Biodynamics of Blast Injury

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2.1 Introduction

Children are not spared from the horrific injuries inflicted by explosives. While exposure can occur in civilian settings such as fireworks or industrial accidents, the majority stem from conflict or terror-related attacks across the globe [1, 2]. Despite increasing recognition of the complex and unique injury patterns sustained following blast exposure in the adult population [3], the physical and psychological impact on the pediatric population is less well understood.

For the purposes of this review, we define children as any person under the age of 18 years (as specified by the United Nations Convention on the Rights of the Child). The heterogeneity of this group is acknowledged, and most studies will further subdivide this population. These classifications vary and are somewhat arbitrary but can be approximated by the following: infants (under 1 year old), young children (1–8 years old), older children (9–15 years old), and adolescents (16–18 years old).

It is essential to understand the epidemiology of blast injuries within this population to demonstrate the effect of explosive weapons on children and the ensuing burden on both domestic and global health systems. Furthermore, insight into the mechanism of childhood blast insults will further efforts to prevent, mitigate, and effectively treat these injuries [3, 4]. This chapter aims to provide an overview of the fundamentals of blast physics and injuries and review how the injury patterns and biomechanical features of explosive blasts may differ between pediatric and adult

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populations. This will aid in defining future research needs for protection, mitigation, acute medical treatment, and rehabilitation. This work is by definition interdisciplinary and as such, covers material that relates to both the biomedical sciences and engineering domains.

2.2 Explosive Blasts

An explosive is a material capable of producing an explosion using its own stored energy [5]. To understand the impact of blast injury on the human body, an examination of these materials' chemical and physical properties is necessary. The injury potential of explosive munitions is dictated by its properties and the physical changes the material and its surroundings undergo. Based on chemical composition and properties, explosives are classified as high or low [6].

Low explosives undergo deflagration, which is the subsonic combustion of the surface chemicals, comparable to a household fire [5]. This chemical phenomenon is seen in gunpowder propellants, smokeless powder, and fireworks. However, these materials may detonate when confined and used in conjunction with another more powerful explosive [5]. Conversely, high explosives detonate without confinement and are subject to a self-sustaining reaction [6] which is propagated by the high-pressure shockwave traveling through the material. These include PETN, HMX, RDX, and nitroglycerine. They have higher wounding potential due in part to the powerful shock waves that can fracture bone and cause soft tissue trauma [6]. Trauma from low explosives is more commonly confined to soft tissues as these do not produce shock waves [5].

When a high or low explosive detonates, it produces a rapid shift from potential to kinetic energy in roughly 1/1000th of a second. This results in the release of a significant amount of heat and gaseous products that are transmitted as a blast (shock) wave. The shock wave propagates out from the center of the blast by the expansion of gases generated by the reaction, with a pressure pulse a few millimeters thick traveling at supersonic speeds [7]. The spherical outer edge of this blast wave yields a disruptive increase in pressure, density, and temperature, known as a shock front, which exercises severe crushing, shattering, and shearing, as well as partially elucidated effects on cells and various tissues, such as the brain, heart muscle, and lungs [7]. As the superheated detonation products rapidly expand, surrounding cool air is compressed (the blast wave) in front of the expanding gas volume, which contains most of the explosive's energy. This leads to the formation of a subatmospheric pressure phase that sucks in the air behind the blast wave. The resulting turbulence aligns any debris into literal projectiles, whether particulate matter from the explosive itself or matter picked up from the environment as the wave progresses [5]. The air molecules are compressed to such a density that the pressure wave itself (a thin layer of compressed air) acts like a solid object propagating spherically in all directions from the explosion's epicenter. This particularly destructive layer of air is also known as the shock front and is capable of striking soft tissues with enormous force [7].

The variations in air pressure with time at a fixed point in space (the Friedlander relationship) describe the physical properties of a blast wave [5, 8]. The blast shock wave has three different physical properties that are responsible for the pathophysiological effects on biological tissue. These are the amplitude of the peak pressure, the impulse (defined as the time integral of pressure), and the duration of the positive phase overpressure. It has also been proposed that the dynamic overpressure of the detonation products (blast wind) and thermal energy released in the explosion contribute to blast injury.

The first event is the high positive pressure phase occurring immediately after the blast, which is the longest portion of a shock wave and causes a rise in ambient air pressure. It is followed by a negative (subatmospheric) pressure phase. This can be represented graphically as an idealized curve (Fig. 2.1) representing the Friedlander equation which is the variation in blast wave pressure with time, at a fixed point in space (the Friedlander relationship) [5, 8]. The curve shows a rapid increase in ambient air pressure following an explosive event over a short period of time. The negative pressure phase is also visualized by the curve reaching a minimum point that is below the ambient air temperature.

Accompanying the positive and negative pressure phases is the blast wind, a mass movement of air caused by the explosion that lags behind the blast wave (a combination of positive and negative phases of the explosion). The expansion of gaseous byproducts of the explosion accelerates molecules of air into a high-speed wind. This phenomenon is potentiated by a countermovement of ambient air filling the vacuum created by the negative phase. This secondary wave is capable of propelling objects and can be as damaging as the original explosion. Once the blast wind resolves, a return to ambient air pressure occurs. These three components (positive and negative pressure phases followed by the blast wind), determined by the physical properties of the explosive and the surrounding environment,

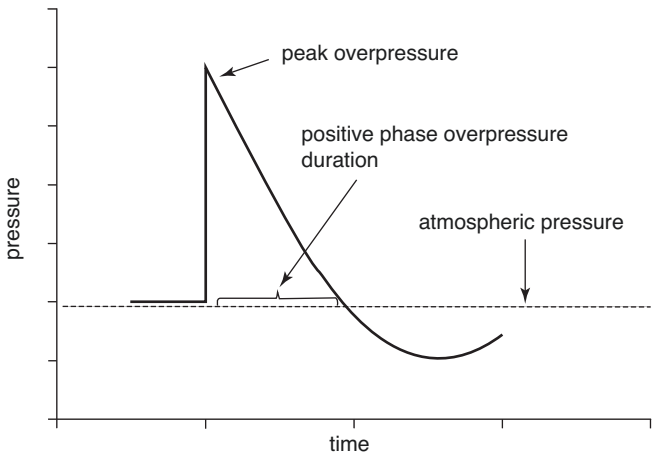

Fig. 2.1 Simplified Friedlander waveform showing pressure changes over time in a free field explosion [5, 8]

collectively form the basis of the injuring processes that act on the human body, which is further explained when considering the human body's response to these rapid changes.

The injuries caused by explosions, whether incurred from blast waves or blast winds, represent a variety of complex traumatic wounds. Various factors such as proximity to the blast determine the complexity and intensity of sustained injuries. The blast wave, for instance, dissipates energy inversely proportionate to the third power of the distance from the detonation point. In CLOSE proximity, injuries are caused directly by the displaced explosive material and its container, rather than the blast wave itself, which is a more common cause of injury further from the epicenter. Because the intensity of the blast (the peak overpressure) decreases rapidly with distance from the detonation point, victims must be in close proximity to sustain primary blast injuries [7]. Context is another important consideration when analyzing trauma from blasts. Many factors need to be considered for a full skeletal assessment such as explosive type, differential survival rates, improved protection (in combat situations), and confined vs. open surroundings.

