Review

Mechanisms and biological consequences of the blast-body/head interactions

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Received: April 3, 2018 Accepted: July 20, 2018

Abstract

Blast injuries, including blast-induced neurotrauma (BINT), are caused by blast waves generated during an explosion. Due to frequent use of explosives both in military and industry, blast injuries show an increasing trend. As the pathobiology and progress of these injuries are extremely complex and unique, there is a pressing need for better understanding the mechanisms of blast-body interactions and their biological consequences. The translational value of experimental biomedical research addressing blast injuries fully depends on the experimental models' capacity to replicate clinically relevant conditions; this in turn hinges on adequate understanding of blast physics. Nevertheless, often, there is a miscommunication between blast physicists, engineers and biomedical researchers mainly due to different professional terminology and limited understanding of biological/physiological or physical principles, respectively. This review provides a short overview on the physical mechanisms of the blast wave-body interactions and the biological effects of such interactions.

Keywords: explosives, blast, blast injuries, biological effects, blast-induced neurotrauma

1. Introduction

A systematic review, based on cohort studies concerning the prevalence and characteristics of battlefield injuries among the North Atlantic Treaty Organization (NATO) coalition forces from Iraq and Afghanistan up to December 20th 2013, showed that 72% of all injuries were caused by explosion[1]. Others also confirmed explosions causing the majority of recent military injuries[2-4]. Thus, there is an unquestionable need for better understanding the mechanisms, progress, and outcomes of injuries caused by explosions. Indeed, the development of improved prevention, diagnosis, treatment and rehabilitation of explosive injuries hinges on how well we grasp the interactions between the injurious forces of explosion and the human body.

This review will use the terminology, definitions, findings and opinions related to blast and its effects, which have been published by biomedical researchers. As such, they could be perceived as incorrect by physicists, chemists, or engineers. Nevertheless, this review reflects the interpretation of blast and blast injuries made by biomedical researchers with a main goal to stimulate multi-disciplinary discussions and knowledge sharing.

2. Explosives and their biological effects

As the explosive environment determines the type and severity of blast injuries, a basic knowledge of explosives and blast physics is necessary to understand the mechanisms of blast-body interactions and their biological consequences[5].

Gunpowder and other explosives are undoubtedly among the legacies of ancient China. Metal barrels have been used to fire lances that propelled gunpowder bombs since the Tang dynasty, circa 618-907 AD[6]. From that time, explosive materials have significantly changed. Today, we can differentiate between low or high explosives[7] (Figure 1). Low, or deflagrating, explosives, such as gunpowder are readily combustible substances, which, when set, burn and produce gas that forces a bullet or shell smoothly out of the barrel. High or detonating explosives (for example, trinitrotoluene/(TNT), cyclonite/RDX or ammonium nitrate, among others) have a

