Fuzzy Knee Proprioception Would be of Benefit

Perspective

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The cartilage lacks histological means to transmit sensations but, at the cellular level [1], it has a (unique) [2] prosperity to transmit mechanical stresses to bone tissue and particularly to the subchondral plate. Apart from lubrication, the main function of cartilage is not to absorb energy through deformation but to distribute the load evenly to the subchondral plate.

The deformation of the cartilage is not an immediate process but take time and requires a static charge and through a mechanism based on the distribution of liquid (ECM).

The subchondral bone is responsible for transforming the mechanical signals that come from cartilage to neurological signals.

Comparing proprioception before and after TKA and PUC, it has been found that proprioception was improved after joint replacement which means that the subchondral bone before arthroplasty has stopped (or reduced) transmit mechanical signals to neurological signals and the bone (under implant) regain, at least partially, its capacity to transmit mechanical signals to neurological signals.

Does osteoarthritis begin with pathology of the subchondral bone that cause the pathology of cartilage due to the problem of translating the mechanical signals to neurological signals i.e. less proprioception leads to less control due to less interaction and finally cartilage damage? Or it is the cartilage with other soft tissue [3,4] which failed to pass the mechanical signals (precisely), so mechanical signals arrived to subchondral bone with disfiguration, in this case the subchondral bone has no choice other than translate that disfigured mechanical signals to disfigured neurological signals.

When we knew that the histological examination showed a loss of neural elements in the ligaments of arthritic knees [5] then we can expect the loss of joint mechanoreceptors of the subchondral bone and reduce proprioceptive acuity in arthritic knees.

To regain normal interaction and knee function, the cartilage's ability to transmit mechanical signals should be precisely regained.

The possibility that implant acquires transmissions functions similar to those of cartilage sounds increasingly fiction. The Improvement of implants in a way to simulate the transmission of mechanical stress by cartilage to subchondral bone is not fiction.

One solution may be resided in implant designing by adding slots in the design to allow what is left from healthy cartilage to continue its function, which would preserve the original qualities of mechanical signals. This seems feasible given that the subchondral bone responds well to pressure by the interface of fluids and not necessarily a directly [6]. In addition, these slots would help economising as much as possible of the healthy subchondral bone. Modification of (surgeries) [7] targeted to improve or regain subchondral bone abilities of translating the mechanical stress to neurological signals is another track to be explored with mini invasive subchondral intervention like concomitant refreshing (pridie) of the subchondral bone.

In considering disease-modifying treatment for osteoarthritis it makes more sense, in our opinion to direct attention to the correction of the underlying mechanical abnormality than to the development of pharmacological or biological agents [8].

We have to look for new ways and indications to prevent mechanical defect from cause subchondral proprioception [9] dysfunction as early as possible rather than correct its consequences (consider all neurological defects [10] in pain assessments).

Slotted implant design, mini invasive intervention would provide highly targeted mechano-neurological managements of arthritic knee.

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