Blast waves occurring in enclosed spaces behave differently from their open-space counterparts, as waves multiply when they bounce off solid objects such as walls. This produces an erratic network of interfering waves and can intensify the effects on biological systems, making injuries more severe. The medium where the blast wave propagates also alters its effect. Immersion blasts, or shock waves produced in water, create waves of higher intensity and longer duration than atmospheric blasts. Intraabdominal injuries are prominent in such cases: laparotomy findings include sub-serosal hemorrhage and tears in the bowel wall [9].

In summary, shock waves have a number of properties that help explain their effects on tissues [8]).

- They generate highly localized forces with small, but rapid distortions. Pathophysiological effects are at the microvascular level; gross lacerations are not typical.
- They affect organs with marked differences in physical properties (acoustic impedance) particularly hollow gas-containing organs.
- Stress concentration: when a stress wave encounters an interface between two media of different physical properties such as bowel wall tissues and gas-filled lumens, a component of the compressive stress wave is reflected back at the interface as a tension wave. Most materials are weaker under tension to compression, disruption at the interface (tissue damage) may result, and this phenomenon can affect organs far from the site of impact. Stress waves through the thoracic and abdominal walls are responsible for blast lung and primary blast injury of the small bowel, respectively.
- Pressure differentials across delicate structures such as alveolar septa or bowels: compression of a segment (implosion) and SDA subsequent expansion damages the wall of the structure.

2.3 Blast Injury Mechanisms

Blasts produce a unique spectrum of devastating injuries classically divided into primary, secondary, tertiary, and quaternary blast injuries [10]. While this classification is useful for its simplicity and mirrors the principal injury mechanisms, it is based on Second World War data of open field bare explosives [11], a model that does not account for modern variations, i.e., explosive types (Mine, IED, Suicide vest) or environmental factors (open air, confined, buried). Furthermore, multiple mechanisms often overlap, leading to complex injury types in the polytraumatized child [12]. Nevertheless, it constitutes a widely accepted theoretical model for understanding blast injuries [5, 13].

2.3.1 Primary Blast Injury

Primary blast damage is largely a function of the character, magnitude, and rate of pressure fluctuations, and the duration of the pressure pulse [14]. Primary blast injuries are caused by the interaction of the blast wave and its components with the body and are therefore a type of non-penetrating trauma that is estimated to constitute 86% of fatal blast injuries [6, 8].

The effect of blast waves is most profound at air and fluid-filled interfaces in the body which are most sensitive to rapid pressure fluctuations as the blast wave propagates [6]. The brain, ears, spinal cord, gastrointestinal tract, lungs, and cardiovascular system are especially susceptible to damage from the primary blast wave [15].

Damage to human tissues is incurred mainly by four mechanisms: spalling, implosion, acceleration/deceleration, and pressure differentials [15].

1. Spalling occurs when particles from a denser fluid are forcibly pushed through a less dense fluid at the interface of two different media.
2. Implosion implies a contraction of gas pockets that occurs as a blast wave propagates through tissue. The expansion follows rapidly, causing injury from multiple miniature internal disruptions.
3. Acceleration/deceleration injury occurs when the body or its internal organs are accelerated in one direction from contact with the blast wave front, followed by an abrupt change in momentum. This can occur when a reflected blast wave collides with the body from a different direction or when meeting a solid object or wall.
4. Pressure differentials between the outer surface of the body and the internal organs during a blast wave can cause internal injury.

Some studies have shown that survivors of explosive blasts were found to have elevations in plasma arachidonic acid metabolites, thromboxane A, prostacyclin, and sulfidopeptide leukotrienes, suggesting that blast waves can cause extensive, measurable pathophysiologic alterations. Traumatic limb amputation from primary blast waves occurs at a blast wave-induced fracture site rather than joints. Other

primary blast wave injuries include traumatic brain injury, tympanic membrane rupture, perforation of the globe of the eye, abdominal hemorrhage, and pulmonary barotraumas. Primary blast injuries in open air tend to be confined to victims in close proximity to the epicenter and are less common than ballistic (secondary) injuries [8]. A casualty close enough to an explosion to sustain serious primary injuries will commonly have lethal secondary and tertiary injuries.

A higher incidence of serious primary blast injury can be observed in the following circumstances:

- Enclosed spaces (vehicles or buildings) where the reflection of the blast wave augments the total blast load.
- Close proximity to the explosion.
- In individuals wearing body armor which confers protection against projectiles but not the blast wave.
- Large-scale explosions.
- Fuel-air explosives and other types of enhanced blast munitions.

2.3.2 Secondary Blast Injury

Secondary blast injury is the most common form encountered in blast-related injuries and is a far greater contributor to mortality in blast victims that survive the primary blast [6, 16]. The mechanism of secondary blast injury is tied to blast winds which lag behind the negative pressure wave that follows the primary blast wave. As the pressure drops below atmospheric levels, the vacuum generates winds that can propel objects in the vicinity of the explosion with considerable destructive force, on par with the blast wave of the initial explosion [15]. The musculoskeletal system is most commonly involved in the form of severe tissue injuries and amputations [15].

Fragmentation is the most common mechanism of secondary blast injuries, capable of damage over large distances. Projectiles, i.e., materials that have been added to the explosive device or built into it by design (fragmentation grenades), are propelled by the blast wave or blast winds [16]. They can travel with varying velocities (up to 2700 feet per second) and emulate ballistic patterns, causing penetrating or blunt ballistic trauma. In addition, blast fragments can carry environmental debris into the wound, seeding wounds with pathogens and impeding proper wound healing [15]. Injury patterns created by penetrating fragments reflect their shape and velocity and are categorized based on their resemblance to ballistic materials (i.e., ball bearings) or irregularly shaped projectiles [6]. The lethality of fragmentation injuries is dependent on the degree of vital structures' penetration. Fragmentation dispersion is chaotic and indiscriminate; the likelihood of penetrating vital organs is increased at shorter distances from the blast epicenter [17]. Small standing individuals such as children will have their vital regions (chest, head, and neck) closer to the epicenter of a buried explosive (mine, IED), increasing their fragmentation

exposure. This explains the increased incidence of thoracic, head, and neck injuries in pediatric victims compared to adults [18], and the increased requirement for operative procedures for these body zones [19]. The introduction of enhanced body armor to adult combatants provides partial protection from direct fragmentation strikes on the thorax, head, and neck [20, 21]. Unfortunately, children are unlikely to benefit from this form of protection.

2.3.3 Tertiary Blast Injury

Tertiary blast injuries are defined as the spectrum of injuries sustained through bodily displacement followed by rapid deceleration of the body or its parts, and impact upon the ground, walls, or objects. Additionally, the damage incurred from collapsing structures such as crush injuries is also considered part of this category. Bodily displacement is caused by an acceleration induced by blast winds or expanding gases in violent explosions. Fractures, crush injuries, traumatic limb amputations, severe soft tissue lacerations, and contusions can result [6, 8, 15]. Blunt head injuries and fractures are the most common types sustained, similar to non-combat or conflict-related trauma, although with greater severity [17]. In fact, head injuries are the second most significant cause of mortality following blasts in the pediatric population [12]. The relatively small body mass of pediatric patients increases their predisposition to bodily displacement and resultant blunt traumatic injuries [3]. In addition to differences in size and shape, pediatric tissues have different biophysical properties than their adult counterparts; the biomechanical response to blast loading and impact may differ. Pediatric bone has been shown to be less mineralized than adult bone. Reduced bone ash content leads to a lower modulus of elasticity and lower bending strength; a child's bone will bend more easily when subjected to the same force [24]. The tendency of pediatric bone to absorb energy, bend more, and deform plastically leads to the phenomenon of "greenstick" fractures. These are characterized by bending and unilateral fracture of the bone; this is in contrast to the adult bone which fails and fractures completely when subjected to a lesser degree of bending [25].

