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Invited review

The past and present of blast injury research in China

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ABSTRACT

With the increasing incidence of blast injury, the research on its mechanisms and protective measures draws more and more attention. Blast injury has many characteristics different from general war injuries or trauma. For example, soldiers often have various degrees of visceral injury without significant surface damage, combined injuries and arterial air embolism. Researchers in China began to investigate blast injury later than the United States and Sweden, but the development is so fast that lots of achievements have been gained, including the development of biological shock tube, the mechanisms and characteristics of blast injury in various organs, as well as protective measures under special environments. This article reviews the past and current situation of blast injury research in China.

Blast injury refers to damage and mortality caused by shock wave induced by a variety of factors in humans or animals. These factors include the nuclear explosions, bombs and other explosive weapons in wartime, as well as the explosions of gas, military and chemical factories, ammunition depot which are more common in peacetime. Blast injury research started from the 1950's in China and has drawn considerable attention since 1964 when China successfully launched its first atomic bomb. A lot of achievements about blast injury research have been gained, and the first monograph, *blast injury*, was published during that time.[1] In the future, the extensive use of a new generation of high-explosive weapons can lead to large quantities of various blast injuries. In peacetime, some unexpected events, such as improvised explosive device used by terrorists and the traffic accidents, could also increase the incidence of blast injury. The present review provides basic knowledge in the development of shock tube, characteristics and mechanisms of the injury, as well as protective measures under specific environments.

1. Development of biological shock tube

In the first two decades, trinitrotoluene (TNT), cyclotrimethylenetrinitramine and detonators were the main compounds used to make outdoor detonation.[2] Explosives have a couple of advantages when used in blast injury research, such as easy processing, no special equipment required, etc. But this type of test needs higher expenses, lots of manpower and material resources, and pathophysiological test is inconvenient to be performed at test site. In addition, making a collection and analysis of data is very difficult because test parameters are not stable and the repeatability might be a problem. To solve this problem, in 1988 the researches in Third Military Medical University (TMMU, China) developed the first biological shock tube, BST-I, to mimic blast injury in cooperation with Institute of Mechanics in Chinese Academy of Sciences. This tube is 39.34 m in length and 1 m in diameter for test segment. The overpressure of the tube can reach up to 219 kPa and 630.3 kPa respectively when the end is opened or closed. In addition to overpressure, this instrument also produces underpressure. The investigators identified that a various extent of injury, from mild to very severe, could be produced by BST instrument using 1459 animals (757 rats, 105 guinea pigs, 335 rabbits, 240 dogs and 22 sheep).[3–5] This device replaces explosive air with compressed air to produce high-speed pressure wave in order to avoid explosion burns caused by the damage resulting from debris to the organism. When the peak pressure reaches 200.2 kPa–254.4 kPa, the tube causes mild injury, and 293 kPa-322.2 kPa and 361.2 kPa–418.3 kPa for moderate and severe injuries respectively with a 15 ms–32 ms duration of positive pressure effect.[6] Since then, BST-II and BST-III type bio-shock tube in different sizes and functions were developed. BST-II type tube is 34.5 m in length and is equipped with a couple of test segments in

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different inner diameter, including 0.77, 1, 2, 3.5 and 6 m. It can simulate plateau environment, underwater explosions, explosive decompression and high-speed airflow effects. BST-III type (0.5 m in length, 2 mm−10 mm in length for test segment) can be used to generate point explosion at local site and is designed with a rack that can be used to regulate the direction of the blast wave.[3] In the United States, the blast injury research has started by employing shock tube since the 1950's,[7,8] and then Jaffin et al[9] developed a shock tube in small size to perform animal experiments in 1987. Compared to these shock tubes, the one designed by Wang and his colleagues made a great progress, who designed a series of tubes (large, medium and small ones) to generate overpressure and underpressure simultaneously. In addition, the length and the diameters could be grouped in different patterns in order to realistically simulate the blast injury under special environments, and these instruments are the most advanced bio-shock tubes in the world in that time.

2. The characteristics of blast injury

Because of diversity of inducing factors and categories, blast injuries are often complicated with multiple injuries. The soldiers exposed to blast shock were seriously debilitated or killed without obvious external symptoms and signs. The target organs are mainly lungs, eardrum, gastrointestinal tract and other air-containing organs.[1,10]

Blast injury usually is divided into four types: the first one, also known as primary blast injury, refers to the shock wave generated by explosives or bio-shock tubes and directly acting on the body. The secondary blast injury refers to damages caused by the high-energy debris, steel casings and bricks blown by blast wave. The third type of blast injury is the blunt force-induced trauma when body is thrown out rapidly and suddenly decelerated after crashing on hard objects. The fourth one refers to the damages caused by building collapse or other factors.[11] The wounded soldiers usually have more than one injury, and blast injury discussed in this article refers to primary blast except the specific instruction.

