Finite Element Modeling of Chiasmal Compression

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It is widely agreed that compression of the optic chiasm by an external pressure causes a visual defect known as bitemporal hemianopia. However, disagreement arises over the mechanism by which this external pressure causes only crossing, nasal nerve fibres within the optic chiasm to be affected, whilst leaving temporal, uncrossed fibres predominantly unaffected. It has recently been proposed that the primary reason for this selective targeting of nasal nerve fibres is due to their crossing geometry amplifying the local pressure experienced by the fibres. This thesis project has extended previous work with the use of Finite Element Analysis (FEA) to model the stress distribution produced in the optic chiasm, when displaced by an abnormal growth in the pituitary gland. Results have shown that in the case of a pre- or post-fixed optic chiasm the region of highest stress produced sits evenly across nasal and temporal nerves. This indicates that the location of the crossing nasal nerves within the optic chiasm can not always be the reason for their being selectively targeted, and therefore some other mechanism must be responsible. Investigation has been conducted into the validity of the material properties used in the FEA and into improving the geometric accuracy of the models through the use of data from magnetic resonance images.

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I. Introduction

The growth of an abnormality, such as a tumour in the pituitary gland, can cause a visual defect known as a bitemporal hemianopia (or hemianopsia) in which a patient will lose their peripheral vision, as shown in Fig 1.

![Image of visual field chart showing bitemporal hemianopia](https://example.com/image)

Figure 1. Bitemporal Hemianopia. The dark areas on the visual field chart above show the regions of visual field loss experienced in bitemporal hemianopia (Snell and Lemp, 1998).

The abnormal growth impinges on the optic chiasm, inducing a pressure on the nerve fibres contained within. The fact that this pressure from an external body causes the loss of vision is widely accepted by the medical community; however the mechanism by which it selectively targets the nasal nerves that carry the information from the temporal visual fields is not agreed upon. Hedges (1969) and Kosmorsky et al. (2008) believe the selective damage to nasal fibres is primarily due to their location within the centre of the optic chiasm. Alternatively, McIlwaine et al. (2005) proposed that the primary reason for the selective damage was due to the crossing geometry of nasal nerve fibres inducing a higher localised stress. The aim of this thesis is to further the base of knowledge surrounding bitemporal hemianopia by testing the theory proposed by McIlwaine et al. (2005), that the selective damage to nasal nerve fibres is due to their crossing geometry. The aims of thesis were met through extending the research conducted by Howard (2008) into Finite Element Analysis (FEA) of an optic chiasm displaced by a tumour of the pituitary gland. Research has been conducted into the use of Magnetic Resonance Image (MRI) data to create a geometrically accurate 3D model of the optic chiasm and into the material models used in the FEA.

II. The Optic Chiasm

To fully understand the mechanisms relevant to bitemporal hemianopia and its causes, an understanding is required of the function and anatomy of the optic chiasm and surrounding structures.

A. Function of the Optic Chiasm

Light enters the eye through the pupil (Gray et al., 1977) in the centre of the frontal surface of the eyeball. The spherical nature of the eyeball means light entering the eye at an angle, other than perpendicular, to the plane of the pupil will travel through to the far side of the eyeball, being registered by nerves attached to the rear of the eyeball. That information is then transferred along the bundles of nerve fibres contained within the optic nerves to the brain. The human brain operates in such a way that the left side of the body is controlled by right side of the brain and right side of the body is controlled by the left side of the brain. This means that information about the right side of the body must be communicated to the left side of the body, and vice versa.

Figure 2 is a diagram of the visual pathway and shows how light from the right temporal visual field, information at the extreme right of a persons vision, progresses through the pupil and eyeball and is incident on nerve fibres on the medial (inner) aspect of the right eye. These nerve fibres are on the inner side of the eyeballs, adjacent to the nose, and are known as the nasal nerve fibres. Similarly, light originating in the centre and slight left of the visual fields, which travels toward the right eye, passes through the pupil and is registered by nerve fibres on the lateral (outer) aspect of the right eye. These nerve fibres are on the outer side of the eyeball, at the side of the head, adjacent to the temple, and are known as the temporal nerve fibres. In this way, the right side nasal nerve fibres contain information about the right hand side of the body, and therefore must transit back to the left side of the brain, requiring them to cross at some point. Similarly, the right side temporal nerve fibres contain information about the left side of the body, and therefore transit directly back to the right side of the brain. The nerve fibres from the left eye progress from the eye to the brain in a mirror image of the nerve fibres from the right eye. Both sets of nasal nerve fibres must therefore cross over each other as they track back to the

![Diagram of visual pathway](https://example.com/diagram)
brain, the point where they cross is the optic chiasm. The optic chiasm is where the two optic nerves split into four parts, the nasal and temporal nerve fibres from each eye, and contains the crossing nasal nerve fibres and the uncrossed temporal nerve fibres. Also shown in Fig 2 is an image explaining the three imaging planes of the body; the transverse, coronal, and sagittal, as they will be referred to from this point forward.

B. Anatomy of the Optic Chiasm and Optical Neural System

Approximately 1.2 million nerve fibres are attached to the rear of the eyeball. These fibres are grouped into approximately 1000 bundles, which then group together to form the optic nerve (Snell and Lemp, 1998). Figure 3 is a cross section of the optic nerve showing the central artery and vein, surrounded by the bundled nerve fibres, and sheaths of pia mater, arachnoid, and finally dura mater. The optic nerve has a diameter of 3 to 4 mm (Snell and Lemp, 1998). The optic nerves pass rearward through the skull via the optic foramen. As they exit the skull, the sheaths of dura mater surrounding optic nerves peel away from the nerves and fuse with a similar dural sheath surrounding the brain, on the inner surface of the skull, thus fixing the optic nerve in place (Gray et al., 1977). The central artery and vein leave the optic nerves before they reach the chiasm. Therefore once the optic nerves reach the optic chiasm they are composed of only the bundles of nerve fibres surrounded by the pia and arachnoid sheaths. The dura mater initially providing a sheath for the optic chiasm is a “thick and dense inelastic fibrous membrane” (Gray et al., 1977), the pia and arachnoid are both described as very thin membranes, the arachnoid being made of fibrous and elastic tissue.

The optic chiasm, also known as the optic commissure (Gray et al., 1977), is formed where the nasal nerve fibres of each nerve separate from the temporal nerve fibres and cross over to join with the temporal nerve fibres of the opposite optic nerve. A greater portion of the nerve fibres within the chiasm (hence from the nerves) are crossing nasal nerve fibres; the ratio is given by O’Connell (1973) as 53:47. In addition to the crossing nasal nerve fibres, nerve fibres exist in the centre of the chiasm, at the rear, which do not transit to the eye and simply pass across the chiasm from one hemisphere of the brain to the other (Gray et al., 1977). The paths of the nerve fibres through the optic chiasm create an x-shape stretched slightly across its width, measuring approximately 12
to 15 mm across, 6 to 8 mm front to back, and 3 to 5 mm in height (Snell and Lemp, 1998; O’Connell, 1973, McIlwaine et al., 2005). The shape of the chiasm can be seen in Fig 4. The new combinations of nasal and temporal nerve fibres from opposite eyes extend to the rear and upwards and are known as the optic tracts. The optic tracts leave the chiasm with a circular cross section and flatten as they approached the brain. The optic tracts are anchored in place by most fibres joining with the lateral geniculate body, as can be seen in Fig 2, which sits around the mid-height of the brain.

C. Location
The optic chiasm sits in the centre of the head, below the frontal lobes and in between the temporal lobes of the brain, as shown in Fig 4. The most important feature of the location of the optic chiasm, for this thesis, are the relative locations of the optic chiasm and the pituitary gland. Figure 5 shows the optic chiasm and many of the surrounding structures. The pituitary body, also known as the hypophysis cerebri (Gray et al., 1977), sits in the sella turcica, a depression in the bone of the skull also known as the pituitary fossa, and is restrained above by a membrane of dura mater with a small hole in the centre known as the diaphragma sellae. The optic chiasm can be seen to sit above the pituitary body; hence a tumour in the pituitary will grow upwards and impinge upon the optic chiasm. Whether the optic chiasm rests directly above the pituitary or not is dependent on an individual, with some individual’s optic chiasm resting significantly forward or rearward of the pituitary, these being known as pre- and post-fixed chiasms respectively.
III. Bitemporal Hemianopia

A. What is Bitemporal Hemianopia

A hemianopia is visual defect in half of one eye, the midline between the halves is usually vertical and the defect may not affect the complete half of the visual field (O’Connell, 1973). Bitemporal refers to a defect affecting both of the temporal fields. Bitemporal hemianopia is therefore blindness in the temporal halves of the visual fields of both eyes. Plainly speaking bitemporal hemianopia is the loss of the lateral (outer) parts of the visual fields in both eyes. Bitemporal hemianopia can be caused by extrinsic compression of the optic chiasm, most often resulting in displacement of the optic chiasm causing stretching of the nerves within. Chiasmal syndromes can be caused by pituitary tumors, suprasellar meningiomas, craniopharyngiomas, and aneurysms (McIlwaine et al., 2005). Figure 6 is an example of the way a tumour of the pituitary gland displaces the optic chiasm; as can be seen the chiasm tracks up and over the tumour, whereas if the tumour were not present the chiasm would sit much lower.

Figure 5. Optic Chiasm and Surrounding Structures. The optic chiasm can be seen to sit above the pituitary body. (Snell and Lemp, 1998).

Figure 6. MRI of the Brain Showing the Optic Chiasm and a Pituitary Tumour. The left image is a coronal MRI and the right image a sagittal MRI of the same patient. The optic chiasm is the slightly ‘dogbone’ shaped body identified by the red arrows and can be seen to stretch over the top of the pituitary tumour, the rounded body shown by the blue arrows. Images courtesy of Dr Glyn Thomas, Radiologist, Palmerston North Hospital, New Zealand.

Figure 1 shows the visual field of a patient with advanced bitemporal hemianopia, it is important to notice the defect’s strong respect of the midline of the visual field. This respect of the midline indicates that the
displacement of the optic chiasm selectively damages only the nasal nerve fibres, whilst leaving the temporal fibres largely unaffected, until much later stages of chiasmal compression. Studies of the literature surrounding bitemporal hemianopia have found three primary theories for the mechanism by which chiasmal compression selectively damages nasal nerve fibres; two primarily mechanical theories and one vascular theory.