2.3.4 Quaternary Blast Injuries

Quaternary blast injuries encompass the wide spectrum of injuries not addressed by the previous three classifications [17]. These include psychological trauma, burns, asphyxia following inhalation of toxic fumes or burned materials, and mucosal edema (oral, nasal, pharyngeal) from high temperatures generated by secondary fires (flammable devices, structures, pavement, vehicles) [16]. Burns are the leading cause of death in children under 15 years old, and alongside head trauma, are the greatest predictor of death in all age groups [26, 27]. Psychological trauma is a complex and underreported issue that often follows a pediatric blast injury.

2.4 Blast Injury Characteristics in the Pediatric Population

While acknowledging that blasts produce a heterogeneous injury pattern within both adult and pediatric populations, epidemiological studies demonstrate certain injury patterns are more common in children. In pediatric populations, multiple body regions are involved in 65–70% of cases [12, 28], with burns and penetrating injuries to the extremities observed in 70–80% of blast victims [12, 18, 22, 27]. Penetrating injuries to the face, head, neck, upper limb, and trunk affects 80% of pediatric patients, significantly higher than the 31% of adult victims [22, 23] (Fig. 2.2). Children experience a high injury burden from blast trauma, as assessed by the injury severity score (ISS—a widely used consensus-based measure of injury severity) [29]. Around 20–36% experience "severe" injury (ISS > 15), while 8-18% are considered "critically injured" (ISS > 25) [10, 18, 19, 30]. Older children have a greater ISS and undergo a higher number of surgical interventions when compared to younger children (<9 years old). Of these, wound debridement and closure are most commonly performed [31], followed by vascular surgeries and exploratory laparotomies. These statistics reflect the prevalence of penetrating trauma as a major mechanism of injury [32].

Pediatric blast victims constitute a disproportionately large resource burden on treatment facilities [32–38], with approximately 56% requiring surgery [19], or twice the requirement of non-blast-related pediatric trauma [35]. Special structural

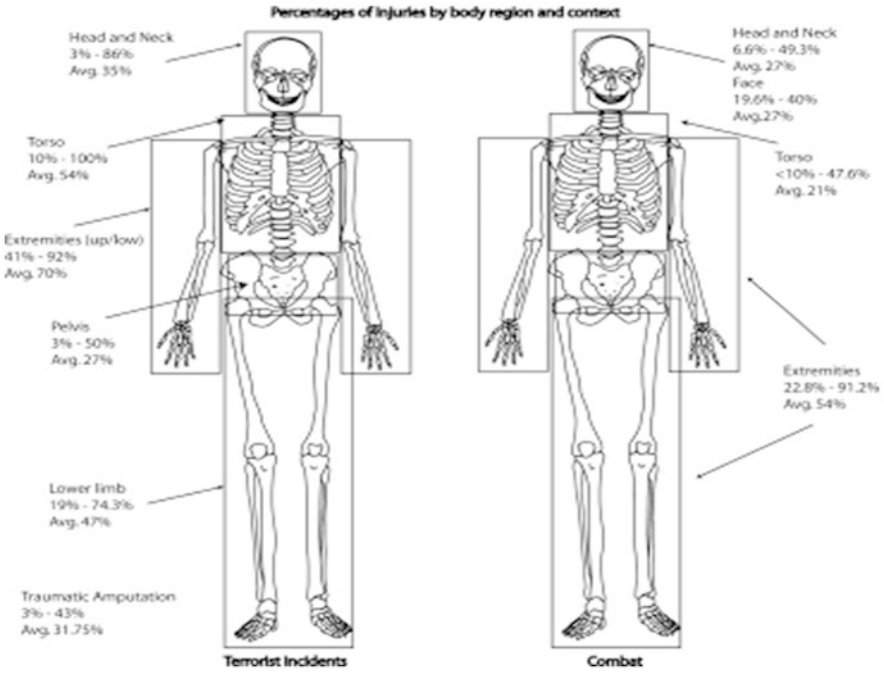

Fig. 2.2 Percentages of injuries by body region and context [22, 23]

Table 2.1 Structural consideration in the pediatric population [39]

Skin	Scalp	The younger a child is, the thinner and the poorer its ability to cushion against external forces
	Epidermis/ Dermis	Fragile and prone to blistering and tearing
	Subcutaneous fat layer	Easily retains water and microvascular breakdown causes a subcutaneous hematoma
	Galea aponeurotica	Blood and exudate can accumulate beneath galea
	Periosteum	Cephalic hematoma can be calcified rarely
Cranium	Cranium	The craniofacial ratio is at its greatest Cranial sutures are loose and highly mobile Calvarium is soft and rich in bone marrow, connected with a periosteum, and strongly attached to the bone cortex. The continuity of the skull tends to be well-maintained. Bone fragments are less likely to occur
Brain and nerve fibers	Nerve fibers	In undeveloped myelin sheaths the water content per unit volume of brain tissue is high. Fibers are pliable and less prone to rupture
	Brain/Cortical veins	Cerebral contusion by a direct external force is high because of its softness. Easily extended with accelerated decelerated motion and can cause subdural hematoma with disruption
Neck and cervical spine	Neck	Undeveloped neck muscle and poor head support
	Vertebrae	The fulcrum of the vertebral body is located in the upper cervical spine

features predispose the pediatric population to sustain multiple injuries, suffer higher ISS, and have a greater risk of death following blasts, as described in Table 2.1.

2.5 Injury-Specific Considerations in Pediatrics

2.5.1 Thermal Burns

Thermal burns are a common injury following blasts, with a prevalence ranging from 56 to 70% in pediatric patients of all ages [10, 40]. Thermal energy released from blasts can result directly from the explosion, through superheated gas products, or by secondary fires igniting surrounding vehicles and buildings [41]. Burn severity has been proposed as a prognostic marker for pediatric blast models [42], and severe burns exceeding 30% of the body surface area are the principal cause of death in children under 15 years old [10, 11, 33]. When a large detonation occurs (hundreds to thousands of pounds of explosive materials), the resulting blast wave is accompanied by a large fireball and surrounding flammable materials. The fireball and other secondary fires consume a significant amount of oxygen from the air, reducing its partial pressure to half the normal atmospheric value of 21% oxygen (10–13%). The fireball that emanates from an explosion can reach temperatures of several thousand degrees and lasts for approximately 500 milliseconds. The

probability of significant secondary fires, particularly burning vehicles, can further potentiate incendiary effects; the victims' open wounds can be complicated by burns and heavy smoke exposure. This compounding of factors complicates the debridement, surgical management, and healing process of sustained blast wounds. The burns typically encountered in explosion victims are flash and flame burns.

