigen, on day — 11 and — 10, respectively. On day 0, enhanced rats received either lymphocytes or heart transplant. Heart transplants survived more than 40 days, whereas lymphocytes were eliminated even faster than in the nonpretreated.

In another study the type of cells which are responsible for induction of immune enhancement was investigated. The most effective were cells rich in class II antigens. In the rat heart class II antigens are expressed by interstitial dendritic cells (IDC). An attempt was also made to modulate these antigens with Poly I:C (interferon alpha and beta stimulator). It was shown that pretreatment with Poly I:C, in a dose of I mg/kg b.w., resulted in an increased number of class II positive IDC's but only of the RT1.B subregion, while the dose of 10 mg/kg b.w. down modulated the number of IDC's of both subregions. Pretreatment of graft donors with 10 mg of Poly I:C did not influence the graft survival, whereas, pretreatment of recipients with 1 mg of Poly I:C resulted in some animals in acceleration, and in others in attenuation of graft rejection.

V. IMMUNE STATUS OF THE IMMUNOSUPPRESSED HOST

An increase in interleukin I production by monocytes and a decrease in IL-2 production by lymphocytes of patients after moderate operative trauma (cholecystectomy) was described. It was found that lymphocytes are able to express IL-2R, therefore, lack of IL-2, a lymphokine triggering cell proliferation and clonal expansion of T lymphocytes, may be responsible for the decreased immune responsiveness of the traumatized patients. A regulating effect of exogenous IL-2 on the immune response in patients after cholecystectomy was found. Exogenous IL-2 caused an increase of PHA triggered proliferation of lymphocytes isolated from patients after surgery. Besides restoration of AMLR response to the preoperative values was observed. The results may encourage an application of IL-2 therapy in patients severely depressed immune responsiveness before surgery.

Pilot studies on immune responsivenes of parenterally nourished patients were performed. Patients recovering from malnutrition showed increased responsivenes to mitogens, however, the recovery did not have any positive influence on the cell subpopulations or activity of suppressor or natural cy-

totoxic cells. The operative trauma superimposed on malnutrition caused a secondary decrease in proliferative responses to mitogens.

See the List of Publications: a) 17, 18, 56, 57, 64; b) 14, 15, 16, 36, 37, 40, 47, 48, 49, 52, 53.

Neuromuscular Unit
Head: Prof. Irena Hausmanowa-Petrusewicz

MECHANISMS LEADING TO HEREDITARY AND ACQUIRED NEUROMUSCULAR DISEASES

This year's investigations concentrated on the hereditary and acquired diseases of the motorneuron, miastenia in adults and children as well as on the congenital myopathies and dystrophies. Experiments on both the muscle and nerve regeneration processes were also carried out.

Changes occuring in myopathies with mosaic disturbances were described and their primary muscle origin has been documented.

In miastenia an apparent selectivity of the neuro-muscular block in the ocular form of this disease was demonstrated. Moreover, it was found that it is generalized, including symptom free muscles. A program for miastenia treatment in adults and children has been elaborated, in which application of immunosupressive drugs is considered the most important.

The results of research done in 400 cases of the spinal muscle atrophy of childhood were summarized. The slowing of sensory nerve conduction and increased excitability of motorneurons was stated.

In the studies on the post-polio syndrome some features of the motor unit instability were revealed many years after the acute phase of this disease. The selectivity of motorneuron involvement and the mechanism of central motor control in spinal atrophy was explained.

The congenital myopathies with types disproportion was confirmed. An analysis of the Ring fibers allowed to classify them into two types: A — occuring in miotonic distrophy, B — in structural myopathies.

The study on experimental degeneration and regeneration of the rat muscles revealed instability of basal membrane structure, of muscle cell appearing in different phases after injury.

See the List of Publications: a) 11, 12, 13, 23, 24, 44, 68; b) 7, 18, 24, 55.

