Flow Induced
Pressure Fluctuation
In A Model Of A
Contorted Carotid Artery

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Abstract

An experimental investigation into the link between the flow induced pressure fluctuations and atherosclerosis is reported in this thesis. A model of a C-shape tortuous channel was used to simulate a contorted cervical portion of the human internal carotid artery (ICA) and air was used as the flow medium. The experiment was carried out under steady flow conditions and pressure fluctuations at various positions along the wall of the ICA model were measured with a calibrated condenser microphone and a digital voltmeter. Dynamic similarity between air flow in the model and blood flow in the artery was scaled by the Reynolds' number and characteristic frequencies of the pressure fluctuations scaled by the Strouhal number.

A Fast Fourier Transform of the measured pressure fluctuations was performed with the aid of a Strobe Acquisition Unit and the resultant frequency spectra analysed. The root-mean-square voltage of the dominant characteristic frequency of the fluctuating pressure was measured and converted to a fluctuating pressure coefficient. The overall results were presented as a map of pressure distribution at various positions on the model.

A dominant characteristic frequency of 34 Hz, as well as periodic doubling and band-broadening were observed. The dominant characteristic frequency was found to increase with flowrate.

These pressure fluctuations may be attributed to the inherent instability in the separation/reattachment shear layer. High amplitude and high frequency pressure fluctuations were found to occur at the proximity of the bends along the flow path and matched the clinically reported sites of atherosclerosis. It could therefore be inferred that flow induced pressure fluctuations influenced the initiation and perhaps the progression of atherosclerosis.
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CHAPTER 1
INTRODUCTION

1.1 Definition of Atherosclerosis

Atherosclerosis may be defined as the degenerative and reparative changes in the blood vessel walls that are the direct consequence of haemodynamically induced fatigue. The atherosclerosis lesion can be described as a fibrous plaque which consists of a cap of smooth muscle cells covered by a layer of endothelium and a core containing lipid. Up to 25% of the lesion consists mainly of esterified cholesterol which is derived from the blood. The affected artery also exhibits calcification, proteoglycan accumulation, loss of elasticity, cracking, stenosis (narrowing of the arterial lumen), aneurysm (ballooning out of the vessel wall) and diminution of elastic tissue. Proliferation of smooth muscle cells in the intimal layer of the arterial wall is referred to as the intimal thickening.

The accumulation of lipid in the tunica intima, which is covered by an intact layer of endothelium signifies the onset of atheroma. At a subsequent stage, raised plaques become visible on the internal surface of the artery. The endothelium overlying these plaques consists of free fat deposits, foam cells (fat-laden) and fibrous (scar) tissue. Later the endothelium may break down, bringing these elements into direct contact with the blood. The endothelium may then regrow and the plaque may increase in size due to fat deposition, scarring and accumulation of thrombus layers, which may result in thrombosis (i.e. malfunction/failure of organs served by the occluded artery). Pieces of thrombus, called emboli, may also be shed into the artery and lodge in a smaller branch downstream, which can result in gangrene, paralysis or sudden death.

There are apparently two types of atheroma lesion (Fee, 1989), depending on the local haemodynamic forces:

a) Proliferative lesion, which is formed in areas where flow separation, low mean shear stress, oscillating shear stress and high fluctuating pressure occurred.

b) Atrophic (non-proliferative) lesion, which is formed in areas where unidirectional high shear stress occurred. The progression of the atheroma is different at each site. In the early stages of atheroma, the proliferative sites are
subjected to lipid accumulation and the atrophic lesion to calcification. The initiation of atherosclerosis is therefore, strongly affected by haemodynamic factors.

1.2 Objective of the Study

Clinical observations have shown that atherosclerosis, a disease of large and medium size arteries, occurs preferentially in regions where the arteries show branching and sharp curvature. From the fluid dynamical point of view, these are zones where the flow is highly disturbed and exhibits phenomena such as flow separation and flow recirculation as well as low arterial wall shear stress. The localised nature of the disease pattern and the geometry dependent anomalies provide indirect evidence for a role of arterial flow phenomena in atherogenesis. Vascular geometry as a risk factor in atherosclerosis has been well recognized. (Ku et al, 1985; Mark et al, 1989)

The present investigation is aimed at:
(i) Characterising pressure fluctuations inherent at various locations along the wall of a contorted tube
(ii) Assessing the likely effects of these pressure fluctuations on atherogenesis, by comparison with clinical observations in similar geometry \textit{in vivo}.

The cervical portion of the human internal carotid artery (ICA), was chosen as the subject of the study due to its simple flow geometry. In normal human beings, this section of the artery is straight, with no bifurcations and relatively free of atheroma. However, in about 15% of the population, an excessive length of the artery develops; forming tortuosity of varying degrees. The tortuous section is prone to a higher incidence of atherosclerosis; which suggests a relationship between the local blood flow characteristics and atherogenesis.

In this study, several simplifications from the \textit{in vivo} artery were made in the model to emphasize flow features that were due to the arterial geometry alone:

(i) The model has rigid walls.
(ii) The ICA channel in the model was of constant cross section throughout.
(iii) The model was symmetrical about its mid-point.
(iv) The tortuosity was confined to one plane.
(v) Air was used as the flow medium in this experiment, to ease pressure fluctuation measurements.

(vi) The experiment was carried out under steady flow conditions, as opposed to the in vivo pulsatile flow. It was thought that most of the flow features under steady flow were qualitatively similar to those observed under pulsatile conditions. (Fee, 1989)

1.3 Physiology and Relevant Haemodynamics

1.3.1 The Tortuous Internal Carotid Artery and the Model

In the in vivo situation, blood flow enters the ICA from the common carotid artery via the carotid sinus. The bifurcation of the common carotid artery into the external and internal carotids, prior to the carotid sinus, causes flow separation which has been associated with proliferative atheroma.

In this study, an idealised model of the tortuous cervical portion of the human internal carotid artery was used. For simplicity, a 'C'-shaped configuration with the tortuosity lying in the plane of symmetry and symmetrical about the midpoint of the 'C'-bend was chosen. The diameter of the 'C'-shaped channel was 20 mm, compared to 8 mm in vivo. Dynamic similarity was scaled by the Reynolds number:

\[ \text{Re} = \frac{D \cdot U \cdot \rho}{\mu} \]

where: \( D \) = vessel diameter, \( U \) = mean velocity, \( \rho \) = fluid density, \( \mu \) = fluid viscosity.

1.3.2 The Relevance of Hydrodynamics to Haemodynamics

Blood is a suspension of cells in a aqueous solution called plasma. About 5% of these cells are platelets which are responsible for blood clotting. About one sixth of 1% of the cells are the white cells which play a role in the body resistance to infection. The red cells, the erythrocytes, make up about 45% of the blood volume in the average person. The red blood cell is a biconcave disk, with a major diameter of about 8 \( \mu \)m and thickness of about 2 \( \mu \)m. Blood may be regarded as an homogenous Newtonian fluid in vessel larger than about 1 mm in
diameter, with a viscosity of about 0.004 Ns m\(^{-1}\) and a density of about 1005 kg m\(^{-3}\) at shear rates above 100 s\(^{-1}\); as in the larger arteries (McDonald, 1974). Blood rheology (non-Newtonian aspects) has been found to play an important role in proliferative atherosclerosis. (Merrill, 1969)

1.3.3 The Arteries and associated Flow Effects

The arteries in vivo are normally of circular cross section. Arterial walls are made up of three characteristic layers:

a) The tunica intima -- consists of an endothelial monolayer and is in contact with the blood ie. the luminal side of the artery.

b) The tunica media -- comprised of smooth muscle cells and surrounds the tunica intima.

c) The tunica adventitia -- consists of connective tissue and elastin. It is the outermost layer of the arterial wall and merges with surrounding tissue at its outer edge.

In contrast to the model used in this study, the arterial wall is not rigid but may be considered to be restrained longitudinally and the radial dilation of the arterial wall was found to be negligible in larger arteries (McDonald, 1974). Blood flow in the ICA in vivo, is likely to be laminar for most parts but secondary flows may cause turbulent spots at various sites. However, arterial blood flow may be approximated to that in rigid pipes (Fee, 1989).

1.3.4 Atherogenesis

A large number of hypotheses concerning the pathogenesis of atherosclerosis exist (refer to Fee, 1989) but none of these is complete on its own. The 'Unified Haemodynamic Hypothesis' is thus proposed (Steinberg, 1983; Fee, 1989); to account for both the biochemical and haemodynamic effects on the biological nature and focality of atheroma predilection.

1.3.5 Relevant Fluid Dynamics

The laminar flow of Newtonian fluid in smooth straight pipes, known as the Poiseuille flow, describes a fully developed laminar flow and exhibits a parabolic axial velocity profile across the pipe, with maximum velocity at the centre of the tube and the zero velocity at the wall. With the introduction of axial curvature a
more complex flow which is dependent on the balance of inertial and viscous forces, occurs. The maximum axial velocity is found to shift towards the outer wall of a curved pipe and a secondary flow develops. Dean (1927/1928) in his theoretical treatment, recognised the flow dependence (in a curved pipe) on both the ratio of pipe radius to axial curvature ratio \( \delta = a/R \), and the Reynolds number. He combined these parameters into a dimensionless number, known now as the Dean number:

\[
K = 2 \delta^{1/2} \text{Re}
\]

For low curvature ratios and \( K \approx 96 \), the fully-developed secondary flow takes the form of two counter-rotating vortices, axisymmetric about the plane of symmetry. Near the bend entrance, the interior 'core' of fluid continues to move under its inertia across the curved tube after the bending starts and the maximum axial velocity is thus shifted towards the outer bends.