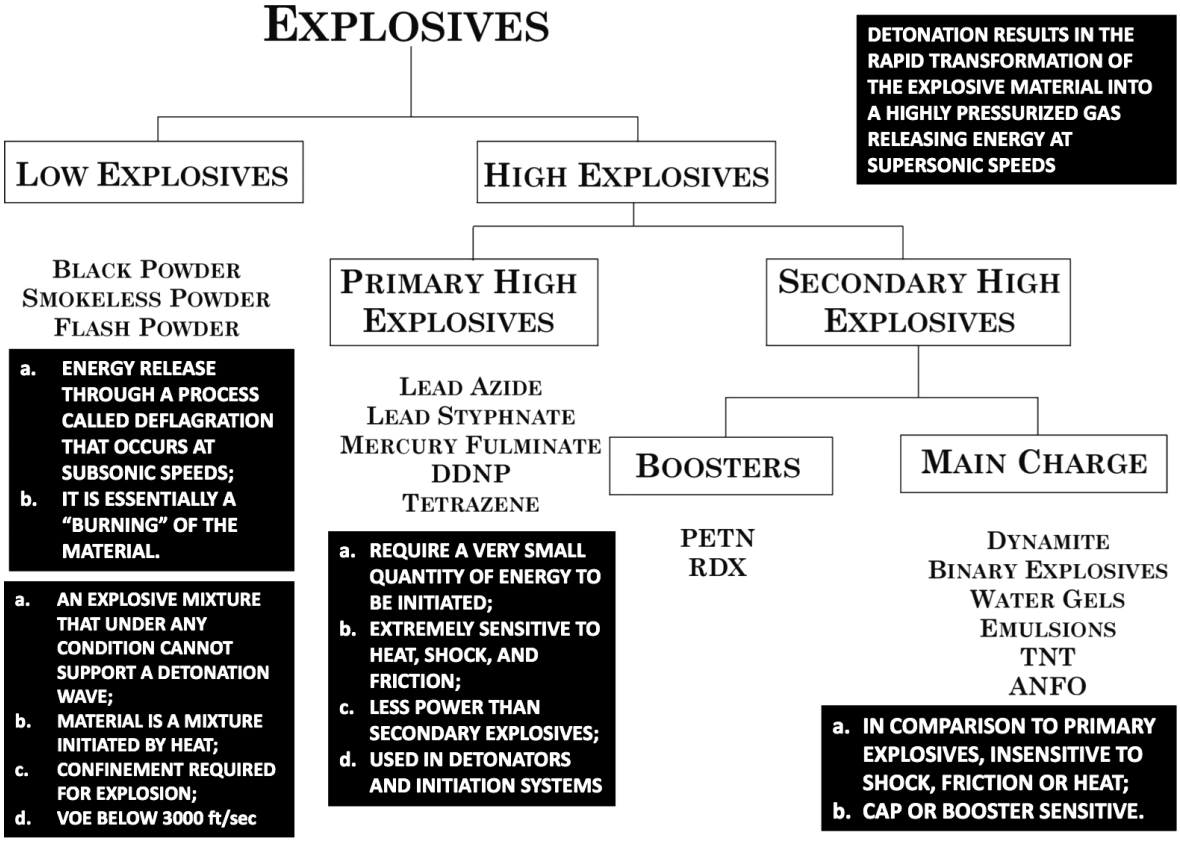

Figure 1 The basic characteristics of the explosives frequently used in military and/or industry.

shattering effect when detonated. Because these materials consist of unstable molecules, their explosive decomposition produces shock waves as end products and they do not require any external source of oxygen. From the perspective of blast injury etiology, it is important to remember that explosive reactions differ from ordinary combustion in the velocity of the reaction. While the combustion velocity of low-energy explosives is slow and may vary within a broad range depending upon the type and physical state of the explosive material, the velocity or time of reaction for high-energy explosives is fast. Consequently, in general, injuries caused by high explosives are more complex and severe as compared with low explosive-induced pathologies.

Historically, the weapon development efforts were directed toward improving the shattering effects of high explosives, i.e., the efficiency to propel metal fragments and shaped charges with high velocity into the surroundings. Consequently, the main strategy focused on overcoming the protective capacity of the enemy's body and/or vehicle armor and on inflicting blunt or penetrating injuries. While previously little attention has been paid on improving blast wave performance, there is a more frequent usage of enhanced blast explosives (for example, fuel-air explosives / FAEs and thermobaric bombs) recently[8]. It is noteworthy that blast waves produced by FAE weapons differ from conventional high explosives. Namely, in the vicinity of explosion, the blast waves generated by conventional high military explosives have a high peak pressure and a relatively short duration, whereas, around the FAE explosions, the peak pressures are lower than for TNT but with a significantly longer duration[8),9)]. The changes in energetic and additive materials incorporated into explosive weaponry significantly modify their injurious effects. This is well illustrated by the shift of injury types from mainly penetrating or blunt injuries during the first three quarters of the 20th century to blast injuries during military actions after that. Consequently, blast injuries should be analyzed from a historical perspective, bearing in mind the material characteristics of explosives used in any particular conflict. For example, the blast injuries of World War I (WWI) vastly differ from those of Operation Iraqi Freedom (OIF), partly because of the significant differences in energetic materials used for explosive weaponry.

3. Mechanisms of blast-body interactions

3.1 Blast exposure features

Blast, one of the products of explosion, is a region of highly compressed gas that expands rapidly to occupy a volume several times greater than that of the original explosive, the solid residues from the explosive or its casing added together[10]. The blast wave travels faster than sound in a form of a sphere of compressed and fast-expanding gases. As it moves from the center of explosion, the blast displaces and subsequently compresses an equal volume of surrounding air at high velocity. This overpressure phase of the blast wave is followed by a short period of negative pressure, the so-called underpressure phase[11].

