BIOLOGICAL AND MECHANICAL CHANGES OF THE BONE-GRAFT-CEMENT INTERFACE AFTER IMPACTION ALLOGRAFTING

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Introduction
The host bone interface after impaction allografting consists of morsellized allograft alone or as a composite with cement and it may be important for the clinical success for this procedure. The purpose of this study was to investigate the temporal changes of these interfaces in a rat bone chamber model.

Methods
The bone chamber model was a modification of one already successfully used to study impacted allograft bone in the rat. In contrast to two subcortical bone ingrowth openings, a microenvironment was created which allowed bone ingrowth only from the endosteal surface to simulate a damaged endosteal circulation (Figure 1). Bone chambers were placed in both tibiae of thirty-three male mature Sprague-Dawley rats (480-550g), containing either a bone allograft or a bone/allograft PMMA cement composite. The morsellized cancellous bone grafts for use in the bone chambers were harvested from the distal femur and proximal tibiae from mature male Sprague-Dawley rats and were compressed in a 2mm diameter metal chamber for 2min with 80N to remove fat and bone marrow and stored at -80°C. After the graft and a spring were inserted, the bone chambers were closed with PMMA lids. The spring which was inserted to keep the samples in place, exerted a constant compressive force of 1N, which was approximately 20% of the rat’s body weight.

The tibiae of three of the eleven rats in each group (0 week, 3 weeks or 6 weeks) were processed for non-decalcified histology. Sections were cut and ground to 50μm and stained according to Goldner. From the mid section of the chamber, graft porosity, percentage fibrous tissue, cortical porosity and cortical thickness were determined with IMAGE-PRO 4.5 (MediaCybernetics, Silver Spring, MD, USA). The other specimens were mechanically tested in torsion. A rotational displacement of 0.5% with compressive dead weight of 1N was applied until failure. After testing, the samples were processed for non-decalcified histology and analysed histomorphometrically as described above.

Results
Only sparse or no graft remodelling was observed at 3 weeks independent of the content of the chamber (Figure 2). No direct bonding of the graft particles in the cement to the endosteal surface was observed. The periosteal surface below the bone chamber was actively remodelled which resulted in a significantly increased cortical porosity and cortical thickness at 3 and 6 weeks compared with the 0 week group (Figure 2). At 6 weeks the periosteal remodelling and endosteal absorption resulted in the formation of a new medullary canal and cancellisation of the endosteal cortex (Figure 2). The composite-host bone interface strength was significantly higher at 3 weeks and was higher than the allograft construct. The construct strength decreased at 6 weeks for both the allograft and the composite construct.

Discussion
The increased interface strength of the composite-host bone interface at 3 weeks was due to fibrous tissue attachment rather than direct bonding of the bone particles. Extensive periosteal remodelling and endosteal absorption was observed which significantly increased cortical porosity and cortical thickness (Figure 2). Increased cortical porosity is known to be caused by a damaged endosteal circulation which results in a necrotic endosteal cortex. The endosteal cancellisation which results in medullary canal widening was observed in primary cemented THR and was correlated with radiological loosening. Theses results suggest that the endosteal circulation may be damaged and impaired after a revision with impaction allografting resulting in medullary widening and potential stem subsidence.


Acknowledgement: The authors acknowledge the George W. Bagby Research Fund, the Canadian Institute of Health Research, the Maurice E. Müller Foundation, the Swiss Academy of Engineering Science and the Robert Mathys Foundation for research funding.