OTHER RESEARCH WORKS

Mental Health Department
Head: Dr Zygfryd Juczyński

1. EVALUATION OF SOME BIOLOGICAL DETERMINANTS OF CHILDREN AND YOUTH WITH LOWERED ADAPTATION CAPABILITY

Over 2 thousand children and youths of the age ranging from 6 to 20 years living in a large city were included into this research. Biological inheritance was found in almost 40% of this population. In younger age groups the biological inheritance was connected with the pregnancy period and the delivery (approx. 26%), in the older groups — with head trauma. Abnormal EEG patterns were found in almost 60% of children showing disfunctional behavior. In the student group over 10% had focal neurological changes.

2. A RELATIONSHIP BETWEEN THE OWN IMAGE OF ILLNESS, DEFENCE MECHANISMS AND LIFE ACTIVITY OF THE PATIENTS WITH THE CANCER DIAGNOSIS

It was found that majority of patients treat their illness as a „challenge" (59%), whereas the rest as a „threat" (24%), or a feeling of harm and loss (17%). The „challenge approach" is combined with the „denial" mechanism and contrary to the other forms of defence it mobilizes the patient to undertake the recovery action and to be more psychically active.

3. ATTITUDE OF PARENTS AND FOSTER-PARENTS TOWARDS DRUG ABUSE BY THE SURVEYED PATIENTS

The catamnestic research included 80 people undergoing treatment. Almost 2/3 of them managed to give up their habits. Duration of drug taking proved to be the best single discriminating factor. When patients gave up their habits it lead to various health and social after-affects. The decisive factors were, however, the social support and the patient's own experience connected with drug taking.

See the List of Publications: b) 56, 57, 58, 74.

The Library
Head: Krystyna Marczakowska

The Library has been organized on July 1st 1967 by an integration of many small medical libraries belonging to the Polish Academy of Sciences. Now, it constitutes one department of the Medical Research Centre and acts as an information source for scientists.

Library structure: main library with affiliated special library in Łódź.

Scope and the subject profile: physiology, neurosciences, and experimental surgery including transplantology.

Present holdings:

books — monographic and serial volumes (Polish and foreign) — 17368, periodicals, newspapers (number of titles) — 469

unpublished documents (dissertations, research reports — SYNABA, in hard microfiches — 4359

Reference aids:

catalogues — alphabetical: books, periodicals and microfiches,
— subject: books,

main card-files — bibliographical list of papers published by scientists of the Medical Research Centre from 1967.

Number of inquiries and services per year:

circulation of documents (original or copies):
reading room and library loan — 9161
interlibrary loan — 1015

direct reference services (in person, by telephone) — 644, circulated news, current books and periodicals for the Departments users — 4205

reprographic services: xerocopies — 1900

systems of the user-oriented information services:
SDI — manual 11 topics
current and retrospective dissemination information — 28
MEDLINE — 14
SYNABA — 48
scientists citation reports — 950 (up to 1984)

Users:

scientific workers of the Medical Research Centre,

interlibrary loans available for all scientific institutes in Poland and abroad.

Bibliography of library: list of new books and current periodicals prepared weekly.

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b) Communications

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4. Borkowski M., Kruk M., Królicki L., Wojtal E., Malmurowicz L., Górewicz R., Laszuk D.: Evaluation of PGE_1 therapic effect in disease of peripheral artery. Abstr. of The Congress on Thoracic Cardiac and Vascular Surgery Polish Surgical Society, Helsinki 1988, 308.
5. Budohoski L., Langfort J.: Exercise-induced changes in muscle insulin sensitivity: the effect of β-blockade. Abstr. of VII International Symposium on Biochemistry of Exercise, Cond. J. Sports Sci. 1988, 13, 7.
6. Domańska-Janik K., Zalewska T.: Calcium, phospholipid-activated protein kinase (PKC) and calcium activated neutral proteases (CANP-s) in gerbils ischemic brain. 7th General Meeting of the European Society for Neurochemistry, Göteborg 12-17 June 1988, Neurochemistry Inter. 1988, 13, 106, F78.
7. Emeryk B., Rowińska K.: Post-polio syndrome. Abstr. of I Europ. Congress Neurol. 18-22 April 1988, Prague.
8. Gordon-Majszak W., Łazarewicz J.: Lipid peroxidation and the ischemic pathology of gerbil brain. International Symposium on Hypoxia, September 12-14 1988, Berlin GDR, Abstracts, 26.
9. Górewicz R., Bendowski P.: Arteriovenous fistulas for dialysis with subcutaneously dislocated basilic vein. Abstr. of The Congress on Thoracic Cardiac and Vascular Surgery, Polish Surgical Society, Helsinki 1988, 321.
10. Grucza R.: Kinetics of sweating and its role in overall thermoregulatory response to heat stress. Abstr. of Inter. Symp. on Work in a Hot Environment and Heat-Related Disorders, Khartoum, January 27-31 1988, 49.
11. Grucza R., Hänninen O.: Importance of dynamics of sweating in men during exercise. Abstr. of International Symp. on Work in a Hot Environment and Heat-Related Disorders, Khartoum, January 27-31 1988, 50.

