Perforation of Arterial Tissue Using Kilohertz Frequency Ultrasound Delivered via Wire Waveguides

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PERFORATION OF ARTERIAL TISSUE USING KILOHERTZ FREQUENCY ULTRASOUND DELIVERED VIA WIRE WAVEGUIDES

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INTRODUCTION

An emerging technology proposes the use of low frequency-high power ultrasound transmitted via wire waveguides for the disruption and ablation of atherosclerotic lesions, more specifically advanced fibrous or calcified plaques such as chronic total occlusions (CTO). This energy delivery selectively ablates rigid diseased tissue by means of direct mechanical contact, cavitational and other forces generated by the intense dynamic pressure fields generated.

The first clinical device using this energy delivery was granted FDA approval in 2007 [1] for the ablation of CTOs and most research to date has focused on ablation and disruption of hard, fibrous or calcified tissues [2]. This work, however, investigates the affects this energy delivery has on the perforation of soft healthy tissue (porcine aorta).

MATERIALS AND METHODS

An ultrasonic apparatus has been developed with operational characteristics similar to clinical devices reported in the literature i.e. frequency of operation (22.5kHz) and distal-tip ultrasonic amplitudes of vibration (~15-50µm). This apparatus delivers ultrasound via 1mm nitinol wire waveguides (132mm in length) with flat distal tips.

An experimental test rig was developed to perform controlled tests (ultrasonic power delivery and feedrates) on tissue samples in a thermostatic tank (37°C). Perforation force measurement was achieved by means of a strain gauge arrangement on a cantilever tissue holder.

A miniature hydrophone was also incorporated for the detection of cavitation by analysing the acoustic spectrum while the device was activated. Sub, super and ultra harmonics of the fundamental are all considered indicative of stable cavitation, whereas an increase in the broadband noise, in regions absent of significant harmonics, are indicative of inertial cavitation [3].

Porcine aorta was exhumed, stored in saline and tested less than 24 hours after death. Connective tissue was removed and samples (10x20mm) were cut from the descending aorta. Wires were advanced towards the tissue at a constant feedrate of 38 mm/min until perforation.

RESULTS

As shown in Figure 1, an increase in distal tip amplitudes of vibration reduced the perforation force. It was found that stable cavitation occurred at all power settings (> 15µm). At the high power displacement amplitude setting of 34.3µm the perforation force was 1.2N when compared with 5.5N with no ultrasonic activation. The inertial cavitation threshold was crossed at distal-tip amplitudes of vibration greater than 30µm. However, no significant decrease in perforation force was evident in the inertial cavitation region. At the macro level, the tissue appears to fail in a similar manner for all distal-tip amplitudes of vibration.

DISCUSSION

Perforation force of soft arterial tissue does not appear to be significantly effected by the onset of inertial cavitation. Further histological examination may be required to determine residual tissue damage from cavitation. Additional studies are needed to determine to what extent tissue is ablated, cut or removed at various power levels. It is suggested, however, that tissue removal using this energy on soft tissue is minimal when compared to that of hard brittle tissue ablation.

REFERENCES