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The electrical impedance of pulsatile blood flowing through rigid tubes: an experimental investigation

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Abstract- Small fluctuations present in an Impedance Cardiogram are often dismissed as noise, but may be due to unknown physiological origins. One such origin suggested in literature is the impedance variation induced by changes in red blood cell orientation during pulsatile blood flow. This study investigated the relationship between the impedance, velocity and acceleration of blood as it pulses during the cardiac cycle. This was achieved experimentally by pumping blood through rigid tubes in a mock circulatory system while measuring the impedance and velocity of the blood. Analysis of collected data confirms that impedance responds to changes in both velocity and acceleration. During acceleration, impedance and velocity are linearly related. However, during deceleration, it was found that the relationship between impedance and velocity is non linear. As velocity increases, the relationship becomes linear with a reducing slope. This indicates that for the same change in acceleration at low velocities, the impedance response is significantly larger than at higher velocities. Experimental data demonstrating these trends is presented for varied pulse rates (20 – 100 beats per minute), stroke volumes (20 – 60 ml) and systolic/diastolic ratios (50/50 - 30/70). These results demonstrate that both the acceleration and velocity are important factors in determining the impedance of pulsing blood. Knowledge of these relationships may prove valuable in identifying the physiological origins of presently unknown fluctuations in an Impedance Cardiogram.

Keywords— Bioimpedance, Red Blood Cell Orientation, Mathematical Model, Pulsatile Blood Flow.

I. INTRODUCTION

Impedance Cardiography is a non-invasive procedure used to monitor cardiac function. It is based on the measurement of impedance changes within the thorax which are related to blood volume changes. However, there exists some variations in an Impedance Cardiogram (ICG), that are not understood physiologically [1]-[6]. Among other sources, literature suggests these variations may be due to the velocity dependant changes in the resistivity of blood [7]-[9].

These flow dependant resistivity changes are thought to be caused by changes in the alignment of red blood cells (RBCs) in response to changing shear forces [7]. As the shear force increases, the cells experience a higher variation in velocity across their surface. To minimise this shearing stress, they align with the minimum cross sectional area facing the direction of flow.

The dependence of blood resistivity on the velocity of blood implies that the impedance waveform in an ICG may contain additional information relating to blood flow and ultimately heart function. If this is the case, a greater understanding of the relationship between impedance and velocity during pulsatile blood flow is an essential first step to improving Impedance Cardiography techniques.

The varying electrical characteristics of flowing blood have been previously reported in the literature [7]-[17]. Theoretical and experimental investigation of the relationship between the impedance of blood and changing flow parameters has been conducted for constant flow rates. Experimental results also demonstrate that the impedance of pulsatile blood behaves in a manner that is different to that of steady flow. This is predicted to be due to the additional effects of accelerating flow. Therefore, this study investigates the experimental relationship between the impedance, velocity and acceleration for pulsatile blood flowing through rigid tubes. This is important because blood flow through the body is pulsatile in nature.

II. METHODS

A. Experimental Measurements

Bovine blood has been used in this study as a model for human blood. At low shear rates, the viscosity of bovine blood (6.6 mPa.s) is considerably less than human blood (33.5 mPa.s) [18]. This difference is thought to be due to the higher degree of aggregation of RBCs to form cylindrical rouleaux occurring in human blood. At higher shear rates, the rouleaux formations are broken up by shear stresses and the viscosity of both human and bovine blood are of similar magnitude (4.8 mPa.s and 6 mPa.s respectively [18]).

Reference [12] suggests that the presence of rouleaux in human blood may alter the impedance in Poiseuille flow at very low shear rates. At low shear rates, the velocity profile of Poiseuille blood flow through venules is flattened rather than parabolic [19]. This effect is less noticeable at high shear rates when rouleaux are disaggregated. As the present study investigates the impedance of pulsatile blood and the effect of pulsatile flow is also to flatten the velocity profile, it is expected that the lack of rouleaux formation in bovine blood will have a negligible effect on measured impedance.

Eight litres of bovine blood were pumped through rigid tubes from one reservoir to another, as shown in figure 1. The blood was pumped through silicon tubing using the Harvard Apparatus Pulsatile Blood Pump for Large Animals and Hemodynamic Studies. The impedance was measured in a Perspex rigid tube (12.7 mm diameter) with 4 platinum electrodes using an Impedimed ImpTM SFB7. This device was modified to make rapid measurements at 1 ms intervals for a period of up to 30 seconds at a single current frequency. Flow rate was measured by a Transonic ME13 PXN Inline Sensor and TS410 Tubing Flow meter. The temperature of the blood was measured.

For this study, the pump parameters were representative of the human heart. The impedance and velocity were measured for pulse rates from 20-100 beats per minute (bpm), stroke volumes from 20-60 ml and systolic/diastolic ratios from 50/50 to 30/70. The current frequency used was 5 kHz and the measured haematocrit was 45 %.