12. Grucza R., Miyamoto Y., Nakazono Y.: The relationship between the maximum aerobic capacity and the rapidity of ventilation and cardiac output in response to a step exercise in healthy men. Abstr. of 65th Annual Meeting of the Physiological Society of Japan, Wakayma, April 1-3 1988, 3.

13. Grucza R., Smorawiński J.: Physiological changes during endurance training. Abstr. of XXI „Interkosmos" Conference, Baranów Sandomierski, June 6-10, 1988.

14. Grzelak I., Olszewski W., Rowiński W.: ILI and IL2 production following moderate trauma. Abstr. of VII Quadrilaterales Symp. of Surg. Res., Rostock 25-27 April 1988.

15. Grzelak I., Olszewski W., Rowiński W.: IL-1 and IL-2 production following surgical trauma. Abstr. of IX Eur. Immunol. Meeting, Roma, 14-17 Sept. 1988, 331.

16. Grzelak I., Olszewski W., Rowiński W.: The effect of moderate operative trauma on the lymphocyte reactivity and interleukins production. Abstr. of I Int. Congress on Immune Consequences of Trauma, Munich, 3-5 March 1988.

17. Haeffner E.W., Strosznajder J.B.: Ascites tumor cell proliferation, cholesterol-induced activation of inositolphospholipid-mediated signal transfer system. Abstr. of 14th International Congress of Biochemistry, July 10-15, 1988, Prague.

18. Hausmanowa-Petrusewicz I.: Remodeling of desintegrated motor unit. Abstr. of Neuromuscular Symposium, 17-20 Oct. 1988, Erfurt GDR.

19. Herbaczyńska-Cedro K., Gordon-Majszak W.: Nisoldipine prevents enhancement of lipid peroxidation induced by coronary occlusion in pig heart. International Soc. Heart Research, Oxford 1988. J. Mol. Cell. Cardiol. 1988, 20, 34, Suppl. 5.

20. Jankowski W., Strosznajder J.: Uptake and metabolism of intracisternally injected dolichol by selected parts of rat brain. Abstracts of 14th International Congress of Biochemistry, July 10-15, 1988, Prague.

21. Jernajczyk U., Kukwa A.: Respiratory activity of the hypoglossal nerve. Abstr. of 9th Europ. Congr. of Sleep Res., Izrael 1988.

22. Kaciuba-Uściłko H., Dubaniewicz A., Nazar K., Budohoski L.: Glucose tolerance and skeletal muscle insulin sensitivity in thyroid hormone deficient rats. Abstr. of VII International Symposium on Biochemistry of Exercise. Canad. J. Sports Sci. 1988, 13, 19.

23. Kaciuba-Uściłko H., Kruk B.: Factors modifying development of exercise hyperthermia. Abstr. of Inter. Symp. on Work in a Hot Environment and Heat-Related Disorders, Khartoum, January 27-31, 1988, 63.

24. Kamińska A., Fidziańska A.: Basement membrane component changes du-

ring experimental skeletal muscle degeneration and regeneration. Europ. J. of Clin. Invest. 1988, 18, No 2, A36.

