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Blunt Traumatic Aortic Injury

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Introduction

Blunt Traumatic Aortic Injury (BTAI) refers to tearing of the wall of the aorta, a serious, often fatal, injury that occurs during severe automobile collisions. BTAI is one of the most common causes of death in road traffic accidents [1], and may be caused by other impacts, such as in fall victims.

Despite 150 years of speculation on the subject, however, there is still widespread disagreement as to the principal mechanisms giving rise to the injury: previously proposed causes include

- stretching of the aorta,
- a sudden rise in blood pressure,
- shear forces, and
- pinching of the aorta by the rib cage.

One of the most hotly debated questions is whether some kind of impact (*e.g.* of a steering wheel with the chest cavity) is required to cause BTAI, or whether a sharp deceleration will suffice.

This question is prompted by reports that many victims of BTAI do not exhibit signs of other serious injury. As noted by Sevitt [11],

> *It is difficult to explain aortic rupture in subjects with little or no evidence of injury to the chest... without involving indirect effects like... deceleration of the moving body. There were many such cases....*

Gotzen[4], by comparing injuries suffered by drivers and passengers who were or were not wearing seat belts, came to the opposite conclusion:

> *Pure deceleration... was of no importance in causing aortic rupture... Seat belt wearers do not suffer aortic rupture unless there is additional direct chest wall trauma.*

Gotzen also referred to the remarkable auto-experiments of Stapp in defending this stance:

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In these unique experiments the author subjected himself to extensive deceleration and there was no thoracic injury.

Clearly some more fundamental understanding of the underlying physical mechanisms is required if progress is to be made in answering such questions. To this end, a finite element model of the aorta and surrounding structures is being developed at the Transport Research Laboratory. The model should be able to predict the motion of, and stresses induced in, the aorta under various body forces and/or displacements applied to the chest cavity.

The aim is to identify the physical features that most influence the occurrence or otherwise of the injury; it may thus be possible to alter the designs of cars and seat restraints so as to minimise the danger.

The chest cavity has many complicated features (*i.e.* organs, bones, ligaments, blood, *etc.*) and, at present, there is little consensus as to which are important in BTAI. Moreover, neither the fluid mechanics of the blood nor the coupling between the aorta and the spine is treated in any detail. If the model is to be extended and improved, the issues to be addressed include the following.

1. How well do the simulations agree with observations from experiments and from real accidents?
2. How realistic are the assumptions made in the current model? What effect do they have on the model's predictions?
3. How important is the fluid mechanics of the blood?
4. How significant are the other anatomical features not presently incorporated?

In this report we suggest some simple sub-models which may lend valuable insight into each of these questions. We begin by using dimensional analysis to estimate which of the many physical processes at work are likely to have a bearing on BTAI. Two separate types of mathematical models are then proposed and preliminary findings are discussed. These are:

1. fluid dynamical models which incorporate bending and the effects of shear flow; and
2. a number of simple as well as complex mechanical models to consider the effects of stress and strain on the aorta.

The idea in either case is, by eliminating all other effects, to understand the influence of each process in isolation. A number of models relevant to these sub-problems already exist in the literature, and we include a summary of these and their respective results.

The report concludes by investigating the way in which the information gained from the sub-models could be fed into the finite element calculation.

Experimental evidence

BTAI manifests itself as a transverse tear in the aorta wall, usually resulting in either rupture or complete severing of the aorta.

Occasionally the wall is damaged but not ruptured; in such cases the evidence is that the tear starts on the inside of the aorta and propagates outwards. In the majority of cases, BTAI occurs in the proximal descending aorta in the neighbourhood of the so-called *isthmus*, a region of high curvature, as shown in

Figure 1. There is a slight narrowing of the aorta in this region, and some believe that the tissue forming the aorta wall may be inherently weaker here. The isthmus also marks the boundary between the relatively free aortic arch and the lower descending aorta which is more-or-less pinned to the spine by the lungs.