Flash burns are produced by the explosion's radiant heat and sustained by victims in close proximity to the detonation. Due to the short exposure time, they are more superficial than flame burns and are typically found on exposed body parts such as the face (most common), neck, hands, and calves of female victims, indicating the protective value of clothing in blast situations. Flash burns are more extensive and severe in victims of confined space blasts due to the containment of the fireball for a longer time period. Interesting characteristics observed in this type of injury are the charred singeing of scalp hair, eyelashes, and eyebrows. Eyelid burns are the most common injury and account for 96% of all flash burns. Lip burns by blast thermal effects on the mucosa present as singed lips, cyanosed, smoked black, covered by a dry crust of blood, and dry when the oral mucosa's minor salivary glands are affected [9, 16].

Burns are multisystemic insults affecting the dermis as well as the pulmonary, cardiovascular, inflammatory, and metabolic systems [2]. Insulin resistance, increased fracture risk, hepatomegaly, cardiac dysfunction, reduced immune function, and hypermetabolic changes, commonly seen in burn patients, have been demonstrated to persist for up to 3 years following burn exposure in adults [43]. Through systemic alternations such as wound healing impairment, nutritional deficits, and infection risk, burn victims carry an increased risk of hospitalization with higher morbidity and mortality, long after the initial insult [30, 43–45].

Children are predisposed to increased burn severity [46]. Anatomical disproportionality increases the lethality of certain burn patterns: a burn to the face and scalp of an adult comprises only 9% of the total body surface area (TBSA), not requiring IV fluid therapy. Conversely, the same injury in a pediatric patient covers 19% TBSA and requires fluid management [23]. Children under 2 years of age have reduced subcutaneous layers and skin thickness compared to older children and adults. Full-thickness burns, and the resulting rapid fluid, protein, and heat loss, can occur at relatively lower thermal energy levels. Consequently, assessment of clinical severity by burn depth has been shown to be less accurate in young children [29]. Subsequent dehydration, nutritional deficiencies, and hypothermia from full-thickness burns increase morbidity significantly in pediatric victims [46].

Thermal inhalation injuries in pediatric victims are difficult to assess, and symptoms of inhalational injuries such as increased respiratory rate may be incorrectly interpreted in the context of physiological age discrepancies. The pediatric subglottis represents the narrowest section of the upper airway and is quickly affected by burn-induced laryngeal edema, especially in the context of failed intubation attempts [46]. Rapid desaturation occurs following upper airway obstruction due to increased oxygen utilization combined with limited functional residual reserves [31].

Children rarely recover from severe burn injuries without functional sequelae, with limited joint mobility and impaired tactile sensation presenting significant

future challenges for rehabilitation [32, 33]. Prolonged rehabilitation and visible aesthetic disfigurement can produce psychosocial morbidity long after the event [34].

Nosocomial infection of the burn eschar, in particular, from *Pseudomonas aeruginosa*, can be prevented by an aggressive debridement and meticulous antimicrobial wound dressings [35]. However, sub-optimal care facilities, delays in wound cleaning, and prolonged transfer times all increase burn infection incidence [36].

In conclusion, pediatric patients should be considered high-risk and require sustained monitoring, optimal treatment, and rehabilitation. Long-term recovery with adequate functional outcomes is achieved by an early return to pre-burn activities and regular multidisciplinary follow-up [43, 45].

2.5.2 Extremity Injuries

Extremity injuries are one of the defining injury patterns observed in adult victims of explosive devices [37]. In the pediatric population, the prevalence of extremity injuries varies greatly from 11 to 85% [12, 38]. The literature establishes a clear age dependence, with only 11% of infants and 20% of children under 7 years old experiencing limb injuries, contrasted with over 50% of older children [38]. Younger children are at increased risk of upper limb injuries, while adolescents predominantly suffer lower limb injuries, in line with data from adult populations [19]. Upper limb traumatic amputations (TA) are commonly associated with the upper torso, neck, and head injuries, and are rarely seen in survivors [40]. Primary, secondary, and tertiary blast injuries result in a variety of extremity wounds, including vascular injuries, long bone fractures, and traumatic amputations.

The long bones of the extremities undergo successive developmental stages which make them more susceptible to trauma when compared to adult bones [41]. The impact tolerance of children's bones is dependent on bone girth and relative proportions of the marrow cavity and bony walls, as well as the relative proportions of organic and inorganic materials that constitute bone tissue (the flexibility or torsional strength of bone is determined by the organic component) [41]. During early bone development, organic components (collagen) outweigh inorganic (minerals) components [41]. The predominance of organic material is maintained through adolescence and is followed by a gradual buildup of inorganic bone materials in early adulthood, with maximal bone strength observed around age 20 years [41]. Children's bones have a lower modulus of elasticity, lower bending strength, and lower bone ash content. They also tend to deflect and absorb more energy, both before and after a fracture starts taking place [42]. That composition may explain the high prevalence of fractures in this demographic following explosive blasts.

Orthopedic trauma resulting from explosive detonations can occur in primary, secondary, tertiary, or quaternary (miscellaneous) blast injuries, in isolation or in combination. Traumatic amputations (TA), or the most severe form of orthopedic trauma, may occur through two mechanisms. The first is diaphyseal stress leading to fracture, flailing of the joint, and transosseous amputation. The second is intra/periarticular stress leading to articular failure, flailing of the joint, and joint

amputation [47]. In an explosion, changes in atmospheric pressure caused by the blast wave can fracture bones, followed by limb avulsion at the fracture site by secondary waves (blast winds), resulting in flailing of the extremity [48]. Some studies report that the most common site of amputation was not at the joint, but at different positions along the long bones based on the blast circumstances. The most frequently observed lower limb amputation site was the lower third of the femur and the upper third of the tibia at the level of the tibial tuberosity. In the upper limb, the distal arm was most commonly amputated. This pattern was explained by the shock wave causing a stretch or bend in the mid diaphysis of long bones as it passes through the victim's body and a complete fracture from the following blast wind [48].

Primary blast waves transfer energy at interfaces between tissues with differing acoustic impedance [5] leading to cellular disruption, soft tissue destruction, and bone micro fractures as the stress wave propagates, prior to any displacement [3]. This builds up shearing and axial stress forces that eventually exceed the tensile strength of bone causing fractures. It is suggested that these stress forces, occurring at the site of blast wave-induced bone fracture, are the probable mechanism of traumatic amputation [3]. For example, the most commonly seen proximal tibia fracture occurs when a blast wave penetrates a tibia from a lateral trajectory, and the bending forces exerted interact with the geometry of the tibia, resulting in peak stress applied to the proximal third [3, 5]. Once the bone is fractured by the blast wave, the detonation products exert on the bone additional bending stress [3], clinically manifesting as a traumatic amputation, with the proximal stump containing a short oblique or transverse fracture morphology [3] (Table 2.2).