25. Kapuściński A.: Blood-brain barrier in the experimental model of clinical death in rats. Abstr. of Annual Conference with the Association of Neuropathologists of GDR and Association of Polish Neuropathologists on „Neuropathology of the blood-brain-barrier and transmission", Görlitz GDR, May 11-14, 1988.

26. Kapuściński A., Hilgier W.: Eicosanoids in brain after resuscitation from clinical death. Experimental study. Abstr. of European Nuclear Medicine Congress, Mediolan, August 29 — September 2, 1988.

27. Kapuściński A., Hilgier W.: Prostaglandins D_2, $F_{2\alpha}$ and thromboxane B_2 in rat brain and plasma after resuscitation from clinical death. Third European Meeting of Neuropathology, Verona, September 27-29, 1988. Abstr. Clinical Neuropathology 1988, 7, 176.

28. Kida E., Matyja E.: Ultrastructural alterations induced by quinolinic acid in organotypic culture of rat hippocampus. Third European Meeting of Neuropathology, Verona, September 27-29, 1988. Abstr. in Clinical Neuropathology 1988, 7, 176.

29. Kida E., Matyja E., Krajewski S.: Morphological and immunohistochemical study of the glial cells response to anoxia and excitotoxin in vitro. Abstr. of 21 Danube Symposium for Neurological Sciences, Varna, October 13-15, 1988, 42.

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Symposium on Thermoregulation, Oulu, Finland, January 15-16 1988 (in Finish).

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36. Kubicka U., Maldyk J., Wierzbicki Z., Olszewski W.: Phenotypic characterization of human peritoneal cells. Abstr. of III International Symposium on the Biology, Immunology and Surgery, Heidelberg, 5-7 May 1988.
37. Kubicka U., Wierzbicki Z., Olszewski W.: Normal human peritonal lymphocytes — phenotypic and functional characteristics. Abstr. of IX European Immunological Meeting, Roma, 14-17 September 1988, 350.
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Poland and IX Socialist Countries Symp. of Child Psychiatry, Kraków, September 1988, 165 (in Polish).

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63. Smorawiński J., Grucza R.: Influence of endurance training on sweting and body temperatures in exercising men. Abstr. of International Symposium on Work in a Hot Environment and Heat-Related Disorders, Khartoum, January 27-31 1988, 101.

64. Stastny F., Hilgier W., Albrecht J., Lisy V.: Gamma-glutamyl transpeptidase in cellular elements isolated from cerebral cortex of rats with hepatic encephalopathy. Abstr. of XIV International Congress of Biochemistry, Praha, July 10-15, 1988.

65. Strosznajder J.: Activation of calcium dependent phospholipase A_2 acting against phosphatidylinositol in brain during ischemia. 7th General Meeting of the European Society for Neurochemistry, Göteborg, 12-17 June 1988; Neurochem Intern. 1988, 175 F287.

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68. Strosznajder J., Samochocki M.: Dolichol alters high-affinity binding of ^3H muscimol to synaptic plasma membranes of rat brain. 7th General Meeting of the European Society for Neurochemistry, Göteborg, 12-17 June 1988; Neurochem. Intern. 1988, 175, F288.

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70. Szumańska G., Mossakowski M.J.: Changes in the activity of alkaline phosphatase and adenylate cyclase in the brain vascular network in experimental postresuscitation syndrome. Abstr. of 21 Danube Symposium for Neurological Sciences, Varna, 13-15 October 1988, 44.

71. Victorov I.V., Khaspekov L.C., Kida E., Mossakowski M.J.: Neurodegenerative action of quinolinic acid on hippocampal neurons in situ and in dissociated cell culture. Third European Meeting of Neuropathology, Verona, September 27-29, 1988. Abstract in Clinical Neuropathology 1988, 7, 219.

72. Waśkiewicz J., Rafałowska U., Wałajtys-Rode E.: In vivo effect of the synthetic glucocorticoid (triamcinol acetonide) on histamine metabolism in rat brain synaptosomes. XXIV Cong. Pol. Biochem. Soc., Poznań, September 12-21 1988, Abstracts, 413 (in Polish).