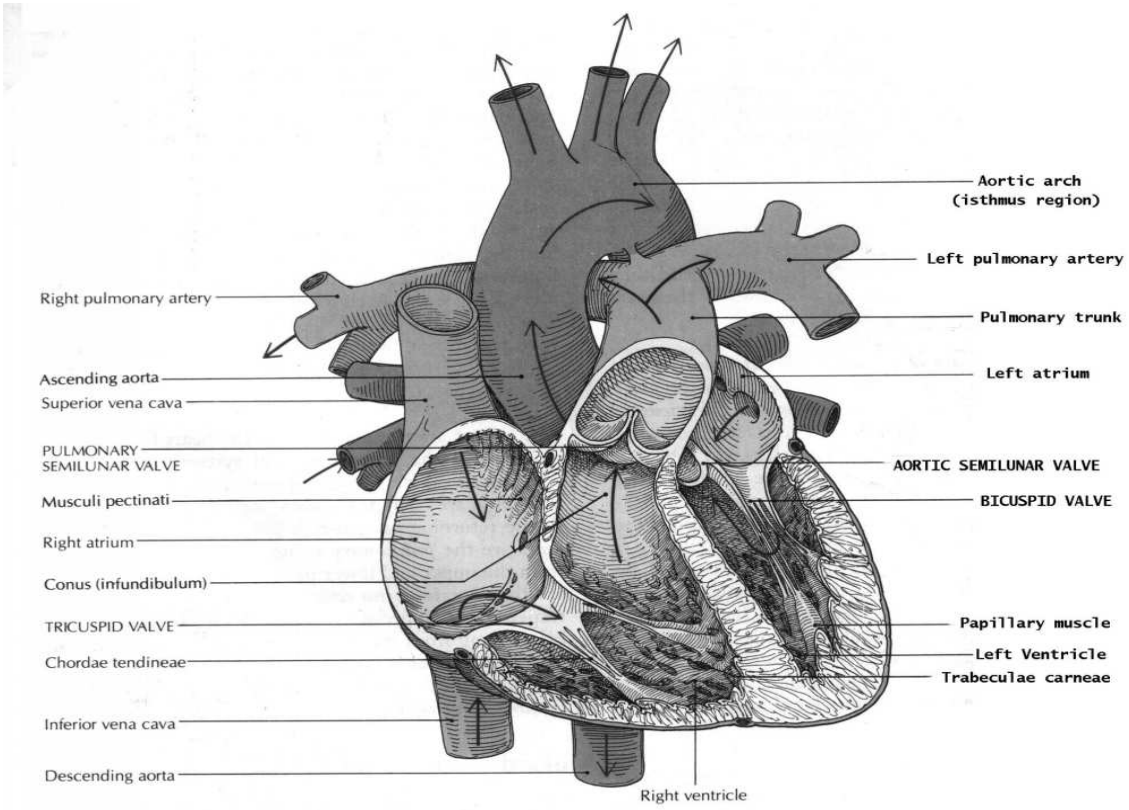

Figure 1: A schematic representation of the heart and aorta showing the aortic arch and isthmus region as taken from [13]. The arrows indicate the direction of the blood flow to and away from the heart.

Some approximate physical data relevant to this problem are summarised in table 1. Estimates for the aorta radius r and wall thickness a have been obtained from values quoted in [5]. The failure stress for human aortic tissue segments was measured during tensile experiments by Mohan & Melvin [7]. It was found that the tissue is in general more susceptible to breaking in the longitudinal than in the transverse direction, although there is a large deviation in these results. Failure stress was also found to be rather strongly rate-dependent. In table 1, we quote a mean value of three atmospheres.

The blood is assumed to have density and viscosity not too different from those of water. The volume V of the heart is found from the estimate in [5] that 7% of the blood in the human circulatory system (total volume 5–8 litres) can be found in the heart at any one time. A heart to isthmus arc length of 6 cm is typical for adult humans [2].

