Do Capsular Pressure and Implant Motion Interact to Cause High Pressure in the Periprosthetic Bone in Cementless Total Hip Replacement?

INTRODUCTION:
Osteolysis is considered as one of the major contributors to aseptic loosening and long term failure in total hip replacements (THR). Osteolytic lesions are more extensive and common in cementless femoral components when there is a lack of seal in the bone-implant interface [1]. To explain the mechanisms involved in osteolysis development, the concept of an ‘effective joint space’, in which joint fluid follows the path of least resistance along interface gaps according to pressure gradients, has been postulated [2]. It has been shown that the presence of adequate number of particles, high fluid pressure, and/or high-velocity fluid flow can cause bone resorption in the effective joint space [3,4]. In all these hypotheses, periprosthetic flow plays a central role in the development of osteolysis and yet this is poorly characterised and the source of elevated fluid pressures has yet to be clearly identified. It has been shown that during physical activities in patients diagnosed for THR revision surgery capsular pressures can be elevated to 69 kPa from rest [5]. Implant micromotion may also induce high fluid pressures and flows at the bone-implant interface [6]. In the current study, the role of capsular pressure, gap dimensions, and the opening and closing motion of the gap during cyclic loadings of an implant will be explored using 2D computational fluid dynamics (CFD) simulations.

METHODS:
The 2D geometry of the model was obtained from a cross-sectional cut of a realistic 3D model of a cementless femoral stem implanted into the proximal femur and the surrounding joint capsule in the coronal plane. Interface gaps between the bone and implant with the desired length (l) and width (w) were embedded in the 2D geometry and then the entire geometry was meshed in GAMBIT. The meshes were then imported to the commercial CFD software Fluent, in which the capsule and gap regions were modeled as fluid continuum, the bone was presented as a porous medium and the implant wall was described as a moving rigid wall. The fluid flowing in the capsule, gap and the bone was modeled with properties of liquid water. Laminar viscous flow was assumed to solve the Navier-Stoke’s equations in these regions. The bone was described as a simple porous medium in which the fluid momentum loss was defined by Darcy’s equation. Fluid pressure inside the capsule was generated by a pressure outlet boundary defined on the soft tissue wall of the capsule. The pressure over the porous bone region was assumed to be zero with respect to the capsule (i.e. 0 kPa). This was achieved by defining a pressure outlet on the endosteal and periosteal surfaces of the bone. The implant motion was displacement-driven and it was simulated by a cosine wave presenting a simplified gait cycle. The parameters varied in this study were capsular pressure p (1 to 60 kPa) gap width w (30 to 500 µm) gap length l (5 to 80 mm) and gap displacement d (30 to 300 µm). A parametric study of 16 models including extreme cases for these parameters was set up. Models were analysed by comparing the steady-state (no micromotion included) solutions and transient solutions at times corresponding to fully closed gap, opening with maximum velocity, fully opened, and closing with maximum velocity. RESULTS:
In general, it can be seen that capsular pressure extends down the gap and into the surrounding bone (Fig.1). The only exception is the model with the longest (80 mm) and narrowest gap (30 µm) in which the capsular pressure does not penetrate the entire length of the gap and drops to approximately one fifth of the capsular pressure at the gap distal tip. Capsular pressure was the dominant force driving the fluid into the bone rather than implant micromotion. While micromotion creates a pumping action in the gap, it does not generate significant flow to the bone tissue and does not cause increase in fluid pressure except for the very long and narrow gaps in which micromotion as small as 30 µm can cause significant high fluid pressure fluctuations between 40 kPa and -40 kPa in opening and closing motions, respectively. Gap dimensions l and w, also influence the flow variables, although to a lesser extent. In the bone, as the fluid passes across the bone-gap interface, there is a significant decrease in velocity. In this region the velocity is of the order of a few mm/s to µm/s depending on the gap dimensions and the capsular pressure. In all the models, as fluid crosses the gap-bone interface, there is always a sudden spike in fluid velocity in the bone at the proximity of the bottom of the gap, which in some cases could be up to ten times larger than the fluid velocity in the neighbouring bone region. These velocity spikes are not affected significantly by changes in gap dimensions and are primarily a function of capsular pressure. Implant micromotion can increase the maximum velocity at these spikes by up to two orders of magnitude only when a gap is very long and narrow.

REFERENCE: