

Mechanisms and Biological Consequences of the Blast-Body/Head Interactions

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Definition of Blast – Body/Head Interactions and Blast Injuries

Nowadays, the number of blast injuries is increasing both in military actions and in industrial accidents.

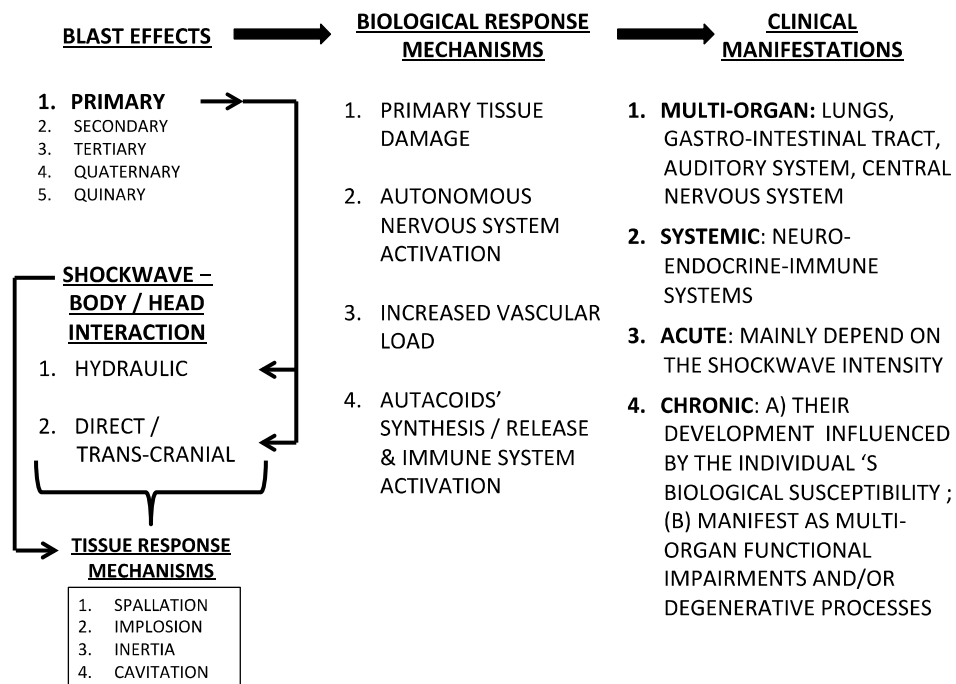
During detonation, which is the first phase of explosion, a rapid chemical reaction transforms a liquid or solid explosive material into gas giving rise to a large amount of energy. Part of this energy compresses the surrounding air generating blast, a sphere of highly compressed gas that rapidly expands and occupies a volume several times greater than that of the original explosive, the solid residues from the explosive, or its casing. It is noteworthy that blast injuries should be assessed from a historical perspective, bearing in mind the material characteristics of explosives used in a particular conflict.

The high explosive shock wave in air travels with supersonic speed. Such a speed is one of the characteristics of a real shock wave. They posited that when entering the body, the original shock wave changes through interactions with the heterogeneous tissue elements causing dispersion, divergence and attenuation. Subsequently, the velocity of the wave reduces so that the main part of the pulse travels with sonic or even subsonic speed. Since it doesn't retain the characteristics of a shock wave in the true sense of the word, the notation of "pressure wave" or "pressure pulse" would be more correct.

Based on his extensive experimental work [1-5], Clemedson postulated that the impulse of the pressure wave and/or the pressure variations along the wave's propagation throughout the body generate tissue-specific responses [5]. The historic explanations of the pressure wave-tissue interactions suggested spalling, inertia, implosion, and cavitation as the main mechanisms [6, 7]. **Spallation** develops at the interface between two media of different densities. As the pressure wave propagates across the denser medium toward a medium of lower density, it reflects from the boundary, creates a defect (i.e., crater) in that denser medium, and spall fractures and fragments from the boundary. **Inertial effects** also happen at the interface of the different densities. Namely, while tissue components with the lightest density travel the fastest, denser elements lag behind. The physical movements' differing velocities cause stretch and strain at the interfaces, and consequently, to displacement, deformation, or rupture of tissues and organs [3, 8, 9]. **Implosion** occurs when the pressure wave passes through a liquid medium containing dissolved gas. The kinetic energy of the pressure wave compresses the gas bubbles, so that the bubbles' pressure becomes higher than the wave's pressure. After the passage of the pressure wave, the bubbles re-expand and burst damaging the surrounding tissue; this mechanism is often called **cavitation** [10]. Besides the tissue damage caused by mechanism explained above, some findings indicate that the damaging effects of the primary blast might also depend on the frequency of the shock wave. For example, it was posited that high-frequency (0.5-1.5 kHz), low-amplitude stress waves target mostly organs consisting of structures with different densities. Examples of such organs include the lungs that contain air, blood, and parenchyma or the brain with multiple interfaces between fluid (blood or cerebrospinal fluid) and parenchyma. On the other hand, low-frequency (< 0.5 kHz), high-amplitude shear waves have been suggested to

induce tissue damage by generating local motions that overcome the tissue's natural elasticity (for example, at the gray-white brain matter interface) [11].

The effects of explosive blasts on the body are five-fold: (1) **primary blast effects** cause injuries (so called, primary blast injuries) solely through interactions between the blast wave and a living body, during which a portion of the shock wave is reflected, while another part of its energy is absorbed and propagates through the body as a tissue-transmitted shock wave; (2) **secondary blast effects** lead to secondary blast injuries, which can be blunt or penetrating, depending on the interactions between the fragments of debris propelled by the explosion and the body (i.e., whether the fragments damage the integrity of the skull or the skin barrier); (3) **tertiary blast effects** inflict tertiary blast injuries as a consequence of acceleration/deceleration of the body or part of the body; (4) **quaternary blast effects** include transient but intense heat of the explosion and cause quaternary blast injuries such as flash burns; and (5) **quinary blast effects** that include a broad variety of potentially injurious factors such as carbon-monoxide, the “post-detonation environmental contaminants” (bacteria and radiation from dirty bombs), and tissue reactions to fuel and metal residues, among others cause quinary blast injuries. Occasionally, especially in the case of moderate-to-severe blast injuries, the multiple blast effects interact with the body in parallel. Some literature sources call such complex injurious environment and related injuries as “blast plus”.



Biological Consequences of Blast Exposures

The complex injurious environment generated by blast initiates multi-phase biological response mechanisms in multiple organs. These include initial activation of the autonomous nervous system; changes in vascular tonus; increase in blood-brain barrier permeability; ultrastructural cellular damages; oxidative stress and potentially necrotic cell death. These processes are followed by delayed cell death, inflammation, and multiple molecular cascades (such as protein misfolding or hyperphosphorylated tau proteins, among others) leading to neurodegeneration.

The resulting changes could manifest with functional impairments (for example cognitive deficits, motor dysfunction or mood disorders) or irreversible morphological damage such as glial scarring or accumulation of neurofibrillary tangles [12].

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