73. Waśkiewicz J., Wałajtys-Rode E., Rafałowska U.: Hypoxia, ischemia and hyperoxia modifies histamine metabolism and transport in brain synaptosomes. International Symposium on Hypoxia, Berlin GDR, September 12-14 1988, Abstracts, 103.

74. Zakrzewski P., Szafrańska M.: Health problems of male alcohol addicts. Problemy Alkoholizmu 1988, 10, 7-8, 23 (in Polish).

75. Zalewska Z., Kasai Y., Kawashima S.: Calcium dependent proteolitic activity in hypoxic rat brain. Abstr. of International Symposium of Hypoxia, Berlin GDR, September 12-14 1988, 108.

76. Zalewska T., Kasai Y., Kawashima S.: The effect of hypoxia on calcium activated neutral proteases in the rat brain. 7th General Meeting of the European Society for Neurochemistry, Göteborg, 12-17 June 1988; Neurochemistry Intern. 1988, 13, 194, F345.

77. Zelman I., Staniaszek A.: Neuropathological findings following cardiac arrest in humans. Abstr. of 21 Danube Symposium for Neurological Sciences, Varna, 13-15 October 1988, 45.

78. Zelman I., Taraszewska A.: Myelin and axonal abnormalities in the hypomyelinated pt rabbit mutant. Third European Meeting of Neuropathology, Verona, September 27-29, 1988. Abstract in Clinical Neuropathology 1988, 7, 222.

SCIENTIFIC MEETINGS ORGANIZED BY THE MEDICAL RESEARCH CENTRE

Course of the techniques of organ transplantation in the rat — February 1988 — sponsored by ETHICON, Warszawa

XXI Conference of Cosmic Biology and Medicine — June 6-11th 1988, Baranów Sandomierski, Poland

Course of the basic microsurgical techniques — June 1988 — sponsored by DAVIES &GECK, Warszawa

Course of the basic microsurgical techniques — November 1988 — sponsored by DAVIES & GECK, Warszawa

Immunological Problems in Surgery and Hematology — November 3rd — sponsored by Rhone Poulenc, Warszawa.

VISITING SCIENTISTS

Department of Neurophysiology

Glebowski W.D.	Dept. of Physiol Pediatric Institute Leningrad, USSR
Jurco M., Petrasowa D.	Safaric University, Košice, Czechoslovakia

Department of Applied Physiology

Gnüchtel U.	Institute of Cardiovascular Research Academy of Sciences, Berlin, GDR
Kapitaniak B.	Dept. of Work Physiology CNRS, Paris, France
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Pyornil A.	Dept. of Zoology, University of Oulu, Finland

Department of Neuropathology

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Hokkanen E.	The National Board of Health, Helsinki, Finland
Kuridze N.	Inst. of Physiology, Georgian Academy of Sciences, Tbilisi, USSR
Stastny F.	Inst. of Physiology, Academy of Sciences, Prague, Czechoslovakia

Department of Neurochemistry

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Department of Neurosurgery

Himirnicki B., Szczerbakowa H.	Budenko Inst. of Neurosurgery, Moscow, USSR
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Department of Surgical Research and Transplantation

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Neuromuscular Unit

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Hokkanen E.	Dept. of Neurology, Helsinki, Finland
Manolow S.	Labor. of Regeneration, Bulg. Ac. Sci., Sofia, Bulgaria
Zaimom K.	Labor. of Regeneration, Bulg. Ac. Sci., Sofia, Bulgaria

VISITS ABROAD

Department of Neurophysiology

Czerwosz L.	St. Georges Hospital Medical School, University of London, U.K.
Głowicki K.	Institute of Physiology, University of Zurich, Switzerland
Karczewski W.	The Royal Society, London, U.K. University of Oxford, U.K.
Kubin L.	State University of Pensylvania, Philadelphia, USA (long term wisit)
Romaniuk J.	Metropolitan General Hospital, Cleveland, USA (long term wisit)
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Cardiovascular Laboratory

Cedro-Ceremużyńska K.	Cardiovascular Laboratories, Bayer, Wuppertal, GFR
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Department of Applied Physiology

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Kaciuba-Uściłko H.	University of Helsinki, Finland University of Oulu, Finland University of Kuopio, Finland University of Syracuse, N.Y., USA
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Department of Neuropathology

Faff-Michalak L.	Institute of Biology, Medical Academy, Magdeburg, GDR
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Laboratory of Developmental Neuropathology

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Department of Neurochemistry

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Department of Neurosurgery

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Witkiewicz B.	Institute of Neurosurgery, Moscow, USSR

Department for Surgical Research and Transplantation

Grzelak J.	Surgical Clinic, University of Saragossa, Spain
Łukomska B.	Department of Experimental Research, La Sapienza University of Roma, Italy Pharmacology Department, School of Medicine, University of Perugia, Italy
Olszewski W.	University of Madras, India Institute of Neurosurgery Research, Munich, GFR Radium Institute, Oslo, Norway
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Neuromuscular Unit

Hausmanowa-Petrusewicz I.	Baylor College, Houston, Tx USA, Columbia University, Neurological Institute, New York USA
Rowińska K.	Institute of Pathophysiology, Clinical Branch Academy of Medical Sciences, Moscow, USSR
Sieradzan K.	Department of Anatomy, University College, London, U.K. (long term visit)

Mental Health Department

Rożeńska R.	University Clinic, Heidelberg, GFR

PARTICIPATIONS IN INTERNATIONAL MEETINGS IN 1988

European Training Program in Brain and Behaviour Research, Zuoz, Switzerland, January 9-16th
Krynicki M., Romaniuk A.

The International Symposium on Work in a Hot Environment and Heat-Related Disorders, Khartoum, Sudan, January 27-31st
Grucza R., Kaciuba-Uściłko H.

Wather and Electrolite Metabolism in Cardiovascular Deseases, Berlin, GDR, February 2-4th
Sadowski J.

Congress of British Physiological Society, Bristol, U.K., February 7-8th
Sieradzan K.

Immunobiology of Passenger Cells in Allografts, Seul, South Korea, February 16-19th
Olszewski W.

First International Congress on the Immune Consequence of Trauma, Shock and Sepsis, Munich, GFR, March 1-5th
Grzelak J., Olszewski W.

International Workshop on Spinal Muscular Atrophy, Taormina, Italy March 27-29th
Hausmanowa-Petrusewicz I.

Congress Brain Research Association, London, U.K. March 29th
Sieradzan K.

Conference of European Group of Nutritionists „Nutrition, Metabolism and Physical Exercises", Prague, Czechoslovakia, March 30th — April Ist
Kaciuba-Uściłko H., Nazar K.

International Conference on Evaluation of Long Term EEG and Behavioral Data in Epilepsy and Sleep Disorders, Budapest, Hungary, April 14-17th
Majkowska J.

Multicentric International Trial with Cordarone in Secondary Prevention of Sudden Death after Myocardial Infarction, Paris, France, April 17-23rd
Cedro-Ceremużyńska K.

Symposium of Society for Clinical and Experimental Immunology „Monoclonal Antibodies in Morphology", Olbersdorf, GDR, April 18-20th
-Ziółkowska B.

I European Congress of Neurology, Prague, Czechoslovakia, April 18-22th
Emeryk B., Hausmanowa-Petrusewicz I., Ryniewicz B.

6th Tbilisi Symposium on Cerebral Circulation „Microcirculation of the Brain", Tbilisi, USSR, April 20-23rd
Czernicki Z.

2nd International Congress on Phlebolymphology, Ferrara, Italy April 20-23rd
Olszewski W.

7th Quadrilateral Congress of Surgical Research, Rostock, GDR, April 25-27th
Bryła P., Grochowicz O., Grzelak J.

Polish-Italian Joint Meeting on Neurology, Warsaw, Poland, May 2-3rd
Mossakowski M.J.

