EFFECT OF HIGH +Gz ACCELERATIONS ON THE LEFT VENTRICLE

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1 SUMMARY

During certain manoeuvers, fighter pilots are subjected to high accelerations reaching 10g levels. The effect of this acceleration on the left ventricle is most severe when it is directed along the body z axis. Under such accelerations it is difficult for the heart to function and supply the body with blood and further more there is concern that the heart may suffer tissue tear as a result of high stresses on the heart tissue.

In this study a detailed finite element analysis is carried out to determine the stress state of the left ventricle under high Gz loading. To develop the FE model, surface geometry data was acquired from view Point Data Lab in Utah. The surface data for the interior and the exterior of the left ventricle was then used with a software from XYZ Scientific Application Inc. of Livermore to develop a 3D FE model. The model is made up of 3830 solid elements with three layers between the inner and the outer surfaces. Finite element results for deflections, strains and stresses are obtained for a number of acceleration levels. The analysis accounts for geometric nonlinearities and uses the updated Lagrangian method in the MARC finite element program.

2 INTRODUCTION

The ongoing development of high performance aircraft, capable of providing positive accelerations under certain maneuvers, has created substantial research work dealing with physiological reactions affecting a pilot's judgement.

The inertia force acting along the vertical axis of an upright body is denoted as +Gz. Under high +Gz loading conditions, the heart and other organs are displaced downwards and depending upon the level of elevated stresses, can be severely damaged.

Physiological responses to the +Gz acceleration range from less severe temporary loss of peripheral vision to unconsciousness and, in the ultimate case, damage to heart tissue. For safety reasons, there is a need to investigate the effects of +Gz inertia forces on the cardiovascular system (see [1,2,3]).

From experiments conducted in a safe environment using a centrifuge, the average blackout level observed for an unprotected individual is between +3.5 to +4.0 Gz. However, when fitted with an anti-G suit and using straining maneuvers (muscle testing with controlled breathing) a pilot can be exposed to accelerations up to +10Gz for short periods of time [4,5,6]. Over longer periods such high loadings may bring about abnormalities in the electrocardiogram in men [7] as well as sub-endocardial hemorrhaging and pathological changes of the myocardial tissue in animals [8].

Reports of endocardial hemorrhaging and myo-fibrillar degradation in tests with swine (see [8,9]), undergoing sustained +Gz accelerations, raise questions regarding the possibility of cardiac tissue damage in humans subjected to high +Gz loading.

In general, the distribution of stress in the ventricles of the heart depend upon
1)the three dimensional geometry and fibrous architecture of ventricular walls.
2)the boundary conditions imposed by the ventricular cavity and pericardial pressures and structures like the fibrous valve ring skeleton at the base of the ventricles.

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3)the three dimensional mechanical properties of the myofibrils and the collagen interactions in the relaxed and actively contracting states. Many of these determining factors have been quantified in experimental studies [10,11,12,13,14].

Difficulties in cardiological techniques used during experiments on the human heart provide insufficient data to determine the presence of any localized cardiac damage. Accordingly, the use of computational schemes becomes necessary to obtain estimates of stress levels in the human heart under high +Gz loading conditions.

The simplest models used by some researchers [15,16,17], are made up of regular geometrical shapes such as spheres, ellipsoids, cones or a combination of such geometries (see also [18]). These models do not describe the geometry of the heart adequately. Further, they are almost always used for small deformations.

For the present research, where deformations are expected to be large, it is essential to adopt a more realistic geometric model. The flexibility and power of the finite element method (FEM) to deal with complicated shapes and take account of the effects of material and geometrical nonlinearities makes it an ideal tool for analyzing the stress-strain behavior of the human heart under +Gz acceleration.

3 FINITE ELEMENT MODEL DEVELOPMENT

A model of 60000 points was bought from View Point Data Lab, Utah (see also [28]). This model describes the interior and exterior surfaces of a life size heart of a male.

