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BONE BIOMECHANICS

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INTRODUCTION: The multi-billion orthopaedic device industry is churning out every year millions of devices, which are fitted to fractured bones to mend them or to bone ends to replace disfunctional joints. Forty years ago Charnley brought about one of the most visible revolutions in the medical profession and the industry catering to it by devising "bone cement" – as we got used to call room-temperature curing resins of polymethyl methacrylate, in its bulk form better known by its trade name Plexiglas (Lucite in the US). Thanks to Charnley, total joint replacement became a household item, admittedly much appreciated by millions of patients grateful to wear (!) one or more of them following, usually, years of suffering from joint pain.

TROUBLES: Not surprisingly, not everything is quite right with cementing a piece of metal in a reamed-out bone cavity by a quickly setting mass of plastic – the so-called revision burden (percentage of surgical procedures done to replace a failed component) is an impressive 20%. Industry did not help -- in spite of decades of R&D on cementless devices -- their overall performance is still inferior to those cemented. Sadly, it seems that all of the efforts to decrease the revision burden have in fact led to its almost dramatic increase. So-called high-demand cementless prostheses are also high-cost items – greatly favored and promoted by the industry. Yet, on average, they appear to loose their anchorage to bone at rates higher than Charnely's.

MOTIVATION: Does industry have anything to learn from biomechanics of bone? Admitting or not, it sure seems in need. This presentation is intended as an encouragement for those who would like to try to research the problems -- no lack here of either the problems or the will to solve them -- and then, hopefully, teach the results to industry. Medical professionals, surgeons in most instances, need just as much, if not more help (to find their way through the maze of offerings). I will present a small collection of hypotheses, some of which have been supported by limited analysis or experimentation, developed by my collaborators and myself at the AO/ASIF Research Institute, Davos, in early nineties. None of it has been published. In a modest way, some of these concepts have been put to use in a total hip prosthesis for dogs, now in some 1'800 dogs, some with 10 years of follow-up, mostly encouraging. But, by and large, these proposals await thorough studies to confirm or to disprove them.

BONE: Even in its most compact, cortical form, bone is a strongly anisotropic composite -- strength of the cortical bone is typically an order of magnitude higher in the longitudinal than in the transverse axes -- stiffness by a factor of three. This difference arises from the osteonal level of organization, itself the result of a continuous process of bone remodeling through coordinated actions of osteoclasts and osteoblasts, the former in the driver's seat. Should the clasts loose their way, bone would quickly loose its load-supporting function. Where is the compass?

BIOMECHANICS: Bone is poroelastic, i.e. its deformation entails fluid transport in all, but most special load/deformation states. Fluid saturates, percolates and convects. All of the bone is affected, its cells not the least. Biomechanics of bone has paid some attention to the impact of fluid movement on mechanical properties of bone, but the effects here, due to stiffness strongly dominating viscous drag (in contrast to, e.g. cartilage), there is not much to write home about. Streaming potentials fared much better, interest in them driven by the potential utility of electrical / magnetic stimulation. And then, there is all of the molecular biology side, almost to exclusion dealing with what molecules are produced and where and, at best, in response to what. But how do they get to the site of action? Through the fluid, no doubt. Perhaps there are bridges to be built here between biology and mechanics – biomechanics is still in need of defending its name (my spelling checker does not like it either). And not only the compass of the clasts is at stake.

COMPASS LOST: Osteoclasts tunnel through the bone at the apex of so-called cutting cones – followed by blasts producing new bone to fill the tunnel. To remove the mineral and digest the bone, the clasts pump H^+ ions through their ruffled border, sealed at the perimeter, causing a strongly acidic environment in which the bone mineral is dissolved. We have proposed that the tunneling process maintains its orientation by the fluid movement in and out of the resorption cavity, driven by the volumetric strain of the surrounding bone. As it turns out, with the boundary conditions approximately representing Haversian bone, the fluid movement at the equator of the cavity is at its strongest and is nearly zero at the apex (pole). Thus, the clasts can do it at the apex because there they *can* maintain their seal and low pH – ions they produce are trapped near the ruffled border, escaping by diffusion only. As it happens, poles of the resorption cavities are (usually, but subject to the boundary conditions) on the main tensile trajectories! Now, what about interfaces to, universally impermeable implants? There can be no flow across these interfaces, hence no dilution of H^+ ions. At and near this enforced no-flow boundary, clasts are left to digest as they please -- the blasts may have difficult time to orient themselves and repair the damage. How about a draining interface? How about neutralizing H^+ ions? Is our ability to provide for an

