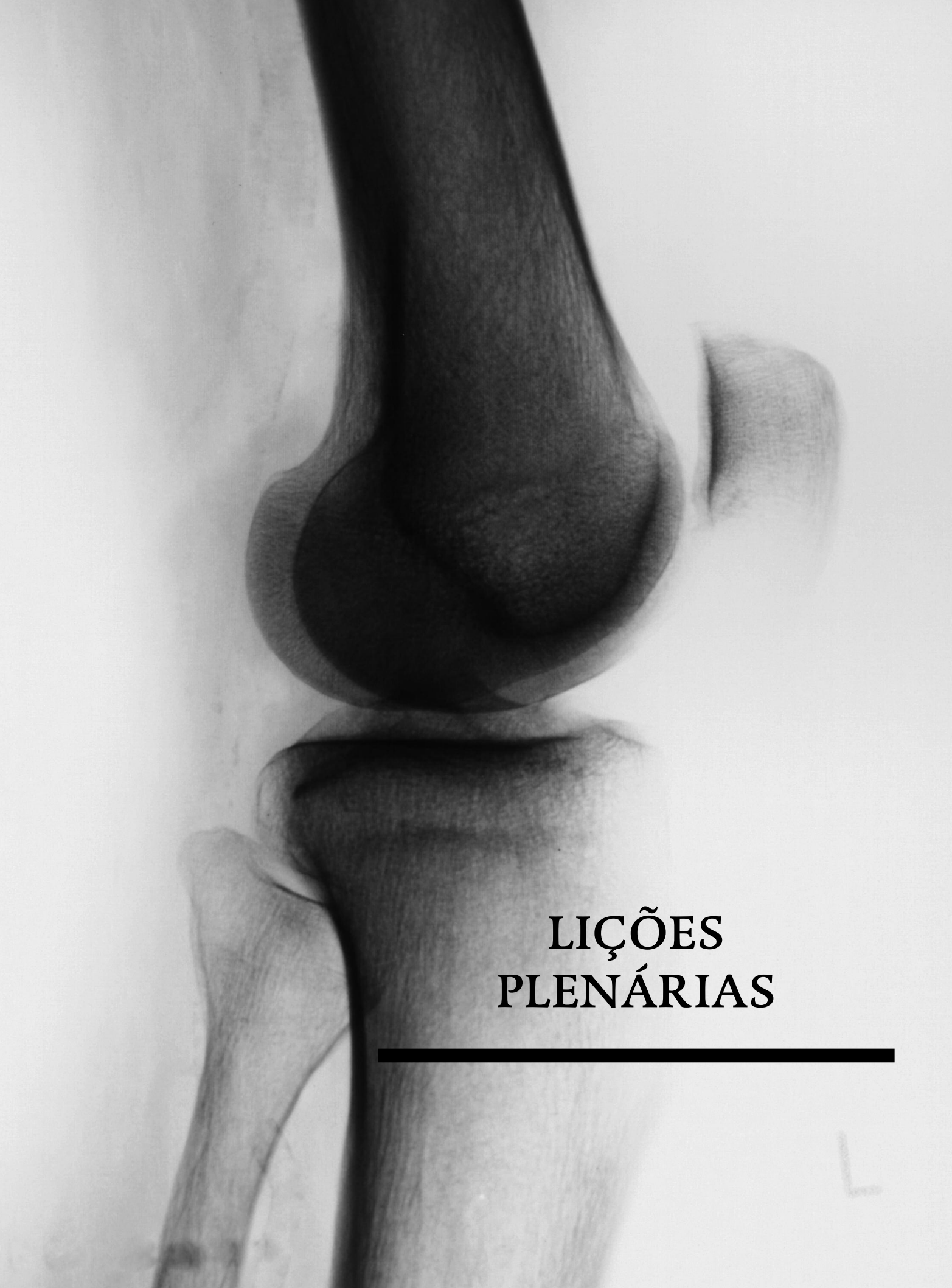

LIÇÕES PLENÁRIAS

Lições plenárias

ACTA REUMATOL PORT. 2012:37:33-35 (SUP)

LIÇÃO PLENÁRIA 1

DIA 3 DE MAIO DE 2012

THE OSTEOBLASTIC FACE OF THE BONE: FROM HARD BONE TO NOVEL TREATMENTS FOR BONE DISEASES.