23rd Congress of European Society for Surgical Research, Bologna, Italy, May 3-5th
Grochowicz P., Łukomska B., Olszewski W., Piotrowicz W., Wąsowska B.

3rd International Symposium on the Immunology and Surgery of the Greater Omentum, Heidelberg, GFR, May 5-7th
Kubicka U.

The Deviated Immune Response — Immunology Course, Poiana Brasov, Rumania, May 10-19th
Sadowska D., Sitnicka E.

Conference of The Association of Neuropathologists of GDR, Görlitz, GDR, May 11-13th
Kapuścińska A., Kroh H., Tubylewicz J.

6th European Colloqium of Renal Physiology, Varna, Bulgaria, May 22-26th
Bądzyńska B., Sadowski J.

International Conference for Metabolism of Phospholipides, Perugia, Italy, May 26-28th
Strosznajder J.

27th Symposium „Outflow of the Cerebro-Spinal Fluid", Copenhagen, Denmark, June 1-5th
Śliwka S.

7th International Biochemistry and Exercise Conference, London-Ontario, Canada, June 1-4th
Budohoski L., Kaciuba-Uściłko H., Nazar K.

International Congress of Swiss Society for Neurosurgery, Aarau, Switzerland, June 6-13th
Jurkiewicz J.

7th Congress of European Neurochemical Society, Göteborg, Sweden, June 12-17th
Albrecht J., Domańska-Janik K., Strosznajder J., Zalewska T.

23th Annual Congress of European Society for Clinical Respiratory Physiology, Athens, Greece, June 20-24th
Janczewski W., Kukwa A., Romaniuk J.

20th Congress of The International Society of Blood Transfusion, London, U.K., July 10-15th
Wąsowska B.

12th Congress of The International Transplantation Society, Sydney, Australia, August 14-20th
Grochowicz P., Olszewski W., Romaniuk A., Wąsowska B.

European Society of Parenteral and Enteral Nutrition Congress, Leipzig, GDR, August 24-26th
Olszewski W.

10th Congress of European Cardiological Society, Wien, Austria, August 28-September Ist
Cedro-Ceremużyńska K.

European Association of Nuclear Medicine Congress, Milano, Italy, August 29-September 2nd
Kapuściński A.

International School of Electromyography, Rovinje, Yougoslavia, September 7-9th
Hausmanowa-Petrusewicz I.

International Symposium on Hypoxia, Berlin, GDR, September 12-14th
Gordon-Majszak W., Puka M., Salińska E., Waśkiewicz J., Zalewska T.

9th European Immunological Meeting, Roma, Italy, September 14-18th
Grzelak J., Krzywicki M., Kubicka U., Łukomska B., Orlewska E.

International Neuromuscular Meeting, Marseille, France, September 15-17th
Hausmanowa-Petrusewicz I.

9th European Meeting of International Society for Heart Research, Oxford, U.K., September 15-17th
Cedro-Ceremużyńska K.

3rd European Meeting of Neuropathology, Verona, Italy, September 26-30th
Dąmbska M., Kida E., Krajewski S., Kroh H., Matyja E., Zelman I.

Salzburg Conference on Cerebrovascular Diseases, Salzburg, Austria, September 30-October Ist
Mossakowski M.J.

„Basic and Applied Aspects of Muscle Physiology", Rheinhardsbrunn, GDR, October 2-6th
Pokorski M.

2lst Danube Symposium for Neurological Sciences, Varna, Bulgaria, October 13-15th
Hausmanowa-Petrusewicz I., Kida E., Kroh H., Majkowska-Wierzbicka J., Szumańska G.

Neuromuscular Symposium, Erfurt, GDR, October 17-20th
Hausmanowa-Petrusewicz I., Rowińska K.

2nd Scientific Meeting of The European Society for Psychosocial Oncology, Amsterdam, Netherland, October 24-25th
Szamburska J.

The 7th Soviet Union Congress of Neurologists, Moscow, USSR, October 25-28th
Hausmanowa-Petrusewicz I., Mossakowski M.J.