Intrasac Pressure Waveforms After Endovascular Aneurysm Repair (EVAR) are a Reliable Marker of Type I Endoleaks, but not Type II or Combined Types: An Experimental Study

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Purpose. To ascertain the nature of the pressure wave transmitted through aneurysm thrombus and the changes produced after endovascular repair and the development of type I and II endoleaks.

Methods. A 25 mm Talent endovascular graft was deployed in a latex model of an abdominal aortic aneurysm, which was incorporated in a pulsatile flow unit. The graft was surrounded by thrombus analogue to simulate conditions in vivo.

Pressure waveforms in the sac were captured over 5 s at 1000 Hz in these settings: (i) no endoleaks (baseline), after introduction of (ii) type I (iii) type II and (iv) combined type I and II endoleaks. The arterial blood pressure settings used were 140/100 and 130/90 mmHg, denoted the high and low settings, respectively. ANOVA in Minitab 13 was applied for statistical analysis.

Results. Pulsatile waveforms were transmitted through the thrombus. Intrasac pressure after stent-grafting reduced to 110/107, 99/96 mmHg (p < 0.001) (high, low settings, respectively). Introduction of a type I endoleak caused this to rise to 120/112, 115/107 mmHg (p < 0.001, vs. baseline); after producing a type II endoleak these were 101/98, 91/88 mmHg (p < 0.001, vs. baseline). A combined type I and II endoleak produced intrasac pressures identical to that of a type I endoleak. Waveforms obtained following type II endoleak simulation resemble the baseline waveform in an attenuated form. Intrasac pressures are, therefore, a reliable marker for type I, but not a type II endoleak. In the case of a combined endoleak, the type I endoleak waveform effectively masks that of the type II. Intrasac thrombus faithfully transmits intrasac pressures.

Conclusions. Intrasac pressure waveforms following EVAR are easily defined following a type I endoleak. Waveforms obtained following type II endoleak simulation resemble the baseline waveform in an attenuated form. Intrasac pressures are, therefore, a reliable marker for type I, but not a type II endoleak. In the case of a combined endoleak, the type I endoleak waveform effectively masks that of the type II. Intrasac thrombus faithfully transmits intrasac pressures.

Keywords: Pressure waveforms; Thrombus; Negative interference; Sac decompression.

Introduction

Monitoring of intrasac pressure (ISP) has been proposed as a tool in assessing success of endovascular aneurysm repair (EVAR).1 A lowering of ISP is reflected by a reduction in aneurysm wall stress, which in turn leads to reduction of aneurysm size that occurs following successful EVAR.2 This has been measured by direct puncture of the aneurysm itself and, therefore, represents a potentially traumatic, invasive procedure.1 This paper seeks to identify the wave patterns and pressures produced by experimental simulation of both successful EVAR, and also failed intervention represented in by type I and II endoleakage. No data exists as to the exact nature and significance of pressure waves transmitted through aneurysm thrombus in relation to each of the settings described above.

Methods

Pressure readings in the high and low arterial blood pressure (ABP) settings (140/100 and 130/90 mmHg, respectively) were taken before stent-grafting of the life-like AAA model, in the absence of thrombus analogue.3 In brief, the model was derived from a 3D computerised tomographic reconstruction of a real non-axisymmetric AAA and has a Young’s modulus of 5.151872 Nmm⁻². The AAA model was incorporated into a pulsatile flow unit for simulation of the cardiac cycle at 70 bpm, which was generated from a pulse generator (Fig. 1).4 A solution of glycerol in water (55:45 v/v, density 1172 kg/m³, viscosity 15.53
centipoise at 25 °C) was used as a blood analogue. The Reynolds’s number (Re) for the PFU exceeded 4000, confirming the flow to be turbulent, as expected in large arteries. The arterial pressure waves (Fig. 2) generated in the system was monitored through a side channel tapped flush into the PFU just above the AAA, while ISP was monitored via a 12 G cannula mounted flush on the AAA wall at the level of the maximum anteroposterior diameter to avoid flow distortion artefacts, ensuring it did not protrude into the thrombus analogue. It was inserted before addition of thrombus to avoid false elevations in the pressure readings. The side channel and the cannula were connected to a pressure transducer (MX 960 LogiCal, Medtronic/World Medical, Sunrise, FL, USA). This was displayed on a standard monitor (Hewlett Packard model HP78353A). The transducer readings on the monitor were calibrated using a mercury sphygmomanometer. The waveform was outputted to a computer using the Wave View platform (Wave View for DOS 1.16(1994), multiboard (3), Eagle Appliances Ltd, UK), which sampled the input over 5 s at a sampling rate of 1000 Hz in volts. This was saved as a text file (.txt) and then converted to a Microsoft Excel numerical and graphic (.xls) file. A conversion equation to mmHg was obtainable using logistic regression in SigmaPlot for Windows.

A 25 mm Talent bifurcated stent-graft was then deployed in the AAA model. The graft was water-proofed by brushing with Tivodex 60 (Evode Ltd, Staffs, UK), a solvent based latex-containing adhesive, which evaporates to leave a fine, membranous latex residue within the warp and weft of the graft. This was leak tested and confirmed to have no porosity endoleak. The aneurysm sac was filled with gelatine solution (Applewolds Jelly, Kerry Food Service, Bucks, UK), which solidified by cooling the model to 12 °C to produce an intrasac thrombus analogue. 

Introduction of a type I endoleak increased the reading to 120/112/115 mmHg (1.254/1.154/1.187 V, SD 0.025) in the high setting, and to 115/107/110 mmHg (1.22/1.11/1.146 V, SD 0.025) in the low setting (p<0.001, relative to the respective baseline values). ISP was 101/98/99 (1.046/0.990/1.009V, SD 0.012) and 91/88/89 mmHg (0.958/0.902/0.920V, SD 0.012) when a type II endoleak was introduced, a significant reduction compared to the respective baselines (p<0.001) (Fig. 4). When a combined type I and II endoleak was introduced, ISPs were 120/112 and

Results

Baseline ISPs (SP/DP/MP, also shown here as the equivalent in volts) of 110/107/108 (1.054/0.995/1.014V, SD 0.011) and 99/96/97 mmHg (0.949/0.863/0.891V, SD 0.012) were obtained after stent-grafting of the AAA model, which denoted a significant drop in ISP relative to ABP (p<0.001). Introduction of a type I endoleak increased the reading to 120/112/115 mmHg (1.254/1.154/1.187 V, SD 0.025) in the high setting, and to 115/107/110 mmHg (1.22/1.11/1.146 V, SD 0.025) in the low setting (p<0.001, relative to the respective baseline values). ISP was 101/98/99 (1.046/0.990/1.009V, SD 0.012) and 91/88/89 mmHg (0.958/0.902/0.920V, SD 0.012) when a type II endoleak was introduced, a significant reduction compared to the respective baselines (p<0.001) (Fig. 4). When a combined type I and II endoleak was introduced, ISPs were 120/112 and
115/107 mmHg for the high and low-pressure settings, mimicking the waveforms noted in trace 1 and 2 (Fig. 4). The waveforms were all broad monophasic in nature, with a rough tabletop appearance to the baseline waveforms and those obtained due to a type 2 endoleak (Table 1).

**Discussion**

The baseline readings suggest that the arterial pressure wave is highly damped by the endograft, and this in turn is an indicator of how the graft itself may reduce wall stress, even in the presence
of thrombus. Our readings are, however, higher than those obtained by Sonesson et al.;1 this may relate to the aneurysm shrinkage that occurred following EVAR. In addition, aneurysm volume (205 ml in this case) is large compared to the volume of the endograft (20 ml within the aneurysm itself). The pressures noted may possibly be due to the interaction of a relatively large volume of thrombus (185 ml) with the sac wall. Our readings may, therefore, indicate ISP changes following early development of endoleaks. The pressures in this study were arbitrarily selected as representative of the pressures that might occur in the age group that would suffer aneurysmal disease. Higher pressures were limited by the apparatus, which only provides gravity-assisted, rather than actively pumped flow. However, the flow characteristic as defined by the Re is sufficient to generate pressures common in the human age group that AAAs present in.