The three consecutive bends in the present model have high curvature ratio (\( \delta = 0.5 \)) and are separated by short straight sections which prohibit fully-developed flow, so that some departures from classical Dean's flow might be expected.

According to Fee (1989), Dean-type flow, with several additional features, was observed, with the present model refer to Figure 2.1):

(i) A secondary flow upstream of the proximal bend.
(ii) A reversed flow region along the outer wall of the proximal bend indicating core flow impingement at the outer wall near the bend. This impingement would give rise to a stagnation zone (ie. a position of zero axial velocity) and cause local high pressure and thickened boundary layer at the outer wall. Similar reversed flows were not observed at the medial or distal bends.
(iii) An axial flow separation from the wall occurred at the inner side of the proximal bend, leading to a complicated recirculation zone downstream along the inner wall of the proximal limb. Axial flow separation also occurred at the inner walls of the medial and distal bends, resulting in relatively simple reversed flow regions downstream. These separations were attributed to the high curvature ratios (Fee, 1989).

The separated flows were subjected to inherent unsteadiness in the distal and proximal limb (eg. vortex formation and shedding from the shear layer bounding the separation region which is known as the free shear layer). Pressure
fluctuation would have consequently been felt at the wall at the points of separation and of reattachment. Analogous to the flow separation over a backward-facing step, there is a back and forth motion of the flow separation point of the shear layer which results in free shear layer oscillations and fluctuating velocities on the wall at the reattachment point (Rockwell, 1979). It is these unsteady vortical motions that may be involved in the etiology of atherosclerosis.

In the presence of a recirculation zone, a shear layer may exhibit well-defined oscillations due to classical hydrodynamic instability or a low-frequency oscillation mechanism associated with imbalance of entrained and return flows of the shear layer (Rockwell, 1983). The unsteady vortical structures in free shear layers were related to a characteristic frequency by the Strouhal number:

$$S_L = \frac{f L}{U}$$

where: $f = \text{frequency (Hz)}$, $L = \text{shear layer impingement length (m)}$, $U = \text{mean velocity in the free-stream}$.

Rockwell (1983) found (with a variety of geometries) a Strouhal number in the range of 0.5 to 2.5 for the shear layer instability and $S_L < 1$ for the low-frequency oscillation mechanism.

1.4 Hypotheses for Transmural Macromolecular Permeability of the Arterial Endothelium

In the Unified Haemodynamic Hypothesis (Steinberg, 1983; Fee, 1989), it was concluded that haemodynamics could initiate atherosclerosis by three mechanisms: (i) stress fluctuation fatigue, (ii) damage to the endothelium or (iii) by altering the endothelial permeability to macromolecules such as Low Density Lipoprotein (LDL).

Three possible hypotheses which account for the trans-endothelial macromolecular transportation have been to date:

(i) The pinocytotic vesicular transport of macromolecules.
(ii) The passage of macromolecules via intercellular crevices between endothelial cells.
(iii) The leaky junction-cell turnover hypothesis.

1.4.1 Macromolecular transport across the Arterial Endothelium via Pinocytotic Vesicular Transport

A hypothesis based on the theory that macromolecular transport into the arterial wall is rate-limited by the endothelial layer (Arminski et al, 1980) and occurs via pinocytotic vesicular transport, was proposed (Weinbaum & Caro, 1976), to explain the effects of haemodynamic forces on the transmural permeability of the artery wall. The pinocytotic vesicles are approximately spherical with an internal diameter of 70 nm, they exist within the cell in constant numbers and are able to diffuse back and forth across the cell by Brownian motion (Casley-Smith, 1969; Caro et al 1976). These vesicles attach to the cell wall via a quasi-steady stalk with an internal diameter and length of approximately 20 nm. The stalk connects the vesicle interior to the cell exterior. Macromolecules (eg. LDL's) in the lumenal (blood) side of the endothelial cell are entrapped in these vesicles together with plasma material. This is known as the vesicle loading process. After a time, the vesicle stalk detaches from the cell wall and the neck is sealed so that the vesicle interior is kept isolated from the cell content. The vesicle then diffuses to the ablumenal side of the cell, attaches to the cell membrane and allows its contents to diffuse from the ablumenal side of the cell (Figure 1.1). Any difference in the concentration of a molecular species between the lumenal (blood) and ablumenal (tissue) sides of the endothelial cell results in a nett transfer, due to the vesicular diffusion, across the cell in the direction of the concentration gradient.

It was argued that the rate-limiting step in this mechanism lies in the vesicle detachment process (Rubin, 1976; Arminski et al, 1979). The driving forces for the migration of free vesicles and the detachment of attached vesicles are derived from mechanical and chemical factors. Macromolecular transport across the endothelial layer is sensitive to temperature (Siflinger et al, 1975), shear stress (Caro, 1974), pressure oscillations and oscillatory stretching of the arterial wall (Chien et al, 1978; Arminski, 1980).
Figure 1.1: The Movement of Vesicles across the Endothelial Cell in the Pinocytotic Vesicular LDL Transport.
1.4.2 Transendothelial Macromolecular Transport through Crevices between Endothelial Cells

Frokjaer-Jensen (1984) suggested that, as an alternative to vesicular transport, the transendothelial LDL transport could occur via passage through the intercellular crevices. Transendothelial macromolecular permeability was shown to be shear dependent and time dependent (Jo et al, 1991) and this observation was attributed to shear-dependent widening of intercellular crevices, which enhanced macromolecular passage. One of the likely mechanisms of this hypothesis is that shear stress experienced on the arterial wall may exaggerate the existing intercellular crevices and the wall pressure fluctuations (attributed to highly disturbed flow) may subsequently convectively enhance the passage of macromolecules into the crevices.

1.4.3 Macromolecular Transport through Leaky Junction – Cell Turnover

According to this theory the transendothelial macromolecular transport occurs via passage through large endothelial pores created by the temporary disruption of the junctional protein strands of a very small of cells that are involved in cell turnover (Weinbaum et al, 1985). The cells associated with the leaky junctions are those which are either in the mitotic phase or in the process of sloughing due to cell death. Macromolecular passage through the leaky junctions may then be enhanced by haemodynamic factors eg. arterial wall pressure oscillations.

1.4.4 Vesicular Transport of Macromolecules: A Theory for the Convective Enhancement of Diffusional Vesicle Loading

In the pinocytotic vesicle transport mechanism, the vesicle loading process could be affected by two different mechanisms:

a) The diffusional loading process for molecules appreciably smaller than the vesicle stalk -- this hypothesis suggested that a concentration gradient (of LDL molecules) between the lumenal (blood) and ablumenal (tissue) sides of the arterial wall induces the diffusion of LDL molecules into the vesicles via the vesicle stalk. To incorporate the effect of molecular sieving in the diffusional loading mechanism, the time of passage of an LDL molecule through the vesicle stalk has been related to an observed diffusion coefficient, \( D_{obs} \), which is lowered from the bulk diffusion coefficient by a geometric partitioning effect in the vesicle stalk (Malone and Anderson, 1978). Local blood flow disturbances which are
postulated to be the source of pressure fluctuations may give rise to fluctuating convection through the vesicle attachment stalk and enhance $D_{obs}$, hence enhancing LDL's diffusion into the vesicle.

b) The convective loading process -- another important effect on the loading of vesicles is offered by the possibility of convective sweeping of LDL molecules and some of the surrounding fluid, through the vesicle attachment stalk, into the vesicle cavity. This process hinges upon the volumetric change of a vesicle which is restricted by the bending stiffness and the spontaneous curvature of the vesicle wall. Since the vesicle interior volume is very close to the volume consisting of an individual LDL molecule and its associated plasma fluid, the presence of one LDL molecule inside a vesicle represents a similar LDL concentration to the plasma concentration. Assuming a pressure fluctuation frequency of 50 Hz and mean vesicle attachment time of 6 to 30 seconds, one LDL molecule could be expected to enter a vesicle before detachment, thus bringing the vesicle interior to an equilibrium concentration equal to the exterior (plasma) LDL concentration. This process is expected to result in a vesicle interior LDL concentration of more than three times that expected by diffusional loading (Fee, 1989). An increase in the equilibrium LDL concentration within the vesicle would lead to an increased transendothelial LDL transport rate.

1.5 The Frequency Characteristics of the Pressure Fluctuations

The frequency characteristics of the wall pressure fluctuations may be predicted by the strange attractor theory of turbulence. Blondeaux and Vittori (1991) used a numerical approach to consider the unsteady flow set up on the vicinity of a wavy wall by an oscillating pressure gradient. A chaotic behaviour was predicted for:

a) A fixed geometrical configuration and an increasing flow Reynold's number, Re (based on shear layer thickness).

b) A fixed characteristic of the oscillatory flow with an increasing amplitude of wall waviness.

In both cases, the flow experienced an infinite sequence of periodic doubling, known as pitchfork bifurcation, in a finite interval of flow Re and in a finite range of the waviness height, respectively. These critical values of finite intervals of Re accumulate to a finite limit with the Feigenbaum rate of convergence. For Re (based on shear layer thickness) larger than about 40, a chaotic flow was
detected where aperiodic flow and broadband component in the frequency spectrum existed.

