

Speaker Abstracts

745

SP-9

TITLE Microgravity: Muscles, Membranes and Molecules

AUTHORS AND COUNTRY D. L. Feedback, M. M. Bamman and M. S. F. Clarke, NASA/JSC, USA

ABSTRACT

We have previously shown that there is a linear relationship between mechanical loading in muscle and the amount of myofiber fibroblast growth factor (FGF) released into the extracellular environment via a phenomenon termed "myofiber wounding". We also demonstrated *in vitro* that the growth-promoting effect of mechanical load upon human skeletal myotubes is specifically inhibited by an anti-FGF antibody. We postulate that a reduction in mechanical load-induced, myofiber wound-mediated release of FGF in microgravity contributes to the initiation of skeletal muscle atrophy during spaceflight. Our hypothesis is supported by experimental data gathered from Space Shuttle crew-members which indicates that circulating levels of a skeletal myofiber-specific wound marker, the MM isoform of creatine kinase (CKMM), is significantly reduced after short duration spaceflight. We have tested our hypothesis in a terrestrial model of spaceflight (i.e. 14 days of 6° degree head-down tilt bedrest) and determined the amount of myofiber wounding and FGF release which occurs during unloading of the human body. In addition, the effects of a resistive exercise countermeasure to skeletal muscle atrophy was studied at both the morphological and biochemical level in order to determine if prevention of myofiber atrophy was correlated with the degree of myofiber wounding and consequent FGF release.

SP-11

MAIN ACHIEVEMENTS OF RUSSIAN SPACE LIFE SCIENCES RESEARCH

A.I. Grigoriev, Russia

The analysis of data of investigations in piloted Salyut and Mir missions and Biosatellite programs revealed the development of a series of consistent shifts in the human body functional systems during and following space flight. Most noticeable are the shifts in sensory systems (space motion sickness, alterations in the motor activity, the structure of motor act, etc.), cardiovascular system (changes in the total and local hemodynamic regulation, and microcirculation leading to orthostatic instability), metabolism including water and electrolyte exchange, changes in body liquids, metabolism of proteins, lipids and carbohydrates; calcium metabolism and mineral density of bone tissues; the structure and function of muscles; immune and blood-forming systems. As was stated, the severity and the depth of these shifts were the function of flight duration, regularity and the extent of countermeasures, and cosmonauts' identity. All the deviations observed disappeared at different points of time after flight completion. No unfavourable delayed biomedical complications were noted after long (up to 14 month) human exposure to space flight. Mechanisms and patterns of human adaptation to micro-g became better understood on the basis of analysis of experimental data obtained with various living species on biosatellites.

New evidences are presented concerning the musculo-skeletal system and metabolism, inferring minimization of the functions in micro-g environment.

SP-10

Modification of Spatial Parameters of Goal-directed Arm Movements during Short- and Longterm Spaceflights

Gerstenbrand F., Berger M., Lechner-Steinleitner S., Kozlovskaya I.* Austria, * Russia

To investigate sensory motor functions in microgravity, goal-directed arm movements (GDAMs) were performed by cosmonauts (age range 31-47) that received one short-term (inflight time one week), eight long-term (inflight time 4-8 months, MV=5.3 months) and one super long-term (14 months) exposures to weightlessness. The ability to reproduce defined motor patterns was examined pre-, in-, and post-flight under two different performance conditions: In a first test the cosmonaut closed his eyes and his outstretched arm was passively moved to trace three times a visually presented pattern by the second cosmonaut. Still with eyes closed, the test person tried to repeat actively the movement sequence (the shape of an isosceles triangle) from memory. In a second test the test person actively traced the figure on the LED's matrix for three times with open eyes and repeated it with eyes closed. The different learning situations had an effect on metric parameters of the memorized stimulus pattern while the influence of the different gravity levels resulted in significant offsets and torsions of the reproduced figures.

