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RESEARCH OF INTERSYSTEM INTEGRATION IN PROCESS OF ADAPTATION TO PSYCHO-EMOTINAL STRESS IN CONDITIONS OF PHYSICAL LOAD

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The article features modern research data about physiological mechanisms of development of resistance to psycho-emotional stress that appears in medical students in dynamics of studies in university (during first 3 years).

The research was carried out within the scientific research work of physiology department of KNMU "Peculiarities of integrative and autonomic functions in process of adaptation to intellectual, emotional and physical loads" (№ of registration 0115U000239, execution period 2015–2017).

Results showed that development of adaptation syndrome in those conditions greatly depends on speed of formation and intensity level of intersystem integration in cardiorespiratory system. That relation is revealed most of all on the background of physical activity (load on bicycle ergometer to the full). The variability of stages of adaptation development was found out.

In first group of students the first stage of adaptive syndrome is characterized by activation of non-specific adaptive mechanisms (1st year of studies), that is naturally replaced by second stage – specific adaptive reactions, when the intellectual work capacity increases on the background of rationalization of its autonomic supply.

In second group of students the second variant of adaptation development occurs – non-specific adaptive mechanisms (1st stage) on the 2nd year of study are changed by the stage of specific adaptation (2nd stage), that is accompanied by excessive psychophysiological indexes. On the 3rd year of study it leads to the depletion of adaptive capabilities (3rd stage).

In third group of students the stage of activation of non-specific adaptive mechanisms was rapidly changed by their depletion that was accompanied by decrease of intersystem integration and intellectual workability.

Therefore, the formation of adaptive optimum occurs only in 40% of students. The majority (60%) of students-young women show either the initially insufficient adaptive capabilities, or excessive adaptation. Both insufficient and excessive adaptation naturally leads to psychological and autonomic disorders followed by appearance of psychosomatic neurotic manifestations.

The prospects of future research from our point of view should lie in further investigation of gender peculiarities of adaptive reactions development up to psycho-emotional stress that develops in conditions of studying in university. That will make an opportunity to reveal the features of mechanisms of resistance formation depending on gender.

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PHLEBOANGIODYSPLASIAS AND POSSIBILITIES OF THEIR CORRECTION AT KLIPPEL-TRENAUNAY-WEBER SYNDROME

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Phleboangiodysplasias relate to rare diseases of lower extremities' veins and combine the pathology of congenital origin in both the veins and in extravenous tissues. They are often connected with the presence of abnormal arteriovenous communications. Klippel-Trenaunay-Weber syndrome (KTWS) is one of the most common forms of hereditary phleboangiodysplasia and represents an abnormality of deep trunk veins of the limbs that appears as the limbs' hypertrophy, varicose veins and hemangiomas.

Objective. To study the clinical manifestations of phleboangiodysplasias and possibilities of their correction at KTWS.

Materials and methods. 17 patients with the clinical diagnosis and the presence of KTWS syndrome and phleboangiodysplasias were observed. The age of patients ranged from 3 to 26 years, female patients dominated (10(59 %) female, 7(41 %) male).

Results and their discussion. All patients were consulted by a vascular surgeon. Patients underwent the duplex scanning of the limbs' venous system, according to which all of observed patients had the symptoms of congenital lesions of the lower limbs' venous system. The choice of therapy was performed individually, taking into account the concomitant genetic background.

Clinical example: patient B., 16 y.o. At the admission he complained to the swelling and varicose veins of left leg from the 5 y.o.age. The phenotype peculiarities are: the nevi on the skin of 2–8 mm size, angiomas on the left knee, the hypertrophy of the left lower extremity with varicose subcutaneous veins, increased skin

stretch, scoliosis. The pedigree's analysis revealed burdened acute cardiovascular pathology. Karyotype is 46, XY, 2 % of chromosomal instability; blood biochemical analysis: hyperhomocysteinemia 19 mmol/L, the reducing of common sulfoglycans' level. The molecular exanimation of polymorphic genes of folate cycle system is: homozygous 677C/T MTNFR/66A/G MTRR compound is revealed. The ultrasound of the lower limb veins: valvular insufficiency of the popliteal and great saphenous veins, varicose changes according to the "rising varicosis" type, the manifestation of venous angiodysplasia on left leg and foot. The diagnosis: KTWS at the background of the deficiency of folate cycle enzymes. A conservative tactics of the patient supervision (compression stockings, venotonic medicine taking) was made. The pathogenetic therapy: restricted diet of food rich in methionine, folic acid, vitamins B6 and B12).