Secondary blast injury is characterized by penetrating trauma from bomb casing fragments, materials implanted within the explosive (e.g., nails and screws), or from local materials displaced by the energy of the explosion [3, 5]. The projectiles rarely cause direct limb amputations [48], but they can cause fractures, directly or indirectly [5]. Aerodynamic drag on irregularly shaped fragments results in rapid deceleration outward from the point of detonation. Therefore, depending on the distance from the blast, fragments can strike the body with varying velocity, contrary to the streamlining seen in bullets fired through a rifled barrel [48]. In addition to their lack of streamlining, low-velocity fragments from explosive munitions

Table 2.2 Injury mechanisms from the explosion and its interaction with bone [49]

Explosion type	Pathophysiology	Fracture characteristics
Primary	Blast wave-mediated fracture	Traumatic amputation, short oblique/transverse fracture
Secondary	Direct impact of fragment	Highly comminuted multi-fragmentary fractures
Primary and secondary	Direct contact with the seat of the explosion, resulting in blast wave and fragment injury (e.g., antipersonnel landmine explosion)	Traumatic or subtotal amputation with significant soft tissue injury and fragments
Tertiary	Displacement of the causality or objects near the causality	Axial loading, 3-point bending, spinal fracture

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behave differently than low-velocity bullets. Upon striking tissue, even at low velocity, these fragments may exhibit a tumbling or so-called shimmy effect that can increase the amount of tissue damage [48]. Blast fragments often carry environmental debris into the wound and can inflict more severe tissue injuries than low-velocity bullets [48]. Furthermore, a large, slower projectile can crush a large amount of tissue, and missile fragmentation that may occur within the body can greatly increase temporary cavity effects [48]. A combination of the factors described above most likely accounts for the qualitative differences in tissue damage often seen with explosive fragments, compared to the damage caused by low-velocity gunshot wounds [48]. The direct impacts of high-energy fragments colliding with bone typically result in a highly comminuted fracture with extensive periosteal stripping [5]. Experimental evidence has shown that these injuries result in multiple bone fragments with no periosteal attachment and thus no blood supply [3]. In addition, these direct high-transfer wounds produce significant contamination of the fracture site and the medullary canal, thereby increasing the risk of developing long-term infective complications (osteomyelitis) [3]. As projectiles pass through tissue, they impart radial velocity to the surrounding medium, thereby creating large temporary cavities [3]. After penetrating the bone cortex, a projectile encounters the marrow-filled cancellous bone and propels the marrow radially at high velocity, fracturing the thin trabeculae. After penetrating the second bone cortex, the exit hole is enlarged by cavitation in the cancellous bone [3]. Due to the relatively inelastic nature of bone, the cavity formed in cancellous bone does not collapse and becomes a permanent cavity [3]. With higher velocity impacts (more than 500 m/s^2), the cavitation phenomenon produces widespread destruction of cancellous bone, with increased fragmentation of the cortical bone on the exit hole [3]. Conversely, at slower velocities, full penetration of the bone does not occur and only a single cortex is breached. In these cases, the classical "drill-hole" fracture is produced [3]. Indirect fractures can be caused by high-energy fragments passing in close proximity to bone. Such injuries are caused by the high pressures exerted on the bone surface by the leading edge of a rapidly expanding temporary cavity [3]. These fractures show no bone loss and the fragments retain periosteal attachments and are likely to remain viable [3]. The configuration in these injuries is usually simple (i.e., transverse or oblique) with little comminution, much like primary blast injuries [3, 5] (Table 2.2).

Secondary soft tissue blast effects are due to a propelled fragment colliding with the body that directly damages soft tissue in its path and, if sufficiently energized, generates a high-radial pressure compression wave in the tissues, just as an explosion does in the atmosphere [5]. The wave creates a temporary cavity of subatmospheric pressure as the fragment traverses, which pulls in external debris, increasing the risk of wound contamination [5]. The proportions of temporary and permanent cavities are determined by the kinetic energy of the causative fragment and the nature of the tissue affected. Areas of devitalized tissue can extend several centimeters and the zone of injury is often much greater than the remaining wound track [5]. The irregular morphology of shrapnel in comparison to a uniform bullet, increases the transfer of kinetic energy to surrounding tissues, thereby dealing greater damage

[5]. Consequently, simple surgical debridement of the wound track may not be sufficient to remove all nonviable tissue [5].

Tertiary blast injuries are caused by blast winds that can accelerate bodies as well as objects but do not reach as far as blast projectiles. The injuries sustained are varying in severity and are largely dependent on the distance from the explosion. Often, victims tumble along the ground, sustaining multiple injuries, or may be hurled through the air and struck by objects or impaled by them. Fractures, crush injuries, amputations, severe soft tissue lacerations, and contusions are all possible [48]. Displacement of the victims with force and sudden deceleration against a solid structure can result in significant tertiary orthopedic blast injuries [5] (Table 2.2).

When bone is subjected to external loads, the pressure distributes unevenly due in part to local osseous imperfections. This leads to nucleation, multiplication, and growth of micro-cracks in the weakest areas, and finally to the formation of macroscopic fissures (fracture) as a result of the coalescence of localized micro-cracks in the most densely damaged area [3]. The fracture pattern is a function of the direction and intensity of the load applied, the geometry of the bone involved, and its specific material properties [3]. When pressure is perpendicular to the axis of the bone, the most common fracture reported was a tension wedge, irrespective of the direction of the impact [3]. Tensile wedge fractures originate at a location directly opposite the point of impact, and the wedge segment radiates back through the bone initially forming a 90-degree vertex angle. This suggests failure from direct stress, i.e., axial loading of the bone in tension at the far cortex [3]. The level of comminution at the fracture site was related to higher impact speeds. Spiral fractures only occur when the bones were subjected to additional torsional loads. Severe axial loading of the lower limbs is common with underground explosions, or victims landing on their feet after being thrown by blasts, with comminuted calcaneal (heel) fractures being a prominent injury [3].

Quaternary (miscellaneous) orthopedic blast injuries are much less common than secondary blast injuries and may include burns from the thermal effects of explosions or from secondary fires [5, 48] (Table 2.2).

One of the important determinants of musculoskeletal injuries is the location of the victim relative to the explosion site. For instance, in close proximity to the detonation site, the effects of shock waves and detonation products occur almost instantaneously, leading to mixed primary and secondary blast injuries. This classically occurs with the detonation of anti-personnel mines, which are designed to release explosive energy at point-blank range, with the goal of maiming rather than killing [3]. The mine's blast wave is transmitted directly into the limb causing a brisance (shattering) effect on bone, within 200 milliseconds of the detonation. 1–2 milliseconds post-detonation, the bomb casing, environmental fragments, and other detonation products contact the limbs, causing destruction of traumatized soft tissue and applying maximal stress on bones previously damaged by the blast wave [3]. The end result is a total or sub-total limb amputation, with a zone of soft tissue injury (and significant amounts of foreign debris and fragments) extending more proximally to the damaged bone [3].

As with other types of blast injuries, orthopedic injuries are affected by environmental factors, especially in enclosed vs. open spaces. When a detonation occurs close to but outside of a structure, the resulting blast wave diffracts around and reflects off the obstacle. It also transmits to the interior of the structure with significantly reduced energy and pressure [49], effectively lowering the risk of blast wave-related injuries substantially [49].

In contrast, blast fragments are more likely to result in fractures of victims caught in the open [49]. Studies show that the lower limb is more frequently affected and sustains more tertiary blast injuries from enclosed space detonations [49]. These observations may be attributed to the momentum effects of the explosion that throw victims into the air for long distances before landing on their feet, or secondary to vertical acceleration and local floor pan deformation [49].

