Aneurysmal Hypertension and its Relationship to Sac Thrombus: A Semi-qualitative Analysis by Experimental Fluid Mechanics

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Objectives. To ascertain the effect of aneurysm thrombus and luminal diameter on arterial blood pressure within the abdominal aortic aneurysm lumen and at the sac wall.

Methods. A life-like abdominal aortic aneurysm was incorporated in a pulsatile flow unit, using systemic blood pressure settings of 140/100 mmHg and 130/90 mmHg (denoted the high and low settings, respectively). Aneurysm sac pressure was measured in the absence of thrombus within the sac. This was repeated after a thrombus analogue (gelatine) was introduced into the aneurysm model in an asymmetric fashion. Luminal and sac wall pressures were compared to the systemic pressure, and to each other, in both blood pressure settings. Statistical analysis was performed using ANOVA in Minitab 13.

Results. In the empty sac, the luminal and sac wall pressures were identical to the systemic pressures at the high and low settings. After introduction of thrombus, pressure was transmitted in a monophasic pulsatile fashion, measuring 166/142/151 mmHg (SP/DP/MP) at the sac wall, while the corresponding intraluminal pressure was 164/136/145 mmHg (p < 0.001, high setting). By contrast, in the low setting, these readings were 157/133/141 (sac wall) and 160/128/138 mmHg (lumen; p < 0.001). The sac wall pressures were significantly higher than the luminal pressures for both high and low settings (p < 0.001).

Conclusions. Thrombus has a significant effect on the intraaneurysmal lumen itself and causes localised hypertension with high intraluminal pressures. The differences between the sac wall/luminal pressures may affect regional aneurysm wall biomechanics, but needs further study.

Key Words: Aneurysm thrombus; Aneurysmal hypertension; Pascal’s Law; Bernoulli’s principle.

Introduction

The relationship of AAA thrombus to aneurysm wall biomechanics has been the subject of several studies.1,2 It has been suggested that aneurysm thrombus transmits pulsatile pressure.3 However, no data exists in the current literature as to the significance of the luminal diameter, or the nature of the pressure waves transmitted through the blood within the lumen of the aneurysm, in relation to the presence of aneurysm thrombus.

Abbreviations: SP, systolic pressure; DP, diastolic pressure; MP, mean pressure; PFU, pulsatile flow unit; AAA, abdominal aortic aneurysm; EVAR, endovascular aneurysm repair; USS, ultrasound scan.

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maximum anteroposterior diameter of the model via a 12 G cannula flush on the posterior and anterior aspect of the AAA wall, respectively, to avoid flow distortion artefacts. The cannulae were placed before addition of thrombus analogue to minimise any artificial increase 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 and then converted to a Microsoft Excel numerical and graphic file. A conversion equation to mmHg was obtainable using logistic regression in SigmaPlot for Windows.

The aneurysm sac was filled anteriorly with gelatine solution (Applefords jelly, Kerry Food Service, Bucks., UK), which solidified by cooling the model to 12 °C to produce an aneurysm thrombus analogue located asymmetrically within the aneurysm sac. This was coated with a membrane of Tivodex 60 (Evode Ltd, Staffs, UK), a solvent based adhesive that evaporates to leave a latex membrane behind, to prevent it from washing out with the flow. This was re-incorporated into the PFU, and intrasac pressure readings in two arterial blood pressure settings (140/100 and 130/90 mmHg, denoted the high and low settings, respectively) in this scenario were taken. The pressure obtained in the lumen and at the sac wall was compared to the systemic pressure at the setting. The sac wall pressure was compared to the intraluminal pressure. Statistical analysis of the pressure readings (derived from the data points on the pressure curves, including the SP and DP, in volts) was performed using ANOVA in Minitab 13 (Table 1).

**Results**

The luminal and sac wall pressures in the empty sac were identical to the systemic pressures at the high and low settings (140/100/113 and 130/90/103 mmHg, respectively, (SP/DP/MP)). In the low setting, intraluminal pressure was 160/128/138 mmHg whilst the pressure measured at the sac wall was 157/133/141 mmHg ($p < 0.001$) (Fig. 2(b) and (c)). The aneurysm sac wall pressure was also significantly higher than the intraluminal pressure at both blood pressure settings ($p < 0.001$) (Fig. 3(a)). This is reflected in the mean pressures. After addition of thrombus analogue, the pressure waves were blunted, losing their triphasic appearance.

**Discussion**

The arterial pressure wave contributes to wall stress within the AAA sac. It is therefore important to study the role of thrombus in modifying the pressure. The heterogenous nature of thrombus suggests that the pressure wave is transmitted in a variable fashion. The homogenous nature of the thrombus analogue used in our ‘idealised’ system would result in pressure transmission in accordance to Pascal’s law and is the basis for pressure measurement in one position. A
Fig. 2. (a) PFU waveforms. A negative excursion is noted due to a water hammer effect as a result of elastic recoil from the latex model. (b) Pressure waveforms transmitted via thrombus. (c) Pressure waveforms within the AAA model lumen.
series where it was suggested that thrombus lowers wall stress did not look at the aneurysm size that might warrant EVAR. It has been suggested that stresses, and therefore the pressures which generate the stresses, may vary positionally in the aneurysm sac. However, the authors do not take into account the possibility of Gore-tex patches and suture lines used in the manufacture of their aneurysm model, which may have contributed to the stress differential. In addition, the baseline waveform obtained using fluid only within the AAA sac is identical to the pressure obtained from the PFU above the AAA neck.

The pressures obtained may be explained qualitatively. The model exhibits complex geometry due to the asymmetrical shape of the aneurysm. The presence of aneurysm thrombus may be causally related to the varying pressures and stresses that have been noted in vivo. However, an idealised situation maybe considered where the AAA might be thought of as a diverging tube with a circular cross section for all ‘x’ described (Fig. 3(b)).

The cross sectional area at ‘x’ is given by:

$A_x = \pi (r_x)^2$  \hspace{1cm} (1)

As volume flow rate ($Q_t$) is a function of time, the mean velocity ($u$) at any given section is denoted by:

$u = \frac{Q_t}{\pi (r_x)^2}$  \hspace{1cm} (2)

This clearly indicates that blood flow velocity is reduced within an arterial channel as it dilates. If it is assumed that Bernoulli’s equation applies approximately along the centre streamline a form of the steady flow equation can be derived (Appendix A).
Multiplying both sides of Eq. (A3) by \(g\), we obtain:

\[
\frac{P}{\rho} + \frac{u^2}{2} = c_2
\]

(3)

As \(\rho\) is a constant for the fluid medium, i.e. blood it therefore follows, from Eq. (3) that as velocity falls pressure must rise. This may be considered in the context of Pillary et al.'s findings, which demonstrated that aneurysm size was inversely related to the volume of thrombus, and also that thrombus was thicker anteriorly. This may, in terms of fluid mechanics, therefore form a basis for the risk of rupture as aneurysmal size, and therefore luminal size and pressure increase. In addition, these findings contradict the preconception that thrombus growth is related to eddy currents and secondary velocity patterns. As we have used only a fixed volume of thrombus volume, the interaction of the flow and thrombus volume is beyond the scope of this experimental setting. However, this will occur only if the diameter of the channel within the AAA is larger than that at the neck, and becomes invalid if the lumen becomes narrowed by thrombus or calcification. The studies of Stenbaek and Wolf et al. suggest a higher risk of rupture in small AAAs as thrombus volume increases, but they have not correlated the thrombus volume with the luminal calibre and intraluminal pressures, and excluded the larger aneurysms that Pillary et al. studied.13,14

There is also generation of turbulence following the abrupt increase in aortic calibre (Fig. 3(b)). This gives rise to eddy currents with loss of kinetic energy (KE, also termed the exit loss in this case) from the blood stream, in accordance with the Borda–Carnot equation.15 We therefore hypothesise that this KE is transmitted through that part of thrombus in contact with the blood stream and therefore all or part of the exit loss manifests as pressure waves with a higher amplitude at the sac wall, whilst the energy loss from the lumen results in a pressure that is comparatively lower than that obtained at the sac wall.

Our findings correlate with other studies, which have demonstrated higher intrathrombic sac pressures in correlation to increased luminal pressures.16 However, Hans et al. demonstrated lower pressure values overall—this is due to the fact that the AAAs had been clamped proximally and distally, and therefore excluded from the systemic pressures. Reduction of the intrathrombic pressure after further clamping of the inferior mesenteric artery supports our hypothesis that KE absorbed from arterial flow causes pressure waves of higher amplitude to be transmitted through thrombus.
Conclusion

Aneurysm thrombus transmits pressure in a pulsatile fashion and may relate to a localised rise in aneurysm intraluminal blood pressure, especially if the aneurysmal lumen continues to be wide. This may add to the risk of rupture, particularly in those aneurysms increasing in size, and needs to be studied in greater detail both in vivo and in vitro.

Appendix

Derivations of the steady flow equation as applied to the flow model

\[
\frac{P}{\rho g} + \frac{u^2}{2g} + z = c \text{ (constant)} \tag{A1}
\]

Then,

\[
\frac{P}{\rho g} + \frac{u^2}{2g} = c - z \tag{A2}
\]

\[
\frac{P}{\rho g} + \frac{u^2}{2g} = c_1 \tag{A3}
\]

References


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