The Friedländer pressure form[12] is frequently used to

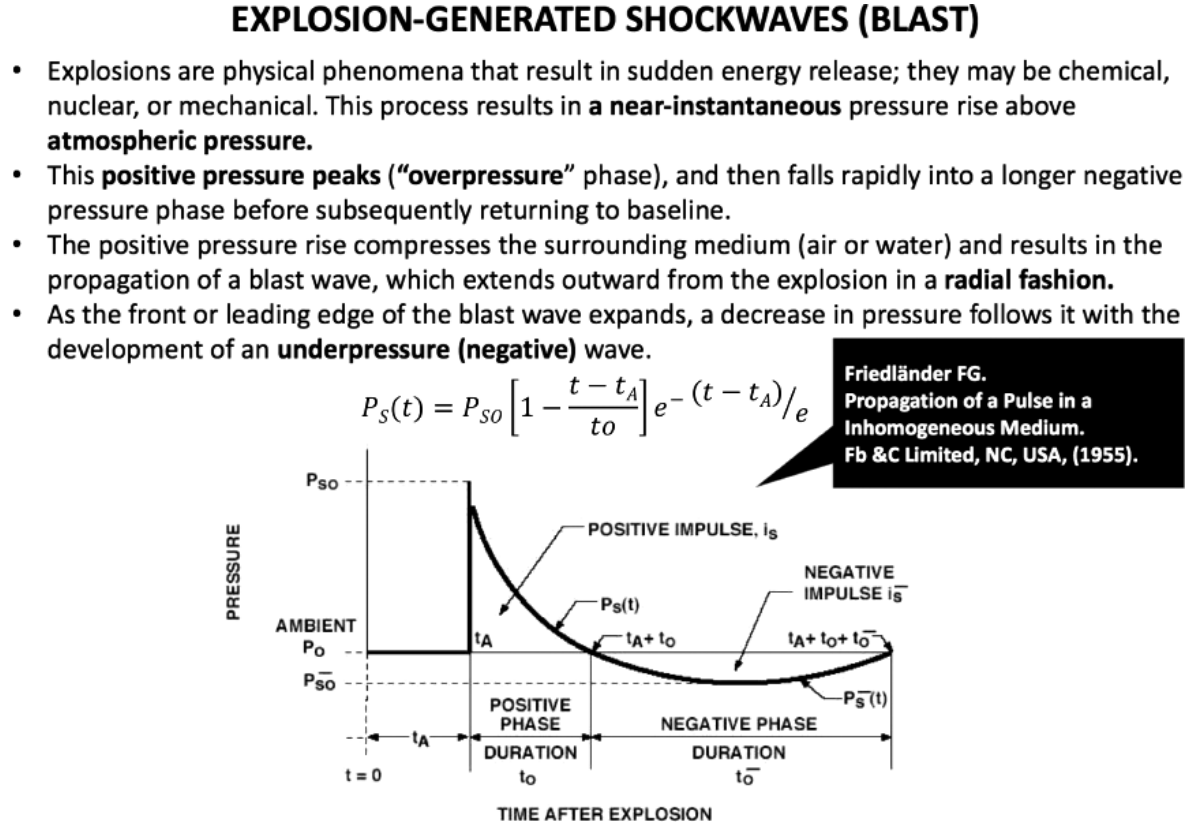

Figure 2 The main components of an idealized, Friedländer waveform as it relates to an explosion-generated shockwave.

graphically illustrate the main components of a blast wave (Figure 2). Nevertheless, it rarely replicates real-life scenarios since even in unobstructed, open field conditions, the blast wave reflects from the ground or from the individual's body generating reflection waves. The subsequent interactions between the primary and reflecting waves amplify the initial pressure wave and makes it more complex. It has been estimated that the resulting pressure can achieve eight-times that of the incident blast wave[13]. The blast wave is followed by a high-velocity, hurricane-like blast wind causing utmost destruction of its surroundings and disintegration, evisceration and traumatic amputation of body parts[14]. The severity of the injuries and the extent of damage caused by a blast wave depend on five main factors[15]: 1) the peak of the initial positive-pressure wave (the overpressure ranges from 690 to 1724 kPa, e.g., 100–250 psi, is considered potentially lethal); 2) the duration of overpressure; 3) the density of the medium in which the explosion occurred (air or water); 4) the distance from the incident blast wave; namely, the intensity of the blast overpressure declines with the cubed root of the distance from the explosion (for example, a person 3 m/10 ft from an explosion is subjected to nine-times more overpressure than a person 6 m or 20 ft away); and 5) the degree of the blast wave's reflection; namely, in complex environments and confined spaces, the intensity of the blast wave can be augmented between two- and nine-times due to reflection from surrounding objects or walls (for example, victims positioned between blast and a building frequently suffer from injuries two- to three-times more severe than a person in an open space).