indefinite, stable to no fault anchor for bone implants fundamentally limited by their (in most cases) impermeable nature?

SMALL SCALE ORIENTATION: A couple of structural levels down from the osteon, collagen fibers and whatever organic part of the matrix is associated with them, are the scaffold of the composite, its reinforcement and its dominant mechanical determinant – deproteinased bone is of no use as a load-carrying material. Collagen in bone, as in other connective tissues, is oriented. By what mechanism? We have studied cartilage and have discovered a plausible mechanism – collagen fibrils can get oriented by oscillating fluid flows, dragging the short fibrils (prior to their incorporation and cross-linking into fibers) against the gel like network of proteoglycans. If at play as well in forming bone (osteoid), there will be a definite problem getting this mechanism to work unperturbed near impermeable interfaces. Again, opening up our implants for fluid flow, at least partially, may be useful.

CONVECTION: Bone is ready and able to fill in a defect such as that created by our preparations of bone to insert a prosthesis. Or to bridge a gap at a fracture site. It seems very plausible that molecular signals driving the process originate at extant bone. How do they move out? Diffusion can of course provide for some of it, but there is a race between bone and other tissue types, which can do the same – grow into defect. If the task is to anchor a prosthesis, bone is at a premium. We have observed in animal studies reluctance of bone to approach our implants – new bone growth seems to readily start out from the extant cancellous bed, but then at a millimeter or so from the implant, to slow down to a crawl, failing to bridge the gap. If convection is at work, it sure cannot work near the impermeable, closed surface of the implant. We have thus tested a perforated nail, as a model, albeit on a millimeter scale, of a hydraulically open "interface". Since bone now readily grew through these perforations, the term interface is rather a misnomer – bone simply engulfed the implant. There are now some 1800 acetabular total hip components implanted in dogs, demonstrating the potential of such hydraulically open anchors.

STABILITY: Some of these have failed to integrate – most likely due to lack of mechanical stability – there is no excuse here – bone will not touch anything that moves. Once a fibrous layer sets in – there is no way to get a satisfying function. Years ago, after numerous failures to provide for a reliable, solid bony integration of cementless total knee prosthesis, a concept of "floating" anchorage was promoted as acceptable – as long as it was not progressive – perhaps it was for undemanding humans, but dogs would not have any of it. Fibrous tissue has no solid matrix to take the load – upon loading its intrastitial fluid is pressurized, much as in cartilage, blocking off any blood supply to the underlying bone. Originally of low density, cancellous bone gradually turns into something very much resembling subchondral bone, sclerotic if not completely dead. Just where the pain originates, is unclear, but it certainly does with any near-normal activity.

GAP PUMP: We have done a large number of fracture healing experiments, addressing design of implants and the issues of treatment protocols, but also the basic mechanisms of bone healing. One of the studies involved gap healing in the context of free space created on purpose around the fracture. The fastest strength recovery was observed in the group with a relatively large inter-fragmentary gap (about a millimeter) and a large space produced on purpose between the bone and the surrounding muscles – the worst results (close to a non-union) were with the same gap size but no space. Intuitive? Perhaps. To help interpret this outcome, we have shown that compressive loading of the early tissue in the gap leads to a net outflow of fluid from the adjacent bone fragments – this would be forced convection bringing out all the good signals; and that mineralization permissive strain distribution closely resembles the conversion of bone precursors to mineralized bone. A statistical outlyer was also detected – a fracture fully healed by three weeks … how about a cluster centered on that outlyer?