Now, to work out the forces experienced by the aorta and its surroundings, we consider the experience of a driver or passenger during a typical traffic accident. Suppose a car travelling at speed $U = 30$ mph ≈ 13 m/s is brought to rest over a distance $d = 2$ feet ≈ 0.6 m. This takes a time of order $2d/U \approx 0.1$ s, and corresponds to a deceleration of magnitude $U^2/(2d) \approx 15g$, where $g \approx 10$ m/s^2 is the acceleration due

Radius of the aorta	r	1.25×10^{-2} m
Aorta wall thickness	a	1.0×10^{-3} m
Tensile strength of aorta tissue	T	3×10^{5} Pa
Blood density	ρ	1×10^{3} kg/m^3
Blood viscosity	ν	1×10^{-6} m^3/s
Volume of the heart	V	5×10^{-4} m^3
Heart-to-isthmus arc length	L	6×10^{-2} m
Impact timescale	t_i	50 ms
Effective deceleration	G	1.5×10^{3} m/s^2

Table 1: Approximate physical parameters for Blunt Traumatic Aorta Injury.

to gravity. The occupants of the car continue at speed U until forced to slow down with the decelerating car; this could be caused by seat-belt restraints or by impact with the steering wheel, the dashboard or an air-bag.

Clearly, the time-scale for such a secondary impact is usually much shorter than $2d/U$. Moreover, the resultant body force exerted on the occupants may be significantly greater than $U^2/(2d)$: the figure $G = 150g$ is widely quoted in the literature [6, 10]. This would correspond to a driver accelerating forwards a distance of order 20 cm before striking the steering wheel and being brought to rest (relative to the moving car) over a timescale t_i of order 50 ms. Note that in what follows we assume the impact time t_i and time for tearing of the aortic wall are of the same order.

Dimensional Analysis

If we suppose that upon impact all the blood in the left ventricle is squeezed into the aorta, then the velocity of the blood is given approximately by

$$v = \frac{V}{\pi r^2 t_i} \approx 20\text{m/s} \tag{1}$$

The Reynolds number (based on the aorta radius) is thus calculated to be

$$Re = \frac{vr}{\nu} = \frac{V}{\pi \nu r t_i} \approx 2.5 \times 10^5. \tag{2}$$

Whilst this high Reynold's number may indicate turbulence, this may not necessarily be the case since the flow is impulsively started, i.e. as a result of the impact.

An interesting factor to consider is the acceleration of the blood in the aorta due to the impact. This is given by

$$a = \frac{V}{\pi r^2 t_i^2} \approx 40g. \tag{3}$$

Calculating the two hydrodynamic parameters

$$\begin{aligned} \rho v^2 &\approx 10^5 \text{ Pa} \\ \rho G L &\approx 10^5 \text{ Pa} \end{aligned}$$

from the above results shows that these pressures, in comparison to the tensile breaking strength of 30×10^5 Pa, are not sufficient alone to cause rupturing of the aorta.

The parameters we have calculated here tend to indicate that fluid dynamical factors are alone not enough to explain BTAI. Other arguments against a fluid dynamical approach include

- a burst of blood in to the aorta predicts maximum pressure near the heart, although the maximum may actually occur at the aortic arch (see Section 4 for details); and
- a change in internal pressures would lead to longitudinal rather than transverse rupturing as is the case for BTAI.

However, fluid dynamical models themselves can not be totally discounted due to a range of phenomena, which as a result of an impact, could occur in the aorta. To this end we discuss the development and modelling of such phenomena, in particular shocks and the effects of blood swirling in a 3-D model, in Section 5.1.

Recent Mathematical Models

Kivity and Collins [6] were the first to consider the application of fluid dynamics to the specific problem of aortic rupture. From the idea that a pulse of blood is pushed into the aorta upon impact, they postulated that shock waves may be created within the aorta and proposed a 1-D model to consider such effects. By including an appropriate expression for the visco-elastic properties of the wall and considering that the impact force acts longitudinally along the length of the aorta, Kivity and Collins where able to show that the formation of shocks was indeed possible. However, conclusions as to whether these shocks would be substantial enough to alone cause rupturing of the aorta were inconclusive.

One of the primary factors neglected by Kivity and Collins was the effect of curvature in the aorta. Whilst they considered shocks generated in a straight tube Ray *et.al.* [10] showed that the effects of curvature were substantial enough to result an almost six fold increase in pressure at the arch of the aorta. As well as this their other main assumptions included : (i) considering the blood to be inviscid but having a very high bulk modulus; (ii) accounting for centrifugal forces resulting from the fluid motion; (iii) taking the de-acceleration field to be horizontal.