The effects of explosive blasts on the body are fivefold[5)–17)]: 1) the primary blast effects (the overpressure phase of the blast wave) causes primary blast injuries. During the interactions between the blast wave and a living body, a portion of the shock wave is reflected, whereas another part of its energy is absorbed and propagates through the body as a tissue-transmitted shock wave[18)]; 2) the secondary blast effects (e.g., the fragments of debris propelled by the explosion and connecting with the body) lead to secondary blast injuries, which can be blunt or penetrating[19)]; 3) tertiary blast effects (e.g., acceleration and deceleration of the body or part of the body moved by the kinetic energy released during explosion) inflict tertiary blast injuries[20),21)]; 4) quaternary blast effects (for example, transient but intense heat of the explosion) cause quaternary blast injuries, such as flash burns[22)]; and 5) quinary blast effects, which include a broad variety of potentially injurious factors, such as, carbon monoxide, the "post-detonation environmental contaminants" (for example, bacteria and radiation from dirty bombs), and tissue reactions to fuel and metal residues, among others, cause quinary blast injuries[23)]. Occasionally, when an individual is close to the center of explosion, multiple blast effects may interact with the body in parallel inducing blast injuries of moderate-to-severe severities. Some literature sources call such complex injurious environment and related injuries as "blast plus"[23),24)].

3.2 Mechanisms of blast-body interactions

The high explosive shock wave in air travels with supersonic speed, and such a speed is one of the

characteristics of a real shock wave. Accumulating experimental work conducted by Clemedson and his colleagues in 1950s and 1960s[18),25)–29)], suggests that when a high explosive shock wave strikes a living body, a number of physical events take place:

1) One fraction of the shock wave is reflected from the body, whereas

2) Another fraction of the shock-wave energy is absorbed or transformed into the kinetic energy of the given medium and propagates through the body as tissue-transmitted pressure waves; and

3) The transferred kinetic energy moves and accelerates the media from their resting state with a speed that depends on the density of the given medium.

Although various organ and body structures differ in their reactions, two main general types of tissue response are observed: a) The first one is caused by the impulse of the shock wave and is of longer duration, whereas b) The other is caused by the pressure variations of the shock wave and is in a form of oscillations or pressure deflections of shorter duration. The tissues in the abdomen and costal interspaces react with typical impulse response (type "a"), whereas the rib and the hind leg show a more or less pure maximal pressure-type curve (type "b").

As the original shock wave enters the body, the interaction with heterogeneous tissue elements lead to its dispersion, divergence and attenuation. As a result, the velocity of the wave reduces so that the main part of the pulse travels with sonic or even subsonic speed. Because it does not retain the characteristics of a shock wave in the true sense of the word, using the term "pressure wave" or "pressure pulse" would be more adequate.

Historically, the pressure wave-tissue interactions have been explained by spalling, inertia, implosion and cavitation phenomena[17),30),31)]. Spallation develops at the interface between two media of different densities. When propagating across the denser medium toward a medium of lower density, the pressure wave reflects from the boundary and creates a defect (i.e., crater) in that denser medium, spall fractures and fragments from the boundary. Inertial effects also occur at the interface of the different densities. These effects are based on the fact that tissue components with the lightest density travel the fastest, whereas denser elements trail behind. Thus, as the pressure wave travels through these components, the lighter components will move faster while the movement of the denser ones' slower; this will cause stretch and strain at the interfaces, subsequently leading to displacement, deformation or rupture of tissues and organs[16),25),29)]. Implosion is a phenomenon that happens when the pressure wave passes through a liquid medium, which contains dissolved gas. The kinetic energy of the passing pressure wave compresses the gas bubbles, so that the bubbles' pressure becomes higher than the wave's pressure. After the pressure wave leaves the medium, the bubbles re-expand and burst damaging the surrounding tissue; this mechanism is often called cavitation.