The data on the coordinates of a large number of points on the exterior and interior surfaces of the heart had to be very extensively processed before a finite element model could be developed. First the proximity of the coordinate points had to be specified. This information was provided through the connectivity of the coordinate points. Thus the coordinates and their connectivity produced polygons on the surface geometries of the heart. To describe the surface geometry semi analytically, for purposes of finite element development, it was necessary to fit specific curves to the coordinate points. This is essentially a Computer Aided Design process. To this end the CAD program TRUE GRID was used for creating multi block stretched FE meshes. The essential idea is to project a block of finite elements in a regular mesh, onto a curved surface.

Making a mesh with TRUE GRID is analogous to sculpting. Pieces of the block mesh are removed so that its topology matches that of the object to be meshed. Portions of the mesh are moved, positioned along curves, projected to surfaces, etc. Interpolation, smoothing, and zoning requirements are used to fine-tune the mesh. Parts created separately are "glued" (merged) together automatically by specifying a tolerance.

Following the above procedure two models of the left ventricle were developed. The first model is comprised of 3300 solid elements (8 nodes) whereas the second model, being far more refined, is made up of 88000 elements. Both models have used three layers of elements through the thickness. The coarser model is small enough to allow computations on a workstation. It was planned to gain experience on the analysis of the coarser model and using this experience to analyse the finer model on a super computer. Typical difficulties encountered include: unacceptable element shapes, errors in node numbering, improper connection of parts etc. much of these errors had to be found and corrected through numerous check runs.
Finite Element Software

3.1 Importing the FEM Model into MARC

Having completed the data processing within the TRUEGRID program [28] it was necessary to import the developed model, with its data files, into the MARC finite element program. Next the objective was to examine the model, from different perspectives, in the MARC program. Figure 1 shows a view of the smaller model.

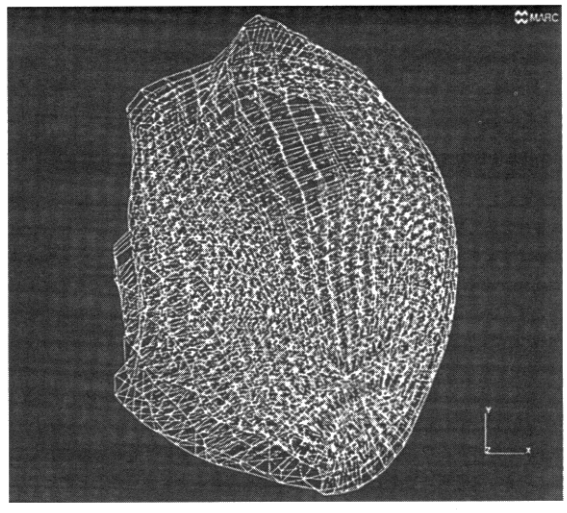

Figure 1: 3300 brick element model viewed in the MARC program.

3.2 Testing the Model within the Frame Work of MARC

Once the model geometry had been verified through a number of check runs in the MARC environment [28], it was necessary to ensure that the material data and boundary conditions were properly registered in MARC. To this end a trial run was carried out under +5Gz acceleration. In this run a linear analysis was carried out where the material density was taken as 9.15×10^{-5} slug/in^{3}and for elastic properties, Hooke's laws were used with E = 142.8 psi and ν = 0.45 , i.e. a soft nearly incompressible material.

The results obtained are shown in Figures 2 and 3. It is emphasized that this analysis was undertaken merely to ensure that the solution process can be carried out on the 3300 element model.

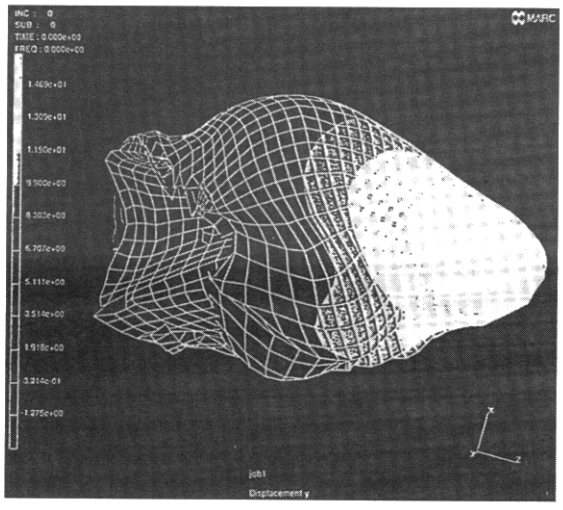

Figure 2: Displacement distribution in the left ventricle, linear check run.