B. Differential Pressure due to Location within the Chiasm

Hedges (1969) conducted an experiment in which he inflated a small balloon under the optic chiasm and observed the effect of the balloon displacing the chiasm. The experiment was conducted on fresh adult necropsy material in which the skull cap, brain, and pituitary were removed, and the optic tracts cut whilst leaving as much of the optic chiasm in place as possible. The optic chiasm was then placed over the sella turcica and the optic tracts pinned in place. A Foley catheter, effectively a long rubber tube with a balloon, able to be filled with saline solution, located at one end, was inserted into the sella turcica and inflated to no more than 2 cm$^3$ to simulate the growth of a tumour.

Upon inflating the balloon Hedges observed that the upper portions of the chiasm were stretched significantly yet the lower portions seemed to relax and were not stretched. Hedges likened this to the bending of a finger, in which the skin on the top of the finger stretches and that underneath is compressed and folds. Hedges’ observations seemed to support his theory that the progression of visual field loss was from upper to lower temporal fields, then lower nasal field, and finally to the upper nasal field. Hedges proposed that the progressive stretching of different nerve fibres caused ischaemia, inadequate blood flow, which interrupted the signal transmission along the nerve, causing the observed visual field loss. However, discussion on Hedges’ paper by Harrington, contained at the end of the paper, indicates that others within the medical community disagree with Hedges progression of visual field loss, the accuracy of the observations made in experimentation, and associated explanation. Particularly Hedges’ analogy of the skin on the underside of a finger folding when bent seems inadequate, as the chiasm has been stretched across a tumour below it, not bent like a finger where the bones would act as some form of neutral axis. Hedges’ theory is primarily a mechanical theory, stating that the stretching of the nerve fibres and the order in which they are stretched is the cause of the initial loss of the temporal visual fields.

Kosmorsky et al. (2008) conducted a very similar experiment to Hedges (1969) in which a Foley catheter was used to displace the optic chiasm in situ, and the pressure generated in the chiasm was measured. This experiment was performed in response to the theory proposed by McIlwaine et al. (2005) as Kosmorsky et al. believed that it relied too heavily on uniform pressure generation across the chiasm. Craniotomies were performed on five cadaveric specimens to expose the optic chiasm. The brains of the specimens were detached and placed on Styrofoam blocks to expose the optic chiasm without it being necessary to damage the chiasm. A pediatric gauge Foley catheter was placed underneath the optic chiasm and inflated. Needles linked to pressure transducers were inserted into the central and temporal aspects of the chiasm and moved simultaneously with the inflating balloon, to avoid puncture. Figure 7 is a photograph of the experiment by Kosmorsky et al. showing the chiasm and pressure sensing needles.

Kosmorsky et al. (2008) experiment obtained usable results in only two of the five specimens, with the three unusable sets of data attributed to deterioration of the specimens prior to testing. The results showed that higher
pressure was consistently generated in the centre of the chiasm over the temporal aspects and bulging was seen to occur in the centre of the chiasm. They concluded that the results showed non-uniform pressure generation, and that greater stress in the centre of the chiasm than in the temporal aspects was the most likely reason for a compressive lesion of the chiasm to cause selective damage to the nasal nerve fibres, and the subsequent visual field defects.

C. Restriction of Blood Supply to the Optic Chiasm

Bergland and Ray (1969) performed inspection of 475 specimens during autopsy to study the vascular supply to the optic chiasm as they felt that current explanations did not adequately explain the fact that most tumours showed significant spreading along the optic chiasm, optic nerves, and optic tracts and did not impress upon only the centre of the chiasm. They found that the temporal aspects of the chiasm were supplied by superior and inferior (above and below) sets of blood vessels, yet the central aspects of the chiasm were supplied by an inferior set only. This led them to conclude that in the presence of a pituitary tumour, or similar abnormality, compressing the chiasm from below that the inferior blood vessels would be constricted, reducing the blood supply to the temporal nerve fibres, and completely removing the supply to the nasal nerve fibres, as can be seen in Fig 8.

Figure 8. Ischaemia of the Central Chiasm. Bergland and Ray (1969) proposed the visual loss was due ischaemia. A. Normal chiasm and blood vessels. B. Chiasm with tumour beneath, restricting blood flow (Kosmorsky et al., 2008)

Bergland and Ray (1969) found a number of inconsistencies with a solely mechanical (stretching) theory for bitemporal hemianopia, including the speed of recovery of vision once compression by a tumour was removed, the lack of altitudinal field defects (significant differences in the loss of vision about a horizontal plane of symmetry through the centre of the eye) though tumours very regularly impressed upon the inferior aspects of the chiasm, and the observation of large variability in the geometry of optic chiasms and tumours whilst the visual field defect remained ‘monotonously similar.’ Kosmorsky et al. (2008) disagreed with Bergland and Ray (1969) on the grounds that abnormalities displacing the chiasm from above produced the same visual field defects as those from below, yet both the temporal and central aspects of the optic chiasm would still have blood supplied by the inferior blood vessels. This however seems in contradiction with the paper by Bergland and Ray in that it states “compression of the chiasm from above by anterior third ventricle tumours is attended by patterns of visual field loss that are much less frequent and much more irregular even though the degree of chiasmal
distortion often equals that seen in pituitary tumours” showing that their theory did, to some extent, account for tumours displacing the optic chiasm from above.

This theory also fails in some respects to explain the distinct midline present in bitemporal hemianopia. Whilst noting the very basic medical knowledge of the author, it would seem if the presence of a tumour below the chiasm cuts off the blood supply to the nasal nerves in the centre of the chiasm, it would be expected that the inferior blood supply to temporal nerve fibres would also be cut-off, therefore causing damage to lower temporal fibres and therefore upper nasal visual fields. If the explanation for the lack of damage to upper nasal visual quadrants is the presence of cross-flow from upper to lower temporal fibres, then what eliminates the possibility of cross-flow from temporal to nasal fibres? It is widely recognised that sufferers of bitemporal hemianopia tend only to present for medical help once the visual defect is relatively advanced, as the initial loss of the furthest peripheral vision goes unnoticed, indicating long periods of time pass before treatment. If, as this theory suggests, the loss of blood supply to the nerve fibres is the reason for the visual defect, then it could be expected that cell death would occur in the long periods before treatment, however as Bergland and Ray (1969) note in their paper, vision rapidly returns in the once compression by a tumour is removed.

D. Crossing Geometry Increasing Local Stress within Nasal Nerves

Dissatisfied with the stretching and vascular theories previously proposed, McIlwaine et al. (2005) proposed a new, mechanically based theory for the selective damage to nasal nerve fibres. The theory proposed is best explained through the use of Fig 9, which shows how crossing nerve fibres, assumed to cross perpendicularly, have a relatively lower area of contact than parallel nerve fibres, causing a locally higher stress in the crossing fibres. If nerves with a diameter, d, are in parallel contact, assuming a flattened area of contact \( p \pi d \), where \( p \) is the proportion of the circumference flattened in contact, it can be shown the surface area in contact is \( p \pi d^2 \). Similarly it can be shown the relative contact area between perpendicular fibres is \( p^2 \pi d^2 \), giving a ratio of contact 1:1 (parallel:perpendicular), where for \( p \) less than approximately one third crossing fibres will have a lesser contact area. The stress on the nerves is given by the force divided by the area; therefore the lower contact area creates a higher stress in the crossing nerves.

Both Hedges (1969) and Kosmorksy et al. (2008) concluded that the most likely mechanism for selective damage to nasal nerve fibres was that their location within the chiasm exposed them to greater deformation and consequent higher stress levels. However, these results were based on experiments modeling a tumour with relatively un-deformable rubber balloons positioned centrally below the chiasm and rely on a tumour impinging in only the centre of the chiasm and not across its width, failing to account for non-uniform growth and decentralised position of a tumour. Figure 6 supports this point, showing a pituitary tumour with an arguably flattened top that pushes somewhat uniformly across almost the entire width of the chiasm. Given their theory of location within the optic chiasm being the primary factor in bitemporal hemianopia, displacing the chiasm from a position other than the centre could be expected to cause a peak stress away from the centre of the chiasm, and therefore not necessarily in a region of only nasal nerve fibres. The theory proposed by McIlwaine et al. (2005) provides a mechanism explaining the selective targeting of nasal nerves even in cases where the loading created
by a tumour is not centralised. It is important to note that these theories are not mutually exclusive, it is apparent that under certain loading conditions nasal nerve fibres will be exposed to higher stress than temporal nerve fibres, combining with the crossing geometry to exacerbate the higher localised stress. Additionally a tumour will likely impinge upon blood vessels in the vicinity, restricting the blood flow to the chiasm, possibly adding to visual field loss due to stress created in the nerve fibres. McIlwaine et al. (2005) and Kosmorksy et al. (2008) both acknowledge the need for further research in this area and recommend the use of FEA to further analyse the structures involved.

IV. Finite Element Analysis of Biological Structures

Finite Element Analysis, also known as Finite Element Modeling, is a method of analysis that allows complex, and otherwise unsolvable, structures and geometries to be broken down into manageable sized pieces for which simple solutions can be obtained. Hutton (2004) describes FEA as obtaining an approximate solution to a boundary value problem. The solution of a boundary value problem is where one or more dependent variables satisfy a differential equation everywhere within a known domain of independent variables, and satisfy specific conditions on the boundary of the domain.

The structure to be analysed, in this case the optic chiasm, is divided up into elements which are given the required material properties. The structure, when divided up into the elements to be analysed, is known as the mesh. Constraints are applied to the model and finally forces are applied and the effect those forces have on each element, whether directly or transmitted from another element, are computed, giving an approximate solution to the problem. The finite number of elements used in the calculations means there will be error present in the solution due to an element approximating the average behavior of the material particles in the region it represents. Therefore, the number of elements used in the solution is proportional to the accuracy, so the more elements used, the more accurate the solution. The accuracy of a solution approaches full accuracy asymptotically, i.e. as more elements are used the accuracy increases, but the increases in accuracy will become smaller. FEA was not a practical tool prior to computers, and though now possible it is still limited by the speed and memory of the computer to be used. An increase in the number of elements obviously creates an increase in the number of calculations to be performed, so as the number of elements increases, so does the computational cost. This means a balance must be found that maximises accuracy and minimises computation.