Blondeaux and Vittori found that for a specific geometrical configuration, small values of Re resulted in a periodic flow where a high frequency peak at frequency $2f$ (which was two-times (superharmonic) the frequency of the forcing term, $f$), existed. The superharmonic can be explained by the fact that a vortex structure is shed each half-cycle from the crest of the waviness, which caused the vertical component of the velocity to be directed upward both when the mainstream flow is from left to right and vice versa. Subsequent increase in Re caused further bifurcation at frequencies of $0.5f$, $0.25f$, $2f$, $3f$ and so on. The system reaches an unpredictable (chaotic) behaviour through a sequence of period doubling. By increasing the Re the non-linear (chaotic) effects become prominent and the superharmonic components increase their amplitude even though the flow remains periodic with period $T/2$. 
CHAPTER 2
EXPERIMENTAL METHODS AND APPARATUS

2.1 The Model

The model used in this experiment was chosen to simulate an abnormal feature of the cervical portion of the human internal carotid artery (it was designed by Fee (1989) and described in detail in his thesis). It consists of two perspex (polymethacrylate, PMA) blocks with highly polished surfaces and a semicircular channel of C-shaped tortuosity machined into each of the blocks. The two blocks were then positioned and joined by pins and screws which resulted in a channel of constant circular cross-section of 20 mm.

The C-shaped configuration of the model, lay in the plane of symmetry and was symmetrical about the mid-point of the C-shaped bend (see Figure 2.1). The bends have inner radii of curvature equal to the tube diameter (20 mm). Straight sections between the three bends were inclined at 70° to the upstream and downstream sections. The length of the straight sections were equal to the tube diameter. This configuration is within the range of geometries found in vivo. (Weibel & Fields, 1965)

The end-pieces consisted of circular brass pieces with the bore slowly tapered from 22 mm to 20 mm diameter and with flanges welded onto both ends. These were joined onto the model with screws to provide a smooth inner surface, with as small a discontinuity to wall flow as possible. RTV (Silicone Adhesive/Sealant, Silastic 732 Room Temperature Vulcaniser) was applied to the flange joints to prevent leakages.

A total of twenty eight pressure measurement ports were machined into one of the perspex blocks, at various positions along the C-shaped channel. These ports were connected to the ICA model wall via ‘V’-shaped channels of 0.4 mm cross sections and 1 mm long. The pressure measuring device was inserted into the pressure ports and thus exposed to the pressure fluctuations on the model wall.
Figure 2.1: The ICA Model Configuration

FLOW

Proximal Stem

Inner Proximal Bend

Outer Proximal Bend

Proximal Limb

Inner Medial Bend

Outer Medial Bend

Outer Distal Bend

Distal Stem

Inner Distal Bend

3000

800

R 20

20

70°
2.2 The Flow System

Figure 2.2 shows a line diagram of the flow system employed in this experiment.

The perspex ICA model was supported free of vibration by a 70 kg 152 mm x 76 mm metal U channel/beam which was rubber mounted horizontally onto brackets fixed to the wall. The support metal channel had low natural frequency (since frequencies between 10 Hz and 20 Hz were observed when the metal channel was subjected to mechanical knock) which, if generated, would not interfere with the expected results. A 2 kg weight was suspended at the midpoint of the beam to balance it in the direction perpendicular to its length.

All the piping involved in the flow system was 20 mm I.D. PVC straight pipes (wall thickness 2 mm) and PVC bends and elbows of similar diameter. A straight and smooth entrance length of 2 m was situated in the flow system prior to entering the upstream end-piece of the model. A straight exit length of 1 m was provided downstream of the model before flow was deviated to pass through other fittings such as a rotameter and valves.

A steady flow of air was sucked through the system by an in-house vacuum system, using a 5.6 kW Nash Hytor vacuum pump (size H4), situated 28 m downstream from the model. A vacuum of about 0.1 bars was normally maintained in the ICA model during the experiment. A Metric size 10 Rotameter with a koranite float, situated 5 m downstream of the model, was used to measured the air flow. The manufacturer’s calibration was used directly for flowrate setting (the experimental results were later adjusted to account for the appropriate lower air density under vacuum). The air flow was controlled by two diaphragm valves located just upstream and about 5 m downstream of the rotameter, respectively. The valve downstream of the rotameter (valve 2) controlled the main size of flow withdrawn by the vacuum system and the one upstream (valve 1) used for minor adjustments in obtaining the required flowrate. These valves generated relatively little noise and flow disturbances when not quite fully opened, which was ideal for the control purposes in this case.

Due to the small amplitude of the fluctuations to be measured, the flow was required to be laminar, steady and ‘quiet’. A 200-litre plastic drum with a silencer was located at the entrance of the experimental system to steady the air flow and eliminate atmospheric noises and pressure disturbances. The silencer
1. Vacuum supplied by vacuum pump
2. Metric 10 Rotameter with Koranite float
3. Diaphragm valves for air flow control
4. ICA model
5. 200 l Plastic drum
6. Silencer
7. Cotton wool filter
Distances in mm
Note: Not drawn to scale

Figure 2.2: Line diagram of the Experimental System
consisted of a 850 mm length of 110 mm diameter PVC pipe which was filled with pieces of fibre glass and cotton wool. The fibre glass pieces were placed such that the air flow followed a sinusoidal pathway, which served to eliminate the higher frequency air-borne noises. The plastic drum, on the other hand, acted as a capacitance to minimise the effects of low frequency atmospheric pressure fluctuations.

Wads of cotton wool measuring about 15 mm to 30 mm in length were inserted at various positions inside the piping. The wads located upstream of the model served to reduce the atmospheric disturbances whereas those downstream, to minimise any disturbances generated by the rotameter and the valves.

As the experimental system was under constant vacuum, all the joints and fittings in the experimental system were sealed by RTV or vaseline to prevent leakage, taking care to prevent any sealant from reaching the flow channel. The pressure ports on the ICA model were sealed with sticky aluminium tape when not in use. It was found that even a tiny leak in the system would generate undesirable disturbances. An air tight system was therefore, required, and this was defined in practice by the vacuum in the section upstream of valve 1, dropping by less than 48 cm³ per hour when isolated, by valve 1 at one end and a rubber bung at the other.

2.3 The Measuring Device

A Bruel & Kjaer calibrated microphone type 4145 was used in this experiment, to measure the wall pressure fluctuations in the ICA model. The microphone was sealed in an air-tight 100 cm³ perspex chamber with a 4 mm diameter opening at one end. Any exposure to atmospheric disturbances on the other side of the sensor was thus avoided. The microphone chamber was supported by three pieces of foamy plastic on a holder which was welded onto the supporting beam.

Measurements were taken with the aid of a measuring probe which consisted of a brass tube (4 mm O.D.) sealed at one end, with a 1 mm circular opening drilled through the tube wall close to the sealed end. (see Figure 2.3) The opened end of the probe was connected to the microphone via a 21 cm length of flexible PVC tubing (4 mm I.D.). During the experiment the probe was inserted into the probe access hole which opened to the 'V'-shape channel. The 1 mm opening on the probe (its position marked by the indicator) was then rotated and aligned with
the 'V'-shape channels thus exposing the microphone directly to the ICA wall (Figure 2.4).

2.4 Data Collection

2.4.1 The Data Collecting Apparatus

The Bruel & Kjaer type 4145 microphone used to measure pressure fluctuations at the wall of the model was powered by a 240 volts power supply/pre-amplifier which also amplified the output signal of the microphone. The output signal was further amplified by one thousand times through a two-stage amplifier.

The amplified signal was then fed three ways:
(i) A data aquisition unit, Strobe Acquisition Unit 901A which collects data and functioned as a spectrum analyser.
(ii) A Krohn-Hite type 3340 electric filter which was subsequently fed into a high-averaging digital voltmeter (DVM). This set-up was aimed at isolating the major frequency of the pressure fluctuations by creating a band-pass filter. The Krohn-Hite filter was operated as a low pass filter while the build-in filter on the DVM acted as a high pass filter thus allowing the Vrms only of the peak around the desired frequency to be recorded. 
(iii) An oscilloscope which gave an instantaneous overall trace of the time spectrum.

The pressure fluctuation measurements were taken individually at each of the port positions. During the experiment all the ports not in current use were sealed by sticky aluminium tape, firmly pressed on top of the probe access holes; which ensured an air-tight condition inside the model.

After positioning the probe into the pressure port, a small amount of RTV was applied around the probe-model interface, to seal up any possible leak points.

2.4.2 Experimental Procedure

1. With both the diaphragm valves closed, the vacuum pump as switched on. The flowrate was set by fully opening valve 1 (the valve upstream of the rotameter) and setting valve 2 (the one downstream of the rotameter) to a flowrate slightly higher than that required. Valve 1, which was located closer to the model, was then used for fine-tuning it.
Indication to mark the location of the opening

Brass tube:
O.D. 4 mm
I.D. 3 mm

1 mm diameter opening

Figure 2.3: The Measuring Probe
1. ICA channel 20 mm I.D.
2. V-shape channel:
   0.4 mm cross section
3. Probe opening:
   1 mm diameter
4. Indicator
5. Flexible PVC tubing
   4 mm I.D.,
   length: 210 mm
6. Perspex chamber
7. O-ring
8. Microphone

Figure 2.4: The Microphone Probe and Pressure Port
Figure 2.5: Flow Diagram for the Data Collecting System
2. The measuring probe was inserted into the first measuring port 8 diameters upstream of the proximal bend. Rotational positioning of the probe was done by 'aurally' detecting the position where maximum flow occurred. RTV was applied around the probe-model interface and the microphone was connected to the probe.