3.3 Biomechanics of The Heart Muscle

The heart consists of two pumps, the right and the left ventricles, connected in series and anatomically intimately linked together into one organ. The right heart maintains blood flow in the systemic circulation. The left heart is by far the stronger of the two heart pumps. It develops a pressure of about 16 kPa which is four to five times the pressure developed by the right heart. Consequently more musculature is developed in the left than the right ventricular myocardium. The initial stages of cardiac disfunction are generally found in the left ventricle.

This study is mainly related to the left ventricular modelling. The mechanical performance of the left ventricle is the result of cooperative contractile action of the muscle cells in the left ventricular wall. Therefore, insight into the mechanics of the left ventricle, at the level of muscle cells, is important for understanding the pumping action of the heart. For that reason, quantifications of local ventricular wall mechanics have been stimulated against the following information:

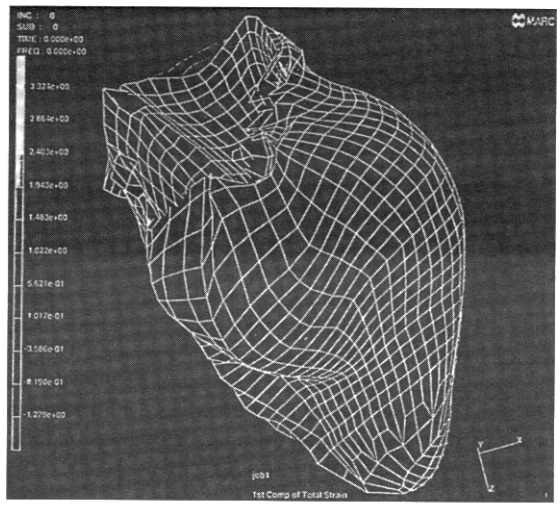

Figure 3: Strain distribution in the left ventricle, linear check run.

1- Myocardial wall stress and deformation are some of the primary determinants of myocardial oxygen consumption.
2- Myocardial oxygen supply is dependent upon coronary blood perfusion, which has a direct dependence upon the mechanical state of the myocardial tissue.
3- Wall deformation is believed to be the feedback signal that governs myocardial growth. For example, ventricular hypertrophy is the result of excessive loading of the heart.
4- Diagnosis of the heart is usually done on the basis of global data of cardiac function. Interpretation of these data in terms of local myocardial disfunction requires a thorough insight into the fundamental mechanisms underlying the ventricular action.

Important quantities for the description of the mechanical state of the left ventricle are the local deformations and stresses. At present, local deformations can be measured accurately only at a limited number of sites in the left ventricular wall. Reliable measurement of left ventricular wall stress is difficult, because insertion of a force transducer damages the tissue.

Here we will present a possible material model for consideration

in the heart muscle biomechanics. First, the main features are sketched for a continuum theory for blood perfused porous medium, largely deformable soft tissue. Next the constitutive law for the myocardial tissue, describing the dependency of stress on tissue deformation, will be addressed.

3.3.1 Continuum Mechanics theory of The Heart Muscle

Heart muscle is a mixture of many different components: muscle fibers, collagen fibers, coronary vessels, coronary blood and interstitial fluid. A rigorous integrated porous medium continuum theory of myocardial deformation and intramyocardial coronary flow has been developed by Huyghe [19]. In this finite deformation mixture theory the differential equations describing the state of the myocardium are derived by means of a formal averaging procedure. The tissue is composed of a solid phase and a spectrum of fluid phases representing the different coronary microcirculatory components.

In this section the major features of the above-mentioned theory will be specified for the simplified situation where the spectrum of fluid phases is condensed into one liquid component, and the transmural pressure differences across blood vessel walls are neglected. This simplification yields considerable computational advantages but retains the salient mechanical aspects of the heart tissue in the resulting so-called deformation model.