Finite Element Analysis is often used in engineering to model structures such as bridges, or components of aircraft, however it can still be applied to the biomedical field, it simply requires the provided material properties to be those of the relevant tissues, however this is not so simple, as will be discussed later. The use of FEA has proven to be very useful in biomedical applications as Cirovic et al. (2006) showed when modeling blunt impact trauma to the eye and surrounding structures.

V. Previous Work

As suggested by McIlwaine et al. (2005) and later by Kosmorksy et al. (2008), Howard (2008) performed FEA simulations of the stresses created in the optic chiasm when displaced by a pituitary tumour. As with this research, Howard’s work was conducted as an undergraduate thesis project at the University of New South Wales at the Australian Defence Force Academy (UNSW@ADFA). Howard’s work primarily involved significant reading around the subject to obtain the data required to set up a finite element model of the optic chiasm. Figure 10 shows the result of Howard’s work in setting up a finite element model of the chiasm. The model represents the chiasm with a single, homogenous nerve fibre given material properties found for neural tissue. The optic chiasm, optic nerves and optic tracts were represented by two perpendicularly crossing cylinders with chamfers at the join.

Howard experimented with using separate nerve fibres, however, the approximately 2.4 million fibres in the chiasm (Snell and Lemp, 1998) make modeling individual fibres a practical impossibility, and using low numbers of fibres proved difficult and would not model the effects adequately. The optic chiasm had a sheath of sclera (the fibrous tissue forming the white of the eye) 0.5 mm thick around the outside. Below the chiasm is a spherical tumour modelled as a balloon of sclera material, resting in the sella turcica modelled as a hemispherical depression in bone. The bone was given material properties of structural steel, to make it undeformable relative to the tumour as its actual behavior was deemed unimportant. The tumour was ‘inflated’, in a manner analogous to Kosmorksy et al. balloon, by applying an arbitrary pressure of 1 Mpa to its inner surface. The model was made symmetrical in two planes to simplify the model and reduce the computational cost, giving the quarter symmetry model shown in Fig 10.

The results obtained from Howard’s model of the optic chiasm are displayed in Fig 11 in comparison with the results obtained by Kosmorksny et al. (2008). As can be seen the results are very similar, verifying that the FEA model and methods used are relevant and usable. Additionally Howard (2008) created models of crossing and non crossing nerves, these models are shown in Fig 12, which showed that, using a nominal 1000 Pa pressure applied to the upper surface of the nerves, crossing nerves developed a peak stress 25% higher than that of non-crossing nerves. Howard’s model contained geometric simplifications and assumed linear elastic material
properties and small deflections. Howard recommended improvement of the geometry and further investigation of the material properties used. Improvement of the geometry could be made through building a model using measured data, such as that from an MRI.

Figure 10. Finite Element Model of Optic Chiasm by Howard (2008). The figure is an image of the model made by Howard, in which can be seen the chiasm (top) consisting of a single homogenous nerve fibre surrounded by a sheath of dura mater, and a tumour (middle) resting in the bone of the sella turcica (bottom). The image on the left is the geometry of the model, and the image on the right is the deflected quarter symmetry model showing the distribution of stresses within.

Figure 11. Results from FEA Model of the Optic Chiasm. The pink and green dots represent Kosmorksy et al. (2008) results. The figure shows that the results obtained by Howard are very close to those obtained by Kosmorksy et al. (Howard, 2008).

Figure 12. Crossing and Non-Crossing Nerves. The model of crossing nerves (left) developed a maximum stress 25% greater than non-crossing nerves (right) (Howard, 2008).
VI. Aims and Methodology

The aim of this thesis is to further the knowledge base surrounding the cause of bitemporal hemianopia. This will be done primarily through extending the work of Howard (2008) in the use of FEA to model the distribution of stresses produced in the optic chiasm when displaced by a pituitary tumour. This thesis aims to determine whether one of the two mechanical theories proposed can be shown to be the primary mechanism for selective damage to nasal nerve fibres. The project aims to deliver modified finite element models in an attempt to improve the geometric accuracy, adapt the behaviour of the structures involved to model more closely what might be expected under loading, and model different loading situations. The project also aims, through investigation, to deliver a method of obtaining a 3D FEA model from MRI data and information on the legitimacy of assuming a linear elastic model for material properties.

A methodology has been developed for this thesis that encompasses the aims whilst recognising some unique requirements and constraints. The methodology recognises that the analysis of this thesis tends toward a more qualitative than quantitative focus, and has been created accordingly. There can be little challenge to the statement that a crossing nerve fibre will experience a higher localised stress than a parallel nerve fibre and this thesis does not aim to prove that point any further. Therefore to determine which theory is the more relevant, involves analysing the distribution of stress within the chiasm to test whether location within the chiasm can account for the selective damage to nasal nerve fibres in all situations.

The comparison of the relative stresses produced within the optic chiasm is the primary aim of the FEA to be conducted in this thesis. Therefore the accuracy of the stress distribution through the cross section of the optic chiasm is of prime importance. In FEA the peak deflection or stress created is often the measure of accuracy, and once the peak deflection or stress shows little change with increasing elements, the solution is said to be accurate. However, in the work of this thesis the peak stress may show little change with large increases in the number of elements, yet the smoothness of the distribution of stress within the optic chiasm may be heavily dependent on the size of the elements used. This thesis will not use the usual approach to verifying the accuracy of the solution of comparing the change in peak stress with change in number of elements. Rather it will increase the number of elements to the limit imposed by the computing power available, in addition to comparing the relative change in stress distribution with increasing elements. This will be done in conjunction with comparing the data produced from FEA to the minimal experimental data available, in an attempt to ensure the stress distributions created are relevant and usable.

VII. Finite Element Analysis of the Optic Chiasm

A. Creation of the Baseline Model

The first step in furthering the work previously conducted by Howard (2008) was to gain a full understanding of his work and the reasons for the choices he made. The best way to gain this understanding was to read his report whilst recreating his final finite element model. Recreation of Howard’s model gave not only an understanding of the programs and procedures used and decisions made, but also served as a quality check to ensure the correct use of the analysis tools. It was assumed that results matching those obtained by Howard, particularly the peak stress and distribution of stress, indicated correct use of the analysis programs. The decisions Howard made relating to the FEA of the optic chiasm are explained in his report. Howard decided to use ANSYS Workbench for the FEA of his thesis. To avoid building a new model from scratch and for continuity with other work done on this topic at the UNSW@ADFA the modeling of this thesis was also done in ANSYS Workbench. From this point forward, unless otherwise specified, references to ANSYS in this thesis refer to ANSYS Workbench, not the classic version of ANSYS. Data extracted from the simulations and used to plot the figures is in Annex A.

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Table 1. Summary of Material Properties. The table, taken from Howard (2008), summarises the material properties used by Howard, including the source of the data.

The geometry used in recreating Howard’s Model is as explained in ‘Previous Work’ above and was taken from one of his later simulations. The cylinders representing the optic chiasm, nerves and tracts were chosen to be 20 mm in length, based on a typical length of optic nerves and tracts, and 5 mm in diameter to give the entire chiasm a typical thickness of 5 mm. The homogenous nerve fibre was given the material properties of neural
tissue. The Material data of human neural tissue is scarce, so it was assumed that neural tissue was similar to that of porcine (pig) neural tissue, as was done by Miller et al. (1999). The sheath of the chiasm was set at a thickness of 0.5 mm and was given the material properties of sclera; as this was taken to be similar to dura mater, has similar properties to pia mater, and was used by Bellezza et al. (2000) to model the sheath of the optic nerve. The tumour was modelled as a balloon with an outer radius of 6 mm and thickness of 1 mm and given the material properties of sclera, as it was assumed that a tumour would have similar properties. The bone was modelled as structural steel, as its behavior was unimportant since its deflection was negligible. Table 1 summarises the data Howard used for material properties.

Recreating Howard’s model and ensuring it ran the same was a relatively time consuming process, however it gave good insights into the use of ANSYS and ensured the author had the necessary skills to further the FEA component of this thesis. The recreated model could not be made to converge with the exact settings used by Howard, most likely due to slight differences between the geometry used in the recreation and from Howard’s original model. The use of a ‘frictionless’ contact setting between the sheath and homogenous nerve fibre in the recreated model caused buckling of the sheath, an unexpected behaviour. To eliminate this buckling, the setting was changed to ‘no separation’ which allowed frictionless movement parallel to the contact face, but did not allow separation of the faces, therefore eliminating the buckling. It is assumed that the change in contact setting did not have a significant impact on the stress distribution.

Figure 13: Howard’s (2008) Model and Recreated Model. Howard’s model (left) and the recreated model (right) show good similarity between distributions of stress.

Figure 13 shows Howard’s original model alongside the recreated model and as can be seen the stress distribution is very similar. The peak stress in Howard’s model was approximately 5200 Pa and the peak stress in the recreated model was 5500 Pa. The similarity between these results was taken to be enough to assume the recreated model was correctly set-up and producing usable results. Howard’s research produced a working model and proved the viability of using FEA to model the stresses present within the optic chiasm, however the model had many areas of concern which limited the usability of the results, for the purposes of this thesis. These areas of concern were the focus for a number of simple modifications made to the model.

As can be seen in Fig 13 the mesh of Howard’s model is very coarse, and the results are very mesh dependent. The mesh dependency can be seen as the different colours representing areas of differing stress levels closely follow the shape of the mesh (black lines), for example the light blue area surrounding the high stress concentration in the centre of the chiasm is quite square in shape, not what might be expected, as it follows the mesh. This thesis is concerned with the distribution of stress within the optic chiasm, so it is desired that results are largely independent on the shape of the mesh. The nominal element size in Howard’s model was 4 mm with refinements on the edge of the chiasm created by quarter symmetry, creating a mesh with 4823 elements. To address the coarseness of the mesh initially the average element size was reduced to one quarter of the original size and the refinements were kept. Refinements reduce the size of the elements in the specified area, creating denser mesh where desired whilst leaving less important areas with larger elements, to reduce the computational cost. The refinements appeared to be useful in creating denser mesh in critical areas of the chiasm, however, it was later discovered that as the refinements were only applied to a face, they were of little use. They increased the mesh density on the face of the critical areas, however sub-surface the mesh was still coarse, producing distributions of stress through the cross section of the chiasm that were still mesh dependent. It is believed possible to apply refinements to a volume section of a body; however the limited skills of the author meant that a viable way to do this was not found. For this reason refinements were removed, except for a refinement on the top corner of the tumour, and on most final models throughout the FEA an element size of 0.5 mm was used on the optic chiasm and sheath.