3.3 Mechanisms of blast-head interactions

Potential mechanisms of blast-head interactions include[16),32)]: (1) direct interaction with the head causing skull flexure[33)] and transmission of the kinetic energy into the brain, as well as potential acceleration and/or rotation of the head; and (2) transfer of kinetic energy from the blast wave through large blood vessels in the abdomen

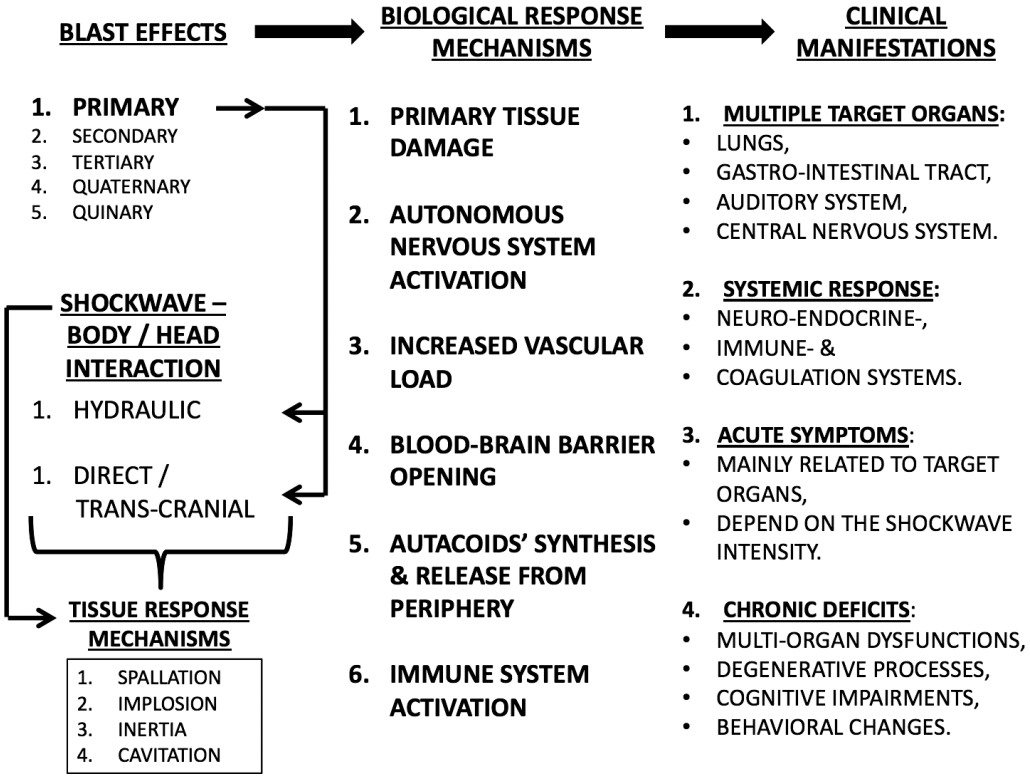

Figure 3 The causal relationship between the blast effects, biological response mechanisms, and consequent clinical manifestations.

and chest, in a form of tissue-transmitted pressure wave, to the central nervous system[27),34)]. The pressure-wave transmitted mainly through the blood initiates oscillating waves that traverse the body at about the speed of sound in water and deliver the kinetic energy of the blast wave to the brain. Once delivered, that kinetic energy causes both morphological and functional damages to distinct brain structures[34)]. These two probable ways of interactions do not exclude each other. The importance of the blast-induced hydrodynamic pulse through venous vasculature has been demonstrated in the experimental work by Simard and colleagues[35)]. It has been suggested that the hydrodynamic pulse spreads from its site of origin away through vasculature, entering the brain via veins without disruption since there are no valves to impede pressure transmission.

4. Biological effects of the blast-body/head interactions

The interactions of the shock wave with the body and head and a consequent passage of a pressure wave through the body and head induce complex response mechanisms (Figure 3), which include: primary tissue damage; autonomous nervous system (ANS) activation; increased vascular load; blood-brain barrier (BBB) opening; effects of locally synthesized and released mediators/modulators (so-called "autacoids"); and immune system activation; among many others. In general, these changes start as reversible functional impairments. Later, often many months and years after the blast exposure(s), the functional impairments might become chronic if they surpass the counteracting defense mechanisms. The chronic impairments trigger neurodegenerative processes as well as other long-term multi-organ deficits such as hormonal insufficiency, cardiovascular instability, dyspepsia and irritable bowel syndrome, among others. If these changes are not identified on time and treated in timely fashion, they might lead to irreversible tissue degeneration and debilitating health impairments.

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