Perspective
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Fuzzy knee proprioception would be of benefit

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Perspective

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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Conflicts of interest

None.

References

1. Eckstein F, Hudelmaier ZM, Putz R The effects of exercise on human articular cartilageJournal compilation. *J Anat.* 2006;208(4):491–512.
2. Sophia Fox AJ, Bedi A, Rodeo SA The Basic Science Of Articular Cartilage Structure Composition and Function. *Sports Health.* 2009;1(6):461–468.
3. van der Esch M1, Knoop J, Hunter DJ et al. The association between reduced knee joint proprioception and medial meniscal abnormalities using MRI in knee osteoarthritis: results from the Amsterdam osteoarthritis cohort. *Osteoarthritis Cartilage.* 2013;21(5):676–681.
4. Cho YR, Hong BY, Lim SH et al. Effects of joint effusion on proprioception in patients with knee osteoarthritis: a single–blind, randomized controlled clinical trial. *Osteoarthritis Cartilage.* 2011;19(1):22–28.
5. Cammarata ML, Schnitzer TJ, Dhaher YY Does Knee Osteoarthritis Differentially Modulate Proprioceptive Acuity In The Frontal And Sagittal Planes of The Knee? *Arthritis Rheum.* 2011;63(9):2681–2689.

6. van Dijk CN, Reilingh ML, Zengerink M et al. Osteochondral defects in the ankle: why painful? Knee Surg *Sports Traumatol Arthrosc.* 2010;18(5):570–580.
7. Gomoll AH, Madry H, Knutsen G et al. The subchondral bone in articular cartilage repair: current problems in the surgical management. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(4):434–447.
8. Brandt KD, Radin EL, Dieppe PA et al. *Yet more evidence that osteoarthritis is not a cartilage disease.* 2006;65(10):1261–1264.
9. Sharma L Role of Proprioceptive Deficits Ligamentous Laxity And Malalignment In Development And Progression Of Knee Osteoarthritis. *J Rheumatol Suppl.* 2004;70:87–92.
10. Barry Wyke Articular Neurology – A Review. *Physiotherapy.* 1972;58(3):94–99.