Whilst fluid dynamical models exist in the literature which have considered effects possibly encountered in BTAI, for example non-linear waves in tubes, transient pulse propagation, etc., no models have considered modelling the mechanical effects of bending stresses, strains or the like.

Proposed Models

In this section we consider a number of important factors which need to be included in any future fluid dynamical based models of BTAI and also seek to elucidate the mechanical properties of the aorta, by first considering a number of simple and then more advanced mechanical based models.

With future work the resultant model may in itself be a 'hybrid' one requiring the inclusion of certain mechanical and fluid dynamically important factors to fully explain the effects of BTAI. However, such a model may be considerably more difficult to implement than those we present in the following sections.

Fluid Dynamical

Simple calculations of the blood flow in the aorta (see Section Three) during an average traffic accident, have shown fluid dynamical effects may alone not be enough to account for BTAI. However, such models can not

be fully discounted as previous ones have neglected to include a number of fundamental factors. Secondly, a number of different phenomena, some already considered to a certain extent in the literature, need to be re-considered in light of recent work.

Any future fluid dynamical models of BTAI should probably include the following :

- the elastic and shearing properties of the aortic wall;
- effects of the basic aorta (vessel) shape, which appears to play little part in the above flow-wall coupling so far, and which in particular is significantly curved. This high degree of curvature should result in an azimuthal focusing of stress, which in itself could lead to a focusing at the most vulnerable point of the aortic arch. Ray *et.al.* [10] have already shown that this to be the case in a simple 1-D model of the problem.

Other phenomena which could also be considered include :

- the effects of shocks in higher dimensions, at least 2-D possibly 3-D. The idea here is to see if shock formation and propagation could cause a tearing of the aortic wall. One proposed model problem is to consider a straight axisymmetric tube, containing incompressible fluid, and hit it in such a way that the cross-sectional area locally falls rapidly, $A_*(t)$, as demonstrated in Figure 2.

  Fluid will be squeezed out, generating an elastic jump which will grow and propagate with speed V, which might vary with time, depending on $A_*(t)$, even in a uniform tube. The basic wave propagation and shock formation can be analysed in the standard one-dimensional way. The shock-jump conditions can also be clearly stated, based on a conservation of mass and momentum as done by Oates [8] and Kivity and Collins [6], and as stated in Pedley [9]. However, these jump conditions cannot be used to evaluate the propagation speed V unless the 1-D model for fluid pressure p and the tube-law $A(p)$ can be used *in the jump itself*. This is because, unlike the case of the hydraulic jump, the pressure on the non-parallel parts of the tube wall exerts a longitudinal force on the fluid, thereby affecting the momentum equation.

  Another requirement of a jump is that energy must be dissipated in it. Kivity and Collins had the undoubtedly correct idea that the energy loss, in the case of the jump propagating into an undisturbed vessel, would take place through viscoelasticity of the wall. They were thereby able to analyse the shock's structure. However, their model retained the 1-D assumption.

  It is therefore proposed that the shock structure should be investigated using a fully 2-D, axisymmetric model. This will involve the complete Euler equations for fluid flow (it is unlikely that fluid viscosity will be important in these circumstances), plus a fully non-linear viscoelastic shell theory.

- the three-dimensionality likely in the rapid quasi-inviscid flow that is squeezed abruptly through the long curved vessel .The three-dimensional flow, being of a longitudinal vortex type with swirling, is influenced by the vessel curvature k far more readily than is a one- or two- dimensional flow. This influence stems for the $k\rho u^2$ centrifugal force which drives, via the momentum balances, the v and w velocity responses which in turn alter the u response via continuity. Here ρ is the fluid density, u is the velocity component along the vessel axis and v, w are in the cross-plane; the long axial length scale compared with the cross-plane scales allows the continuity balance between relatively large u values and relatively small v,w.

