New ideas - Aortic and aneurysmal

Three strikes – don’t die of a broken heart

Mark L. Fielda,*, Priya Sastrya, David Richensa

a The Liverpool Heart and Chest Hospital, Liverpool, UK
b The Trent Cardiac Centre, Nottingham, UK

Received 16 May 2009; received in revised form 14 September 2009; accepted 15 September 2009

Abstract

There are multiple layers of complexity in prevention of vehicle related blunt traumatic aortic rupture (BTAR), many of which are enshrined within government policy and car design. We present a ‘layers of protection analysis’ (LOPA) based loosely on original work by Professor John Doyle, which describes these attempts to ‘design out’ the risk of BTAR following a vehicle collision. We have modified this approach to include a physiological dimension suggesting that this may be a factor in susceptibility to aortic injury following trauma. Understanding processes involved in BTAR following vehicle collisions is key to designing preventative processes.

Keywords: Trauma; Aorta; Layers of protection

1. Introduction

The long running ‘Think!’ campaign by the Government Department for Trade (DfT) (UK) publicizing the risk reduction benefits of seat belt use has introduced a new initiative for 2008/2009 (www.dft.gov.uk). The campaign highlights the added risk of blunt traumatic aortic rupture (BTAR) in victims of vehicle collisions who are not seat belted. Interestingly, the campaign mantra suggests a mechanism of action for BTAR; ‘three strikes – don’t die from a broken heart’. At its heart is a video (http://www.dft.gov.uk/think/mediacentre/237144/seatbelts) presentation showing a head-on vehicle collision with the driver sustaining BTAR. The following narrative underpins the clip: ‘Richard didn’t want to die but he couldn’t stop himself. The collision with the car didn’t kill him but he wasn’t wearing a seat belt so he continued on his journey. When he hit the inside of the car that didn’t kill him either. But his internal organs carried on travelling until they hit his rib cage, and his lungs were punctured, and the main artery from his heart was torn and that’s what killed Richard’.

This advertisement is not only powerful in its presentation and therefore, effective in relaying its message, but it is also apparently insightful in describing a putative mechanism of injury, which even to-date remains controversial. This paper addresses the evidence base for this mechanism of action for BTAR and applies the technique of ‘layers of protection analysis’ (LOPA) to examine preventative strategies in addition to seat belt use. We propose that the dynamic interplay between physical and physiological processes during a collision is key to a LOPA and design methods to prevent this injury.

2. What are the current statistics on road traffic accidents and BTAR (UK)?

In 2007 there were 247,780 road casualties within the UK according to the DfT (www.Dft.gov.uk). Of these, 27,774 were seriously injured and 2946 died. One thousand four hundred and thirty-two of these deaths involved car users, of which 34% were not wearing seat belts which has been shown to double your likelihood of death (www.ukccis.org.uk). The DfT estimate that full seat belt compliance would have saved 300 lives in 2007 within the UK. Following blunt vehicle trauma, aortic injury is the second most common cause of death after head injury and thought to account for ~20% of road fatalities. In this subgroup, seat belt use has been shown to be even more efficacious with a relative risk ratio of ~3.0 [1, 2]. The DfT have previously estimated fatalities from BTAR related to automotive accidents to be 342 annually – roughly one every day of the year in the UK. Most of these deaths occur at the scene, however, for the few who reach hospital alive (5%) the outcome is poor with an overall survival of 2%.

3. Three strikes: is the propaganda evidenced based?

As we have previously stated [3], the characteristics of BTAR have been well documented, including the circumstance under which it occurs, the transduction of impact energy through the thorax, and the evolution of the injury to the aortic wall, however, the exact aetiology which accounts for injury characteristics remains unclear. In particular, it is uncertain why despite a range of trauma...
scenarios; the injury profiles of victims are very similar with the majority of tears being initiated in the intima and occurring in a transverse fashion in the peri-isthmus region. Several hypotheses have been put forward to account for this characteristic set of lesions and these broadly centre around mechanical deformation and intraluminal hypertension. Mechanical deformation and injury of the peri-isthmus aorta during trauma may occur by a number of mechanisms including: tension, torsion, bending, and their combinations \[4, 5\]. These forces may result from a complex combination of both relative motion of structures within the thorax and local loading of tissues either as a result of their anatomy or the nature of the impact. Compounding mechanical deformations in loading the aorta following trauma are a number of mechanisms resulting in intraluminal hypertension including: luminal compression, either by osseous or diaphragmatic pinch, sympathetic discharge and the Valsalva effect \[6\]. We believe however, that supra-paressurization of the aorta occurs during anticipation of impact and the brace response, and simply serves to place the aorta in a high tensile, vulnerable state, with vessel deformation secondary to relative organ movement during trauma ultimately initiating injury \[5\]. The exact process by which some or all of these mechanisms are channelled through a ‘final common pathway’ to cause consistent injury to the isthmus, despite a wide range of trauma scenarios, remains unknown. The putative three strikes model is discussed below in the context of a LOPA.