2.5.3 Torso Injuries

Chest and abdominal trauma are common following blasts, with an incidence varying between 32 and 50% and a peak in children aged 5–10 years [12, 38]. Primary, secondary, and tertiary blast injuries may impact the abdominal or thoracic structures and viscera. A comparison with unarmored adults [28] suggests that the torso is far more commonly injured in children following landmine and UXO (Unexploded Explosive Ordnance) explosions [28]. This may be due to anatomical susceptibility or unintentional high-risk behavior around explosives. Several structural considerations in pediatric populations lead to distinct patterns of chest injuries [50]. Pediatric thoracic blast injuries usually affect the internal organs due to the lesser protection provided by the developing rib cage [41]. In infants and young children, the thoracic walls are thinner and the ribs are more elastic than their adult counterparts. This greater flexibility is due to incomplete bony ossification, more flexible ligamentous attachments, and less developed supportive musculature, and makes it possible for anterior ribs to be compressed all the way to meet posterior ribs [51]. The anatomical features of the young thorax increase the likelihood of suffering severe parenchymal thoracic injuries such as heart contusion, dislocation, transection, or angulation of the great vessels, tracheal compression, and angulation, esophageal rupture [52], as well as pulmonary contusions with minimal or no signs on superficial examination or admission chest X-rays [41, 50, 51]. Over 80% of children who sustain a thoracic aortic tear will have significant associated injuries to the lung, heart, long bones, abdominal viscera, and central nervous system, although only 50% will present with external evidence of thoracic injury. Rib fractures are much more likely to occur from secondary or tertiary injuries [52]. Primary blast waves powerful enough to fracture flexible ribs usually result in fatal pulmonary trauma [52].

In addition to the structural and size differences, thoracic organs in children exhibit different physiologic characteristics [51]. In early life, the trachea is narrow, short, more compressible, and narrowest at the level of the cricoid cartilage. Therefore, small changes in airway diameter, seemingly inconsequential wounds in

the thoracic cage, or small foreign bodies may rapidly lead to respiratory distress [51]. Children also have a lower functional residual capacity coupled with higher oxygen consumption per unit of body mass and are therefore more susceptible to rapid deterioration from hypoxemia [51]. Moreover, they are at particularly high risk for airway obstruction [53] due to several oropharyngeal anatomic features: their tongues are relatively large for their oral cavity, as are the soft palate, oropharyngeal tissues, and the epiglottis, which are relatively longer and stiffer [53]. To complicate matters further, their large heads and occiput relative to body size allow their necks to flex easily when lying supine, exacerbating airway obstruction. These factors make children more susceptible to airway irritation and asphyxiation from the copious amounts of hot dust, sand, particles, debris, smoke, toxic fumes, and gases produced by explosions [7]. Lower respiratory tract and lung burns are rare but can be caused by superheated steam; signs include dyspnea, cough, and crackles from pulmonary edema [7].

Primary blast lung injury (PBLI) is the most common fatal injury following exposure to overpressure waves [2] and presents essentially as pulmonary contusions [52]. The degree of pulmonary pathology is proportional to the velocity of chest wall displacement [52]. A slow steadily applied force to the lungs allows compressed air to vent out through the trachea [52], while the abrupt chest wall compression induced by blast waves does not allow for this equilibration [52]. Pressures within the lung parenchyma and air spaces can match or greatly exceed the blast pressures because lung tissue compresses more slowly than the air in the respiratory tract [52]. Air-tissue interfaces of the pulmonary system are vulnerable to spalling, compression, and shear forces [17]. Barotrauma and volutrauma (overexpansion damage) lead to alveolar hemorrhage, pulmonary contusions, widespread edema, and pneumothoraxes [2, 21]. Depending on the blast load, this varies from scattered petechiae to large confluent hemorrhages involving the entire lung. Pulmonary contusions are more severe on the impact side of blast waves in the open air but tend to be bilateral and diffuse in victims of confined space blasts [52]. Pleural and subpleural petechiae are the mildest pathologies described. Ecchymoses, often in parallel bands corresponding to intercostal spaces, may be seen with larger blast loads [52]. Pneumothoraxes from blast injury are at increased risk of tensioning in infants due to the inherent mobility of the pediatric mediastinum, causing additional mortality [31].

2.5.4 Head and Spinal Injuries

The reported prevalence of pediatric head injuries following blasts varies between 15% and 60% [18, 38]. Patients under 7 years old are almost twice as likely to present with head injuries as older children (28% vs 15%) [18]. Blast-induced traumatic brain injury (bTBI) is more common in victims under 10 years of age compared to adolescents [38]. Head and cervical spine injury is the second most common cause of death in all age groups [18], with one retrospective study conducted on postmortem data reporting skull fractures in 90% of pediatric casualties [54].

Neurosurgical decompression is the most common surgical intervention for blast-related head injuries in children under 3 years old [55].

Child cranial and spinal anatomy undergoes many changes, from the closure of fontanels and cranial sutures to changes in the thickness and pliability of the cranium, anatomy of the vertebra, and the maturity of cervical ligaments and muscles [53]. These structural differences change the fundamental injury mechanism for an infant compared to that of an older child or adult [56]. The child's head and brain are also fundamentally different from adults physiologically and anatomically [53]. Children have larger heads relative to body size, increasing the likelihood of head injuries in pediatric victims [39, 53]. Furthermore, the head is relatively heavy compared to the rest of the body and is supported by a weaker cervical musculature [57], making the head more vulnerable to TBI and resulting in different dynamics of head acceleration in response to external forces [39]. The ratio of head-to-body size gradually declines with age [39].

As children grow and develop, facial development and expansion of the paranasal sinuses progressively provide protection from brain damage [39]: the sinuses play a role as air-filled shock absorbers, partially absorbing energy directed at the skull and brain [39]. At birth the facial portion of the head is smaller than the cranium, with a face-to-cranium ratio of 1:8 (vs. adult ratio of 1:2.5). This pattern is notable in children up to age 8 years [41]. Moreover, the newborn forehead is high and bulged relative to the facial profile, due to the large size of the frontal lobe of the brain [41]. Thus, in newborns and infants, the face is tucked below the larger brain case, and their protruding forehead increases the probability that a force directly impacts the frontal skull and underlying cerebral parenchyma [39]. Furthermore, they lack the protection of fully pneumatized sinus cavities.

The mechanical characteristics of infant and adult skulls are significantly different. Anatomical immaturity in skull composition may increase the risk of bTBI from primary blast waves. Infant and child skulls are considerably more pliable, due to the segmental development and arrangement of skull bones, in addition to the flexibility and thinness of individual bones, possibly leading to greater shear stress and subsequent injury to the underlying brain structures [58]. Reduced calvarium thickness is also likely to provide less protection from penetrating and blunt traumatic injury. Another material property of the calvarium is its elastic modulus: fiber orientations parallel to the long axis have significantly higher elastic moduli than those with fibers perpendicular to the long axis [59]. In adults, cranial bones and sutures have similar properties and adult calvarias deform very little prior to fracture [59]. In contrast, pediatric cranial bones are 35 times stiffer than their cranial sutures and are able to deform 30 times more than older children's cranial bones before failure, and 243 times more than adult bones [59]. The large strains in pediatric bones and sutures result in a skull case that can undergo dramatic shape changes before fracture, potentially causing devastating damage to the brain [59]. In addition, fontanelles are extremely vulnerable to trauma. The skull develops as a loosely joined system of bones formed in the soft tissue matrix surrounding the brain. Interosseous junctions are relatively broad and large, leaving certain areas of the brain covered by a thin fibrous sheath and somewhat exposed to the external

environment. They are most obvious in the frontal and posterior skull regions and make the head of the child less resistant to impact trauma [41].