According to the clinical manifestation, blast injuries are divided into the following four categories: mild injury: with symptoms of hearing impairment, surface scratches, scattered punctate hemorrhage; moderate injury: presence of severe hearing impairment (fracture of ossicles, hemotympanum), large areas of concussion in soft tissue, multiple patch hemorrhage or hematoma formation, mild interstitial pulmonary edema, simple dislocation or fracture and brain concussion; severe injury: severe pulmonary hemorrhage and edema (interstitial and alveolar), ruptured liver, spleen, stomach or bladder, the multiple fractures of limb, spine, pelvis, skull or rib, and severe crush injuries, etc; Very severe injury: presence of an extensive pulmonary hemorrhage, severe pulmonary edema, head injury, the ruptured thorax, abdomen, internal organs and large blood vessels. The mortality is very high for patients with severe and very severe blast injury. The causes of death are very complicated, and early death is mostly due to severe brain and spinal cord injuries, severe hemorrhage caused by internal organ rupture (hemorrhagic shock) and multiple fractures (shock and fat embolism). Overpressure could directly lead to death because it causes coronary air embolism, acute respiratory and circulatory failure. The causes of death in the late period are mainly perforative peritonitis, bronchial pneumonia, sepsis or other secondary infections.[1]

Impulse and increasing duration of pressure are the major physical parameters determining the severity of injury. Impulse depends on the duration of positive pressure and value of pressure. Within a certain range, the duration of positive pressure and value of pressure positively correlated with mortality (Table 1). Wang et al[12] performed blast injury experiments using 256 dogs and summarized the relationship between the peak overpressure and the injury (Table 2).

Table 1
Overpressure duration and overpressure value of different lethal dose (LD).

Overpressure duration (ms)	LD		
	LD1	LD50	LD99
400	2.6	3.7	5.1
60	2.9	4.1	5.6
30	3.2	4.5	6.2
10	4.9	6.9	9.5
5	9.2	13	17.5
3	21.9	30.4	42.3

In addition, repeated blast wave at low intensity displays an additive effect and reduces threshold for damage. When using 10 kg TNT to analog muzzle shock, the injury threshold for lung, gastrointestinal and upper respiratory tract was 37.27 kPa, 41.0 kPa and 77.0 kPa respectively, and it reduces to 20.2, 20.2 and 40.89 kPa after several explosions. Lung is the most vulnerable organ in blast injury.[13,14]

2.1. Pulmonary blast injury

Lung is the most important target organ in blast injury because it is air-containing. The pathological changes in pulmonary injury mainly manifest as pulmonary hemorrhage, edema, rupture, bullae, collapse and emphysema. Among these symptoms, pulmonary hemorrhage has the highest incidence (79.1%) and is prone to occur in the left lung, followed by pulmonary edema (65.1%), bullae (48.5%) and rupture (36.7%).[15] In 1989, Wang et al[14,16] proved that mutually parallel bleeding stripes are not the indentation of rib, but bleeding stripes between ribs, which is the characteristic of pulmonary blast injury. This result was included in the US military medical textbook. Pulmonary bleeding is more serious at the side towards the center of explosion and is obvious along the distribution of deep bronchi.[17] Low-intensity blast wave makes the gap between endothelial cells widen. Under moderate- and high-intensity blast wave, lung endothelial cells drop off and tight junction between epithelial cells and capillary endothelial cells is destroyed.[18] Zhao et al[19–22] found that the arithmetic mean thickness of blood-air barrier increased in rat alveoli by a quantitative research on morphology changes of lung, mainly due to the increase in the endothelial thickness. In diagnosis of the mild and moderate injuries, observing the change of blood shunt is more significant than blood gas analysis.[23] In addition, the biomechanical research in pulmonary blast injury provides data to support the establishment of the models and the explanation of mechanism.[24–26] Chen et al[27,28] found that in addition to overpressure effects on the lungs, decompression of the blast wave would cause over-expansion and over-speed expansion of lung which could lead to alveolar edema, tearing and other injuries.

2.2. Auditory blast injury

The ear is the most frequently damaged organ in blast injury in wartime. The overpressure of blast wave is the main cause of eardrum perforation which can be used to determine the farthest boundary of the damage caused by blast wave.[1] But this method is not very accurate, and sometimes lung damage occurs without eardrum injury, indicating large differences in eardrum damage. In addition to damage to the eardrum, some patients had inner ear impairment, manifested as hearing loss which is different from noise-induced hearing loss, characterized by decrease in high-