3.3.1.1 Equilibrium Equations

The state of stress in the liquid component of the biphasic mixture is described by an intramyocardial pressure p. The stress in the solid is a full 3D stress tensor. The stress in the deformed mixture is best understood as follows. Say the solid phase is subjected to a deformation in the absence of the fluid phase. The stress σ_{ij}^{eff} in the solid induced by this deformation and measured per unit bulk surface area is called the effective stress. The word effective indicates that this part of the stress is the only part depending directly on the deformation. Now we inject a fluid at a pressure p into the deformed solid matrix. The pressure p will effect both the fluid and the solid. So, the total Cauchy stress in the mixture may be written as:

$$\sigma_{ij}^{tot} = \sigma_{ij}^{eff} - p\ \delta_{ij} \qquad i,j = 1,2,3 \tag{1}$$

In the absence of inertia forces, the equilibrium equation of the deformed myocardium may be written as:

$$\sigma_{ij,\ j}^{eff} - p_{,i} = 0 \tag{2}$$

In the above equations we have used the conventional notations in continuum theories.

3.3.1.2 Blood Flow through Porous Myocardium

To simulate the redistribution of coronary blood in the ventricular wall during deformation, the liquid phase is allowed to flow relative to the solid. To generalize the relationship to the porous myocardium, where the pore length is a variable, we use Darcy's law, i.e., the fluid flow is proportional to the local intramyocardial pressure gradient.

Neglecting transmural pressure differences across blood vessel walls, Darcy's law yields the following equation in the Eulerian form:

$$Q_j = -\ K_{ij}\ p_{,i} \tag{3}$$

where Q_j is the Eulerian spatial fluid flow vector and K_{ij} is the permeability tensor with respect to the current configuration. Neglecting the stretching of blood vessels and assuming the ratio of the current and the initial blood volume fractions to be independent of the vessel diameter, the following expression holds for K_{ij}, [20],:

$$K_{ij} = \left(\frac{J-1}{R_{av}} + 1\right)^2 K_{ij}^{o} \tag{4}$$

where K_{ij}^{o} and R_{av} are the permeability and the averaged porosity of the undeformed tissue, respectively, while J is the jacobian of the deformation gradient tensor.

3.3.1.3 Continuity Equation

The cardiac tissue is considered as a mixture of an intrinsically incompressible solid and an incompressible fluid. Consequently any change in the volume of this solid-fluid mixture is equal to the amount of fluid being squeezed out or sucked in. Then, conservation of mass requires:

$$\dot{U}_{i,i} + Q_{i,i} = 0 \tag{5}$$

in which $\dot{U}$ is the material time derivative of the displacement of the solid.

3.3.2 Constitutive Behavior of Myocardial Tissue

The constitutive law for the myocardial tissue is expressed as a relationship between the effective second Piola-Kirchhoff stress tensor S_{IJ}^{eff}, the Green-Lagrange strain tensor E_{IJ} and time t, where:

$$S_{IJ}^{eff} = J\ F_{mI}^{-1}\ \sigma_{ij}^{eff}\ F_{mI} \tag{6}$$

and

$$E_{IJ} = \frac{1}{2}\left(F_{mI}\ F_{mJ} - \delta_{IJ}\right) \tag{7}$$

Here F_{mI} is the deformation gradient tensor.

The effective stress S^{eff}_{IJ} may be written as the sum of a passive stress S^{p}_{IJ} and an active stress S^{a}_{IJ}:

$$S^{eff}_{IJ} = S^{p}_{IJ} + S^{a}_{IJ} \tag{8}$$

3.3.2.1 Passive Constitutive Behavior

The stress tensor S^{p}_{IJ} is split into two components, one resulting from elastic volume change of the myocardial tissue S^{v}_{IJ}, the other from viscoelastic behavior of the myocardial tissue S^{ve}_{IJ}, i.e.:

$$S^{p}_{IJ} = S^{v}_{IJ}\left(E_{IJ}\right) + S^{ve}_{IJ}\left(E_{IJ}, t\right) \tag{9}$$

The elastic stress S^{v}_{IJ}, due to volume change, may be derived from the isotropic strain energy function $\mathcal{W}$ as:

$$S^{v}_{IJ}\left(E_{IJ}\right) = \frac{\partial \mathcal{W}}{\partial E_{IJ}} \quad ; \quad \mathcal{W} = \frac{c_v}{2}\ (J-1)^2 \tag{10}$$

where c_v is the volumetric modulus of the empty solid matrix.