3. All the measuring devices were switched on. The Krohn-Hite filter was set to low pass with a roll-off point at 40 Hz and the DVM filter was set to high pass roll-off at 20 Hz; thus 'isolating' the 34 Hz peak. The DVM was also set to 10 seconds averaging.

4. A Fast Fourier Transform (FFT) was performed using the Strobe Aacquisition Unit 901A at a sampling rate of 500 per second and a total of 4096 samples. The resultant frequency spectra were stored on a computer disk. A manual recording of the Vrms (ie the DVM read-out) was also carried out simultaneously.

5. After each measurement the flow rate was increased and the experiment repeated. The flowrate tested corresponded to Reynolds numbers of 1087, 1189, 1257, 1325, 1393 and 1461.

6. A further test was done to investigate the hysteretic nature of the pressure fluctuations. In this test the flowrate was decreased gradually until the major peak (34 Hz) disappeared from the frequency spectrum, and subsequently increased until the peak reappeared. The flowrate(s) at which the major peak disappeared and reappeared were recorded.

7. The FFT and Vrms measurement were carried out at each pressure port, in turn. The results were collected to provide an overall pressure fluctuation distribution of the C-shape model.

8. Frequent checks were made on the upstream FFT to ensure that background noise level remained constant.
Plate 1: The Experimental System
Plate 2: The ICA Model and Brass End-pieces supported on the supporting beam.
Plate 3: The Pressure Measurement Probe.
Plate 4: The Microphone Support.
CHAPTER 3
EXPERIMENTAL RESULTS

The Fast Fourier Transforms (FFT's) of the pressure fluctuations and the root-mean-square voltage (Vrms) of the 34 Hz peak were examined for six steady flowrates: Re = 1087, 1188, 1257, 1325, 1393, 1461; at various positions along both the $\alpha = 0^\circ$ and $\alpha = 180^\circ$ azimuth of the model (Figure 3.1). The rotameter was calibrated for air flow under the appropriate vacuum (see Appendix A).

The major frequency of pressure fluctuations observed in this study was 34 Hz. At positions downstream of the mid-medial bend, harmonics and subharmonics of the major frequency were evident at 17 Hz, 68 Hz and 102 Hz (periodic doubling) and a band-broadening phenomenon was also observed at these positions. Due to the presence of low frequency background disturbances, a band-pass electrical filter was created with the Krohn-Hite 3340 filter (sixth order filter) set to eliminate all frequencies above 40 Hz (cutoff frequency $\pm 10\%$) and the built-in filter of the digital voltmeter (DVM) (cutoff frequency $\pm 20\%$) set to eliminate frequencies below 20 Hz. The Vrms of the major frequency, 34 Hz, was thus electrically ‘isolated’ and manually recorded.

4096 measurements were analysed at each measurement point by a Strobe Acquisition Unit 901A. Due to the effects of intermittency, the pressure fluctuations were found to erratically jump between metastable states over times of one or two seconds and the resultant Vrms readings could not be recorded accurately on a voltmeter with a time constant of less than a second. Therefore, the Vrms data was averaged by the DVM over a 10 seconds sampling period, to ‘average out’ the effects of intermittency. With this precaution, fluctuations of $\pm 0.02$ mV were obtained. Before each run the microphone was connected at the pressure port 8 diameters upstream of the proximal bend and a spectrum observed to assess the back ground disturbances.

The microphone chamber and pressure measuring port on the model essentially formed a Helmholtz resonator of volume 32 cm$^3$ and length of 1 mm, with a calculated resonant frequency of about 600 Hz (see Appendix B). Frequency ratios of (measured frequency)/(resonant frequency) of 0.06, 0.11 and 0.17 were
Figure 3.1: Pressure Port Locations for Wall Pressure Fluctuation Measurements.
thus obtained for the 34 Hz, 68 Hz and 102 Hz frequencies respectively. The resultant pressure attenuation was negligible since the frequency ratios were less than 0.33 (Kinsler & Frey, 1962). A test for pressure attenuation was also carried out by exposing the microphone: a) directly and b) with the connecting tube and measuring probe, to a signal source and a time trace was observed on the oscilloscope. The resultant peak amplitudes were identical for both cases ie. no attenuation was observed.

Using the microphone calibration (46.2 mV per Pa) allowing for the further amplification, the Vrns data were converted to pressure fluctuations. The resultant pressure fluctuations were then presented as pressure coefficients,

\[ c_p = \frac{<p^2>^{0.5}}{0.5 \rho U^2} \]

where: 
- \( c_p \) = pressure coefficient,
- \(<p^2>^{0.5}\) = root-mean-square (rms) pressure (Pa),
- \( \rho \) = density of air (kg/m\(^3\))
- \( U \) = free stream velocity (m/s).

Figure 3.2 showed the rms pressure distribution drawn on the geometry of the model.

Amplitude ratios of harmonics to major peak were calculated to give an estimation of strength of the harmonic frequencies present.

3.1 An Analysis of the Frequency Spectra and Vrns Results at a Constant Flowrate (Re = 1087)

3.1.1 The Proximal Stem

The pressure fluctuations observed in the proximal stem for both azimuth \( \alpha = 0^\circ \) and \( \alpha = 180^\circ \) sides were relatively small (about 2 x 10\(^{-3}\) Pa). The major frequency (34 Hz) peak was not prominent on the frequency spectra.

However, some low frequency fluctuations were observed. These were the remnant of the atmospheric disturbances. As mentioned in Chapter 2, most of the disturbances of higher frequencies (> 20 Hz) were filtered off physically by
Figure 3.2: Root-mean-square Pressure Distribution (X $10^{-3}$ Pa) of the major frequency peak (34 Hz)

$\alpha = 180^\circ$

$\alpha = 0^\circ$

FLOW

Scale 1 mm : $1 \times 10^{-3}$ Pa

Run 1

Run 2
Figure 3.3: Amplitude Distribution (mV) of the Harmonic frequency (68Hz)
the silencer at the entrance of the flow system and the cotton wool filters along the piping. The lower frequency disturbances, being long-wavelength, could not be eliminated completely (see Figure 3.4) and had to be filtered off electrically. The frequency spectrum observed in the proximal stem could therefore be used as an assessment of the background disturbances being admitted to the flow system.

3.1.2 The Proximal Bend

Pressure fluctuations were small at the positions just before (ie upstream) both the inner and outer proximal bends (inner bend being the one with the smaller radius of curvature and vice versa). Their magnitude increased along the bends but the major frequency peak was still not prominent on the frequency spectra.

3.1.3 The Proximal Limb

The pressure fluctuations began to increase after the proximal bend and the 34 Hz peak first appeared on the frequency spectra taken at the mid-proximal limb. Vrms values of the major peak at the azimuth $\alpha = 0^\circ$ were generally larger than those at $\alpha = 180^\circ$ and the major peak (on the frequency spectra) became sharper and more well-defined, downstream of the mid-proximal limb.

3.1.4 The Mid-Medial Bend

The major peak was prominent (about $5.2 \times 10^{-3}$ Pa) at the mid-medial bend and its harmonics at 68 Hz, 102 Hz and 136 Hz were observed on the frequency spectra. Vrms (of the 34 Hz peak) measured on the outer bend were higher than those of the inner bend. However, much higher amplitude ratios were found on the inner mid-medial bend.

Amplitude ratios:

<table>
<thead>
<tr>
<th>Hz Ratio</th>
<th>Inner Bend</th>
<th>Outer Bend</th>
</tr>
</thead>
<tbody>
<tr>
<td>68 Hz : 34 Hz</td>
<td>0.9</td>
<td>0.05</td>
</tr>
<tr>
<td>102 Hz : 34 Hz</td>
<td>0.2</td>
<td>-</td>
</tr>
<tr>
<td>136 Hz : 34 Hz</td>
<td>-</td>
<td>0.01</td>
</tr>
</tbody>
</table>
3.1.5 The Distal Limb

Periodic doubling seemed to be a prominent feature at both the azimuth $\alpha = 0^\circ$ and $\alpha = 180^\circ$ sides of the distal limb. $\text{Vrms}$ of the major peak (34 Hz) was higher at the azimuth $\alpha = 180^\circ$.

3.1.6 The Distal Bend

The frequencies observed at the distal bend were 34 Hz, 68 Hz and 17 Hz. The 34 Hz peak was strongest at the outer distal bend (about 0.03 Pa). Band-broadening was observed on both the inner and outer distal bends, especially at the lower frequency end of the spectra (ie. the 17 Hz peak). Amplitude ratios of 68 Hz : 34 Hz and 17 Hz : 34 Hz were higher at the inner bend (0.3 and 0.5 respectively) than the outer bend (0.1 and 0.2 respectively).

3.1.7 The Distal Stem

Peaks at 34 Hz, 68 Hz and 17 Hz were observed at the distal stem. The $\text{Vrms}$ (34 Hz) was higher at the azimuth $\alpha = 0^\circ$ side (about 0.01 Pa) than the $\alpha = 180^\circ$ side. Band-broadening of the 17 Hz peak was observed. Amplitude ratios for the 68 Hz : 34 Hz and 17 Hz : 34 Hz were 0.1 and 0.5 respectively for the azimuth $\alpha = 0^\circ$ side and 0.1 and 0.2 respectively for $\alpha = 180^\circ$ side.

