

Controlled Cortical Impact: A New Experimental Brain Injury Model

JAMES W. LIGHTHALL

PAPER PRESENTED BY JOHN MELVIN

ABSTRACT

A new experimental model of mechanical brain injury was produced in the laboratory ferret (*Mustela putorius furo*) using a stroke-constrained pneumatic impactor. Cortical impacts were made on vertex to the intact dura mater overlying the cerebral cortex with contact velocities ranging from 2.0 to 4.0 m/sec and with deformations of 2.0 to 5.0 mm. The dwell time of the impact and the stability of the skull during impact were verified with high speed (1000 to 3000 frames/sec) cineradiography. Systemic arterial blood pressure, heart rate, and respiration were monitored, and postinjury changes were recorded. Anatomic brain injury, including subdural hematoma, subarachnoid hemorrhage, tears or rents of the dura mater, and contusions of the cortex, brainstem, cervical spinal cord, and cerebellum. Injury responses ranged from no apparent anatomic injury or alterations in the systemic physiology at low severity impact (2.0 m/sec, 2.0 mm) to immediate fatality in the highest severity impact groups (4.0 m/sec, 4.0 mm). The range of changes in systemic physiology and of pathology in the brain, brainstem, and spinal cord was a function of both contact velocity and the amount of brain deformation. In two cases where postinjury time was 8-10 h, diffuse axonal injury, indicated by beaded axons and retraction balls, was present in subcortical regions underlying the site of impact. The spectrum of anatomic injury and systemic physiologic responses closely resembled aspects of closed head injury seen clinically. This procedure complements and improves on existing techniques by allowing independent control of contact velocity and level of deformation of the brain to facilitate biomechanical and analytic modeling of brain trauma. Graded cortical contusions and subcortical injury are produced by precisely controlled brain deformations, thereby allowing questions to be addressed regarding the influence of contact velocity and level of deformation on the anatomic and functional severity of brain injury.

Biomedical Science Department, General Motors Research Laboratories, Warren, Michigan.

DISCUSSION

PAPER: Controlled Cortical Impact, A New Experimental Brain Injury Model

SPEAKER: John Melvin, General Motors Research Lab

Q: Tony Sances, Medical College at Wisconsin

John, that's a very elegant experiment you have. You're to be congratulated. It's very nice.

A: Melvin

Congratulate Jim Lighthall, I'm just presenting it for the audience here but it's Jim's work.

Q: I was wondering if you think that the trauma that you are seeing, at least the histological changes down into the brain stem and spinal cord, could be secondary to tension propagated responses. Have you been able to identify that with your high speed x-ray?

A: No, we need to target this system. The original experiments, which I presented here were to find out if we could produce, in particular, the diffuse axonal injury. That was really what we were concerned about. The experiments are really not biomechanical at this point because we really didn't measure anything other than the preset velocity and stroke, which were not continuously measured. The next series of experiments will involve animals that are exposed to certain test conditions and then similar animals with a lot of instrumentation and targeting built in, to look at exactly the kinds of things you're talking about: Where are things going, what's moving, what's being stretched, what's being sheared, what are the pressures associated with this involvement, obviously pressures created in this process as well as shearing stresses. Many things remain to be done.

Q: Colleagues in our laboratory have found that there have been distal propagated tension responses by putting markers on the spinal cord. The radioopaque markers, show the propagation of substantial distance from the action site, particularly if you're dropping a ball on the spinal cord or if you're using the tension model.

A: Yes.

Q: Dorothy Richardson, Ford Motor Company

What was the load that you could impact on the tissue of the brain before you'd start seeing hypertension brain pressure?

A: We didn't measure any load in those experiments, unfortunately. That's one of the things we are going to measure in the next series. We felt that we needed to explore whether or not the kind of injury we wanted could be produced in this limited series, before we went through all the extra difficulty of measuring the load. Measuring the load is very difficult. If you've ever had anything to do with brain tissue it deforms at very low force levels. We have a problem here, that we stop this stroke and turn it around very quickly. We have to subtract out an inertial component. The load cell has to be inertially compensated, yet small. and it's quite a design problem. We're in the process of solving that right now. We've been fairly successful but we have not applied it to the experiment yet. We really can't say what the forces are.

Q: Marc Weiss, Naval Biodynamics Lab

On the defuse injury, you mentioned that it took some period of time to develop. I have a technical question. What was the optimal time between the time of inducing the injury and the time you perfused the animal to get maximum detectable pathology of the defused sort?

A: This occurred in about four to five days. I don't think that there's an optimal time. If you do it too soon you won't see anything. If you wait longer, more may even develop. It is a developing process and its been explained to me as simply a clogging of the axon and the building up of that matter in the form of a wall.