The viscoelastic component S^{ve}_{IJ} is described in a spectral form of quasi-linear viscoelasticity as proposed by Fung, 1993, i.e.:

$$S^{ve}_{IJ}\left(E_{IJ}, t\right) = \int_{-\infty}^{t} H(t-\tau)\frac{d}{d\tau}S^{e}_{IJ}\left(E_{IJ}(\tau)\right)\, d\tau \tag{11}$$

where S^{e}_{IJ} is the anisotropic elastic response of the material.

The reduced relaxation function $H(t)$ is a scalar function derived from a continuous relaxation spectrum.

The elastic response S^{e}_{IJ} is generally taken to be orthotropic. The three axes of orthotropy are specified in Figure 4 by the three orthonormal vectors e_1, e_2 and e_3. The vector e_3 is parallel to the initial local fibre direction.

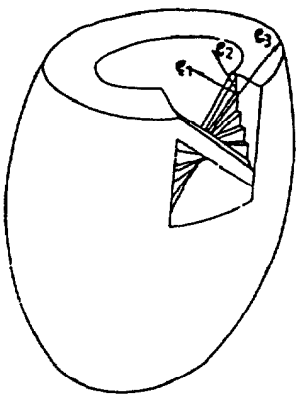

Figure 4: Orthonormal vectors e_1, e_2 and e_3 specifying axes of orthotropy. e_3 is parallel to the initial local fibre orientation. e_1 is perpendicular to the wall.

In this local coordinate system the components of the stress S^{e} and the Green-Lagrange strain E are written as:

$$S^{e}_{IJ} = e_I \cdot \left(S^{e}\ e_J\right) \quad ; \quad E_{IJ} = e_I \cdot \left(E\ e_J\right) \tag{12}$$

The passive constitutive behavior is described by a strain energy function $W\left(E_{IJ}\right)$ that relates S^{e}_{IJ} to E_{IJ}:

$$S^{e}_{IJ} = \frac{\partial W}{\partial E_{IJ}} \tag{13}$$

The functional forms of the strain energy function $\mathcal{W}$ (describing volumetric changes) and W are chosen so that:

$\mathcal{W}$ and W are zero in the unstrained state and positive elsewhere.

S^{v}_{IJ} and S^{e}_{IJ} are zero in the undeformed state.

The expression (10) for $\mathcal{W}$ satisfies these conditions.

The most comprehensive expression for $W\left(E_{IJ}\right)$, satisfying the above conditions, has been proposed by Huyghe [21], and is considered as a full orthotropic description of the tissue behavior.

3.3.2.2 Active (Contractile) Constitutive Behavior

Cardiac muscle is striated across the fibre direction. The sarcomere length is the distance between the striations, and may be used as an objective measure of muscle fibre length. In experiments it has been found that active stress, generated by cardiac muscle cells, depends on time, sarcomere length and the velocity of shortening of the sarcomeres (Fung [22]).

The active stress generated by the sarcomeres is directed parallel to the fibre orientation. Consequently the active stress tensor S^a_{IJ} can be written as:

$$S^a = e_3 \cdot \left(S^a\ e_3\right) \tag{14}$$

Since the first Piola-Kirchhoff stress tensor P^a_{IJ} is easily obtainable in experiments, we may use this stress measure instead of the second Piola-Kirchhoff stress S^a_{IJ} and therefore we have:

$$\begin{aligned} P^a &= \left\|F \cdot e_3\right\|\ S^a \\ &= \frac{l_s}{L_s}\ S^a \end{aligned} \tag{15}$$

where l_s and L_s are the current and the initial local sarcomere lengths, respectively.

We may write the first Piola-Kirchhoff stress P^a as (see Huyghe [23]):

$$P^a = P^a_o\ A_t\left(t_s\right)\ A_l\left(l_s\right)\ A_v\left(v_s\right) \tag{16}$$

where the constant P^a_o is associated with the load of maximum isometric stress. The factors $A_t\left(t_s\right)$, $A_l\left(l_s\right)$ and $A_v\left(v_s\right)$ represent the time dependency and the length dependency and the velocity dependency, respectively, of the active stress development ($v_s = dl_s/dt$). Figure 5 shows the above relationship. More detail on these coefficients can be found in Huyghe [23].

