Micromechanics of Minor Cervical Spine Injuries*

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Minor soft tissue injuries of the cervical spine are of increasing significance in public health. They may in particular be associated with long-term impairment. Such injuries are observed primarily in rear-end automobile collisions at low impact speeds and are attributed to a "whiplash"-type event. The question with respect to injury mechanisms of the cervical spine in cases of impacts of a low severity have raised controversial views in the past. Among proposed injury mechanisms, interactions between fluid and solid structures have been postulated: Viscous shear stresses or pressure gradients which arise in the deforming anatomical structures may have an adverse influence, e.g., on cellular membranes. In this communication, mathematical modeling approaches are presented which allow for a quantification of fluid/solid interactions under typical loading conditions of interest here. It is found, that the shear stresses caused by fluids and acting on accelerated surfaces of fluid-filled bodies depend largely on the size of the fluid space under consideration. Accelerations exhibit a stronger influence than their duration. It cannot be excluded that critical levels are reached even in a low speed impact scenario.

Key Words: Whiplash, Cervical Spine Injuries, Low-Speed Impacts, Fluid/Solid Interaction

1. Introduction

The term "whiplash" is used in connection with a wide range of traumatic loading conditions which may lead to cervical spine injuries. Strictly speaking, whiplash denotes a particular motion pattern where a head executes an unimpeded rapid excursion due to a sudden force acting on the upper torso during an accident, i.e., a true whiplash-type head motion develops itself only in situations where no severe contact of the head with an object of its surrounding (e.g., car interior) occurs. An exception is usually made in rear-end automobile impacts where a contact (as soft as possible) with the head restraint is intended to restrict the head motion⁽¹⁾⁻⁽⁵⁾. Whenever there is a hard head contact other than with the head restraint, a whiplash motion cannot evolve.

Whiplash may cause injuries of various degrees. A particularly difficult problem in trauma biomechanics thereby exists with respect to minor soft tissue injuries of the cervical spine. Such injuries are primarily sustained in low-speed rear-end automobile impacts, but, although less frequently, they are also observed in sports, work or household accidents^{(6),(7)}. The loading levels prevailing under the impact conditions which are of concern in this context are low and the injuries are, according to the Abbreviated Injury Scale (AIS⁽⁸⁾), mostly classified as type AIS=1 (minor injuries). They can in particular often not be detected morphologically by way of standard medical imaging methods (MRI, CT, ultrasound, PET, thermography etc.). In contrast to the minor cervical spine injuries considered here, injury patterns, mechanisms, treatment and prevention strategies as well as long-term outcome associated with moderate and severe neck injuries in general have been discussed and analyzed in depth⁽⁹⁾⁻⁽¹⁵⁾.

Health problems which are reported by patients who had been exposed to a low-speed rear impact or some other sudden force on the shoulder from behind are generally denoted as Whiplash-Associated Di-

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sorders (WAD), even in cases where it is questionable whether a whiplash in the strict sense had developed. Typical manifestations of WAD include cervical or related pain, muscular stiffness, dizziness, fatigue, depression and autonomic disturbances. In spite of the marginal severity of such disorders, it is known that long-term impairment may nevertheless ensue. It is furthermore known that psychosomatic factors may have a substantial influence or give rise to an aggravation of the typical symptoms associated with WAD, however, quantitative information is lacking^{(16)–(18)}.

In rear-end automobile collisions, a critical impact severity exists whenever Δv^{*1} values of the vehicle under consideration lie between about 2.5 m/s ($\cong 10$ km/h) and 4 m/s ($\cong 15$ km/h). Mean accelerations amount to 30 m/s² and 45 m/s², respectively, under the assumption of typical collision durations. Below the lower threshold, a causality between mechanical trauma and clinical symptoms cannot readily be explained except for special circumstances, e.g., if the cervical spine had been injured before. Above the higher threshold, in turn, the mechanical trauma can be assumed to be sufficiently severe to cause physical damage and injuries of a severity of AIS ≥ 2 . At higher Δv levels, furthermore, head contacts are frequent.