3.2 Results Analysis for Increased Flowrates

By increasing the flow Reynolds number (Re) from Re = 1087 to Re = 1461 at Re = 70 steps (approximately), several features were noted:

(i) The $\text{Vrms}$ values generally increased with higher flowrate (see Figure 3.4), indicating higher-amplitude pressure fluctuations.

(ii) With increased flowrate higher amplitudes of low frequency peaks were observed on the frequency spectra.

(iii) The effects of periodic doubling were more prominent at increased flowrates -- for instance at the distal limb at azimuth $\alpha = 0^\circ$ side, peaks at 0.25f, 0.5f, 2f, 3f and 4f, where f was the major frequency, were observed at higher flowrates (Re = 1461).
(iv) At higher flowrates the effects of band-broadening were more prominent for all the peaks.

(v) The dominant fluctuation frequency shifted to the higher frequency end of the spectra -- for instance the major frequency at the inner mid-medial bend moved from 34 Hz at $Re = 1087$ to 45 Hz at $Re = 1461$.

3.3 Test for Hysterisis

A test was carried out to examine any hysterisis of the pressure fluctuation with respect to the flow (refer to Chapter 2). It was found that the major frequency peak appeared on the frequency spectra at about $Re = 1005$ consistently throughout the model when flow was increased. This peak also disappeared at the same Re. It could therefore be inferred that the wall pressure fluctuations were independent of hysterisis (Rockwell and Naudascher, 1979).
Graph of Vrms of Major Frequency Peak (34 Hz) vs. Renolds Number

Figure 3.4: Strength of Peak with Increasing Flowrate
Figure 3.5: Frequency Spectrum at 8 diameters upstream of the Proximal Bend (Re = 1087) which served as the assessment of the background atmospheric pressure disturbance.
Figure 3.6: Frequency spectrum at the Inner Mid-Medial bend (pressure port position 12 T) (Re = 1087) showing strong peaks at higher harmonic frequencies.

Figure 3.7: Frequency spectrum at the Outer Mid-Medial bend (pressure port position 10 B) (Re = 1087) showing less harmonics than the Inner Mid-Medial bend.
Figure 3.8: Frequency spectrum at the Distal limb (pressure port position 11 B) at increased flowrate (Re = 1461) showing periodic doubling, band broadening and shift of major frequency.

Figure 3.9: Frequency spectrum at the Inner Distal bend (pressure port 12 B) showing linear and nonlinear periodic doubling of the subharmonic frequency.
CHAPTER 4
DISCUSSION

4.1 Experimental Techniques and Apparatus

4.1.1 The Model

The C-shaped tortuosity of the ICA model was chosen for simplicity of configuration; since simple flow geometry could reduce any ambiguity in the relationship between atherosclerosis and haemodynamics. Flow disturbances, due to second order wall waviness, were minimised by the constant diameter ICA tube.

The presence of the crack along the interface of the two-halves of the perspex (polymethacrylate, PMA) blocks which made up the model posed a threat for leakage. Other possible leakage points include pressure measurement ports that were not in use and the threads for screws and pins (which hold the model together). All of these potential leakage points were sealed with sticky aluminium tape, firmly pressed onto the outer surface to eliminate any possible clefts.

To detect leakages, a test was carried out by exposing all potential leak points (eg. the cracks along the interface between the perspex blocks) directly to a signal source of a specific frequency, with the microphone and measuring probe in place in one of the pressure ports (on the model). The resultant frequency spectrum and time trace (on the oscilloscope) were monitored simultaneously. If a peak at the applied frequency was observed on the frequency spectrum at a particular potential leak point then a leakage was established. The leak point was promptly sealed, either with RTV (Silicone Adhesive/Sealant, Silastic 732 Room Temperature Vulcaniser), if the joining surfaces were internal (eg. the sealing surfaces of the brass end-pieces and the model); or with sticky aluminium tape, if the surfaces were external (eg. the crack along the symmetry interface).

The brass end-pieces were joined onto the perspex model with screws and RTV was applied around the sealing surfaces to ensure maximum security against leakage. The end-pieces were originally made of perspex but were found to be
unsatisfactory due to the bending load applied by the perspex model itself, which made surface-sealing difficult. They were later replaced by the present brass end-pieces. Reshaping and polishing were done within the brass sections to match their inner flow channels to the entrance and exit of the model, to within an accuracy of 0.5 % of the ICA channel diameter, to minimize any flow disturbance.

A straight tube with a similar diameter and pressure measurement port was machined out of perspex and was initially used for assessing the atmospheric noise and pressure disturbance. The straight section was connected to the flow system during these initial tests thus eliminating the presence of the C-shape tortuosity which may interfere with the upstream flow pattern. It was expected to produce a smooth and laminar flow. Any disturbance observed could then be attributed to atmospheric perturbation.

The experimental system was tested for leakage by applying a vacuum of 0.8 bars absolute pressure to the sealed system. The leak rate was found to be about 48 cm³/hour. A small hole (1 mm diameter) was tapped onto the downstream end-piece to enable a vacuum gauge connection. This hole was sealed with a rubber bung when not in use.

4.1.2 The Flow System

Some of the major objectives that ensured the success of this experiment were:
(i) The elimination of structural vibrations.
(ii) The prevention of leakage.
(iii) The elimination of atmospheric pressure disturbance.
(iv) The elimination of flow disturbance.

Structural vibrations were found to severely affect the flow system by introducing amplified low frequency vibrations. The model was initially mounted onto a square section sheet metal beam which was mounted directly off the concrete building wall. The resultant frequency spectrum was swamped by low frequency (1 Hz to 30 Hz) peaks of high amplitude (up to 50 mV). It was also sensitive to the structural vibrations generated on the floor above. To overcome this problem, the model was mounted on a very heavy beam (weighing about 70 kg), with felt-lined metal holders to hold the model in place. The straight section of the piping was mounted onto the beam through holder bats (circular clamps) which were lined with a foamy elastomeric material. The beam was in turn
mounted onto the concrete wall by a rubber lined (rubber lining about 2 cm thick) metal bracket. The weight of the beam increased the inertia of the flow system and lowered its natural frequency. The microphone was also rubber mounted on a holder which was welded onto the same supporting beam, which ensured an identical natural frequency for both the flow system and the measuring device.

Each section of the flow system was tested individually for leakage and flow/atmospheric disturbances, prior to the main series of experiments. An appreciable resistance to flow was introduced at the flow entrance, due to atmospheric disturbance precautions (see below) and this meant that the model was operated under appreciable vacuum (absolute pressure of 0.8 bars). It was found that due to the constant vacuum, the system was very susceptible to leakage. This was corrected by sealing all the joints with RTV.

The atmospheric disturbance introduced to the flow system was first found to be unacceptably high. Various flow systems were experimented on, to minimise this effect:

a) A flow system identical to the present one without the 200 litres plastic drum and silencer -- a high level (amplitudes up to 50 mV) of disturbance (up to 60 Hz), was recorded.

b) The same flow system with the silencer connected directly to the entrance -- the resultant frequency spectra was still swamped by atmospheric pressure disturbance which was similar in amplitude and frequency to those mentioned above.

c) A 'closed' flow system with the vacuum on the downstream end and an air supply (supplied by a blower) on the upstream end -- this essentially eliminated atmospheric disturbances of all frequencies but introduced noise (frequency: 35 Hz to 50 Hz, maximum amplitude: 70 mV)) generated by the blower.

d) The present system except with a steel drum and silencer at the entrance -- the steel drum resonated (at 10 to 20 Hz) with the atmospheric pressure fluctuations.

The present flow system was found to be optimal, with a 200 litre plastic drum and silencer connected to the entrance of the flow system. The atmospheric disturbance, though not completely eliminated, was reduced to an acceptable level (background noise: frequency < 15 Hz and maximum amplitude < 20 mV; with maximum amplitude at about 5 Hz)). All the high-frequency disturbance (20
Hz and above) was filtered off by the silencer (maximum amplitude was reduced to less than 5 mV).

The air flowrate was found to remain constant within measurement errors of 5 % once set. During the experiment, the valve upstream of the rotameter (valve 1) was used only for fine-tuning the flowrate and was nearly always fully opened to minimise flow disturbance. Two chunks of cotton wool were located inside the pipeline downstream of the model to filter off any flow disturbance generated by the rotameter and the valves.

4.1.3 The Measuring System

The calibrated Bruel & Kjaer type 4145 condenser microphone used in this experiment has a sensitivity of 46.2 mV per Pa. The measuring probe and flexible tubing which connected the microphone to the pressure ports essentially formed a Helmholtz resonator. Since the ratio of measured frequency to the calculated resonator’s natural frequency was very small (see Appendix B), the resonator’s effect on the measuring system was expected to be negligible. The pressure attenuation test carried out with the microphone-probe set up (Chapter 3) confirmed this assumption.

During the experiment, the probe was inserted into the probe access hole of the pressure port and a small amount of RTV was smeared around the interface between the probe and the perspex (PMA) model to ensure air-tight sealing. The pressure ports and probe access holes were cleaned with a small piece of paper towel wrapped around a syringe needle, after each run.
4.2 Experimental Results

In the present study, various interesting features were observed with the C-shaped, rigid-wall, ICA model:

(i) The existence of pressure fluctuations which were related to the C-shaped tortuosity.
(ii) The dominant frequency of pressure fluctuations and its magnitude.
(iii) The period doubling of the resultant frequency spectra.