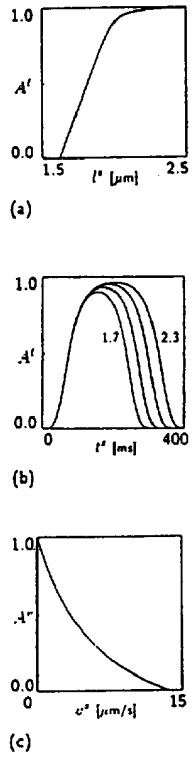

Figure 5: Active material behavior: (a) length dependence of active stress, (b) time dependence of active stress for sarcomere length of 1.7, 1.9, 2.1, and 2.3 μm , (c) stress-velocity relation.

3.4 Sources of Nonlinearities in Finite Element Analysis

Nonlinearities in finite element analysis arise due to
1- significant changes in geometry,
2- nonlinear constitutive relations,
3- contact problems.

For the present project, all three sources of nonlinearity are present. Certainly the geometry of the heart changes significantly under high +Gz loading. The tissue constitutive relations, found experimentally, are nonlinear and under large deformations the heart will contact other parts in the body. However for this study, nonlinear boundary conditions, due to contacts, will be neglected. This is in part due to the fact that the information on the geometry of other parts, in the vicinity of the heart, is not available.

By neglecting the nonlinear boundary conditions the computed results will be on the conservative side, i.e. the deformations will be larger than those resulting when the heart contacts other

parts.

3.5 Finite Element Results of Deformation and Stresses in the Human Heart under High +Gz Acceleration

In this study, a quasi-static analysis is performed for the diastolic phase of the left ventricle under +Gz loading conditions (see also [28]). After checking the 3300 elements model of the left ventricle, a geometric nonlinear analysis was carried out for different levels of acceleration. A full nonlinear formulation, including nonlinearities in the material of the human heart, has been outlined in the previous sections. Due to complication of the model, the initial analysis was carried out for geometrical nonlinearities alone. Here several test runs were made under different loading conditions. For this analysis, the myocardium is modelled as an isotropic soft elastic material with a Young modulus of 14.28 psi and Poisson's ratio 0.3.

Results obtained for the +5G loading are shown in Figures 6, 7, and 8. In Figure 6 one can see that displacements increase cumulatively as one proceeds from the constrained nodes, at the top to the bottom nodes. In Figure 7 the Von Mises stresses are shown across a vertical cut through the left ventricle wall. The highest stresses arise in the vicinity of the constrained nodes, at the top of the model. Figure 8 depicts the distribution for the first component of stress, σ_{xx}, in the global coordinate system (X).

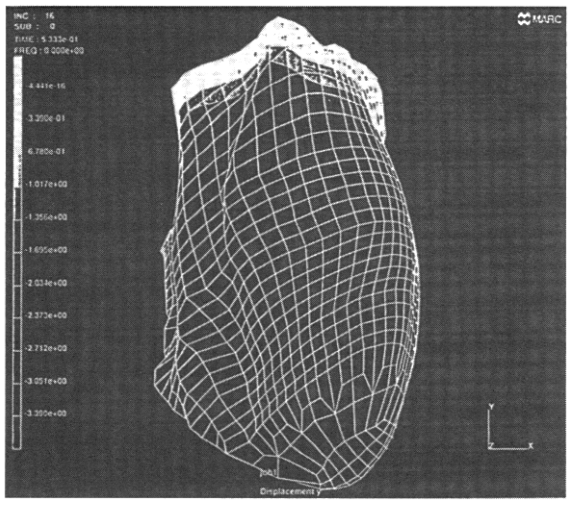

Figure 6: Displacement distribution, geometrically nonlinear analysis.

