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P1_8 Turbulence, Atherosclerosis and the Carotid Bruit

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Abstract

This article discusses turbulence in the human carotid artery, its links to narrowing of the artery due to atherosclerosis and the amplitude of the carotid bruit. The Reynolds number at the narrowest part of the artery is found as a function of the degree of stenosis which is then related to the amplitude of the carotid bruit. It is demonstrated that the amplitude of the bruit could be used as an estimate of the degree of stenosis of the artery.

Introduction

Turbulent blood flow in the carotid artery produces vibrations in the surrounding tissue creating noises, detected with a stethoscope, known as the carotid bruit [1]. When considering fluid flow through a tube, the turbulent (chaotic) or laminar (non-chaotic) behaviour of the fluid is commonly characterised using a dimensionless Reynolds number. An increase in the Reynolds number (more turbulent flow) in a section of an artery could indicate that the size of the artery has decreased.

A common cause of stroke is carotid atherosclerosis where the artery is narrowed due to fatty deposits (plaque) [2]. The degree of narrowing, or stenosis, of the artery is the ratio of the size of the plaque to the normal artery diameter [2]. A large stenosis (>70%) is usually connected to a high risk of stroke [2]. The degree of stenosis of the artery can be related to the Reynolds number and a higher Reynolds number (more turbulent flow) manifests itself as a louder bruit [1]. Thus, it is theoretically possible to estimate the degree of stenosis by simply listening to the bruit. This article finds the relationship between the degree of stenosis of the human right internal carotid artery (R-ICA) and the Reynolds number, finally linking this to the amplitude of the detected bruit.

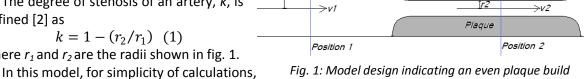
Model and Reynolds Number Calculations

The degree of stenosis of an artery, k, is defined [2] as

 $k = 1 - (r_2/r_1)$ (1)

where r_1 and r_2 are the radii shown in fig. 1.

it is assumed that the blood flows through



Plaque

Fig. 1: Model design indicating an even plaque build up on the artery walls.

straight, rigid arteries and that the blood velocity is constant over time. For a viscous fluid, the velocity of the blood decreases from a maximum at the centre of the artery to a minimum at the walls. This maximum central velocity (shown in fig. 1) is considered in the following calculations. This means that the general Reynolds number [3] is

$$Re = \frac{2rv\rho}{\eta} \quad (2)$$

where *Re* is the Reynolds number at some arbitrary point at which r is the radius of the artery, v is the maximum blood velocity, ρ is the blood density and η is the blood viscosity. Thus *Re* at position 1 in fig.1 would require $r=r_1$ and $v=v_1$ in (2). These values (r_1, v_1) are easily found from clinical data [4, 5], thus it is convenient to relate Re at position 2 in fig.1 to r_1 and v_1 . It is assumed that the volume flow rate (m³s⁻¹) of blood at position 1 is equal to that at 2 (this is the continuity equation for an incompressible fluid flow) so,

$$\pi r_1^2 v_1 = \pi r_2^2 v_2$$
$$v_2 = \frac{r_1^2}{r_2^2} v_1 = \frac{1}{(1-k)^2} v_1 \quad (3)$$

where (1) has been used to write (3) in terms of k and v_1 . Using (1) and (3), the Reynolds number at position 2, Re_2 , can be written in terms of r_1 and v_1 :

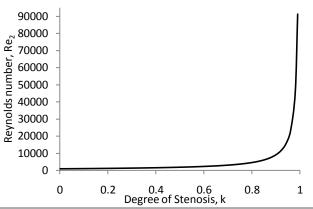
$$Re_{2} = \frac{2r_{2}v_{2}\rho}{\eta} = \frac{2r_{1}v_{1}\rho}{\eta} \cdot \frac{1}{(1-k)}$$
(4)

For the R-ICA, clinical measurements have found r_1 =0.002m [5] and v_1 =0.65m/s [4]. The density of blood, ρ , is 1053kg/m³ and the viscosity of

blood, η , is 3mPa.s [6]. Using these values in (4) and plotting Re_2 for $0 \le k < 1$ gives graph 1.

The Bruit Amplitude and Discussion

A previous study [1] has related the amplitude of the bruit (its loudness) to the Reynolds number of the blood. It was found that there was no bruit for Re<800, a small amplitude bruit for $800 \le Re<1200$, an average amplitude bruit for $1200 \le Re<1800$ and a high amplitude bruit for $Re \ge 1800$. A comparison between these values and data used to plot



graph 1 shows that a high amplitude bruit is heard if k>0.49. A small amplitude bruit would relate to k<0.24.



Interestingly, according to this model, a very low amplitude bruit (Re=912.6) should be heard even at k=0. This could be present in the real system, although perhaps too quiet for the human ear to distinguish it from the background noises of the body and surroundings. Alternatively it could be indicative of flaws in the model, particularly in the assumption that the blood flows through straight, rigid tubes and its velocity is constant over time. This allowed the Reynolds number (2) to be used. In reality, blood flow in the body is pulsatile and the arteries are elastic. The Reynolds number used here is a reasonable approximation to the real situation [3], however some discrepancy between real Reynolds numbers and those found with this model would be expected. The mathematics for pulsatile flow Reynolds numbers is very complex and best implemented through numerical modelling [3] which would be infeasible here. This could provide an interesting avenue for further study. Finally, whilst the assumption that the flow rate at position 1 is the same as at 2 is reasonable for a small k (little difference in artery size), as k continues to increase, the artery's resistance to the flow will increase meaning that the volume flow rate will probably decrease; this would affect the calculated Reynolds number.

Conclusion

It has been shown that since the amplitude of the bruit heard is dependent upon the Reynolds number, it is possible to relate the amplitude of the bruit to the degree of stenosis within an artery. For example, a high amplitude bruit suggests the degree of stenosis is greater than 0.49. With further adaptations to the model such as use of a pulsatile Reynolds number, this concept could be used by doctors to roughly estimate the degree of stenosis in the carotid artery simply by listening. In particular, since the bruit is heard when the degree of stenosis is less than 0.5, it could be a way to catch atherosclerosis early when the patient may not show symptoms.

References

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