The way in which Howard smoothed the contact region between the two cylinders, to give the chiasm its width and length, was to use chamfers. Chamfers draw a straight line between the two surfaces to be joined, at the radius specified by the designer. The chamfer removes the 90° “hard” corner at the join but the straight line...
creates two 135° ‘hard’ corners, as shown in Fig 14. These ‘hard’ corners are discontinuities in the surface of the structure, and therefore possible stress concentrations. Although the chamfers were considered unlikely to have significant effect, they were easily replaced with blends, which replace the 90° corner with a smooth radius between the two surfaces. The blends created a smooth surface on the chiasm removing the possible stress concentration. The result of the change from chamfers to blends is shown in Fig 14, and it can be seen the chamfer produces a much more realistic looking geometry, as it produces a smooth and continuous surface as observed on a real optic chiasm.

Figure 14. Chamfer and Blend. The right image is that of the optic chiasm with a chamfer, the left image a blend, creating a smooth surface.

The optic chiasm, as modelled by Howard, had a homogenous nerve fibre surrounded by a sheath of sclera with a thickness of 1 mm, or as found in some models 0.5 mm, this is however somewhat thicker than the pial and arachnoid sheaths actually surrounding the optic chiasm. Figure 3 was used to estimate the thickness of the pial and arachnoid sheaths surrounding the optic nerves, and gave a thickness of 1/16 of the radius, and therefore 1/32 of the diameter. The diameter of the optic nerves in the model was set as 5 mm hence giving a sheath thickness of 0.16 mm. The thickness of the actual sheaths may be some what less than this as much of the measured ‘sheath’ was the sub-arachnoid space, between the pia and arachnoid, which is filled with mainly fluid. The thickness of the sheath to be used was rounded to 0.2 mm, for simplicity and to assist with the modeling, as a very thin sheath caused problems with convergence of the model and also required many more elements to be used to mesh the sheath, increasing the computational cost.

Table 2. Summary of Optic Nerve Head Material Properties. The table quotes the Young’s Modulus for various brain related tissues from a number of sources (Sigal et al., 2004)

<table>
<thead>
<tr>
<th>Tissue/Species</th>
<th>Author(s)</th>
<th>Young’s Modulus (MPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sclera Tree Shrew</td>
<td>Philips and McBrien</td>
<td>2.28</td>
</tr>
<tr>
<td>Sclera Tree Shrew</td>
<td>Siegwart and Norton</td>
<td>0.69–18.3</td>
</tr>
<tr>
<td>Bovine</td>
<td>Smolak</td>
<td>3.8–9.0</td>
</tr>
<tr>
<td>Human</td>
<td>Woo et al.</td>
<td>5.5</td>
</tr>
<tr>
<td>Human</td>
<td>Friberg and Lace</td>
<td>1.8–2.9</td>
</tr>
<tr>
<td>Monkey</td>
<td>Downs et al.</td>
<td>2.9–5.5</td>
</tr>
<tr>
<td>Porcine</td>
<td>Sport E, et al. JOVS 2003;44:ARVO EAbstract 33:18</td>
<td>0.5</td>
</tr>
<tr>
<td>Human</td>
<td>Battaglioni and Kamn</td>
<td>4.76</td>
</tr>
<tr>
<td>Human</td>
<td>Kobayashi et al. 10</td>
<td>5.5</td>
</tr>
<tr>
<td>Neural tissue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Porcine brain</td>
<td>Müller</td>
<td>0.03</td>
</tr>
<tr>
<td>Bovine brain</td>
<td>Guillaume et al.</td>
<td>0.046</td>
</tr>
<tr>
<td>Monkey brain</td>
<td>Merz et al.</td>
<td>0.010</td>
</tr>
<tr>
<td>Bovine retina</td>
<td>Jones et al.</td>
<td>0.020</td>
</tr>
<tr>
<td>Cat spinal cord</td>
<td>Chang et al.</td>
<td>0.2–0.6</td>
</tr>
<tr>
<td>Rabbit spinal cord</td>
<td>Ozawa et al.</td>
<td>0.035</td>
</tr>
<tr>
<td>Lamina cribrosa</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Porcine</td>
<td>Sport E, et al. JOVS 2003;44:ARVO EAbstract 33:18</td>
<td>0.1</td>
</tr>
<tr>
<td>Fit to human</td>
<td>Edwards and Good</td>
<td>0.14–0.38</td>
</tr>
<tr>
<td>Monkey</td>
<td>Bellezza et al.</td>
<td>0.077–0.405</td>
</tr>
<tr>
<td>Pia mater</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Human</td>
<td>Zhivodrov et al.</td>
<td>1.44–4.65</td>
</tr>
<tr>
<td>Human</td>
<td>Our computations based on measurements by Mazuchowski and Thibault</td>
<td>2.5–65</td>
</tr>
<tr>
<td>Human</td>
<td>Brands</td>
<td>1.86 (Shear modulus)</td>
</tr>
</tbody>
</table>

The Young’s moduli chosen for this study were: sclera, 3 MPa; lamina cribrosa, 0.3 MPa; neural tissue, 0.03 MPa; pia mater, 3 MPa; and central retinal vessels, 0.3 MPa. Results of a parametric study based on these values are shown in Figure 7.

*As cited by Kleiven.†

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A review of the anatomy and properties of the optic chiasm late in the work of the thesis revealed that material properties of pia mater are listed in Table 2, taken from Sigal et al. (2004). The sheath was originally modelled as sclera material, due to its assumed similarity to dura mater. However, given that the sheath surrounding the optic chiasm is made of only pia mater and arachnoid the material properties of the sheath were changed from sclera to pia mater. Table 2 shows there is significant variation in the value of Young’s Modulus for pia mater, so a typical mid-range value of 3 MPa has been used, as was used by Sigal et al. (2004).

The results of the modifications to the model made by Howard (2008) are shown in Fig 15, showing the thinner sheath, blends, and denser mesh. As can be seen the distribution of stress is much the same as that from the original model, but is much less mesh dependent, as the size of the elements is much smaller than the chiasm, so results in a particularly area are not dominated by a single element. This model will be referred to as the baseline model, as it gives a benchmark against which other, more major modifications can be compared. The results from the baseline model are compared to Howard’s model in a graph of stress vs location in Fig 16. Despite a much more coarse mesh, Howard’s model shows quite a smooth distribution to stress, that maintains high stress levels for much of the lateral distance away from the centre. This higher level is likely due to the thicker and stiffer sheath, surrounding the homogenous nerve fibre, absorbing much of the deforming energy and in a way regulating the deformation experienced by the nerve fibre.

Figure 15. Baseline Model. Shows the basic model of this thesis, to which other models and modifications can be compared.

Figure 16: Baseline Model Compared to Howard’s (2008) model.
B. Spreading of Contact Between Tumour and Optic Chiasm

The experiments conducted by Hedges (1969) and Kosmorsky (2008) both used relatively un-deformable rubber balloons to displace the chiasm. These balloons appeared to push right in the centre of the chiasm and lend credence to the theory that the location within the chiasm was the primary reason for the selective damage to nasal nerve fibres. The modeling of the tumour as a balloon continued in the research by Howard (2008), and the results show that the tumour pushes quite locally in the centre of the chiasm, with little deformation to the shape of the chiasm. The discussion by Harrington (at the end of Hedges, 1969) and by Bergland and Ray (1969) explain that the growth of a tumour, and hence the way in which it impinges upon the chiasm is rather non-uniform and often not centralised, certainly it can be taken that the tumour will not always push very directly and locally in the centre of the chiasm. Information received from a number of sources in the course of this thesis have suggested that a tumour is softer than how it has previously been modelled, and deforms much more to the shape of the chiasm as it grows. MRI data, from a number of different sources, viewed in the course of this thesis has shown that a tumour tends to grow and push on the chiasm across much of its width. An example of this is displayed in Fig 17, showing a tumour growing slightly to the left of centre of the chiasm (from the patient’s perspective, right side from the reader’s perspective). It is important to note the shape of the tumour, as it can be seen that the radius of the surface in contact with the chiasm is much larger that the radius of the actual tumour, that is the top of the tumour is much flatter than the top of a spherical balloon.

The behaviour of tumours deforming to the shape of the chiasm could be explained by the nature of tumours as a mass of dividing cells, where an abnormality has caused a loss of control of the cell division (Evan and Vousden, 2001). As the tumour contacts the chiasm, dividing cells from the inside of the tumour will push the tumour outwards, analogous to the behaviour of an expanding balloon, but in addition to this, those cells at and/or near the surface of the tumour will divide, quite likely taking the path of least resistance, i.e. around the chiasm, therefore causing the tumour to deform to the shape of the chiasm. This indicates that the current method of modeling the tumour needs to be modified. As can be seen in Fig 15, the current area of contact between the chiasm and the tumour is quite small, and there is limited deformation of the tumour around the chiasm. The aim of this section of work was to spread the region of contact between the tumour and chiasm by altering the properties of the tumour and causing it deform more to the shape of the chiasm.

Deforming the shape of the tumour could be done in three different ways. The first was to decrease the thickness of the balloon, reducing its strength, and therefore causing greater deflection. The second was to reduce the stiffness of the balloon, by reducing the Young’s modulus of the material. The third method was to increase the radius of the balloon, so the initial radius that came into contact with the chiasm was more like that expected at full deflection. Decreasing the thickness of the balloon was a very simple method intended to increase the area of the tumour in contact with the underside of the optic chiasm. The intention was the reduced thickness of the balloon wall would reduce its overall rigidity. The thinner the balloon wall, the more it would be expected to deform around the chiasm. The application of this method was more limited than expected, as once the balloon wall thickness was reduced the meshed elements forming the balloon were subject to severe stretching and large deformation, making converged solutions hard to achieve. No usable results were obtained.