Mathew L. Warman[1]
1. Investigator, Howard Hughes Medical Institute; Director, Orthopaedic Research Laboratories, Childrens Hospital, Boston; Professor of Orthopaedic Surgery and Genetics, Harvard Medical School

Osteoporosis-Pseudoglioma Syndrome, Autosomal Dominant High Bone Mass, van Buchem disease, and Sclerosteosis are Mendelian genetic human diseases in which patients produce too little or too much bone. The discoveries that these diseases are caused by mutations in the cell surface receptor LRP5 and the secreted protein SOST led to the identification of a signaling pathway that osteocytes use to regulate bone mass and to respond to changes in mechanical load. I will describe the studies in humans, mice, and frogs that identified the components of this signaling pathway and the strategies scientists are currently using to modulate this pathway in order to improve bone health. At least one biologic agent that targets this pathway is in Phase II clinical trials for patients with osteopenia and several others are in pharmaceutical company pipelines.

LIÇÃO PLENÁRIA 2

DIA 4 DE MAIO DE 2012

CARDIOVASCULAR RISK AND THE EFFECT OF TNF INHIBITORS

Daniel Solomon[1]
1. Harvard medical School

Patients with rheumatoid arthritis (RA) are known to be at an increased risk of cardiovascular disease, at least in part because of the systemic inflammation associated with RA. TNF is an important cytokine associated with cardiovascular disease. While TNF blockade was not found effective for heart failure, these agents appear to reduce the risk of ischemic cardiovascular endpoints in epidemiologic studies. As well, TNF blockade is associated with improvement in several cardiovascular risk factors. This lecture will review the biologic basis of the potential role of TNF blockade in cardiovascular disease and examine published epidemiologic data. As well, the importance of randomized controlled trials of TNF blockade will be emphasized.

LIÇÃO PLENÁRIA 3

DIA 5 DE MAIO DE 2012

NEW APPROACHES TO PAIN MANAGEMENT – A PARADIGM SHIFT

Michael Spaeth[1]
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Acute and chronic pain are based on different mechanisms. Whereas acute pain is related to tissue injury or inflammation (protective), chronic pain is not just longer lasting acute pain, but is a disease process with different underlaying mechanisms. Chronic pain lacks consistent tissue abnormalities, but displays features of hyperalgesia and allodynia.

Many recent studies on chronic pain states have demonstrated central nervous system pain processing abnormalities, such as sensitization and persistent pain ("wind-up"), including a loss of inhibitory neurons ("neuroplasticity"). These mechanisms are triggered by the release of excitatory amino acids such as glutamate, substance P and nerve growth factor. After central sensitization has been established, only minimal peripheral input is required for the maintenance of a chronic pain state. The system of „descending inhibition" inhibits nociceptive neurons by releasing (among other neuropeptides) serotonin. Noradrenergic neurons in the medulla are involved.

Furthermore, other receptors participate in the regulation of the pain system, such as alpha-2-delta receptors and serotonin receptors. Alterations of the en-

docrine system, indicating chronic stress, among others, have been shown to contribute to chronic pain states. Abnormalities in sleep architecture may result in non-restorative sleep. Chronic pain patients often report sleep disturbances and fatigue.

Consequently, drug treatment of chronic pain now modulates these different mechanisms and their interactions. Thus, compounds which primarily have not been thought to be used as pain medications, have now been shown to be effective in the treatment of chronic pain. Furthermore, pain management is a multidisciplinary challenge.