hand, the velamentous insertion represents 0.27% and the marginal insertion - 0.12%. Both of them are associated with massive blood loss at birth, hemorrhagic complications in the antenatal period, miscarriage, hypotonia and cyanosis in the newborn, difficulties in approaching the birth and sudden intrauterine death of the newborn caused by hypoxia. Furcate insertion of the umbilical cord was not found, because it has the lowest incidence, as other studies have shown.

Conclusion. Even if the incidence of the pathological types of the umbilical cord insertion is low, the associated abnormalities are severe and must be taken into consideration. Thus, punctilious and continuous monitoring of the development of the pregnancy, including the analysis of the insertion type could help the obstetricians in providing professional medical assistance. It should be said that the formation of the umbilical cord is finished at 7 weeks of pregnancy. So, by using an ultrasonographic examination in the 1st trimester, between 12 and 15 weeks, it is possible to determine the insertion type of the umbilical cord. To sum up, all the necessary precautions can be taken in time and pregnancy/birth-giving can occur physiologically, with no harm to the mother and fetus.

Key words: Physiology, obstetrics, umbilical cord, development, pregnancy.

DEPARTMENT OF PATHOPHYSIOLOGY AND CLINICAL PATHOPHYSIOLOGY

285. PLACEBO AND NOCEBO EFFECTS - A SYNTHESIS OF MAIN UNDERLYING MECHANISMS

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Introduction. Although the effects of placebo have been known in medicine for several centuries, the research of the underlying mechanisms has developed relatively recently. Consequently, in the last decades, numerous studies and researches have been published, most of them focused on symptoms such as pain, fatigue, nausea and itching.

Aim of the study. To disclosure the main mechanisms underlying the placebo and nocebo effects.

Materials and methods. The study was performed in the base of reviewes of various researches and scientific materials (articles in specialized journals, monographs and articles on the Internet etc.) that refer to placebo/nocebo effects description and observation.

Results. The classical conditioning theory and the response expectancy model were considered for a long time the most accepted theories explaining the underlying processes of placebo/nocebo phenomena. Numerous researches revealed that suggestions, thoughts and beliefs could have an important influence on human body, thus giving rise to specific therapeutic processes. However, placebo and nocebo effects are not mediated only by psychological mechanisms. There is a clear evidence of neurobiological changes at different levels and areas of the brain, involving endogenous opioids, as well as dopamine, especially in the case of the analgesic placebo effect. Similarly, such neuromodulators as cholecystokinin play a significant role in the nocebo effect. The findings concerning the involvement of the genetic mechanisms in the process of manifesting placebo/nocebo effects cannot be neglected. In the last years, the studies reveal that genetic variations in the brain's neurotransmitter

pathways could influence placebo effects, the incipient evidence being provided with regard to existence of genes that mediate placebo effects in individual patients. Placebo/nocebo genetics is an area in which research is only in a quit early phase.

Conclusions. The study results evoke the complexity of the placebo and implicitly nocebo phenomena and the difficulty of formulating a generally valid theory, which could explain in a clear and complete manner how they function. The described mechanisms have to be regarded in a complementary manner and must be analyzed and regarded as a whole. It is worth to continue the investigations of placebo/nocebo mechanisms so as to optimize the therapeutic interventions and to improve the design of clinical trials.

Key words: placebo effect, nocebo effect, response expectancy, unconditioned factors, placebo genetics

DEPARTMENT OF BIOCHEMISTRY AND CLINICAL BIOCHEMISTRY

286. IS LACTATE A USEFUL MARKER OF CARDIAC HYPOXIA?

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Introduction. According to World Health Organization report, cardiovascular diseases are the leading cause of mortality, accounting for 31% worldwide. Cardiac ischemia is the key-mechanism that underlies acute coronary syndrome and induces cellular necrosis. Prolonged ischemia and hypoxia switch the myocardial metabolism from aerobic to anaerobic glucose degradation with increased lactate formation. The accumulation of lactate in cardiac myocytes results in acidosis, altered energy metabolism and cellular membrane damage. We suppose that myocardial ischemia could cause an elevation of circulating lactate level.

Aim of the study. The research purpose was to evaluate serum lactate levels in experimental acute myocardial infarction.

Materials and methods. Forty healthy adult male rats were randomly divided into five groups: (L1) - sham; (L2) - control NaCl 0.9% solution; and with acute myocardial infarction induced by subcutaneous injection of isoproterenol hydrochloride 100 mg/ kg (one dose), and sacrificed after 6 h (L3), 24 h (L4) and 7 days (L5) respectively. Serum lactate concentration was determined using standard kit (ELITech, France). Obtained data were represented by median and interquartile range. For group comparison, the Kruskal-Wallis and Dunn nonparametric tests were performed (SPSS 23.0).

Results. The investigated groups have shown statistically insignificant difference for serum levels of lactate ($p < 0.05$). Initially in L3 was identified a slight decrease, followed by a significant increase in L4, with a repeated fall in L5 group.

Conclusions. The obtained data denote the possible release of lactate from the ischemic cardiomyocytes to the blood. Serum lactate level, following acute myocardial infarction is a useful biomarker that reflects the severity of tissue hypoxia.

Key words: acute coronary syndrome, lactate, hypoxia, myocardial metabolism