The second method of spreading the region of contact was to decrease the stiffness of the tumour balloon by decreasing the Young’s Modulus of the material. The tumour was originally given the material properties of sclera, as no data has been found for the material properties of a tumour. This assumption was necessary for the initial model and it has been assumed in this thesis that using the material properties for sclera gave the tumour a more rigid structure than expected. Sinkus et al. (2005) reported that the ratio of the shear modulus of a tumour
to that of healthy tissue varied from 1.4 to 3.3, therefore implying a similar ratio between Young’s Modulus. This indicates that the Young’s Modulus used for the tumour in this modeling is much too large, as it is over two orders of magnitude larger than that of neural tissue. For this reason the Young’s Modulus for the material used in the tumour was reduced from its original value, of 5.5 Mpa, in several steps down to the value used for neural tissue, 0.03 Mpa. The model could not be made to converge to a solution when the Young’s Modulus for the tumour was dropped to 0.03 MPa, presumably as the very soft material could not withstand the high pressure applied to its inner surface. Figure 18 shows the progression of softening the tumour, with Young’s Modulus values of 5.5 MPa, 4.1 MPa, 2.7 MPa, and 1.3 MPa. It can be seen that the area of maximum stress within the chiasm spreads as the tumour is softened, although it must be noted that the apparent area of maximum stress is heavily dependent on the user altering the colour levels. The area of contact between the sheath and chiasm does not increase as expected, in fact there appears to be less contact in the models with softened tumour properties. This result is not what was expected. Additionally, the model with a Young’s Modulus of 1.3 Mpa for the tumour shows a very odd stress distribution. This, combined with the area of contact not spreading as expected, leads to the conclusion that the FEA may be subject to error from assuming linear behaviour, that being that the modeling has been conducted with large deflection turned off. This assumption and its ramifications will be discussed later. It is noted that the sheath of the chiasm in these models has the original thickness of 0.5 mm, as this modeling was conducted prior to the reduction of the sheath thickness.

Figure 18. Progressively Softened Tumour. Images of a deflected models where the tumour has been softened progressively. Clockwise from top left the Youngs Modulus of the tumour changes from 5.5 MPa (the normal model) to 4.1 MPa, 2.7 MPa, and finally 1.3 MPa. All models had a tumour internal pressure of 1 MPa.

The final method of increasing the area of contact between the tumour and chiasm was to increase the radius of the balloon representing the tumour. The tumour was originally modelled as a balloon of radius 6 mm sitting in a bone with a hemispherical depression of radius 6 mm. Howard (2008) based this on the geometry of the diaphragma sellae and sella turcica, to give the likely size of a tumour. However, Fig 6 shows a tumour that has become ‘peanut’ shaped due to it being constricted around its middle by the top of the sella turcica and the diaphragma sellae, thus indicating that the radius of the tumour is larger than the geometry of these structures. The tumour in Fig 6 appears almost as wide as the optic chiasm, giving a radius of up to 7.5 mm. Additionally, Hedges (1969) inflated the balloon of the Foley catheter to a maximum of 2 cm³ which, assuming a spherical shape, gives a radius of 7.8 mm. Additionally, the top of the tumours in Fig 6 and Fig 17 appear to have flattened, so the radius of the surface in contact with the chiasm is greater than the tumour’s radius. The radius of the tumour and the hemispherical depression used in modeling was increased to 10 mm, as trial and error showed that this radius appeared to give a contact area more like that seen in MRI data. This tumour radius is
larger than real, typical tumour radii, making the tumour much larger overall, however the only region of interest is where the tumour and chiasm meet, and this area shows a radius much more like that seen in MRIs. The same pressure of 1 MPa was applied to the inner surface of the tumour to ‘inflate’ it. Figure 19 shows the result of increasing the radius of the tumour, and it can be seen that the area of contact between the chiasm and tumour has increased significantly along the optic nerves. This model was a relatively early model and as a result the mesh is coarser than that on later models, meaning results from this model will be somewhat mesh dependent.

![Figure 19. Larger Tumour.](image)

Figure 19. Larger Tumour. The figure shows that the increased tumour radius has increased the area of contact between the tumour and chiasm.

The effects of softening the tumour and increasing the radius were combined in an attempt to get the best spreading of contact between the chiasm and tumour. An attempt was made to combine this with the effects of thinning the tumour wall; however the large size of the tumour, and thin wall created large deflection in the elements, causing problems with convergence of the model, most likely as the analysis assumed no large deflection. The combined model had a tumour radius of 10 mm, the same as previous model, and used a Young’s Modulus of 4 Mpa, at which it was considered the tumour would be softer, without causing convergence problems. The combined model is shown in Fig 20, where it can be seen the area of contact is similar to that of the model with only a larger tumour radius. This model has a much denser mesh, and it can be seen that the results appear much less mesh dependent.

![Figure 20. Big Soft Tumour.](image)

Figure 20. Big Soft Tumour. The figure shows the deflected shape of a model with a larger tumour in which the Young’s Modulus was reduced to 4 MPa. The right hand side is a lateral (coronal) section through the chiasm showing the contact area.

The results show that the area of contact has increased, however not exactly as hoped, as Fig 20 shows that the chiasm and tumour are not in contact for much of the lateral distance across the chiasm. The way in which
the geometry has been created means the bottom of the circular optic nerves, and the point where they meet in the centre, are the lowest points, and the tumour spreads along these as hoped. However the sides of the chiasm, as they have been created with blends between cylinders, taper upwards immediately from the centre and therefore do not come into contact with the tumour. This is not like the contact seen in Fig 6 and Fig 17, where a coronal slice (equivalent to the lateral slice shown in Fig 20) shows the tumour in contact across most of the chiasm.

Figure 21 compares the stress distribution though the chiasm for the progression of tumour softening, increase in tumour radius, and combination. The data was taken from a lateral (coronal) slice of the chiasm on a line through the centre of the peak stress region to the edge of the chiasm at mid-height, and has been plotted as a percentage of max stress within the chiasm vs. location in mm from the centre. The reason for plotting percentage of maximum stress is that as the tumour is softened it absorbs less of the energy of the pressure inside, so will deform further and therefore deflects the chiasm further. The greater deflection is likely to cause larger stress values within the chiasm so one model cannot be directly compared with another, therefore percentages have been used. It can be seen that reducing the Young’s Modulus of the tumour material had little effect on the distribution of stress within the chiasm. This is not overly surprising, as the contact area between the tumour and chiasm did not appear to increase, although it was expected it would. It can also be seen that increasing the radius of the tumour had no significant impact on stress distribution; this was unexpected as the contact area had increased. However as explained above, the contact did not increase in the intended area across the chiasm, giving the likely reason for the lack of change in results. The results show that in the case of a central loading nasal nerves are still exposed to higher stress levels due to their location within the chiasm, and comparing the results to those of Howard (2008) it would appear that the location with the chiasm has a greater effect than crossing vs non-crossing geometry, as Howard’s model showed a stress increase of only 25% in a crossing nerve. The peak stress developed in the models ranged from 5900 to 7800 Pa, showing the models all developed similar maximum stress to the baseline model and there were no significantly outlying results.

Figure 22 shows the distribution of stress along the centre of the optic nerve and as can be seen there is a significant difference in stress through a lateral cross-section between the normal model and models with modified tumours. The ‘Big Tumour’ results refer to models with increased radii tumours. Results with ‘E= x MPa’ refer to a model with a reduced Young’s Modulus for the tumour.

Figure 21. Plot of Percentage of Maximum Stress vs Location Across Optic Chiasm. The plot shows no significant difference in stress through a lateral cross-section between the normal model and models with modified tumours. The ‘Big Tumour’ results refer to models with increased radii tumours. Results with ‘E= x MPa’ refer to a model with a reduced Young’s Modulus for the tumour.
impinging in the centre would progressively deflect into the chiasm as it grew, but in this case for a tumour to deflect into the chiasm as it grows, it needs to grow higher than the centre, an unlikely behaviour. The high pressure in the balloon makes the tumour ‘grow’ however it also gives the tumour much of its rigidity. This can be seen by blowing up a normal rubber balloon, with only a little air in the balloon, it is still soft and deformable, however, as the pressure increases the balloon gets stiffer, and deforms less. These results show that the geometries and modeling methods need to be modified before meaningful results about the stress differential between nasal and temporal nerves can be obtained. It has led to the realisation that the method of deflecting the chiasm may need to be rethought. A method such as pushing a tumour shaped structure, with appropriate material properties and no internal pressure, into the inferior aspect of the chiasm could allow the tumour to deform more, or perhaps creating a tumour that is initially much closer to the desired final geometry then using a very low pressure to ‘inflate’ it would allow it to deform more. Problems with the geometry of the optic chiasm created and the method of ‘growing’ the tumour limit the usability of this modeling.

C. Offset of Optic Chiasm and Tumour

As discussed the experiments and modeling done to date have modelled a tumour as a balloon loaded directly underneath the chiasm. The reason for this is that in 91% of the population the optic chiasm sits almost directly atop the pituitary gland. However, in 5% of the population the optic nerves are significantly shorter meaning the optic chiasm sits anteriorly (forward) of the sella turcica and pituitary, and in 4% of the population the optic nerves are longer so the optic chiasm sits posteriorly (to the rear) of the sella turcica and pituitary (O’Connell, 1973), as is shown in Fig 23. The length of the intracranial portion of the optic nerves (between the optic chiasm and where the optic nerve enters the skull) can vary from 6 to 21 mm (O’Connell, 1973), therefore causing a significant difference in the location of the optic chiasm with relation to the pituitary gland, and hence a pituitary tumour. The theory of the location of the nasal nerve fibres within the optic chiasm predisposing them to a higher stress seems to rely greatly on the central loading. The purpose of this section is to test the effect the relative locations of the optic chiasm and a pituitary tumour have on the stress distribution created within the optic chiasm.

The variation in length of the optic nerves from 6 to 21 mm gives a total travel of 15 mm, and assuming this is spread evenly about a mid-point of the optic chiasm resting directly above a pituitary tumour, this gives a travel of up to 7.5 mm forward or rear. The baseline model was modified so that optic chiasm sat 6 mm forward (or rearward, the symmetry makes this irrelevant) of its original position, as shown in Fig 24. This placed the centre of the optic chiasm near the edge of the tumour. The optic nerves were shortened and optic tracts lengthened accordingly. The movement of the chiasm with respect to the tumour caused the loss of the second plane of symmetry, so the model now only has symmetry across its width.