Injury patterns are also determined by the mechanical properties of brain tissue, which is stiffer in children [53]. Infant cerebral white matter contains little myelin, and its distribution is very different compared with adults [39]. The neonatal brain is watery and has a low density, while the fully myelinated adult brain has a much higher density [39]. Different brain regions myelinate at different rates and the resulting density variations can be pronounced at different developmental stages [39]. The degree of myelination results in different absorptions of traumatic forces, with increased susceptibility to TBI in unmyelinated areas [39]. When intracranial volume increases rapidly, as, in blast trauma, the acute increase in ICP can be life threatening. It may be more dangerous in young children than in older children and adults because of the lower normal range of ICP in this age group [53]. Cerebral compliance observed in young children as a result of open fontanelles and unfused sutures can only provide protection to a certain extent [53]. It is also determined by cerebral blood flow (CBF) and volume, and the ratio of cerebrospinal fluid (CSF) volume to brain volume, all of which are age dependent [53]. The CSF-brain ratio reflects the balance between brain tissue and CSF in the ventricles and subarachnoid cisterns of the brain. Although this has not been formally quantified across the age range, radiologists and pediatric specialists are aware of the differences between very young children, older children, and adults with respect to the amount of intracranial CSF that is expected, to reflect the growth of the brain from the neonatal stage through childhood and the gradual atrophy with age [53]. Post-mortem pathoanatomical data provides the majority of evidence for the pathophysiological effects of blast, which include edema, contusions, vasospasm of the internal carotid and anterior cerebral arteries, diffuse axonal injuries, and hematomas [60–64]. Following the blast, cerebral concussion is common, with increasing evidence of association with post-traumatic stress disorder (PTSD) [65–68].

The mechanism of bTBI following primary blast injuries remains incompletely understood [69, 70]; most experimental data originate from laboratory or computer models [66, 69, 71–73]. The overall hypothesis is that brain injuries can occur following overpressure oscillations from the primary blast, pressure on the cranium by the secondary blast winds, and tertiary blast injuries in the form of blunt traumatic or coup-countercoup injuries [74].

Blunt Traumatic brain injury (TBI) in children is caused by one of two mechanisms [56]:

1. Impulsive loading, where the head moves as the result of motion imparted by some other part of the body (e.g., “whiplash”).
2. Impact loading, where the head either strikes a stationary object or is struck by a moving object.

These events are mechanically distinct and have very different clinical consequences. Both cannot occur simultaneously, although they may happen sequentially. Impulsive loading of an unsupported head will cause it to rotate around some point

in the cervical spine, from the occipital condyles to C7/T1. With such a rotation, the skull will receive the transmitted force faster than the brain, which lags behind because the brain and skull are not rigidly linked [56]. This differential displacement may result in tensile failure of the bridging veins, which can withstand a force up to 30% stronger than their average baseline stretch [56]. It has also been suggested that following brain trauma, pediatric patients are at greater risk of brain injury from enhanced excitotoxicity and impaired cerebral blood flow. Excitotoxic effects may lead to increased neuronal apoptosis [75]. Experimental data using pediatric neurons subjected to non-blast TBI (nbTBI) demonstrated that extrasynaptic N-methyl-D aspartate (NDMA) channels were excited, leading to increased calcium channel influx [76, 77]. Calcium influx is associated with enhancing intracellular cascades and promoting neuroapoptosis [78]. Furthermore, severe nbTBI in children has been associated with impaired cerebral autoregulation and subsequent poor outcomes [79, 80]. In a later study, those under 4 years old were found to be at risk of impaired autoregulation, regardless of nbTBI severity, suggesting an enhanced susceptibility in younger patients. This correlates with animal studies demonstrating prolonged reductions in cerebral blood flow in newborn pigs compared to juvenile pigs following diffuse nbTBI [81].

Significant cognitive, intellectual, and functional sequelae arising from pediatric nbTBI have been described [82–87] and there is a clear need for specific studies on the long-term prognosis of pediatric bTBI. Controversy exists as to whether mild nbTBI is analogous to moderate bTBI in adults [88], and the paucity of pediatric data makes this comparison difficult. Extrapolation of bTBI results from nbTBI data is limited by variable follow-up times, more segmented age groups in pediatric populations, developmental milestones which complicate assessment and differing TBI mechanisms. Early nbTBI data suggest pediatric patients benefit from increased neuroplasticity in the developing brain, allowing recovery of cognitive and intellectual function [82]. However, conflicting studies demonstrated reduced educational performance, increased impulsivity, hyperactivity, and learning disabilities after 2–5 years in children with brain injury [83–86]. A recent study by Shaklai et al. [87] assessed 77 children of ages 2–17 over 10 years following moderate to severe nbTBI and found that 69% were able to fully reintegrate back into regular education following extended rehabilitation. The remaining 31% required additional help (19%) or special education (12%). Previous studies report reintegration of 24–59% of cases [89, 90]. A Higher Glasgow Coma Scale at admission and shorter loss of consciousness correlate with a positive outcome, which is consistent with other reports [91–93].

Pediatric spine injuries affect a modest percentage of children following blast injury (1–3%) [12, 27], with its presentation being almost ubiquitously associated with concurrent head injuries [18]. No data exists for blast-specific pediatric spinal injuries; the literature does, however, describe patterns for non-blast-related spinal trauma: the cervical spine is affected in 60–80% of total pediatric spinal injuries [94], while only in 15–45% of adult spinal injuries [95–97]. This pattern may be explained by progressive changes at the level of the epiphyses which fuse progressively at different times [53]. The biomechanical maturation of the spine only begins

to resemble the adult spine after age 8–9 [53]. Prior to the age of 10 years, the relatively large head places the fulcrum of flexion and extension at the upper cervical region, potentially increasing injuries [53]. Cervical spinal fractures are rare while ligamental dislocations are much more common, due to underdeveloped neck musculature, lax interspinous ligaments, and incomplete vertebral ossification. The absence of fractures may partially explain the high rate of spinal cord injury without radiographic abnormalities (SCIWORA) in infants (17%) compared to adolescents (5%) [98]. Other factors also explain the weakness of pediatric cervical spines and their tendency to deform: increased water content of intervertebral disks, unfused epiphyses, shallow facet joints, anteriorly wedged vertebral bodies, and undeveloped uncinate processes [53]. All these contribute to a more malleable spine that puts neural structures at risk, even without bony injury evident on radiographs [53]. Neurological sequelae are largely dependent on the degree of spinal cord injury (SCI) sustained. A high degree of clinical suspicion is thus warranted for pediatric blast victims.

2.5.5 Facial Injuries

Primary blast waves inflict different types of injuries on the maxillofacial region that result from interactions of the blast shockwave with these tissues, resulting in barotrauma [16]. The stress on impacted areas may be concentrated at certain locations called stress points; when tension exceeds their tensile strength, collagen fibers will fracture, and tissues will tear [16]. Primary blast wave impact on the face may result in transverse mandibular fractures, eye rupture, orbital fractures, tympanic membrane rupture, fracture of paranasal sinus walls, facial soft tissue injuries, and scalping injuries. They are usually associated with injuries to the lung, brain, and hollow organs; it is relatively uncommon to find isolated facial injuries in survivors [16]. Exposed wound surfaces are then hit by thermal gases (fireballs) and suffer burns on top of trauma. Blast winds also carry sand and other particles into the damaged soft tissues and exposed fractured bones [16].