In view of the relatively low severity of the loading in the collision situations considered here (i.e., Δv between 2.5 m/s and 4 m/s) the question arises with respect to possible injury mechanisms. In the situation of a car occupant who is subjected to a low-speed rear-end impact a typical motion sequence evolves as is seen in Fig. 1. During the initial phase (phase 1) the head executes a translatory motion because only minor forces act on it such that it keeps its state of motion. Second, a typical "S"-shape of the cervical spine results (phase 2). Rotation and rebound occurs afterwards (phases 3 and 4, respectively).

Various injury mechanisms have been discussed in the literature. First, it is hypothesized that the intravascular and interstitial fluid spaces in the cervical spine may undergo volume changes during the "S"-shape phase of the motion (phase 2). Since the body fluids are incompressible, the formation of pressure peaks accompanied by rapid fluid displacements are to be expected which may interfere adversely with nerve endings. Corresponding experiments were performed with pigs^{(19),(20)} and with PMHS's (*post mor-*

Table 1 Tests performed on cell cultures (adopted from Morrison et. al.⁽²⁵⁾)

Loading	Cell Type	Reference
Hydrostatic pressure	cell lines	Murphy & Horrocks 1993 (J Neurotrauma 10, p. 431)
	glia cells	Shepard et al. 1991 (J Surg Res 51, p. 417)
	brain tissue	Wallis & Panizzon 1995 (Eur J Pharmacol 294, p. 475)
Acceleration	neurons, glia cells	Lucas and Wolf 1991 (Brain Res 543, p. 181)
Strain	neurons, glia cells	Ellis et al. 1995 (J Neurotrauma 12, p. 325)
	differentiated cell line	Cargill & Thibault 1996 (J Neurotrauma 13, p. 396)
	brain tissue	Morrison et al. 1998 (Ann Biomed Eng 26, p. 381)
Hydrodynamic shear	differentiated cell line	LaPlaca & Thibault 1997 (Ann Biomed Eng 25, p. 665)

Fig. 1 Typical motion sequence during rear-end impact. The typical "S" shape of the cervical spine is seen in phase 2

tem human subjects, i.e., cadavers, by Eichberger et al.⁽²¹⁾). Possible harmful interactions are attributed either to pressure peaks or due to impact-induced fluid shear stresses acting at the fluid/solid, i.e., interstitial fluid/cell membrane interface or to local deformation patterns. Second, it has been found on the basis of high-speed X-ray analysis^{(5),(22)} that the zygapophysial joints are exposed to compression (during phases 2 and 3, respectively⁽²³⁾). Long-lasting pain may be the result of this mechanical trauma⁽²⁴⁾.

A number of experiments has been performed on cell cultures in order to determine tolerance limits on a microscopic level. An overview of the published tests performed with various types of nerve cells in vitro is given in Morrison et al.⁽²⁵⁾ Table 1 is reproduced in part from these authors. Loading conditions including the target values applied in these tests differ considerably and in particular, a systematic tolerance criterion can hardly be derived at this time. In the first place, however, it is generally difficult to relate the laboratory loading patterns to those prevailing in humans under realistic impact conditions.

Mathematical models are presented in the following which are intended to allow for an analysis of fluid/solid interactions under loading conditions which have to be expected in a typical low-speed impact scenario. To this end, an idealized fluid and solid system is first considered. It is subjected to a constant

*1 Δv denotes the change of the velocity of the vehicle under consideration which is due to the primary collision forces. This parameter is defined only in essentially uniaxial collisions and has proven to be a valid quantity to characterize the severity of car crashes.

acceleration and the geometry is chosen such that the equations can be solved analytically and as such allow for general conclusions. The geometry corresponds furthermore to a configuration which leads to maximal fluid-solid interactions in order to characterize the maximal possible effect. Shear stresses acting between the fluid and solid surface are calculated in order to determine their potential with respect to injury causation. Second, a detailed neck model is presented which includes vertebrae, discs, muscles, tendons as well as a typical fluid space (plexus venosus). This model is in part based on earlier work by Yang et al.⁽²⁶⁾, Schmitt et al.⁽²⁷⁾, Schmitt et al.⁽²⁸⁾. Boundary conditions mimic a typical low-speed rear-end impact and the pressure in the venous vessel is determined. The results are compared with the tests performed by Svensson et al.⁽¹⁹⁾, Oertengren et al.⁽²⁰⁾ as well as Eichberger et al.⁽²¹⁾

2. An Analytical Model

As a model to study fluid-solid interactions, in particular viscous shear stresses, we consider a cylindrical fluid-filled tube which is subjected to a sudden acceleration in the axial direction. This configuration is expected to lead, in comparison with other orientations of the vessel, to maximal gradients in the flow field and therefore to maximal shear stresses at the tube wall.