Though the ISPs after endografting do not seem much lower numerically than the ABP, they do represent pressures that do not normally lead to rupture and more importantly may reduce wall stress significantly, though this was not a subject of this study.

ISP was lower than ABP when a type I endoleak was induced, which may be due to the resistance provided by the thrombus itself, or the calibre of the endoleak. A complete disappearance of the pressure waveform due to the presence of intrasac thrombus, as suggested by Schurink et al., was not noted.6 This in fact conforms with their later paper, which suggests that thrombus does not reduce pressure transmission.

![Fig. 4. Intrasac pressure waveforms (in volts) with type I, type II and no endoleaks. These are numbered: (1) type I endoleak, ABP 140/100 mmHg (2) type I endoleak, ABP 130/90 mmHg (3) no endoleak, ABP 140/100 mmHg (4) no endoleak, ABP 130/90 mmHg (5) type II endoleak, ABP 140/100 mmHg (6) type II endoleak, ABP 130/90 mmHg.](image-url)
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to the aneurysm wall. This could possibly be better examined using differential pressure wave values against the thrombus, which is probably the effect of using endoleaks of varying sizes. However, given that a rise in ISP parallels that of the ABP, it is possible that uncontrolled hypertension in the background of a type I endoleak may contribute to further rises in ISP with the risk of rupture. The presence of a type I endoleak seems to counteract any pressure wave generated from the type II endoleak, presumably due to negative interference between wave fronts. In addition, there is no outflow, i.e. it is a type IA endoleak, which may contribute to the higher pressures noted. In the case of a type II endoleak (type IIB, in this case), the same phenomenon, or even decompression via the collateral leaks, due to an ‘open valve’ effect might also account for the waveform that resembles an attenuated version of the baseline. This may form the physical basis for the less obvious rise of pressure as noted for a type I endoleak, resulting in inconsistencies in management approaches to this type of endoleak. We accept that the setting for the type II endoleak, though positionally correct, is somewhat arbitrary in terms of pressure characteristics and may not truly resemble the in vivo situation. This could be further studied using data from in vivo monitoring of arterial pressure within back-bleeding lumbar of inferior mesenteric arteries. Arterial pressure wave damping has been noted in other studies, which have also shown a reduction of ISP with a type II endoleak. This would imply that the pressure generated from a type II endoleak is actually quite low. It must also be considered that the diameter of the endoleak channels, and the difference in pressurisation alone may affect the pressures irrespective of the presence of thrombus.

It has been suggested that ISP monitoring may predict the success or failure of EVAR. The heterogenous nature of thrombus implies that the pressure wave is also transmitted in a variable fashion. We have chosen to measure pressure in only one location in the sac because of the homogenous nature of the gelatine used in such an idealised system, which would result in pressure transmission in accordance to Pascal’s law. It has also been suggested that pressures may vary positionally in the aneurysm sac. However, the authors do not take into the account the possibility of Gore-tex patches and suture lines used in making the aneurysm model contributing to the stress differential.

Though it is possible that the method used may produce artefacts due to the use of a cannula, the pressures obtained in this series are near systemic, which conforms to results from other studies that also demonstrate that endoleaks transmit pulsatile pressure. We, therefore, feel that the readings obtained are an accurate representation of the pulse pressure wave, and also validate the use of a latex coating in maintaining the diaphragm-like properties of the stent-graft.

**Conclusion**

ISPs, and more importantly, the pressure waveforms can accurately reveal the presence of type I endoleak after EVAR. ISP measurements cannot reveal the presence of a pure type II endoleak, or one such in the presence of a concurrent type I endoleak due to the masking effect of negative interference. The endograft itself possibly produces its therapeutic effects by mechanisms that involve damping of the arterial pressure wave through the aneurysm thrombus. Changes in ISPs parallel changes in systemic ABP and, therefore, may help guide treatment decisions especially in patients undergoing EVAR who develop uncontrolled hypertension, as the damping effect of the graft is thereby lost. However, if only a type I endoleak is accurately detectable, invasive procedures as such ISP monitoring may not be entirely justifiable.

**References**


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