4.2.1 Relationship between Pressure Fluctuation and C-shaped Tortuosity

Pressure fluctuations were observed at various positions on the test model especially along the proximal limb, mid-medial bend, distal limb, distal bend and distal stem. On the other hand, no significant signals were observed at the proximal stem where the ICA tube was smooth and straight. It could be deduced that the pressure fluctuations were related to secondary flows induced by the C-shaped tortuosity.

At constant flowrate it was found that the fluctuations intensified (as measured by the Vrms) as the measurement position moved from the proximal limb downstream, towards the distal bend. The frequency spectrum for the distal bend was very complex due to a highly turbulent flow which was generated by three consecutive bends on the flow path. The increased tortuosity (ie. the number of bends) seemed to be responsible for both the complexity and increased intensity of the pressure fluctuations.

Fee (1989) concluded that flow separations at various positions in the model were subjected to inherent unsteadiness. Vortex formation and shedding from the shear layer bounding the separation regions occurred in both the distal limb and distal stem. In the proximal limb, Fee (1989) found that the flow downstream of the vortex structure exhibited a periodic wavering. Consequent pressure fluctuations would undoubtedly have been felt at the walls of the tube near the separation and reattachment regions.

With respect to the separation/impingement flow configuration, as observed in this case at the bends of the model tortuosity, Rockwell (1979) concluded that many types of impingement geometry were capable of inducing a self-sustaining oscillation which arises from instability of the free shear layer in conjunction with disturbance feedback. Generation of the instability wave at the impingement...
edge was probably triggered by nonlinear and viscous effects. Rockwell (1979) pointed out that highly organised oscillations of an separating/impinging flow were sustained through a series of interacting events:

a) feedback of disturbances from the impingement region to the area of the free shear layer near the separation,

b) inducement of localised vorticity fluctuations in this region by the arriving perturbations,

c) amplification of these vorticity fluctuations in the shear layer between impingement,

d) production of organised disturbances at impingement.

It could therefore, be inferred that the C-shaped tortuosity was responsible for flow separation, reattachment and recirculation which in turn, caused the observed pressure fluctuations at the ICA wall.

4.2. The Dominant Frequency of Fluctuation and its Magnitude

Driver et al (1987) concluded that, in a reattaching turbulent shear layer behind a back-facing step, the major part of the energy in the separated flow resided in the frequency characteristic of roll-up and pairing of vortical structures seen in free shear layers. Rockwell (1979) stated that for a given shear impingement configuration and length, the frequency of oscillation was a strong function of conditions at separation. Typically, the frequency of oscillation was expressed in terms of the global Strouhal number, $S_L$, based on impingement length:

$$S_L = \frac{f}{L} \frac{L}{U}$$  \hspace{1cm} (4.1)

where: $f =$ frequency, $L =$ shear layer impingement length and $U =$ mean velocity in the free-stream.

The impingement enhanced the organisation of the flow along the entire length of the shear layer due to the strong influence of disturbance feedback; thus creating the characteristic frequency of the fluctuation. Vortex shedding at the impingement edge also contributed to the amplitude of the wall pressure fluctuations. The impingement length of a recirculation region was also found to be $Re$ dependent (Fotea et al, 1991).
Rockwell (1983) found that oscillations triggered by the inherent instability of the shear layer have Strouhal numbers in the range of 0.5 to 2.5. In this experiment the dominant fluctuating frequency was observed to be 34 Hz (at Re = 1087) and Strouhal numbers of 2.1, 0.9 and 0.9 were calculated for the inner medial, proximal and distal bends, at the dominant frequency; using Fee's (1989) results on separation-impingement length (L) measured along the inner wall. This frequency scaled to an in vivo pressure oscillation of about 53 Hz at Re = 1087 and 74 Hz at Re = 1500 (see Appendix C). The impingement length/tube diameter ratios measured from Fee's (1989) experiment ranged from 1 to 2.7 which were of the same magnitude with Fotea's (1991) results (about 3, at Re = 1500).

The root-mean-square voltage, $V_{rms}$, of the dominant fluctuating frequency (34 Hz), which relates to the rms pressure, $<p^2>^{0.5}$, were measured. As the measurements were taken by a calibrated microphone with a sensitivity of 46.2 mV per Pa, the resultant $V_{rms}$ could be readily converted into rms pressure fluctuations, $<p^2>^{0.5}$.

At the proximal bend $rms$ pressure fluctuation, $Prms$, were found to be very small. This was probably attributed to the existence of two separation regions which led to two areas of low shear at the wall. $Prms$ increased downstream of the proximal limb. The outer medial bend (ie larger radius of curvature) showed a higher wall $Prms$ (caused by impingement) compared to the inner medial bend (separation region). The maximum $Prms$ were found on the outer distal bend and inner distal stem where impingment occurred.

Several attempts have been made to relate the wall pressure fluctuations in a separated flow to the shear layer bounding the flow. Ignoring radiation of pressure from other parts of the flow, the wall pressure fluctuations in the recirculation region was due either to the recirculating flow itself or the shear layer.

The rms pressure $<p^2>^{0.5}$ of the reversed flow (with negative velocity) in a recirculating flow boundary layer could be estimated by using the ordinary boundary layer result (Fricke and Stevenson, 1970):

$$\frac{<p^2>^{0.5}}{q} = 0.006$$

(4.2)
where \( q = \) kinetic head = \( 0.5 \rho u^2 \)
\[ u = \text{reversed flow velocity}. \]

With this calculation, the wall pressure fluctuations from a reversed flow boundary layer was shown to be very small.

Another source of hydrodynamic pressure fluctuations at the wall in the recirculation zone is the shear layer. Roos and Kegelman (1986) measured a maximum fluctuating pressure coefficient

\[ c_{p1} = \frac{p^1}{q_o} = 0.04 \quad (4.3) \]

at reattachment downstream of a backward-facing step, where \( q_o = \) the kinetic head based on the free stream velocity and \( p^1 = \langle p^2 \rangle^{0.5} \).

Driver et al (1987) measured the static pressure fluctuations at the wall under a separated shear layer behind a rearward-facing step and found a maximum static pressure fluctuation coefficient, \( c_p \), of 0.2 near the reattachment point, where:

\[ c_p = \frac{P_s - P_{sref}}{0.5 \rho U_{ref}^2} \quad (4.4) \]

and \( P_s \) = static pressure at the wall,
\( P_{sref} \) = reference pressure,
\( q \) = fluid density and
\( U_{ref} \) = velocity at the reference station.

In the present experiment, it was deduced that \( c_{p1} \) ranged from 0.003 to 0.07 which falls within the magnitude of equations (4.2) and (4.3); although it was on the lower side of equation (4.4).

The above estimates of wall pressure fluctuations (equations (4.2) to (4.4)) which were associated with separated flows in the model, were wide ranging. The backward-facing step used by Roos and Kegelman (1986) was the closest match to the present flow configuration but was effectively a two dimensional flow.

4.2.3 The Periodic Doubling
The periodic doubling and band-broadening phenomena observed on the frequency spectra from the mid-medial bend downstream, towards the distal stem, can perhaps be described by the strange attractor theory of turbulence (Blondeaux and Vittori, 1991) which stated that by increasing the flow Reynold’s number, Re (based on shear layer thickness), the flow experienced an infinite sequence of period doubling (pitchfork bifurcation), in a finite interval of flow Re. These critical values (ie. the finite intervals) of Re accumulate to a finite limit with the Feigenbaum rate of convergence. For Re larger than about 40, a chaotic flow is detected where aperiodic flow and broadband component in the frequency spectrum existed.

According to Blondeaux and Vittori (1991), in the phase space, the limit cycle characteristic of an increasing Re loses stability. In doing so, it produces a new attracting periodic orbit which tracks the old one closely but goes around it twice before closing, thus produces the pitchfork bifurcation. Subsequent increase in Re causes further pitchfork bifurcation at frequencies of 0.5 f, 0.25 f, 2 f, 3 f and so on. The system reaches an unpredictable behavior through a sequence of period doubling.

Moreover, Rockwell (1983) observed that streamwise disturbance growth in the shear layer was usually characterised by initially exponential amplification (ie. ‘linear growth’ on semi-log coordinates), followed by nonlinear distortion involving energy transfer between the fundamental and its higher harmonics. Frequency spectra of impinging shear layer were found to exhibit a number of organised peaks which represented frequencies higher and lower than the fundamental instability frequency of the shear layer.

Rockwell noted that for purely hydrodynamic oscillations, formation of highly coherent waves at the fundamental/dominant frequency, f, led to vortex formation and occurrence of 2 f, 3 f components at lower amplitude. This is known as the nonlinear distortion of the fundamental. Vortical coalescence of two adjacent vortices initially formed at frequency f, subsequently yields a subharmonic component at frequency 0.5 f and occasionally its nonlinear subharmonic 1.5 f. Simultaneous or successive coalescence of a number of vortices produces a corresponding reduction in the predominant frequency.

It was indeed found, in this experiment, that periodic doubling occurred, peak frequency increased and broadened as the flowrate increased. As in the mid-medial bend and distal limb, the dominant frequency, 34 Hz, was shown to
'double' up to 238 Hz ie six cycles of periodic doubling, as the flow Re increased from 1087 to 1461. Moreover, linear and nonlinear streamwise amplification of the disturbance was substantiated by: a) the increase in amplitude of the dominant frequency (34 Hz) and b) the appearance of higher and lower harmonics of the dominant frequency; at downstream of the proximal limb (eg. the medial and distal bends).

