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АКТУАЛЬНІ ПРОБЛЕМИ ТА СУЧАСНІ ДОСЯГНЕННЯ**

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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 examination 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

the recently exposed surface of the mineralized matrix. This new lamellar bone is in the form of trabecular bone. Eventually, all of the woven bone and cartilage of the original fracture callus is replaced by trabecular bone, restoring most of the bone's original strength.

The remodeling process substitutes the trabecular bone with compact bone. The trabecular bone is first resorbed by osteoclasts, creating a shallow resorption pit known as a "Howship's lacuna". Then osteoblasts deposit compact bone within the resorption pit. Eventually, the fracture callus is remodeled into a new shape which closely duplicates the bone's original shape and strength.

Conclusion. Processes that provide normal bone healing are complex and depend one on another. Change of any of its components will lead to its breakdown and make the outcome unsuccessful. Moreover, some patients will need a repeat of surgery or other treatment in that case, thus physiological process of bone healing and factors that may improve or prevent it have a great importance in traumatology and orthopedics.

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FUNCTIONAL CONDITIONS OF THE RAT KIDNEYS UNDER THE SALT LOAD

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Human and animal kidneys are the efferent organs which provide support of the organism with water electrolytic balance, acid-base and osmotic homeostasis.

Kidney is one of the organs with a clear circadian functional organization. It is proved that various external and internal factors can influence their functioning. A chronological structure of the kidney's acid, ionic and excretion functions is being changed in course of ontogenesis and the ionic regulation function is the one that undergoes most significant changes, which manifest themselves through a sharp decrease of the sodium ions excretion because of activation of their proximal transportation. It has been found that the night tubular reabsorption of water is more active than the day one. Investigation of the rhythms of urea osmolarity, electrolytes excretion, hydrogen ions concentration, glomerular filtration and tubular reabsorption of water proves that the daily rhythms depend on interconnection between the glomerular and tubular systems. This conclusion has been drawn from the results of investigation carried out in the group of children suffering of enuresis and hypercalcinuria.

It is known that salt stress leads to disruption of ion-regulatory function (concentration and excretion of sodium increases) and acid-regulatory function (increase in excretion of titrative acids and ammonia) of kidneys.

A response of kidney to the water and salt loads depends on the content of sodium in meals and this is an evidence of the stable adaptive changes formed in the rat's organism and directed onto homeostasis maintenance. The kidney functional reserve plays an important role in this process. This is the main physiological mechanism governing depth of the kidney adjustment towards the water/salt homeostasis regulation. On the other hand, tubular reabsorption and secretion process is responsible for accuracy and specificity of this adjustment.

All experiments were carried out with white pubertal nonlinear male rats, weight 180 ± 10 g. All experimental animals were divided into three groups. First group (6 animals) – control, no load; second group (6 animals) – 5 % water load calculated as 5 ml of water per 100 g weight and third group (6 animals) – 3 % salt load in the form of infusion of the 3 % NaCl solution calculated as 2.56 mmoles (59 mg) of Na per 100 g weight.

All types of the load were applied 2 hours before euthanasia by the metal intragastric probe. The ion regulation function was evaluated by the sodium ions excretion values as well as by Na concentration in urea, its reabsorption value and filtration charge while pH of urea, excretion of the titratable acids and ammonia were used to evaluate the acid regulation function. Folin's method was employed to measure concentration of creatinine in urea while its concentration in the blood plasma was determined by photolorimetry.

It has been shown that both types of the load cause changes in the kidney functional activity indexes. For instance, it is proved by investigation of the ion-adjusting function of the rat kidneys that the 3 % salt load causes increase in sodium concentration in urea by 82 % comparing to the control group while this concentration rises by 28 % at the 0.75 % load. Excretion of sodium ions has also risen twice at the 3 % salt load and by 43 % at the 0.75 % load. Finally, it was found that the 0.75 % salt load caused a 27 % increase in the sodium filtration charge.

A rise in sodium concentration and excretion was provided mainly by decrease in its tubular reabsorption. On the other hand, changes in creatinine excretion and calculated glomerular filtration for the animals exposed to NaCl infusion prove that the filtration was experiencing increase simultaneously with rise in the sodium filtration charge. These changes in the kidneys functional activity under salt load can be caused by rise of sodium ions concentration in the organism and higher osmolarity after exposition to the salt load. It can also be noted that almost no changes in pH values after the salt load were registered except shifting pH for 16 % down after the 0.75 % load. Ammonia excretion rose by 24 % and 15 % while excretion of the titratable acids also increased by 19 % and 32 % for the 3 % and 0.75 % salt loads simultaneously.