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BIODEGRADABLE TRACHEAL PROSTHESIS USING HYDROXY-APATITE(HAP) AND CARBON FIBER(CF)

To solve the problem of infection, abnormal granulation and extrusion of the tracheal prosthesis, and to give good epithelialization on the inner surface, we developed a new biodegradable tracheal prosthesis consisting of HAP rings as artificial tracheal cartilage rings, and CF mesh tube coated by fibrin glue as tracheal tube.

On five mongrel dogs, this new tracheal prosthesis was implanted to the cervical trachea after resecting five tracheal cartilage rings. And six to ten weeks later, the specimen was taken out and examined by light(LM) and scanning electron microscope(SEM).

One of five implanted tracheal prosthesis was infected and extruded 2 weeks after operation. The other four were attached satisfactorily, and at a macroscopic observation, the HAP rings were firmly anchored to the cartilaginous tissue and overgrowth of the tracheal mucosa was recognized on the inner surface of the CF tube.

LM findings revealed fibroblasts and cartilaginous tissue growing into the pore of HAP rings, and the overgrowth of the tracheal epithelium was observed to some extent on the inner surface of CF tubes. In SEM findings, etching of HAP grains was observed as a sign of resorption, and biodegradation of CF was also observed.

In conclusion, HAP ring has excellent histocompatibility to the cartilage and the overgrowth of epithelium could be seen on the CF tube. And sign of biodegradation of HAP and CF was recognized.

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EXPERIENCE WITH COMPLIANT POLYURETHANE AND POLYURETHANE/FIBRIN MICROVASCULAR PROSTHESES

Compliance is viewed as a critical factor for the success of microvascular prostheses. In the present study, biodegradable, porous, distensible grafts were compared to partially resorbable, porous, distensible grafts under the assumption that a bioresorbable compound (e.g. fibrin) could stimulate the progressive replacement of the graft material by the cellular elements of a normal artery. Porous tubes (1.5 mm ID, 2.0 mm OD) were fabricated by spraying either a polyurethane solution (PU) or a 1:1 blend of polyurethane and human fibrin (PU-F) over a rotating mandrel according to a phase inversion technique. The prostheses, 1.5 cm long, were inserted in the rat infrarenal aortic position using microsurgical techniques. All 6 PU and PU-F grafts appeared functional and pulsating at the time of implantation. Upon retrieval 4 weeks later, 5 out of 6 PU grafts were patent and free of intraluminal clot and one was occluded. Three out of 6 PU-F grafts were patent, but 2 of those were aneurysmal and partially thrombosed. The internal tissue capsule was 20 μ m thick, well organized and consisted of layers of mainly longitudinally oriented smooth muscle-like cells with substantial elastin deposition. A lining of endothelial-like cells covered most of the luminal surface. PU-F grafts showed significant resorption of the graft material with marked tissue invasion. However, the regenerated arterial wall was not as well organized and differentiated as in the PU grafts. In the rat model, porous, distensible micro-prostheses allow a high patency rate with the development of a thin, well organized internal capsule with significant elastin deposition. Blending the elastomer with fibrin leads to greater tissue invasion but poorer organization and patency. An optimal balance between prosthesis degradation and tissue replacement remains to be identified.

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EFFECTS OF SHEAR INDUCED RBC DAMAGE ON THROMBOSIS

This in-vitro study investigates low-stress shear-induced release of ADP from RBC as well as RBCs' mass transport effect on thrombosis through potentiation of platelet aggregation (PAG) and adhesion (PAD) to artificial surfaces. Anticoagulated samples of whole blood, suspensions of RBC in platelet (P1) free plasma or Tyrode albumin solution, and suspensions of P1 and RBC ghosts are exposed to shear rates ($\dot{\gamma}$) to 5680 s^{-1} . Resulting shear induced damage is characterized by single P1 loss, hemoglobin release, ADP and total phospholipid release, size distribution of P1/P1 aggregates adhered to the viscometer surfaces, surface material used and surface-to-volume ratio. The effects of thrombin on (PAG) and (PAD) are eliminated by pretreating some samples with hirudin.

Results obtained thus far show that: 1) application of shear results in the highest rate of single P1 loss in the $\dot{\gamma}$ range to 720 s^{-1} ; 2) the amount of shear-induced ADP release is in a bulk concentration range where P1 aggregation can be potentiated, suggesting that surface concentration of ADP can be considerably greater assuming P1 and RBC damage is a surface initiated phenomenon; and 3) pretreating the blood samples with hirudin (up to 4 U/ml) has little effect (less than 5%) on shear-induced single P1 loss, however a hirudin concentration of 8 U/ml increases P1 sensitivity to shear; also the hirudin pretreated samples show less shear-induced ADP release compared to those without hirudin.

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PERFORMANCE OF BLOOD COAGULATION ASSAYS:

NEUTRALIZATION OF HEPARIN IN TEST SAMPLES

Heparin in citrated blood samples inhibits specific reactions in the coagulation cascade and interferes with coagulation assays. Protamine neutralization of heparin is impractical because an excess of protamine is undesirable. Other antiheparin agents are known to bind coagulation factors. To resolve the problem of heparin neutralization, we propose a new method. Polylysine, an anionic polymer, is cross-linked to agarose beads under appropriate conditions. After extensive washing to remove free polylysine, the beads are treated with albumin, washed, and sterile-stored in saline. Aliquots of beads are added to the citrated blood sample and gently mixed by inverting the collection tube 10 times. The blood is centrifuged, removing the beads, and the collected plasma is heparin free. Results of a comparative activated partial thromboplastin time (APTT) and prothrombin time (PT) tests are given in the following table and illustrate that treatment of blood with polylysine-agarose beads does not affect the coagulation tests while it effectively removes heparin.

Test medium	Heparin added U/ml	APTT(Sec)	PT(Sec)
Plasma (P)	0	37*	12*
P + PLL-agarose	0	36	12
P + PLL-agarose	1	37	11
P + PLL-agarose	5	37	12

*Means of 10 separate experiments
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