![Figure 22. Plot of Percentage of Maximum Stress vs Location Along Optic Nerve. The plot shows a significant difference between the normal model and the models modified to increase chiasm-tumour contact. ‘Big Tumour’ refers to increased tumour radius. ‘E=x MPa’ refers to a model with a reduced Young’s Modulus for the tumour.]
The model of the offset chiasm is shown in Fig 24. The loss of the second plane of symmetry and the high density mesh created a model with a larger number of elements, and therefore longer solution times. The number of elements in the model was 57028, models with greater numbers of elements were attempted but could not be made to solve due to limitations in computing power. The maximum stress developed in the chiasm was approximately 8200 Pa, greater than that previously found, but still of the same order. The results show that the region of maximum stress has moved away from the centre of the chiasm, as might be expected. Importantly, the region of maximum stress no longer sits solely in an area occupied by only crossing nerve fibres. This indicates

Figure 24. Offset Chiasm. The Optic Chiasm can be seen to sit forward (or rearward) of the tumour (top right). The image of horizontal slice through the chiasm (bottom right) shows that the region of peak stress is distributed equally over the nasal and temporal nerves. The dashed black line indicates the approximate division of nasal and temporal nerves.

The model of the offset chiasm is shown in Fig 24. The loss of the second plane of symmetry and the high density mesh created a model with a larger number of elements, and therefore longer solution times. The number of elements in the model was 57028, models with greater numbers of elements were attempted but could not be made to solve due to limitations in computing power. The maximum stress developed in the chiasm was approximately 8200 Pa, greater than that previously found, but still of the same order. The results show that the region of maximum stress has moved away from the centre of the chiasm, as might be expected. Importantly, the region of maximum stress no longer sits solely in an area occupied by only crossing nerve fibres. This indicates
that in the case of a pre- or post-fixed optic chiasm, the reason for the selective damage to nasal nerve fibres is not their location within the chiasm; therefore it must be a different mechanism. The stress across the optic tract in the pre-fixed chiasm has been graphed and is shown in Fig 25. The data was taken from a horizontal section through the region of peak stress, as indicated by section a-a in Fig 24, and as can be seen there is no significant difference between the stress experienced by nasal nerve fibres and temporal nerve fibres.

**Figure 25. Stress Across the Optic Tract.** The graph shows there is no significant difference between the stress in the nasal and temporal nerve fibres.

### D. Parametric Studies

To test the dependency of the results on the particular variables of each model, a number of parametric studies have been conducted. The mesh density, material properties, and pressure within the tumour balloon have been varied over a range of values to test the sensitivity of the results.

The mesh density was varied on the model of a pre-fixed chiasm and stress distribution of the different meshes compared. The stress distribution was again taken from a lateral section through the centre of the chiasm. In the pre-fixed model this was not the region of highest stress and the results were deliberately taken from this region, as the lower overall stress levels should have meant the results were relatively smooth if the mesh density did not affect them significantly. Figure 26 is a graph of stress levels vs distance from the centre of the chiasm. The nominal element size used for the homogenous nerve fibre within the chiasm was doubled for each new model, whilst the element size of the other structures in the model were left the same to help with convergence. For the model with the greatest mesh density (0.0005 m element size) the results show a reasonably steady reduction in stress with increasing distance from the centre of the chiasm, as would be expected. Whilst there is some variation from a perfectly smooth reduction, much of this may be due to the method of gathering the data. It is expected that as the mesh density is increased the results of the different models will converge to a single stress distribution, so that above some particularly mesh density there will be no significant change in results. As the element size decreased (mesh density increased) the results tended to converge towards those of the model with the smallest element size, indicating the models and results are mesh dependent, and that the densest mesh should be used. Models with less dense mesh showed greater variation from the results of the densest mesh, with large flat spots indicating a single element dominating the reading.

The value of 30,000 Pa for the Young’s Modulus (E) of neural tissue was taken from Table 2. Research in the course of this thesis has indicated that in situations of very low strain rates that this value is much too high. Miller and Taylor (2003) investigated the strain rate dependence of the Young’s Modulus for brain tissue and found that in low strain rate conditions the values are significantly lower. This will be discussed in further detail later in Section IX. Taylor and Miller obtained a value of E=3184 Pa at a strain rate of 0.64 s$^{-1}$ and for a very low strain rate a value of E=584 Pa, almost two orders of magnitude lower that what has previously been used. To identify the effect this has on the results of this FEA the new values of Young’s Modulus were used in the model. As expected, the values of the stress created, decreased along with the Young’s Modulus. With E=3184 Pa the peak stress developed in the model was approximately 900 Pa, and with E=584.4 Pa the peak stress was approximately 150 Pa. These values are significantly lower than those obtained by Kosmorsky *et al.* (2008) and indicate that if these new values for Young’s Modulus are correct then the deflection of the model is below what it should be. The effect of reducing the Young’s Modulus on the distribution of stress is shown in Fig 27.

**Figure 27. Stress Distribution Across the Optic Tract.** The graph shows the stress distribution across the optic tract with different Young’s Modulus values.
result of this is very interesting, as it shows that with a decreased Young’s Modulus of neural tissue, the stress decreases at a much lower rate with distance from the centre of the chiasm. The change in deflection of the chiasm with varying neural properties was measured and found to be negligible.

The pressure within the tumour balloon was originally set at a nominal pressure of 1 Mpa, which gave the deflection seen in the models. Video footage of the experiment by Kosmorsky et al. (2008) show an estimated deflection of the chiasm of approximately 2-3 times its height, much more than the deflection present in our model. An attempt was made to increase this deflection by increasing the pressure in the balloon, testing the effect pressure in the balloon has on the deflection of the chiasm. The pressure in the tumour balloon was

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**Figure 26.** Graph of Stress vs Location for Varying Mesh Density. The results are shown for 4 different element sizes used in the optic chiasm. The first number in the key is the element size used and the number in brackets is the total number of elements in the model.

**Figure 27.** Changes in Stress Distribution with Neural Tissue Properties. The figure shows that the stress distribution laterally across the chiasm changes significantly with decreasing the Young’s Modulus of neural tissue.
increased to 2 MPa and 3 MPa. Table 3 shows the results of increasing the balloon pressure, as can be seen the deflection and peak stress increased, as might be expected. The increase of the tumour balloon pressure increased the stress levels created in the chiasm. If this was used to create deflection more like that seen in Kosmorsky et al.’s experiment, and combined with reduced Young’s Modulus of neural tissue (to reduce the stress levels) the stress values may decrease to values corresponding more closely to those measured by Kosmorsky et al.

<table>
<thead>
<tr>
<th>Balloon Inner Pressure (MPa)</th>
<th>Deflection (mm)</th>
<th>Peak Stress (Pa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.7</td>
<td>6200</td>
</tr>
<tr>
<td>2</td>
<td>3.3</td>
<td>8000</td>
</tr>
<tr>
<td>3</td>
<td>5.0</td>
<td>11000</td>
</tr>
</tbody>
</table>

Table 3. Effects of Tumour Balloon Pressure

E. Assumptions, Simplifications and Limitations

A number of assumptions and simplifications have been made during the FEA in this thesis and there are a number of limitations in the results. Many of these have been identified at the beginning of this section, for example the simplified geometry, and will not be repeated. The results have been compared to those obtained by Kosmorsky et al. (2008). The pressure measured by Kosmorsky et al. was obtained with pressure transducers attached to needles inserted into the optic chiasm. This measured the hydrostatic pressure in the chiasm, and it has been assumed that this can be compared to the von Mises stress of the neural tissue. No way to test this assumption has been identified at this time.

The structures and tissues in the model have all been modelled with linear elastic material models from data on human tissues where possible and otherwise using data from pig tissue. Research has shown that the assumption of linear stress-strain relationships may not adequately model biological tissues, and this will be discussed later in more detail.

The mesh densities of the FEA models in this thesis are significantly greater than that of Howard’s (2008) models; however the modeling in this thesis has still been limited by the computing power available. The length of time to calculate solutions became very large, and often models with the desired mesh density could not be made to solve due to insufficient memory. The source of this problem could not be identified as different computers; with what was thought should be sufficient memory, were tried but did not work. This became particularly apparent once the pre-fixed models were run, as the loss of the second symmetry plane meant models of the same mesh density suddenly had twice the number of elements, and therefore computational cost, of the original models.

What could be the biggest, or most significant, assumption in the FEA conducted in this thesis is the simplifying assumption of no large deflection. ANSYS has a setting ‘Large Deflection’ which has mostly been set to off in this modeling. The ANSYS release 11.0 simulation help states the following regarding large deflection:

> Setting Large Deflection to On will take into account stiffness changes resulting from changes in element shape and orientation due to large deflection, large rotation, and large strain. Therefore the results will be more accurate. However this effect requires an iterative solution. In addition it may also need the load to be applied in small increments. Therefore, the solution may take longer to solve.

Initially, no large deflection was assumed for simplicity, and to reduce the computational times of the model. It was attempted in this thesis to create model which would converge with large deflection set to ‘on’, and it was found to be possible only once the pressure in the tumour balloon was significantly reduced, and therefore did not deflect the optic chiasm as far as necessary. The stretching present in the walls of the tumour balloon is considered to be the most likely reason for the model not converging. There is obviously large deflection present in this model and further work is needed to see whether this has a significant impact on the results, and if so to make the models converge with large deflection on.