At the distal limb, a broadband low frequency peak of approximately 17 Hz (subharmonic of the major frequency) was observed, which could be attributed to vortex coalescence. This peak increased in amplitude and became less well-defined downstream of the distal limb. The non-linearity (chaotic nature) of the subharmonic peak was amplified streamwise.

The haemodynamic instability may be further enhanced by the occurrence of oscillating reattachment point of the shear layer and vortex shedding in vivo, due to the pulsating nature of the flow (Mullin et al, 1980; Olson et al, 1991).

4.3 Haemodynamics and Atheromic Sites

Fee (1989) deduced that the in vivo sites which corresponded to all the bends in the ICA model were affected by atheroma. The inner bends (bends with smaller radius of curvature) matched with the in vivo proliferative atheromic sites and the outer bends (larger radius of curvature), to the atrophic lesion (Figure 4.1).

4.3.1 The Proliferative Atheroma

Sites for proliferative atheroma were predicted to experience low mean shear stress, oscillating shear stress and high frequency-high magnitude pressure fluctuations (Fee, 1989). Caro et al (1971) suggested that the accumulation of LDL at the arterial wall was attributed to:

(i) the active inward transport of the LDL's from the blood to the arterial wall,
(ii) the synthesis of LDL in situ (at the arterial wall) which was regulated by the rate of inward transendothelial LDL transport (ie. from blood to wall)
(iii) the passive outward diffusion of LDL from arterial wall to the blood.

Increase in transendothelial macromolecular permeability was also found to be shear dependent (Caro et al, 1971; Jo et al, 1991). At low shear stress the in situ synthesis of LDL was not inhibited and since the passive outward diffusion of LDL was inhibited by elevated blood LDL levels, a nett accumulation of LDL at the arterial wall may result, given sufficient time.
Inherent haemodynamic instabilities such as oscillating shear stress may contribute significantly to the transendothelial LDL transport from blood to arterial wall. Moreover, for pinocytotic vesicular transport of LDL (see Chapter 1) high frequency-high magnitude fluctuating pressure experienced on the arterial wall may enhance the inward transendothelial LDL transport rate (and thus atherogenesis) by:

(i) enhancing the vesicle loading process and
(ii) fracturing the vesicular stalk and thus enhancing the vesicle detachment process (Weinbaum & Caro, 1976). These observations supported the association of proliferative atheromic sites to areas of low shear stress, oscillating shear stress and high frequency pressure fluctuations in vivo.

4.3.2 The Atrophic Atheroma

Atrophic atheroma, on the other hand, was commonly related to areas of arterial wall which were subjected to unidirectional, high shear stress and lower amplitude pressure fluctuations. Since high shear stress was reported to retard progression of atheroma lesion by inhibiting the in situ LDL synthesis (Caro et al, 1971), thus the relationship between high shear stress and atropic atheroma was established.

4.3.3 Pressure Fluctuations and Atheromic Sites

In this experiment, the pressure fluctuations observed upstream of the proximal stem were small compared to that further downstream. This is consistent with the fact that atheroma is seldom found in straight sections of the ICA in vivo.

The inner mid-medial bend showed a high amplitude peak of 68 Hz, even though the Vrms measured (for the 34 Hz peak) was lower than that of the outer mid-medial bend (refer to Figure 3.2 and 3.3). Prediction of proliferative atheroma at this site was not supported if only the 34 Hz peak was considered to contribute to the overall pressure fluctuation, since the magnitude of this peak, at the inner medial bend, was lower than that at the outer bend. On the other hand, if the higher frequency (ie. 68 Hz) peak was taken into consideration than the inner medial bend would have a higher magnitude pressure fluctuation than the outer bend and was thus subjected to atheroma of proliferative nature in vivo.
The outer distal bend showed an exceptionally high $V_{\text{rms}}$ value, in contrast to the prediction of atrophic atheroma. This may be accounted for by the high level of low frequencies signals (up to 34 Hz) observed on the frequency spectrum. The high pass filter on the DVM (second order filter) had a very gradual (i.e., not sharp) frequency response and a rolled-off point at about 40 Hz when set to 'high pass > 20 Hz'; with a cutoff of $\pm 20\%$ at frequencies between 34 Hz and 45 Hz. However, the Krohn-Hite sixth order filter had a very sharp frequency response and a rolled-off point at 50 Hz when set to 'low pass maximumly flat < 40 Hz'. Due to the frequency response characteristics of the band-pass filter (made up of the DVM filter and the Krohn-Hite filter) the major frequency peak (34 Hz) was not strictly 'isolated' in the presence of high amplitude low frequency pressure fluctuations.

Only low levels of pressure fluctuations occurred at the proximal bend (due to the separation regions) and no prominent distinction could be made with regards to the proliferative and atrophic atherotic sites.
Figure 4.1: Atheromic sites in the tortuous Internal Carotid Artery.
CHAPTER 5
CONCLUSIONS

5.1 Experimental Conclusion

In this study with the C-shaped ICA model, it was established that the occurrence of wall pressure fluctuation was attributed to the geometry of the flow path. The major fluctuation frequency was observed to be about 34 Hz at Re = 1057 which scale to a frequency of 53 Hz in vivo, using the Strouhal's number (Appendix C). The maximum pressure fluctuation found on the outer distal bend was scaled to 10.7 Pa in vivo (Appendix D). Extrapolating to Re = 1500 the in vivo frequency was scaled to 74 Hz and pressure fluctuation to 8 Hz. Due to the high Reynolds number and high curvature ratios of the bends (of the model) periodic doubling was observed in the frequency spectra.

The highest probability of atherosclerosis occurrence has been reported to lie in the proximity of bends in the flow path (Stehbens, 1987). The regions of the model subjected to the higher frequency (68 Hz, 102 Hz etc.) pressure fluctuations (ie. on the inner mid-medial and distal bends) were associated with reported sites of proliferative atheroma in vivo. Atrophic atheroma, on the other hand, was associated with areas subjected to low frequency (34 Hz) but high amplitude pressure fluctuations (at the outer mid-medial and distal bends of the model).

5.2 Atherogenesis and LDL Transport

It is now generally accepted that a haemodynamic factor is involved in atherogenesis but questions still remain over which of the flow features are important in the progression of the diseases and the mechanisms by which flow disturbances may lead to atherosclerosis. In particular, the mechanism of transendothelial LDL transport in relation to the pathogenesis of the disease remains a disputable issue.
In regard to this study, three possible mechanisms could account for the transendothelial LDL transport:

(i) The pinocytic vesicular transport.
(ii) The intercellular junction convective transport.
(iii) The leaky junction-cell turnover convective/diffusive transport.

In the pinocytic vesicular transport theory (refer to Chapter 1), the arterial wall pressure fluctuations may play an important role in the convective loading of the vesicles and in the vesicle stalk detachment process as well (Weinbaum and Caro, 1976), hence enhancing the transport rate of the vesicles across the cell. Step changes in shear stress (both in increasing and decreasing directions) has also been found to enhance pinocytic vesicular transport (Davies et al, 1984). These features corresponded well with the predicted sites for proliferative atheroma. The validity of the pinocytotic vesicular transport, however, has been subjected to criticism (Frokjaer-Jensen, 1984).

Convective passage of the LDL molecules through intercellular junctions, on the other hand, is possible only if the junctions are widened by constant shear stress experienced on the arterial wall (Jo et al, 1991). Once a LDL molecule is lodged in the intercellular junction which is widened by shear stress, arterial wall pressure fluctuations may enhance the convective transport of the molecule to the ablumenal side of the cell. Low shear stress was found to retard the recovery of pre-shear transendothelial permeability level (Jo et al, 1991) and hence contributes to proliferative atheroma. However, this mechanism have been contradicted by Born & Shafi (1989).

The leaky junction-cell turnover hypothesis postulates that the LDL molecules enter the artery wall via the leaky junctions between dying or regenerating cells involves in the turnover process. Non-uniform subendothelial pressure fields arise from the different hydraulic resistances of normal and leaky endothelial clefts which enhances the horizontal transport (ie parallel to the endothelial layer) of macromolecules after they have passed through the leaky clefts. The inherent arterial wall pressure fluctuations may, in this case, affect the subendothelial pressure field and enhance the macromolecule transport. Further, it was found that while low levels of turbulent shear stress stimulates cell turnover (Davies et al, 1986) high constant shear stress has no effect on the cell turnover. On the other hand, the cells associated with leaky junctions comprise less than 1 % of the endothelial cell population in nearly all the major blood vessels and the leaky
junctional area through which macromolecules might pass involves less than $10^{-5}$ of the endothelial surface. Therefore its contribution towards transendothelial LDL transport may well be questionable.

In the light of the mechanisms of atherogenesis proposed, it could be concluded that constant, as well as oscillating, shear stress and also arterial wall pressure fluctuations may be contributive factors to the initiation and progression of the disease. Each of the above LDL transport hypotheses is alone insufficient to account for atherosclerosis due to the complex interactions between the haemodynamical and biochemical factors involved.

The study of haemodynamics and the possible relationship between blood flow and atherosclerosis remains an important area of research.
References


Appendix A: Rotameter Calibration

The Metric size 10 rotameter used in this experiment was calibrated against a known air flowrate, with the aid of a gasometer.

Method:
Valve 1 (upstream of the rotameter) was set to give a specific rotameter reading. With the vacuum pump operating (ie set by valve 1), the gasometer was connected to the entrance of the experimental flow system; thus creating a system where the gasometer supplied air directly to the flow system. The air flowrate from the gasometer was measured.