Fig. 1 Mock Circulatory System Experimental Set Up

B. Data Analysis

For each trial the collected impedance and velocity signals were averaged over a number of pulses to create an ensemble averaged representative pulse for the trial parameters. From this, the derivative of the blood velocity with respect to time was calculated to determine the acceleration.

To aid in analysis, two distinct flow phases were identified and analysed. These were: a) the acceleration phase, defined from the time when the blood velocity begins to rise to the time where the peak of the pulse occurs, and b) the deceleration phase, defined as the time from the peak of the pulse to the time the flow returns to the initial velocity. The remaining flow phase in which the flow fluctuates around zero while impedance is still changing has not been analysed.

III. RESULTS

Figure 2 shows an example of the ensemble averaged experimental results for impedance and velocity of pulsatile bovine blood. The average velocity is represented by the dashed line and the impedance is represented by the dotdashed line. These results share common characteristics with previously published data such as those in [12]. It can be seen that the impedance responds instantaneously to velocity changes during the acceleration phase while a decayed response occurs during the deceleration phase. It can also be seen that the impedance is different for the same velocity, during acceleration and deceleration such as at time (a) and (b) indicated by the vertical dotted lines. Results collected over a range of cardiac parameters such as pulse rate, stroke volume and systolic/diastolic ratio demonstrate similar characteristics.



Fig. 2 Ensemble averaged velocity and impedance of blood over one pulse, ($\pm \sigma$ also shown) h = 45%, d = 12.7mm, pulse rate = 60 bpm, frequency = 5 kHz, ratio = 35/65 (a) t = 0.1 s and (b) = 0.42 s.

Figure 3 displays the ensemble averaged velocity (a) and corresponding impedance (b) for a pulse rate of 20 bpm and varying stroke volume. This figure shows that for velocities below approximately 30 cm.s⁻¹, small pulses in velocity are repeated in impedance waveforms. However, at velocities above this the small pulses in velocity are not repeated in impedance. At the peak of all velocity pulses shown in figure 2, there exists a "double" pulse. This variation is only seen in the impedance waveform for a stroke volume of 20 ml as the peak velocity is low. The remaining impedance waveforms do not respond to these velocity changes.



Fig. 3 Ensemble averaged velocity and impedance of blood over one pulse, h = 45%, d = 12.7mm, pulse rate = 20 bpm, frequency = 5 kHz, ratio = 35/65, varying stroke volume

IV. DISCUSSION

The results demonstrate that the relationship between impedance, velocity and acceleration varies through out the cardiac cycle. Figure 4 shows an example of this relationship for the pulse shown in figure 2. This plot shows explicitly the relationship between impedance and velocity. The acceleration is indicated by a variation in colour according to the colour bar. Figure 4 shows that the relationship between impedance and velocity is difficult to define over the duration of the pulse. It appear to be non linear at low velocities and during deceleration, but is clearly linear during the acceleration phase.

The relationship between impedance and velocity during the acceleration phase has been further investigated by performing a linear regression analysis for each trial. This relationship has been found to be highly linear over all parameters (e.g. $r^2 = 0.99$, for a pulse rate of 60 bpm).

Similar trends have been found in [8]. Stop flow experiments in which the velocity is made to flow in a repeating square waveform demonstrate that impedance responds instantaneously to velocity increases or accelerations.

Closer analysis of the linear regression coefficients shows that the magnitude of the slope of the best fit line decreases as peak velocity increases. This can be seen in figure 5. It shows the linear relationship between the best fit slope and the peak pulse velocity (Pearson's correlation coefficient = 0.92). This relationship demonstrates that changes in velocity have less effect in changing the impedance at higher velocities than they do at low velocities. This is explained by a saturation point that occurs at high velocities when all red blood cells are aligned. Thus any increases in velocity will no longer affect the impedance. Such a saturation point has been previously reported in published literature [8-11, 13-15]. These results for the acceleration phase suggest the relationship between impedance and velocity shows similar trends as that during steady flow.



Fig. 4 Impedance vs. Velocity with Acceleration indicated by the colour map, h = 45%, d = 12.7mm, pulse rate = 60 bpm, frequency = 5 kHz, ratio = 35/65



Fig. 5 Relationship between best fit slope and peak velocity for each trial

During deceleration, the relationship between impedance and velocity is clearly not linear in figure 4. To investigate this relationship, the data for all parameters has been collated and searched for points of the same velocity. For each of these points, the impedance and acceleration of the blood has been recorded. For each velocity (from 0-140 cm.s⁻¹), the impedance is plotted against acceleration. This data (which is not included here due to page limits) shows some general trends which suggest that acceleration plays an important role in determining the impedance. From these plots, it appears that there is no definite relationship between impedance and acceleration at velocities below about 30 cm.s⁻¹. However, as the velocity increases, the relationship becomes more linear with a reducing slope. This indicates that at high velocities, changes in acceleration no longer affect the impedance of flowing blood.