The fluid is assumed as Newtonian and the tube is sufficiently long such that entrance effects can be ignored. The tube and the fluid are initially at rest and at $t=0$, an acceleration $a(t)$ sets in whose level is constant, a_0 , for $t>0$ (Fig. 2):

$$a(t) = a_0 \theta(t) \quad (1)$$

with the Heaviside function, $\theta(t)$. For this geometry an analytical solution can be found and some general conclusions can be drawn.

We solve the Navier-Stokes equation in axial direction under the assumptions of one-dimensional, laminar, incompressible flow (Fig. 3):

$$\partial v_z / \partial t = \nu \partial^2 v_z / \partial r^2 \quad (2)$$

whereby

z, r : denote the axial and radial coordinate, respectively,

$v_z(r, t)$: the axial fluid velocity and

$\nu = \mu / \rho$: the kinematic viscosity (viscosity μ over density ρ).

v_z depends on t and r only, because the problem is invariant with respect to z . The pressure, p , furthermore, is assumed to be (and remain) constant, i.e., $\text{grad } p = 0$ (there is no fluid motion other than that induced by the accelerated tube).

Upon performing a Laplace transformation,

$$a(t) = a_0 \theta(t); \quad \tilde{a}(s) = a_0 / s \quad (3)$$

Fig. 2 Acceleration as function of time

Fig. 3 Fluid in accelerated tube

the Eq. (2) reads

$$\tilde{v}_z'' + (1/r) \tilde{v}_z' - (s/\nu) \tilde{v}_z = 0 \quad (4)$$

A Bessel equation is obtained when the substitution

$q = ir\sqrt{(s/\nu)}$ is made

$$q^2 \tilde{v}_z'' + q \tilde{v}_z' + q^2 = 0 \quad (5)$$

The solution is

$$\tilde{v}_z(r, s) = [a_0 / (s^2)] \{ J_0(ir\sqrt{(s/\nu)}) / J_0(ir_0\sqrt{(s/\nu)}) \} \quad (6)$$

whereby J_0 denotes the zeroth Bessel function and r_0 the radius of the tube. An inverse transform cannot readily be found in closed form. The shear stress can nevertheless be calculated as (J_1 is the first Bessel function)

$$\tilde{T}(r, s) = -i [a_0 \sqrt{(\mu\rho)} / (s^2)] \times \{ J_1(ir\sqrt{(s/\nu)}) / J_1(ir_0\sqrt{(s/\nu)}) \} \quad (7)$$

For the limiting case, $r_0 \rightarrow \infty$, one finds, after some calculations,

$$\tau(t) = \mu \partial v_z / \partial r|_{r=\infty} = [-2a_0 / (\sqrt{\pi})] \sqrt{(\mu\rho t)} \quad (8)$$

Accordingly, the shear stress increases with time because the shear rate at the surface increases as the fluid lags behind the accelerated surface.

We now use the Δv parameter in order to interpret this result in terms of crash severity. Δv equals the integral of the acceleration of the vehicle under consideration over the collision time, Δt . At a constant acceleration the integration is straightforward and yields $\Delta v = a_0 \Delta t$. The shear stress is maximal ($\tau_{\max}$) at the end of the time interval Δt . The above formula (8) furnishes

$$\begin{aligned}\tau_{\max} &= [-2\sqrt{(\mu\rho/\pi)}]\Delta v/\sqrt{\Delta t} \text{ or} \\ \tau_{\max} &= [-2\sqrt{(\mu\rho/\pi)}]a_0\sqrt{\Delta t}\end{aligned}\quad (9)$$

It follows that the maximal shear stresses at a given Δv increase with the accelerations and Δt , thereby exhibiting a more pronounced dependence on the (mean) acceleration level than on Δt . Although a direct comparison is not straightforward, this result to some extent parallels the concept underlying the Head Injury Criterion (HIC) where the accelerations are given a higher weight than the duration during which they act.