Assuming a linear relationship between air flowrate and rotameter reading at constant pressure and temperature, all subsequent (higher) flowrates and Reynolds numbers, Re, were calculated.

\[
Re = \frac{D \cdot U \cdot \rho}{\mu}
\]  
(A-1)

where:  
\(Re\) = Reynolds number,  
\(D\) = Diameter of the tube (m) = \(20 \times 10^{-3}\) m  
\(U\) = Mean velocity (m/s)  
\(\rho\) = Density of flow fluid (kgm\(^{-3}\)) = 1.1055 kgm\(^{-3}\)  
\(\mu\) = Dynamic viscosity of fluid (kgm\(^{-1}\)s\(^{-1}\))  
= \(1.75 \times 10^{-5}\) kgm\(^{-1}\)s\(^{-1}\)

At a rotameter reading of 16 cm, the air flowrate was found to be 0.27 l/s and \(Re = 1087\), from equation A-1.

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Appendix B: The Helmholtz Resonator

The transient response of pressure-measuring instruments is dependent on the response of the pressure transmitting fluid and the connecting tubing etc., which determines the overall frequency response of a pressure measurement system. The configuration of the pressure measurement port and microphone probe used in this experiment may be thought of as comprising a Helmholtz resonator, consisting of a rigid enclosed volume $V$, communicating with the fluctuating pressure source via an opening of radius, $r$, and length, $L$ (Figure B-1).

Consider the system shown in Figure B-1. The mass of transmitting fluid vibrates under the influence of friction in the connecting tube, which tends to dampen the motion. If the conventional formula for laminar friction resistance in tube flow is used to represent this friction, the resulting expression for the pressure-amplitude ratio is

$$\left| \frac{p}{p_0} \right| = \frac{1}{\left[ \frac{1}{2} \left( \frac{v}{\omega \rho} \right)^2 + 4 \eta^2 \left( \frac{v}{\omega \rho} \right)^2 \right]^{1/2}}$$  \hspace{1cm} (B-1)
where:

\( p \) is the amplitude of the pressure signal impressed on the microphone

\( p_0 \) is the amplitude of the measured pressure signal

\( \omega \) is the frequency of the measured fluctuating pressure

\( \omega_n \) is the natural frequency of the Helmholtz resonator and is given by:

\[
\omega_n = \left[ \frac{3\pi r^2 c^2}{4LV} \right]^{1/2}
\]  

(B-2)

\( h \) is the damping ratio given by:

\[
h = \frac{2\mu}{\rho c r^3} \left[ \frac{3LV}{\pi} \right]^{1/2}
\]  

(B-3)

In the above formulas:

\( c \) is the velocity of sound in the transmitting fluid

\( \mu \) is the dynamic viscosity of the fluid and

\( \rho \) is the fluid density.

The transmitting fluid, in this case, is air at 20° C and 0.9 bars, with: \( \rho = 1.1055 \) kg/m³; \( \mu = 1.75 \times 10^{-5} \) kgm⁻¹s⁻¹ and \( c = 343 \) ms⁻¹

The connecting channel (which connects the fluctuating pressure source to the pressure transducer) is the V-shaped opening with cross section, \( r = 0.4 \) mm and length, \( L = 1 \) mm The connection volume, \( V \), consists of:

(i) volume of the probe, \( V_1 = 3.82 \times 10^{-7} \) m³

(ii) volume of the flexible PVC tubing, \( V_2 = 2.66 \times 10^{-6} \) m³

(iii) volume enclosed in the microphone chamber (i.e., the volume in front of the microphone), \( V_3 = 2.89 \times 10^{-5} \) m³

Therefore, \( V = V_1 + V_2 + V_3 = 3.19 \times 10^{-5} \) m³
From equation B-2, \( \omega = 594 \text{ Hz} \) and \( \omega/\omega_n = 0.06 \), for \( \omega = 34 \text{ Hz} \) (the major/dominant frequency peak). Damping ratio, \( h = 2.01 \), from equation B-3. Therefore, from equation B-1,

\[
\left| \frac{P}{P_0} \right| = 0.97
\]

Repeating the procedure for \( \omega = 68 \text{ Hz} \) (harmonics), \( 102 \text{ Hz} \) and \( 17 \text{ Hz} \) (subharmonics), values of 0.92, 0.84 and 0.99 are obtained from equation B-1. It could therefore be concluded that amplitude attenuation for the present pressure fluctuation measuring set-up is negligible.
Appendix C: Calculations for In Vivo Characteristic Frequency of Pressure Fluctuation

The characteristic frequency of the pressure fluctuations caused by inherent instability of the shear layer was scaled by the Strouhal number,

\[ S_L = \frac{f}{L} \frac{L}{U} \]  \hspace{1cm} (C-1)

where: \( f \) = characteristic frequency (Hz)  
\( L \) = shear layer impingement length (m)  
\( U \) = mean velocity of the free-stream (m/s)

The mean velocity of free-stream was scaled by the Reynolds number,

\[ Re = \frac{D \ U \ \rho}{\mu} \]  \hspace{1cm} (C-2)

where: \( D \) = tube diameter (m) = 20 \times 10^{-3} m  
\( U \) = mean linear velocity (m/s)  
\( \rho \) = density of the flow fluid at the operating pressure = 1.1055 kg m^{-3}  
\( \mu \) = dynamic viscosity of the flow fluid at the operating pressure = 1.75 \times 10^{-5} kg m^{-1} s^{-1}

Using equation C-2, \( U = 0.86 \) m/s.

From Fee's experimental results (1989) the ratio of impingement length/tube diameter, was measured along the inner wall of the model. These were found to be 1.21, 2.66 and 1.1 for the proximal, medial and distal bends respectively. The impingement length, \( L \), was simply calculated by multiplying the impingement length/tube diameter ratio by the tube diameter.

Assuming a characteristic frequency of 34 Hz (as observed in this experiment), \( S_L = 2.1 \) for medial bend, using equation C-1.

For the in vivo conditions:

blood vessel diameter (D) = 8 mm, \( \rho_{\text{blood}} = 1005 \) kg m^{-3} and \( \mu_{\text{blood}} = 0.004 \) kg m^{-1} s^{-1}.  
At \( Re = 1087, U = 0.54 \) m/s, using equation C-2.
From equation C-1 substituting $S_L = 2.1$ and the appropriate $L$ (ie impingement length/tube diameter x blood vessel diameter), the \textit{in vivo} characteristic frequency was calculated to be 53 Hz at $Re = 1087$. For $Re = 1500$, assuming a linear increase of the major frequency, $f$ was calculated to be 47 Hz \textit{(in vitro)} and $S_L = 2.1$. Using the same treatment, an \textit{in vivo} frequency of 74 Hz was obtained.
Appendix D: Fluctuating Pressure Coefficient and In Vivo Pressure Fluctuation

Fluctuating Pressure Coefficient:

The results of this experiment are presented as fluctuating pressure coefficients,

\[ C_P = \frac{<p^2>_{1/2}}{0.5 \rho U^2} \]  \hspace{1cm} (D-1)

where: \( C_p \) = Fluctuating pressure coefficient
\( <p^2>_{1/2} \) = Root-mean-square pressure fluctuation (Pa)
\( \rho \) = Density of fluid (kgm\(^{-3}\))
\( U \) = Mean linear flowrate (m/s)

At \( Re = 1087 \), \( U = 0.86 \text{ m/s} \) and maximum root-mean-square pressure fluctuation, \( <p^2>_{\text{max}}^{1/2} = 0.03 \text{ Pa} \). Using equation D-1, maximum fluctuating pressure coefficient, \( C_{p_{\text{max}}} = 0.07 \). Extrapolating to \( Re = 1500 \) (Figure 3.4), \( <p^2>_{\text{max}}^{1/2} = 0.02 \text{ Pa} \) and therefore \( C_{p_{\text{max}}} = 0.03 \).

In Vivo Pressure Fluctuation:

At \( Re = 1087 \), \( U_{\text{blood}} = 0.54 \text{ m/s} \) (see Appendix C) and \( C_{p_{\text{max}}} = 0.07 \); therefore using equation D-1, \( <p^2>_{\text{max}}^{1/2} = 10.7 \text{ Pa} \). At \( Re = 1500 \) (which is the average blood flow Reynolds number), \( U_{\text{blood}} = 0.75 \text{ m/s} \), \( C_{p_{\text{max}}} = 0.03 \) and \( <p^2>_{\text{max}}^{1/2} = 7.9 \text{ Pa} \).
### Appendix E: Raw Data

**Vrms Data for \( k = 0 \)**

**KROHN-HITE 3340 FILTER**: Low pass upper limit

**DVM: Avg 10 s**  
**DVM High Pass Filter > 20Hz**

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<th>Vrms (mV)</th>
<th>Discrepancy (%)</th>
<th>Filter upper limit (Hz)</th>
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Vrms Data for  = 180

KROHN-HITE Filter 3340 Low Pass upper limit

DVM : 10 s avg DVM filter > 20 Hz

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Appendix F: Distribution of root-mean-square pressure fluctuation on the ICA model

Figure F-1: Root-mean-square Pressure Distribution ($10^{-5}$) of the major frequency peak (34 Hz) Run 1
Figure F-2: Root-mean-square Pressure Distribution 
($x \times 10^{-3}$) of the major frequency peak (34 Hz) 
Run 2