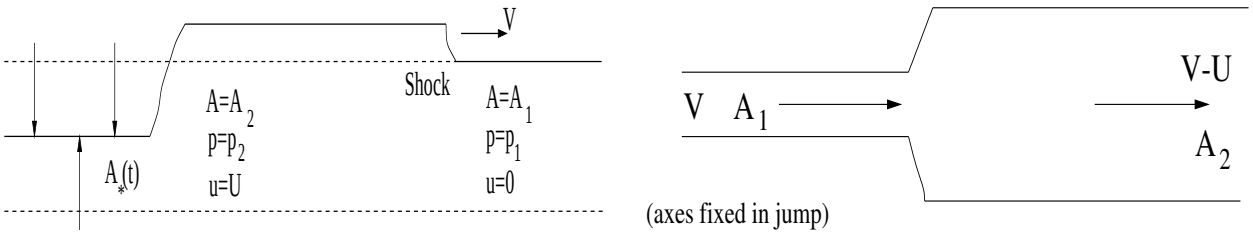

Figure 2: A schematic representation of the proposed model to consider the effects of shock waves generated in the aorta.

The pressures so produced in the three-dimensional flow through the curved vessel can be relatively large (for example see [3]) and likewise for extreme wall stresses. This could yield a pressure or stress focusing at the inner bend, enhancing the wall movement there. A study along these lines has recently been completed by Smith and Li [12].

Mechanical

In light of the lack of mechanical models regarding BTAI, we present in this section firstly a number of simple models which could provide important insights in to the effects of forces on the aorta. These lead to the more advanced models of an elastic rod and a weakly-curved, fluid filled elastic tube.

The "jerk" mechanism

This is the simplest mechanism that could arguably give rise to BTAI. When a person is subjected to the large body force G, the comparatively massive heart will be accelerated relative to the chest cavity. As

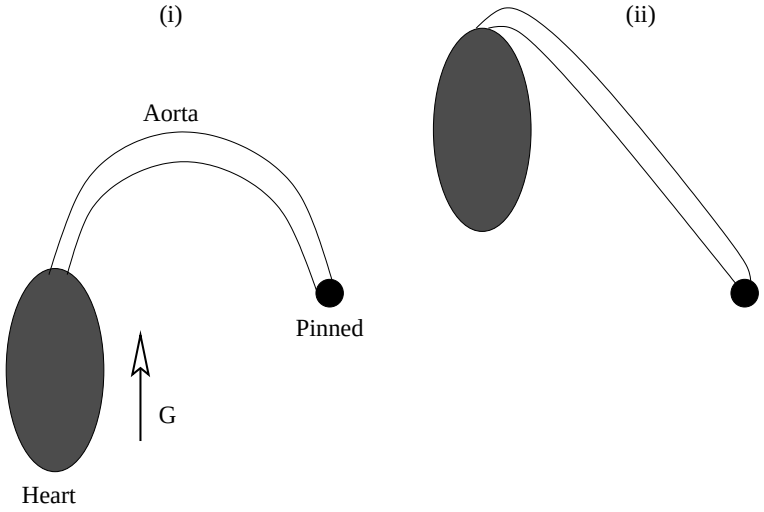

Figure 3: A schematic diagram of the aorta being "jerked" via acceleration of the heart.

indicated in Figure 3, if the heart moves far enough, this will have the effect of "jerking" the aorta which, recall, is (at least partly) pinned to the spine near the isthmus. The force experienced by the heart is of

order ρVG. If this is transmitted as tension along the aorta as in Figure 3(ii), it translates into a stress of order

$$\frac{\rho VG}{2\pi ar} \sim 10^7 \text{ Pa}$$

in the aorta wall, which is two orders of magnitude higher than the failure stress. Notice also that this kind of stretching would tend to cause the observed transverse tearing.

Experiments performed on pigs [11] indicate that the heart can move a distance of at least 5 cm during extreme acceleration, which would certainly be sufficient for this mechanism to come into play. It is also supported by the evidence [1] that accelerations directed towards the head are particularly dangerous so far as BTAI is concerned. This suggests that seat restraints which prevent the occupants from sliding forward into a horizontal position may help to reduce occurrence of the injury.

A simple model for this mechanism could be obtained by treating the heart as a rigid massive body and the aorta as an extensible string that is pinned at one end. It would be relatively straightforward to incorporate other details of the chest cavity in such a picture.

Torsion

As the heart is accelerated by the applied body force G, it will in general transmit a torsion as well as a tension along the aorta. Excessive twisting of the aorta may also give rise to the observed transverse failure. The next level of sophistication, therefore, would be to treat the aorta as an elastic rod rather than a string. This would enable us to determine the stresses due to both stretching and twisting of the aorta.