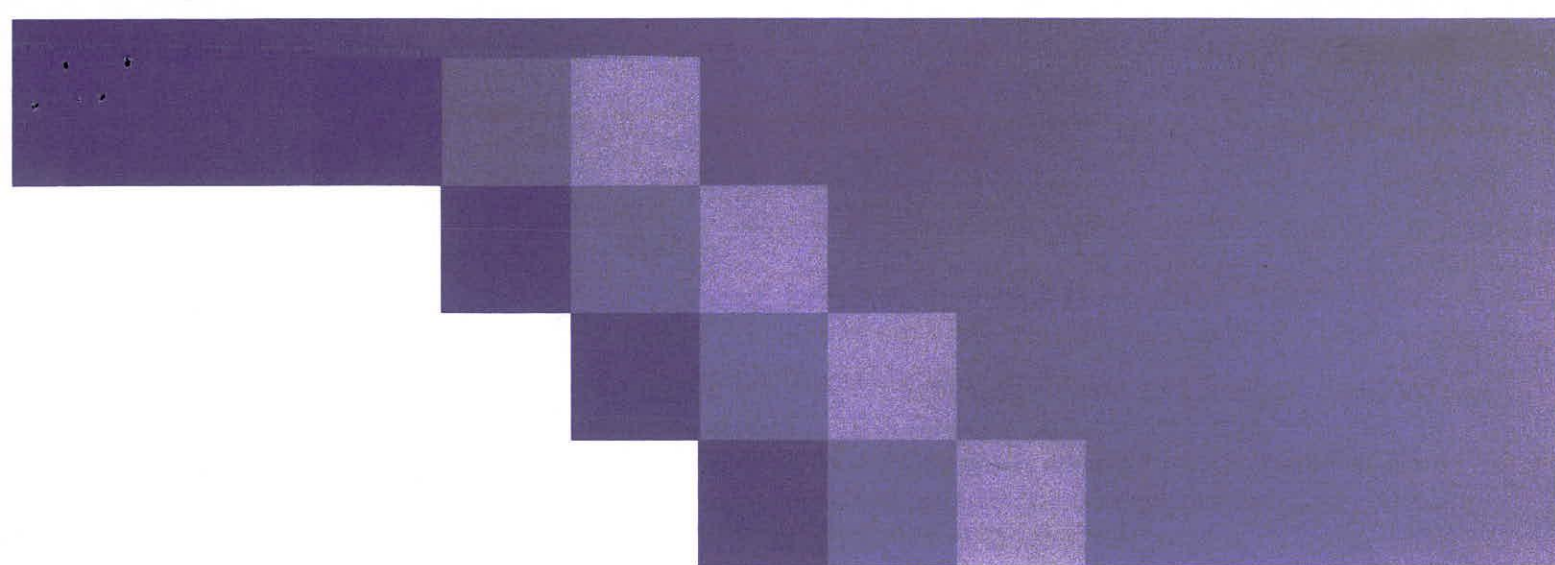
SP-12

Hyperbaric Oxygen Therapy: Pharmacology and Effects on Pathophysiology in Acute Central Nervous System Ischemia and Anoxia

Paul G. Harch, M.D., USA

Hyperbaric oxygen therapy (HBOT) is the use of high pressure oxygen as a drug to treat pathophysiologic processes and diseases. In acute cerebral anoxia/ ischemia, anoxia, ischemia, edema, and reperfusion injury are present. HBOT is known to reverse anoxia and edema and has been shown to powerfully inhibit reperfusion injury in two animal models (acute carbon monoxide poisoning and peripheral ischemia) and has suggested such an effect in two others (acute myocardial infarction and cerebral decompression illness). HBOT also has beneficial effects on vasospasm and energy and glucose metabolism in injured brain, yet causes vasoconstriction in normal brain tissue. Most of the above effects are dose and time dependent, such that hyperacute intervention has very potent results. Later intervention requires repetitive HBOT to cause trophic and possibly immunomodulatory changes.

The literature will be reviewed on the above topics, particularly with respect to the hyperacute (0-3 hour) period post-neurological insult and two illustrative cases presented, a 60 year old comatose male, status post cardiac arrest and an 11 year old near-drowning adolescent. A proposal for HBOT in acute stroke is offered with high pressure for the acute/hyperacute period and lower pressures thereafter.



**Congress on Cerebral Ischemia, Vascular
Dementia, Epilepsy and CNS Injury**

**New Aspects of Prevention and Treatment from
Space and Underwater Explorations**

World Federation of Neurology

May 9–13, 1998

Washington Marriott Wardman Park Hotel

2660 Woodley Road at Connecticut Avenue, NW • Washington, DC 20008