4. What measures other than seat belt use may be preventative?

Government policy and car design contribute significantly to the ‘layers of protection’ against BTAR following an automotive collision. Professor John Doyle (Caltech) has published widely on control theory and complex systems. As part of that interest he has lectured on collision analysis (http://www.cds.caltech.edu/~doyle/CmplxNets). Protection includes legislative measures such as lane control, speed limits, appropriate luminescence and traffic calming measures. Additional car design including anti-lock breaks, collision avoidance systems, seat belts, air bags and cages add to layers of protection. In certain circumstances, particularly motor racing, protective clothing including helmets reduce the injury of contact trauma. Ideally these systems are designed to direct the driver or passenger into a ‘safe zone’ thus reducing the possibility of injury. In addition to these physical factors we hypothesize that there is an interplay between the victim’s physiology at the time of impact and the susceptibility to aortic rupture.

5. A novel LOPA

We propose that the basic LOPA mentioned above ignores the interplay of the physical processes and the physiological processes of the victim at the time of impact. We believe the analysis should in fact be three dimensional (x, vehicle; y, safety systems and z, physiology). This is depicted in Fig. 1 as a conventional LOPA. At each stage in the process we suggest there are physiological variables of importance. Underlying all these processes are other variables such as weather conditions, response time of drivers and vehicle road worthiness.

5.1. Transition Zone 5 to Zone 4

Zone 5 represents the vehicle as a safe mode of transport, functioning within legislative limits in terms of car design and road usage appropriate to road conditions. The transition from normal processes to danger involves anticipatory processes dominated by the ‘flight or fright response’. Under normal circumstances the identification of danger will elicit a range of physiological responses amounting to anticipation and leading to avoidance behaviour. These responses will naturally be blunted by the presence of drugs and/or alcohol and affected by factors such as weather. A favourable response to danger will lead to re-entry into Zone 5, while failure to manage the danger appropriately will result in transition from Zone 4 to Zone 3.

5.2. Transition from Zone 4 to Zone 3

Zone 3 represents the moment of contact between car and object. This is ‘Strike 1’. Physiologically the victim will be bracing with associated Valsalva-type response. Clearly at this point the usage of seat belts and air bags is key to dispersing impact energy away from the victim. A factor in the outcome at this point will be the make and model of car. Speed of impact will be a critical factor in the end result. Low impact speed may allow successful dispersion of energy with no injury to the victim. There is no option to return to Zone 4, however, the victim may pass through to Zone 2.

5.3. Transition from Zone 3 to Zone 2

Zone 2 represents the moment of impact between body and vehicle or ‘Strike 2’. In terms of BTAR variables such as side, front or rear impact, as well as speed and position in vehicle. Crucial at this point in preventing transition to Zone 1 is dispersal of energy through the thorax and a key variable in this is inevitably speed of impact. Other intrinsic factors in determining outcome will be comorbid pathophy-
siology of the victim such as age, hypertension and atherosclerotic disease. The putative impact of contact between organs and the thoracic cage represents 'Strike 3', however, the exact mechanism leading to BTAR remains controversial. Our belief is that a pressurized aorta, as a consequence of anticipation, undergoes mechanical distortion (bending, tension and torsion) through a relative movement of organs, and this results in BTAR rather than organ-cage contact per se. Whether Strike 3 represents an actual impact or a relative motion is a point of contention.

5.4. Transition from Zone 2 to Zone 1

Zone 1 represents the injury, in this case blunt traumatic aortic injury. Whether this results in death depends on the nature of the aortic injury.

The route from Zone 5 to Zone 1 may be direct or indirect. Following a high impact vehicle collision, it might be expected that the BTAR will occur irrespective of any preventative measures or the physiology at impact. The force of impact punches through the layers of protection producing multiple lethal injuries including BTAR. However, following low impact collision, other factors such as physiological processes at the moment of impact will be more contributory. With low impact BTAR the route to Zone 5 may be more circuitous with opportunities to intervene and modify the process of energy transduction, reducing the probability of BTAR. Intelligent impact management systems may be able to shift the moment of body contact a few milliseconds to a more favourable point.

The end result of Zone 1 will depend on the position of aorta in the spectrum that represents acute aortic syndrome which spans from intimal tear, through intra-mural haematoma or localized dissection, through to complete transection – contained or not.

6. Conclusions

The current UK government campaign to encourage seat belt use as a means to reduce BTAR is evidenced-based and insightful. There are, however, multiple layers of complexity in prevention of BTAR encompassed within government policy and car design. We have presented a ‘LOPA’ based loosely on original work by Professor John Doyle, which includes these attempts to ‘design out’ the risk of BTAR following a vehicle collision. We have modified this approach to include a physiological dimension suggesting that this may be a factor in susceptibility to aortic injury following trauma. Although no direct evidence exists for these myriad of mechanisms we believe they are intuitive. This work serves as a platform for further investigation. Understanding all processes involved in BTAR following vehicle collisions is key to designing preventative processes.

References