INJURY MECHANISMS AND BIOFIDELITY OF DUMMIES

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Abstract

The principal aim of impact biomechanics is the prevention of injury through environmental modification, such as the provision of an airbag for automotive occupants to protect them during a frontal crash. To achieve this aim effectively, it is necessary that workers in the field have a clear understanding of the mechanisms of injury, be able to describe the mechanical response of the tissues involved, have a basic understanding of human tolerance to impact, and be in possession of tools which can be used as human surrogates to assess a particular injury. The tools can be in the form of anthropometric test dummies or mathematical models for simulation of impact events and evaluation of injury risks. This article addresses the biomechanics of head and neck, thorocolumbar spine, and chest and abdomen. It provides basic information on injury mechanisms, biomechanical responses, human tolerances and surrogates to evaluate safety systems for injury prevention.

1. Head and Neck

1.1. INJURY MECHANISMS

1.1.1. Head Injury Mechanisms
It is postulated that the relative motion of the brain surface with respect to the rough inner surface of the skull results in surface contusions on the inferior surfaces of the frontal and temporal lobes and the tearing of bridging veins between the brain and the dura mater, the principal membrane protecting the brain beneath the skull. The irregular geometry and surface of intracranial bones and membranes contributes to deformation of the brain upon severe head impact that can result in injury. Gennarelli et al. [1] have found that rotational acceleration of the head can cause a diffuse injury
to the white matter of the brain in animal models, as evidenced by retraction balls developing along the axons of injured nerves. This injury was described by Strich [2] as diffuse axonal injury (DAI) in the white matter of autopsied human brains. Other researchers, including Lighthall et al. [3] have been able to cause DAI in the brain of a ferret by the application of direct impact to the brain without an associated head angular acceleration. Adams et al. [4] indicated that DAI is the most important factor in severe head injury, as it is irreversible and leads to incapacitation and dementia. It is postulated that DAI occurs as a result of the mechanical insult but cannot be detected by staining techniques at autopsy unless the patient survives the injury for at least several hours.

Among the other theories of brain injury due to blunt impact, are changes in intracranial pressure and the development of shear strains in the brain. Positive pressure increases are found in the brain behind the site of impact on the skull. Rapid acceleration of the head, in-bending of the skull and the propagation of a compressive pressure wave are proposed as mechanisms for the generation of intracranial compression which causes local contusion of the brain tissue. At the contrecoup site, there is an opposite response in the form of a negative pressure pulse which also causes bruising. It is not clear as to whether the injury is due to the negative pressure itself (tensile loading) or to a cavitation phenomenon similar to that seen on the surfaces of propellers of ships (compression loading). The pressure differential across the brain necessarily results in a pressure gradient which can give rise to shear strains developing within the deep structures of the brain. Furthermore, when the head is impacted, it not only translates but also rotates about the neck and in reaction to the orientation of the impact load.

1.1.2. Neck Injury Mechanisms

Injuries to the upper cervical spine, particularly at the atlanto-occipital joint, are considered to be more serious and life-threatening than those at the lower level. The atlanto-occipital joint can be dislocated either by an axial torsional load or a shear force applied in the anteroposterior direction or vice versa. A large compression force can cause the arches of C1 to fracture, breaking it up into two to four sections. The odontoid process of C2 is also a vulnerable area.

Hyperflexion of the neck is a common cause of odontoid fractures, and a large percentage of these injuries are related to automotive crashes of largely unrestrained occupants [5]. Fractures through the pars interarticularis of C2, commonly known as "hangman's" fractures in automotive collisions, are the result of a combined axial compression and extension (rearward bending) of the cervical spine. Impact of the forehead and face of unrestrained occupants with the windshield can result in this injury. Garfin and Rothman [6] discussed this injury in relation to hanging and traced the history of this mode of execution. It was estimated by a British judiciary committee that the energy required to cause a hangman's fracture was 1,708 N.m (1,260 ft-lb).

In automotive crashes, the loading on the neck due to head contact force is usually a combination of an axial or shear load with bending. Bending loads are almost