VIII. Building FEA Model from MRI Data

One of the major aims of this thesis was to improve the geometric accuracy of the FEA model of the optic chiasm. The FEA is still limited by the geometric accuracy of the model, as the current model prevents the modeling of accurate contact between tumour and chiasm and the smaller lateral distances involved mean that the stress at the edge of the chiasm may be higher than in a wider chiasm. The building of a highly geometrically accurate optic chiasm using CAD tools could prove difficult due to the irregular shapes present, and it was considered that the most reliable way to get an accurate geometry was to use real data. Real data on the geometry of the optic chiasm can be provided by MRI, and a method to use this data in building a 3D model was investigated.
Magnetic Resonance Imaging uses high powered magnets to align the nuclear magnetisation of atoms within the body, and when radio frequencies are passed through the object magnetic field changes are produced which can be measured and recorded. Different tissues in the body have different nuclear magnetic properties, and so produce varying magnetic fields for the scanner to register and record. In this way MRI is used to build up a picture of the internal structure of the body, allowing accurate 3D and 2D visualisations to be built without invasive surgery (Filler, 2009). Figure 6 and Figure 16 are typical MRI images. MRI data is obtained in a 2D dimensional format, so ‘slices’ are taken at known locations in the third dimension. This means that processing of the data can be done to find adjacent pixels of the same intensity value that can then be used to build 3D structures. The use of MRI data to build 3D FEA models has been done before by Liu et al. (2005), Todd and Wang (1996), and Kaazempur-Mofrad et al. (2004), among others. MRI data is obtained and stored in the DICOM format, which contains the image data as well as other information such as patient data, details of the settings of the MRI etc. DICOM images can be saved into JPEG or BITMAP format using readily available DICOM readers. The building of a FEA model from MRI data had four primary steps; these form the headings of the following sections and are shown in Fig 28, a flow chart of conversion from MRI to 3D FEA. The MRI data used in this thesis has been provided by our collaborator, Associate Professor Christian Lueck from Canberra Hospital and the Australian National University (ANU).

A. Segregation of the Chiasm

MRI data registers and records the magnetic field changes from the tissues in the body and converts this data into an intensity value that is set for the pixel at the particular location. The material properties of the optic chiasm are very similar to those of the brain, and so both have very similar intensity values. This can be seen in Fig 29, which shows a transverse (horizontal) slice through the head showing the optic chiasm in between the temporal lobes of the brain. To build the model of the optic chiasm we must first segregate the optic chiasm from the surrounding structures using image processing. The similarity between intensity values makes this very difficult. It was initially attempted to separate the optic chiasm from the surrounding structures using automated methods. With significant assistance from SBLT Matthew Corner, RAN (Corner, 2009), MATLAB programs

Figure 29. MRI Image of the Brain and Optic Chiasm. The similarity between the intensity values of the brain and the optic chiasm are shown in the figure on the left. The image on the right shows the chiasm manually separated from the other structures. MRI courtesy of Associate Professor Christian Lueck, Neurologist, Canberra Hospital, Australia.

† Sub-Lieutenant
‡ Royal Australian Navy
written by Corner, for the purposes of segregating white matter lesions in the brain, were used to segregate the chiasm. These programs set upper and lower limits of intensity, intending to isolate the range of intensity values containing the chiasm. Unfortunately the optic chiasm was too similar to surrounding structures for these methods to work. Edge detection methods that search for changes in intensity at the edges of a structure were also used; however the close proximity of the chiasm to the brain prevented these from being successful. The human brain is a very effective image processor, more so than the computer programs used, so that the optic chiasm was visible to the author unfortunately did not mean that it was ‘visible’ to the programs used. The failure of these methods to isolate the chiasm from the surrounding structures meant that segregation of the optic chiasm had to be done manually. This was done by converting the image to a JPEG format and manually blacking out all other structures using Microsoft Paint as shown in Fig 29. This was done for all nine slices that cut through the chiasm. An automated method of image segmentation is desired, as it will allow models to be easily built from multiple patients, reducing the likelihood of one particular, abnormal patient significantly affecting results.

B. Extraction of Data

The next step was to extract the data of the location of the chiasm. This was done using the program jpegloader.m written by Corner (2009), a copy of which can be found in Annex B. The program processed the image, changing the colours, which all appear grey, to an actual greyscale, then to black and white by setting every pixel with an intensity value below a certain level to black, and every pixel above the set level to white, providing a stark contrast for the chiasm. The program finally cropped the image to remove white areas added in the transformation to a JPEG format. To extract the data the program created a matrix of zeros the size (resolution) of the image, and then checked every pixel to identify which were white and which were black, placing a one in the matrix at the location of every white pixel (representing the chiasm). This data was then vectorised by taking the \([x, y, z]\) data (z being the height/slide number) of each ‘one’ in the matrix and placing this in a 3 column matrix.

C. Building a 3D Model

The vector created in extracting the data contained the three dimensional co-ordinates of every pixel from the MRI data which contained the chiasm. The next step was to take this data and build a 3D model. With significant help from Dr Murat Tahtali of UNSW@ADFA the program runMRI.m was written, a copy of this program can be found in Annex B. This program used other built in MATLAB functions to take the vectorised pixel data and join all pixels within a certain radius of each other together to form larger bodies rather than individual pixels. The initial result was a very rough surface, due to the individual pixels, but with the use of smoothing functions the model surfaced was smoothed out. Caps were added to the model to close off the surface, making an enclosed volume. The programs used to create this model outputted the three dimensional locations of the vertices on its surface and the connectivity matrix connecting sets of three adjacent vertices to form triangular surface elements. The resultant model is shown in Fig 30. The distance between adjacent MRI slides was 1.0 mm, and each pixel was 0.5 mm wide by 0.5 mm tall. The data from each slide had to be extracted and recorded in integers, the result of this being that the 3D locations pulled straight from the data were a factor of two larger.

Figure 30. 3D Optic Chiasm Model in Matlab. The image shows the surface created from MRI data.
in the horizontal dimensions, so the model was stretched horizontally by a factor of two. To counter this effect, the vertical dimensions were multiplied by two. This caused the whole model to be a factor of two oversized but had to be done, as the division of the horizontal dimensions would result in decimal places. This oversize was rectified in the next step.

D. Conversion into ANSYS

The final step in the building of a 3D model for FEA was the conversion of the model into ANSYS so that FEA can be performed. Unfortunately neither ANSYS Workbench nor CATIA had the functionality to build a surface from a set of 3D data points, so this step had to be done in ANSYS Classic. Again with help from Dr Murat Tahtali, the program ansyssurf.m was created, which ran matlab code to write the vertices and connectivity matrix previously mentioned to a .txt file that could be read by ANSYS Classic. A copy of ansyssurf.m can be found in Annex B. ANSYS Classic allows 3D co-ordinates to be read in to the program to create keypoints, these keypoints can then be selected and surface elements built between them. The coding for this was copied from the ANSYS Classic log file into the matlab and written to the .txt file, this allowed ANSYS Classic to automatically read in and create all the keypoints, then use connectivity matrix to create the triangular surface elements making the 3D surface. The result of this is shown in Fig 31.

The hardest steps in creating a 3D FEA model are complete, however it is not yet ready to be used. The model at the moment is only a single surface, so before it can be stressed as intended it must be changed into a solid volume, where all the points within the solid have the given material properties, not just the outside. The model needs to have a second surface placed along the inside of the current surface to model the sheath of the chiasm. This will require creating an offset surface, which may not be easy given the irregular faces on the model. This could be done through further coding be done in MATLAB to obtain the normals of each face, then at each vertex average the normals of the adjacent faces, to give a vertex normal. The required offset distance could then be travelled along the vertex normals to create a new set of keypoints that can be connected using the original connectivity matrix. Some how the two surfaces would need to be placed one inside the other and made into volumes, giving the outer volume the sheath properties and the inner volume neural tissue properties.

The FEA done thus far has been conducted in ANSYS Workbench and it would be preferred to continue doing it this way. This means conversion from ANSYS Classic to ANSYS Workbench. The simplest way to do this would be to export the model from ANSYS Classic in an Initial Graphics Exchange Specification (.iges) format, a generic 3D modeling standard recognised by both types of ANSYS. This has been attempted using the export feature in ANSYS Classic file menu; however ANSYS Workbench did not recognise all the faces in the model, so more work needs to be done to make the models compatible. This may be a problem with the number of faces created in the surface building in MATLAB. The density of the surface mesh created is far greater than necessary, particularly for the given resolution of the MRI data, creating an extremely large number of vertices and faces. Investigation into whether this can be reduced should be done as it would assist in reducing the running time of the code and possibly the export of the model.

![Figure 31. 3D Model of Optic Chiasm in ANSYS. The model shows the surface made of triangular elements.](image)

The biggest barrier to the usage of the MRI built 3D model in FEA is currently the resolution of the supplied MRI data. Figure 30 and Figure 31 show that the model is very ‘stepped’, the individual MRI images can be seen as layers in the model. To get a usable model MRI data will be required that cuts through the chiasm probably
more than 25-30 times, as the angle of the chiasm means that at any particular horizontal location there will be less slices through the chiasm. Also the MRI data should be taken whilst focused on the chiasm, if possible, so that of the 512 pixels in the image much more than 20 to 30 contain the chiasm, as this will assist in regularity of the image.

IX. Research into Material Properties

The FEA conducted thus far, into the stress created in the optic chiasm as a result of displacement from a tumour, has used a single value for the Young’s Modulus of the material properties of the structures involved, and therefore assumes the tissues involved exhibit a linear stress-strain relationship. Research in the course of this thesis has shown that for human neural tissue this assumption may be incorrect. Wittek et al. (2009) state that “the brain and other soft tissues exhibit non-linear stress-strain relationship and strain rate dependency.” No specific data has been found for the material properties of human optical or neural tissue, as obvious ethical and practical considerations limit the number and scope of experiments that can be conducted. As mentioned, the neural tissue in this thesis has been modelled with that of porcine neural tissue, as it is generally considered the most similar to human neural tissue.

Research has been conducted by Miller et al., (2000), Miller and Chinzei (1997, 2002), Miller (1999, 2001), Taylor and Miller (2004), Dutta-Roy et al. (2008), Wittek et al. (2009), and Galle et al. (2007), among others, into the material properties and behaviour of neural tissue from humans and some animals. Miller et al., (1997) performed uni-axial unconfined compression tests of porcine brain matter in an attempt to fit a linear-viscoelastic mathematical model to the data, and found a significantly non-linear stress-strain relationship. This led to Miller (2000) attempting to fit a hyper-viscoelastic model for brain tissue to the data obtained. Miller et al. (2002) noted the lack of data concerning the behaviour of brain tissue in tension, as most experiments had until then tested the response of the brain in compression, and performed uni-axial tension tests on the brain showing that porcine brain tissue exhibited a bi-modal response (different properties in compression and tension). Galle et al. (2007) performed extension tests on spinal nerves, and found significant non-linear behaviour. These experiments have shown that a simple linear elastic model is likely to be inadequate in modeling the tissues present in the FEA of this thesis.