Upon evaluating Eq. (9) for blood as fluid ($\mu = 0.0035 \text{ kg/ms}$, $\rho = 1000 \text{ kg/m}^3$), and choosing as a representative low-speed crash pulse a mean acceleration of 50 m/s^2 and a duration of the loading phase of $\Delta t = 0.1$, $\tau_{\max} = 33 \text{ N/m}^2$ is obtained. This corresponds, e.g., to about the twentyfold value of the shear stress which acts typically on endothelial cells of arterial blood vessels in a healthy adult at rest. The shear stresses determined by LaPlaca and Thibault in their experiments (see Table 1) are furthermore substantially exceeded. Accordingly, it cannot be excluded that the above stresses are in excess of a critical level, yet, a conclusive answer cannot be given at this time since on the one hand the available experimental evidence is hardly sufficient and on the other, the mathematical model presented here is insufficiently refined and adapted to realistic physiological fluid spaces. Because in particular the shear stresses according to Eq. (8) become smaller with decreasing size of the fluid space under consideration, the conclusion may further be relativized, as typical biological fluid spaces are always confined and often minute.

The latter extend in humans over several orders of magnitude, from submicron dimensions to centimeters. In order to qualify the assumption leading to Eq. (8) ($r_0 \rightarrow \infty$), boundary layer thicknesses may be used for comparison. The simplest case of a steady-state boundary layer develops over an oscillating plate and has a thickness of $\sqrt{(2\mu/\rho\omega)}$. This is less than 1 mm, if the frequency, ω , is derived from the loading duration, Δt . Since transient boundary layers are generally smaller than those under corresponding steady-state conditions, it is seen that the assumption $r_0 \rightarrow \infty$ is not really restrictive.

3. An Anatomical Model of the Cervical Spine

In the aforementioned mathematical analysis, a regular geometry has been defined which allows to analyse the effects of fluid-solid interactions formally. A relative orientation of the acceleration pulse along the vessel axis has thereby been chosen because this leads to maximal fluid-solid interactions at the tube

Fig. 4 Finite element model of the cervical spine. A typical venous vessel (right, part of the plexus venosus) is included.

wall and as such can serve as an upper bound for the effect to be examined. Yet, in real neck, the main vessels are oriented in a cranial-caudal direction such that they undergo an acceleration which exhibits a transverse direction with respect to the vessel axis. The following analysis is confined to such a vessel.

A number of mathematical models of the cervical spine have been put forward in the literature^{(26),(29)-(31)}. In view of the inhomogeneous geometry and the non-linear constitutive properties of the involved materials, Finite Element (FE) techniques were usually applied. Realistic results in terms of motion patterns are obtained. Yet, the documented models are generally confined to the solid constituents of the cervical spine, viz., vertebrae, discs, tendons, muscle and connective tissue. Since these constituents represent biological materials and are as such fluid-filled, poroelastic equations have furthermore been applied⁽³²⁾. However, explicit fluid spaces are absent in all of the published models, such that they cannot be used to study the particular question of fluid/solid interactions.

The present new FE model of the cervical spine is based on earlier work by Yang et. al.⁽²⁶⁾. Several extensions were made (Fig. 6^{(27),(28),(31)}) such that the solid part comprises vertebrae, muscles, ligaments and intervertebral discs, furthermore, a representative fluid space (venous vessel as part of the *plexus venosus*) has been implemented. It has to be noted, however, that in a typical rear-end impact scenario, the vessel is accelerated in a perpendicular direction with respect to the vessel axis which is in contrast to the tube considered in the theoretical model before.

The solid part is modeled using the FE system PAMCrash®, while for the fluid model use is made of PAMFlow®. An explicit integration scheme in combination with a weak coupling algorithm is used to integrate concurrent solid and fluid motions.

Fig. 5 Calculated motion sequence for a $\Delta v=15$ km/h rear-end impact. The acceleration at the lower end of the cervical spine was used as input.

The input consisted of an acceleration pulse which is applied at the lower end of the cervical spine. It corresponds to the acceleration measured at T1 in a $\Delta v=15$ km/h rear-end impact⁽²¹⁾. The initial condition for the flow in the sample vessel is 0.2 m/sec in the caudal direction; the pressure gradient due to gravity was neglected. The resulting motion sequence is highlighted in Fig. 7.