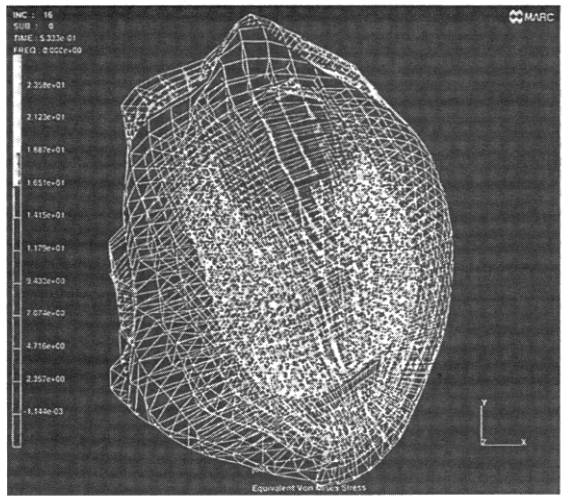

Figure 7: Von Mises stresses, distribution, geometrically nonlinear analysis.

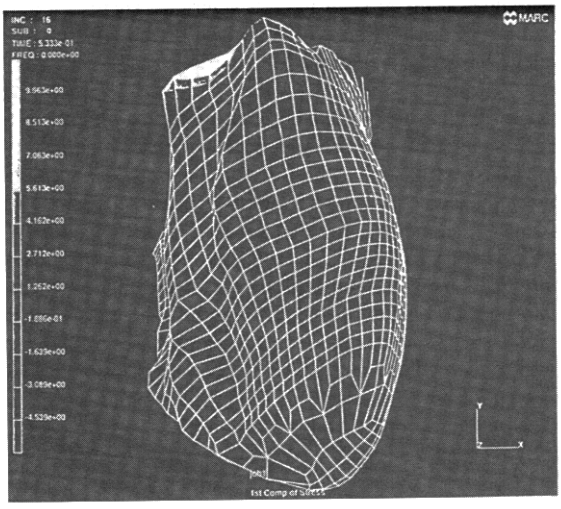

Figure 8: First component of stress, geometrically nonlinear model.

4 CONCLUSIONS AND FUTURE WORKS

The key objective in this study has been the investigation of the +Gz loading on the human heart. To this end it was necessary to generate an accurate geometrical model of the left ventricle. This data was processed by an advanced CAD tool named TRUEGRID. Next the model was discretized via the projection technique introduced in the TRUEGRID software. Two finite element models were developed, one with approximately 88000 eight-node brick elements and the other with 3300 eight-node brick elements.

For numerical processing it was decided to use the MARC finite element program. To run the model on the MARC program it was essential to find and correct poorly shaped elements. Such elements can give rise to ill conditioning during the solution process.

Once the model was suitably improved for element connectivity and the poor shaped elements were eliminated, a linear run was carried out. This run took about 10 minutes on a Sun Ultra work station. The results obtained were consistent with anticipated deformations in the model. Next a series of cases were run for different levels of accelerations. In these runs geometric nonlinearities were taken into account. From the results obtained one can see that under high acceleration the left ventricle becomes longer while its "diameter" diminishes. This is in agreement with one's expectations on physical grounds. The resulting stresses show high values at the constrained end and relatively low values at the apex of the left ventricle. There is a nonuniform stress distribution on the surface of the left ventricle as well as across the wall of the left ventricle.

Due to its high flexibility the heart elongates and changes its shape significantly, under high accelerations. If the distortions are not excessive; that is the case for relatively low acceleration levels, the elements almost retain their initial shapes. However, at high accelerations the element shapes become highly distorted leading to numerical problems. To overcome this difficulty it is essential to remesh the heart model when the distortions become excessive. This requires the transfer of information, such as nodal displacements and stresses from the initial mesh to the updated mesh. This is done by interpolation of the information to be transferred. This task remains to be undertaken in the next phase of this project.

To incorporate the material nonlinearities a detailed literature search was carried out. At this stage it appears that the Ogden model [24] is the most suitable for describing the nonlinear elastic behavior of the heart tissue. This model includes several parameters which must be determined by the "best fit" method to the experimental data presented in the literature [25,26,27]. This task and further refinements to the model will form the main focus of the future studies. Subsequent to that the porous media (section 3.3.2) will be used in the modeling.

5 ACKNOWLEDGEMENT

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