Symposium 2

'Temporomandibular Disorder and Anti-inflammation Treatment'

Articular Inflammation and Molecular Pathology in the Geneses of the Temporomandibular Joint Disorders

Jun-Ichi Ishimaru, DDS, PhD

Temporomandibular joint disorders (TMDs) have been considered as non-inflammatory disease, however, recent studies have disclosed lots of evidences of articular inflammation associated with TMDs. They may be categorized mainly into four groups. The first is by inflammatory cytokines such as IL-β and TNF-α. The second group is by inflammatory articular markers such as MMP-3. The third is by neuropeptides such as neurokinins or substance-P. Final one is by free radicals such as superoxide and nitric oxide. There have been many reports to identify those markers in the synovial fluid of the patients with TMDs, which highly suggest inflammatory pathology in the genesis of the TMDs. The author and co-workers have been engaged in the clinical study to identify and analyze MMP and TIMP in both sera and synovial fluid of the patients with TMDs. We have also quantitatively analyzed the degree of intra-articular oxidation. The details of our results will be discussed correlated with the articular inflammation to consider the possible mechanism in the genesis of the TMDs.

Arthrocentesis: Its Use for the Elimination of Inflammation from the TMJ, Facilitating its Function Rehabilitation in a Variety of Disorders

Dorrit W. Nitzan, DMD

Overloading of extrinsic or intrinsic origin is potentially harmful to the joint. During inflammatory event, the joint is perfused and physiologic load becomes pathologic resulting in increased intra-articular pressure (IAP). Such an event, if not controlled, may initiate a cascade that may end up in degenerative changes of the joint via either disruption of the lubrication system or, alternatively, by causing stiffness of the sub-chondral bone that reduces its shock absorbance ability.

The central role of inflammation in the initiation of various TMJ disorders clarify the need for its elimination when treating these patients. A way to accomplish this mission is by arthrocentesis, a non-arthroscopic lavage of the joint’s compartments. Arthrocentesis and mobilization became a treatment of choice in a variety of disorders.

Phospholipase A₂, a very primary product of the inflammatory cascade, is an existing threat to the lubrication system of the joint. When uncontrolled, various disorders may develop. One disorder is ‘anchored disc phenomenon’ in which sudden inability to open the mouth occurs. It is now known that, as a result of the absence of lubricants, adhesive forces are adhering the disc to the fossa, totally preventing its sliding. Arthrocentesis releases the disc and eliminates the inflammation thereby rehabilitating the joint function. Mobilization of the joint is then critically used for the final elimination of inflammation and function rehabilitation.

Similarly, a beneficial use of arthrocentesis in painful TMJ, intermittent clicking, open lock, osteoarthritis etc. takes place and will be discussed.

Finally, the primary role of over loading and inflammation in synovial joint osteoarthritis will be presented by new animal models. Such models may lead to new treatment modalities for osteoarthritis.

Peripheral and Central Pain of Temporomandibular Disorders

Gary M. Heir, DMD

Can pain of peripheral inflammation initiate chronic, central pain?
Can we predict when an inflammatory process will generate chronic pain?
What are the mechanisms?
What is the relation to temporomandibular disorders?
What is the treatment?

The temporomandibular joint is a highly innervated structure with significant intracapsular vascularity. It is subject to injury, dysfunction, pathological processes and the inflammation that accompanies these conditions.

Inflammation is a fundamental pathologic process consisting of a dynamic complex of cytological and histological reactions that occurs in affected blood vessels and adjacent tissue as a response to an injury or abnormal stimulation caused by physical, chemical or biological agents. The initiation of pain is the result of lowered firing thresholds of nociceptors. This phenomenon, secondary to tissue damage, normally resolves once the tissue damaging or pathologic process ends.

Peripheral inflammation is appropriately and effectively treated with various anti-inflammatory medications. However, in some cases pain continues after healing and local inflammation resolves. Continuous pain can have both peripheral and central etiologies resulting in more widespread, chronic pain. Peripheral receptor sensitization results in second order neurons becoming more sensitive to normally non-painful stimuli. Central sensitization occurs, non-noxious input is perceived as painful, and pain spreads over a wider receptive field. As chronic peripheral pain input results in expansion of the receptive field of pain it assumes a neuropathic quality which is not responsive to NSAIDs. Alternate treatment strategies must be considered.