The calculated pressures exhibit a significant dependence on the axial location along the neck. This finding can be explained by the differences in the motion pattern as well as by the way the boundary conditions were defined (open at the top, constant pressure at the bottom). While at T2 the pressure deviations from ambient pressure were found to be minimal, the largest values are obtained at the T4 level. The associated curve is seen in Fig. 8 where also two experimental curves are shown. The pressure levels are found to lie within physiological boundaries.

The shear stresses which can be calculated from the velocity profiles in the representative vessel and which act on the vessel wall remain below 1 N/m^2 . This low level, in particular in comparison with the results obtained before, is mainly due to the fact mentioned above that the vessel is accelerated perpendicular to its axis. While all major vessels in the neck have a mainly cranial-caudal direction, there are extravascular fluid spaces, e.g., the cerebrospinal fluid space with other spatial orientations.

4. Discussion and Conclusions

It has been postulated that whiplash occurring under low-speed impact conditions may induce minor soft tissue injuries in the cervical spine whereby the relevant injury mechanisms involve microscopic events. Such injuries can therefore not be detected by routine diagnostic imaging methods such as MRI or

Fig. 6 Pressure pulse obtained in hypothetical venous vessel (cf. Fig. 4). Measurements were made at corresponding locations in the animal and PMHS tests, respectively.

PET. In view of a possible injury mechanism, the significance of fluid-induced viscous shear stresses which are to be expected in certain fluid-filled compartments of the cervical spine during a whiplash-type event has been investigated on the basis of mathematical models.

First, an idealized fluid and solid system (fluid-filled tube) has been analyzed which is exposed to a constant acceleration. The geometry and boundary conditions were chosen such that an analytic solution could be obtained. Second, a neck model was utilized which includes vertebrae, discs, muscles, tendons as well as a representative fluid space (part of the *plexus venosus*). The model was subjected to boundary conditions which correspond to a typical low-speed rear-end impact.

The results from the two models cannot readily be compared, because the fluid-filled tube was assumed to be accelerated in a direction parallel to its orientation, whereas the venous vessel in the neck model was exposed to an acceleration whose primary direction is perpendicular to the vessel axis. The two cases can be regarded as extremes, in that we expect maximal fluid-solid interactions to occur in the first case and minimal in the second. As such, the two models can be considered to span the range of possible physiologic conditions.

We conclude from the models:

— In a crash environment, to which an automobile occupant is exposed under typical low-speed rear-end impact conditions, shear stresses due to fluid/solid interactions can occur in biological fluid spaces of the neck whose level are such that it cannot be excluded that critical limits are reached.

— The larger the fluid spaces, the higher are the shear stresses acting at the surface at a given constant acceleration.

— Given a constant acceleration, a_0 , during the time interval Δt , the height of the acceleration has a stronger influence on the generation of shear stresses than the duration during which it acts. However, at a fixed $\Delta v = a_0 \Delta t$, the exposure of a surface, i.e., of a cell membrane to the shear stress is shorter at higher accelerations.

In the presented theoretical models a number of possibly important effects could not be studied:

— A simple vessel geometry was chosen in both the tube and the neck models. Yet, intravascular and interstitial spaces are often highly tortuous such that more complicated flow patterns as considered here will arise. In particular, inertial effects caused by accelerated fluids within highly tortuous and deformable conduits could lead to critical cellular deformations.

— The boundary conditions chosen for the sample vessel in the FE model (constant initial flow, constant pressure at the lower outlet) are basically physiologic in nature, however, they could likewise exhibit some dependence on the crash pulse. Yet, there is neither experimental nor theoretical evidence which could be used for a possibly more realistic assumption.

— The influence of the main orientation of the vessel axis has not been studied. Major vessels in the neck are oriented mainly in a cranial direction, accordingly, the FE model is representative for such fluid spaces. In such spaces, load-induced shear stresses were found to be insignificant. Yet, in extravascular regions, fluid spaces may have any orientation and geometric configuration. Although their sizes generally are of microscopic dimensions, critical shear stresses can therefore not a priori be excluded based on the results obtained in this communication. This applies in particular for cerebrospinal fluid spaces which are not necessarily microscopic and whose orientation may deviate substantially from a cranial-caudal direction.

— The models are highly nonlinear and therefore not scalable. An extrapolation to include the complexity of cellular spaces is not straightforward.

Further work will have to concentrate on a geometrical refinement of the models in order to mimic cellular dimensions more realistically than is presently possible.

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