The term viscoelasticity refers to a strain-rate dependence of the material properties, i.e. that the stress-strain relationship of a material is dependent on the speed at which it is deformed. Human tissues show viscoelastic behaviour in that when loaded rapidly they are stiffer than when loaded slowly (Hukins, 1990). Hyperelastic behaviour refers to a non-linear stress response to deformation. The values for Poisson’s ratio were 0.47 or 0.499, as used by Howard (2008), depending on the tissues in question, and all tissues were given a density of 1000 kg/m³ as human tissue is predominantly water.

The effect of the viscoelasticity of neural tissue is limited in its impact on this thesis. Aoki (1988) describes a case where a patient suffered a rapid onset bitemporal hemianopia caused by partial thrombosis associated with enlargement of an unruptured anterior communicating artery aneurysm. This case was described as rapid onset bitemporal hemianopia, however, three months elapsed between the patient initially presenting with a burning sensation in the face and headaches, and the returning to the hospital complaining of blurred vision (upon examination found to be bitemporal hemianopia). This rapid onset case could be considered one of the highest strain rates likely to occur in bitemporal hemianopia, yet the three month period means that the overall strain rate is very low, in the order of 10⁻⁷ s⁻¹. The importance of this to the material models used in this thesis is that they must calculated from data obtained with very low strain rates, the particular relationship between stress and strain rate is less important. Taylor and Miller (2004) explain that the values for Young’s Modulus of brain tissue used in previous studies (3000 to10000 Pa) correspond to strain rates much higher than those present in their study of hydrocephalic brains, and hence to this study of the optic chiasm. Using data from Miller (1999), Miller and Taylor (2004) calculate a Young’s Modulus of E=3184 Pa for strain rates of approximately 0.64 s⁻¹ and E=584.4 Pa for ‘very low’ strain rates. This indicates that if linear elastic models are to be used, the Young’s Modulus must be significantly reduced from its current value. The effect of reducing the stiffness of the neural tissue has been modelled and explained earlier in ‘parametric studies’, and showed that the reduction of the Young’s modulus had a significant effect on not only the peak stress developed, but also the percentage distribution of stress throughout the optic chiasm. Taylor and Miller also quote a Poisson’s ratio of ν=0.35 for the solid phase of their analysis when they complete a bi-phasic analysis, recognising the ability of fluid to move throughout the tissue, making it locally stiffer and softer. This is significantly lower than the value used in this thesis, and investigation should be made in future as to its relevance to this thesis.

Wittek et al. (2009) conducted a study to determine the importance of non-linear constitutive models to the deformation of the brain under craniotomy. They used three different models for brain mechanical properties, one hyper-viscoelastic, one hyper-elastic model, and one linear elastic model. The results of this study were that the calculated deformation of the brain showed no significant difference regardless of the model used, and led Wittek et al. to recommend the use of a linear elastic model in order to minimise computational cost. However they note that, although their results show no significant change in total deformation, this cannot be assumed to
infer no significant variation in the stress created by different models. Thus it would appear that it is important for this research to test the impact of a hyperelastic material model on the stresses calculated in an FEA model of the optic chiasm. Wittek et al. propose a hyperelastic model for very low strain rate conditions that is described by equation 1; a potential function where $W$ is the potential, $\mu$ the instantaneous shear modulus, $\alpha$ a material constant, and $\lambda_i$’s are the principal stretches.

$$W = \frac{2\mu}{\alpha^2} \left( \lambda_1^\alpha + \lambda_2^\alpha + \lambda_3^\alpha - 3 \right)$$

Wittek et al. provide values of $\mu=842$ Pa, and $\alpha=-4.7$. Initial inspection of the built in material models in ANSYS show that it appears the model could be applied as a first order Ogden function, which is an option available when selecting the type of non-linear material model to be used in ANSYS. This can be done by using the provided values of $\mu$ and $\alpha$ for the ‘Material Constant MU1 Pa’ and ‘Material Constant A1’ inputs respectively, then assuming incompressibility (as the tissues are predominantly water) and using a value of zero for the ‘Incompressibility Parameter D1 1/Pa.’ It must be noted that the author has limited knowledge in this subject area, and it is therefore recommended that further research be conducted in this area to clarify the relevance of the model to this thesis and also its correct application.

X. Conclusions

The displacement of the optic chiasm by an abnormality such as a pituitary tumour has been modelled using FEA techniques. Improvements have been made to the geometry of the model of the optic chiasm, new scenarios have been modelled, and research has been conducted into the use of MRI data to build an accurate 3D model and into the material models used for the mechanical properties of the relevant tissues. The results obtained have been shown to correspond well to the limited experimental data available.

The results of this thesis show that in the situation of a tumour positioned directly below the optic chiasm there is significantly greater pressure created in the central aspects of the chiasm than the temporal aspects. This shows that, in this situation, the nasal nerve fibres are exposed to a selectively higher stress due to their location within the optic chiasm. This pressure difference is exaggerated by the local increase in stress due to the crossing geometry of the nasal nerve fibres, as shown by Howard (2008). This thesis has shown that the distribution of stress created in the optic chiasm is significantly affected by the characteristics of the tumour impinging upon its lower surface and the Young’s Modulus used for the neural tissue of the chiasm.

The results also show that in the case of decentralised loading of the optic chiasm, such as could be expected in the case of a pre- or post-fixed chiasm, the temporal and nasal nerves may be exposed to equal pressure distributions. This indicates that their location within the chiasm cannot be the primary mechanism by which nasal nerve fibres are selectively damaged, causing Bitemporal Hemianopia.

Research has provided a method to import data from MRIs that can be used to build a 3D model capable of being used in FEA. This method requires further work on modifying the surface created into a volume and importing it into ANSYS workbench for FEA, and is currently limited by the resolution of the MRI data available.

Research has also shown that the use of linear elastic material models for neural tissue may not accurately model the stress within the optic chiasm. A suggested hyperelastic model has been provided for further research.

XI. Recommendations

The work of this thesis has addressed a number of areas of concern in the work done by Howard (2008), however there are still many simplifications and many areas of concern present in this model that are the focus for recommended improvements.

The biggest concern to the legitimacy of the results obtained is the assumption of linear behaviour of the model. The setting of large deflection to ‘off’ in ANSYS ignores the effect of changes in element shape on the stiffness of a model, reducing the accuracy of the analysis. It is recommended that future work focus on modifying the model, particularly the analysis settings to create a model which will converge with large deflection set to ‘on.’ This will likely require research into the finer workings of ANSYS to gain a greater understanding of the way in which it solves and the effect of features such as weak springs and contact formulations.

The research into the material properties of brain tissue has showed that under low strain rates the Young’s Modulus is significantly reduced. The reduction of the Young’s Modulus of neural tissue was shown to decrease the peak stress developed in the model to values significantly below those measured by Kosmorksy et al. (2008). It is recommended that the deflection of the model be increased to more closely reflect that observed in the experiment by Kosmorsky et al. and to see whether this increases the stress created in the optic chiasm back to the levels measured by Kosmorksy et al.
It is acknowledged that further research needs to be conducted into the material models used in the FEA of this thesis. It is recommended that further investigation be made of the proposed hyperelastic model as to its suitability for this research and its application in ANSYS. This model, if appropriate, should then be implemented in the FEA to determine whether it has a significant impact on the stress created within the optic chiasm.

The work of this thesis has provided a method to create a 3D model from MRI data and import it into ANSYS (classic). The use of MRI data is considered the most likely way to obtain a geometrically accurate model, so it is recommended that the work in this be continued. Research into export of the model as an IGES file, creation of a volume from the surface, the creation of a sheath around the surface and, most importantly, the acquiring of higher resolution MRI data will provide a usable and highly geometrically accurate model.

As the material models and geometries used for the FEA of this research improve then the focus can move away from the comparative distribution of the stress created within the optic chiasm to the specific stress created at points. This will allow prediction of which nerves are exposed to stress levels expected to cause damage and signal destruction. It is recommended that for this type of analysis research be conducted into the method of signal destruction and the particular pressures at which it is expected that signals will no longer transmit. Investigation is recommended into the work of Galle et al. (2007) on the extension of spinal nerves and the quoted level of deformation at which signal loss occurs. Parametric studies of crossing and non-crossing nerves could identify typical pressures at which nerves experience this deformation, giving approximate pressures at which signal loss occurs.

As this work progresses research will need to be conducted into the location of particular nerve fibres within the optic nerves, chiasm, and tracts. Figure 32 shows that the locations of temporal and nasal nerve fibres do not exhibit a precise midline division, a simplification assumed in this project, and their relative positions change along the length of the optic nerve. Thus research must be conducted to determine which particular fibres are affected by the stress created in the optic chiasm.

This thesis has assumed that the visual field defects present in bitemporal hemianopia always exhibit a strong respect of the midline of the visual fields, as the evidence from the medical community seems to suggest. However to further verify the results; investigation should be made into the regularity of the precise midline cut-off of visual field defects, with varying positions and geometries of both tumours and optic chiasm.

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I would like to acknowledge the supervision and guidance of my supervisor Dr Andrew Neely of the UNSW@ADFA. His patience and assistance in explaining concepts, providing clarification of issues, identifying the ‘elephants in the corner,’ and guiding my work in the right direction has been invaluable. The medical expertise and assistance of Dr Christian Lueck has been vital to my understanding the anatomy and characteristics of the structures involved. His explanations have been of great help to me in writing this report and the work it details. I would like to acknowledge the generous assistance of SBLT Matthew Corner, RAN, and Dr Murat Tahtali of the UNSW@ADFA in the creation of a 3D model from MRI data. Without their help and vast knowledge of MATLAB I would not have been able to complete this part of the thesis. Finally to my family and my partner Lou, I would like to acknowledge your patience and understanding through the last four years. The information, contacts, images, explanations, and proof reading provided by my father have been of great assistance to the successful completion of this work and the writing of this report. To these people, thank you.

Figure 32. Positions of Nasal and Temporal Fibres. The areas of occupied by nasal and temporal nerve fibres change along the length of the optic nerves (Clinical Neuro-Ophthalmology, Miller et al., 1999)