When the wave impacts bony processes such as the zygomatic process or mandibular body and symphysis, the energy released can crush soft tissues between the compressed air wave contacting the skin surface, and the internal bone surface, causing skin and subcutaneous contusions. These wounds are characterized by ragged, tattered, and ecchymosed edges [16]. Because facial skin has strong resistance to primary blast waves, most injuries seen on the cheeks, eyelids, and lips are due to the combination of primary and secondary biophysical effects [16]. The suspended hot particles in the blast wave or winds impact a maximally stretched skin at high velocity, resulting in traumatic and deep scratches [16]. These abrasions facilitate the tearing of tightly stretched collagen fibers, resulting in shredding, laceration, or multiple punctures of all layers of skin in the affected area [16]. Scalping blast injuries occur when the blast wave strikes the front of the victim's helmet or scalp and the resultant maximal stretching exceeds the elastic limits of the skin at weak points near the eyelids. This leads to the skin tearing along a line between the

eyebrows and eyelashes consisting of the thinnest skin in the region. This can be followed by a degloving of the full thickness of the scalp tissue with separation occurring at another weak attachment at the pericranium and calvarias [16]. The scalping in this case extends posterior to the coronal suture and usually indicates a very powerful blast.

2.5.6 Maxillary Sinus Fracture

Implosion of maxillary sinuses has been proposed as a mechanism of "crushed eggshell" fractures of the midface, but experimental evidence for primary blasts causing facial fractures directly is lacking. Whether caused by primary blasts or secondary blunt trauma, these types of injuries do occur in victims close to explosions [99]. Rapid external loading of pressure onto the sinus structures compresses the sinus walls and causes them to splinter [6]. Once the high pressure has abated, the air re-expands, effectively creating a miniature explosion within the sinuses [6]. This causes more damage to the delicate structures of the nasal area, and this type of injury occurs when shock waves hit the midface area directly [6]. Alternatively, when a lateral wave impacts the skeletal structures of the cranium, the lateral portion of the maxillary sinuses is less affected due to the thicker zygomatic buttresses deflecting the shock wave more effectively than the thinner maxillary bone with a perpendicular force directly to the front of the face [6].

2.5.7 Mandibular Fracture

The pathophysiology of shock wave impacts at the lateral surface of the body of the mandible is different from non-blast-related trauma and results in a new type of fracture seen only in mandibular blast injuries [100]. Most civilian mandibular fractures are vertical to the longitudinal axis of the mandible, as seen in the body, angle, symphysis, ramus, condyle, and coronoid processes. These fractures are produced by high tensile strain caused by a traumatic impact, which leads to vertically orientated deformation patterns at points of weakness, causing tensile failure [100]. In the case of a transverse impact from a blast, wave-particle displacement is perpendicular to the direction of propagation of the wave, and they oscillate up and down around their individual axis. When the wave encounters the transverse middle part of the body of the mandible, part of it is reflected at the rigid boundaries (upper and lower borders) and the other part is transmitted across a less rigid middle part. A structural difference exists between the different mandibular sections because of solid cortical bone and the alveolar region, which is reinforced by cylindrical sockets and the strength of dental roots [100]. This causes a shearing fragmentation of the mandible at the mylohyoid ridge, a weak area in the mandible and attachment point of the mylohyoid muscle, with separation of muscle and bone [6]. The cancellous and cortical bone at this weaker point split transversely due to the differing shock absorbing properties of the impacted bone structures, provided the blast wave

is powerful enough [6]. Because of the factors explained above, blast mandibular fractures are a unique type of transverse split fracture occurring at the angle of the mandible [100]. They can manifest as a single line or multiple, almost parallel shearing lines, in the same region where fragmentation occurs [100].

Teeth are designed to withstand vertical forces; the impact of the blast, however, strikes the lateral surfaces uniformly, and much of the energy is reflected because of the convexity of the buccal surface and the hardness of tooth enamel [100]. The root is protected by the cortical bone of the alveolar socket and by its cylindrical shape which can deflect some of the energy. A powerful enough blast wave can lead to flexural failure and shearing (direct or punching shear) at the cementoenamel junction, resulting in sharp transections at the gingival margins. This type of tooth fracture parallels the displacement of the transverse mandibular fracture segments [100]. The effects of the blast and the tooth's structural response depend mainly on the pressure loading rate, the incoming angle of the blast, and the condition of the bony structures [100].

2.5.8 Acoustic Injury

Hearing loss following blast exposure is the most prevalent primary blast injury [101, 102] persisting well after the initial insult [103]. Blast pressures exceeding 104 kPa, approximately 1/5th the pressure required for a lethal injury, damage the tympanic membrane (TM) at the air–tissue interface with a 50% chance of rupture, in addition to middle ear ossicular damage and subsequent conductive hearing loss [104, 105]. The pars tensa is the TM area most frequently injured. Although much less common, dislocation of the incudomalleal or incudostapedial joints can occur, with or without fractures of the individual ossicles. Orientation of the head relative to the blast wave could possibly alter the severity for smaller blast loads that cause isolated auditory injury. Disruption of the ossicular chain may also protect the inner ear from permanent damage by absorbing the bulk of the pressure wave. In most cases, inner ear injury is reversible or treatable; temporary hearing loss and tinnitus are quite common, the severity of which typically decreases at farther distances from the blast. However, severe cochlear damage may occur: sensorineural hearing loss is usually permanent in these cases and occurs following excessive pressure mechanotransduction to the sensitive cochlear hair cells, or through bTBI damaging the auditory cortex [105].

2.5.9 Eye Injury

Despite representing only 0.3% of the anterior body surface area, eyes are often injured following blasts, with as many as 60% of blast victims undergoing minor clearance operations [27, 54, 106]. Eye injuries, including globe perforation, are commonly caused by secondary projectiles after explosions of all sizes. Interestingly, only one case of ocular primary blast injury (causing hyphemia) has

been reported in the literature, likely because of the eye's nearly homogenous density. Damage to the eye can result from overpressure waves reflecting off the bony orbit, causing optic nerve and anterior/posterior segment disruption, from secondary injuries caused by bone fragmentation, tertiary facial trauma, and chemical or thermal burns [107, 108]. Mine blasts are thought to cause a high incidence of eye injuries due to high concentrations of explosive particles, especially affecting children whose eyes are closer to the detonation point [7, 106, 109]. As it was described for the torso and upper limb injuries, the curiosity of children and high-risk behavior in the vicinity of explosives may predispose them to facial and ocular injuries. Vision loss confers significant long-term morbidity in children. In infants, visual processing plasticity and binocular vision develop in the first year of life. Monocular visual impairment can lead to further morbidity through amblyopia and visual defects [110, 111]. Without adequate social support, these victims are likely to suffer from developmental and educational deficiencies. This happens because 75% of early learning occurs through vision, and visual impairment at this age translates into future social and economic challenges to both the individual and the society [110, 112].

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