Buckling

At a still higher level of sophistication, the aorta could be treated as an elastic shell. As well as stretching and twisting, such a shell will fold if distorted too far. Again, this would tend to occur in the transverse plane, and so provides another possible mechanism for the observed failure of the aorta wall.

Canonical problem

Study of the following canonical problem will shed further light on which, if any, of the mechanisms suggested above may explain BTAI. Take a weakly-curved cylinder with thin elastic walls. Now put it under stress by applying tension, torsion and bending at the ends as demonstrated in Figure 4 and by imposing a hydrostatic pressure in the fluid inside the cylinder. Now the following questions can be addressed.

- Where are the stresses maximum?
- Where, and in which direction, is failure most likely to occur?
- Will the shell buckle?
- Is stretching, torsion, bending or internal pressure most likely to cause failure?

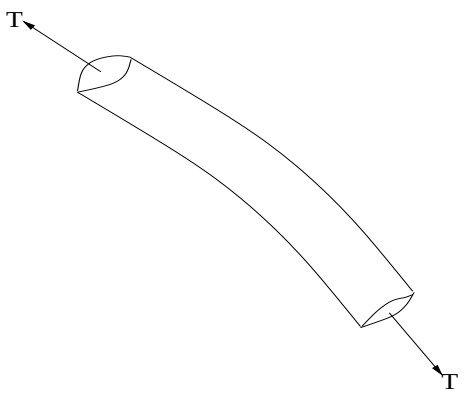

Figure 4: The problem of the fluid filled elastic rod.

Conclusions

This report has considered a number of models (fluid dynamical and mechanical) which could be included in a finite element model of Blunt Traumatic Aortic Injury (BTAI) being developed by Dr. Mike Neale at the Transport Research Laboratory.

An analysis of some of the important parameters relevant to blood flow in the aorta following an average traffic accident, has shown that whilst fluid dynamical effects may not be solely responsible for tearing of the aorta, such models cannot be discounted. This stems from the fact that previous models of BTAI have neglected a number of important factors. These include

- the elastic and shearing properties of the aortic wall; and
- the effects of the curvature in the aorta.

Further to this, two specific models to consider other important fluid dynamical phenomena have been proposed. The first is to consider whether the effects of shocks could cause a tearing in the aorta by considering a 2-D axisymmetric model using the inviscid Euler equations and non-linear viscoelastic shell theory. The second model seeks to include the effects of three-dimensionality as a result of the rapid quasi-inviscid flow being squeezed through the long curved aortic vessel.

In order to investigate the effects of stress and strain on the aorta during an impact, a number of simple and somewhat more advanced mechanical models of the aorta have been proposed. These include

1. the "jerk" mechanism;
2. treating the aorta as an elastic rod (torsion);
3. treating the aorta as an elastic shell (buckling); and
4. modelling the aorta as a thinly curved, fluid filled elastic tube (canonical).

A simple calculation of the stress exerted on the aorta in the case of the "jerk" model has shown that the tension experienced by the aorta as a result of a road traffic accident, is two orders of magnitude greater than that of the failure stress. In light of this the other models, particularly two and four, have been proposed in order to elucidate whether stresses, internal pressures or bending would constitute tearing or rupturing

of the aorta. Models two and four are proposed in such a form that they could be incorporated in to the current finite element model being developed by the TRL.

The causes of BTAI are difficult to postulate. Whilst fluid dynamical effects as a result of a burst of blood into the aorta following a large deaccelerative force, as in the common cases of road traffic accidents or falls, may cause some rupturing, the stress and strains experienced by the aorta must also be taken into account. In proposing the mathematical models here to account for such phenomena it may well be that the 'ideal' model includes both the mechanical properties of the wall as well as considering important fluid dynamical features relevant to this problem. In terms of a finite element model, possible ways forward include

- accounting for the curvature of the aorta;
- ensuring the elastic properties of the aorta are accounted for in the relevant constitutive equations; and
- accounting for the stresses and strains experienced by the aorta by incorporating either the torsion or canonical problems presented in this report.

Once these factors have been accounted for, fluid dynamical phenomena such as those mentioned above could then be included.

Acknowledgements

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