ON THE MECHANISM OF CELLULAR DAMAGE BY SHOCK WAVES

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ABSTRACT

The present study addresses the mechanism by which shock waves damage cells and may lead to traumatic brain injury (TBI). Fibroblasts (3T3) cell cultures were exposed to pressure waves produced by a reacting mechanically activated thermite mixture of Al+CuO in water. The shock over-pressures were approximately 200-300 kPa and the peak pressures obtained were 1 MPa. Fluorescent images of the cells before and after pressure wave exposure were used to determine the type of damage suffered by the cells and whether the cells have died. Immediately after the exposure to the pressure waves, the cells were found to have their actin filament de-polymerized. If allowed to recover for 60 minutes the 3T3 cells were found to have their actin re-polymerized. While the link to TBI is not clear at present, the re-polymerization stage is known to be a period during which the cell ceases its normal functions and must reconstruct its filament. The pressure threshold for cellular modification by shock waves was also addressed by considering the shock thickness that is comparable with the characteristic dimension of cellular components. The shock thickness associated with the shock overpressure investigated in the present study correspond approximately to the dimension of the cells, approximately 30 m. Stronger shocks known to cause damage to the neurons have a thickness comparable to the nucleus. The coincidence of the shock thickness with the dimension of the cellular component damaged by the shock supports the view that cell damage or death may be related to the localized mechanical deformation of cell components for sufficiently strong and thin shocks.