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2009-01-01

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Recommended Citation

Wyley, M., McGuinness, G., Gavin, G.: Ultrasonic Angioplasty: Assessing the risk of arterial perforation. 8th annual conference UK Society for Biomaterials, Jordanstown, Northern Ireland, 2009. doi:10.21427/7m9h-nh04

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Ultrasonic Angioplasty: Assessing the risk of arterial perforation

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Introduction:

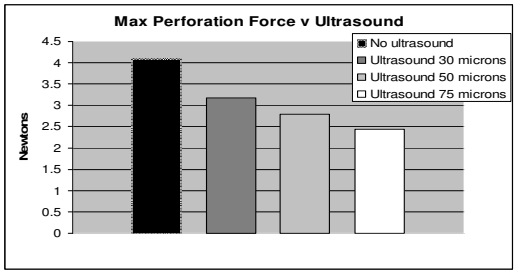
Atherosclerosis is a cardiovascular disease that effects large and medium muscular arteries (such as coronary and iliac) and also large elastic arteries (such as aorta) [1]. It causes thickening of the arterial wall and over time can result in a completely blocked artery or chronic total occlusion (CTO). While the majority of atherosclerotic lesions can be attempted by typical Percutaneous Transluminal Coronary Angioplasty (PTCA) such as balloon and stent implantation, calcified CTOs are often problematic as they do not lend themselves to be accessed by the guidewire which is required to implant the balloon and stent. Excessive guidewire pushing force may result in arterial perforation with CTOs often requiring invasive by-pass surgery. An alternative method proposes the use of low frequency high power ultrasound transmitted through wire waveguides for the removal of the calcified material from advanced atherosclerotic lesions. This type of energy manifests itself as a mechanical vibration at the distal tip of the wave guide with amplitudes of up to 100 microns and frequencies ranging between 20-45 kHz commonly reported. The ultrasound acts to disrupt calcified diseased tissue by means of direct contact ablation, cavitation, acoustic steaming and other pressure wave components while the elastic tissue remains largely unaffected [2]. In this study the effects of this form of ultrasound on healthy arterial tissue (porcine aorta) is examined. Experiments were carried out to determine the force required to perforate healthy porcine arterial tissue both with and without ultrasound at various distal tip displacements.

Material and Methods

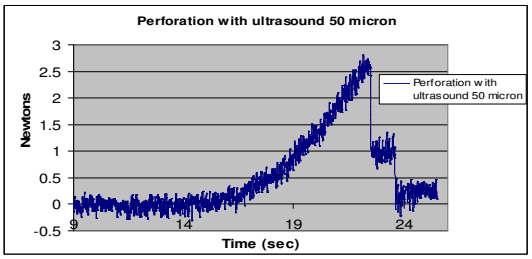
A device was developed that could transmit ultrasound through 1mm NiTi wire waveguides at 22.5 KHz similar to those in surgical use. The 1mm vibrating wire is cut to an anti-resonant length of 146mm and can deliver peak-to-peak vibrations up to 80 microns according to previous studies by Gavin *et al* [3]. A cantilever beam was designed to hold the tissue samples and read force by means of a half-bridge temperature compensation strain gauge arrangement. This could then be placed in a thermostatic bath of water approximately 300x150x200 mm in dimensions. Approximately 150mm of porcine aorta was harvested less than 24 hours after death. The samples were cut and placed securely in the cantilever submersed in a thermostatic tank at 37^{o}C. Tests were carried out to determine the force required for arterial perforation by the wire wave guides both with and without ultrasound at various displacements (32, 50 and 80 microns p-p). The device was advanced vertically penetrating samples at a constant federate of 0.5mm/sec. The various instruments were controlled by LabView (SC068 DAC hardware).

Results and Discussion:

At the constant feed-rate the reduction in maximum force required to perforate the artery wall with ultrasound and increasing wire tip displacements can be seen below (Figure 1). The tests revealed a distinct two-stage penetration.



[Figure 1, Max perforation Force vs ultrasonic wire tip displacements peak-to-peak. Freq = 22.5 kHz]



[Figure 2, Force V Time for tissue penetration with ultrasound at 50 micron distal wire tip displacement. Feed-rate = 0.5mm/min]

Conclusion:

The two-stage penetration recorded may be associated with the composition of the arterial wall, namely the two structural layers of varying elasticity (intima- media and adventitia). It has been reported that the intima-media region can sustain trauma while arterial integrity is still maintained [4]. It is therefore important to define the penetration force for both layers to establish safety thresholds both with and without ultrasonic energy.

References:

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