Micro Air Embolism Contribution to Blast-Induced Mild Traumatic Brain Injury

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RESULTS (CON’T)

Massive air embolism (AE) from lung disruption is the accepted principal etiology of mortality in blast injury (White et al., 1971). Blast-induced AE is recognized as the chief complication of primary blast injuries (Buamoul, 2009). For mammals that die promptly from either air or underwater blast, air embolism has long been recognized as the primary cause of death (Bauman et al., 2009). Recent work by Yang et al., 1996 (sheep model) suggests the lung damage threshold pressure may be as low as 50% of the 50% mortality level (White et al., 1971). Recent data (Yang et al., 1996) suggests the threshold pressures for lung damage may be lower (circa 50%) than those shown.

Blast-related bubble production, when it does occur, has been shown to be transient, lasting only 15 minutes to 3 hours (Richmond,1968). Data shown is for a single reflection where the total overpressure is ~2x incident pressure. Total pressures can be up to 8x incident pressure if dispersion of the blast gases, and by longer exposure times. It is further lowered by repeat exposures in less than 24 hours (Stuhmiller, Phillips & Richmond, 1990).

Maison (1971) outfitted a dog with a Doppler bubble detector on the carotid artery, exposed the dog to an LD50 air blast, and subsequently observed bubble detections going up the carotid correlating with respirations for approximately 30 minutes post-blast. The dog’s chest blood flow was observed to temporarily drop to near zero following each group of echoes, possibly indicating reduced blood velocity due to temporary local occlusions (Fig. 2). The dog initially showed severe respiratory distress, but recovered. Positronenium exam showed evidence of residual lung hemorrhage, but no other damage. Maison concluded that the bubbles were “clinically silent.”

A conceptual model of how AE sequelae to blast exposure occurs, confirmed with rat model data, can be found in Richmond (1971). Any fast-rising blast pressure wave long enough to produce significant chest compression is likely to produce some AE.

Evidence that microbubbles are NOT harmless

Microbubbles were first recognized as a medical hazard in open-heart surgery decades ago (Barak & Katz 2005). An embol from various sources in the extracorporeal circulation (ECC) set and tuben can drift into the heart and systemic circulation, especially if the suction in the myocardial side of the heart-lung machine is not functioning. ENM (endocardial) injury, neurologic injury, pulmonary hypertension and other injuries were well known in the literature before the advent of ECC. For many years, microbubbles were considered to be a negligible hazard to patients, even over a period of many years.

A possible complication of exposure to air blast. However, neither author addresses the possibility of neurocognitive sequelae from AE.

Microbubbles have been found to cause significant neurocognitive sequelae in humans. Patients with neuropsychological deficits 5 to 7 days after coronary bypass graft surgery averaged nearly twice the number of emboli compared to those without deficits (Stump, et al., 1996). In mechanical heart valve carriers, microbubbles are chronically delivered into the arterial system at variable rates, which can rise as high as 1000 per hour in the cerebral circulation. Patients with these devices have been found to have impairment in episodic memory and deficits in working memory (Deklunder et al., 1998). Multiple brain lesions in dogs with a reported history of neurological DCS have been found to be strongly correlated with repeated episodes of high oxygen/hemoglobin saturation. This finding led the authors to a hypothesis that the brain lesions were the consequence of subclinical central gas embolism (Knauf et al., 1997).

A number of 145 cases of delayed DCS treatment (acute, delay 33.3 hrs) reported findings of neurological and neuropsychological injury, including severe and extended cognitive dysfunction, memory and test scores in the 5% range. Patients with these devices have been found to have impairment in episodic memory and deficits in working memory (Deklunder et al., 1998). Microbubble exposure has been found to cause significant neurocognitive sequelae in humans. Patients with neuropsychological deficits 5 to 7 days after coronary bypass graft surgery averaged nearly twice the number of emboli compared to those without deficits (Stump, et al., 1996).

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The contribution of micro air embolism to blast-related brain injury may be significantly greater than has been previously believed.

SUMMARY/CONCLUSIONS

• In hemorrhagic, CIG, or cervicodorsal to microbubbles have been correlated with the duration of delayed DCS treatment. Barak & Katz (2005) attributed the abnormalities to microbubbles and stated “a small quantity of microbubbles may be trivially incident, while recurrent exposure has a more, embolizing, chronic effect” (p. 2021)

• Baumert et al. (2009) provides a summary of the test conditions and initial results from the PREVENT (Preventing Violent Explosive Neurotrauma) research program being conducted by DARPA. In the tests reported (same model), the lung and upper abdomen were protected to minimize the probability of brain injury by indirect pathways. Some neurological damage was observed, and its significance is still being determined. However, the best conditions are of interest as they are also ones where lung injury can readily occur. Point C on Fig. 1 represents a typical Friedenberger model expected for the blast tube. Test set-ups used were to deposit embol in the crow compartments of a Hamster with a blast under its head and an open gutter port and in semi-confined spaces-open top room with dimensions as shown in Fig. 1. In both cases the pressure durations from a moderate-sized shock wave charge were reported to be about 4 ms. The pressure data was reported in general form only with numerical values. However, if 4 ms duration, then pressure required to produce lung injury is not large. In situations where the Hamster or building were to be fully closed, both the magnitude and duration of blast overpressures can be expected to be greater; 2001. (2003) reports results from a computer model developed by Defense R & C (CRDC) for estimating the blast damage to the lungs of sheep and humans. He reports the intrathoracic pressure range currently accepted as the ‘threshold’ for lung damage is 7 kPa (55 cmH2O) to 113 kPa (850 cmH2O), which corresponds quite well to the intra-thoracic pressures predicted by the model at exposures near the lung damage threshold line on the Bowden chart. The intra-thoracic pressures produced by even moderate size blasts can be very substantial (They may exceed 100 kPa [750 cmH2O] for 1-2 ms duration if the blast is in a dense volume). The model also indicates that complete (full-compressed) blast waves can produce higher lung pressures, and therefore greater risk of lung damage than an implosive phase. The Friedenberger waves of the same implosion.

• Recent work by Yang et al., 1996 (sheep model) suggests the lung damage threshold pressure may be as low as 50% compared to the lower end of the Bowden chart. The contribution of blast-related AE is a continuum ranging from no bubbles, to a few bubbles, to many bubbles, to a full compression of the lungs. It is reasonable to expect that the degree of blast-related AE is a continuum ranging from no bubbles, to a few bubbles, to many bubbles, to a full compression of the lungs. It is reasonable to expect that the degree of blast-related AE is a continuum ranging from no bubbles, to a few bubbles, to many bubbles, to a full compression of the lungs.

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