Conclusions: 1) timely medical and genetic consultation helps to improve the prediction and prevention of re-birth of sick children in families with KTWS; 2) timely instrumental verification of the volume and location of the venous system congenital abnormalities allows to foresee and prevent the development of serious complications.

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PHYSIOLOGY OF BONE HEALING

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Background. Success of patient's recovery after fractures, arthrodesis, osteotomies and bone grafting surgeries depends on the normal process of bone healing. The cells that make intercellular substances necessary for each stage of bone healing are not present in sufficient numbers at the moment of fracture or surgery, they appear under the influence of local and systemic agents. Errors in any of links of that process may lead to various kinds of retarded or abnormal bone healing, thus proving the importance of research of normal, physiological processes.

In the process of fracture healing, several phases of recovery facilitate the proliferation and protection of the areas surrounding fractures and dislocations. The length of the process depends on the extent of the injury, and usual margins of two to three weeks are given for the reparation of most upper bodily fractures; anywhere above four weeks given for lower bodily injury.

The process of the entire regeneration of the bone can depend on the angle of dislocation or fracture. While the bone formation usually spans the entire duration of the healing process, in some instances, bone marrow within the fracture having healed two or fewer weeks before the final remodeling phase.

While immobilization and surgery may facilitate healing, a fracture ultimately heals through physiological processes. The healing process is mainly determined by the periosteum (the connective tissue membrane covering the bone). The periosteum is the primary source of precursor cells which develop into chondroblasts and osteoblasts that are essential to the healing of bone. The bone marrow (when present), endosteum, small blood vessels, and fibroblasts are secondary sources of precursor cells.

There are three major phases of fracture healing, two of which can be further sub-divided to make a total of five phases – reactive phase, including fracture and inflammatory phase and granulation tissue formation; reparative phase, that consists of callus formation and lamellar bone deposition; and remodeling phase when the remodeling to original bone contour occurs.

After fracture, the first change seen by light and electron microscopy is the presence of blood cells within the tissues which are adjacent to the injury site. Soon after fracture, the blood vessels constrict, stopping any further bleeding. Within a few hours after fracture, the extravascular blood cells, known as a "hematoma", form a blood clot. All of the cells within the blood clot degenerate and die. Some of the cells outside of the blood clot, but adjacent to the injury site, also degenerate and die. Within this same area, the fibroblasts survive and replicate. They form a loose aggregate of cells, interspersed with small blood vessels, known as granulation tissue.

Days after fracture, the cells of the periosteum replicate and transform. The periosteal cells proximal to the fracture gap develop into chondroblasts and form hyaline cartilage. The periosteal cells distal to the fracture gap develop into osteoblasts and form woven bone. The fibroblasts within the granulation tissue also develop into chondroblasts and form hyaline cartilage. These two new tissues grow in size until they unite with their counterparts from other pieces of the fracture. This process forms the *fracture callus*. Eventually, the fracture gap is bridged by the hyaline cartilage and woven bone, restoring some of its original strength.

The next phase is the replacement of the hyaline cartilage and woven bone with lamellar bone. The replacement process is known as endochondral ossification with respect to the hyaline cartilage and "bony substitution" with respect to the woven bone. Substitution of the woven bone with lamellar bone precedes the substitution of the hyaline cartilage with lamellar bone. The lamellar bone begins forming soon after the collagen matrix of either tissue becomes mineralized. At this point, "vascular channels" with many accompanying osteoblasts penetrate the mineralized matrix. The osteoblasts form new lamellar bone upon