Military trauma

The aim of a research was to analyse military trauma publications over the last 16 years of armed conflict in order to highlight the most important lessons that have translated into civilian practice and military doctrine as well as identify emerging areas of importance.

A systematic search of research published between January 2000 and December 2016 was conducted using the Thompson Reuters Web of Science database. Both primary evidence and review publications were included. Results were categorised according to relevance and topic and the 30 most cited publications were reviewed in full. The h-index, impact factors, citation counts and citation analysis were used to evaluate results.

A plateau in the number of annual publications on military trauma was found, as was a shift away from publications on wound and mortality epidemiology to publications on traumatic brain injury (TBI), neurosurgery or blast injury to the head. Extensive collaboration networks exist between highly contributing authors and institutions, but less collaboration between authors from different countries. The USA produced the majority of recent publications, followed by the UK, Germany and Israel.

In recent years, the number of publications on TBI, neurosurgery or blast injury to the head has increased. It is likely that the lessons of recent conflicts will continue to influence civilian medical practice, particularly regarding the long-term effects of blast-related TBI¹⁾.

Modeling this disease in rodents to pre-clinically evaluate potential therapeutics has been challenging because of inconsistency between models. Although the effects of primary blast wave injury have been extensively studied, little is known regarding the effects of noncontact rotational TBIs independent of the blast wave. To model this type of injury, we generated an air cannon system that does not produce a blast wave, but generates enough air pressure to cause rotational TBI. Mice exposed to this type of injury showed deficits in cognitive and motor task acquisition within 1-2 weeks post-injury, but mice tested 7-8 weeks post-injury did not retain any deficits. This suggests that the effects of a single, noncontact rotational TBI are not long lasting. Despite the transient nature of the behavioral deficits, increased levels of phosphorylated tau were observed at 2 and 8 weeks post-injury; however, this tau did not adopt typical pathological structures that have been observed in other TBI models that incorporate blast waves. This was possibly attributed to the fact that this injury was insufficient to induce changes in microglial activation, which was not affected at 2 or 8 weeks post-injury. Taken together, these data suggest that exposure to noncontact, rotational head injury only produces transient cognitive anomalies, but elicits some minor lasting neuropathological changes².

1)

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