Again, this suggests the presence of a saturation point at which changes in velocity no longer effect the orientation of the red blood cells and thus the impedance. This saturation point also provides an explanation for the observed difference in impedance response to flow variations at different velocities seen in figure 3. Results for the deceleration phase demonstrate that the relationship between impedance and velocity is much more complex than for the acceleration phase and simple steady flows.

V. CONCLUSIONS

Previous investigations on the electrical characteristics of blood have concentrated on constant flow rates and ignored the effect of acceleration. This investigation has demonstrated that acceleration is an important factor in determining the impedance of flowing blood through investigation of experimental relationships between impedance, velocity and acceleration during acceleration and deceleration Impedance responses during acceleration demonstrate characteristics similar to constant flow responses, however during deceleration, the relationship between impedance and velocity changes. These results show that the impedance of blood responds to changes in both the velocity and acceleration of blood and as a result Impedance Cardiography techniques may be able to generate more information relating to blood flow from impedance waveforms.

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References

1. R. P. Patterson (1989) Fundamentals of impedance cardiograhy. Engineering in Medicine and Biology Magazine, IEEE, 8:35-8

- L. Wang and R. Patterson (1995) Multiple sources of the impedance 2 cardiogram based on 3-D finite difference human thorax models. IEEE Trans Biomed Eng 42:141-8
- 3 L. A. H. Critchley (1998) Impedance cardiography: the impact of new technology. Anaesthesia 53:677-684
- R. K. Kauppinen, J. A. Hyttinen, and J. A. Malmivuo (1998) Sensitiv-4. ity distributions of impedance cardiography using band and spot electrodes analyzed by a three-dimensional computer model. Ann Biomed Eng 26:694-702
- 5. F. Skrabal, H. Mayer, E. Hopfgartner, G. Gratze, G. Haitchi, and A. Holler (2005) Multi-site-frequency electromechanocardiography for the prediction of ejection fraction and stroke volume in heart failure. Eur J Heart Fail 7:974-83
- G. Cotter, A. Schachner, L. Sasson, H. Dekel, and Y. Moshkovitz (2006) Impedance cardiography revisited. Physiol Meas, 27:817-27
- K. R. Visser, R. Lamberts, and W. G. Zijlstra (1988) Origin of the impedance cardiogram. VOL 2, 10th Annu. Int. Conf. of the IEEE En-7. gineering in Medicine and Biology Society, pp. 763-65
- K. R. Visser (1989) Electric properties of flowing blood and impedance cardiography. Ann Biomed Eng, 17:463-73
- 9. P. M. de Vries, J. W. Langendijk, and P. M. Kouw (1995) The influence of alternating current frequency on flow related admittance changes of blood: a concept for improvement of impedance cardiography. *Physiol Meas* 16:63-9 10. F. M. Liebman, J. Pearl, and S. Bagno (1962) The electrical conduc-
- tance properties of blood in motion. Phys Med Biol 7:177-94
- 11 F. M. Liebman and S. Bagno (1968) The behaviour of red blood cells in flowing blood which accounts for conductivity changes. Biomed Sci Instrum 4:25-35
- 12. J. W. Dellimore and R. G. Gosling (1975) Change in blood conductivity with flow rate. Med Biol Eng 13:904-13
- K. Sakamoto and H. Kanai (1979) Electrical characteristics of flow-13. ing blood. IEEE Trans Biomed Eng 26:686-95
- K. R. Visser (1992) Electric conductivity of stationary and flowing human blood at low frequencies. Med Biol Eng Comput 30:636-40
- 15. A. E. Hoetink, T. J. C. Faes, K. R. Visser, and R. M. Heethaar (2004) On the flow dependency of the electrical conductivity of blood. IEEE Trans Biomed Eng 51:1251-61
- E. Raaijmakers, J. T. Marcus, H. G. Goovaerts, P. M. J. M. de Vries, 16. Th. J. C. Faes, and R. M. Heethaar (1996) The influence of pulsatile flow on blood resistivity in impedance cardiography. 18th Annu. Int. Conf. of the IEEE Engineering in Medicine and Biology Society, pp 1957-8
- 17. R. A. Peura, B. C. Penney, J. Arcuri, F. A. Anderson Jr, and H. B. Wheeler (1978) Influence of erythrocyte velocity on impedance plethysmographic measurements, Med Biol Eng Comput 16:147-54
- U. Windberger, A Bartholovitsch, R Plasenzotti, K. J. Korak, and G 18. Heinze (2003) Whole blood viscosity, plasma viscosity and erythrocyte aggregation in nine mammalian species: reference values and comparison of data. Exp Physiol 88:431-40
- 19 J. Bishop, P. Nance, A Popel, M. Intaglietta, and P Johnson (2001) Effect of erythrocyte aggregation on velocity profiles through venules. Am J Physiol Heart Circ Physiol 280:H222-H236

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